IARS AUA SOCCA
ANNUAL MEETINGS • 2022

IARS & SOCCA

2022 Annual Meetings

Medically Challenging Cases

TABLE OF CONTENTS

Airway Management

Airway Management - 1 Induction of General Anesthesia in a Patient with Left Diaphragmatic Rupture and Herniation

Madeline Savage[1], R. Alok Gupta[1]

[1]Northwestern University Feinberg School of Medicine, Chicago, IL

Introduction: Diaphragmatic hernia is most often congenital or the result of traumatic injury, but cases caused by metastatic disease have been described (1). Diaphragmatic rupture and herniation present many challenges for the induction of anesthesia and subsequent ventilation given that only one lung may be effectively ventilated. The purpose of this medically challenging case presentation is to describe the anesthetic risks in a patient with lung collapse due to diaphragmatic hernia. This case reinforces the difficulty of induction, ventilation, and extubation of a patient with significantly reduced FRC and the need for careful pre-operative assessment to consider potential outcomes.

Methods: Case Report: A 56 year-old female with history of bilateral breast cancer suspicious for metastatic disease was admitted with shortness of breath and found to have left diaphragmatic rupture with herniation of visceral contents into the thorax, causing complete left lung collapse and associated rightward mediastinal shift. She had a history of chemotherapy, radiation, and bilateral lumpectomy. She was scheduled for port placement with interventional radiology for palliative hormonal therapy, as she was not a candidate for chemotherapy. The procedure was requested to be completed under general anesthesia because the patient could not tolerate lying at less than a 45 degree angle without dyspnea. She required 3L of oxygen delivered via nasal cannula while sitting up at rest. She was pre-oxygenated and induced while sitting up. An RSI was planned. After induction, the patient was placed supine. The intubation was performed immediately, but she desaturated quickly to 20-30% and became hypotensive requiring norepinephrine infusion after intubation. The patient was not able to be extubated following the procedure given her hemodynamic instability and ventilatory requirements.

Conclusion: This case highlights the importance of pre-oxygenation prior to induction. However, the patient had severely reduced FRC due to her lung collapse which significantly contributed to the desaturation on induction. In addition, the mediastinal shift and collapsed lung may have caused airway obstruction, leading to autoPEEP which contributed to her hypotension by decreasing venous return and cardiac output. An awake fiberoptic intubation in patients with diaphragmatic rupture could be considered to prevent similar situations in the future.

References: 1. Chaudhry MS. Intraabdominal Herniation Caused by Metastatic Disease to the Diaphragm. Am J Respir Crit Care Med. 2015 Nov 15;192(10):1247-8. doi: 10.1164/rccm.201506-1095IM. PMID: 26244824.

Airway Management - 2 The bronchoscopic findings in tension pneumothorax during surgery

Akiko Tomita[1]

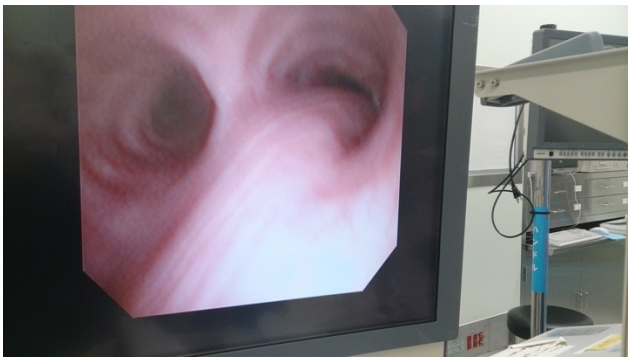
[1]Hokkaido University Hospital, Sapporo, Japan

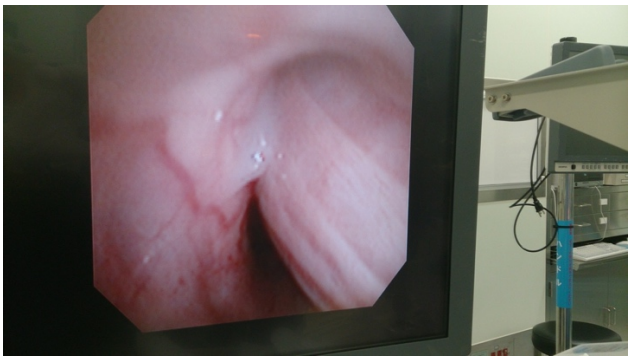
Introduction: Pneumothorax occurs rarely during surgery. However, pneumothorax occurring during surgery may lead to tension pneumothorax due to mechanical ventilation. In such cases, rapid diagnosis and treatment via insertion of a thoracic drain are imperative. The diagnosis of pneumothorax is usually made using chest radiographic findings. However, acquiring a chest radiograph during surgery hinders the progress of the surgery and the operative position might make rapid imaging difficult.

Methods: We present two cases of pneumothorax during surgery. 83 year old female. Laparoscopic colectomy was scheduled for colorectal cancer. An epidural catheter was inserted to Th10 / 11, and after general anesthesia was introduced, a central venous catheter was placed from the right internal jugular vein and surgery was started. After the start of Laparoscopy, the tidal volume decreased and the EtCO2 increased. Subsequently, the SpO2 decreased and EtCO2 and pulse increased. Auscultation revealed that the breath sounds in the right lung were diminished. Sputum accumulation was suspected and aspiration was performed, but there was no improvement, and bronchoscopy was performed. Catecholamines were used because blood pressure decreased. Postoperative Xp revealed right pneumothorax. 93 year old male. A trial laparotomy was scheduled for intra-abdominal bleeding due to traffic trauma. Preoperative CT showed mild right pneumothorax, and a right chest tube was inserted after induction of anesthesia. During the operation, a decrease in the tidal volume, an increase in the EtCO2, and a decrease in SpO2 were observed. Norepinephrine was used because of increased pulse rate and decreased blood pressure. Auscultation showed diminished right breath sounds, and air leaks from the drain were reduced. Considering tension pneumothorax due to drain obstruction, when the tube was optimized, an air leak appeared and the situation improved.

Conclusion: Bronchoscopy performed during surgery revealed a bulge in the membranous portion. This is thought to indicate positive pressure in the thoracic cavity. Diagnosis of pneumothorax also based on signs such as differences in breath sounds on the left side and right side, emphysema, and vital signs indicative of obstructive shock. Although a method for confirming the 'sea shore sign' with pulmonary echo has been proposed recently. Intraoperative bronchoscopy is often performed by anesthesiologists for diagnosis of respiratory failure (DOPE), and bronchoscopy is easy to prepare for operating room staff. In cases of tension pneumothorax, the image depicts inward compression of membranous portion. In this report, we present the bronchoscopic findings of two pneumothorax cases that occurred during surgery. Sharing these findings may help diagnose intraoperative pneumothorax.

References: Chan AS, Manninen PH. Bronchoscopic finding of a tension pneumothorax. Anesth Analg 1995;80:628-9. Sundeep M, William AS, et al. Contralateral Tension Pneumothorax During One-Lung Ventilation for Lobectomy: Diagnosis Aided by Fiberoptic Bronchoscopy Anesth Analg 2002;95:570-2 Dosios TJ. Bronchoscopic findings in traumatic rupture of the aorta. J Thorac Cardiovasc Surg 1988;96:342-3. Gorman RB, Merritt WT, Greesspun H, et al. Aneurysmal compression of the trachea and right mainstem bronchus complicating thoracoabdominal aneurysm repair. Anesthesiology 1993;79:1424-7. Prakash UBS, Abel MD, Hubmayr RD. Mediastinal mass and tracheal obstruction during general anesthesia. Mayo Clin Proc 1988;63:1004-11.





Airway Management - 3 A Simple Assembly of a Pediatric Facemask and a Self-Inflating Bag Provided Continuous Assisted Nasal Ventilation/Oxygenation in Obese Patients during Difficult Emergency VL Intubation in ER/Cath Lab

Thomas C Gliozzo[1], Dennis B Hall[2], Supreet Grewal[3], Samantha Malanowski[4], Alexander Kahan[5], James Tse[6]

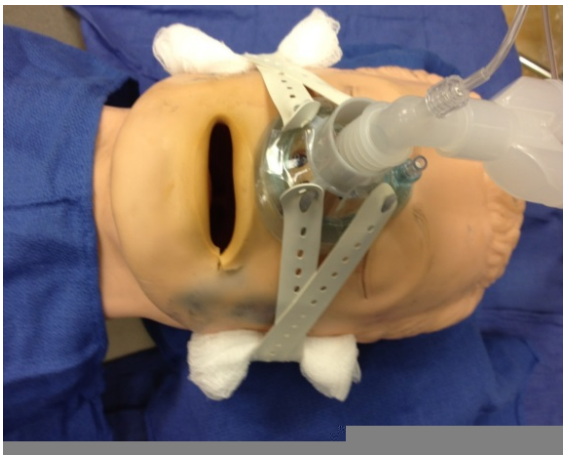
[1]Rutgers - Robert Wood Johnson Medical School, New Brunswick, NJ, [2]Robert Wood Johnson University Hospital, New Brunswick, NJ, [3]2St. John,Äôs Dignity Health, Oxnand, CA, [4]Mike O,ÄôCallaghan Military Medical Center, Las Vegas, NV, [5]Hackensack Meridian Jersey Shore University Medical Center,, Neptune, NJ, [6]Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ

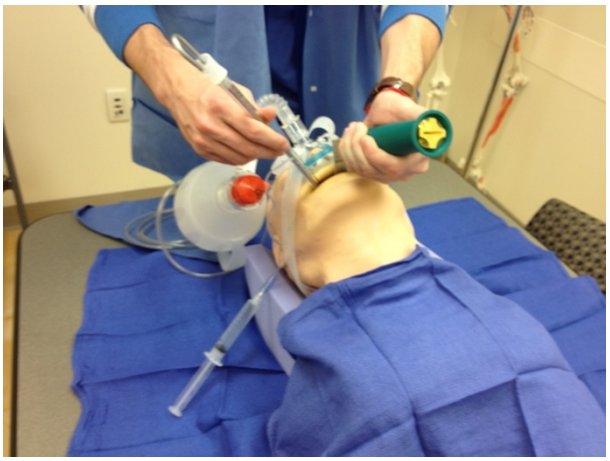
Introduction: Obese patients with respiratory distress often present difficult face-mask ventilation and challenging endotracheal intubation. Furthermore, removing face mask and O2 supply often causes severe O2 desaturation during emergency intubation. A pediatric facemask connected to an anesthesia circuit/machine has been shown to provide nasal CPAP ventilation and improve oxygenation in obese patients with OSA during procedural sedation (Fig. 1) or GA induction/intubation (Fig. 2).1-4 We combined the nasal mask and a self-inflating bag to improve oxygenation in obese patients during difficult emergency intubation at NORA locations.

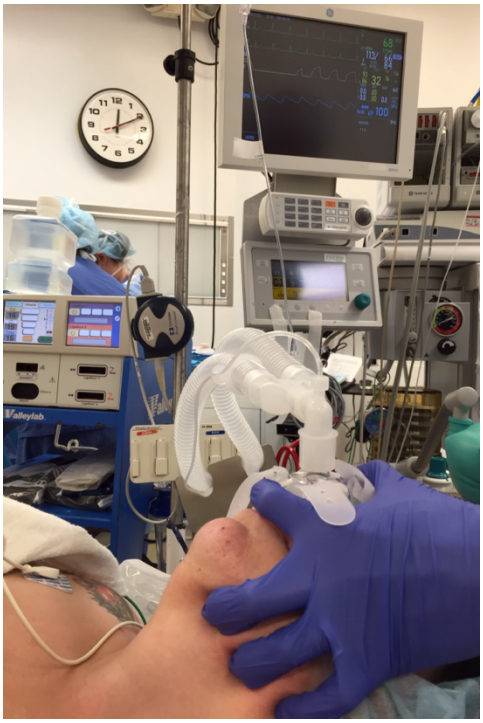
Methods: Case Report Case 1: An obese male presented to ER with respiratory failure. ER physicians administrated iv ketamine and failed to perform intubation despite multiple attempts using direct laryngoscope and video-laryngoscope (VL). 'Anesthesia STAT' was then called. Upon arrival of the Anesthesia Intubation Team, a facemask assisted ventilation was performed by a respiratory therapist and his SpO2 was in mid 80's%. A modified infant mask was secured over his nose and connected to a flexible connector and a self-inflating bag (Fig. 3). Assisted nasal mask ventilation with his mouth closed was started with a closed PEEP valve and 10 L O2/min. His SpO2 improved to 100%. Following titration of etomidate, VL revealed copious bloody oral secretions and redundant tissues. Oral suctioning was performed while assisted nasal ventilation/oxygenation continued. After several attempts and change of anesthesia provider, VL intubation was accomplished without desaturation (99-100% SpO2 throughout). Case 2: Following receiving midazolam and fentanyl during percutaneous transluminal coronary angioplasty, a morbidly obese patient desaturated and subsequently suffered cardiac arrest. 'Code Blue' was called. Upon arrival of the Anesthesia Intubation Team, his SpO2 was 50-60% with assisted ventilation using a facemask and a self-inflating bag during chest compression. An infant facemask connected with a flexible connector and the self-inflating bag was immediately secured over his nose. Manual nasal ventilation was started with a closed PEEP valve and 15 L O2/min. With his mouth closed and chest compressions, his SpO2 improved to 80-90's%. Manual nasal ventilation was continued during chest compressions and difficult oral VL intubation attempts. Following successful intubation, the patient regained spontaneous rhythm and was transported to the OR for emergency coronary bypass surgery.

Conclusion: This simple nasal mask-self-inflating bag assembly provided un-interrupted assisted nasal ventilation/oxygenation during difficult emergency VL intubation in morbidly obese patients in ER and Cath Lab. It maintained continuous active oxygenation during intubation attempts. It could also be used during direct laryngoscopy (Fig. 4) or procedural sedation at NORA locations without anesthesia circuit/machine (Fig. 5). This assembly can be easily prepared and may improve patient safety at a low cost.

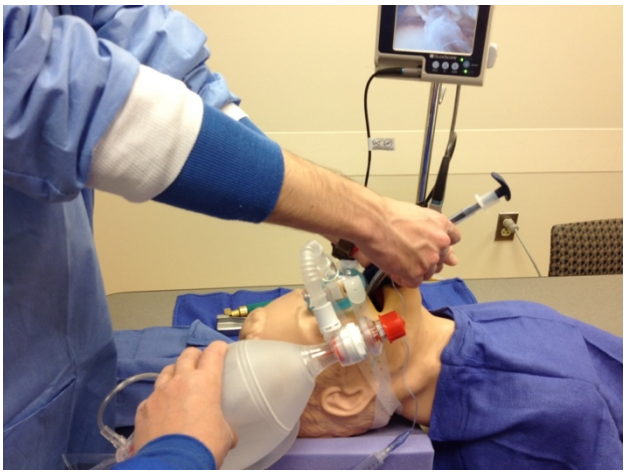
References: 1. www.TSEMask.com; 2. SAMBA 28th Annual Meeting MCC, 2013; 3. SAM Annual Meeting MCC, 2014; 4. ASA Annual Meeting MC1026, 2014








110
97



Airway Management - 4 Oral High Flow Oxygenation for Difficult Airway Fiberoptic Intubation: A Case Report

Thao Bui[1], Sharma Ila N[2], Andrzej P Kwater[3], Techecia Idowu[2], Acsa Zavala[4]

[1]University of Texas MD Anderson Cancer Center, HOUSTON, TX, [2]Universtiy of Texas at MD Anderson, Houston, TX, [3]UTHealth McGovern Medical School, HOUSTON, TX, [4]University of Texas MD Anderson Cancer Center, Houston, TX

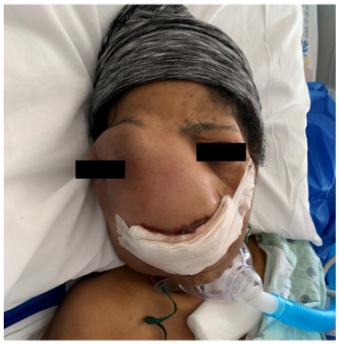
Introduction: High flow nasal oxygenation (HFNO) during fiberoptic intubation has been shown to limit hypoxia secondary to patient comorbidities, airway pathology, or effects of sedation. This technique was adapted for difficult airway management of a nasal tumor.

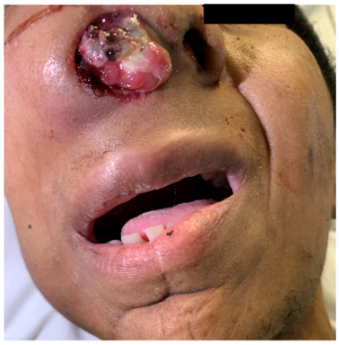
Methods: A 50 year-old woman with recurrent salivary myoepithelial carcinoma presented for a tracheostomy in the setting of seizures and diffuse nasal bleeding (Image 1 & 2). History included prior resections, partial pharyngectomy, tracheostomy, free flap, and radiation. CT and MRI demonstrated a large expansile sinonasal mass extending to the skull base (Figure 1 & 2). Initially, 4% nebulized lidocaine was administered during transport to the OR, where sedation was provided with remifentanil 0.05 mcg/kg/min, propofol 20 mcg/kg/hr, and midazolam 2 mg IV.

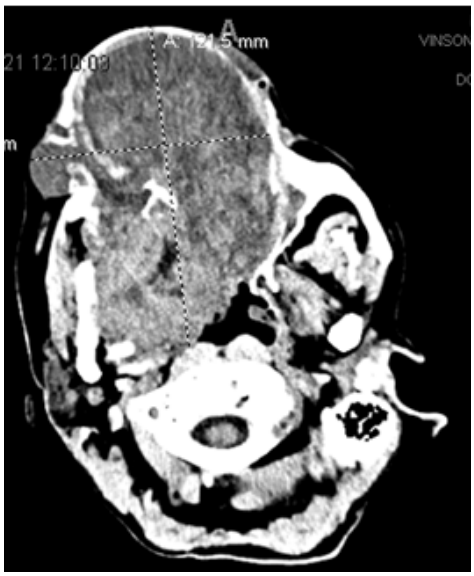
High flow oxygenation (Optiflow) was applied to the mouth at 30 L/min and the oropharynx was further topicalized using atomizer spray. The patient was able to protrude her tongue for passage of the fiberoptic scope into the retropharyngeal space. Upon visualization of the vocal cords, 4% lidocaine was injected via the fiberoptic port. After advancing the scope into the trachea, the patient was successfully intubated using a preloaded 6.0 reinforced ETT.

Conclusion: HFNO provides titratable oxygen therapy to improve oxygenation. Low level continuous positive airway pressure is generated to facilitate washout of pharyngeal dead space, reduce airway resistance, increase alveolar recruitment, and prevent atelectasis. This case highlighted similar benefits when this technique is adapted to oral administration during awake fiberoptic intubation.

References: 1.Badiger S, M. John, R.A. Fearnley and I. Ahmad Optimizing oxygenation and intubation conditions during awake fibre-optic intubation using a high-flownasal oxygen-delivery system. British Journal of Anaesthesia, 115 (4): 629-32 (2015). 2.Vionarica G and H Vaghadia. Pre-oxygenation with Optiflow THRIVE™ (transnasal humidified rapid insufflation ventilatory exchange) in a patient with impossible bag mask ventilation due to large facial tumor. Journal of Clinical Anesthesia Volume 64, September 2020, 109847.






A
A 121.5 mm
VINSON

Airway Management - 5 Occult Nasogastric Tube-Induced Laryngopharyngeal Edema Resulting in Unanticipated Difficult Airway

Elizabeth Rieth[1]

[1]*Memorial Sloan Kettering Cancer Center, New York, NY*

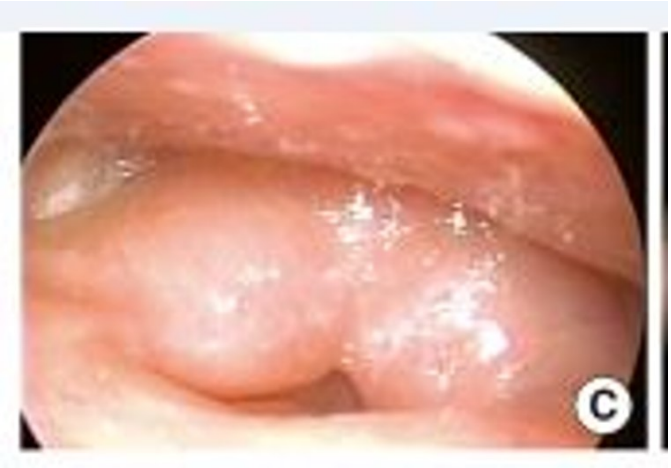
Introduction: Patients may present for anesthesia with indwelling nasogastric tubes (NGTs). Often these procedures are urgent or emergent in the setting gastrointestinal pathophysiology and risk for bowel ischemia if the case is delayed. Patients frequently complain of pain with indwelling nasogastric tubes, expressing the desire for their removal. Presented here is a gentleman who presented for elective incisional hernioplasty in the setting of small bowel obstruction. The case was complicated by unanticipated difficult airway manipulation due to severe laryngopharyngeal edema associated with an indwelling NGT.

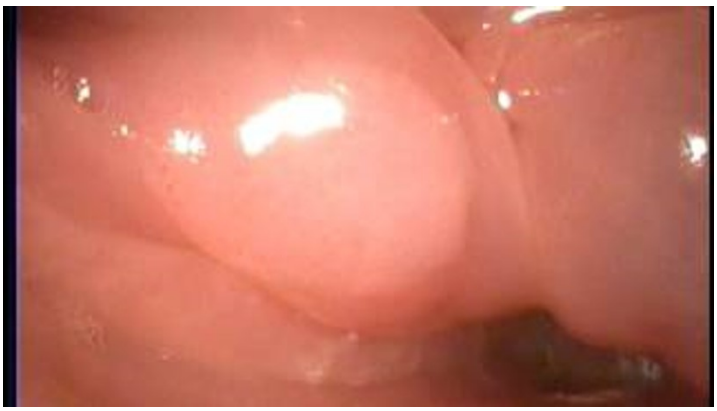
Methods: This 52-year old man presented for hernioplasty in the setting of small bowel obstruction. Medical history included BMI 40 and a history of easy direct laryngoscopy and tracheal intubation for colectomy 8 days prior. Preanesthesia evaluation revealed a left nasal 18 Fr nasogastric tube indwelling for 3 days to 60 cm, pharyngodynia, Mallampati Class 3, and an obese neck. On induction, UESCOPE® video laryngoscopy revealed an edematous oropharynx with no achievable view of the glottis. Direct laryngoscopy with a Mac 4 blade revealed the same poor view. Help was called, and 2-handed mask ventilation with an oral airway was not achievable. An i-gel® size 5 supraglottic airway was placed with success in achieving an airway. Tracheal intubation was achieved with a standard Sheridan® HVT 5.5 endotracheal tube (ETT) over an Aintree catheter (Cook Medical) placed via flexible fiberoptic exam (FFE), however the ETT was dislodged from the airway on removal of the i-gel® airway. The i-gel® airway was replaced, and a wire reinforced 5.5 longer-length 400 mm ETT (Fuji Systems Corporation) was placed via the i-gel® airway, with placement in the trachea confirmed via FFE. Given difficulty in achieving the airway, and the possibility of dislodging ETT if i-gel® airway removal was attempted, the i-gel® airway was left in place with the ETT for surgery, which proceeded uneventfully. The patient remained intubated and sedated in PACU for approximately 6 hrs with the head of the bed elevated and steroids administered intra- and post-operatively. Prior to extubation, an ETT cuff leak was noted, indicating adequate airway caliber for a trial of extubation. Extubation was successful, with the patient able to main his natural airway. "Nasogastric tube syndrome" is characterized by acute upper airway obstruction due to bilateral vocal cord paralysis, with local irritation, edema, and eventual ulceration of the tissues, leading to impaired vocal cord function. It is typically associated with respiratory distress. In our case, laryngoscopy revealed severe edema of the entire posterior oropharynx (similar to but more profound than the views seen in Fig. 1). An indwelling NGT is a risk factor for occult laryngopharyngeal edema. Odynophagia may predict possible laryngopharyngeal edema, however this symptom is often present with indwelling NGTs. Respiratory distress associated with an indwelling NGT should prompt preoperative diagnostic evaluation. Preoperative diagnostic evaluation can be performed by awake FFE by an otolaryngologist or anesthesiologist; if present, laryngopharyngeal edema should be treated with steroids and racemic epinephrine, and surgery delayed if feasible. Strategies for intubation of the difficult airway in the setting of occult laryngopharyngeal edema include consideration of awake fiberoptic intubation, availability of difficult airway equipment, including small caliber, LONG ETTs; as well as preparation for securing a surgical airway if indicated.

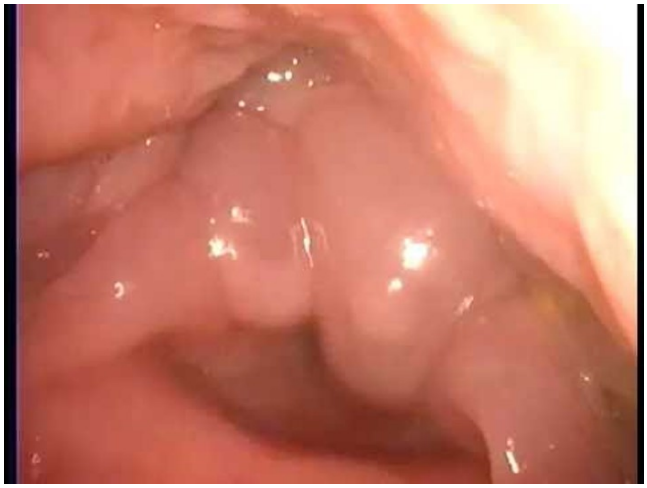
Conclusion: Significant nasogastric tube-associated airway trauma is typically accompanied by respiratory distress. However, this case reveals that significant laryngopharyngeal edema may be occult and may be associated with an unanticipated difficult airway.

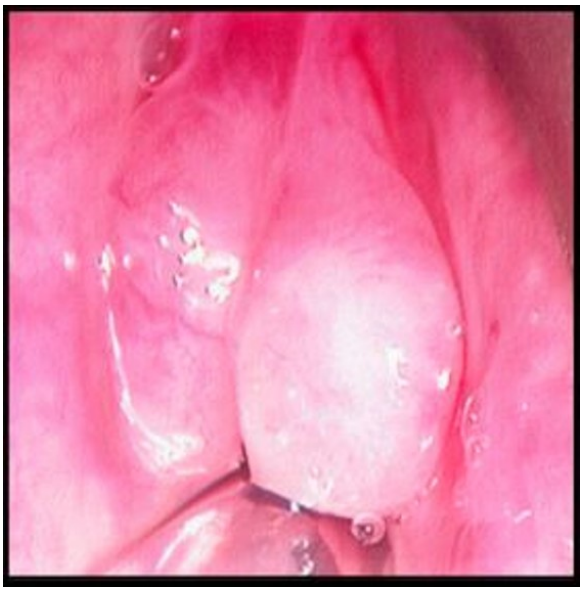
References: 1. Head Neck 2001; 23: 59-63 2. Anesthesiology 1976; 45: 555-7 3. Otolaryngol Head Neck Surg 2006; 135: 677-9 4. J Otolaryngol 2007; 36: E96-7 5. Int J Pediatr Otorhinolaryngol 2014; 78: 882-4 6. Intern Med 2005; 44: 1286-90 7. Ann Fr Anesth Reanim 2002; 21: 306-9 8. Age Ageing 2006;

35: 538-9 9. Anestesiol Reanim 2006; 53: 197-8 10. Med Princ Pract 2003; 12: 44-6 11. Anesth Analg 1993; 77: 1306 12. Masui 2010; 59: 495-7 13. Int J Pediatr Otorhinolaryngol 2014; 78: 1410-2 14. J Clin Gastroenterol 2004; 38: 913 15. World J Gastroenterol 2016; 22: 4057-61 16. Laryngoscope 1990; 100: 962-8 17. Acta Otorrinolaringol Esp 2010; 61: 85-6









Airway Management - 6 Failed Decannulation: Post-traumatic Partial Vocal Cord Fusion with Abnormal Fistula to Subglottic Space - A Medically Challenging Case Report

Anna J Miller[1], Amin Tafti[2], Praveen Prasanna[1], R. Wes Shepherd[1]

[1]Virginia Commonwealth University, Richmond, VA, [2]Virginia Commonwealth University School of Medicine, Richmond, United States of America

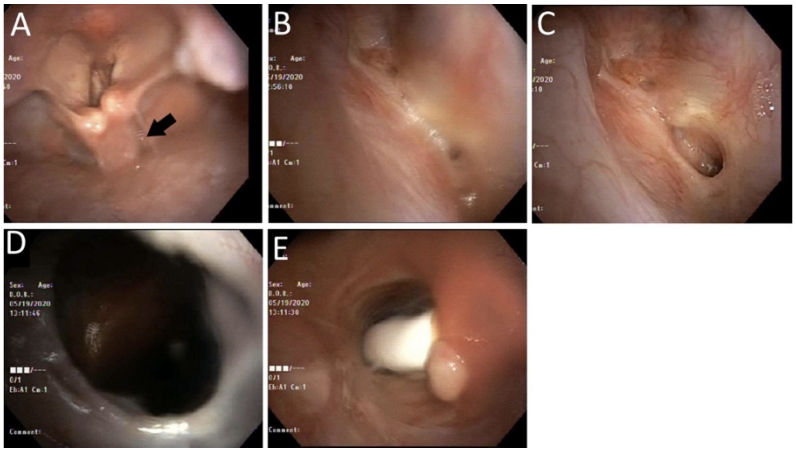
Introduction: Tracheostomy tubes are commonly placed after failing to wean from mechanical ventilation, vocal cord paralysis, laryngeal injury, severe neck and mouth injury, and concern for burns near the airway. Decannulation is the removal of a tracheostomy tube and failure of decannulation may occur due to abnormal upper airway anatomy. Bronchoscopy, particularly in patients where the indication for intubation/tracheostomy was airway obstruction, is done to determine patency of upper airway.

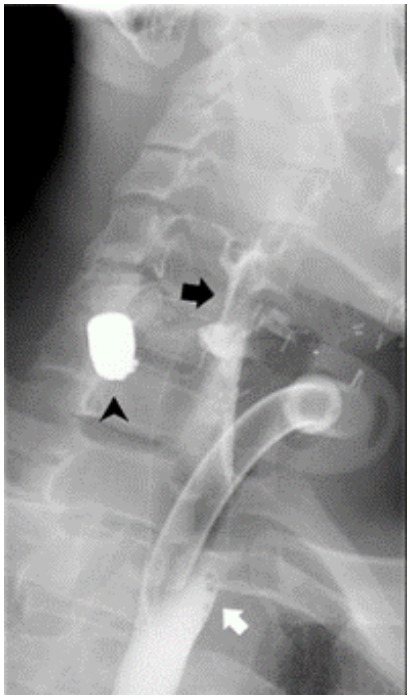
Methods: A 31-year-old chronic tracheostomy patient presented to pulmonology clinic for tracheostomy management 4 years after GSW to neck. He had been lost to follow up from his outside otolaryngologist. A bronchoscopy was performed as an outpatient procedure to evaluate for tracheostomy decannulation and demonstrated near-complete vocal cord fusion (Figure 1A, 1B, 1C). This had resulted in an orifice smaller than the diameter of a 5mm bronchoscope that existed posteriorly. Additionally, with head extension and direct laryngoscopy an abnormal tract was observed to the right and just inferior to the arytenoid tissue (Figure 1D). This aberrant, cartilaginous tract passed directly from the hypopharynx into the subglottic space where the trachea was observed to be without stenosis and with an appropriately placed tracheostomy tube (Figure 1D and 1E). A subsequent esophagram (Figure 2) confirmed a fistula arising from the upper larynx to the subglottic space with evidence of aspiration. Surgical notes from at the time of injury described a bullet entry wound on the left neck originating at the inferior border of the thyroid alar cartilage, obliterating the left vocal fold, and traversing to the posterior right hypopharynx, further disrupting the right vocal fold. Figure Legend: Figure 1. Bronchoscopy images: A. Laryngoscopic view with arrow locating fistula B. Image of vocal cords C. Image of vocal cords - anterior fusion with small posterior orifice D. Cartilage fistula E. Inspection of supraglottic space with bronchoscope inserted from the abnormal thyroid fistula Figure 2. Esophogram demonstrating abnormal fistulous tract is seen at the level of the upper larynx with contrast outlining the tract and coursing caudally (black arrow), retained bullet fragment (black arrowhead)

Conclusion: On evaluation with direct laryngoscopy and bronchoscopy, the patient had two pathways into the subglottic trachea. The midline structure appeared to be his anatomically true, albeit fused, vocal cords. He had developed a cartilaginous tract inferior and lateral to the true anatomical airway that led to the trachea. It was later confirmed to be his source of chronic aspiration on esophagram. This unique anatomic variation is likely a natural result of his original glottic trauma. Though this patient was able to breathe through his mouth, this anatomic variation precluded decannulation given his significant stenosis. This case reemphasized the importance of ensuring the presence of normal anatomy above the site of the tracheostomy prior to decannulation, especially important in patients at risk for abnormal airway anatomy such as those with history of trauma, severe burn, surgical manipulation of this anatomic region. Bronchoscopy involves direct visualization of the upper airway including the larynx and trachea, such that abnormalities including complications from previous trans-laryngeal endotracheal tubes, tracheostomy, or other neck injuries can be diagnosed.

References: Raimonde AJ, Westhoven N, Winters R. Tracheostomy. [Updated 2020 Nov 30]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan. Christopher KL.Tracheostomy decannulation. Respir Care. 2005 Apr;50(4):538-41. Li Y, Garrett G, Zealear D. Current Treatment Options for Bilateral Vocal Fold Paralysis: A State-of-the-Art Review. Clin Exp Otorhinolaryngol. 2017;10(3):203-212 Sabri A, Dabbous H, Dowli A, Barazi R. The airway in inhalational injury: diagnosis and management. Ann Burns Fire Disasters.

2017;30(1):24-29. Ceriana, P., Carlucci, A., Navalesi, P. et al. Weaning from tracheotomy in long-term mechanically ventilated patients: feasibility of a decisional flowchart and clinical outcome. Intensive Care Med 29, 845-848 (2003). Heffner JE, Hess D (2001) Tracheostomy management in the critically ill patient. Clin Chest Med 22:55-69 Lee TS, Wu Y. Bedside fiberoptic bronchoscopy for tracheostomy decannulation. Respir Med. 1995 Sep;89(8):571-5.





Airway Management - 7 Emergency Airway Management in a Patient with Trismus due to Spastic Hypertonia: a medically challenging case.

Charles D Arno[1], Laura Webb[1], Timothy Wills[1]

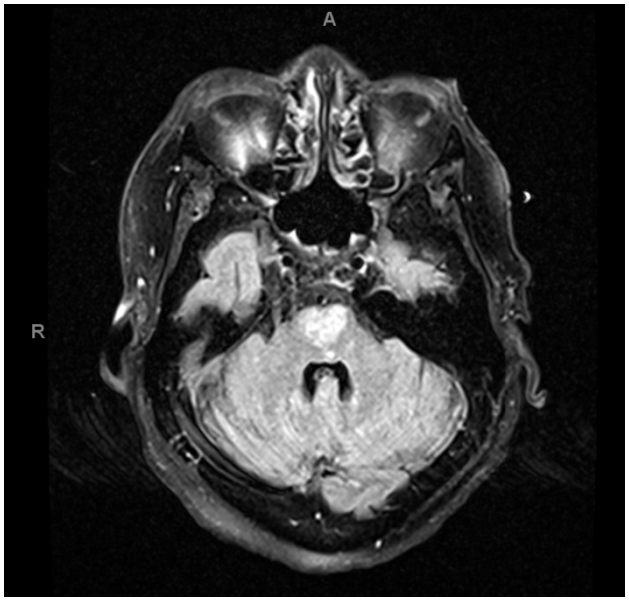
[1]VCU Health System, Richmond, VA

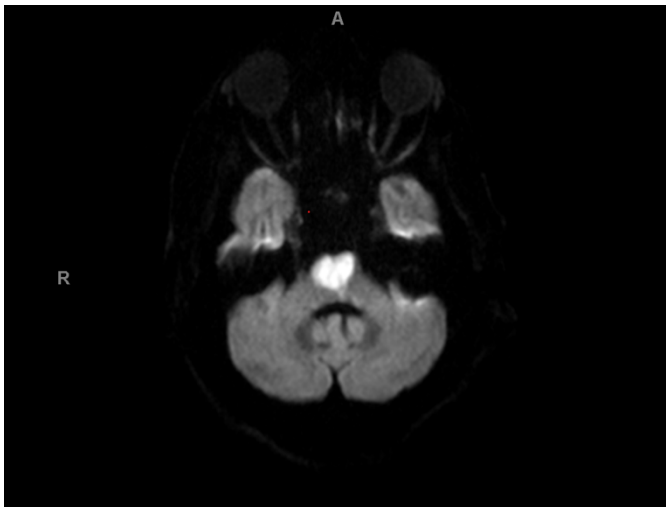
Introduction: Trismus is broadly defined as a restricted opening of the oral cavity and has a wide array of etiologies that anesthesiologists may encounter. The diagnostic criterion for trismus is that the maximum incisal opening (MIO) must be less than 40mm with assistance. In patients with trismus who are unable to adequately protect their airway, this is of particular concern as direct laryngoscopy requires an MIO of greater than 40mm (1). In this report, we will discuss the airway management of a patient who presented with initial symptoms of altered mental status who subsequently developed additional neurological symptoms and trismus in the emergency department.

Methods: The patient is a 70-year-old female with past medical history of diabetes, hypertension, breast cancer, and previous TIA, who presented to the emergency department with symptoms of altered mental status and bilateral lower extremity weakness. She was evaluated by neurology and underwent a complete workup for CVA, however initial CT scans obtained in the emergency department did not demonstrate signs of acute stroke. Several hours after initial presentation, the patient began to demonstrate increased gurgling and on exam was unable to open her mouth as well as worsening of her mental status. She was examined by physicians from the hospital medicine team and neurology and was found to have decreased oral opening despite assistance with a tongue depressor. Initial differential diagnosis included worsening of possible CVA, seizure, and infectious etiologies including oral infection, rabies, and tetanus. Given the patient's mental status change and concern that she would be incapable of protecting her airway, anesthesia was consulted to evaluate for airway management. On exam the patient had minimal mouth opening with an MIO of 1mm and was noticeably gurgling and drooling. Due to concern that the patient would potentially aspirate secretions as well as the potential for rapid worsening of her symptoms, the decision was made to pursue intubation in the operating room. The safest route for intubation was determined to be awake nasal fiberoptic intubation to minimize the chance of airway loss. Once transported to the operating room, the patient was moved to the operating table and connected to standard ASA monitors. She received a nebulized treatment of 4% lidocaine and anesthesia was induced with 1mg of midazolam and 50mg of propofol. Following induction, some loosening of the jaw muscles was noted, however it was not sufficient to allow oral intubation. Using a disposable bronchoscope, the patient's nasopharynx was entered and the vocal cords were identified, after which the bronchoscope was passed into the trachea. The patient was then intubated with a 7.0 nasal RAE endotracheal tube. After securement of the ETT, the patient was started on a propofol infusion for sedation and successfully transported to the intensive care unit.

Conclusion: There are very few case reports of trismus after neurologic injury. The proposed pathophysiology is from spastic hypertonia of the masticatory muscles due to an upper motor neuron disinhibition (2). In our case; disinhibition of the motor trigeminal area of the pons. Papers published in the 1970s by Jelasic and Frietag and a study in 1985 by Schwerdtfeger and Jelasic described a syndrome of trismus from the paradoxical activation of jaw closing muscles during attempted opening of the mouth. This syndrome is clinically important for anesthesiologists to be aware of as the paradoxical nature of the muscle contraction means that mechanical stretching will not be effective in increasing the interincisor distance of affected patients (3, 4). The decision to intubate a patient who is being treated for an ischemic stroke depends on several factors that must be considered by providers. While stroke and trismus in and of themselves are not indications for intubation, the decision to pursue intubation in this patient centered around her inability to effectively clear secretions as well as concerns that the underlying diagnosis was not a stroke but potentially status epilepticus or an infectious etiology such as tetanus. It was not until after the patient was able to have a secure airway and receive sedating medications that she was able to undergo an MRI which revealed the underlying ischemic stroke.

References: Bouloux G.F. (eds) Office Based Anesthesia Complications. Springer, Cham. Ann Rehabil Med. 36 (2012), 154-158. J. Neurol. Neurosurg. Psych. 41 (1978), 798-804. Acta Neurochirurgica 76 (1985), 62-66.





Airway Management - 8 Perioperative Management Considerations of Laryngotracheal Trauma: A Case Highlighting Laryngotracheal Complications Following a Gunshot Injury

Trishul Kapoor[1], Samuel A Schechtman[2], Andrew P Stein[3], Robbi Kupfer[1]

[1]University of Michigan Medicine, Ann Arbor, MI, [2]University of Michigan, Ann Arbor, MI, [3]Northwestern University, Evanston, IL

Introduction: Airway complications are a major cause of early death in patients presenting with traumatic injuries. [1-6] Current literature estimates the mortality rate to be between 16-36% in the setting of blunt or penetrating airway trauma leading to life-threatening airway obstruction. [1,2,4] This is also due to the impact of blunt or penetrating neck trauma on other vital structures of the respiratory, vascular, digestive, endocrine, and neurologic systems. [1,4] The upper airway consists of the nasal cavity, pharynx, and larynx. The larynx is a complex structure that consists of nine cartilages as well as five intrinsic and numerous extrinsic muscles. There are multiple associated ligaments and membranes that divide the larynx into important spaces, which could be impacted during a traumatic injury. The lower airway consists of the trachea, primary bronchi, and segmental bronchi. Any combination of structures can be injured within the laryngotracheal complex, but the most immediate mortality results from complete laryngotracheal separation. [5,6] In this medically challenging case of airway complications following a gunshot injury to the neck, we present several educational points from the perioperative management of complex laryngotracheal trauma.

Methods: A 48-year-old male presented to an emergency department after a close-range gunshot to the face and neck. Initial evaluation demonstrated a large, open wound of the oral cavity and tongue, comminuted fracture of the right body of the mandible, and a complex laryngeal fracture involving the thyroid cartilage and cricothyroid membrane with retained bullet fragments. On imaging, he was noted to have additional retained bullet fragments in the left mainstem bronchus, esophagus, and stomach without the presence of free or subcutaneous air. Initial attempts at oral endotracheal intubation were unsuccessful due to supraglottic airway edema and profound bleeding. The patient then developed severe hypoxia leading to hypoxic cardiac arrest. Cardiopulmonary resuscitation was performed with return of spontaneous circulation after an emergent tracheostomy was performed, and a 6.5mm endotracheal tube was placed through the tracheotomy stoma. The patient subsequently proceeded to the operating room for direct laryngoscopy, neck exploration, repair of laryngeal fractures, and revision tracheostomy with stent placement. The patient was transferred to our tertiary academic medical center for further care. Upon arrival, the patient's neck was erythematous, tender to palpation, and patient developed fevers. A computed tomography scan demonstrated concern for abscess formation within the anterior neck as well as extra-luminal placement of the laryngeal stent. After discussion regarding the patient's case between the intensive care team, anesthesia, and otolaryngology, the patient returned to the operating room. He underwent direct laryngoscopy, removal and replacement of laryngeal stent, neck re-exploration including incision and drainage of abscess as well as revision open reduction and internal fixation of complex laryngeal framework fractures.

Conclusion: Laryngotracheal trauma is uncommon, but it is vital to have a risk-stratified approach to manage these patients due to the high risk of mortality. If a laryngotracheal injury is missed after initial presentation, patients can suffer long-term complications including airway stenosis, chronic aspiration, and even death. [5] In recent literature, airway management recommendations are largely dictated by four stages of laryngeal injury. [5,6] In the past, orotracheal intubation was primarily recommended for airway control. [5] However, it is now recommended to avoid orotracheal intubation in the setting of known laryngeal trauma due to risk of further injury and possible cricotracheal separation. [5,6] The current recommendation for airway management, in those classified as grade 2 and beyond, is an awake tracheostomy. Diagnostic assessment including computed tomography scans and flexible nasoaryngoscopy can help guide the classification and

management. [5] Through discussion of this medically challenging case, we aim to provide an understanding of the pathophysiology of laryngotracheal trauma, surgical considerations, airway management approaches, and the important aspect of team communication in the management of laryngotracheal trauma.

References: 1. Anesthesiology, 124, 199-206, 2016 2. Injury, 38, 27-33, 2007 3. J Thorac Cadiovasc Surg, 130, 426-432, 2005. 4. StatPearls Publishing, updated 24 June 2021, accessed 9 August 2021. 5. Anesthesis and Analgesia, 129, e52-54, 2019. 6. Oral and Maxillofacial Surgery Clinics of North America, 20, 415-430, 2008.

Airway Management - 9 Successful airway management of unexpected tracheal mass during stenting for superior vena cava syndrome - A Case Report

Elise H Sullivan[1], Hart Donahue[2], Acsa Zavala[3], Alisha Sansguiri[4], Kevin Huynh[5], Olakunle Idowu[6], Jens Tan[7], Nina Castro-Koshy[2], Roberto Casal[2], Joshua Kuban[1]

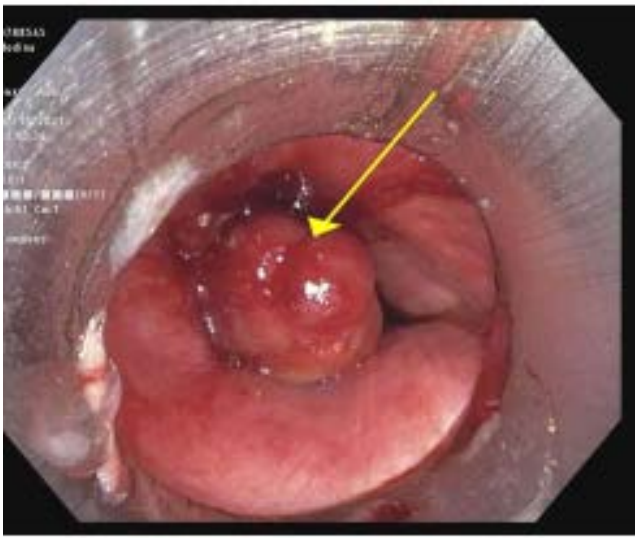
[1]The University of Texas MD Anderson Cancer Center, Houston, TX, [2]The University of Texas MD Anderson Cancer Center, Houston, United States of America, [3]University of Texas MD Anderson Cancer Center, Houston, TX, [4]The University of Texas Health Science Center at Houston, Houston, TX, [5]Sam Houston State University College of Osteopathic Medicine, Conroe, TX, [6]UT MD Anderson Cancer Center, Houston, TX, [7]MD Anderson Cancer Center, Houston, TX

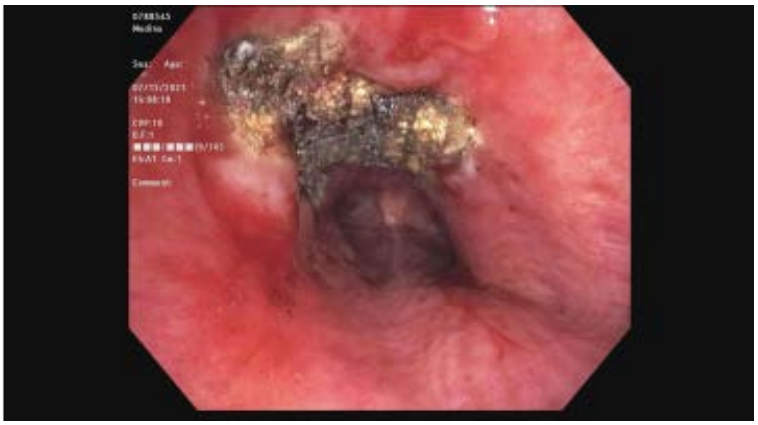
Introduction: Superior vena cava (SVC) syndrome is a known complication of advanced lung cancer. Stenting is frequently performed for symptomatic patients. Anesthesia considerations typically include external compression by tumor, but not invasion into the airways. However, in patients with severe orthopnea precluding interval imaging, it is imperative to prepare for possible obstructive metastasis or tumor erosion into the airway in patients with rapidly progressive disease prior to management of the airway.

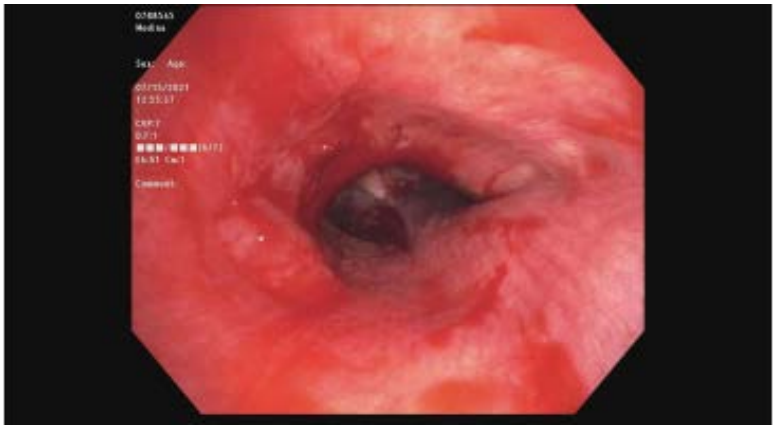
Methods: A 54-year-old male with stage 4 atypical carcinoid tumor in the right upper lobe presented for an SVC stent due to symptomatic SVC syndrome. He maintained oxygen saturation of 95% on room air at rest, though experienced progressively worsening dyspnea on exertion and critical orthopnea. This precluded repeat imaging of the chest for three months prior to planned SVC stenting. We planned an awake flexible bronchoscopic intubation. The bronchoscope was easily passed through the vocal cords but immediately afterwards a tracheal mass was visualized, occluding nearly 100% of the tracheal lumen (Figure 1). The endotracheal tube was secured proximal to the mass and the procedure proceeded uneventfully. Interventional pulmonology resected the previously unidentified tracheal tumor the following day (Figure 2,3).

Conclusion: Atypical carcinoid tumors are a rare, aggressive class of pulmonary neuroendocrine tumor and compose 1% to 5% of all lung cancers.(1,2) SVC syndrome is reported in up to 2-4% of all lung cancers during the course of their disease(3) with SVC stenting frequently performed for symptomatic relief.(4) Awake intubation decreases the risk of loss of airway due to upper airway engorgement, tracheal compression and hemodynamic compromise during induction of general anesthesia. This case report cautions that, in advanced lung cancers causing SVC syndrome, clinicians should consider the possibility of tumor invasion into the tracheobronchial tree, since blind endotracheal insertion into the trachea may dislodge a friable tumor and cause catastrophic complete obstruction or bleeding.

References: 1. Steuer CE, Behera M, Kim S, et al. Atypical carcinoid tumor of the lung: a surveillance, epidemiology, and end results database analysis. J Thorac Oncol. 2015;10(3):479-485. 2. Garg R, Kumar R, Singh P, Kshetrimayum S. Atypical carcinoid tumor of the lung: A rare entity. Lung India. 2019;36(3):236-238. 3. Hinton J, Cerra-Franco A, Shiue K, et al. Superior vena cava syndrome in a patient with locally advanced lung cancer with good response to definitive chemoradiation: a case report. J Med Case Rep. 2018;12(1):301. 4. Wei, S., Liu, J., Li, X., Song, Z., Dong, M., Zhao, H., Zhao, Q., Chen, G. and Chen, J. (2020), A retrospective stenting study on superior vena cava syndrome caused by lung cancer. Thorac Cancer, 11: 1835-1839.







Airway Management - 10 Difficult airway management secondary to laryngeal malignancy and superimposed abscess: Double trouble!

Yerin Koh[1], Avneep Aggarwal[2]

[1]Cleveland Clinic Foundation, Cleveland, United States of America, [2]Cleveland Clinic, Cleveland, OH

Introduction: Approximately 60% of Squamous cell carcinoma (SCC) Head and Neck cancers are diagnosed at advanced stage (Stage 3 or 4) for which total laryngectomy is often the only definitive treatment.[1] As the airway serves as both the operative field and means of ventilation, the anesthesiologist's role in facilitating a successful intubation becomes particularly crucial. Large airway masses can distort standard airway anatomy and predispose to challenging intubation scenarios. We present a case of difficult airway management in a patient with severe tracheal compression from Stage 4 laryngeal cancer complicated by a superimposed laryngeal abscess.

Methods: A 42-year-old male with history of hypertension, chronic smoking, heavy alcohol use and Stage T4aN2cMx laryngeal cancer presented to the ED for worsening dysphagia. Upon admission, patient was febrile (101.1°C), tachycardic (HR 130s) with a leukocytosis (WBC 19K) concerning for sepsis. Neck CT showed infiltrative multi-spatial necrotic collection centered at the right glottis measuring approximately 5.5 x 5.1 x 6.6 cm. CT scan confirmed the significant interval enlargement of the necrotic collection exerting mass effect with leftward displacement of the larynx and severe narrowing. CT scan was suggestive of superinfection with abscess formation, in the setting of mucosal necrosis, given foci of air within the collection. Smallest tracheal diameter was reported as 2 mm. He was urgently transferred from the ED to ICU for risk of acute respiratory failure.

The otolaryngology team was consulted and the decision was made to proceed with an emergent tracheostomy to protect his airway until definitive surgery. Head and neck examination revealed a visible right neck mass, approximately 0.5 cm past midline, that was firm to palpation. Airway evaluation revealed Mallampati Score of 3 and the patient had full range of motion of the neck. Patient was emergently transferred to OR for tracheostomy.

The patient was very anxious and also reported significant dyspnea with lying supine. The patient was administered 0.4 mg IV glycopyrrolate and 20 ml of 4% lidocaine through a nebulizer in the operating room. Dexmedetomidine infusion was started and 2 mg IV midazolam and 50 mcg IV fentanyl were additionally administered for comfort and anxiolysis. Lidocaine was administered with an atomizer to topicalize the oropharynx. The gag reflex was abolished when tested with a tongue depressor. An Ovassapian airway was placed in the mouth. The supraglottic mass prevented visualization of the vocal cords. The first attempt of passing the fiberoptic catheter was unsuccessful. During the second attempt, edema of the right arytenoid and AE fold hooding over the glottis was visualized with shifting of the larynx to the left. The immediate subglottic area was unable to be visualized. The fiberoptic scope was advanced and passed through an approximately 2 mm glottis opening without complication. Subsequently, a lubricated 5.0 microlaryngoscopy tube (MLT) was successfully advanced through the vocal cords over the scope. The placement of the MLT was confirmed with end tidal CO2 on capnography. Patient was sedated with IV propofol and placed on mechanical ventilation with isoflurane for the remainder of the surgery. Tracheostomy was completed uneventfully and postoperatively the patient was brought to the ICU. He was successfully discharged home with a total laryngectomy scheduled in a month.

Conclusion: Head & Neck cancers continue to present unique challenges due to unpredictable distortions of airway anatomy.[2,3] Significant tracheal compression is defined as radiological measurements of <15mm and critical compression as <5mm at its minimal diameter. Additionally, tracheal deviation (>1cm) has been reported as the strongest predictor of difficult intubation in these patients. More severe stenosis and deviation is often associated with more prominent physical exam findings. [4,5] Our case was further complicated by the presence of a superimposed laryngeal abscess on an already compromised airway. Devastating airway obstructions can be reduced by allowing spontaneous ventilation until the airway is definitely secured.[6] The primary advantage of the

awake fiberoptic intubation is that it allows direct visualization of the laryngopharyngeal anatomy and facilitates navigating a smaller-lumen tube through a critical airway stenosis.

References: 1. Elsevier. Chapter 35 Larynx and Hypopharynx Cancer. In Clinical radiation oncology (4th ed., pp. 649-672). 2016. 2. Lippincott Williams & Wilkins. Bailey's head and neck Surgery: OTOLARYNGOLOGY 5th edition (5th ed., Vol. 2, pp. 1961-1977). (2013).

3. International surgery. Fiberoptic bronchoscopy-assisted endotracheal intubation in a patient with a large tracheal tumor. Volume 100(4), 589-592. (2015). 4. Anesthesia Essays Researches. Airway management in patients with tracheal compression undergoing thyroidectomy: A retrospective analysis. Volume 11 (1): 110-116. 2017. 5. Elsevier. Goitre and difficulty of tracheal intubation. Volume 29: 436-439. 2010. 6. Anesthesiology. Practice guidelines for management of the difficult airway: An updated report by the American Society of anesthesiologist task force on Management of the Difficult Airway. Volume 118: 251-270. 2013.

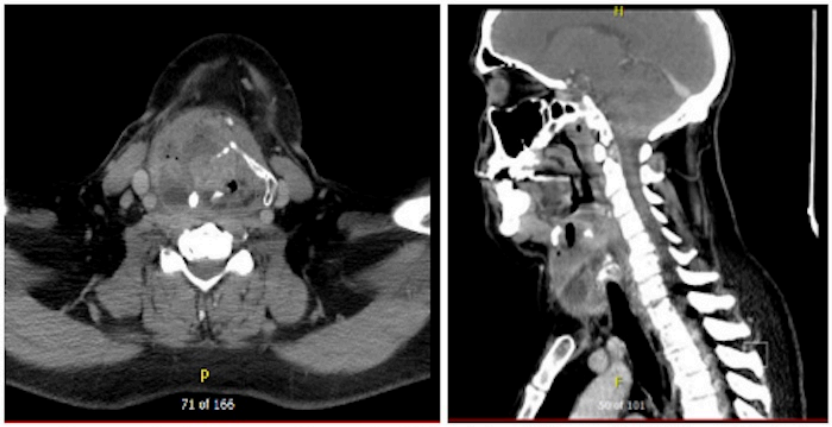


Figure 1. Laryngeal cancer lesion measuring up to 5.5 x 5.1 x 6.6 cm. Axial & sagittal view of laryngeal malignancy & abscess causing stenosis and deviation of airway

Airway Management - 11 Using a Custom Designed Bifurcated Airway Stent as a Rescue Tracheostomy Tube in a Patient with Non-Operative Tracheal Erosion - A Case Report

Jacob Cole[1], George Frendl[2]

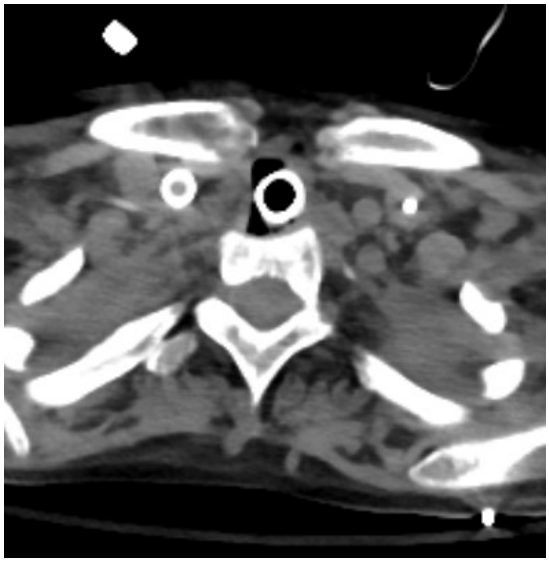
[1]Brigham and Women's Hospital, Boston, MA, [2]Brigham and Women's Hospital, Harvard Medical School, Boston, MA

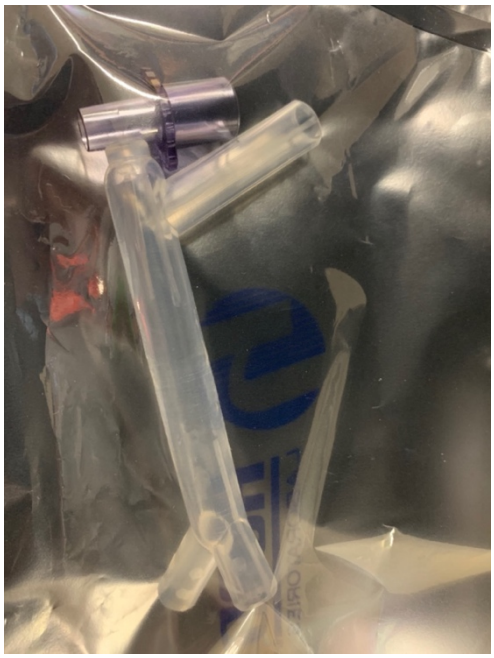
Introduction: Custom airway devices and tracheostomy tubes are potentially lifesaving tools that are heavily regulated by the US Food and Drug Administration (FDA). To be granted a Custom Device Exemption, these devices necessarily deviate from generically available products and are unique to the point that clinical investigations are impractical. They are made at the request of a qualified physician, and firms are generally limited to the production of custom devices for only 5 patients per calendar year. This case describes the clinical need, design, and manufacture of a custom bifurcated airway stent that is used as a rescue tracheostomy tube in a patient with a non-operative tracheal erosion.

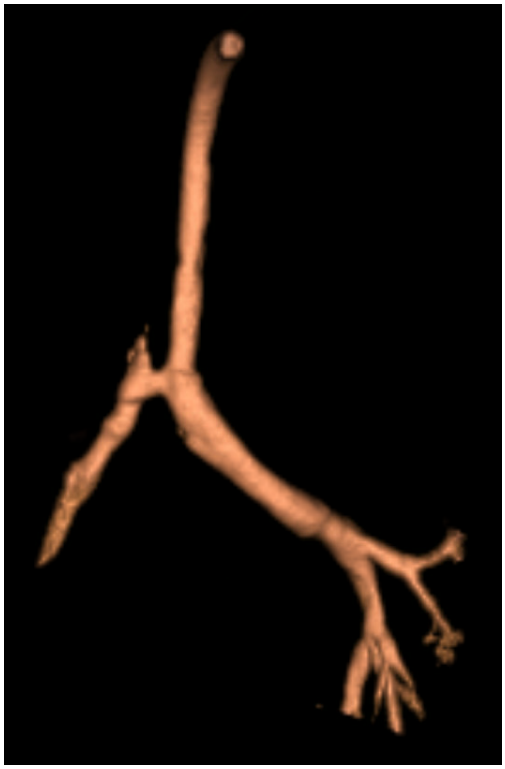
Methods: This case describes a 23-year-old woman with a history of severe idiopathic ARDS and no other notable past medical history who was initially admitted over one year prior to the placement of this custom airway stent. Her hospital course was complicated by rapid pulmonary decompensation requiring intubation, cannulation for venovenous extracorporeal membrane oxygenation (VV ECMO), conversion to venoarterial-venous ECMO due to progressive right ventricular failure, multiple surgeries for decortication of empyema and follow-on surgical morbidities, renal failure requiring renal replacement therapy, and eventual bilateral orthotopic lung transplant and deceased-donor renal transplant. Following the transplant surgery, she required ongoing VV ECMO support and eventually developed protracted tracheal hemorrhage from progressive tracheal ulceration at the site of her tracheostomy tube. On investigation, this ulceration had eroded through the posterior membrane of her trachea and incorporated portions of the T1-T3 vertebral bodies. Multiple surgical and procedural services were consulted who agreed that this pathology was non-operative. Given this ulceration, all efforts were made to avoid pressure on her trachea to allow the erosion to heal. However, the erosion was so distal in the airway that the only way to relieve this pressure was to deflate the cuff of the tracheostomy tube and allow spontaneous ventilation. During the multiple periods that this was attempted throughout her months-long recovery after the transplant surgeries, the patient rapidly developed pneumonia with the withdrawal of positive pressure ventilation requiring aggressive antimicrobial and toilet bronchoscopy therapies. It was recognized that a ventilatory solution was needed to allow for positive pressure ventilation of the lungs while avoiding tracheal pressure to allow the tracheal erosion to scar and heal. Using 3D reconstructions of high-definition CT scans as well as bronchoscopic measurements, a custom bifurcated tracheal stent was designed. Hood Laboratories (Pembroke, MA, USA) was contracted to manufacture this device as a truly custom device that was similar to a bifurcated t-tube stent. This stent acts as an uncuffed bifurcated endobronchial tube that allowed for positive pressure ventilation without the need for a tracheal balloon, allowing the patient to maintain adequate recruitment within their transplanted lungs. It was placed via rigid bronchoscopy without difficulty, and its uncuffed nature allows adequate internal lumen diameter to facilitate ongoing toilet bronchoscopy for secretion clearance as clinically indicated.

Conclusion: The US FDA can grant Custom Device Exemptions for medical device manufacturers to create truly custom products in exceptional circumstances. In this case, a truly custom, long-term airway stent was successfully designed, manufactured, and implanted in a patient with unique and complex tracheobronchial pathology. This process of unique device manufacture can potentially be used for other patients with unique medical pathology to facilitate their treatment and recovery.

References: 1. US Food and Drug Administration. Food, Drug and Cosmetic Act, Section 530(b). 2. US Food and Drug Administration. Custom Device Exemption - Guidance for Industry and Food and Drug Administration Staff. 2014.







Airway Management - 12 Prolonged Apnea after Awake Tracheostomy for Obstructive Laryngeal Cancer

Pulsar Li[1], Annandita Kumar[2]

[1]Loyola University Medical Center, Maywood, IL,
[2]Loyola University Medical Center, Chicago, IL

Introduction: Tracheostomy is offered to patients with laryngeal cancer in order to relieve stridor as well as maintain tracheobronchial access during surgical debulking and chemoradiation therapy, which can cause airway swelling. Awake tracheostomy is indicated for severe malignancies and external compression of the airway by deep neck abscesses or trauma since general anesthesia can decrease airway tone, worsening airway obstruction. We describe a woman presenting with stridor from advanced laryngeal cancer undergoing awake tracheostomy, complicated by delayed emergence and prolonged apnea.

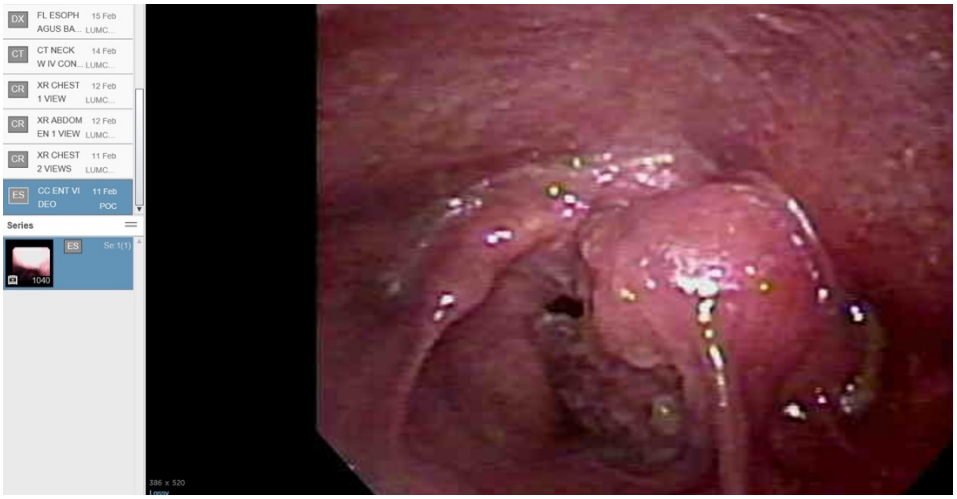
Methods: Case Report: A 54 year old female (80kg, 35 BMI) with medical history of hypertension, obesity, anxiety, former 30+ pack-year smoker, and difficult sleep for three weeks presented to the emergency department with a laryngeal mass causing severe orthopnea, respiratory distress, and hypoxia. Earlier that afternoon, she visited ENT, who performed flexible endoscopic nasopharyngolaryngoscopy revealing left glottic lesion (video 1). ENT planned for panendoscopy with biopsies the next day, however that evening, she was taken to EMS for dyspnea and 'panic attack.' In the ED, COVID test was negative, bicarbonate elevated at 31, and breathing treatments were unhelpful. 4L nasal cannula O2 improved her O2 saturation from mid-80s to 93% as well as her work of breathing. She continued to demonstrate severe stridor and increased work of breathing while sitting upright due to orthopnea. She was taken to the OR for awake tracheostomy and panendoscopy with biopsies. Light sedation was achieved with midazolam and dexmedetomidine infusion and a cuffed tracheostomy tube was placed uneventfully. During emergence, the patient remained apneic and unresponsive for thirty minutes despite total elimination of sevoflurane and administration of 0.2mg IV flumazenil. Just prior to transport to the PACU, she became agitated with irregular breathing. A 40mcg dexmedetomidine IV bolus was administered, her arms restrained, and an arterial blood gas revealed hypoxemia and respiratory acidosis with metabolic compensation (pH 7.17, pCO2 104, HCO3 38, pO2 100 on 100% FiO2). Glucose, sodium, and body temperature were normal. In the PACU, she remained apneic as minute ventilation was increased with subsequent ABG showing hypoxemia and mixed respiratory-metabolic alkalosis (pH 7.53, pCO2 43, HCO3 36, pO2 65 on 80% FiO2). Post tracheostomy CXR showed subtle pneumomediastinum tracking into tissues of the neck and bilateral perihilar/R basilar opacities from atelectasis (figure 1). The patient did not become alert and calm until POD1, when she was converted to supplemental oxygen via trach collar. She was discharged home on POD4 and completed chemoradiation for T3N0 laryngeal squamous cell carcinoma with cisplatin 3 months later.

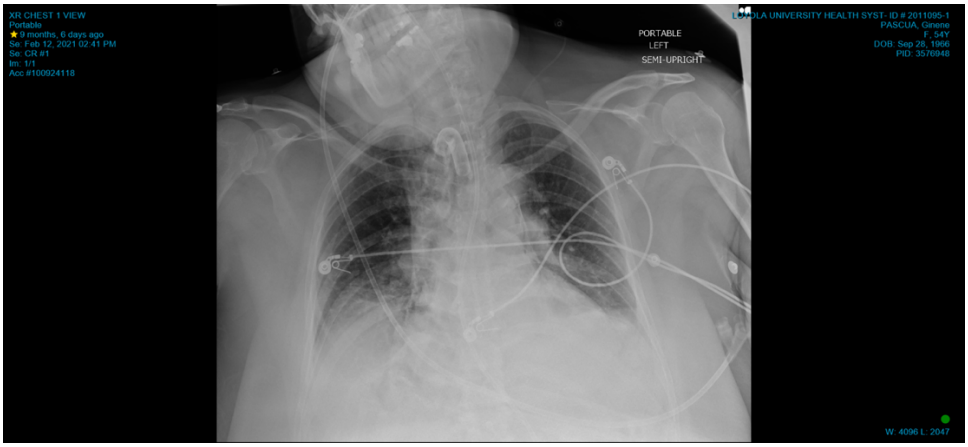
Conclusion: 1. Awake tracheostomy was chosen due to non reassuring nasopharyngolaryngoscopy in addition to the patient's need for tracheostomy during chemo-radiation. If a patient is uncooperative, spontaneous ventilation can be preserved with titration of anxiolytics and even inhaled induction with volatile anesthetics, however clear communication, availability of advanced airway equipment, and experienced providers are vital to minimizing airway loss. 2. The prolonged apnea, agitation, and delayed emergence observed after tracheostomy was likely due to her three-week history of stridor resulting in hypercapnic narcosis. Moderate elevation of PaCO2 can cause anxiety and agitation while levels exceeding 100 mmHg can alter consciousness resulting in delayed emergence (2). As described in COPD patients, blunted sensitivity to CO2 (ie, pH) can lead patients to rely on their hypoxic ventilatory drive, which itself is diminished with the administration of oxygen leading to decreased minute ventilation (3). Lastly, decreased binding affinity of hemoglobin for CO2 (the Haldane effect) in the presence of oxygen may contribute (3). 3. The hypoxemia persisting after tracheostomy was likely due to negative pressure pulmonary edema (NPPE) from the obstructing laryngeal mass. NPPE is caused by transvascular fluid filtration into alveoli and pulmonary interstitium after intense inspiratory effort against an obstructed airway and can be treated with positive pressure mechanical ventilation, continuous

positive airway pressure (CPAP), and diuretics if hypervolemic.

References: 1. Fang, C.H., Friedman, R., White, P.E., Mady, L.J. and Kalyoussef, E. (2015), Emergent Awake tracheostomy—The five-year experience at an urban tertiary care center. The Laryngoscope, 125: 2476-2479. https://doi-org.archer.luhs.org/10.1002/lary.25348

2. Mecca RS. Coma and delayed emergence. In: Lobato EB, Graventein N, Kirby RR, editors. Complications in Anesthesiology. Philadelphia, USA: Lippinoct Williams and Wilkins; 2008. p. 351-64. 3. Hanson CW 3rd, Marshall BE, Frasch HF, Marshall C. Causes of hypercarbia with oxygen therapy in patients with chronic obstructive pulmonary disease. Crit Care Med. 1996 Jan;24(1):23-8. doi: 10.1097/00003246-199601000-00007. PMID: 8565533. 4. Cascella M, Bimonte S, Di Napoli R. Delayed Emergence from Anesthesia: What We Know and How We Act. Local Reg Anesth. 2020;13:195-206. Published 2020 Nov 5. doi:10.2147/LRA.S230728





Airway Management - 13 Complications of Tracheal Resection and a Novel Airway Stent Application for Tracheal Reconstruction

Michael Tan[1], Danielle Holland[2], Lundy Campbell[1]

[1]University of California, San Francisco, San Francisco, CA, [2]UCSF, San Francisco, CA

Introduction: Providing anesthesia for tracheal surgery is a complex and infrequently performed area of airway management, requiring detailed planning with the surgical team. Evolving techniques with airway stents also provide new potentials in tracheal surgery. Here, we present a case of tracheal resection that reviews conventional intra-operative anesthetic management and highlights key complications of tracheoinnominate and tracheocutaneous fistula, along with a novel tracheal reconstruction technique using an airway stent.

Methods: A 62-year-old man with history of Type 2 Diabetes and Hypertension suffered blast fire burns to over 30% body surface area on his torso and extremities, requiring multiple intubations for airway protection. One month after discharge from hospitalization for his burn injuries, he presented with progressive dyspnea concerning for subglottic stenosis. Examination in the operating room revealed a 2.1cm length tracheal stenosis (5mm diameter), located 2.5cm below the vocal cords. Serial balloon dilations and electrocautery were used to dilate the tracheal narrowing to 12mm. Given ongoing dyspnea despite serial dilations, the patient returned to the operating room for tracheal resection. He had an uneventful induction and oral intubation with a 6.5mm endotracheal tube (ETT), and a maintenance anesthetic of Propofol and Remifentanil infusions. The ETT was advanced easily past the recently dilated stenosis under fiberoptic guidance. After dissection down to the trachea, the oral ETT was partially withdrawn, and an anterior tracheal incision was made at the distal end of the stenosis. A sterile, armored ETT was inserted into the distal trachea and connected to a cross-table ventilatory circuit. 3cm of tracheal resection was performed, then anastomosis of the posterior, membranous trachea was completed. At this point, the distal, armored ETT was removed, and the oral ETT was re-advanced past the anastomosis under direct vision (Figure 1). Closure was completed, and he was extubated uneventfully. On post-operative day 8, the patient developed a neck hematoma with massive hemoptysis, and an urgent nasal intubation was performed in the intensive care unit. Subsequent neck exploration revealed bleeding from a posterior ulceration in the innominate artery, which was divided and ligated. Right carotid stump pressures were adequate at 60-65 mmHg. The patient was extubated two days later, but unfortunately developed a tracheal leak and was re-intubated after one day (Figure 2). Repeat neck exploration showed complete dehiscence of the tracheal anastomosis. Five days later, after careful multi-disciplinary surgical planning was complete, repair of the 3cm tracheal dehiscence was accomplished by placing a 15mm x 6cm silicone stent via rigid bronchoscope to bridge and overlap the proximal and distal ends of trachea (Figure 3). Ventilation was maintained with sterile cross-table ventilation during deployment and suturing of the stent; then, a 5.0mm oral ETT was advanced with fiberoptic guidance through the stent, into the distal trachea. A pectoralis muscle flap was used for tracheal reconstruction around the stent. The patient was extubated successfully after two weeks. A gastrojejunostomy feeding tube was placed for pharyngeal dysphagia, and he was discharged to acute rehabilitation.

Conclusion: Close communication and understanding of potential complications are critical in anesthetic management of tracheal resection. In this case, prompt recognition and management of two separate high mortality post-operative complications allowed for successful interventions. We believe this is the first described instance of combining a silicone airway stent and multi-disciplinary tracheal reconstruction to create a unique solution for complete tracheal dehiscence.

References: 1. Journal of Cardiothoracic and Vascular Anesthesia. 2020;34(7):1902-1913. 2. Anesthesiology Clinics. 2012;30(4):709-730. 3. The Annals of Thoracic Surgery. 2019;108(5):1471-1477.

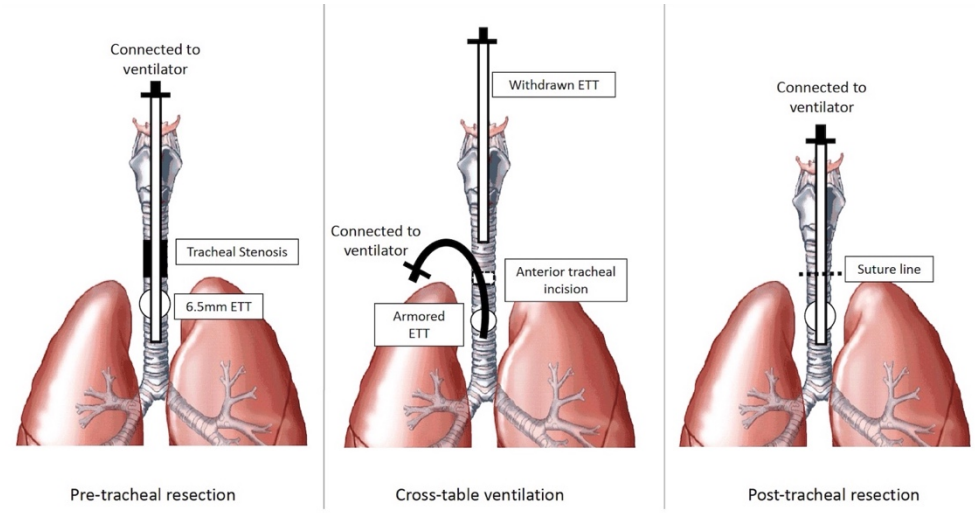


Figure 1: Cross Table Ventilation

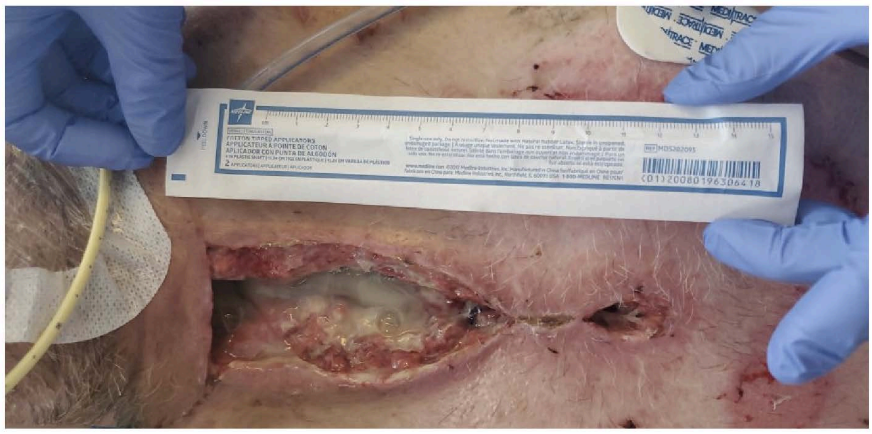
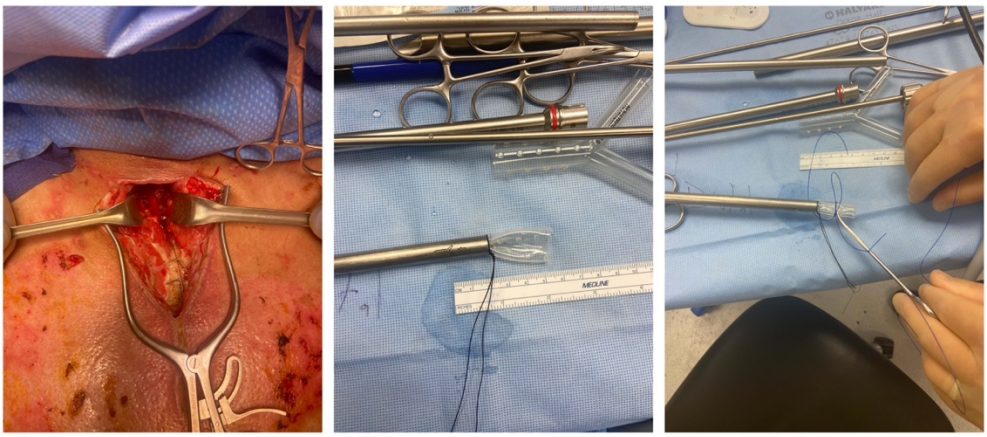


Figure 2: Tracheocutaneous Fistula



(A) Tracheal Dehiscence (B) Silicone Airway Stent (C) Stent with Sutures

Figure 3: (A) Complete dehiscence of tracheal anastomosis with exposed endotracheal tube. (B) Silicone airway stent (15mm x 6cm) prepared on rigid bronchoscope prior to insertion. (C) Sutures attached to airway stent for securement to proximal and distal ends of trachea.

Airway Management - 14 Laryngeal Mask Airway For Control Of Bleeding And Difficult Airway Management In A Patient Undergoing Mechanical Thrombectomy For Basilar Artery Occlusion Status Post Systemic tPA

Nishita N Patel[1], Allen Wang[2], Wissam Mustafa[2]

[1]Boston University School of Medicine, boston, MA, [2]Boston University School of Medicine, Boston, MA

Introduction: The Laryngeal Mask Airway has an established role in elective and rescue airway management in the difficult airway algorithm [1]. Anesthesiologists are oftentimes required to secure the airway under emergent conditions where laryngoscopy and tracheal intubation can be challenging. Here we describe a case of a difficult airway complicated by severe airway bleeding that was successfully managed by using an LMA supreme.

Methods: This is a 39-year-old, 153 kg man with uncontrolled hypertension presented for mechanical thrombectomy in the setting of basilar artery occlusion status post systemic tPA. Rapid sequence induction and attempted Glidescope intubation complicated by an obscured glottic view due to brisk oropharyngeal bleeding, a large epiglottis, and redundant tissue resulted in esophageal intubation. Two-handed, two-provider mask ventilation with an oral airway was difficult. An LMA Supreme was placed successfully. An oral endotracheal tube was advanced through the LMA over a fiberoptic bronchoscope. The LMA was left in place overnight with the cuff inflated to tamponade oropharyngeal bleeding. On postoperative day 1, the endotracheal tube was exchanged over an airway exchange catheter uneventfully. The patient was extubated on postoperative day 4 with no further airway complications.

Conclusion: The Laryngeal Mask Airway has been successfully used as a means of rescue for 'cannot intubate and cannot ventilate' situations within the field of Anesthesiology. The use of an LMA during an unanticipated difficult airway provides anesthesiologists with an alternative mode to successfully ventilate the patient until a more definitive airway is secured. In our case, we describe a situation in which a difficult intubation further complicated by bleeding was managed by the placement of an LMA. In turn, the cuff of the LMA tamponaded the oropharyngeal bleeding and allowed adequate ventilation until the airway was secured with an endotracheal tube. Realizing that the LMA served a dual purpose, the decision was made to leave it in place overnight (with the cuff inflated) until adequate hemostasis was achieved. Whenever instrumenting the airway, an LMA should always be readily available for rescue in the event that a difficult airway situation arises.

References: 1. Onyekwulu FA, Nwosu A. Emergency airway management with laryngeal mask airway. Niger J Clin Pract. 2011;14(1):95-97 2. Pearson DM, Young PJ. Use of the LMA-Supreme for airway rescue. Anesthesiology. 2008;109(2):356-357 3. Timmermann A, Cremer S, Eich C, et al. Prospective clinical and fiberoptic evaluation of the Supreme laryngeal mask airway. Anesthesiology. 2009;110(2):262-265. 4. Sch√§uble JC, Heidegger T. Erratum zu: Management des schwierigen Atemwegs : √úbersicht √ºber die aktuellen Leitlinien [Erratum to: Management of the difficult airway : Overview of the current guidelines]. Anaesthesist. 2019;68(1):29.

Airway Management - 15 Are You All In or All Out? Successful use of endotracheal tube "push-in" technique to maintain ventilation with high FiO2 and PEEP during open tracheostomy in a critically ill morbidly obese patient

Joseph Hodapp[1], Carlos Brun[2]

[1]Stanford University, Stanford, CA, CA, [2]Stanford University, Stanford, CA

Introduction: Conventional technique for a surgical tracheostomy involves withdrawing the endotracheal tube (ETT) immediately prior to formation of the tracheal stoma, in order to ensure the cuff is not within the surgical field1. In order to perform this technique, ventilation and oxygenation must be ceased upon tracheal incision. The patient will be apneic, and have no provision of oxygen or positive-end expiratory pressure (PEEP) for up to several minutes. However, in patients where continued ventilation/oxygenation is a physiologic necessity (hypoxemia requiring high fraction of inspired oxygen [FiO2], high PEEP requirements, aerosolization risk in respiratory infections), this conventional technique may not be able to be safely performed. Our alternative method combines the ETT 'push-in' technique2,3 and high-flow nasal cannula apneic oxygenation ('HFNC APOX')[4,5] and 'clamp' technique6 to allow continued, safe ventilation/oxygenation/PEEP maintenance throughout formation of the tracheal stoma[2,3] and until a tracheostomy tube is placed. This is the first case report employing all three of these techniques to optimize safety during surgical tracheostomy.

Methods: A 68 year-old man with a history of Class III morbid obesity (BMI 60), severe OSA and Gold 1 COPD on nocturnal BiPAP 20/10 presented with urosepsis complicated by acute on chronic hypoxic-hypercapnic respiratory failure requiring mechanical ventilation with high PEEP requirements (PEEP min 13-max 20) and low FiO2 requirements (30-50%). ENT was consulted for a surgical tracheostomy. Given the patient's high PEEP requirement, we decided to pursue a push-in technique with the ETT during tracheal stoma formation. We utilized a fiberoptic bronchoscope to guide the ETT tip to the carina under direct visualization. During the dissection, we maintained low FiO2 (<30%) and continued to ventilate on a high PEEP (20 cm H2O). We maintained this ventilatory technique throughout incision into the airway, formation of the tracheal stoma, and suturing of the Bjork flap -- allowing our surgical colleagues ample time with minimal stress to perform the tracheostomy safely. When it was time to extubate the orotracheal tube and place the tracheostomy tube, we preoxygenated at an FiO2 of 100%, initiated nasal high flow oxygen at 70 lpm, then paused respirations. We removed the ETT under direct FOB guidance while the ENT surgeons placed an 8.0 cuffed tracheostomy tube. This portion of the procedure took roughly 12 seconds. Vital signs remained stable throughout this brief apneic period, and his SpO2 actually increased from 97% to 99% upon re-initiation of mechanical ventilation. The tracheostomy tube was sutured in place and the patient was uneventfully transported back to the ICU in stable condition.

Conclusion: The primary benefit of the push-in technique described above is the maintenance of oxygenation and ventilation throughout the formation of the tracheal stoma, decreasing surgical time pressure and decreasing the risk of critical desaturation events associated with prolonged apnea times. A critical step to ensure that this method can be performed safely is to evaluate pre-operative chest imaging to measure the distance from the carina to the sternal notch to ensure that your ETT cuff will be distal to the surgical field when you advance the ETT to the carina. We also employed the use of nasal high flow oxygen at 70 lpm to provide some maintenance PEEP (~7 cm H2O at 70 lpm) and apneic oxygenation during the brief period that our patient was removed from ventilator support to extubate the orotracheal tube and place the tracheostomy tube. During the duration of the surgical tracheostomy, utilizing these techniques can minimize apnea time, effectively reducing the risk of critical desaturation.

References: Journal of Oral and Maxillofacial Surgery, vol. 57, no. 3, 1999, pp. 310-15 Auris, Nasus, Larynx, vol. 47, no. 3, June 2020, pp. 472-76 BJA: British Journal of Anaesthesia, vol. 99, no. eLetters Supplement, Jan. 2007 StatPearls, StatPearls Publishing, 2021, http://www.ncbi.nlm.nih.gov/books/NBK526071/.

	Starting Parameters	First Stage	Second Stage	Resaturation Time
FiO2 Challenge (PEEP maintained)	FiO2 35%/PEEP 13 SpO2 94%	Decreased FiO2 to 28% SpO2 91% after 15 minutes	Decreased FiO2 to 21% SpO2 85% after 1 minute	Increased FiO2 to 100% SpO2 100% after 1 minute
PEEP Challenge (FiO2 maintained)	FiO2 50%/PEEP 13 SpO2 97%	Decreased PEEP to 5 SpO2 94% after 15 minutes	Decreased PEEP to 0 SpO2 to 87% after 2 minutes	Increased PEEP to 10 SpO2 97% after 90 seconds

Table 1. PEEP/FiO2 Challenge Table

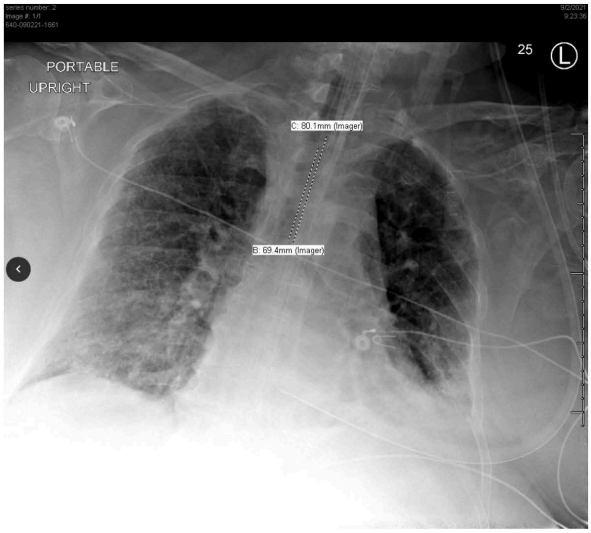


Figure 1. Pre-operative chest radiograph

Figure 2. Intraoperative ventilator settings

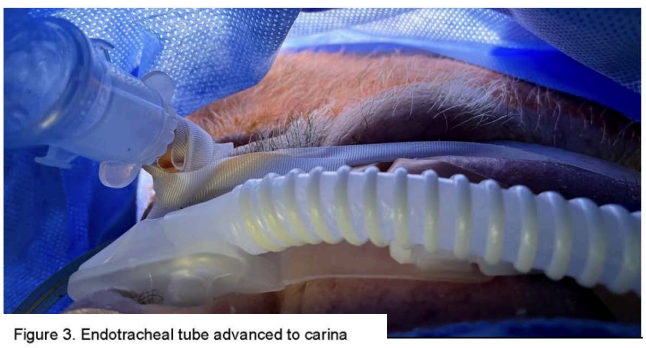


Figure 3. Endotracheal tube advanced to carina

Ambulatory Anesthesia

Ambulatory Anesthesia - 1 Superficial Thrombophlebitis after Caldolor (IV Ibuprofen) Administration

Iyabo Muse[1]

[1]*Westchester Medical Center/ New York Medical College, Valhalla, NY*

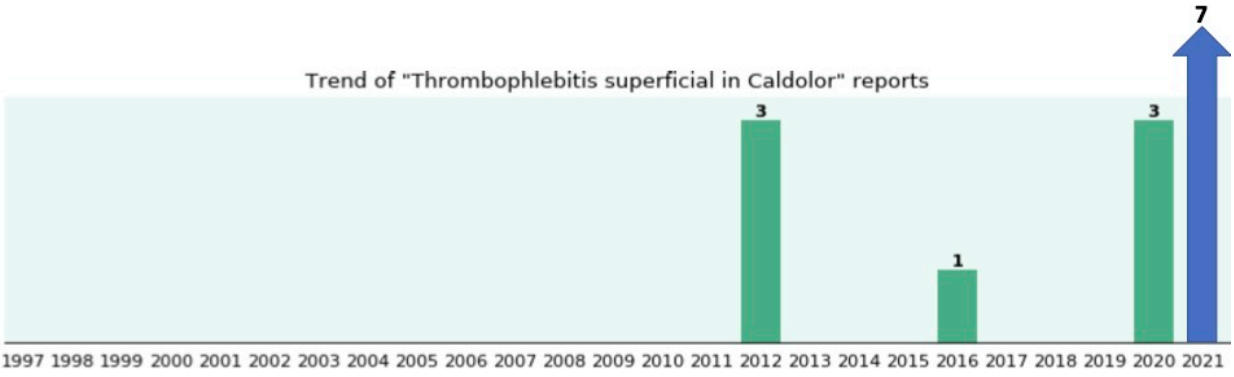
Introduction: Caldolor is an intravenous formulation of ibuprofen that was FDA approved in June 2009 for adult use. Caldolor must be diluted to a concentration of 4mg/ml or less in 0.9% normal saline or 5% glucose prior to infusion over 30 minutes. Its utilization for perioperative pain management has steadily increase thru the years and especially in ambulatory anesthesia where the goal is to minimize opioid utilization and its adverse side effects. Although IV ibuprofen as compared to oral ibuprofen has been shown to be more effective in reducing postoperative pain and opioid utilization, it does carry a risk of causing infusion site pain (5-15%) and superficial thrombophlebitis (Figure 1).1,2 Superficial thrombophlebitis is the swelling, inflammation, and thrombosis of a superficial vein caused by a blood clot. We present a case of swelling, pain, and erythema of the hand in outpatients undergoing breast mammoplasty at an ambulatory surgery center with one patient requiring inpatient admission.

Methods: A 32 year old female with previous history of gastric sleeve surgery and current history of GERD. She presents to a free standing ambulatory surgery center for breast mammoplasty/augmentation. A 22G peripheral IV was placed at the back of the left hand. The patient received preoperative oral acetaminophen 975mg, Lyrica 50mg, and aprepitant 40mg. She also received preoperative bilateral paravertebral nerve block for postop management. Intraoperatively, the patient received general anesthesia with endotracheal tube. Medications given intravenously included midazolam, fentanyl, rocuronium, cefazolin, dexamethasone, hydromorphone, ondansetron, sugammadex, and caldolor in sequential order. Caldolor 800mg was given without dilution in 2ml increments over 10 minutes with normal saline infusing thru the 22G IV at the end of the procedure. Patient was dropped off in the PACU asleep but arousable. Within an hour after arrival to PACU, the nurse noted that the patients' hand was swollen and erythematous. Patient began to complain of moderate pain of the hand. The IV was removed, warm compress and oral oxycodone was given to the patient. The patient hand was also elevated to improve drainage. Patient's pulses were intact, she was able to move all fingers but complained of some tingling in the ulnar distribution of the hand. Patient was observed in the PACU for another 3 hours with swelling still present but pain improving with medication. However due to the redness and edema, the patient was transferred to the hospital for 24 hour observation. On POD#1, the patient was seen by hand surgeon, who ruled out compartment syndrome. A hand vascular duplex was obtained which showed superficial and deep vein thrombosis. The patient was started on Apixaban 5mg twice a day. On POD#2, patient was discharged with 2 weeks supply of apixaban after reporting minimal pain and continue improvement of hand edema.

Conclusion: Caldolor is a good drug to reduce postoperative pain and decreased opioid use and its adverse effect in the perioperative setting. However, one must be vigilant and follow the instructions on the drug packet. Caldolor must be diluted and given at a rate of 4mg/ml or less to reduce the risk of infusion site pain and superficial thrombophlebitis. This is especially important when doing cases in free standing ambulatory surgery center because unexpected admissions to inpatient facility after surgery is reported to the state medical board, increases the cost of care for the patient, and the hospital may not be reimbursed by the insurance company. However, most importantly an unexpected admission may cause increase patient anxiety and dissatisfaction with their medical care.

References: 1. Southworth SR, Sellers JA. Narrative Summary of Recently Published Literature on Intravenous Ibuprofen. Clin Ther. 2020 Jul;42(7):1210-1221. Epub 2020 Jun 3. PMID: 32505495. 2. Bergese SD, Candiotti K, Ayad SS, Soghomonyan S, Gan TJ; Intravenous Ibuprofen Surveillance Trial Investigational Sites. The shortened infusion time of intravenous ibuprofen part 1: a multicenter, open-label, surveillance trial to evaluate safety and efficacy. Clin Ther. 2015 Feb 1;37(2):360-7. Epub 2015 Jan 8. PMID: 25577542.

Figure 1: Cases reported with superficial thrombophlebitis after the administration of Caldolor from FDA data, a Phase IV clinical study. https://www.ehealthme.com/ds/caldolor/thrombophlebitis-superficial/



Ambulatory Anesthesia - 2 A Novel Nasal Mask-Face Tent and Self-Inflating Bag Maintained Spontaneous CPAP Ventilation/Oxygenation and Reduced Aerosol/Droplet Spread in a Super Obese OSA Patient during Outpatient TEE/CV at NORA

James Tse[1], Nathaniel Hughes[1], Julie Wise[1], Antonio Chiricolo[1], Sylviana Barsoum[1]

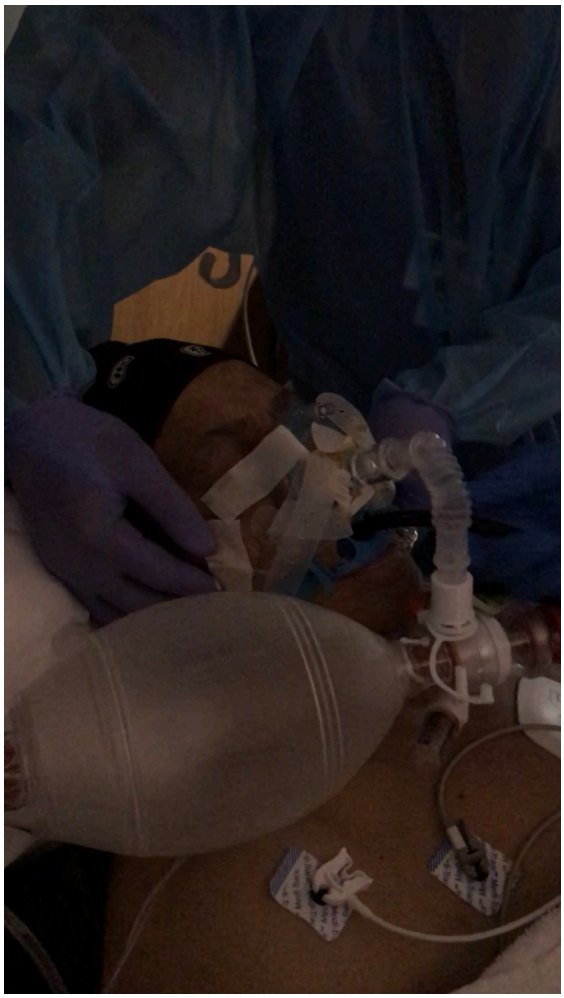
[1]Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ

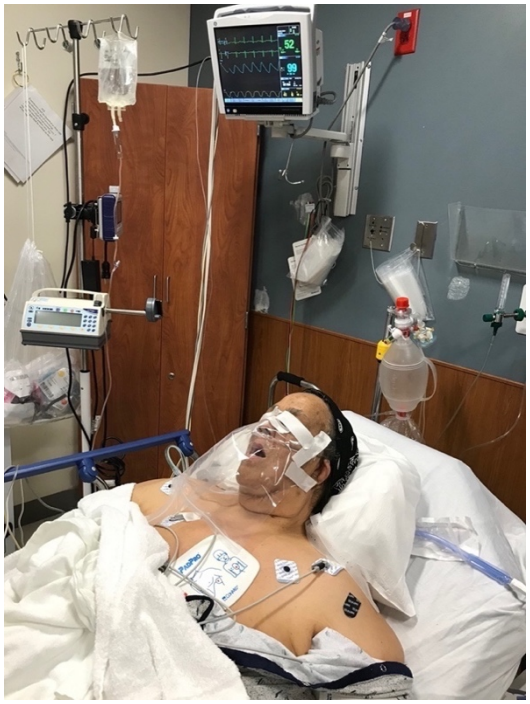
Introduction: It is always very challenging to provide MAC for patients at non-OR anesthesia (NORA) locations without an anesthesia machine. Over-sedation and/or airway obstruction may result in severe desaturation, especially in obese patients with obstructive sleep apnea (OSA). A simple modified infant facemask has been shown to provide spontaneous nasal CPAP ventilation and improve oxygenation using an anesthesia machine in deeply sedated obese OSA patients during various procedures. (1-6) Amid COVID pandemic, a combined nasal mask-face tent provided pre/apneic nasal oxygenation and reduced aerosol/droplet spread during RSI, intubation and extubation in a COVID-19 positive patient. (7) We used this technique combining with a self-inflating bag to provide nasal CPAP ventilation/oxygenation and reduce aerosol/droplet spread in a super obese patient during outpatient TEE/cardioversion (CV) in the Echocardiography Suite.

Methods: Case Report: A 68-year-old female, 5'5', 319 lbs, BMI 53.1 kg/m2, with HTN, COPD, OSA, cardiomyopathy, dyspnea and atrial fibrillation, presented for outpatient TEE/cardioversion. She had a Mallampati Class III airway and 99% SpO2 on 2 L O2/min via nasal cannula. She gave her consent for nasal mask, photography and case report. A modified infant facemask was secured over her nose with elastic head-straps and connected to a self-inflating bag via a flexible connector (Fig. 1). She was pre-oxygenated with the PEEP valve closed and 10L O2/min from wall O2 supply line. Following insertion of TEE probe, her mouth was covered with a clear plastic sheet (face tent) to reduce aerosol/droplet spread (Fig. 2-3). Deep sedation was titrated with lidocaine/propofol. The patient tolerated TEE and cardioversion well and maintained spontaneous nasal CPAP ventilation and 100% SpO2 throughout without airway obstruction or the need for airway manipulation (Fig. 4). She recovered promptly while on 4L O2/min via nasal cannula and a face tent covering her mouth to reduce aerosol/droplet spread (Fig. 5).(1) .

Conclusion: This simple nasal mask-face tent combined with a self-inflating bag provided spontaneous nasal CPAP ventilation/oxygenation in a super obese patient with OSA, COPD and atrial fibrillation during outpatient TEE/cardioversion at a NORA location without an anesthesia machine. It also reduced aerosol/droplet spread during the procedure. Amid the ongoing COVID-19 pandemic, this technique may improve patient safety and provide additional provider protection.

References: 1. www.TSEmask.com; 2. SAMBA 28th AM, MCC, 2013; 3. SASM MC,Äôs, 2013; 4. ASA AM (MC536 & 1100), 2013; 5. NYSSA 67th PGA (MCC 7094, 7115, 7120, 7129, 7189, 7199 & 7203), 2013; 6. SAM AM MCC,Äôs, 2014; 7. ASA virtual Annual Meeting: (MC1280), 2020





Ambulatory Anesthesia - 3 Episodic Enervations: A Case Postoperative Weakness

John L Csiha[1], Howard Lee[1]

[1]Northwestern Memorial Hospital, Chicago, IL

Introduction: Postoperative weakness can lead to patient discomfort, delayed discharge, and prolonged hospital stay. It has many risk factors and causes, both intrinsic and extrinsic, that should be addressed and reviewed in the pre-, intra-, and postoperative settings. When encountered, it requires immediate treatment to prevent further harm to the patient, and investigation of the source to prevent recurrence.

Methods: A 31-year-old male with a history of Crohn's, eosinophilic esophagitis, migraines, and episodes of rhabdomyolysis after multiple procedures with different anesthetic techniques underwent an esophagogastroduodenoscopy and colonoscopy. Previously, he underwent a lipoma removal under sedation with propofol and developed rhabdomyolysis; and had a similar experience after a GI procedure where he received general anesthesia with etomidate, succinylcholine, and sevoflurane. Past malignant hyperthermia testing was inconclusive. He subsequently had multiple general anesthetics with etomidate, rocuronium, and volatile anesthetic without complication. It was decided to provide the patient with a similar anesthetic. No issues occurred during the procedure. He was reversed with sugammadex. Train of four pre/post reversal was 4/4. He was extubated and brought to post anesthesia care unit. Upon arrival, he complained and had signs of diffuse body weakness and pain. He underwent a full diagnostic workup in which only his creatinine kinase was elevated. Over several hours, his weakness improved, and he was admitted for further care. His symptoms completely resolved after a 24 hours, and his creatinine kinase normalized. He described this incident as 'mild' compared to prior.

Conclusion: Isolated or recurrent postoperative weakness requires diligence to ensure the patient's immediate and future health and safety. Common causes of postoperative weakness include inhaled anesthetics, hypothermia, electrolyte disturbances, anemia, prolonged immobility, inflammation, or neurologic injury. This case report explores the various causes and risks that lead to these symptoms and provides insight on how to approach and manage a patient with postoperative weakness. We hypothesize our patient most likely has an undiagnosed myopathy or mitochondrial disorder resulting in his weakness and prior rhabdomyolysis events.

Ambulatory Anesthesia - 4 Case Report: Cushing's Triad as a Complication of Sinus Surgery at an Ambulatory Surgical Center

Jonathan Vachon[1], Javier Marull[1]

[1]UT Southwestern Medical School, Dallas, TX

Introduction: Cushing's Triad, also known as Cushing's Reflex (CR), describes a physiological state of significantly elevated blood pressures, apnea, and bradycardia in response to raised intracranial pressures (ICP). Increased ICP are commonly due to hemorrhage, tumors, cerebral edema, and acute trauma. In the setting of increased ICP, the cerebral perfusion pressure (CPP), which is calculated from the difference of the mean arterial pressure (MAP) and the ICP, is not strong enough to overcome the increased ICP and therefore cannot perfuse the brain adequately [1]. A primary literature search revealed only two other cases of CR related to sinus surgery, none of which were elective procedures [2, 3]. Here, we describe an unusual case of a patient undergoing Functional Endoscopic Sinus Surgery (FESS) who exhibited CR and her subsequent emergency treatment.

Methods: A 53-year-old female with no past medical history presented to the outpatient surgical center for a FESS procedure for definitive treatment of chronic sinusitis. She is an ASA 1 patient. It was a standard induction with propofol, rocuronium, fentanyl, and sevoflurane, with midazolam used as a sedative on the way to the operating room. Standard ASA monitors were applies, and it was an easy airway and intubation. Her vitals at the point of induction and intubation were all within normal limits. Through the first 45 minutes of the procedure, her heart rate (HR) slowly dropped from 70 to a bradycardic 50 BPM, and MAP had a slight bump to the 90s from its baseline of 70. At the one-hour mark, the patient developed a sharp increase in her MAP, from 70 to 160 mmHg (BP = 210/120) in the span of 7 minutes, and HR rose to 90 BPM. At this time, additional fentanyl, 100% Oxygen, and increased flow of sevoflurane was given to the patient. After treatment, MAP precipitously dropped to around the mid 50s, but the HR stayed elevated at around 90 BPM. There were no changes to vitals for the remainder of the procedure. After completion of the surgery, the patient was transferred to a nearby hospital for further evaluation.

Conclusion: Cushing's reflex is a rare but severe physiological nervous system response that can happen not only in acute surgery centers, but ambulatory surgical settings as well. The steps for management must be swift and judicious. Management targets the physiological idea of decreasing the ICP and elevating MAP to restore perfusion to the brain (Cerebral Perfusion Pressure). Initial emergency treatments for CR include hyperventilation, elevation of the head and bed to 30°, and diuresis by mannitol (1mg/kg) and furosemide to lower ICP and the risk of herniation. In some cases, steroids or rarely cerebrospinal fluid drainage via ventriculostomy are also administered [4-6]. Transfer of patient to a hospital for imaging and evaluation is also needed for further management.

References: 1. Dinallo S, Waseem M. Cushing Reflex. May 24, 2020. https://www.ncbi.nlm.nih.gov/books/NBK549801/ 2. Zabalo San Juan, G., Vázquez Míguez, et al (2019). Intracranial hypertension caused by superior sagittal sinus stenosis secondary to a depressed skull fracture: Case report and review of the literature. Neurocirugia (Asturias, Spain), 30(5), 243–249. 3. Neves MWF, Pontelli LOC, et al. Giant mucopyocele associated with intracranial hypertension: Case report and literature review. Surg Neurol Int. 2017;8:242. 4. Francony G, et al. Equimolar doses of mannitol and hypertonic saline in the treatment of increased intracranial pressure. Crit Care Med. 2008;36:795. 5. Stocchetti N, Maas AI, Chieregato A, van der Plas AA. Hyperventilation in head injury: a review. Chest. 2005;127:1812. 6. Hoff JT, Reis DJ. Localization of regions mediating the Cushing response in CNS of cat. Arch Neurol. 1970;23:228–240.

Anesthetic Pharmacology

Anesthetic Pharmacology - 1 Case Report: Subacute Combined Degeneration following Nitrous Oxide Abuse

Jeffery DeGrauw[1], David Takehana[2], Robert Camarillo[2], Jay Patel[3]

[1]HCA Healthcare, Las Vegas, NV, [2]Western University of Health Sciences, Pomona, CA, [3]PIH Downey, Downey, CA

Introduction: Nitrous oxide is a commonly available inhalant used commercially in whipped cream canisters, in the automobile industry, and as a sedative for medical and dental procedures. Younger populations abuse the substance for its euphoric and psychedelic effects, which can lead to a dangerous inactivation of vitamin B12, causing megaloblastic anemia, permanent neurologic deficits, and psychiatric changes. Swift recognition of this form of myeloneuropathy lessens the morbidity of the disease but is frequently complicated by normal vitamin B12 levels on lab reports and patients, as well as doctors, unfamiliarity with the abuse of nitrous oxide.

Methods: An 18-year-old man developed subacute combined degeneration over a six-month period of frequent and consistent nitrous oxide abuse. Diagnostic workup revealed a normal vitamin B12 level in the setting of elevated folate, methylmalonic acid, and homocysteine levels. Additionally, imaging revealed expanded cervicothoracic spinal cord with diffuse abnormal signal and normal caliber. The patient showed rapid improvement with intramuscular vitamin B12 repletion and elected for an outpatient continuation of treatment.

Conclusion: Nitrous oxide abuse is an emerging phenomenon amongst adolescents, presenting with symptoms of ataxia, paresthesia, hypoesthesia, urinary retention, cognitive decline, and muscle weakness. Laboratory diagnosis is made with decreased vitamin B12 levels with elevated methylmalonic acid levels; however, this is not always the case as seen in this patient. Therefore, it is important to take a detailed substance use history in patients with these symptoms regardless of the lab results. Treatment involves vitamin B12 intramuscular injections with the cessation of nitrous oxide use.

References: The CBHSQ Report, (2017) Regulatory Toxicology and Pharmacology, 73(3), 790-796, (2015) The American Journal on Addictions, 25(5), 358-369, (2016) Journal of Thrombosis and Thrombolysis, 49, 501-503 (2020) World Journal of Clinical Cases, 7, 4057-4062 (2019) Clinical Toxicology 58, 476-481 (2020) Journal of Clinical Psychopharmacology, 40, 80-83 (2020) Journal of Clinical Neuroscience, 72, 277-280 (2020) Brain and Development, 41, 428-435 (2019) Medical Journal of Australia, 211, 428 (2019) Frontiers in Psychiatry, 8, 312 (2018) Anesthesia & Analgesia, 58, 104-106 (1979) Practical Neurology 15, 207 (2015)

Anesthetic Pharmacology - 2 A case of recurrent propofol-induced dystonia

Federico Ciardi[1], Michael R Fettiplace[2]

[1]Massachusetts General Hospital, Boston, MA,
[2]Massachusetts General Hospital, BOSTON, MA

Introduction: Propofol-induced dystonia (PID) is a rare adverse effect of propofol, easily misattributed to epileptiform or functional disorders[1-4]. We describe a case of suspected PID successfully managed perioperatively with the avoidance of GABAergic hypnotic agents.

Methods: A 32-year-old female with a past medical history of supraventricular tachycardia, migraine, seronegative inflammatory arthritis with temporomandibular joint involvement, and prior post-anesthesia dystonic reactions, who presented for temporomandibular joint arthroscopy. The first episode of postoperative dystonia occurred in 2008 following catheter ablation: two days after surgery the patient developed eye pain along with nausea and vomiting. Computed tomography and spinal fluid analysis were unremarkable, and she was treated with morphine and ondansetron. Following discharge she developed dystonia, dysarthria, and ataxia. Neurology consultation suggested a diagnosis of drug-induced parkinsonism due to ondansetron and she was successfully treated with diphenhydramine. The second episode occurred in January 2020 following ankle arthroscopy under regional block and propofol sedation. Following emergence, the patient noticed abnormal movements and was treated with diphenhydramine, diazepam, and clonidine. A final episode occurred in September 2020 following a propofol/remifentanil TIVA. The dystonic reaction developed immediately after emergence and lasted ten days. She responded to diphenhydramine, benztropine and benzodiazepines. Neurology consult advised the symptoms were functional and recommended discontinuation of medications. For the current surgery, propofol was avoided. The patient received diphenhydramine, hydromorphone, midazolam and dexmedetomidine as premedication. We induced general anesthesia via single-breath induction, maintained the patient on sevoflurane, relaxed the patient with rocuronium, used dexamethasone for post-operative nausea and vomiting prophylaxis and avoided anti-dopaminergic agents. Post-operatively, the patient experienced no involuntary movements. PONV did occur and was treated successfully with prochlorperazine.

Conclusion: Given the utility and safety of propofol, it is used ubiquitously in anesthetic practice. Prior literature recommends the use of diphenhydramine, benztropine and lipid emulsion to treat propofol-induced dystonia[4]. However, our patient previously experienced dystonic reactions even with appropriate pre-treatment. In response, we utilized a propofol sparing anesthetic that successfully prevented dystonic reactions. Involuntary movements may occur via a mechanism distinct from other drug-induced dystonic reactions, as evidenced by the tolerance of anti-dopaminergic agents such as prochlorperazine. Recognition of this rare adverse effect must be emphasized as appropriate management is easily employed to avoid patient harm in the perioperative period.

References: 1. Neurology. 2002;58(9):1327-1332. 2. Br J Anaesth. 1993;70(4):459-461. 3. Can J Anaesth. 1995;42(8):774-776. 4. Clin Res Regul Aff. 2000;17:19-26

Anesthetic Pharmacology - 3

Intraoperative Management of Type 2 Protamine Reaction

Uchenna Ubozoh[1], Kenneth Sutin[1]

[1]NYU Grossman School of Medicine, New York, NY

Introduction: Protamine sulfate is a common reversal agent for heparin anticoagulation after vascular or cardiac surgery. Protamine reactions are a rare but potentially devastating adverse effect of administration with a mortality rate of approximately 23.5% in patients suffering a severe event during that hospital admission. (1) This medically challenging case will highlight diagnosis and intraoperative management of an anaphylactic protamine reaction.

Methods: 68 year old male with severe peripheral artery disease with multiple vascular interventions including bilateral deep femoral endarterectomies and right to left fem-fem bypass graft complicated by surgical site infection, MRSA bacteremia, and continued claudication presenting for fem - fem graft excision, wound exploration, and bilateral sartorial flap closure. He had a past medical history of hypertension, insulin dependent diabetes, coronary artery disease, and heart failure with reduced ejection fraction (40-45%). On arrival to the operating room from the ICU, standard ASA monitors were placed on the patient and preinduction a-line placed in the right radial artery. Following a routine induction and intubation, an 8.5 Fr introducer was placed for additional access. Maintenance was achieved using sevoflurane and precedex. The patient required phenylephrine drip throughout the case. Patient was heparinized for the angiogram and at the request of the vascular surgery team patient was reversed using protamine. He quickly developed hypotension (60s/40s), elevated peak airway pressures, and hypoxia. Hypotension was refractory to phenylephrine pushes and wheezing noted on lung exam prompting treatment of type 2 protamine reaction with total 40 mcg epinephrine and 250 mg solumedrol with resolution of symptoms. Case was continued and completed without issue and patient taken back to SICU intubated and on a phenylephrine drip. Patient was quickly weaned of pressors and extubated on POD1 and had a prolonged hospital course complicated by development of AKI and cutaneous vasculitis. Patient had PICC placed to continue 4 week treatment course of IV Vancomycin for MRSA bacteremia and discharged to SAR on POD 20.

Conclusion: Protamine sulfate is an alkaline polycationic protein comprised in two-thirds part by arginine subgroups. When exposed to negatively charged heparin, protamine binds and forms complexes that effectively neutralize anticoagulant activity. Protamine is also used to slow insulin absorption when used in intermediate and long acting insulin formulations. Those with a history of IDDM and previous exposure such as seen in the case of this patient are at an increased risk of developing a Type 2 protamine reaction. The high morbidity and mortality associated with protamine reactions warrant prompt recognition and aggressive supportive care.

References: Mortality and adverse events after protamine administration in patients undergoing cardiopulmonary bypass, Anesth Analg, 94 (2002), pp. 1402-1408 'Protamine and Protamine Reactions.' International Anesthesiology Clinics, vol. 42, no. 3, 2004

Blood Management

Blood Management - 1 58-year-old Woman Presenting for Open Abdominal Aorta Aneurysm Requiring Massive Transfusion in the Setting of Anti-K1 Antibodies

Ryan Dodge[1]

[1]*Massachusetts General Hospital, Boston, MA*

Introduction: The purpose of this case report is to describe the management of a patient requiring massive transfusion in the setting of Anti-K1 antibodies.

Methods: A 58-year-old woman with peripheral artery disease (PAD) and complicated interventional history presented to the emergency department with right groin pain and CT imaging concerning for enlarging aneurysm at the inferior aspect of her aortic endograft and new pseudoaneurysm. Her medical history was remarkable for early onset PAD of unknown etiology requiring initial revascularization with an aortobifemoral bypass 28 years prior to her current presentation at the age of 30. This was followed by a complicated revascularization history over the subsequent decades including multiple pseudoaneurysm repairs and graft infections, and two redo aortobifemoral bypass grafts procedures. Her medical history also included hypertension, hyperlipidemia, non-small cell lung carcinoma status post right lower lobectomy, right atrophic kidney, and gastritis. For this presentation, she was admitted from the ED to the surgical ICU on nicardipine and esmolol infusions with plan to proceed urgently to the operating room for redo open aortobifemoral bypass. Her type and screen on the day of surgery was noted to have minor antibodies and a note from the transfusion medicine team from a prior admission confirmed the presence of anti-K1 antibodies. In the OR, arterial and central access was obtained, and a rapid infuser was connected to her central line. The blood bank was instructed to prepare packed red blood cells (pRBCs), fresh frozen plasma (FFP), platelets, and cryoprecipitate for this patient. They blood bank confirmed the request and informed the OR team that there would be an approximately 45-minute lead time on preparation of blood product for this patient in the setting of anti-K1 antibodies. Throughout the case, the patient had an estimate blood loss of 25 L. There was constant communication between the OR anesthesia team and the blood bank as they worked in parallel to care for this patient with significant transfusion requirements. Overall, the patient received 6 L of cell saver autotransfusion, 21 units of pRBCs, 27 units of FFP, 4 'six packs' of random donor platelets, and one unit of cryoprecipitate in addition to albumin and crystalloid. Due to time constraints with cross-matching, uncrossed-matched blood was required and the patient received five units each of uncross-matched pRBCs and FFP. At the completion of the case, the patient was transported to the ICU intubated and sedated, hemodynamically stable not requiring any vasoactive medications.

Conclusion: This report highlights some of the challenges encountered in cases involving significant blood loss. Transfusion medicine was pioneered by anesthesiologists in the early 1900's, specifically by Dr. John Silas Lundy. He recommended the first 'transfusion level' for anemia (hemoglobin of 8-10 g/dL) in 1942 (1), and over the subsequent decades this threshold had been studied extensively in surgical populations. Data support a transfusion threshold of 7-8 g/dL to decrease morbidity and mortality among surgical patients (2). Antigens on the surface of red blood cells must be accounted for when transfusing blood products to reduce the risk of hemolytic reactions. Non-ABO blood group antigens - such as Kell, Duffy, and Lewis - are important to consider when selecting blood products. In patients that have circulating antibodies to these non-ABO blood group antigens, special care must be taken by blood banks to ensure the products delivered do not contain these antigens. However, patients receiving non-ABO blood group incompatible units in hemorrhage scenarios only rarely have clinically significant hemolytic reactions (3). The Kell blood group, which is composed of 25 separate antigens, is the third most immunogenic blood group following ABO and Rh (4). They can cause both hemolytic transfusion reactions (HTR) and hemolytic disease of the newborn (HDN). The anti-Kell antibodies are most commonly IgG (IgM is uncommon) and thus typically cause extravascular hemolysis if hemolysis does occur. However, some anti-Kell antibodies can cause severe HTR and HDN. In this case report, the anesthesiology providers utilized their expertise in transfusion medicine, cardiovascular physiology, and resource management to provide

effective care for a complicated patient in a massive transfusion scenario.

References: 1 Lundy JS; Anesthesia in Cases of Poor Surgical Risk. Anesthesiology 3, 603-607 (1942). 2 Mazer CD et al.; Restrictive or Liberal Red-Cell Transfusion for Cardiac Surgery. New Engl J Medicine 377, 2133-2144 (2017). 3 Yazer M et al.; Use of Uncrossmatched Erythrocytes in Emergency Bleeding Situations. Anesthesiology 128, 650-656 (2018). 4 Dean L. The Kell Blood Group. Blood Groups and Red Cell Antigens Chapter 8 (2005).

Cardiovascular Anesthesiology

Cardiovascular Anesthesiology - 1

Perforation of the innominate vein by a left sided single-lumen infusion catheter

Alec James[1], Zhen Deng[1], Satoshi Hanada[1]

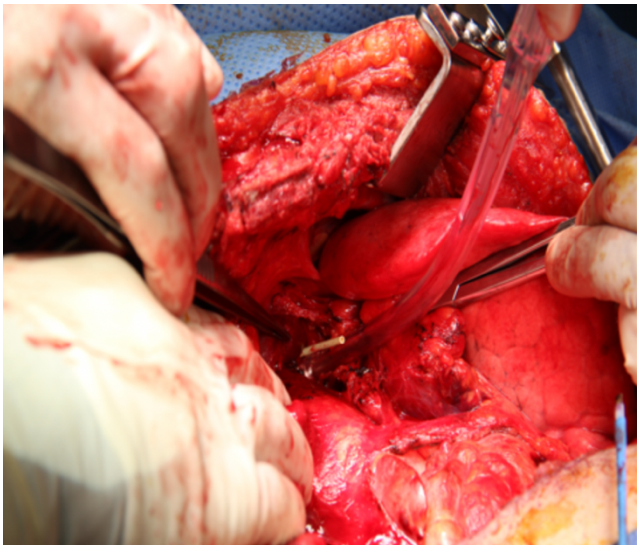
[1]University of Iowa, Iowa City, IA

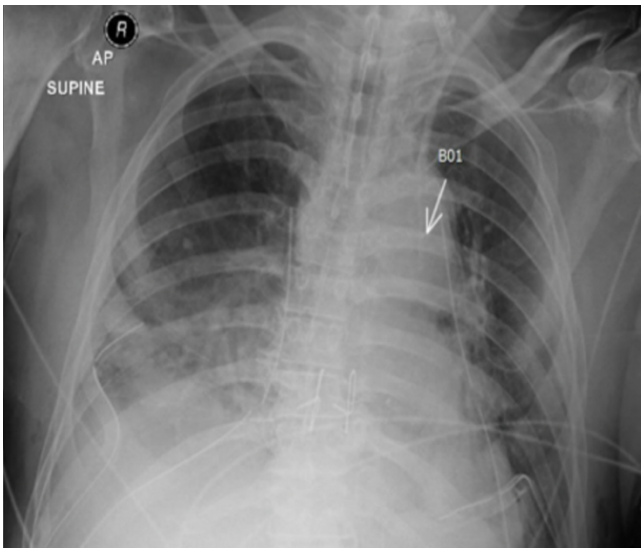
Introduction: While there have been multiple reports of vascular complications with hemodialysis catheter placement (1-9), there are few reports with a single-lumen infusion catheter (SLIC, Arrow International, Reading, PA, USA) placement.

Methods: A 60-year-old male with history of idiopathic pulmonary fibrosis underwent bilateral lung transplant. A sheath introducer (9 Fr multi-lumen access catheter [MAC] [Arrow International, Reading, PA, USA]) was placed via the left internal jugular vein (LIJV) in the operating room (OR), and then, a pulmonary artery catheter (PAC) was advanced through the MAC transducer port. The PAC placement was eventually abandoned after multiple failed attempts because there was increased resistance at approximately 17cm once the tip of PAC reached the length of the MAC transducer. Instead of a PAC, an 8-inch (20cm) SLIC was inserted via the MAC introducer port to monitor central venous pressure during lung transplant surgery. On postoperative day 3, a blood transfusion was administered via the LIJV MAC to treat anemia. At the time of transfusion, there was a sudden increase in sanguineous output from the left chest tube, and the patient was taken emergently to the OR for mediastinal exploration. Intraoperatively, bleeding subsided after cessation of all transfusions via the MAC, and surgical investigation revealed a clear perforation of the innominate vein with approximately 2 inches (5cm) of the SLIC protruding into the mediastinum (Image1). The SLIC was then removed by the anesthesia team, and the perforation was surgically repaired.

Conclusion: Although a SLIC has a smaller diameter, it is structurally sharper and firmer than a hemodialysis catheter, which could contribute to vascular complications. Furthermore, prior difficulty of PAC placement, especially when a PAC cannot be advanced beyond the tip of MAC, may indicate increased risk of vascular perforation as the tip may already be abutting the vascular wall. This may occur more frequently during the LIJV CVC approach because of the acutely angulated venous anatomy at the conjunction of the innominate vein and LIJV (1, 6). Although the manufacturer does not recommend the alteration of the SLIC, insertion of a modified SLIC (manually trimmed 2 inches (5cm)) could have prevented vascular perforation in this present case, since the tip of the trimmed SLIC would not extend the end of the MAC. This case also highlights the importance of thorough evaluation of post-operative imaging after CVC placement. In fact, prior to mediastinal exploration, the postoperative chest radiograph clearly demonstrated the SLIC not following the expected course of the innominate vein, indicating it had already perforated into the mediastinum (Image2). However, the radiographic findings were ignored, and blood transfusion via the MAC allowed the transfused blood to empty into the mediastinum.

References: Endovascular repair of an iatrogenic superior vena caval injury. 2007;46(3):569-71 Successful treatment of superior vena cava rupture with placement of a covered stent. 2011;34(3):667-71 Complications and failures of subclavian-vein catheterization. 1994;331(26):1735-8 Dilator-associated complications of central vein catheter insertion: possible mechanisms of injury and suggestions for prevention. 1996;10(5):634-7 Pneumothorax as a complication of central venous catheter insertion. 2015;3(3) The perforation of the superior vena cava secondary to the left subclavian dialysis catheter. 2017;51(2):95-7 Bedside ultrasonic detection of massive hemothorax due to superior vena cava perforation after hemodialysis catheter insertion. 2009;47(2):95-8 Management of venous perforation during central venous catheterization in hemodialysis patients. 2019;98(25) Treatment of a central venous perforation caused by dialysis intubation using coils and cyanoacrylate glue. 2019;18(4):2979-83




R
AP
SUPINE
B01

Cardiovascular Anesthesiology - 2

Cor Triatriatum Dexter In A Teenager

Ryker Saunders[1], Dien Vu[1]

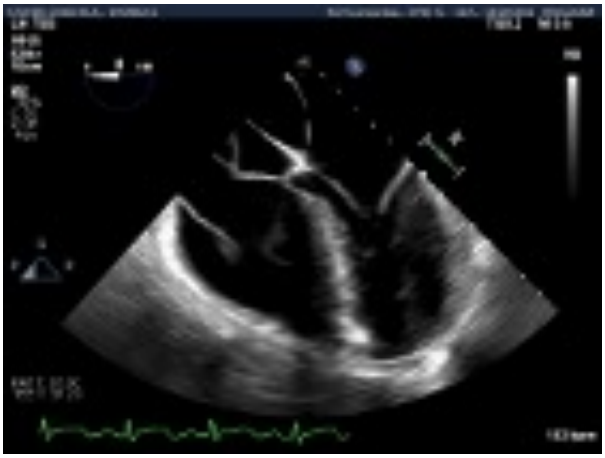
[1]University of Kentucky, Lexington, KY

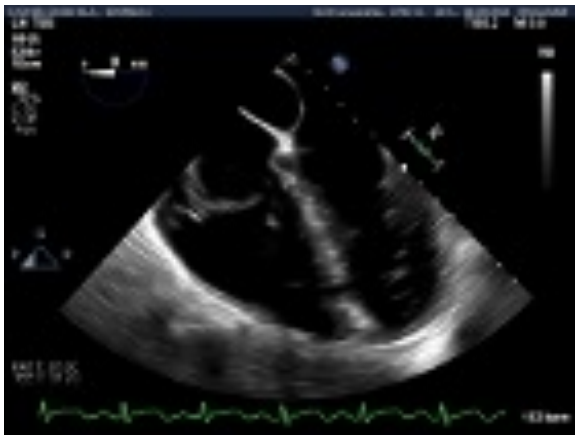
Introduction: Cor triatriatum dexter (CTD) is one of the rarest of congenital heart defects seen in childhood with overall incidence estimated to be approximately 0.1% of all congenital heart defects3. CTD is caused by a preserved right valve of the sinus venosus causing a two-chambered right atrium, separated by a thin membrane1,2. Functionality of this membrane in utero helps to divert blood from the sinoatrial junction across the foramen ovale to the left atrium, however this tissue usually regresses around the 12th week of gestation. Without complete regression, this tissue can widely range in size from reticulum to a significant layer of tissue potentially causing an obstruction of flow into the RV2.

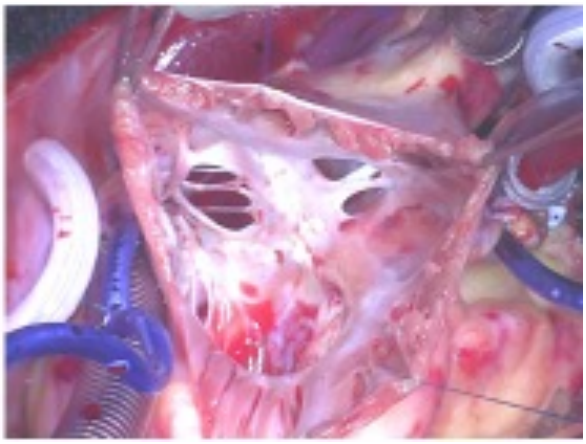
Methods: We present a case of a 17 year-old male with cor triatriatum dexter (CTD) and atrial septal defect scheduled for repair and TV ring replacement. His medical history includes hypertension, obesity, and obstructive sleep apnea not requiring BiPAP. His only scheduled medication was lisinopril 10mg daily. He was being followed by his cardiologist for a known VSD that underwent spontaneous closure, however it was discovered on serial TTEs that he was continuing to have tricuspid valve prolapse with a tethered septal leaflet and moderate tricuspid regurgitation. TTE was limited due to his habitus yielding limited viewing windows. Subsequent exercise testing demonstrated easy fatigue and poor tolerance with a baseline oxygen saturation of 93% and exertional desaturation to 88%. Transesophageal echo was performed and demonstrated CTD with right to left shunting across a small ASD. TEE noted aneurysmal tissue prolapsing in and out of the tricuspid valve orifice and extending to the TV annulus. Accompanying the CTD was a secundum ASD with very significant right to left atrial level shunting. A dilated TV annulus of 4.2cm with poor coaptation due to the tethered septal leaflet was noted, along with severe TR. Resection of the membrane and repair of the CTD and ASD were successful, along with a TV ring reenforcement.

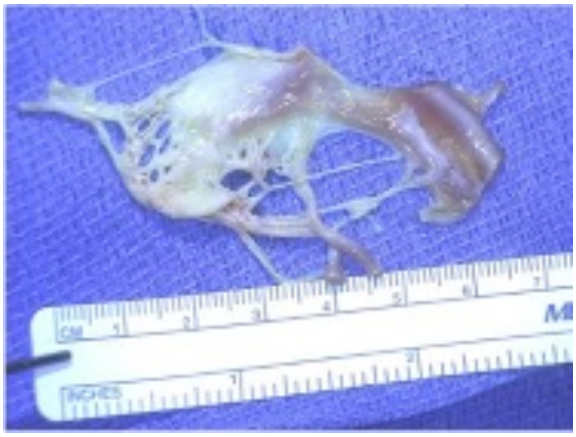
Conclusion: CTD is formed by an embryologic anomaly where the embryologic right valve of the sinus venosus that shunted blood to the foramen ovale does not regress around the 12th week of gestation,Äã - CTD can present as an isolated anomaly or concurrently with other malformations like Ebstein anomaly, ASD, pulmonary artery stenosis or atresia,Äã - The degree of cyanosis in CTD can be associated with the degree of RV inflow obstruction or other congenital defects such as ASD,Äã - Most CTD is diagnosed postmortem, as most people remain asymptomatic, however those that are symptomatic are usually diagnosed by echo, angiography, or MRI.

References: 1. Cor triatriatum dexter: A rare cause of childhood cyanosis. Ann Pediatr Cardiol. 2012;5(1):92-94.2. Cor triatriatum, pulmonary vein stenosis, atresia of the common pulmonary vein. Pediatric cardiac surgery. 2nd ed. St Louis: Mosby-Year Book; 1994. pp. 503–4.3. Cor triatriatum: preoperative diagnosis and successful surgical correction in a ten-week-old infant.1973 Jun; 63(6):1027-8.









Cardiovascular Anesthesiology - 3 Acute Right Atrial Wall Perforation by Atrial Lead After Placement of Implantable Cardiac Defibrillator.

Armel Ngeugaum Silenou[1], Mohammad Sadat[2]

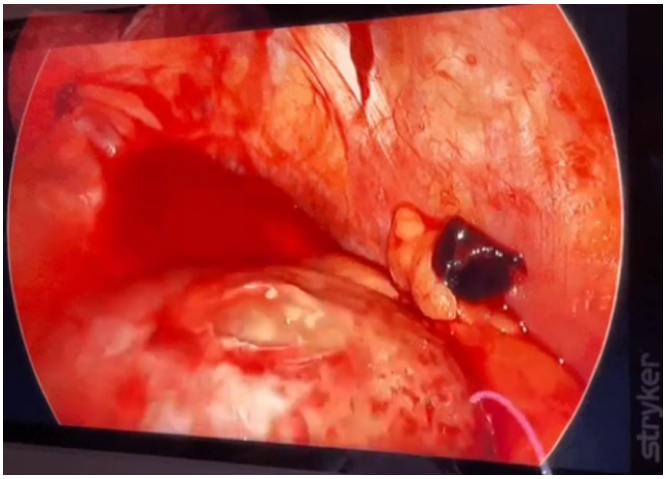
[1]Maimonides Medical Center, Brooklyn, NY, [2]Columbia University Medical Center, New York, United States of America

Introduction: Automatic Implantable Cardiac Defibrillators (AICDs) and pacemakers have been proven to be effective for the management of patients with arrhythmias, congestive heart failure, and those at risk of sudden cardiac death. However, these devices also have potential life-threatening complications. Such complications include pneumothorax, lead malposition, infections, thrombotic events, or cardiac perforation (1). The reported rate of occurrence of pacemaker placement leading to perforation is 0.1–3% (2). Perforations that occur within twenty-four hours after implantation are labeled as acute; those occurring within one month after implantation are subacute while perforations which occur after one month are chronic (3). Pacemaker perforations may occur through the walls of the large veins, atria, or ventricles. We describe a case of acute right atrial wall perforation that occurred after the placement of a biventricular implantable defibrillator. The patient presented with a right hemothorax and hemorrhagic shock.

Methods: A 73-year-old woman presented to the emergency department twelve hours after placement of a biventricular implantable cardiac defibrillator complaining of dyspnea and right pleuritic chest pain. The patient's past medical history included hypertension, hyperlipidemia, diabetes mellitus type 2, chronic kidney disease, chronic anemia, and non-ischemic cardiomyopathy with an ejection fraction of 21%. Upon initial presentation, the patient was hypotensive and hypoxic on room air. Chest X-ray showed no signs of pneumothorax. Chest computer tomography (CT) scan revealed a large right hemothorax and penetration of the right atrial lead of the defibrillator through the wall of the right atrium. AICD interrogation showed normal function. Transthoracic echocardiogram (TTE) showed severely decreased left ventricular function and a small pericardial effusion which was unchanged from previous studies. A right sided chest tube was placed in the emergency room and the patient received vigorous fluid resuscitation with crystalloids and transfusion of blood products. After the patient was hemodynamically stabilized, she was urgently taken to the operating room for video assisted thoracoscopy surgery and drainage of the hemothorax. Intraoperatively it was confirmed that the atrial lead had protruded through the right atrium and was likely the cause of the bleeding. The perforated area had already thrombosed and there was no active bleeding (see figure 1, which shows the thrombosed area on right atrium after resolution of bleed). The atrial lead was left in place by the surgical team, as the bleeding had already resolved. Intraoperative transesophageal echocardiogram (TEE) showed no signs of pericardial effusion. The patient remained hemodynamically stable intraoperatively and had an uneventful hospital course postoperatively.

Conclusion: This case describes a complication rarely seen after placement of ICDs and pacemakers. Atrial perforation is more common than ventricular perforation as the right atrial wall is very thin, averaging only 2 mm in thickness, whilst the right ventricular wall is twice as thick (1). Normal function on device interrogation does not rule out perforation as evidenced in this patient. Chest CT scan is the diagnostic gold standard for the diagnosis of cardiac lead perforation (3).

References: 1) “A Life Threatening Perforation of Pacemaker Atrial Lead through the Right Atrium and the Right Lung”. J Cardiol Curr Res 2(6): 00079, 2015 2) “Cardiac pacing systems and implantable cardiac defibrillators: a radiological perspective of equipment, anatomy and complications,” Clinical Radiology, vol. 59, no. 8, pp. 699–708, 2004 3) "Subacute Right Ventricle Perforation by Pacemaker Lead Presenting with Left Hemothorax and Shock", Case Reports in Cardiology, vol. 2015, Article ID 983930, 4 pages.


stryker

Cardiovascular Anesthesiology - 4

Difficulty with weaning off cardiopulmonary bypass in a case of VSD

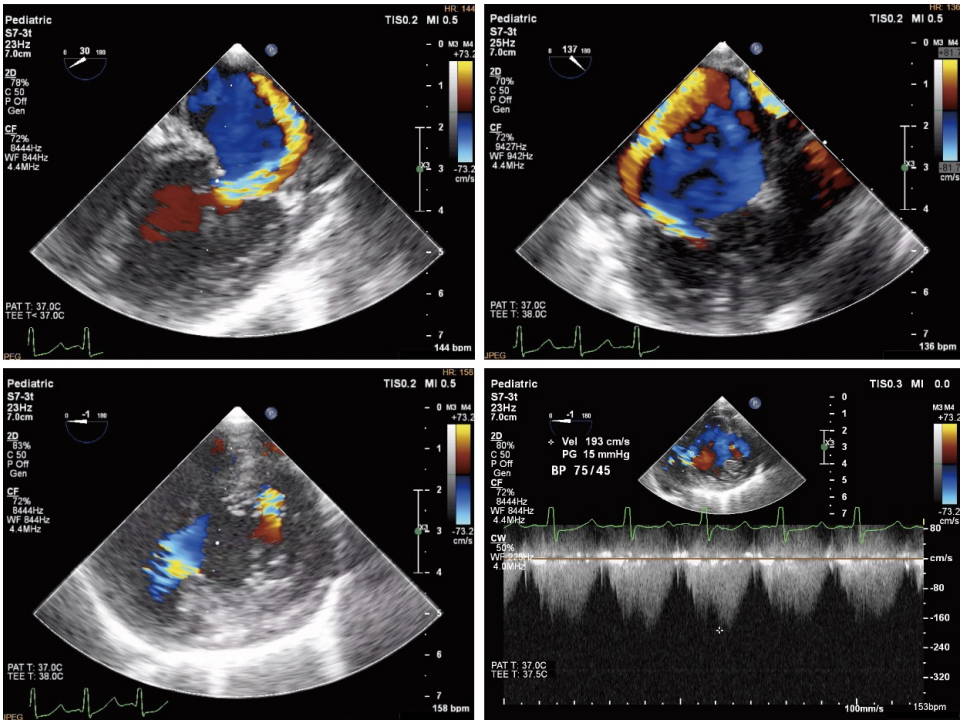
Koichi Akiyama[1], Hirokazu Muneishi[2], Yu Hirase[3], Tomoya Sato[4], Yurie Obata[3], Risa Kawaguchi[4], Teiji Sawa[5]

[1]Yodogawa Christian Hospital, Osaka, Osaka, [2]University of Fukui, Yoshidagun, Fukui, [3]Yodogawa Christian Hospital, Osaka city, Japan, [4]Yodogawa Christian Hospital, Osaka, Japan, [5]Kyoto Prefectural University of medicine, Kyoto city, Kyoto prefecture

Introduction: A 7month-old infant was referred to our hospital due to cardiac murmur and diagnosed with ventricular septal defect (VSD) and mitral regurgitation. She was scheduled to undergo VSD repair and mitral valve repair.

Methods: Intraoperative transesophageal echocardiography revealed large VSD (10 mm in diameter) that was located at perimembranous outlet. Color Doppler images showed severe mitral regurgitation directing from posterior commissure to anterolateral wall and from A3 to posterior wall. The etiology was assessed as atrial functional mitral regurgitation due to the enlarged left atrium. Systolic pulmonary artery pressure was estimated 60 mmHg derived from the pressure gradient of VSD flow (jet velocity: 1.93 m/s, systemic blood pressure 75/45 mmHg). The operational procedure was performed without any trouble (VSD patch closure, mitral valve repair, tricuspid annuloplasty). Dobutamine (5 mcg/kg/min), nitroglycerin (1.6 mcg/kg/mi), and olprinone (0.4 mcg/kg/mi) were started to be administered 15minutes before aortic declamping.

Conclusion: Cardiac wall motion was severely impaired even 20 minutes passed after aortic declamping, which was considered to be due to afterload increase owing to the eliminating leaks (VSD, mitral regurgitation). Therefore, we waited for the situation to improve further 15 minutes under cardiopulmonary bypass and started to administer adrenaline (0.07 mcg/kg/min). Wall motion improved to the form of diffuse hypokinesis, and we could manage to wean off the cardiopulmonary bypass. Blocking preoperative left ventricular escape route for both large VSD and mitral regurgitation could induce impaired wall motion even after a short aortic clamping time.



Cardiovascular Anesthesiology - 5 Use of Transesophageal Echocardiography to Diagnose an Uncommon Cause of Sudden Cardiovascular Collapse in the Prone Position

Samuel J Aldous[1], Scott McQuilkin[2], Emmett Whitaker[3], Dani Bliss[2]

[1]Larner College of Medicine at the University of Vermont, Burlington, VT, [2]University of Vermont Medical Center, Burlington, VT, [3]University of Vermont, Burlington, VT

Introduction: A surgery that accesses posterior anatomy often requires prone positioning, causing many physiologic changes that can include hemodynamic compromise [1]. In rare situations, hemodynamic collapse or cardiac arrest can occur. Here, we present a case in which transesophageal echocardiography (TEE) was used to diagnose an uncommon cause of severe hypotension and guide further management.

Methods: A 54-year-old male with known right-sided C7-8 radiculopathy presented to the emergency department with worsening neck pain and was admitted for an open posterior laminoforaminotomy and partial facetectomy. His initial presentation and hospital course prior to surgery was complicated by elevated blood pressures that were relieved with pain control and hydralazine as needed. The last hydralazine dose was eight hours before surgery. The patient denied having any cardiovascular history and scored more than 4 metabolic equivalents. In the operating room, general anesthesia was provided with sevoflurane, fentanyl, rocuronium, ketamine, and propofol. After intubation and prone positioning on the Jackson table, it suddenly became difficult to obtain a blood pressure in any extremity and oxygen saturation began to decrease. The few blood pressures recorded indicated profound hypotension that was unresponsive to phenylephrine boluses. The patient was quickly supinated and a normal blood pressure was easily obtained; an arterial line was then inserted. When prone positioning was repeated, rapid hypotension recurred and necessitated immediate resolution with supination (Figure 1). An intraoperative TEE was then performed. No abnormalities were noted while supine (Figure 2), but prone positioning revealed right ventricular (RV) external compression and tamponade physiology without evidence of pericardial/pleural effusions, masses, or increased intrathoracic pressures (Figure 3). With the TEE probe still in place, the support struts on the surgical bed were repositioned away from the mid/low sternum, relieving the tamponade physiology (Figure 4). Continued TEE monitoring for 15 minutes did not show any signs of hemodynamic collapse, and the procedure progressed without complication. The patient was discharged 24 hours post procedure without further sequelae.

Conclusion: Prone positioning for surgery can cause unique physiologic changes that may risk hemodynamic stability. In this case, prone positioning was intolerable due to direct pressure from the thoracic brace on the sternum. The use of TEE identified RV compression as the cause of hypotension while prone and helped determine positional changes that alleviated hemodynamic instability, allowing for continuation of the procedure. This case highlights the value of point-of-care echocardiography as a diagnostic tool, even in acute and life-threatening circumstances.

References: [1] The prone position during surgery and its complications: a systematic review and evidence-based guidelines. 100(2):292-303. 2015.

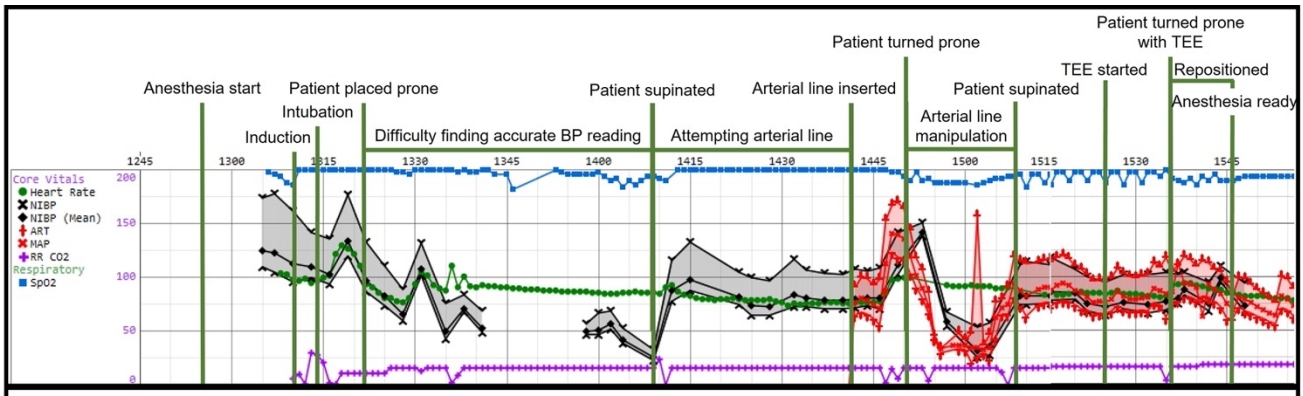


Figure 1: Anesthesia timeline showing periods of rapid hypotension with prone positioning

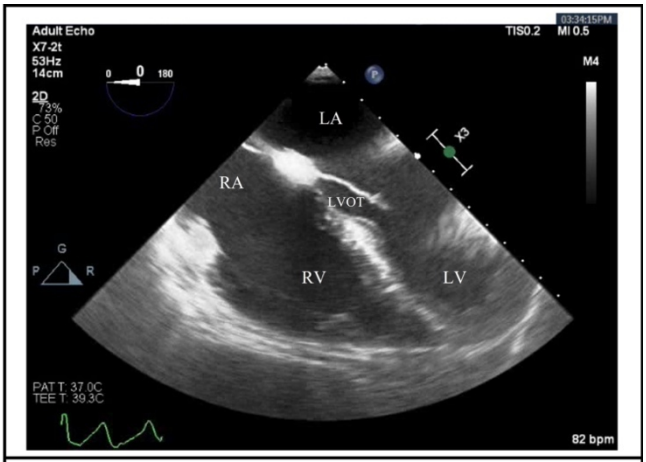


Figure 2: Mid-esophageal five chamber view showing RV chamber volume in supine position

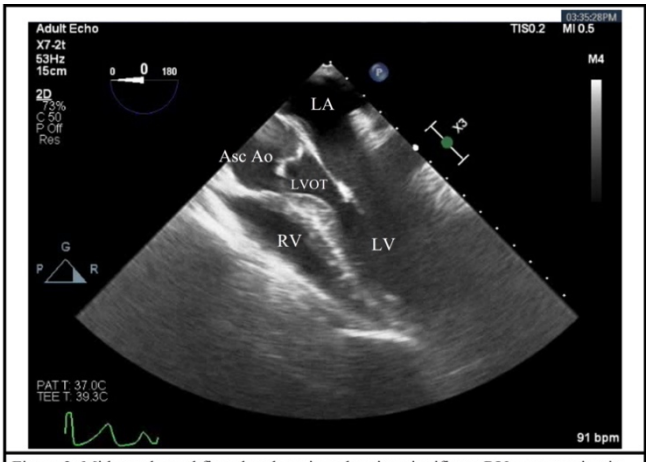


Figure 3: Mid-esophageal five chamber view showing significant RV compression in prone position

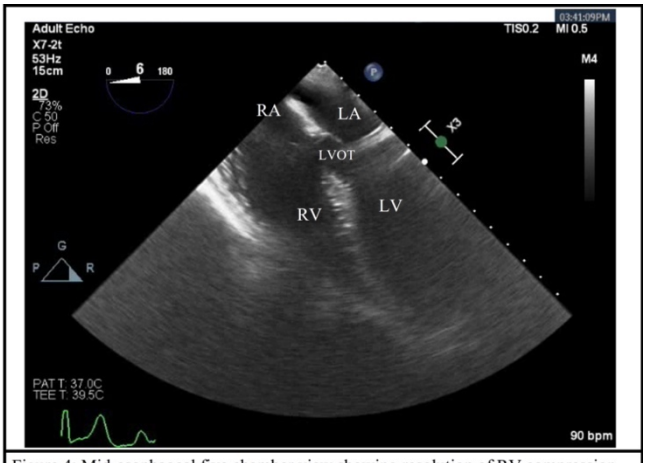


Figure 4: Mid-esophageal five chamber view showing resolution of RV compression with prone position adjustments

Cardiovascular Anesthesiology - 6

Anesthetic Management of a Patient with a Massive Mediastinal Intrapericardial Tumor

Emiley Tou[1], Jenessa Job[1], Manxu Zhao[2]

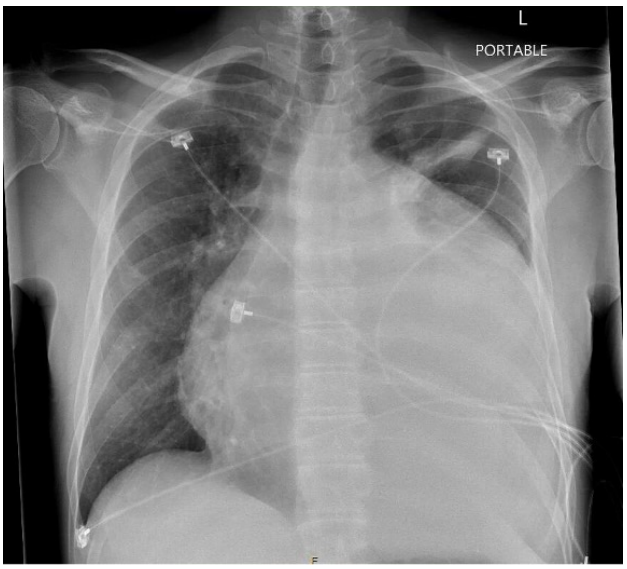
[1]Cedars-Sinai Medical Center, Los Angeles, United States of America, [2]Cedars Sinai, Los Angeles, CA

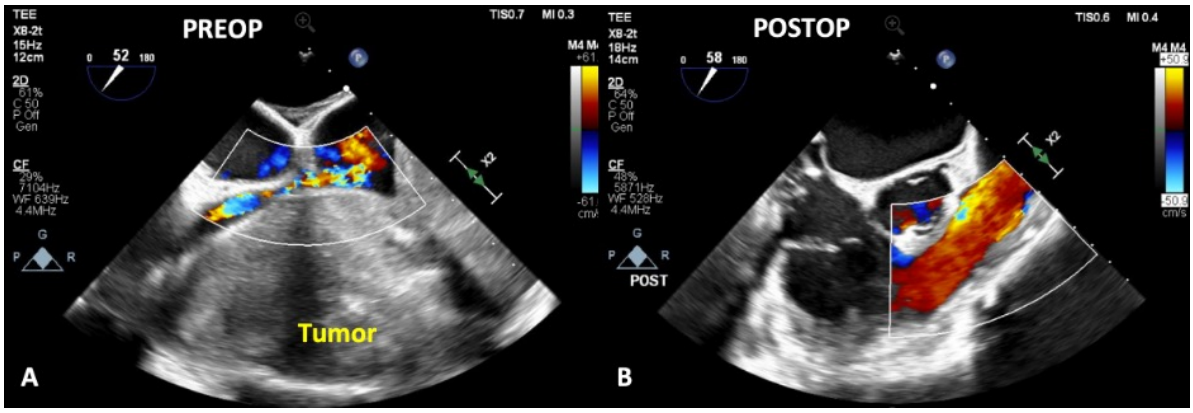
Introduction: There is scarce literature describing management of adults with massive mediastinal mass due to their rarity. This case report describes the perioperative management of a patient with a mediastinal intrapericardial mass.

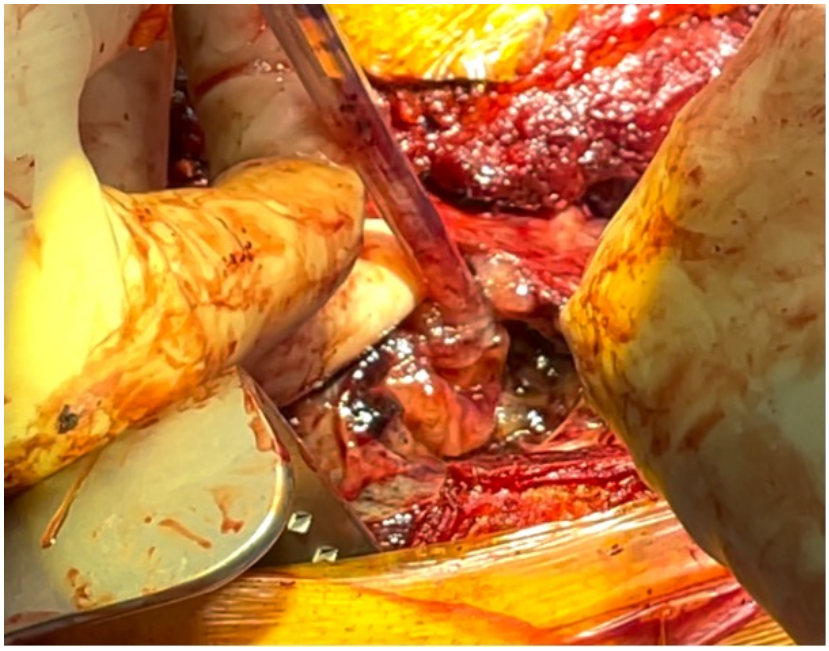
Methods: A 45-year-old male with history of left hip myxoid liposarcoma status post resection and radiation presented for tumor debulking of a 20 x 15cm mediastinal liposarcoma compressing the main pulmonary artery and left lung; and deviating heart, trachea, and esophagus to the right (Fig.1). Patient endorsed symptoms of supine chest discomfort requiring 2-3 pillows at night, orthopnea that improved in left lateral position, and 30lbs weight loss over a 6-month period. After discussion between the anesthesiologists and surgeons, pre-induction radial arterial line and wires in the femoral artery and vein for potential cardiopulmonary bypass (CPB) were placed with perfusionist on standby. Patient was intubated with a single lumen ETT after induction with propofol, ketamine, and rocuronium in a sitting position. Endobronchial blocker and central lines were placed. Intraoperative TEE confirmed the mass compressing both ventricles and the right ventricular outflow tract (RVOT) (Fig. 2A). Intraoperatively, the mass was noted to be intrapericardial with encasement of the LAD and SVC (Fig.3). Dissection and manipulation of the adherent mass resulted in profound hypotension and cardiac arrest requiring cardiac massage and prompt initiation of CPB. Patient required inotropic support and multiple blood products. Only 50% of the mass was able to be resected due to the extensive tumor invasion into the myocardium and vasculature. Post-operative TEE revealed significant improvement of the RVOT (Fig. 2B) and improved biventricular function. Patient successfully came off CPB and was transferred to the ICU intubated and sedated. Later that day, patient was extubated and discharged home on post-op day 9.

Conclusion: Patients with mediastinal tumors pose a challenge to anesthesiologists especially if the mass is associated with mediastinal mass syndrome (MMS), characterized by respiratory and hemodynamic decompensation from mediastinal structure compression.1 On induction and during surgery, these patients can have sudden decompensation so it is recommended to have the surgical team perform femorofemoral CPB cannulation or wire placement prior to induction.2 Preoperative evaluation of symptoms and adequate positioning of patient prior to induction can significantly offload the compression of the mediastinal structures. Additionally, it is important to have all equipment and personnel available in the operating room. We had perfusionists, surgeons, rapid infuser, and blood products in the room prior to induction. Overall, having a multidisciplinary discussion and plan preoperatively is essential for safe, adequate management of these patients.

References: 1. Li, Wilson W L et al. Management of large mediastinal masses: surgical and anesthesiological considerations. Journal of thoracic disease. 2016; 8(3): E175-84. 2. Rath, Lindsey et al. Anterior mediastinal mass biopsy and resection: anesthetic techniques and perioperative concerns. Seminars in Cardiothoracic and Vascular Anesthesia. 2012; 16 (4): 235-242.




PREOP
POSTOP
Tumor
A
B



Cardiovascular Anesthesiology - 7

Anesthetic Management of a Combined Radical Nephrectomy and Intracardiac Thrombectomy

Jeffrey D Mimbs[1], Dev Vyas[2], Zach Arnett[3], Katherine Cox[1]

[1]Tulane University School of Medicine, New Orleans, LA, [2]Tulane University School of Medicine, NEW ORLEANS, LA, [3]Tulane Medical Center, New Orleans, LA

Introduction: A patient presented for combined radical nephrectomy and cardiac thrombectomy with a PMH of HTN, Type II DM, right renal mass with extensive extension through the IVC and into the cavoatrial junction. TEE findings were significant for an EF of 55-60%, mild tricuspid regurgitation, no diastolic dysfunction, and significant large mass that originates from the IVC, obliterating the right atrium cavity and protruding through the tricuspid valve. Anesthetic Technique: General endotracheal anesthesia technique was initiated. The patient was given 4 mg midazolam and transported to the OR. Standard ASA monitors were applied in supine position. The patient was pre-oxygenated. Induction through large bore peripheral IV access using 150 mcg fentanyl, 80 mg lidocaine, 50 mg propofol, 8 mg etomidate, and 60 mg rocuronium. The patient was intubated with video laryngoscopy using a 7.5 cuffed ETT. Sevoflurane was started (2% et) while further access was established. IV access x 2 was obtained. A right radial arterial line and right internal jugular 9 Fr double lumen central venous catheter was placed. Given the location of the thrombus, a pulmonary arterial catheter was contraindicated. Following the placement of invasive lines and monitors, a TEE probe was placed. Sevoflurane was discontinued and isoflurane was initiated (0.7% et).

Methods: Intraoperative Course: Isoflurane was maintained throughout the case. Muscle relaxation was monitored and rocuronium was redosed as needed. Upon TEE probe insertion, a significant thrombus was evident in midesophageal long axis and bicaval views in the right atrium and could be visualized touching the tricuspid valve as seen in pre-operative TTE. The urology team began radical dissection of the right kidney. Significant blood loss was encountered until the renal artery could be clamped. The hepatobiliary surgeon dissected and reflected segments of the liver to gain adequate exposure of the IVC. The aortic cannula was placed and IV heparin was administered. The IVC distal to the renal veins was cannulated, as was the SVC. The cannulae were secured, primed, and the patient was placed on cardiopulmonary bypass for a total of 18 minutes. Cardioplegia solution was not administered. A right atriotomy incision was made, as well as a longitudinal incision of the IVC allowing for mobilization and removal of the thrombus from the renal vein, IVC and right atrium in two pieces. The IVC and atrial incisions were closed. The patient was weaned from cardiopulmonary bypass. Infusions of vasopressin (0.04 U/min; total 0.1 units) and norepinephrine (0.07 mcg/kg/min; total 0.14 mg) were given as needed to achieve MAP goals of greater than or equal to 65 mmHg based on invasive arterial blood pressure monitoring. The patient received 10 U pRBCs, 2000 mL albumin 5%, 8 U FFP, 2 U platelets, and 1500 mL cell saver. Estimated blood loss was approximately 3 L. Postoperative Course: The patient was transferred to the SICU intubated with continuous monitoring and without vasopressor requirement. The patient was extubated on POD1, stepped down to med/surg floor POD2, and discharged on POD5.

Conclusion: Renal cell carcinoma invading the IVC is a surgery in which complexity varies with how far the tumor has invaded.[1] Subhepatic spread has been documented to have a 35% 5-year-survival, 18% for intrahepatic, and 0% for those masses with atrial invasion, whereas other studies have shown the extent of IVC spread does not significantly impact survival.[2] [3] [4] However, regardless of these documented studies, the invasion does significantly impact surgical and anesthetic technique. With atrial or valvular involvement, cardiopulmonary bypass is warranted.[5] This could have been done with hypothermic circulatory arrest, but cardiopulmonary bypass was used instead due to short estimated length of use.[6] [7]

While uncommon, atrial thrombus formation in the setting of IVC invasion of renal cell carcinoma requires unique surgical and anesthetic considerations. It is likely multiple surgical teams will need to work in tandem when the extent of IVC involvement is as extensive as this patient's. These patients are likely to

be high risk surgical candidates, and though they may have extensive disease involving multiple organ systems, it is indeed possible to keep a patient stable through vigilance of surgical technique and strong communication from all teams involved.[9]

References: 1) Renal Cell Carcinoma with IVC Thrombi; Current Concepts and Future Perspectives. Clin Med Oncol. 2008;2:247-256. 2) Vena caval involvement by renal cell carcinoma. Surgical resection provides meaningful long-term survival. Ann Surg. 1989;210:387-94. 3) Long term results of renal cell cancer with extension into IVC. J Urol. 1987;137:21-4. 4) Surgical management and prognosis of renal cell carcinoma invading the vena cava. J Urol. 1991;145:20-3. 5) Resection of the abdominal IVC for complicated renal cell carcinoma with tumor thrombus. BJU Int. 2005;96:815-8. 6) Experience with cardiopulmonary bypass and deep hypothermic circulatory arrest in the management of retroperitoneal tumors with large vena caval thrombi. Ann Surg. 1990;212:472-7. 7) Long-term outcomes of the surgical management of renal cell carcinoma. World J Urol. 2006;24:255-66. 8) Renal cell carcinoma with infrarenal vena caval tumor thrombus. Urology. 1999;54:561-5.

Cardiovascular Anesthesiology - 8

Anesthetic Management: Leaking Mycotic Thoracic Aortic Aneurysm Impinging on Trachea

Brandon Duffin[1], Yong G Peng[1]

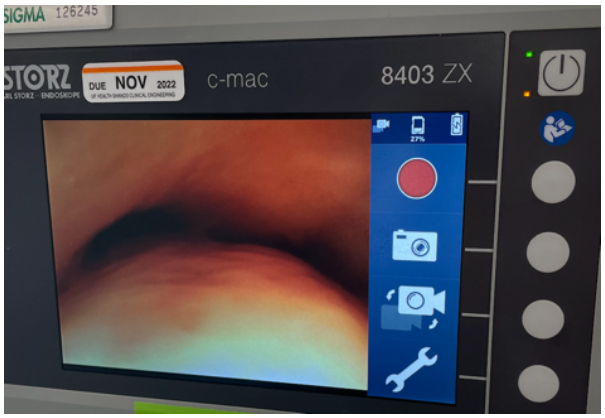
[1]University of Florida, Gainesville, FL

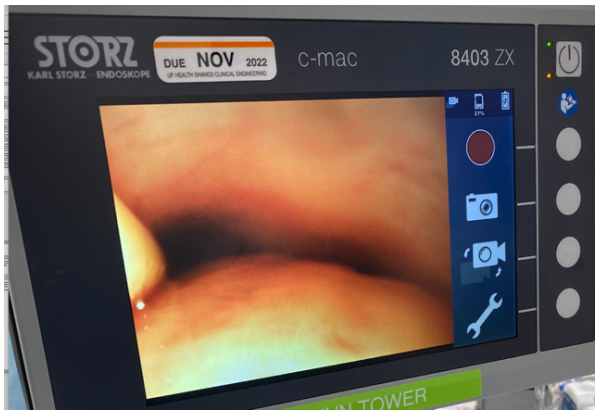
Introduction: Mycotic aneurysm is a life threatening disorder with an infectious etiology that manifests as variable symptoms (1). It confers a high risk of morbidity and mortality if a contained ruptured mycotic aneurysm is not promptly surgically repaired. The mass effect from a thoracic aortic aneurysm can compromise airway, pulmonary vasculature and cardiac function (2). We present a case of a mycotic thoracic aortic aneurysm with an imminent leak impinging on the trachea and creating a significant challenge for one lung isolation.

Methods: A 77-year-old male with history of hypertension, atrial fibrillation on Apixaban, who had colon resection surgery for cancer one month ago. He presented to the emergency department following a fall. CT exam revealed an extremely large air pocket filled descending thoracic aorta mycotic aneurysm. The irregular surface of the aortic intimal layer in conjunction with an episode of hypotension, lead to the suspicion of a leaking mycotic aneurysm. There is also concern that the contained rupture of the mycotic aneurysm compressed the trachea, possibly forming an aortoesophageal fistula. The decision was made to proceed with immediate surgical intervention. After uneventful induction, a single lumen endotracheal tube was placed. Fiberoptic bronchoscopy revealed a significant narrowing of the distal trachea. Posterior tracheal compression from the aneurysm made it almost impossible to visualize the carina. Bronchial blocker in lieu of a double lumen tube for lung isolation was selected. After several attempts and some difficulty, a bronchial blocker was placed into the left main bronchus, confirmed with fiberoptic bronchoscopy. TEE exam revealed LVEF 45%, minimal valvular pathologies and large mycotic thoracic aortic aneurysm with a maximal diameter of 6.3 cm. Surgical operation required partial femoral-femoral bypass support while the mycotic aneurysm was resected and replaced with a Dacron graft. Upon closer surgical examination, no aortoesophageal fistula was appreciated. After separation from cardiopulmonary bypass, point of care Quantra testing suggested tissue bleeding was due to coagulation factor deficiency. Hemostasis was achieved with administration of platelets, cryoprecipitate and 50 mcg/kg of prothrombin complex concentrate. The patient was transferred to the ICU with minimal vasoactive infusions and extubated post-operative day one.

Conclusion: Although thoracic aorta mycotic aneurysm is rare, the nature of disease process may exclude endovascular approaches, thus requiring open surgical repair (3). Mass effect from the aneurysm compressed the airway creating significant challenge on the optimal approach for lung isolation. Despite concern for an aortoesophageal fistula, TEE was placed per surgeon's request and provided valuable information for intraoperative management. Point of care testing with Quantra provided quick and reliable guidance to replace coagulation factors to achieve hemostasis.

References: 1) Surgical innovation 2019, 26:260-264 2) American Journal of Emergency Medicine 2021, 260e1-260e2 3) Journal of Cardiothoracic and Vascular Anesthesia 2018, 32:550-557


SIGMA 126245
STORZ
DUE NOV 2022
c-mac
8403 ZX


STORZ
KARL STORZ - ENDOSKOPE
DUE NOV 2022
c-mac
8403 ZX

Cardiovascular Anesthesiology - 9

Perioperative management of intravenous and intracardiac leiomyomatosis: a case report

Miguel E Perez-Viloria[1], Marco A Castillo-Rodriguez[1], Nazir Noor[2], Joshua Durbach[1], Howard S Goldman[1]

[1]Mount Sinai Medical Center of Florida, Miami Beach, FL, [2]Moun Sinai Medical Center of Florida, Miami Beach, FL

Introduction: Leiomyomas are one of the most common gynecological benign neoplasias with estimated incidence of 5.4% to 77% of women of reproductive age. They are characterized as benign smooth muscle neoplasms inside the uterus1. Leiomyomas beyond the uterus are subclassified as intravenous leiomyomatosis (IVL), benign metastasizing leiomyomas, diffuse peritoneal leiomyomatosis, retroperitoneal leiomyomas and parasitic leiomyomas2. Intravenous leiomyomatosis with right intracardiac extension is rare. Surgical treatment of the tumor is still controversial because of the high postoperative risk of morbidity and mortality3. In this case report we present a complicated case of intravenous and intracardiac leiomyomatosis (ICL) diagnosed incidentally during a hysterectomy and managed in a second surgery as a single stage procedure with thoracotomy, laparotomy and excision of tumor from the inferior vena cava (IVC), right atrium (RA) and right ventricle (RV).

Methods: A 43-year-old female with a history of fibromas and abnormal uterine bleeding underwent robotic hysterectomy, bilateral salpingectomy, and right oophorectomy. During the procedure the patient was found to have a retroperitoneal lesion extending intravascularly into the inferior vena cava. Upon further questioning, the patient also referred to episodes of shortness of breath, back pain, dizziness, and an episode of syncope. Computer Tomography scan demonstrated a retroperitoneal solid mass extending into the inferior vena cava and further into the right atrium and ventricle (Figure 1). Transesophageal echocardiogram (TEE) showed normal ejection fraction of 64%, an echodense homogeneous mass extending from the visualized portions of the IVC and occupying the near entirety of the right atrium, with extension across the tricuspid valve into the proximal right ventricular infundibulum. A narrow channel of blood flow around the lateral wall of the RA was propagating into the RV. Both right heart chambers were dilated, and the RV systolic function was impaired. The patient was then referred to Cardiothoracic, Vascular, and Gyn-oncology surgery teams for a sternotomy with cardiopulmonary bypass and resection of the intra-hepatic inferior vena cava, right atrial and right ventricular tumor, tricuspid valve repair with annuloplasty and left oophorectomy. A detailed preoperative review of the available images, such as CT scan and TEE, was done by the anesthesia team and discussed with the surgery team to determine the vascular access needed for the case, to prevent complications from these procedures such as embolization of fragments of the tumor. Pathology demonstrated vascularized myxoid fibrous tissue, cardiac muscle and fat which is leiomyomatosis (Figures 2,3). Patient was discharged home after 8 days.

Conclusion: Intravenous leiomyomatosis, a rare disease that is histologically benign but clinically aggressive, is characterized by the intraluminal growth of leiomyomas in intrauterine and systemic veins[3]. IVL and ICL are benign intravascular proliferations of smooth muscle cells arising from the uterine vein wall or the myometrium. The tumor has the potential to grow inside the veins of broad ligament, extending to the IVC through different routes, reaching up to the right heart chambers and occasionally the pulmonary artery[4]. An adequate diagnosis should provide detailed information about the tumor localization, extravascular and intravascular diameters, site of vascular entry, and patency of iliac and femoral veins. Accurate preoperative assessment of the tumor size and different routes of extension by a multidisciplinary medical team is essential to enable successful complete tumor excision[3]. Successful clinical management is dependent on total surgical excision, which may necessarily include cardiotomy. Optimum and safest strategy is a single or 2-stage procedure with an abdominal and thoracic approach. The long-term prognosis is good because the growths are hormonally responsive.[2]

References: 1. An International Journal of Obstetrics and Gynaecology. 124 (10), 1501-1512, 2017 2. Radiographics. 28 (7), 1931-1948, 2008 3. Annals of Thoracic Surgery. 89. 1641-3. 2010 4. Journal of Thoracic and Cardiovascular Surgery. 130 (6), 1724-1726, 2005 5. Gynecology Oncology. 89, 175-80, 2003

Figure 1. CT scan

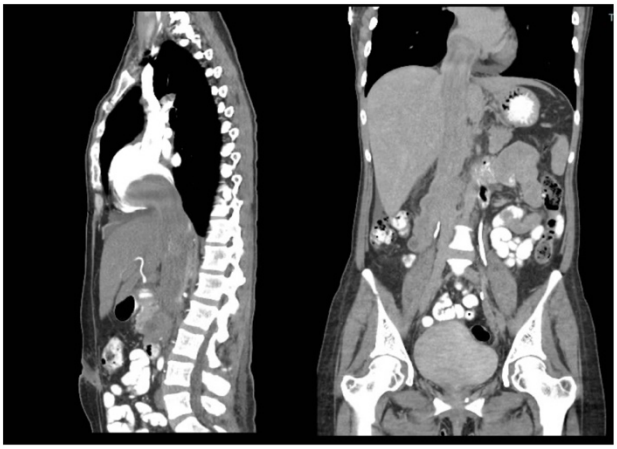


Figure 2. Gross pathology

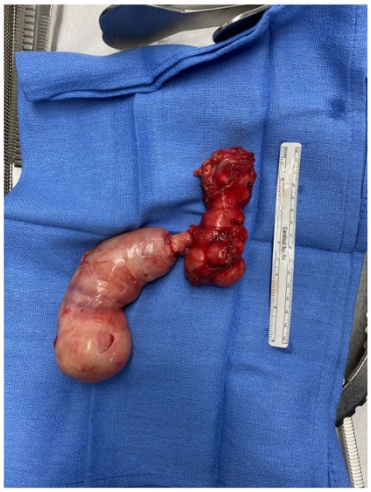
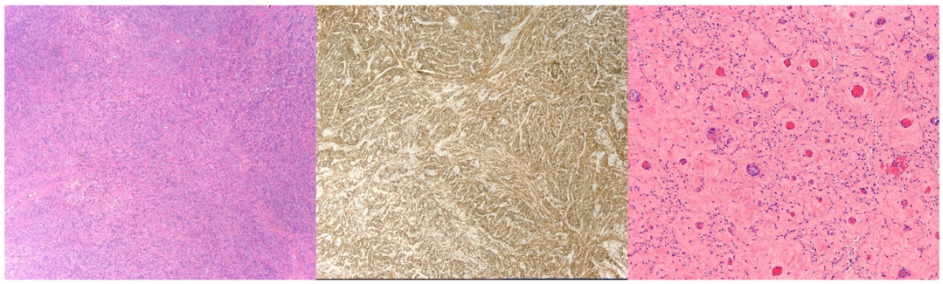


Figure 3. Histologic images



Cardiovascular Anesthesiology - 10

Perioperative management of a complicated case of pulmonic and aortic valves infective endocarditis: a case report

Miguel E Perez-Viloria[1], Fayeza Malik[2], Robert Zusman[1], Marco A Castillo-Rodriguez[1], Gerald Rosen[1]

[1]Mount Sinai Medical Center of Florida, Miami Beach, FL, [2]Florida International University, Miami, FL

Introduction: Infective endocarditis (IE) is a microbial infection that most commonly involves the endocardial surface of native valves, prosthetic valves, or implanted cardiac devices1. IE is most commonly found in the aortic valve followed by the mitral valve2. Right-sided endocarditis is in itself rare, accounting for 5-10% of all cases of infective endocarditis (IE) with the majority of these cases involving the tricuspid valve3. Pulmonic valve (PV) involvement in IE is uncommon, accounting for 1.5-2.0% of total admissions for IE, and may be missed if patients do not have the typical risk factors or features of right-sided endocarditis4. While intravenous (IV) drug abuse is the most important factor that predisposes to pulmonic valve endocarditis (PVE), central venous catheter placement, alcoholism, dental extraction, congenital heart disease (CHD), and idiopathic causes have been reported4,5,6. PVE is a condition that warrants early management and frequent follow-up to prevent long-term systemic complications and to reduce the risk of mortality. Diagnosis of PVE is based on clinical findings that include persistent fever, pulmonary symptoms (cough and dyspnea), pulmonic regurgitant murmur, positive blood cultures, and transesophageal echocardiographic findings of pulmonary vegetation7. The most common causative pathogens involved in PVE include Staphylococcus aureus, coagulase-negative Staphylococci, and group B Streptococci8. Intravenous antibiotics administration is the primary treatment modality, with surgical intervention reserved for cases with persistent bacteremia, complications (recurrent septic pulmonary embolism), and vegetations > 10 mm9. This case report aims to highlight the importance of early recognition and careful management of this rarely reported condition perioperatively.

Methods: A 66-year-old man with a past medical history of congenital pulmonic stenosis status post pulmonic valve repair via sternotomy at 5 years of age, moderate aortic regurgitation, May-Thurner syndrome, and hypertension was admitted for a five-month history cough, one-month history of weight loss, and diaphoresis after dental cleaning. He did not receive antibiotic prophylaxis for the dental cleaning. Blood cultures were drawn, and he was diagnosed with gram positive bacteremia (S. mitis). Transesophageal echocardiography showed a 1x0.7cm mobile vegetation attached to the ventricular side of the right coronary aortic cusp (Figure 1) and a 1.1x0.5cm mobile vegetation attached to the arterial side of the pulmonic valve cusp (Figure 2) and he was started on ceftriaxone and underwent re-operative sternotomy, aortic valve replacement and pulmonary valve replacement using cadaveric pulmonary homograft. During his post-operative course, he developed a moderate pericardial effusion with echocardiographic signs of early cardiac tamponade as well as atrial flutter. He underwent an urgent subxiphoid pericardial window. Later he underwent synchronized cardioversion and converted back to normal sinus rhythm. The patient was later discharged to inpatient rehabilitation in stable condition.

Conclusion: PVE has a low incidence, representing only 1.5-2.0% of total cases of IE3. Literature on PVE is limited to a few small case series and case reports 3,10,11,12. IE may involve solely the pulmonic valve or may simultaneously involve the mitral or aortic valve2. The scarcity of PVE cases may be due to factors including lower oxygen content of venous blood and lower pressures within the right heart. Additionally, the differences in the endothelial lining and vascularization of the valve, as well as lower incidence of PV congenital malformations or acquired valvular abnormalities may account for the rarity of PVE 4,13. In this case, the patient had both aortic and pulmonic valve replacement, and post-operatively developed pericardial effusion with echocardiographic signs of early cardiac tamponade as well as atrial flutter, which were subsequently resolved with an urgent subxiphoid pericardial window. He also underwent synchronized cardioversion and was converted back to normal sinus rhythm. Due to its rarity and complexity of presentation,

PVE requires a multidisciplinary approach to its perioperative management to prevent systemic complications.

References: 1. Lancet. 387(10021), 882-893, 2016 2. International Journal of Clinical and Experimental Medicine. 7(1), 199-218, 2014 3. American Heart Journal. 117(5), 1140-1146. 1989 4. Canadian Journal of Cardiology. 16(10), 1282-1288, 2000 5. Journal of Cardiology Cases. 8(5), 161-163, 2013 6. Lancet. 339(8786), 135-139, 1992 7. Journal of the American Society. 18(2), 197-198, 2005 8. International Journal of Clinical and Experimental Medicine. 7(1), 199-218, 2014 9. Journal of Thoracic and Cardiovascular Surgery. 153(6), 1241-1258, 2017 10. The American Journal of Surgery. 75(12):1265-1266, 2009 11. American Heart Journal. 109(3 Pt 1), 558-567, 1985 12. The American Journal of Cardiology. 56(10), 610-613, 1985 13. Journal of Community Hospital Internal Medicine Perspectives. 5(6), 29647, 2015

Figure 1. Aortic valve vegetations

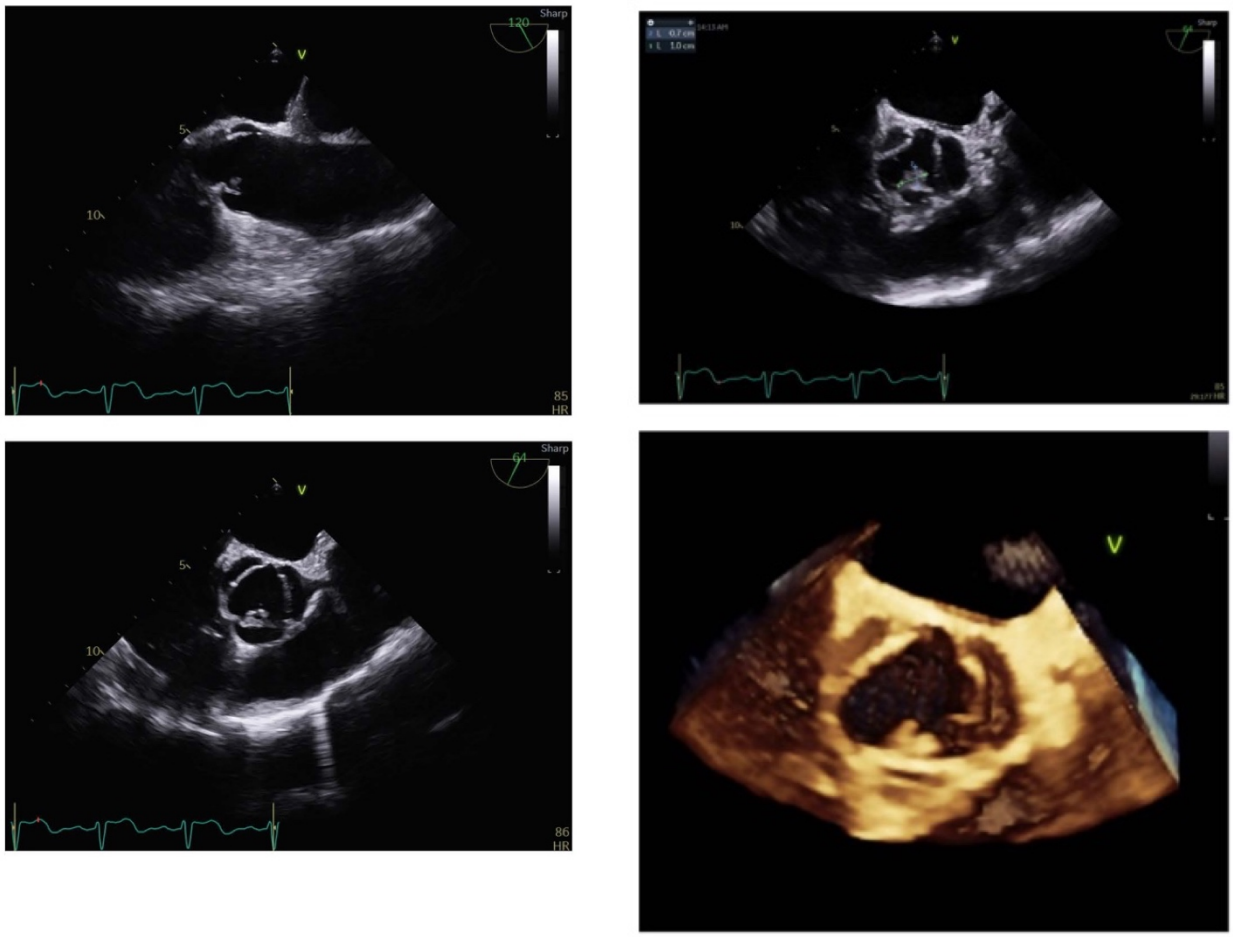
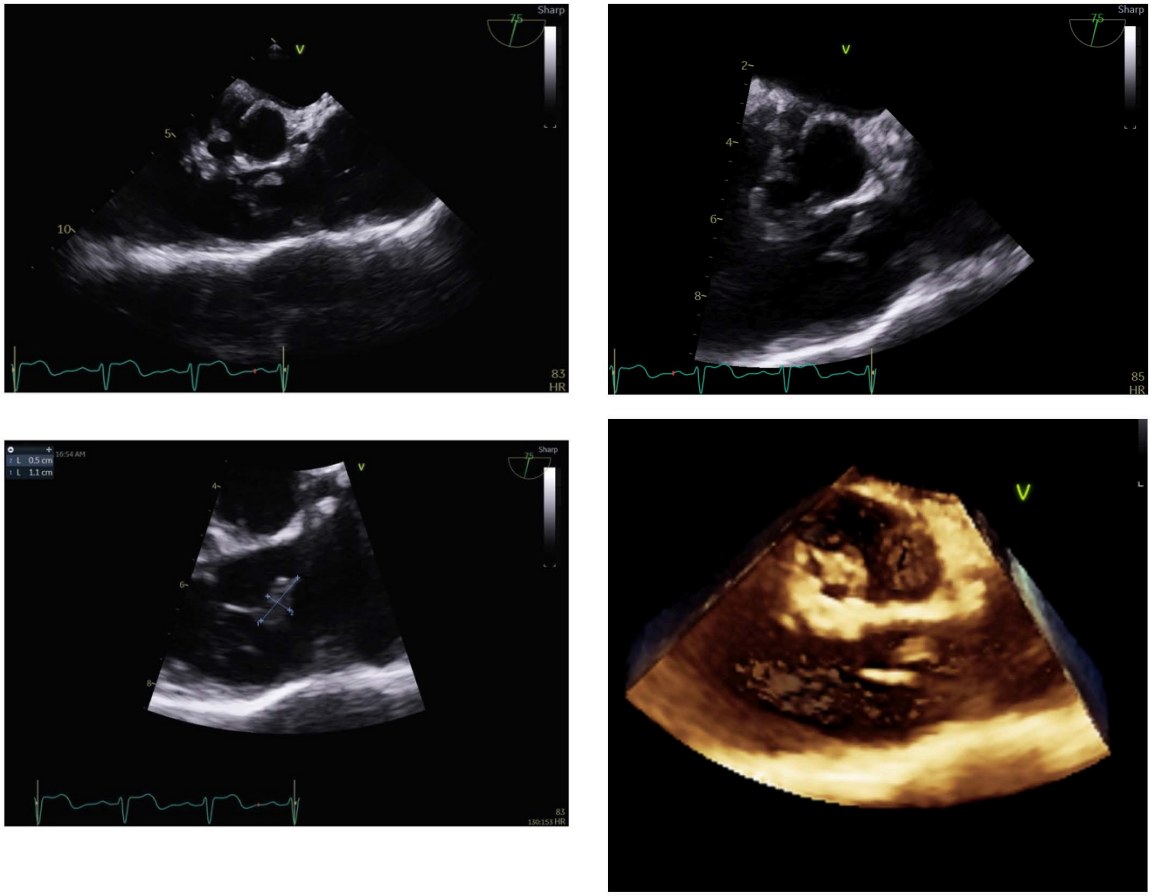


Figure 2. Pulmonic valve vegetations



Cardiovascular Anesthesiology - 11

Rapid Onset Acute Primary Graft Dysfunction after Right Lung Transplant Requiring Veno-Venous ECMO

Sean M Baskin[1], Nicole Tomkowich[1]

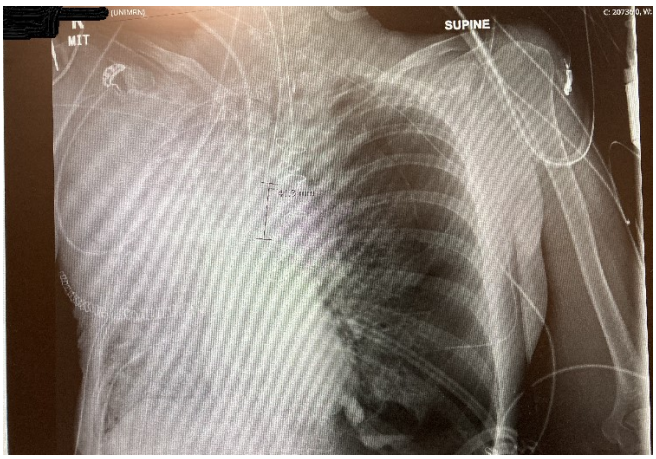
[1]Temple University Hospital, Philadelphia, PA

Introduction: Primary graft dysfunction (PGD) is a devastating complication of organ transplantation, occurring in up to 30% of lung transplants1. Defined by hypoxia, decreased pulmonary compliance, increased PVR and pulmonary edema occurring within 72 hours of transplant, PGD can increase 30-day mortality 6-fold, and 1-year mortality 10%2. The treatment for PGD is supportive, and confers a higher rate of chronic allograft dysfunction and reduced functional status1,2. We present a 69-year-old female who underwent right lung transplant complicated by severe PGD less than 2 hours after reperfusion.

Methods: A 69-year-old female with severe Emphysema and COPD was scheduled for right single lung transplant. Induction of anesthesia was uneventful and induction immunosuppression was Basiliximab. Singe lung ventilation was accomplished by a double lumen tube (DLTL). Inhaled Nitric Oxide (iNO) was utilized for the entirety of the surgery at 40ppm. The transplant was performed without the use of mechanical circulatory support and reperfusion occurred 289 minutes after donor cross clamp. Immediately prior to reperfusion, 250mg Methylprednisolone was given. Double-lung ventilation resumed after 154 minutes of single lung ventilation. Prior to DLT exchange, SpO2 was 98%, FiO2 34% and PaO2 151mmHg. In preparation for transport to the ICU, the DLT was exchanged for a single lumen endotracheal tube without difficulty. A brief bronchoscopy was performed by the surgeon to assess the bronchial suture line and assess for any bleeding. The patient rapidly become hypoxic and despite 100% FiO2 and multiple ventilator adjustments, we were unable to meaningfully increase SpO2. Copious frothy pink secretions were noted in the endotracheal tube and could not be cleared by suctioning. At this time, she was peripherally cannulated for V-V ECMO with an appropriate recovery of her oxygen status (Flow 4LPM,FdO2 100%, PaO2 250mmHg). 89 minutes elapsed between allograft reperfusion and ECMO cannulation. After arrival in the ICU, she was found to have a completely opacitifed right lung and an left pneumothorax (image 1). She was decannulated on POD 3, extubated on POD 10. She was eventually discharged home on POD 31.

Conclusion: PGD is a devastating complication of lung transplant conveying a 6-fold increase in short term mortality and 10-year survival as low as 11% respectively2. Our patient experienced rapidly progressive PGD 89 minutes after reperfusion, requiring V-V ECMO to restore oxygenation. Additionally, her left pneumothorax may be due to a ruptured bleb during recruitment breaths for the allograft or during PEEP adjustments prior to the initiation of ECMO. While a pneumothorax is unlikely to be responsible for the development of PGD, it likely contributed to her hypoxia and hemodynamic instability. Anesthesiologists and members of a thoracic surgical transplant team should consider both PTX and PGD in early onset hypoxia and not hesitate to utilize mechanical circulatory support for oxygenation.

References: 1-Chest 2003;124:1232-1241 2-Turk J Anaesthesiol Reanim. 2015;43(6):418-423



Cardiovascular Anesthesiology - 12

Anesthesia for non-cardiac surgery in an adult patient with an underlying non repaired aortic Coarctation: A case report

Beatriz A Deras[1], Deny L Avelar[2], Ana L Guerrero[1]

[1]Hospital Nacional Rosales, San Salvador, El Salvador, [2]Hospital Nacional Zacamil, San Salvador, El Salvador

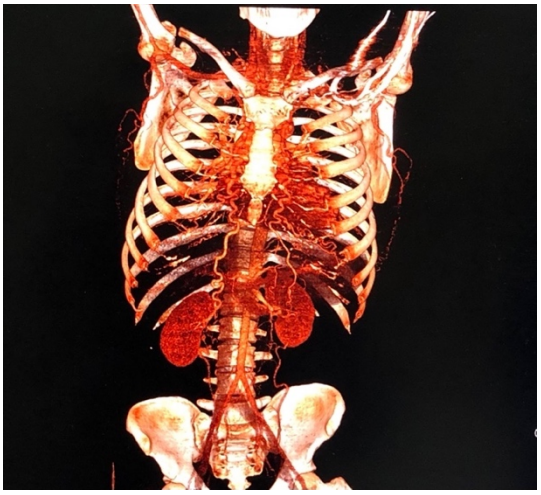
Introduction: Aortic Coarctation is a congenital cardiac anomaly consisting of a constricted aortic segment. Its prevalence varies between 5-8%. Nowadays, more adults are living with congenital heart disease (CHD) (1) requiring anesthesia for cardiac and non-cardiac surgeries. The distinctive physiology of these patients presents as an anesthesia challenge.

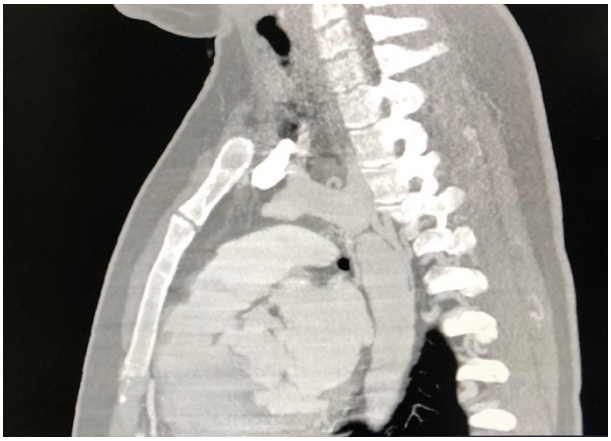
Methods: A 20-year-old man, normal body mass index, presented to the emergency room with multiple trauma. No life threatening injuries, and the following vital signs: blood pressure (BP) 190/90 mmHg, Heart rate (HR) of 110 bpm, Respiratory Rate (RR) of 24, 97% SaO (2) and Visual Analog Scale (VAS) for pain: 8/10. Head, abdominal and thoracic trauma were excluded, but a closed fracture in his left femur. The patient was scheduled for an elective osteosynthesis of his femur and managed with a non-peptide angiotensin II receptor antagonists. Preoperative assessment: Patient at age 15 presented shortness of breath and angina with exercise, a murmur was found at consultation. He was prescribed with an angiotensin-converting-enzyme inhibitor. No more tests were done and he has being non-compliant to his treatment. He denied worsening of symptoms. Cardiovascular exam: localized holosystolic aortic murmur. No enlargement of intercostal arteries. Delayed femoral pulses in both lower extremities. Electrocardiogram: Sinusal Rhythm, 100 bpm, 30° axis, P 0.08s, QRS 0.10s, T wave upslope in V1-V2. Echocardiogram: left aortic arch with a coarctation area of 0.33 cm with a 80 mmHg peak flow in descendent aorta, and 28 mmHg mean gradient, mild mitral valve insufficiency, concentric hypertrophy LVEF 45%, PCWP 30 mmHg. Kidney ultrasound: No abnormalities. Angio- Computed Tomography: Descendent Aorta with a stenotic area 2.5 cm distal to the origin of the left subclavian artery, with a diameter of 9 mm. Distally enlargement of the intercostal and internal mammary arteries. No patency of ductus arteriosus. (FIG.1-3)
For surgery the patient was monitored, BP: upper left arm: 153/72mmHg, upper right arm: 152/62mmHg, right lower limb: 115/78 mmHg. HR: 47 bpm. SaO (2):99%, FiO2: 21%. The choice for anesthesia was general anesthesia: premedication with Midazolam 5mg IV, a right arterial radial line was placed for invasive arterial pressure monitoring. For Induction etomidate 20mg, fentanyl 200 mcg and cisatracurium 10mg was used. Remifentanil 0.4 mcg/kg/h and Propofol 4mg/kg/h was used for maintenance. Vital signs monitor record during surgery is shown in Table 1. A blood loss of 800 ml was estimated and recovered with Hartman 1L and 1 unit of pRBC. The total surgery time was three hours. Written informed consent was obtained from the patient for publication of this case report.

Conclusion: Aortic coarctation produces an increase in the afterload of the left ventricle, heart failure, hypoperfusion below the defect with its consequences. During adulthood this defect can be detected by hypertension in the upper limbs (2). Our case, is an incidental diagnosis of non repaired aortic coarctation during preparation for osteosynthesis. CHD intervention was postponed. Published literature for anesthesia management had little information. It was decided to avoid regional anesthesia which could increase the risk of bleeding (3) and monitor parameters that could compromise coronary and cerebral perfusion and left ventricle function. Nowdays it is important to identify CHD, and choose the adequate anesthestic management to avoid increasing the perioperative risk in these patients.

References: (1) Cannesson M, Earing MG, Collange V, Kersten JR. Anesthesia for noncardiac surgery in adults with congenital heart disease. Anesthesiology. 2009 Aug;111(2):432-40. doi: 10.1097/ALN.0b013e3181ae51a6. PMID: 19602959. (2) Warnes CA, Liberthson R, Danielson GK, Dore A, Harris L, Hoffman JI, Somerville J, Williams RG, Webb GD. Task force 1: the changing profile of congenital heart disease in adult life. J Am Coll Cardiol. 2001 Apr;37(5):1170-5. doi: 10.1016/s0735-

1097(01)01272-4. PMID: 11300418. (3) Howard-Quijano, K., Smith, M. & Schwarzenberger, J.C. Perioperative Care of Adults with Congenital Heart Disease for Non-cardiac Surgery. Curr Anesthesiol Rep 3, 144-150 (2013). https://doi.org/10.1007/s40140-013-0023-8





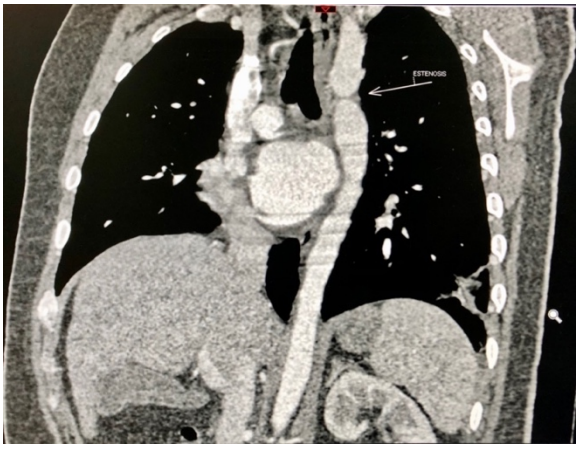


Table 1. Main parameters monitored during the surgery

Time of the surgery	Arterial line blood pressure	Cuff in Lower right extremity blood pressure	Heart rate	Biespectral index	Saturation with FIO2 100%	ETCO2
15 min	202/100 mmHg	112/67 mmHg	43 rpm	44	98%	25
30 min	174/73 mmHg	100/70 mmHg	46 rpm	40	99%	27
1st hour	133/105 mmHg	111/68 mmHg	52 rpm	56	99%	35
2nd hour	120/70 mmHg	100/70 mmHg	65 rpm	50	100%	30
3rd hour	98/72 mmHg	70/40 mmHg	73 rpm	48	100%	28
Awake patient	160/70 mmHg	140/63 mmHg	80 rpm	88	100%	n/a
PACU 20 min	n/a	137/79 mmHg	75 rpm	n/a	100%	n/a

PACU: Postanesthetic care unit.

Cardiovascular Anesthesiology - 13

Intraoperative considerations in a patient on intravenous epoprostenol undergoing minimally invasive cardiac surgery

Christina M Tull[1], Anand Abraham[1], John W MacArthur[1], Matthew W Vanneman[1], T. Robert Feng[1]

[1]Stanford University Medical Center, Palo Alto, CA

Introduction: Intravenous (IV) epoprostenol is frequently used to treat severe pulmonary hypertension (1) but is less commonly seen in the perioperative setting. Minimally invasive cardiac surgery via thoracotomy in patients on IV epoprostenol presents a unique anesthetic challenge and insufficient literature exists regarding best practices. Here, we highlight the expected intraoperative challenges in patients on IV epoprostenol where rebound pulmonary hypertension and acute right ventricular failure from abrupt discontinuation (2) are a significant concern.

Methods: A 35-year-old male was simultaneously diagnosed with a left atrial myxoma and World Health Organization Group I pulmonary hypertension with severe right ventricular (RV) dysfunction. He was started on IV epoprostenol one month prior to scheduled myxoma resection via minimally invasive thoracotomy requiring one-lung ventilation on peripheral cardiopulmonary bypass (CPB). Intraoperatively, IV epoprostenol was continued and 20 ppm of inhaled nitric oxide and an epinephrine infusion were started prior to induction. Induction of anesthesia was uneventful. A bronchial blocker was placed through endotracheal tube, and intraoperative lines included an arterial line, a sheath introducer, and a pulmonary artery (PA) catheter (initial PA pressure 74/43 mmHg). Transesophageal echocardiography confirmed a 2.5 cm mass on the interatrial septum and a severely dilated RV with severely decreased function. Vasopressin was started due to hypotension post-induction. With one-lung ventilation (OLV), acute, severe hypoxemia occurred requiring immediate resumption of two-lung ventilation. Due to an inability to tolerate OLV, CPB was initiated following femoral arterial and venous cannulation. Upon initiation of CPB, severe hypoxemia with decreased cerebral oximetry saturations re-occurred in the setting of an excessively full venous reservoir and an inability to completely empty the heart despite functioning cannulas. An additional superior vena cava cannula was placed for drainage and reservoir volume was diverted with resolution of the hypoxemia. IV epoprostenol was discontinued due to refractory hypotension requiring norepinephrine and vasopressin infusions. Upon weaning of CPB, 2 units of platelets were transfused for coagulopathy. IV epoprostenol was restarted at a lower dose after achieving hemodynamic stability and the remainder of the case was uneventful.

Conclusion: Management of patients on IV epoprostenol presenting for cardiac surgery via thoracotomy on peripheral CPB is complex and understanding the physiological challenges is important. Potential anesthetic difficulties include ventilation/perfusion mismatch from non-selective pulmonary vasodilation, refractory hypotension, volume overload resulting in dual circulation when initiating peripheral CPB, and epoprostenol-induced platelet dysfunction. (3,4,5) Open communication with the surgical team is vital to minimize IV epoprostenol-related complications.

References: 1. A Comparison of Continuous Intravenous Epoprostenol (Prostacyclin) with Conventional Therapy for Primary Pulmonary Hypertension. 334:296-301, 1996 2. Rebound Pulmonary Hypertension and Cardiogenic Shock after Withdrawal of Inhaled Prostacyclin. 100:1023-5, 2004 3. Aerosolized and Intravenous Prostacyclin During One-lung ventilation. Hemodynamic and Pulmonary Effects. 57:612-20, 2004 4. A Novel Veno-arteriovenous Extracorporeal Membrane Oxygenation with Double Pump for the Treatment of Harlequin Syndrome. 35:65-72, 2020 5. Management of Urgent High-risk Cardiopulmonary Bypass in Patients with Heparin-induced Thrombocytopenia Type II and Coexisting Disorders of Renal Function: Use of Heparin and Epoprostenol Combined with On-line Monitoring of Platelet Function. 14:304-8, 2000

Cardiovascular Anesthesiology - 14

Neuraxial Dilemmas in Complex Hybrid Aortic Repair: A Case Report

Matthew Orlowski[1], Ashley P Oliver[1], Andrew Schober[1], Michael P Bokoch[1], Pedram Aleshi[1]

[1]University of California, San Francisco, San Francisco, CA

Introduction: Critical neuraxial procedures (lumbar drain ± thoracic epidural) may improve safety and recovery of patients undergoing thoracoabdominal aortic aneurysm (TAAA) repair. However, the burden of comorbid disease in this patient population, as well as complex anticoagulation requirements, make neuraxial techniques challenging to implement safely. New strategies are needed to manage these often-competing goals. We describe the management of a patient undergoing a complex staged open and endovascular repair of a chronic TAAA. We emphasize the interplay between prevention of spinal cord ischemia (SCI), analgesia, coagulation status, and safe neuraxial techniques.

Methods: A 57-year-old man with history of type A dissection and multiple previous open and endovascular aortic procedures presented with an expanding TAAA and chronic type B dissection. Additional history included ischemic cardiomyopathy and mechanical aortic valve replacement with warfarin anticoagulation. He was scheduled for a two-stage procedure: (1) open aortic visceral debranching, followed by (2) extension of an endovascular stent graft from the thoracic to infrarenal aorta to exclude both the dissection and aneurysm. Notably, prior lumbar drain placement in this patient was complicated by a spinal hematoma requiring T7-L3 laminectomy, from which he recovered without long-term neurologic deficits. Warfarin was bridged with enoxaparin perioperatively. Given the high risk for SCI and perceived risk of traumatic placement, CT guidance was used to aid placement of a 5 Fr lumbar drain. The drain remained in place for 8 days for use during both stages of the procedure. We used a combination of vasopressors and prophylactic CSF drainage to optimize spinal cord perfusion pressure. The patient was extubated in the operating room for a detailed neurologic after each stage of his procedure. The patient's mechanical aortic valve necessitated maintenance of therapeutic anticoagulation with a low dose IV heparin infusion between procedures. Out of concern for compounding the risk of neuraxial hematoma, we elected to place bilateral T7 erector spinae plane (ESP) catheters before the first procedure in lieu of a thoracic epidural. This technique resulted in low pain scores, minimal opioid consumption, and no catheter related complications during the hospitalization. Ultimately, the patient underwent a successful staged aortic repair and was discharged without significant neurologic injury and near his functional baseline.

Conclusion: Complex hybrid aortic repair poses unique risks and anesthetic challenges when balancing anticoagulation needs with critical neuraxial procedures. This case highlights specific management strategies (CT-guided lumbar drain placement and ESP catheters as an alternative to thoracic epidural analgesia) that can be employed to improve patient safety and outcomes. Furthermore, effective care of these patients requires a multidisciplinary approach at all stages of the perioperative period in order to address their unique needs.

Cardiovascular Anesthesiology - 15

Intraoperative Diagnosis of an Immobile Mechanical Aortic Valve Leaflet Using Transesophageal Echocardiography: A Case Report

Allan L Yau[1], Duc P Le[2], Steven Haddy[2]

[1]LAC+USC Medical Center, Los Angeles, CA, [2]Keck Medicine of USC, Los Angeles, CA

Introduction: Valve obstruction is a serious complication after implantation of a mechanical valve, most commonly secondary to pannus formation, thrombosis, or vegetation [1]. The causes of intraoperative valve obstruction have yet to be fully defined due to infrequent occurrence but include anatomic interference and suture placement [2]. The use of intraoperative transesophageal echocardiography (TEE) plays a critical role in determining pre- and post-operative cardiac function in addition to prosthetic valve function. This case report presents a rare occurrence of an intraoperative stuck mechanical aortic valve diagnosed by TEE. A multifaceted approach to diagnosis with TEE was utilized, ultimately leading to surgical correction prior to closure.

Methods: A 31-year-old female with a history of Takayasu's arteritis presented for redo aortic root replacement and mechanical aortic valve replacement for severe aortic insufficiency (AI). After uneventful intubation, a TEE probe was placed. Sternotomy was performed and cardiopulmonary bypass (CPB) was initiated. The previous aortic root graft and native aortic valve were excised and replaced with a new aortic root graft and a 25mm St. Jude Medical (SJM) Masters Series aortic valved graft. Prior to CPB decannulation, TEE showed a poorly visualized but nonmobile leaflet with effective orifice area (EOA) of 0.6 cm2 by continuity equation. Color flow Doppler showed turbulent flow in the aortic root without AI (Figure 1). Maximum transvalvular aortic velocity (Vmax) was measured to be 4.64 m/s with a mean pressure gradient (PG) of 52.8 mmHg. Dimensionless valve index (DVI) was 0.15 and the spectral Doppler contour was parabolic with an acceleration/ejection time (A/E) ratio of 0.38 (Figure 2). CPB was reinitiated and fibrinous material was found to obstruct the opening of one leaflet, treated with cautery and 45-degree valve rotation. After weaning from CPB, Vmax was 2.78 m/s with a mean PG of 17 mmHg and EOA of 1.6 cm2 (Figure 3). Patient was transported to ICU intubated, sedated, with epinephrine for inotropic support.

Conclusion: This case report describes the rare occurrence of a mechanical aortic valve leaflet with obstruction to systolic opening primarily diagnosed by intraoperative TEE. Several echocardiographic modalities were used to characterize valve function. Valve flow dynamics were abnormal with unexpectedly high Vmax and PG that met criteria for severe outflow obstruction (Figure 2) [3]. Though there are several causes for high PGs, in the immediate post-CPB setting with the lack of structural disease preoperatively and poor valve visualization on TEE, the differential included acute valvular obstruction versus a high-flow state induced by a hyperdynamic left ventricle on inotrope infusion [4,5]. The findings of a low EOA and DVI in combination with a parabolic Doppler contour, turbulent aortic root flow, and an elevated A/E ratio suggested an etiology of valvular obstruction (Figure 2). After repair, postoperative valve flow dynamics were more aligned with documented values of SJM mechanical aortic valves (Figure 3) [6].

References: 1. J Am Coll Cardiol. 1991;17:646-50. 2. JA Clin Rep 2020;6:9. 3. J Am Coll Cardiol 2014;63:e57. 4. J Am Coll Cardiol Img. 2010;3:296-304. 5. Circulation 1959;20:181-189. 6. Circulation 1991;83:213-23.

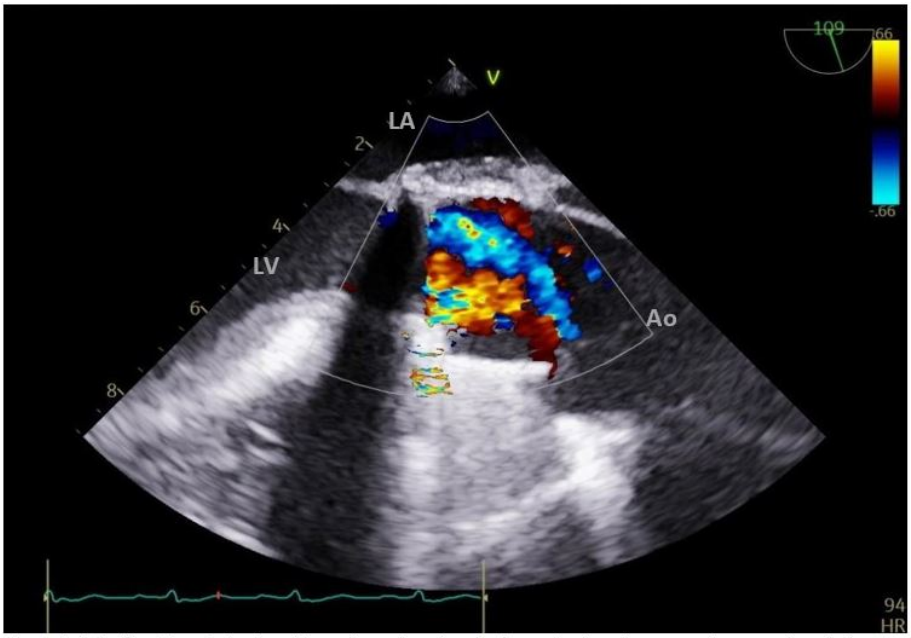


Figure 1. Color flow Doppler in the mid-esophageal aortic valve long axis view demonstrating severely turbulent flow through the mechanical aortic valve immediately post cardiopulmonary bypass after initial implantation. LA = Left Atrium; LV = Left Ventricle; Ao = Aorta.

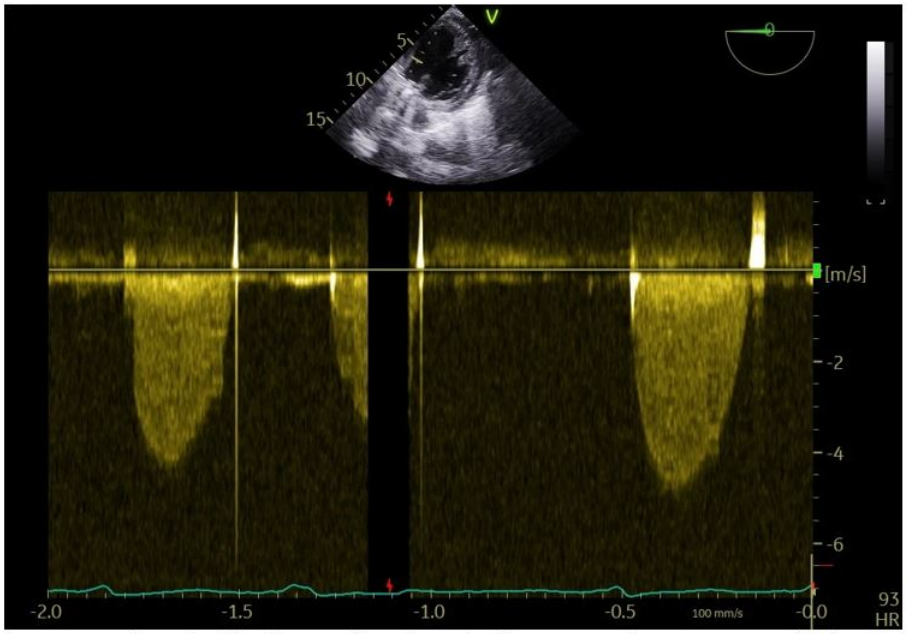


Figure 2. Spectral Doppler of the left ventricular outflow in the deep transgastric long axis view immediately post cardiopulmonary bypass after initial mechanical aortic valve implantation.

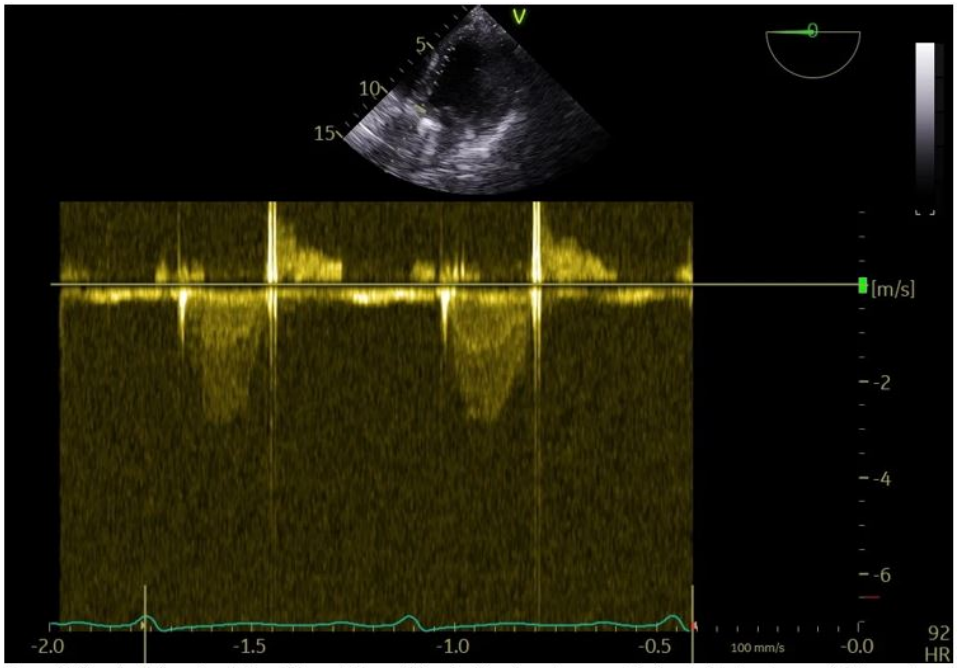


Figure 3. Spectral Doppler of the left ventricle outflow in the deep transgastric long axis view immediately post cardiopulmonary bypass after the obstructed mechanical aortic valve leaflet was repaired.

Cardiovascular Anesthesiology - 16

Careful Consideration of Thrombus in Transit in Cardiac Surgery after Acute Ischemic Stroke

Alyssa K Streff[1], Chris Lensing[1], Kelly Mishra[1]

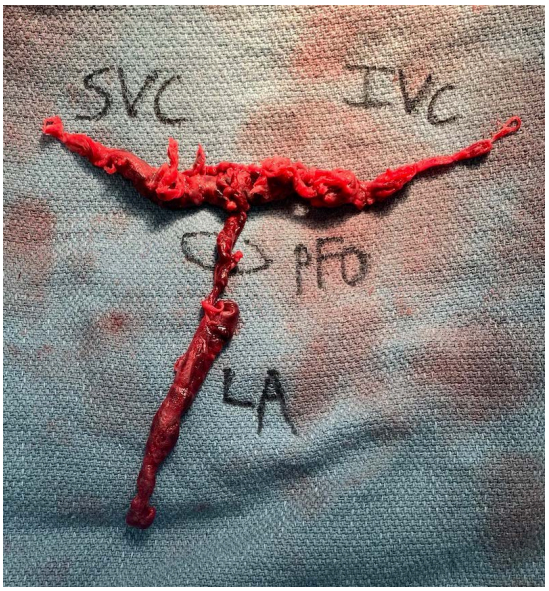
[1]Vanderbilt University Medical Center, Nashville, TN

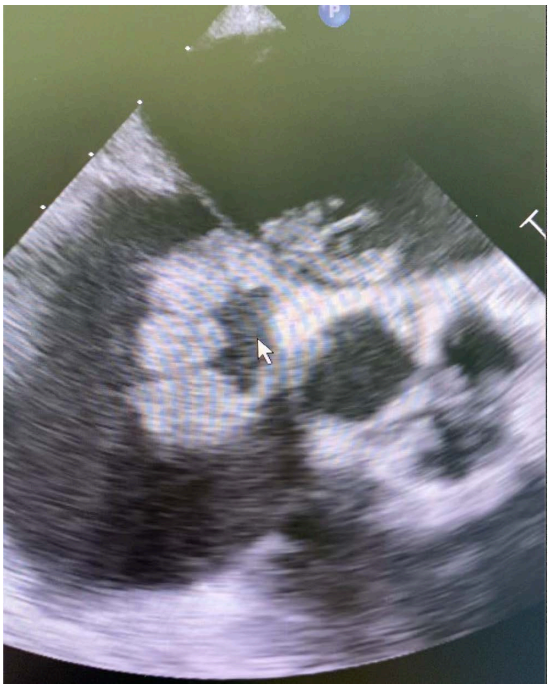
Introduction: Thrombus in transit through a patient foramen ovale (PFO) is extremely rare. This phenomenon has only been reported in limited case reports or series and is often not diagnosed until autopsy (1,2). Thrombus extending through a PFO is associated with high risk of complications including pulmonary embolism (PE) and ischemic stroke (2). In one study, a PFO was found to be present in 43.9% of patients with cryptogenic stroke (3). We present a case of ischemic stroke and extensive thrombus that involved the inferior and superior vena cava (IVC, SVC), right atrium (RA), PFO and left atrium (LA). Management of thrombus crossing a PFO is a major challenge, especially in the setting of acute ischemic stroke requiring heparin infusion with high risk of hemorrhagic conversion.

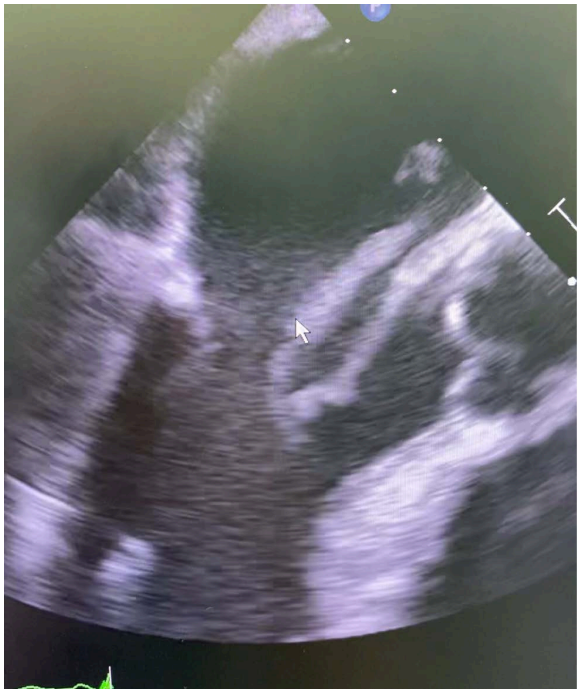
Methods: 68-year-old male with history of coronary artery disease status post 4 vessel coronary artery bypass ten years prior, hypertension, and type 2 diabetes, presented with altered mental status (AMS) initially thought to be due to hypoglycemia. A code stroke was called for worsening AMS, new left sided weakness and left facial droop. Pertinent labs included troponin 0.41, BNP 1,774, K 5.3. Work up for stroke revealed multiple deep vein thromboses (DVT) in both lower and upper extremities, transthoracic echocardiogram (TTE) showed large RA thrombus in transit to LA extending to the mitral valve. The patient was placed on a heparin infusion and transferred to CVICU. He underwent urgent mini right thoracotomy for extensive thrombectomy of IVC, SVC, RA, LA and PFO closure (Figure 1). Vascular access was placed in right internal jugular vein under TEE guidance to avoid embolization of thrombus. Transesophageal echocardiogram (TEE) at the time of surgery showed normal LV function, moderately reduced RV function, mild MR and large bi-atrial clot burden (right 2.98cm x 1.11cm, left 3.19cm length) (Figure 2) extending to the mitral valve (figure 3), but no evidence of main pulmonary artery (PA) thrombus. It was imperative to confirm absence of main PA thrombus which allowed for right mini thoracotomy approach instead of repeat sternotomy. On postop day one, neurology exam had improved to only mild residual left sided weakness, however, CT PE revealed acute PE involving distal left and right main pulmonary arteries. The patient remained on heparin infusion and CT angiogram head and neck showed evolving stroke. Hematology was consulted and recommended reducing heparin infusion dose to reduce hemorrhagic conversion risk while bridging to warfarin. Further hematology workup revealed possible ascending colon mass, but no definitive cause of multiple DVTs and thrombus. Patient to follow up with hematology outpatient.

Conclusion: There were multiple challenges in caring for this patient with competing acute pathologies and chronic comorbidities: safe placement of vascular access, TEE guidance of surgical approach, and postoperative management of remaining DVTs and distal PA thrombus in the setting of evolving ischemic stroke with high risk of hemorrhagic conversion. Surgical removal was preferred in this case due to extreme risk for continued embolization of thrombus. A multi-disciplinary team approach including cardiothoracic surgery, CVICU, cardiothoracic anesthesiology and hematology with careful consideration of risks vs benefits was necessary for safe and complete care of this patient.

References: (1) Thrombus in Transit through Patent Foramen Ovale. Case Rep Cardiol. 2013;2013:395879. (2) A ticking time bomb: thrombus straddling a patent foramen ovale, QJM: An International Journal of Medicine, Volume 113, Issue 7, July 2020, Pages 483-484. (3) Patent foramen ovale and cryptogenic stroke in older patients. N Engl J Med. 2007;357(22):2262-2268.


SVC
IVC
PFO
LA





Cardiovascular Anesthesiology - 17

Refractory Vasoplegia under General Anesthesia in the Setting of Renin-Angiotensin Axis Blockade in Major Non-cardiac Surgery

Daniel Lazzareschi[1], Julin F Tang[2], Abigail Howard[3]

[1]Zuckerberg San Francisco General Hospital and Trauma Center, San Francisco, CA, [2]UCSF/ZSFGH, San Francisco, CA, [3]UCSF, San Francisco, CA

Introduction: A 93 y/o male with PMH of HTN, T2DM, and CAD was admitted for GLF complicated by C1-2 fractures and taken to the OR for posterior spinal arthrodesis. The patient,Äôs outpatient medications included aspirin, amlodipine, lovastatin, and enalapril. He was induced with etomidate and intubated uneventfully, however following incision and instrumentation, the patient became hypotensive with mean MAPs in the 50 mmHg range despite minimal blood loss and modest intravenous anesthetic. Phenylephrine and norepinephrine vasopressor infusions were escalated accordingly, the latter reaching 14 mcg/min, with pressures minimally responsive to norepinephrine boluses of 32 mcg at a time. Finally, transesophageal echocardiography was initiated for hemodynamic monitoring, revealing expected left ventricular hypertrophy but otherwise normal systolic function without evidence of obstructive pathophysiology. The patient was supported with aggressive vasopressor therapy throughout the case to maintain MAPs greater than 65 mmHg, most effectively with boluses of vasopressin. The procedure concluded after 5 hours, and he was extubated with improved hemodynamics following emergence from general anesthesia (GA). This case demonstrates the important and controversial phenomenon of refractory hypotension under GA in the setting of renin-angiotensin axis blockade by outpatient angiotensin converting enzyme inhibitors (ACEIs), ostensibly by the patient,Äôs use of enalapril in this example. The existence and pathophysiology of this phenomenon has long been debated and is best understood in the context of cardiac surgery involving cardiopulmonary bypass (CPB), whereby increased inflammatory mediators are thought to mediate vasopressor-refractory vasodilation leading to severe hypotension. In cardiac surgery, this vasoplegic syndrome is often treated with methylene blue, a tricyclic phenothiazine known to inhibit guanylate cyclase, an important enzyme in the nitric oxide (NO) signaling cascade that mediates vasodilation by the vascular endothelium. Recent retrospective studies failed to identify a significant difference in episodes of perioperative vasoplegia between ACEI and non-ACEI users in non-cardiac surgery. Further research in this area is needed to identify perioperative vasoplegia in the setting of ACEI use among patients undergoing non-cardiac surgery, examine potential risks outside of the use of CPB, and explore treatment strategies involving modulation of the NO signaling pathway.

Methods: This case demonstrates the important and controversial phenomenon of refractory hypotension under GA in the setting of renin-angiotensin axis blockade by outpatient angiotensin converting enzyme inhibitors (ACEIs), ostensibly by the patient's use of enalapril in this example.

Conclusion: The existence and pathophysiology of this phenomenon has long been debated and is best understood in the context of cardiac surgery involving cardiopulmonary bypass (CPB), whereby increased inflammatory mediators are thought to mediate vasopressor-refractory vasodilation leading to severe hypotension. In cardiac surgery, this vasoplegic syndrome is often treated with methylene blue, a tricyclic phenothiazine known to inhibit guanylate cyclase, an important enzyme in the nitric oxide (NO) signaling cascade that mediates vasodilation by the vascular endothelium. Recent retrospective studies failed to identify a significant difference in episodes of perioperative vasoplegia between ACEI and non-ACEI users in non-cardiac surgery. Further research in this area is needed to identify perioperative vasoplegia in the setting of ACEI use among patients undergoing non-cardiac surgery, examine potential risks outside of the use of CPB, and explore treatment strategies involving modulation of the NO signaling pathway.

References: 1. Bertrand M, Godet G, Meersschaert K, Brun L, Salcedo E, Coriat P. Should the

angiotensin II antagonists be discontinued before surgery? Anesth Analg. 2001 Jan;92(1):26-30. doi: 10.1097/00000539-200101000-00006. PMID: 11133595. 2. Booth AT, Melmer PD, Tribble B, Mehaffey JH, Tribble C. Methylene Blue for Vasoplegic Syndrome. Heart Surg Forum. 2017 Oct 31;20(5):E234-E238. doi: 10.1532/hsf.1806. PMID: 29087290. 3. Colson P, Ryckwaert F, Coriat P. Renin angiotensin system antagonists and anesthesia. Anesth Analg. 1999 Nov;89(5):1143-55. PMID: 10553825.

Cardiovascular Anesthesiology - 18

Management of Myasthenia Gravis without Neuromuscular Blockade during Coronary Artery Bypass Grafting

Erica Zanath[1], Daniel Wyler[2]

[1]University Hospitals Cleveland Medical Center, Pepper Pike, OH, [2]University Hospitals Cleveland Medical Center, Cleveland, OH

Introduction: Myasthenia gravis (MG) is a disorder of neuromuscular weakness due to antibodies to acetylcholine receptors. There are many challenges in caring for patients with MG perioperatively. We present the successful management of a patient with MG undergoing coronary artery bypass grafting (CABG).

Methods: A 77-year-old male with MG on daily pyridostigmine was transferred to our hospital for coronary artery bypass (CABG) after NSTEMI with workup showing 90% LAD stenosis and 90% left circumflex stenosis. He was evaluated by neurology pre-operatively and deemed to have stable MG with high risk of myasthenic crisis due to lack of chronic immunosuppression. Pre-operative treatment with IVIG or plasma exchange (PLEX) was deferred due to the associated risks of inducing prothrombic state and fluid shifts with hypotension, respectively. Intraoperatively, MG in the setting of cardiac surgery posed several challenges, including 1. Avoiding neuromuscular blockade, 2. Hypothermia and risk for shivering, and 3. Avoiding medications that can exacerbate MG. Anesthesia was induced with fentanyl 100 mcg, lidocaine 80 mg, propofol 30 mg, and etomidate 14 mg. Anesthesia was maintained with isoflurane, remifentanil infusion, and dexmedetomidine infusion. He also received ketamine 140 mg and 4 mg midazolam, both in divided doses. No neuromuscular blockade was needed throughout the case. To decreased the risk of shivering during hypothermic periods, meperidine 12.5 mg was administered as an adjunct to the effects of dexmedetomidine and ketamine. During this case, medications which may exacerbate MG, including magnesium infusion, were avoided. Post-operatively, the patient was transferred to the cardiothoracic intensive care unit (CTICU) intubated, mechanically ventilated, and sedated on propofol and dexmedetomidine. On postoperative day two, the patient underwent PLEX and was subsequently extubated later that day.

Conclusion: Myasthenia gravis poses challenges for clinicians perioperatively. Treatments that may prevent myasthenic crisis, such as IVIG and PLEX, have significant risks for critically ill patients. Patients with MG have unique pharmacologic considerations - 1. sensitivity to nondepolarizing neuromuscular blockade (NMB) and 2. Risk of exacerbation with many medication classes, including magnesium, calcium channel blockers, and beta blockers (medications commonly used perioperatively in cardiac surgery). Unique to this case were the additional considerations for cardiopulmonary bypass (CPB). CPB for CABG utilizes hypothermia which may lead to shivering without NMB. Perioperative shivering causes increased oxygen demand. Several medications can decrease shivering including meperidine, dexmedetomidine, and ketamine; all of which were utilized for this purpose during our case. This case demonstrates the successful management of a critically ill patient with myasthenia gravis undergoing coronary artery bypass grafting.

References: Annals of Cardiac Anesthesia. 2020, 23(2): 209-211. J Clin Med. 2021. 10(7). Neurology. 2011. 76(23): 2017-2023. Cochrane Database Syst Rev. 2012. 12(12).

Cardiovascular Anesthesiology - 19

Massive Saddle Pulmonary Embolus during Resection of Adrenocortical Carcinoma: A Case Report

Niklas Glenesk[1], Sungyoung Lee[2], Daniel An[1], Sin Yeong An[1], Rishi Kumar[1]

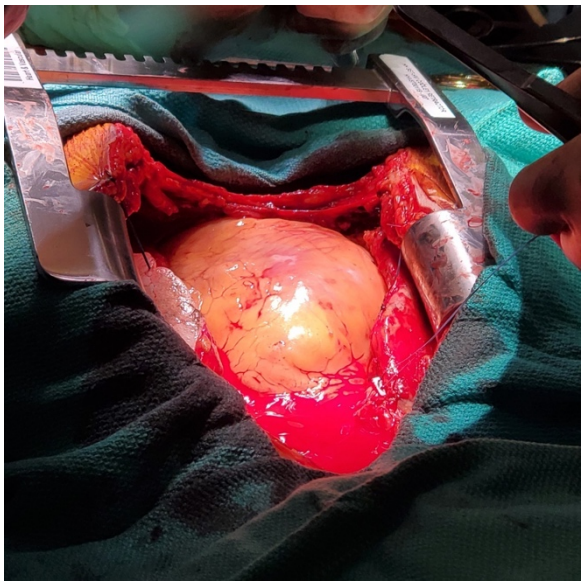
[1]University of Texas Health Science Center at Houston, Houston, TX, [2]American University of the Caribbean, Miami, FL

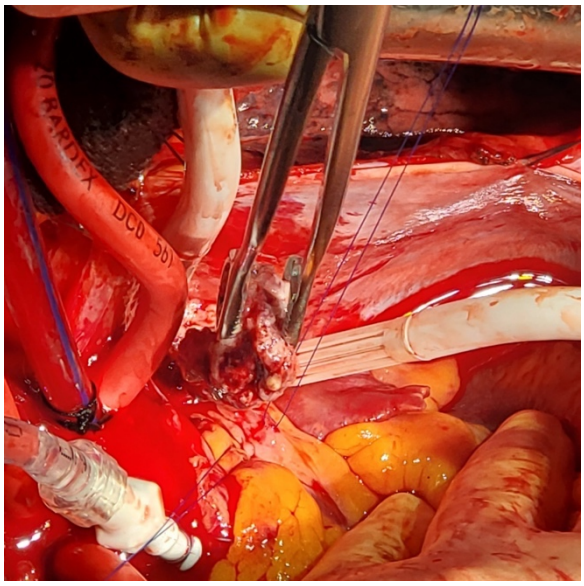
Introduction: The incidence of adrenocortical carcinomas (ACC) is extremely rare, occurring in approximately 1 in a million per year in the United States. Surgical resection is the primary recommended treatment; however, relatively little is written regarding intraoperative management of ACC resections. Our challenging case report details important anesthetic considerations for complex removal of an ACC invading the inferior vena cava (IVC) in the setting of massive pulmonary embolism (PE) during surgical resection.

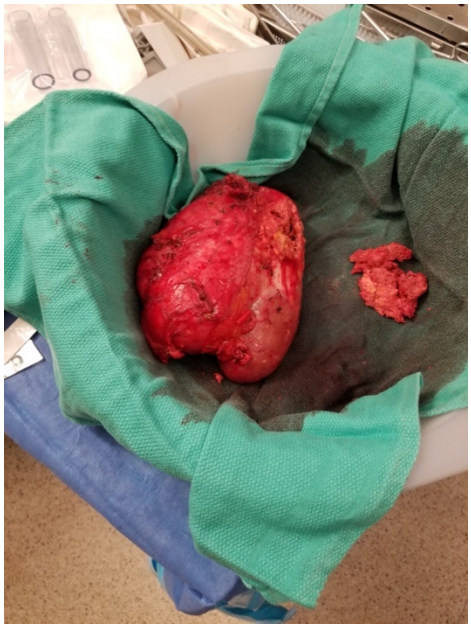
Methods: A 58-year-old female presented with abdominal pain and was found to have a large left adrenal mass (8 x 13 x 12 cm). She was transferred to a tertiary hospital for a higher level of care and scheduled for surgical tumor resection. After inducing general endotracheal anesthesia, a large-bore peripheral catheter, a 9 French introducer sheath, and an invasive arterial catheter were placed for access and monitoring. After determining the tumor was surgically resectable by initial exploration, an IVC bypass with interposition graft was planned. The tumor was observed to invade the IVC, spleen, left kidney, and liver during dissection. After further mobilization, the patient suddenly became hemodynamically unstable and went into PEA arrest. Given the high suspicion for tumor embolus, ACLS was immediately initiated with prompt ROSC. Transesophageal echocardiography (TEE) confirmed a new massive saddle PE, severe right ventricular dysfunction, and high right atrial pressure. Cardiac surgery was consulted, and the patient was placed on VA ECMO. An emergent sternotomy, pulmonary embolectomy, and PFO closure were done to remove the tumor burden from the heart. Her postoperative course was complicated by cardiogenic shock and limb ischemia from the prolonged need for ECMO. The patient passed away two weeks later.

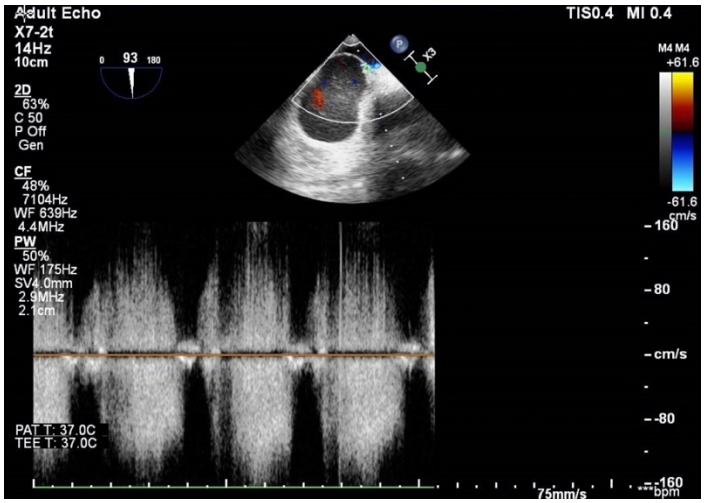
Conclusion: Though there are case reports detailing palliative surgical intervention for PE from ACC[1], or documentation of PE as an adverse sequela of ACC [2], our case is unique in its discussion of anesthetic considerations and management for ACC resection in the setting of tumor embolus. A preoperative meeting between the anesthesia and surgical teams is essential in a complex single-stage ACC resection to ensure proactive management. Standard ASA monitors, invasive arterial blood pressure monitoring, and adequate vascular access are essential to prepare for acute blood loss, labile hemodynamics, and, in the worst-case scenario, tumor embolization. TEE has been shown to provide valuable insight into PE risk in patients with IVC invading tumors[3]. The survival rate of patients with ACC resulting in IVC thrombus undergoing complete resection justifies performing this operation[4]. As seen in this case, the risk of emboli remains high in patients with advanced ACC despite preventative measures.

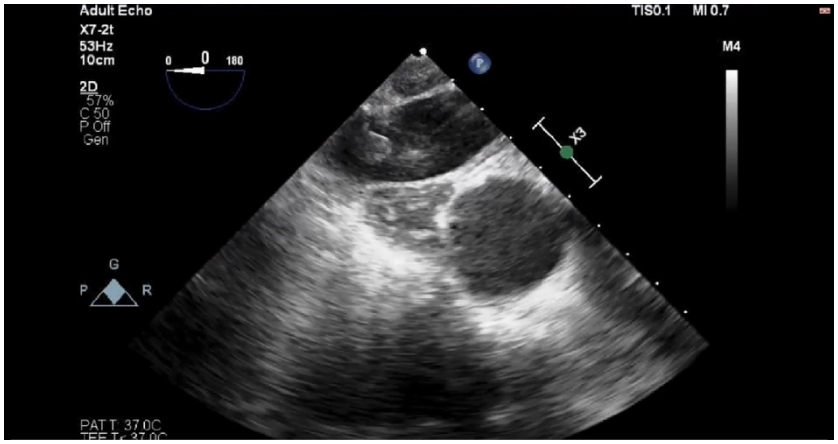
References: 1. Yunoki K, Wada T, Miyawaki I, Yamazaki K, Mima H. Anesthetic management of one-stage scheduled surgery for adrenal cortical carcinoma complicated by massive pulmonary tumor embolism. JA Clin Rep. 2017;3(1):48. doi:10.1186/s40981-017-0115-4 2. Hekhar, Skand, Sriram Gubbi, Georgios Z Papadakis, Naris Nilubol, and Fady Hannah-Shmouni. ' Adrenocortical carcinoma and pulmonary embolism from tumoral extension'. Endocrinology, Diabetes & Metabolism Case Reports 2019 (2019), 19-0095. < https://doi.org/10.1530/EDM-19-0095>. Web. 11 Nov. 2021. 3. Else T, Kim AC, Sabolch A, et al. Adrenocortical carcinoma. Endocr Rev. 2014;35(2):282-326. doi:10.1210/er.2013-1029 4. Neves RJ, Zincke H. Surgical treatment of renal cancer with vena cava extension. Br J Urol. 1987;59:390-395. doi: 10.1111/j.1464-410X.1987.tb04832.x 5. Laan DV, Thiels CA, Glasgow A, et al. Adrenocortical carcinoma with inferior vena cava tumor thrombus. Surgery. 2017;161:240-248. doi: 10.1016/j.surg.2016.07.040.








Adult Echo
TIS0.4 MI 0.4
X7-2t
14Hz
10cm
2D
63%
C 50
P Off
Gen
CF
48%
7104Hz
WF 839Hz
4.4MHz
PW
50%
WF 175Hz
SV4.0mm
2.9MHz
2.1cm
M4 M4
+61.6
-61.6
cm/s
160
80
cm/s
-80
-160
PAT T: 37.0C
TEE T: 37.0C
75mm/s
bpm


Adult Echo
TIS0.1 MI 0.7
X7-2t
53Hz
10cm
2D
57%
C 50
P Off
Gen
M4
G
P
R
PAT T: 37.0C

Cardiovascular Anesthesiology - 20

Anterior mediastinal mass anesthetic management in an adult

Jocelyn Wang[1], Michael L Hall[2]

[1]Stanford, Palo Alto, CA, [2]University of Washington Medical Center, Seattle, WA

Introduction: Anterior mediastinal masses (AMM) present many anesthetic challenges given the compression of the great vessels, airway, or heart. The supine position can obstruct the airway and/or vasculature, impeding ventilation and/or cardiac function (1). Life-threatening complications during positive pressure ventilation (PPV) are reported in 20% of cases, so moderate sedation is preferred where applicable (2).

Methods: A 36 yo female with known AMM presented with dyspnea, increase in O2 requirements, and atrial fibrillation with rapid ventricular response. Chest CT showed rapid increase in mass size and pre-existing pulmonary artery PE with new severe tracheal/heart deviation, cardiac compression, restricted ventilation, and left lung collapse (fig 1). The patient was orthopneic, sleeping only in a tripod or sitting position. IR embolization of vessels to the mass was done under sedation with spontaneous ventilation. Back-up plans, general anesthesia (GA) or ECMO, were not used. On hospital day 4, she underwent resection via clamshell incision. She was sedated with head above 30¬∞ to avoid mediastinal compression as femoral access was obtained. The surgeons remained scrubbed in case of urgent cardiopulmonary bypass (CPB) while GA was induced with ketamine. A small dose of succinylcholine was given and once bag mask ventilation was achieved, the full intubating dose was given. Her head stayed elevated and direct laryngoscopy (DL) and intubation was uncomplicated. A central venous catheter (CVC) was placed in the right internal jugular (IJ) vein with careful ultrasound (US) and fluoroscopy confirmation of the wire prior to dilation and placement of the catheter. The surgery was uncomplicated. She was transported to the cardiothoracic intensive care unit still intubated, where post-operative course was uncomplicated.

Conclusion: AMM management for pediatric cases is common, but management for adults is not prevalent in the literature and this case highlights different types of anesthetic management. PPV is associated with worse outcomes but spontaneous ventilation has also led to cardiopulmonary collapse, so risks and benefits of GA must be considered (3). For the IR procedure, sedation was sufficient, but for the surgery, GA was the only option. This case also demonstrates successful DL in AMM, despite the size of the mass. The few reported adult cases performed awake fiberoptic intubations. Symptomatic adults with CT measured tracheal diameter <50% of normal have a higher risk of complications, and the tracheal diameter was 17mm, which was >50% of normal (fig 2). Given her overwhelmed state and lack of other difficult airway risk factors, we elected to do DL incrementally: ketamine induction to avoid hemodynamic changes, test succinylcholine and bag mask ventilation before giving the full dose, and intubation with head of bed at 30¬∞. Vascular compression is a known complication of AMM and US and fluoroscopy should be used intra-operatively if concerns arise. US confirmed patency of the right IJ vein and wire placement was checked with US and fluoroscopy prior to dilation/placement of the catheter. A pulmonary artery catheter was not used due to the concern for vessel tortuosity and compression. Lastly, CPB should always be considered for AMMs. One other case prepared for femorofemoral CPB prior to induction and electively initiated it (4). While the surgical team was ready for urgent CPB, we were able to avoid bypass for the entire case.

References: 1. Canadian Journal of Anesthesia. 2011; 58.9: 853. 2. The Journal of the American Society of Anesthesiologists. 2004;100.4: 826-834. 3. Anesthesia & Analgesia. 2011;113.2: 239-242. 4. Indian Journal of Anaesthesia. 2010; 54.6: 565.

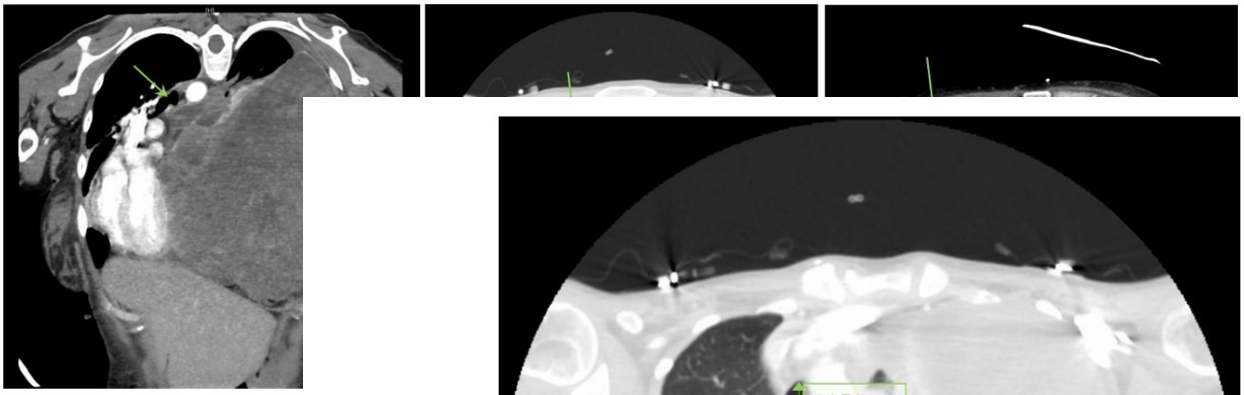


Figure 1 (a) Coronal view
(b) Transverse view de
carina. (c) Transverse vi

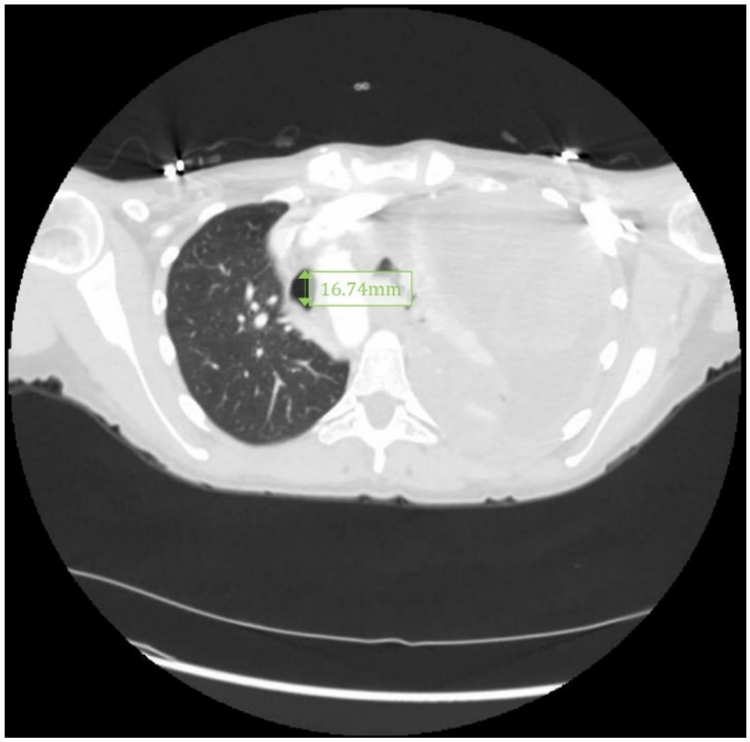


Figure 2 Transverse cross-sectional view of trachea, measuring 16.74mm in diameter

Critical Care

Critical Care - 1 Case of Kodamaea ohmeri Native Aortic Valve Infective Endocarditis in Immunocompetent Patient

Andrew J Gallagher[1], McKenna Schimmel[2], Cindy Noyes[1], Fuyuki Hirashima[1], John Klick[1]

[1]University of Vermont Medical Center, Burlington, VT, [2]University of Vermont, Burlington , VT

Introduction: Kodamaea ohmeri, formerly Pichia ohmeri or Yamadazyma ohmeri, is a rare cause of invasive, fungal infection in humans. K. ohmeri is the telomorphic form of Candida guilliermondii complex and is often misidentified as Candida albicans. This uncommon pathogen has been isolated from sand, seawater, pools, and fruit.1 Known risk factors for K. ohmeri infection include catheter insertion, diabetes mellitus, severe burns, cancer, parental nutrition, and immunosuppression. It has previously been identified as the infectious culprit in cases of fungemia and onychomycosis.1,2 However, K. ohmeri infective endocarditis has seldom been described in the literature, and native valve infection is even more obscure. Herein, we describe the 19-day hospital course of a patient with K. ohmeri native aortic valve endocarditis, with a course complicated by two episodes of Torsades de Pointes arrest attributed to stress cardiomyopathy.

Methods: A 40-year-old-female with a history of depression on fluoxetine and intravenous opiate abuse, presented with worsening shortness of breath. On exam, she was hypotensive, tachypneic, with bilateral lower extremity edema. Labs were significant for lactic acidosis and elevated BNP. EKG demonstrated sinus tachycardia with QTc of 488mS. TTE demonstrated a left ventricular ejection fraction of 10% with aortic valve vegetations and severe aortic regurgitation. Blood cultures grew yeast, and empiric micafungin was started. She was admitted and underwent tissue aortic valve replacement. Blood and valve cultures (Figure 1) grew K. ohmeri, therefore liposomal amphotericin was started. On POD3, fluoxetine was re-started. On POD4, the patient developed ventricular bigeminy which progressed to Torsades de pointes (TdP) cardiac arrest with return of spontaneous circulation after one round of compressions and 200J defibrillation. The QTc on ECG was 515mS at this time. She was transitioned back to micafungin and fluoxetine was discontinued, over concerns this agents may prolong QTc. The patient continued to have daily 6-12 beat runs of ventricular tachycardia. On POD8, she again had TdP arrest, with ROSC after a 200J shock. QTc was 636ms at this time. Isoproterenol was started, and later a temporary atrial pacer was placed for a goal of maintaining heart rates above 90BPM, thereby shortening QTc. A repeat echocardiogram showed an EF of 35% and the patient underwent left heart catheterization, showing only mild coronary artery disease. She was diagnosed with stress cardiomyopathy. The patient left against medical advice on POD16, therefore intravenous micafungin was discontinued and oral isavuconazonium was initiated.

Conclusion: Fungal endocarditis remains rare, though particularly deadly. It is associated with intravenous drug use, urinary catheters, immunosuppression, and prolonged use of antibiotics.3 Most commonly, fungal endocarditis is caused by Candida and Aspergillus species, though correct identification often proves difficult due to nonspecific clinical manifestations. Recently, K. ohmeri has been increasingly isolated as an opportunistic pathogen. A recent review in the publication Mycoses, (Ioannou, 2020) included 35 publications of patients with K. ohmeri infections, which included 44 patients. Sole fungemias were described in 36 of the 44 cases. Cases of infective endocarditis were described in four of the forty-four cases, yet our report describes the first native aortic valve K. ohmeri infectious endocarditis in an immunocompetent individual. Treatment options for K. ohmeri include amphotericin, echinocandins, flucytosine, and azoles, though this organism is generally resistant to fluconazole. However, susceptibility testing has found amphotericin to have the lowest MIC90 when treating K. ohmeri.4,5 For this reason, the patient was transitioned from micafungin, which was initially selected due to her renal dysfunction, to liposomal amphotericin once K. ohmeri was identified. Subsequently, the patient experienced two TdP arrests despite electrolytes being within normal limits. Therefore, micafungin was re-initiated due to concerns over cardiotoxic effects and possible QTc prolongation by amphotericin. Though typically used for invasive aspergillosis and mucormycosis, the

patient was given oral isavuconazonium, the only azole believed not to prolong QTc, upon eventual outpatient follow up after leaving against medical advice.

References: 1. Ioannou P, Papakitsou I. Kodamaea ohmeri infections in humans: A systematic review. Mycoses. 2020;63(7):636-643. 2. Tashiro A, Nei T, Sugimoto R, et al. Kodamaea ohmeri fungemia in severe burn: Case study and literature review. Medical Mycology Case Reports. 2018; 22:21-23. 3. Negi N, Ahmad A. Current updates on fungal endocarditis. Fungal Biology Reviews. 2018;32(1):1-9. 4. Shang, ST, The Emerging Life-threatening Opportunistic Fungal Pathogen Kodamaea ohmeri: Optimal Treatment and Literature Review. Journal of Microbiology, Immunology, and Infection. 2010;43(3):200-206. 5. Zhou M, Yu S, Kudinha T, Meng X, Wang H, Xu Y, Zhao H. Identification and antifungal susceptibility profiles of Kodamaea ohmeri based on a seven-year multicenter surveillance study. Infect. Drug Resist. 2019; 12:1657-1664.

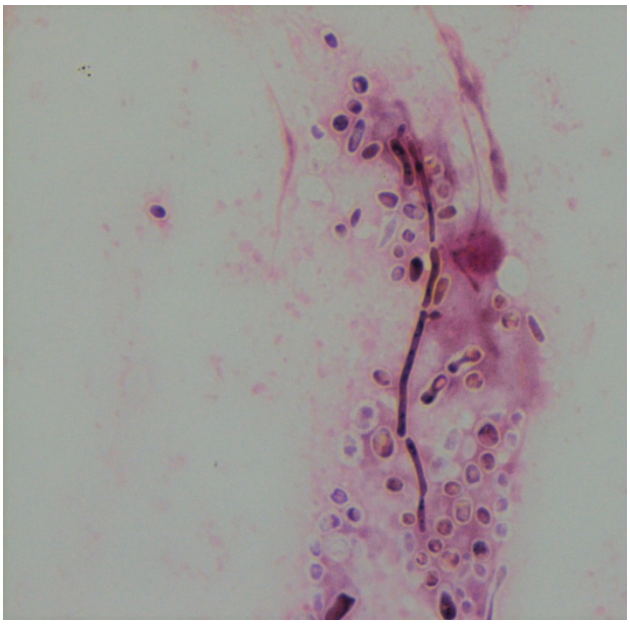


Figure 1: Gram stain (400X) prepared from the patient's aortic valve tissue showing gram positive pseudohyphae and gram variable budding yeast. Courtesy of Drs Cindy Noyes, MD and Jonathon Wilcock, DO.

Critical Care - 2 Successful utilization of V-PA ECMO; a new approach for circulatory and respiratory failure; for a patient with pulmonary edema following intraoperative cardiopulmonary arrest due to uterine rupture.

Takeshi Nagaoka[1], Tomohide Takei[2], Hitoshi Sato[3], Hiromasa Kawakami[3]

[1]Yokohama City University Medical Center, Yokohama City, Japan, [2]Yokosuka Kyosai Hospital, Yokosuka, Japan, [3]Yokohama City University Medical Center, Yokohama, Japan

Introduction: ECMO (extra corporeal membrane oxygenation) is a mechanical supportive device of providing prolonged cardiac and respiratory support to a patient with life threatening circulatory and/or respiratory failure. The site where the oxygenated blood is returned to lies on the patient's pathophysiology, which are mostly V-V(veno-venous) or V-A(veno-arterial). However, both approaches have disadvantages as well as advantages. V-PA (veno-pulmonary arterial) ECMO has recently arisen as a novel approach which compensates for the weakness of the traditional approaches. Herein, we report a case which V-PA ECMO was thought beneficial as a mechanical support for respiratory failure accompanied by pulmonary edema after CPA (cardiopulmonary arrest) during urgent hysterectomy. To our knowledge, no cases have been reported establishing V-PA ECMO in a gynecologic surgery.

Methods: A 41-year-old healthy female was transferred to our hospital with the diagnosis of uterine rupture after vaginal delivery representing shock. Subsequently, emergent abdominal total hysterectomy was planned. Soon after her surgery had begun, her electrocardiogram waveform changed to wide QRS complex and subsequently progressed to asystole. In addition to immediate cardiopulmonary resuscitation, treatment for detected hyperkalemia was conducted and thoracotomy was simultaneously performed in order to introduce central ECMO. As a result, ROSC (return of spontaneous circulation) was achieved 24 minutes after asystole recognition, before ECMO introduction. Although circulation after ROSC was stable, progressive hypoxemia and hypercapnia presumably related to pulmonary edema, accompanied by massive blood transfusion, was observed. As hemodynamics including cardiac function had recovered, optimal blood oxygenation with less recirculation was considered more essential at this moment and having the fact that thoracotomy had already been performed, we decided to establish V-PA ECMO and the operation was accomplished free from any other adverse events. Respiratory function gradually improved after active diuretic therapy. On POD(postoperative day) 7, V-PA ECMO was explanted and subsequently she was extubated on POD 9. Finally on POD 21, she was discharged without physical nor neurological impairment.

Conclusion: Although the patient once became CPA, its cause, which we considered to have been hypovolemia and hyperkalemia, was treatable and hemodynamics actually got stable soon after ROSC. In addition, anticoagulant therapy was of hesitation after experiencing hemorrhagic shock. Furthermore, under the circumstances of pulmonary edema, less preload to the right ventricle and less recirculation was considered to be ideal for optimal blood oxygenation and systemic circulation. Thus, we selected V-PA ECMO instead of V-A or V-V. V-PA ECMO was effective for respiratory failure accompanied by pulmonary edema following intraoperative cardiopulmonary arrest caused by uterine rupture.

Critical Care - 3 Cardiac arrest secondary to sulfur hexafluoride lipid-type A anaphylactic shock

Charlota Jurcik[1], Syed Afzal[2], Reza Garajehdaghi[3], Carmine Gianatiempo[4], Michael Basir[2]

[1]NJMS Rutgers, Bayonne, NJ, [2]Englewood Hospital Medical Center, Englewood, NJ, [3]Rutgers NJMS, Newark, NJ, [4]Englewood Hospital Medical Center, Englewood, United States of America

Introduction: Review a case of adverse reaction to sulfur hexafluoride lipid-type A microspheres contrast in the indication for echocardiograms.

Methods: 33 year old male with a past medical history of obesity, HTN, NIDDM2, HLD, OSA, asthma presented for an outpatient echocardiogram as part of his workup for bariatric surgery clearance. Due to patient's habitus, anatomical structures were not appreciated, as a result sulfur hexafluoride IV contrast was injected. However, right after injecting the contrast, patient became hypoxic with wheezing, diaphoretic, and cyanotic. Before any interventions could be performed, patient went into cardiac arrest. CPR was commenced and after one round of CPR and one dose of epinephrine, ROSC was obtained. Patient remained diaphoretic, hypoxic and agitated and the decision was made to intubate patient for airway protection after which patient was transferred to the ICU. Patient remained hypotensive and required both norepinephrine and epinephrine to maintain his MAPs. Patients hemodynamics however improved quickly over the next 24 hours and he was weaned of pressors and successfully extubated the next day. Patient underwent diagnostic catherization the following day which demonstrated patent coronaries and patient was cleared for discharge to home the next day.

Conclusion: Sulfur hexafluoride lipid-type A microspheres is an injectable suspension used intravenously or intravesically as a contrast used for ultrasonography. It provides signal intensity for two minutes; its peak effect occurs in two minutes and its half life last about ten minutes. It has been indicated for use in echocardiography to opacify the left ventricle with the intent to improve suboptimal echocardiograms. It is also indicated to obtain sonographic characterization of focal hepatic lesions and to evaluate vesicoureteral reflux in children. The producers of this contrast have reported possible but uncommon serious cardiopulmonary reactions which includes fatalities. It is mention that reactions most commonly presents within thirty minutes after administration. It also states that risk for these reactions commonly occurs among patients with unstable cardiopulmonary conditions such as acute MI, acute coronary artery syndrome, worsening or unstable congestive heart failure. It is also contraindicated on patient with known reaction to it or to its similar component, such as polyethylene glycol (PEG). Our patient however had no such allergy history, nor did he have any evidence of heart failure or coronary occlusions. He presented to an ambulatory setting for an echocardiogram as part of his cardiac clearance for bariatic surgery. This case report highlights the importance of further studying sulfur hexafluoride as the potential risks of this agent can be significant even in patients with relatively healthy underlying physiology.

References: LUMASON® (sulfur hexafluoride lipid-type A microspheres) for injectable suspension, for intravenous use or intravesical use. November 5, 2021. https://imaging.bracco.com/us-en/products/contrast-enhanced-ultrasound/lumason
Lumason (sulfur hexafluoride) dosing, indications, interactions, adverse effects, and more. Medscape. November 5, 2021. https://reference.medscape.com/drug/lumason-sulfur-hexafluoride-999971

Critical Care - 4 A Pitfall of Intravenous Sedative Route in patients with VA-ECMO: A Case Report

Mirei Kubota[1], Junichi Saito[2], Satoshi Uchida[2], Daiki Takekawa[2], Satoko Noguchi[3], Kazuyoshi Hirota[4]

[1]Hirosaki university graduate school of medicine, Hirosaki, Japan, [2]Hirosaki University Hospital, Hirosaki, Japan, [3]Hirosaki University Hospital, Hirosaki-shi, Aomori-ken, [4]Hirosaki University Graduate School of Medicine, Hirosaki, Japan

Introduction: Sedation and analgesia during veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is essential to minimize patient discomfort associated with treatments including mechanical ventilation and to secure the ECMO circuit. We report a case in which an inadequate level of sedation occurred as a patient's cardiac output (CO) increased during VA-ECMO support. This may have been due to the increase in the recirculation of blood in the lower body with sedatives through the VA-ECMO circuit, as we changed the sedatives' intravenous (i.v.) route from the lower extremity to the right internal jugular vein, and an adequate level of sedation was then obtained.

Methods: A 70-year-old Japanese man who received an implantable cardioverter defibrillator (ICD) against ventricular tachycardia (VT) following an old myocardial infarction developed a VT storm after suffering enterogastritis and in-stent re-stenosis of the right coronary artery at another hospital. An emergency percutaneous coronary intervention (PCI) against the re-stenosis was performed, but immediately after the PCI the VT storm was refractory. VA-ECMO was introduced with a blood drainage cannula in the right femoral vein and a blood return cannula in the left femoral artery. The patient was then transferred to our hospital for catheter ablation therapy against the refractory VT.
After the ablation therapy he was admitted to the ICU. His clinical course in the ICU is depicted in Figure 1. Just after the ICU admission he was deeply sedated with the Glasgow Coma Scale (GCS) value E1V1M1 and the Richmond Agitation Sedation Scale (RASS) value −5 under a continuous administration of propofol (2 mg・kg−1・h−1), dexmedetomidine (0.4 μg・kg−1・h−1), and fentanyl (20 μg・h−1). At that time, his CO was 2.7 L・min−1, mixed venous oxygen saturation (SvO2) was 64%, and the saturation of the blood in the drainage cannula (SV-ECMO O2) of VA-ECMO was 65% with the assist of VA-ECMO flow at 2.9 L・min−1. On the second day in the ICU, his CO gradually increased to 4.8 L・min−1 and the ECMO flow decreased to 2.5 L・min−1 with a continuous i.v. administration of noradrenalin (0.025 μg・kg−1・min−1), dobutamine (2 μg・kg−1・min−1), and olprinone (0.15 μg・kg−1・min−1). The patient became fully awake (GCS E3V5M6 and RASS 0–1) even though the doses of sedatives and analgesics were increased (propofol 3 mg・kg−1・h−1, dexmedetomidine 0.6 μg・kg−1・h−1, fentanyl 26 μg・h−1, and midazolam 0.1 mg・kg−1・h−1). He was able to communicate in writing even after a bolus administration of morphine (5 mg), propofol (60 mg), and midazolam (8 mg) through the peripheral route in the right lower extremity. We observed a dissociation of venous oxygen saturation between the SVO2 and SV-ECMOO2 with the values of 74% and 95%, respectively, indicating a certain amount of recirculation of blood in the lower body through the VA-ECMO circuit. As there was a possibility that the upper body received sedative-poor blood because the sedatives were administered into a peripheral vein in the right lower extremity, we changed the i.v. route of sedatives from the lower extremity to the right internal jugular vein. The patient then instantly lost consciousness without a bolus administration of sedatives. After that we were able to control the sedation levels without any difficulty. The patient was weaned from VA-ECMO on day 3 in the ICU, without VT arrhythmia. He was moved to the general ward on day 5 in the ICU and discharged to home 20 days after the ICU admission.

Conclusion: The intravenous route of sedatives for patients being treated with VA-ECMO with a femoral-vein drainage cannula and a femoral-artery return cannula should be in the upper body, because recirculation of blood in the lower body through the VA-ECMO circuit may occur after a patient's cardiac output increases.

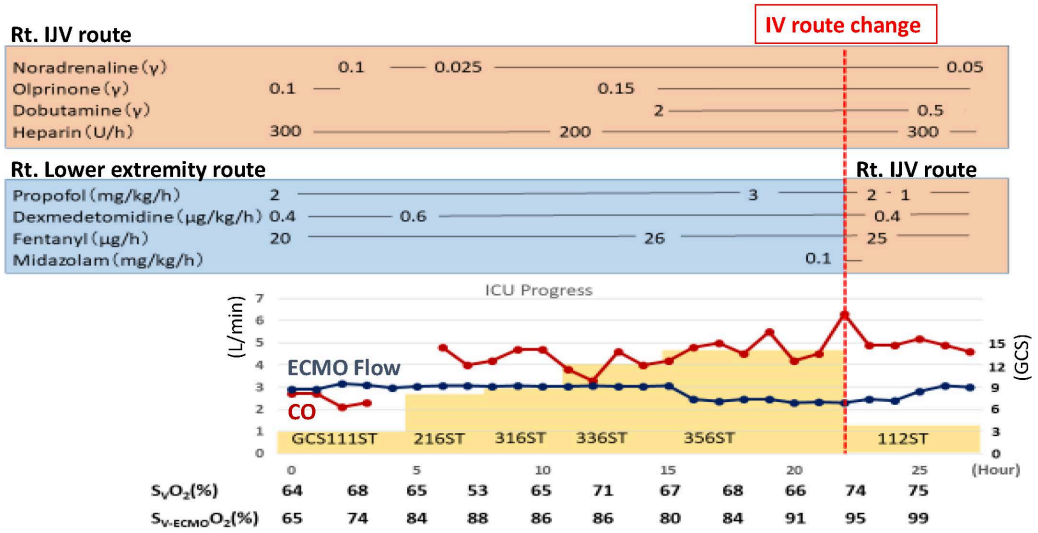


Fig. 1 Changes in the patient's consciousness levels before and after the change in the i.v. route for sedatives. At his ICU admission, he was deeply sedated and his CO was low; the S_VO_2 was 64% and the $S_{V\text{-}ECMO}$ O_2 of VA-ECMO was 65%. After his CO increased, he became fully awake despite the sufficient dose of sedatives. At the time, the S_VO_2 was 74% and the $S_{V\text{-}ECMO}$ O_2 was 95%. After the i.v. route of sedatives was changed from the lower extremity to the right internal jugular vein, he instantly lost consciousness. CO: cardiac output, ECMO: extracorporeal membrane oxygenation, GCS: Glasgow Coma Scale, S_VO_2: mixed venous oxygen saturation, $S_{V\text{-}ECMO}$ O_2: the saturation of the blood in the drainage cannula.

Critical Care - 5 COVID-19 Related Uncontrolled Psychosis in Well-controlled Schizophrenia Patient

Sungyoung Lee[1], Yaroslav Buryk[2]

[1]American University of the Caribbean, Miami, FL, [2]University of Miami, Miami, FL

Introduction: The management of patients with coronavirus disease 19 (COVID-19) comorbidities has become an area of increased study, particularly as the global disease burden of COVID-19 has reached unprecedented levels (1). Growing bodies of evidence have shown the increased risk of COVID-19 complications among patients with mental health disorders such as schizophrenia (1). The identification of best practices when treating these patients is essential in reducing COVID-19 disease burden, as well as ensuring safety for the patient and healthcare providers. Here, we report a complex case demonstrating the challenges of intubation and airway management of a patient with COVID-19 related uncontrolled psychosis and diagnosed schizophrenia.

Methods: A 30-year-old African American female was admitted for care based on suspicion of COVID-19. The patient's medical history was significant for hypertension, diabetes, and schizophrenia. Upon admission, the patient was initially placed on risperidone 2mg TID, prednisone IV 6mg BID, Depakote 500mg BID, two IV treatments of Haldol, as well as olanzapine 5mg IM twice per day. The patient continued Precedex drip, which was eventually increased to a maximum dosage. Depakote was increased to 750mg BID on day 6 of treatment. The vaccination status for COVID-19 was negative and suspected COVID-19 related symptoms included 1 week history of fever, cough, shortness of breath and sore throat. After subsequent testing, the patient reported as COVID-19 positive. The patient rapidly developed severe hypoxia, evidenced by O2 saturation levels in the forty percentile. The patient was placed on high flow nasal cannula oxygen on the maximal settings. Initially, the treatment was well received but the patient began to demonstrate signs of agitation and confusion. Eventually the patient demonstrated uncontrolled psychosis, becoming combative and requiring restraint to prevent harm to themselves and others. She remained delusional and confused, believing that she was pregnant and describing multiple hallucinations. Additional hospital staff were immediately called to assist in restraining the patient, as she had physically overpowered her sole 1:1 sitter. Psychiatric consultation recommended careful sedation of the patient to prevent harm and allow continued COVID-19 related management. Intervention with Depakote 25mg IM was ineffective, necessitating additional intervention. Additional medical measures were immediately taken including administering maximal levels of dexmedetomidine drip and increased dosages of antipsychotics (Thorazine, risperidone). After successful sedation, the patient was intubated. Additional testing including lumbar puncture, brain CT and blood work were taken at this time to rule out other potential causes of psychosis. Brain CT without contrast showed no acute intercranial hemorrhage, extra-axial collection, mass effect, hydrocephalus, or infarction. Brain CT scan and lumbar puncture results presented with no findings that correlated with the new onset of psychosis seen in the patient.

Conclusion: Early models of dexmedetomidine in COVID-19 patients suggests its effects reduce ischemia-reperfusion injury in the brain (2). Scientific literature has shown evidence that human coronaviruses possess the capacity to be invasive neurologically by spreading to the central nervous system from the respiratory tract (3). This case demonstrated the use of dexmedetomidine drip combined with antipsychotics to successfully treat COVID-19 related uncontrolled psychosis in a critical care setting. The sedation of the patient allowed psychiatric testing (lumbar puncture, CT scan, blood work) in addition to managing the COVID-19 associated hypoxia symptoms. Because the novelty of the COVID-19 virus, physicians should not hesitate to obtain peer consultation in patients with multi-system involvement and form a multi-disciplinary treatment plan. A multi-disciplinary approach provided an effective response to the unexpected psychotic episodes demonstrated by the patient. Psychiatric consult, critical care, pulmonology, technicians, and nurses all provided valuable contributions in ensuring a safe, successful outcome. This perspective of care should be used universally during the COVID-19 pandemic to improve patient outcomes and provider safety.

References: 1. Increased risk of COVID-19 infection and mortality in people with mental disorders: analysis from electronic health records in the United States. World Psychiatry. 2021 Feb;20(1):124-130. 2. The effect of dexmedetomidine and clonidine on the inflammatory response in critical illness: a systematic review of animal and human studies. Crit Care. 2019;23(1):402. 3. Human coronaviruses: viral and cellular factors involved in neuroinvasiveness and neuropathogenesis. Virus Res. 2014;194:145-158.

Critical Care - 6 Fibrosing Mediastinitis Presenting with SVC Syndrome

Jee Ha Park[1], Jon Lucaj[1], Krassimir Denchev[1]

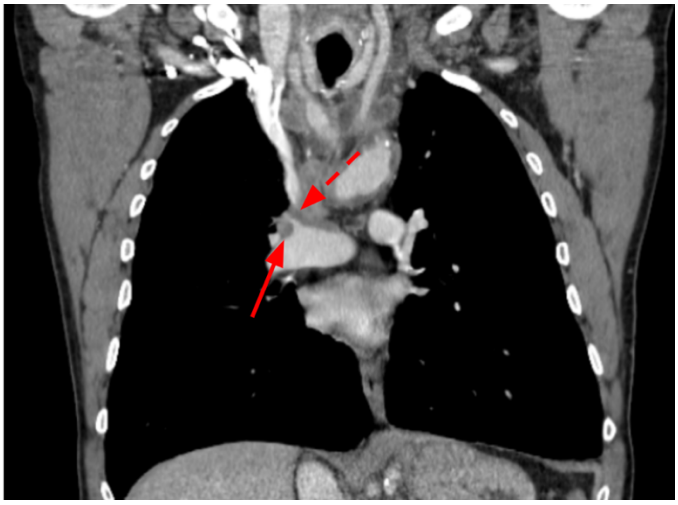
[1]Wayne State University - Detroit, Detroit, MI

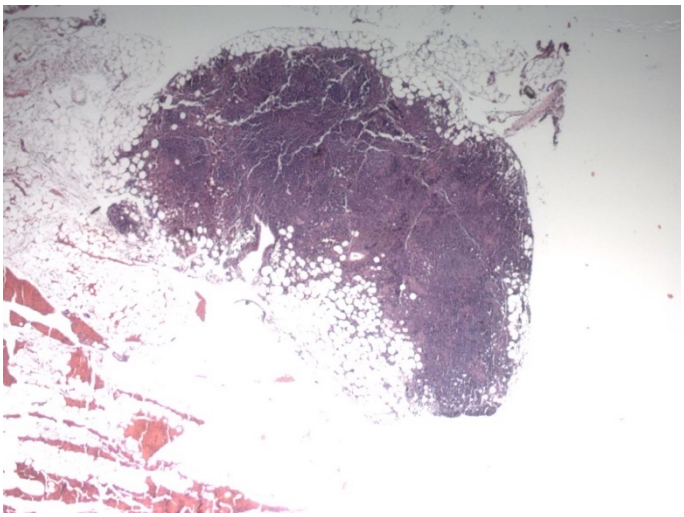
Introduction: Fibrosing mediastinitis (FM) is an uncommon but fatal disease without a clear treatment plan. It is a rare complication of Histoplasma capsulatum characterized by excessive fibrotic reaction in the mediastinum resulting in compromise of surrounding anatomy. It accounts for less than 1% of people infected [1], induced by hypersensitivity immune response to antigen released during the exposure rather than acute granulomatosis [2,3]. Other causes include tuberculosis, blastomycosis, sarcoidosis, radiation, and idiopathic.

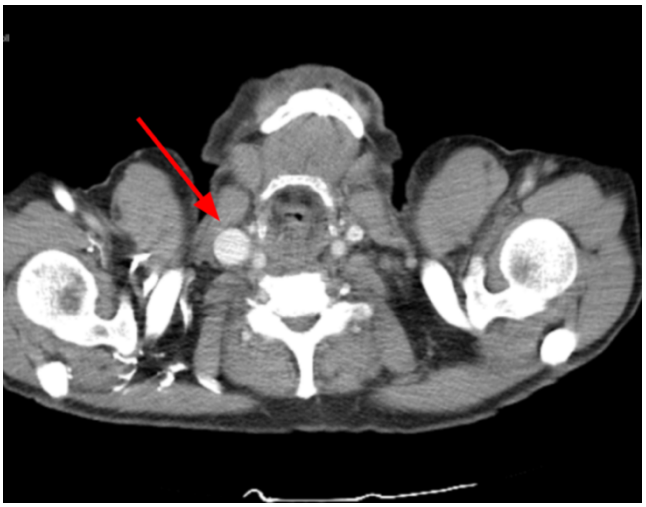
Methods: We present a case of fibrosing mediastinitis in a patient with superior vena cava (SVC) syndrome. A 66-years old Caucasian male presented with a one-week history of progressively worsening facial swelling associated with dysphonia, bilateral ptosis, dyspnea on exertion, and unintentional weight loss of 30 pounds. Computer topography of the chest demonstrated nonspecific soft tissue extending throughout the mediastinum and towards the right hilar region complicated by severe attenuation of SVC and a 2.4x1.6 centimeter necrotic lymph node. The mediastinum had hyperemic and desmoplastic changes heavily encased in venous collaterals. Pathological evaluation demonstrated sinus histiocytosis and reactive lymphoid hyperplasia without signs of malignancy or atypia. Patient was treated with corticosteroid and diuretics, which failed to prevent further progression of the disease.

Conclusion: Our case demonstrates rapid deterioration of FM presenting with SVC syndrome. The patient did not respond adequately to the treatments provided. Only case reports have demonstrated mixed symptomatic and radiologic responses to anti-inflammatory and/or antifungal treatment. Nonsurgical interventional option for patients with SVC syndrome is endovascular balloon angioplasty with or without stenting; whereas, possible surgical intervention includes resection of the mass and/or vascular reconstruction. Even in successful cases, restenosis and re-exploration are often warranted.

References: 1. Wheat, L. J., Slama, T. G., Eitzen, H. E., Kohler, R. B., French, M. L., & Biesecker, J. L. (1981). A large urban outbreak of histoplasmosis: clinical features. Annals of internal medicine, 94(3), 331-337. https://doi.org/10.7326/0003-4819-94-3-331 2. Strock, S. B., Gaudieri, S., Mallal, S., Yu, C., Mitchell, D., Cogan, J., Mason, W., Crowe, D., & Loyd, J. E. (2015). Fibrosing mediastinitis complicating prior histoplasmosis is associated with human leukocyte antigen DQB1*04:02 - a case control study. BMC infectious diseases, 15, 206. https://doi.org/10.1186/s12879-015-0943-7 3. Loyd, J. E., Tillman, B. F., Atkinson, J. B., & Des Prez, R. M. (1988). Mediastinal fibrosis complicating histoplasmosis. Medicine, 67(5), 295-310. https://doi.org/10.1097/00005792-198809000-00002







Summary of Treatment Modalities	
Medical	
Anti-inflammatory (corticosteroid)	• May control the inflammatory process • Mixed success with unclear efficacy
Anti-fungal	• FM is not an acute fungal infection • Mixed success with unclear efficacy
Interventions	
Nonsurgical	• Balloon angioplasty with or without stent • Good initial symptom relief but high rate of reintervention
Surgical	• Perioperative risk • Resection vs. Vascular reconstruction • Good longer term symptomatic relief

Table 1: Summary of treatment modalities.

Critical Care - 7 Case of VV ECMO for Near Drowning ARDS Complicated by Aspergillus Pneumonia

Piper L Nash[1], Tichaendepi Mundangepfupfu[2]

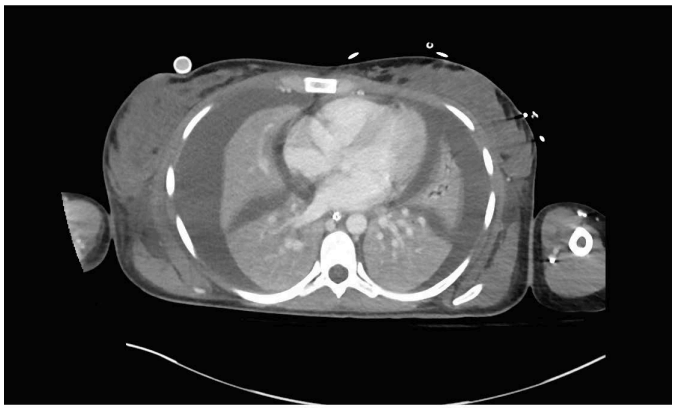
[1]University of Washington, Seattle, WA, [2]University of Rochester, Medical Center, Rochester, NY

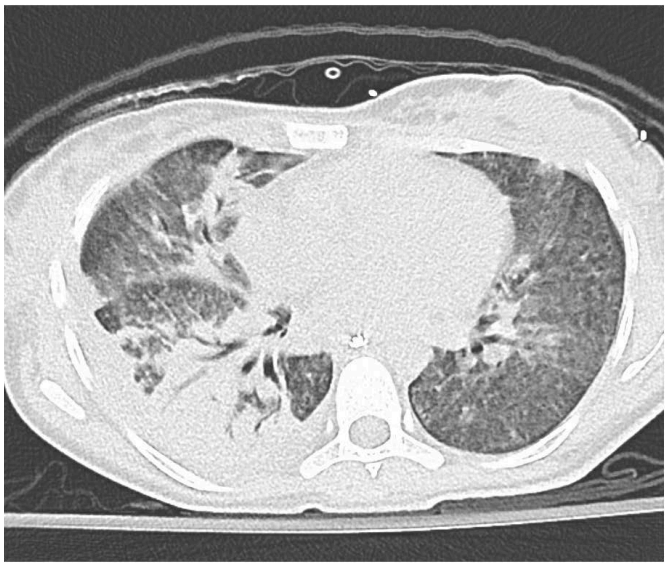
Introduction: Veno-Venous ECLS is indicated for acute, severe, reversible respiratory failure. Current indications include drowning and pneumonia. This case report details Aspergillus fumigates as a severe and likely pathogen in near drowning aspiration and pneumonia.

Methods: A 21 year old healthy female was resuscitated after MVC rollover into a water-filled ditch. Paramedics performed CPR and intubated her for PEA due to respiratory arrest. She was transferred to our hospital for VV-ECLS. She arrived on volume control with SpO2 86% on 100% FiO2 and PEEP 5, heart rate 128, and normal blood pressure. The initial chest CT demonstrated significant bilateral opacities and pleural effusions. Due to refractory respiratory failure she was cannulated onto VV-ECLS in the ED. TTE demonstrated global LV hypokinesis treated with dobutamine while on ECLS. Overnight, our patient's pulmonary and cardiac function improved with improved lung compliance, oxygenation, pulmonary edema, and off dobutamine so the team planned to decannulate on hospital day 3 (HD3). Unfortunately, she acutely worsened with Tmax 37.7, tachypnea, hypoxia, reduced lung compliance, and chest radiograph concerning for acute pneumonia. She underwent BAL which was initially unrevealing so testing was broadened. BAL fluid PCR was positive for Aspergillus and she was broadened to voriconazole, meropenem, and micafungin. After this she clinically improved and on HD16 she was decannulated and extubated. She was discharged from the ICU on HD 19 and went home in stable condition and neurologically intact on HD25.

Conclusion: Annually, drowning effects about 4000 individuals in the United States and globally approximately 88,000 people die from drowning. The estimated survival rate is 2-8% in those who experience cardiac arrest after drowning. Notably, our patient's pulmonary function rapidly improved on VV-ECLS until she acutely decompensated overnight due to acute pneumonia. This diagnosis may have been delayed as her temperature was controlled on VV-ECLS with ice preventing the patient from mounting a fever. Steadily improved after treatment for Aspergillus. Pneumonia is a common complication of near-drowning due to aspiration of foreign materials or upper airway material. Aspergillus is a ubiquitous mold in soil, seawater, polluted water, and sewage but this organism is classically associated with immunocompromised hosts. Aspergillus is a more common pathogen in pneumonia complicating near drowning events among immunocompetent hosts than may be widely believed. Case reports of near drowning demonstrate Aspergillus as a common organism causing life threatening invasive pneumonia. Therefore, after a near drowning event, a broad infectious workup including fungal organisms should be sent.

References: Resuscitation, volume 104, pages 19-23, 2016. Infectious Disease Clinical Practice, volume 23, pages 227-230 2015. Annals of Pharmacotherapy, volume 48, pages 1125-1129, 14.





Critical Care - 8 Intermittent Superior Mesenteric Artery Occlusion by Axillary Intra-Aortic Balloon Pump: An Unusual Cause of Bowel Necrosis

Kumar Seelam[1], Miguel A Yaport[1]

[1]Tufts Medical Center, Boston, MA

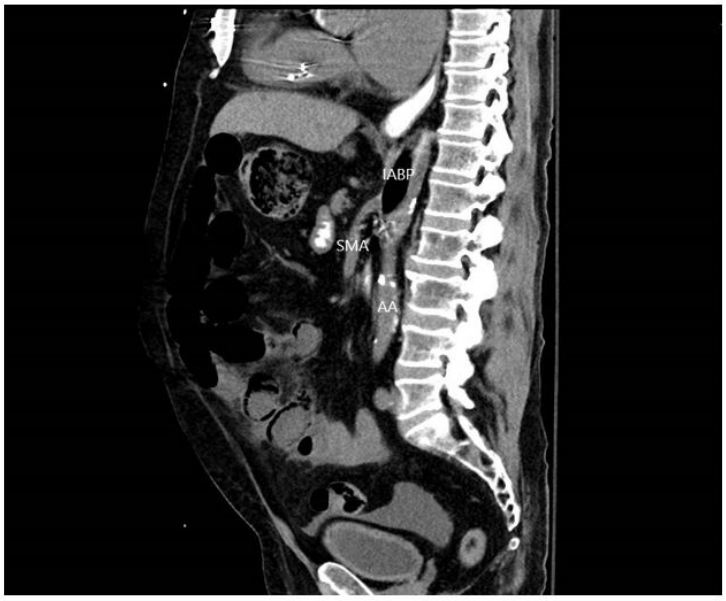
Introduction: Intra-aortic balloon pump (IABP) use has increased significantly since the United Network for Organ Sharing (UNOS) allocation criteria on heart transplant practices changed in 2018, prioritizing the sickest patients. Traditionally, IABPs have been placed percutaneously through the transfemoral artery approach. Femoral placement is associated with many disadvantages, including risk for infection and, most notably, the limitation of patient mobility resulting in physical deconditioning. Inserting an IABP through the axillary artery is a well-tolerated and feasible strategy to bridge patients with end-stage heart failure to heart transplantation or ventricular assist devices. This form of mechanical circulatory support permits the patient to sit upright and ambulate, optimizing the potential for extended periods of rehabilitation. Numerous studies have shown the effectiveness of ambulatory IABPs in managing advanced heart failure patients waiting for advanced heart failure therapies. The major complications of IAPBs, include severe bleeding, infection, acute thromboembolic events, arterial injury, balloon leak, IABP failure, and death. Most commonly however, IABPs require repositioning, this can occur in up to 44% of patients.

Methods: A 64-year-old male with a past medical history of non-ischemic cardiomyopathy, bicuspid aortic valve status post mechanical aortic valve replacement, ventricular tachycardia status post implantable cardioverter-defibrillator placement, chronic kidney disease, and multiple hospitalizations for acute decompensated heart failure presented with dyspnea on exertion, orthopnea, fatigue, and dizziness. These symptoms were consistent with previous heart failure exacerbation presentations. He underwent right heart catheterization, which showed elevated biventricular pressures, pulmonary hypertension, and a cardiac index of 1.91 L/min/m2. A left axillary intra-aortic balloon pump was placed to augment his cardiac output. Two days after his IABP placement, the patient complained of severe abdominal pain and was found to have a mild lactic acidosis. Due to poor baseline renal function, computed tomography (CT) of the abdomen and pelvis without intra-venous contrast was done. The scan showed the tip of the IABP just inferior to the origin of the superior mesenteric artery as well as bowel pneumatosis involving segments of the ileum and ascending colon concerning for bowel ischemia, and he was booked for emergent surgery. The surgical team requested a CT angiography of the abdomen and pelvis for better evaluation of mesenteric vasculature, which was obtained immediately prior to the operating room. The subsequent study showed the tip of the IABP within the ostium of the superior mesenteric artery. The left axillary IABP was removed along with 160cm of necrotic small bowel. Post-operatively, the patient had significant improvement in his hemodynamics. His hospital course was complicated by refractory ventricular tachycardia and respiratory failure. He was ultimately transitioned to comfort measures.

Conclusion: Axillary IABPs provide a safe and efficient method of mechanical circulatory support for selected patients as a bridge to heart transplantation. Malposition and thromboembolic phenomenon are well-known complications of IABP placement. Only three reported cases are documented in the medical literature of axillary and subclavian IABPs migrating into the superior mesenteric artery (SMA), resulting in occlusion and bowel ischemia. Abdominal pain in a patient with an IABP includes decreased perfusion due to heart failure, embolic phenomenon, and mechanical complications from the IABP itself. Theoretically, an axillary IABP can obstruct the mesenteric vasculature with the patient presenting with only mild or no overt clinical symptomatology. However, the incidence of balloon pump tip migration and intermittently occluding the Celiac, SMA, or inferior mesenteric arteries is unknown. Catastrophic consequences of IABP are limited to case reports and post-marketing data. In addition to CT imaging, there is a role for using point of care ultrasonography (POCUS) to evaluate vascular flow and IABP tip location. Management can include either repositioning or removing the IABP as deemed clinically appropriate. Further investigation is needed to determine the optimal use and study the rate of complications associated with axillary IABPs.

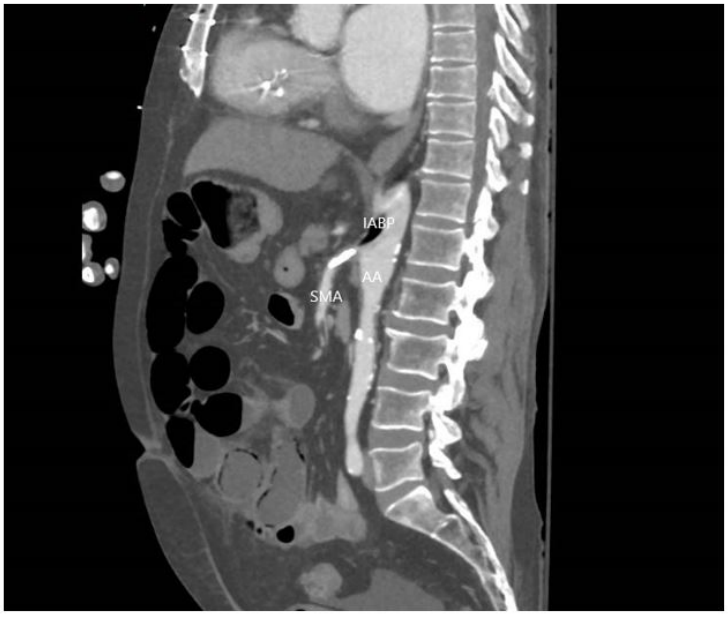
References: 1.Journal of the American College of Cardiology: Heart Failure. 2013 Oct;1(5):382-8. 2. Cardiac Failure Review. 2018 May;4(1):43-45. 3. Circulation Heart Failure. 2013;6:606-19 4. The Journal of Thoracic and Cardiovascular Surgery. 1978;76:61-3 5. The Annals of Thoracic Surgery. 2015;100:2151-7. 6. Journal of the American College of Cardiology 2001;38:1456-62 7. Circulation. 2010;122:S92-S99 8. The Annals of Thoracic Surgery. 2021 Apr;111(4):1264-1270. 9. The Journal of Thoracic and Cardiovascular Surgery. 2012; 143: 1193-1197 10. Transplantation Direct. 2020;7(1):e642. Published 2020 Dec 15. 11. Journal of the American College of Cardiology. 2020 Mar, 75 (11_Supplement_1) 2995 12. Circulation: Heart Failure. 2014;7:864-867

Image 1:



A sagittal section of a non-contrast computed tomography of the abdomen and pelvis showing the tip of the Intra-Aortic Balloon Pump (IABP) distal to the origin of the Superior Mesenteric (SMA) from the Abdominal Aorta (AA). Intestinal pneumatosis is also noted in the image.

Image 2:



A sagittal section of a computed tomography angiography of the abdomen and pelvis showing the tip of the Intra-Aortic Balloon Pump (IABP) that has migrated into the Superior Mesenteric (SMA) originating from the Abdominal Aorta (AA). Intestinal pneumatosis is again noted in the image

Critical Care - 9 Development of a Colopericardial Fistula after Left Ventricular Assist Device

John J Barnes[1], Sathappan Karuppiah[1], Monica I Lupei[2]

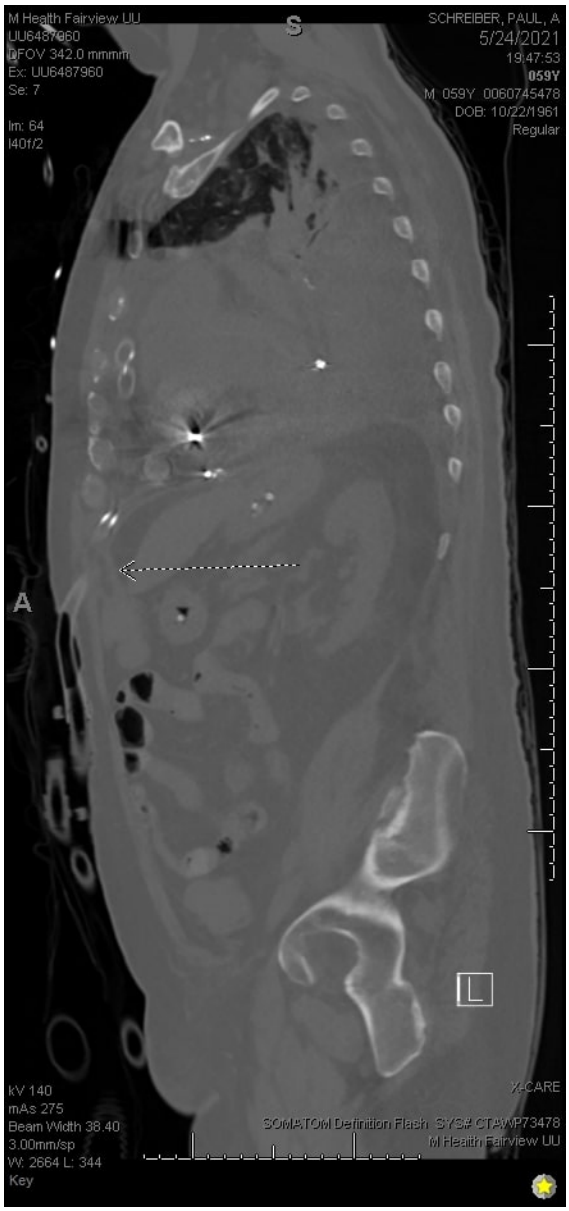
[1]University of Minnesota, Minneapolis , MN,
[2]University of Minnesota, Saint Paul, MN

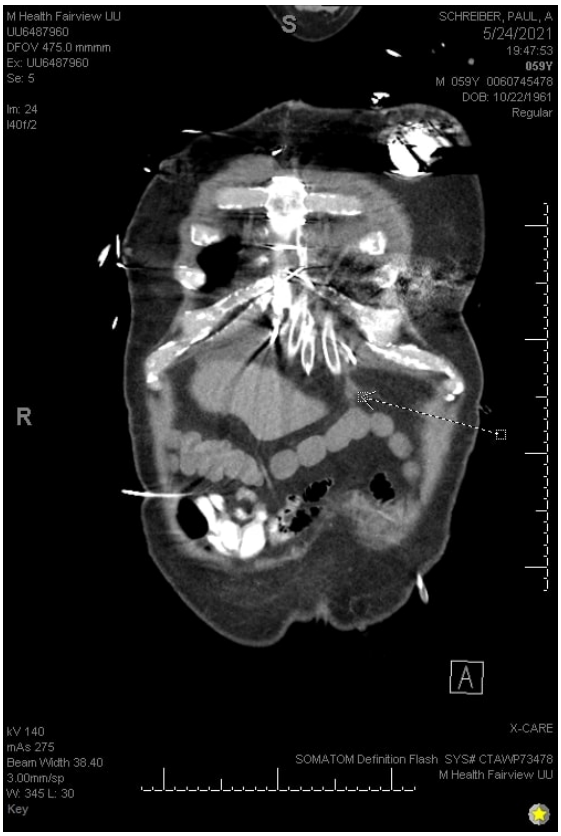
Introduction: Fistulas between the pericardium and gastrointestinal tract are extraordinarily rare and present challenges in management. There have been few case reports dating back to the 1980s describing abnormal communications between the gastrointestinal tract and the heart. These cases have a mortality rate of ~50%.(1) The majority of these case reports are related to esophageal cancer or after major surgeries involving the gastrointestinal tract. To our knowledge, the development of a colopericardial fistula after cardiac surgery has never been published in the literature. Patients with pericardial-gastrointestinal fistulas are typically critically ill and are often unable to tolerate major surgery to repair the fistula. This makes management of these conditions extremely complex for the intensive care team.

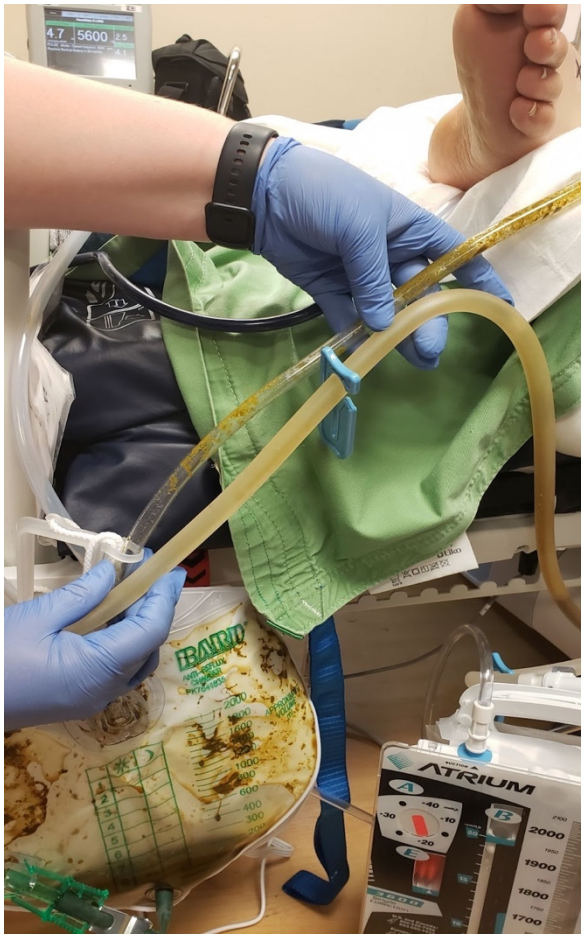
Methods: A 59-year-old man with a history of a prior mitral valve replacement for mitral regurgitation, atrial fibrillation, and dilated cardiomyopathy underwent placement of a left ventricular assist device. His intraoperative course was unremarkable; however, the patient had a complicated postoperative course including an acute middle cerebral artery stroke requiring emergency thrombectomy, several intubations for airway protection, persistent hypotension requiring vasoactive medications, and an empyema requiring chest washout in which chest tubes were placed. Two days after his chest washout, the mediastinal chest tube output increased significantly and appeared feculent (Figure 1). The chest computed tomography (CT) (Figures 2 and 3) showed a colopericardial fistula originating from the transverse colon. As the patient did not suffer clinical decompensation, a multidisciplinary panel including general surgery, cardiothoracic surgery, and the intensive care unit collectively decided that surgical management would carry significant mortality risk. The family pursued non-operative treatment so the patient was transitioned from tube feeding to total parenteral nutrition and was started on six weeks of intravenous antibiotics. The following week, tube feedings were resumed with the mediastinal chest tube in place, and there were no further signs of fistula on clinical examination or on a follow-up CT scan. The patient improved clinically over the next few weeks and was extubated and discharged to a rehabilitation facility.

Conclusion: Enteropericardial fistulas are a devastating complication after gastrointestinal surgery and, now, cardiac surgery. Literature review suggests this condition is frequently lethal. Treatment options include conservative management with bowel rest or surgical interventions such as fistula closure or gastrointestinal diversion, all of which have been successfully used to treat this condition.(2,3) Our patient who developed a colopericardial fistula after placement of a left ventricular assisted device was treated with conservative medical management and had a good outcome.

References: 1) Zuluaga-Zapata N, Lopera-Valle JS, Suarez-Poveda T. Colopericardial fistula presenting with massive rectal bleeding. J Radiol Case Rep. 2019;13(11):18-23. 2) Wetstein L, Ergin MA, Griepp RB. Colo-pericardial fistula: complication of colonic interposition. Tex Heart Inst J. 1982;9(3):373-376. 3) Imran Hamid U, Booth K, McManus K. Is the way to a man's heart through his stomach? Enteropericardial fistula: case series and literature review. Dis Esophagus. 2013;26(5):457-464.


M Health Fairview UU
UU6487960
DFOV 342.0 mmmm
Ex: UU6487960
Se: 7
Im: 64
I40f/2
S
SCHREIBER, PAUL, A
5/24/2021
19:47:53
059Y
M 059Y 0060745478
DOB: 10/22/1961
Regular
A
L
kV 140
mAs 275
Beam Width 38.40
3.00mm/sp
W: 2664 L: 344
Key
X-CARE
SOMATOM Definition Flash SYS# CTAWP73478
M Health Fairview UU


M Health Fairview UU
UU6487960
DFOV 475.0 mmmm
Ex: UU6487960
Se: 5
Im: 24
I40f/2
S
SCHREIBER, PAUL, A
5/24/2021
19:47:53
059Y
M 059Y 0060745478
DOB: 10/22/1961
Regular
R
A
kV 140
mAs 275
Beam Width 38.40
3.00mm/sp
W: 345 L: 30
Key
X-CARE
SOMATOM Definition Flash SYS# CTAWP73478
M Health Fairview UU


4.7
5600
BARD
ATRIUM

Critical Care - 10 Hyperhemolysis Syndrome: To Transfuse or Not to Transfuse?

Adeola Omojola[1], Daniela Lazea[1], Roxanne Favis[2]

[1]Brigham and Women's Hospital, Boston, MA, [2]Riverside University Health System, Moreno Valley, CA

Introduction: A 29 year old female with a medical history of well-managed sickle cell disease presented four days after undergoing successful total hip arthroplasty for avascular necrosis of the femoral head. She had received cross-matched red blood cells prior to the scheduled surgery to minimize a sickle cell crisis. However she developed delayed hemolytic transfusion reaction and hyperhemolysis syndrome requiring admission to the intensive care unit. This case report will discuss the course of hyperhemolysis syndrome, its complications, and current critical care management. Hyperhemolysis syndrome will also be compared with delayed hemolytic transfusion reaction.

Methods: A 29-year-old female with history of well-managed sickle cell disease (SCD), and post-operative day four from a successful total arthroplasty of the left hip due to avascular necrosis, presented to the emergency department with pain in her right upper and lower extremities. Notably, she received prophylactic pre-operative exchange transfusion with three units of cross-matched red blood cells (RBC). Her admission studies were significant for pulmonary embolus, deep vein thrombus, fevers, positive blood culture, elevated bilirubin, and positive direct anti-globulin. She was diagnosed with delayed hemolytic transfusion reaction (DHTR) and appropriate management initiated. After admission, she developed significant anemia and worsening hyperbilirubinemia, and hyperhemolysis syndrome (HS) was diagnosed. High-dose steroids, Erythropoietin, Eculizumab, and IVIg were administered. She was not a candidate for RBC transfusion, to prevent exacerbation. Her hematocrit reached a nadir of 5% and mental status declined warranting intubation. Due to low hematocrit, she was transfused two units of RBCs and received five more units during her ICU admission. Her course was complicated by thrombocytopenia, coagulopathy, and subdural hematoma. She was extubated when Hemoglobin, platelets, and mental status improved.

Conclusion: Hyperhemolysis syndrome is a rare but severe complication of transfusion often associated with SCD. It is likely due to frequent blood transfusions and the subsequent development of antibodies. Though the syndrome is not well-understood, the pathophysiology is theorized to be multifactorial: bystander hemolysis, antibodies, erythropoiesis suppression, and macrophage activation. It can be difficult to differentiate HS and DHTR as they present similarly with hemolysis, fever, and pain. Yet, identification is critical because their managements differ. Anti-ABO alloantibodies primarily destroy transfused RBCs in DHTR, while transfused and native RBCs are hemolyzed via anti-ABO alloantibodies and bystander hemolysis in HS. Bystander Hemolysis occurs when non-anti-ABO antibodies react with transfused proteins to activate complement and promote hemolysis. Reticulocytopenia, a hallmark of HS, could be due to erythropoiesis suppression by RBC transfusion or peripheral consumption of premature cells. DHTR is not associated with low reticulocytes. It appears, HS is a macrophage-driven inflammatory condition as patients can present with pain and fever, symptoms of inflammation. Macrophages bind, and destroy, Hb S cells more easily than Hb A cells. This mechanism is like that used by reticulocytes to bind endothelium. Thus, it is hypothesized that macrophages also bind, and destroy, reticulocytes. Management includes anti-inflammatory agents: IVIg and steroids to suppress macrophage activity, and Eculizumab to suppress complement-driven inflammation. On the other hand, the management of DHTR is transfusion of cross-matched RBCs.

References: 1. Win, N. (2009). Hyperhemolysis syndrome in sickle cell disease. Expert Review of Hematology, 2(2), 111-115. https://doi.org/10.1586/ehm.09.2 2. Karafin, M. S., Singavi, A., Johnson, S. T., & Field, J. J. (2017). A Fatal Case of Immune Hyperhemolysis with Bone Marrow Necrosis in a Patient with Sickle Cell Disease. Hematology Reports, 9(1), 6934-6934. https://doi.org/10.4081/hr.2017.6934 3. Starr, D., Hermelin, D., & Blackall, D. (2020). Hyperhemolysis Syndrome Following Red Cell Exchange in a Newly

Diagnosed Sickle Cell Disease Patient With Spinal Cord Infarction. Journal of Medical Cases, 11(9), 263-266. https://doi.org/10.14740/jmc3541

Critical Care - 11 TRALI in PACU after 1 unit of platelets: a case report.

Miguel E Perez-Viloria[1], Fayeza Malik[2], Marco A Castillo-Rodriguez[3], Robert Zusman[1], Guillermo Garcia[1]

[1]Mount Sinai Medical Center of Florida, Miami, FL, [2]Florida International University, Miami, FL, [3]Mount Sinai Medical Center of Florida, Miramar, FL

Introduction: Transfusion-related acute lung injury (TRALI) is a rare but serious syndrome characterized by the acute onset of noncardiogenic pulmonary edema with severe hypoxemia within 6 hours of receiving transfusion of any blood product1-2. It is the leading cause of mortality associated with transfused blood components reported to the FDA4. Risk factors include history of smoking and alcohol use, liver disease, systemic inflammation, and increased WBC levels in the pulmonary vasculature due to pre-existing lung injury. It has been hypothesized that a second hit from the presence of cytokines and lipids in the blood product being transfused activates neutrophils causing pulmonary damage, leading to TRALI5. Diagnosis relies on excluding other causes of acute pulmonary edema following transfusion, including sepsis, transfusion-associated circulatory overload (TACO), anaphylaxis, and cardiogenic pulmonary edema6. Clinical signs include dyspnea, tachypnea, fever, frothy sputum, hypotension, and cyanosis7. Diagnostic criteria include acute onset hypoxemia within 6 hours of transfusion; PAO2/FiO2 ratio <300mm Hg or oxygen saturation <90% on room air; bilateral pulmonary infiltrates on chest radiograph; and PAWP< 18mm Hg without evidence of left atrial hypertension8. This case report describes an unusual case of TRALI in a 66-year-old female after receiving one unit of platelets, highlighting the importance of identifying predisposing risk factors prior to the utilization of blood products.

Methods: 66yo F with PMH of HTN, HLD, hepatitis C, tobacco abuse, metastatic spindle cell carcinoma of the lung s/p chemotherapy, recurrent pleural effusion, and PET positive and enlarged mediastinal lymph nodes. She presented to the OR for fiberoptic bronchoscopy with US biopsy, robotic VATS, right lower lobe wedge resection and pleurodesis. Preop workup showed a platelet count of 55,000 and CXR showing trace small bilateral pleural effusions with associated bibasilar atelectasis (Fig 1). TIVA was used with propofol and remifentanil. A bag of platelets was transfused when the surgeon observed excessive bleeding at the biopsy sites. No issues with upper airway obstruction were encountered. Shortly after extubation, she had frothy secretions. Her RR was 35bpm and acute hypoxia. She was re-intubated in the OR. Auscultation revealed bilateral rales in all lung fields. She was transferred to the PACU intubated on mechanical ventilation. Workup included labs (WBC 19.2 and platelets 132,000), Swan-Ganz catheter placement (PAP 27/18 mmHg, CVP 7mmHg) and CXR (interval development of diffuse bilateral interstitial and airspace opacities suggestive of pulmonary edema) (Fig 2). TTE revealed moderately impaired LV systolic function with an LVEF of 40%, mild LV concentric remodeling. Right ventricular size and systolic function were normal. Troponin levels were normal. Given these findings, we believe the patient's acute decompensation was due to TRALI. The patient was then transferred to the SICU where she continued to deteriorate, progressing to multi-organ failure, and expiring on POD6.

Conclusion: TRALI is difficult to evaluate and warrants careful approach during blood transfusions4. TACO is the second leading cause of transfusion-related fatality and is difficult to discern from TRALI. TRALI may be recognized by the absence of signs of circulatory overload9. In this case, the patient had normal CVP, normal PAWP, no S3, and no signs of peripheral edema. Allergic and anaphylactic transfusion reactions also present with respiratory distress and hypoxia due to bronchospasm and laryngeal edema but can be ruled out due to the absence of wheezing, urticarial rash, and hypotension combined with CXR findings. Negative pressure pulmonary edema (NPPE) is caused by intense inspiratory effort against obstruction leading to very negative airway pressures10. Risk factors are young age, male sex, generally healthy, difficult intubation, use of irritant volatile agents, obesity, and recent upper airway infection11-13. The patient did not have any of the risk factors or experience upper airway obstruction after extubation, ruling out NPPE. This case report highlights the importance of identifying predisposing risk factors prior to the utilization of blood products. Her risk factors included liver disease, history of smoking

and alcohol use, and lung disease, predisposing her to develop TRALI after a single unit of platelets.

References: 1. Transfusion. 25(6). 573-577.1985 2. Hematology. Education Program of the American Society of Hematology. 1. 585-594. 2018 3. Blood Reviews. 23(6). 245-255. 2009 4. Transfusion Medicine Reviews. 18(3). 184-188. 2004 5. Vox Sanguinis. 112(8). 694-703. 2017 6. Technical Manual. 272-762. 2011 7. Blood Reviews. 20(3). 139-159. 2006 8. Critical Care Medicine. 36(11). 3080-3084. 2008 9. American Journal of Medicine. 126(4). 357.e29-357. 2013 10. Medicine. 98(17). e15389. 2019 11. Chest. 90. 802-5. 1968 12. Journal of Anesthesia and Clinical Research. 3. 252. 2013 13. Anesthesiology. 113. 200-207. 2010

Figure 1. Preoperative CXR

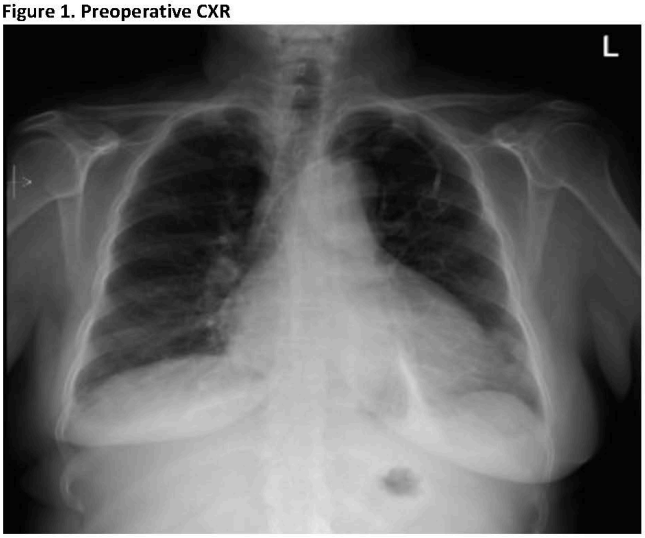
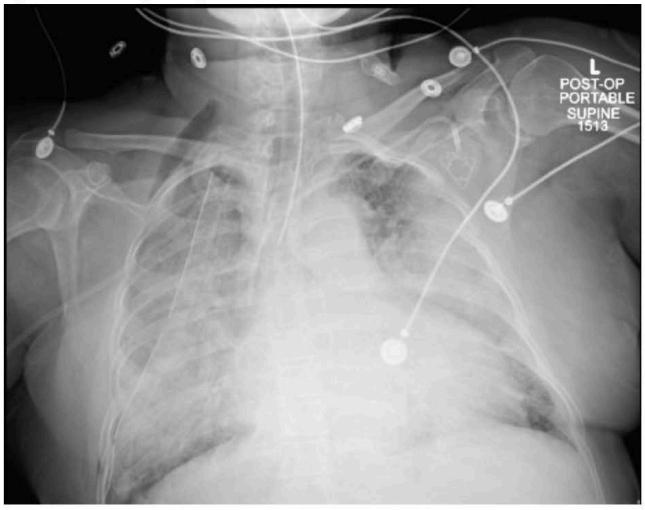


Figure 2. 1-hour post-op CXR



Critical Care - 12 Use of Visceral Organ Doppler by a Modified Transhepatic View for Volume Assessment and ECMO weaning

Jacqueline Sohn[1], Mourad H Senussi[2]

[1]Baylor College of Medicine CHI St. Luke's Medical Center/Texas Heart Institute, Houston, TX, [2]Baylor College of Medicine CHI St. Luke,Äôs Medical Center/Texas Heart Institute, Houston, TX

Introduction: Critical Care Echocardiography (CCE) has become an instrumental tool in the management of critically ill patients in the cardiac intensive care unit. However, obtaining traditional echocardiographic views is technically difficult and may not be feasible in post-sternotomy patients or patients on mechanical circulatory support (MCS) due to residual air in the thoracic cavity or multiple chest tubes, drains, and dressings. In these patients, a novel modified transhepatic view can be used to assess patient's right heart function and filling pressures. Evaluation of hepatic and portal vein Doppler can provide useful clinical information. This can be useful in assessing volume status and congestion in ECMO patients and guide clinical decision making. In this case report, we present an example of how this can be used to guide in fluid management in ECMO patient, and in ECMO weaning.

Methods: 47-year-old male with a past medical history of ESRD, CAD with stents, diabetes and hypertension initially presented with right sided pleural effusion and loculation. The Patient required coronary artery bypass graft (CABG) while undergoing evaluation for video assisted thoracoscopic surgery (VATS). Postoperatively, patient required high doses of vasopressors and inotropes due to persistent acute on chronic right heart failure eventually requiring VA-ECMO support. While on ECMO, we managed his volume status using the novel modified transhepatic view and evaluating the hepatic vein and portal vein Doppler. Initially, due to his right heart failure and volume overload, hepatic vein and portal vein congestion was noted on the Doppler images. After fluid removal by continuous renal replacement therapy (CRRT), we were able to observe normalization in hepatic vein and portal Doppler. Furthermore, we utilized the view and bedside CCE to evaluate his readiness for wean off of ECMO by obtaining hepatic vein Doppler and portal Doppler while incrementally decreasing ECMO flow. When we were able to wean down to 1L flow on the ECMO without any signs of reversal of flow on hepatic vein or increased portal vein Doppler pulsatility, we made the decision to de-cannulate the patient. Patient was de-cannulated successfully and eventually recovered enough to be transferred to a long term acute care hospital.

Conclusion: Visceral Organ Doppler of liver vasculature (hepatic vein and portal vein) can be obtained in both subcostal and novel modified transhepatic views. The novel modified transhepatic view is especially useful in patients with MCS and post cardiac surgery who have very limited area on the body for traditional CCE views. The normal hepatic vein Doppler is mostly antegrade flow back to the heart with a retrograde flow during atrial contraction and atrial overfilling. In right-sided CHF without tricuspid regurgitation, increased pulsatility and retrograde flow indicates volume overload. The normal portal vein Doppler is monophasic flow above the baseline (antegrade) with sometimes minimal undulation. When there is a pulsatility in the flow (pulsatility index (PI) > 0.5) this may indicate right-sided CHF due to volume overload. These Doppler images can be used to guide volume removal on MCS patients where volume management can be challenging. These Doppler images can be further used to guide ECMO weaning. This is advantageous since it can be quickly performed, interpreted and therapeutic measures instituted by the critical care physician at the bedside. Therefore, critical decisions can be made much more promptly.

References: 1. McNaughton, Dean Alexander, and Monzer M. Abu-Yousef. Doppler Us of the Liver Made Simple. RadioGraphics, vol. 31, no. 1, 2011, pp. 161-188., doi:10.1148/rg.311105093. 2. Prada-Ruiz A, Baker-Smith C, Beaty C et al. Echocardiographic assessment of mechanical circulatory support and heart transplant. Prog Pediatr Cardiol. 2020;58:101272. doi:10.1016/j.ppedcard.2020.101272

3. Koratala A. Venous congestion assessment using point-of-case Doppler ultrasound: Welcome to the future of volume status assessment. Clin Case Rep. 2021; 9:1805-1807. 4. Valette X, Ribstein P, Ramakers M, du Cheyron D. Subcostal versus transhepatic view to assess the inferior vena cava in critically ill patients. Echocardiography. 2020; 37:1171-1176.

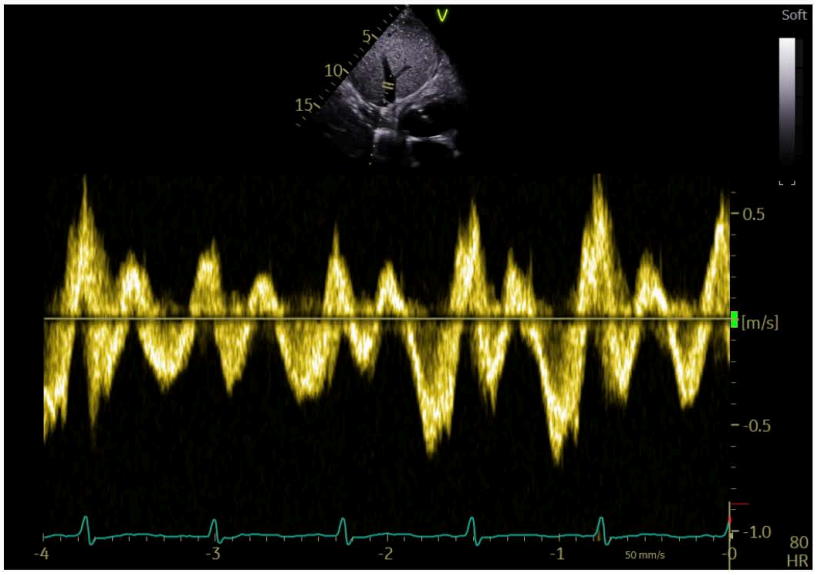


Figure 1. Hepatic Vein Doppler showing blunting of S-wave consistent with congestion and right heart failure

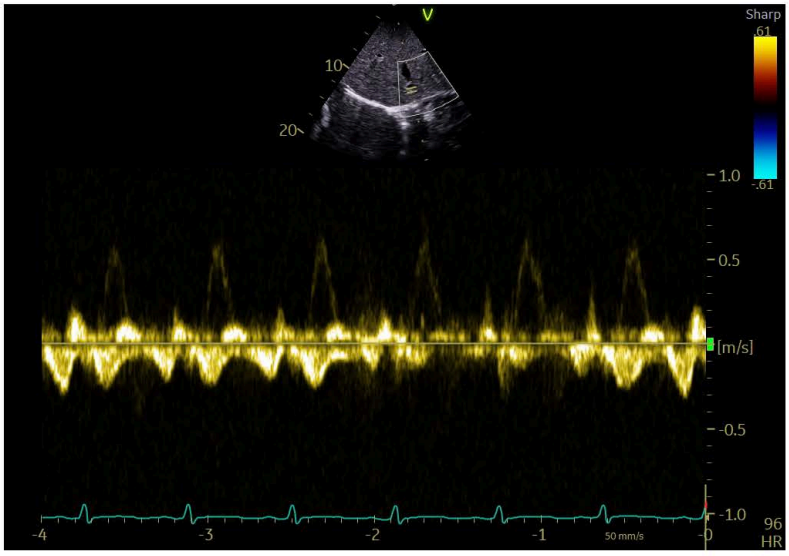


Figure 2. normalization of hepatic vein Doppler flow after fluid removal.

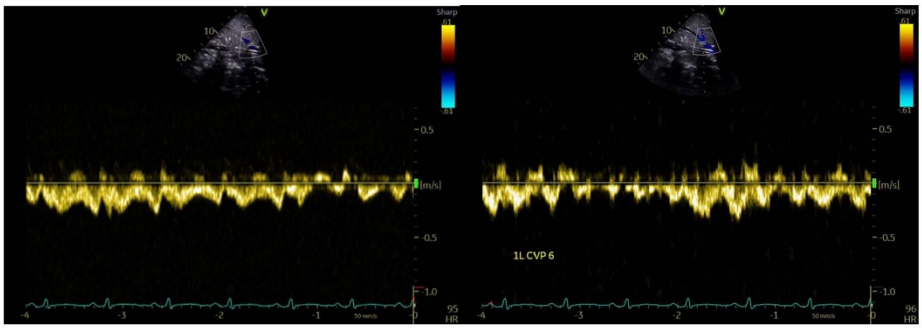


Figure 3. (Left) ECMO 3.4L flow baseline. (Right) ECMO 1L flow continues to show normal hepatic vein Doppler suggesting no congestion or RV failure.

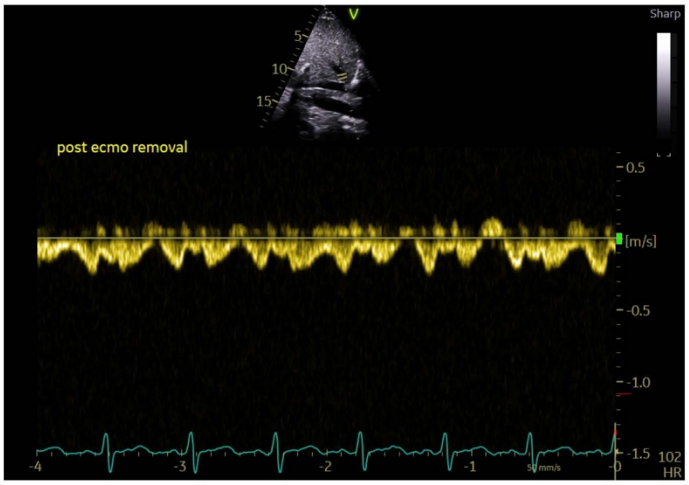


Figure 4. Post de-cannulation, hepatic vein Doppler.

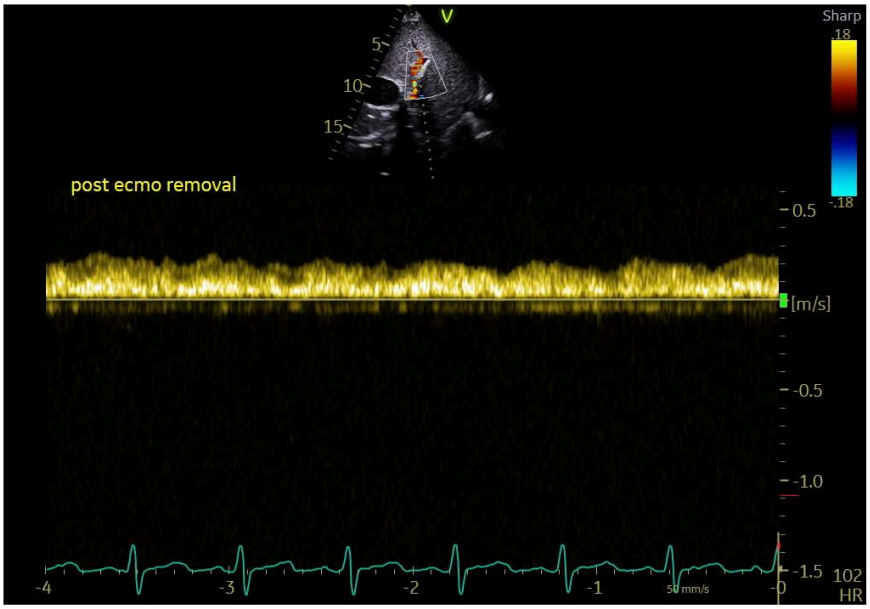


Figure 5. Post de-cannulation, Portal Vein Doppler

Critical Care - 13 Acute traumatic lung injury in Fontan physiology

Jack Chan[1], S. Patrick Bender[1]

[1]University of Vermont Medical Center, Burlington, VT

Introduction: Repaired congenital cardiopulmonary pathophysiology, specifically Fontan physiology, can complicate trauma resuscitation. Fontan physiology predisposes patients to decreased cardiac output if placed on positive pressure ventilation, as the increased intrathoracic pressure impedes the passive flow thorough the cavopulmonary circulation. We witnessed a clinical encounter where the ED trauma protocol was not optimal given the patient's Fontan physiology. Additionally, her care in the surgical ICU was complicated by the conflicting cardiopulmonary goals of Fontan physiology and acute lung injury (ALI).

Methods: This 19 year old female was status post a Fontan procedure in 2007 for pulmonary atresia. She presented as a trauma after a high speed MVC. The patient was hypotensive (SBP = 80) en route to the hospital with a GCS of 13. She underwent a full body CT scan and was found to have extensive rib fractures, bilateral pneumothoraces, and pulmonary contusions. She was hypoxic (spO2 = 74%) via face mask and was transitioned to HFNC. Bilateral chest tubes were placed with no oxygenation improvement, and spO2 subsequently decreased to 50%. The decision was made to intubate, after which the oxygenation improved to 80%, but SBP decreased to the 40s. The surgical ICU was consulted, and treatment specific to the patient's Fontan physiology was commenced. The patient's treatment was transitioned to 1) low tidal volume and low PEEP to decrease mean airway pressures to avoid worsening cavopulmonary flow, 2) inhaled pulmonary vasodilators to support oxygenation, and 3) fluids and norepinephrine to augment cardiac output. Blood pressure and oxygenation normalized. Early decision was made to transfer to an ECMO facility, as the goals for treating pulmonary contusions and acute lung injury (ALI), namely elevated PEEP and mean airway pressure, would be counterproductive for this patients Fontan physiology. It was believed that early transfer would be better, as any worsening of the ALI would be challenging to address emergently without ECMO. The patient underwent a prolonged course of intubation with ECMO in the other facility, and is continuing to recover from her medical course.

Conclusion: This case illustrates how conventional treatment of trauma and acute lung injury can be detrimental to patients with Fontan physiology. Positive pressure ventilation may lead to hemodynamic compromise and worsened oxygenation. Preload resuscitation prior to intubation, along with instituting lower mean airway pressures and pulmonary vasodilators earlier would have been prudent. Additionally, it demonstrates the importance of early transfer to an ECMO facility, as ECMO may be the only option for balancing the conflicting cardiopulmonary goals of ALI and Fontan physiology.

References: Rychik, J., Atz, A.M., Celermajer, D.S., Deal, B.J., Gatzoulis, M.A., Gewillig, M.H., Hsia, T.-Y., Hsu, D.T., Kovacs, A.H., McCrindle, B.W., Newburger, J.W., Pike, N.A., Rodefeld, M., Rosenthal, D.N., Schumacher, K.R., Marino, B.S., Stout, K., Veldtman, G., Younoszai, A.K. and d'Udekem, Y. (2019). Evaluation and Management of the Child and Adult With Fontan Circulation: A Scientific Statement From the American Heart Association. Circulation, 140(6). Khairy P, Fernandes SM, Mayer JE Jr, et al. Long-term survival, modes of death, and predictors of mortality in patients with Fontan surgery. Circulation 2008; 117:85. Jolley, Matthew MD*†; Colan, Steven D. MD†; Rhodes, Jonathan MD†; DiNardo, James MD* Fontan Physiology Revisited, Anesthesia & Analgesia: July 2015 - Volume 121 - Issue 1 - p 172-182. doi: 10.1213/ANE.0000000000000717 Miller, R.D. (2015). Miller's anesthesia. Philadelphia, Pa: Elsevier/Saunders.

Critical Care - 14 Intraoperative Management Of Reconstructive Flap In A Patient With Extensive Total Body Burns And Known Difficult Airway

Elisa C Walsh[1], Jamie Sparling[2]

[1]Massachusetts General Hospital, Boston, MA, [2]Massachusetts General Hospital, Melrose, MA

Introduction: Approximately 450,000 patients per year in the United States seek treatment for burn injuries (1). Acute care for major burn injuries revolves around airway management, vascular access, hemodynamic and pulmonary support, and analgesia. As patients transition to the reconstructive phase of treatment, intraoperative management remains challenging due to difficult airway and vascular access as well as risk of graft malperfusion.

Methods: A 34-year-old woman with a distant history of 80% total body surface area burns status-post multiple fascial excisions and skin grafting procedures complicated by persistent torticollis and bilateral lower extremity edema presented for a omental free flap to the right lower extremity to reestablish lymphatic drainage. Given a history of difficult airway, she underwent an inhalational induction with sevoflurane/nitrous oxide and a 6.5 Parker endotracheal tube was advanced through a size 3.5 laryngeal mask airway with bronchoscopic verification. For optimal flap perfusion, a high lumbar epidural was placed for lower extremity vasodilation which was dosed with bupivacaine 0.25% in 3 milliliter increments throughout the case. Generous fluid resuscitation totaling 4 liters of Lactated Ringer's and 1 liter of 5% albumin and a continuous ketamine infusion at 7.5-10 mcg/kg/min for analgesia without hypotension were used in place of vasopressor support for a MAP goal greater than 60mmHg. After case completion, she was admitted to the intensive care unit and remained intubated for an anticipated free flap closure 48 hours after the initial procedure. The team continued to use her epidural postoperatively to provide analgesia and maximal flap vasodilation. The patient ultimately underwent a planned takeback that was complicated by a flap avulsion requiring reanastomosis and Integra coverage. She had a prolonged hospitalization requiring multiple debridements and split-thickness skin grafts but was ultimately discharged to a rehabilitation facility where she continues to recover.

Conclusion: Optimal perioperative care of burn-injured patients in the reconstructive phase of treatment requires thorough preoperative assessment and attention to long-term complications including difficult airway and vascular access to ensure that appropriate equipment is available. Intraoperatively, the predominant goal is to optimize perfusion to the vascularized free flap through reduced systemic vascular resistance, increased cardiac output, generous preload, and effective analgesia. The use of intraoperative vasopressors has traditionally been avoided due to concern for peripheral vasospasm causing reduced flap perfusion and ultimately failure, although there is no prospective clinical evidence to support absolute contraindication (2). Furthermore, excessive fluid resuscitation to maintain or increase cardiac output may be associated with edema, venous stasis, and thrombosis in some patient populations (3). Goal-directed fluid resuscitation may be improved through invasive hemodynamic monitoring as well as novel non-invasive technologies to estimate cardiac output and systemic vascular resistance. Central neuraxial anesthesia results in a sympathectomy below the level of placement that selectively increases vasodilation of the pedicle artery and microvasculature, in addition to providing intraoperative and postoperative analgesia.

References: 1. Bittner EA et al. Acute and Perioperative Care of the Burn-injured Patient. Anesthesiology; 122:448-64. 2015. 2. Ibrahim AMS et al. Vasopressors and Reconstructive Flap Perfusion. Annals of Plastic Surgery; 73:245-248. 2013. 3. Booi DI. Perioperative fluid overload increases anastomosis thrombosis in the free TRAM flap used for breast reconstruction. Eur J Plastic Surg; 34:81-86. 2011.

Critical Care - 15 Management of pregnant patients with VV-ECMO for severe acute respiratory distress syndrome due to COVID-19.

Ryan Grieger[1], Lidia Csernak[1], Jarod Speer[1], Michael Lyaker[1]

[1]Ohio State University Medical Center, Columbus, OH

Introduction: Pregnancy is a risk factor for severe disease in patients infected with the SARS-CoV-2 virus and is associated with higher rates of ICU admission and mechanical ventilation (MV)(1). COVID-19 disease during pregnancy is also associated with increased rates of pre-term birth, pre-eclampsia, and stillbirth (2). Extracorporeal Membrane Oxygenation (ECMO) has been utilized throughout the pandemic to rescue patients with severe acute respiratory distress syndrome (ARDS) refractory to mechanical ventilation, prone positioning and paralysis. ECMO has also been used to manage pregnant patients with ARDS, including during the H1N1 influenza outbreak. However, less is known about maternal and fetal ECMO outcomes with severe COVID-19 disease. We present two cases of pregnant patients with COVID-19 who required VV-ECMO at a large academic medical center.

Methods: Patient 1 was a 35-year-old female who presented at 19w0d gestation, and Patient 2 was a 32-year-old female who presented at 26w3d gestation. Both women were not vaccinated against COVID-19 and presented with respiratory failure. Despite recommended COVID-19 therapies, MV and prone positioning, they required VV-ECMO cannulation for refractory hypoxemia. Maternal welfare was prioritized, but fetal-protective measures were also instituted when feasible. Patient 1 had a pre-viable spontaneous abortion at 20 weeks. Her course was further complicated by large volume epistaxis, coagulation factor depletion, and increasing ECMO support requirements. Patient 2 underwent elective cesarian section at 27w5d gestation while on ECMO due to increasing maternal support requirements to avoid an emergent delivery if mother or fetus became acutely unstable. The infant did well and was transferred to a nearby pediatric hospital, but the mother's course was further complicated by hemoperitoneum requiring uterine artery embolization. Ultimately, comfort care measures were pursued by the families of both patients due to worsening respiratory status, increasing ECMO requirements, and persistent complications with lack of progress. Patient 1 expired on ECMO day 14, and Patient 2 expired on ECMO day 57.

Conclusion: Management of pregnant COVID patients on VV-ECMO requires careful consideration of the physiologic changes of pregnancy. The goal in both cases was bridging towards recovery of lung function. We chose to insert a single dual-lumen cannula via the right internal jugular vein to avoid compromise to uterine perfusion and alterations in ECMO flow from the gravid uterus. For fetal protection, oxygen saturation goals were increased when possible. Preference was given to sedatives without known adverse effects on fetal development. The anticoagulation plan considered the elevated risk of thrombosis from pregnancy and COVID against the risk of factor depletion and platelet destruction from the ECMO circuit. In contrast to earlier published reports of largely favorable outcomes in pregnant women requiring VV-ECMO (3,4,5), both of our patients succumbed to complications of their disease. This is consistent with an observed decrease in ECMO survival later in COVID-19 pandemic and may be partially explained by improvement in COVID-19 therapies, resulting in fewer but sicker patients requiring ECMO support (6).

References: 1. Update: Characteristics of Symptomatic Women of Reproductive Age with Laboratory-Confirmed SARS-CoV-2 Infection by Pregnancy Status - United States, January 22-October 3, 2020. MMWR Morb Mortal Wkly Rep. 2020;69(44):1641-1647. 2. The impact of COVID-19 on pregnancy outcomes: a systematic review and meta-analysis. CMAJ. 2021; 193(16): E540-E548 3. Survival of Pregnant Coronavirus Patient on Extracorporeal Membrane Oxygenation. Ann Thor Surg. 2021;111(3): E151-E152. 4. First successful treatment of a COVID-19 pregnant woman with severe ARDS by combining early mechanical ventilation and ECMO. Heart & Lung. 2021; 50(1): 33-36. 5. Successful Treatment of Pregnant and Postpartum Women With Severe COVID-19

Associated Acute Respiratory Distress Syndrome With Extracorporeal Membrane Oxygenation. ASAIO J. 2021; 67(2): 132-136. 6. Evolving outcomes of extracorporeal membrane oxygenation support for severe COVID-19 ARDS in Sorbonne hospitals, Paris. Crit Care 2021; 25(355).

Critical Care - 16 Cold and Dead? Prolonged Resuscitation Utilizing an Automated Compression Device and ECMO in a Patient Presenting with Severe Hypothermia and Cardiac Arrest

Adam Pagryzinski[1], Justin Tawil[1]

[1]Medical College of Wisconsin, Milwaukee, WI

Introduction: Primary hypothermia from environmental exposure is a relatively common occurrence in cold climates. (1) Severe hypothermia may be associated with significant metabolic derangements, coma, circulatory collapse, and multisystem organ failure. In severe cases patients may present with asystole, lack of vital signs, and loss of brainstem reflexes that may mimic death. (2) There are however numerous case reports of survival with favorable neurological outcomes after prolonged resuscitation in patients initially presenting with profound hypothermia and cardiac arrest. (3)

Methods: A 61-year-old male with significant cardiac history presented to an outside hospital with severe hypothermia (82o F) and asystole. Last known well time was nearly 4 hours prior. Cardiopulmonary resuscitation (CPR) was initiated with an automated compression device and warming was started with modalities including forced air, warmed intravenous fluids, gastric and rectal lavage, and femoral venous warming catheter. After several hours of rewarming at a rate of 0.4 degrees per hour and persistent asystole the patient was transferred to a tertiary care center for further care. Upon arrival to the cardiovascular intensive care unit bedside venoarterial extracorporeal membrane oxygenation (VA ECMO) cannulation was attempted by landmark-based approach, but accidental venovenous (VV) cannulation was completed. CPR with the automated compression device was continued while the patient was rewarmed via VV ECMO. Laboratory values were frequently monitored and were in extremis despite aggressive therapy. As core temperature approached normothermia the patient remained in asystole with a physical examination consistent with brain and cardiac death. He was pronounced dead after more than 10 hours of resuscitative efforts. A single automated compressive device was used for the duration of the resuscitation, only overheating and failing after 10 hours of continuous use.

Conclusion: This case illustrates the importance of an algorithmic approach to treatment of severe hypothermia. (4) The intensivist practicing in cold climates should be familiar with the spectrum of treatment options including non-invasive and invasive modalities. While VA ECMO remains the gold standard for rewarming in severe hypothermia, VV ECMO proved to be a slow and inefficient warming modality. (5) Arterial cannulation may have been facilitated by the use of ultrasound given the difficulty of landmark based techniques, especially during CPR. (6) Given the potential for recovery with favorable outcomes, even in patients with prolonged cardiac arrest, attention must be paid to prognostic indicators so the potential for resuscitation and recovery can be used to guide the extent of rewarming efforts. (7) Critical care providers must also familiarize themselves with the presence, or lack thereof, of any hospital policies, local, or state laws guiding the pronouncement of death in hypothermic patients with cardiac arrest. (8, 9)

References: 1. Brown, D. et al. (2012) N Engl J Med 367: 1930-1938 2. Hilmo, J. et al (2014) Resuscitation 85(9): 1204-11 3. Vretenar, D. et al. (1994) Ann Thorac Surg 58: 895-8 4. Maeder, M. et al. (2011) Injury 42: 539-543 5. Mazur, P. et al (2019) Ann Cardiothorac Surg 8(1): 137-142 6. Nanjayya, V et al (2015) Extracorporeal Life Support Organization 7. Svendsen, O. et al. (2017) Ann Thorac Surg 103: 920-925 8. Greer, D. et al (2008) Neurology 70(4): 284-289 9. Nikas, N. et al (2016) J Med Philos. 41(3): 237-256

Critical Care - 17 Right Ventricular Failure Secondary to Vitamin C Deficiency-Associated Pulmonary Hypertension

Eduardo Soares Cirne de Toledo[1], Miguel A Yaport[1]

[1]Tufts Medical Center, Boston, MA

Introduction: Pulmonary Hypertension (PH) is associated with increased perioperative morbidity and mortality. Prevalence of PH is increasing with 1000 or more new cases diagnosed each year in the United States (US) alone. In the cardiac surgical population, PH can lead to right ventricular (RV) dysfunction or frank postoperative RV failure. As the majority of cases of PH in the US are secondary to, or associated with another disease state, managing comorbid pathology is paramount in the treatment of pulmonary hypertension. Most commonly the American patient population presents with PH secondary to heart failure, however, unrecognized causes of PH can include undiagnosed intracardiac shunts, anorectic agents, and nutritional deficiencies. Critical illness, a catabolic state, is associated with reduced Vitamin C levels. Even despite appropriate recommended nutritional intake as many as 70% of critically ill patients have hypovitaminosis C and 30% will reach nutritional deficiency. This micronutrient is an essential cofactor for important enzymatic processes, of particular interest is its role in nitric oxide synthesis. We present a case of a patient with recalcitrant PH complicated by RV failure, with an unrecognized ascorbic acid deficiency. After vitamin supplementation he had recovery of RV function and improvement of the PA pressure.

Methods: A 51 year old man with a history of ischemic cardiomyopathy, heart failure with reduced ejection fraction, peripheral vascular disease complicated by mesenteric ischemia and insulin dependent diabetes mellitus was transferred to our institution for advanced heart failure therapies. He required placement of a superior mesenteric artery stent, after which he underwent implantation of a durable Left Ventricular Assist Device (LVAD). Postoperative course was complicated by persistent right ventricular dysfunction. The patient was placed on a Dobutamine drip at 10 mcg/kg/min, Milrinone drip at 0.5 mcg/kg/min and inhaled Nitric Oxide at 40ppm. Daily hemodynamic measurements were obtained using a pulmonary artery catheter. On average, mean pulmonary artery pressure (mPAP) was 29mmHg with a pulmonary artery occlusion pressure (PAOP) of 14mmHg. Average Indices of pulmonary arterial hypertension were a pulmonary vascular resistance (PVR) of 265 dyn*s-1cm-5m-2, Transpulmonary gradient (TPG) of 15mmHg and a Diastolic pulmonary gradient (DPG) of 7mmHg Weekly transesophageal echocardiography showed a persistently dilated RV and moderate to severe RV dysfunction. Three weeks after LVAD placement he was started on continuous veno-venous hemodialysis (CVVHD) for volume overload and oliguria. Shortly thereafter, the patient was noted to be increasingly agitated, newly thrombocytopenic, and an abdominal rash with petechiae were noted. A punch biopsy of the rash revealed 'telangiectasia with extravasated red blood cells in background of perifolliculitis' Labs at the time were notable for a Vitamin C level of <0.1mg/dL (Normal = 0.2-2mg/dL). The patient was subsequently started on vitamin C supplementation with 1g BID. Within 5 days of starting Vitamin C supplementation he was weaned off inhaled pulmonary vasodilator therapy, Dobutamine weaned from 10 to 2.5 and Milrinone weaned from 0.5 to 0.125. At this time, average hemodynamic measurements were mPAP decreased to 26mmHg, PAOP stable at 15mmHg, PVR decreased to 184 dyn*s-1cm-5m-2, TPG decreased to 11mmHg and DPG decreased to 6mmHg. Vitamin C level was measured at 0.8mg/dL.

Conclusion: Here we present a case of recalcitrant PH that despite optimal medical management, was not responsive to medical therapy. There was an unrecognized ascorbic acid deficiency that improved with supplementation. Our case is unique in several ways. Firstly, we document a temporal relationship between starting CVVHD and symptomatic worsening. CVVHD and iHD have both been shown to be associated with a reduction in water soluble vitamins, notably Vitamin C. Secondly, his altered mental status, thrombocytopenia and rash after starting CVVHD may have represented a scurvy-like disease. Lastly, to our knowledge this is the first case that describes patient clinical improvement with measured vitamin C levels and hemodynamic measurements. Finally, In a critically ill patient experiencing recalcitrant PH, especially in someone with evidence of pre-existing

malnutrition, consider measuring micronutrients in the ICU to rule out vitamin C deficiency as a potential cause.

References: 1. A global view of pulmonary hypertension. Lancet Respir Med. 2016 Apr;4(4):306-22. 2. Impact of Pulmonary Hypertension on the Outcomes of Noncardiac Surgery: Predictors of Perioperative Morbidity and Mortality ; J Am Coll Cardiol. 2005 May 17;45(10):1691-9. 3. Secondary pulmonary hypertension; Respiratory Medicine CME, Volume 3, Issue 1, 2010, Pages 1-9. 4. Intravenous iron therapy in patients with idiopathic pulmonary arterial hypertension and iron deficiency. Pulm Circ. 2015 Sep;5(3):466-72. 5. Vitamin C: should we supplement? Curr Opin Crit Care. 2018 Aug;24(4):248-255. 6. Association between vitamin C deficiency and dialysis modalities; Nephrology (Carlton). 2012 Jul;17(5):452-7.

Vit C Status	Vit C Level	Date	CVP	PAS	PAD	PA(m)	Wedge
		3-Feb	8	52	20	31	
		2-Feb	10	50	20	30	
		1-Feb	8	48	20	29,333333	
		30-Jan	10	44	22	29	
		29-Jan	8	40	18	23	9
		28-Jan	10	28	20	23	16
		27-Jan	5	36	12	20	8
Vit C On		26-Jan	4	38	20	26	8
		25-Jan	10	37	17	24	10
	wedge fron	24-Jan	8	38	18	25	10
		23-Jan	6	38	18	25	10
		22-Jan	7	39	18	26	10
		21-Jan	4	48	12	24	8
		20-Jan	6	40	16	24	10
		19-Jan	10	42	20	27	11
		18-Jan					
		17-Jan	10	32	18	23	12
		16-Jan					
	Lab res 0.2	15-Jan	6	24	12	16	6
	Lab Res	13-Jan	8	36	24	28	24
		12-Jan	10	46	20	29	20
		11-Jan	10	38	20	26	10
	0,2	9-Jan	8	32	15	21	6
		8-Jan	8	31	18	22	11
		7-Jan	12	34	24	26	13
	0,4	6-Jan	10	36	24	28	12
		5-Jan	back to ICU				
		3-Jan	Swan d/c 2/2 infection				
Vit C Off	Lab Res	2-Jan	12	46	22	28	10
		1-Jan	12	40	21	30	15
		44196	10	32	20	24	13
cut dose		30-Dec	11	34	18	23	13
Vit C on		44194	11	34	22	26	15
	0,8	28-Dec	11	34	18	23	14
		44192	11	32	16	21	12
Vit C off		26-Dec	22	40	30	33	26
		44190	8	32	16	21	12
		24-Dec	14	48	24	32	18
		44188					
		22-Dec	11	47	22	31	18
Vit C On		44186	10	38	20	27	14
		20-Dec					
		44184					
	Lab res	18-Dec	10	30	20	23	13
		44182	8	34	17	23	13
		44181					
		15-Dec	15	44	24	31	14

	0,1	44179	14	49	23	32	15
		44178	12	48	24	32	16
		12-Dec	9	52	20	31	12
		44176	11	39	19	26	14
		44175	12	47	21	28	15
		9-Dec	16	52	24	34	16
		44173	16	50	24	33	18
		44172	12	46	18	28	14
		6-Dec	14	44	20	31	16
		44170	14	46	18	27	14
		44169	6	32	16	21	6
		3-Dec	8	42	20	27	12
		44167	8	42	20	28	15
		44166	6	32	20	25	12
		30-Nov	8	36	20	25	14
		44164	8	56	24	35	20
		44163	8	48	20	29	16
		27-Nov	18	60	28	39	24
		44161	10	44	24	31	8

TPGc	DPGc	PVRc	CO	CI	AvgPAm	AvgW	AvgTPG	AvgDPG
		187,75510	9,8	4,83			11	5,6666666
		120,30075	13,3	6,5				
		172,73954	9,88	4,87				
		193,38422	7,86	3,87				
14	9	150,56461	7,97	3,91				
7	4	157,57575	6,6	3,25				
12	4	159,15119	7,54	3,71				
18	12	196,20958	8,97	4,2			14,4	8,2
14	7	167,41405	6,69	3,3				
15	8	279,83539	4,86	2,39				
15	8	226,86567	6,7	3,3				
16	8	195,12195	7,79	3,84				
16	4	314,34184	5,09	2,51				
14	6	220,85889	6,52	3,21				
16	9	206,68693	6,58	3,21				
11	6	193,66852	5,37	2,65				
10	6	208,33333	3,84	1,89				
		318,09145	5,03	2,47				
		348,62385	4,36	2,15				
16	10	221,07081	5,79	2,84				
15	9	247,61904	4,2	2,03				
11	7	257,47126	4,35	2,82				
13	11	207,02402	5,41	2,83				
16	12	274,80916	5,24	2,74				
18	12	182,33618	7,02	3,68	26,5454541	15,0909091	11,4545455	5,7272727
15	6	263,73626	5,46	2,86				
11	7	189,83050	5,9	3,1				
10	5	151,65876	6,33	3,37				
11	7	188,67924	6,36	3,12				
9	4	197,93814	4,85	2,37				
9	4	130,50709	6,13	3				
7	4	162,96296	5,4	2,65				
9	4	175,37942	5,93	2,91				
14	6	187	6	2,9				
13	4	201	6,38	3,13				
13	6	249,54128	5,45	2,59	29,0454541	14,4090909	14,6363636	6,6818181
10	7	184,39716	5,64	2,76				
10	4	187,5	6,4	3,14				
17	10	200,31298	6,39	3,13				

17	8	246,99828	5,83	2,86				
16	8	294,11764	5,44	2,67				
19	8	341,08527	5,16	2,52				
12	5	229,44550	5,23	2,56				
13	6	254,47316	5,03	2,47				
18	8	276,39155	5,21	2,55				
15	6	256,60377	5,3	2,6				
14	4	263,91752	4,85	2,38				
15	4	259,04761	5,25	2,57				
13	4	195,85687	5,31	2,06				
15	10	250	4,8	2,35				
15	8	298,03921	5,1	2,5				
13	5	259,74025	6,16	3,02				
13	8	257,62711	5,9	2,89				
11	6	261,03646	5,21	2,55				
15	4	380,28169	5,68	2,78				
13	4	269,66292	6,23	3,05				
15	4	303,24909	5,54	2,72				
23	16	380,95238	4,41	2,16				

						795
		Vel			CVVH yes	955
			11	80TID		1038
						963
			15	60TID	CVVH ?	1129
			15	40 TID	Bumex gtt	1013
						935
						1022
						1005
						1072
			40			1099
						1142
			15			961
						962
				?		1197
						924
						938
	10	0,5	40			1342

AvgPVR	DBA	Mil	iNO/Vel	Sildenafil	Renal	SVR
163,06731						
240,23798						
						2,0926756
						2,5555555
						693
184,63883						
	2,5	0,125		80		908
						859
	2,5				iHD (numbers then rise)	893
						887
	2,5					
	5					782
	7,5	0,125	off			869
			Vel	On		790
265,94256					Petechial Rash	1073
					Delirious	922
					Thrombocytopenia	887
		0,25		OFF		738

		253		56,40%				
		294		55,60%				
		183		55,20%				
		206		54,50%				
		276		55,70%				
		226		58,20%				
		212		53,40%				
		228		57,20%				
		250		54%				
		300		58%				
		169		63,90%				
		176		63,70%				
		169		58%				
				59,8				
				62%				
				47,40%				

SVRc		PVR		SVO22				
167,2								
		194		67		ABG	7.43/30/82/19927406	
				54				
				53%				
		126						
		138						
		148						
		117						
				50%				
		121		54,8				
		187		55%				
		201		66				
		154		60,50%				
		142		62,10%				
		125		64,60%				
		213		63,30%				

Critical Care - 18 A Unique Case of Unintentional Isopropyl Alcohol Toxicity

James D Bacon[1], Meredith Owens[2], Manpreet Datta[2], Blair Lawless[2]

[1]University of Kentucky, Lexington, KY, [2]University of Kentucky Albert B Chandler Hospital, Lexington, KY

Introduction: Isopropyl alcohol (IPA) is the most common toxic alcohol ingestion reported to the US Poison Control Centers each year. IPA toxicity results in a unique set of lab findings in relation to other toxic alcohol ingestions, and can result in a falsely elevated creatinine level. Although toxic oral ingestion is common, transdermal toxicity is more rare. We present a unique case of an unintentional transdermal IPA toxicity.

Methods: A 79-year-old male with past medical history of CKD presented to our hospital with acute encephalopathy requiring intubation after being found unresponsive in his bed. Twelve empty bottles of IPA were found in the kitchen sink concerning for toxic ingestion. Laboratory findings included IPA level 110 units and acetone level 131 units. Additional laboratory values included an anion gap 14.5, osmolar gap 25, and a creatinine level of 1.72 (baseline 1.1-1.4). Despite an elevated creatinine level, urine output remained adequate. Toxicology was consulted without additional recommendations. He was extubated hospital day one and admitted to pouring IPA on his mattress to kill bed bugs prior to laying down to sleep resulting in a diagnosis of transdermal IPA toxicity. On hospital day two, the patient was deemed to have decisional capacity by behavioral health and signed out against medical advice. IPA poisoning represents a unique presentation when compared to other alcohol poisonings. While all alcohol toxicities typically result in altered mental status, IPA toxicity displays unique laboratory findings including an elevated osmolar gap, ketosis without an anion gap, and ketonuria. A positive isopropyl level can aid in making the diagnosis as in our case. Creatinine levels may be falsely elevated in laboratories that use the Jaffe reaction to measure creatinine levels. The transdermal route of absorption of IPA often leads to prolonged symptoms and may require skin decontamination. Regardless of the route of IPA poisoning, the standard treatment of care is supportive care. Hemodialysis in some cases can be considered if significant hemodynamic compromise remains after fluid resuscitation as well as an IPA level of >500 mg/dL (80 mmol/L). Fomepizole and ethanol should be avoided in the treatment of IPA toxicity as blocking alcohol dehydrogenase with prolong intoxication.

Conclusion: Not applicable.

Critical Care - 19 Peripartum Veno-venous ECMO for Severe Refractory COVID-19 ARDS

Jonathan A Molina Cifuentes[1], Burak Zeybek[1], Bhoumesh Patel[1]

[1]Yale New Haven Hospital, New Haven, CT

Introduction: Since January 2020, the prevalence of COVID-19 among pregnant women is 141,499 cases, with only 0.42% (606 cases reported) requiring admission to the ICU1. From those, only 4.4% have required extracorporeal membrane oxygenation (ECMO). Prior to COVID-19, some studies evaluated the maternal and fetal survival rates of ECMO during pregnancy and reported rates up to 77.2% and 69.1% for mother and the fetus, respectively2. We present a patient at 32 weeks gestation with severe acute respiratory distress syndrome (ARDS) due to COVID-19 pneumonia, who was successfully treated with peripartum initiation of veno-venous ECMO (V-V ECMO) after emergency cesarean delivery.

Methods: The patient is a 27-year-old G5P1112 with a 32-week pregnancy and asthma, who was transferred to our center with hypoxemic respiratory failure in the setting of COVID-19 pneumonia. She arrived intubated with a PaO2/FiO2 of 76. Fetal monitoring was established and was initially reassuring. On day 3 of admission, she had episodes of refractory hypoxemia associated with signs of fetal distress despite proning, initiation of nitric oxide and paralysis. She underwent an emergent cesarean section followed by the initiation of V-V-ECMO. ECMO configuration was right femoral vein to right internal jugular vein. ECMO settings were RPM 3800 with 4-4.3 LPM, Sweep gas flow 2-6 LPM. Heparin was used for anticoagulation while on ECMO with PTT GOAL 50-70 and Anti Xa 0.3-0.7. Patient underwent tracheostomy on ECMO Day 7. Ultra-lung protective ventilation (Plateau pressure < 25, RR < 12, FiO2 40%) strategy was utilized while on ECMO. ICU course was complicated by left pulmonary alveolar hemorrhage on ECMO Day 8 which was initially treated with lung isolation and intrapulmonary tranexamic acid (Figure 1). However, due to persistent alveolar hemorrhage patient underwent left bronchial artery embolization. She was weaned off ECMO on day 24. Over the following weeks, the patient gradually recovered her pulmonary function. She came off mechanical ventilation on day 36 and the tracheostomy was decannulated on day 57. She was discharged home on day hospital 70.

Conclusion: Extracorporeal membrane oxygenation should be considered early when conventional therapy is ineffective. The fundamental role of ECMO is the temporary support of cardiac and/or lung function in the management of potentially reversible effects, providing time necessary for recovery of cardiac and/or respiratory function. ECMO has been successfully use for the management of critical illness in pregnant and postpartum patients, including during the previous H1N1 influenza pandemic. There is very high-maternal survival rates with ECMO support in the management of COVID-19 associated severe ARDS, highlighting that pregnant and postpartum patients should be supported with ECMO in severe refractory ARDS. After the COVID-19 pandemic, some societies such as the Society of Maternal Fetal Medicine (SMFM) have developed recommendations regarding ECMO in pregnancy (Figure 2) 2. The timing and mode of ECMO for these patients require a careful discussion of risks and benefits among multidisciplinary team members and their families. Although delivery may have some benefits due to reduction on physiological demands of patients with severe disease such as COVID-19 related myocarditis or refractory hypoxemia, the decision of delivery is still made based on obstetrical indications 2,3

References: 1. Prevention C for DC and. COVID Data Tracker. Published March 28, 2020. Accessed November 2, 2021. https://covid.cdc.gov/covid-data-tracker/#pregnant-population. 2. Halscott T, Vaught MJ, Force SC 19 T. Management Considerations for Pregnant Patients With COVID-19. Society for Maternal-Fetal Medicine. Published online 2021. https://www.smfm.org/covidclinical. 3. Douglass K, Strobel K, Richley M, et al. Maternal-Neonatal Dyad Outcomes of Maternal COVID-19 Requiring Extracorporeal Membrane Support: A Case Series. Am J Perinat. 2020;38(01):082-087.

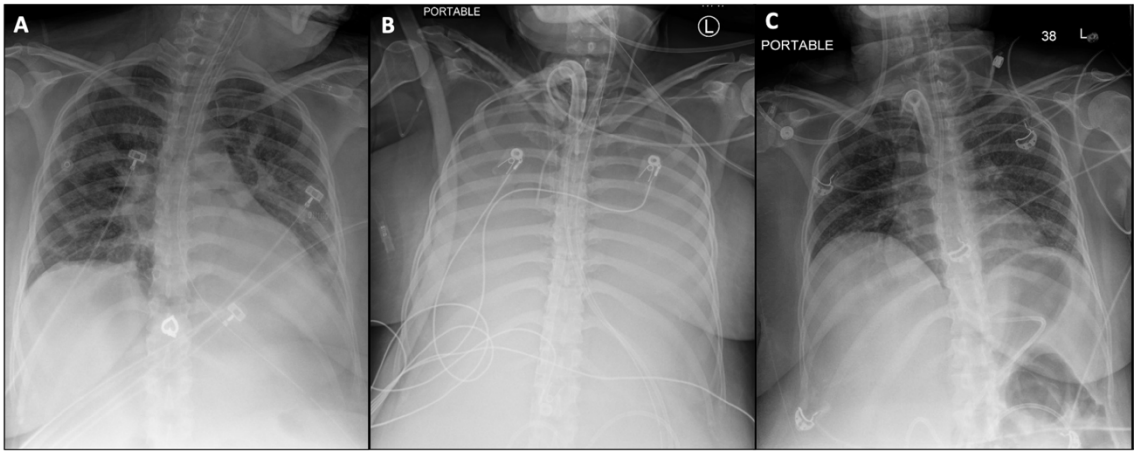


Figure 1. Radiographic progression of chest X-Ray (CXR). A: CXR on admission. B: CXR during left pulmonary alveolar hemorrhage. C: CXR before tracheostomy decannulation.

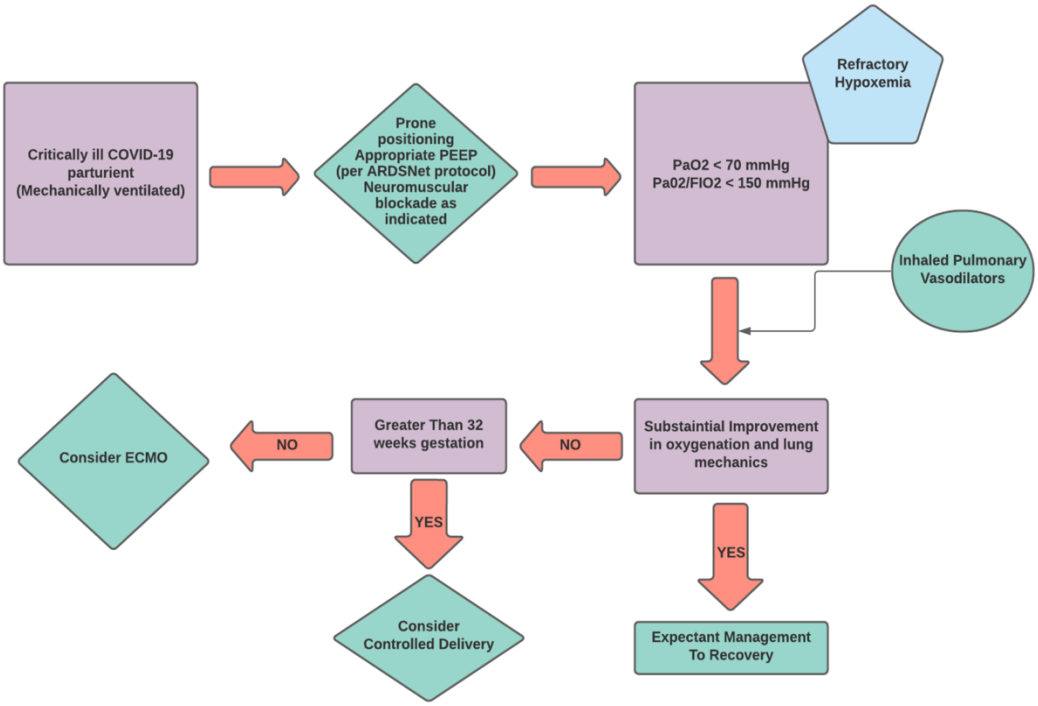


Figure 2. Suggested algorithm for refractory hypoxemia[2].

Critical Care - 20 Recognition and Management of High-Grade Tracheal Stenosis: A Post-COVID Phenomenon and an Anesthesiologist's Potential Nightmare

Grace Donzelli[1], Hisashi Tsukada[1], Alissa Sodickson[1], Gyorgy Frendl[1]

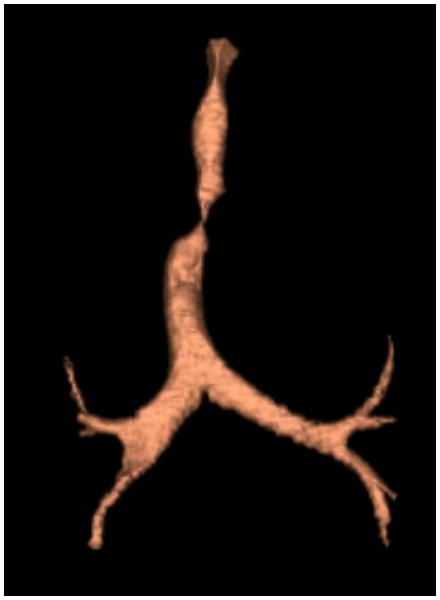
[1]Brigham and Women's Hospital, Boston, MA

Introduction: Tracheal stenosis can be a serious complication of prolonged endotracheal intubation, originally described post-polio epidemic. The COVID-19 pandemic created an unprecedented need for mechanical ventilation and proning, conditions that increase the risk of developing this condition.[1] Patients may subsequently present with high-grade lesions that are difficult to treat.[2,3] Tracheal stenosis, especially if unrecognized, poses a challenge to anesthesiologists, as it may render endotracheal intubation difficult or impossible. We present a case of post-COVID high-grade proximal tracheal stenosis and its management.

Methods: A 60 year-old woman with recent history of severe COVID pneumonia requiring a 13-day intubation presented with respiratory distress and stridor. In the three months following her COVID infection, the patient had experienced increasing dyspnea on exertion, worse for three days prior to admission. CT scan revealed narrowing of the proximal trachea measuring 6 mm x 9 mm in diameter (Fig. 1; normal female trachea measures 15 x 21 mm). The patient was taken to the OR for rigid bronchoscopy, which showed stenosis at 2-3 cm below the vocal cords. The stenosis was mechanically dilated, and an hourglass-shaped silicone stent was placed across the narrowing and balloon dilated. The patient was discharged home on POD1. Over the following sixteen months, the patient had recurrent respiratory symptoms, requiring a total of 25 (including 10 emergent) endoscopic interventions to maintain airway patency. Modalities used in treatment included mechanical and balloon dilatation, stent placement, laser ablation, cryoablation, and topical steroid injections. She continues to have monthly bronchoscopic treatments with steroid injections and dilatation as an outpatient.

Conclusion: Clinicians should maintain a high index of suspicion for tracheal stenosis in patients who required prolonged intubation for severe COVID infection, especially if prone position was utilized. Optimal treatment for tracheal stenosis has not yet been clearly defined, but options include interventional bronchoscopy, surgical resection, and bypassing tracheostomy.[2,3,4] While tracheal resection is considered the definitive treatment for tracheal stenosis of distal lesions, many cases of post-COVID tracheal stenosis are found in the proximal, subglottic region and not ideal for surgical resection. We describe the case of a patient who developed a high grade, complex, proximal tracheal stenosis after intubation for COVID. The patient was treated with serial endoscopic interventions including laser, cryotherapy and steroid injections, and was able to resume activities of daily living. As patients with tracheal stenosis remain asymptomatic until high-grade stenosis cause stridor, anesthesiologists need to be concerned about unrecognized cases where difficulties with endotracheal intubation could present in an unanticipated manner.

References: 1. Eur Arch Otorhinolarygo, 278(1): 1-7, 2021. 2. Eur Respir J, 13: 888-893, 1999. 3. BMC Pulm Med, 8:18, 2008. 4. J Thorac Cardiovasc Surg, 109: 486-493, 1995.



Critical Care - 21 ECPella Use in COVID-19 Myocarditis: A Case Series

Jordan Holloway[1], Matthew C Henn[1], Veena Satyapriya[1]

[1]Ohio State University Wexner Medical Center, Columbus, OH

Introduction: Viral myocarditis in COVID-19 infection has a prevalence ranging from 0.3% when screened for symptoms to 2.3% when screened with imaging (1). Symptoms can range from mild to cardiogenic shock. This series outlines two cases of COVID-19 myocarditis who presented in cardiogenic shock requiring veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and Impella (i.e. ECPella) and survived treatment with improvement in ejection fraction (EF) prior to discharge. These cases demonstrate ECPella as a successful treatment for patients who present with cardiogenic shock due to COVID-19 myocarditis.

Methods: Case A: 55 year old male with hypertension who presented to an outside institution with dyspnea and COVID pneumonia (PNA). He developed atrial fibrillation with rapid ventricular response and arrested due to hypoxia and acidosis with return of spontaneous circulation. Bedside transthoracic echocardiography (TTE) demonstrated severe left ventricular (LV) dilatation with an EF of <10% with diffuse akinesis and mild to moderate right ventricular (RV) dysfunction. He was cannulated for VA-ECMO and an Impella CP was placed in preparation for transfer to our institution. Five days later, his Impella was removed, followed five days later by decannulation from VA-ECMO. He was extubated three days after decannulation to high-flow nasal cannula. His course was complicated by new onset renal failure, gastrointestinal bleed, and secondary PNA. He was discharged to inpatient rehabilitation. TTE prior to discharge demonstrated a EF of 30% with LV dilated dysfunction and mild RV dysfunction. Case B: 37 year old male who presented to an outside institution with COVID-19 myocarditis in cardiogenic shock and acute hypoxic respiratory failure. His TTE demonstrated a dilated LV with an EF of <20% and RV free wall hypokinesis. He was subsequently cannulated for V-A ECMO and an Impella CP was placed prior to transfer to our institution. His Impella was removed five days later with VA-ECMO decannulation five days following. He developed refractory hypoxic respiratory failure following decannulation, prompting re-cannulation for veno-venous ECMO (VV-ECMO) and bedside tracheostomy. He was then weaned off of VV-ECMO. His course was complicated by cannulation site hematoma, acute renal failure, bilateral lower extremity paralysis due to spinal cord ischemia, secondary PNA, and deep venous thromboses. He had sufficient recovery for discharge to a long-term acute care hospital for ventilator wean with TTE prior to discharge demonstrative of mild, global LV hypokinesis with an EF of 45% and mildly reduced RV function.

Conclusion: We present two successful cases of the use of ECPella as a bridge to recovery for patients suffering from COVID-19 myocarditis. ECPella, i.e. the use of Impella in patients with VA-ECMO, has demonstrated mortality benefits for patients in cardiogenic shock, with a matched cohort demonstrating a hazard ratio of 0.79 for 30-day mortality (2). Increased afterload from VA-ECMO leads to worsening LV function with smaller stroke volumes, increased pulmonary capillary wedge pressure, cardiogenic edema, and RV failure (3). By utilizing Impella to overcome increased afterload associated with VA-ECMO, patients have improved forward flow, improved oxygen supply, improved LV unloading (reducing wall tension and microvascular resistance to improve myocardial oxygen supply and mechanical work), and reduced myocardial oxygen demand (3). Overall, this improves myocardial oxygen versus supply balance. For patients with viral myocarditis, this assists in the setting of cardiogenic shock, as the natural phases generally result in recovery or development of dilated cardiomyopathy (4). In this series, both patients survived to discharge despite onset of cardiogenic shock and experienced improvement in EF as measured by TTE, though will need longitudinal follow-up and may require future intervention. Benefits aside, compared to patients on VA-ECMO alone, those with ECPella have higher rates of bleeding, hemolysis, access site-related ischemia, abdominal compartment syndrome, and dialysis requirements (2). In this series, both patients had bleeding and renal complications. We were able to identify one case report of ECPella use in COVID-19 in the literature, though they did not survive (5). In summary, this demonstrates that ECPella can assist

patients in cardiogenic shock due to COVID-19 myocarditis and bridge to recovery or advanced treatment.

References: 1. JAMA Cardiol., Vol. 6, 1078-1087, 2021. 2. Circulation, Vol. 142, 2095-2106, 2020. 3. Journal of Cardiothoracic and Vascular Anesthesia, No volume available - in press, 1-10, 2021. 4. Nature Reviews Cardiology, Vol. 12, 670-680, 2015. 5. European Heart Journal - Case Reports, Vol. 4, 1-6, 2020.

Critical Care - 22 Veno Arterial Extracorporeal Membrane Oxygenation as a 'Bridge-to-Surgery' in Refractory Septic Shock

Aaron Whitby[1], Mahfuzul Majumdar[2], Matthew Wiepking[2], Se Fum Wong[3], Abirami Kumaresan[4]

[1]USC Keck Hospital, Los Angeles, CA, [2]USC Keck Hospital, Los Angeles, United States of America, [3]University of Southern California, Los ANgeles, CA, [4]Cedars-Sinai Medical Center, Los Angeles, CA

Introduction: The use of VA-ECMO as a bridge-to-surgery in refractory septic shock is a controversial topic. A 2021 meta-analysis which examined outcomes of VA-ECMO with refractory septic shock placed overall survival at 36.4%(1) in comparison to 45% for those with cardiogenic shock alone(2). We present here a case of a 69 year old man with a past medical history of GERD and hypertension who required veno arterial extracorporeal membrane oxygenation (V-A ECMO) following complications of an elective laparoscopic inguinal hernia repair.

Methods: This patient underwent an elective laparoscopic inguinal hernia repair at an outside hospital and was subsequently discharged home. Four days later, he presented to the same outside facility complaining of severe nausea, vomiting, and chest pain. Imaging at the outside facility found an incarcerated umbilical hernia from his laparoscopic port site as well as an esophageal perforation with mediastinal air and fluid likely secondary to retching. The patient acutely decompensated requiring vasopressors, intubation, bilateral chest tubes, and emergent abdominal surgery to repair his hernia. The patient also required a stent placed via EGD to seal off a 1cm perforation in his distal esophagus. Due to increasing pressor requirements and hemodynamic collapse the patient was unable to undergo thoracotomy for source control of his presumed intrathoracic infection. He was then transferred to our tertiary hospital for ECMO consideration secondary to refractory septic shock. The patient arrived at our facility on maximal vasopressor therapy. Transthoracic echocardiography demonstrated a depressed left ventricle with an ejection fraction of 28%. 12 hours after arrival the decision was made by our ECMO team to place the patient on V-A ECMO. Soon after initiation of V-A ECMO the patient was taken for thoracotomy and washout of his chest. Subsequently the patient's vasopressor requirement improved significantly and after four days the patient was successfully decannulated from V-A ECMO.

Conclusion: The decision to place this patient on V-A ECMO highlights the importance of patient selection and multi-disciplinary teams to facilitate ECMO cannulation. Although in refractory septic shock our teams decision was based on how the patient was otherwise healthy, had an elective procedure, and a definitive source of his infection with the potential for surgical control. In addition, the patient presented with septic shock combined with left ventricular failure which may respond better to V-A ECMO than septic shock alone (3). Multi-disciplinary teams used to facilitate and decide the appropriateness of ECMO have been shown to improve survival (4,5). Lastly this case brings up ethical considerations such as if we place a patient on ECMO as a 'bridge-to-surgery' how do we proceed if our intervention fails and it becomes a 'bridge-to-nowhere'?

References: 1. Crit Care 25, 246 (2021) 2. CJC Open. 2020 Sep 16 ;3(1):71-81. 3. Crit Care Med. 2019;47(8):1097-1105. 4. J Cardiothorac Vasc Anesth. 2019;33(4):902-907. 5. Ann. Intensive Care 10, 83 (2020).

Critical Care - 23 Right Ventricular Assist Device with an Oxygenator in a Patient with Diffuse Pulmonary Hemorrhage

Andrew Anderson[1], Babar Fiza[2]

[1]Emory University Hospital, Atlanta, GA, [2]Emory University, Atlanta, GA

Introduction: The clinical applications of extracorporeal circulatory and respiratory support devices have continued to evolve since their introduction in the mid-20th century. In this case report, we present a patient who needed an emergent addition of a membrane oxygenator to their right ventricular assist device (RVAD) circuit in the setting of diffuse pulmonary hemorrhage impeding ventilation.

Methods: The patient is a 52-year-old male with a medical history of recently diagnosed congestive heart failure with reduced ejection fraction (16%) after suffering from a stroke and saddle pulmonary embolus. The patient presented to our hospital with congestive heart failure exacerbation and cardiogenic shock requiring Impella® device implantation. The Impella® needed to be replaced with LVAD Heart-Mate 3® due to minimal clinical improvement. During the LVAD insertion, the patient could not be weaned from cardiopulmonary bypass due to severe right ventricular dysfunction. Thus a CentriMag® RVAD was placed using a 25F multistage venous cannula placed in the right atrium and a graft was placed for the pulmonary artery. The patient was extubated on postoperative day four but continued to have issues with coagulopathy, septic shock, cardiogenic shock, and renal failure post-operatively. On post-operative day 12, he developed an acute deterioration in his respiratory status with the development of significant hemothorax requiring chest tube insertion. Shortly after insertion of the chest tube, he developed significant hemoptysis requiring emergent intubation. Immediately following intubation, the patient was unable to be ventilated due to the lumen of the endotracheal tube being obstructed by a clot. Even with the constant retrieval of the large clots, and aggressive suctioning, he continued to form clots in the lumen of the endotracheal tube as well as in his tracheobronchial tree. At this time, the decision was made to emergently add a membrane oxygenator into the RVAD circuit. This allowed for stabilization of the patient's respiratory function while he received multiple blood products to address the massive bleeding. This technique provided near-apneic ventilation until coagulopathy was corrected and the clots obstructing the endotracheal tube and larger airways were cleared in the days that followed. This was essential as the extent of the clots resulted in multiple prolonged clot extractions and cryoablations with a bronchoscope. The patient was eventually able to be ventilated reliably days after the diffuse pulmonary hemorrhage occurred. The oxygenator was weaned and discontinued, and he was decannulated from the RVAD as well. Unfortunately, the patient continued to suffer from sepsis and a month later elected to transition to hospice care.

Conclusion: The indications for the addition of a membrane oxygenator to a RVAD circuit often involve respiratory failure following cardiac surgery which commonly happens secondary to pulmonary edema in the setting of cardiogenic shock (Mohite, 2015). Our case is unique in that the extracorporeal oxygenation allowed for gas exchange due to the constant obstruction of the endotracheal tube and the larger airways in the setting of massive pulmonary alveolar hemorrhage. Thus, an addition of a membrane oxygenator to the RVAD circuit can be utilized in cases of respiratory failure secondary to the inability to ventilate.

References: 1. Mohite, P. N., Sabashnikov, A., De Robertis, F., Popov, A. F., & Simon, A. R. (2015). Oxy-RVAD: rescue in pulmonary complications after LVAD implantation. Perfusion, 30(7), 596-599.

Critical Care - 24 Covid-19 Associated LVAD Thrombus

Ethan Garrigan[1], Abbas Al-Qamari[2]

[1]Northwestern, chicago, IL, [2]Northwestern University, Chicago, IL

Introduction: LVAD patients experience unique coagulation physiology and must balance the risk of thrombosis versus bleeding resulting in a narrow therapeutic anticoagulation range . Even though LVAD patients are at risk for a multitude of complications, pump thrombosis remains one of the largest contributors to mortality and morbidity. Fortunately complete LVAD outflow obstruction remains an exceedingly rare phenomenon. Thromboembolism has been reported in 20-30% of symptomatic Covid-19 infections. To the best of our knowledge, our case represents the first known Covid-19 pump thrombosis in the setting of appropriate anticoagulation in a patient fully vaccinated.

Methods: We present a case of a 71 year old male with a history of Stage D heart failure secondary to ischemic cardiomyopathy status post HVAD placement in December 2018. Other significant PMH includes CAD s/p LCx and RCA DES, multiple GI bleeds , AAA s/p EVAR, and CKD stage 3. He was admitted from the ED with persistent low-flow LVAD alarms, shortness of breath, cough and diarrhea approximately 2 years and 9 months after LVAD implantation.. Despite being fully vaccinated as of February 2021, he was found to be Covid-19 positive. On arrival VAD flows were noted to be zero. He was resuscitated, intubated and started on inotropic support and sent for a cardiac imaging. Imaging revealed complete obstruction of the LVAD outflow tract. He underwent emergent exchange from HVAD to Heartmate 3. Intra-op, HVAD outflow tract noted to have complete obstruction with a well organized thrombus. Post-operatively, his course was complicated by prolonged respiratory failure and copious secretions necessitating tracheostomy and prolonged ventilator weaning. Additionally he experienced acute on chronic renal failure requiring CRRT/HD prior to eventual renal recovery. He continues his recovery and remains hospitalized.

Conclusion: This case illustrates difficulties LVAD patients may experience with Covid-19 infection. Even at greater than two years from LVAD implantation, reports of complete outflow tract thrombosis are rare as incomplete pump thrombosis rates are reported at 8.4% at two years after LVAD implantation independent of anticoagulation status. Data on pump thrombosis in the setting of appropriate anticoagulation remains scant. Data on LVAD anticoagulation strategies is ever evolving and while most governing bodies recommend anticoagulation therapy in conjunction with antiplatelet therapy, this becomes less clear when the patient has suffered from multiple significant bleeding events in the past. In regards to Covid-19, our patient was fully vaccinated, however still suffered significant morbidity due to pump thrombosis, symptomatic Covid-19 pneumonia and renal failure requiring renal replacement therapy. Our patient was six months out from vaccination and would currently qualify for a booster dose. This case emphasizes the importance of optimally managing our most at-risk populations during the pandemic. Key points to take from this case are the importance of balancing thromboembolic risks against bleeding risks and how Covid-19 infection may influence this balance. Additionally, as Covid-19 data continues to evolve, the ideal vaccination schedule to minimize the risk of complications in LVAD patients should be considered.

References: Dealing with surgical left ventricular assist device complications. J Thorac Dis 2015;7(12):2158-2164 Prevalence of Venous Thromboembolism in Critically Ill COVID-19 Patients: Systematic Review and Meta-Analysis. Frontiers in Cardiovascular Medicine. 2021;7:338 Ann Cardiothorac Surg. 2014 Sep; 3(5): 450-471

Critical Care - 25 Non-Traumatic Subclavian Artery Dissection Causing Hemothorax

Khanant Desai[1], George Frendl[2]

[1]Brigham & Women's Hospital/Harvard Medical School, Boston, MA, [2]Brigham and Women's Hospital, Harvard Medical School, Boston, MA

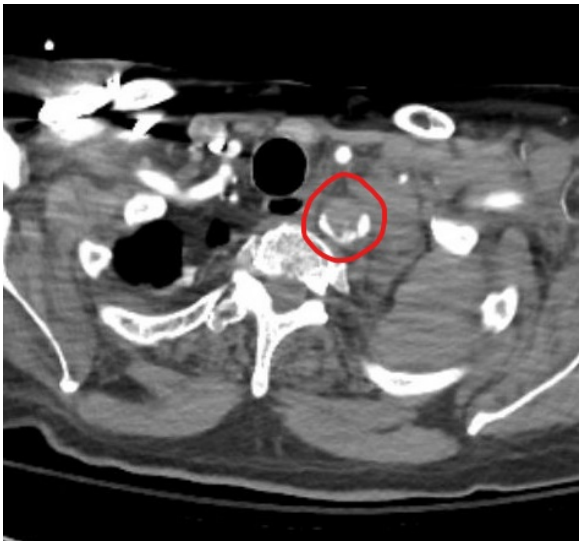
Introduction: Spontaneous subclavian artery dissection is an exceedingly rare finding in the absence of trauma or recent catheterization [1]. The differential diagnosis is broad, and presentation ranges widely from absence of symptoms to catastrophic hemorrhage, thrombosis, or end-organ malperfusion. Here, we present a patient found to have spontaneous hemothorax in association with subclavian artery dissection and intramural hematoma, in addition to dissections and vessel irregularity in multiple other arterial beds.

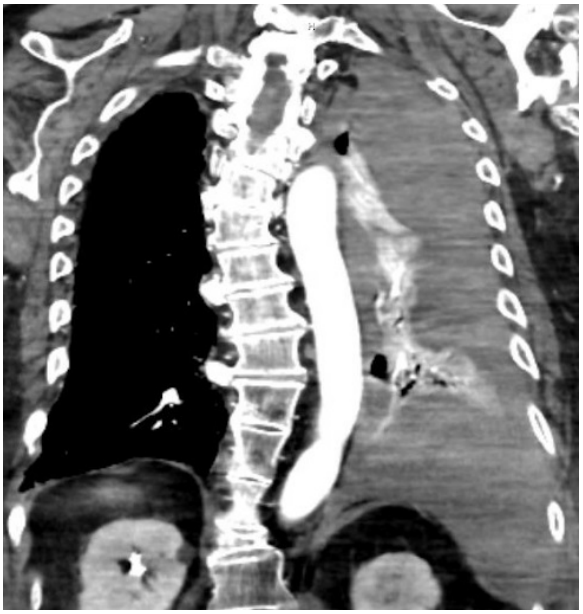
Methods: A 74 year-old male with history of paraplegia secondary to remote trauma may years prior to this admission, COPD and OSA, and remote spontaneous right pneumothorax s/p talc pleurodesis was admitted with sudden onset of dyspnea and left-sided pleuritic chest pain. He had recently developed a right mid-abdominal Zoster infection. On admission, he was found to have bacteremia with gram positive cocci, later speciating to viridans group streptococci. Blood pressure and heart rate were within normal limits. CT angiography (CTA) revealed left subclavian artery dissection and intramural hematoma, left hemothorax and extrapleural hematoma, and dissections with vessel irregularities in the celiac and bilateral external iliac arteries. Notably, affected arterial segments did not display significant atherosclerotic calcification. Transfusion was required, and despite ongoing bacteremia, a covered stent was deployed in the left subclavian artery due to potential for further massive hemorrhage. Left thoracoscopy and evacuation was performed twice due to hemothorax reaccumulation, after which the patient stabilized. Rheumatologic serologic workup including IgG4, ANA, anti-dsDNA, anti-Sm, ESR, ANCA, anti-CCP, RF was performed and was negative. TTE did not show valvular vegetation. Carotid duplex showed no stenosis, although neck MRA raised concern for focal non-flow-limiting left internal carotid artery dissection. Apixaban was initiated upon discharge and the patient will be followed in Vascular Medicine clinic.

Conclusion: While trauma, atherosclerosis, and hypertension are widely implicated in arterial pathology such as aneurysm formation and dissection, vascular abnormalities may rarely still arise in their absence. Spontaneous non-aortic arterial dissections may be asymptomatic or present with pain, particularly if there is rupture and hemorrhage, such as the large extrapleural hematoma and hemothorax our patient developed. Thrombosis and embolism may occur and cause stroke, limb ischemia, or other end-organ ischemia [2,3]. Physical exam should include a multi-point pulse examination and bilateral blood pressure measurement [1]. Multifocal arterial dissections suggest a systemic etiology such as infection, connective tissue disease, or vasculitis - these may be diagnosed with a combination of rheumatologic/infectious/inflammatory serum markers, CTA/MRA/duplex ultrasound imaging, and biopsy. Much rarer primary arteriopathies such as segmental arterial mediolysis and fibromuscular dysplasia display a distinct pattern of multifocal aneurysmal dilation and/or dissection [4]. Definitive treatment ranges from anticoagulation with surveillance imaging, to endovascular stenting, to surgery, and largely depends on severity of presentation [5].

References: 1. Catanese V, Alberto Pegorer M, Bissacco D, Di Gregorio S, Dallatana R, Settembrini P. Conservative management in a young woman affected by isolated left subclavian artery dissection. Ann Vasc Dis. 2014;7(3):347-349. doi:10.3400/avd.cr.14-00060 2. Winblad JB, Grolie T, Ali K. Subclavian artery dissection. Radiol Case Rep. 2015;7(4):626. Published 2015 Dec 7. doi:10.2484/rcr.v7i4.626 3. Marik PE, Mclaughlin MT. Spontaneous subclavian artery dissection: a pain in the neck diagnosis. BMJ Case Rep. 2013;2013:bcr2013201223. Published 2013 Nov 21. doi:10.1136/bcr-2013-201223 4. Skeik N, Olson SL, Hari G, Pavia ML. Segmental arterial mediolysis (SAM): Systematic review and analysis of 143 cases. Vasc Med. 2019;24(6):549-563. doi:10.1177/1358863X19873410 5. Williams S, Pagan Jaramillo A, Posch ML, Prince SA, Hamera L.

Idiopathic Left Subclavian Artery Dissection. Cureus. 2020;12(12):e12151. Published 2020 Dec 18. doi:10.7759/cureus.12151





Critical Care - 26 The spontaneous development of a retroperitoneal hematoma in a patient with a left ventricular assist device.

Mahfuzul Majumdar[1], Matthew Wiepking[1], Abirami Kumaresan[2], Se Fum Wong[3], Aaron Whitby[4]

[1]USC Keck Hospital, Los Angeles, United States of America, [2]Cedars-Sinai Medical Center, Los Angeles, CA, [3]University of Southern California, Los Angeles, United States of America, [4]USC Keck Hospital, Los Angeles, CA

Introduction: Anticoagulation is a mainstay of therapy in patients requiring mechanical circulatory support. Left ventricular assist devices (LVAD) are becoming increasingly prevalent, as they provide not only destination therapy but also a bridge to heart transplant for patients with end stage heart failure. LVADs are associated with numerous coagulopathic complications, including bleeding, pump thrombosis, embolic strokes, and iatrogenic platelet dysfunction while simultaneously creating increasing bleeding risk. This case demonstrates a patient with end stage heart disease, who developed a spontaneous retroperitoneal hematoma three weeks after LVAD placement.

Methods: Our index patient is a 59-year-old man with a past medical history of end stage ischemic cardiomyopathy, who was transferred from an outside hospital to our institution for advanced heart failure therapies. He underwent placement of a Heart Mate 3 (Abbot) LVAD as destination therapy after a trial of inotropes and Impella 5.5 (Abiomed) support. His postoperative course was complicated by development of a hemothorax on postoperative day (POD) # 3, requiring reintubation and ultimately tracheostomy on POD # 14. His initial anticoagulation strategy with a heparin drip was modified to a lower partial thromboplastin time (PTT) goal secondary to his hemothorax. On POD # 22, our patient was noted to have increased pressor requirements, decreased flows on his LVAD, a lactic acidosis, an acutely unstable hemoglobin requiring multiple blood transfusions with associated right lower abdominal pain. There was a significant concern for ischemic colitis- and emergent imaging of the abdomen and pelvis demonstrated an unexpected large spontaneous retroperitoneal hematoma (RPH) measuring 21 cm in diameter. He underwent a successful gelfoam arterial embolization by interventional radiology of the lumbar arteries via a left femoral approach. The evening following the embolization, our patient was noted to have a pulseless left lower extremity. An emergent arterial duplex demonstrated an occlusion of the left femoral vasculature. On POD #23, he was taken for a thrombectomy and endarterectomy of his left femoral arterial system. He subsequently underwent a below the knee amputation (BKA) on POD #55, redemonstrating the delicate balance between bleeding and thrombosis in the LVAD patient.

Conclusion: The advent of advanced heart failure therapies has led to a new generation of complications. LVAD patients are prone to bleeding, but continuously teeter on the precipice of a prothrombotic state. LVAD recipients have complex anticoagulation needs; some require dual antiplatelet therapy (DAPT) for recent cardiac stenting, while others may require systemic anticoagulation for a mechanical valve or atrial fibrillation. In the coming years LVAD patients will become increasingly prevalent, as will their associated complications. Our patient developed a RPH, and a subsequent BKA from the complications of addressing this hemorrhage. His postoperative hemothorax dictated a lower PTT goal, and he was managed with aspirin and systemic heparin alone. Current anticoagulation strategies in LVAD patients are varied. Some institutions have been known to start warfarin without a heparin bridge. Heparin can be associated with the development of heparin induced thrombocytopenia (HIT) in approximately 4% of patients[1]. Aspirin is almost universally a mainstay of therapy, but platelet dysfunction is intrinsic to LVAD patients as they develop an acquired von Willebrand syndrome from the shearing forces of the impeller[2]. As per our institutional best practices, our anticoagulation strategies are managed by our pharmacy department in tandem with our cardiothoracic surgeons as we recognize the complexity of our patients. Despite our cautious approach, our patient developed a retroperitoneal hematoma which led to an eventual BKA. LVAD patients are far from homogenous, and their anticoagulation strategies require intricate consideration.

Unfortunately, this makes it difficult to establish standardized protocols, which in turn may lead to an increase in patient morbidity and mortality.

References: Expert Review of Cardiovascular Therapy, Volume 18, Issue 6, 363-372, 2020

Critical Care - 27 Parallel veno-venous extracorporeal membrane circuits for refractory hypoxemia in extremely morbidly obese patient

Melina Shoni[1], Sofiane Lazar[1], Andrea Jackson[1], Mary Tonetti[1], Jiri Horak[1], Jacob Gutsche[1]

[1]Hospital of the University of Pennsylvania, Philadelphia, PA

Introduction: The expanded indications for extracorporeal membrane oxygenation (ECMO) have allowed higher-risk patients with challenging clinical scenarios to be successfully managed with this platform. Traditionally, extreme morbid obesity has been considered a contraindication to ECMO due to the risk of inadequate flows for the body surface area (BSA) as well as hemolysis with secondary coagulopathy from attempted high ECMO flows (1). The complex cardiovascular and pulmonary physiology associated with morbid obesity may necessitate alternative ECMO configurations to overcome the limitations in oxygen delivery encountered with the conventional peripheral ECMO circuit (2, 3).

Methods: We present the case of a 31-year-old female with class III morbid obesity (Body Mass Index 77 kg/m², BSA 2.85 m²), uncontrolled hypertension, diabetes mellitus type II, tobacco use, anemia and recent COVID-19 pneumonia, who presents for elective hysteroscopy and dilation and curettage in the setting of recurrent abnormal uterine bleeding. The intraoperative course was complicated by difficult intubation and ventilation, hypoxemic respiratory failure and inability to extubate at the end of the procedure. The patient was transferred to the surgical intensive care unit where lung protective ventilation with high positive end-expiratory pressure and inhaled epoprostenol were initiated for severe acute respiratory distress syndrome [arterial partial pressure of oxygen (PaO2) to fraction of inspired oxygen (FiO2) ratio 69) and associated moderate right ventricular systolic dysfunction. Despite maximal ventilatory support, the patient experienced worsening hypoxemia and hypercapnia attributed to MSSA/H.influenza pneumonia and possible residual COVID-related lung injury. Proning was not attempted due to body habitus and airway concerns. On postoperative day 5, refractory hypoxemia led to the institution of veno-venous (VV) ECMO via a 29-French right femoral vein drainage cannula and a 19-French right internal jugular vein return cannula recognizing the risk of inadequate flows in the setting of her extremely high BMI. The patient had a transient improvement in oxygenation and hypercapnia after ECMO implementation but over the next eight days developed profound hypoxemia with PaO2 as low as 45 mmHg, acidemia and lactic acidosis while maintaining ECMO pump flows up to 7 L/min, sweep flows up to 11 L/min and FiO2 100%. The patient also developed a large spontaneous left hemothorax as well as retiform purpura in the setting of disseminated intravascular coagulopathy and thrombotic microangiopathy. On postoperative day 13, and after having ruled out oxygenator malfunctioning, cannula malposition, recirculation and abdominal compartment syndrome, the decision was made to implement a second VV ECMO circuit with a 25-French left femoral vein drainage cannula and a 15-French left internal jugular vein return cannula in parallel with the right-sided circuit. A total flow of 8.7 L/min was achieved with gradual improvement of the PaO2, allowing for ECMO wean with decannulation of the first and second circuit on postoperative day 16 and 26, respectively.

Conclusion: This report demonstrates that parallel VV ECMO circuits can be used to treat refractory hypoxemia in patients with acute respiratory failure and extreme morbid obesity. Despite her high BMI, our patient was considered an ECMO candidate provided her young age, the reversibility of her acute disease, the timeline of her respiratory failure and a sonographic assessment indicating an approximation of the required ECMO flows for her BSA, as well as a promising vasculature to accommodate the optimal cannula size for her features. Our efforts focused first on maximizing the potential of conventional VV ECMO by manipulating the ventilator, maintaining deep sedation and paralysis, and minimizing the risk of recirculation and shunt. The conventional VV ECMO circuit failed to meet her oxygen needs due to insufficient flows. The parallel VV ECMO circuit improved oxygen delivery by achieving higher additive flows.

References: 1. Brodie D, Bacchetta M. Extracorporeal membrane oxygenation for ARDS in adults. N Engl J Med. 2011;365(20):1905-14. 2. Malik A, Shears LL, Zubkus D, Kaczorowski DJ. Parallel circuits for refractory hypoxemia on venovenous extracorporeal membrane oxygenation. J Thorac Cardiovasc Surg. 2017;153(3):e49-e51. 3. Sorokin V, MacLaren G, Vidanapathirana PC, Delnoij T, Lorusso R. Choosing the appropriate configuration and cannulation strategies for extracorporeal membrane oxygenation: the potential dynamic process of organ support and importance of hybrid modes. Eur J Heart Fail. 2017;19 Suppl 2:75-83.

Critical Care - 28 Intracranial hemorrhage in a pregnant patient with HELLP syndrome

Mohammed A Hussain[1], Lindsay D Nowak[1], Allison Mullins[2], Rakesh Vadhera[1], Beth M Teegarden[3]

[1]University of Texas Medical Branch, Galveston, TX, [2]University of Texas Medical Branch, Galveston, United States of America, [3]The University of Texas Medical Branch at Galveston, Galveston, TX

Introduction: Intracranial hemorrhage (ICH) is relatively uncommon in pregnancy, with an estimated incidence of 0.01 to 0.05%, with vascular malformations as the most common cause of hemorrhagic stroke in this patient population [1-3]. Often, the presenting symptom is new onset tonic-clonic seizures. In contrast, the incidence of eclampsia is relatively higher at 0.08%, which also has a similar presentation of generalized tonic-clonic seizures in a woman with preeclampsia [4]. Treatment and diagnostics are very different for the two. The added urgency of fetal wellbeing compounded by the diagnostic limitations associated with pregnancy often pigeonholes providers into a narrower differential diagnosis that can lead to devastating morbidity and mortality. With no established prior medical or obstetric care, a pregnant patient presents to a community Emergency Department with new onset seizures. She is transferred to our institution for a higher level of care. Emergency caesarean delivery is then complicated by HELLP syndrome. The patient further has delayed emergence postpartum; workup reveals an intracerebral hemorrhage with devastating neurologic injury. This is a medically challenging case for obstetric anesthesia and critical care.

Methods: A 32-year-old and 26 weeks pregnant female was transferred to our institution for severe ranging blood pressures and seizures concerning for eclampsia. The patient arrived obtunded after administration of 55 mg valium by an outside institution and non-reassuring fetal heart tones. Shortly after arrival, and prior to labs resulting, she was taken for an emergent c-section under general anesthesia. During surgery a coagulopathy was noted on labs, later diagnosed as HELLP syndrome. She was left intubated and sedated due to a poor neurologic exam, and admitted to the Surgical ICU. Despite sedation being stopped her neurological function did not improve, and she was further evaluated with an EEG and head CT. This revealed a large intraparenchymal hemorrhage with midline shift. Before any neurosurgical intervention, the patient's neurologic status abruptly worsened due to herniation. The family decided on a palliative extubation and autopsy, which noted her cause of death as ICH due to eclampsia.

Conclusion: This case of a parturient with ICH in the setting of HELLP syndrome demonstrates a few crucial points in managing a high risk parturient with neurologic abnormalities. The medical team should seek to stabilize the mother prior to delivery if possible. While the seizures in this case were initially attributed to eclampsia, given the difficulty in treating the seizures compounded by HELLP syndrome, further investigation into an underlying etiology is warranted. Perhaps if cerebral imaging had occurred earlier in her presentation, the ICH would have been detected and acted upon prior to devastating neurologic injury.

References: 1. Sadasivan B, Malik GM, Lee C, Ausman JI. Vascular malformations and pregnancy. Surg Neurol 1990;33:305-313 2. Robinson JL, Hall CJ, Sedzimir CB. Subarachnoid hemorrhage in pregnancy. J Neurosurg 1972;36:2 3. Ascanio, L et al. Neurocritical Care 2019; 30:5-15. doi.org/10.1007/s12028-018-0501-4 4. Ananth CV, Keyes KM, Wapner RJ. Pre-eclampsia rates in the United States, 1980-2010: age-period-cohort analysis. BMJ. 2013;347:f6564. doi:10.1136/bmj.f6564 5. Leeman L, Dresang LT, Fontaine P. Hypertensive Disorders of Pregnancy. Am Fam Physician. 2016 Jan 15;93(2):121-7. [PubMed: 26926408] 6. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists,Äô Task Force on Hypertension in Pregnancy. Obstet Gynecol. 2013 Nov;122(5):1122-1131. [PubMed: 24150027] 7. De Herdt V, et al. Early seizures in intracerebral hemorrhage: incidence, associated factors, and outcome. Neurology 2011:77(29) [PubMed: 21975203]

Critical Care - 29 A Case of Severe Amlodipine Toxicity Requiring Mechanical Circulatory Support

Roxanne Favis[1], Adeola Martins[1], Crystal Kyaw[1], Raghu R Seethala[2], Kathleen Haley[3]

[1]Brigham and Women's Hospital, Boston, MA, [2]Brigham and Women,Äôs Hospital, Westwood, MA, [3]Brigham and Women's Hospital, Boston, United States of America

Introduction: In the National Poison Data System Report, calcium antagonists (CCB) were among the top 6 fatal toxicities, with amlodipine being the most common. First line therapy for CCB toxicity is high-dose insulin euglycemic therapy to improve impaired sensitivity to insulin and reduced insulin secretion due to calcium antagonism. Insulin increases myocardial glucose uptake, enhancing ventricular contractility by increasing extracellular calcium and intracellular cAMP concentrations. We present a patient with amlodipine toxicity who also required VA-ECMO. The potential effects of ECMO on pharmacokinetics are not defined, and can complicate the management of toxicities.

Methods: A 38-year-old man with a history of hypertension, diabetes, depression, and obesity (BMI 44 kg/m2) was transferred in multipressor shock with and multisystem failure after initially complaining of chest pain. Empty amlodipine bottles (180 tablets each) were discovered at home. The patient had PEA arrest twice and was placed on VA-ECMO for circulatory support. During each arrest, intralipid and methylene blue was administered. The patient was started on a high-dose insulin (HDI) drip at 1 U/kg/hr with titration based on hemodynamics. Euglycemia was maintained with dextrose. His highest requirement was 350 U/hour (2.6U/kg/hr). A calcium chloride drip was started to maintain ionized calcium of 2mmol/L. The next day, he had an abrupt loss in pulsatility. Echocardiogram showed severe biventricular failure, whereas pre- and post-cannulation evaluation showed hyperdynamic function. An Impella (Abiomed Danvers, MA) vent was placed. Additional complications included respiratory distress syndrome (ARDS) with hypoxemia, renal failure requiring renal replacement therapy, rhabdomyolysis , and a soft tissue injury due to methylene blue extravasation. Despite aggressive interventions, he continued to decline and was transitioned to comfort care.

Conclusion: Though amlodipine is a common cardiovascular drug toxicity observed, severe cases are rare. This case was further notable for the additional complexity of mechanical circulatory support, refractory acidosis, musculoskeletal injuries, renal failure, and ARDS. Euglycemic HDI is effective therapy for CCB overdose. This patient's euglycemic HDI was within the dose range reported for CCB overdose (1 - 10 U/kg/hr), although HDI is frequently effective at lower doses than this patient required. His HDI required a large infusion volume for delivery despite maximally concentrating all drips. In the setting of the patient's cardiopulmonary failure, renal failure, and high infusion requirement, managing the patient's volume status was challenging. Hypervolemia complicates ECMO management and can worsen cardiac function. Our intention was to extend survival beyond drug clearance (30 - 50hr half-life) and await signs of recovery. This case highlighted the complexities of CCB overdose management and the limitations of available mechanical support.

References: 1. Gummin et al. 2019 Annual Report of the American Association of Poison Control Centers,Äô National Poison Data System (NPDS): 37th Annual Report. CLINICAL TOXICOLOGY. 2020. 58 (12):1360-1541. 2. S.J. Rietjens et al. Practical recommendations for calcium channel antagonist poisoning. The Netherlands Journal of Medicine. 2016. 74 (2): 60-67. 3. R. Masson et al. A comparison of survival with and without extracorporeal life support treatment for severe poisoning due to drug intoxication. Resuscitation. 2012. 83: 1413-1417 4. J.B. Cole el al. High dose insulin for beta-blocker and calcium channel-blocker poisoning. American Journal of Emergency Medicine. 2018 36: 1817-1824

Critical Care - 30 Tamponade physiology caused by tense ascites and bilateral pleural effusions.

Mena Isnassuos[1], Babar Fiza[1]

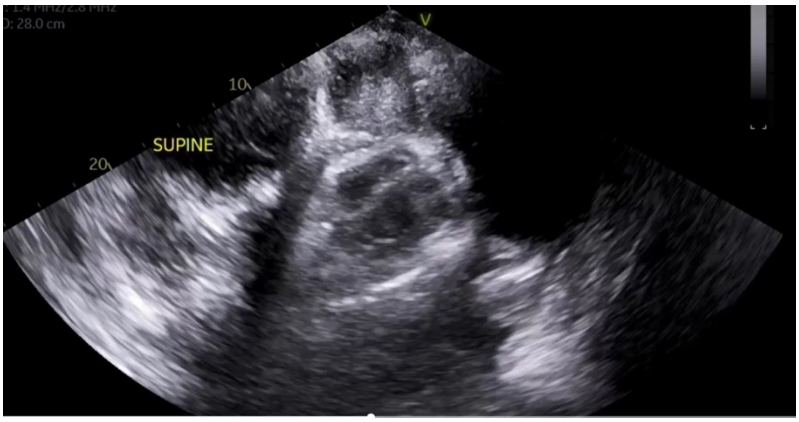
[1]Emory University School of Medicine, Atlanta, GA

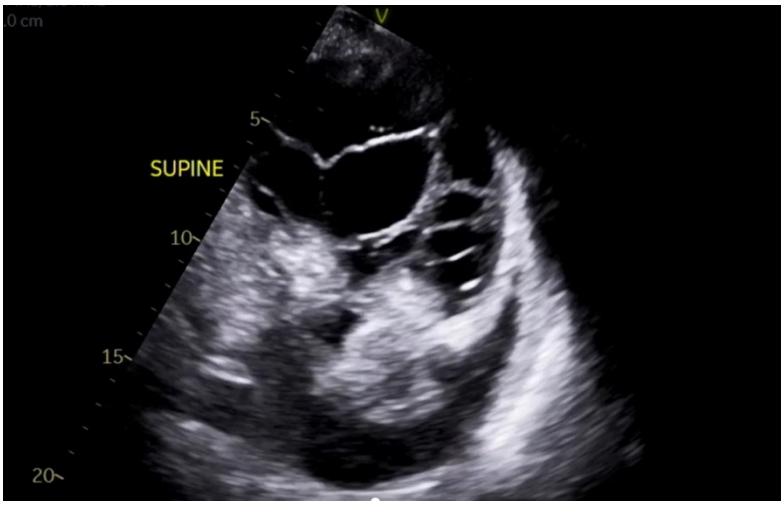
Introduction: We present a case of a 46-year-old female who developed tamponade physiology due to tense massive ascites and large bilateral pleural effusions in the presence of a small pericardial effusion. Previous case reports have detailed either ascites or pleural effusions as the cause of tamponade physiology. Although, there are no case reports of the simultaneous presence of ascites and pleural effusions as the cause of tamponade.

Methods: The patient is a 46-year-old female with alcoholic cirrhosis, recurrent pleural effusions, and ascites who presented with decompensated liver failure and worsening renal function. The patient presented to the intensive care unit with hyperammonemia and uremia. The patient was placed on continuous renal replacement therapy and progressively developed worsening hypotension and shock. A therapeutic paracentesis was attempted, but only a small amount of ascitic fluid could be removed due to the loculated nature of the ascites. The patient was started on empiric antibiotics as the shock state was presumed due to an underlying infection. There were no clinical improvements despite the antibiotic therapy. On physical examination, the patient was noted to have distended neck veins, pulsus paradoxus, tachycardia, and hypotension, indicating tamponade physiology. In addition, there was the presence of a distended and tense abdominal cavity. A bedside cardiac ultrasound was performed. The ultrasound was notable for large bilateral pleural effusion, loculated ascites, and a small pericardial effusion on the subcostal four-chamber view. The tamponade physiology was confirmed with the presence of right atrial systolic collapse. Given these findings, the patient underwent bilateral chest tube placement with the removal of 3 liters of pleural fluid. A fluoroscopy-guided paracentesis was performed, and four liters of ascitic fluid was removed, leading to shock resolution.

Conclusion: Cardiac tamponade can be a fatal process if not detected promptly. Large pleural effusions can lead to increased intrapleural pressure, which can be transmitted to the heart resulting in tamponade physiology. Tense ascites, common in patients with advanced liver disease, can result in external cardiac compression and impair venous return leading to tamponade-like physiology. This case illustrates the need to maintain a high vigilance for the extracardiac pathologies as the cause of tamponade physiology. This is essential in patients with advanced liver disease who commonly suffer from recurrent pleural effusions and ascites.

References: Gowani SA, Danielian A, Villaneuva J, Pickett C. Large pleural effusions causing cardiac tamponade: a case report and review of the literature. Conn Med. 2014 Mar;78(3):149-52. PMID: 24772831. Khaja, M., Santana, Y., Rodriguez Guerra, M. A., Rehmani, A., & Perez Lara, J. L. (2020). Isolated Left Atrial Cardiac Tamponade Caused by Pleural Effusion. Cureus, 12(11), e11578. https://doi.org/10.7759/cureus.11578 Ahmad S, Jamali HK, Waqar F, Harris DM. Cardiac Tamponade Physiology Secondary to Tense Ascites. Cardiology. 2016;134(4):423-5. doi: 10.1159/000445048. Epub 2016 Apr 28. PMID: 27120437.


V
10
SUPINE
20


V
5
SUPINE
10
15
20

Critical Care - 31 Nicardipine-Induced Hypoxemia After Pulmonary Thromboendarterectomy

Lakshmi Nemani[1], Abbas Al-Qamari[1]

[1]Northwestern University, Chicago, IL

Introduction: Hypoxic pulmonary vasoconstriction (HPV) is a normal physiologic mechanism whereby alveolar hypoxia triggers pulmonary arterial vasoconstriction to optimize ventilation/perfusion (V/Q) matching. HPV is a locally mediated response inhibiting blood flow to poorly oxygenated lung parenchyma, improving systemic oxygenation in certain disease states including asthma, chronic obstructive pulmonary disease (COPD), and acute lung injury [1]. HPV can be impaired by certain medications such as volatile anesthetics, phosphodiesterase inhibitors, and calcium channel blockers, causing worsening V/Q mismatch and oxygenation in patients with pulmonary disease [2]. Here we present a case of hypoxemia in a hypertensive patient on a nicardipine infusion following pulmonary thromboendoarterectomy (PTE) for chronic thromboembolic pulmonary hypertension (CTEPH).

Methods: An 83 year-old with a past medical history of CTEPH, coronary artery disease, hypertension, and chronic kidney disease stage 3 successfully underwent a pulmonary thromboendarterectomy. He was extubated on post-operative day (POD) 1 and quickly weaned to 40% fraction of inspired oxygen (FIO2) via high flow nasal cannula (HFNC) without issues. He became hypertensive with mean arterial pressure (MAP) greater than 100 mmHg so his home carvedilol was restarted and a nicardipine infusion was initiated with dosages ranging from 5-7.5 milligrams/hour. His oxygen saturation decreased from 98% to 92% and his arterial partial pressure of oxygen (PaO2) decreased from 88 mmHg to 63 mmHg on FIO2 40% after initiation of nicardipine. A chest x-ray demonstrated stable, small bilateral pleural effusions, mild bibasilar atelectasis, no pulmonary edema, and no pneumothorax. PaO2 improved from 86 mmHg to 122 mmHg on FIO2 70% after discontinuation of nicardipine and he was weaned down to FIO2 50% by the next morning with stable oxygen saturations ranging from 98-100%.

Conclusion: The differential for hypoxemia following PTE is broad, including pleural effusion, atelectasis, infection, pulmonary edema, and lung injury - which manifests as non-cardiogenic, reperfusion-related pulmonary edema [3]. Hypoxic pulmonary vasoconstriction can help attenuate hypoxemia by redirecting blood away from pathologic, poorly oxygenated lung tissue [1]. Calcium channel blockers have been shown in limited studies and case reports to inhibit HPV, worsen V/Q matching, and significantly impact PaO2 in disease states such as advanced COPD [4], pneumonia, and even primary graft dysfunction after lung transplantation [5]. This is the first case report demonstrating rapid development of hypoxemia in a post-PTE patient after initiation of nicardipine. Future areas of study may include identifying optimal anti-hypertensive agents in patients with underlying lung pathology to avoid precipitating hypoxemia through inhibition of HPV, as well as better describing the role of HPV in reperfused lung.

References: 1. Hypoxic Pulmonary Vasoconstriction: From Molecular Mechanisms to Medicine. Chest. 2017; 151(1):181-192. 2. Hypoxic Pulmonary Vasoconstriction: Physiology and Anesthetic Implications. Anesthesiology. April 2015, Vol. 122, 932-946. 3. Chronic Thromoboembolic Pulmonary Hypertension: Evolving Therapeutic Approaches for Operable and Inoperable Disease. Journal of the American College of Cardiology. 2018 May, 71 (21): 2468-2486. Vol. 71 No. 21. J Am Coll Cardiol. 4. Deleterious effect of nifedipine on pulmonary gas exchange in chronic obstructive pulmonary disease. Am Rev Respir Dis. 1984 Oct; 130(4):612-6. 5. Severe, Rapidly Reversible Hypoxemia in the Early Period after Bilateral Lung Transplantation. Annals of the American Thoracic Society. March 2016, Vol 13, Issue 6. 979-985.

Critical Care - 32 Atypical Presentation in Patient with Behçet's Syndrome

Reed S Geisler[1], Ty Gilkey[1], Chelsea Leipold[1], Nicholas Romano[2], Asok Dasgupta[2], Paresh Timbadia[2]

[1]The Ohio State University, Columbus, OH, [2]Mount Carmel East Hospital, Columbus, OH

Introduction: Behçet's Syndrome is a systemic vasculitis that clinically presents with recurrent oral aphthous ulcers, genital ulcers, & uveitis. The prevalence of Behçet's varies significantly by region & is most commonly seen in Turkish patients (80 to 370 cases per 100,000). An estimated 30%-40% of patients with Behçet's syndrome experience vascular involvement, which causes significant morbidity & mortality. 1 and 5 year mortality of Behçet's Syndrome are 1.2% and 3.3% respectively, with increased mortality in patients aged 15-24 years.

We report a case of rapidly developing, recurrent, & extensive vascular pathology in a young male with previously undiagnosed Behçet's Syndrome.

Methods: 19 year old African American male was admitted with scrotal pain, dyspnea, palpitations, fatigue, fever with chills, & non-bloody diarrhea. He had a four month history of scrotal pain & ulcerations requiring incision & drainage followed by multiple antibiotic courses.

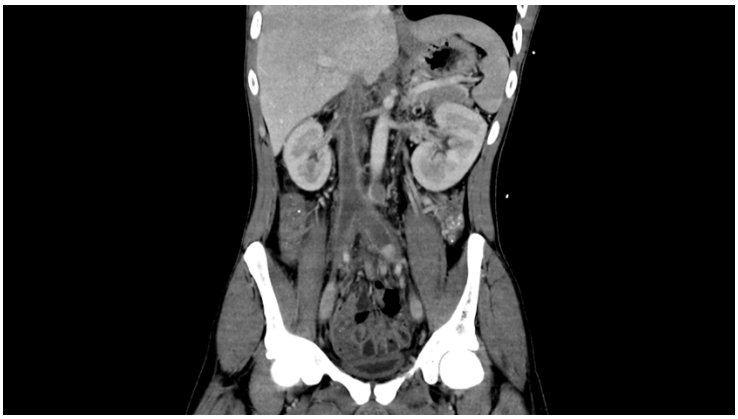
Past history was negative for venous thromboembolism (VTE) or smoking. Family history was negative for VTE. Physical exam showed critically ill male with hypotension & scrotal ulcerations. Labs showed lactic acidosis, anemia, coagulopathy, & acute kidney injury. Scrotal ultrasound was unremarkable for torsion or abscess. Computed Tomography (CT) of abdomen & pelvis revealed extensive thrombosis of the renal veins, inferior vena cava (IVC), & common iliac veins, along with bilateral lower lobe pulmonary emboli & small pulmonary infarctions. Echocardiogram was unremarkable. He was started on systemic anticoagulation, broad-spectrum antibiotics, intravenous fluids, & vasopressor therapy. Hypercoagulability workup was negative.

Mechanical thrombectomy of the IVC was performed with extensive thrombi removal & restoration of IVC patency. His wound cultures grew Methicillin-resistant Staphylococcus aureus & Pseudomonas aeruginosa resistant to fluoroquinolones. He was discharged with aspirin & apixaban for VTE, and colchicine & prednisone for Behçet's. Three months following discharge, the patient presented again with pulmonary thromboembolism and was switched from apixaban to warfarin following left iliofemoral recanalization, venoplasty, and stenting. Four months following initial discharge, the patient was started on apremilast per rheumatology. The patient has presented with left lower extremity deep vein thrombosis and/or pulmonary embolism for a total of five times in the past year and is actively followed for ongoing management.

Conclusion: Extensive VTE with IVC involvement is typically a late finding in Behçet's Syndrome, developing in a median of 5 years following disease onset. Our patient presented with extensive VTE and was subsequently diagnosed with Behçet's Syndrome. Although Behçet's Syndrome is typically seen in Middle Eastern/Turkish patients, it has been uncommonly described in Central African and Afro-Caribbean populations. Our case highlights the importance of recognizing an atypical initial presentation of Behçet's Syndrome in African American population. Unfortunately, the patient continues to experience recurrent VTE despite optimal management.

References: Seyahi E, Yurdakul S. Behçet's Syndrome and Thrombosis. Mediterr J Hematol Infect Dis. 2011;3(1):e2011026. doi:10.4084/MJHID.2011.026 Houman MH, Ben Ghorbel I, Khiari Ben Salah I, Lamloum M, Ben Ahmed M, Miled M. Deep vein thrombosis in Behçet's disease. Clin Exp Rheumatol. 2001 Sep-Oct;19(5 Suppl 24):S48-50. PMID: 11760399. Wu X, Li G, Huang X, Wang L, Liu W, Zhao Y, Zheng W. Behçet's disease complicated with thrombosis: a report of 93 Chinese cases. Medicine (Baltimore). 2014 Dec;93(28):e263. doi: 10.1097/MD.0000000000000263. PMID: 25526452; PMCID: PMC4603137. Hatemi G, Christensen R, Bang D, et al2018 update of the EULAR recommendations for the management of Behçet's

syndrome. Annals of the Rheumatic Diseases 2018;77:808-818.



Critical Care - 33 Utilization of Voxelotor to Left-Shift the Hemoglobin-Oxygen Disassociation Curve, Enabling Successful Liberation of a Patient from VV-ECMO: A Medically Challenging Case Report

Karl Kristiansen[1], Ian Welsby[2], John Haney[3], John Reynolds[4]

[1]Duke University, Durham, NC, [2]Duke University School of Medicine, Durham, NC, [3]Duke University Medical Center, Durham, NC, [4]Duke University, Durham, United States of America

Introduction: The current standard of care for treating acute lung injury (ALI)-induced hypoxemia focusses on escalation of supplemental oxygen modality including deploying intubation and mechanical ventilation or ECMO. (1,2) Voxelotor is a small-molecule, allosteric modifier of hemoglobin (Hb) designed to stabilize oxy-Hb (thereby increasing the affinity of Hb for oxygen) and ameliorate sickling in patients with sickle cell disease. An off-target effect may allow a higher SaO2 to be achieved for a given pO2 even at lower oxygen concentrations, oxygen can be delivered effectively throughout the body.(3-6) In preclinical studies a voxelotor analogue, GBT1118, increased hemoglobin affinity (lower p50) shifting the oxygen-hemoglobin curve and improving SaO2 and survival in hypoxic (5%-10% O2) environments.(7) Voxelotor was also shown to improve oxygen saturation at maximal exercise by 3.6 percentage points with a rapid onset of action in patients with idiopathic pulmonary fibrosis related hypoxemia (8.9) Based on these results, we obtained compassionate approval to use voxelotor (under emergency IND and IRB approval) to assist with liberation from VV ECMO for a patient with post-orthotopic liver transplant acute lung injury (ALI)-induced hypoxemia, exacerbating hepato-pulmonary syndrome. By left shifting p50 and increasing SaO2 for disease-limited pO2, we postulate that this approach can de-escalate the invasiveness of treatment for ALI .

Methods: A 61-year-old Caucasian female with a past medical history of non-cirrhotic portal hypertension, hereditary hemorrhagic telangiectasia, hypothyroidism and idiopathic pulmonary fibrosis underwent orthotropic liver transplantation. While thought to be primarily driven by hepatopulmonary syndrome, her chronic hypoxemic respiratory failure required 10L oxygen at rest and up to 20L with ambulation. The intraoperative course was significant for progressive hypoxemia unresponsive to increased FiO2 requiring veno-venous ECMO indicated for a p02 of 50mmHg on 100% oxygen. Over the course of over 3 months, the patient became dialysis dependent and returned to the operating room at least a dozen times due to infectious, bleeding, and abdominal vascular complications remaining VV ECMO dependent, with weaning efforts failing due to low SpO2 confirmed by low pO2 on arterial blood gas analysis. Voxelotor was initiated at 500mg Q8hrs, crushed and administered parenterally and continued until decannulation was successfully performed after 30 days of therapy and stable SpO2 during weaning trials. There were no observed deleterious effects attributed to voxelotor. In an attempt to quantify the effect of voxelotor during this time and noting the fact that a P/F ratio on VV ECMO does not reflect global pulmonary function, we instead used SpO2/FiO2 (S/F) ratio. This measurement has been validated as an alternative to P/F ratio in many studies(10). Unfortunately, in this retrospective review of charted data, we were unable to discern a clear trend in these values during which voxelotor was started. Nonetheless, sweep trials were increasingly successful without desaturation during exercise or agitation; tolerance of physical therapy was a pre-requisite to decannulation.

Conclusion: To our knowledge, this is the first case report describing the use of voxelotor, a novel agent to left-shift p50 in an attempt to liberate a patient suffering from hypoxemic respiratory failure from VV ECMO. The limitations of this case report are the inability to exclude confounding variables (infections, surgical revisions), lack of ability to generalize these findings, and inability to establish a cause-effect relationship. Nonetheless, exploring this physiological manipulation in the setting of hypoxemic respiratory failure is novel and a prospective pilot study will begin in 2022, the challenge will be choosing relevant and familiar physiological parameters that evaluate any benefit of approaches to manipulate the p50 to optimize the pathway from inspired oxygen concentration to tissue pO2.

References: 1. Acute lung injury: epidemiology, pathogenesis.... J Aerosol Med Pulm Drug Deliv. 2010;23(4):243-252. 2. Drug repurposing of histone deacetylase inhibitors.... J Med Chem. 2017;60:1817-1828. 3. A phase 3 randomized trial of voxelotor.... N Engl J Med. 2019;381:509-519. 4. Voxelotor in adolescents and adults with sickle cell disease (HOPE):.... Lancet Haematol. 2021; 8(5):e323-e333. 5. Pharmacokinetics and pharmacodynamics of voxelotor (GBT440)..2019 6. Oxbryta. Global Blood Therapeutics, Inc; January 2021. 7. Putz ND, Shaver CM, Dufu K, et al. GBT1118...J Appl Physiol. 2018 8. Vlahakis NE, Dixon S, Lehrer J, Beck K. Voxelotor, which left shifts the oxy-hemoglobin dissociation curve... Eur Respir J. 2019;54:PA1636. 9. Fell CD, Liu LX, Motika C, et al. The prognostic value of cardiopulmonary.... Am J Respir Crit Care Med. 2009;179(5):402-407. 10. Bilan N, Dastranji A, Ghalehgolab Behbahani A.Comparison of the spo2/fio2 ratio....2015.

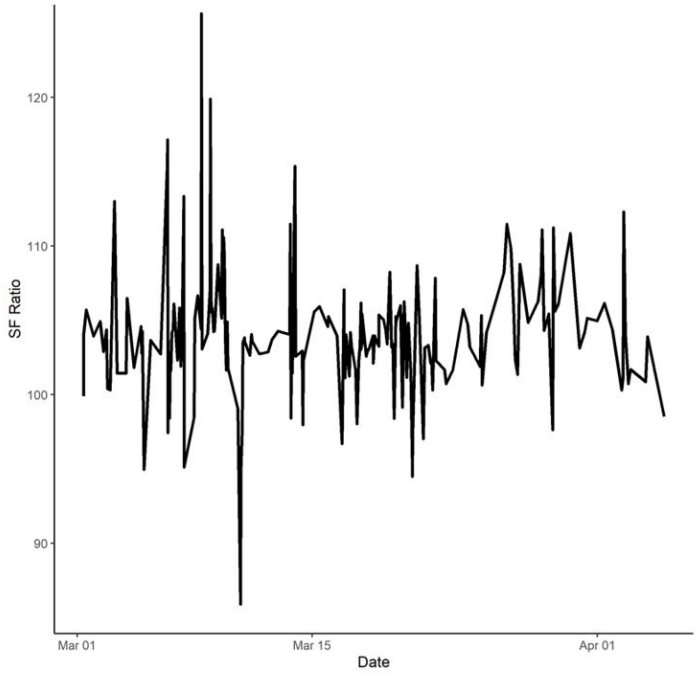
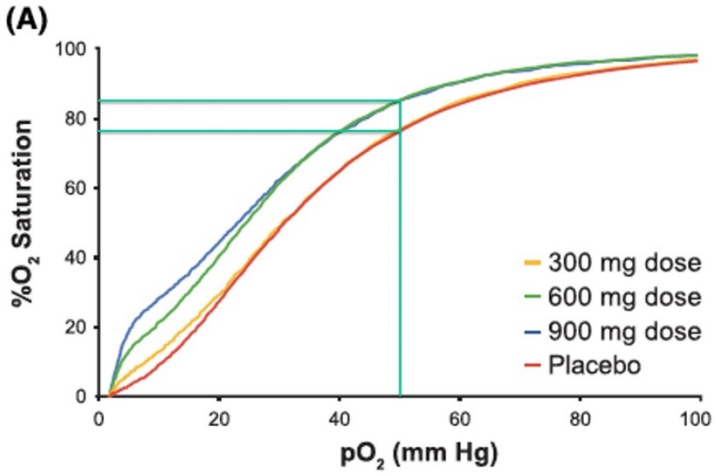


Figure 2: Calculated S/F ratio over time. While there did not appear to be a clear trend in S/F ratio after the administration of voxelotor, the method of oxygen delivery deescalated from ventilator to high flow nasal cannula to 2-4 liters per minute nasal cannula.



Critical Care - 34 Suspected anaphylactic reaction to fresh frozen plasma in an alpha-gal patient

Alexander Neilson[1], Christin Kim[2], Nicholas Malki[3]

[1]VCU Health System, Richmond, VA, [2]VCU Health, Richmond, VA, [3]VCU Health System, Richmond, United States of America

Introduction: We present the case of a 76 year old male with a known history of alpha-gal syndrome who developed an anaphylactic reaction confirmed by tryptase levels during transfusion of fresh frozen plasma (FFP).

Methods: Alpha-gal syndrome is an allergy mediated by an IgE antibody response to mammalian oligosaccharide, galactose-alpha-1,3-galactose (alpha-gal). Alpha-gal is present on meat from non-primate mammals. Lone star ticks, which are endemic to the Southeastern United States, are suspected in the transmission of this antigen.(1) Patients with alpha-gal syndrome must avoid eating mammalian meats and products derived from it. Most notably gelatin based products such as capsules and vaccines. Heparin is another agent to avoid.(2) To date there have been no published cases linking FFP to anaphylaxis in alpha-gal patients. Our patient was a 76 year old male with a history of lung cancer and atrial fibrillation (on apixaban) who was transferred from an outside hospital with dyspnea secondary to a mediastinal mass compressing the trachea. He was admitted to the cardiac surgery ICU in preparation for a rigid bronchoscopy and tracheal stent placement. The decision was made to transfuse one unit of FFP to reverse any residual coagulopathy. During transfusion he became acutely hypoxic, developed atrial fibrillation with rapid ventricular response, and became hypotensive and unresponsive. He required epinephrine boluses and was ultimately intubated at bedside. After intubation his oxygenation status improved but he required multiple vasopressors and inotropes for continued hypotension. He ultimately stabilized and was able to have the aforementioned procedure the following day. He was discharged 2 days after his procedure with no further incident. Tryptase levels were sent, which confirmed anaphylaxis.(Figure 1) Anaphylaxis is a rare complication of blood product transfusion, it is unknown whether this is increased in patients with alpha-gal syndrome. The most common type of transfusion reaction is a febrile non-hemolytic reaction. Other reactions include hemolytic reactions (delayed and immediate), transfusion-related acute lung injury(TRALI), and transfusion associated circulatory overload (TACO). TRALI was ruled out given immediate onset.(3) There was no evidence of TACO on bedside echocardiogram. Hemolysis labs were also sent which were negative. Careful consideration was made to avoid medications that could induce a reaction. While blood products are not currently known to be a risk for alpha-gal patients, there is anecdotal evidence of alpha-gal patients having an anaphylactic response to albumin, which is prepared from human pool plasma.(4)(5) Cases of tick-borne illnesses being transmitted by transfusion poses a theoretical risk for alpha-gal patients.(6)There have been case reports of patients with peanut allergies having anaphylactic reactions to blood products secondary to passive transfer of antigens.(7) It is possible that our patient received product from a donor who ingested meat or was bitten by a lone-star tick, which are endemic to the case location, prior to donation. This could explain the alpha-gal antigen being present in the product.

Conclusion: Currently, literature does not link blood product transfusion to anaphylactic reactions in alpha-gal patients. There is a theoretical risk for alpha-gal antigen transmission with transfusion and our case suggests a link. In conclusion, caution should be taken with any transfusion of blood products in alpha-gal patients

References: 1. The alpha-gal story: lessons learned from connecting the dots. J Allergy Clin Immunol. 2015;135(3):589-597 2. Diagnosis & management of alpha-gal syndrome: lessons from 2,500 patients. Expert Rev Clin Immunol. 2020;16(7):667-677. 3. Transfusion reactions: prevention, diagnosis, and treatment. The Lancet. 2016; 388(61);2825-2836 4. Perioperative challenges in patients with alpha-gal allergy. J Clin Anesth Pain Manag. 2019;3(1):70-78. 5. Impact of Newly Emerging Alpha-Gal Allergies on Cardiac Surgery: A Case Series. Clinics in Surgery. 2017 | Volume 2 | Article 1477 6. Transfusion-transmitted tick-borne infections: a cornucopia of

threats. Transfus Med Rev. 2004 Oct;18(4):293-306
7. Anaphylaxis from passive transfer of peanut allergen in a blood product. N Engl J Med. 2011 May 19; 364(20):1981-2.

Figure 1

Hours after event	Tryptase (ug/L) reference range (2.2-13.2)
2	39.2
9	12.4
26	6.2

Critical Care - 35 Right Coronary Artery Occlusion after Tricuspid Valve Annuloplasty

Shamik Patel[1], Howard Lee[2]

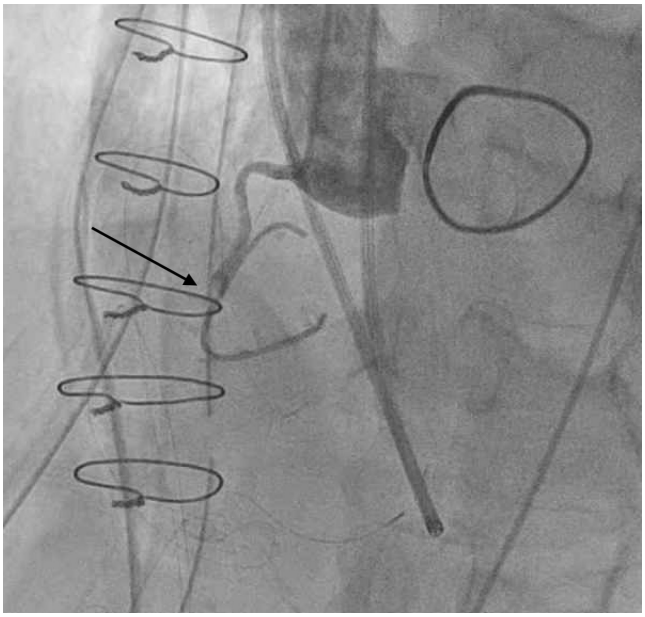
[1]McGaw Medical Center at Northwestern University, Chicago, IL, [2]Northwestern Memorial Hospital, Chicago, IL

Introduction: Tricuspid valve repair is a common surgical procedure for the management of tricuspid valve regurgitation, stenosis, or dilated annulus and patients are routinely admitting to the intensive care unit (ICU) for post-operative monitoring. A rare potential complication is occlusion of the right coronary artery (RCA) which may have a delayed presentation after the patient has left the operating room. Prompt recognition is imperative to survival as a patient can quickly develop hemodynamic collapse and electrical disturbances.

Methods: We report a case of a 66-year-old male who presents for mitral valve repair for severe mitral regurgitation due to mitral valve prolapse. Intraoperative echocardiogram showed mild tricuspid regurgitation with enlarged tricuspid annulus which indicated tricuspid valve annuloplasty be performed as well.[1] Intraoperative course was unremarkable and a valve ring was sutured in for the tricuspid valve repair. Intraoperative echocardiogram was unremarkable with no wall motion abnormalities during closure. The patient was taken to the intensive care unit where initial ECG showed ST elevations in the inferior leads. The patient subsequently went into cardiac arrest due to ventricular fibrillation. Return of spontaneous circulation was obtained after multiple defibrillations and amiodarone bolus. The patient was taken emergently for cardiac catherization which revealed complete occlusion of the RCA due to kinking from a suture placed on the tricuspid ring (Image 1). He then went to the operative room for urgent coronary artery bypass followed by transfer back to the intensive care unit where he had a quick recovery and was discharged home a week later.

Conclusion: There have been only a few case reports of RCA occlusion after tricuspid valve annuloplasty, but it is a potentially fatal complication. Based on a small case series, severely dilated tricuspid annulus, severe pulmonary hypertension, dilated right ventricle, and dilated right atrium seem to be risk factors. The portion of the RCA at highest risk appears to be between the right marginal artery and the crux of the heart where the distance between the annulus and the RCA in approximately 80% patients is less than 5 mm.[2] Presentation may be delayed and therefore any hemodynamic instability, ECG changes in the inferior leads, or arrhythmia after tricuspid valve annuloplasty should raise the concern for RCA occlusion. Immediate recognition and management with percutaneous coronary intervention or coronary artery bypass surgery can lead to recovery of cardiac function.

References: [1] Indications for Surgery for Tricuspid Regurgitation. Interv Cardiol. 2015;10(1):58-60. [2] Direct Injury to Right Coronary Artery in Patients Undergoing Tricuspid Annuloplasty. Ann Thorac Surg. 2014;97(4):1300-5



Critical Care - 36 Critical Care Management of a Type B Aortic Dissection Amidst Workup of a Catecholamine-Secreting Tumor

Megan R Spitz[1], Amar M Bhatt[1]

[1]Wexner Medical Center at The Ohio State University, Columbus, OH

Introduction: We present a case of a 38-year old African American male admitted with chest pain, shortness of breath and hypertensive emergency found to have a Stanford Type B Aortic Dissection (AD). On further workup, elevated urinary and plasma metanephrines raised suspicion for a catecholamine-secreting tumor (CST). The findings complicated urgent surgical planning due to the dangerous side effects if left untreated perioperatively. Although CSTs are exceedingly rare, occurring in 0.2-0.6% of patients with HTN, it is a diagnosis that could lead to catastrophic consequences if left untreated.

Methods: A 38yo male with history of uncontrolled HTN and medication non-compliance presented to the ER in hypertensive emergency, blood pressure was 271/151mmHg, complaining of chest pain and shortness of breath. CTA demonstrated a Stanford Type B AD. The patient was started on esmolol and nicardipine infusions and invasive blood pressure monitoring was established. Routine lab work was notable for creatinine of 1.56, CK of 1169, and lactate of 2.01. Given episodic hemodynamics and history of uncontrolled HTN, urine and plasma catecholamine studies were sent. Due to worsening symptoms, repeat imaging was obtained showing progression of his AD. Operative intervention was complicated by elevated urinary and plasma metanephrines. The clinical dilemma and risk-benefit analysis of urgent surgery in the face of positive serum lab values consistent with CST makes critical care management of this case complex. Following extensive discussion between the multi-disciplinary teams the risk of waiting for confirmatory testing was outweighed by the benefit of proceeding with definitive operative repair of a worsening AD. Surgical intervention is typically delayed 10-14 days to establish complete alpha and subsequent beta-blockade, however this was not possible in our case. Invasive arterial BP monitoring was maintained throughout the patient's ICU stay and central venous access was placed pre-operatively. Strict BP control was achieved pre-operatively with an extensive regimen including nicardipine and nitroglycerine infusions in addition to six oral agents. The patient successfully underwent operative repair of his AD. At the time of submission, dotatate scan results were pending. While it is well recognized that stress can increase catecholamine levels and routine biochemical testing for suspected CSTs in critically ill patients should be avoided; there could be catastrophic consequences should the diagnosis go amiss. Therefore, as demonstrated by our case, when there is a strong clinical suspicion for a CST a diagnostic workup should be pursued in cases of hypertensive emergency resulting in AD.

Conclusion: In conclusion, a CST should always be considered in the differential diagnosis of uncontrolled, treatment-resistant HTN in the setting of a type B AD. Diagnosis of a CST can take time and should not delay definitive surgical repair of a life-threatening and clinically worsening Type B AD.

References: Zuber SM, Kantorovich V, Pacak K. Hypertension in pheochromocytoma: characteristics and treatment. Endocrinol Metab Clin North Am. 2011;40(2):295-vii. Young WF Jr. Pheochromocytoma: 1926-1993. Trends Endocrinol Metab. 1993 May-Jun;4(4):122-7. doi: 10.1016/1043-2760(93)90035-d. PMID: 18407145.

Critical Care - 37 Aortic Rupture and Cardiac Tamponade in a Patient with Subacute Type A Aortic Dissection Presenting with Isolated Cerebral Ischemia

David R Busse[1], Bryan Romito[1]

[1]University of Texas Southwestern, Dallas, TX

Introduction: Acute type A aortic dissection (ATAAD) represents a true surgical emergency with both high pre-hospital and in-hospital mortality rates. While ATAAD frequently presents with sudden, intense chest pain, in rare cases, cerebral ischemia or paraplegia are the initial presenting symptoms. Diagnosis and treatment may be delayed in patients with pain-free dissections, increasing the risk of life-threatening complications like aortic rupture, cardiac tamponade, aortic valve regurgitation, and myocardial ischemia. Because ATAAD is often fatal and rarely evolves naturally into the subacute (15-90 days) or chronic (>90 days) phases, data on management and outcomes are limited. We describe a case of aortic rupture and cardiac tamponade in a patient with subacute type A aortic dissection who presented with cerebral ischemia.

Methods: A 61-year-old man with a past medical history of essential hypertension presented to the emergency department of an outside hospital with one day of sudden-onset left-sided hemiplegia, confusion, and expressive aphasia. Head computed tomography angiography (CTA) revealed an occluded A2 segment of the right anterior cerebral artery (ACA). Because his symptoms developed more than six hours prior to presentation, the patient was deemed a poor candidate for mechanical thrombectomy and intravenous thrombolytics. He was medically managed with antiplatelets. Four days later, the patient was transferred to our hospital for continued management and rehabilitation following his apparent ischemic stroke. During his evaluation, a transthoracic echocardiogram (TTE) revealed an aortic dissection extending from the sinotubular junction through the aortic arch. CT angiography of his aorta confirmed the presence of the dissection, identifying the origin of the dissection flap just distal to the takeoff of the coronary arteries, and terminating slightly beyond the subclavian artery origin. As his dissection had progressed to the subacute phase with hemodynamic stability, and with the hope of obtaining some neurologic recovery, the cardiovascular surgery service opted to pursue a medical management strategy followed by a delayed elective surgical repair. Over the next two weeks, his dissection was managed conservatively with oral beta blockers and antiplatelets. The patient's neurologic deficits improved, and he regained the ability to speak and ambulate. On hospital day 16, the patient became profoundly hypotensive and obtunded. Bedside TTE revealed a large pericardial effusion with evidence of tamponade physiology. His airway was emergently secured, and an ultrasound-guided pericardial drain was placed at the bedside approximately two hours after the onset of hypotension. His blood pressure improved following removal of approximately 900 mL of blood from the pericardial sac. Immediately afterwards, he was taken to the operating room for successful repair of an acute ascending aortic rupture as well as his previously identified aortic dissection. The following day he underwent mediastinal washout and sternal closure. He was ultimately discharged to a long-term acute care facility on hospital day 33 in good condition.

Conclusion: While acute aortic dissections are typically thought of as 'chest pain plus' syndromes, they can be masked by the presence of altered mental status or cerebral malperfusion. Up to 1/3 of ascending dissections present with isolated neurologic symptoms. Ischemic strokes occurring secondary to aortic dissections most commonly involve the anterior circulation. In this case, the ACA occlusion was likely a direct result of the dissection, arising from arterial embolization from the dissected aorta. Non-acute dissections represent small subsets of patients who survive the acute phase with hemodynamic stability. Our patient was initially managed conservatively for several weeks, allowing time for neurologic recovery and rehabilitation. The benefits of delayed surgical management in patients presenting with both cerebral ischemia and stable ascending dissections remain unclear, as well as a point of controversy in the published literature. Given the inconclusive and limited evidence, this case highlights the diagnostic and therapeutic challenges in patients presenting with painless, hemodynamically stable type A dissections with concomitant neurologic findings and describes the

successful management of an aortic rupture following evolution to the subacute phase.

References: 1. Predicting death in patients with acute type a aortic dissection. International Registry of Acute Aortic Dissection (IRAD) Investigators. Circulation. 2002;105(2):200-206. 2. Contemporary results of surgery in acute type A aortic dissection: The International Registry of Acute Aortic Dissection experience. J Thorac Cardiovasc Surg. 2005;129(1):112-122. 3. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. Circulation. 2013;127(20):2031-2037. 4. Acute type A aortic dissection - a review. Scand Cardiovasc J. 2020;54(1):1-13. 5. Management of acute aortic dissection. Lancet. 2015;385(9970):800-811. 6. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology

Critical Care - 38 Blunt Cardiac Left Ventricular Rupture and Left Anterior Descending Artery Injury: A Medically Managed Post-Operative ST Segment Elevation Myocardial Infarction in a Multitrauma Patient

Patrick J Coleman[1], Samuel Galvagno[2]

[1]University of Maryland Medical Center R. Adams Cowley Shock Trauma Center, Annapolis, MD, [2]University of Maryland Baltimore, Baltimore, MD

Introduction: Blunt cardiac injury from trauma requiring operative intervention due to atrial or ventricular injuries portends a poor prognosis with mortality ranging from 40% to 70% (1). If ventricular rupture is suspected, expedient surgical evaluation and management in the operating room with potential for institution of cardiopulmonary bypass is imperative for the patient to survive this type of injury (2). Initial post-operative management in the intensive care unit can be challenging considering the nature of cardiac injury and mixed shock states present in multitrauma patients.

Methods: Case Presentation: A 51-year-old female who was an unrestrained passenger in a high speed motor vehicular accident with two feet intrusion into the passenger side requiring extraction presented to the trauma resuscitation unit with a small pericardial effusion noted on transthoracic echocardiogram (TTE). Additionally, a widened mediastinum was seen on chest x-ray and at this time the patient was hemodynamically stable. However, after initial examination the patient started to complain of chest pain, become dyspneic and hemodynamically unstable with repeat TTE revealing an enlarging effusion. The patient was taken emergently to the operating room for median sternotomy and chest exploration which revealed a large, 1.5cm defect in the left ventricle near the mid-left anterior descending artery with exsanguinating hemorrhage. This defect was primarily repaired and the hemorrhage was controlled with no other significant injuries noted, however the patient experienced an intraoperative arrest with return of spontaneous circulation after brief CPR. The post-operative CT Scan showed non-visualization of the LAD and the patient was also noted to have ST segment elevations on EKG. The patient was taken for emergent cardiac catheterization which revealed total occlusion of the early mid LAD, possibly from the injury and repair, and attempts to revascularize were unsuccessful. Cardiothoracic surgery was consulted, however in the setting of the patient's multiple injuries, and likely unrecoverable myocardium open revascularization was deferred. The patient was then medically managed with vasoactive medications titrated by utilization of a pulmonary artery catheter, serial focused critical care echocardiograms, and careful volume management via continuous renal replacement therapy in the setting of her mixed shock state from multiple traumatic injuries. The patient's initial troponin was 11.9 and it peaked at 253 on hospital day 3. The patient's TTE's showed severely reduced systolic function with estimated LVEF 20-25% with an akinetic anterior wall, septum and apex consistent with LAD culprit distribution. The patient was discharged to home from the hospital after a 48 day admission.

Conclusion: Discussion: While blunt cardiac rupture carries a high mortality, the patients who arrive to the hospital and receive prompt surgical intervention can survive this rare type of traumatic injury with appropriate interventions. As the injury may be operatively fixed, the post-operative intensive care course proves quite challenging to manage in the setting of multiple traumatic injuries due to the nature of a mixed shock state, and injuries that may be prohibitive for utilization of mechanical circulatory support devices typically utilized for left or right ventricular failure.

References: 1) Surg Clin N Am 97 (2017) 1065-1068 2) Ann Thorac Surg 44 (1987) 532-535

Critical Care - 39 Perioperative Management of Abdominal Compartment Syndrome

Tara P Mojtahed[1], Lisa Solomon[2]

[1]Rush Medical Center, chicago, IL, [2]Rush Medical Center, Chicago, IL

Introduction: The purpose of this study was to review the challenging case of diagnosing and treating abdominal compartment syndrome perioperatively. This study sheds light on the risks that predispose one to the condition, symptoms, and management options.

Methods: Abdominal compartment syndrome occurs predominantly in critically ill patients, with early recognition of its risks factors and symptoms critical to reduction of morbidity and mortality. The condition is seen in a wide variety of patient populations from ruptured aortic aneurysms to acute pancreatitis, typically manifesting with patients in shock requiring large quantities of vasopressors, resuscitation fluids, and blood. The combination of increased intra-abdominal pressure and end-organ damage leads to a high mortality, making early detection and close surveillance peri-operatively vital. A 74 year old female status post right robotic-assisted nephrectomy complicated by retroperitoneal hematoma and hemoperitoneum requiring two rounds of renal artery embolization presented with abdominal distension and hemorrhagic shock. CT imaging confirmed retroperitoneal hematoma with concern for active bleeding. She was urgently brought to the IR suite for angiogram and embolization where the renal arterial stump was accessed and successfully embolized. The patient's growing pressor requirements decreased with embolization and resuscitation with numerous blood products and fluids. However, the patient experienced significant amount of bleeding with notable worsening of her abdominal distension, sudden increase in her peak airway pressures and difficulty with ventilation, and development of shock liver and acute renal failure. Upon transfer to the ICU, bladder pressure was measured to be 40, and high peak inspiratory pressures were necessary for adequate ventilation. With the combination of multiple end-organ damage, the diagnosis of abdominal compartment syndrome was confirmed. Therapeutic paracentesis was performed with improvement of intra-abdominal pressures and ventilation requirements. This case demonstrates that in patients at extremely high risk for decompressive laparotomy in the setting of multisystem organ failure, paracentesis is a successful alternative for resuscitation-induced abdominal compartment syndrome and that early detection intra-operatively was vital to the prompt treatment.

Conclusion: In conclusion, early diagnosis of abdominal compartment syndrome reduces morbidity and mortality in critically ill patients. It it more likely to occur in patients complicated with profound shock, requiring large amounts of vasopressors, resuscitation fluids, and blood. Thus, in the perioperative period involving patients at high risk for abdominal compartment syndrome, the clinician should be aware of sudden increases in intra-abdominal pressure, increased peak inspiratory pressure, and decreased urinary output. Management tailors to lowering intra-abdominal pressure and organ support. While the main treatment has typically been decompressive laparotomy, this study shows non-surgical solutions for critically ill patients unable to undergo a procedure.

References: Hunter JD, Damani Z. Intra-abdominal hypertension and the abdominal compartment syndrome. Anaesthesia. 2004. Papavramidis TS, Marinis AD, Pliakos I, Kesisoglou I, Papavramidou N. Abdominal compartment syndrome - Intra-abdominal hypertension: Defining, diagnosing, and managing. J Emerg Trauma Shock. 2011.

Critical Care - 40 Massive upper gastrointestinal hemorrhage caused by an unrecognized left subclavian artery-esophageal fistula secondary to compression by anterior cervical spine fusion hardware

Jennifer Neva[1]

[1]*Massachusetts General Hospital, Boston, MA*

Introduction: Upper gastrointestinal bleeding due to arterio-esophageal fistula is a rare event but associated with significant morbidity and mortality. In the case of subclavian artery-esophageal fistulas, the most common etiology is an aberrant right subclavian artery (ARSA) that originates distal to the left subclavian artery and passes posterior to the esophagus en route to the right axilla, present in approximately 0.5-1.8% of the population. When patients with this abnormal anatomy have prolonged nasogastric tube or endotracheal tube placement, compression of the esophagus between the foreign device and the aberrant artery can lead to ischemia and consequent focal necrosis of the esophagus (1). Less common causes of subclavian artery-esophageal fistulas include previous esophageal or thoracic surgery, neoplasm, vascular conduits, infection, and the ingestion of foreign bodies (2). This medically challenging case reports on the case of a patient with an unrecognized left subclavian artery-esophageal fistula, likely secondary to extrinsic compression by anterior cervical spine fusion hardware, that resulted in fatal exsanguination. While anterior cervical spinal fusion can cause esophageal fistula, even many years after surgery, its contribution to the development of a subclavian artery-esophageal fistula has previously been unreported (3).

Methods: A 66-year-old female with severe cervical dystonia, cervical spondylosis treated previously with an anterior cervical spinal fusion, chronic obstructive pulmonary disease on home oxygen therapy, atrial fibrillation on anticoagulation, and lung cancer status post a right upper lobe lobectomy was initially admitted to an outside hospital with altered mental status and worsening neck pain after a fall. During that hospitalization, she was found to have methicillin-resistant Staphylococcus aureus bacteremia of unclear etiology and was transferred to our institution for magnetic resonance imaging of her spine after approximately ten days. She underwent further evaluation and treatment of her presenting complaints, including a transthoracic echocardiogram, a nondiagnostic spine and brain MRI due to motion artifact, intravenous antibiotic therapy, and pain control. Almost one month after transfer to our institution, she developed an episode of acute hematemesis overnight associated with hypotension, which was initially responsive to fluid resuscitation and blood product administration. However, ongoing resuscitation became less effective and led to worsening respiratory status, prompting the decision to transfer her to the intensive care unit (ICU) for elective intubation and evaluation by gastroenterology with esophagogastroduodenoscopy at bedside. Prior to transfer, she underwent a computed tomography angiogram (CTA) of the chest. Shortly after transfer to the ICU, she had another episode of massive hematemesis requiring intubation for airway protection. Immediately after intubation, she became pulseless, requiring cardiopulmonary resuscitation and initiation of the massive transfusion protocol. Pulses were regained, although lost again multiple times throughout her volume resuscitation. Evaluation by gastroenterology revealed red arterial blood in the esophagus with clotted blood in the gastric fundus and the entire examined duodenum. Formal interpretation of the CTA then revealed marked inflammatory changes of the esophagus where it passed between the arch vessels and spinal fixation hardware with a small outpouching of the left subclavian artery, concerning for subclavian artery-esophageal fistula. Given the patient's hemodynamic instability, no intervention was offered, and she was transitioned to comfort measures-only, dying shortly thereafter.

Conclusion: This case report is unique in that it presents the case of a patient with massive upper gastrointestinal hemorrhage, leading to fatal exsanguination, secondary to a left subclavian artery-esophageal fistula, a rare pathology documented in the literature (4). This patient's fistula was thought to be secondary to extrinsic compression of the esophagus between her left subclavian artery and anterior cervical spine fusion hardware as evidenced by her CTA. This case emphasizes the importance of recognizing

previous anterior cervical spinal fusion as a risk factor for the development of a subclavian artery-esophageal fistula, a risk factor that was previously unacknowledged in the literature.

References: 1. Upper gastrointestinal bleeding secondary to an aberrant right subclavian artery-esophageal fistula: A case report and review of the literature. Canadian Journal of Gastroenterology, 21(6): 389-392. 2007. 2. Anomalous right subclavian artery-esophageal fistulae. Case Reports in Vascular Medicine, 7541904. 2018. 3. Esophageal perforation after anterior cervical spine surgery: a systematic review of the literature. Journal of Neurosurgery: Spine SPI, 25(3), 285-291. 2016. 4. Left subclavian artery-esophageal fistula induced by a paper star: a case report. Biomedicine (Taipei), 6(3):18. 2016.

Critical Care - 41 Transient Diabetes Insipidus After Vasopressin Withdrawal - A Persistent Phenomenon

Jeffrey Carness[1], Rania El Fekih[2], Suzanne Klainer[3], Saif Muhsin[2], Ernest Mandel[2]

[1]Naval Medical Center, San Diego, CA, [2]Brigham and Women's Hospital, Boston, MA, [3]Brigham Health - Harvard Medical School, Boston, MA

Introduction: Vasopressin is often used for the treatment of shock. Case reports describing diabetes insipidus after discontinuation of vasopressin infusion are increasing in number. (1-13) Although, the mechanism is not completely clear, it is reasonably associated with central and peripheral modulation of the hypothalamic / pituitary / renal axis, with effects manifested via alteration in the activity of the vasopressin V2 receptor. This may result in a mixed type of central and nephrogenic diabetes insipidus whereby the administration of exogenous vasopressin triggers renal receptor downregulation and the withdrawal of exogenous vasopressin results in the absence of intrinsic antidiuretic hormone. (14) This pathology is believed to occur more frequently than is described in the literature due to underreporting. The exact incidence of diabetes insipidus after discontinuation of vasopressin is largely unknown though recent reporting has made attempts to better quantify this epidemiologic variable. (4) In the absence of a depth of reporting, and without further clinical guidance in the literature, clinicians are left with little resource to guide patient care delivery.

Methods: Here we present a case series to add to the current body of literature regarding this phenomenon. We have included a table with patient characteristics. Notably, the baseline sodium levels were all within the normal range upon admission. Subsequent deviations are noted in Table 1. Urine osmolality was measured upon recognition of initial polyuria after vasopressin discontinuation. Desmopressin response was arbitrarily defined as a reduction in hourly urine output by a value greater than 50%. Reportedly, post desmopressin urine osmolality testing will likely result in a significant increase to >750 mOsm/kg. (15) Unfortunately, these tests were not readily available during our chart review. Copeptin, a surrogate marker for AVP release that is released into circulation at an equimolar amount to AVP, may serve as a useful biomarker in distinguishing central versus nephrogenic diabetes insipidus. (16) Serum copeptin levels were measured, from the time of vasopressin infusion discontinuation (12.4 --> 23.0 --> 45.6 pmol/L), in only one patient in our cohort. They rose after vasopressin discontinuation suggesting a resumption of intrinsic ADH secretion.

Conclusion: Additional studies should be performed, both retrospective and prospective, to better elucidate the epidemiology, risk factors, specific pathophysiology, and treatment approach for patients who experience this adverse effect.

References: 1. AACE Clinical Case Reports (2021), doi: https://doi.org/10.1016/j.aace.2021.06.004. 2. Journal of the Endocrine Society, Volume 5, Issue Supplement_1, April-May 2021, Pages A572-A573, https://doi.org/10.1210/jendso/bvab048.1167 3. Journal of the Endocrine Society. Volume 5, Issue Supplement_1, April-May 2021, Page A571 https://doi.org/10.1210/jendso/bvab048.1164 4. Crit Care Med. 2019 Dec;47(12):e1008-e1013. 5. Case Rep Endocrinol. 2019 Dec 11;2019:4189525 6. J Clin Pharm Ther. 2018 Apr;43(2):287-290 7. World Neurosurg. 2017 Jan;97:479-488 8. Open J Clin Med Case Rep 2015;1:1042 9. J Cardiothorac Vasc Anesth. 2014 Jun;28(3):e24-5 10. J Thorac Cardiovasc Surg. 2009 Aug;138(2):491-2. 11. Chest 2005; 128(4_MeetingAbstracts):454S-455S 12. Pharmacotherapy. 2004 Apr;24(4):541-5 13. Ann Am Thorac Soc. 2016 Oct;13(10):1760-1767 14. Am J Physiol. 1959;196:451-456. 15. Clin Endocrinol (Oxf). 2019 Jan;90(1):23-30. 16. J Endocrinol Invest. 2020 Jan;43(1):21-30.

Table 1: Diabetes Insipidus after Vasopressin Withdrawal - Patient Characteristics

Age / Sex	Diagnosis	Sodium - Minimum During Infusion	Sodium - Maximum During Infusion	Infusion Duration - Days	Hourly Polyuria - Maximum after Discontinuation	Urine Osmolality	DDAVP	Response
64 / F	Idiopathic Pulmonary Fibrosis	136	160	18	800	179	2 mcg	+
22 / F	Idiopathic Pulmonary Fibrosis	126	145	6	1235	79	2mcg	+
60 / F	Esophageal Perforation	120	139	5	725	*SG 1.007	---	---
40 / F	Inhalation Burn	125	143	2	1050	142	---	---
58 / F	Necrotizing Fasciitis	133	146	9	700	22	1 mcg	+
51 / F	Myocardial Infarction	132	147	4	1000	153	4 mcg	+

*SG - Specific gravity reported as urine osmolality was not recorded for this patient

Critical Care - 42 Gradual vision Loss in a Thirsty Adult

Jonathan Bond[1], Jasdeep Dhaliwal[1]

[1]University of Kentucky, Lexington, KY

Introduction: 34 yo female presented with one week history of progressively worse vision loss over the preceding week along with persistent nausea and vomiting associated with 30 lb weight loss over the last 3 months.

Methods: 34 yo female with a history of anxiety, depression and remote traumatic brain injury presents with a 3 month history of nausea. vomiting and an unintentional 30 lb weight loss. Over the preceding week she had progressive vision loss to the point on physical exam she was essentially blind. CT of her head revealed left frontal encephalomalacia, while CT of her chest/abdomen/pelvis revealed right hilar and periarotic adenopathy. An endobronchial biopsy of the hilar mass was performed and MRI of the head revealed prominent pituitary with thickened pituitary stock. Soon after presentation she developed polyuria and hypernatremia concerning for diabetes insipidus and treated with DDAVP and crystalloid replacement. Other notable labs included a TSH of <0.01 uIU/mL. Ophthalmology consult and exam revealed a normal exam without disc edema but with significant vision loss concerning for optic chiasm compression. Neurosurgery and endocrinology were also consulted with the concern for pituitary apoplexy. With no obvious hemorrhage or mass the differential diagnosis of her hypophysitis included sarcoidosis, histiocytosis x and autoimmune hypophysitis. Further history revealed she was consuming up to a case of water a day for the last several months along with up to 3 bottles of water in the middle of the night. Further workup revealed LH<0.3. Biopsy from the perihilar adenopathy revealed reactive lymphadenopathy with no granuolmas. Her vision loss gradually resolved and she had no visual field deficits on discharge. She was started on prednisone and scheduled for follow up MRI and visits with endocrinology, neurology and neurosurgery. While no final diagnosis has been given, likely she presented with autoimmune hypophsitis

Conclusion: New onset vision changes can be difficult. Quick decision making to rule out causes of permanent vision loss is necessary. Pituitary apoplexy can present in interesting ways

Critical Care - 43 The use of veno-venous extracorporeal membrane oxygenation for pregnant patients with COVID related acute respiratory distress syndrome: a case series

Clare O Hasken[1], Samuel Jensen[2], Benjamin Abrams[1], Cristina Wood[1], Maung Hlaing[1]

[1]University of Colorado School of Medicine, Aurora, CO, [2]University of Colorado Anesthesiology Department, Aurora, CO

Introduction: The use of extracorporeal membrane oxygenation (ECMO) for critically ill patients with acute respiratory distress syndrome (ARDS) is often indicated when medical management fails [1-2]. However, little data exists regarding the use of ECMO in pregnant patients with COVID associated ARDS. The National COVID-19 Clinical Evidence Taskforce of Australia provides recommendations for evidence-based care of pregnant patients with severe COVID infections [3]. Data supports the use of certain medical therapies in this population including the administration of steroids [3]. However, the risks and benefits of other medical therapies, i.e. remdesivir, azithromycin, and convalescent plasma, in pregnant patients remain poorly understood as they are often excluded from clinical trials [3]. Additionally, the impact of interventions such as prone positioning and ECMO therapy in pregnancy is unclear [3-8].

Methods: At a large tertiary care academic center in the US, two pregnant patients were identified from March 1, 2020 through August 30, 2021 in which veno-venous (VV) ECMO was initiated due to severe ARDS from COVID. The first patient was a 29F, 27 weeks 3 days pregnant, admitted to an intensive care unit in April 2020 with diabetic ketoacidosis and subsequently tested positive for COVID. She was intubated on hospital day 2 for acute hypoxic respiratory failure but improved and was extubated on day 3. The patient and fetus remained stable until day 9 when the patient required reintubation for worsening hypoxia. Due to fetal distress, a Cesarean delivery was emergently performed. The patient's respiratory status did not improve despite paralysis, proning, and inhaled epoprostenol therapy. She was cannulated for VV ECMO on day 10. The patient gradually improved and on day 19 she was decannulated. She was extubated on day 23 and discharged to acute rehab on day 38 on supplemental oxygen. ECMO complications included multiple right upper extremity deep vein thromboses (DVTs) as well as a segmental pulmonary embolus. The patient's overall treatment for COVID included azithromycin, ceftriaxone, hydroxychloroquine, convalescent plasma, remdesivir, and sarilumab. The second patient was a 32F previously healthy but unvaccinated, 28 weeks 5 days pregnant, who tested positive for COVID as an outpatient in August 2021. She presented to the hospital several days later and was subsequently intubated on hospital day 1 due to acute hypoxic respiratory failure. She continued to decompensate despite paralysis, proning, and inhaled epoprostenol therapy. On day 5 she underwent emergent VV ECMO cannulation followed immediately by uncomplicated Cesarean delivery. She was extubated on day 7, ambulated on day 8, and was decannulated on day 9. The patient was discharged home on day 12 with supplemental oxygen. The patient suffered no major complications related to ECMO. She was treated with steroids, azithromycin, ceftriaxone, and remdesivir during her hospital course.

Conclusion: Despite little evidence regarding the use of VV ECMO therapy for pregnant patients with severe ARDS secondary to COVID, multiple case reports have suggested VV ECMO can be a relatively safe and effective treatment option during pregnancy if warranted [4,6-8]. One patient in our case series did suffer from multiple DVTs. However, both the mothers and newborns survived with minimal morbidity. Multiple case reports in the literature report no maternal deaths from VV ECMO use [4,6-8]. Further evaluation is needed to fully determine the risks and benefits of VV ECMO therapy for pregnant patients with severe ARDS secondary to COVID, including the risks and benefits of proning during pregnancy and initiation of ECMO prior to versus after delivery.

References: 1. Extracorporeal Membrane Oxygenation for Severe ARDS. N Engl J Med 2018; 378:1965-1975. 2. CESAR: conventional ventilator support vs ECMO for severe adult respiratory failure. BMC Health Serv Res 2006; 6:163. 3. Clinical care of pregnant and postpartum women with COVID-19:

Living recommendations from the National COVID-19 Clinical Evidence Taskforce. Aust N Z J Obstet Gynaecol 2020; 60: 840-851. 4. Successful treatment of pregnant and postpartum women with severe COVID-19 associated ARDS with ECMO. ASAIO J 2021; 67: 132-136. 5. ECMO during pregnancy and postpartum. Sem in Perinat 2018; 42: 21-25. 6. Analysis of maternal coronavirus infections and neonates born to mothers with 2019-nCoV; a systematic review. Arch Acad Emerg Med 2020; 8: e49. 7. Clinical Findings and Disease Severity in Hospitalized Pregnant Women with COVID-19. Obstet Gynecol 2020; 136: 252-258. 8. Coronavirus in pregnancy and delivery: a rapid review. Ultrasound Obstet Gynecol 2020; 55:586-592.

Critical Care - 44 Acute EMCO circuit thrombosis due to IVC thrombus in a lung transplant candidate with COVID-19 pneumonia: diagnosis and management considerations

Kevin K Kim[1], Cynthia Williams[2], Sarah Alber[1]

[1]University of Colorado, Aurora, CO, [2]University of Colorado, Denver, CO

Introduction: Patients with the novel coronavirus (COVID-19) can develop severe acute respiratory distress syndrome (ARDS), necessitating veno-venous extracorporeal membrane oxygenation (VV-ECMO). Not only are patients with COVID-19 hypercoagulable[1, 2], but ECMO independently promotes a prothrombotic state[3]. ECMO circuit thrombosis is a potentially lethal complication, and many management challenges arise in this scenario. This author will be illustrating the following case of an acute ECMO circuit thrombosis caused by IVC thrombosis in a lung transplant candidate who was on VV-ECMO.

Methods: A 33-year-old male with no past medical history presented with severe COVID-19 pneumonia complicated by ARDS. He was intubated on hospital day 1 and quickly required proning, paralysis, and inhaled pulmonary vasodilators to maintain adequate oxygenation. He was cannulated for VV ECMO using a Crescent cannula in the right internal jugular (IJ) vein on hospital day 20 as a bridge to lung transplant. Lab values of note at the time of cannulation were INR 2.6, ACT 488, and platelet count of 810. Thrombus was soon noted in the outflow cannula and oxygenator of the ECMO circuit despite adequate anticoagulation therapy, and on ECMO day 1 (hospital day 21), the circuit and oxygenator were exchanged. On hospital day 25, the patient underwent tracheostomy placement, which was complicated by significant bleeding at the tracheostomy site requiring transfusion and temporary suspension of anticoagulation. The patient's respiratory function steadily improved, however on hospital day 26 ECMO flows dropped acutely and the air-in-circuit alarm was triggered due to air in the pump head. The air was quickly aspirated from the circuit, along with some thrombus, and after careful inspection, no source of air entrainment was identified. A bedside transesophageal echocardiogram (TEE) was performed due to ongoing severe fluctuations in ECMO flows without an obvious cause. The TEE demonstrated a large thrombus with very limited ECMO flow by color flow doppler within the distal portion of the cannula, a smaller mobile thrombus in the superior aspect of the cannula, as well as thrombus external to the cannula in the inferior vena cava.

Conclusion: Our case highlights some significant challenges in the management of patients on ECMO who develop circuit thromboses that threaten its continued function and patency. In the above case, due to the high burden of thrombosis both within and external to the inflow cannula and its ongoing impact on ECMO flows, our team considered several management strategies. First, we discussed rewiring and replacing the Crescent cannula, however, we were concerned that in doing so we could cause a large pulmonary embolism and worsen his overall cardiopulmonary function. We also questioned the patient's ability to tolerate the abrupt removal of VV-ECMO in order to replace the cannula and circuit. We also considered a reconfiguration of the ECMO circuit to fem-fem prior to removal of the IJ cannula to minimize interruptions in ECMO support, but as the patient was undergoing a lung transplant workup at the time we were concerned about the impact this cannulation strategy would have on his mobility and subsequent candidacy. A final consideration was whether an endovascular Angiovac procedure could feasibly remove the thrombus from the IVC at the time of ECMO cannula exchange, or whether there would be a role for thrombolytic therapy in order to maintain his original cannulas and minimize ECMO interruptions. Fortunately, our patient was weaning from ECMO support when the inflow cannula thrombosis was discovered, so the cannulas and the majority of thrombus were removed uneventfully and he was successfully transitioned off ECMO support and maintained on therapeutic anticoagulation. One point our team could have considered earlier was whether the patient's thrombocytosis and early circuit thrombus warranted treatment with an antiplatelet agent after the initial ECMO circuit exchange. While his anticoagulation was paused for a short while due to tracheostomy bleeding, the thrombus noted in the cannula was well organized and we suspect it was

likely present before the brief interruption in anticoagulation.

References: 1. Thachil, J., et al., ISTH interim guidance on recognition and management of coagulopathy in COVID-19. J Thromb Haemost, 2020. 18(5): p. 1023-1026. 2. Kowalewski, M., et al., COVID-19 and ECMO: the interplay between coagulation and inflammation-a narrative review. Crit Care, 2020. 24(1): p. 205. 3. Sperling, C., et al., Blood coagulation on biomaterials requires the combination of distinct activation processes. Biomaterials, 2009. 30(27): p. 4447-56.

Critical Care - 45 Atypical Neuroleptic Malignant Syndrome in COVID-19 Intensive Care Unit

Joshua Durbach[1], Gerald Rosen[2], Carolina De La Questa[3], Seth Gottlieb[3]

[1]Mount Sinai Medical Center, Miami Beach, FL, [2]Mt. Sinai Medical Center, Miami Beach, FL, Miami Beach, FL, [3]Mount Sinai Medical Center, Miami Beach, United States of America

Introduction: Neuroleptic malignant syndrome (NMS) is particularly rare and disastrous drug-induced febrile life-threatening neurologic emergency involving multiple systems, with incident rates ranging 0.02 - 3 percent amid patients taking antipsychotic medications1. One usually suspects NMS when a patient presents with a tetrad of clinical findings: 1) fever, 2) muscle rigidity, 3) encephalopathy and 4) autonomic instability (tachycardia, labile or high blood pressures)2 often seen with high-potency (first-generation) antipsychotic medications and may occur after a single dose. The presentation of NMS can vary without a specific diagnostic test, making the diagnosis extremely challenging. There have been conflicting reports in the literature of an Atypical Neuroleptic Malignant Syndrome (ANMS), associated with lower-potency agents (second generation) antipsychotic medications. We report a case of a patient admitted for Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV2) pneumonia induced acute respiratory distress syndrome (ARDS) who developed probable ANMS.

Methods: Case: 68-year-old male admitted to ICU due to SARS-CoV2 pneumonia, requiring endotracheal intubation and mechanical ventilation. Several days following the intubation, weaning trials were started, however, sedation was restarted for agitation & the patient was started on Quetiapine (ICU delirium). Within twenty four hours of starting Quetiapine, the patient developed tachycardia, ventilator desynchrony with worsening hypoxia, increased peaked pressures and low grade fever. A concern for possible pneumothorax was high on the differential along with the possibility of pulmonary embolism (PE). A Computer Topography pulmonary angiography (CTPA) was ordered to rule out the aforementioned. The CTPA scan diagnosed of a large left pneumothorax but no PE. Under sterile technique a chest tube was inserted. Following this, the patient developed a persistent fever >38.2¬∞C. He was pan cultured and started on broad spectrum antibiotics. The patients fever continued to increase peaking at 42¬∞C with a rectal temperature probe. He was treated with acetaminophen, cooling blankets & cool saline gastric lavage but these failed to control his pyrexia. Due to the patient recently starting Quetiapine prior to these new features, a concern for neuromuscular malignant syndrome (NMS) was raised except the patient lacked the muscular rigidity. The patients core body temperature remained elevated between 40-42¬∞C. A decision was made to treat for NMS. Quetiapine was discontinued and two doses of Dantrolene were administered. Within a couple of hours, the patient's vital signs normalized with resolution of his pyrexia, hemodynamic instability and ventilator desynchrony. Discussion: Causes of NMS are currently unknown but given the correlation with antipsychotic medications it is likely to be associated with dopamine receptor blockade, along with nigrostriatal dopamine pathway activation induced rigidity and tremors 2,3,4. There is likely a genetic genetic predisposition, given reports of families experiencing NMS5; via higher Dopamine-2 receptor gene alle6. There are no specific laboratory tests to confirm diagnosis, rather to rule out other etiologies. NMS is typically characterized by muscle rigidity, pyrexia, autonomic dysfunction and encephalopathy, however, none of these is pathognomic and not all four of the aforementioned are required for diagnosis 7. The lack of rigidity, in our case, is thought to be due the midazolam infusion as benzodiazepines have been used as an adjunctive therapy in NMS 1,7. We were hesitant to accept this diagnosis, due to the absence of rigidity, however, the patient responded (resolution of pyrexia) only once Dantrolene was administered; supporting the theory of atypical NMS. Unfortunately the patient succumbed two weeks later.

Conclusion: Neuroleptic malignant syndrome (NMS) is a rare and a disastrous antipsychotic drug induced febrile state that requires early recognition and diagnosis. NMS is diagnosed clinically although none of the tetrad are required for diagnosis, as mentioned in our case. Moreover, our incomplete understanding of COVID-19 pathogenesis adds another angle of complexity to patient care. With this case, we promote

vigilance and to maintain broad differentials when managing critically ill patients, given the disease process, multiple medications administered and their potential interactions.

References: 1. Neuroleptic malignant syndrome. Recognition, prevention and management. Drug Saf. 1998;19(1):73. 2. Neuroleptic malignant syndrome. Am J Psychiatry. 1985;142(10):1137 3. Neuroleptic malignant syndrome: a pathogenetic role for dopamine receptor blockade? Neurology. 1981;31(2):132 4. Neuroleptic malignant syndrome. Br J Anaesth. 2000;85(1):129. 5. The predisposition to neuroleptic malignant syndrome genetically transmitted? Br J Psychiatry. 1991;158:850. 6. Relationship between functional dopamine D2 and D3 receptors gene polymorphisms and neuroleptic malignant syndrome. Am J Med Genet B Neuropsychiatr Genet. 2003;117B(1):57 7. Atypical neuroleptic malignant syndrome: diagnosis controversies and considerations. Pharmacotherapy 2008;28(4):530-535

Critical Care - 46 Chronic Lithium Toxicity Presenting as Chronic Diarrhea

Eric Y Chen[1], Tichaendepi Mundangepfupfu[2], Emily Gore[1]

[1]University of Rochester Medical Center, Rochester, NY, [2]University of Rochester, Rochester, NY

Introduction: Lithium, a mood stabilizer commonly prescribed for bipolar disorder, has a narrow therapeutic index. The risk of lithium toxicity is further increased in settings of volume depletion or decreased renal excretion, as can be seen in patients concurrently taking ACE inhibitors.

Methods: A 28-year-old male with bipolar disorder on lithium, type 2 diabetes mellitus, and hypertension started on lisinopril six months prior to presentation, presented to the Emergency Department with a two-month history of worsening nausea, malaise, and diarrhea. History and physical exam were significant for a normal electrocardiogram (ECG) and a lack of myoclonus, seizures, or other neurological symptoms. Labs were notable for a lithium level of 2.48 mmol/L, sodium of 120 mmol/L, serum bicarbonate of 13 mmol/L, and creatinine of 13.97 mg/dL. Renal ultrasound demonstrated bilaterally enlarged kidneys (see Figure 1). He was admitted to the ICU for emergent dialysis to treat his lithium toxicity, metabolic acidosis, and hyponatremia. Continuous renal replacement therapy (CRRT) was initiated, and once a sodium level of 126 mmol/L was achieved on hospital day 2, he was switched to intermittent hemodialysis (iHD). He was transferred to the floor on hospital day 3 with a lithium level of 1.28 mmol/L and sodium level of 134 mmol/L. He was discharged on hospital day 7. By discharge, his lithium had normalized to 0.62 mmol/L, and his symptoms of nausea, malaise, and diarrhea had resolved.

Conclusion: Patients who concurrently take lithium and medications that affect renal elimination of lithium (such as ACE inhibitors) are at risk for developing lithium toxicity, which may present with chronic, insidious symptoms such as diarrhea and lethargy, rather than the classic neurological symptoms seen with acute lithium toxicity. Symptoms such as diarrhea exacerbate volume depletion and further decreases renal excretion, causing a cycle of worsening lithium toxicity. Patients with chronic lithium toxicity may be at risk of developing nephromegaly and renal cysts, although these findings are currently limited to case reports. , Given our patient also had hyponatremia and did not have cardiac and neurological symptoms of lithium toxicity, our primary goal in the first 24 hours of his admission was to prevent to prevent overly rapid correction of his serum sodium level to prevent the development of osmotic demyelination syndrome. His serum sodium level was allowed to increase to 126 mmol/L, after which iHD was initiated. His serum sodium level continued to safely correct to normal and his lithium level decreased to within its therapeutic index. Upon transfer to the hospital medicine service, we advised providers to avoid initiating thiazides or ACE inhibitors for the management of hypertension, as these agents may decrease renal elimination of lithium.

References: Alexander MP, Farag YM, Mittal BV, Rennke HG, Singh AK. Lithium toxicity: a double-edged sword. Kidney Int. 2008;73(2):233-237. doi:10.1038/sj.ki.5002578 Slaughter A, Pandey T, Jambhekar K. MRI findings in chronic lithium nephropathy: a case report. J Radiol Case Rep. 2010;4(8):15-21. doi:10.3941/jrcr.v4i8.470

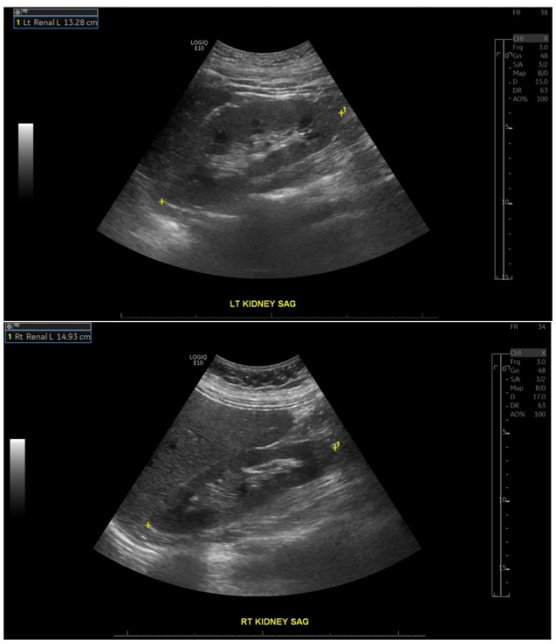


Figure 1. Bilateral renal enlargement on renal ultrasound with the left kidney measuring 13.28cm and the right kidney measuring 14.93cm.

Critical Care - 47 Unexpected Methemoglobinemia in a Septic Patient with Lidocaine Patches

Ashley Miller[1], Allan Migirov[1], Lynden Baesch[1], Trisha McGinnis[1], Piyush Mathur[1]

[1]Cleveland Clinic, Cleveland, OH

Introduction: Methemoglobinemia is an uncommon condition in the ICU caused by an increase in blood levels of oxyhemoglobin containing oxidized ferric iron (Fe^{3+}). [1] Acquired forms are typically related to drugs that increase oxidative stress, including cocaine-derived local anesthetics such as benzocaine and lidocaine, dapsone, sulfonamides, and nitric oxide. [2] The signs and symptoms of methemoglobinemia can include cyanosis, nausea, dyspnea, tachycardia, altered mental status, acidosis, dark-colored blood, a discrepancy between SpO2 and SaO2 refractory to oxygen therapy, seizures, coma, and death. [1,2] We present the case of a decompensating patient in the surgical intensive care unit with elevated blood methemoglobin.

Methods: A 63-year-old male patient presented to the hospital for elective ileostomy reversal after subtotal colectomy for volvulus of the transverse colon. His past medical history included COPD, HTN, CVA, carotid stenosis, CAD, MI, PAD, and atrial fibrillation. On POD # 16 he was re-admitted to the SICU for respiratory distress, tachycardia, fever, and hypotension, presumed to be septic shock secondary to pneumonia. His SpO2 was persistently in the low 90s despite escalation of supplemental oxygen therapy including tracheal intubation and mechanical ventilation. Arterial blood gas analysis demonstrated several surprising findings including dark-colored blood, higher-than-expected arterial pO2, and elevated methemoglobin (peak of 10.6%). The unexpectedly elevated methemoglobin resulted in an investigation into the patient's medications to elicit the cause. The patient had 4% lidocaine patches covering the previous ileostomy site which remained an open wound measuring about 1.5cm in diameter. The patient was treated with three doses of methylene blue and one dose of intravenous vitamin C, throughout a period of 18 hours, resulting in a marked decrease in methemoglobin to 1.7% and improvement of SpO2. The patient eventually improved, leading to extubation and transfer out of the ICU.

Conclusion: The presentation of methemoglobinemia can be widely varied. In our report, the presentation was insidious and blood levels of methemoglobin were unlikely to be directly causing the patient's deterioration, especially given his other chronic and acute conditions. However, failure to recognize the methemoglobinemia could have led to worsening of blood methemoglobin to toxic levels. Additionally, due to the falsely low SpO2, over-estimation of his true disease severity could have resulted in non-optimal management. Very few case reports that describe methemoglobinemia from transdermal lidocaine are not also confounded by other medications, such as intravenous or tracheal administration of a local anesthetic. [3,4] The novelty of our case presentation involves the development of clinically significant methemoglobinemia from transdermal lidocaine only, with two additional details: two patches were used simultaneously and one of the patches was placed over an open wound.

References: 1. Methemoglobinemia in the Operating Room and Intensive Care Unit: Early Recognition, Pathophysiology, and Management. Adv Ther. 2020 May;37(5):1714-1723. 2. Methemoglobinemia: pathogenesis, diagnosis, and management. South Med J. 2011 Nov;104(11):757-61. 3. Rare cause of delirium and hypoxemia after coronary bypass surgery: transdermal lidocaine patch-associated methemoglobinemia. Int J Legal Med. 2018 May;132(3):767-769. 4. Methemoglobinemia in the setting of chronic transdermal lidocaine patch use. Pain Med. 2012 Jul;13(7):976-7.

Critical Care - 48 Venoarterial Extracorporeal Membrane Oxygen as a Bridge to Recovery Following Biventricular Failure in COVID-19 Related Myocarditis: A Case Report

Rebecca Piland[1], Hannah Viroslav[1]

[1]University of Texas Southwestern, Dallas, TX

Introduction: COVID-19 has proven to have catastrophic effects on every organ in the body. There are a variety of well documented cardiovascular complications, which include myocardial infarction, myocarditis, pericarditis, and acute decompensated heart failure. These complications can result in cardiogenic shock resistant to typical medical resuscitative therapies. Venoarterial extracorporeal membrane oxygenation (VA ECMO) is an invasive and previously scarcely available resource that has expanded in its utility over the past 18 months through its implementation as salvage therapy for acutely decompensated COVID-19 associated cardiovascular disease. VA ECMO requires a specialized team to initiate and manage with the goal of bridging the patient to full recovery, ventricular-assisted device, or heart transplant. Here we present a successful study of a previously healthy, 27-year-old female who developed COVID-19 viral myocarditis, resulting in fulminant heart failure and cardiogenic shock requiring rescue, centrally cannulated, VA-ECMO approximately 60 days after vaccination. This case report aims to show an example of the severe cardiac complications related to COVID-19 infection that can result even after vaccination while demonstrating the lifesaving utility of VA-ECMO in this patient population.

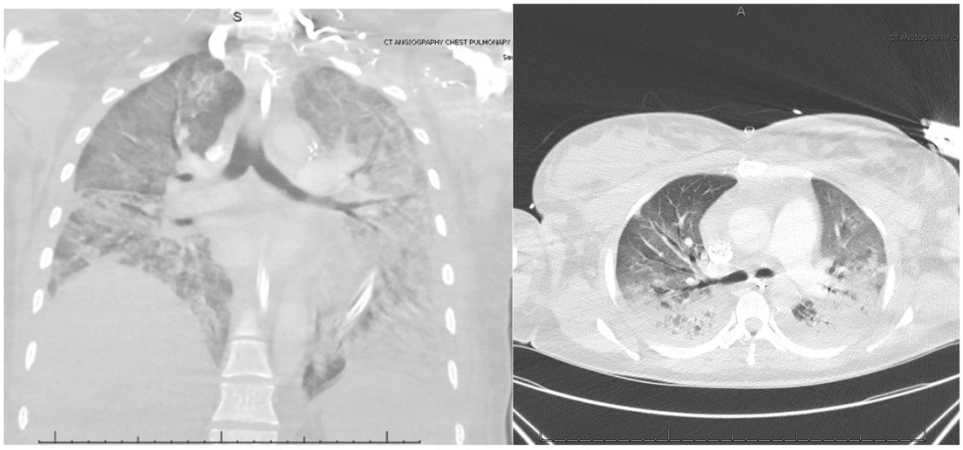
Methods: A 27-year-old female, vaccinated against COVID-19 with the J&J vaccine in April, tested positive for COVID-19 in June 2021. Three days later, she presented to the emergency department with chest pain, dyspnea, fever, and mild single lead ST elevations. Her troponin levels progressively increased, and a left heart catheterization showed normal coronary arteries, but severely depressed ejection fraction (EF) at 20%. With a presumed myocarditis diagnosis, she was admitted and started on appropriate treatment. She then acutely decompensated with diffuse ST elevations and rising troponins, developing cardiogenic shock requiring vasopressor support. She was transferred to a second facility and intubated for planned ventricular assist placement. Due to small, incompatible, femoral vessels, the procedure was aborted and she was transferred to our facility for emergent VA ECMO cannulation. On arrival, her lactate was 17.1 mmol/L and her arterial blood gas showed a pH of 7.0, pCO2 of 40 mmHg, bicarbonate of 9 mmol/L and pO2 of 77 mmHg on norepinephrine 3.5 mcg/kg/min, epinephrine 0.07 mcg/kg/min, vasopressin 0.06 units/hr and dobutamine 10 mcg/kg/min. Her high sensitivity troponin was >50,000 ng/L (Figure 1). She was centrally cannulated for VA ECMO with her EF of 10%. Given her medical history, illness presentation, and positive COVID-19 PCR, she was treated as an assumed COVID-19 related myocarditis. Treatment was initiated with tocilizumab, methylprednisolone, remdesivir and IVIG. Also, she was enrolled in a clinical trial and received plasma transfusion exchange for four days. After 10 days, TEE showed an improving EF from 15% to 30% and complete right ventricular recovery. Her course was complicated by acute renal failure and volume overload requiring initiation of dialysis, ARDS with P:F ratio of 167 (Images 1, 2), pneumonia, and ECMO catheter-associated right atrial thrombus. She was decannulated from VA ECMO after 13 days. Two weeks after admission a TEE showed complete biventricular recovery. She was extubated after 24 days, however, two days later she was found to have a right interlobar pulmonary artery embolism with contiguous extension into the middle and lower lobar pulmonary arteries and required re-intubation and re-initiation of a heparin infusion. This was attributed to dislodgment of the serpiginous right atrial clot. When medically able she was transferred to an active-military hospital for ongoing rehabilitation on room air, where she was weaned from dialysis and discharged.

Conclusion: In this report, we presented a young patient who successfully recovered from fulminant cardiogenic shock due to COVID-19 myocarditis through central cannulation of VA ECMO, combined with multimodal proven, postulated, and experimental treatments for COVID-19 and related complications. Our patient proved that VA ECMO is a valuable resource for COVID-19 related cardiac complications, but not a benign intervention. Complications such as coagulopathy, increased sedation requirements, lack

of mobility, and absence of pulsatile blood flow to the major organs should be considered as each of these had long term consequences

References: Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA Cardiol 5:802-810, Duration of veno-arterial extracorporeal life support (VA ECMO) and outcome: an analysis of the Extracorporeal Life Support Organization (ELSO) registry. Crit Care 21, 45 (2017).

Rescue Venoarterial Extracorporeal Membrane Oxygenation After Cardiac Arrest in COVID-19 Myopericarditis: A Case Report, Cardiovascular Revascularization Medicine, Volume 28, Supplement, 2021, Pages 57-60, ISSN 1553-8389, Potential of therapeutic plasmapheresis in treatment of COVID-19 patients: Immunopathogenesis and coagulopathy, Transfusion and Apheresis Science, Volume 59, Issue 6, 2020



Images 1, 2. Computed tomography (CT) scans in coronal (left) and axial (right) views demonstrating air bronchograms, crazy paving infiltrates and dense bilateral pulmonary consolidations. Taken August 16th, 2021, or 25 days after diagnosis with COVID-19.

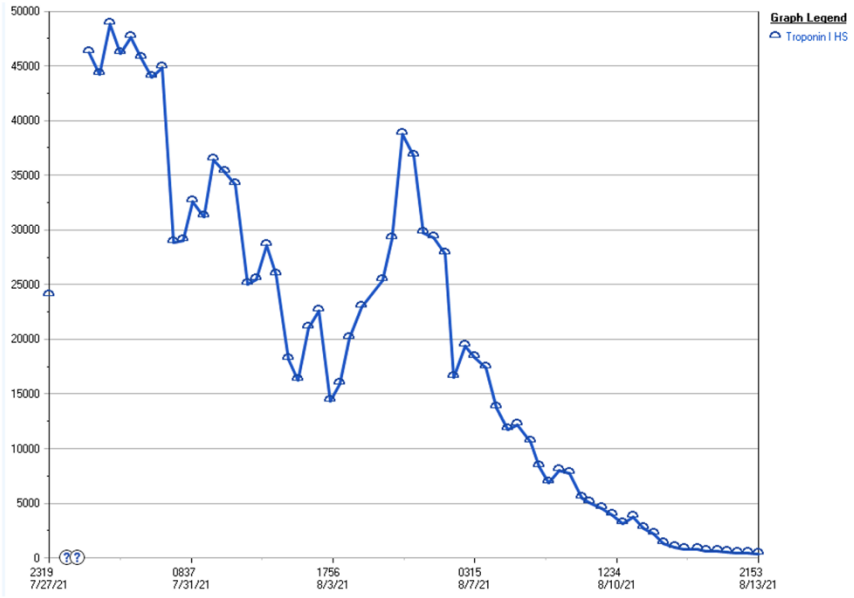


Figure 1. Troponin I high sensitivity trend from admission on July 27th 2021 through August 13th 2021. Normal Troponin I HS <52 ng/L. Plasmapheresis was initiated on August 2nd and completed August 6th.

Critical Care - 49 Case Of Multisystem Inflammatory Syndrome In Adult Requiring Impella Related To COVID-19

Lynden Baesch[1], Emily T Poynton[2]

[1]Cleveland Clinic, Cleveland Heights, OH, [2]University Hospitals Cleveland Medical Center, Cleveland, OH

Introduction: Intro: Throughout the course of the COVID-19 pandemic starting in 2019, several reports of a multisystem inflammatory syndrome in children (MIS-C) had been reported, which presented with symptoms similar to that of Kawasaki disease in children1,2,3. Symptoms included shock, cardiac dysfunction, abdominal pain, and elevated inflammatory markers, including elevated CRP, D-Dimer, and ferritin2. Patients also exhibit positive IgG antibodies to COVID-191. Currently, more and more reports are occurring which reveal a multisystem inflammatory syndrome in adults (MIS-A) related to COVID-19 infection.

Methods: Case Discussion: A 23-year-old female with no past medical history had previous COVID-19 infection with minimal symptoms of headaches one month prior to presentation. On presentation to hospital, patient had complaints of several days of fever, shortness of breath, and pleuritic chest pain. She was admitted to hospital at this time and was tachycardic with persistent fever. Patient had persistent hypotension despite aggressive IV fluid resuscitation. CT scan for pulmonary embolus was negative, but revealed bilateral pleural effusions, cardiac enlargement, and bilateral atelectasis. Transthoracic echocardiogram was done which showed severely reduced EF of 30-35%, normal right ventricular function, and mild mitral regurgitation. Her cardiac index was 1.8 L/min/m2. Given severe cardiogenic shock despite high dose vasoactive medications, plan was to place percutaneous Impella for cardiac support. Impella placement initially failed due to small vessels, so patient underwent a right axillary cutdown with Impella placement. Symptoms of high fevers and cardiogenic shock lead to a diagnosis of multisystem inflammatory syndrome in adults (MSI-A). This diagnosis was made as the patient had IgG positivity to COVID-19 and had elevated inflammatory markers, including elevated D-Dimer and CRP. The patient was treated with IVIG at dose of 2g/kg (based on ideal body weight) in two divided doses due to high volume. She was also treated with dexamethasone 6mg every 24 hours for ten days. Impella was removed after seven days as patient had cardiac recovery. Patient was later successfully extubated, weaned off vasoactive medications, and discharged from ICU. She was later discharged from hospital and continued to show full recovery at cardiology follow-up appointment.

Conclusion: Conclusion: During the COVID-19 pandemic, more and more reports are occurring of MIS-A. Patients have presented with varying symptoms of viral myocarditis, endothelitis, and vasculitis1,2. It is important to recognize this syndrome and appropriate treatment so that more can be learned about the effects of COVID-19 and how to adequately treat patients. More studies and reports need to be described in order to develop treatment protocols for conditions like MIS-A.

References: 1. MMWR Morb Mortal Weekly Report. 2020;69:1450-1456. 2. Annals of Internal Medicine. 2020 Dec 15;173(12):1025-1027. 3. European Heart Journal Cardiovascular Imaging. 2020 Sep 4:jeaa232.

Critical Care - 50 Decompensating Pregnant Patient With COVID-19 Pneumonia Delivered in the Medical Intensive Care Unit.

Anesah Elhaddi[1], Katya Chiong[2]

[1]CWRU/University Hospitals, Cleveland, OH, [2]University Hospitals Cleveland Medical Center/CWRU School of Medicine, Cleveland, OH

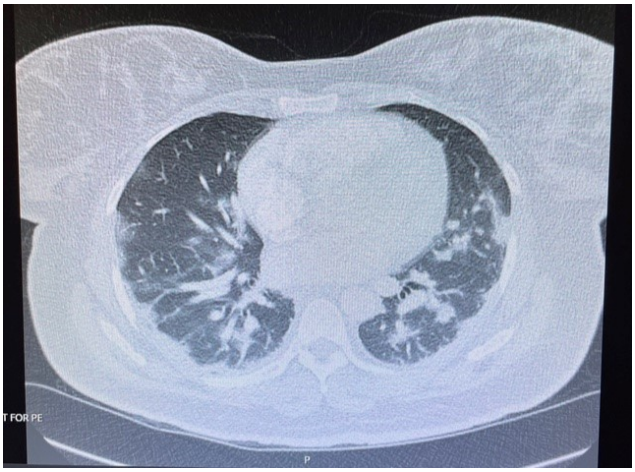
Introduction: Coronavirus disease 2019 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus infecting individuals and burdening healthcare systems worldwide. As more cases are discovered every day, data on disease severity, treatment and complications are investigated.¬π Although limited data exists on the clinical characteristics of SARS-CoV-2 on pregnant women, it is a topic of discussion as more pregnant women with COVID-19 are diagnosed and may be at a higher risk of being infected due to physiologic, anatomic and immunological changes. Acute respiratory distress due to COVID-19 in pregnant women carries severe complications as these women carry a higher risk of miscarriage, preterm delivery and fetal growth restriction which are detrimental to the mother and the fetus. A systematic review reported maternal mortality, stillbirth and neonatal mortality rates of 1.6%, 1.4% and 1% respectively. The purpose of this case report is to highlight the importance of multidisciplinary approaches to complex and critical illness with rapid and effective decision-making, understanding the implication of performing a cesarean section in the medical intensive care unit.

Methods: We report a 40 year old female G6P3 at 35 weeks pregnant who presented to labor and delivery triage with several days of body aches, fever, headaches and non-productive cough. Pregnancy had been notable for GDMA1, AMA and class II obesity. Patient tested positive for SARS-CoV-2 virus and was admitted for close monitoring and management. Initially, the patient was tachycardic, hypotensive and with an SpO2 of 96% on room air. Patient began experiencing worsening dyspnea and tachypnea with increased oxygen requirements and was placed on a NRB at 10 L. At that time, she was started on remdesivir and dexamethasone treatment per COVID-19 institutional guidelines. Chest CT for evaluation of PE was negative but showed extensive bilateral diffuse infiltrates and was started on prophylactic anticoagulation. On hospital day 3, a rapid response was called and the patient became increasingly tachypneic with a RR in the 40s and increasing dyspnea. At that time, she was placed on Airvo with the benefit of heated and higher volume of oxygen. The decision to transfer to the medical intensive care unit (MICU) was warranted for escalation of care. As fetal status remained overall reassuring, the initial expectation was for the patient to recover from a respiratory standpoint in preparation for an induction of labor. However due to worsening clinical status of the patient, a multidisciplinary approach ensued consisting of the MFM, OB anesthesia, and critical care teams as well as nursing and respiratory therapy. On hospital day 4, the multidisciplinary discussion teams met to reassess the patient's worrisome respiratory status and concerns for barriers to care with transport to even the nearest OR. The safest plan for both the patient and the fetus was deemed delivery by cesarean section under general anesthesia, necessitating a TIVA in the MICU. The patient and the fetus both tolerated the procedure well.

Conclusion: Management of critical illness in maternal populations is important and serves many concerns to the mother and the fetus. Due to the worsening respiratory status of our patient, our multidisciplinary team deemed that the patient was too unstable to be transported safely to the main operating room for delivery. After careful review, the decision to intubate and place the patient on mechanical ventilation with immediate cesarean delivery to follow was the appropriate next step. Prior to intubation, fetal monitoring was reassuring and all teams were prepared to begin. Neuraxial anesthesia would not have been an appropriate option due the patient's decompensation and her current anticoagulation prophylaxis onboard. The importance of mode and timing of delivery should be individualized based on severity of disease, co-existing comorbidities and obstetrical indications. Although delivery in the MICU has risks, all teams had the appropriate staff members, equipment and medications prepared to deal with any emergencies. Early communication and planning by the multidisciplinary teams was crucial to delivering safe and excellent care to a decompensating pregnant

patient with severe-COVID-19 pneumonia outside the norm, away from the operating room and in the medical intensive care unit.

References: 1. American Journal of Reproductive Immunology, volume 84, 1-11, 2020 2. International Journal of Medical Sciences, volume 18, 763-767, 2021 3. International Journal of Gynecology and Obstetrics, volume 151, 7-16, 2020



Critical Care - 51 The Use of Double Oxygenators in VV-ECMO for Severe Burns

Faryal Ahmad[1], Elio Beta[2]

[1]Loyola University Medical Center, Chicago, IL, [2]McGaw Medical Center of Northwestern University, Chicago, IL

Introduction: We present a case of a severe burn patient with >50% BSA requiring VV ECMO with two oxygenators in parallel, due to the severe refractory hypoxemic respiratory failure and high metabolic demand as a result of the extensive burns.

Methods: Our case is of a 28-year-old male with no significant medical history presenting after >50% body surface area burns due to a chemical being thrown into an open flame. Severe refractory hypoxemic failure ensued presumably due to a combination of factors including chemical inhalational injury, extensive burns, and pulmonary edema secondary to resuscitation or pneumonia. Patient was placed on VV ECMO through femoral veins access but remained hypoxic presumably secondary to high metabolic demand due to a high burn burden. An additional cannula was inserted into the right internal jugular with VVV ECMO configuration to increase the volume of blood running through the oxygenator. However, it was observed that blood oxygenation was not optimized, likely due to rapid blood flow and insufficient contact time with oxygenator membrane. To overcome this, an additional oxygenator was added in parallel, with right and left femoral cannula inflows to separate oxygenators and a joined right IJ outflow. Splitting the flow allowed for greater contact time with oxygenator membrane which translated in significant clinical improvement. Patient was subsequently successfully decannulated 7 days later.

Conclusion: Triple cannulation ECMO is a newer technique being employed more frequently in cases requiring increased circulatory and respiratory support, such as our case. Depending on the clinical situation requiring increased support, there are recommendations for how triple cannulation should be executed.1 Our patient developed multifactorial, severe, refractory hypoxic respiratory failure in the setting of extensive burn injury. Two-oxygenator approach has previously been successful in resolving refractory hypoxemia in burns patients.2 We argue that in this setting the cardiac output and metabolic demand were higher than what could be met with a single oxygenator configuration and additional oxygenator was needed to meet the high metabolic demand. Support for ECMO in ARDS associated with burns is not widespread and there are limited case reports outlining its effectiveness. Here we outline both the use of triple cannulation and double oxygenators in parallel with significant improvement in a case of severe burns.

References: 1. Napp LC (2016). Clin Res Cardiol. 105(4):283-296 2.Szentgyorgyi L (2018). Burns. 44(5):1091-1099

Critical Care - 52 Drug Induced Immune Hemolytic Anemia secondary to Piperacillin and Tazobactam

Hatim D Ali[1], Avneep Aggarwal[1]

[1]Cleveland Clinic, Cleveland, OH

Introduction: The incidence of Drug-induced immune hemolytic anemia (DIIHA) is rare with an incidence of about 1 in 1 million. Many medications have been implicated in the diagnosis. Due to increased administration over the years, Piperacillin-Tazobactam is now the third most common drug to cause Hemolytic anemia. Our case illustrates the interesting presentation of a patient presenting to the ICU in hemorrhagic shock secondary to hemolytic anemia due to Piperacillin-Tazobactam induced hemolysis with possible super-imposed TTP.

Methods: A 69-year-old female with past medical history significant for coronary artery disease, diastolic heart failure, cerebrovascular accident, and chronic kidney disease presented for removal of an infected hernia mesh. On post-operative day 1, the patient was found to be anemic requiring blood transfusion. On day 2, the patient was transferred to the surgical ICU in hemorrhagic shock despite multi-unit blood transfusion. Initially suspicion was that patient was having a post-surgical bleed. But, there were no signs of active bleeding on exam and imaging. Lab work was consistent with hemolytic anemia. Concomitantly, the patient developed worsening thrombocytopenia, acute kidney injury and altered mental status. Following a complex workup in conjunction with the American Red Cross, the patient was diagnosed with Drug-Induced Immune Hemolytic Anemia. The patients' blood sample was positive for anti-piperacillin antibodies and her serum was DAT positive. Patient was given methylprednisolone and antibiotic therapy was changed. Interestingly, the patient sample also tested low for ADAMTS 13 activity with the presence of ADAMTS13 inhibitor. Due to the concern for TTP, urgent plasmapheresis was initiated with correction of ADAMTS 13 activity and undetectable levels of inhibitor. Additional serum tests were ordered to assess for cross-reactivity between the patient's inherent anti-bodies and of those biomarkers that would paint the picture of TTP. Eventually, patient was successfully discharged to a skilled nursing facility after a prolonged course in ICU.

Conclusion: Drug-induced immune hemolytic anemia may be a life-threatening complication of antibiotic therapy. While Drug-Induced hemolytic anemia as a cause is well documented, documentation of hemolysis secondary to Piperacillin and Tazobactam administration specifically is limited to individual case reports. This case illustrates the development of an immune hemolytic anemia caused by the presence of warm, DAT+ anti-piperacillin antibodies with pan agglutinin e-like reactivity. Piperacillin-Tazobactam induced hemolysis should be considered as a differential diagnosis in patients with hemolytic anemia as an early suspicion and diagnosis is crucial for successful outcome. Although no conclusions can be made based on the experience of a single patient, we believe this particular case presents the opportunity for further discussion and a valuable mean for heightening initial clinical suspicion.

References: 1. George Garratty; Drug-induced immune hemolytic anemia. Hematology Am Soc Hematol Educ Program 2009; 2009 (1): 73-79. 2. George Garratty, Immune hemolytic anemia associated with drug therapy. Blood Reviews, Volume 24, Issues 4-5, 2010, Pages 143-150. 3. Leger RM, Arndt PA, Garratty G. Serological studies of piperacillin antibodies. Transfusion. 2008 Nov;48(11):2429-34.

Critical Care - 53 A Case of Acute Right Heart Failure after Pericardial Window

Anastasia Meleties[1], Liang Shen[2], Sagar Navare[3]

[1]NYP Weill Cornell Medical Center, New York, NY, [2]Weill Cornell Medical College, New York, NY, [3]NYP Weill-Cornell Medical Center, New York, United States of America

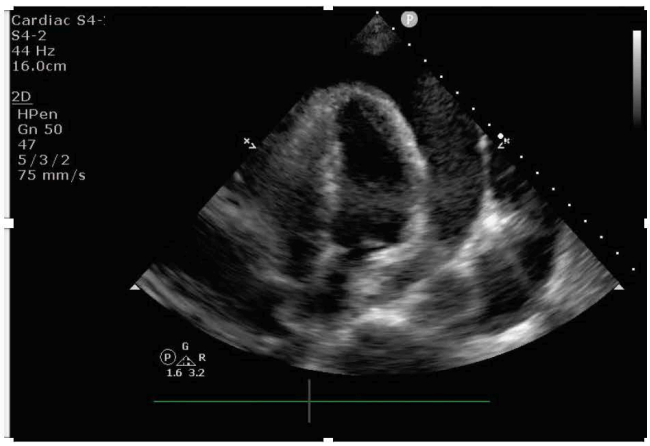
Introduction: We describe a case of acute post-operative right heart failure after a pericardial window in a patient with no previous history of cardiovascular disease that was attributed to pericardial decompression syndrome, a rare complication of pericardial effusion drainage. We focus on diagnosis and management strategies of right heart failure in the acute setting, emphasizing the need for a high index of suspicion and attention to clinical findings.

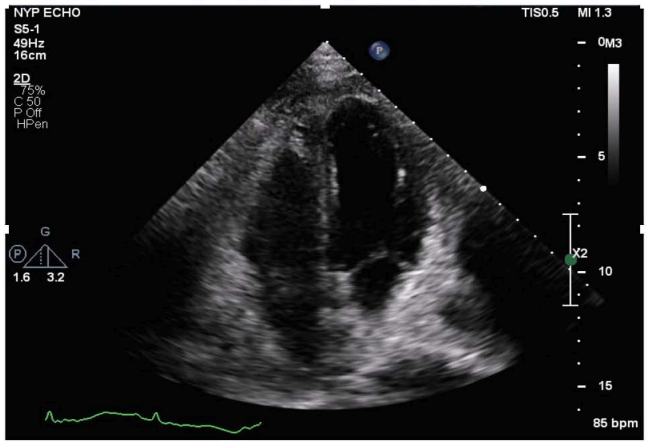
Methods: A 70-year-old female with no significant past medical or surgical history initially presented to an outpatient clinic complaining of dyspnea and decreased exercise tolerance for approximately two weeks. An EKG was done that showed new-onset atrial fibrillation with rapid ventricular response; this finding in the setting of dyspnea prompted transfer to the emergency room. Pulmonary embolism was ruled out by the ER staff with a CT scan, though the scan was remarkable for a new peri-hilar mass and a large pericardial effusion. She was then transferred to a tertiary care center for evaluation by Cardiothoracic Surgery. She was admitted directly to a CTICU where a bedside ultrasound was done that showed evidence of diastolic collapse of the right ventricle, so she was taken emergently for decompression via pericardial window. TEE performed during the case showed resolution of the effusion after drainage of 800cc of pericardial fluid, but noted global hypokinesis and dilation of the right ventricle. Shortly after arrival to the CTICU post-operatively, her vasopressor requirement significantly increased, along with her lactate. A PA catheter was placed in the ICU and helped to confirm that the patient was in cardiogenic shock (cardiac index 1.1) due to right heart failure (CVP 16, dilated and hypokinetic RV seen on TTE compared to underfilled LV with no apparent valvulopathy). Transaminitis and bilirubinemia further supported a shock diagnosis. The patient was supported hemodynamically with vasopressors, inotropes, and diuresis while her progress was monitored via labs, measurements from the PA catheter, and serial echocardiograms. Shock work-up did not reveal new ischemic cardiomyopathy as the cause, nor was the cause thought to be hypovolemia (hemorrhagic or otherwise), obstructive (pericardial effusion did not recur, no pulmonary embolism on imaging), infection, or anaphylaxis. Her shock was attributed to pericardial decompression syndrome as a diagnosis of exclusion. The patient recovered to near-normal cardiac function within two weeks and was discharged home, at which point she began outpatient treatment for her newly diagnosed lung malignancy (the most likely etiology of her pericardial effusion). The pathophysiology of pericardial decompression syndrome is not entirely understood given its rarity and likely under-reporting. One current hypothesis is interventricular volume mismatch; preload is suddenly increased in the setting of high SVR and tachycardia. Others suggest myocardial stunning as a cause, given that the elevated interventricular pressues may impede coronary artery filling. Finally, others point to an imbalance of the sympathetic and parasympathetic system after having been in a high-catecholamine state for a prolonged period of time.

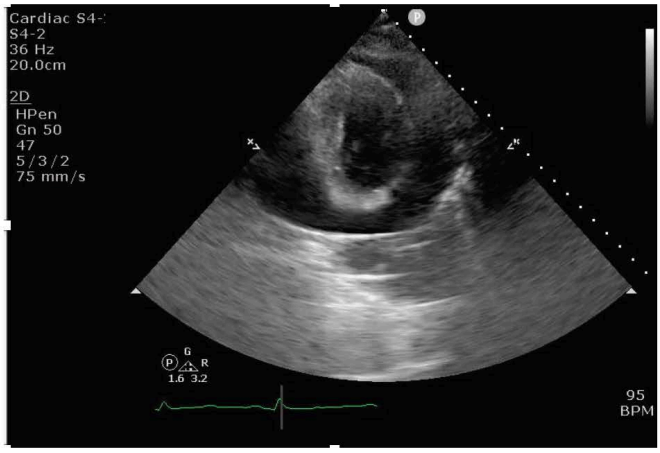
Conclusion: This case highlights the idea that a high index of suspicion necessary to diagnose quickly evolving pathology such as acute right heart failure or cardiogenic shock in general. Echocardiography is a useful tool for rapid diagnosis and serial monitoring of recovery progress. Finally, swift intervention and decisive action are key to ICU management .

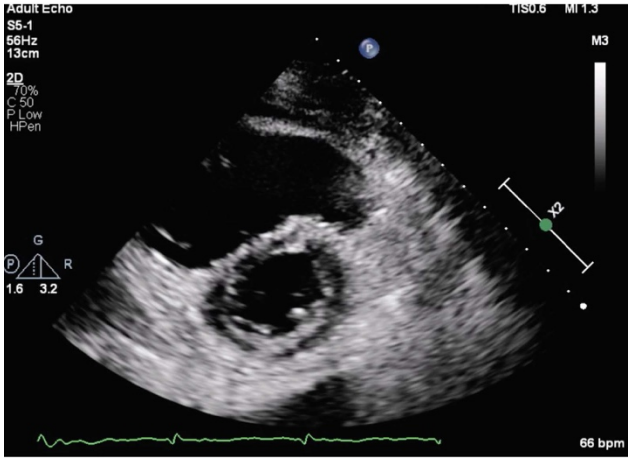
References: Kearns MJ, Walley KR. Tamponade: Hemodynamic and Echocardiographic Diagnosis. Chest. 2018;153(5):1266-1275. doi:10.1016/j.chest.2017.11.003 Konstam MA, Kiernan MS, Bernstein D, et al. Evaluation and Management of Right-Sided Heart Failure: A Scientific Statement From the American Heart Association. Circulation. 2018;137(20):e578-e622. doi:10.1161/CIR.0000000000000560 Prabhakar Y, Goyal A, Khalid N, et al. Pericardial decompression syndrome: A comprehensive review. World J Cardiol. 2019;11(12):282-291. doi:10.4330/wjc.v11.i12.282 Acute Right Ventricular Failure After Surgical

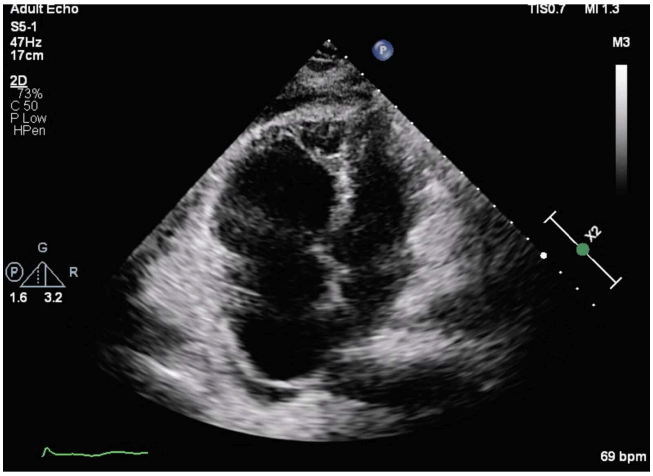
Drainage of Pericardial Tamponade: A Case Report of Pericardial Decompression Syndrome and Review of the Literature Chung et al. - Journal of Cardiothoracic and Vascular Anesthesia - 2019











Critical Care - 54 Recurrent Euglycemic Diabetic Ketoacidosis in the Setting of SGLT-2 Inhibitor Use

Chase Elkin[1], Kitae Chang[1], Robert F Groff[1], Amit Prabhakar[1], Craig S Jabaley[1], Samer Melhem[1]

[1]Emory University School of Medicine, Atlanta, GA

Introduction: Euglycemic diabetic ketoacidosis (eDKA) is a well-known, but poorly understood complication of sodium-glucose cotransporter 2 inhibitor (SGLT-2i) use in the management of type 2 diabetes mellitus (T2DM). However, there is scant literature that has described cases of recurrence of eDKA despite early, aggressive treatment.

Methods: A 47 year-old male with uncontrolled T2DM who was recently started on an SGLT-2i presented to a community hospital with chest pain and was found to have ST-elevation myocardial infarction (STEMI). He was emergently taken for a left heart catheterization (LHC) which demonstrated severe multi-vessel coronary artery disease (mvCAD) and underwent a plain old balloon angioplasty as a temporization strategy prior to definitive revascularization. He was transferred to our community based academic medical center for coronary artery bypass graft (CABG) surgery evaluation. On the day prior to transfer, the patient received dapagliflozin 10 mg at the outside facility along with medical management for STEMI. On arrival, an intra-aortic balloon pump (IABP) was placed to augment coronary artery perfusion and the patient was admitted to the cardiothoracic surgery ICU. Shortly after arrival, the patient became altered and hemodynamically unstable requiring endotracheal intubation and high dose vasopressor therapy. Initial labs demonstrated a profound anion gap acidosis with elevated beta-hydroxybutyrate in the setting of near-normal blood glucose and significant glucosuria. The patient was initiated on an aggressive insulin infusion protocol and remained on high dose vasopressor support for a mixed shock picture driven by eDKA. On day 3 of hospitalization, the anion gap closed and the patient was able to be weaned from vasopressor support. With improvement in overall clinical status, the patient underwent percutaneous coronary intervention (PCI) and placement of a drug-eluting stent on day 5 without complications. The IABP was removed on day 6. Later that day, the patient was again found to have recurrent eDKA despite ongoing parenteral insulin and dextrose infusions. The insulin and dextrose infusions were uptitrated under the guidance of the endocrinology service with gradual correction of DKA. The remainder of this patient's care was uneventful, and he was discharged from the ICU on day 9.

Conclusion: Recurrent, refractory eDKA is an uncommon, but life-threatening side effect of SGLT-2i therapy which may be provoked by critical illness or surgery. The duration of action of certain SGLT-2i medications may exceed 7 days. and requires vigilance and a high index of suspicion by the critical care treatment team.

Critical Care - 55 Colorado's First Coronavirus-19 (COVID-19) Lung Transplant Following Veno-venous Extracorporeal Membrane Oxygenation (VV-ECMO)

Kenji Tanabe[1], Sarah Alber[2], Samuel Gilliland[2], Andrew Hennigan[3], Maung Hlaing[1], Meghan Prin[4], Breandan Sullivan[4], Marianne Wallis[4], Tim Tran[2]

[1]University of Colorado, Denver, CO, [2]University of Colorado, Aurora, CO, [3]University of Colorado School of Medicine, Aurora, United States of America, [4]University of Colorado School of Medicine, Aurora, CO

Introduction: It is estimated that 5-8% of the patients infected with COVID-19 will require mechanical ventilation, which carries a mortality rate of approximately 35%. Lung transplant is a lifesaving treatment for refractory COVID-19 that has only previously been described in small case series. In this report, we present Colorado's first case of a bilateral lung transplant performed for a patient with severe COVID-19 pneumonia bridged with VV-ECMO.

Methods: A thirty-seven year old male with multiple sclerosis well controlled on ocrelizumab presented to a rural hospital with profound hypoxia after being diagnosed with COVID-19 pneumonia nine days prior. He was managed with bilevel positive airway pressure and medical therapies including dexamethasone, remdesivir, and monoclonal antibody infusion. On hospital day 7 the patient rapidly decompensated and required intubation, chemical paralysis, prone positioning, and VV-ECMO cannulation (right femoral to right internal jugular vein). He underwent tracheostomy on hospital day 29 and was transferred to a tertiary hospital in Colorado on hospital day 37 for lung transplant consideration. At the tertiary care hospital, the patient's ECMO was reconfigured to a dual lumen right internal jugular cannula and his sedation weaned to facilitate rehabilitation and communication. He was supported for an additional month using lung protective ventilation and VV-ECMO with no improvement in lung compliance. On hospital day 78, the patient was listed for lung transplantation. A suitable deceased cardiac donor became available and on hospital day 88 the patient underwent a bilateral lung transplant performed on cardiopulmonary bypass. Following a chest washout on postoperative day (POD) 1, the patient separated from ECMO support on POD 4, transferred to the floor on POD 8, and discharged to acute inpatient rehabilitation on POD 21 (hospital day 110).

Conclusion: Bilateral lung transplant is a lifesaving intervention for a subset of patients with severe COVID-19 pneumonia requiring VV-ECMO, despite limited evidence. The largest published case series, which followed twelve COVID-19 lung transplant recipients, reported an 80-day survival of 91.6%. Specific pre-transplant clinical criteria met by this patient included age under 65, single organ dysfunction, radiologic evidence of irreversible lung fibrosis, intact capacity, and active participation in rehabilitation. Interestingly, this patient's time on ECMO prior to transplant (81 days) was substantially longer than the reported average (49 days). Despite the risks that accompany ECMO support, this was felt necessary to facilitate the patient's rehabilitation and ensure that his lung damage was truly irrecoverable prior to transplant.

References: 1. COVID-19: Epidemiology, clinical features, and prognosis of the critically ill adult. In: Uptodate (2021). 2. 'ICU and ventilator mortality among critically ill adults with coronavirus disease 2019.' Critical care medicine. 48(9):e799-e804 (2020). 3. 'Lung Transplantation in a Patient With COVID-19-Associated Acute Respiratory Failure.' Cureus 13.8 (2021). 4. 'When to consider lung transplantation for COVID-19.' The Lancet Respiratory Medicine 8.10: 944-946 (2020). 5. 'Early outcomes after lung transplantation for severe COVID-19: a series of the first consecutive cases from four countries.' The Lancet Respiratory Medicine 9.5: 487-497 (2021).

Critical Care - 56 E CPR And VA ECMO With TEE Guidance In A Case Of Peripartum Hemodynamic Collapse

Alexander Lewis[1], Ranjani Venkataramani[1]

[1]University of New Mexico, Albuquerque, NM

Introduction: Peripartum hemodynamic instability has multiple etiologies including cardiovascular disease, postpartum hemorrhage, sepsis and hypertensive disorders.1 Peripartum cardiomyopathy is a rare form of systolic heart failure presenting at the end of pregnancy and has a higher incidence in African American women, advanced maternal age, concurrent hypertensive disorders, and multiple gestation.2 The utility of TEE in cardiac and high risk populations is well established however its use in critically presenting patients is not well described.3 Presented here is the use of resuscitative TEE to diagnose of a peripartum patient with sudden cardiorespiratory failure.

Methods: A G4P3 28-week antenatal woman with limited prenatal care and a history of chronic and current methamphetamine and current and chronic alcohol use was airlifted and brought to our ICU. She presented to the outside hospital in acute respiratory distress from pulmonary edema and was intubated for hypoxemia (72% saturation on 10 L oxygen). Fetal bradycardia ensued and fetal heart tone was absent upon arrival in our ICU. Systemic hypotension with <80 mm Hg SBP quickly led to a brief episode of cardiac arrest with a PEA. She was resuscitated per ACLS protocol with epinephrine and a bolus of fluid in the form of 500 mL of LR while adequate access was obtained. Transthoracic POCUS did not yield adequate images. At this point we initiated TEE guided resuscitation which revealed the following findings: acute LV dilatation and dysfunction with an LVEF of 10 to 15% with severe MR and lack of co-optation of the mitral leaflets. Left uterine displacement was performed. Veno-arterial ECMO (VA-ECMO) was then initiated using a 20 Fr femoral arterial cannula and a 23 Fr femoral venous cannula. During this time hemodynamics were supported by epinephrine and norepinephrine drips. PEEP was set at 12 mm Hg to circumvent pink frothy sputum from pulmonary edema. Once VA ECMO flows were established at 3-3.5L/min, TEE was used to adjust flows to be able to see just enough aortic valve opening with every cardiac contraction and maintain a pulse pressure of at least 20 mm Hg to prevent further LV distension and stasis of blood in the left atrium to mitigate the amount of mitral regurgitation. The fetal demise and remains were delivered via vaginal route without a need for a cesarean section. The patient remained on VA ECMO for 48 hours during which pulmonary edema resolved and we were able to see an improvement in her ejection fraction to 30% while doing a ramp down study of her VA ECMO flows at about 48 hours. She was decannulated after 72 hours on ECMO with a marginal improvement in LV function to approximately 35-40% on minimal dose of inotropes. Unfortunately, her neurological status remained a GCS 3T2 with an MRI confirming hypoxic brain injury. She was transferred out to an LTAC facility for ongoing care.

Conclusion: This case highlights the importance of resuscitative TEE and maintaining a high index of suspicion for a broad differential for cardiac arrest in the peripartum period. TEE was also used to help direct urgent ECMO cannulation at the bedside by visualizing wires in the IVC and descending aorta enabling swift cannulation.

References: 1. Kolovetsiou-Kreiner V, Moertl MG, Papousek I, et al.: Maternal cardiovascular and endothelial function from first trimester to postpartum. PLoS One. 13:e0197748, 2018. 2. Davis MB, Arany Z, McNamara DM, et al.: Peripartum Cardiomyopathy: JACC State-of-the-Art Review. J Am Coll Cardiol. 75:207-221, 2020. 3. Burrage PS, Shernan SK, Tsen LC, et al.: Emergent transesophageal echocardiography in hemodynamically unstable obstetric patients. Int J Obstet Anesth. 24:131-136, 2015.

Critical Care - 57 Bedside Echo Uses Beyond Diagnosis in the Critically Ill: A Case Series of Dynamic Takotsubo

Alexander Lewis[1], Ranjani Venkataramani[1]

[1]University of New Mexico, Albuquerque, NM

Introduction: Stress induced cardiomyopathy also known as Takotsubo cardiomyopathy or broken heart syndrome is a rare diagnosis and presents with an acute dramatic hemodynamic instability mimicking myocardial infarction.1 Patients are typically elderly females with recent stress provoking syndromes. The comorbidities and presenting symptoms dictate its treatment with proposed pathophysiology including catecholamine cardiotoxicity, metabolic disturbances, coronary microvascular impairment and multivessel epicardial coronary artery spasm.2 Presented here are two separate cases where point of care ultrasound (POCUS) was used to diagnose an unknown etiology of hemodynamic instability and which changed the treatment algorithm and care of each patient.

Methods: Presented here are two separate cases of Takotsubo cardiomyopathy with drastically different presentations that have implications for perioperative and critical care physicians. The first patient presented with acute flaccid quadriparesis prompting a working diagnosis of Guillain Barre Syndrome (GBS). The patient was admitted to the neurocritical care unit with refractory hypotension. Hypotension was attributed to autonomic instability and vasodilation with intravenous fluids subsequently being administered. Concurrently, ST-T wave elevations were noted on an EKG during this time. The ST-elevations were diffuse over several leads not meeting criteria for STEMI. A POCUS cardiac exam demonstrated global hypokinesis with apical ballooning of the LV function (estimated LVEF 20-25%) with preserved RV function. The cardiac angiogram was negative for coronary occlusion confirming diagnosis of Takotsubo cardiomyopathy. Hemodynamics were optimized using a low dose inotrope. The second patient was admitted to the medical intensive care unit from the nephrology service following treatment with pulse steroids and basiliximab for acute antibody mediated rejection that was complicated by hypotension. Similar EKG changes with ST-T changes were noticed in several leads without an anatomic correlation to a coronary territory. Bedside POCUS revealed global hypokinesis of the LV but marked hyperkinesis of the basal segments and apical ballooning leading to dynamic LVOT obstruction and systolic anterior motion of the mitral valve (SAM). This finding led us to administer fluids, treat hypotension with an alpha agonist to raise SVR and avoid inotropic agents. Once the MAP stabilized, low dose carvedilol was introduced over the next 24 hours.

Conclusion: These cases highlight the importance of resuscitative TTE and the use of this diagnostic tool. Stress induced cardiomyopathy or Takotsubo arises from hyperadrenergic states. The diagnosis however can have different implications based on the pathophysiologic type. While the classic pattern of apical ballooning is common several other types of Takotsubo have been reported. These include the midventricular, basal, and focal wall motion patterns. Besides the four major types, other morphological variants have been described including the biventricular apical type and right ventricular involvement, isolated right ventricular, and global form. While one type required the use of inotropes, the other type would have become worse with inotropy due to worsening of LVOT obstruction. Identifying the type of Takotsubo cardiomyopathy is key in managing these patients in the acute care setting and when subjected to anesthesia in the perioperative period as using inotropes or fluids may not be the answer to all hypotensive episodes.3

References: 1. Dawson DK: Acute stress-induced (takotsubo) cardiomyopathy. Heart. 104:96-102, 2018. 2. Ono R, Falcao LM: Takotsubo cardiomyopathy systematic review: Pathophysiologic process, clinical presentation and diagnostic approach to Takotsubo cardiomyopathy. Int J Cardiol. 209:196-205, 2016. 3. Ghadri JR, Wittstein IS, Prasad A, et al.: International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. Eur Heart J. 39:2032-2046, 2018.

Critical Care - 58 Inappropriate Shock Therapy due to Electromagnetic Interference from an Implanted Left Ventricular Assist Device in a Patient with Subcutaneous Implantable Cardioverter-Defibrillator

Jeffrey Lin[1], Gene Kurosawa[1], Mandeep Singh[1]

[1]Department of Anesthesiology, Keck School of Medicine, University of Southern California, Los Angeles, CA

Introduction: The prevalence of left ventricular assist devices (LVADs) continues to rise both as a bridge to transplantation and as destination therapy for patients with left ventricular heart failure. Many patients undergoing LVAD implantation also have an implantable cardioverter-defibrillator (ICD) to prevent sudden cardiac death [1]. Transvenous ICD implantation has several potential complications including pocket hematoma, endocarditis, lead fracture, and venous obstruction [2]. A subcutaneous implantable cardioverter-defibrillator (S-ICD) is an attractive option to avoid these lead-related complications, especially in patients undergoing LVAD implantation since device-related endocarditis could require prolonged antibiotics or pump exchange [3]. However, electromagnetic interference (EMI) from the LVAD can lead to sensing dysfunction in the S-ICD [4]. Here we report a case of inappropriate shocks delivered from a patient's S-ICD following LVAD implantation.

Methods: A 54 year-old male with non-ischemic cardiomyopathy on chronic intravenous milrinone and with an S-ICD placed 3 years prior, was transferred to our institution with acute decompensated heart failure. S-ICD interrogation prior to transfer revealed no treated episodes and no shocks since the S-ICD was implanted (Fig 1, Fig 2). Of the 3 sensing vectors available, the patient's S-ICD was configured to sense the secondary vector (Fig 3). The patient was admitted to the ICU and continued on milrinone in addition to a bumetanide drip. Transthoracic echo showed an ejection fraction of 15-20%, mild mitral regurgitation, and mild tricuspid regurgitation. Approximately two weeks following admission, the patient underwent Heartmate 3 (HM3) LVAD implantation along with tricuspid valve repair via median sternotomy. A magnet was placed over the S-ICD throughout the case. The patient exited the operating room on milrinone and epinephrine drips and maintained an intrinsic rhythm of sinus tachycardia with a heart rate in the 110s. On postoperative day 1, the patient was awake and close to extubation, but suddenly experienced consecutive shocks from his S-ICD. The telemetry rhythm strip from his five-lead EKG at the time displayed sinus tachycardia with a heart rate in the 100s. A magnet was immediately placed over the S-ICD, preventing further shocks. The in-house device representative was contacted, and S-ICD interrogation showed the device was oversensing at >300 bpm or undersensing with noise from the LVAD (Fig 4). It appeared that the low-amplitude R wave and EMI oversensing in the secondary sensing configuration allowed for the delivery of inappropriate shocks despite the underlying rhythm being sinus tachycardia (Fig 5). The S-ICD therapy was subsequently turned off. The patient was then slowly weaned off epinephrine and extubated post-operative day 2. The HM3 was noted to be placed between the lead and the S-ICD, and it would be possible to use the alternate sensing vector (Fig 3) to distinguish the R wave amplitude from HM3 noise. The patient transferred out of the ICU with the defibrillator function turned off, pending further discussions to either continue using the S-ICD with alternate sensing or converting to a transvenous system.

Conclusion: LVAD implantation may incite EMI oversensing in patients with S-ICDs and result in inappropriate shock therapy. Based on the electrode ring to the can, there are three possible sensing vectors in the S-ICD system: primary, secondary, and alternate (Fig 3). Our patient's S-ICD detected EMI in both the primary and secondary vectors leading to inappropriate shocks. Two reports of inappropriate sensing due to EMI in patients with S-ICDs leading to inappropriate shocks also demonstrated oversensing in the primary and secondary vectors triggering multiple shocks after HM3 implantation, but there was no EMI observed in the alternate vector similar to our case [2]. It has been shown that the shorter distance between the S-ICD and the LVAD correlates with EMI which is a potential explanation as to why the distant alternate vector has less EMI [5]. There should be strong consideration to avoiding S-ICD placement in patients who are potential

LVAD candidates given the risk of oversensing and inappropriate shock therapy. LVAD candidates who already have S-ICDs implanted should have the device reprogrammed to sense the most appropriate vector.

References: 1. Cardiac implantable electronic devices in patients with left ventricular assist systems. J Am Coll Cardiol. 2018;71(13):1483-1493. 2. Inappropriate shocks in the subcutaneous ICD: Incidence, predictors and management. Int J Cardiol. 2015;195:126-133. 3. Subcutaneous implantable cardioverter-defibrillator troubleshooting in patients with a left ventricular assist device: A case series and systematic review. Heart Rhythm. 2020;17(9):1536-1544. 4. De novo subcutaneous implantable cardioverter-defibrillator in patient with left ventricular assist device. HeartRhythm Case Rep. 2020;6(10):774-776. 5. Electromagnetic interference from left ventricular assist devices in patients with subcutaneous implantable cardioverter-defibrillators. J Cardiovasc Electrophysiol. 2020;31(5):1195-1201. 6. Subcutaneous ICD: Current standards and future perspective. Int J Cardiol Heart Vasc. 2019;24:100409

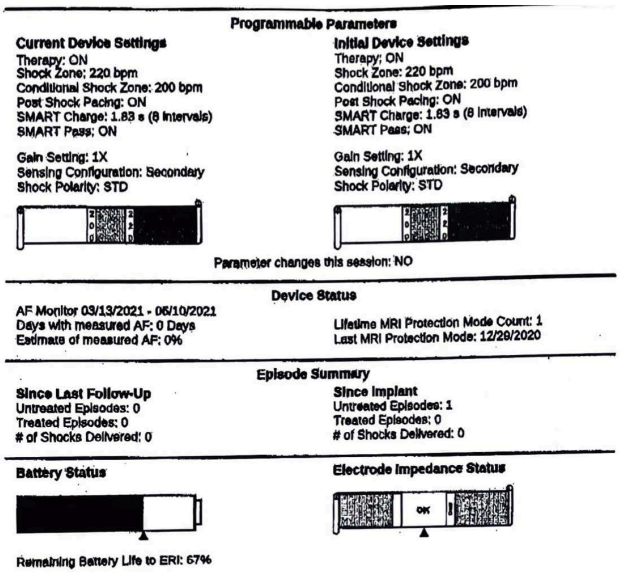


Figure 1: Interrogation summary of S-ICD prior to LVAD implantation

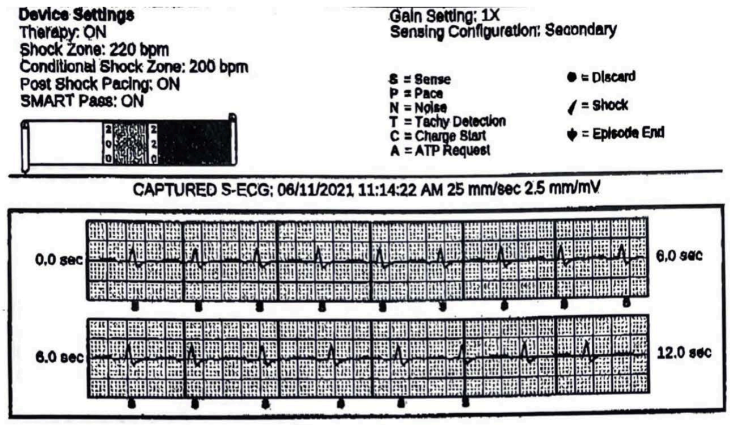


Figure 2: Interrogation capture/sensing data of the S-ICD prior to LVAD implantation

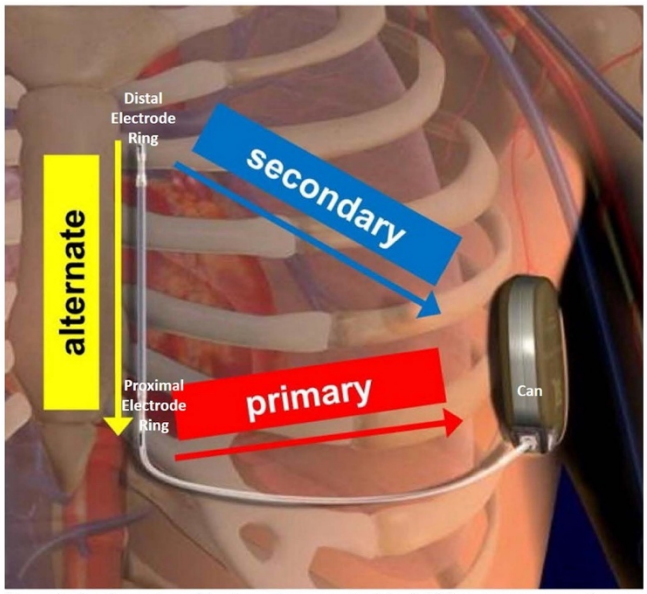


Figure 3: Configuration of the 3 sensing vectors of the S-ICD: primary vector (from proximal electrode ring to can), secondary vector (from distal electrode ring to can) and alternate vector (from distal to proximal electrode) [6]

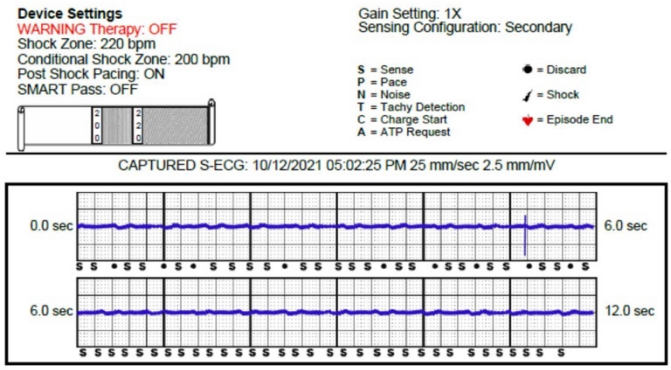


Figure 4: Captured sensing data of the S-ICD following LVAD implantation

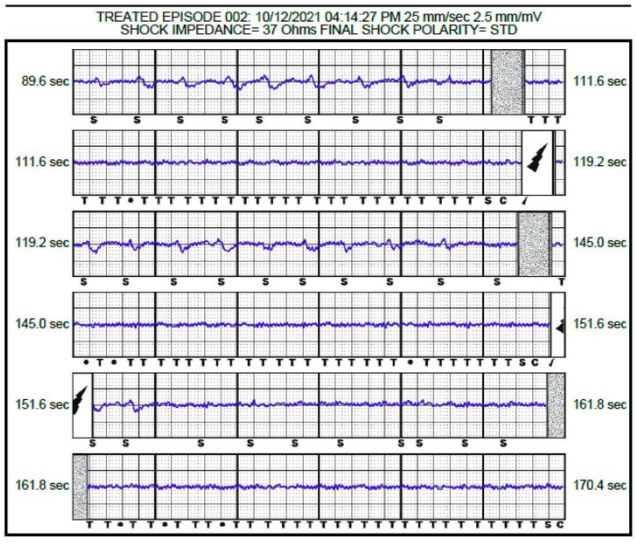


Figure 5: Captured waveforms from the S-ICD during inappropriate shock therapy following LVAD implantation

Critical Care - 59 A rare post-pancreas transplant arterioenteric fistula presenting as a near-fatal GI bleed and requiring aggressive surgical intervention

Soyun M Hwang[1], Benjamin Philosophe[1], Aliaksei Pustavoitau[2], Sergio B Navarrete[3]

[1]Johns Hopkins University School of Medicine, Baltimore, MD, [2]Johns Hopkins University, Baltimore, MD, [3]Johns Hopkins Hospital, Baltimore, MD

Introduction: Arterioenteric (AE) fistula is a rare but critical complication after pancreas transplant with less than 30 cases reported in the past 20 years [1, 2]. Pathophysiology includes peripancreatic enzyme leak, chronic rejection, surgical trauma or biopsy [3]. Although massive GI bleed is the cardinal outcome, clinical presentation of AE fistula varies widely, making prompt diagnosis and intervention challenging [4, 5]. This is a rare case of an AE fistula after simultaneous pancreas-kidney (SPK) transplant that initially presented with intermittent bloody stools and negative diagnostic work-up. This elusive presentation rapidly progressed to near-fatal GI bleed with ongoing shock despite interventional radiology (IR) stenting and required emergent surgical repair with graft pancreatectomy.

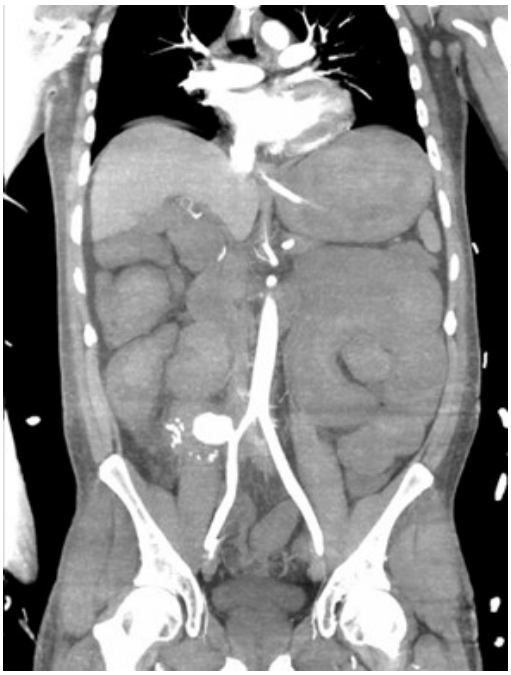
Methods: A 37-year-old male with T1DM and ESRD secondary to lupus nephritis underwent SPK transplant in 2019. Immediate post-transplant period was unremarkable without delay in graft function. 4 months post-transplant, the patient presented with abdominal pain and was found to have increased amylase and lipase and pancreatic stranding on CT. Pancreas biopsy showed grade II acute rejection. Patient was treated with high dose steroids and antithymocyte globulin and followed closely every 3 months. Labs improved and immunosuppressive therapy was appropriately titrated. 23 months post-transplant, on the first day of initial presentation, the patient was admitted to an outside hospital after 7 days of bloody stools and a near syncopal episode. He was hemodynamically stable. Initial CT, tagged RBC study, and small bowel enteroscopy were negative. On the second day, the patient had several bloody stools with significant hypotension; he was given 5u RBC, 1u FFP, and 1u cryoprecipitate and transferred to another hospital, where his hemoglobin was 2.6 g/dL. Massive transfusion protocol was initiated, and patient received 38u RBC, 16u FFP, 2u platelets, and 2u cryoprecipitate. He was intubated and found in shock requiring multiple pressors. CTA revealed active bleed within the small bowel loops adjacent to the right common iliac artery, suggestive of an AE fistula. Due to ongoing hemodynamic instability, despite high risk of pancreatic necrosis, the patient underwent IR stenting of the fistulous tract on the third day and was transferred to our hospital. At our hospital, the patient was taken to the OR as level 1. The mucosa of the graft duodenum encircled the right common iliac artery and a 1cm anterior wall defect was found distal to the donor Y graft implantation. The pancreas graft was ischemic. Graft pancreatectomy was performed and the bowel was left in discontinuity. On post-operative day (POD) 2, patient returned to the OR; the right common iliac artery was repaired with an interposition graft using donor iliac artery and the small bowel was anastomosed. The patient was extubated on POD 3. The NG tube was removed on POD 4 and diet was advanced. The patient was transferred to floor by POD 7 and discharged by POD 14 with a stable hemoglobin trend and on antimicrobial regimen and intermittent hemodialysis.

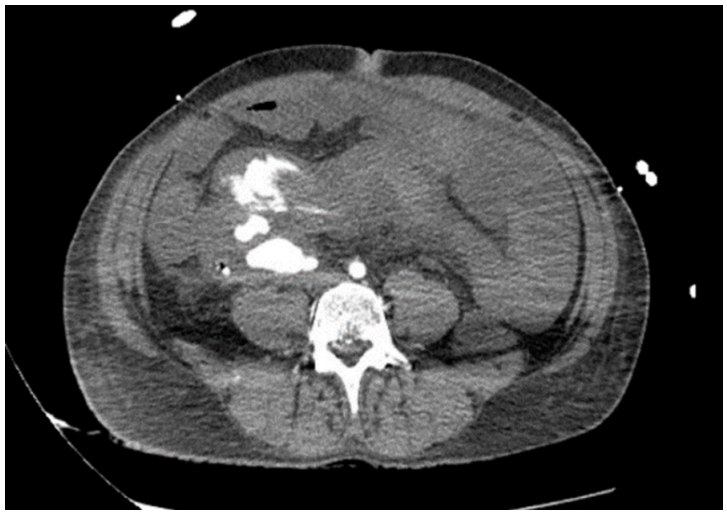
Conclusion: This is a rare case report of a near-fatal AE fistula after SPK transplant where the hemodynamic instability continued despite IR intervention and was successfully rescued by surgical repair. IR stenting seemed to control the bleed, but it also likely compromised perfusion to the vulnerable graft and caused pancreatic necrosis, resulting in significant inflammatory response that required continued pressor support. While a few prior case reports recommend endovascular management of AE fistula, our case supports surgical intervention for severe clinical presentations. Prompt surgery allows for visual inspection of the pancreas graft in addition to definitive repair of complex fistulous connection. This case also further exemplifies variable pathophysiology and clinical presentation of post-pancreas transplant AE fistula. Since our patient's initial acute graft rejection was well managed, the mechanical trauma from pancreas biopsy may have contributed to AE fistula creation. Similar to prior reports, this case demonstrates how unpredictable the degree of GI bleed can be from an AE fistula and how it can be

missed by multiple diagnostic tools. Therefore, despite its rare occurrence, it is crucial to include AE fistula in the differential for post-pancreas transplant patients presenting with GI bleed.

References: 1. Significant arterial complications after pancreas transplantation-A single-center experience and review of literature. Clin Transplant, 2017. 31(10)

2. Successful Bridge Therapy with Initial Endovascular Repair for Arterioenteric Fistula Resulting from Pseudoaneurysm Rupture with Massive Gastrointestinal Hemorrhage after Pancreas Transplantation. Ann Vasc Surg, 2019. 58: p379 e15-379 e22 3. Late complications of pancreas transplant. World J Transplant, 2020. 10(12): p404-414 4. Anterio-colonic Fistula as a Complication of Failed Pancreatic Transplant. Int J Surg Res Pract, 2018. 5(2) 5. Critical presentation and management of an acute gastrointestinal haemorrhage as a result of an arterioenteric fistula secondary to a failed pancreatic allograft. BMJ Case Rep, 2021. 14(7)





Critical Care - 60 Post-pneumonectomy Respiratory Failure Successfully Managed with Veno-Venous ECMO

Kyle Gronbeck[1], Erin Etoll[1]

[1]University of Nebraska Medical Center, Omaha, NE

Introduction: A pneumonectomy is an operation that is intended to remove one lung, typically secondary to lung cancers such as non-small cell lung cancer (NSCLC).1 Pneumonectomy carries a significant perioperative mortality risk secondary to pulmonary complications.2 Extracorporeal membrane oxygenation (ECMO) is a form of life support that has been proven to be beneficial when conventional mechanical ventilation fails.3 ECMO has been utilized to improve outcomes in post-pneumonectomy patients whose post-operative course is complicated by acute respiratory failure of the remaining lung.4 ECMO provides better lung protection for severe post-pneumonectomy noncardiogenic respiratory failure and improves overall mortality.5 We present a case of post-pneumonectomy respiratory failure successfully treated with femoral-femoral veno-venous (VV) ECMO transitioned to single site dual lumen VV ECMO.

Methods: The patient is a 60-year-old female with a history of NSCLC who initially presented for a scheduled pneumonectomy. In the immediate post-operative period, the patient went into profound respiratory failure secondary to superimposed infection and post pneumonectomy pulmonary edema. She failed conventional mechanical ventilation and was initiated on femoral-femoral VV ECMO on hospital day four. Her femoral-femoral ECMO sites were switched out for a single site dual lumen cannula in the right internal jugular vein on hospital day eight to allow for participation in physical therapy. A tracheostomy and percutaneous gastrostomy tube were placed on hospital day thirteen. An echocardiogram performed during her VV ECMO run showed evidence of severe pulmonary hypertension with pulmonary arterial pressures as high as 70 mmHG. Her oxygenation and hemodynamics were managed with inhaled epoprostenol, and her hypotension was treated with epinephrine and vasopressin infusions. She was slowly weaned from VV ECMO and decannulated on hospital day forty-one. In total she spent thirty-seven days on ECMO. On hospital day fifty-two she was discharged to a long-term acute care hospital for a slow ventilator wean and rehabilitation.

Conclusion: This case represents successful treatment of post pneumonectomy respiratory failure utilizing VV ECMO. Failure of conventional mechanical ventilation was recognized early, before increasing ventilator settings could cause further damage to the remaining lung. Patients with one lung ventilation are particularly susceptible to mechanical stress caused by mechanical ventilation and carry a significant mortality risk.6 Early recognition to identify which patients ECMO may benefit is crucial to allow for early lung protection. Recognizing patients that will benefit from ECMO who have post-pneumonectomy respiratory failure is only the first step in management. This case also highlights early tracheostomy during ECMO. Tracheostomy in non-transplant surgical patients has been proposed to avoid complications related to mechanical ventilation, sedation and immobilization and may lead to improved outcomes.7 There are mixed results in patients undergoing tracheostomy while on ECMO, but it has been consistently proven to be safe and reduces morbidity. If longer ECMO runs are anticipated, our practice is to move to single site dual lumen ECMO cannulation with tracheostomy and an emphasis on early involved physical therapy. Another intriguing aspect of this case is the patient's development of pulmonary hypertension post-operatively. Post pneumonectomy pulmonary hypertension (PH) has been reported in the literature.8 PH in these patients can be partially explained by vascular changes caused by altered pulmonary blood flow. Pulmonary arterial pressures are typically only moderately elevated but carry an increased risk of poor outcomes. In our case PH was managed by supporting right ventricular function, utilizing vasopressin for hypotension, aggressive diuresis, and inhaled pulmonary vasodilators. Post-pneumonectomy ECMO is not well represented in the literature but has been reported in published case reports. We present a unique case of post-pneumonectomy respiratory failure managed with single site dual lumen ECMO complicated by pulmonary hypertension. These patients can be challenging to manage but with the popularity of EMCO on the rise, more patients can be successfully managed than previous.

References: Graham and the First Pneumonectomy. 1984;251(2) An analysis, systematic review, and meta-analysis of the perioperative mortality after neoadjuvant therapy and pneumonectomy for non–small cell lung cancer. Volume 143, Issue 1, 2012. Extracorporeal Membrane Oxygenation in Adult Acute Respiratory Distress Syndrome. Volume 27, Issue 3. Veno-venous ECMO in ARDS after post-traumatic pneumonectomy. 39, 2235–2236 (2013) Extracorporeal lung membrane provides better lung protection than conventional treatment for severe postpneumonectomy noncardiogenic acute respiratory distress syndrome. 2008 Jun;135(6):1362-71 Protective Ventilatory Approaches to One-Lung Ventilation: More than Reduction of Tidal Volume. Rep 4, 150–159 (2014) Tracheostomy as a bridge to spontaneous breathing and awake-ECMO in non-transplant surgical patients. 2017 May;19 Suppl 2:120-123 Vascular remodelling in the pulmonary circulation after major lung resection Aug 2017, 50 (2) 1700806

Critical Care - 61 Use of Volatile Anesthetics for the Management of Intractable Status Asthmaticus in the ICU - Logistics and Regulatory Challenges

Leslie A Vargas[1], Brian Sun Sou[2], Adam Smith[2], Gyorgy Frendl[2]

[1]Brigham and Women's Hospital, Boston,MA, [2]Brigham and Women's Hospital, Boston, MA

Introduction: Not Applicable - Medically Challenging Case Report

Methods: The use of volatile anesthetics as a therapeutic agent or as an alternative for intravenous sedation in the intensive care units (ICUs) is a relatively uncommon practice in the United States. This could be due to several factors. First, the need for anesthesia trained personnel for titration and monitoring the delivery of anesthetic gases outside of the operating room. Second, ICUs are not routinely engineered to deliver anesthetic gases safely. To do so there are multiple components needed: a vaporizer that is compatible with the ICU ventilator, a sampling line to reliably measure the inhaled and exhaled concentrations of the anesthetic gases and a scavenging system to appropriately dispose of the waste anesthetic gas. We present the case of a 46-year-old African American female with a past medical history of anxiety and asthma who presented to the emergency department with a severe asthma exacerbation. She was initially treated with maximal medical therapy and was eventually intubated for severe and refractory respiratory acidosis due to life-threatening status asthmaticus. Given the inability to ventilate the patient she was emergently cannulated for peripheral VV ECMO. Despite being on ECMO and continued maximum medical treatment there was no improvement in her ventilatory parameters. The decision was made to start the patient on inhaled isoflurane. An anesthesia machine was setup in the ICU room to deliver isoflurane at 1% and provide ventilatory support. After 24 hours of initiation of isoflurane, her ventilatory parameters gradually improved towards normal. The patient successfully passed a spontaneous breathing trial and was extubated the next day. Twenty-four hours after extubation, VV ECMO was weaned and later decannulated. There have been previous reports in the medical literature regarding the use of isoflurane as a therapeutic modality in cases of severe status asthmaticus. These reports, however, fail to convey the challenges these clinicians may encounter while attempting to coordinate the delivery of anesthetic gases in the ICU. The main challenges we faced involved: 1. The stressor of an unconventional situation to many team members who lacked training and familiarity with the use and delivery of volatile anesthetics (board certified anesthesiologists were on site). 2. Inappropriate systems for documenting the administration of isoflurane, the absence of a medical order for isoflurane, and subsequent billing issues (in EPIC). 3. Inability to measure end-tidal isoflurane concentrations (a standard of care in the operating room). 4. Finally, the lack of a standardized protocol at our institution to guide the overall delivery and management of the anesthetic gases in the ICU. We not only propose the proactive development of protocols to address the gap in care but most importantly we believe there is a place for volatile anesthetics in the ICU that should be further explored.

Critical Care - 62 Parainfluenza and COVID-19 myocarditis managed with VA ECMO

Chandni Ravi[1], Miguel Artzner[1], Amanda Arnzen[1]

[1]University of Nebraska Medical Center, Omaha, United States of America

Introduction: Fulminant myocarditis is sudden in onset and life-threatening with a 50% predicted mortality with conventional treatment [1]. Usual management is supportive and includes the administration of high-dose inotropic agents and pacing. Survival of patients in recent years have seen significant improvement owing to the use of mechanical circulatory devices [2]. We describe here a case of parainfluenza and COVID-19 myocarditis successfully managed with Veno-Arterial (VA) ECMO.

Methods: A 35-year-old male with no significant prior medical history presented with an acute onset shortness of breath and fever. Acute clinical decompensation shortly after admission required the initiation of vasopressors and broad-spectrum antibiotics. On hospital day 2, his echocardiogram demonstrated an left-ventricular ejection fraction (LVEF) of under 15% with a BNP of 16,165 pg/mL. His clinical status deteriorated and was significant for marked acidemia which required intubation. On hospital day 3, he sustained two pulseless-electrical-activity cardiac arrests with a total down time of 10 minutes prior to return of spontaneous circulation. Following this, he was peripherally cannulated for Veno-Arterial extra-corporeal membrane oxygenation (VA ECMO). Pertinent lab work included leukocytosis to 22 x 103/µL with 29% bands, lactic acidosis to 4.7 mmol/L and a procalcitonin of 222 ng/mL. A respiratory viral panel returned positive for parainfluenza virus 4 and COVID-19. His clinical course was complicated by rhabdomyolysis resulting in acute kidney injury requiring continuous veno-venous hemodialysis (CVVHD). Over the next few days, his vasopressors were able to be weaned off and he experienced renal recovery. A repeat echocardiogram during an ECMO turn-down study demonstrated drastic recovery with the LVEF measured at 60-65%. On day 6, He was successfully de-cannulated and subsequently extubated. A follow-up echo on day 14 demonstrated return of normal cardiac function. He was discharged to an acute rehab facility on day 15.

Conclusion: Fulminant viral myocarditis may be associated with rapidly progressive cardiovascular compromise leading to refractory shock or cardiac arrest. Mechanical circulatory support can greatly improve mortality by bridging to clinical recovery or transplantation. We have described a case of viral (parainfluenza and COVID-19) myocarditis successfully managed with VA ECMO.

References: 1) Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. European heart journal. 2013 Sep 1;34(33):2636-48. 2) Acute mechanical circulatory support for fulminant myocarditis complicated by cardiogenic shock. Journal of cardiovascular translational research. 2014 Mar;7(2):156-64.

Critical Care - 63 Treatment of severe infective endocarditis leads to Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS syndrome) following cardiac surgery: A case report

Luna Chaaban[1], Madison D Tenbarge[2], Tarek Elshazly[3], Francis T Lytle[4]

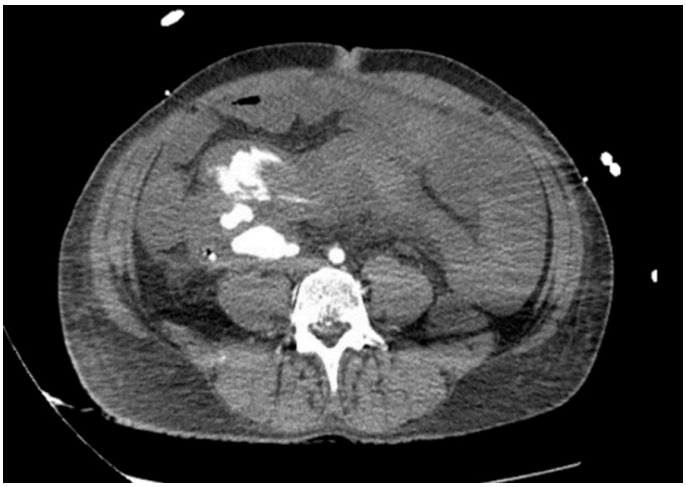
[1]University hospitals cleveland medical center, cleveland, United States of America, [2]Univesity hospitals Cleveland Medical Center, Cleveland, United States of America, [3]University hospitals Cleveland medical center, Cleveland, United States of America, [4]University Hospitals Cleveland Medical Center, Cleveland, OH

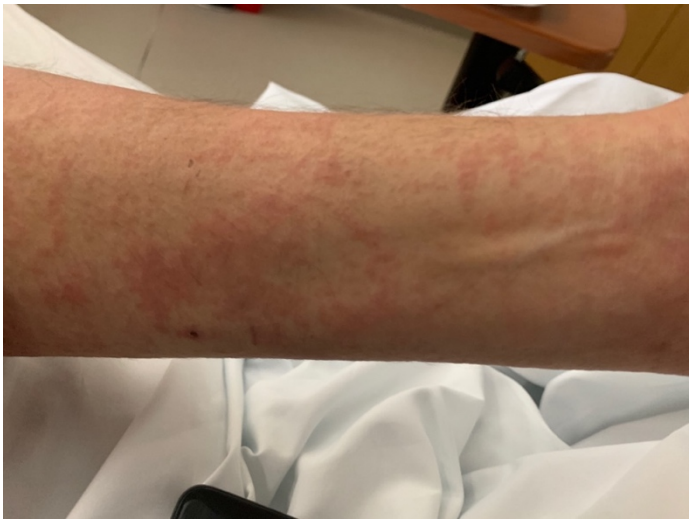
Introduction: Drug reaction with eosinophilia and systemic symptoms (DRESS syndrome) is a rare, life-threatening and often overlooked drug-induced reaction with a mortality rate as high as 10%, that appears 2 to 6 weeks after initiation of treatment [1][2]. It is mainly caused by anti-epileptics, and rarely by antibiotics [2]. It is a diagnosis of exclusion, however, the RegiSCAR scoring system can aid in posing the diagnosis of DRESS syndrome [1][2]. We present a rare case of antibiotic-induced DRESS syndrome following cardiac surgery for severe infective endocarditis (IE).

Methods: Case: 27YOM with history of IV drug use and HCV, who presented with severe AI and reduced EF (40%) in the setting of perforated leaflet causing severe regurgitation. Given the concern for IE and likely etiology of aortic root abscess, the patient was initiated on empiric antibiotic therapy with vancomycin and ceftriaxone; and subsequently underwent aortic valve replacement and aortic root reconstruction. His postoperative course was complicated by acute heart failure and complete heart block requiring permanent pacemaker placement. On POD 18, he developed a generalized pruritic maculopapular rash thought to be related to vancomycin that was discontinued. The rash initially improved but worsened again on POD 23 along with fevers and lymphadenopathy, AKI, transaminitis and eosinophilia which increased his RegiSCAR score from 0 to 5. Ceftriaxone was discontinued, and the patient was transitioned to Ciprofloxacin. The patient was treated with IV steroids with significant improvement of his rash and resolution of his eosinophilia, AKI, and liver dysfunction. He was then transitioned to an oral steroid taper regimen.

Conclusion: Antibiotics can rarely cause DRESS syndrome [2]. In our literature review, only 3 cases related to Ceftriaxone have been reported [3][2]. There is an increase in vancomycin-induced DRESS syndrome which may be attributable to its more frequent use as empiric therapy for many infections, most importantly in this case, infective endocarditis [1]. DRESS syndrome can severely affect multiple organs including the liver, the kidneys, heart, lungs and GI tract, making it a formidable complication of prolonged antibiotic therapy [1]. It is often a challenging diagnosis because of its variable cutaneous presentations and common systemic features with other etiologies, shock states especially in context of recent complicated cardiac surgery as in our case [2]. Treatment requires a multidisciplinary approach including dermatology and infectious diseases expertise and involves long-term steroids, a non-benign therapy, as well as immediate withdrawal of causing agent which can be difficult, as is this case where long term antibiotics are needed [1].

References: 1. Littlehales E et al. Vancomycin-Induced DRESS Syndrome: An Important Concern in Orthopedic Surgery. Case Rep Orthop. 2018 Jun;1439073. 2. Sharifzadeh S et al. Antibacterial antibiotic-induced drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome: a literature review. Eur J Clin Pharmacol. 2021 Mar; 77(3):275-289. 3. Guleria VS et al. Ceftriaxone induced drug rash with eosinophilia and systemic symptoms. J Res Pharm Pract. 2014 Apr;3(2):72-74.





Critical Care - 64 A Focal Neurologic Deficit in a Patient with Altered Mental Status: Rhabdomyolysis With Acute Compartment Syndrome and Acute Kidney Injury

Tiffany Otero[1], Erin Hennessey[2], Jocelyn Wang[3]

[1]Stanford Health Care, Palo Alto, CA, [2]Stanford University Medical Center, Palo Alto, CA, [3]Stanford, Palo Alto, CA

Introduction: Rhabdomyolysis (rhabdo) is a syndrome of skeletal muscle necrosis that results in the intracellular release of creatinine kinase (CK) and myoglobin into the peripheral circulation. The presence of these muscle proteins may lead to metabolic acidosis, acute kidney injury (AKI) and acute compartment syndrome (ACS). Rhabdo is diagnosed through laboratory studies, whereas ACS is a clinical diagnosis considered to be a surgical emergency. Pain is usually the earliest clinical symptom of ACS, whereas paralysis and pulselessness are late findings associated with irreversible injury. The recognition of ACS can be challenging in the critically ill, where patients are sedated or on infusions of neuromuscular blocking agents. We describe a case of rhabdo leading to ACS and AKI, where the initial clinical symptom was a focal neurologic deficit of the distal lower extremity (LE).

Methods: TT was a middle-aged black male with no known past medical history who was found down with altered mental status. Vital signs in the ED were stable; pH: 6.94, Co2: 115; potassium: 7.0, bicarbonate: 16, serum creatinine (SCr): 2.09, anion gap: 28; CK: 7,500; troponin:1.58; EKG: sinus, peaked T waves; urine toxicology positive for cocaine. Primary and secondary survey negative for traumatic injuries; head CT negative. TT was intubated for acute hypercapnic respiratory failure. In the ICU TT became hemodynamically unstable, and oliguric, requiring vasopressors, and fluid boluses. Continuous renal replacement therapy (CVVHDF) was started at 20mg/kg/hr. Pt initially required a propofol infusion and paralysis for vent desynchrony and hypoxemia. TT continued to have uptrending troponins with ST changes consistent with NSTEMI. Transthoracic ECHO was notable for a reduced left ventricular systolic function (ejection fraction = 35%).

On hospital day (HOD) 2 serum CK was above the reportable laboratory limit (22,000 unit(s)/L). CVVHDF clearance increased to 40mg/kg/hr. On physical exam, TT was found to have isolated right LE deficits not present on admission. A stat repeat head CT was negative for intracranial bleed. Vascular ultrasound of the right LE notable for a monophasic signal; left LE vasculature was triphasic. LE compartments were checked periodically and remained soft. Serial lactates were within normal limits.On HOD 3, CK remained >22,000 units/L. Manual evaluation of CK was 174,867 units/L. On physical exam, the right LE was tenser to palpation with dopplerable pulses. Orthopedic surgery was consulted and compartment pressures of the right leg were elevated prompting an emergent fasciotomy. Non-viable muscle tissue in the deep and superficial posterior compartments was removed. TT underwent serial irrigation and debridement procedures of the right LE during his ICU stay. TT was discharged to the medicine floor on HOD 11.

Conclusion: We describe a case of rhabdo in a critically ill patient whose clinical course was complicated by ACS and AKI, in the setting of an NSTEMI and a neurological exam that prompted a code stroke. Our patient highlights the challenges involved in diagnosing ACS in the ICU setting, as well as the significance of an ongoing elevated CK despite CVVHDF. The management of rhabdo involves reducing ongoing muscular destruction by identifying precipitating factors. Aggressive fluid resuscitation remains the mainstay treatment used to circumvent renal insult. The use of bicarbonate infusions to alkalize the urine to reduce tubular damage has also been described. Mannitol has also been proposed as a therapeutic agent for myoglobinuria as a free radical scavenger. The resolution of muscle injury is demonstrated by a down-trending serum CK. Therefore, CK levels that exceed the reportable laboratory limit may make clinical improvement difficult to ascertain. And while the manual evaluation of serum CK is possible, it is labor-intensive, with results that may be delayed. In this case, discerning the degree of ongoing muscular injury was impeded due to CK's that were not immediately quantifiable. Moreover, the significance of requiring increased dialysis clearance was not immediately recognized by the ICU team,

given renal replacement therapy is facilitated with the help of our nephrology colleagues. In sum, rhabdo is a serious medical condition that may result in multi-organ dysfunction. The prompt recognition and treatment of rhabdo is vitally important in the ICU setting.

References: Scharman EJ, et al. Prevention of kidney injury following rhabdomyolysis: a systematic review. Ann Pharmacother. 2013 Jan;47(1):90-105. doi: 10.1345/aph.1R215. Epub 2013 Jan 16. PMID: 23324509. Torres PA, et al. Rhabdomyolysis: pathogenesis, diagnosis, and treatment. Ochsner J. 2015;15(1):58-69. Somagutta MR, et al. Role of Bicarbonates and Mannitol in Rhabdomyolysis: A Comprehensive Review. Cureus. 2020;12(8):e9742. Published 2020 Aug 14. doi:10.7759/cureus.9742 Yiannis S. Chatzizisis, et al.The syndrome of rhabdomyolysis: Complications and treatment, European Journal of Internal Medicine,Volume 19, Issue 8, 2008, Pages 568-574, https://doi.org/10.1016/j.ejim.2007.06.037. West H, et al. Rhabdomyolysis associated with compartment syndrome resulting in acute renal failure. Eur J Emerg Med. 2007 Dec;14(6):368-70. doi: 10.1097/MEJ.0b013e32823a3c8b. PMID: 17968208.

Critical Care - 65 Use of Early Veno-Venous Extracorporeal Membrane Oxygenation in COVID-19 ARDS with Idiopathic Giant Bullous Emphysema

Patrick Ward[1], Shaun Thompson[1], Chandni Ravi[2], Elizabeth Mahal[1], Eric Asbe[1]

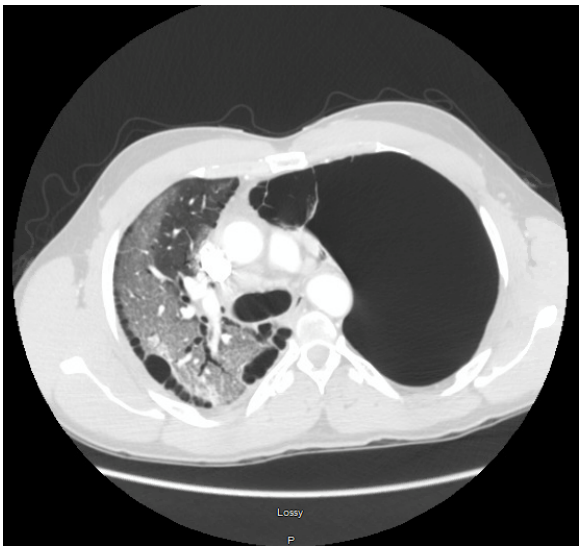
[1]University of Nebraska Medical Center, Omaha, NE, [2]University of Nebraska Medical Center, Omaha, United States of America

Introduction: Veno-venous extracorporeal membrane oxygenation (VV ECMO) has been a rescue measure of last resort for patients with refractory hypoxia/hypercapnia despite maximal medical therapy. VV ECMO allows for lung rest and recovery in patients with acute respiratory distress syndrome (ARDS) in order to avoid continued lung injury from mechanical ventilation. Cannulation for VV ECMO has increased during the COVID-19 pandemic with 8677 COVID-19 patients being cannulated as of August 20211. Of those cannulated for VV ECMO, approximately 74-76% of the patients had some sort of non-invasive ventilatory support and an average of 3.2 days of mechanical ventilation before cannulation1. Given the highly contagious nature of COVID-19 and vaccine hesitancy that is seen in the United States, more patients with co-morbid illness such as bullous emphysema and COPD, are presenting with severe COVID-19 illness1. Mechanical ventilation can prove to be difficult and, in some cases, may not be beneficial as further injury in the form of pneumothorax may occur. This can continue the spiral of lung injury that can lead to worsening ARDS from COVID-19 infection. In this scenario, a patient with vanishing lung syndrome presented with COVID-19 infection devolving to ARDS. Due to the risks of positive pressure ventilation in this patient with a significant bullae, the risk of intubation and mechanical ventilation was felt to be significant and could possibly lead to life threatening issues such as a pneumothorax and a bronchopleural fistula if a chest tube was inserted into this bullous area. Because of this, an awake VV ECMO cannulation was performed in an attempt to avoid mechanical ventilation and avoid positive pressure ventilation. This presents a unique scenario where standard protocols of mechanical ventilation with VV ECMO rescue for hypoxemia puts the patient at an increase risk from initiation of positive pressure ventilation.

Methods: The patient is a 44 year old male who was admitted with acute respiratory failure and sepsis secondary to COVID-19 Infection. Significant past medical history was pertinent for idiopathic bullous emphysema of his left upper lobe of his lung (Fig 1a and 1b). The patient presented to the emergency department with shortness of breath, fevers, chills, nausea, and vomiting 6 days after testing positive for COVID-19. The patient was unvaccinated. Upon admission, he was started on remdesivir and dexamethasone with supplemental oxygen as needed for treatment of his hypoxia. Unfortunately, on hospital day 5, he developed a significant increase in his oxygen requirement and the need for non-invasive or even invasive mechanical ventilation was deemed to be needed. Due to his significant bullous disease involving the left lung, avoidance of positive pressure ventilation was felt to be the safest route of treatment and deployment of VV ECMO would be beneficial prior to initiation of these ventilation modalities. After discussion with the patient and family, informed consent was obtained, and the patient was prepared for an awake cannulation onto VV ECMO. With the use of moderate sedation, the patient was successfully cannulated via the femoral veins and VV ECMO support was initiated. While cannulated he required only supplemental oxygen for comfort and required minimal to no sedation. The patient was able to participate in rehabilitation therapies despite bedrest secondary to VV ECMO cannulas in the groins. On cannulation day 7, the patient developed a spontaneous pneumothorax of his right lung likely from coughing. He was able to remain hemodynamically stable and also stable from a respiratory standpoint without increased oxygen needs or other ventilation assistance. A chest tube was placed under CT guidance with complete resolution of the pneumothorax. On day 10 of cannulation via the femoral veins, VV ECMO support was transitioned to right internal jugular vein via a dual lumen catheter. His clinical course improved and he was successfully decannulated from ECMO after 25 days of support. He was discharged to an acute rehabilitation center on hospital day 37 requiring two liters of supplemental oxygen.

Conclusion: This case presents an ECMO first strategy for respiratory failure in COVID-19 illness where positive pressure ventilation via non-invasive or invasive mechanical ventilation could have been deleterious

References: Extracoporeal Life Support Organization: ECMO in COVID-19: Full COVID-19 Registry Dashboard. 30 September 2021. Available from: https://www.elso.org/Registry/FullCOVID19RegistryDashboard.aspx Aziz, J. E., DellaVolpe, J., Aziz, S. & Sterling, R. An extracorporeal membrane oxygenation first strategy in COVID-19 acute respiratory distress syndrome. ASAIO Journal Publish Ahead of Print, (2021). Loyalka, P., Cheema, F. H., Rao, H., Rame, J. E. & Rajagopal, K. Early usage of extracorporeal membrane oxygenation in the absence of invasive mechanical ventilation to treat COVID-19-related hypoxemic respiratory failure. ASAIO Journal 67, 392,Äì394 (2021). Tang, J., Li, W., Jiang, F. & Wang, T. Successfully treatment of application awake extracorporeal membrane oxygenation in critical COVID-19 patient: A case report.Journal of Cardiothoracic Surgery 15, (2020).



Critical Care - 66 The Domino Effect of a Gastrointestinal Bleed and Ornithine Transcarbamylase Deficiency

Jesse Calestine[1], Colin Sheehan[1]

[1]Geisinger Medical Center, Danville, PA

Introduction: Ornithine transcarbamylase deficiency (OTCD) is an x-linked urea cycle disorder affecting 1:14,000 people resulting in hyperammonemia (HA). Though fatal in male newborns, female gene expression varies 1. Literature reports OTCD HA complications of seizure, cerebral edema, and encephalopathy 2,3. There are reports of gastrointestinal bleeding (GIB) resulting in OTCD associated encephalopathy1. We highlight a case of OTCD who encountered a perfect storm of events (i.e. status epilepticus, encephalopathy and subacute cerebral infarcts) originating from a GIB.

Methods: 58 year old female with history of OTCD, ischemic stroke on plavix and remote post infarction seizures presents with coffee ground emesis and admitted for GIB. Endoscopy showed non bleeding gastric ulcer and cameron lesion despite recurrent emesis during hospital stay. Up trending ammonia precipitated altered mental status, hypoxia with mucus plugging and kussmaul respirations leading to intubation. Left upper extremity tremors along with electroencephalogram suggested focal motor status epilepticus. Mental status remained non responsive prompting a CT head which revealed new subacute multifocal infarcts without clear etiology. Catabolic state related to prolonged lack of oral access (i.e. delayed nutrition and oral antiepileptics), seizures, septic shock and hyperosmolar hyperglycemic state contributed to persistent HA. Ultimately the patient did not respond to ammonul and arginine therapy leading to continuous renal replacement therapy in the setting of oliguric acute renal failure. Mental status failed to recover, resulting in withdrawal of care.

Conclusion: An initial GIB insult led to HA sequelae in our patient with known OTCD. Catabolism was a main driver toward persistent elevated ammonia4. Our patient had a prolonged lack of oral access given endoscopic procedure and poor mental status limiting nutrition and medical therapy which could have been addressed with early use of parenteral therapies. Recurrent coffee ground emesis in the setting of non bleeding lesions may have been indicative of improperly cleared blood from the GI tract that could have been mitigated with an intensive bowel regimen. This patient had new subacute infarcts compared to admission imaging that could not be explained given lack of atrial fibrillation and structural disease on echocardiogram. Etiology may have been related to hyperinflammatory state secondary to SARS-COV-2 infection that was initially thought to be incidental5. Even if the infectious process was a contributor, management would be limited given contraindication to steroids in OTCD and remdesivir in renal failure. This case highlights the importance of precipitants to HA and emphasis on early management of these etiologies to decrease intensive care mortality.

References: Ornithine transcarbamylase deficiency unmasked because of gastrointestinal bleeding. J Clin Gastroenterol. 2001 Severe Hyperammonemia in Late-Onset Ornithine Transcarbamylase Deficiency Triggered by Steroid Administration", Case Reports in Neurological Medicine, vol. 2015, Article ID 453752, 3 pages Hemodialysis for hyperammonemia associated with ornithine transcarbamylase deficiency. The application of clinical genetics, July 2008, Pages 1-5 Hyperammonemia: What Urea-lly Need to Know: Case Report of Severe Noncirrhotic Hyperammonemic Encephalopathy and Review of the Literature. Case Rep Med. 2016;2016:8512721 Acute Ischemic Stroke and COVID-19. American Heart Association, February 2021, Stroke 52:905–912

Critical Care - 67 A "shocking" diagnosis: Acute pericardial tamponade from hemorrhagic conversion of a pre-existing pericardial effusion

MacGregor J Hodgson[1], Anne L Donovan[2]

[1]University of California, San Francisco, San Francisco, CA, [2]UCSF, San Francisco, CA

Introduction: Pericardial tamponade is a potentially devastating condition that develops rapidly due to abnormal fluid accumulation under pressure from a myriad of etiologies.1,2,3 In this case report, we describe the diagnosis and management of hemodynamic collapse from acute pericardial tamponade in a medically complicated, critically ill patient.

Methods: A 69-year-old man with a complex medical history was transferred to our ICU from the community for vasopressor support, mechanical ventilation, and continuous-renal-replacement-therapy (CRRT) in the setting of septic shock, volume overload, oliguric renal failure, and hypoxemic, hypercapnic respiratory failure. His history was specifically notable for Factor V Leiden complicated by recurrent deep vein thromboses (DVT) and pulmonary emboli. A transthoracic echocardiogram (TTE) early in his admission showed grossly normal left and right ventricular function as well as a small-to-moderate pericardial effusion that was not present on a TTE from six months prior. His initial ICU course was notable for fluid removal via CRRT, extubation to bilevel positive airway pressure, and S. epidermidis central line-associated bloodstream infection. Lower extremity Doppler ultrasound showed bilateral DVT, so he was restarted on a heparin infusion. Two days after re-initiation of heparin, the patient complained of abrupt onset and rapidly worsening dyspnea without associated chest pain. He quickly developed worsening shock requiring two vasopressors and new onset sinus tachycardia. His oxygen saturation was unchanged from baseline at 96% on 4 liters nasal cannula. His arterial blood pressure waveform showed exaggerated decreases in systolic pressure with inspiration. A bedside point-of-care cardiac ultrasound revealed evidence of diastolic chamber collapse with a larger pericardial effusion when compared with his admission TTE. His heparin infusion was paused and an emergent, bedside pericardiocentesis was performed. Approximately 1.1L of blood-tinged serous fluid was drained from the pericardium. The patient's hemodynamics and symptoms rapidly improved to their baseline. All pericardial fluid lab studies were negative except for the presence of non-specific inflammatory cells, blood, and bacterial culture positivity for S. epidermidis. His pericardial drain was removed after six days with repeat focused TTE showing resolution of the effusion; he was then successfully restarted on a heparin infusion with no further complications. He was discharged from the ICU back to his referring facility two weeks later due to need for continued management and care coordination of multiple other comorbidities.

Conclusion: The progression of pericardial effusion to cardiac tamponade depends on the rate and volume of fluid collection. As little as 150mL of rapid fluid buildup can cause tamponade, whereas up to 2L of slow collection may not lead to tamponade.4 Our patient's initial drainage of 1.1L likely reflected an acute-on-chronic process given his known pericardial effusion. Given our patient's medical complexity, it was important to maintain a simple approach focused on the five main classifications of shock: distributive, cardiogenic, hypovolemic, obstructive, and mixed/unknown. Our patient had risk factors that raised the pre-test probability for any one of these domains; however, the key initial diagnostic step was recognition of the brisk tempo of his decompensation. The immediacy of his shock points to a newly depressed cardiac output which elevates obstructive or cardiogenic shock to the top of the differential, as cardiac output depression is typically a late finding of distributive or hypovolemic shock. It was also equally as important to examine the arterial waveform changes. In cardiac tamponade, although pulsus paradoxus reported incidence varies widely from 12-75% of cases, the sensitivity of this finding exceeds 80% and is higher than any other single physical finding.5,6 The presence of pulsus paradoxus greater than 10 mmHg increases the likelihood of tamponade by 3.3-fold, while its absence greatly lowers but does not eliminate the possibility, thus highlighting the importance of bedside ultrasound.7 Major echocardiographic signs of cardiac tamponade involve the presence of any chamber collapse (sensitivity and specificity of 90 and 65%, respectively), respiratory

variation in volumes and flows, as well as inferior vena cava plethora.8

References: Acute pericardial disease: approach to the aetiologic diagnosis.2004; 90(3):252-4 Spectrum of hemodynamic changes in cardiac tamponade.1990; 66(2):1487-91 Acute cardiac tamponade.2003; 249(7):284-90 Constrictive pericarditis as the first sign of lung cancer.2006 Nov; 42(11):608-610 Acute pericarditis: Current Concepts and Practice.2003;289(9):1150 Does this patient with a pericardial effusion have cardiac tamponade?2007;297(16):1810 Pulsus paradoxus: definition and relation to the severity of cardiac tamponade.1988;115(2):391 Correlation between clinical and Doppler echocardiographic findings in patients with moderate and large pericardial effusion: Implications for cardiac tampoande.1999;138(4):759

Critical Care - 68 ECMO Cannula-Associated Inferior Vena Cava Thromboembolism Leading to Pulmonary Emboli and Abscess While on Therapeutic Anticoagulation

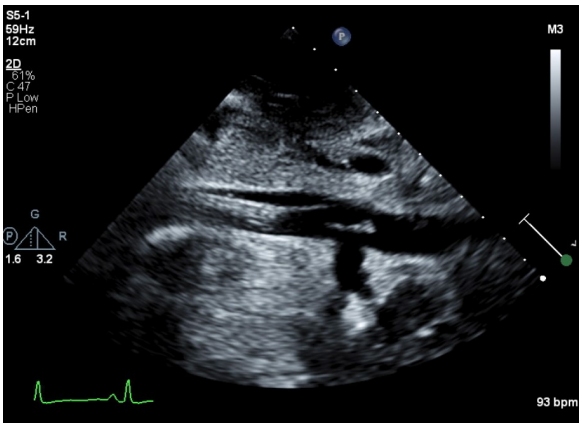
James M Urness[1], George Frendl[2]

[1]Brigham and Women's Hospital, Boston, MA, [2]Brigham and Women's Hospital, Harvard Medical School, Boston, MA

Introduction: Not Applicable - Medically Challenging Case Report

Methods: Cannula-associated thrombus involving the inferior vena cava after venous-venous extracorporeal membrane oxygenation in the setting of therapeutic anticoagulation is an infrequently reported complication. A 34 year old female with history of bronchiectasis and group 3 pulmonary hypertension on home oxygen presented with progressive functional decline and worsening respiratory decompensation. She had refractory hypoxemic and hypercarbic respiratory failure during admission leading to initiation of venous-venous extracorporeal membrane oxygenation (ECMO) utilizing the right internal jugular and femoral veins as a bridge for transplant. The patient had an uncomplicated bilateral lung transplant performed with consecutive decannulation of ECMO after a total of 70 days of support at the conclusion of the case. The patient was hemodynamically stable and extubated on supplemental oxygen following the transplant. On post-operative day 4 (both post-transplant and post-ECMO cannula removal), upon routine bedside ultrasound a large mobile linear thrombus was visualized in the inferior vena cava and the patient was started on therapeutic heparin infusion. The following day, the patient went into acute respiratory failure requiring reintubation and segmental pulmonary emboli were found in the left lower lobe and lingula on computed tomography eventually leading to left lung abscess, requiring prolonged antibiotic therapy and mechanical ventilatory support. The patient received a tracheostomy with a slow wean from the ventilator and was successfully discharged to rehab 44 days after transplant and was discharged home 17 days later functioning independently. The use of ECMO support is complicated by a high risk of thromboembolisms directly correlated to length of therapy, although anticoagulation reduces this risk, residual thrombus from the cannula should still be considered after removal. At the time of decannulation the inferior vena cava lumen can easily be investigated by bedside ultrasound or intraoperative transesophageal echography for any retained thrombus or fibrin sheath. Early detection of a cannula-associated thrombus can prevent further morbidity or mortality in an already fragile patient population.

Conclusion: Not Applicable - Medically Challenging Case Report



Critical Care - 69 Perioperative VV-ECMO for Management of Severe Esophageal and Tracheal Injury from Liquid Drain Cleaner

Ian Paterson[1], Connor McNamara[2]

[1]University Hospitals Cleveland Medical Center, Cleveland, OH, [2]University Hospitals, Cleveland, OH

Introduction: Ingestion of caustic alkalis such as household cleaning products is a method of attempted suicide with often disastrous consequences. GI tract damage can be extensive and involve surrounding structures. Tracheo-esophageal fistula (TEF) is a rare but recognized complication. Here we describe a patient who developed TEF and required ECMO to facilitate thoracotomy with esophagectomy and tracheal defect repair.

Methods: A 31 year old female with a history of depression and polysubstance abuse presented to the ED after a suicide attempt by ingestion of ¬º gallon of liquid drain cleaner. She had burns to her mouth, tongue, and lips and was drooling. She was intubated for airway protection and transferred to our Surgical ICU. CT showed diffuse wall thickening consistent with esophagitis, gastritis, and small bowel inflammation without free fluid or free air. In addition, she had evidence of tracheobronchial aspiration with subsequent lung injury. Thoracic surgery was consulted and initial management was conservative with maintenance of intubation and sedation, careful hemodynamic monitoring, strict NPO, and broad spectrum antibiotics. On ICU Day 4 the patient self-extubated and the airway was then secured with tracheostomy by ENT in the OR due to extensive ulceration and necrosis of the oropharynx. On ICU Day 9 bilious material was suctioned from the trach and CT findings were concerning for TEF. She went to the OR for bronchoscopy and EGD. Oral endotracheal intubation with a 6.5 armored ETT was performed using video laryngoscope. The trach cuff was deflated, a pediatric fiberoptic scope advanced past the tracheal defect and into the left mainstem bronchus, ETT passed over the bronchoscope and positioned distal to the defect and the cuff inflated. Multiple attempts at one lung ventilation resulted in desaturation. It was clear that the patient would not tolerate one lung ventilation, much less the apneic episodes required for repair of the airway defect. It was then decided to place the patient on VV-ECMO. 4L of flow was sufficient to supplement ventilation and facilitate the surgery. Due to extensive full thickness necrosis throughout the upper GI tract, the patient underwent esophagectomy, total gastrectomy, and duodenal resection of D1 as well as tracheal defect repair with intercostal muscle flap. The patient returned to ICU in stable but critical condition. ECMO was weaned and decannulated the next day.

Conclusion: Caustic ingestion causes mucosal injury via liquefactive necrosis with deep penetration into tissues and risk of full thickness injury (1). Damage begins within minutes and continues for 4-7 days, and perforated viscus remains a risk for up to 3 weeks (1). Surgery is generally reserved until a change in clinical condition necessitates exploration and intervention (2). TEF after caustic ingestion is a rare complication with two possible mechanisms, namely aspiration and necrotic extension from the esophagus, with the latter being more common (3). Lesions tend to occur on the left posterior wall of the trachea and/or the proximal left main bronchus, representing the areas of anatomic contact with or proximity to the esophagus (3). Perioperative management of the airway and ventilation can be challenging due to severe damage to the oropharynx, the need to avoid positive pressure ventilation in TEF, and associated respiratory complications such as pneumonia, bronchiolitis, and ARDS. Long and complex surgeries such as thoracotomy, esophagectomy, and TEF repair, especially those requiring periods of apnea, are often impossible due to airway and ventilation issues (3,4). In the past the only option would have been cardiopulmonary bypass, with its associated pulmonary complications as well as the undesirable risks of high dose systemic anticoagulation. Here we describe the use of VV-ECMO to facilitate complex apneic surgery that would have been otherwise impossible. Several recent case reports also describe similar use of ECMO in surgeries such as TEF repair and esophagectomy (4). With the increasing availability of ECMO and its expanding list of potential applications we are likely to see it used more frequently in difficult airway and ventilation situations such as TEF.

References: 1. World J Gastrointest Pharmacol Ther 2017 May 6; 8(2): 90-98 2. World J Gastroenterol 2013 July 7; 19(25): 3918-3930 3. J Thorac Cardiovasc Surg 1992 Mar;103(3):412-3 4. J Thorac Dis 2017; 9(9):E735-E738

Critical Care - 70 The Management of an Aortoenteric Fistula using a Sengstaken Blakemore Tube

Klint J Smart[1], Thomas A Nahass[2], Brian Wengerter[3], Mayur Narayan[4]

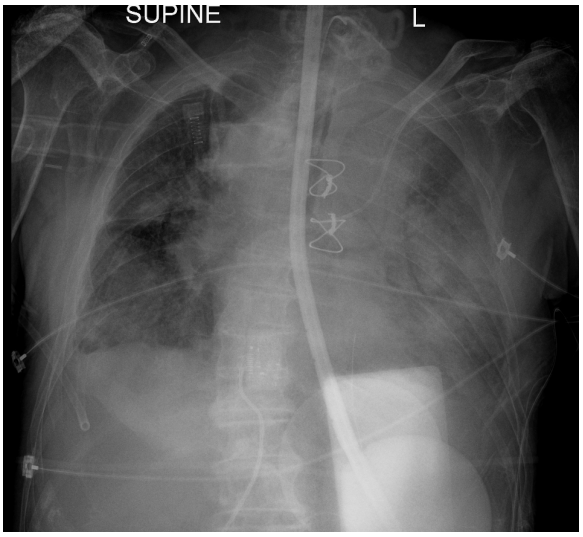
[1]Weill Cornell Medical Center/New York Presbyterian, New York, NY, [2]Memorial Sloan Kettering Cancer Center, New York, NY, [3]Weill Cornell Medical Center/ New York Presbyterian Hospital, New York , NY, [4]Weill Cornell Medical Center/New York Presbyterian Hospital, New York, NY

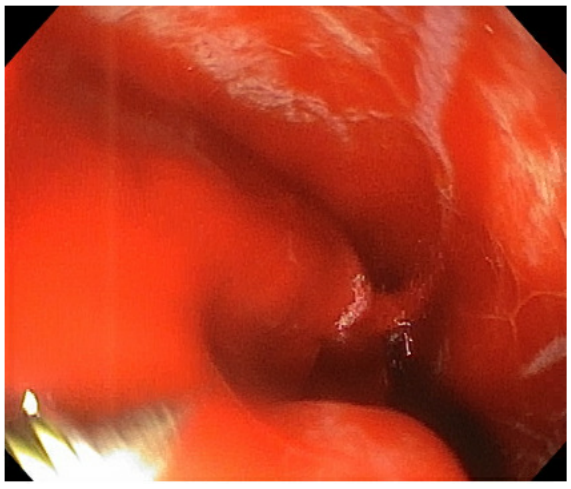
Introduction: An aortoenteric fistula (AEF) is a rare but life-threatening abnormal connection between the aorta and gastrointestinal (GI) tract. These fistulae can be classified into primary and secondary AEFs based on the etiology. Primary AEFs are rare and arise de novo between the aorta and the GI tract. The causes of primary AEFs include aneurysm, foreign body, tumor, radiation therapy and infection. Secondary AEFs occur after aortic reconstruction and other aortic procedures. This is an important differential for upper GI bleeding since the overall mortality rate is nearly 100 percent if it is misdiagnosed. We present the case of a 76 year-old man who suffered cardiac arrest from acute massive bleeding from an aortoesophageal fistula 13 weeks after he underwent resection of a multinodular goiter. This case describes the management of the GI bleeding with cardiopulmonary resuscitation, a Sengstaken-Blakemore tube and massive blood product transfusions.

Methods: A 76-year-old man was admitted to the intensive care unit (ICU) after suffering cardiac arrest secondary to hypoxia respiratory failure. At that time, return of spontaneous circulation (ROSC) was achieved after cardiopulmonary resuscitation (CPR) and emergent cricothyroidotomy. On computer tomography (CT) scan he was found to have a large substernal multinodular goiter with tracheal compression. As a result, he underwent a sternotomy, total thyroidectomy, along with an IVC filter placement. His past medical history was significant for a pulmonary embolism (PE) and hyperthyroidism due to a multinodular goiter. A right-sided aortic arch with aberrant left subclavian artery coursing behind the esophagus was incidentally noted on CT angiogram in August 2021 for PE evaluation. His postoperative course was complicated by chronic respiratory failure with mechanical ventilatory dependence, pneumonia, acute kidney injury and sepsis. On week 13 of his admission, he had an episode of acute massive hematemesis followed by cardiac arrest. ROSC was achieved after 30 minutes of CPR and massive transfusion of blood products. During that time, emergent bronchoscopy and laryngoscopy showed no source bleeding in the upper and lower airways. Apart from the administration of 1g of tranexamic acid, no anticoagulant reversal was administered since he received subcutaneous Enoxaparin 12 hours prior to this event. Octreotide was administered and a pantoprazole drip was initiated. He also required high dose vasopressor support with vasopressin, norepinephrine and phenylephrine infusions. A Sengstaken Blakemore tube was placed in the esophagus and inflated by the surgical team after which the hematemesis stopped. The initial attempt to perform an Esophagogastroduodenoscopy (EGD) was aborted due to massive bleeding, hypotension then cardiac arrest with ROSC after 10 mins of CPR. After adequate resuscitation, the EGD was successfully performed with the Sengstaken Blakemore tube deflated. The EGD showed active pulsatile bleeding about 25 cm from the incisors on the posterior wall of the esophagus. The balloon was re-inflated and the patient remained hemodynamically stable thereafter. Vascular surgery and cardiothoracic surgery specialties were consulted; however, no operative intervention was offered given the patient's poor prognosis and Glasgow coma scale of 3. After discussion with his family, they expressed wishes to not resuscitate him further or perform any surgical interventions and they requested to withdaw all life saving therapies. The Blakemore tube was subsequently deflated and the patient expired under confort measures.

Conclusion: It is essential to have a high index of clinical suspicion for an AEF in any patient presenting with hematemesis or massive upper gastrointestinal bleeding. The overall prognosis and outcome in patients who develop AEFs depend of the speed at which the AEF is diagnosed, the anatomical site of the aorta invloved and the patient's comorbidities. Delays in diagnosis or treatment will result in a high mortality rate.

References: Primary Aortoenteric Fistula after Nissen Fundoplication. Cureus. 2018;10(3):e2386
Aortoenteric fistula: Recognition and management. UptoDate. 2020.





Critical Care - 71 Disseminated Meningococcemia resulting in Myocarditis, Cardiogenic Shock, Thrombogenic Vasculitis and Disseminated Intravascular Coagulation

Klint J Smart[1], Ankur Srivastava[2]

[1]Weill Cornell Medical Center/New York Presbyterian, New York, NY, [2]New York Presbyterian Weil Cornell Medical Center, New York, NY

Introduction: Acute meningococcal disease is a life-threatening systemic infection caused by the bacteria Neisseria Meningitidis. It typically manifests by three syndromes: meningitis, meningitis with accompanying meningococcemia, or meningococcemia without clinical evidence of meningitis. Meningococcemia may have devastating clinical consequences hence it requires prompt identification and appropriate treatment. Severe meningococcemia may precipitate hypotension, disseminated intravascular coagulation (DIC), multi-organ failure as well as immune complex associated complications. Also, more than 50 percent of the mortality from meningococcemia have varying degrees of myocarditis seen on postmortem examination. We present the case of a 44 year-old man with no significant past medical history who developed disseminated meningococcal infection and cardiogenic shock requiring Veno-Arterial extracorporeal membrane oxygenation (ECMO).

Methods: 44 year-old man with no significant past medical history presented to an outside hospital with 2 days of night sweats, fever, fatigue and chills. He developed deteriorating mental status and hypoxic respiratory failure requiring intubation. Chest CT scan showed multifocal pneumonia. COVID PCR testing was negative. Respiratory viral panel and blood cultures were sent. Broad spectrum antibiotics (Vancomycin and piperacillin/tazobactam) were initiated. However, patient's hypoxic respiratory failure and hypotension continued to worsen. Bedside TTE showed severe left ventricular failure resulting in initiation of inotropes and consult for mechanical circulatory support. At this time, cardiogenic shock team was consulted and based on the presumed diagnosis of myocarditis with failing medical management, young age and stable neuro status decision was made to place the patient on Veno-Arterial ECMO. Further evidence of cardiogenic shock was reflected by pale, cold extremities, low cardiac output, blood lactate level of 8 mmol/L and brain natriuretic peptide level of 20,000 pg/mL. He also developed oliguric renal failure requiring continuous veno-venous hemodialysis. Left femoral veno-arterial ECMO was initiated with a flow of 4 L/min and he was transferred to our hospital for further management. He underwent extensive infective, autoimmune and heart failure workup. His course was complicated by multi-organ dysfunction and diffuse purpura fulminans confirmed by a punch biopsy. Next day, blood cultures were positive for Neisseria Meningitidis, so ceftriaxone was started for improved CNS penetration. This made our diagnosis likely myocarditis secondary to meningococcal infection. He continued to develop progressive thrombogenic vasculitis of his upper and lower extremities resulting in diffuse limb ischemia and mottling. Over the course of 3 days since admission he rapidly deteriorated clinically and had poor neurological function. After discussion with his family, the decision was made to withdraw lifesaving therapies and he subsequently expired under comfort care measures.

Conclusion: Meningococcal infections can have various clinical manifestations from fevers and bacteremia to fulminant disease with death occurring within hours of the onset of symptoms. It is important to identify this organism and initiate prompt treatment which includes antibiotics and systemic support.

References: 1. Meningococcemia in Adults: A Review of the Literature. (2016). Intern Med;55(6):567-72 2. Clinical manifestations of meningococcal infection. (2021). UptoDate.

Critical Care - 72 Rumpel-Leede phenomenon in a patient with hepatic abscess post liver transplant

Bryan L Benson[1], Avneep Aggarwal[1]

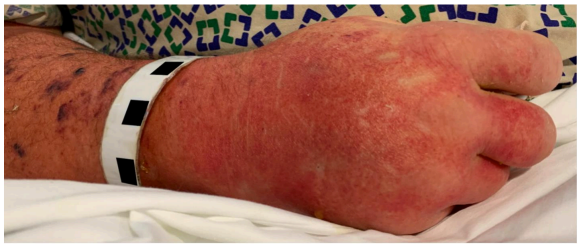
[1]Cleveland Clinic Foundation, Cleveland, OH

Introduction: Rumpel-Leede phenomenon refers to a petechial purpuric rash caused by extravasation of blood from dermal capillaries. This is induced by capillary hypertension through tourniqueting or the use of noninvasive blood pressure monitoring, and occurs in states of microvascular fragility and bleeding diathesis. First described by Rumpel in 1909 and Leede in 1911 as a diagnostic test for Scarlet Fever using a tourniquet, it has since been linked with diabetes, hypertension, thrombocytopenia and noninvasive blood pressure monitoring. We describe a case of Rumpel-Leede phenomenon in a patient presenting with a hepatic abscess post liver transplantation.

Methods: A 53 year old male was hospitalized in the surgical intensive care unit for hepatic abscess after liver transplantation. He had a history of hypertension, cirrhosis, and perforated gastric ulcer. He was in septic shock on presentation requiring vasopressor support and was being treated with broad spectrum antibiotics after sending blood cultures. His antibiotic regimen included daptomycin, meropenem, micafungin, ciprofloxacin, rifaximin, and pentamidine. Lab work was significant for Platelets of 26, BUN of 104, INR 1.6, aPTT 58, procalcitonin 13, CRP of 28. After few days in ICU, he developed a rash on his left upper limb which mimicked a drug reaction, but was restricted only to the left hand (see photograph). Medications were reviewed and no precipitant was identified. Dermatology was consulted and opined that this most likely represented Rumpel-Leede phenomenon and needed no specific treatment. The rash closely mimicked a morbilliform drug rash, but was distinguished by a typically clear demarcation distal to the tourniquet, unilateral distribution, and the fact that it was non-blanching. Over a course of few days, the rash became better with limb elevation and avoidance of tourniquets on the affected limb.

Conclusion: Rumpel-Leede phenomenon can easily be induced by tourniqueting of the arm as is commonly done when drawing blood cultures, and multiple factors common in critically ill patients such as thrombocytopenia, coagulopathy, and microvascular fragility make its appearance much more likely. The same set of clinical circumstances also necessitate initiation or escalation of antibiotic therapy. Knowledge of this phenomenon and its characteristics which distinguish it from drug rash can prevent unnecessary discontinuations or modifications of antimicrobial therapy and unnecessary investigations. Individual case reports are a valuable means for highlighting this phenomenon and encouraging one to consider it in the differential diagnosis.

References: 1: Stobbe H, Rürup C. Zum Nachweis von Kapillarschäden im Rahmen der Mikroangiopathie-Diagnostik beim Diabetes mellitus mittels Stauversuchs [Determination of capillary lesions during diagnosis by means of the tourniquet test of microangiopathies in diabetes mellitus]. Schweiz Med Wochenschr. 1979;109(46):1808-1810. 2: Varela D, Tran D, Ngamdu KS, Trullender B, Mukherjee D, Abbas A. Rumpel-Leede phenomenon presenting as a hypertensive urgency. Proc (Bayl Univ Med Cent). 2016;29(2):200-201. 3: Hess AF. The blood and the blood vessels in hemophilia and other hemorrhagic diseases. Arch Intern Med (Chic). 1916;XVII(2):203–220. 4: White WB. The Rumpel-Leede sign associated with a noninvasive ambulatory blood pressure monitor. JAMA. 1985;253(12):1724. 5: Nguyen TA, Garcia D, Wang AS, Friedlander SF, Krakowski AC. Rumpel-Leede Phenomenon Associated With Tourniquet-like Forces of Baby Carriers in Otherwise Healthy Infants: Baby Carrier Purpura. JAMA Dermatol. 2016;152(6):728-730.



Critical Care - 73 Acute coronary syndrome with cardiogenic shock in acute lymphoblastic leukemia with severe baseline thrombocytopenia

Lyubov Tsytsikova[1], Daniela Lazea[1]

[1]Brigham and Women's Hospital, Boston, MA

Introduction: Acquired thrombocytopenia is a known phenomenon in patients with acute coronary syndrome (ACS) and has been shown to carry a significantly higher risk of 30-day mortality, as well as increase in both hemorrhagic and thrombotic events (1). However, the implications of baseline thrombocytopenia in the setting of ACS is less well understood. Cancer patients are at risk of developing chronic thrombocytopenia secondary to hematologic etiology as well as certain chemotherapy regimens (2). Given that malignancy is also a known risk factor for acute coronary events, it is important to address the incidence and management of ACS in cancer patients with baseline thrombocytopenia. In this case report, a leukemia patient with severe baseline thrombocytopenia develops myocardial infarction with cardiogenic shock, for which cardiac catheterization is not pursued.

Methods: A 53-year old male with a history of B-cell acute lymphoblastic leukemia, status-post multiple chemotherapy regimens, radiation, cervical spine debulking, and allogeneic stem cell transplant, is found to have progressive disease to multiple organs, and thus admitted for potential CAR-T cell therapy. This was delayed due to altered mental status and question of ongoing infectious process given leukocytosis. CT chest and abdomen showed bilateral peribronchial opacities, sigmoid thickening, and moderate ascites, for which the patient underwent 3L paracentesis. He was started on broad intravenous antibiotics and antifungals, however then became leukopenic and progressively more thrombocytopenic. Platelet transfusion was initiated but discontinued due to dyspnea and hypoxic respiratory failure for which the patient was intubated. He was admitted to ICU intubated but hemodynamically stable. Bedside TTE revealed normal biventricular function on ICU admission. Two hours after admission patient went into atrial fibrillation with RVR and progressive shock. The EKG revealed new ST elevation in anterior leads and hemodynamic status rapidly deteriorated; serum troponin was 4887 (from 691 a few hours prior). Bedside echocardiogram showed new global LV hypokinesis. Patient was diagnosed with STEMI, however cardiology recommended no coronary intervention given severe baseline thrombocytopenia (8000) and poor oncologic prognosis. Patient expired 1 hour later. Blood cultures drawn earlier that day ultimately resulted with Candida fungemia.

Conclusion: Baseline thrombocytopenia in the setting of ACS significantly increases rates of 1-year mortality (3), major adverse cardiac events after coronary angiography (4), and both ischemic and bleeding events after coronary intervention (5, 6). Whether the severity of the thrombocytopenia further worsens outcomes has yet to be clearly studied. There are no existing guidelines at this time in regards to the use of coronary intervention and dual antiplatelet therapy for patients with severe baseline thrombocytopenia who develop ACS.

References: 1. Oikonomou et al. (2016) The effect of in-hospital acquired thrombocytopenia on the outcome of patients with acute coronary syndromes: A systematic review and meta-analysis. Thrombosis Research; 147, p64-71. 2. Leader et al. (2021) Challenges and Advances in Managing Thrombocytopenic Cancer Patients. J Clin Med; 10(6), p1169. 3. Chao et al. (2021) Baseline thrombocytopenia in acute coronary syndrome: The lower, the worse. Int J Cardiology; 332, p1-7. 4. Yadav et al. (2016) Effect of Baseline Thrombocytopenia on Ischemic Outcomes in Patients With ACS Who Undergo PCI. Can J Cardiology; 32(2), p226-33. 5. Hakim et al. (2011) Impact of baseline thrombocytopenia on the early and late outcomes after STEMI treated with primary angioplasty: analysis from HORIZONS-AMI trial. Am Heart J; 161(2), p391-6. 6. Ayoub et al. (2018) Impact of Chronic Thrombocytopenia on In-Hospital Outcomes After PCI. JACC Card Inv; 11(18), p1862-8.

Critical Care - 74 Late Post-partum Eclampsia Presentation to Cardiac ICU

Ken Oba[1], Brent Kidd[1]

[1]University of Kansas Medical Center, Kansas City, KS

Introduction: Preeclampsia is a leading cause of maternal mortality worldwide, and preeclampsia and eclampsia are responsible for a combined 10-15% of direct maternal deaths [1,2]. Postpartum preeclampsia occurs within 48 hours to 6 weeks after delivery [2], while eclampsia occurs within one week of delivery approximately 90% of the time [3]. Our case highlights the importance of early inclusion of preeclampsia in differential of postpartum patients presenting with signs of heart failure.

Methods: A 25-year-old G2P2 PPD8 female presented to outside hospital with 2 days of right upper quadrant abdominal pain and difficulty breathing. She was normotensive throughout her pregnancy but developed proteinuria and delivered at 37 weeks gestational age via cesarean. Her blood pressure was 149/99mmHg and bilateral pleural effusions were found on CTA. Labs were notable for hemoglobin of 6.4g/dL and Pro BNP of 4750. She was started on furosemide for suspected postpartum decompensated cardiomyopathy. Patient was initiated on Bipap 60% FiO2 and transferred to our institution's cardiac ICU, where she required 100% FiO2 on BiPap and was emergently intubated. Of note, her systolic blood pressure was consistently between 180-220mmHg. High-risk obstetrics was consulted for concern for preeclampsia. Patient was started on magnesium and given labetalol. While magnesium bolus was infusing, patient had a witnessed seizure which was aborted with 4mg lorazepam. CT of the head and HELLP labs obtained were negative. Bedside point-of-care ultrasound demonstrated an estimated left ventricular ejection fraction (LVEF) of 40%. Electroencephalogram monitoring showed no further seizure activity. Diuresis was aggressively continued. A formal echo the next morning showed LVEF 60% without valvular pathology. IV magnesium was stopped after 24 hours with no recurrence of seizures or hypertension. Patient was extubated and transferred to the floor. After 48 hours of normotensive blood pressures, she was discharged with close OB follow-up.

Conclusion: Postpartum eclampsia is possibly due to an increased inflammatory state, potentially triggered by infection, delayed clearance of antiangiogenic factors and proinflammatory cytokines, or activation of the complement system after delivery [2,4]. Generalized endothelial dysfunction in target organs underlies the clinical features of headaches, seizures, visual symptoms, epigastric pain, and pleural effusions [5]. Preeclampsia is associated with increased risk for cardiovascular events such as heart attack, stroke, heart failure and pulmonary edema [6,7]. Peripartum cardiomyopathy is an important consideration [5,6]. Thus, echocardiography is critical to evaluate for cardiac dysfunction contributing to pulmonary edema. In our patient, her clinical course in conjunction with labs, CT imaging, and echocardiography, were effective in guiding diagnosis and narrowing the differential.

References: 1. Obstet Gynecol 135, 1492-5, 2020 2. Am J Perinatol 37, 1217, 2020 3. Obstet Gynecol, 118, 1102-7, 2011 4. Circulation, 132, 1726-33, 2015 5. Semin Perinatol, 33, 158-65, 2009 6. Handb Clin Neurol, 141, 657-74, 2017 7.Circulation, 138, 2359-66, 2018 8. J Am Coll Cardiol, 72, 1-11, 2018

Critical Care - 75 Severe Hypertriglyceridemia: An impending disaster averted with a continuous Insulin Infusion

Gyan N Das[1], Avneep Aggarwal[2]

[1]Cleveland Clinic Foundation, Cleveland, OH, [2]Cleveland Clinic, Cleveland, OH

Introduction: Severe hypertriglyceridemia is a medical emergency secondary to associated risk of acute pancreatitis. It has significant morbidity and mortality but there are no established guidelines for management in a critically ill patient. We present a case of severe hypertriglyceridemia successfully treated with a continuous insulin infusion.

Methods: A 61 year-old-male with a history of alcohol abuse and findings of alcoholic hepatitis with hepatic cirrhosis, presented to ED with chest pain, shortness of breath, and vomiting. Workup for cardiopulmonary causes of chest pain was unremarkable. The laboratory results were significant for lipemic blood, a sodium level of 127 mmol/dL, AST 136 units/L, ALT 75 units/L, lipase 53 units/L, and triglyceride 3835 mg/dL. The patient was admitted to the ICU for management of his severe hypertriglyceridemia and alcohol withdrawal. Upon admission, the patient was kept NPO and started on insulin and dextrose 10% in water infusion. The insulin infusion was dosed from 1-7 units/hr and was maintained for a total duration of 34 hours. 7 hours after initiation of insulin therapy, serum triglyceride had decreased to 2303 mg/dL and 30 hours after initiation of insulin therapy, serum triglyceride had decreased to 485 mg/dL. Close monitoring of blood glucose was kept per protocol for intravenous insulin therapy. After resolution of severe hypertriglyceridemia, the patient's diet was advanced and was started on fenofibrate 200 mg once daily as well as maintaining the patient's prior statin therapy. At time of discharge, the patient's serum triglyceride level remained stable and within normal limits at 97 mg/dL.

Conclusion: Severe hypertriglyceridemia especially triglyceride levels greater than 1000 mg/dL, can lead to acute pancreatitis and requires urgent treatment. Without clearly defined management guidelines, multiple treatments for severe hypertriglyceridemia have been tried including insulin, heparin, octreotide, and plasmapheresis. Insulin specifically increases synthesis of lipoprotein lipase which acts to remove triglycerides from plasma. The rarity of this condition makes standardization of treatment very challenging. Individual case reports are a valuable means for providing new insights to effective treatment guidelines.

References: Rodríguez Santana Y, Nimo Román A, García Sáez I, López Alvarez JM, Consuegra Llapur E, González Jorge R. Treatment of severe hypertriglyceridemia with continuous insulin infusion. Case Rep Crit Care. 2011;2011:293917. doi: 10.1155/2011/293917. Jain D, Zimmerschied J. Heparin and insulin for hypertriglyceridemia-induced pancreatitis: case report. ScientificWorldJournal. 2009 Nov 1;9:1230-2. doi: 10.1100/tsw.2009.142.

Critical Care - 76 Managing a single ventricle LVAD

Maria Betances[1], Mandisa-Maia Jones[1], James Littlejohn[1]

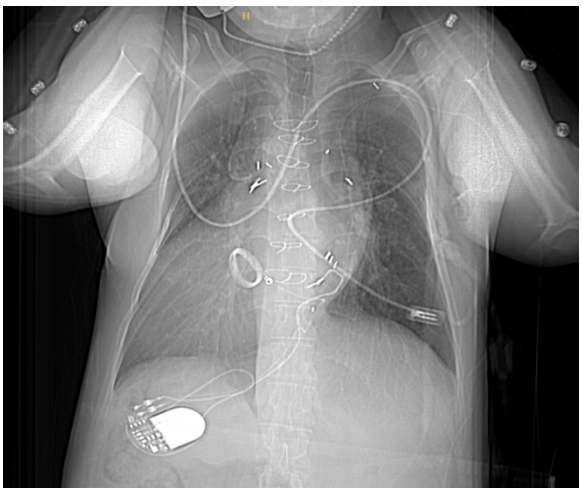
[1]Weill Cornell Medicine, New York, NY

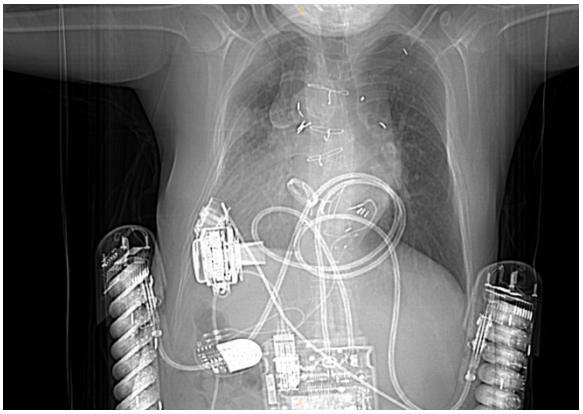
Introduction: Due to advances in surgical techniques and pharmacologic options, more patients born with single ventricles, are living longer and our insight into the progression of the single-ventricle physiology continues to expand. Unfortunately, a percentage of these patients will develop advanced heart failure. Secondary to physiologic and molecular differences between the failing adult heart versus the failing single ventricle heart, pharmacologic therapy has its limitations in this population (1). For this reason, mechanical circulatory support devices (MCSD) play a significant role in the management of these patients, and for some, it is the only therapeutic option before heart transplantation (2)

Methods: A 41-year-old male with multiple heart congenital abnormalities including dextrocardia, left unbalanced atrioventricular septal defect, situs ambiguous, right atrial isomerism, bilateral superior vena cava, and most recently status post bilateral Blalock-Taussig, central aortopulmonary shunt, Glenn, extracardiac Fontan and more recently a 33-mm mechanical common atrioventricular valve at 21 years of age. He presented in cardiogenic shock to his outpatient cardiologist for follow up visit 3 days after discharge from the hospital due to an episode of decompensated heart failure. He was admitted to the cardiology intensive care unit for further management, medication titration and heart transplant evaluation. His transesophageal echocardiogram revealed an ejection fraction (EF) of 25-30%, moderate aortic insufficiency and a non-obstructive mechanical valve prosthesis. Right heart catheterization (RHC) revealed Fontan pressures of 16-17mmHg along with severely reduced cardiac output (CO) (2.8 L/min) and cardiac index (CI) (1.6 L/min/m2). His hospital course was complicated by oliguria in the setting of increasing inotropic support with cardiogenic shock evolving to INTERMACS 1, prompting repeat RHC that showed elevation in Fontan pressures to 20 – 21 mmHg, reduced MVO2 51% with worsening of CO/CI (2.6 L/min and 1.4 L/min/m2). Given his worsening hemodynamics, he underwent a fifth time reoperation, HeartMate 3 (Abbot, Chicago, IL) Left Ventricular Assist Device (LVAD) placement and a Park Stitch aortic valve repair via upper hemisternotomy and right thoracotomy as bridge to decision. Intra-operatively, a pulmonary artery catheter was placed at the Left Superior Vena Cava (LSVC) Left Pulmonary Artery Junction and echocardiographic imaging revealed severe left ventricular dysfunction, moderate aortic insufficiency and a normal atrioventricular mechanical prosthesis function. The device was initially started at 3200 RPM and gradually increased to 5200 RPM while closely monitoring the central venous pressure (CVP), mean arterial pressure and left ventricular size. Post-operatively, the patient had evolving AKI that required initiation of renal replacement therapy (RRT), but LVAD function was excellent with adequate flows, pulsatility index (PI) and minimal PI events. We decided to guide our hemodynamics based on previous cath data and we targeted a CVP of 16-18mmHg to accommodate appropriate passive pulmonary circuit filling and a Mean Arterial Pressure (MAP) of 70 – 80mmHg. We provided inotropic support utilizing Milrinone, and utilized inhaled nitric oxide as a bridge back to sildenafil which the patient was on chronically to optimize Fontan efficiency and flow and used central venous oxygen saturation (ScvO2) as our method to calculate cardiac output. Despite maintaining hemodynamic parameters at goal

Conclusion: The complexity of single-ventricle physiology no longer only poses a challenge to pediatric care teams only as it has now expanded to the adult population due to advancements in the care of these patients resulting in increased life expectancy. This has created the need to expand the applications of advanced heart failure therapy to this unique population. With altered systemic venous return and arterial connections, usage of MCSD is a challenging endeavor that requires careful consideration on a case by case basis. In terms of outcomes, the data is scarce and difficult to interpret (2). Specific echocardiographic benchmarks have been identified in patients with normal heart anatomy and extrapolation of these parameter to single ventricle patients has the potential to misguide management. This reinforces the need for a multidisciplinary approach when treating this population.

References: Heart failure in single right ventricle congenital heart disease: physiological and molecular considerations. Am J Physiol Heart Circ Physiol. 2020 Apr 1;318(4):H947-H965. An overview of mechanical circulatory support in single-ventricle patients. Transl Pediatr. 2018 Apr;7(2):151-161 First Use of HeartMate 3 in a Failing Fontan Circulation. Ann Thorac Surg. 2018 Nov;106(5):e233-e234.





Critical Care - 77 Dialysis Disequilibrium Syndrome After Temporary Discontinuation of Hemodialysis

Emily A Fitzgerald[1], Robert Tainsh[1], Robert Pranaat[1], Tichaendepi Mundangepfupfu[1]

[1]University of Rochester, Rochester, NY

Introduction: Dialysis Disequilibrium Syndrome (DDS) is a rare complication of dialysis leading to neurologic complications ranging in severity from headache and muscle cramps to seizure, coma, and death [1]. These are symptomatic manifestations of underlying cerebral edema [2]. Management consists of measures to decrease cerebral edema and includes reduction or cessation of dialysis [1].

Methods: A 74 year old woman with CREST syndrome resulting in severe pulmonary hypertension and end-stage liver disease from primary biliary cirrhosis presented with hepatic encephalopathy requiring intubation. Her course was complicated by ongoing respiratory failure requiring tracheostomy placement and acute renal failure requiring continuous renal replacement therapy (CRRT). After 50 days of CRRT, she was transitioned to intermittent hemodialysis (iHD) due to stable hemodynamics. The next day, she began having urine output which prompting discontinuation of iHD for 11 days. During this time period, her BUN and serum osmolarity rose from 59mg/dL to 232mg/dL and 308mOsm/kg to 377mOsm/kg, respectively. She was anemic with Hematocrit 18% due to a GI bleed. Dialysis was resumed due to oliguria with an elevated BUN. During this 3-hour session with blood flow rate (BFR) 300mL/hr, hypotension prohibited fluid removal. Two hours and forty minutes into the session, she had a tonic-clonic episode which self-aborted after 3 minutes followed by a 3-hour long post-ictal period. This was her first life-time seizure. CT head showed no acute abnormalities. Labs 3-hours post-iHD demonstrated a BUN of 109mg/dL, a serum osmolarity of 324mOsm/kg, and otherwise non-explanatory electrolytes. Her ammonia level was 82umol/L pre-session and 33umol/L post-session. After consultation with neurology and nephrology, the team concluded that DDS was the most likely explanation for this seizure and the she was transitioned to CRRT to prevent recurrent DDS.

Conclusion: There is active debate in the literature regarding the pathophysiology of DDS [1-3]. Risk factors for DDS include, advanced age, hemodialysis, the initiation of dialysis, markedly elevated BUN and serum osmolarity pre-treatment and rapid correction of these factors during treatment, severe metabolic acidosis, and anemia [1-3]. Already a rare phenomenon, incidences of dialysis disequilibrium syndrome have continued to decrease as dialysis protocols have been improved over time.

Our case does not represent a "typical" presentation of DDS as our patient was not dialysis-naïve and her neurologic symptoms began almost 3 hours into her dialysis session. However, she did have a both a markedly elevated starting BUN and was of advanced age. Her long and complicated hospital course raised concern for a number of etiologies for her seizure, but quick action to pause dialysis and consult nephrology likely spared the patient from further injury. DDS is a diagnosis of exclusion, and many of the predisposing risk factors themselves pose risk for the same neurologic symptoms seen in DDS via different mechanisms. The picture is especially complicated for ICU patients who may have multiple confounding problems. Renal replacement therapy is common in the ICU, with a wide variety of patients from those starting on RRT for the first time to those who have been on iHD for many years. This case demonstrates the necessity of vigilance for all patients undergoing RRT in the critical care setting.

References: 1. Mistry, K. (2019). Dialysis disequilibrium syndrome prevention and management. International Journal of Nephrology and Renovascular Disease, Volume 12, 69–77. https://doi.org/10.2147/ijnrd.s165925 2. Zepeda-Orozco, D., & Quigley, R. (2012). Dialysis disequilibrium syndrome. Pediatric Nephrology, 27(12), 2205–2211. https://doi.org/10.1007/s00467-012-2199-4 3. Arieff, A. I. (1994). Dialysis disequilibrium syndrome: Current concepts on pathogenesis and prevention. Kidney International, 45(3), 629–635. https://doi.org/10.1038/ki.1994.84

Critical Care - 78 A Case of Phlegmasia Cerulea Dolens-Like Phenomenon Following ECMO Cannulation

Hinna Malik[1], Raghu R Seethala[2]

[1]Harvard Medical School, Brigham and Women's Hospital, Boston, MA, [2]Brigham and Women,Äôs Hospital, Westwood, MA

Introduction: Veno-venous extracorporeal membrane oxygenation (VV-ECMO) has become increasingly more utilized since the onset of COVID 19 pneumonia refractory to conventional lung-protective ventilation strategies, both as a bridge to recovery and as a bridge to lung transplant. Cannula location and size are based on a multitude of patient factors including height and weight. The most common cannulation sites for peripheral VV-ECMO are the internal jugular (return) and femoral (drainage) veins. The desired flow diversion in VV-ECMO is 80%, and larger cannulas will help to augment this. For IJ cannulas, most adults will fall into the 19-21Fr range; and for femoral cannulas, the range is usually 21-27Fr. Literature supports the high incidence of cannula-associated DVT (CaDVT), however the majority of studies looked at DVT incidence after de-cannulation. We present a case of acute deep venous obstruction seen immediately after initial cannulation.

Methods: A 60 year old male was transferred from an outside hospital with diagnosed COVID 19 acute respiratory distress syndrome for ECMO consideration as a bridge to lung transplant. On arrival, he had been intubated for 13 days already with radiographic evidence of extensive parenchymal lung disease. Ultrasound of his femoral and internal jugular veins showed widely patent vessels. He was selected for a 27Fr right femoral vein cannula and 21Fr right internal jugular vein cannula. These were based on his anatomy, his weight of 78kg, and his height of 172cm. He was prepped and draped in the usual sterile fashion, and with real-time ultrasound guidance his right femoral and right IJ veins were accessed via a 5Fr micropuncture kit. 5000 units of heparin were administered. Serial dilations were performed over an Amplatz stiff wire in the right IJ vein until a 21Fr cannula was placed with ultrasound confirmation of tip in the SVC. The right femoral vein was then serially dilated in a similar fashion to accommodate a 27Fr cannula with confirmation of tip in the IVC 2cm distal to the right atrium. Upon undraping, the right lower extremity was immediately noted to be cyanotic with diffuse swelling. The patient had bilateral palpable pulses, however ultrasound of the right deep venous system revealed a right popliteal vein with completely static flow concerning for impending thrombosis. Given the risk for limb ischemia from venous obstruction, the femoral cannula was removed and replaced with a 21Fr cannula in the left femoral vein. Immediately upon removing the right femoral cannula, the right lower extremity returned to its normal coloration. The patient was started on a therapeutic heparin drip. A formal lower extremity non-invasive study obtained the next morning revealed no evidence of DVT.

Conclusion: Cannula-associated DVT is a known complication of ECMO cannulation. Most literature observes the incidence in post-decannulation DVT, and one case study showed an incidence as high as 85%. These DVTs were commonly found to be located at the tip of the cannula itself. A study looking at the prevalence of thrombotic complications in VV ECMO by obtaining a CT scan within 4 days of de-cannulation found an incidence of DVT as high as 71.4%. Femorofemoral cannulation had a higher incidence of femoral DVT compared to femorojugular cannulation (69.2% vs 63.1%). Contributing factors include coagulation abnormalities, large cannula size, time on VV ECMO, renal failure, and underlying malignant disease. Introduction of a foreign body (cannula and circuit) to the patient's circulation poses an immediate risk for thrombosis, and anticoagulation will help to prevent this. As a general rule, full anticoagulation is recommended in both VV and VA ECMO. In our case above, we encountered immediate obstruction of the deep venous plexus after right femoral vein cannulation. Two major contributing factors are cannula size and cannulation location close to the great saphenous vein (GSV) takeoff. Both the deep and superficial venous plexuses appeared to be affected, concerning for impending limb ischemia. Despite a negative DVT study, the patient showed all signs and symptoms of Phlegmasia Cerulea Dolens. Literature associated with post-cannulation CaDVT enforces the practice of fully anticoagulating patients on VV and VA ECMO. While peri-cannulation DVT has been rarely reported on, the practice of anticoagulating these patients still applies.

References: 1. Incidence of Cannula-Associated Deep Vein Thrombosis After Veno-Venous Extracorporeal Membrane Oxygenation. ASAIO J. 2017 Sep/Oct;63(5):588-591 2. Prevalence and Risk Factors for Thrombotic Complications Following Venovenous Extracorporeal Membrane Oxygenation: A CT Scan Study. Crit Care Med. 2020 Feb;48(2):192-199 3. Incidence and Risk Factors for Cannula-Related Venous Thrombosis After Venovenous Extracorporeal Membrane Oxygenation in Adult Patients With Acute Respiratory Failure. Crit Care Med. 2019 Apr;47(4):e332-e339

Critical Care - 79 Fem-Fem verses TandemLife Protek Duo Extracorporeal Membrane Oxygenation for COVID-19 ARDS

Raphael Keegan[1], Clara Chu[1], Cyrus Khoyilar[1], Kirsten Young[2], Sonali Rao[1], Phat T Dang[1], Jack Sun[1], Fabio Sagebin[1], Kei Togashi[1]

[1]University of California Irvine Medical Center, Orange, United States of America, [2]University of California Irvine School of Medicine, Irvine, CA

Introduction: One of the most complex complications from COVID-19 acute respiratory distress syndrome (ARDS) is right ventricular dysfunction (RVD) and heart failure (RVHF). Progression to RVHF can be secondary to either intrinsic injury to the right ventricle (RV) or increased RV afterload. The prevalence of RVD in COVID-19 patients has been reported around 20.4% and can be one of the deadliest complications of COVID-19.[1] The most critically ill COVID-19 patients are often consulted to undergo veno-venous extracorporeal membrane oxygenation (VV ECMO)-therapy. Vascular access can be achieved via either femo-femoral (fem-fem) or right internal jugular vein with a TandemLife Protek Duo (TPD, TandemLife, Pittsburg, PA) cannulas. TPD dual cannula offers the capability of providing temporary right ventricular assist device support.[2] The inflow lumen opens in the right atrium and drains venous return to the ECMO oxygenator. The outflow lumen opens beyond the pulmonic valve in the main pulmonary artery - by circumventing the right ventricle TPD theoretically protects against increases in RV afterload.[3,4] In contrast, fem-fem ECMO cannulation does not assist the RV during times of increases in afterload. Both drainage and return cannulas are positioned in the right atrium. To date, no institution has compared clinical outcomes between fem-fem and TPD ECMO cannulations. In this case report series, we assessed patient outcomes of COVID-19 ARDS patients who underwent either fem-fem ECMO or TPD ECMO cannulation. The purpose of this case series is to report the incidence of RVHF, the percentage of days spent spontaneously breathing after VVECMO initiation, ratio of intensive care unit (ICU) days spent in refractory shock and overall survivability of fem-fem and TPD ECMO cannulation.

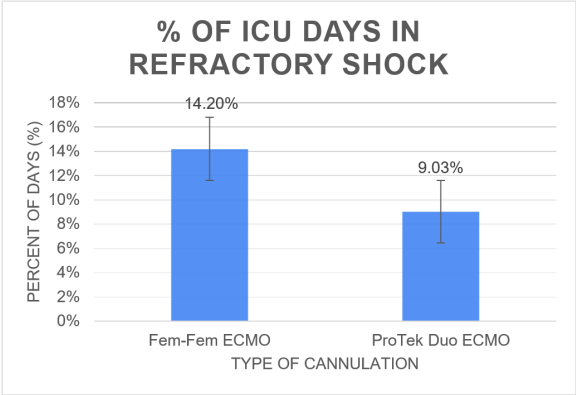
Methods: 26 patients with COVID-19 pneumonia exacerbation underwent VVECMO management from March 2020 to September 2021. Selection for cannulation was based on age (<65 years old), body mass index (BMI <45), number of days intubated (<14 days), and no other organ failure besides respiratory failure. Midway through the COVID pandemic (January 2021), we have discovered that increasing number of patients developed RVD due to pulmonary hypertension from respiratory failure. With this observation, we transitioned from fem-fem VVECMO cannulation to TPD cannulation for right ventricular support. As a result, 14 patients were managed with fem-fem cannulation and 12 patients were managed with TPD cannula. We then assessed existence of RVHF by transthoracic echocardiography (TTE) assessment before and after cannulation. The TTE parameters were right ventricular size, systolic function and tricuspid annular plane systolic excursion (TAPSE). Secondary outcomes included percentage of days spent spontaneously breathing during VVECMO management, the ratio of ICU days spent in refractory shock and in-hospital mortality. The ratio of ICU days spent in refractory shock was defined as the number of ICU days while receiving infusions of either epinephrine or norepinephrine (≥15 mcg/min) divided by total days spent in the ICU.

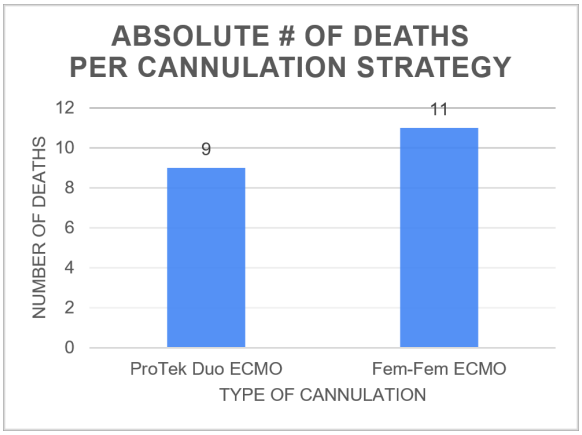
Conclusion: Twenty six patients underwent VVECMO treatment. Of these 14 patients received fem-fem ECMO while 12 patients received TPD ECMO. A total of 15 patients received a TTE before and after cannulation. Nine of the 15 patients underwent fem-fem cannulation while the remaining six underwent TPD cannulation. Pre-existing RVHF was greater in the fem-fem ECMO group (n=3, 33.3%, SD ± 50) compared to the TPD ECMO group (n=2, 33.3, SD ± 55.6). New onset RVHF was also greater in the fem-fem ECMO group (n=5, 55.6%, SD ± 52.7) versus the TPD ECMO group (n=3, 50%, SD ± 54.8). Out of the 26 ECMO patients selected, the percentage of days spent spontaneously breathing after VVECMO initiation was lower in the fem-fem ECMO group (9.0%, SD±16.2) compared to the TPD ECMO group (37.6%, SD±40.4), and the ratio of ICU days spent in refractory shock was greater in the fem-fem ECMO group (13.9 days, SD ± 14) compared to TPD ECMO group (9 days,

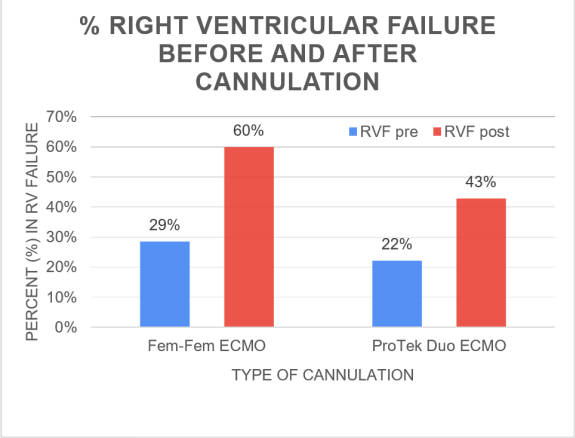
SD ± 9). Overall survivability was greater in TPD ECMO group where the number of deaths only nine (69.2%, SD ± 48) compared to 11 the fem-fem ECMO group (78.6%, SD ± 42.6).

Patients with COVID-19 ARDS who underwent TDP ECMO cannulation had a decreased incidence of new onset RVHF in our institution. These patients also spent fewer ICU days in refractory shock, more days breathing spontaneously and fewer patients expired.

References: 1. Sci Rep 11, 17774 (2021). 2. Circulation. 2017 Jul 18;136(3):314-326. 3. J Surg Res. 2021 Aug;264:81-89. 4. ASAIO Journal: July/August 2018 - Volume 64 - Issue 4 - p 570-572.







Critical Care - 80 Durable LVAD - ethical at 26 years of age?

Maria Betances[1], Liang Shen[1]

[1]Weill Cornell Medical College, New York, NY

Introduction: Deciding which patients are suitable for mechanical circulatory support devices (MCSD) can pose an ethical challenge to the team making the decision, especially in the setting of emergent decompensations and without knowing the patient's wishes. Shock states can compromise the ability of a patient to determine his own care (1). These devices are generally considered a last resort for a specific group of patients that meets certain criteria, and therefore data is very limited. The utilization of MCSD outside of the studied population presents a complex question that requires thorough, multidisciplinary evaluation of the patient's conditions along with risks and benefits.

Methods: A 26-year-old man with a history of morbid obesity, non-ischemic cardiomyopathy with recovery of left ventricular ejection fraction (LVEF) and schizophrenia with multiple suicide attempts presents to the cardiothoracic ICU after VA ECMO cannulation at an outside hospital. He initially presented with shortness of breath but rapidly decompensated in the setting of cardiogenic shock, based on severely reduced left and right heart function on cardiac catheterization, of unclear etiology. Concurrently, upon presentation the patient was diagnosed with shock liver, acute kidney injury, ventricular tachycardia, acute respiratory failure, and pneumonia. Initial echocardiogram revealed a LVEF of 10%, with severe global hypokinesis. Over the next several days, his cardiac function minimally improved on ECMO and the need for a post-decannulation plan became apparent. Ongoing severe agitation required high doses of sedatives, which precluded the patients' participation in goals of care discussions. The patient was deemed not a transplant candidate because his BMI was >55. Due to his concerning psychiatric history, conflicting reports from family members, and multiple severe comorbidities, the medical team was concerned about the risks and benefits of offering a durable mechanical circulatory support device such as LVAD. Data available for patients with similar characteristics, but older age, predicted low chance of survival after LVAD implant, but there was uncertainty that this data accurately translates to a patient of younger age. It was unclear whether the patient's age advantage could overcome his range of co-morbidities and risk factors for noncompliance with the stringent care required to live successfully with a device. After multiple multidisciplinary meetings including involving the ethics department, the patient underwent a successful placement of a LVAD as a bridge to recovery/destination therapy along with a right ventricular assist device (RVAD) to support the right ventricular after LVAD implantation. His post-operative course was complicated by mediastinal bleeding requiring washout twice, but the RVAD was able to be weaned and removed on post-operative day 14. The patient required tracheostomy, continues to require low doses of inotropes and vasopressors, and has been able to participate in physical therapy efforts at the time of this report.

Conclusion: The use of LVAD has been shown to be effective as a therapeutic option in patients with heart failure as a bridge to recovery (2) but, having an LVAD is accompanied by many risks and complications that can severely impair the patient's life. Worse, its placement could be potentially harmful when implanted in an inappropriate patient. Important ethical considerations include the patients' known wishes, support for the patient's autonomy, and ensuring non-maleficence in the treatments that are offered, one way to ensure this is by using the criterion of proportionality and the method of four boxes described by Roggi et al(3). Despite the development of indications and contraindications to the use of MCSD, the decision-making process can still prove extremely challenging, and decisions should be made on a case-by-case basis with the involvement of a multidisciplinary team.

References: Emergent use of mechanical circulatory support devices, Current Opinion in Cardiology: May 2014 - Volume 29 - Issue 3 - p 281-284 Left Ventricular Assist Device as a Bridge to Recovery for Patients With Advanced Heart Failure. J Am Coll Cardiol. 2017;69(15):1924-1933.

Critical Care - 81 Anticoagulation Considerations in an LVAD Patient with Intraparenchymal Hemorrhage Needing Emergent Noncardiac Surgery

Lauren Rego-Cherian[1], Brent Kidd[2]

[1]University of Kansas Medical Center, Kansas City, United States of America, [2]University of Kansas Medical Center, Kansas City, KS

Introduction: Patients with left ventricular assist devices (LVADs) can find themselves in a tenuous position when needing emergency surgery or other invasive procedures. The majority of these cases, excluding cardiac surgeries, are usually gastrointestinal endoscopies . Currently, there are no guidelines regarding reversal of anticoagulation in these patients. In addition, retrospective studies have demonstrated a 13% incidence of intracranial hemorrhagic (ICH) events in patients with LVAD [1]. We present a case demonstrating anticoagulation and reversal strategies in a patient with a LVAD who, originally admitted with intraparenchymal hemorrhage (IPH) and subarachnoid hemorrhage (SAH) in the setting of supratherapeutic INR , also suffered a splenic rupture necessitating an emergent laparotomy.

Methods: A 50-year-old male with a LVAD presented to the hospital with a three day history of headache and double vision. He had a past medical history notable for chronic obstructive pulmonary disease and coronary artery disease with multiple percutaneous interventions complicated by ischemic cardiomyopathy. Upon admission, his vitals were within normal limits and he had no focal deficits. A CT of the head without contrast demonstrated a SAH and multiple IPHs in the setting of an INR of 7.1. He received 5mg of vitamin K. Neurosurgery was consulted and recommended an INR goal of 1.4, but no acute neurosurgical intervention was warranted. The patient was reversed with 5 units/kg of PCC. However, 12 hours after reversal, a repeat CT showed an interval increase in his IPH. He was given 2 units of platelets to counteract the effects of ASA. On the evening of admission day 1, the patient became hypotensive and with PI events – an emergent CT of the chest/abdomen/and pelvis demonstrated a spontaneous splenic rupture as the cause of hemorrhagic shock. The patient was taken to the operating room emergent for splenectomy, where he was placed on three pressors for support and received 3 units of RBCs, 3 units of FFP, and 1 unit of platelets. A postoperative thromoboelastogram demonstrated a normal curve. His postoperative course was complicated by atrial fibrillation, an upper extremity DVT, and combined hemorrhagic and septic shock presumably from rhinovirus. On POD 2, he was started on an IV heparin drip without a bolus, and an interval head CT showed no changes in the IPH. On POD 6, he was transitioned to argatroban due to low antitrypsin 3 levels and ASA started, and this was used as a bridge to warfarin.

Conclusion: Reversal strategies for patients with LVADs and ICHs are not well defined. Small retrospective studies comparing PCC vs. non-PCC in patients on vitamin K antagonists showed no difference in thrombotic events, however reversal was more quickly achieved in patients receiving PCC [2]. In this case, the patient was reversed with both vitamin K and PCC, and did not suffer any cardioembolic events. A thromboelastogram to guide reversal in these patients may be prudent to avoid inducing a hypercoagulable state. A retrospective study of 8118 LVAD patients who needed noncardiac surgery showed that those who needed emergent surgery do have an increased risk of major adverse cardiac events (16.9%) and is associated with a higher mortality. In addition, patients with an LVAD who experience cranial hemorrhage have survival rates of 41% for IPH and 83% for SDH [1]. More studies regarding both the timing and methodology of reversal and resumption of anticoagulation are needed in this high-risk patient population to help improve outcomes.

References: 1. J Neurosurg 2019;132:1133-9. 2. ASAIO J 2016;62:552-7.

Critical Care - 82 Successful Treatment of Severe COVID-19-Associated ARDS in a Postpartum Patient with Prolonged VV-ECMO

Johnny Wei[1], Brigid C Flynn[2], Brent Kidd[3]

[1]University of Kansas, Kansas City, KS, [2]University of Kanas, Kanas City, KS, [3]University of Kansas Medical Center, Kansas City, KS

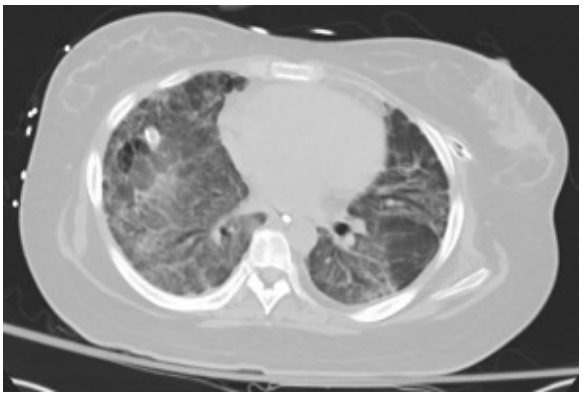
Introduction: Although the use of extracorporeal life support (ECLS) in the peripartum setting is extremely rare, acute respiratory distress syndrome (ARDS) represents nearly half of the indicating pathology (1). The COVID-19 pandemic and the concomitant rise of severe respiratory infection in pregnant women internationally have highlighted the unique challenges of initiating ECLS in this challenging patient population. We present the case of a pregnant patient who required postpartum venovenous extracorporeal membrane oxygenation (VV-ECMO) for severe COVID-19 pneumonia.

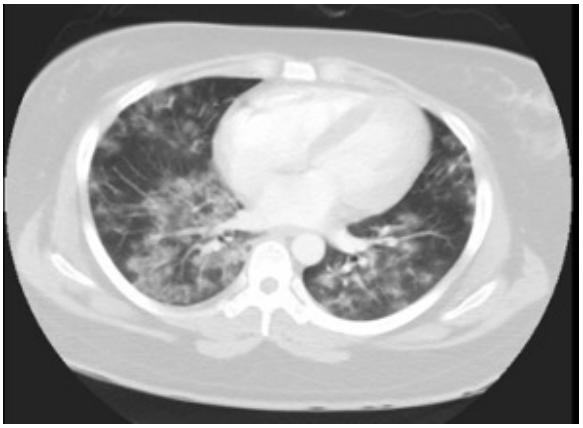
Methods: A 24 year old female patient at 33 weeks gestation presented to an outside hospital with COVID-19 pneumonia. She was started on antiviral and adjunctive therapy but continued to decompensate and was intubated and the fetus delivered via cesarean section at 34 weeks. Her hypoxemia continued to worsen, with a P/F ratio of 53 and chest imaging revealing multifocal infiltrates. She was subsequently initiated on VV-ECMO and transferred to our institution for further care. Our patient required ECMO for over seven weeks and experienced a complex clinical course with multi-organ dysfunction. She had significant hypoxemia secondary to extensive pulmonary injury with fibrosis and pneumatocele development and required tracheostomy cannulation, neuromuscular blockade, and heavy sedation to maintain ventilator synchrony and promote lung recovery. Inhaled nitric oxide was also initiated to maintain oxygenation and improve right heart dysfunction. She developed anuric renal failure and required renal replacement therapy. She developed bacterial superinfection of her viral pneumonia with Stenotrophomonas and Acinetobacter. Throughout her hospitalization, multiple family meetings were held to discuss the patient's poor recovery trajectory and prognosis. She was ultimately decannulated on hospital day 51, transferred out of the ICU on day 77, and discharged to inpatient rehabilitation on day 88. She was also successfully decannulated from her tracheostomy tube and transitioned off of hemodialysis. At time of discharge from rehabilitation, patient was able to mobilize independently with a walker with supplemental oxygen and had no notable neurologic deficits.

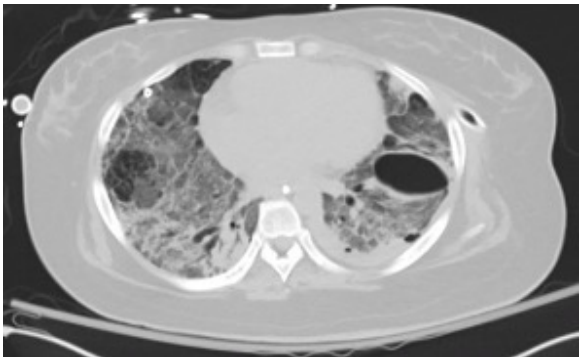
Conclusion: COVID-19 has emphasized the importance of appropriate patient selection and resource allocation for initiating VV-ECMO for ARDS (2), and peripartum patients present a unique challenge in this calculus. Normal immunologic changes in pregnancy combined and the prevalence of pregnancy-related conditions such as hypertension, diabetes, and obesity place pregnant women at high-risk for severe COVID-19 infection (3). However, pregnant patients also tend to be younger and healthier which portends a higher probability of recovery with ECLS, with one-year survival rates of nearly 75% in some large-scale reviews and some single-center reports of full survival in pregnant patients with COVID-19 (1,3). This can complicate discussions regarding withdrawal of ECLS, particularly during a pandemic when scarce resources make maintenance of life-sustaining support a crucial opportunity cost. In patients with ARDS due to COVID-19, the mean duration of ECMO use and ICU length of stay is 16 and 29 days respectively, but a growing body of literature demonstrates recovery despite ECMO use exceeding 28 days (4,5). However, longer ECMO and ICU duration also exposes patients to complications from ECLS and critical illness. Although our patient far exceeded these averages and fortunately made a remarkable recovery, the ability to predict those who may benefit from prolonged use of ECMO remains elusive for intensivists. In the future, use of ECLS for ARDS will continue to require careful patient selection, rigorous guidelines for use, and clear communication between physician and decision makers regarding when the use of those resources may no longer be in line with the patient's goals of care (2).

References: 1. Naoum EE et al. “Extracorporeal Life Support in Pregnancy: A Systematic Review.” J Am

Heart Assoc. 2020 Jul 7; 9(13): e016072. 2. Murugappan KR et al. “Veno-venous extracorporeal membrane oxygenation allocation in the COVID-19 pandemic.” J Crit Care. 2021 Feb; 61: 221–226. 3. Barrantes JH et al. “Successful Treatment of Pregnant and Postpartum Women With Severe COVID-19 Associated Acute Respiratory Distress Syndrome With Extracorporeal Membrane Oxygenation.” ASAIO J. 2021 Feb; 67(2): 132–136. 4. Ramanathan K et al. “Extracorporeal membrane oxygenation for COVID-19: a systematic review and meta-analysis.” Critical Care 25, 211 (2021). 5. Dreier E et al. “ECMO in COVID-19-prolonged therapy needed? A retrospective analysis of outcome and prognostic factors.” Perfusion. 2021 Sep;36(6):582-591.







Critical Care - 83 Bullous Lung Disease and Tension Pneumothorax in a Patient with Recent COVID-19 Pneumonia

Patrick J Binetti[1], Sarah Jarjour[2], Michelle Yu[3], Melissa Coleman[4]

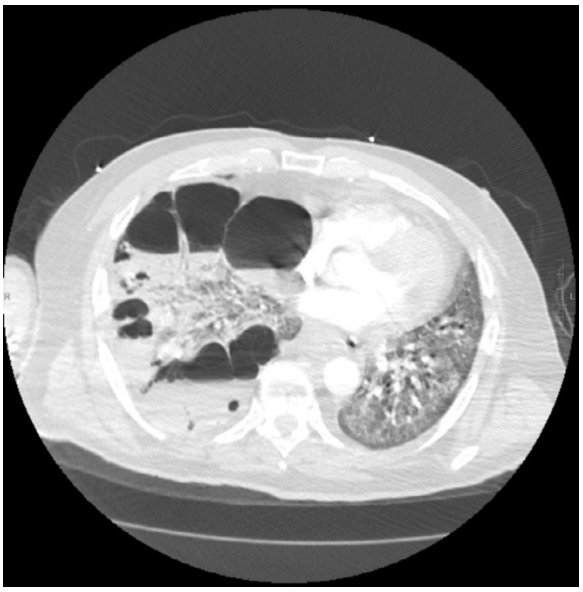
[1]University of California San Francisco, San Francisco, CA, [2]University of California, San Francisco, San Francisco, CA, [3]University of California San Francisco, San Francisco, United States of America, [4]University of California San Francisco, San Fracisco, United States of America

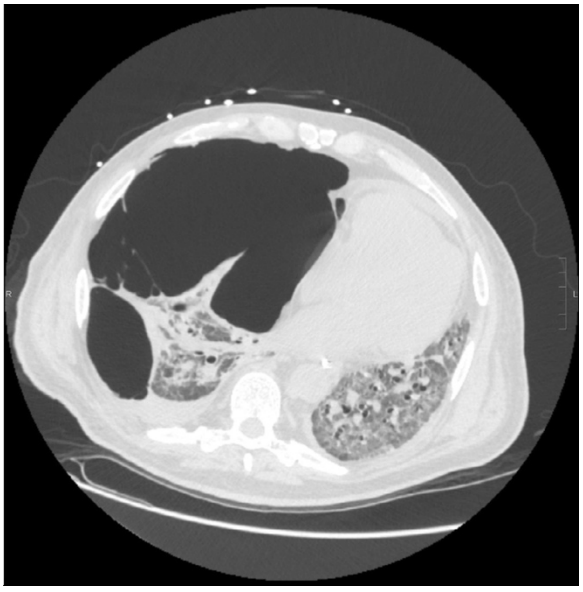
Introduction: The COVID-19 pandemic persists as a significant cause of hypoxemic respiratory failure and acute respiratory distress syndrome with an estimated 32% adult of patients hospitalized for COVID-19 requiring ICU admission and 19% requiring invasive mechanical ventilation (1). Mechanically ventilated patients with COVID-19 may experience barotrauma complications at a higher rate than those without COVID-19 (2). We present a case of COVID-19 complicated by empyema, necrotizing pneumonia leading to bullous changes, and tension pneumothorax.

Methods: A 60-year-old male with a past medical history of smoking, asthma, congestive heart failure, atrial fibrillation (Afib), and recovered from COVID pneumonia was readmitted with severe shortness of breath, hypoxia, and right pleuritic chest pain. CT pulmonary embolism (PE) study was negative for PE but was significant for diffuse ground glass opacities, a moderate right complex hydropneumothorax concerning for an empyema, and extensive cystic lesions with air-fluid levels in the right lung (Figure 1). He was started on broad spectrum antibiotics but progressed to septic shock and required intubation for hypoxemic respiratory failure. He was transferred to our center for surgical evaluation. A right 16Fr thoracostomy tube was placed with significant cloudy serosanguinous output. Pleural fluid studies were consistent with empyema, but no organisms were isolated. He remained on broad spectrum antibiotics but continued to decompensate with persistent fevers, frequent episodes of rapid Afib, worsening shock, and high airway pressures. Repeat CT chest showed a resolution of the fluid component but worsening of the air component of the previously seen hydropneumothorax, with almost complete collapse of the right lung and concern for tension physiology (Figure 2). Given his extensive bullous disease on presentation, the differential for the expanding air component seen on scan included giant bullae vs pneumothorax. After discussion with thoracic surgery and radiology, a right pigtail catheter was placed, which improved hemodynamics and airway pressures without additional air leaks. He ultimately underwent tracheostomy placement for ventilator weaning and was eventually discharged to a skilled rehabilitation facility.

Conclusion: Unstable COVID-19 patients represent a difficult population to evaluate. Parenchymal damage, such as cystic or bullous changes, and barotrauma should always be on the differential for clinically deteriorating patients (2,3). Our case highlights a challenging scenario of worsening obstructive shock in the setting of possible pneumothorax vs giant bullae. Decompression with thoracostomy tube placement, while appropriate for pneumothorax, would be a serious and potentially fatal complication if placed into a bulla. A multidisciplinary approach with thoracic surgery, interventional radiology, pulmonology, and ICU was critical in the safe and effective management of this complex case.

References: 1. Kim et al. Risk Factors for Intensive Care Unit Admission and In-hospital Mortality Among Hospitalized Adults Identified through the US Coronavirus Disease 2019 (COVID-19) Associated Hospitalization Surveillance Network (COVID-NET). Clin Inf Disease. 2021 May; 72(9): e206-e214 2. McGuinness et al. Increased Incidence of Barotrauma in Patients with COVID-19 on Invasive Mechanical Ventilation. Radiology. 2020; 972:e252–e262 3. Berhane et al. Development of bullous lung disease in a patient with severe COVID-19 pneumonitis. BMJ Case Rep. 2020 Sep; 13:e237455





Critical Care - 84 Fatal Acute Hyperammonemic Encephalopathy: A Case Report

Adriana S Martini[1], Lawrence Nolan[1], Garrett Thomas[1], Vivek Sabharwal[1]

[1]Cleveland Clinic, Cleveland, OH

Introduction: Acute hyperammonemic encephalopathy (AHE) is a potentially life-threatening condition that can initially be mistaken for hypoxic ischemic encephalopathy or other stroke syndromes leading to delayed treatment. Patients can present with impaired consciousness, generalized weakness, seizures, and death1,2. The high mortality with severe cases is directly correlated to elevated ammonia that can lead to cerebral edema. Liver failure is the primarily etiology, but less commonly congenital enzyme deficiencies of the urea cycle, medications such as valproic acid, barbiturates, acetaminophen, urease-producing GI or GU organisms, parenteral nutrition and septic shock are other sources3,4.

Methods: We present the case of a 54-year-old male with history of cirrhosis from congenital hepatic fibrosis, portal hypertension, and hereditary hypophosphatemic rickets leading to ESRD on peritoneal dialysis admitted to the neuro-ICU for management of suspected left MCA syndrome after presenting with 4-day history of tremors, altered mental status, right sided weakness, and facial droop. Initial neuroimaging was not suggestive of stroke. Bloodwork was significant for ammonia level of 261 umol/L, BUN 105 mg/dl and creatinine 15.13 mg/dl. The ensuing hospital course was complicated by septic shock requiring broad spectrum antibiotics and vasopressors, coagulopathy requiring multiple transfusions, and seizures refractory to initial therapy that eventually required burst suppression with IV sedation. His ammonia up-trended to a maximum of 1649 umol/L despite traditional therapy and ultimately responded to L-Carnitine started for concern of a genetic etiology. On day 3 of the hospital course, an MRI brain performed for encephalopathy demonstrated multiple areas of restricted diffusion in cortical and subcortical regions. IV sedation used for seizure suppression was weaned several days later, unfortunately the patient's exam was poor with minimal brain stem reflexes. Follow-up neuroimaging was suggestive of cerebral edema secondary to metabolic encephalopathy with several diagnostic possibilities explored including genetic, vascular, traumatic. Repeat imaging demonstrated worsening edema with associated herniation. Due to dire clinical picture, patient was made DNR-CCA after family discussion.

Conclusion: Early diagnosis and aggressive treatment of acute hepatic encephalopathy are critical to decrease mortality and should be considered in the appropriate clinical setting. Our case demonstrates the importance of distinguishing and differentiating acute vascular neurologic manifestations from metabolic syndromes. We also hope to highlight the need to carefully consider any underlying pathophysiology when evaluating patients in these situations. Early identification of these patients with history, appropriate labs, and involvement of multidisciplinary consultant is essential while also evaluating and ruling out other neurologic etiologies3,4,5.

References: 1. Ito H, Ogawa Y, Shimojo N, et al. (September 08, 2020) Delayed Cerebral Abnormalities in Acute Hyperammonemic Encephalopathy. Cureus 12(9): e10306. 2. Younes K, Gonzales N, R, Sarraj A, Bonfante E, Jagolino-Cole A: Hepatic Encephalopathy Mimicking Acute Dominant Middle Cerebral Artery Ischemic Stroke: A Case Report. Case Rep Neurol 2019;11:304-311. 3. Machado, M.C., Pinheiro da Silva, F. Hyperammonemia due to urea cycle disorders: a potentially fatal condition in the intensive care setting. j intensive care 2, 22 (2014). 4. Kalra A, Norvell JP. Cause for Confusion: Noncirrhotic Hyperammonemic Encephalopathy. Clin Liver Dis (Hoboken). 2020 Jun 30;15(6):223-227. 5. Pulivarthi, S., & Gurram, M. K. (2016). Magnetic resonance imaging of brain findings in hyperammonemic encephalopathy. Journal of neurosciences in rural practice, 7(3), 469–471.

Critical Care - 85 Successful Intralipid Rescue in Cardiac Arrest Secondary to Loperamide Toxicity

Kelsey Harder[1], Matthew Johnson[1], Joel Grigsby[1], Elizabeth Cotter[1]

[1]University of Kansas Medical Center, Kansas City, KS

Introduction: Opioid misuse in the United States continues to be a growing problem. Its involvement in overdose related deaths increased six times since 1999.[1] While loperamide misuse likely accounts for a negligible proportion of these deaths, its use as a drug of abuse has also been on the rise.[2] This case highlights the unique cardiotoxic effects of loperamide toxicity and describes the use of intralipid as salvage therapy in the case of cardiac arrest.

Methods: A 25-year-old woman with history of recurrent syncope and Brugada pattern presented in transfer following an electrophysiologic study and implantation of pacemaker and ICD. On hospital day 6, the patient displayed worsening pre-syncopal symptoms, hypotension and lactic acidosis. Repeat echocardiogram showed a left ventricular EF of 30%. The next 24 hours were notable for rapid swings in blood pressure, with periods of profound hypotension refractory to vasopressors and intermittent runs of monomorphic and polymorphic ventricular tachycardia. Given the lack of response to traditional therapies her medical history was revisited with family and it was discovered that had a long history of loperamide misuse. Subsequently, the patient suffered a cardiac arrest with pulseless electrical activity and ventricular tachycardia. After approximately 30 minutes of advanced cardiac life support without return of circulation, 100 grams of intralipid fat emulsion was administered. Shortly after, sinus rhythm was restored. Her hemodynamic profile rapidly improved over the next 12 hours. Loperamide levels were obtained both before and after intralipid administration and demonstrated extremely elevated plasma concentrations. (Table 1) While the patient did suffer an acute kidney injury requiring initiation of hemodialysis; she otherwise made a remarkable recovery. Unfortunately, she declined opioid addiction treatment, left the hospital against medical advice and has failed to follow up.

Conclusion: Loperamide is a phenylpiperidine opioid used as an over the counter anti-diarrheal. The maximum recommended dose is 16mg/day. Extremely high doses are required to achieve euphoria and even higher doses to cause cardiotoxicity. Rhythm disturbance is the most commonly described cardiac manifestation with prolongation of the QT interval and widening of the QRS. Notably, amiodarone is a known inhibitor of P-gp, which when taken with loperamide can increase systemic absorption as well as increase penetration into the central nervous system.[2] It is unclear to what degree amiodarone administration during her stay contributed to the clinical picture. Treatment of loperamide toxicity is largely supportive. Several case reports suggest that lipid therapy may help in refractory cases of cardiotoxicity but supportive information was limited.2 The administration of Intralipid in this case may have contributed to successful resuscitation and subsequent recovery.

References: 1. Center for Disease Control and Prevention. March 10, 2021. https://www.cdc.gov/opioids/data/analysis-resources.html. Accessed Nov 21 2021. 2. Annals of Emergency Medicine 70(2):245-252. 2017.

Table 1: Loperamide and Desmethyloperamide Levels

	Reporting limit	Time relative to cardiac arrest		
		- 40 hours	- 30 minutes	+ 5 days
Loperamide (ng/mL)	5	190	120	Not detected
Desmethyloperamide (ng/mL)	5	520	390	100

*Approximately 12 hours prior to profound hemodynamic instability

Critical Care - 86 Stellate Ganglion block for Upper extremity Ischemia following failed surgical revascularization.

Karuna Puttur Rajkumar[1], Arun Ganesh[2]

[1]Duke, Cary, NC, [2]Duke University Medical Center, Durham, United States of America

Introduction: We describe the successful use of stellate ganglion blockade to salvage an acutely ischemic hand following failed surgical revascularization.

Methods: 51 year old female with an aberrant right subclavian artery presented with right upper extremity paresthesias and weakness from ischemia. After undergoing axillary to ulnar artery bypass with the ipsilateral cephalic vein, she had absent radial and ulnar artery Doppler signals post-operatively which required repeat emergent surgical revascularization. While arterial flows were initially restored, Doppler signals again abated and another surgical revascularization was deemed not possible. After medical treatment with intravenous nitroglycerin and calcium channel blockers failed to improve ischemia, a right stellate ganglion block was performed to vasodilate the extremity and a catheter was placed near the ganglion for repeat dosing. Four hours after the block, arterial Doppler signals returned and pulses were palpable. The stellate ganglion catheter was bolused intermittently with local anesthetic for eight days with improvement of the ischemic hand.

Conclusion: Stellate ganglion block should be considered for upper extremity ischemia that is refractory to medical and surgical intervention.

References: 1.Soni KD, Sawhney C, Kaur M, Ramchandani S, Singhal M. Stellate ganglion block as a limb salvaging technique. Indian J Anaesth. 2012;56(3):307-308. doi:10.4103/0019-5049.98792
2.Matche P, Poorna M S. Continuous stellate ganglion block for the effective management of ischemic pain following compartment syndrome of upper limb due to accidental intra-arterial injection. Indian J Pain 2020;34:50-2

Critical Care - 87 TPA-induced Angioedema and Airway Management

Andrew Lee[1], Ryan Nowatzke[2], Mohammadali Chokr[2], Roy Soto[2]

[1]Oakland University William Beaumont, Royal Oak, MI, [2]Beaumont Health, Royal Oak, MI

Introduction: The use of tissue plasminogen activator (tPA) is common in patients who present with ischemic cerebral vascular accidents (CVAs). In about 1.3% to 5% of cases, tPA has been shown to cause angioedema1. Angioedema is a severe allergic reaction which is mediated by both the complement and kinin pathway. Increased levels of bradykinin, a powerful vasodilator, can increase vascular permeability and consequently lower blood pressure2. Common localization of angioedema includes swelling of the face, tongue, lip and oral mucosa, but can progress to a severe, life-threatening upper airway emergency. This case will discuss the management of a stroke patient who experienced angioedema after the administration of tPA.

Methods: A 52-year-old female with a history of T2DM, subarachnoid hemorrhage and HTN treated with an ACE-inhibitor presented to the ED with acute onset L sided weakness and slurred speech. On arrival to the ED around 0100, she also presented with left-sided facial droop and left-sided visual field loss. The husband reported that the patient was last seen normal at 0030, and her last intake of ACE-inhibitor was 15 hours prior to arrival. Patient was evaluated by the stroke team and NIHSS was 10. CT head was negative for hemorrhage and CTA showed no major vessel occlusion. Patient was administered tissue plasminogen activator (tPA) at 0150 and 30-minutes post-tPA infusion, NIHSS dropped to 3. Around 0315, the patient began to complain of tongue swelling and increasingly muffled speech. Shortly afterwards, her tongue doubled in size and she had difficulty swallowing. Diphenhydramine, methylprednisolone and epinephrine were given without improvement. Level 2 emergent airway was activated and the patient was transferred to the OR for emergent fiberoptic intubation. MRI head done thereafter, showing subacute ischemia involving the posterior limb of the right internal capsule. She remained on full ventilatory support and stable blood pressure control. The patient was extubated 10 days later and direct laryngoscopy noted no edema in the tongue, larynx or pharynx. Repeat CT head showed improvement in edema of right hemispheric infarcts without hemorrhagic transformation. Patient gradually improved in following commands with occasional slurred speech and left sided extremity weakness. Patient was finally discharged home one month later but has yet to have followed up since hospitalization.

Conclusion: Patients on ACE-inhibitors who are given tPA should be monitored closely for tPA-induced angioedema due to similar mechanisms. Although there are no currently approved algorithms or conclusive treatments for tPA-induced angioedema, physicians should be prepared to administer supportive medications or intubate in cases of airway compromise. It has been recommended by other authors that it may be beneficial to routinely observe patients 30-60 minutes post-tPA administration to check for signs of angioedema3. This specific case may serve as an example to extend patient observation post-tPA for 1-2 hours. Additionally, patients who have had an episode of tPA-induced angioedema and are currently taking an ACE-inhibitor should have the risks and benefits weighed prior to the administration of tPA4. If possible, it should be later advised for the patient to transition from ACE-inhibitors to angiotensin receptor blockers (ARBs) to avoid future adverse reactions.

References: 1. Harris D, Harter D. A Case Report of Unilateral Orolingual Angioedema Secondary to Alteplase Administration. Cureus. Published online June 23, 2018. doi:10.7759/cureus.2869 2. Obtu≈Çowicz K. Bradykinin-mediated angioedema. Pol Arch Med WEWNƒòTRZNEJ.:10. 3. Foster-Goldman A, McCarthy D. Angioedema From Recombinant TPA Administration: Case Report and Pathophysiology Review. :3. 4. Burd M, McPheeters C, Scherrer LA. Orolingual Angioedema After Tissue Plasminogen Activator Administration in Patients Taking Angiotensin-Converting Enzyme Inhibitors. Adv Emerg Nurs J. 2019;41(3):204-214. doi:10.1097/TME.0000000000000250

Geriatric Anesthesia

Geriatric Anesthesia - 1 Intraoperative Application of Pulmonary Venous Doppler Flow to monitor Left Atrial Pressure on an Elderly Patient with Aortic Stenosis Undergoing Right Hip Open Reduction and Internal Fixation

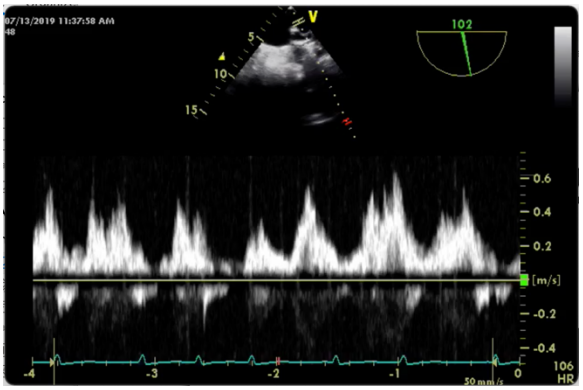
Daniel Lazzareschi[1], Julin F Tang[2], Abigail Howard[3]

[1]Zuckerberg San Francisco General Hospital and Trauma Center, San Francisco, CA, [2]UCSF/ZSFGH, San Francisco, CA, [3]UCSF, San Francisco, CA

Introduction: A 100-year-old woman underwent open reduction and internal fixation for a right hip fracture after a ground-level fall requiring careful hemodynamic monitoring and targeted therapeutic interventions. The patient had a PMH notable for hypertension, hyperlipidemia, CKD, and CHF. Upon presentation to the hospital, initial surveys and workup revealed small epidural and subarachnoid hemorrhages in the brain as well as a right femoral intertrochanteric fracture. Abdominal CT was notable for active extravasation requiring emergent embolization with monitored anesthetic care. Preoperative assessment revealed atrial fibrillation on EKG with evidence of left anterior fascicular block. A prior transthoracic echocardiogram (TTE) confirmed atrial fibrillation and heart failure with reduced ejection fraction, and the pulmonary arterial systolic pressure (PASP) was 33 mmHg plus CVP. Of note, the TTE showed the left atrial dimension to be moderately dilated (5.2 cm). Aortic valves were sclerotic, and the aortic valve area was 1.5 cm2 with mean gradient of 6 mmHg consistent with mild aortic stenosis (AS). An arterial line was placed prior to induction with 12mg of Etomidate, 50 mcg of Fentanyl, and 100 mg of Rocuronium, and subsequent endotracheal intubation was successful. Transesophageal echocardiography (TEE) was then placed for hemodynamic monitoring. The procedure took three hours, and a total of 32 mcg of norepinephrine was administered to mitigate hypotension. Fluid resuscitation was monitored closely via the transgastric short-axis view (TG SAX) whereby left ventricular internal diastolic dimension (LVIDd), ejection fraction (EF), and stroke volume (SV) were calculated and dynamically reassessed. Additionally, pulmonary vein doppler flow (PVDF) was utilized periodically during fluid boluses to estimate left ventricular filling pressures. The systolic-to-diastolic ratio of PVDF in the mid-esophageal two-chamber view were imaged immediately before and after fluid boluses, titrating resuscitation efforts to the delicate and sometimes competing demands of traumatic injury and preload-dependent AS under general anesthesia. Arterial Blood Gas (ABG) readings remained stable. Serum troponins drawn before and after surgery were negative (<0.04 ng/mL), and she was admitted to the surgical ICU after the procedure for a gradual weaning of mechanical ventilation. She was successfully extubated six hours later and did well thereafter, ultimately being discharged from the ICU.

Conclusion: It was a useful intraoperative application of pulmonary venous doppler flow to monitor left atrial pressure on an elderly patient with aortic stenosis undergoing right hip open reduction and internal fixation.

References: Tomotsugu Tabata, MD, FACC, James D. Thomas, MD, FACC, Allan L. Klein, MD, FACC. Pulmonary Venous Flow by Doppler Echocardiography: Revisited 12 Years Later. JACC 2003; Vol.41, No.8:1243,Äì50



Liver

Liver – 1 Repeated doses of Heparin fail to correct Hypercoagulability - deficiency of Antithrombin due to poor synthetic function of the Liver

William P Dillon[1], Imad Riazuddin[1], Vanessa Wall[1], Ranganathan Govindaraj[1]

[1]University of Texas Health Science Center, McGovern Medical School, Houston, TX

Introduction: Hypercoagulability in a patient with end stage liver disease is not very common. Here we present the case of a cirrhotic patient with TIPS who was hypercoagulable and had clotting of her intrahepatic shunts. She was scheduled for mechanical clearance of the clotted shunts by interventional radiologists. During the procedure her portal vein, stent, and sheath clotted repeatedly despite multiple administrations of unfractionated IV heparin. Resistance to heparin in this patient was most likely due to a deficiency of antithrombin due to end stage liver disease. We used bivalirudin, fixed the hypercoagulable state, and the patient went into bleeding diathesis. Patients presenting with liver cirrhosis have problems with coagulation due to poor synthetic function, leading to a deficiency of the factors necessary for coagulation. Generally we look at the extrinsic pathway factors responsible for coagulation (2, 7, 9, and 10) and monitor the PT, INR, and albumin levels to assess the synthetic function of the liver. Patients with liver cirrhosis and portal hypertension are also at an increased risk of bleeding due to increased sequestration of platelets by the spleen. A TIPS is used to reduce portal hypertension and allow portal blood an alternate path to return to systemic circulation[1].

Methods: The patient is a 67-year-old woman with a past medical history of hepatocellular carcinoma, decompensated liver cirrhosis secondary to chronic hepatitis C, portal hypertension, and history of prior TIPS procedure. The patient presented with several days of abdominal pain, intractable vomiting, and melena. She was found to be anemic and acidotic with bleeding esophageal varices and admitted to the ICU in view of her variceal bleed. She underwent doppler ultrasound of her liver to evaluate TIPS patency which revealed thrombosis of her TIPS and main portal vein. During her stay in the ICU she developed acute worsening of her anemia requiring transfusion of multiple units of PRBCs. She underwent EGD which revealed significant duodenopathy thought to be secondary to her portal vein thrombosis which was due to shunt thrombosis. Her episodes of melena continued and interventional radiology decided to proceed with mechanical thrombectomy of her shunt. During the procedure the patient's portal vein, stent, and sheath clotted repeatedly. We used several doses of unfractionated heparin to prevent new clot formation. Multiple attempts were made to clear the clots (22000u of heparin in total) but despite this, the clots persisted and ACT values did not improve (Figure 1). The decision was made to start bivalirudin with an initial bolus of 0.75 mg/kg followed by an infusion at 1.75 mg/kg/min with subsequent achievement of anticoagulation. The procedure was completed without recurrence of thrombosis. Mechanical thrombectomy was achieved but post-procedure the patient developed bleeding diathesis at all access sites and had elevated clotting analyses (INR >10, PT >100, PTT >200). Hematology was consulted for assistance in managing post-op anticoagulation to prevent TIPS re-thrombosis in the setting of coagulopathy. All anticoagulation was held and the patient was given 10 mg Vitamin K, 1 unit of fresh frozen plasma, and 1 unit of cryoprecipitate. The patient was monitored in the ICU and over the next 24 hours INR returned to normal therapeutic levels (2.45, see Table 1). The patient was started on a heparin drip which was tolerated well. She was transferred to the floor and was later discharged on Eliquis. She is currently pending liver transplant evaluation.

Conclusion: Thrombosis of TIPS is possible when it is malpositioned, causing a slowing of circulation in the portal venous system[2]. Hypercoagulable state is rarely a concern in end stage liver disease patient and given the resistance to heparin seen during the thrombectomy, a lack of antithrombin due to poor synthetic function of the liver is the only possible explanation. Bivalirudin, which acts by preventing thrombin formation, worked efficiently and increased the patient's INR and PTT, which prevented further clot formation and maintained shunt patency.

References: 1) Luca A, Miraglia R, Caruso S, et al. Short- and long-term effects of the transjugular intrahepatic portosystemic shunt on portal vein thrombosis in patients with cirrhosis. Gut 2011;60:846-52. 2) Lv, Yong, Guohong Han, & Daiming Fan. 'Thrombosis after transjugular intrahepatic portosystemic shunt: an ominous sign?.' AME Medical Journal [Online], 2.3 (2017)

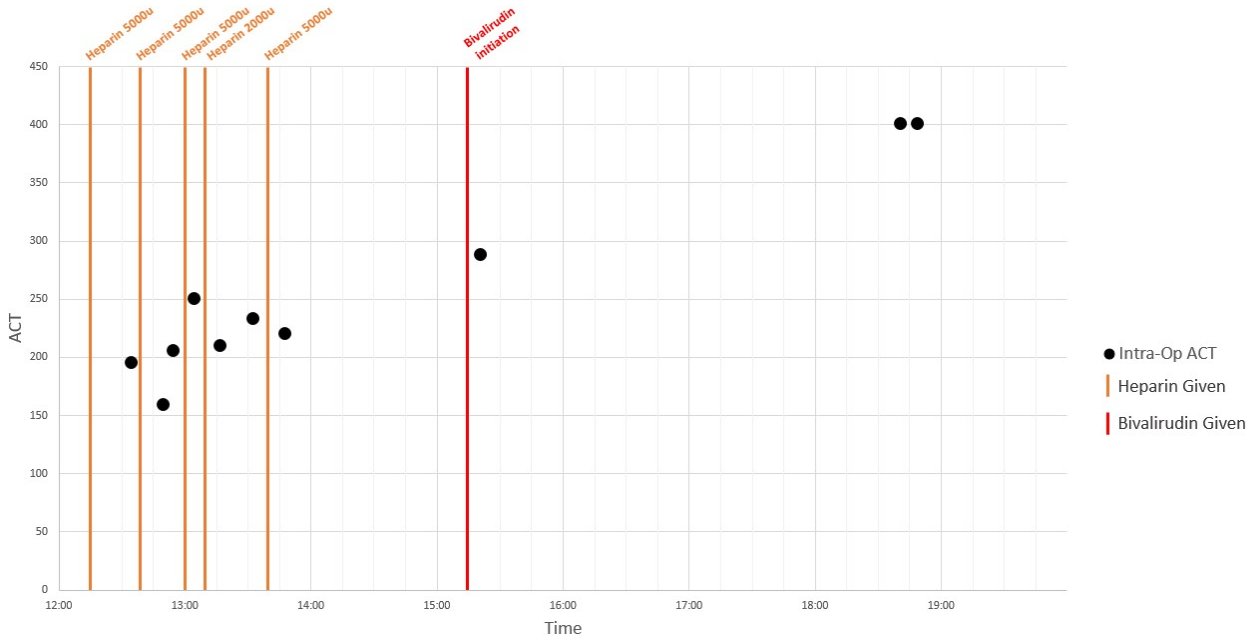


Figure 1: Intra- and post-operative ACT values during TIPS revision. The patient's portal vein, TIPS, and sheath repeatedly clotted even with repeated administrations of IV unfractionated heparin. Anticoagulation was achieved after giving bivalirudin with an initial bolus of 0.75 mg/kg followed by an infusion at 1.75 mg/kg/min. The procedure was completed without recurrence of thrombosis.

Coagulation Parameter	Baseline	Post-Operative	s/p Vit K, Cryo, FFP
PT	18.7	>100	25.7
PTT	34.3	>200	61.2
INR	1.61	>10	2.45

Table 1: Elevated clotting analyses after initiation of bivalirudin for intra-operative anticoagulation. Post-operatively the patient developed bleeding diathesis at all access sites. Coagulation parameters returned to therapeutic levels after administering 10 mg Vitamin K, 1 unit of Cryoprecipitate, 1 unit of Fresh Frozen Plasma, and holding all anticoagulation for 24 hours.

Liver - 2 Successful Intraoperative Implementation of VA-ECMO for Acute Cardiogenic Shock as a Bridge Through Orthotopic Liver Transplantation

Milap Rakholia[1], Amy Kloosterboer[1], Marianne C Chen[1]

[1]Stanford University, Stanford, CA

Introduction: Extracorporeal membrane oxygenation (ECMO) has been used successfully to bridge end stage liver disease patients to orthotopic liver transplantation (OLT) or used as rescue therapy in the post-transplant period. Few cases have been reported on initiation of intraoperative veno-arterial ECMO during liver transplantation. Here we describe the successful utilization of and recovery from VA-ECMO for unexpected intraoperative cardiogenic shock in a patient without significant cardiac or pulmonary comorbidities as a bridge to get the patient safely through OLT.

Methods: A 54-year-old man with a history of alcoholic cirrhosis, stage 2 kidney disease, and mild hepatopulmonary syndrome on room air presented from home for OLT. He had an unremarkable 12-lead electrocardiogram (EKG), normal findings on a transthoracic echocardiogram, and a negative coronary CT angiogram (CCTA). Following induction of anesthesia, intubation, and initiation of CRRT for kidney disease, incision was made and 9.2L of ascites was drained. Fifteen minutes later, the patient became hypotensive with minimal response to multiple boluses of fluid, vasopressin, and epinephrine. ST depressions were noted on EKG. Septal wall motion was depressed with newly reduced LV and RV systolic function on transesophageal echocardiogram (TEE). His mean arterial pressure (MAP) improved with epinephrine 1mg which then led to a wide complex tachycardia, HR 130s-140s, requiring increasing doses of vasopressors to maintain MAP. Amiodarone and lidocaine were administered with conversion to sinus bradycardia in the 40s requiring 1mg atropine. Arterial blood gas showed no significant electrolyte abnormalities. Formal TEE by a cardiac anesthesiologist demonstrated reduced LVEF to 15-20%, non-territorial wall motion abnormalities (WMA), and a mildly dilated right ventricle. Cardiology consultants deemed there was no indication for coronary angiography at this time due to a low suspicion for a coronary artery lesion given a normal pre-operative CCTA. The patient was supported with epinephrine infusion, milrinone infusion, and inhaled nitric oxide. It was unclear the etiology of his acute cardiogenic shock. A discussion between the transplant surgeon, primary anesthesiologist, cardiac anesthesiologist, and cardiac surgeon determined that there was no indication of irreversible cardiac dysfunction at this point. However, it was uncertain if the patient would be able to hemodynamically tolerate reperfusion of the new liver graft. Thus, we placed the patient on femoral-femoral VA-ECMO to support him through OLT. 4,000 units of heparin were given to prevent circuit clot. Portal-venous bypass was achieved via the ECMO circuit and transplantation proceeded uneventfully. On 0.12 mcg/kg/min epinephrine, his LVEF improved to 40%. After graft reperfusion, he was weaned off ECMO support (total time 143 mins). In total he was transfused 43 units packed red blood cells, 41 units fresh frozen plasma, 11 units platelets, and 8 units cryoprecipitate. His cardiac function improved further and he was transferred to the ICU without ionotropic support. TTE on POD 0 revealed normal biventricular function. Amiodarone was weaned without repeat tachyarrhythmias. He was extubated and weaned off iNO POD 1, off dialysis by POD 3, transferred to the floor POD 5, and discharged home on POD 11.

Conclusion: VA-ECMO provided a bridge for OLT and cardiac recovery from acute cardiogenic shock in this patient. Most case reports and case series on perioperative ECMO involve its utilization to manage pre- and post-transplantation complications.1-4 The need for VA-ECMO intraoperatively during OLT is rare with one case series describing only one instance in 1,792 OLTs.1 By initiating a multidisciplinary discussion in our patient, we determined we could either cancel the surgery and recover the patient in the ICU, initiate VA-ECMO but not proceed with transplantation, or utilize ECMO as a bridge through transplantation. Our patient, without prior cardiac comorbidities, was expected to recover function and delaying OLT would only serve to complicate his cardiac recovery with ongoing hepatic dysfunction. As such, we mobilized multiple teams to initiate VA-ECMO and proceeded with the operation. Our case provides a useful addition to evidence that patients with acute

hemodynamic collapse during OLT should be considered for VA-ECMO given the chance of successful recovery of cardiopulmonary function.

References: 1.Transplantation. 2019 Aug;103(8):1568-1573 2. Crit Care. 2011;15(5):R234 3. Minerva Anestesiol. 2017 Dec;83(12):1336-1337 4. BMJ Case Rep. 2017 Nov 1;2017:bcr2017221381

Liver - 3 Intraoperative management of hyponatremia in a liver transplant patient with fluid selection and CRRT dialysis

Matthew Palascak[1], William J Daly[1]

[1]Ochsner, Jefferson, LA

Introduction: A 60-year-old male with decompensated NASH cirrhosis and a MELD of 24 along with treated hypothyroidism presented for liver transplant surgery. Patient had decompensated hepatic cirrhosis complicated by hepatic encephalopathy and was admitted approximately one month prior to surgery for suspected small bowel obstruction that resolved without surgical intervention and allowed him recovery time to be listed for liver transplantation. He also had a complex abdominal surgery history that included an appendectomy, open cholecystectomy, ostomy creation (from prior small bowel obstruction) and takedown, and hernia repair. The patient was admitted from home and was found to have a serum sodium of 125 mmol/L and creatinine was 1.2 mg/dL and had no symptoms of encephalopathy. Initial sodium in the operating room was 123 mmol/L. Intraoperative dialysis was discussed and used during the case to limit the rise in serum sodium. Additionally, lactated ringers and D5W with 0.45% saline were used as intraoperative fluids for the case, both serving to limit the amount of sodium administered to the patient. The goal of this case report is to discuss the management of hyponatremia in a liver transplant patient with the use of hemodialysis and selective fluid management. The initial sodium upon arrival to the intensive care unit was 129 mmol/L. Perioperative concerns for the case included central pontine myelinolysis after liver transplantation, secondary to rapid change in serum sodium. By limiting the change in sodium, the patient remained neurologically intact, followed commands when off sedation, and was extubated without issue on post-operative day five.

Methods: A case report of one patient with hyponatremia presenting for liver transplantation surgery and intraoperative management of hyponatremia with dialysis and fluid selection.

Conclusion: By limiting the change in sodium, the patient remained neurologically intact, followed commands when off sedation, and was extubated without issue on post-operative day five.

References: Ginès P, Guevara M. Hyponatremia in cirrhosis: pathogenesis, clinical significance, and management. Hepatology. 2008 Sep;48(3):1002-10. doi: 10.1002/hep.22418. PMID: 18671303. Londoño MC, Guevara M, Rimola A, Navasa M, Taurà P, Mas A, et al. Hyponatremia impairs early posttransplantation outcome in patients with cirrhosis undergoing liver transplantation. Gastroenterology 2006; 130: 1135–1143. Dawwas MF, Lewsey JD, Neuberger JM, Gimson AE. The impact of serum sodium concentration on mortality after liver transplantation: a cohort multicenter study. Liver Transpl 2007; 13: 1115–1124.

Liver - 4 Spontaneous Intracerebral Hemorrhage after Orthotopic Liver Transplant and Duodenal Switch Reversal

Ayden D Zarkhah[1], Hokuto Nishioka[2]

[1]University of Illinois at Chicago, Chicago, IL,
[2]University of Illinois Hospital, Chicago, IL

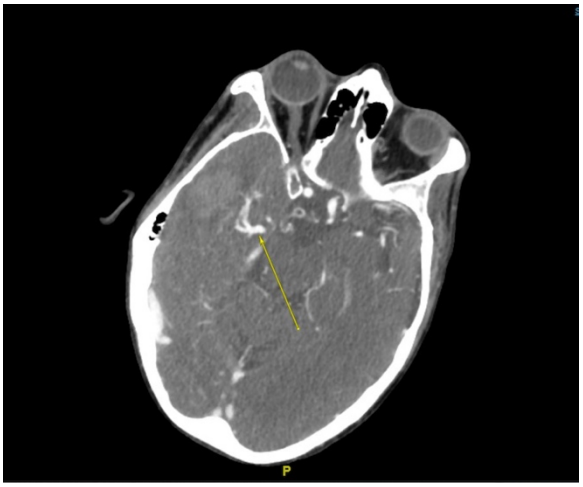
Introduction: Developmental venous anomalies(DVA) are estimated to have an incidence of up to 2.6% in the general population (1). DVAs are dilated medullary veins which converge centrally into a larger collecting venous system that drain into superficial or deep venous system within the brain. These anomalies are generally benign and asymptomatic until certain conditions arise causing altered hemodynamics – including orthotopic liver transplantation (OLT) - resulting in venous hypertension and congestion with a possible devastating complication of intracerebral hemorrhage (ICH).

Methods: A 44 year-old male presented for OLT for end stage liver disease secondary to non-alcoholic steatohepatitis - Model for End-Stage Liver Disease 32. Past medical history also included chronic kidney disease, anemia of chronic disease, and history of morbid obesity treated with a duodenal switch. Pre-operative workup for liver transplantation was unrevealing in regards to a transthoracic echocardiogram, dobutamine stress test, electrocardiogram, and routine chest imaging. Neuroimaging was not part of the preoperative work up for transplant. Intraoperatively, the patient was appropriately resuscitated with the use of blood products, crystalloids and colloids, and hemodynamically supported with vasopressors. The patient was transported to a surgical intensive care unit and extubated on postoperative day (POD)3 to room air. On POD 9 a rapid response was initiated for unresponsiveness, left facial droop, and two subsequent generalized tonic-clonic seizures. The patient was emergently intubated for airway protection and treated with benzodiazepines and anti-epileptics. Computed tomography (CT) head demonstrated a right anterior temporal cortical hemorrhage, and a CT angiography showed a concern for a right sided vascular malformation. Lab values around this acute event were platelets of 161 and an INR of 1.1. Vitals surrounding this event were systolic blood pressures (SBP) less than 140. The day after the ICH - POD 10 - patient was alert and following commands while intubated and off sedation. Diagnostic cerebral angiogram showed excessive perfusion throughout the right temporal lobe and a presence of a slightly early draining vein within the lobe, representing a DVA. However, no vascular malformation was identified. The patient was subsequently extubated with his remaining recovery complicated by waxing and waning of his mentation with recurrent subclinical seizures on EEG. Ultimately, the patient was discharged to a skilled nursing facility and no further intervention was performed regarding the DVA. The resulting sequelae from the ICH included periodic somnolence and agitation, but a functioning transplanted liver.

Conclusion: New-onset hypertension is an important element of the post-liver transplant recovery. The developing hypertension is attributed to the use of immunosuppressive agents in addition to the vast changes to the circulatory abnormalities preceding transplant (2). As demonstrated in this patient, unmasking of pathologies susceptible to hypertension can lead to devastating consequences. Within a single high volume institution, 5.2% of OLT recipients developed an intracranial hemorrhage within 12 months ,Äì the majority within 1 month of transplant and an overall mortality of 45% within 1 year (3). Additionally, the study indicated that blood pressure intervention might be reasonable when the change of SBP is greater than 28 mmHg pre and post-transplant. Ultimately, post-operative blood pressure control is a significant modifiable risk factor as demonstrated by this patient as intracranial vascular abnormalities are often undetected.

References: 1. Mullaguri, N., Battineni, A., Krishnaiah, B., Migdady, I., & Newey, C. R. (2019). Developmental Venous Anomaly Presenting with Spontaneous Intracerebral Hemorrhage, Acute Ischemic Stroke, and Seizure. Cureus, 11(8), e5412. https://doi.org/10.7759/cureus.5412 2. Sandra J. Taler, Stephen C. Textor, Vincent J. Canzanello, Lora Schwartz, Michael K. Porayko, Russell H. Wiesner, Ruud A. F. Krom, Hypertension after liver transplantation: A Predictive role for pretreatment hemodynamics and effects of isradipine on the

systemic and renal circulations, American Journal of Hypertension, Volume 13, Issue 3, March 2000, Pages 231–239, https://doi.org/10.1016/S0895-7061(99)00171-5 3. Gallagher, T. K., Thomas, K. A., Ladner, D. P., Ganger, D., Sorond, F. A., Prabhakaran, S., Abecassis, M. M., Fryer, J. P., & Liotta, E. M. (2018). Incidence and Risk Factors of Intracranial Hemorrhage in Liver Transplant Recipients. Transplantation, 102(3), 448–453. https://doi.org/10.1097/TP.0000000000002005



Liver - 5 Refractory post-operative hepatic encephalopathy treated with Molecular Adsorbent Recirculating System (MARS)

Jordan S Pogu[1], Mark Keegan[1]

[1]Mayo Clinic, Rochester, United States of America

Introduction: Detoxification, biosynthesis, and hemodynamic regulation are impaired in the absence of hepatic integrity (1), causing significant morbidity and mortality (2). Temporizing to spontaneous hepatic recovery or, when applicable, liver transplant, is the goal of extracorporeal liver support devices (3). Molecular Adsorbent Recirculating System (MARS) filters protein-bound and water-soluble compounds across a membrane via an albumin-enriched dialysate (4). MARS has been shown to reduce serum ammonia and bilirubin, with improved recovery from hepatic encephalopathy (HE), although no survival benefit has been proven (5) and there remains a paucity of data with heterogeneous results (2).

Methods: A 57-year-old female with pancreatic adenocarcinoma underwent a complex total pancreatectomy with peri-aortic lymphadenectomy, portal SMV confluence resection and reconstruction, splenectomy, gastrojejunostomy, and hepaticojejunostomy. Intraoperative liver biopsy showed severe steatosis with minimally active steatohepatitis and focal pericellular fibrosis; hepatectomy was not performed. After an unremarkable initial post-operative recovery, she was transferred to the ICU on post-operative day (POD) 11 for progressively worsening mental status associated with hyperammonemia and hyperbilirubinemia (Figures 1-3). A head CT demonstrated mild cerebral edema. She was diagnosed with grade IV HE and intubated for airway protection. It was hypothesized that perioperative impairment of liver blood flow, coupled with 10 days of total parenteral nutrition, exacerbated her hepatocellular dysfunction, precipitating an episode of severe HE. Despite administration of lactulose with adequate stool production, rifaximin and zinc, her clinical and biochemical status remained unchanged. MARS therapy was initiated with 7-hours of daily treatment over 3 sequential days, in addition to standard medical therapy. After the third MARS treatment, her HE improved to grade I with downtrending bilirubin and ammonia, allowing for extubation on the subsequent day. She was discharged from the ICU after 7 days and dismissed from the hospital in satisfactory clinical condition on POD 28.

Conclusion: This case is an example of a patient with acute on chronic liver failure whose hyperammonemia, hyperbilirubinemia and HE improved following MARS therapy coupled with standard medical treatment. While MARS has not demonstrated survival benefit (5), there may be a role for MARS in patients who have proven refractory to conventional therapy alone, and in whom hyperammonemia and hyperbilirubinemia are potentially reversible. MARS may represent an advance in palliating liver failure, but additional research remains necessary to ensure efficacy, standardization and cost-effectiveness (2).

References: 1. N Engl J Med. 2016 Oct 27;375(17):1660-1670. 2. Clin Liver Dis. 2014 Nov;18(4):945-56. 3. Best Pract Res Clin Gastroenterol 2012; 26: pp. 17-26. 4. Hepatology 2007; 46: pp. 1853-1862. 5. JAMA 2003; 289: pp. 217-222.

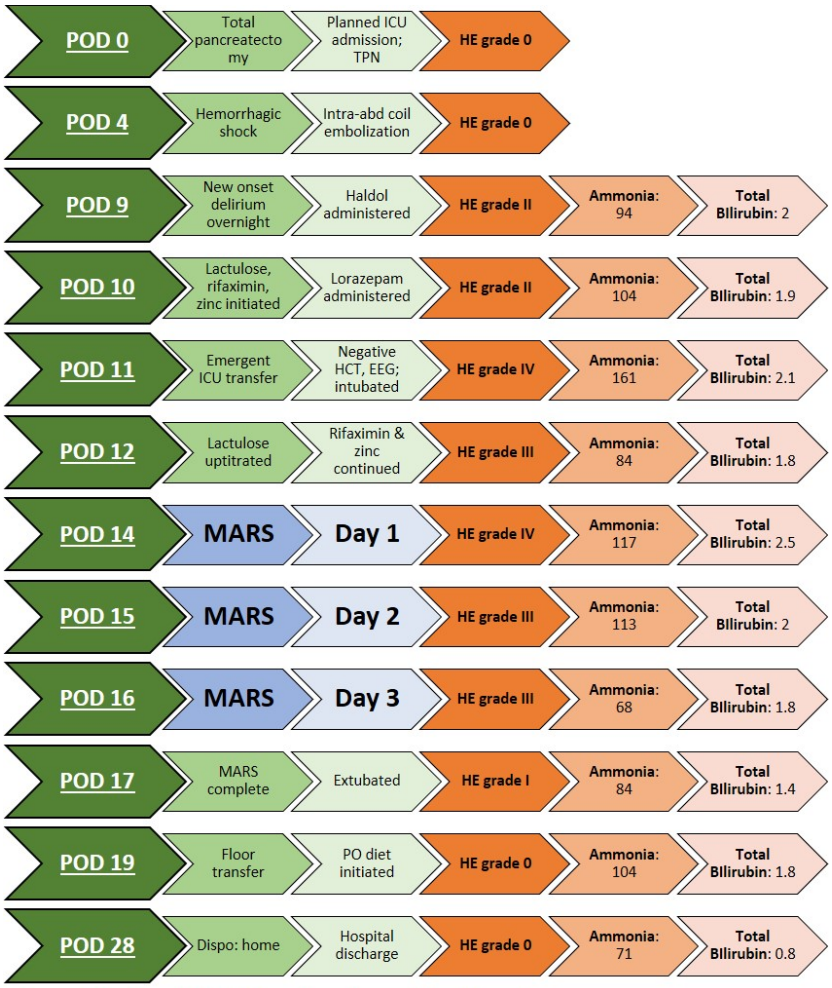


Figure 1: Pertinent clinical events and associated lab values.

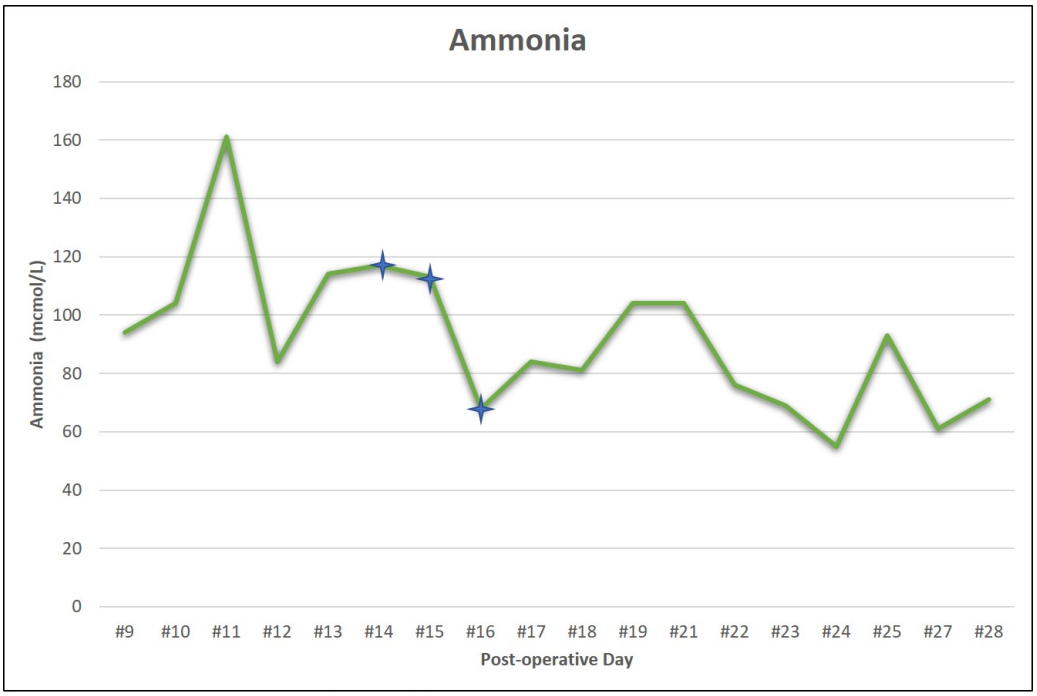


Figure 2: Ammonia trend during hospitalization – ✦ denotes MARS therapy.

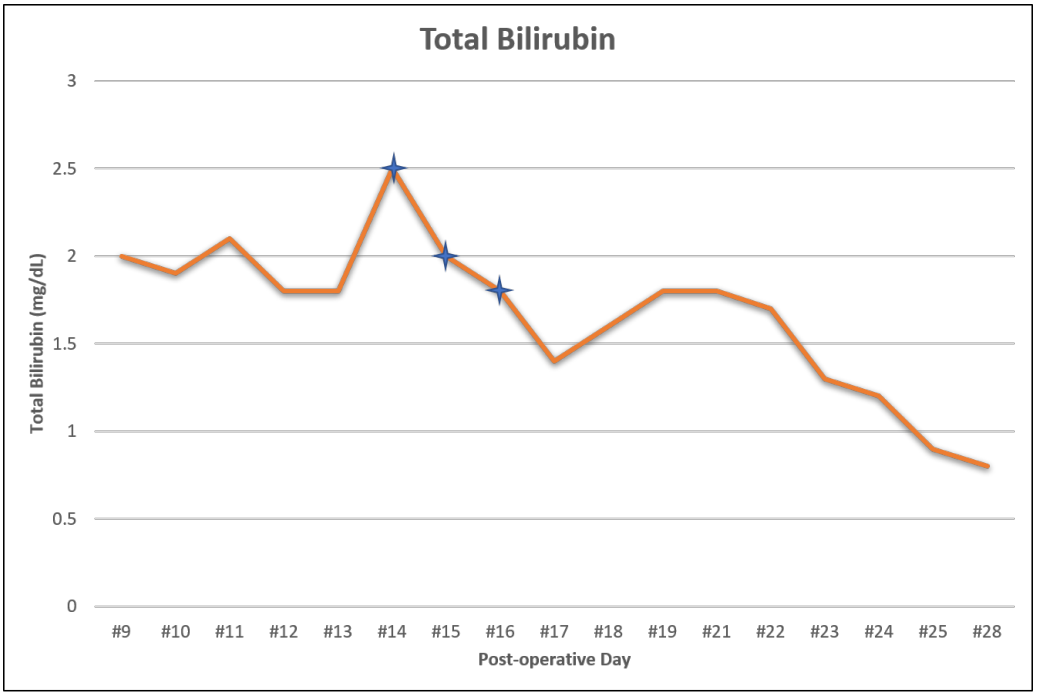


Figure 3: Total bilirubin trend during hospitalization – ✦ denotes MARS therapy.

Neuroscience in Anesthesiology and Perioperative Medicine

Neuroscience in Anesthesiology and Perioperative Medicine - 1 Adult Mitochondrial Disease: The Anesthetic Management Conundrum

Adrienne Ligouri[1]

[1]*Temple University Hospital, Haddon Heights, NJ*

Introduction: Case Report

Methods: 46 year old female with multiple sclerosis and mitochondrial disease with exacerbation of mitochondrial encephalopathy due to port site infection causing bacteremia and left shoulder infection with a planned procedure for an arthroscopic shoulder incision and drainage. Her mitochondrial encephalopathy included symptoms of migraines, stroke like episodes, episodes of confusion with possible seizures, peripheral nerve involvement, optic nerve involvement and fatigue. Mitochondrial diseases typically cause impaired energy metabolism that lead to metabolic acidosis and cell injury. Like this patient, the symptoms tend to involve various organ systems, such as the heart, brain, skeletal muscle, sensory organs and endocrine cells. She was 5'4', and 68kg. Current medications included: Oxygen 4L, Advair HFA, Albuterol, Alprazolam, L- Arginine, Diclofenac, Misoprostol, Bupropion, Carisoprodol, Coenzyme Q10, Ubiquinol, Dexlansoprazole, Duloxetine, Esomeprazole, Folic Acid, Hydromorphone, Levocarnitine, Magnesium Oxide, Montelukast, Niacin, Ocrelizumab, Restoril, Sodium Chloride Infusions, Thiamine, Topiramate, Zofran, Zolpidem. Most patients with mitochondrial disease, depending on their prognosis and severity of disease, die before the age of 16. There are very few case reports of anesthesia management in adult patients, which makes the preferred anesthesia technique unclear.1 Upon review of patient history, she stated she had general anesthesia in the past for a cholecystectomy and tonsillectomy. This gave some reassurance she will do fine, despite her current encephalopathic state. The patient had been kept npo, and was continued on D5 ¬Ω normal saline, to decrease risk of hypoglycemia.2 Patient was premedicated with Midazolam 2mg and Fentanyl 50mcg for anxiolysis. The goal of the anesthetic plan was to maintain core temperature, normal blood glucose, maintain oxygen saturation and blood pressure. Plan was for general anesthesia with endotracheal intubation. Nerve block was not considered due to her bacteremia and septic joint. Induction proceeded with Ketamine 60mg, Propofol 50mg, Fentanyl 150mg, and Lidocaine 100mg. No neuromuscular blockade was given. Patient maintained respiratory drive. Intubation was achieved using a glidescope and endotracheal tube was placed when vocal cords were visually open. Anesthesia was maintained using Isoflurane, and no further analgesia was needed. Hydrocortisone 100mg stress dose was given, and Ondansetron 4mg antiemetic was given. D5 ½ NS was maintained throughout the procedure.3 Normothermia was maintained with a forced air warming blanket. Preoperative blood glucose was 87, and postoperative was 139. Anesthetic gas was stopped, and patient awakened with minimal pain and discomfort. Upon chart review patient did well post-op without any worsening of her mitochondrial encephalopathic symptoms, and sent home with intravenous antibiotics.

Conclusion: Successful anesthetic management of patient with mitochondrial disease without any worsening of her mitochondrial encephalopathic symptoms, and sent home with intravenous antibiotics for presenting bacteremia.

References: Mitochondrion. 2016 Jan 1;26:26-32 Journal of Inborn Errors of Metabolism and Screening. January 2017 Paediatr Anaesth. 2013 Sep;23(9):785-93

Neuroscience in Anesthesiology and Perioperative Medicine - 2 The safety use of sugammadex in a patient with dermatomyositis : A case report

Daiki Takekawa[1], Satoshi Uchida[1], Kazuyoshi Hirota[2]

[1]Hirosaki University Hospital, Hirosaki, Japan, [2]Hirosaki University Graduate School of Medicine, Hirosaki, Japan

Introduction: Dermatomyositis is an inflammatory myopathy characterized by muscle weakness and typical skin symptoms (1). The patients with some types of neuromuscular disorders are markedly sensitive to muscle relaxants (2), and the safety of sugammadex in these patients has not been established. Thus, anesthetic management for patients with dermatomyositis must be done with caution. Here, we report a successful anesthetic management for a patient with dermatomyositis, which presents the safety of sugammadex to reverse rocuronium with the use of muscle relaxant monitor.

Methods: We obtained a written informed consent from the patient for the publication of this case report. The patient was a 57-year-old female (163 cm, 50 kg) with a history of dermatomyositis diagnosed several years ago. Although she has mild lower limb weakness and dysphagia, she doesn't require any assistances with her activities of daily living. The patient was scheduled to undergo the abdominal total hysterectomy with bilateral salpingo-oophorectomy and pelvic lymphadenectomy for suspected ovarian cancer. She had anemia (hemoglobin, 9.0 g/dL), but she did not have any other abnormal medical history or laboratory results. Anesthesia was uncomplicatedly induced by propofol (120 mg), remifentanil (0.5 μg/kg/min), and rocuronium (30 mg) followed by tracheal intubation. Rocuronium was administered in 3 divided doses while confirming that there was no abnormal reaction to rocuronium by using train-of-four (TOF) monitoring. Anesthesia was maintained by propofol (5–6 mg/kg/h), remifentanil (0.1–0.3 μg/kg/min), and rocuronium (5–6μg/kg/min). Additionally, we administered fentanyl (200 μg) and acetaminophen (750 mg) intravenously for postoperative analgesia. We maintained TOF counts 0-1 during the surgery. The tumor has invaded the rectum, and rectal resection and colostomy were added. The operation lasted 6 h and 12 min and the total blood loss was 2750 g. The patient got blood transfusion of red blood cell (6 units) and fresh frozen plasma (6 units) during the surgery. We stopped the continuous infusion of rocuronium before the skin suture, and the TOF ratio was 29 % at the end of the surgery. Thus, we administered sugammadex (100 mg) and confirmed TOF ratio 100 %. After then, we stopped the continuous infusion of propfol and remifentanil, and the patient emerged from anesthesia and extubated uncomplicatedly. The postoperative course was uneventful without any respiratory complications and progression of muscle weakness. The patient was discharged on the 17th postoperative day.

Conclusion: We successfully managed the anesthesia of a patient with dermatomyositis undergoing abdominal surgery using sugammadex to reverse rocuronium with the use of TOF monitor. Further research to confirm the safety of rocuronium and sugammadex in the patients with dermatomyositis are needed.

References: 1. Findlay AR, Goyal NA, Mozaffar T. An overview of polymyositis and dermatomyositis. Muscle Nerve. 2015;51:638-56. 2. Schmitt HJ, Muenster T. Anesthesia in patients with neuromuscular disorders. Minerva Anestesiol. 2009;75:632–7.

Neuroscience in Anesthesiology and Perioperative Medicine - 3 Transnasal humidified rapid insufflation ventilatory exchange (THRIVE) to improve patient safety and surgical field during awake craniotomies in obese patients

Sara Feldman[1], Vijay Ramaiah[1]

[1]Duke University School of Medicine, Durham, NC

Introduction: Transnasal humidified rapid insufflation ventilatory exchange (THRIVE) has been used during difficult airway management and laryngeal surgeries due to prolonged apnea time and enhanced clearance of carbon-dioxide (CO2).1,2 Awake craniotomy in patients with high body mass index (BMI) presents unique challenges of airway management and maintaining patient comfort, while simultaneously requiring intraoperative mapping of intricate cerebral cortex and brain relaxation. We describe two cases of awake craniotomy during which THRIVE was deployed.

Methods: Case 1: Patient is a 33-year-old female, BMI 37.8 (99.8 kg), with diabetes, seizures, mood changes and speech issues, with a (6 x 5.5 cm) left frontal-temporal insular tumor. She was scheduled for awake craniotomy (Asleep-Awake-Asleep). After establishing standard ASA monitors, anesthesia was induced with propofol and fentanyl. An LMA Supreme size 4 was placed, and position confirmed with flexible intubating bronchoscope (FIB). Positive pressure ventilation (PPV) with pressure control ventilation (PCV) was used, with peak inspiratory pressure (PIP) 25 cmH2O. Arterial line was placed and patient placed in left lateral position with Mayfield head pins. Anesthesia was maintained with propofol, remifentanil and dexmedetomidine infusions. After dural opening and completion of intraoperative asleep motor mapping, all anesthetic agents were turned off, and the LMA removed when patient breathing adequately and following commands. We deployed THRIVE (Optiflow, Fisher and Paykel) as soon as the LMA was removed at 60 L/min oxygen. Patient required fentanyl bolus and remifentanil infusion during awake motor and speech mapping. THRIVE decreased to 30-40L/min during awake state. There were no significant changes in brain relaxation compared to PPV. After mapping, patient was pre-oxygenated with THRIVE 60-70 L/min for 10 minutes, and after induction and neuromuscular blockade, a Fastrach LMA (Teleflex) Size 4 was placed, and regular endotracheal tube placed through it with aid of FIB and Aintree exchange catheter (Cook Medical). THRIVE was used for entirety of airway management. Patient had uneventful surgery with resection of tumor and intraoperative MRI, and extubated at end of case. Case 2: Patient is a 34-year-old male, BMI 36.5 (102.5 kg), with recent seizure and posterior left frontal brain tumor scheduled for awake craniotomy (Asleep-Awake). After establishing standard ASA monitors, anesthesia was induced with propofol and fentanyl. LMA Supreme size 5 was placed. Patient placed in left lateral position with head in Mayfield pins. Anesthesia was maintained with propofol and remifentanil infusions. Patient ventilated with PCV with PIP 16-20 cmH2O. After dural opening and brain cortex exposure, all anesthetic agents were stopped and LMA Supreme was removed when patient was breathing adequately and following commands. We deployed THRIVE as soon as LMA was removed. THRIVE started at 60 L/min and decreased to 40 L/min gradually with FiO2 0.5 during motor/language testing and tumor resection. Patient was comfortable for awake motor and speech testing with fentanyl supplementation. After tumor resection, sedation with propofol and remifentanil infusions was initiated. THRIVE with maintained with oxygen flow 60 L/min with patient spontaneously ventilating. Oxygen was decreased to 30% during electrocautery use. Patient had good brain relaxation conditions and remained comfortably sedated. At the end of surgery, sedation was stopped and THRIVE discontinued.

Conclusion: We deployed THRIVE successfully in two patients with high BMI undergoing awake craniotomy. THRIVE helped increase patient comfort during sedation while maintaining adequate oxygenation and ventilation in spontaneously breathing patients. The brain was adequately relaxed and we had no airway obstruction. In awake patients, humidified gas decreases patient discomfort, while additionally limiting mucosal injury and impairment of ciliary functions. Good intraoperative functional mapping of intricate brain cortex was obtained. In patients with asleep-awake-asleep craniotomy, margin of safety was increased by providing apneic oxygenation and

ventilation. We were able to safely perform intubation in lateral positioning with head stabilized by Mayfield pins comfortably without any need to hurry.

References: 1.Anaesthesia 2015; 70(3): 323-9 2. British Journal of Anaesthesia 2017; 118(4): 610-617

Neuroscience in Anesthesiology and Perioperative Medicine - 4 Alpha matters - The correlation between alpha oscillatory activity during emergence from anesthesia and PACU delirium

Rieke Lutz[1], Gerhard Schneider[2], Paul S Garcia[3], Matthias Kreuzer[4], Stephan A Kratzer[5]

[1]Technical University of Munich (TUM), Munich, Germany, [2]Technical University Munich, Munich, na., [3]Columbia University, New York, NY, [4]Klinikum rechts der Isar, Technical University of Munich, Munich, Germany, [5]Technical University Munich School of Medicine, Munich, BY

Introduction: Previous research described the relationship between type of electroencephalogram (EEG) pattern during the emergence from general anesthesia (GA) and the risk to develop delirium in the postoperative anesthesia care unit (PACU-D) [1]. To understand the difference between patients starting anesthesia emergence with an alpha-dominant and a delta-dominant EEG, we observe EEG patterns from patients undergoing elective surgery. We also monitored whether patients developed PACU-D in the postoperative period.

Methods: We included 167 patients who underwent a surgical procedure under GA. After being induced with propofol, 80 patients received volatile anesthetics and 87 patients received intravenous anesthetics for anesthesia maintenance. We equipped each patient with a 10-electrode EEG cap to record the EEG with 250Hz sampling rate throughout the intervention. We evaluated a possible case of PACU-D by using the CAM-ICU score to survey cognitive status 15 and 60 minutes after the emergence from GA.

Conclusion: 51 of the 167 patients started emergence with alpha-dominant EEG and 116 started with a delta-dominant EEG. From the 116 delta-dominant patients, 25 developed PACU-D. From the alpha-dominant patients, 7 out of 51 developed PACU-D. There was no significant in the occurrence of PACU-D between patients with either alpha- or delta- dominant EEG pattens (p=0.237). In the density spectral arrays (DSA, normalized) of anesthesia emergence, we found significant differences between patients with and without PACU-D in both groups. In the delta-dominant group, we observed that patients with PACU-D had significantly less alpha power throughout the first half of emergence (Figure 1A). This observation also applies for the patients starting their emergence with an alpha-dominant EEG. In addition, patients with PACU-D and alpha-dominant EEG showed significantly stronger power in the higher frequencies in the late stages of emergence earlier than the patients without PACU-D (Figure 1B).

We confirm the protective effect of strong alpha oscillatory activity during emergence from GA on postoperative neurocognitive disturbance [2], [3]. Further, we find that PACU-D patients may show an earlier cortical activation reflected by an earlier increase in the EEG power in the higher frequencies. Hence, our investigation helps to understand neurophysiological activity during anesthesia emergence and to describe EEG features that may be associated with the development of a postoperative neurocognitive disorder.

References: [1] Br J Anaesth. 2019 May;122(5):622-634, [2] Front Syst Neurosci 2017 May 8;11:24. [3] Front Syst Neurosci 2019 Oct 18;13:56.

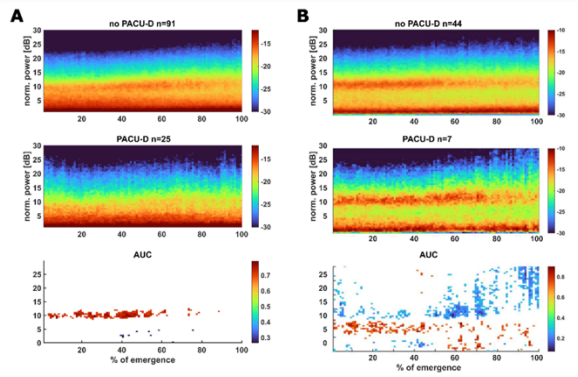


Figure 1: normalized density spectral array of patients without (top) and with (center) PACU-D. Red or blue colors in the bottom plots indicate significant differences between patients with and without PACU-D.
A) Patients starting anesthesia emergence with a delta-dominant EEG
B) Patients starting anesthesia emergence with a alpha-dominant EEG

Neuroscience in Anesthesiology and Perioperative Medicine - 5 Anesthetic Challenges for Deep Brain Stimulator Placement in a Chronic Pain Patient

Meredith Kushner[1], Neha Dasmunshi[2]

[1]Loyola University Chicago, Stritch School of Medicine, Chicago, IL, [2]Loyola University Medical Center, Chicago, IL

Introduction: Deep brain stimulator (DBS) placement can be an effective surgical treatment for a variety of movement disorders, psychiatric disorders, as well as chronic pain. To optimize target placement, DBS placement is commonly performed under MAC (Monitored Anesthesia Care) to allow for a period of an awake state during microelectrode recordings and stimulation testing. The anesthetic care for patients undergoing DBS placement under MAC can be challenging. The anesthetic plan must consider the patient's safety and comfort as well as medical comorbidities. This case describes the anesthetic challenges for a DBS placement in a patient with chronic pain related to intractable headaches and obstructive sleep apnea (OSA).

Methods: A 54-year-old male with past medical history significant for numerous concussions secondary to football and child abuse, OSA, and intractable chronic cluster headaches with bilateral tinnitus and facial pain presented for left posterior hypothalamic DBS lead placement under MAC. The patient was given 2 mg intravenous (IV) midazolam and 100 mcg IV fentanyl in the preoperative holding area to allow for placement of a stereotactic four-pin head fixation frame. After the patient's head computed tomography (CT) was completed, the patient was brought to the operating room and standard ASA monitors were placed. He was then positioned supine with his head elevated and the stereotactic headframe was secured. The bed was turned 180 degrees away from the anesthesia machine. He received a dexmedetomidine induction dose of 0.5mcg/kg (40 mcg) infused slowly over 10 minutes. The patient was breathing spontaneously with supplemental oxygen administered via face mask at 2 L/min. Procedural sedation was maintained with a dexmedetomidine infusion of 0.5 mcg/kg/hr and intermittent 10-20 mg IV propofol boluses. Prior to burr hole drilling by the surgeon, the patient was deeply sedated safely. Oxygen was temporarily discontinued with electrocautery use due to fire risk. Intraoperative microelectrode recording to confirm accurate lead placement was accomplished by reducing sedation intermittently so the patient could awaken and respond to the surgeon. After successful DBS placement, dexmedetomidine infusion was resumed and an additional propofol bolus was administered for deeper sedation during surgical closing of burr holes. The patient was transferred to the post-anesthesia care unit in stable condition. There were no intraoperative complications.

Conclusion: This case highlights the unique challenges for patients with chronic pain undergoing neurological interventions with MAC. Additionally, this case underlines considerations for safe airway management with stereotactic headframe in a patient with pre-existing OSA. Because this patient suffered from severe chronic pain due to his poorly-controlled daily headaches, which was refractory to years of failed medical management, we considered that he may require higher levels of sedation in order to adequately control his pain intraoperatively. This necessary depth of sedation was balanced with the need to rapidly wake the patient for key moments of communication with the surgeon during microelectrode recording to ensure safe and accurate DBS placement. The need to intermittently verbally communicate with the patient prohibited the anesthesia team from securing an artificial airway. Oversedation in this setting was concerning for respiratory depression and possible airway obstruction, particularly in a patient with OSA. Emergently securing an airway would have been difficult as the patient's head was fixed in the stereotactic frame and the surgical table was rotated 180 degrees away from the anesthesiologist. Extreme care was taken by both anesthesia and surgery teams to collaboratively position the patient so as to maximize airway safety and maintain spontaneous breathing while under varying degrees of MAC sedation with fixed head positioning. Dexmedetomidine infusion was selected for this case as it posed a low risk for respiratory depression. Propofol boluses were delivered in coordination with the surgery team to specifically maximize sedation depth during burr hole drilling, lead placement, and skin closure, while

minimizing sedation during microelectrode recordings. Ultimately, constant communication and strong rapport between the anesthesiologist, surgeon, and patient was critical to successfully navigating this case and ensuring patient safety.

References: 1. Continuing Education in Anaesthesia Critical Care & Pain. 9, 157–161 (2009). 2. Minerva Anestesiologica. 74, 393-408 (2008). 3. Canadian Journal of Anesthesia/Journal canadien d'anesthésie. 64, 517–529 (2017). 4. British Journal of Anaesthesia, 103, 152-165 (2009). 5. Current Opinion in Anesthesiology. 34, 563-568 (2021).

Neuroscience in Anesthesiology and Perioperative Medicine - 6 Delayed Diagnosis of anti-NMDAR Encephalitis

Catherine Janszen[1], Matthew J Hernandez[2]

[1]University of Kentucky, Lexington, KY, [2]University of Florida, Gainesville, FL

Introduction: Anti-N-methyl D-aspartate (NMDA) receptor (anti-NMDAR) encephalitis is an autoimmune encephalitis that occurs when antibodies against the NR1 subunit of the NMDAR cause internalization of the receptor. As a result, this can cause progressive decline of NMDAR associated synaptic functions, which may lead to neurologic and psychiatric alterations. Clinical presentations of anti-NMDAR can imitate schizophrenia and other psychotic spectrum disorders. This disorder can also be paraneoplastic and has been linked in young females presenting with ovarian teratoma. This case report describes a 24 year old female who presented with new onset seizures and auditory and visual hallucinations who was discovered to have anti-NMDAR encephalitis due to ovarian teratoma.

Methods: 24 year old female with past medical history of HTN, obesity, and prior ovarian teratoma who presented with migraine, new onset seizures, and auditory and visual hallucinations. She was started on levetiracetam for seizure prevention and admitted to the neurology service. There was initial concern for posterior reversible encephalopathy syndrome (PRES) due to CT head showing possible vasogenic edema in the occipital lobes but subsequent MRI brain was negative for PRES. EEG showed beta background, intermittent rhythmic theta and delta activity in L frontal-temporal region, but no clear epileptiform activity or seizure during the study. Urine drug screen was negative and withdraw from a substance was deemed unlikely. One week later, patient developed stupor, mutism, and oral dyskinesias. Further work up included lumbar puncture that showed lymphocyte predominance and an opening pressure of 54. Patient was started on acetazolamide and a fundoscopic exam was performed that showed bilateral grade IV disc edema. She was also started on acyclovir. Repeat MRI showed possible hyperintensity of the medial temporal lobes consistent with limbic encephalitis. Two days later, the patient had an episode of apnea associated with bradycardia and worsening stupor, resulting in intubation. Levetiracetam was increased due to concern that the episode was due to a seizure. Eventually, the patient's mother revealed she had a history of teratoma which prompted a CT C/A/P that showed a 12mm fat-containing nodule on the left ovary, making anti-NMDAR encephalitis likely. Patient completed a 5 day course of methylprednisolone and 6 rounds of IVIG. She went to the OR with surgical gynecology that week for diagnostic laparoscopy with left oophorectomy, and partial left salpingectomy. Pathology confirmed teratoma. General anesthesia with isoflurane was utilized for the case. CSF anti-NMDA Ab resulted positive a week later. Patient was ultimately hospitalized for one month. On discharge she was referred to genetics due to family history of teratoma. Four months post discharge, patient denied any further seizures and endorsed doing well overall.

Conclusion: Anti-NMDR encephalitis can be autoimmune or paraneoplastic and is more prevalent in females. Clinical presentation often mimics psychotic disorders such as schizophrenia or substance-induced psychosis, thus proper diagnosis is often delayed. Long term prognosis improves with early diagnosis and treatment and no admission into the ICU. Diagnosis ultimately requires confirmation of anti-NMDAR antibodies in the serum or CSF. Additional workup often includes MRI, EEG studies, and ultrasound to screen for teratoma. If discovered, early tumor resection can result in significant improvement of neurologic status. Several anesthetic drugs that interact with the NMDAR, like ketamine, propofol, and inhaled agents including nitrous oxide and sevoflurane, may pose risk in these patients as they may act unpredictably and affect activation of the CNS by excitatory or inhibitory neurons. Sevoflurane inhibits NMDA-induced mitochondrial membrane depolarization and NMDA-gated currents, which may cause clinical deterioration. In these cases, TIVA should be considered as there is less effect on NMDAR by propofol compared to volatile anesthetics. If appropriate for the procedure, regional anesthesia should also be considered in these patients. Regardless of tumor presence, patients should be treated with immunotherapy including IV immunoglobulins, corticosteroids, or plasma exchange. Overall, anti-NMDAR encephalitis a disease

that has a good prognosis when treated early and increased awareness among providers is imperative.

References: Mitra AD, Afify A. Ovarian teratoma associated Anti-N-methyl-D-aspartate receptor encephalitis: a difficult diagnosis with a favorable prognosis. Autops Case Rep. 2018;8(2):e2018019. Published 2018 Apr 18. Ford B, McDonald A, Srinivasan S. Anti-NMDA receptor encephalitis: a case study and illness overview. Drugs Context. 2019;8:212589. Published 2019 Aug 30. Yan, Bin et al. “Teratoma-associated anti-N-methyl-D-aspartate receptor encephalitis: A case report and literature review.” Medicine vol. 98,21 (2019): e15765. Wada N, Tashima K, Motoyasu A, et al. Anesthesia for patient with anti-N-methyl-D-aspartate receptor encephalitis: A case report with a brief review of the literature. Medicine (Baltimore). 2018;97(50):e13651. Shaikh MA, Dhansura T, Gandhi S, Shaikh T. Anaesthetic management of a patient with anti-NMDA receptor encephalitis. Indian J Anaesth. 2015;59(4):248-250.

Obstetric Anesthesiology

Obstetric Anesthesiology - 1 Obstetric Anesthetic Management in a Patient with Concurrent Klippel-Feil and Goldenhar Syndrome.

Westin R Tom[1], David T Chin[1], Peter Yeh[1]

[1]University of California San Francisco, San Francisco, United States of America

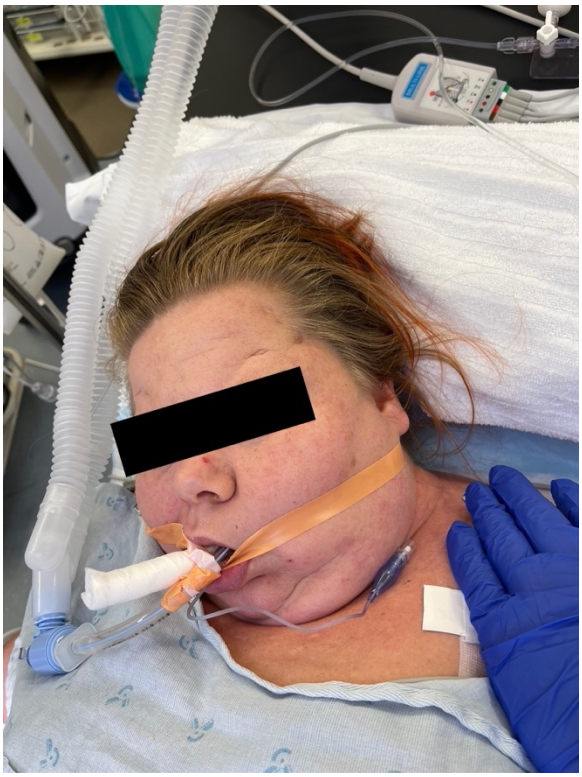
Introduction: Klippel-Feil syndrome is a rare congenital condition leading to fusion of the cervical vertebrae resulting in a short hypomobile neck and associated anomalies [1]. Goldenhar syndrome is a rare congenital condition with abnormal development of the branchial arches and vertebral column resulting in hemifacial microsomia of the ear, soft palate, lip, and mandible, and can be associated with congenital scoliosis, limbal dermoids, hearing loss, blindness, or rare cardiac or visceral anomalies [2]. Rarely these syndromes can co-occur [3]. The purpose of this medically challenging case report is to highlight the unique perioperative and anesthetic considerations in obstetric patients with Klippel-Feil and Goldenhar syndrome.

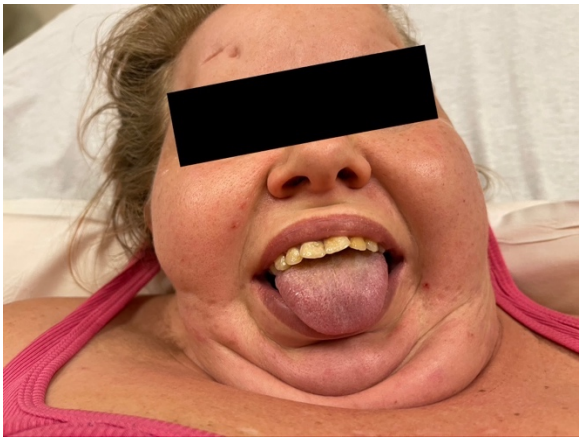
Methods: A 31-year-old G1P0 with Klippel-Feil and Goldenhar syndrome presents at 38w4d from an outside hospital for management of complex obstetrical and anesthetic concerns. Her medical history is significant for congenital scoliosis corrected by multiple spinal fusions and known difficult intubation with prior successful awake fiberoptic nasal intubation. Her pregnancy is complicated by lower back pain with intermittent lower extremity paresthesias. On physical exam, her mouth opening is one finger breadth and her neck is in fixed flexion with a nonpalpable cricoid cartilage (Fig 1). With preexisting parethesias, severe scoliosis, and prior back surgeries, the patient was not felt to be a candidate for neuraxial anesthesia. She was also not considered a candidate for trial of labor without neuraxial analgesia given the high airway risk and the need for safe and rapid intubation in the event of an emergent caesarian section. Through multidisciplinary discussions, it was agreed that in order to mitigate these risks, the patient should be offered a semi-urgent caesarian section under general anesthesia. Risks, benefits, and alternatives were explained to the patient who agreed that a controlled induction and intubation would be the safest option. The patient was premedicated with oral sodium citrate, intravenous glycopyrrolate, and topicalized with oropharyngeal lidocaine. A low dose remifentanil infusion was initiated with supplemental oxygen via nasal cannula. The patient was successfully intubated via an awake oral fiberoptic approach and subsequently induced with intravenous propofol and rocuronium. Mechanical ventilation was started with sevoflurane and quickly transitioned to propofol infusion to facilitate uterine tone. The surgery was uncomplicated and sugammadex was administered for reversal of neuromuscular blockade. Prior to extubation, an airway exchange catheter was placed. The patient was extubated awake, and the airway exchange catheter was removed. The patient recovered in PACU without complication.

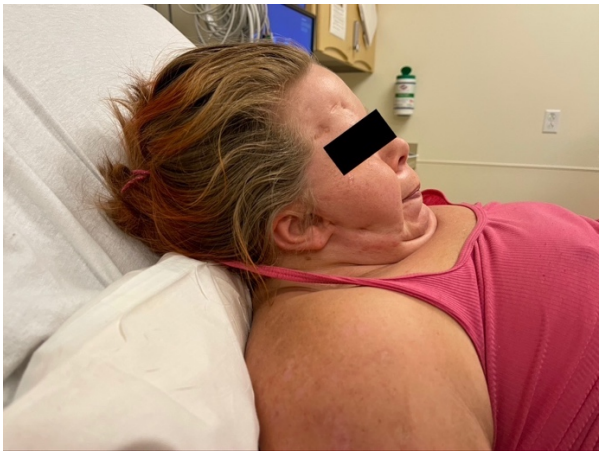
Conclusion: Obstetric patients with Klippel Feil syndrome and/or Goldenhar syndrome require a multidisciplinary approach and thorough preoperative evaluation to mitigate both obstetric and anesthetic risks. While neuraxial techniques have been demonstrated in a few case reports [4,5,6], severe scoliosis, prior spine surgeries, or preexisting neurologic deficits in this population may preclude successful, reliable, or safe neuraxial placement. The potential need for emergent airway instrumentation should always be considered in all obstetric patients. However, hypomobile or unstable cervical spines, distorted facial anatomy, or prior craniofacial surgeries can lead to difficult mask ventilation and intubation. Safety of rapid sequence intubation and feasibility of rescue front-of-neck airway should be assessed. Awake oral fiberoptic intubation should be considered as nasal intubation is associated with higher bleeding risk [7]. For this patient, the anesthesia team elected to undergo general anesthesia with awake oral fiberoptic intubation to secure the airway in a controlled manner. Discussions surrounding a complex patient's candidacy to undergo vaginal delivery should be discussed early in pregnancy with both obstetric and anesthesia teams. Early referral to tertiary medical centers should also be considered for the purpose of safe labor and delivery planning. Complex patients should consider scheduled cesarean sections prior to delivery dates. ECMO can also be considered as a backup plan for patients with complex pulmonary and cardiac concerns. However, ECMO can be a complex

process that requires a multidisciplinary approach and a robust institutional protocol.

References: 1. Can Anaesth Soc J. 1986;33(1):66-70. 2. Anaesthesia. 1990 Jan;45(1):49-52. 3. Spine (Phila Pa 1976). 2006;31(13):E400-E407. 4. Int J Obstet Anesth. 2013;22(4):343-348. 5. Indian J Anaesth. 2014;58(3):341-343. 6. J Clin Anesth. 2019;52:78-79. 7. Anaesthesia. 2020;75(7):945-961.







Obstetric Anesthesiology - 2 Inhalation induction for emergency cesarean section

Domenic Valentino[1], Jeffrey Gross[2], Dhamodaran Palaniappan[3]

[1]University of Connecticut, Farmington, CT, [2]University of Connecticut, -, United States of America, [3]UConn Health, Farmington, CT

Introduction: Inhalation induction as alternative means of achieving anesthesia in pregnant patients with difficult I.V. access.

Methods: Recreational drug abuse during pregnancy has been steadily increasing with incidence ranging from 0.4-27% and can present unique challenges to anesthesia providers. A 24yo G1P0 women at 34-week gestation with a history of beta-thalassemia, polysubstance abuse, acute intermittent porphyria, and seizure disorder presented for emergent cesarean section secondary to biophysical profile score 0/8 during antenatal visit. Patient had heroin prior to antenatal visit and en route from clinic to obstetrics suite she ate a full meal. Consent to deliver the fetus was obtained from patient's mother who was the conservator, the patient was considered altered at the time of encounter. The risk management service of the hospital was notified. General endotracheal anesthesia with rapid sequence intubation was planned given the scenario. Unfortunately, because of prior i.v. drug abuse, i.v access could not be obtained despite attempts with ultrasound and vein finder. Patient became more agitated and required restraints. Decision was made to induce anesthesia with sevoflurane/N20/O2. After inhalation induction with continuous cricoid pressure, saphenous vein was cannulated. Paralysis was then achieved with i.v. succinylcholine and trachea was intubated successfully. The fetus was quickly delivered and did not require resuscitation. Left internal jugular central line was placed. At conclusion of surgery, patient remained intubated overnight. She was extubated on POD#1 making full recovery. Discussion: Mother's life is the priority over fetal life. The mother was at no risk of harm if left alone. The fetus was at great risk for morbidity and mortality if delivery did not proceed. In this scenario, general endotracheal anesthesia with rapid sequence intubation is the standard of care. Because of the difficult i.v. access and uncooperative patient, though not ideal, inhalation induction with cricoid pressure can be considered in the above scenario. Over several breaths, sevoflurane concentration should be increased to achieve general anesthesia. Immediately after, intravenous access should be sought either peripherally or centrally. If unable to acquire i.v. access, intubation [with i.m. succinylcholine] for airway control is recommended. In cases where intravenous access is difficult to obtain, inhalation induction may be used to either improve chance of i.v. access or as means of securing the airway in rapidly deteriorating situations with strategies to reduce the risk of aspiration.

Conclusion: Inhalation induction with continuous cricoid pressure in a pregnant patient with no IV access though associated with risk can be considered for emergent situations.

References: Anesthesiology 1997; 86:1392-4.
Acta Anaesthesiol Scand. 2003; 47:1181-2.

Obstetric Anesthesiology - 3 Malnutrition on labor and delivery. What to expect with prolonged nausea, vomiting and poor oral intake.

Romon Neely[1], Beata Evans[1]

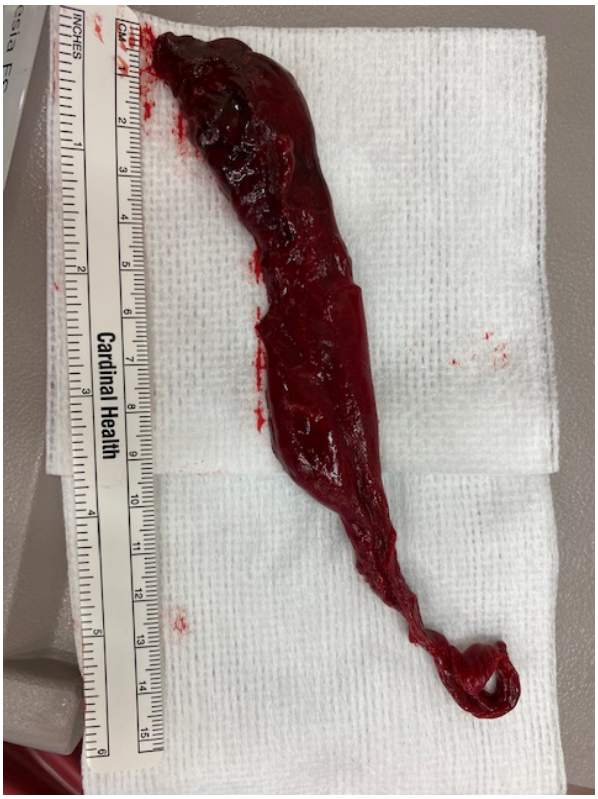
[1]Virginia Commonwealth University, Children Hospital of Richmond, Richmond, VA

Introduction: 39-year-old G5P2214 at 34 weeks 5 days gestation with a BMI of 29, weight of 84 kg returned to the labor and delivery unit with intractable nausea and vomiting. In addition to ongoing abdominal discomfort, she reported symptoms of odynophagia. This was attributed to acidic irritation to her esophagus from ongoing vomiting. The patient was admitted to the antepartum unit where she was treated with intravenous proton pump inhibitors, ondansetron, sucralfate, and intravenous fluids. She reported a mild improvement with the initiation of gabapentin. Further workup indicated elevated lipase and changes on CT of the abdomen consistent with acute pancreatitis. This diagnosis was attributed to gallstone versus idiopathic versus iatrogenic. While receiving treatment for the above, the patient was diagnosed with gestational hypertension and preeclampsia with significant features. The patient underwent primary, low transverse cesarean section for fetal malpresentation at 36 weeks gestation. She tolerated the C-section under single shot spinal well. The estimated blood loss was one liter. Postoperatively, she had an acute three-point drop in hemoglobin. Images of the abdomen and pelvis were obtained to evaluate for bleeding. With concerns for incisional bleeding, the patient went back to the operating room for an emergent exploratory laparotomy under general anesthesia. There was no significant active bleeding during the surgery, the hematoma was evacuated, and the hysterotomy defect was repaired. Prior to the emergence, the stomach was decompressed with an orogastric tube. Upon removal of the orogastric tube, there was a large, well-organized blood clot noted. The gastroenterology team was consulted for immediate evaluation with esophagogastroduodenoscopy while under general anesthesia. The gastroenterology team reported esophageal ulcers without bleeding or stigmata of recent bleed; however, there was red blood in the esophagus, clotted blood in the stomach, and the duodenum. Biopsies were obtained. Intraoperatively, coagulation studies indicated abnormalities (INR 3.1, PT 31.8, PTT 101). Upon completion of the exploratory laparotomy, hematology consult was obtained for concerns of coagulopathy. The patient was evaluated for a factor deficiency and the possible presence of inhibitors. Upon workup the hematologist attributed the changes to sequelae of pregnancy and dietary causes. The patient was prescribed vitamin K course and started on tube feeds for nutrition.

Methods: This is a case of a parturient who had a long history of intractable nausea and vomiting with multiple visits to labor and delivery triage for treatment. During admission to the antepartum unit, the patient received a diagnosis of acute pancreatitis. While in treatment for acute pancreatitis, the patient developed preeclampsia with severe features prompting urgent delivery of the fetus. Postoperatively, the patient developed anemia and coagulopathy. The hematology workup indicated that the coagulopathy was related to ongoing vomiting, prolonged poor nutrition, and subsequent vitamin K deficiency.

Conclusion: The parturient frequently experienced nausea and vomiting. These symptoms are often acknowledged and accepted as a normal part of the pregnancy. We have a case of a parturient with prolonged nausea, vomiting, and poor oral intake who developed coagulopathy. Her coagulopathy contributed to an increased blood loss associated with the delivery of the fetus, development of hematoma at the surgical site (subjecting the patient to other surgical interventions), and bleeding from esophageal ulcers. She required multiple blood product transfusions to stabilize her while in recovery from the birth of her baby. All of these triggered by prolonged nausea, vomiting, and poor nutrition leading to vitamin K deficiency. We would like to bring up to the attention of the conference attendees that despite living in a well-developed country where most of our patients have relatively good and easy access to the healthcare system, there is still work to do in order to improve patient care. As shown by this case report, nausea and vomiting are not benign and must not be accepted as a normal part of pregnancy. They can carry significant risks and morbidities to the parturient.

References: Lee, N, Saha, S, Nausea and Vomiting of Pregnancy, Gastroenterol Clin North Am., Gastroenterol Clin North Am. 2011 Jun; 40(2): 309,Äìvii, PMID: 21601782; Cappell, M, Gastric and duodenal ulcers during pregnancy; Gastroenterol Clin N Am. 2003 32 263-308; Sobral, M, Granja, C, Sampaio, M, Guerreiro, F, Bleeding from oesophageal varices in pregnancy, BMJ Case Rep 2013;



Obstetric Anesthesiology - 4 Ultrasound Guided Caudal Epidural Analgesia for Labor in a Parturient with Posterior Thoraco-Lumbar Spinal Fusion

Anna Gitterman[1], Anna Gabrielian[1], Zachary Janik[1], Karen Lindeman[1]

[1]Johns Hopkins University, Baltimore, MD

Introduction: Labor analgesia remains challenging in parturients with previous lumbar spinal fusions. Considerations for epidural placement in this population include difficulties with positioning, alterations in surface anatomy, bone grafts that obscure the intervertebral space, changes in sensation of loss of resistance that may increase the risk of accidental dural puncture, and post-surgical epidural scarring that may alter the spread of local anesthetics. Although caudal epidural catheters are readily utilized in pediatric anesthesia and in chronic pain clinics, they are less commonly used for labor analgesia. Reasons for this are multifactorial and include unfamiliarity with the procedure by providers, difficulty with positioning in the gravid patient, inability to locate the sacral hiatus with palpation, significant anatomical variation of the sacral hiatus in adults, and concern that cephalad spread of the local anesthetic may not be sufficient to provide surgical anesthesia for cesarean delivery. Here we present the use of a caudal epidural catheter placed under ultrasound guidance to provide labor analgesia in a G1P0 parturient with a history of both anterior (L3-S1) and posterior (T12-S1) spinal fusions secondary to idiopathic scoliosis.

Methods: Our patient presented for antenatal anesthesiology consultation given corrective surgery for idiopathic scoliosis 17 years prior. Surgical procedures included anterior discectomy L2 - S1 and instrumentation L3 - S1, which was complicated by a right foot drop suggestive of a L5 radiculopathy requiring lumbar laminectomy and decompression. A second staged procedure followed with posterior segmental instrumentation and fusion from T12-S1. She presented for induction of labor at 39 weeks gestational age to coordinate caudal epidural catheter placement. Following sterile preparation, Sims' position was utilized, and an ultrasound transducer was placed transversely to locate the sacral cornua, the sacrococcygeal ligament, and sacral hiatus. Lidocaine (1%) was injected in the skin, and the transducer was rotated 90 degrees between the two sacral cornua. Under direct ultrasound guidance, a 17 gauge Tuohy needle was advanced through the sacrococcygeal ligament into the sacral hiatus. No fluid or blood returned through the needle, and normal saline was easily injected. A 19 gauge epidural catheter was advanced 10 cm without resistance, and a test dose (1.5% lidocaine with 1:200,000 epinephrine) was negative for intrathecal or intravascular administration. Following injection of 15 ml bupivacaine (0.125%), we detected a T10 sensory block to temperature bilaterally. Patient controlled epidural analgesia (PCEA), bupivacaine (0.125%) and fentanyl (2 mcg/ml) at 8 mL per hour with a 5 ml bolus every 20 minutes as needed, maintained an adequate sensory level throughout labor. Ultimately, the patient had an uncomplicated spontaneous vaginal delivery with excellent analgesia, 26 hours after caudal epidural catheter placement.

Conclusion: Caudal epidural analgesia remains a consideration in parturients who otherwise may not be candidates for lumbar epidural placement due to lumbar spinal abnormalities. Anesthesia providers should be familiar with this technique as with proper dosing and catheter placement, excellent labor analgesia can be achieved. The availability of ultrasound can facilitate both needle visualization and imaging of reference structures to improve success and to limit the likelihood of complications, such as dural puncture and fetal head injection.

References: “Scoliosis: Repaired and Unrepaired”. In: Mankowitz S. (eds) Consults in Obstetric Anesthesiology. Springer, Cham. 2018 “Caudal Anesthesia”. NYSORA, 11 Feb. 2021, https://www.nysora.com/techniques/neuraxial-and-perineuraxial-techniques/caudal-anesthesia/. “Caudal Epidural Block: An Updated Review of Anatomy and Techniques”. BioMed Research International, vol. 2017, 2017.

Obstetric Anesthesiology - 5 Fever of Unknown Origin in a Parturient: An Unexpected Case of Plasmodium Falciparum Malaria

Amanda Berry[1], Reine Zbeidy[1]

[1]Jackson Memorial Hospital/University of Miami, Miami, FL

Introduction: Globally, more than 125 million women each year are at risk of malaria during pregnancy. Malaria caused by P.falciparum is the most dangerous form of malaria. Early diagnosis and multidisciplinary management are essential. Malaria can cause abortion, preterm labor, stillbirth, and severe maternal morbidity and mortality.[1] Pregnant women are at increased risk of malaria, and they act as a parasite reservoir in the community. Semi-immune women often carry P. falciparum with low peripheral parasite burdens and few acute symptoms, hindering diagnosis and complicating efforts to use targeted treatment as a strategy. In populations with low transmission, women with no immunity are at increased risk of acute severe disease and mortality during P. falciparum infection. Therefore, active surveillance and prompt treatment of malaria in these women are crucial. [2]

Methods: A 32 year-old, G2P0010, at 39-weeks of gestation presented to a Miami, FL hospital with a one-week history of intermittent fever, shaking chills, headache, and cough. Between febrile episodes, she reported malaise, but was otherwise asymptomatic. She had no other significant medical history and her pregnancy was uncomplicated. The only recent travel was a trip to Haiti five months earlier. On admission, her blood pressure was 95/50 mmHg, heart rate 160, 103°F. Blood culture, urine culture, chest x-ray, and malaria blood smear were ordered. She was started on broad-spectrum intravenous antibiotics of vancomycin and piperacillin-tazobactam in addition to intravenous fluids. No maternal anemia or thrombocytopenia was reported. Kidney and liver functions and coagulation profile were normal. She developed severe, late fetal heart rate decelerations after approximately 6 hours of labor, and she underwent an emergent cesarean section with general anesthesia. Rapid sequence induction using etomidate and succinylcholine was performed. Monitoring consisted of pulse oximetry, electrocardiography, and direct intra-arterial blood pressure measurement. She remained stable throughout the surgery and was extubated in the operating room. She was subsequently transferred to the intensive care unit where a central line was placed. She was started on the medications quinidine and doxycycline because of a clinical suspicion of malaria although microbiology results were not immediately available. A peripheral blood smear confirmed the diagnosis of malaria demonstrating plasmodium falciparum infestation and a parasitic index of 1%. The patient was switched to chloroquine one gram orally for 48 hours for a total of four doses. Complete recovery was achieved in one week. For P. falciparum infections acquired in areas without chloroquine-resistant strains, which include Central America west of the Panama Canal, Haiti, and the Dominican Republic, patients can be treated with oral chloroquine, or, alternatively, hydroxychloroquine at recommended doses per CDC guidelines. [3]

Conclusion: After the third month of pregnancy, the placenta is highly susceptible to malaria infection. The maternal sinusoids allow parasites to develop in the sequestered erythrocytes. Although this patient was a US citizen admitted to a hospital in Miami, she traveled to a malaria-infested area when she was nineteen weeks pregnant. General anesthesia was chosen because she exhibited signs of systemic sepsis before confirming the diagnosis of malaria. The role of regional anesthesia is controversial because of the possibility of seeding the central nervous system with the parasite, especially in patients pending test results for malaria. In a similar case report from the UK, a patient infected with malaria was given a spinal anesthetic for an emergency cesarean, however, the patient remained stable and did not appear to be in severe sepsis. [4] In conclusion, we believe that the safest option for an unstable, suspected malaria-infected patient requiring emergency cesarean should be general anesthesia.

References: [1] Obstet Gynecol Surv. 74(9): 546–556 2019 [2] Cold Spring Harb Perspect Med. 7(6): a025551. 2017 [3] “CDC - Malaria - Diagnosis & Treatment (United States) - Treatment (U.S.) - Guidelines for Clinicians.” Centers for Disease Control and Prevention, Centers for Disease Control and Prevention, 2020 [4] Int J Obstet Anesth. 24(1):91. 2015

Obstetric Anesthesiology - 6 The question of when to restart full-dose anticoagulation after neuraxial blockade in obstetric patients with cerebral venous thrombosis? A case report.

David Kim[1], Baiping Lei[1]

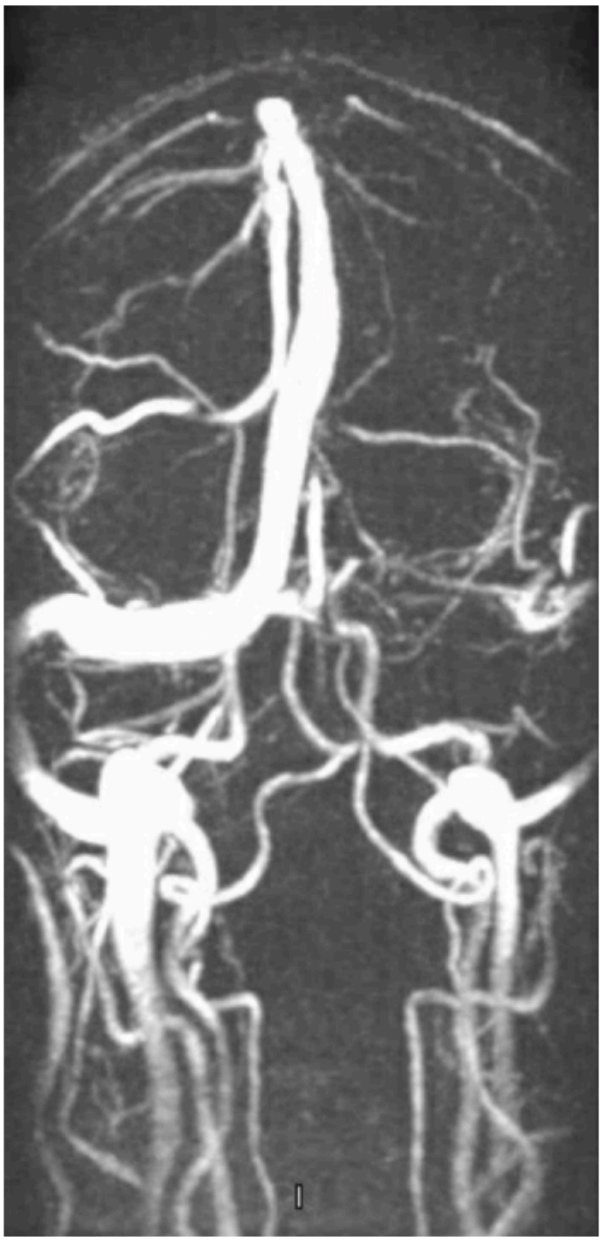
[1]SUNY Downstate Medical Center, Brooklyn, NY

Introduction: The treatment of venous sinus thrombosis in the obstetric population can be a challenge in the perioperative setting. Patients who present for a scheduled cesarean usually receive a neuraxial blockade as general anesthesia may pose harm to the fetus. The current ASRA guidelines suggest that clinicians should not restart full dose anticoagulation for until at least 24 hours after administration of neuraxial blockade to prevent the possibility of epidural bleeding/hematoma. It is known that epidural hematoma is a relatively rare complication after neuraxial blockade in the obstetric population (1 in 200,000), however it is a complication that can potentially lead to permanent neurological damage. On the other hand, holding the anticoagulation for an extended period of time may lead to propagation of thrombosis. The incidence of thromboembolic disease related to pregnancy is 0.13% and in developed countries, it constitutes 10% of maternal morbidity and mortality. It is known that pregnant females are at a higher risk of developing thromboembolism in the peripartum period. In one case report, a 37 year old female patient with an unremarkable history except for a previous cesarean, underwent a cesarean under spinal anesthesia. In the third postoperative hour, she developed nausea, vomiting, generalized tonic-clonic convulsion and respiratory arrest. On imaging, she was found to have left-sided cerebral vein thrombosis. She was eventually intubated and admitted to the intensive care unit for further care.

Methods: This is a 30 year old pregnant female at 37 weeks who comes in to labor and delivery for an elective scheduled cesarean for gestational hypertension. She has a history of ulcerative colitis, hyperlipidemia, chronic migraines, and obesity. In her prior pregnancy four years ago, patient was admitted in another hospital for intractable headache, nausea, vomiting. MRV brain at that time showed severe narrowing of the proximal left transverse sinus without complete obstruction. At that time, she was also found to have left-sided popliteal DVT. She was subsequently treated with lovenox throughout the rest of her pregnancy and 6 weeks postpartum. Her pregnancy was complicated by painless third trimester bleeding and gestational hypertension, in which she received a cesarean. She was instructed to repeat MRV brain 6 weeks postpartum and follow-up with the neurosurgery service, however she did not do so. Of note, she had two unintentional abortions in the past (2016 and 2019). She is a current everyday smoker and also reports daily marijuana use. She has no family history of thrombophilia. Two weeks prior to her elective cesarean, patient had a similar presentation in the ED with intractable headache, nausea, vomiting, and an MRI/MRA/MRV at this time showed total loss of signal in left transverse sinus with faint signal in left sigmoid sinus, findings suggestive of venous sinus thrombosis. She was subsequently admitted for recurrent left venous sinus thrombosis. Her physical exam was unremarkable. Her labs, including PT, PTT, INR, platelets were all WNL. She was started on intravenous heparin drip until she showed clinical improvement and was transitioned to lovenox 1mg/kg BID upon discharge. She was advised to not take lovenox the day prior to the cesarean due to the risk of extensive bleeding. On the day of surgery, patient was given combined spinal epidural and underwent a successful cesarean. The primary team was advised to restart the patient on lovenox the following day (24 hours after neuraxial blockade), and to continue indefinite anticoagulation in view of recurrent cerebral venous thrombosis as long as benefits of anticoagulation outweigh the risks.

Conclusion: Venous sinus thrombosis can present with generalized tonic-clonic seizures and respiratory arrest. In this young female patient, anticoagulation was held for 24 hours after administration of neuraxial blockade, following ASRA guidelines. However, she has significant risk factors for thrombophilia. Furthermore, obstetric patients are shown to have a lower incidence of spinal hematoma than the general population due to the hypercoagulable state of pregnancy, wider capacity of epidural space, and higher intra-epidural pressure. One can argue that the benefit of restarting her full-dose lovenox earlier than

the recommended period outweighs the risk of potential spinal hematoma formation.



Obstetric Anesthesiology - 7

Preconception COVID-19 Acute Respiratory Distress Syndrome (ARDS) Requiring VV-ECMO Presenting with Antenatal Tracheal Stenosis: A Case Report

Ashlee Gourdine[1], Katelyn Scharf[1], John Pallan[2], Shobana Bharadwaj[3], Bhavani Shankar Kodali[3], Jessica Galey[4]

[1]University of Maryland, Baltimore, MD, [2]University of Maryland, Baltimore, United States of America, [3]University of Maryland Medical Center, Baltimore, MD, [4]University of Maryland School of Medicine, Baltimore, MD

Introduction: We report a case of vaginal delivery in a parturient with a past medical history significant for COVID-19 acute respiratory distress syndrome requiring Veno-venous Extracorporeal Membrane Oxygenation (VV-ECMO) and tracheostomy resulting in severe restrictive lung disease and post-decannulation tracheal stenosis. Her other medical history included prior recurrent ventricular tachycardia, morbid obesity (BMI 51) and obstructive sleep apnea on continuous positive airway pressure (CPAP) therapy. Anesthetic management included neuraxial anesthesia with a lumbar epidural and facilitation of assisted second stage of labor in the operative setting. Considerations of the cardiopulmonary physiology changes of pregnancy was essential in risk stratifying and delivery planning. Being a high-risk labor candidate, the patient underwent extensive preoperative evaluation and multispecialty coordination for a successful labor course. Optimal anesthetic management was challenging- including the avoidance of arrhythmogenic or bronchoconstricting agents, preparation of Heliox for reduced airway resistance and continued oxygenation during valsalva, early epidural for reduced catecholamine excretion secondary to pain, and continual monitoring of maternal-fetal hemodynamics. We present this case to elucidate a new category of peripartum patients who are post-COVID infection and who require unique considerations for delivery planning.

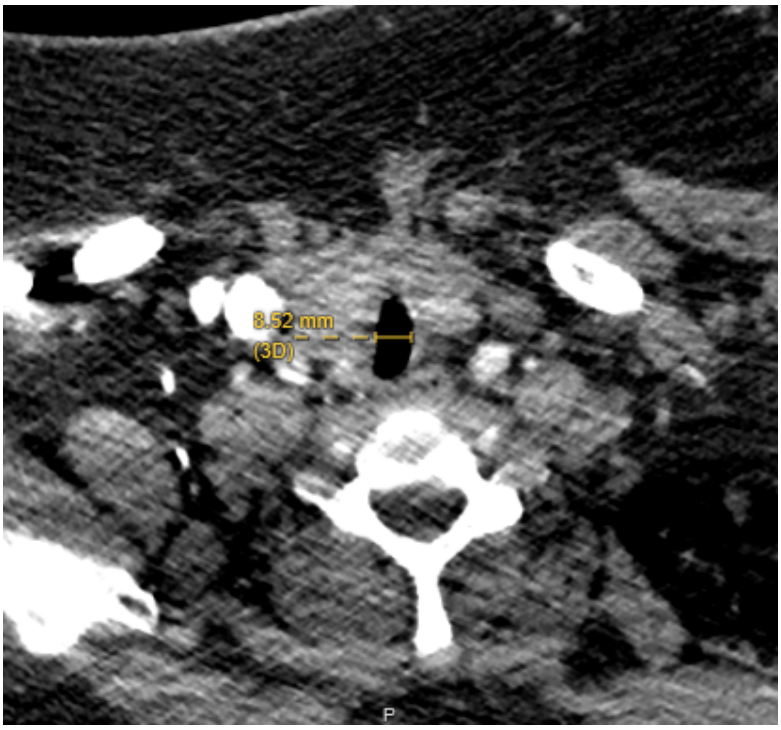
Methods: A 27-year-old female G1P0 at 37w2d presents for induction of labor post COVID-19 infection. The patient was diagnosed after presenting with cough, dyspnea, chest pain, vomiting, and anosmia. Her respiratory status quickly declined, requiring supplemental oxygenation with high-flow nasal cannula transitioning to BiPAP and then mechanical ventilation. After worsening clinical status despite proning and mechanical ventilation optimization, the patient was cannulated for Veno venous extracorporeal membrane oxygenation (VV-ECMO for 31 days) during which she was also transitioned from oral endotracheal intubation to tracheostomy for 36 days. Shortly after her hospitalization and recovery, the patient conceived her first intrauterine pregnancy with an estimated delivery date approximately 10 months post resolution of her COVID-19 hospitalization. Her past medical history was also significant for chronic hypertension, polycystic ovarian syndrome, severe restrictive lung disease, obstructive sleep apnea (AHI 13.4), history of ventricular tachycardia with residual paroxysmal tachycardia, tracheal stenosis, class III obesity with a BMI 51, and a history of pulmonary embolism and right internal jugular vein deep venous thromboembolism status post three months of anticoagulation. Her physical examination was significant for tachypnea with speaking, orthopnea, and a Mallampati III airway. Her most recent pulmonary function test showed restrictive lung disease. The patient was scheduled for induction of labor in the setting of her cardiopulmonary comorbidities and to allow for multidisciplinary planning. She was followed closely after her hospitalization by pulmonology and cardiology given her residual post-COVID pneumonia sequela. Computerized tomography angiography (CTA) neck showed short segment stenosis of the subglottic trachea measuring 1 cm in length and showed 8 mm diameter at its narrowest portion. Fiberoptic laryngoscopy was performed showing 60% stenosis of the trachea. As her pregnancy progressed, the patient became more tachypneic and her sleep disordered breathing worsened, requiring multiple antepartum admissions for evaluation by ear nose and throat specialists, pulmonologists, and cardiologists for optimization.

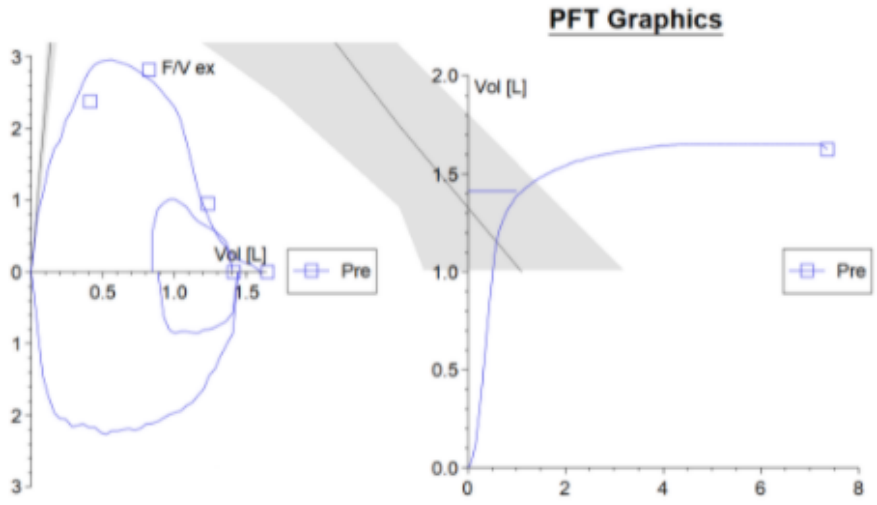
Conclusion: The COVID-19 pandemic has produced a new subset of patients that merit multidisciplinary preparation and evaluation: the post-COVID parturient. Parturients with a history of COVID ARDS are a population of patients that require unique risk stratifying for avoidance of peripartum cardiopulmonary complications. These populations, especially in the setting of COVID pneumonia with ARDS, frequently have a coinciding history of prolonged oral endotracheal intubations or tracheostomy with higher risk of subsequent tracheal stenosis. The use of appropriate antenatal monitoring, emergency planning, and specialty formulations like heliox allow for successful avoidance of morbidity and mortality.

References: 1. Kepley JM, Bates K, Mohiuddin SS. Physiology, Maternal Changes. [Updated 2021 Sep 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. https://www.ncbi.nlm.nih.gov/books/NBK539766/ 2. Ernst A, Feller-Kopman D, Becker HD, Mehta AC. Central airway obstruction. Am J Respir Crit Care Med 2004; 169:1278. 3. Nouraei SA, Winterborn C, Nouraei SM, et al. Quantifying the physiology of laryngotracheal stenosis: changes in pulmonary dynamics in response to graded extrathoracic resistive loading. Laryngoscope 2007; 117:581. 4. Barach, AL. The use of helium in the treatment of asthma and obstructive lesions in the larynx and trachea. Ann Intern Med 1935; 9:739. 5. Milner QJ, Abdy S, Allen JG. Management of severe tracheal obstruction with helium/oxygen and a laryngeal mask airway. Anaesthesia 1997; 52:1087. 6. Branch AL. The therapeutic use of helium. JAMA 1936;107:1273 – 80.

Spirometry:

		Ref	LLN	Pre	Pre%Ref
FVC	L	3.42	2.73	1.64	48.1
FEV 1	L	2.93	2.35	1.41	48.2
FEV1/FVC	%	86	74	86	100.0
FEV 6	L	3.41	2.80	1.64	48.1
FEF 25-75%	L/s	3.35	2.23	2.20	65.7
ISOFEF 25-75%	L/s			2.20	
PEF	L/s	6.53	4.98	3.08	47.2
FIVC	L	3.43	2.80	1.46	42.6
PIF	L/s	5.54	3.14	2.26	40.8


8.52 mm
(3D)
P


PFT Graphics
F/V ex
Vol [L]
Pre
Vol [L]
Pre

Obstetric Anesthesiology - 8 Cesarean Section In A Patient With Marfan Syndrome After Multiple Cardiovascular Surgical Interventions

Ryker Saunders[1], Regina Y Fragneto[1], Sanjay Dwarakanath[1]

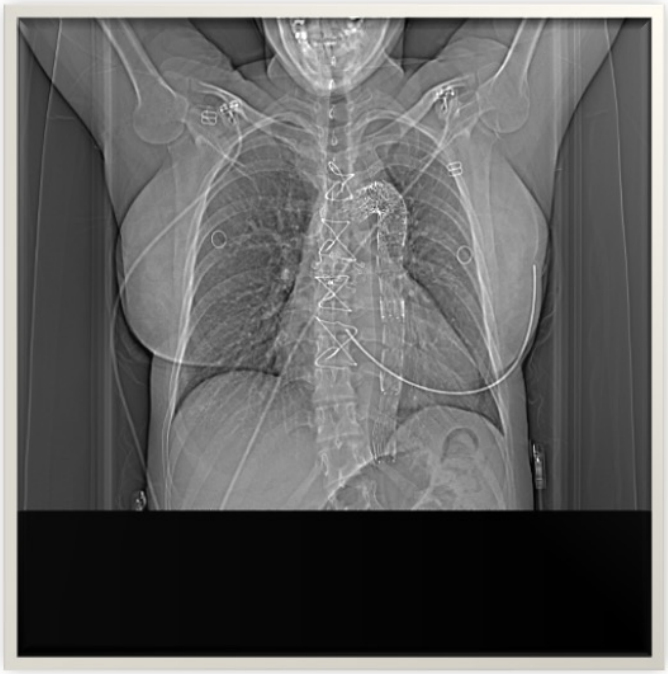
[1]University of Kentucky, Lexington, KY

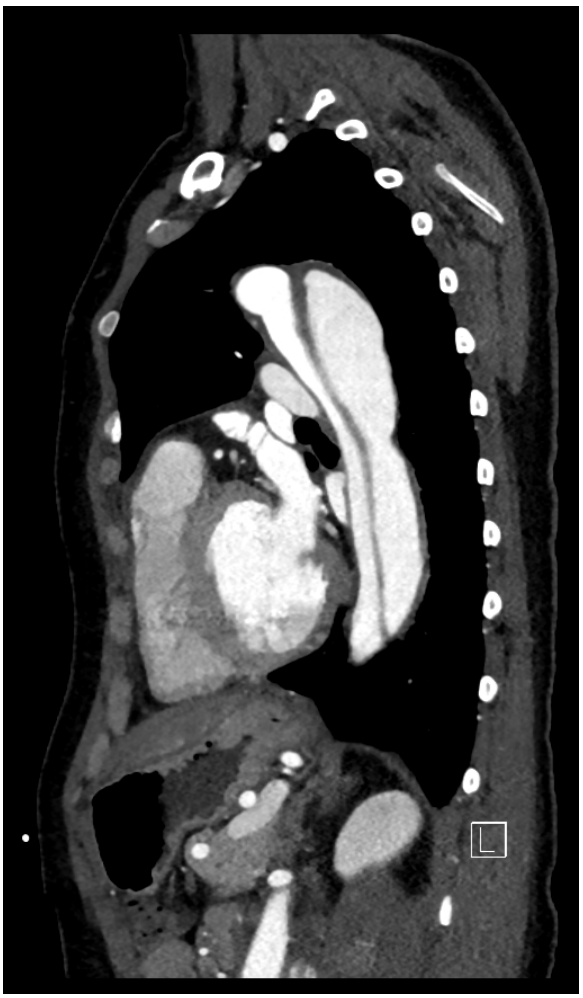
Introduction: Marfan Syndrome (MFS) is an autosomal dominant connective tissues disorder of fibrillin-1 that forms abnormal and weak structural connections of microfibrils1. Particularly affected structures include the aorta, heart, lungs, and skin. The tunica media of the aorta contains the abnormal microfibrils, which predisposes to dissections and aneurysms throughout the vasculature. Coupled with the hemodynamic changes with pregnancy, the fragile state of the cardiovascular system poses a significant risk of morbidity and mortality to the parturient and the fetus. An increased cardiac output during both vaginal and cesarean deliveries increases the shear stress along the aorta that can potentiate root dilation, aneurysm, dissection, and rupture.

Methods: We present a case of a 30-year-old woman with a history of Marfan Syndrome who had aortic root dilation s/p two separate valve-sparing root reconstructions and Type B thoracoabdominal aortic dissection s/p TEVAR. Other significant medical history included GERD, scoliosis, unilateral deafness, and was a regular tobacco and marijuana smoker. Her only medications included 75mg PO metoprolol, 81mg aspirin, PO iron and prenatal vitamin supplements. The fetus had an uncomplicated prenatal course with reactive nonstress tests, measuring in the 44th percentile in weight, and had assumed a vertex position. The placenta was implanted in the posterior wall of the uterus. It was discovered that she was pregnant during a follow up CT scan of her aortic dissection, and subsequently underwent a TEVAR while she was 16 weeks pregnant. At 33 weeks and 1 day, she presented to labor & delivery for an artificial induction of labor as she was needing an additional aortic root repair after delivery. Upon arrival, she received an echocardiogram which was unchanged from prior echo prior to and earlier in her pregnancy. A discussion was had about whether to approach delivery either vaginally or by cesarean section. Further, if cesarean, would it be under a combined spina-epidural technique (CSE) or under general anesthesia? It was decided to proceed with cesarean delivery under combined spinal-epidural with cardiovascular anesthesiology. She was continued on her scheduled metoprolol the morning of her cesarian section. Prior to the CSE, an arterial line was placed in the right radial artery with a 20g 1-¬æ inch arrow catheter to monitor hemodynamic changes with the spinal. The CSE was performed on the operating table with the patient in the upright, sitting position. Esmolol and nicardipine infusions were prepared for impulse and hemodynamic control. The spinal dose consisted of 1.8mL of 0.75% hypertonic bupivacaine, 25mcg of fentanyl, and an epinephrine 1:200,000 washout. After the spinal was given, an epidural catheter was threaded into the epidural space and secured. The patient was laid supine with slight left uterine displacement. Adequate sensory blockade was determined and little hemodynamic change was noted. The cesarean section started and was tolerated very well by the patient, lasting about two hours. Her blood pressures never varied more than 10% from her pre-operative baseline, and never required any infusions of esmolol or nicardipine. She was brought to PACU and had spontaneous recovery of sensory and motor function. Her hemodynamics were consistent throughout the perioperative period and did not require significant intervention.

Conclusion: Strict control of hemodynamics is paramount in pregnant Marfan patients especially during the delivery period, as there can be significant fluctuations of heart rate and blood pressure dependent on labor pain and other factors. Without impulse control with beta blockade, hemodynamic shifts can exacerbate and worsen existing aneurysms and dissections. Prophylactic beta blockade is also useful for preventing aortic root dilation3. Spinal anesthesia is not only beneficial in avoiding hemodynamic shifts seen with pain, but also by inducing a sympathectomy of the vasculature in the lower extremity. Both help contribute to lowering blood pressure by decreasing preload and endogenous catecholamine surges

References: 1. Genetics, clinical features, and diagnosis of Marfan syndrome and related disorders. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. Retrieved October 30th, 2020.2. Cardiac hemodynamics before, during and after elective cesarean delivery, do we really know it all? American Journal of Obstetrics and Gynecology. January 2016. 3. Cardiovascular problems in pregnant women with the Marfan syndrome. Ann Intern Med. 1995 Jul 15;123(2):117-22.4. Maternal Adaptations to Pregnancy: Cardiovascular and Hemodynamic Changes. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. Retrieved November 2, 2020.5. Pregnancy and Marfan Syndrome. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. Retrieved October 30th, 2020.





Obstetric Anesthesiology - 9 Anesthetic Management for Placenta Accreta: Neuraxial versus General Anesthesia

Michael Wen[1], Mutmainnah Bello[1], Ronnie Stotts[1], Jenny E Pennycuff[1]

[1]The University of Texas Medical Branch at Galveston, Galveston, TX

Introduction: Placenta accreta spectrum disorder (PAS) describes a range of pathological conditions where an abnormally invasive placenta is unable to detach spontaneously after delivery and cannot be forcibly removed without significant hemorrhage. The depth of placenta invasion can range from the uterine decidua (accreata) to the myometrium (increta), or in severe cases to the serosa and adjacent organs (percreta). Placenta accreta has a mortality rate ranging from 0.6% to 7% and increasing to 30% in the absence of antenatal diagnosis [1]. The incidence has increased from approximately 0.8 per 1,000 deliveries in the 1980s to 3 per 1,000 deliveries in the past decade [2]. Here we discuss a case of cesarean hysterectomy for placenta accreta and the anesthetic management.

Methods: A 29-year-old, 60 kg, and gravida five para four female at 35 weeks 0 days gestation presented for a planned cesarean hysterectomy. The patient has history of four previous c-sections, one conducted under general anesthesia and all others under spinal anesthesia, without complications. Prenatal workup was significant for placenta accreta and positive anti K antibodies on type and screen. Due to the likely need for transfusion, prior planning and coordination with blood bank was necessary to cross eight units. The patient's pre-operative hemoglobin was 9.7 g/dL and platelets were 312,000. Routine monitoring was started with blood pressure, electrocardiography, and pulse oximetry. A 16-gauge peripheral IV was secured as well as a pre-induction 20-gauge left radial arterial line for continuous blood pressure monitoring and blood sampling. Her right internal jugular vein was cannulated with a 9 French multi-lumen access catheter under ultrasound guidance. A combined spinal-epidural was performed with 12 mg of Bupivacaine 0.75%, 15 mcg of fentanyl, and 150 mcg of morphine given intrathecally, followed by epidural catheter placement. Bilateral ureteral stents were placed by the urologist for improved ability to identify the ureter during the surgery. Next, a vascular surgeon gained access to the right common femoral artery in the event of uncontrollable hemorrhage and the need for resuscitative endovascular balloon occlusion of the aorta (REBOA). The surgery proceeded with the cesarean delivery of a 2,400 gram female baby, with Apgar scores of 7 and 8 at 0 and 5 minutes, respectively. The patient was started on an oxytocin IV infusion of 40 units. During the surgery, the arterial line malfunctioned, however blood pressure was able to transduce through the femoral arterial line already obtained by vascular surgery. Shortly after delivery of the infant, the surgeons were working quickly to achieve control of the hemorrhage. At this time, the patient endorsed discomfort and the surgeons requested full relaxation for optimal surgical conditions, so the decision was made to convert to general anesthesia. The patient was induced with 150 mg of propofol and 40 mg of rocuronium and intubated. Anesthesia was then maintained with sevoflurane. The hysterectomy portion was completed, with total blood loss estimated at 3,000 milliliters. Intraoperative course was complicated by dense adhesions and accidental cystostomy, requiring repair. A total of 4.3 L of crystalloid, 1.5 L of albumin, 4 units of packed red blood cells, and 2 units of cell saver were given to the patient. The patient was successfully extubated in the OR, 3 mg of morphine was given via the epidural, and the epidural catheter was left in place for post-operative analgesia.

Conclusion: With an increase in c-section rates, there has been an increase in incidence of placenta accreta, which is one of the leading causes of peripartum hemorrhage. Our case demonstrates the importance of interdisciplinary coordination and an anesthetic plan that was flexible and centered on patient care. We allowed the mother to be awake and see the birth of her child with neuraxial anesthesia, but then allowed for patient comfort and optimal surgical conditions with general anesthesia. While general anesthesia is usually preferred for significant hemorrhage and hemodynamic instability, the c-section portion of the surgery had reasonable blood loss and the patient's hemodynamics were stable, allowing for safe neuraxial anesthesia. However, when the hysterectomy portion of the surgery started, the surgeons were quickly

working to achieve hemostasis which made general anesthesia the safe anesthetic option.

References: 1. Maternal morbidity and mortality due to placental accrete spectrum disorders. Best Practice and Research Clinical Obstetircs and Gyneocology 2021; 72:84-91. 2. Placenta Accreta Spectrum Disorders: Challenges, Risks, and Management Strategies. Int J Womens Health 2020; 12:1033-1045. 3. Management of massive hemorrhage in pregnant women with placenta previa. Anesth Pain Med (Seoul) 2020; 15(4):409-416. 4. Anesthetic management of patients with placenta accrete and resuscitation strategies for associated massive hemorrhage. Current Opinion in Anesthesiology 2011; 24:274-281. 5. A prospective study of awareness and recall associated with general anesthesia for cesarean section. Obstetric Anesthesia Digest 2009; 29: 155-156. 6. Anesthesia-related maternal mortality. Clinical Obstetrics and Gynecology 2003; 46:679-687.

Obstetric Anesthesiology - 10 Anesthesia management for cesarean section in a patient with secondary immune thrombocytopenia associated with primary Sjögren's Syndrome: a case report

Chaoxuan Dong[1], Xi Tan[1], Wenyi Sun[1], Li Zhang[2]

[1]The First Affiliated Hospital of Jinan University, Guangzhou, Guangdong, [2]International School of Jinan University, Guangzhou, Guangdong

Introduction: Primary Sjogren's syndrome (pSS), an autoimmune disorder, can be manifested with secondary immune thrombocytopenia (ITP). ITP is commonly associated with autoimmune diseases and has been reported in approximately 5-15% of PSS patients. Severe thrombocytopenia in a pregnant woman with pSS is a life-threatening risk during the labor and delivery, which brings a huge challenge to the management of anesthesia, especially to the choice of epidural or general anesthesia. We present a case of patient with secondary ITP associated with pSS who successfully underwent cesarean section under general anesthesia without any complications.

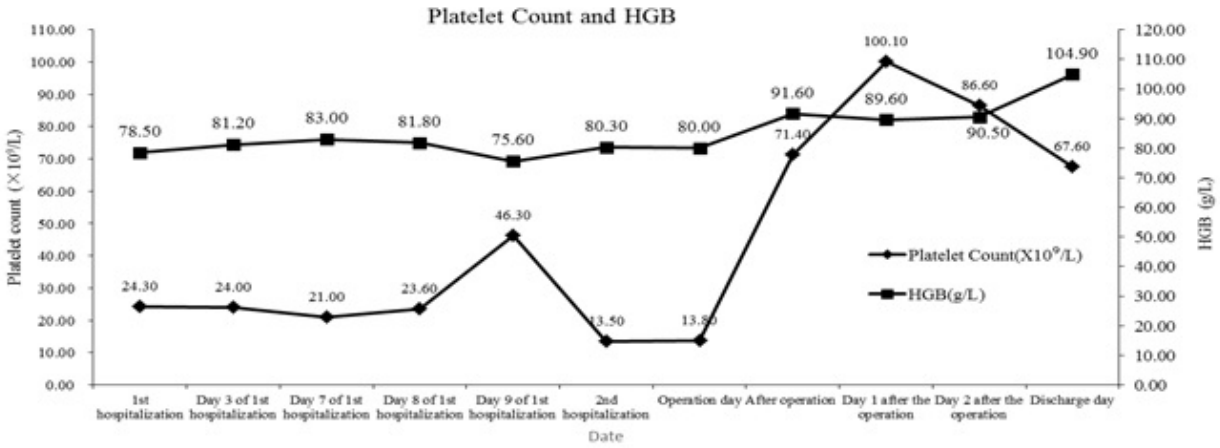
Methods: A 25-year-old woman (weight, 73.4 kg; height, 156.9 cm) was found to have a platelet count of 24×109/L out of the range of normal platelet count (190-524×109/L) on routine screening at 26+3 weeks' gestation of her first pregnancy. The patient was admitted to hospital at 27+6 weeks, abnormal laboratory findings included: Hb 78.5 g/L, WBC 5.54×109/L, platelets 24.3×109/L, albumin 31.6 g/L. (Figure 1) She was diagnosed with Sjogren's syndrome based on laboratory tests of anti-SSA, anti-SSB and anti Ro-52 antibodies and other clinical manifestations. An elective cesarean section was scheduled at 37+1 weeks of the gestation. Electrocardiogram (ECG) and chest X-ray were normal, preoperative laboratory tests reported a platelet count of 13.7×109/L, albumin 33.4 g/L. (Figure 1) After arriving the operating room, noninvasive blood pressure, ECG, and pulse oximetry monitoring commenced. Her initial blood pressure was 128/68 mmHg, heart rate was 98 beats/min, respiratory rate was 15 breaths/min, and oxygen saturation (SpO2) was 100%. Oxygen at 4L/min was supplied via a facial mask. An arterial line and a large-bore peripheral IV (intravenous) catheters were placed, 80 mg methylprednisolone and 350 ml albumin were given intravenously, 1 unit of platelets were immediately transfused. General anesthesia was induced via a rapid sequence induction with IV midazolam 2.5 mg (0.035 mg/kg) and esketamine 35 mg (0.48 mg/kg), 12 units of platelets were transfused simultaneously. Seven minutes after the skin incision, a male neonate (weight, 3.1 kg) was delivered with his Apgar scores of 9 at the first minute and 10 at the fifth minute. The patient was injected 50 mg rocuronium and 15 mg esketamine immediately. One minute after the injection, an oral endotracheal tube was intubated. Remifentanil at 5.4 mg/(kg•h) and propofol at 2.7 mg/(kg•h) were infused continuously. The placenta was removed and an intravenous infusion of 20 International Units (IU) of oxytocin mixed with 250 ml of 0.9% saline was started. Then, 1.5 unit of red blood cells and 200 ml plasma were transfused. The patient's vital signs were stable within the normal range during the operation, the operation ended 54 minutes after delivery. Postoperative platelet count was 71.4×109/L. (Figure 1) In total, the patient received 13 units of platelets, 350 ml albumin, 1.5 unit of red blood cells, 200 ml plasma, 1000 mL of IV multiple electrolytes injection and 250 mL of 0.9% sodium chloride solution

Conclusion: After the surgery, the patient's platelet count reduced continuously: 100.1×109/L on the postoperative day 1, 86.60×109/L on the postoperative day two, and 67.60×109/L on the postoperative day four (Figure 1). The patient and her neonate were discharged on the fifth postoperative day without any complication. The initial neonatal heart rate was 150 beats/min, and the neonate was transferred to the neonatal intensive care unit (NICU) immediately for continuous monitoring. During the NICU stay, the neonate's ECG findings showed no abnormality, and the heart rate was approximately 120-160 beats/min. The neonate's laboratory examination results revealed that antinuclear antibodies, anti-SSA, anti-SSB and anti-Ro-52 antibodies were also positive, there was no abnormal signs of blood routine examination.

Pregnancy with the Sjögren syndrome is more likely with a complication of thrombocytopenia. Women with this underlying autoimmune disorder must undergo prenatal counseling explaining all risks involved and

the need to control thrombocytopenia before conception. This case suggests that a pregnant woman with pSS and secondary ITP at a very low platelet count can safely deliver the baby by undergoing a cesarean section under general anesthesia.

References: 1. Neunert C, Lim W, Crowther M, Cohen A, Solberg L, Jr., Crowther MA, et al. The American Society of Hematology 2011 evidence-based practice guideline for immune thrombocytopenia. Blood. 2011;117(16):4190-207. 2. Ramakrishna R, Chaudhuri K, Sturgess A, Manoharan A. Haematological manifestations of primary Sjogren's syndrome: a clinicopathological study. Q J Med. 1992;83(303):547-54.



Obstetric Anesthesiology - 11 Labor Analgesia in a Pregnant Patient with Acute Hypoxemic Respiratory Failure Secondary to COVID-19 in the ICU

Alice Chu[1], Tichaendepi Mundangepfupfu[1]

[1]University of Rochester, Rochester, NY

Introduction: Pregnant patients are at high risk for infection with COVID-19. In pregnant patients with COVID-19 who develop acute respiratory failure, the physiologic changes associated with pregnancy, such as decreased FRC, can be associated with worsening of hypoxemic respiratory failure [1]. We describe an approach to management of a medically challenging case of a pregnant patient with acute fetal demise and acute hypoxemic respiratory failure secondary to COVID-19 requesting labor analgesia.

Methods: A 38-year-old female G3P1 with a history of hypertension, obesity, and type 2 diabetes at 31 weeks gestational age presented to another hospital with shortness of breath, chest tightness, cough, and sputum production, and noticed that her fetus had stopped moving. She was diagnosed with preeclampsia and treated with magnesium. She was also diagnosed with COVID-19, and intrauterine fetal demise was discovered, so she was transferred to the MICU at our hospital for further management. On arrival to the MICU, she was tachypneic with a respiratory rate in the 40s. She was placed on a high flow nasal cannula at 1.0 FiO2 and 40L/min. Chest CT showed extensive bilateral peripheral and lower lung ground glass opacities consistent with multifocal infection. Based on her symptom severity and an arterial blood gas with a pH of 7.31, PaCO2 17, and PaO2 113, the MICU team were concerned for impending respiratory collapse and recommended intubation. The patient refused and only wanted intubation if she were to code. Given her respiratory status, intrauterine fetal demise, and refusal of intubation, the MICU and OB teams discussed induction of labor. Due to concerns for contractions and labor worsening her respiratory status, OB Anesthesiology was consulted for a labor epidural. Platelet count was 185,000 per ŒºL and INR was 1.4. The patient had not received any anticoagulation for more than 24hrs proceeding the Anesthesiology consult. An 18G Tuohy was used at the L4-L5 and L3-L4 interspaces with 4 attempts. There was an unintentional dural puncture at L3-L4, and an intrathecal catheter was placed. Labor analgesia was started with a 0.5mL bolus of 0.25% bupivacaine, and an infusion of 0.125% bupivacaine was started at 1mL/hr. All providers were informed of the intrathecal catheter to prevent medication errors. The patient had painless passive fetal descent, and spontaneous vaginal delivery occurred 12 hours later without issues. Following delivery, her hypoxemia improved, and she was eventually weaned down to 2L of oxygen and discharged home after a 10-day hospitalization.

Conclusion: Symptomatic COVID-19 during pregnancy is associated with a substantial increase in the risk for severe maternal and neonatal morbidity and mortality [2]. Our patient's fetus had an intrauterine demise likely due to her hypoxemia from acute hypoxemic respiratory failure. Epidural placement early in labor is desirable in pregnant patients with COVID-19 to avoid exacerbation of respiratory symptoms with labor pain [3]. Given the severity of her respiratory distress and the ICU team's concerns for the patient progressing to respiratory collapse, management of this patient was further complicated by her refusal to be intubated. The patient was able to avoid intubation given good neuraxial analgesia, which prevented her from feeling her contractions and trying to push, both of which could have precipitated respiratory collapse.

References: 1). Dashraath P, Wong JLJ, Lim MXK, Lim LM, Li S, Biswas A, Choolani M, Mattar C, Su LL. Coronavirus disease 2019 (COVID-19) pandemic and pregnancy. Am J Obstet Gynecol. 2020 Jun;222(6):521-531. 2). Villar J, Ariff S, Gunier RB, et al. Maternal and Neonatal Morbidity and Mortality Among Pregnant Women With and Without COVID-19 Infection: The INTERCOVID Multinational Cohort Study. JAMA Pediatr. 2021;175(8):817,Äì826. 3). Bauer ME, Bernstein K, Dinges E, et al. Obstetric Anesthesia During the COVID-19 Pandemic. Anesth Analg. 2020;131(1):7-15.

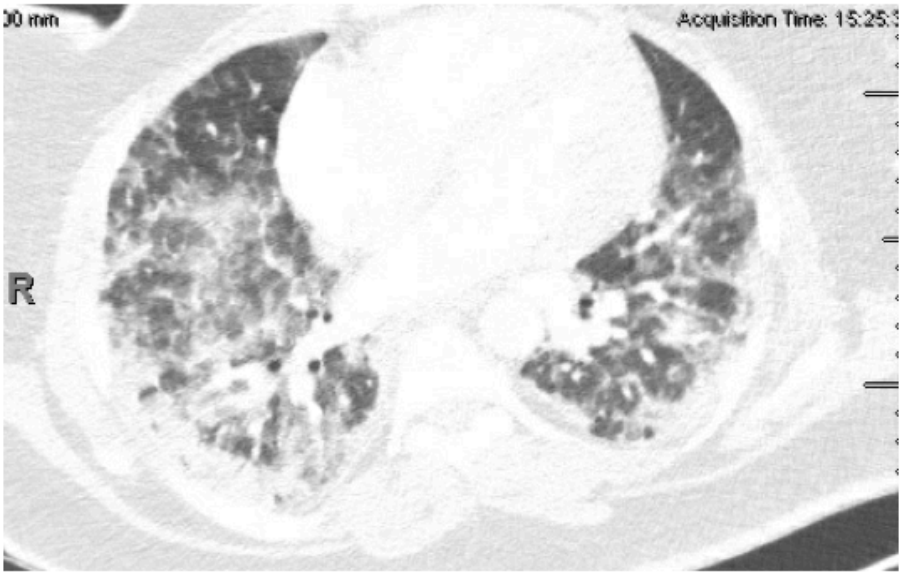


Image: CT Chest demonstrating ground glass opacities and consolidative changes.

Obstetric Anesthesiology - 12

Multidisciplinary Management of 23-Week Pregnant Woman with Cerebellar Hematoma and Obstructive Hydrocephalus: Case Report

Liliana Z Goelkel-Garcia[1], Vinh Nguyen[1]

[1]University of Minnesota, Minneapolis, MN

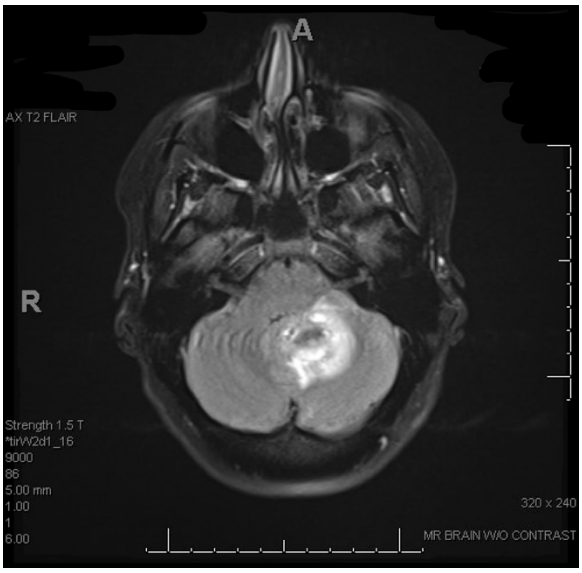
Introduction: Intracerebral hemorrhage (ICH) is a rare event during pregancy, with potentially devastating consequences for the mother and the unborn child. Most common causes of ICH include aneurysmal subarachnoid hemorrhage (SAH), and rupture of arteriovenous malformation (AVM).(1),(2),(3),(4) Other causes include preeclampsia/elampsia, coagulopathy, trauma, and cerebral venous thrombosis. While ICH risk might be elevated throughout the pregnancy, the rate of ICH increases during 3rd trimester, with the greatest risk during partum and puerperium.(5),(6),(7) Presentation is usually with headaches or seizures, with or without focal deficits. We here present a case of an otherwise healthy pregnant patient who presented with a non-traaumatic ICH.

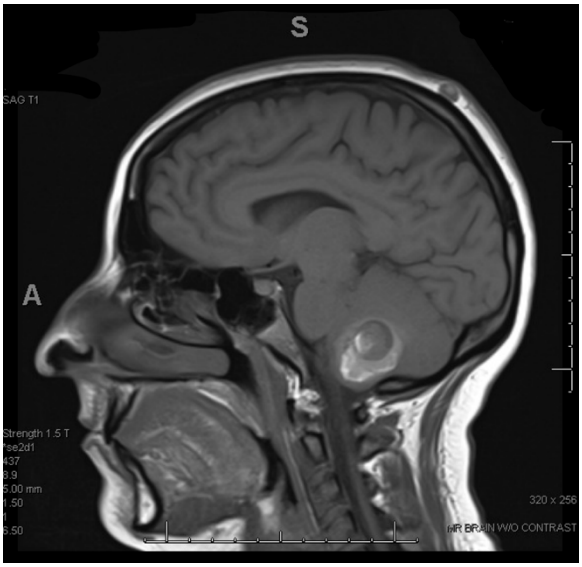
Methods: CASE REPORT: 30-year-old G2P1001, at 23wks, with three-day history of headache, nausea, and vomiting, blurry vision and dizziness. She denied fever or recent trauma. Uneventful pregnancy 1 year prior delivered vaginally with epidural catheter. Patient class I obese, non-smoker, with no drug use, and no relevant personal or family history. Vitals and physical were normal. MRI showed 28x28x23mm hemorrhage in left cerebellar hemisphere with surrounding vasogenic edema, compressing fourth ventricle, without hydrocephalus. Normal MRA of the circle of Willis and neck. Focused neuroexam revealed no neurodeficits for Intracerebral Hemorrhage score of 1. Conservative management with repeat CT at 6 hours, and then serial non-contrast MRIs, neuroexams, dexamethasone, pain control, and tight blood pressure control. Management involved Neurosurgery, ObGyn, Maternofetal Medicine (MFM), and Anesthesiologists. Patient remained neurounchanged, but with headache and nausea. On Hospital day (HD) 12, MRI showed new hydrocephalus. Options discussed included cerebrospinal fluid diversion via endoscopic 3rd-ventriculostomy, vs VP shunt placement, vs craniotomy with ventriculostomy to address the hemorrhage and any underlying lesion. Patient decided to proceed with craniotomy. Preoperative angiogram revealed no aneurysm or vascular malformation, and no vascular Blush to the mass. MFM recommended continuous electronic fetal monitoring (cEFM) during craniotomy, with plans for STAT Cesarean as backup. Magnesium sulfate was deferred unless fetal status became concerning. On HD13 patient underwent craniotomy, open ventriculostomy, right frontal external ventricular drain, and excision of friable cerebellar mass - likely cavernous malformation. Procedure done under general anesthesia with endotracheal tube, standard monitoring, and an arterial line. Patient positioned in right lateral decubitus to grant access to the abdomen if cesarean section needed. Surgery was uneventful with 25ml estimated blood loss. MFM remained on standby throughout the surgery and cEFM remained non-concerning. Patient extubated in the operating room and ransfered to Neuro-ICU. EVD was removed on HD17, and she was discharged home on HD20 with no residual neurological deficits. Her pregnancy continued otherwise uncomplicated.

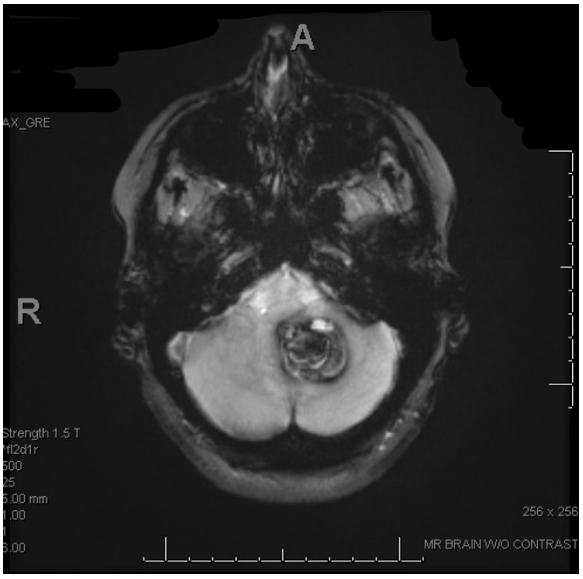
Conclusion: Our patient presented with a non-traumatic ICH of unclear etiology, suspected to be secondary to a cavernous malformation that ruptured. Diagnosis requires brain imaging with CT or MRI. When ICH is suspected, the benefit of a promp diagnosis outweights the small risk of fetal malformation. In our patient initial CT scan was folowed by repeated surveillance with non-contrast MRI to minimize ionizing radiation exposure to the fetus. Contrast MRI was not obtained since the safety of gadolinium-based contrast agents for the fetus has not been proven. Surgical intervention of ICH is clearly indicated for progressive neurological deficits, while conservative approach is preferred for devastating non-operable ICH. The decision between interventional and conservative management is harder to make in stable ICH in a neurologically intact patient, since there is not enough evidence to favor earlier surgical intervention. Potential complications of surgery under general anesthesia during pregnancy, the early gestational age of our patient, and lack of clinical indication for urgent surgical removal weighted in favor of our expectant management on admission.

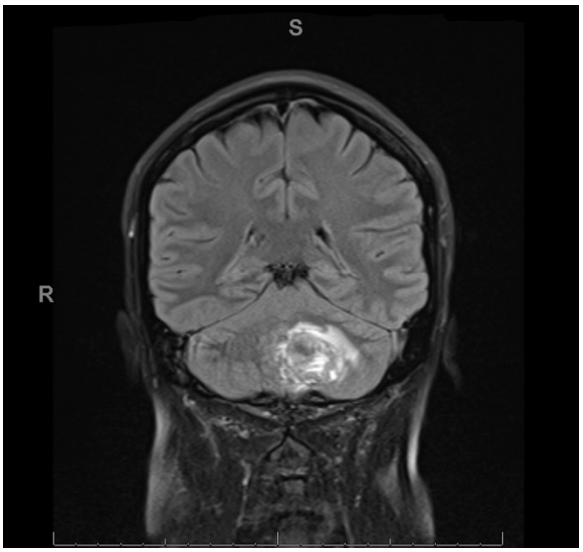
Management of ICH follows same standard neurosurgical principles, with additional considerations needed in pregnant patients such as early OBGyn and MFM involvement, and multimodal approach to decision making.

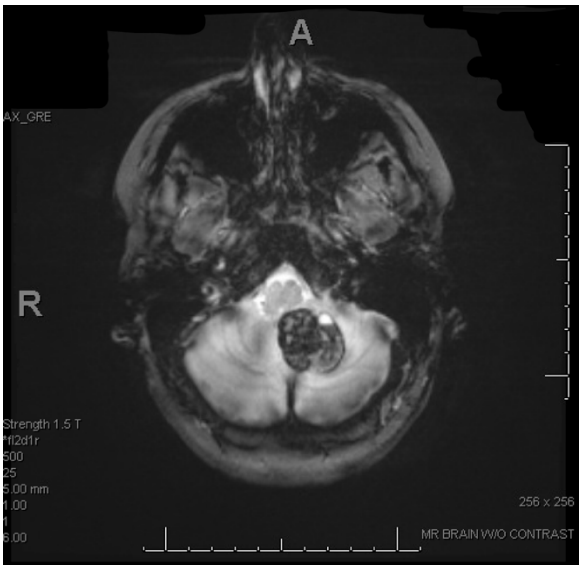
References: (1). Stroke and pregnancy. Stroke. 2000 Dec;31(12):2948-51. PMID: 1110875 (2). Incidence and etiologies of stroke during pregnancy and puerperium as evidenced in Taiwanese women. Cerebrovasc Dis. 2004;18(4):290-5. Epub 2004 Aug 24. PMID: 15331875. (3). Intracranial hemorrhage from aneurysms and arteriovenous malformations during pregnancy and the puerperium. Neurosurgery. 1990 Dec;27(6):855-65; disc 865-6. PMID: 2274125. (4). Intracranial haemorrhage in pregnancy. Obstet Med. 2009 Dec;2(4):142-8. Epub 2009 Nov 30. PMID: 27579059; PMCID: PMC4989666. (5). Intracerebral hemorrhage in pregnancy: frequency, risk factors, and outcome. Neurology. 2006 Aug 8;67(3):424-9. Erratum in: Neurology. 2007 Apr 3;68(14):1165. PMID: 16894102. (6). Stroke and pregnancy. J Neurol Neurosurg Psychiatry. 2008 Mar;79(3):240-5. Epub 2007 Nov 6. PMID: 17986502. (7). Kittner SJ, Stern BJ, Feeser BR, et al. Pregnancy and the risk of stroke. N Engl J Med 1996;335:768-74.








S
R


A
AX_GRE
R
Strength 1.5 T
rfl2d1r
500
25
5.00 mm
1.00
5.00
256 x 256
MR BRAIN W/O CONTRAST

Obstetric Anesthesiology - 13 It is all in the timing: Interesting neuromuscular reversal for patient in first trimester pregnancy with current lower respiratory infection undergoing a laparoscopic cholecystectomy

Fernando Alquicira-Macedo[1], Tewfeek Abu-Shami[2]

[1]UTMB, Galveston, TX, [2]UTMB, Galveston, United States of America

Introduction: It is common practice to proceed with neuromuscular reversal of a pregnant patient undergoing a non obstetric surgical procedure with neostigmine and atropine. However, what do you do when paralysis is necessary and the patient has a lower respiratory tract infection for which you want to avoid neostigmine? We present an interesting solution to this dilemma with our case presentation.

Methods: This is a case of a 25 year old G1P0 at 12 weeks gestational age undergoing an urgent laparoscopic cholecystectomy that had an active lower respiratory tract infection. Aside from the typical management of a pregnant patient needing general anesthesia for a non-obstetric surgical procedure, we also had this respiratory infection to worry about. Given that paralysis was asked for the case we had to think about the reversal. Normally a sugammadex reversal would be thought of these days. However, the SOAP task force recommendations suggest to avoid sugammadex early in pregnancy given that the literature is insufficient to recommend. Furthermore, the potential for sugammadex to bind to progesterone as has been seen in invitro studies could be disastrous given the need for progesterone to maintain pregnancy. (1) This leaves us with the use of neostigmine and atropine. Atropine may be preferred instead of glycopyrrolate because of the potential fetal bradycardia that can occur secondary to neostigmine. (2) Atropine crosses the placenta and can help minimize the potential fetal bradycardia. However, the use of neostigmine in respiratory infection could have significant bronchoconstriction. (3) Given the significant symptoms including fever, productive cough, and preoperative crackles/wheezing, neostigmine was not an option. We were stuck in this dilemma which brought us to consider the possibility of avoiding deep paralysis. After discussing with the surgical team, we came to an agreement and proceeded with paralysis with rocuronium however at only a moderate block with Train of Four (TOF) ratio 1:4. (4) An accelerometer was attached, and patient was kept at a moderate block with TOF ratio 1:4 for the whole case. As soon as insufflation was released and the trocars were taken out of the belly, close visualization was kept on the TOF. Closing was done by a medical student and resident which provided some time benefit. At the end of closure, the TOF ratio was >0.9 and no reversal agents were given. (5) An awake extubation was performed with patient following commands, spontaneously breathing, 5 second head lift, and adequate respiratory effort. No post operative complications were noted.

Conclusion: This case illustrates the option of waiting for neuromuscular relaxant to wear off in the rare instance where reversal agents might be contraindicated. Everything we do as anesthesiologists has a risk-benefit ratio and in our case the timing worked out perfectly. If the patient's infection was not as severe as she presented, the risk-benefit ratio might have favored in the direction of utilizing neostigmine.

References: 1.Society for Obstetric Anesthesia and Perinatology Statement on Sugammadex during pregnancy and lactation Ad Hoc task force: April 22,2019 2. Neostigmine, Atropine, and Glycopyrrolate: Does Neostigmine Cross the Placenta?. Anesthesiology 1996; 84:450–452. 3.Neostigmine. In: StatPearls ; 2021 Jan-. 4. Laparoscopic surgery and muscle relaxants: is deep block helpful? Anesth Analg. 2015 Jan;120(1):51-58. 5 Intraoperative Acceleromyography Monitoring Reduces Symptoms of Muscle Weakness and Improves Quality of Recovery in the Early Postoperative Period. Anesthesiology 2011; 115:946–954

Obstetric Anesthesiology - 14 Protein S Deficiency in Early Pregnancy with Ischemic Stroke and Saddle Pulmonary Embolism

Michael Chang[1], Iwan Sofjan[2]

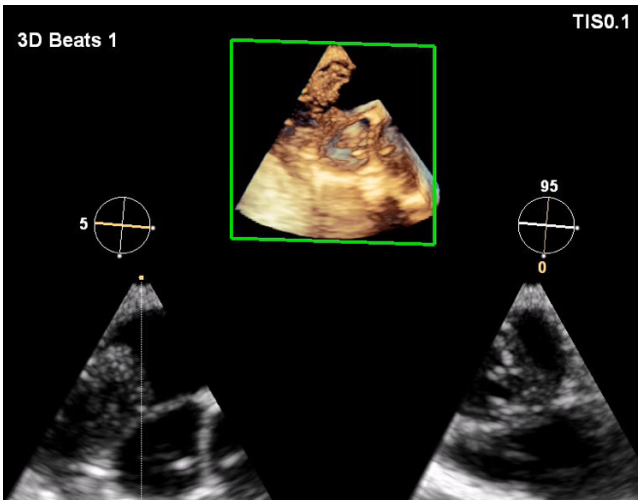
[1]Westchester Medical Center, Fresh Meadows, United States of America, [2]Westchester Medical Center, Valhalla, United States of America

Introduction: Thrombophillia is a state of hypercoagulation and may be due to inherited or acquired causes. One of the common causes of acquired thrombophillia is the state of pregnancy. This is due to physiologic changes in the hemostatic system which includes a functional resistance to activated protein C, decrease in protein S and antithrombin, and reduction of pathways that normally decrease fibrinolysis and platelet activation. Other factors such as advanced maternal age, obesity, immobilization, and the presence of inherited throbophillias increase the likelihood for arterial or venous thrombosis events in pregnancy. Thrombotic event continues to be a leading cause of maternal mortality in the United States as well as the world. According to the CDC's Pregnancy Mortality Surveillance System, 'thrombotic pulmonary or other embolism' and 'cerebrovascular accidents' account for 9.6 and 8.2% of pregnancy related deaths respectively. The combination of increasing maternal age and the obesity epidemic translates to a higher overall number of chronic medical co-morbidities accompanying the parturient, leading to increased risk of adverse maternal events. This is a case of a patient presenting in early pregnancy with a cerebrovascular event who then developed a massive saddle pulmonary embolism later in her hospital course.

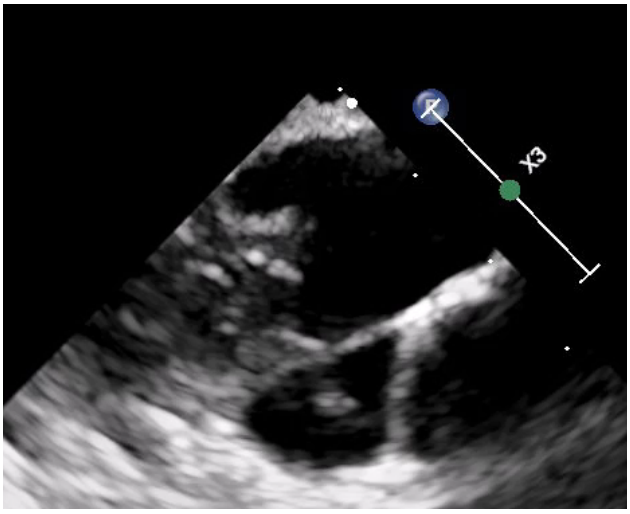
Methods: The patient was a 40-year-old female G2P0010 with a history of chronic migraine with aura and a right salpingo-oophorectomy. She also had a history of infertility and had 7 rounds of IVF treatment with preceding oral contraceptives (OCP). She initially presented to an outside hospital for recurrent left-sided upper and lower extremity hemiplegia. CTA showed a terminal R-ICA occlusion. She received tPa promptly and was transferred to our institution for endovascular thrombectomy (EVT). The EVT was successful and the patient had a progressive neurological improvement. Anticoagulation for acute ischemic stroke (AIS) prevention per institution protocol was initated after unremarkable repeat CTH 24 hours post EVT. Investigation to work up the cause of AIS was initially unremarkable. Unfortunately, on the 13th day of hospitalization, the patient suffered a major cardiopulmonary collapse requiring ACLS. Post-resuscitation TTE showed McConnell's sign consistent with right ventricular pressure/volume overload. She was brought emergently to the OR for a VA ECMO placement. Intraoperative TEE showed a right pulmonary artery embolism and a right-to-left shunt through a patent foramen ovale (PFO). The thrombophilia workup returned positive for protein S deficiency (level 29% of normal).

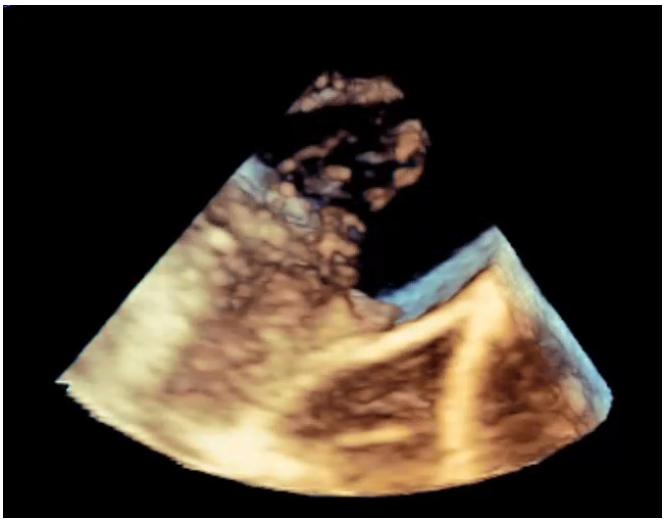
Conclusion: The body's hemostatic state is a fine balance of clotting proteins that are activated to promote fibrin clots when necessary and to reverse the process as needed. Studies looking at the physiological changes of pregnancy have described a decreased protein S level starting in the early stages of pregnancy. The main clinical implications of this thrombophilia include VTE, arterial thrombosis, and miscarriage during pregnancy. The patient did not have a known history of thrombophilia in her family, but due to her history of multiple IVF failure events, inherited protein S deficiency may be able to explain the multiple thrombosis events and the very low protein S level. Data in the literature suggests that inherited thrombophilia may play a role in the etiology of repeated IVF failures (4). Studies have attempted to tie migraine with aura to a thrombophilic state. In a study of nearly 1000 young adults with ischemic stroke, data demonstrated that migraine with aura was associated with an underlying thrombophilic state as well as with PFO. This is an area of ongoing research and debate. Of note, this patient did have a PFO with a right-to-left shunt during her acute pulmonary embolism. In summary, thrombophilia is more common during pregnancy and should be worked up thoroughly, especially in the presence of clinically relevant symptoms. Other significant thrombophilia-related complications such as pulmonary or other vascular embolisms should at least be considered and ruled out when possible. Our patient initially had a stroke, and an earlier diagnosis of protein S deficiency may or may not have altered her clinical course.

References: 1: Reprod Biol Endocrinol. 2003;1:111. Published 2003 Nov 14. doi:10.1186/1477-7827-1-111 2: MMWR Morb Mortal Wkly Rep. 2019;68:423–429. 3: Hematology Am Soc Hematol Educ Program. 2016;2016(1):243-250. doi:10.1182/asheducation-2016.1.243 4: Hum Reprod. 2004 Feb;19(2):368-70. doi: 10.1093/humrep/deh069. Erratum in: Hum Reprod. 2004 May;19(5):1238. Ben Ami, Ido [added]. PMID: 14747183. 5: Cephalalgia. 1998;18:618–621. 6: Clin App Thromb Hemostasis. 2014;20:851–856. 7: Stroke. 2011;42:17–21.

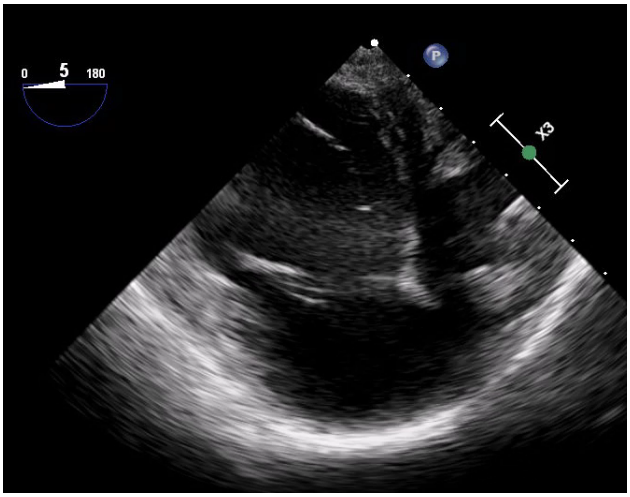


Right PA Clot

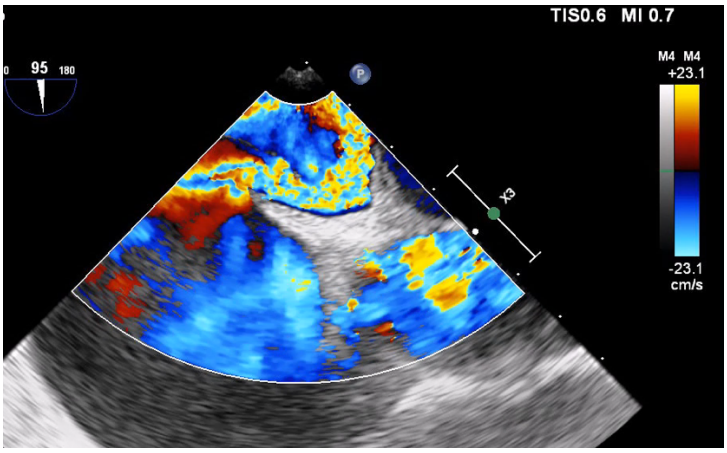


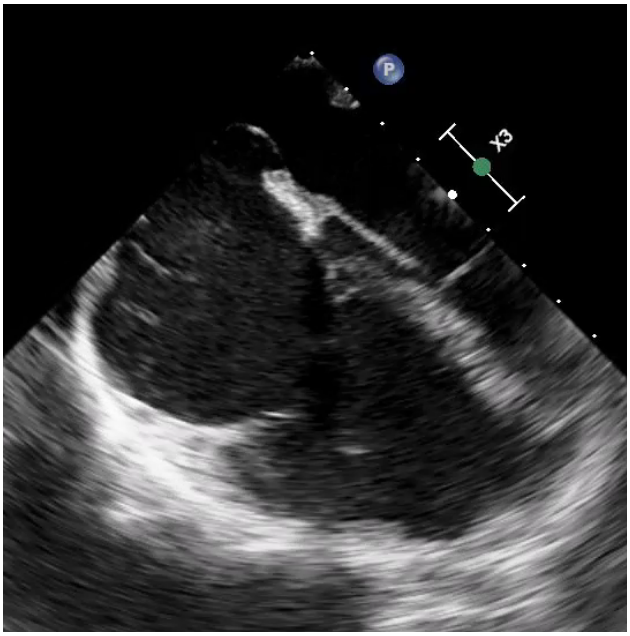


Dilated RV:



Right to left shunt through PFO;





Obstetric Anesthesiology – 15

Paradoxical embolism in a postpartum patient with Anti-phospholipid Syndrome & Atrial Septal Defect

Ioannis (Yanni) Angelidis[1], Amit Pawale[2], Michael Ault[3]

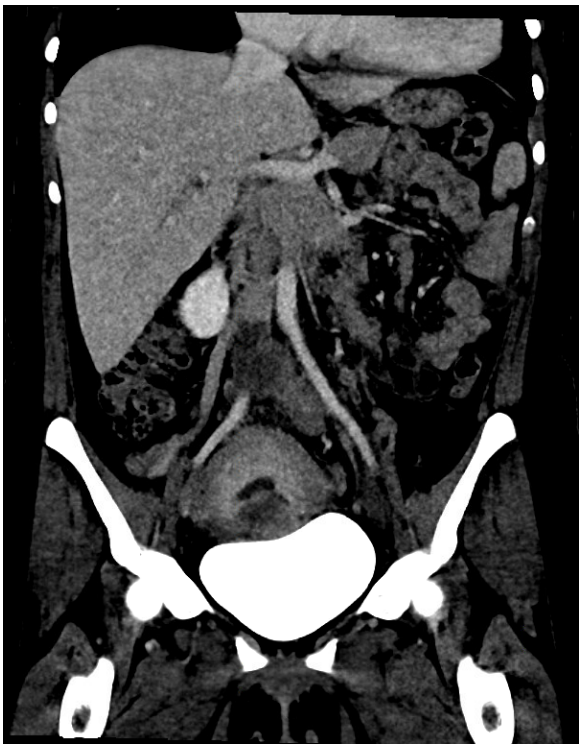
[1]University of Chicago, Pritzker school of medicine, Chicago, IL, [2]Northwestern University, Feinberg School of Medicine, Chicago, United States of America, [3]Northwestern University, McGaw Medical Center, Chicago, United States of America

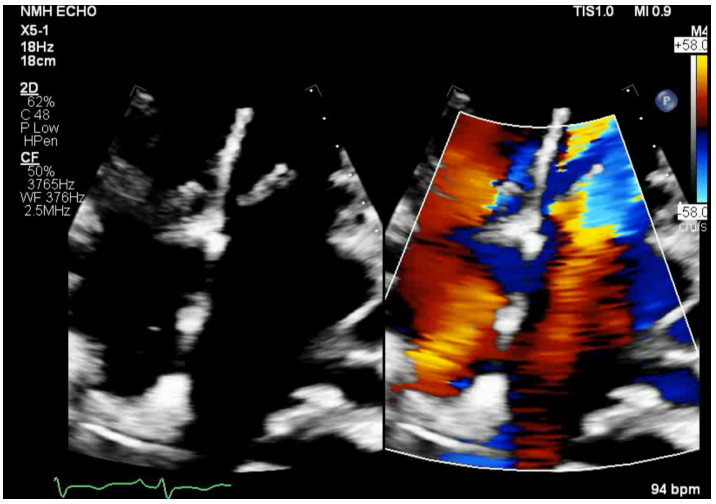
Introduction: Anti-phospholipid syndrome (APS) is an autoimmune disease and a severe prothrombotic condition. The incidence of APS in Caucasians is approximately 2 to 5 per 100,000 individuals per year and the prevalence is approximately 40 to 50 per 100,000.1

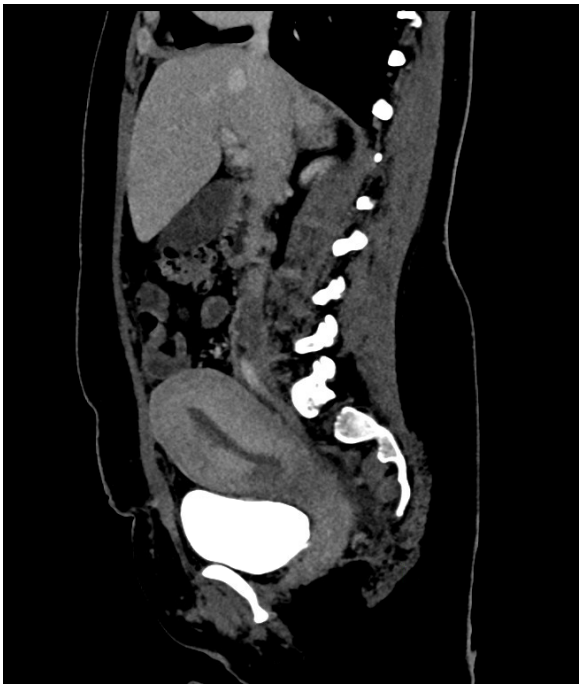
Methods: We present a case of a 19-year-old woman with a history of APS, preeclampsia with severe features, thrombocytopenia and atrial septal defect (ASD) who had a recent cesarean-section. She was admitted six days after delivery with left leg swelling and found to have deep venous thrombosis extending into the inferior vena cava (IVC) (FIG 1 and 2). MRI of the brain showed cerebellar strokes despite being on systemic anticoagulation. The transesophageal echocardiogram showed a large five cm secundum ASD (FIG 3) with severe pulmonary hypertension, severely dilated right atrium and right ventricle. Due to its large size, the ASD couldn't be repaired with a transcatheter occluder device so the patient was taken to the operating room for closure. The large thrombus in the IVC and her previous stroke increased her risk for more severe cerebrovascular accidents. The ASD was repaired, and the patient was transferred to the intensive care unit on no vasopressors where she was monitored for one day. APS can present with thrombosis, recurrent early pregnancy loss, fetal death and preterm delivery due to severe preeclampsia. It can be confirmed by one or more repeatedly positive antiphospholipid (aPL) antibodies on two or more occasions at least 12 weeks apart.1,2 Clinical assessment is important in diagnosis as only a small proportion of individuals who are positive for aPL antibodies develop APS.3 Occurrence of stroke due to paradoxical embolism in patients with APS and congenital heart defects (CHD) has been previously documented. Tanaka et al. showed a statistically significant association between patent foramen ovale and atrial septal aneurysm and stroke in patients with APS.3 A heparin agent is the recommended treatment during pregnancy, and while it improves outcomes over 70%, around 30% of patients can suffer severe pregnancy outcomes.1

Conclusion: Physicians should be aware of the possibility of the presence of CHDs in patients with APS. Transthoracic echocardiography is a good initial assessment tool, and a multi-disciplinary approach that includes maternal and fetal medicine, rheumatological, and hematological expertise would be beneficial.3

References: 1. Arslan E, Branch DW, Scott JR. Antiphospholipid syndrome: Diagnosis and management in the obstetric patient. Best Practice & Research Clinical Obstetrics and Gynaecology 64 (2020) 31-40. 2. Czwalinna A, Bergmann F. Prevention of Pregnancy Complications in Antiphospholipid Syndrome. Hämostaseologie 2020;40:174–183. 3. Tanaka Y, Ueno Y, Miyamoto N et al. Patent foramen ovale and atrial septal aneurysm can cause ischemic stroke in patients with antiphospholipid syndrome. J Neurol (2013) 260:189–196.




NMH ECHO
X5-1
18Hz
18cm
2D
62%
C 48
P Low
HPen
CF
50%
3765Hz
WF 376Hz
2.5MHz
TIS1.0 MI 0.9
94 bpm



Obstetric Anesthesiology - 16 ICU Management in a Case of Amniotic Fluid Embolism after an Uneventful Vaginal Delivery: A Case Report.

Rosa Lopez-Rincon[1], Miguel Plaza-Lloret[2], Julio Medina[3], Efrain Riveros Perez[4], Akbar Herekar[3]

[1]Medical College of Georgia - Augusta University, Augusta, GA, [2]Medical College of Georgia - Augusta University, Evans, GA, [3]Mcg Augusta University, Augusta, GA, [4]Medical College of Georgia. Augusta University, Augusta, GA

Introduction: Amniotic fluid embolism (AFE) is a rare and catastrophic complication unique to pregnancy and delivery. The characteristic cardiovascular collapse related to this anaphylactoid reaction is remarkable for a maternal mortality rate of 13-26% per recent studies. We present a case of amniotic fluid embolism in a patient admitted to ICU who successfully recovered from this fatal syndrome. [1][3][5]

Methods: The 39-year-old female with a past medical history of Placenta Previa and smoker presented to the hospital for elective vaginal delivery at 39.1-week gestation, which was uneventful. Extensive vaginal bleeding post-delivery required IV Oxytocin and Carboprost for uterine atony. The patient ultimately became short of breath, experienced chest pain, and went into respiratory distress with an episode of seizure-like activity around 20 minutes postpartum. Hypotension of SBP 85 mmHg and tachycardia were also noted. Emergent oral intubation and arterial line placement were performed with raising concerns for Amniotic fluid embolism (AFE) Vs Pulmonary embolism (PE) Vs Eclampsia. A bedside Transthoracic Echo revealed mild right ventricle (RV) dilation, left ventricular ejection fraction (LVEF) 40-45% and hyperdynamic left ventricle (LV). Her laboratory results were remarkable for platelets 150 th/mm3, Hb 8.8 g/dl, lactic acid 2.5 mmol/L, fibrinogen 557 mg/dl, INR 1, PT 11.3 secs and PTT 25.5 secs and troponin < 0.01 ng/ml. She was transferred intubated to ICU and supported with IV crystalloids. TEG (Thromboelastogram) showed increased LY30 but Tranexamic acid (TXA) administration was held at that time. IV Norepinephrine and Epinephrine infusions were started for persistent hypotension and empiric IV Heparin drip was added until a CTA chest ruled out PE. Then, it was switched to SQ Heparin, and TXA was administered. The patient also experienced fever in the postpartum period with a temperature of 41 degrees Celsius and thus empiric antibiotics were ordered. A complete Transthoracic echo showed mild concentric left ventricular hypertrophy with LV systolic function severely reduced and LVEF 28%. TEG then was compatible with DIC. Hemodynamic support was optimized, diuretics were added and she continued mechanically ventilated. Around 30 hs after the episode, the patient was extubated and vasopressors requirements weaned off. Guideline-directed medical therapy (GDMT) was started. On day 4 the LVEF showed a significant improvement to 45-50%, the patient remained asymptomatic and she was transferred to the floor.

Conclusion: AFE, also known as the anaphylactoid syndrome of pregnancy, has an incidence in the US from 2 to 8 cases per 100,000 deliveries causing multiorgan failure [1][4][5][6]. The finding of fetal elements or amniotic fluid into the pulmonary circulation is the most accepted cause but the pathophysiology remains unknown [2][6]. The American Society for Maternal-Fetal Medicine (SMFM) described four diagnostic criteria for AFE: DIC, acute cardiorespiratory arrest or both hypotension (SBP < 90 mmHg) with respiratory dysfunction, no fever present during labor and clinical onset during labor or within 30 minutes of placental delivery [6]. The patient in this case met the four proposed criteria: severe hemodynamic instability with respiratory failure and biventricular heart failure, delayed DIC, clinical onset 20 minutes after placental delivery and afebrile. The cornerstone treatment for this syndrome is supportive, adequate control of the airway, correcting the coagulopathy, and prompt aggressive intensive care management [2][3][4]. Transthoracic, or transesophageal echocardiography is essential to help to guide the resuscitation. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) turn to be very useful in decision-making. The use of recombinant factor VIIa (rfVIIa) in recent articles has shown worse outcomes [5].

AFE is a life-threatening disorder during pregnancy associated classically with a poor maternal prognosis. Early recognition, diagnosis, and optimum

resuscitative management may impact in declining the risk for maternal mortality secondary to this event [3][4][5]. A multidisciplinary team including neonatologists, obstetricians, intensivists, labor and delivery nurses, and anesthesiologists should be prepared to manage this uncommon syndrome.

References: 1. Amniotic Fluid Embolism, Obstetric Anesthesia Digest: December 2010-Volume 30-Issue 4-p 205-206. 2. Amniotic-fluid embolism and medical induction of labor: a retrospective, population-based cohort study. The Lancet, Volume 368, Issue 9545, 2006, Pages 1444-1448. 3. Amniotic fluid embolism. J Anaesthesiol Clin Pharmacol. 2016;32(2):153-159. 4. Proposed diagnostic criteria for the case definition of amniotic fluid embolism in research studies. Am J Obstet Gynecol. 2016 October; 215 (4): 408-412. 5. Amniotic fluid embolism: antepartum, intrapartum and demographic factors. The journal of maternal-fetal & neonatal medicine: the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians. 2015 May. 6. Society for Maternal-Fetal Medicine (SMFM). Amniotic fluid embolism: diagnosis and management. Am J Obtet Gynecol 2016; 215:B16-24.

Pain Medicine

Pain Medicine - 1 A Unique Case of Complex Regional Pain Syndrome after COVID-19 Infection

Chris Sobey[1], Samir Khan[1], Christian Roehmer[1], John Corey[1], Puneet Mishra[1]

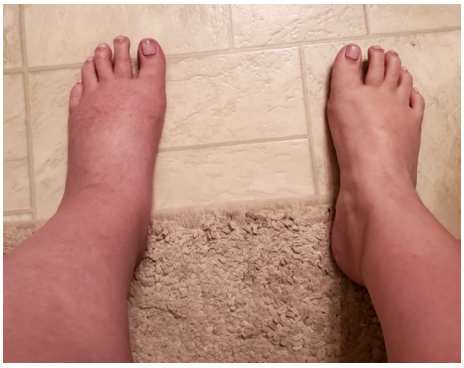
[1]Vanderbilt University Medical Center, Nashville, TN

Introduction: The long-term complications of infection from the novel coronavirus SARS-CoV-2 (COVID-19) are unknown. Although primarily spread through respiratory droplets, most commonly resulting in respiratory tract pathology, recent data suggest that virtually no organ system is spared [1]. The nervous system may be affected through direct invasion of nerves via axonal or retrograde transport [2]. When COVID-19 symptoms persist for more than four weeks, the infection is considered to be 'long COVID,' also known as post-COVID syndrome (PCS) [3]. The purpose of this study is to share a unique case of complex regional pain syndrome (CRPS) that developed after COVID-19.

Methods: A 53-year-old female with a history notable for hypertension, obesity, and COVID-19 was referred for persistent bilateral lower extremity pain that had emerged one year prior, after having contracted a severe case of COVID-19. The pain was localized to her distal lower extremities in a non-dermatomal distribution and was worse on the right. Pain was described as constant, shooting, 'electric,' and was rated 10/10 in severity. Additionally, she related symptoms of intermittent swelling, temperature asymmetry, and color changes to both lower extremities. Her painful symptoms worsened with weightbearing and had been refractory to several medications and interventional therapies, including lumbar sympathetic block. Her pain improved modestly with a combination of gabapentin, amitriptyline, and duloxetine. Her physical exam was notable for lower extremity edema, allodynia, erythema, warmth, and weakness, which was more prominent on the left. Previous imaging modalities had ruled out acute pathologies such as fracture or venous thromboembolism. Notably, due to persistent supplemental O2 requirements and weakness necessitating a walker, she had been diagnosed with PCS by an outside provider.

Conclusion: Given the patient's disproportionate pain, along with sudomotor, vasomotor, and sensory changes, the patient met Budapest Criteria for a diagnosis of bilateral lower extremity CRPS, with no alternative explanatory diagnosis. Initially, critical illness polyneuropathy was contemplated, but this did not explain the patient's sudomotor and vasomotor symptoms. Electromyography was considered but deferred due to patient intolerance. CRPS Type I was diagnosed due to lack of evidence of direct nerve damage. Spinal cord stimulation was offered as the next step in treatment [4]. Several viruses are known to induce pain syndromes, specifically neuropathic pain, and SARS-CoV-2 has been theorized to carry similar potential [5,6]. To our knowledge, this is the first reported case of CRPS as a sequela of COVID-19. It is important for physicians to recognize CRPS as one possible condition associated with PCS, adding to the broad spectrum of pathologies related to COVID-19.

References: 1. Mechanisms of SARS-CoV-2 Transmission and Pathogenesis. Trends Immunol. 2020;41(12):1100-1115. 2. Update on neurological manifestations of COVID-19. Life Sci. 2020;257:118063. 3. Post-acute COVID-19 syndrome. Nat Med. 2021;27(4):601-615. 4. Spinal cord stimulation for complex regional pain syndrome: a systematic review of the clinical and cost-effectiveness literature and assessment of prognostic factors. Eur J Pain. 2006;10(2):91-101. 5. Potential for increased prevalence of neuropathic pain after the COVID-19 pandemic. Pain Rep. 2021;6(1):e884. 6. Considering the potential for an increase in chronic pain after the COVID-19 pandemic. Pain. 2020;161(8):1694-1697.



Pain Medicine - 2 Spinal Cord Stimulation for Sciatic Pain in Seronegative Neuromyelitis Optica Spectrum Disorder

Shayla Mena[1], Ramanjot S Kang[2], William Caldwell[1]

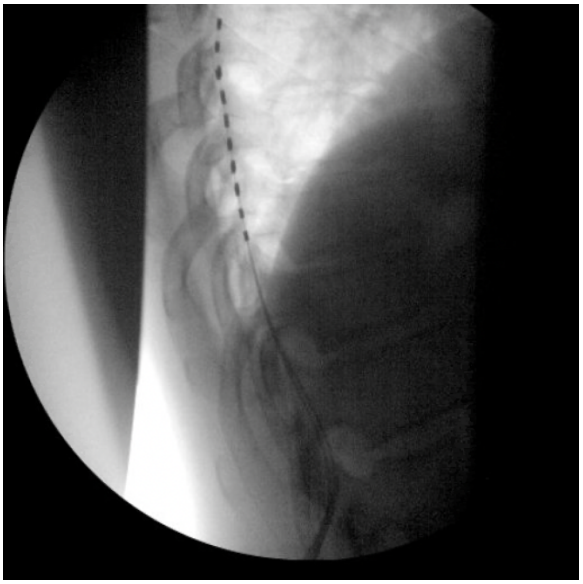
[1]Stony Brook Medicine, Stony Brook, NY, [2]Stony Brook University, Stony Brook, NY

Introduction: With the success of spinal cord stimulation (SCS) in treating chronic pain conditions, there has been a considerable increase in the off-label use of SCS to treat other pain conditions. Specifically, there are reports in the literature of SCS being using to treat pain related to rare autoimmune, inflammatory, and demyelinating diseases. For example, SCS has been used to treat spasticity and improve motor function in patients with multiple sclerosis and Parkinson's disease.[1, 2] More recently, new types of SCS, including high-frequency technology, dorsal root ganglion stimulation, burst stimulation, and other waveforms, have opened the door for treating other chronic pain conditions such as painful diabetic neuropathy.[3, 4] As SCS technology develops further, an increasing number of patients with rare diseases may benefit from this treatment modality. Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune disease of the central nervous system that primarily affects the optic nerves and spinal cord. The majority of patients test positive for serum antibodies targeting the aquaporin-4 channel expressed on astrocytes, and it is estimated that 1-2 people per 100,000 suffer from this disorder.[5] Some patients may test negative for aquaporin-4 channel antibodies, and still others present with varying degrees of radicular pain and paroxysmal tonic spasm.[6, 7] The treatment for this disorder involves immunosuppressive therapy and there is no cure. Here we present the case of a patient with NMOSD that was treated with SCS.

Methods: The patient was a 32 year-old wheelchair-bound female that presented to our clinic in 2019 with complaints of sciatic pain in the left hip and leg. The patient had originally been misdiagnosed with multiple sclerosis in 2011 and was later diagnosed with seronegative NMOSD and Ehlers-Danlos syndrome. Her pain had gradually worsened since onset a few years prior, and the pain was rated as the worst pain she had experienced in her life. She described it as a sharp and shooting sensation and had attempted treatment with over the counter medications, muscle relaxers, ice packs, and light stretches which did not alleviate her pain. Although she was able to complete some of her activities of daily living, she endorsed overall limitations due to the pain. A magnetic resonance imaging study demonstrated degenerative disc disease and disc herniation at the L4-L5 level. After careful consideration of alternative treatment options, the patient opted for bilateral transforaminal epidural steroid injections (ESIs) at L4-L5. The procedure was efficacious in abating her pain severity and radiculopathy. However, over the following two years, she required repeat injections and her degenerative disc disease progressed into her cervical spine. After a total of eight bilateral transforaminal ESIs and two cervical ESIs, the patient reported decreasing efficacy. At that time, it was explained that she could continue to receive ESIs to a maximum of four per year while continuing conservative management, continue with conservative measures only, or consider a SCS trial. The patient desired an additional bilateral transforaminal L4-L5 ESI, after which she consented to moving forward with a SCS trial. Under monitored anesthesia care in the ambulatory surgery center, a spinal cord stimulator (Medtronic) was trialed with leads placed to T8 and the patient was given one week to assess improvement in pain severity. During follow up, she endorsed excellent relief of her pain and increased mobility due to the improvement in her pain level. The trial leads were pulled, and she was subsequently scheduled for permanent implantation of a spinal cord stimulator.

Conclusion: Our cases demonstrate successful use of SCS for the treatment of sciatic pain in a female with seronegative NMOSD. To our knowledge, there are no cases reported in the literature where SCS proved to be an effective treatment for pain in patients diagnosed with this rare pathology. In a study regarding the association of pain and quality of life in patients with NMOSD, pain severity proved to be the strongest negative predictor of quality of life.[8] Therefore, clinicians should be aware that some patients diagnosed with NMOSD may benefit from receiving a spinal cord stimulator.

References: 1. Spinal cord stimulation for Parkinson's disease: a systematic review. Neurosurg Rev. 2016;39:27-35 2. Spinal Cord Stimulation for Spasticity: Historical Approaches, Current Status, and Future Directions. Neuromodulation. 2017;20:307-21 3. Spinal Cord Stimulation: Clinical Efficacy and Potential Mechanisms. Pain Pract. 2018;18:1048-67 4. 10-kHz spinal cord stimulation treatment for painful diabetic neuropathy: results from post-hoc analysis of the SENZA-PPN study. Pain Manag. 2020;10:291-300 5. New therapies for neuromyelitis optica spectrum disorder. Lancet Neurol. 2021;20:60-7 6. Neuromyelitis optica spectrum disorder: an overview. Acta Neurobiol Exp (Wars). 2020;80:256-72 7. Seronegative neuromyelitis optica spectrum disorder: severe polysymptomatic presentation with successful treatment response. BMJ Case Rep. 2019;12 8. Patient-reported burden of symptoms in neuromyelitis optica: A secondary analysis on pain and quality of life. J Neurol Sci. 2021;428:117546



Pain Medicine - 3 Management of Headache After Removal of Intrathecal Drug Delivery System

Kruti Parikh[1], Nahla Mosleh[2], Surjya Sen[2]

[1]Rutgers New Jersey Medical School, Newark, NJ, [2]Hackensack University Medical Center, Hackensack, NJ

Introduction: Intrathecal drug delivery systems (IDDS) were first developed for management of chronic malignant pain, but have since been used to manage resistant nonmalignant pain states and spasticity. For placement of an IDDS, a 14-gauge Tuohy needle is used to enter the lumbar intrathecal space via an oblique paramedian approach. Once the Tuohy needle pierces the dura and cerebrospinal fluid (CSF) flow is noted, a catheter is advanced through the needle to the appropriate level under fluoroscopic guidance. Once the needle is removed, a suture is used to create a purse-string suture around the needle entry point to help prevent catheter movement and potential CSF leakage (1). A pump is then surgically placed into an abdominal pocket, the pump catheter is subcutaneously tunneled, and then connected to the intrathecal catheter. This pump then delivers preservative free medications directly into the CSF (2). We report the case of patient who had an IDDS in place for six years that was administering high dose opioid, and subsequently developed headache after its removal. We discuss our management options based on our differential diagnosis of post dural puncture headache (PDPH) vs. opioid withdrawal headache.

Methods: A 52-year-old male patient with past medical history of complex regional pain syndrome (CRPS) and Parsonage Turner syndrome presented with severe headache several hours after surgical removal of his intrathecal pump which had been in place for six years. Per patient, his pump was delivering 4mg Dilaudid daily as well as bupivacaine. Prior to removal, he was weaned off his high dose opioid in a step-wise fashion with the help of an outpatient pain management specialist. Per surgical report, a small laminectomy was required at L1-2 to expose the catheter entry into the thecal sac since there was scar tissue surrounding the catheter making it difficult to remove. No CSF leak was noted during the procedure. The dura was closed with figure-8 sutures, and an adjunct hemostatic agent was used over the small L1-2 laminectomy site. Our pain service was consulted hours after removal of the intrathecal pump for severe headache and continued generalized pain. The patient described his headache as 10/10 in severity, started 2 hours after surgery, and somewhat relieved with laying flat. Our differential diagnosis for his headache included PDPH vs opioid withdrawal headache. Our management included starting a medication regimen of PO hydromorphone, butalbital, baclofen, gabapentin, ketorolac, diazepam; advising bed rest, laying flat, and encouraging caffeine intake. Ultimately, the patient's headache resolved in 36 hours with our conservative management measures.

Conclusion: Headache after removal of a longstanding IDDS that had been administering high dose opioid could be a result of two main etiologies: PDPH or opioid withdrawal headache. In this patient, the dural fibers likely developed scar tissue and created risk for ongoing CSF leak even after surgical closure, predisposing the patient to PDPH. Alternatively, the patient was at risk of developing a headache secondary to opioid withdrawal because of his high pump dose. PDPH typically presents as bilateral frontal or occipital headache that is worse in the upright position and can be associated with nausea, visual changes, and neck pain. PDPH is initially managed conservatively, but the most definite therapy for PDPH is epidural blood patch, which has a success rate of 77-96% (3). A recent retrospective review of cases showed that up to 23% of patients developed PDPH symptoms after IDDS implantation (4). Opioid withdrawal typically presents as nausea, rhinorrhea, diarrhea, muscle cramps, and headache. A Short Opioid Withdrawal Scale (SOWS) (TABLE 1) has been developed as a scoring system to monitor symptoms of withdrawal and is used to guide management (5). In conclusion, we report the case of a patient who presented with headache of unknown etiology after removal of an intrathecal pump. There are limited reports in the literature addressing the optimal management of headache after IDDS removal, and we believe that further investigation is justified on this topic. We wonder if prophylactic epidural blood patch should become standard of care after every insertion, exchange, and removal of IDDS to best prevent CSF leaks. Additionally, it is important that

providers realize that optimal opioid weaning should be facilitated prior to removal of IDDS.

References: 1. Belverud S, Mogilner A, Schulder M. Intrathecal pumps. Neurotherapeutics. 2008;5(1):114–22. 2. Ghafoor VL, Epshteyn M, Carlson GH, Terhaar DM, Charry O, Phelps PK. Intrathecal drug therapy for long-term pain management. Am J Health Syst Pharm. 2007;64(23):2447–61. 3. Bezov D, Ashina S, Lipton R. Post-dural puncture headache: Part II--prevention, management, and prognosis. Headache. 2010;50(9):1482–98. 4. Neuman SA, Eldrige JS, Qu W, Freeman ED, Hoelzer BC. Post dural puncture headache following intrathecal drug delivery system placement. Pain Physician. 2013;16(2):101–7. 5. Gossop M. The development of a short opiate withdrawal scale. Addictive Behaviors. 1990;15:487–490.

Symptom	Not present	Mild	Moderate	Severe
Feeling sick	0	1	2	3
Stomach cramps	0	1	2	3
Muscle spasms or twitching	0	1	2	3
Feeling cold	0	1	2	3
Heart pounding	0	1	2	3
Muscular tension	0	1	2	3
Aches and pains	0	1	2	3
Yawning	0	1	2	3
Runny/watery eyes	0	1	2	3
Difficulty sleeping	0	1	2	3

Score	Suggested withdrawal management
0-10	Mild withdrawal; symptomatic medication only
10-20	Moderate withdrawal; symptomatic or opioid medication
20-30	Severe withdrawal; opioid medication

TABLE 1. Short Opioid Withdrawal Scale (SOWS) has been developed as a scoring system to monitor symptoms of withdrawal and is used to guide management.

Pain Medicine - 4 Meralgia Paresthetica caused by Hibernoma in the thigh: a rare diagnosis

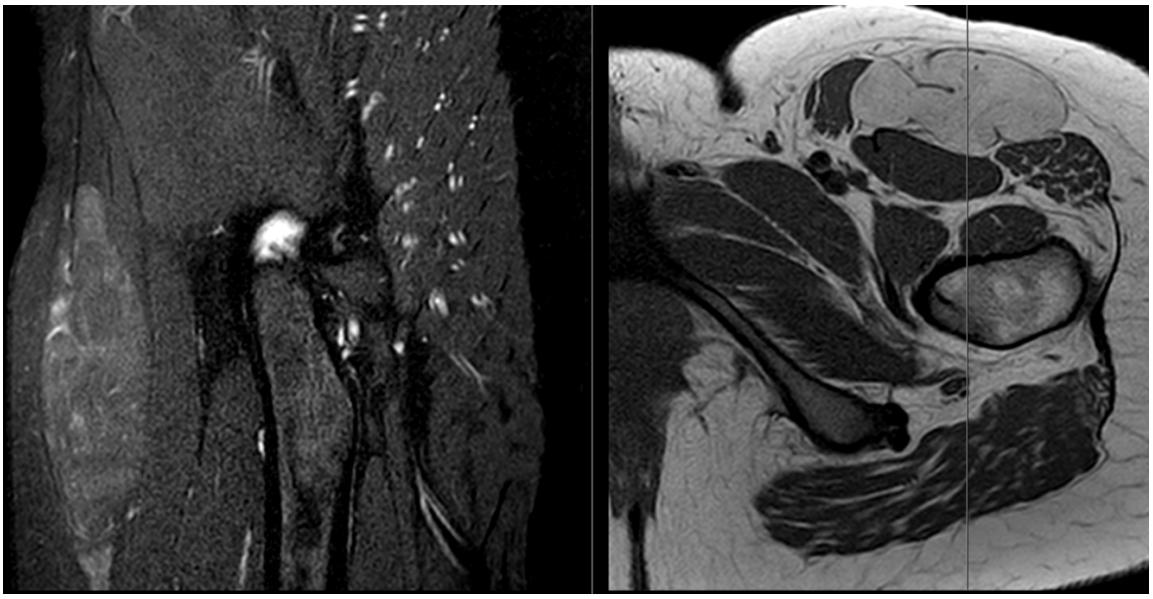
Krishna Shah[1], Sean Fletcher[2]

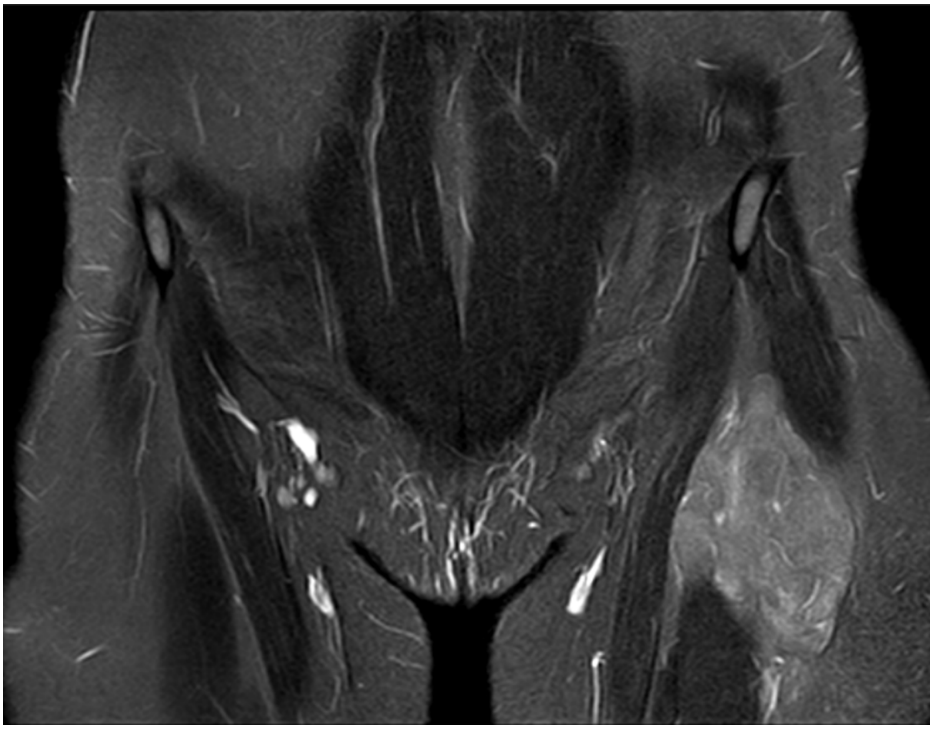
[1]Medical College of Georgia, Milton, GA, [2]Dwight D. Eisenhower Army Medical Center, Augusta, GA

Introduction: Young previously healthy female with no reported trauma presented with insidious onset lateral thigh numbness and groin pain, gradually worsening over time. This pain was not relieved by pain relieving techniques, and was refractory to conservative management. Imaging showed a large soft tissue mass with local mass effect, concerning for malignant sarcoma. Wide local excision biopsy was performed, with histologic features consistent with Hibernoma, a rare soft tissue brown fat tumor. Post-operatively, she had dense sensory deficits in the distribution of the LFCN, and the pain had not subsided.

Methods: A 26-year-old previously healthy female with no pre-existing co-morbid conditions suffers from three years of atraumatic left hip pain. Pain and numbness began at the left lateral groin which gradually increased and worsened with usage and time. Upon physical exam, Tinel's sign over inguinal crease recreated thigh paresthesia, FABER/FADIR test, Log Roll test, Stinchfield test were positive. Normal sensation to light touch was present bilaterally, except for loss of sensation over left anterolateral thigh to the area just proximal to knee. She notes continued 3/10 stabbing knife like groin pain that increases to 5/10 with prolonged sitting and 7-8/10 with repetitive squatting motions. Non-steroidal anti-inflammatory medications and repeated icing provided no relief. Upon MRI imaging, a soft tissue mass in left anterior thigh was found and excised, and histopathology confirmed a Hibernoma. The fatty tissue measured 11.9x8.7x5.6cm and weighed 131 grams, which was likely causing mass effect, resulting in meralgia paresthetica. After resection, no relief in pain was found, and incidentally, new onset distal thigh paresthesia's and hypersensitivity was noted. Conservative management of ibuprofen, physical therapy for 8 months, as well as repeat intra-articular corticosteroid injections under fluoroscopy did not provide relief.

Conclusion: Meralgia paresthetic, a painful mononeuropathy, is a common diagnosis, typically caused by external compression of the LFCN, due to excess body weight, seat belt injuries, and tight clothing. Hypoesthesia and pin and needle pain are common symptoms of this syndrome. Although LFCN has no motor function, it does have sensory function, as it travels under the inguinal ligament, and travels superficially on the sartorius muscle, and transverses across the anterior thigh. This patients tumor that was located in the anterior compartment of the thigh, anterior to the rectus femoris muscle, and between the vastus medialis and lateralis muscles, caused local mass effect, resulting in meralgia paresthetica due to direct compression. It was believed that surgical excision would relieve the compression of the LFCN. In the literature, meralgia paresthetica is noted to occur spontaneously with a reported incidence of 32.6 per 100,000 in the general population and 23.8% of postoperative patients undergoing posterior spine surgery. However, due to consistent pain post-operatively, it is more likely that during excisional biopsy, the LFCN likely underwent neurotmesis resulting in dense sensory deficits. As a secondary form of pain treatment, after failed therapies of nonsteroidal anti-inflammatory drugs, continuous physical therapy, and avoidance of external triggers, corticosteroid injections under fluoroscopy was considered. Intra-articular corticosteroid injections at the L2 and L3 facet have a success rate of about 56%, however, did not prove to be successful for this patient, who had LFCN secondary to a rare tumor. This patient has further irritation of the LFCN that has been resistant to multiple pain management techniques, that could be idiopathic, a result of the hibernoma, or a result of the surgical excision of the hibernoma, alleviating the original pain that the patient experienced, but then aggravating other peripheral branches in the thigh during surgery. This case demonstrates a rare etiology for a relatively common pain diagnosis.





Pain Medicine - 5 "What do you mean I can't have 3 mg of Hydromorphone IV push every 3 hours?" An ethical case of institutional change after a patient services phone call

Fernando Alquicira-Macedo[1], Siyun Xie[1], Axel Rodriguez[1], Beth M Teegarden[2]

[1]UTMB, Galveston, TX, [2]The University of Texas Medical Branch at Galveston, Galveston, TX

Introduction: Acute intermittent porphyria (AIP) is an autosomal dominant disorder caused by partial deficiency of porphobilinogen deaminase (PBGD), an enzyme in the heme biosynthetic pathway, leading to accumulation of PBG and delta-aminolevulinic acid (ALA) and uncontrolled upregulation of ALA synthase (1). This accumulation of porphyrin precursors and ALA synthase (ALAS), as well as a deficiency in heme, is what leads to acute attacks of neurovisceral symptoms including abdominal pain, nausea/vomiting, and peripheral neuropathy (2). Rapid treatment of pain is critical since the stress from pain can contribute to neuroendocrine reactions, activating ALAS and possibly exacerbating attacks (3). Pain secondary to attacks is often treated with intravenous (IV) narcotics as oral medications are limited by nausea/vomiting. Patients suffering from recurrent attacks requiring chronic use of opioids are at risk of opioid dependence (3). An increase in tolerance to narcotics may lead to providers feeling unsafe with administration of escalating doses, especially during an opioid epidemic. This scenario created an ethical dilemma of inadequately treating a patient's pain because it required doses that significantly exceed usual dosing ranges. It also led to institutional change with the implementation of a subanesthetic ketamine infusion protocol outside of the Intensive Care Unit (ICU) for the treatment of acute pain.

Methods: We present a case of a 30-year-old female admitted for an exacerbation of AIP who called patient services because of inadequate pain control. The patient was requesting 3 mg of hydromorphone IV push q3H and refusing acute pain service recommendations that included hydromorphone patient-controlled analgesia (PCA). The advantage of a PCA is that it can be programmed conservatively to minimize risks of respiratory depression. A 2018 systematic literature review on PCA use in cancer pain found many studies that supported the safe use of PCA, with only rare occurrences of severe sedation and respiratory depression (4). Although the patient was amenable to a subanesthetic ketamine infusion, it was not possible due to high COVID-19 admissions and limited ICU beds; ketamine infusion at the time was only available in the ICU at our institution. We attempted to administer ketamine infusion on the floor, but significant pushback was presented by nursing staff given their lack of experience with this medication. The most common misconception was the thought that ketamine could only be used as an anesthetic, although it is a potent analgesic at subanesthetic doses (5). The benefit of ketamine is that it maintains one's respiratory drive and has minimal concern for respiratory depression as opposed to the large doses of narcotics (5). The patient was also found to be exhibiting highly suspicious behavior concerning for drug-seeking and addiction, prompting an ethics consult. While we were limited in what we could offer our AIP patient secondary to lack of ICU beds and patient refusal of other modalities, we have since trained floor nursing staff and successfully implemented a ketamine infusion protocol for use on intermediate bed units. For the creation of the protocol, we utilized the 2018 consensus guidelines on the use of intravenous ketamine infusion for acute pain management, which recommends acute pain infusions of 1.67-8.33 µg/kg/min to provide an adequate balance between analgesia and adverse effects (6). Our protocol is very conservative and has a range of 0.5-3 µg/kg/min (Figure 1).

Conclusion: This case highlights many dilemmas in anesthesiology practice including challenges with acute pain management in opioid-tolerant patients. This subset of patients commonly refuses the PCA and multimodal approaches to pain management and instead, requests high-dose IV narcotic boluses. Ketamine is increasingly being used in the inpatient setting under the acute pain service to manage such patients (6). Given our institution's lack of a ketamine infusion protocol outside of the ICU, a multidisciplinary team was created to implement a protocol (Figure 1). This case also highlights the continued stress that COVID-19 is imposing on our healthcare system given the ramifications of limited ICU space. Despite these

challenges, implementation of a subanesthetic ketamine infusion protocol can be an effective adjunct in combating the ethical dilemmas that come with treating opioid-tolerant patients.

References: 1. Acute Intermittent Porphyria: Predicted Pathogenicity of HMBS Variants Indicates Extremely Low Penetrance of the Autosomal Dominant Disease. Hum Mutat. 2016 Nov;37(11):1215-1222. 2. Acute porphyrias in the USA: features of 108 subjects from porphyrias consortium. Am J Med. 2014 Dec;127(12):1233-41. 3. Therapeutic strategies for acute intermittent porphyria. Intractable Rare Dis Res. 2020 Nov;9(4):205-216. 4. Subcutaneous or intravenous opioid administration by patient-controlled analgesia in cancer pain. Support Care Cancer. 2019 Jan;27(1):33-42. 5. Impact of Low-Dose Ketamine on the Usage of Continuous Opioid Infusion for the Treatment of Pain in Adult MechanicallyVentilated Patients in Surgical Intensive Care Units. J Intensive Care Units. J Intensive Care Med. 2019 Aug;34(8):646-651. 6. Consensus Guidelines on the Use of Intravenous Ketamine Infusions for Acute Pain Management From the ASRA and Pain Medicine, the AAPM, and the ASA. Reg Anesth Pain Med. 2018 Jul;43(5):456-466.

INTRAVENOUS KETAMINE REFERENCE for NURSING- Intermediate Units

Drug	Ketamine (Schedule III Control)
Class	Non-opioid, N-methyl-D-aspartate (NMDA) receptor antagonist and opioid receptors agonist
Indications	▪ **FDA approved:** General anesthetic ▪ **Common Off Label:** Analgesia, refractory major depressive disorder, procedural sedation ▪ **New Off Label:** Neuroprotection, bronchospasms, migraines
Pharmacology/ Pharmacokinetics	▪ **Mechanism:** Non-competitive NMDA-receptor antagonist acting on CNS to block pain message ▪ **Metabolism:** Hepatic ▪ **Onset:** IV (~30 seconds) & IM (3 – 4 minutes) ▪ **Duration of Action:** 5 – 30 minutes ▪ **Half-life:** 10 – 15 minutes ▪ No renal dose adjustments needed ▪ Continuation of infusion should be re-evaluated at 3 days
Precautions	**Caution in:** ▪ Cardiovascular diseases (coronary artery disease, catecholamine depletion, hypertension, tachycardia) ▪ History of substance abuse ▪ History of psychosis & seizure disorders ▪ History of increased intracranial pressure ▪ Active pregnancy ▪ History of head injury ▪ Uncontrolled hypertension

Adverse Effects

CARDIAC	NEUROLOGICAL	GASTROINTESTINAL	OTHER
• Tachycardia • Hypertension • Bradycardia • Hypotension • Arrhythmias	• Vivid dreams • Hallucinations • Emergence reactions • Increased ICP	• Nausea • Vomiting • Excess oral secretions	• Dependence • Withdrawal • Respiratory depression • Urinary frequency

Side Effect Management	**Neurological:** Floating sensation, hallucinations, vivid dreams or illusions, delirium ▪ Benzodiazepines may reduce these effects **Glycopyrrolate:** Can be used to mitigate excessive secretory processes

Analgesic Dosing

INDICATION	DOSING	PEARLS
Pain Management	• Continuous IV Infusion: **0.5 - 3 mcg/kg/minute**	• Analgesia outlasts sedative effects • Given through central or peripheral line

Nursing Implications	▪ 2018 published guidelines have recommended the more frequent use of IV ketamine in select patients ▪ Shown to reduce opioid requirements in inpatient perioperative pain management ▪ CANNOT use Sigma pumps on basic mode to infuse ketamine and requires dual sign off in EPIC by nursing ▪ Restricted to Intermediate Units: 9A, 9D ▪ Non-opioid, schedule III medication with dissociative sedation and analgesic properties ▪ Monitoring is important with ketamine infusions ▪ Every 2 Hours: Vital signs, pulse oximetry, pain intensity ▪ Psychotomimetic Effects: Hallucinations, vivid dreams, dysphoria should be reported to physician ▪ **Activate Rapid Response (and/or code) if needed: Extension 24000**

References

1. Kosharskyy B, et al. *Pain Physician*. 2013;16(3):231-249.
2. US Food and Drug Administration. 2014. KETALAR-ketamine hydrochloride injection; 2013.
3. Ahern T, et al. *Am J Emerg Med*. 2015;33(2):197-201
4. Vadivelu N, et al. *J Anaesthesiol Clin Pharmacol*. 2016;32(3):298-306
5. Bell RF, et al. *Acta Anaesthesiol Scand*. 2005;49(10):1405-28
6. Perumal DK, et al. *J Res Pharm Pract*. 2015;4(2):89-93
7. Motov S, et al. *Am J Emerg Med*. 2019;37(2):220-227
8. Elia N, Tramer MR. *Pain*. 2005;113(1-2):61-70
9. Schwenk ES, et al. *Reg Anesth Pain Med*. 2018;43(5):456-466
10. Cohen SP, et al. *Reg Anesth Pain Med*. 2018;43(5):521-546

July 2021

Pain Medicine - 6 Sphenopalatine Ganglion Block with Lidocaine Relieves Lower Extremity Complex Regional Pain Syndrome

Danielle Levin[1], Martin Acquadro[1], Olusola Ackenroye[1], Joseph James[1], Bishoy Michaels[1], Frederic Gerges[1]

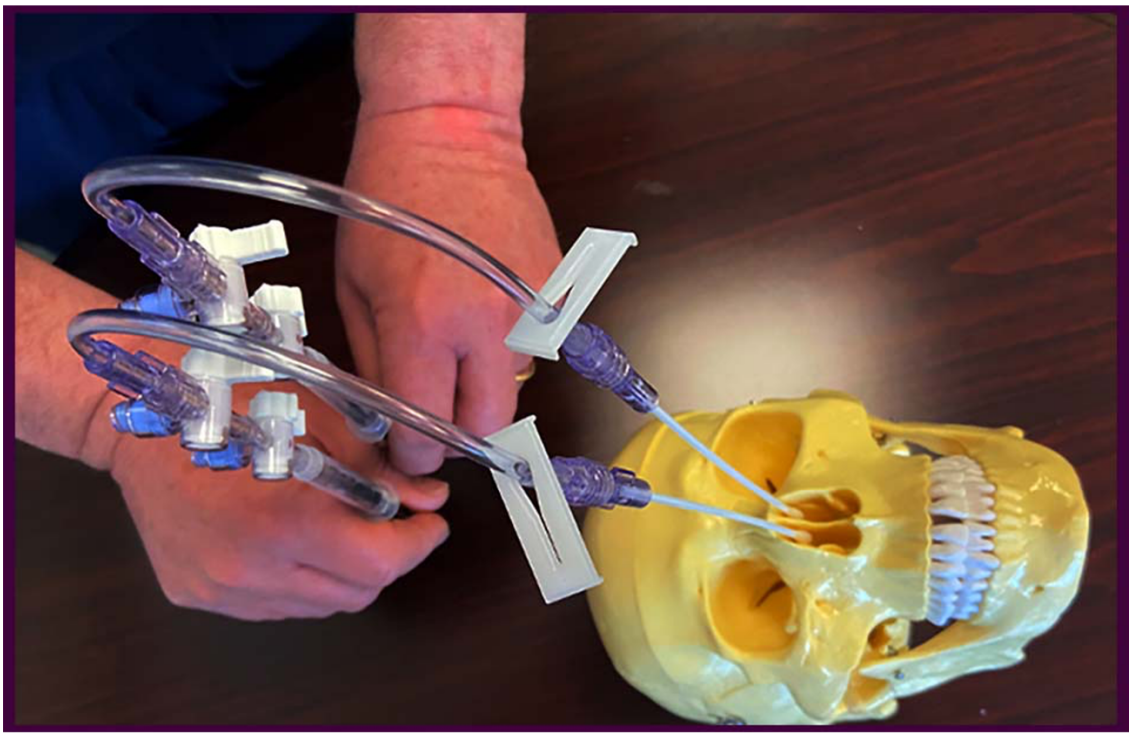
[1]St. Elizabeth's Medical Center, Boston, MA

Introduction: Complex regional pain syndrome is a debilitating, painful disorder that is often difficult to treat. Various pharmacological medications, spinal blocks, intravenous lidocaine infusions, physical therapy, and spinal cord stimulators have been utilized with not guaranteed effectiveness. Currently, no gold standard therapy exists. We report the first case of a transnasal sphenopalatine ganglion block with lidocaine drops that provided 100% pain relief for a patient suffering from lower extremity complex regional pain syndrome. The patient provided verbal and written informed consent for publication of his case.

Methods: A 48-year-old male suffered a work related accident 10 years ago that was followed by a right ankle reconstruction surgery. Since then, he has been experiencing edema, skin color changes, and allodynia in his right foot. He described the pain as moderate to often severe, causing functional limitation in his life with significant interference with his ability to do activities of daily life including household chores, yard work, and shopping, as well as affecting his sleep and mood. Through the years, the Pt has been managed with a multidisciplinary regimen consisting of spinal blocks, physical therapy, spinal cord stimulator, buprenorphine transdermal patches, gabapentin, oxcarbazepine, and monthly IV lidocaine infusions. Unfortunately, with this regimen, the Pt was never pain free. After the IV lidocaine infusions, the Pt would report partial pain relief, which would last at most 2.5 weeks. Even though the IV lidocaine infusions were providing some relief, the Pt was unable to continue receiving these infusions secondary to insurance difficulties. Considering that transnasal sphenopalatine ganglion block has minimal to no side effects and has been previously reported to be effective in relieving multiple painful conditions, we offered this treatment as an option for our Pt. At the start of the appointment, the Pt reported a tingling/sensitive area underneath the medial malleolus of the right foot and radiating to the sole of the foot. He rated the pain as 5/10. For the procedure, the Pt was in supine position with his chin-up Hemodynamic monitors consisting of non-invasive blood pressure and pulse oximetry were applied and the Pt remained hemodynamically stable throughout the procedure. Long hollow cotton-tip applicators, dipped into lidocaine ointment USP, 5%, were placed atraumatically into both nasal sinuses. They were advanced until gentle resistance was met at the back of the nasopharynx. Lidocaine 4% was dripped drop by drop through the hollow cotton-tip applicators into each nostril until the Pt felt the medication in the back of the throat. The cotton-tip applicators were left place in for a total of 15 min, and then the Pt was asked to sit up. After the first 15 min, the Pt pain decreased to 4/10. The procedure was a repeated a 2nd time. When the Pt sat up, his pain was entirely gone. He kept moving his right ankle around in disbelief, but the pain was not there. The Pt was followed up one month later. He reported that for the first time since his initial injury, he was 100% pain free for 5 days post the sphenopalatine ganglion block treatment. The Pt's right foot pain started to gradually return after the 5 days, but he felt that the pain that returned was significantly less than his original pain. When asked whether he had any other effects from the treatment, the Pt reported that since the sphenopalatine ganglion block treatment, his arthritic knee pain also significantly improved.

Conclusion: The sphenopalatine ganglion block is a safe, inexpensive, and well-tolerated treatment for various painful conditions. This case report demonstrates that the sphenopalatine ganglion block with lidocaine may be a new effective analgesic option for those suffering from lower extremity complex regional pain syndrome.


Lidocaine Ointment USP, 5%



Patient Safety

Patient Safety - 1 Suspected Venous Air Embolism in a Left Frontal Craniotomy

Kathryn M Black[1], Kamilla Esfahani[1]

[1]University of Virginia, Charlottesville, VA

Introduction: A venous air embolism (VAE) can be a potentially catastrophic intraoperative complication and lead to rapid and total cardiovascular collapse (1). Certain surgeries, such as a sitting craniotomy are well known to be high risk, however VAEs can occur in many other types of cases and procedures (2). Monitoring modalities are limited and may only detect VAE's when clinically significant changes have already occurred. Therefore, knowledge of high risk scenarios for VAE's, quick recognition, and rapid and coordinated management in the OR are essential for patient survival (1).

Methods: A 78 year-old otherwise healthy female presented for a left frontal craniotomy for a large left sided meningioma. She was induced with lidocaine, propofol, and rocuronium and her airway was secured with an endotracheal tube. Standard monitors and an arterial line were used for monitoring. She was placed in lawn-chair positioning. She was maintained on less than 0.5% sevoflurane and lidocaine at 2mg/min, remifentanil at 0.7mcg/kg/min, and propofol at 50mcg/kg/min. Her ventilator settings were titrated to an end tidal carbon dioxide in the low to mid 30s. During scalp dissection, she settled into a stable plane of anesthesia with mean arterial pressures in the high 60s and 100% oxygen saturations on 50% fractional inspired oxygen. As the surgeons began drilling through the skull, her end tidal dropped acutely from 35 to 22 mmHg. There were no changes in her breathing pattern, peak airway pressures, EKG, or blood pressure. We immediately suspected a VAE and notified the surgical team, who flooded the field with saline. We lowered the head of the bed and switched to 100% oxygen as the surgeons looked for a vein they had transected when opening the skull. We called for additional help and started a norepinephrine infusion. We paused the remifentanil infusion, decreased the sevoflurane, and administered midazolam. We drew an arterial blood gas that showed a PaCO2 of 40.8mmHg, 10mmHg higher compared to the end tidal, indicative of a large A-a gradient possibly due to VAE or pulmonary embolism (PE). After a few minutes, her end tidal carbon dioxide steadily rose back to the mid 30s. With the norepinephrine infusion at 4 mcg/min, she maintained her blood pressures in a normal range and we agreed to proceed with surgery with her head still in the lower position. She remained stable throughout the remainder of the case with minimal blood loss, but had ongoing vasopressor requirements. We took her to the intensive care unit intubated, sedated, and on 2 mcg/min of norepinephrine. Bedside transthoracic echocardiogram did not show right ventricular strain. In the coming days, she was found to have small acute segmental and subsegmental PEs in the right lung base. She was weaned off of pressors, extubated, and sent home soon after.

Conclusion: In this case, we noticed changes in end tidal CO2 and immediately intervened to stop the entrainment of air by removing the interface between air and the venous circulation (flooding the field) and decreasing the pressure gradient between the two (lowering the head of the bed). End tidal carbon dioxide may only detect a venous air embolism after some mild physiologic changes have occurred and blood pressure and EKG changes may only occur after cardiovascular collapse (1). These interventions likely stopped the rapid entrainment of air. We decreased her anesthesia and supported the right heart with norepinephrine as it pushed the air through the right ventricle and into the pulmonary circulation until it was eventually absorbed. Had our interventions not worked and she became more unstable with a potential airlock phenomenon, we were prepared to place her in left side down positioning, apply pressure to the ipsilateral jugular vein to block further air entrapment, or perform chest compressions to break up a potential air bubble and maintain coronary perfusion. This case outlines a very subtle change (drop in end tidal CO2) that if not recognized and intervened on immediately, would have resulted in significant morbidity. While a PE was maintained in the differential throughout the case, this did not affect or delay our management of a potential VAE. Additionally, prompt coordinated action from the entire operating room team and recruitment of additional help was essential during a suspected VAE.

References: 1. Diagnosis and treatment of vascular air embolism. Anesthesiology. Vol. 106. 164-177. 2007. 2. Patient positioning in neurosurgery, principles, and complications. Acta marisiensis. Vol. 66. 9-14. 2020. 3. Vascular air embolism: a silent hazard to patient safety. Journal of Critical Care. Vol. 42. 255-263. 2017.

Patient Safety - 2 Anesthetic Management of a Challenging Case of Klippel Trenaunay Syndrome

Ahmed A Abouarab[1], Indranil Chakraborty[2]

[1]UAMS, Little Rock, AR, [2]University of Arkansas for Medical Sciences, Little Rock, AR

Introduction: Klippel-Trenaunay Syndrome (KTS) is a rare (3/100,000) congenital disorder first described by Klippel and Trenaunay in France in 1900. [1] Diagnoses is made based on the presence of 1- Port-wine vascular lesions, 2- lateral venous malformation and/or dilatation, and 3- underlying soft tissue and/or extremity bone hypertrophy. These features may or may not be accompanied by lymphatic malformations. [2] Given the variable distribution and severity of lesions, anesthetic management of these patients can be challenging, especially for relatively major procedures such as laparoscopic bowel resections. The presence of soft tissue hypertrophy and unrecognized pharyngeal and airway vascular malformations can lead to an increased risk of bleeding and difficult intubation. The presence of lumbar vascular lesions can also preclude utilization of neuraxial anesthesia due to increased risk of hematoma formation from abnormal vessels in spinal canal. Capillary malformations and venous varicosities may also complicate securing an intravenous access. [3]

Methods: Case Report We report a KTS case in a 29-year-old female (60 kg, BMI 20) with past medical history of Iron deficiency anemia and sinus bradycardia. Computed tomographic angiography showed a duplicated IVC and prominent venous tributaries to the ileocolic pedicle. She presented for laparoscopic (possible open) right hemicolectomy due to multiple partial small bowel obstructions at the terminal ileum (TI). Initially, she underwent colonoscopy and esophagogastroduodenoscopy to rule out Crohn's disease. The scope was unable to be traversed past the terminal ileum despite multiple attempts. Pathology report showed chronic mildly active colitis at the ileo-cecal valve and inactive colitis in the ascending colon and descending colon. According to gastroenterology team, her symptoms were attributed to the KTS over Crohn's disease. MR enterography also showed wall thickening involving the cecum and TI with prominence of the mesenteric vessels including a dilated draining vein that could represent Crohn's or related to KTS. The KTS diagnosis was made earlier by the hematology team who recommended to defer surgery to rule out risk for coagulopathy and treat Iron deficiency anemia. The patient reported that slow venous GI bleeds were the reason for her persistent anemia and that she was started on Sirolimus to decrease the frequency of bleeding episodes and control her vascular malformations. Anesthetic Management Preoperatively, careful airway assessment was conducted, Mallampati class II and no soft tissue hypertrophy or visible vascular structures were noticed. Multiple peripheral IV insertion attempts were required due to fragile peripheral veins. The patient was typed and cross matched for blood products. Preoperative labs were within normal limits. Intraoperatively, induction was performed using fentanyl 100 mg, lidocaine 2% 100 mg, propofol 2 mg/kg, and rocuronium 50 mg. Careful direct laryngoscopy was performed using a mac 3 blade followed by insertion of a 7mm cuffed endotracheal tube. Anesthesia was maintained with oxygen and sevoflurane. The case was nearly uneventful except for a few episodes of hypotension responding well to phenylephrine boluses. The procedure concluded as planned entirely laparoscopic without excessive unexpected hemorrhage. After the surgery team closed the skin, local bilateral nerve block using 30 ml of bupivacaine 0.25% was performed with no complications and routine recovery and inpatient management was implemented. The patient was discharged on postoperative day 3 with no complications, pain free and with normal labs.

Conclusion: In this experience, we believe that multidisciplinary management and aggressive preoperative assessment and preparation were key factors for safety and success. Even in the presence of aberrant intraperitoneal and pericolic vascular anatomy, relatively routine anesthetic management was possible with no excessive bleeding or other complications.

References: [[1] Klippel Trenaunay syndrome. E J Indian Soc Teledermatol 2008;2(4):7–14. [2] Vascular malformations. Part II: associated syndromes. J Am Acad Dermatol. 2007 Apr;56(4):541-64. [3] Anesthetic challenges in a case of Klippel-Trenaunay Syndrome with severe anaemia. J Clin Anesth. 2019 Mar; 53:27-28. [4] An anesthetic management of a patient with Klippel-Trenaunay syndrome. Korean J Anesthesiol. 2012;63(1):90-91. [5] Anesthesia for surgery related to Klippel-Trenaunay syndrome: a review of 136 anesthetics. Anesth Analg. 2011 Jul;113(1):98-102.

Patient Safety - 3 Late Onset Malignant Hyperthermia

Riju Dasgupta[1], Ebony Hilton[2], Kamilla Esfahani[2]

[1]University of Virginia Health System, Charlottesville, VA, [2]University of Virginia, Charlottesville, VA

Introduction: Most anesthesiologists have not seen a case of malignant hyperthermia (MH). However, 1 in every 2000 patients harbors a genetic variant that makes them susceptible to MH. This autosomal dominant disease manifests itself 1 in every 10,000 to 200,000 anesthetics¬π. On average, patients are exposed to three anesthetics before developing fulminant MH symptoms. This is partly explained by the diseases' incomplete penetrance and variable expressivity across the patient population¬≤. We present two MH cases at our institution, four months apart, and the challenges of clinically diagnosing and managing variable penetrance MH.

Methods: Case 1: 52 yo woman with past medical history of ESRD, hypertension, morbid obesity, cerebrovascular accident, and COVID-19 infection four months prior presented for robotic kidney transplant. Orotracheal intubation, placement of radial arterial line, and additional peripheral IV were accomplished with ease, and the patient was maintained under anesthesia with sevoflurane. Insufflation of the peritoneum proceeded without complications. Four hours into the case, end-tidal CO2 gradually began to rise to a level in the mid 50's, despite multiple interventions, including hyperventilation and reducing insufflation. Over the next 30 minutes, it suddenly climbed to 88 mmHg. Similarly, the patient's temperature steadily climbed, until it reached a maximum of 39.6 degrees Celsius. MH was suspected at this time. The anesthetic was converted to a TIVA. Inhalational anesthetics and the old circuit were removed from the ventilator, and replaced with charcoal filters. Treatment of MH was initiated with a 2.5 mg/kg bolus of dantrolene. Patient had severe metabolic acidosis, with pH 7.08, and hyperkalemia of 7.8 mmol/L. The groin and bilateral axilla were packed with ice. Temperature and ETCO2 gradually improved with dantrolene treatment. Hyperkalemia persisted despite multiple rounds of sodium bicarbonate, insulin, dextrose, and furosemide. Decision was made to emergently initiate intraoperative dialysis through the patients existing HD line. A second central venous catheter was placed for ongoing resuscitation. Patient remained hemodynamically stable with the help of six grams of calcium chloride and eventually was transported to the ICU. She was extubated on POD #1 and discharged on POD #12 with improved renal function, producing her own urine and off dialysis. Case 2: 67 yo man with past medical history of asthma, CAD, and numerous past uncomplicated anesthetics presented for robotic assisted prostatectomy. Orotracheal intubation, placement of radial arterial line, and addition peripheral venous access were established at the beginning of the case. Sevoflurane was used to maintain general anesthesia. After several hours into the case, end-tidal CO2 had risen to a peak of 88 with temperature increasing to a peak of 38.8 degrees Celsius. MH was suspected. Inhalational anesthetics and the old circuit were removed from the ventilator, and replaced with charcoal filters, and TIVA initiated. EKG showed T wave inversions, and the patient became progressively more hypotensive. Blood gas was notable for lactate of 7.9 mmol/L and peak potassium of 4.8 mmol/L. Calcium chloride and vasopressin were given, closely followed by full MH treatment with 2.5 mg/kg bolus of dantrolene. End-tidal CO2 and temperature gradually improved. Decision was made to abort the surgery and transfer the patient to the ICU for ongoing management. He was extubated on POD #1 and discharged on POD #3

Conclusion: These two cases demonstrate the variable presentation of malignant hyperthermia and examples of management of the disease once diagnosis is suspected. On further questioning, the first patient had a history of a similar incident with a prior anesthetic but failed to disclose this, despite direct questioning during the preoperative interview. Conversely, the second patient had three anesthetics with sevoflurane exposure Both of these cases highlight the management of suspected MH. Once initial suspicion is made, it is crucial to administer dantrolene as 2.5mg/kg bolus and to discontinue all triggering agents. Anesthetic should be converted to a TIVA and the ventilatory circuit should be flushed, and exchanged for one with charcoal filters applied. In addition to dantrolene, treatment should focus on correcting life-threatening hyperkalemia and metabolic acidosis.

References: 1) Rosenberg, H., Pollock, N., Schiemann, A., Bulger, T., & Stowell, K. (2015). Malignant hyperthermia: A Review. Orphanet Journal of Rare Diseases, 10(1). 2) Litman, R. S., Griggs, S. M., Dowling, J. J., & Riazi, S. (2018). Malignant hyperthermia susceptibility and related diseases. Anesthesiology, 128(1), 159–167.

Patient Safety - 4 Knowledge of Line Isolation Monitor and Electrocution Hazard Among Operating Room Staff

Daivyd A Palencia Pacheco[1], Lisa Ross[1], Michael Wong[1]

[1]NYC Health + Hospitals/Harlem, New York, NY

Introduction: Since 1984, line isolation monitors (LIM) have been installed in operating rooms to prevent electrical shock to operating room staff and patients. The LIM constantly scans for potential electrical current connections, and predicts how much current could develop if a second short-circuit were to be added. If this predicted current is unacceptable, the LIM will alarm. In other terms, the alarm signals that a piece of equipment, most likely the last piece of equipment plugged into the wall or tower, has created a 'fault'. If another fault were to be added, it could potentially lead to an electrical shock. [1,2] While education related to the LIM is part of training during anesthesia residency, it is important that all operating room staff are able to recognize the alarm from the LIM and how to correct the situation. We present a case in which the LIM alarmed during an urgent cesarean section, where it became evident that there was a significant gap in knowledge of the role of a LIM among the staff in the room.

Methods: A 23-year-old primiparous patient was admitted to the labor and delivery suite for induction of labor due to post-term pregnancy. After induction of labor, she requested a labor epidural which was inserted without incident. Eventually the patient underwent an urgent cesarean section due to failure to progress. The cesarean section was performed successfully, and a healthy neonate was delivered. During surgical closure of the incision, the LIM, located in the wall behind the anesthesiologist, alarmed. In the room, two obstetricians, the neonatologist, and four nurses did not recognize the alarm, or the purpose of the alarm. The alarm was temporarily silenced by the anesthesiologist to communicate to the staff that there was an electrical 'fault' and they needed to search and identify which piece of equipment was responsible. The staff did not understand the necessity of this search, and could not identify which piece of equipment had been last plugged into an electrical outlet. At this time, the anesthesiologist began to unplug all unnecessary equipment from the electrical outlets in the room. After a period of time the alarm stopped but the exact offending piece of equipment was not identified. Post-operatively the supervising administrator on duty was alerted to the issue in the operating room and the engineering department was notified. The room was put out of service immediately. The engineer on duty responded but was not familiar with the LIM, nor did he know how to interrogate the equipment. The room was kept closed until biomedical services and engineering could inspect all of the equipment in the room as well as the LIM itself. This task was completed and it was determined that all equipment was working properly, the panel was working properly and there was no further risk of electrical shock. The room was put back into service with no further incidences. Afterwards, a poll regarding general knowledge of LIM was taken of faculty anesthesiologists, certified registered nurse anesthetist (CRNA), as well as operating room nursing staff. A total of 51 responses were recorded. Findings from this poll demonstrated that 93% of anesthesiologists, 50% of CRNAs, <10% of operating room nurses in both the main operating room and the labor and delivery operating room knew what the LIM was and/or its purpose.

Conclusion: The line isolation monitor helps to prevent electrical shock in the operating room. While education regarding LIMs are common in anesthesia training, it is important that all operating room staff understand the role of the LIM to avoid potential workplace hazards. This can be done with standardized training in LIMs or by placing information about LIMs and electrical hazards in emergency manuals. In this case, we highlight a lack of knowledge of this monitor in an institution's operating room staff. Further studies into the prevalence of LIM education in operating room staff may help reduce the chance of electrical shock and improve staff and patient safety.

References: Electrocution in the Operating Room. Anesthesiology.1973, Vol 38, p 181-183.
Electrocution during Anaesthesia. Anaesthesia, 1979, Vol 34, p 173-175

Patient Safety - 5 The Importance of the Intensivist in the Management of Conversion Disorder

Logan Hollman[1], Jonathan Bond[2], James D Bacon[2]

[1]University of Kentucky Albert B Chandler Hospital, Lexington, KY, [2]University of Kentucky, Lexington, KY

Introduction: Conversion disorder (CD), i.e. functional neurological disorder, is a complex psychiatric disorder distinguished by sensory, motor, and/or cognitive deficits that are not explained by any recognized neurologic condition. More common in women, CD is believed to be the manifestation of abnormalities in brain structure, physiology, and function as well as psychological underpinnings. Symptoms may be involuntary or voluntary and may or may not be preceded by psychological stressors. Surprisingly, the misdiagnosis rate is reported at only 4%, but coordinated treatment can be challenging. We describe a patient who presented with hemiplegia three times before a diagnosis of CD was made, and propose intensivists orchestrate care for patients with CD in the ICU.

Methods: A 69-year-old woman with generalized anxiety disorder presented with left arm and leg weakness, sensory deficit, and dysarthria despite a negative CT head and CTA head/neck. tPA was administered, the patient complained of headache, and ultimately required intubation for airway protection. Repeat CT head was negative. A chart review revealed two previous admissions with identical presentations. She was extubated, and despite a normal MRI, continued to display left hemiparesis and slurred speech. The diagnosis of conversion disorder was made. With neurology, psychiatry, and narrative medicine following the patient, symptoms improved with only one recurrent episode of symptoms prior to discharge. Undiagnosed CD can put patients at risk for harm as illustrated by our case. Further, the overlap of neurologic and psychiatric specialties in the management of CD can result in neither desiring to take the lead in treatment, and suboptimal care can result. As such, in the ICU the intensivist must be able to diagnose CD and assemble and direct a multidisciplinary team including neurology, psychiatry, and even PT. This collaborative care approach helps the patient better understand that their disorder is 'functional' and 'treatable' and a full recovery is possible. Additionally, appropriate treatment can be started in hospital, and follow up care after discharge established. For the patient, this approach should result in a better understanding CD, fewer subsequent ED visits, and avoidance of needless high-risk treatments.

Conclusion: Not applicable.

Pediatric Anesthesiology

Pediatric Anesthesiology - 1 Anesthetic Considerations for Separation of Conjoined Twins- A Case Report

Claire Naus[1], Henry W Davis[2], Teeda Pinyavat[3], Philipp Houck[3]

[1]Texas Children's Hospital/Baylor College of Medicine, Houston, TX, [2]Memorial Sloan Kettering, New York, NY, [3]Columbia University Medical Center, New York, NY

Introduction: Conjoined twins are quite rare with an approximate incidence of 1:200,000 live births and a female to male ratio of 3:1.[1] [2] Conjoined twins are classified based on where they are joined together; thoracopagus twins are most common, making up 40-45%, followed by omphalopagus (33%), pyopagus (19%), ischiopagus (6%), and craniopagus (2%).[2] [3] The unique and specific anatomy of conjoined twins has significant ramifications for anesthetic management due to concerns about airway management, pharmacological and hemodynamic effects of possible cross-circulation, positioning, and the logistics of running two anesthetics (with two anesthesia machines, two sets of monitors, and two anesthesia teams) in a single operating room.[3]

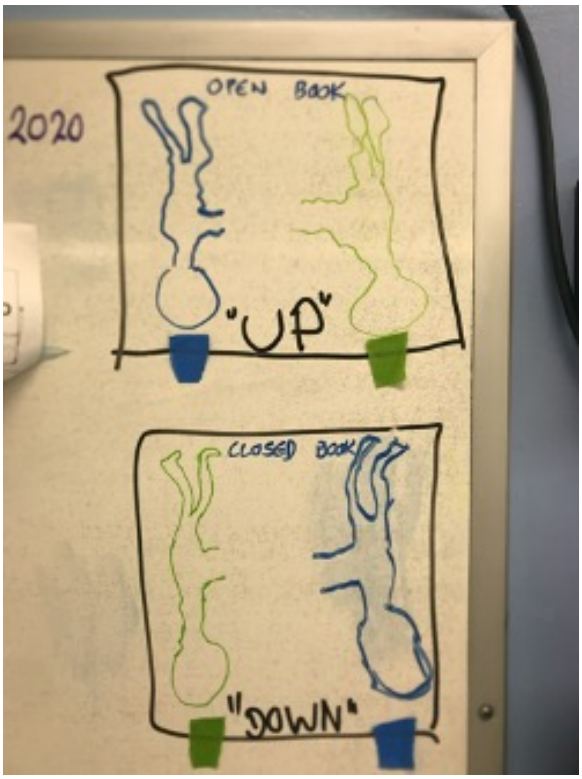
Methods: This set of thoraco-omphalopagus conjoined twins were born via elective caesarean section at an outside hospital at 38 weeks gestation. They were fused from the xiphisternum to the abdomen with a shared band of liver and structurally normal hearts with a shared pericardium. The twins underwent multiple preoperative imaging studies to identify shared anatomy and elucidate the degree of cross-circulation. After 4 weeks of nutritional optimization for failure to thrive, they were brought to the operating room at 4 months of age for placement of tissue expanders in preparation for the eventual separation. Intravenous (IV) access was secured in both twins under nitrous oxide prior to induction. IV atropine was given to one twin with no change in heart rate in the other twin, signifying that the degree of cross-circulation was likely minimal. They were placed on a special, custom-made pillow for optimal positioning ("open book" configuration, see images) and then induced and intubated one at a time. Anesthesia was maintained with sevoflurane and remifentanil. Monitors, medications, equipment, and anesthesia teams for each twin were differentiated by color, which was particularly important for turning the twins halfway through the case to place tissue expanders on the opposite side. At the end of the case, the twins were turned back to their original positions and extubated sequentially.

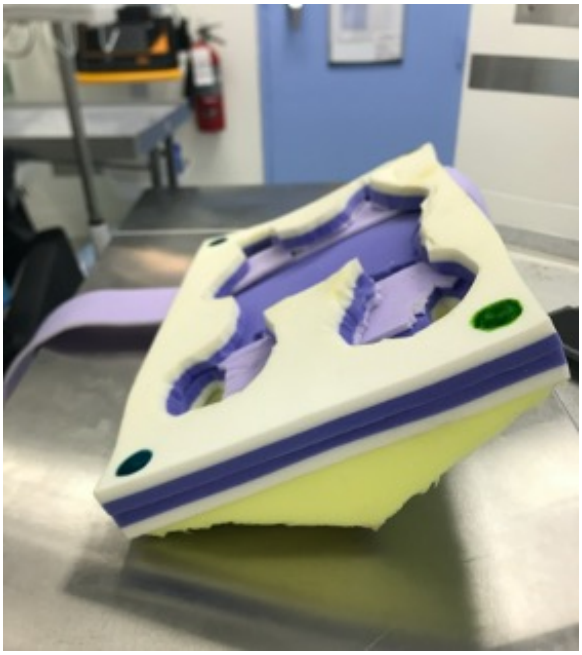
The twins returned to the operating room at 5 months of age for the separation. Pre-induction IV access and intubation were performed in the same manner. Additional IVs, radial arterial lines, and internal jugular central venous catheters were placed for each twin. Anesthesia was maintained with sevoflurane and fentanyl. Multiple surgical teams – general, plastics, transplant/liver, and cardio-thoracic – were involved in the separation. After they were uneventfully separated, the twins were moved to different tables in the same operating room. Both patients received approximately 10cc/kg of packed red blood cells, but neither required vasopressors. Given the large defects and the limited available skin, closures for both twins were completed with a patch. The twins remained intubated at the end of the separation and were transported to the pediatric intensive care unit for postoperative care. One twin remained intubated for 8 days, and the other for 11 days. Their postoperative course was complicated by compartment syndrome, multiple infections requiring the placement of peripherally inserted central catheters and removal of an infected pericardial patch, persistent tachypnea, and swallowing issues.

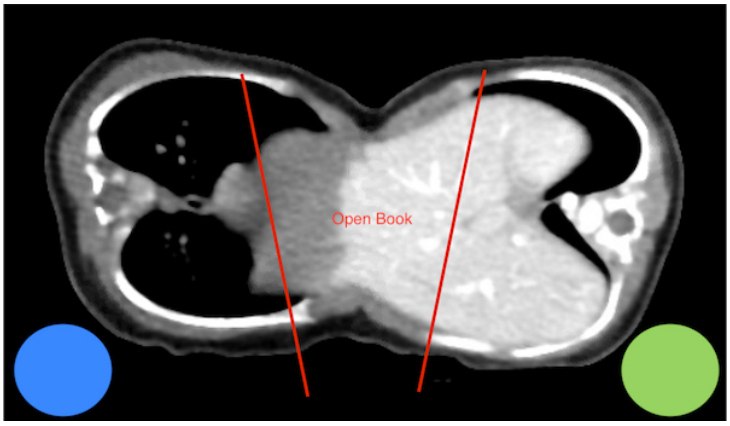
Conclusion: This case demonstrates the complexities involved in providing anesthesia for conjoined twins. Careful planning is necessary, particularly for critical aspects of the case, namely, being prepared to manage two airways simultaneously, determining how giving medication to one twin affects the other, ensuring that lines are not in the surgical field, and addressing the logistics of position changes. Additionally, the seemingly simple step of color coding the lines, monitors, medications, equipment, and personnel not only defined the teams for each twin but helped to keep a confined space organized, optimizing patient care for the placement of tissue expanders and the separation. Lastly, given the multiple postoperative complications and prolonged hospital stays for both twins, this case highlights the ethical issues involved in separating conjoined twins and subjecting otherwise

healthy children to a potentially life-threatening operation.

References: A&A Practice 2018; 11: 71-72. Pediatric Anesthesia 2004; 14: 117-129. A&A Practice 2018: 10: 298-301.







Pediatric Anesthesiology - 2 Respiratory Acidosis With to Failure to Wean from Ventilator in Down's Syndrome Patient Following Adenotonsillectomy

Elisabeth Hughes[1], Carrie Menser[2]

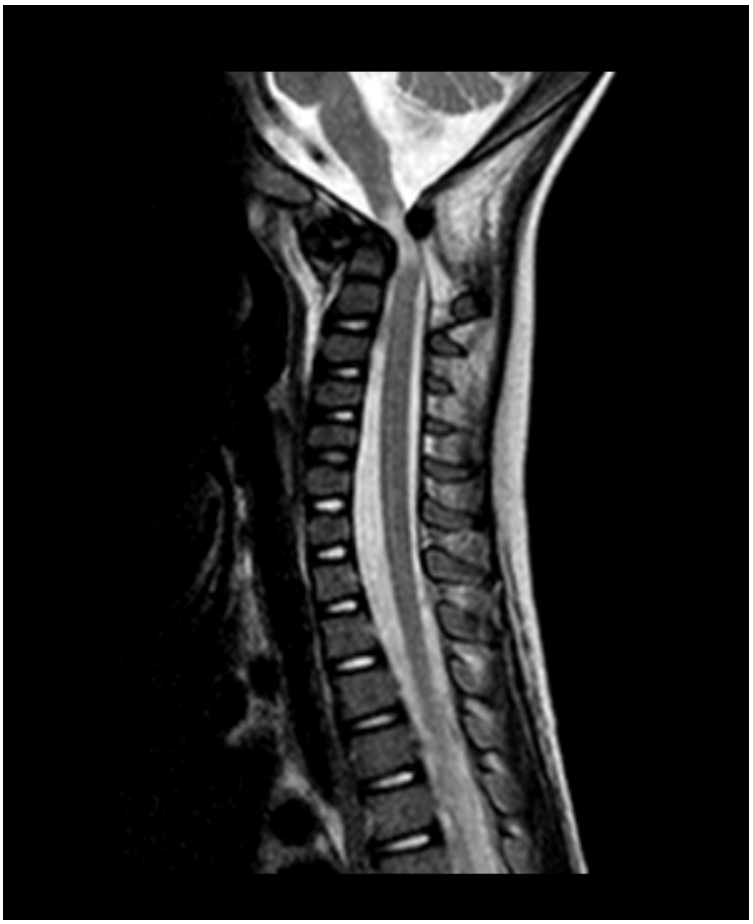
[1]Vanderbilt University Medical Center, Nashville, TN, [2]Vanderbilt University Hospital, Nashville, TN

Introduction: A 2 yo patient with Trisomy 21 presented with somnolence 3 weeks post tonsillectomy and was found, after extensive workup in the ICU, to have C1-2 instability with cord impingement. The purpose of this Case Report is to review and describe the potential neurologic risks associated with surgery in a pediatric patient with Down's Syndrome. We will review the preoperative pediatric guidelines for patients with Down's Syndrome. We will also describe the medical sequelae that can be seen with neurologic complications follow surgery.

Methods: 2yo female with Trisomy 21 presents to our hospital after difficulty with feeding and swallowing as well as being difficult to arouse in the morning from sleep. Of note, she underwent adenotonsillectomy 3 weeks prior, but until this point had been recovering well and had returned to daycare. On arrival to the ED, the patient was somnolent and found to have a venous CO2 of 110. She was intubated in the ED and admitted to the PICU. Over the course of 3 weeks, there were multiple failed attempts to wean and extubate with continued hypercarbic respiratory failure. A pulmonary consult revealed no obvious reason for respiratory failure, neurology consult did not relate the respiratory failure to central hypotonia from Trisomy 21, multiple bedside ENT airway flexible scope exams were relatively normal and an intraoperative DLB was performed which was not revealing for a cause. At ICU week 3, a repeat ENT airway evaluation in the OR was combined with a MRI of the brain and c-spine. Airway evaluation was again normal. Her C-spine MRI revealed AO subluxation of C1-C2 causing severe stenosis with cord compression and associated cord signal abnormality. Two days later the patient underwent a posterior spinal fusion of C1-C2 and halo placement. Despite cervical spine stabilization, she required tracheostomy and gastrostomy tube placement 1 month later due to continued respiratory support and aspiration risk. 6 weeks following these procedures, the patient left the ICU for rehabilitation with a home ventilator. Our case highlights the importance of understanding the neurologic risks and presentations associated with patients with Trisomy 21 undergoing surgery, otolaryngology surgery specifically, and we intend to review these implications and risks with this case presentation.

Conclusion: Children with Down's Syndrome are not likely to be able to self report neurologic symptoms preoperatively and a neurologic exam may be difficult to obtain given developmental delays as well as relative hypotonia associated with the syndrome. The AAP no longer recommends routine c-spine films prior to surgery and practices surrounding c-spine stabilization during intubation or surgery are variable. AO instability is a widely known occurrence in Trisomy 21 and respiratory distress or weakness should signal investigation of AO stability. In our case, a thorough workup for respiratory insufficiency and airway abnormalities was negative. Clear causation of symptoms was not identified until the MRI of the cervical spine was obtained. It is important to keep AO instability and cord compression in the differential for respiratory failure in a patient with Down's Syndrome.

References: AAP updates guidance on caring for children with Down syndrome. Alyson Sulaski Wyckoff, AAP News July 2011, E110725-3; DOI: https://doi.org/10.1542/aapnews.20110725-3 Is it Safe to Perform Adenotonsillectomy in Children with Down's Syndrome? Yumusakhuylu et al. Eur Arch Otorhinolaryngol (2016) 273: 2819-2823. Neurologic Sequelae Secondary to Atlantoaxial Instability in Down Syndrome. Implications in Otolaryngologic Surgery. Harley et al. Arch Otolaryngol Head Neck Surg. 1994; 120: 159-165.



Pediatric Anesthesiology - 3

Cor Triatriatum Sinister in a Pediatric Patient

Tamra N Kelly[1], David Vener[1]

[1]Baylor College of Medicine, Texas Children's Hospital, Houston, TX

Introduction: Cor triatriatum sinister (CTS) is a rare congenital anomaly accounting for 0.1-0.5% of all congenital heart disease(1) where the left atrium is divided into two chambers by a membrane impairing blood flow to the left ventricle.(2,3) CTS can be difficult to diagnose as the patients present with respiratory symptoms or reflux progressing to cardiogenic shock; if left untreated mortality is 75%.(4,3) We describe a case of a 5 month old female who presented for urgent repair of undiagnosed CTS.

Methods: Preoperative: The patient presented to the emergency room with a history of reflux in respiratory distress. Echocardiogram showed CTS with severe obstruction 2.8mm orifice (Image 1), severe pulmonary hypertension, mildly depressed right ventricle (RV) function, and normal left ventricle (LV) function. Induction: The patient was intubated with a central line and PIV in the cardiac intensive care unit. Fentanyl 2 mcg/kg, midazolam 0.1 mg/kg and vecuronium 0.1mg/kg were given prior to transport to the operating room (OR). Intraoperative: On arrival to the OR, the blood pressure and heart rate dropped, and the ECG showed ST depression. Resuscitation was started with multiple epinephrine, CaCl, and bicarbonate boluses. Epinephrine and vasopressin infusions were initiated. An arterial line was placed urgently in left femoral artery. The patient recovered after 20 minutes of resuscitative efforts. (See Figure 1) Incision was made 8 minutes after anesthesia ready. Nitric oxide (iNO) was initiated after the cross clamp was removed. The patient came off bypass on dexmedetomidine, milrinone, and epinephrine. The pulmonary artery pressure was measured in the field at 51/15(30) mmHg. The epinephrine infusion was weaned prior to procedure end. Postoperative: The patient was transferred to CVICU intubated on dexmedetomidine and milrinone infusions and iNO. She was extubated on postoperative day 2 and remained hemodynamically stable. Discharge echocardiogram showed non-obstructive residual tissue in the left atrium and no evidence of elevated RV pressures.

Conclusion: Discussion The anesthesia provider must be acutely aware of the potential hemodynamic instability of the patient with CTS. Clinical instability is dependent on the size of the hole in the membrane.(1)If the orifice is less than 4 mm there is a high risk for cardiogenic shock (1) and instability perioperatively due to pulmonary hypertension and underfilling of the LV. The anesthesia provider must be prepared with vasoactive infusions and emergency drugs pre-bypass. A multidisciplinary meeting prior to bringing the patient to the OR is necessary to discuss the potential urgency to go on bypass. Post bypass, inhaled nitric oxide is indicated to treat pulmonary hypertension caused by CTS. Conclusion This case report describes the hemodynamic instability of a patient with CTS. A thorough understanding of the anatomy and physiology of this rare congenital anomaly is necessary for the anesthesia provider to anticipate potential complications and be prepared to manage accordingly.

References: 1. “Wheezing as a sign of cor triatriatum sinister culminating in multiple organ failure” 56:92-95, 2014 2. ”Surgical Repair of Cor Triatriatum Sinister: The Mayo Clinic 50-Year Experience”, 97:1659-1663, 2014 3. “Cor Triatriatum Sinistrum A rare disease with a common presentation” 21:324-326, 2021 4. “A Rare Cause of Shock in an Infant Cor Triatriatum” 00:00-00, 2017

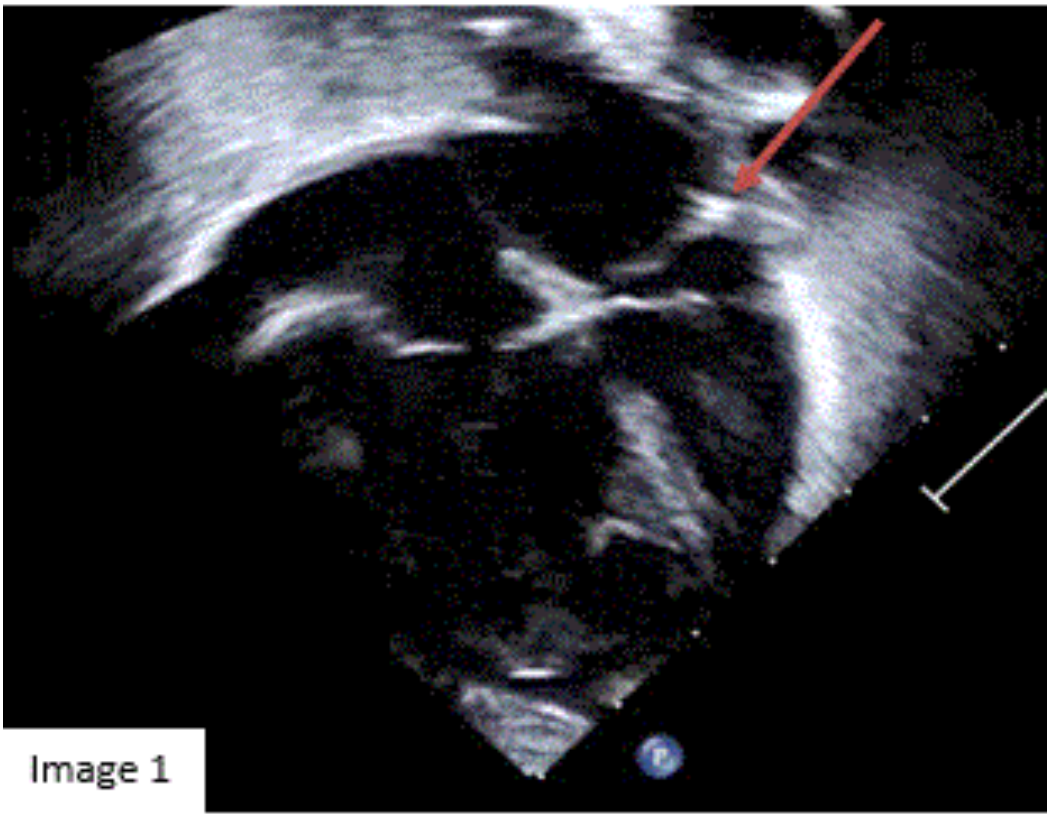


Image 1

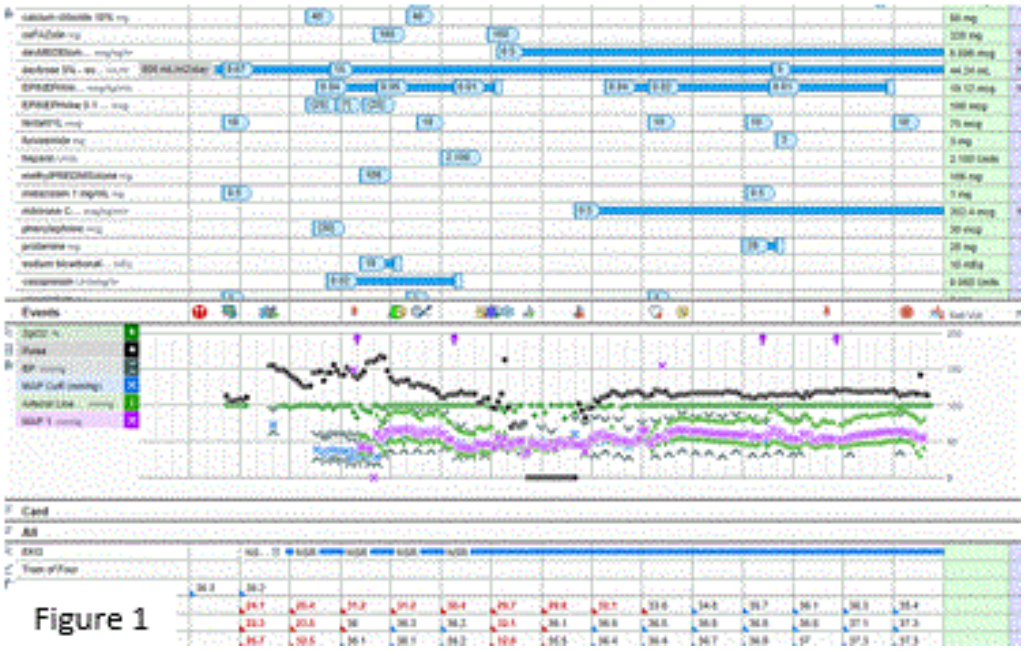


Figure 1

Pediatric Anesthesiology - 4

Endovascular fenestration of intimal flap in an adolescent patient with Type A aortic dissection

Jingjing Sparrow[1], David Vener[1]

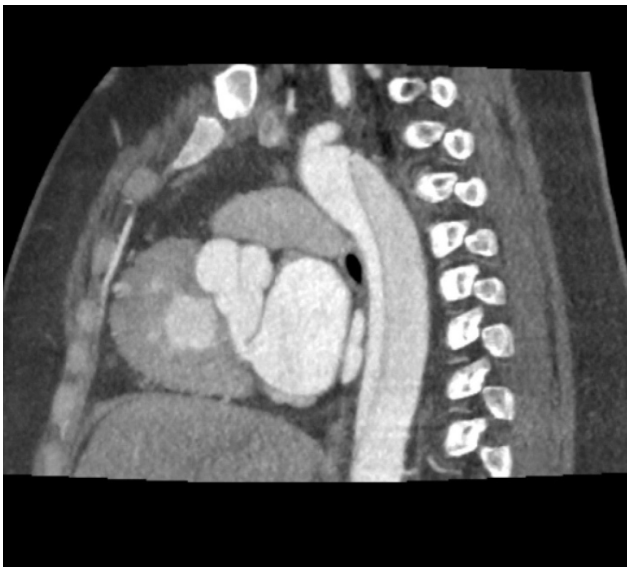
[1]Baylor College of Medicine, Texas Children's Hospital, Houston, TX

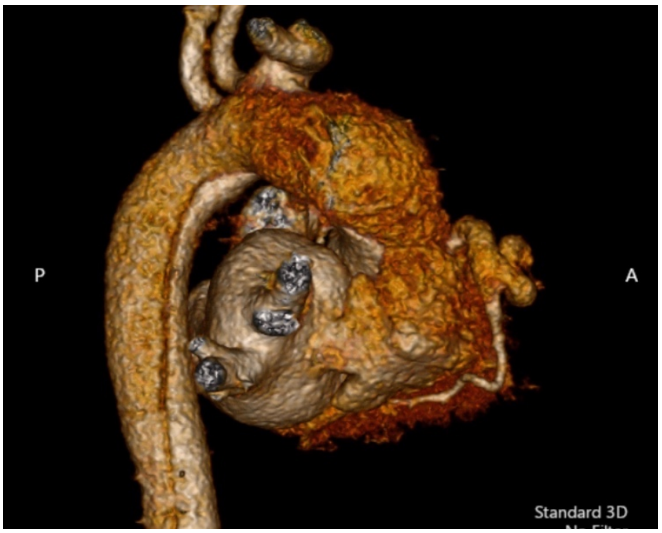
Introduction: Aortic dissection is a rare but life-threatening disease in which an intimal tear results in dissection between the intima and media layers of the aorta, creating a false lumen for blood entry. Acute Type A dissection is often fatal without emergent surgical repair. Chronic Type A dissection (CTAD) may go unnoticed initially. It is diagnosed more than 14 days since symptom onset or found incidentally. CTAD can be managed by surgical repair or endovascular techniques.1¬†We describe a case of a 16 year-old female undergoing laser fenestration of the intimal flap for CTAD with distal malperfusion.

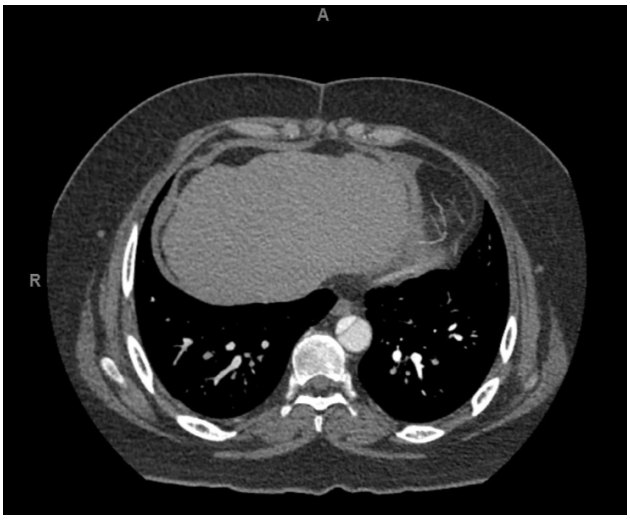
Methods: Pertinent medical history: Congenital heart defects: Pulmonary atresia with intact ventricular septum (PA/IVS), RV dependent coronaries with three RV-RCA fistulas. S/P single ventricle palliation: BT shunt (2003), bidirectional Glenn (2003), and non-fenestrated Fontan (2007). Asymptomatic from RV-RCA fistulas until 2019 when a murmur and exercise intolerance developed. Cath closure of RV-RCA fistulas complicated by RCA dissection, s/p RCA angioplasty with restored RCA flow (2019). Planned for follow-up CT in three months. Claudication and increased fatigue started about two weeks after cath. Follow-up CT: Type A aortic dissection from within the mid RCA extending distally to just below the left renal artery. All head and neck vessels and main abdominal aortic branches arise from the true lumen. Left renal artery likely arises from the false lumen. Echo: normal LV function (single ventricle). Plan for endovascular fenestration of intimal flap. A standard IV induction was performed with midazolam, fentanyl, lidocaine, propofol and vecuronium. Esmolol 1mg/kg was administered prior to intubation. A right radial arterial line and two 18G PIVs were placed thereafter. Esmolol and nicardipine infusions were used to control heart rate (HR) and blood pressure (BP). Hypertension and tachycardia due to procedural stimulation were treated with fentanyl and propofol. The team of cardiologist and vascular surgeon advanced catheters from the left and right femoral arteries into the false and true lumens respectively. The false lumen pressure was about 30-40 mmHg higher than the true lumen. Laser fenestration of intimal flap was performed in the distal descending aorta, followed by serial balloon dilations. Post-intervention pressures in the false and true lumens were equalized. The patient was extubated in the cath lab and transported to CVICU in stable condition. Esmolol and nicardipine infusions were weaned off overnight. She reported resolution of claudication on POD 1 and was discharged home with metoprolol.

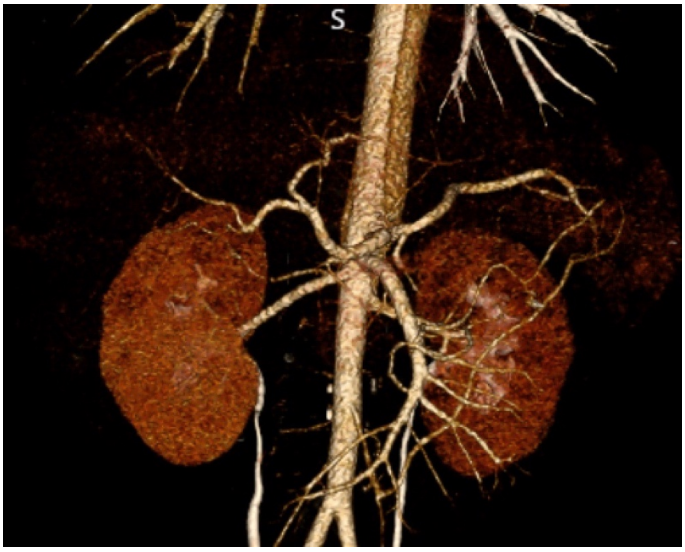
Conclusion: Patients with stable CTAD may benefit from endovascular fenestration/stenting of the aorta to improve perfusion to affected organs, so that open aortic repair can take place when patient is optimized.2 Knowledge of the disease pathophysiology, procedural risks, and the patient's unique medical history is essential for safe and effective anesthetic management. In this case, the patient's HR was targeted to 60 bpm and SBP to 100 mmHg to reduce the shearing force from blood flow. Blood products, fluids, large-bore PIVs, and vasoactive infusions were available in case of bleeding or the rare need for conversion to open surgery. With stable hemodynamics, early extubation is favorable given the patient's Fontan physiology. Additionally, communication with the cardiologist and vascular surgeon during the procedure is crucial in identifying changes in clinical status and assessing effectiveness of intervention.

References: 1. Hynes, C. F., Greenberg, M. D., Sarin, S., & Trachiotis, G. D. (2016). Chronic Type A Aortic Dissection: Two Cases and a Review of Current Management Strategies. Aorta (Stamford, Conn.), 4(1), 16–21. 2. Yang, B., Rosati, C. M., Norton, E. L., Kim, K. M., Khaja, M. S., Dasika, N., Wu, X., Hornsby, W. E., Patel, H. J., Deeb, G. M., & Williams, D. M. (2018). Endovascular Fenestration/Stenting First Followed by Delayed Open Aortic Repair for Acute Type A Aortic Dissection With Malperfusion Syndrome. Circulation (New York, N.Y.), 138(19), 2091–2103.




P
A
Standard 3D


A
R


S

Pediatric Anesthesiology - 5 Foreign Body Detected During Magnetic Resonance Imaging: A Case Report

Ramanjot S Kang[1], Alexa Christophides[1], Shayla Mena[1], Vahe Tateosian[1]

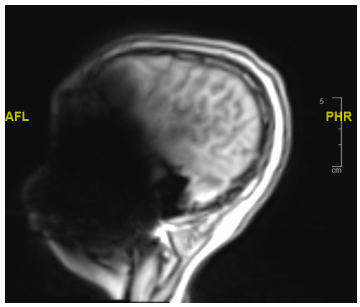
[1]Stony Brook University, Stony Brook, NY

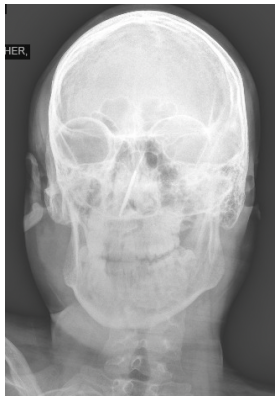
Introduction: There has been a considerable increase in the number of procedures performed outside of the operating room requiring sedation or anesthesia. There are special considerations for non-operating room anesthesia (NORA) given the unique challenges these cases pose to anesthesiologists (1). Diagnostic imaging studies performed for pediatric and special needs patients are often performed under general anesthesia and thus require anesthesiologists to be aware of not only the considerations for these patients, but also those of NORA. Magnetic resonance imaging (MRI) is one of the most common diagnostic imaging studies performed for these patients under general anesthesia because the test requires that patients remain stationary for a prolonged period of time. Contraindications for MRI include ferromagnetic foreign bodies, which can be attracted or accelerated by the static magnetic field of the MRI scanner (2), implants, and pacemakers. For these patients, it is particularly important to rule out foreign bodies. It is common for these patients to insert small objects in their mouths, external auditory canal (EAC), or nares - all of which may go unnoticed prior to imaging despite a proper physical exam (3, 4). Moreover, since these patients routinely require general anesthesia for imaging, the early warning signs that typically indicate that a ferromagnetic foreign body is present may be concealed, preventing early termination of the MRI. We present the case of a pediatric patient undergoing an MRI that was found to have an unknown foreign body.

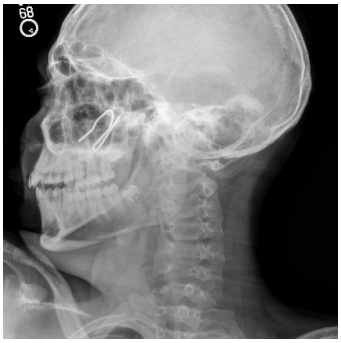
Methods: A 13 year old male with a history of autism and epilepsy presented for an MRI of the brain. Pre-admission testing and MRI safety assessment were completed per institutional protocol. The patient's parents reported that he did not have any implants, devices, or objects that would be hazardous or interfere with the study. Prior to induction, a pulse oximeter was applied. The patient underwent a mask induction with Sevoflurane. Eyes were protected and a 20g peripheral IV was placed in the left hand. A non-invasive blood pressure cuff was applied to the left lower extremity and EKG monitoring was established. A laryngeal mask airway size 3 was placed in the patient's mouth and secured. Anesthesia was maintained with Sevoflurane. The MRI was started and a foreign body was noticed on initial scout images. The study was immediately stopped. The patient was examined again in order to identify the foreign body. An otoscopic exam was performed but no foreign object was found in the mouth, nares, or EAC. The patient's parents denied the possibility of metal in the head or face; however, did disclose that the patient had been known to insert foreign bodies into his nares. A plain film x-ray was ordered, and a metal object was seen on imaging. Pediatric otolaryngology was consulted and the patient was brought to the pediatric emergency room for further workup. The timing of insertion was unknown and the patient had no symptoms of pain, swelling, or redness. The patient went to the operating room for removal of the foreign body under general anesthesia. A 3 cm bent rusted metal object was seen between the middle turbinate and septum under nasal speculum exam and was removed with a straight grasper. At the end of the procedure, the patient was successfully extubated and transported to PACU.

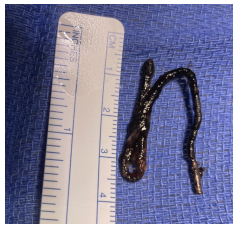
Conclusion: Given the dangers of performing an MRI with a ferromagnetic foreign body, there are several safeguards in place to protect patients undergoing an MRI. However, pediatric and developmentally delayed patients pose a unique challenge. Firstly, they often require general anesthesia for MRI given the need to remain still for the imaging study. This prevents us from identifying some of the early warning signs, such as pain or discomfort, that a ferromagnetic foreign body may be present. Secondly, they are more likely to have a foreign body that may be missed on a routine physical exam. Despite a preoperative physical exam by the anesthesiologist and a questionnaire completed by the patient's parents, the object was not discovered until the MRI exam was initiated. Our case demonstrates the need for additional safeguards to protect pediatric patients undergoing MRI under general anesthesia.

References: (1) v. Paczynski, S., Braun, K., Müller-Forell, W. et al. Fallgruben in der Magnetresonanztomographie. Anaesthesist 56, 797–804 (2007). (2) Ghadimi, M., & Sapra A. Magnetic Resonance Imaging Contraindications. StatPearls. (2021). (3) Metterlein, T., Haubner, F., Knoppke, B. et al. An unexpected ferromagnetic foreign body detected during emergency magnetic resonance imaging: a case report. BMC Research Notes 7, 808 (2014). (4) Kiger, J. R., Brenkert, T. E., & Losek, J. D. (2008). Nasal foreign body removal in children. Pediatric Emergency Care, 24(11), 785–792.









Pediatric Anesthesiology - 6 A Case of Intraoperative Anaphylaxis Due to Factor IX Infusion in a 15-Month-Old Patient with Hemophilia B

Tiffany Chen[1], Stephanie Sutter[1], Jina Sinskey[1]

[1]UCSF, San Francisco, CA

Introduction: Patients with inherited bleeding disorders due to coagulation factor deficiency often receive infusions of plasma-derived recombinant factors. Anesthesiologists should be aware that 1.5-3% of patients develop inhibitors which can cause life-threatening anaphylactic reactions to factor IX even when previously tolerated [1].

Methods: A 15-month-old boy born at 24 weeks with severe hemophilia B presented for elective permanent port placement for frequent infusions. Family history was significant for a twin brother and older half-brother with severe factor IX (FIX) deficiency (<1% FIX). His half-brother had angioedema with FIX infusion. The patient received FIX once for bruising two months prior without issue. Intraoperatively, general anesthesia was induced and a supraglottic airway device was placed. 1,500 units of recombinant FIX were given prior to incision. Within one minute, the end tidal CO2 tracing flattened. He desaturated to 40% with high peak pressures and difficulty ventilating. Pulmonary exam revealed minimal breath sounds and coarse wheezing bilaterally. Inhaled albuterol was given with improved oxygen saturation and diffuse urticaria was seen on >50% of his body. He received intravenous epinephrine, dexamethasone, diphenhydramine, and crystalloid fluids. An epinephrine infusion was started for ongoing hypotension despite multiple bolus doses. Port placement was postponed and the patient was intubated and transported to the pediatric intensive care unit (PICU). In the PICU, he was hemodynamically stable, the epinephrine infusion was weaned off over a few hours, and the patient was extubated. The patient's course was complicated by compartment syndrome in his right upper extremity, after an arterial blood gas draw, requiring urgent fasciotomy and multiple wound explorations. Prior to each procedure, he was given factor VII with close observation for anaphylaxis. FIX inhibitor titers were drawn and he underwent a desensitization protocol. While receiving higher concentrations of factor over shorter infusion duration, the patient developed urticaria despite previous tolerance. Inhibitor titers were redrawn and showed a significant increase from prior to desensitization. Given his increased inhibitor level, he was no longer a candidate for re-desensitization and was restarted on factor VII infusions for bleeding episodes.

Conclusion: Patients can develop anaphylaxis to FIX despite previous administration without issue [2]. If this occurs, prompt consultation with hematology is recommended. If patients are at risk for bleeding, first-line treatment typically involves activated factor VII which can bypass factors VIII and IX and directly activate factor X [3]. Hemophilia occurs in 1 of 5,000 male births [4]. Children with hemophilia have a higher risk of uncontrolled or serious bleeding. Anesthesiologists should be aware of the risk of anaphylactic reactions to coagulation factors and should have a high index of suspicion for this serious complication.

References: 1. Inhibitors in Hemophilia B. Seminars in Thrombosis and Hemostasis. Sept 2018;44(6):578-589. 2. Factor IX inhibitors and anaphylaxis in hemophilia B. Journal of Pediatric Hematology/Oncology. Jan-Feb 1997;19(1):23-7. 3. Management of haemophilia B patients with inhibitors and anaphylaxis. Haemophilia. 1998 Jul;4(4):574-6. 4. Occurrence of hemophilia in the United States. The Hemophilia Surveillance System Project Investigators. American Journal of Hematology. 1998 Dec;59(4):288-94.

Pediatric Anesthesiology - 7 Spontaneous brachial arterial thromboses in a day-old neonate.

Nicole Ribeiro Marques[1], Anita Akbar Ali[2], Thejovathi Edala[3]

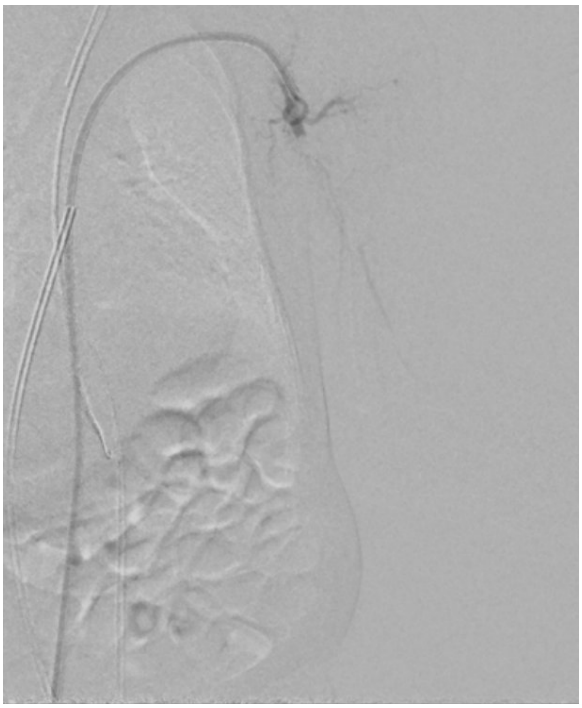
[1]University of Arkansas Medical Science, Little Rock, AR, [2]1 Children's Way, Slot # 203, Little Rock, AR, [3]University of Arkansas of Medical sciences, Little Rock, AR

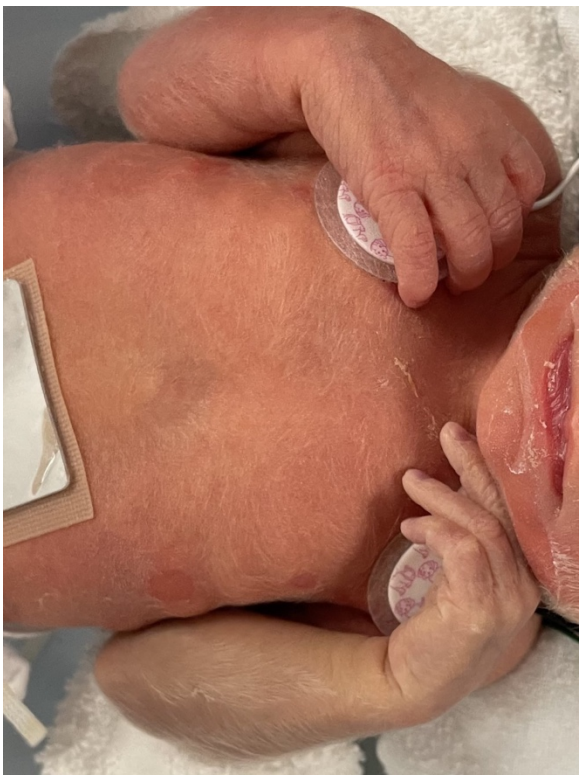
Introduction: Spontaneous arterial thromboses in neonates is rare without the presence of an additional risk factor for thrombosis. In this case report, we describe an unusual case of a day-old neonate presenting with left arterial thromboses involving the left brachial artery.

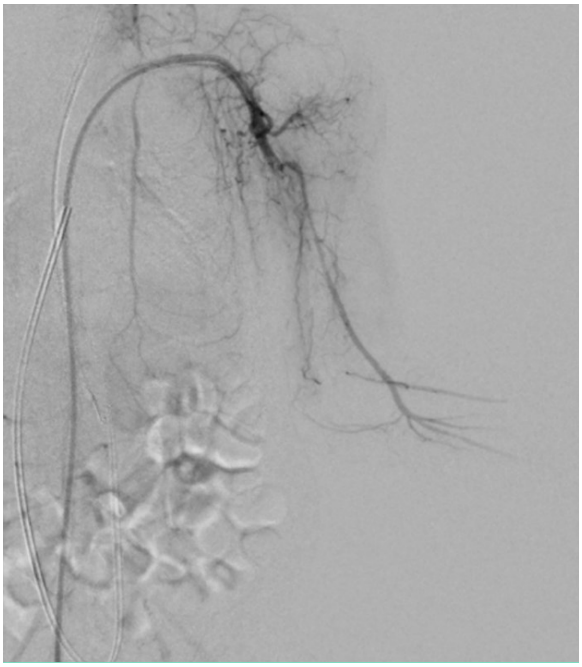
Methods: A male baby weighted at 1080-g, premature of 32 weeks gestational age, was delivered via C-section due to non-reassuring fetal heart tones and breech presentation to a 39-year G1P1 woman. Pregnancy was complicated by gestational hypertension, advanced maternal age, assisted reproductive technology, fetal growth restriction, abnormal dopplers and oligohydramnios. Mother was RH negative and received rhogam at 28 weeks. Antenatal steroids were completed. At birth CPAP was initiated, umbilical vein (UVC) and artery (UAC) catheters placed. Apgar scores were 3 at 1 minute and 7 at 5 minutes. No bleeding or stretch injury to arms/neck reported. Admission to NICU was complicated by hyperbilirubinemia (10.2), respiratory distress of the newborn requiring oxygen via nasal cannula and threatened limb. Approximately 25 hours after delivery, left arm and hand were noticed to be discolored, cool to touch, with weaker pulses and significant delay in capillary refill. Motor function was preserved. Vascular scan revealed occlusion of left proximal brachial artery consistent with acute thrombus. After transcranial ultrasound showed to be within normal limits, heparin infusion was initiate and patient was transferred to Arkansas Children's Hospital for endovascular thrombolysis and Alteplase (tPA) infusion catheter placement. Patient received on oxygen via nasal cannula, hemodynamically stable with purple discoloration of left forearm and hand. Maintenance fluid through UCV and arterial pressure monitoring through UAC. ASA standard monitors were applied before patient was preoxygenated, and was induced with a mixture of Sevoflurane and oxygen. Intravenous fentanyl (1 mcg/kg) and rocuronium (1mg/kg) were administered before direct laryngoscopy. Miller 0 blade and a 2.5 mm endotracheal tube used for intubation. Pressure controlled ventilation was initiated with tidal volume of 6 ml/kg. Anesthesia maintained with mixture of Sevoflurane, air and oxygen. Fluid maintenance with dextrose 10% at 4.6 ml/hr and albumin bolus was used for vascular expansion. Intraprocedural guidewire manipulations performed within the thrombus then 0.1 mL aliquots of tPA given over 30 minutes duration to dose of 0.8 mg tPA. Arterial vasospasm at site of thrombus was noticed at the end of the procedure. Patient was transferred to NICU intubated and with endovascular catheter within the left brachial artery for overnight tPA infusion of 0.1 mg/kg/hour and intravenous heparin at 10 units/kg/hour. Patient returned to intervention radiology for angiogram 12 hours later for removal of infusion catheter. Arterial narrowing remained at the left brachial artery with marked improvement in left extremity collateral flow. Anticoagulation with intravenous heparin was continued then transitioned to low molecular weight heparin. Infant's anti-Xa level was trended during hospital course and dosing adjusted accordingly to maintain goal of 0.5-1.

Conclusion: Spontaneous neonatal arterial thromboembolism is associated with a relatively high risk of morbidity and mortality. Preterm birth and low birth weight are reported as risk factors. Dramatic changes in circulation occurring during transition from fetal to neonatal life within the context of relative hemostatic immaturity attributed to these changes . In this case report the etiology is not clear as there is no family history of thrombophilia, not known shoulder dystocia, no anatomic abnormality noted on angiogram and negative genetic mutation of factors V Leiden and prothrombin 20210. Possible contributing factor was prematurity, low birth weight, maternal oligohydroamnios, possible compression by an intrauterine fibroid, previous COVID-19 exposure, and maternal COVID-19 vaccination. Further workup for thrombophilia could not be completed due to ongoing anticoagulation.

References: 1- Roles of the Microsurgeon and Anesthesiologist in the Treatment of Neonatal Upper Extremity Arterial Thrombosis: A Case Report. Keane AM, Santosa KB, Shahrawat SM et al. A A Pract. 2018 Mar 1;10(5):113-117. 2- Left brachial artery thrombus, left axillary vein thrombus, and stroke in a neonate with factor V Leiden mutation. Broxterman KJ, Mathew P, Chicoine L. J Pediatr Hematol Oncol. 2000 Sep-Oct;22(5):472-5. 3- Spontaneous neonatal arterial thromboembolism: infants at risk, diagnosis, treatment, and outcomes. Rashish G, Paes BA, Nagel K et al. Thrombosis and Hemostasis in Newborns (THiN) Group. Blood Coagul Fibrinolysis. 2013 Dec;24(8):787-97.







Pediatric Anesthesiology - 8 Anesthetic Considerations for Children with TANGO2-Related Metabolic Encephalopathy and Arrythmias

Gale K Goodlow[1], Patrick Young[2]

[1]University of Michigan, Ann Arbor, MI, [2]University of Michigan Health System, Ann Arbor, MI

Introduction: Transport and golgi organization 2 homolog (TANGO2) is a protein in humans encoded by the TANGO2 gene. Variants in the TANGO2 gene can lead to a rare genetic disorder known as TANGO2-Releated Metabolic Encephalopathy and Arrythmias (TRMEA). TRMEA in children can cause a number of conditions such as developmental delay, metabolic crises, malignant arrythmias – especially, QT prolongation- leading to cardiac arrest. Because of the recent discovery of this disease, there is a paucity of literature regarding the anesthetic management of affected patients.

Methods: Our medically challenging case involves a 4-year-old boy with a history of TRMEA who presented for PICC insertion and Transthoracic Echocardiogram with sedation. Three days prior, the patient arrived in our ED with two weeks of worsening ataxia and weakness. Further investigation of the patient's illness revealed severe rhabdomyolysis, transaminitis, and newly-diagnosed hypothyroidism. After obtaining informed consent, we provided anesthesia for PICC line placement and Transthroacic Echocardiography.

Conclusion: Because volatile anesthetics are known to prolong the QT interval, we elected to administer a mixture of Nitrous Oxide and Oxygen for peripheral IV placement. After placing the IV, we administered an IV lidocaine bolus and ran a propofol infusion for the Transthoracic Echocardiogram and PICC line placement. During the case, we avoided arrhythmogenic medications and any medications with the potential to prolong the QT-interval. The anesthetic and the procedures were tolerated well. The patient was the returned to the Pediatric ICU without incident.

Anesthetic management of children with TRMEA presents a unique challenge for Pediatric Anesthesiologists. Intricate knowledge of the pathophysiology and clinical manifestations necessitate adjustments and deviations from commonly practiced techniques in Anesthesia. Prior to administering an anesthetic to these patients, it is prudent for the Anesthesiologist to be familiar with the techniques utilized in the successful management of these patients. Specifically, volatile anesthetics, arrhythmogenic agents, and any drug with the potential to prolong the QT interval should be avoided. Anesthesiologists should be aware that patients with TRMEA are at high risk for malignant tachyarrhythmias in the perioperative period and should be prepared to treat issues should they arise.

References: 1. Multi-system disorder and severe recurrent rhabdomyolysis due to TANGO2 mutations in a 3 year-old child (online article), 2017. 2. TANGO2-Related Metabolic Encephalopathy and Arrhythmias (online article), 2018.

Pediatric Anesthesiology - 9 Sudden code blue during MRI scan in an 3 year-old patient with AML

Michael P Tulio[1], Zuhair A Siddiqui[1], Mary A Felberg[1], Thanh V Dang[2], Kelley N Gonzalvo[2]

[1]Baylor College of Medicine/Texas Children's Hospital, Houston, TX, [2]Baylor College of Medicine, Houston, TX

Introduction: Childhood cancer is the second most common cause of death in children younger than 15, with leukemias being the most frequent childhood malignancy. Acute myeloid leukemia (AML) is characterized by a clonal proliferation of myeloid precursors that can be life-threatening, potentially leading to coagulopathy, infections, thrombosis, metabolic derangements, and hemodynamic instability. In the unstable patient, tumor lysis syndrome (TLS) is often suspected and may trigger life-threatening arrhythmias. Many common procedures for this population occur in remote locations. The MRI scanner is a particularly formidable setting, as the fluctuating fields can interfere with monitors, it is difficult to communicate outside the room, and the magnet precludes availability of many valuable resources such as a defibrillator. Here we present a 3-year-old girl with AML who experienced a sudden cardiac arrest during an MRI under general anesthesia.

Methods: A 3 year-old, 15kg female presented with 1 month of right facial weakness and 1 week of left body weakness, fatigue, bruising, and lymphadenopathy. After diagnosis of AML, concern for TLS resulted in intravenous fluids at double the maintenance rate and frequent blood draws for chemistry panels. She presented for PICC line placement followed by brain/spine MRI. Standard induction and intubation preceded uneventful PICC insertion; EBL was minimal. Intraoperative hemoglobin was 7 g/dL, thus her fluids were stopped. During the MRI, increasing tachycardia and hypotension were noted. An hour into the MRI, pulse oximeter rate audibly slowed, EKG had unclear morphology, end tidal CO2 dropped to 15 mmHg, and BP was 60/20 mmHg, so the scan was stopped. The patient turned grey as the pulse oximeter and EKG waveforms disappeared. She was placed on 100% oxygen, sevoflurane was turned off, and 1mcg/kg of epinephrine was given without effect. The patient was moved into a safe zone for continued resuscitation, with ROSC after 10 minutes. Exam showed unequal pupils and frank blood in the nasogastric tube. Labs exhibited severe metabolic acidosis, DIC, and worsening anemia (Hgb 5.2 g/dL). Massive transfusion protocol was initiated; she received 35mL/kg pRBCs, 60mL/kg FFP, 5mL/kg cryoprecipitate, and 27mL/kg platelets. The ICU course was prolonged, with eventual discharge to home after chemotherapy for AML.

Conclusion: This case presented unique challenges, from a rapidly changing clinical picture resulting in cardiac arrest, to the challenges of emergencies during non-operating room anesthetics. There are several possible causes of her cardiac arrest. Since she had hyperkalemia and acidosis, worsening tumor lysis with arrhythmia is a possibility. A mechanical disturbance, such as an inappropriately placed PICC line, could also provoke arrhythmia, although chest X-ray confirmed appropriate placement. Anemia is also high on the differential, as her hemoglobin fell from 9.5 to 5.2 g/dL over 8 hours. Other factors contributing to the anemia were DIC, several invasive procedures, frequent lab draws, and a large amount of crystalloid given to avoid TLS. Additionally, thrombosis due to DIC or cell sludging from hyperleukocytosis could have led to cardiopulmonary collapse. However, the post code echocardiogram was non-explanatory. Given the challenges in MRI, treatment was delayed in recognizing and responding to the patient's deteriorating status. For example, although the EKG was abnormal just prior to the code, it was difficult to discern if it was an arrhythmia or interference from the magnet. MRI also precludes immediate access to the patient, hinders the ability to call for help, and limits availability of equipment that could prove useful in case of emergency. When encountering an emergency in the MRI, the patient should be moved from the scanner room to the adjacent area to have better access to equipment and personnel. Providers should also familiarize themselves with the location of emergency resources in this setting and consider drawing up emergency drugs ahead of time. Finally, an awareness of how the environment may bias treatment of the patient (e.g. not giving blood during a bloodless MRI) can guide more appropriate care for patients in these settings.

References: 1. A Practice of Anesthesia for Infants and Children. 240-256. 2019 2. J Thromb Haemost. 8(7):1477-1482. 2010 3. Cureus. 12(8):e9551. 2020 4. Anesthesiology. 122:495–520. 2015 5. “Overview of the Complications of Acute Myeloid Leukemia.” Uptodate, 24 May 2021, https://www.uptodate.com/contents/overview-of-the-complications-of-acute-myeloid-leukemia#H28. Accessed 24 May 2021. 6. “Clinical Manifestations, Pathologic Features, and Diagnosis of Acute Myeloid Leukemia.” Uptodate, 4 June 2021, https://www.uptodate.com/contents/clinical-manifestations-pathologic-features-and-diagnosis-of-acute-myeloid-leukemia#H1. Accessed 4 June 2021. 7. “Acute Myeloid Leukemia in Children and Adolescents.” Uptodate, 21 Apr. 2021, https://www.uptodate.com/contents/acute-myeloid-leukemia-in-children-and-adolescents. Accessed 12 May 2021.

Pediatric Anesthesiology - 10 Incidentally Found Complete Heart Block in Toddler Presenting for Urgent Surgery, Now What?

Amanda Williams[1], Harrison Wade[1], Beata Evans[1], Jeannette Kierce[1]

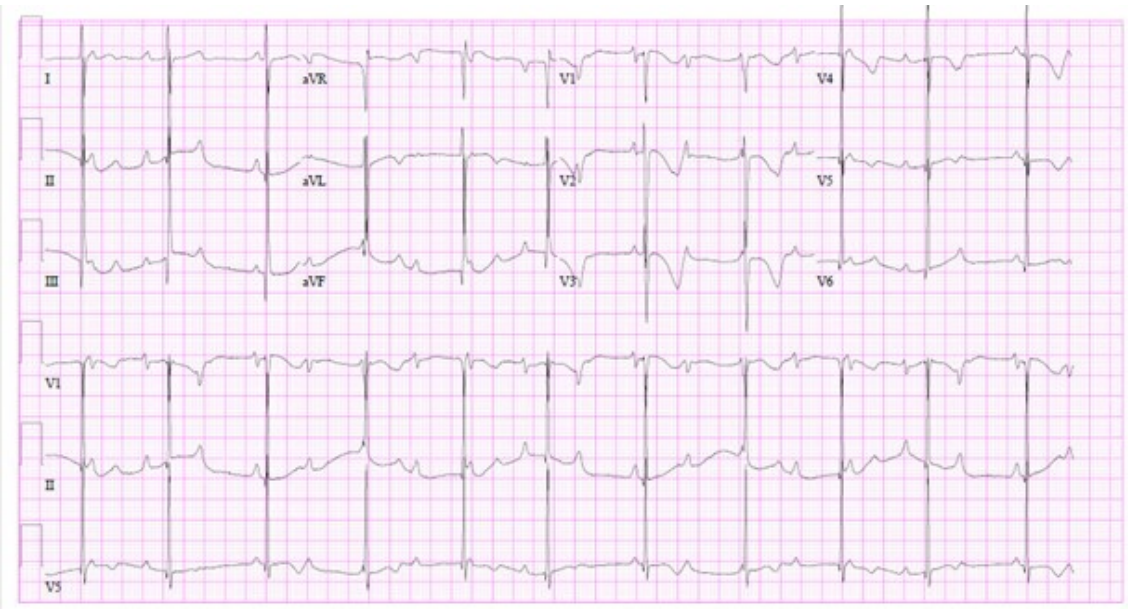
[1]Children Hospital of Richmond at Virginia Commonwealth University, Richmond, VA

Introduction: The purpose of this case report is to discuss preoperative and intraoperative management as well as anesthetic considerations for a 2-year-old with an incidental finding of complete heart block presenting for an urgent surgery.

Methods: This is the case of a 2-year-old female admitted with abdominal pain. The patient was born at 30 weeks gestation with a birth weight of 1.77 kg and required a 7-week stay in the neonatal ICU during which she required supplemental oxygen. Her diagnoses at discharge included patent ductus arteriosus (PDA) and bronchopulmonary dysplasia which had resolved by the time of presentation. She did not have any other illnesses, hospitalizations or surgeries and was not on any home medications. Her family history was negative for congenital heart defects, sudden cardiac death, early coronary artery disease or arrhythmias. While living in the United Kingdom, she was followed by a pediatric cardiologist for her PDA but had yet to establish routine health care upon recent relocation to the United States. The patient initially presented to the emergency department with a four day history of abdominal pain, concerning at the time for perforated appendicitis versus Meckel's diverticulum and was admitted for observation and IV antibiotics. During this admission she was noted to have a low heart rate and elevated blood pressure for her age. She was placed on telemetry and an electrocardiogram was ordered which showed an abnormal waveform, concerning for heart block. Cardiology was consulted and further ECGs revealed a high grade heart block with occasional conduction of atrial beats and the patient was newly diagnosed with complete heart block. Despite her cardiac history, her parents stated this condition had never been mentioned to them before. Given the novelty of the diagnosis, pediatric rheumatology was consulted to evaluate for possible underlying autoimmune disease. While admitted, she wore a 72-hour Holter monitor which showed an average heart rate of 57 beats per minute, with the lowest recorded being 35 beats per minute. The patient was initially treated conservatively with antibiotics for her intraabdominal process; however, due to persistent fevers, leukocytosis, abdominal pain and pelvic fluid collection, she was scheduled for an urgent appendectomy. As per pediatric cardiology recommendations, close follow up is necessary but a cardiac device is not warranted at this time. The development of signs or symptoms such as exercise intolerance, syncope, ventricular enlargement or hypertrophy, or heart failure would be indications to move forward with pacemaker implantation.

Conclusion: During this case presentation, we will discuss causes and frequency of high-grade atrioventricular blocks in children, temporary and definitive management of a pediatric patient with high-grade heart block who is scheduled for an urgent/emergent surgery and discuss the anesthetic implications of such. We will discuss the frequency of pacemaker implantation in children (as opposed to adults) and the risks associated with early pacemaker implantation.

References: Saleh, F, Greene, E, Mathison, D, Evaluation and management of atrioventricular block in children. Current Opinion Pediatrics. Emergency and critical care medicine. 2014, 26:279-285; Sykes, J, Lubega, J, Ezetendu, C et al. Asymptomatic Complete Atrioventricular Block in a 13-year old girl. Pediatric Emergency Care, November 2011, 27:1081-1083; Playfor, S, Khader, A, Case report. Arrhythmias associated with respiratory syncytial virus infection. Pediatric anesthesia, 2005, 15:1016-1018


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Pediatric Anesthesiology - 11

Perioperative Considerations for Pediatric Patient with Heartmate 3 Left Ventricular Assist Device

Federico Almonacid-Cardenas[1], Fabio Rodriguez-Patarroyo[1], Mateo Montalvo-Campana[1], Surendrasingh Chhabada[1]

[1]Cleveland Clinic Foundation, Cleveland, OH

Introduction: Children with medically refractory congestive heart failure require ventricular assist device (VAD) or heart transplant to survive. Historically, Berlin Heart® EXCOR pump was the only available option in these children. This device is a paracorporeal pneumatic driven pump with remarkable disadvantages, such as; damage to blood component, infection and it has a bulky controller console. These complications may lead to mobility restrictions and an increase in hospital length stay.[1] However, in December 2020, FDA approved Abbott's HearMate 3 device to be used in pediatric patients. HeartMate 3 is a newer continuous flow centrifugal pump consists of small inflow cannula, full magnetic levitation (MagLevTM) technology pump and outflow cannula.[2] Tunnel driveline is connected to the smaller controller. Furthermore, magnetic levitation pump has been engineered with wide blood-flow pathway, friction-free movement and intrinsic pulsatility to reduce shear stress and stasis of blood. Recently, this device was approved by the FDA in pediatric population. We present a case of pediatric patient schedule for Heartmate 3 VAD placement.[3]

Methods: CASE REPORT A 9-year-old boy with left ventricular (LV) non-compaction cardiomyopathy with congestive heart failure (CHF) was admitted to intensive care unit. Despite of inotropic support with milrinone and dobutamine infusions, the patient had compromised end-organ perfusion, that was evident by decrease mixed venous saturation and urine output. After multidisciplinary discussion, the treatment plan was to place HeartMate 3 VAD as bridge to transplant. Induction was performed with opioids, benzodiazepine and muscle relaxant; the prebypass course remain fairly stable. Transesophageal echocardiography was performed to rule out LV apical thrombus, patent foramen ovale and baseline right ventricular function. In addition to central line and arterial line, surgical team placed a left atrial (LA) line to further guide postoperative management. Apical LV inflow cannula connected to centrifugal pump and outflow graft to ascending aorta were placed on beating heart with cardiopulmonary bypass (CPB). Driveline was tunneled to left lower abdominal quadrant. After de-airing, the pump was started at 4000 rpm and slowly increased to unload LV, and separation from CPB was achieved with milrinone and epinephrine infusion. However, with increasing VAD speed, we noticed remarkable leftward interventricular septal shift, increase CVP and decrease LA pressure- raising the concern of worsening RV function. Consequently, VAD speed was reduced and inhaled nitric oxide therapy was initiated with immediate improvement of RV function. Postoperative course remain uncomplicated.

Conclusion: Although, some studies have addressed the optimal speed for VAD in adults, there is no guideline in pediatric population yet. It is critical to determine optimal VAD speed wherein the LV hemodynamics are favorable without causing an imbalance in the septal interaction, RV dysfunction and suction events. Furthermore, ideal speed allows midline interventricular septum, intermittent aortic valve opening and improved LV chamber dimensions. HeartMate 3 device is dependent on preload and extremely sensitive to afterload. Thus, the target is to avoid hypovolemia and excessive blood pressures changes. Ideal speed in VAD for pediatric population have not been set yet. We further discuss ideal pump speed and various causes of extreme VAD flow (Figure 1) and echocardiography imaging in VAD patients (Figure 2).

References: 1. Hansen G, Conradi A, Lequier L, Guerra GG, Quiñonez LG, Ross DB, Rebeyka IM, Buchholz H: Safety of Berlin Heart EXCOR Pump Changes in the PICU. Pediatric Critical Care Medicine 2013, 14(1):50-54. 2. O'Connor MJ, Lorts A, Davies RR, Fynn-Thompson F, Joong A, Maeda K, Mascio CE, McConnell PI, Mongé MC, Nandi D et al: Early experience with the HeartMate 3 continuous-flow ventricular assist device in pediatric patients and patients with congenital heart disease: A multicenter registry analysis. J Heart Lung Transplant 2020,

39(6):573-579. 3. Goldstein DJ, Naka Y, Horstmanshof D, Ravichandran AK, Schroder J, Ransom J, Itoh A, Uriel N, Cleveland JC, Jr, Raval NY et al: Association of Clinical Outcomes With Left Ventricular Assist Device Use by Bridge to Transplant or Destination Therapy Intent: The Multicenter Study of MagLev Technology in Patients Undergoing Mechanical Circulatory Support Therapy With HeartMate 3 (MOMENTUM 3) Randomized Clinical Trial.

Pediatric Anesthesiology - 12 How to ensure pediatric epidural catheter is in the correct space. It works for adults, will it work for children? Pediatric epidural placement conformation.

Beata Evans[1], Seamus Dore[2], Bryant Tran[3]

[1]Children Hospital of Richmond at Virginia Commonwealth University, Richmond, VA, [2]Children Hospital of Richmond at Virginia Commonwealth University, Richmond , VA, [3]Virginia Commonwealth University Medical Center, Richmond, VA

Introduction: The purpose of this case report is to discuss preoperative and intraoperative management for a 5 year-old 25 kg patient coming for a unilateral nephrectomy. Our anesthetic plan included general anesthesia with tracheal intubation and thoracic epidural for both intra and post operative pain control.

Methods: Epidural placement is a blind technique with many factors contributing to the successful placement. The loss of resistance technique to saline or air is nonspecific. In children, the epidural space is more superficial, and piercing ligamentum flavum is very subtle. Dural puncture with a spinal needle is too risky because of the low-lying spinal cord in pediatric patients, length of the spinal needle when compared to the length of the Tuohy needle, and easy dislodgement of the Tuohy with minimal manipulation. We have used the epidural gravity flow technique safely and successfully with epidural placements in adults. This technique transformed a blind procedure into a procedure that provides objective feedback. With this confirmatory technique, the epidural failure rate reached nearly zero in this patient group. We have a case of a 5-year-old 25 kg child for a scheduled unilateral nephrectomy. Our anesthetic plan included general anesthesia with a tracheal tube and thoracic epidural for intraoperative and postoperative pain control. After our patient was anesthetized and intubated, we positioned her in left lateral decubitus position with neck and hip flexion. After sterile preparation, proper handwashing, donning sterile gloves, we identified T8/T9 space using anatomical landmarks. We seated 19G Tuohy needle. Once we obtained, what we perceived as a loss of resistance with saline, we disconnected the glass syringe from the Tuohy hub. We carefully connected a sterile intravenous tubing extension filled with sterile saline to the Tuohy hub. The tubing was elevated above the level of the needle insertion site allowing some air to be entrained into the tubing. At that point, we observed slight variation in the saline column and continuous movement of the meniscus. This continuous movement of the saline meniscus was indicative of epidural pressure variation therefore Tuohy bevel being in the epidural space. This test allowed us to verify that the Tuohy bevel was in the epidural space, a catheter was safe to thread, and our epidural would provide excellent analgesia during and after the surgery.

Conclusion: During this case presentation, we will discuss epidural placement techniques and different confirmatory tests available. Based on this case, we found the epidural gravity flow technique is invaluable for confirming the proper position of the Tuohy bevel in the epidural space and eliminating epidural failures in children. In the future, we would like to evaluate if this approach can be applied to even smaller children and infants. We would like to evaluate if the size of the Tuohy needle, length and size of intravenous extension tubing affects this confirmatory test.

References: Grant, G, Echevarria, G, Agoliati, A, et al. Research letter. Epidural gravity flow technique for labor analgesia. Reg Anesth Pain Med. September 2020 Vol 45 No 9; Sethi, N, Chaturvedi, R. Editorial. Pediatric epidural. Journal of anesthesiology clinical pharmacology. Jan-March 2021, vol 28 issue 1

Pediatric Anesthesiology - 13 Protamine reaction in a pediatric patient during atrioventricular canal repair - strategies for early detection, management, and prevention

A Rebecca L Hamilton[1], David Whiting[2], Annette Schure[2]

[1]Boston Childrens Hospital, Boston, MA, [2]Boston Children's Hospital, Boston, MA

Introduction: Introduction Protamine reactions may present as pulmonary hypertension, systemic arterial hypotension and cardiopulmonary collapse in the adult population but are rarely reported in pediatric patients (1, 2). While protamine-induced hypotension has been estimated to occur in 1-3% of all pediatric cardiopulmonary bypass (CPB) cases, the true incidence of protamine reactions leading to right ventricle (RV) collapse in children has not yet been established (2).

Methods: Case Report A 3 year old girl presented with poor weight gain and a new murmur. She was found to have a partial atrioventricular canal defect with a medium-large primum atrial septal defect (ASD), cleft mitral valve and moderate regurgitation. She was scheduled for an elective surgical repair under general anesthesia. After an uneventful induction and line placement, the patient underwent ASD and cleft mitral valve repair on bypass. Unfortunately, the post bypass TEE showed a significant AV valve gradient which required a second bypass run. After surgical repositioning, she was again weaned from CPB with good biventricular systolic function. Systemic heparinization was reversed with protamine diluted 1:10 in normal saline (NS) and administered slowly by hand. The injection was stopped when peak inspiratory pressures began to rise. Saturation and ETCO2 then dropped precipitously, followed by progressive hypotension and bradycardia. The patient was given 100% FiO2, Albuterol, Calcium Gluconate and Epinephrine with poor response. Internal cardiac massage was initiated, the patient was re-heparinized and CPB re-instituted. The RV function eventually recovered and the patient was successfully separated from CPB. Protamine was diluted in 1:20 NS and administered over 20 minutes without any hemodynamic compromise. The patient was transferred to the ICU on inotropic support but in an otherwise stable condition. She was found to have mild pulmonary edema treated with diuretics and a temporary heart block requiring pacing which resolved within a few days. She recovered without any neurological deficits and was discharged home on postoperative day ten.

Conclusion: Conclusions: This case demonstrates the need for vigilant monitoring and proactive management of early signs of cardiopulmonary compromise following Protamine administration. Following this case, our division standardized Protamine delivery by implementing a policy to administer the dilute dose as an infusion over 15 minutes. We hope that this policy will reduce the risks associated with heparin reversal for our pediatric cardiac patients.

References: 1)Pediatric Anesthesia 2001; 11; 729-732 2) Basic & clinical pharmacology & toxicology 2008; 103: 192-196 3) Anesthesia & Analgesia 2003: 97: 383-389

Pediatric Anesthesiology - 14 Truncus Arteriosus repair in a patient with Smith-Lemli-Optiz syndrome: an up-hill perioperative battle against unforeseen challenges

A Rebecca L Hamilton[1], Alfonso Casta[1]

[1]Boston Children's Hospital, Boston, MA

Introduction: Truncus arteriosus (TA) is a cyanotic defect accounting for 1-3% of patients with congenital heart disease (CHD) (1). It is characterized by a single arterial trunk arising from the ventricles by means of a semilunar valve and is classified based on the origin of the pulmonary branches from the common trunk. TA is associated with DiGeorge's syndrome and without surgical repair up to 90% of patients die in infancy (2). Smith-Lemli-Optiz syndrome (SLOS) is a heterogenous autosomal recessive disease caused by an abnormal cholesterol metabolism. Its prevalence is 1 in 20,000-60,000 births in Caucasians with a carrier frequency of 1 in 30. While the phenotype varies greatly amongst affected individuals, its characterized by growth retardation, microcephaly, dysmorphic facial features, cleft palate, heart defects and underdeveloped genitalia (3).

Methods: Case report: A 2-day old full-term boy with a family history of SLOS presented with a TA type 1A, cleft palate, facial deformities, hypothyroidism and adrenal hypoplasia. On DOL2 the patient was taken to the OR for a primary truncus repair. The patient was a suspected difficult airway due to a cleft palate, micrognathia, limited mouth opening and mid-face hypoplasia. Mask ventilation was difficult but possible with an oral airway. A C-MAC was electively used for intubation obtaining a grade 1 indirect view after IV induction with muscle relaxation. Due to the abnormal vascular anatomy (including an aberrant right SVC and right subclavian artery) vascular access posed a challenge. The patient was loaded with stress dose steroids preoperatively and re-dosed throughout the case. Despite steroid loading, the intraoperative course was marked by autonomic instability requiring multiple pressors going on cardio-pulmonary bypass (CPB) as well as Procainamide boluses for recurring runs of SVT. Coming off CPB the patient was again hemodynamically labile with junctional bradyarrhythmia. Multiple vasopressors, aggressive resuscitation and AV pacing were required for hemodynamic support. The patient was severely hypocalcemic, hypomagnesemic and hyperglycemic and electrolytes were repleted as needed. Upon returning to the ICU, the patient remained intubated, sedated and treated with Epinephrine and Norepinephrine infusions to raise SVR and iNO to decrease PVR.

Conclusion: While TA patients are challenging from a cardiopulmonary standpoint due to pulmonary overload, heart failure and escalating PVR, TA patients with SLOS offer a whole new dimension of perioperative challenges. Main concerns in this case were a challenging airway and autonomic dysfunction due to adrenal insufficiency. By sharing these experiences we hope to offer some insight into how to approach the perioperative management of these highly complex neonates undergoing high-risk cardiac surgery.

References: 1) Seminars in Thoracic and Cardiovascular Surgery 2019; 23: 225-236 2) Seminars in Cardiothoracic and Vascular Anesthesia 2018; 2: 285-293 3) Anesthesiology 2002; 97: 1015-1019

Pediatric Anesthesiology - 15 Multiple Episodes of Intraoperative Cardiovascular Collapse During Posterior Spinal Fusions

Nnamdi Abalihi[1], Mariam Ashraf[2], Teeda Pinyavat[2]

[1]Columbia University Irving Medical Center - Department of Anesthesiology, New York, NY, [2]Columbia University Irving Medical Center, New York, NY

Introduction: Intraoperative anaphylaxis has been reported in 1:4000 to 1:25,000 cases (Agarwal 2015). Neuromuscular blocking agents, antibiotics, opioids, and blood transfusions are common culprits of intraoperative anaphylaxis (Khoriaty 2012). However, products used by the surgical team can also be allergenic; for example, gelatin-based hemostatic agents such as Floseal, derived from animal skin and bone products, have been implicated in incidents of intraoperative anaphylaxis.

Methods: A 12-year-old boy presented for scoliosis repair in July 2020 at a local community hospital. His medical history included TAPVR, secundum ASD and PDA status post repair, fungal meningitis requiring a VP shunt, seizure disorder, solitary kidney, and heterozygous Factor V Leiden mutation. Intraoperatively, he had a cardiac arrest shortly after the second screw was placed by the surgical team. He was stabilized after chest compressions multiple rounds of epinephrine. He found to have a right lower lobe subsegmental pulmonary embolism and moderately reduced biventricular function. He was managed in the ICU with inotropic support, anticoagulated, and discharged after one month with normal cardiac function. The patient presented again in June 2021 for scoliosis repair at our institution. His anticoagulation was held. In addition to standard monitors, arterial and central lines were placed. Anesthetic agents used during the case were midazolam, fentanyl, propofol, rocuronium and remifentanil. Tobramycin and cefazolin were used for antibiotic prophylaxis. During the first screw placement, the patient again developed severe hypotension. Elevated peak airway pressures were also noted. No rash was found but anaphylaxis was high on the differential. The surgery was aborted, he was quickly proned, and managed with epinephrine, dexamethasone, methylprednisolon, diphenhydramine, and albuterol. A TTE demonstrated normal biventricular function. He was maintained on a low dose epinephrine infusion and monitored in the ICU. The pediatric allergy and immunology team was consulted. The aforementioned anesthetic agents did not elicit a reaction on skin prick testing. The tryptase level sent intraoperatively was elevated at 37.5 mcg (normal <5) and antibody studies showed elevated IgE levels to beef, porcine gelatin, and bovine gelatin. These results suggest that anaphylaxis was the cause of hypotension and that Floseal was allergen. He returned to the OR the following week and underwent an uneventful scoliosis repair without the use of Floseal.

Conclusion: The cause of intraoperative anaphylaxis can be extremely difficult to identify due to the variety of agents used by both the anesthesia and surgical teams. A literature review from March 2021 found gelatin-based hemostatic agents as a cause of anaphylaxis in 21 patients, all of whom demonstrated elevated trypase level, positive porcine and bovine IgE, and positive to skin prick testing. Although this patient had a similar response during spine surgery one year prior, he did not undergo allergy work up earlier because pulmonary embolism was identified as the primary mechanism for the hemodynamic collapse. In retrospect, the sequence of events and the acuity of hemodynamic collapse during the first scoliosis repair also points to an anaphylactic reaction. Patients presenting for surgery should be asked a detailed history of their allergies including an intolerance to gelatin-containing or meat products which may warrant diagnostic testing to avoid intraoperative anaphylaxis.

References: Agarwal NS, Spalding C, Nassef M. Life-threatening intraoperative anaphylaxis to gelatin in Floseal during pediatric spinal surgery. J Allergy Clin Immunol Pract. 2015 Jan-Feb;3(1):110-1
Khoriaty E, McClain CD, Permaul P, Smith ER, Rachid R. Intraoperative anaphylaxis induced by the gelatin component of thrombin-soaked gelfoam in a pediatric patient. Ann Allergy Asthma Immunol. 2012 Mar;108(3):209-10.

Pediatric Anesthesiology - 16

Perioperative morbidity in a child with severe obstructive sleep apnea

Michael Hafeman[1], Viktorya Poghosyan[2], Sidrah M Ahmad[1], Vahe Tateosian[1]

[1]Stony Brook University, Stony Brook, NY, [2]Stony Brook University, Stony Brook, United States of America

Introduction: Pediatric obstructive sleep apnea (OSA) is associated with behavioral and neurocognitive dysfunction and cardiovascular morbidity [1]. Diagnosis is often complex and may be delayed [2]. It's pathophysiology differs from that of adults and adenotonsillectomy is commonly the first-line treatment. These patients often present with significant perioperative challenges.

Methods: A 6 year-old male, BMI 35, with a history of chronic rhinitis, was referred to Otolaryngologist for adenotonsillar hypertrophy. He had OSA signs/symptoms including ADHD, unrefreshed sleep, stertor when awake and witnessed apneas. Nocturnal polysomnogram (PSG) revealed severe sleep disordered breathing with apnea/hypopnea index (AHI) of 168 (severe: >10) and desaturations to <70%. He did not tolerate CPAP or nasal cannula. On initial presentation to Otolaryngologist his tonsils were noted to be small. Sleep endoscopy with adenoidectomy, possible tonsillectomy, possible lingual tonsillectomy vs adenotonsillectomy was discussed. Decision was made to proceed with sleep endoscopy to address the sites of obstruction. The patient underwent a mask induction with volatile anesthetic agent, Sevofluorane. He had significant upper airway obstruction during the induction process, which was relieved partially by oral airway insertion. On sleep endoscopy he was noted to have complete obstruction at level of adenoids and complete collapse at level of tonsils. He had minimal collapse at the level of the base of tongue. He underwent adenotonsillectomy with pharyngoplasty. Fentanyl 50 mcg and IV Acetaminophen was administered for analgesia during the procedure. Surgery duration was 59 minutes; emergence was prolonged with 47 minutes prior to extubation. He was extubated and taken to PACU with oxygen via non-rebreather mask. Oxygen saturation was 90%; he was transitioned to NIPPV. Arterial blood gas (ABG) analysis performed 75 minutes post-extubation showed 7.15/104/134/36. After a multi-disciplinary discussion with the family, reintubation was considered, however, the parents refused. The patient remained somnolent for approximately 2 hours, after which appeared more alert and responding appropriately. Overnight the patient remained on NIPPV. The following morning he removed the mask and refused nasal oxygen. On POD-1 he was noted to have mucosal oozing from anterior pillars which ultimately self resolved. He refused NIPPV or oxygen overnight and desaturated to low 80s while sleeping on room air His O2 saturations maintained in high 80s to low 90s on blow-by oxygen. On POD-2, ABG was improved to 7.41/53/58/33 on room air. Due to patient not having oxygen at home and not being able to determine safe level of oxygen requirement (NIPPV vs nasal O2 vs blow by) he was kept one additional night for observation on blow-by. He desaturated to high 80's with self resolution. He was discharged home on POD-4 with instructions to use supplemental oxygen via nasal canula or blow by if unable to tolerate nasal canula. Over the next week, parents reported improved oxygenation with no desaturations below 90%. One month post operatively, parents reported patient with oxygen level above 95% throughout the night even without using oxygen.

Conclusion: This patient had multiple risk factor for post-operative respiratory complications after adenotonsillectomy: high apnea-hypopnea index, low SpO2 nadir on PSG, rhinitis, and CO2 retention on PSG [3]. A clear relationship between adenotonsillar size and AHI has not been established, especially in obese children [4], and recurrence of OSA is common in the years after surgery [5]. These suggest that the cause of OSA is often multifactorial. Reintubation was suggested by a multi-disciplinary team consisting of pediatric anesthesiology, ENT, pulmonary and ICU teams. Use of NIPPV after adenotonsillectomy carries risk of post-operative hemorrhage by drying out the tonsillar bed and sheering force from the positive pressure. Although preliminary investigations suggest it's safety [6], this has not been well studied. It's use was limited in this case but was without complication. Areas for further study include predictors of pulmonary complications after adenotonsillectomy in pediatric patients with severe OSA. The safety of post-operative use of NIPPV, including what settings may be

considered 'safe' in the immediate post-operative time period, especially with respect to hemostasis.

References: 1. Pediatric obstructive sleep apnea: complications, management, and long-term outcomes. Proc Am Thorac Soc. 2008;5(2):274-282. 2. Prolonged morbidity due to delays in the diagnosis and treatment of obstructive sleep apnea in children. Clin Pediatr (Phila). 2000;39(2):103-108. 3. Polysomnography variables associated with postoperative respiratory issues in children <3 Years of age undergoing adenotonsillectomy for obstructive sleep apnea. Int J Pediatr Otorhinolaryngol. 2020;137:110215. 4. Systematic review of pediatric tonsil size and polysomnogram-measured obstructive sleep apnea severity. Otolaryngol Head Neck Surg. 2011;144(6):844-850. 5. Correlations between obstructive sleep apnea and adenotonsillar hypertrophy in children of different weight status. Sci Rep. 2019;9(1):11455. 6. Treatment outcomes of adenotonsillectomy for children with obstructive sleep apnea: a prospective longitudinal study. Sleep. 2014;37(1):71-76.

Pediatric Anesthesiology - 17 No Source Control? No Problem! Sticking to the Principles of the Massive Transfusion Protocol Following Hepatic Subcapsular Rupture and Hemorrhage

Mohammed Waqar[1], Chyongjy Liu[2], Haley DeLaGarza[3], Monica Chen[4]

[1]U.T. McGovern Medical School, Houston, United States of America, [2]Baylor College of Medicine, Texas Children's Hospital, Houston, TX, [3]Texas Children's Hospital, Houston, United States of America, [4]Texas Children's Hospital / Baylor College of Medicine, Houston, TX

Introduction: Hemorrhagic shock is associated with high morbidity and mortality. A massive transfusion protocol (MTP) can guide temporary hemostatic resuscitation before definitive surgical intervention is achieved, though standards for MTP vary across institutions. Hemodynamic instability, electrolytes disturbances (e.g. hyperkalemia, hypocalcemia), and acidosis are often seen in those patients undergoing resuscitation for hemorrhagic shock.

Methods: Case Report: A 20-month-old female, 11.6kg, with a history of portal hypertension secondary to chronic portal vein thrombosis, on therapeutic enoxaparin, presented with acute hypotension and tachycardia. The patient's medical history was significant for recent mesocaval and splenorenal shunts status post angioplasty and stent placement due to thromboses. Initial resuscitation efforts by the intensive care unit (ICU) were directed towards presumed septic shock due to a simultaneous fever. However, progressive distention and rigidity of the patient's abdomen presented a new concern for abdominal compartment syndrome in the setting of hemorrhagic shock. Point-of-care ultrasound showed active bleeding from the liver. MTP was activated, and the patient was brought to the operating room for an emergent exploratory laparotomy. In the operating room, a multidisciplinary-developed MTP protocol was used to guide resuscitation efforts in the setting of profound hemoperitoneum. Following incision, 2 liters of blood were immediately removed. Multiple surgical attempts to obtain source control failed. Ultimately, interventional radiology-guided embolization of the right hepatic artery was required to achieve hemostasis. Total blood loss was estimated to be more than 5 liters. At the conclusion of the case, the patient received 13 units of packed red blood cells (PRBC), 8 units of fresh frozen plasma (FFP), 6 units of platelets, and 20cc/kg of cryoprecipitate. The patient was transported to the ICU appropriately resuscitated, with stable labs, and on minimal vasopressor support.

Conclusion: An institutional MTP protocol plays a vital role in guiding resuscitation. A collaborative effort from multiple stakeholders is required to develop an institutional MTP protocol. Coordinated resource management is crucial for effective and efficient resuscitation in emergent situations. And likewise, early recognition of blood loss, in conjunction with clinical judgment, allows for timely MTP activation. Initial resuscitation efforts should begin with administered products at a 1:1 (PRBC:FFP) ratio. This ratio has been associated with the highest survival in severely injured children receiving massive transfusion. Once lab results are available, 'goal-directed' transfusion can ensue with real-time rotational thromboelastometry. Hemostatic adjuncts like tranexamic acid (TXA) have excellent application in hemorrhagic shock. TXA is a safe and effective adjunct in most MTP cases. However, it was not used for this patient given concern over her multiple active intravascular thromboses of unknown etiology. Contraindications to the use of TXA in MTP can include allergy, subarachnoid hemorrhage, hematuria, and active intravascular thrombosis. Additionally, it remains debatable whether partial reversal of enoxaparin with protamine would have offered any benefit. Careful attention was necessary to prevent hyperkalemia and hypocalcemia, correct acidosis, and address ongoing hypothermia. Though the patient received over 6 liters of blood product, she never had significant metabolic derangements due to aggressive treatment of electrolyte abnormalities, any of which would have been potentially fatal in the setting of this prolonged and extensive resuscitation.

References: 1. Journal of Blood Medicine (2020). 11, 163–172. 2. Journal of Pediatric Surgery (2019). 54(2), 345–349. 3. Pediatric Emergency Care (2018). 34(8), 594-598.

Pediatric Anesthesiology - 18 Palliative Anesthesia for an Effusive Pediatric Cancer Patient

pulsar Ii[1], Kevin Choi[2], Mihael Rosenbaum[1]

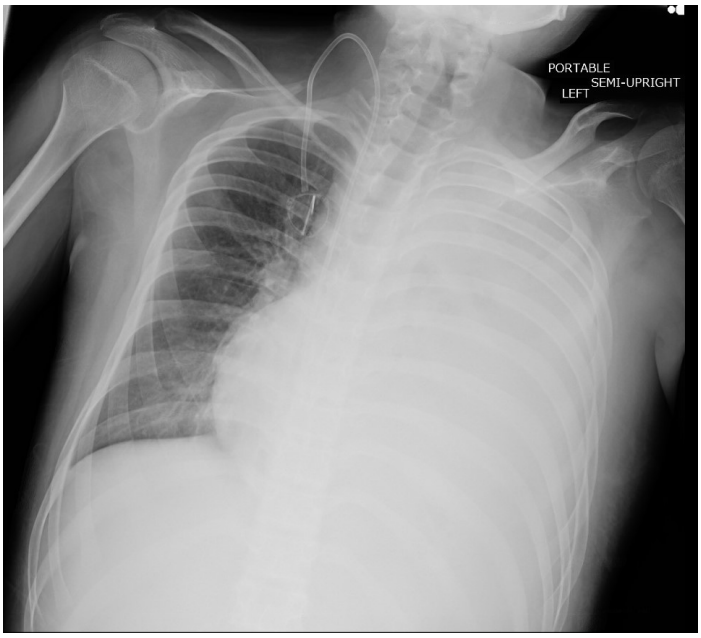
[1]Loyola University Medical Center, Maywood, IL, [2]Loyola University Medical Center, Chicago, IL

Introduction: Providing anesthesia for an unstable pediatric patient demands thoughtful, compassionate, and collaborative care. We describe the anesthetic and palliative considerations for a terminally-ill child undergoing thora-pericardiocentesis.

Methods: Case: A 12 year old Latino male (46 kg) was admitted with refractory progressive T-cell lymphoblastic leukemia (LBL) relapse, now complicated by both pleural and pericardial effusions with tamponade physiology and severe left lung collapse requiring O2 (figure 1). Anesthesia was consulted to provide sedation for emergent pericardiocentesis and left-sided chest tube placement due to severe orthopnea, as he only tolerated the seated left lateral decubitus position. During preoperative assessment with Spanish interpretation, the risk of cardiovascular collapse was emphasized with patient and mother, who wished to proceed since Christmas was only days away. In the operating room, the patient was gradually positioned into the Semi-Fowler's position for comfort and surgical access. Moderate IV sedation was achieved with midazolam, dexmedetomidine, and ketamine. Pericardiocentesis and thoracentesis produced 300 mL and 60 mL serosanguinous fluid, respectively. During his 3-week postop course, chemotherapy was restarted, code status switched to DNR, and he was discharged home with physical therapy. One month after his discharge, he passed away of late complications. Discussion: Refractory LBL often localizes as a massive mediastinal mass and management is difficult with a 14% (SE = 6%) overall survival (1). In light of his tamponade physiology, we favored a 'fast' heart rate and maintained intravascular volume ('full'), contractility, as well as spontaneous ventilation since positive airway pressure can decrease venous return. Our patient's family initially resisted palliative care during frank multi-disciplinary code discussions. Underutilization of palliative care services has been reported among ethnic minorities specifically Latino, Indian, Native and African-Americans, and is often attributed to lack of the family's familiarity with hospice and palliative care services, language barriers, religious differences, difficulties in accessing insurance, distrust of the healthcare services, and physicians' discomfort (2). Although our care did not impart long term survival, our care relieved symptoms and the wishes of the child and mother were met at their emotional pace.

Conclusion: 1) Palliative care services is often underutilized in ethnic minorities for reasons listed above. 2) At the end of a child's life, the focus needs to be on quality of life as defined by the family, not the provider. 3) Anesthetic management for tamponade physiology should favor high heart rate, volume, and contractility.

References: 1) Wiener, L et al. Cultural and religious considerations in pediatric palliative care. Palliat Support Care. 2013 Feb;11(1):47-67. 2) Burkhardt, B et al. Poor outcome for children and adolescents with progressive disease or relapse of lymphoblastic lymphoma. J Clin Oncol. 2009 Jul 10;27(20):3363-9.



Perioperative Anesthesia

Perioperative Anesthesia - 1 TPA to The Rescue: A Case of Intraoperative Pulmonary Embolism During Emergent Hip Arthroplasty

Jay A Sanford[1], Ivy Forkner[2]

[1]Emory University, Atlanta, GA, [2]Emory University Hosptial Midtown, Atlanta, GA

Introduction: We present a report of extreme intraoperative hemodynamic instability instigated by a large pulmonary embolism, in a patient undergoing emergent hip arthroplasty.

Methods: The patient was a 43yo female, brought to the OR for ORIF/THA of a fracture sustained from a ground level fall. Medical history notable for likely metastatic lung CA, polysubstance abuse, morbid obesity, and hypertension. Prior to being brought to the OR, patient had been bed-bound for over a week given the extreme pain she experienced with her fracture. Induction was uneventful, however after 30 minutes, some desaturation was noted. A discussion between the anesthesiologist and the attending surgeon determined that these desaturations were unlikely reoccur and the surgery proceeded. One hour into the surgery, a precipitous fall in end-tidal CO2(ETCO2) was noted, and a palpable pulse was not detected. Three rounds of CPR and defibrillation were completed, with only fleeting return of circulation. In the ensuing twenty minutes since hemodynamic collapse, multiple vasopressors boluses were given and infusions were initiated, including vasopressin, norepinephrine, and epinephrine. A tenuous return of circulation was achieved after 30 minutes of resuscitative efforts. Given the persistent hemodynamic instability, the likelihood that this was a pulmonary embolism (PE) was discussed. With our facilities lack of available mechanical support (i.e. Extracorporeal Membrane Oxygenation ECMO), the decision was made to administer tissue-plasminogen-activator (tPA). tPA was administered at 100mg over two hours, per the pharmacy issued dosing protocol. The surgery was completed, and patient was transferred intubated, on vasopressor therapy to the ICU. A bedside transthoracic echocardiogram (TTE) and CT scan was ordered, demonstrating a normal myocardium on TTE, but multiple pulmonary emboli were noted on CT scan. Patient's status improved dramatically and quickly post-operatively. The patient was extubated and weaned off vasopressors within 24 hours, and subsequently discharged from the hospital on day seven.

Conclusion: This case report illustrates two important concepts. First and foremost, given the extremely high mortality of intraoperative PE, as high as 50% in trauma patients, the need to keep pulmonary embolism high on your intraoperative differential for hemodynamic collapse, can't be overstated. (1) This is especially true in patients undergoing long-bone orthopedic surgery with a history of medical comorbidities that tend toward hypercoagulability (obesity, metastatic cancer, immobility, orthopedic injuries). (2,3) Secondly, given that several studies have demonstrated the effectiveness and improvement in mortality in massive PE when TPA is administered, the need to standardize the management of intraoperative TPA administration in this setting, especially in centers where immediate mechanical cardiopulmonary support (i.e. ECMO) is not available, is critical. The time from hemodynamic compromise to administration of TPA was nearly 30 minutes, despite the anesthesiologist managing the case understanding the possibility that this was an embolic event. With standardized intraoperative protocol regarding PE diagnosis and TPA usage, the time from PE diagnosis to TPA being delivered could be decreased considerably, leading to saved lives and quicker decision making. (2) The presence of another attending anesthesiologist who was assisting with the resuscitation, who had previous experience with intraoperative TPA administration under an identical scenario, likely also led to TPA being used. A protocol could replace the need for experience, and perhaps lead even the inexperienced provider to the same conclusion. We believe that with quick recognition of the diagnosis and the subsequent treatment of the PE with tPA via a robust protocol, lives can be saved. The incorporation of a protocolized approach to intraoperative PE management, especially in centers with limited resources (i.e. ECMO) could further hasten this process, and serve to guide decision making, especially in situations where experience, personnel, and equipment with might be lacking.

References: 1. Okoronkwo, T. et. al. “Early Detection and Management of Massive Intraoperative Pulmonary Embolism in a Patient Undergoing Repair of a Traumatic Acetabular Fracture.” Case Reports in Anesthesiology. 2018. 2. Alan M. Smeltz, Lavinia M. Kolarczyk, Robert S. Isaak. “Update on Perioperative Pulmonary Embolism Management: A Decision Support Tool to Aid in Diagnosis and Treatment.” Advances in Anesthesia, Vol 35, Issue 1, 2017, 213-228, ISSN 0737-6146. 3. Q. Hao, R. Dong Bi, J. Yue, et al. “Thrombolytic therapy for PE.” Cochrane Database Syst Rev Vol 9, 2015.

Perioperative Anesthesia - 2 Anesthetic management of a patient with severe history of postoperative nausea and vomiting undergoing liver resection.

Jeffrey D Mimbs[1], Zach Arnett[2], Jayachandra Induru[1]

[1]Tulane University School of Medicine, New Orleans, LA, [2]Tulane Medical Center, New Orleans, LA

Introduction: Post-operative nausea and vomiting (PONV) is defined as nausea, vomiting or retching in the immediate 24 hours postoperative. In this case, we present a patient with severe history of PONV and the strategies utilized to mitigate these symptoms. A patient presented for liver lobectomy with PMH of liver malignancy, HTN, and severe PONV. The patient had 7 prior surgeries, all resulting in severe PONV, lasting up to 30+ hours. One instance was so severe that the patient experienced submucosal esophageal tearing. Anesthetic Technique: For this case, we used an opioid free total intravenous anesthetic with a lumbar epidural for postoperative pain control and to avoid postoperative narcotics. The patient was given 2mg midazolam and taken to the OR. A midline lumbar epidural was placed at the L1-L2 interspace. Following a negative test dose, the epidural catheter was bolused with 10cc 0.25% plain bupivacaine. Standard ASA monitors were placed in supine position. General endotracheal anesthesia was induced with 25mg ketamine, 60mg lidocaine, 100mg propofol, 10mcg dexmedetomidine, and 40mg rocuronium. The patient was intubated with video laryngoscopy and 7.0 cuffed ETT. Sevoflurane was initiated while access was established. A right radial arterial line and right internal jugular triple lumen central venous catheter were placed. Infusions of propofol (100mcg/kg/min), dexmedetomidine (0.2mcg/kg/hr) and ketamine (0.2mcg/kg/hr) were begun and continued throughout the intraoperative course.

Methods: Intraoperative Course: The patient was maintained with the above infusions. Muscle relaxation was monitored and rocuronium was redosed as needed. A scopolamine patch was applied preoperatively to the right post-auricular surface. 8mg dexamethasone was given prior to incision. 4mg ondansetron was given halfway through the case and 15 minutes prior to the end of the procedure. 2.5mg olanzapine was given intramuscularly 45 minutes prior to the end of the procedure, as our institution does not have droperidol or phenothiazines on formulary. Postoperative Course: The patient was transferred to the SICU and extubated within an hour of arrival. The patient reported transient nausea that resolved without intervention. The patient did not experience vomiting or retching at any point in the postoperative period. At the 30-hour mark, the epidural catheter was removed intact and 8 hours later DVT prophylaxis was begun. During the 30-hour period that the epidural was infusing 12 cc/hr of 0.125% plain bupivacaine, the patient received one dose of 15mg ketorolac IV for breakthrough pain. The patient was transferred to the surgical floor and then discharged home.

Conclusion: PONV is considered one of the most significant postoperative complications following general anesthesia, being reported in up to 30% or more of all patients.1 Some studies even suggest that patients place as high of a value in PONV as they do in postoperative pain.1-2 PONV is commonly unrecognized as it can occur following discharge to home, often from same day surgeries. PONV is usually multifactorial in etiology, with factors such as agents used, type of surgical procedure performed, and patient risk factors strongly leading to the increase in incidence of PONV. Preventing PONV is important as it frequently impairs quality of recovery, decreases patient satisfaction scores, delays discharge, and may cause unanticipated admissions, leading to increased healthcare costs and spending. The Apfel scoring system is commonly used to help predict the pre-operative risk for PONV. Other important factors include surgical site (intraperitoneal surgeries, strabismus, ENT), use of volatile anesthetics or nitrous oxide, large doses of neostigmine, duration of surgery, and gastric distention.2-3 Regarding therapy, it's important to develop a prophylactic regimen to limit the risk of PONV. For antiemetics, using drugs from separate classes is beneficial. Some studies suggest a single intervention in a patient with 4 risk factors resulted in an absolute risk reduction of 21% compared with 3% in patients with 1 risk factor.4 As demonstrated above, we successfully delivered an opioid free anesthetic that limited the amount of PONV this patient experienced to a minimal level. By choosing a TIVA technique, using multimodal pain strategies, and avoiding the use of inhalational agents

and opioids, we were able to help prevent PONV in a high-risk patient.

References: 1. Clinical Anesthesiology. New York: Lange Medical Books/McGraw Hill Medical Pub. Division, 2006. Print. 2. Miller's Anesthesia. 7th ed. Philadelphia, PA: Churchill Livingstone/Elsevier, 2010. 3. Faust's anesthesiology review. 2015. 4. Postoperative nausea and vomiting following inpatient surgeries in a teaching hospital: a retrospective database analysis, Current Medical Research and Opinion, 22:6, 1093-1099, 2006.

Perioperative Anesthesia - 3 A Case of Serotonin Toxicity Following Intraoperative Fentanyl Administration

Christabel Jaiyeola[1], Amy E Kim[1], Alexander Papangelou[1]

[1]Emory School of Medicine, Atlanta, GA

Introduction: Serotonin toxicity, aka serotonin syndrome, is a rare, potentially life-threatening disease characterized by serotonergic overactivity. Presentation ranges from mild tremors to life threatening hyperthermia and death. Classically, it has been described as a triad of altered mental status, neuromuscular activity, and autonomic hyperactivity.

Methods: A 35-year-old woman with asthma, anxiety, and sciatica presents for hysteroscopy for abnormal uterine bleeding. Patient denied history of anesthesia complications. Home medications included lorazepam, quetiapine, vortioxetine, albuterol, none of which were taken the morning of surgery. The patient received midazolam 2mg for anxiolysis. Patient was induced with lidocaine 60mg, fentanyl 50mcg, and propofol 150mg. An i-gel supraglottic airway (Intersurgical Ltd, Burlington, ON) was placed and sevoflurane was used for maintenance of anesthesia. Immediately following i-gel placement, patient exhibited spontaneous myoclonic jerks and inducible myoclonus in response to neck palpation while evaluating i-gel position. The myoclonus improved spontaneously, and the procedure continued. The procedure was completed with no operative interventions. No additional opioids were administered. Given the post-intubation observations, ondansetron was not given and the patient received dexamethasone 4mg and diphenhydramine 12.5mg for PONV prophylaxis. While being taken out of lithotomy position, patient demonstrated spontaneous myoclonus and prolonged stimuli-induced clonus in the bilateral lower extremities at ankles and knees. Patient's upper and lower extremities and neck were rigid on physical exam. Vital signs revealed tachycardia and slight increase in blood pressure, but temperature and end-tidal CO2 remains normal. Frontal processed EEG (Root with Sedline, Masimo Corporation, Irvine, California) was placed and raw EEG was not consistent with seizure activity. Due to the suspicion for serotonin syndrome and anticipated clinical course, the patient's airway was secured. She was induced with propofol and rocuronium and video laryngoscopy employed to replace the i-gel with an endotracheal tube. Lorazepam 2mg was administered. The patient was transferred to the SICU where she continued to have inducible myoclonus and agitation. Cyproheptadine is administered until symptoms resolved. CMP, CBC, coagulation panel, creatinine kinase, and urinalysis are normal. Urine toxicology analysis was negative including for methadone. A urine 5-HIAA level and a 24-hour 5-HIAA level started 2 hours after ICU admission were all normal. The patient was extubated the following day and had no recall of her entire clinical course.

Conclusion: Serotonin toxicity is caused by ingestion of one or more drugs that increase 5-HT or activate 5-HT1A and 5-HT2A receptors. Many drug classes have been implicated in serotonin toxicity including SSRIs, SNRIs, TCAs, opioids, amphetamines, antiemetics, triptans, second-generation antipsychotics and MAO-inhibitors. Our patient's serotonin toxicity may have been precipitated by fentanyl administered during induction in the setting of being on multiple serotonergic agents at home. Serotonin toxicity has a wide range of presenting symptoms and is a clinical diagnosis. The Hunter Serotonin Toxicity Criteria (HSTC) has the highest sensitivity (84%) and specificity (97%) for diagnosis. Lower extremity clonus combined with known serotonergic insult has been shown to be strong supporting evidence for a diagnosis of serotonin toxicity. The onset and resolution of serotonin toxicity is usually rapid, occurring within the first 24 hours of exposure to the insulting agents(s). Lab testing can be variable. Serotonin serum and urinary 5-HIAA (the principal metabolite of serotonin) levels do not correlate well with the presence or severity of disease. Accurate measurement of 5-HIAA levels require specific processing and can be influenced by diet. Evidence of kidney or liver failure can be present, but is often not. Treatment involves administration of benzodiazepines, supportive care, cyproheptadine. Overall, the case highlights the importance of exercising caution with patients presenting for surgery that potentially have high serotonergic tone. Some of the most commonly used intraoperative agents can trigger serotonin toxicity even in modest doses. Awareness of the signs and

symptoms of serotonin toxicity is becoming crucial knowledge for the astute clinical anesthesiologist.

References: 1. Serotonin syndrome. Ochsner J. 2013;13(4):533-540. 2. Serotonin Syndrome: Pathophysiology, Clinical Features, Management, and Potential Future Directions. Int J Tryptophan Res. 2019;12:1178646919873925. Published 2019 Sep 9. 3. Serotonin Syndrome in the Perioperative Setting. Am J Case Rep. 2018;19:833-835. Published 2018 Jul 16. 4. The Hunter Serotonin Toxicity Criteria: simple and accurate diagnostic decision rules for serotonin toxicity, QJM: An International Journal of Medicine, Volume 96, Issue 9, September 2003, Pages 635–642 5. Serotonin syndrome. Presentation of 2 cases and review of the literature. Medicine, 79(4), 201–209. 6. Serotonin Syndrome and Other Serotonergic Disorders, Pain Medicine, Volume 4, Issue 1, March 2003, Pages 63–74.

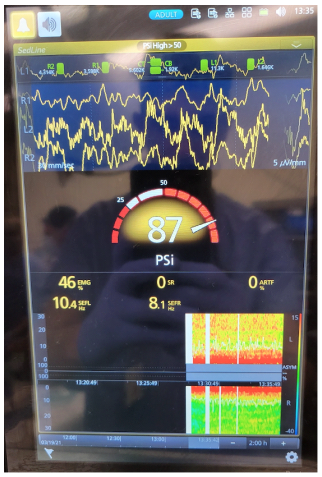


Figure 1. Image of Frontal processed EEG (Root with Sedline, Masimo Corporation, Irvine, California) with raw EEG show no evidence of seizure activity.

Hunter Serotonin Toxicity Criteria				
Presence of a serotonergic agent *plus* (one of the following)				
Spontaneous Clonus	Inducible clonus *and* Agitation *or* Diaphoresis	Ocular Clonus *and* Agitation *or* Diaphoresis	Tremor *and* hyperreflexia	Temperature >38°C *and* Hypertonia *or* Ocular clonus

Table 1. The Hunter Serotonin Toxicity Criteria. Adapted from E.J.C. Dunkley, et al. The Hunter Serotonin Toxicity Criteria: simple and accurate diagnostic decision rules for serotonin toxicity, *QJM: An International Journal of Medicine*.

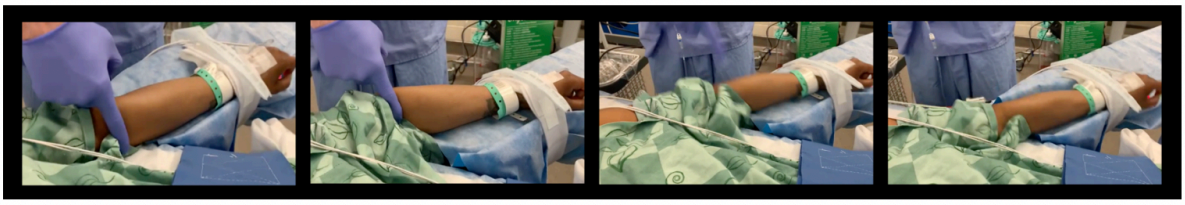


Figure 2. Photo showing “rubber-band-like” upper extremity rigidity at the end of the case.

Perioperative Anesthesia - 4 Two cases of circulatory collapse suspected due to remimazolam anaphylaxis

Satoshi Uchida[1], Daiki Takekawa[1], Kazuyoshi Hirota[2]

[1]Hirosaki University Hospital, Hirosaki, Japan, [2]Hirosaki University Graduate School of Medicine, Hirosaki, Japan

Introduction: Remimazolam, a novel benzodiazepine characterized by its ultra-short acting property with flumazenil as a specific antagonist, was approved as a general anesthetic in Japan in January 2020. Although the safety of remimazolam has been evaluated, the prevalence of its anaphylaxis has unknown yet because it's still a relatively new drug. Actually, our PubMed search could reveal only a report describing remimazolam anaphylaxis (1). Here, we report two cases of circulatory collapse suspected due to remimazolam anaphylaxis during anesthetic induction with elevated serum tryptase.

Methods: We obtained a written informed consent from the patients for publication of this case report. Case 1: A 74-year-old male (height, 157 cm: weight 78 kg) was scheduled for debridement and skin grafting for severe burn injury. Anesthesia was induced with 4 mg of remimazolam. Following then, systolic blood pressure dropped to 30-40 mmHg and SpO2 dropped to 73 % without any respiratory symptoms. We administrated 100 μg of noradrenaline in multiple divided doses, but hemodynamics did not change drastically. We administered 250 μg of intravenous adrenaline and 2,000 ml of crystalloid, following then his blood pressure and SpO2 returned to 115/70 mmHg and 98 %, respectively. At this point, we couldn't diagnose anaphylaxis yet because skin symptoms couldn't be confirmed due to burn injury. Anesthesia was maintained with propofol, ketamine and fentanyl instead of remimazolam during the surgery. We maintained blood pressure by continuous intravenous infusion of adrenaline. The surgery was completed, and a subsequent blood test suggested that circulatory collapse after anesthetic induction was due to anaphylaxis. Serum tryptase was elevated from baseline sample 2.9 μg/L to acute sample 8.3μg/L.

Case 2: A 59-year-old male (height, 176cm; body weight, 52kg) was scheduled for laparoscopic-assisted sigmoidectomy. Anesthesia was induced with 9 mg of remimazolam divided into three times. Following then, the patient complained of discomfort. Within a few minutes, the patient developed sinus tachycardia (105 bpm) and systolic blood pressure dropped to 30-40 mmHg without skin and respiratory symptoms. Ephedrine and phenylephrine were administered repeatedly, but hemodynamics did not change. Thus, we administered 300μg of intravenous adrenaline, following then his blood pressure returned to 85/45mmHg. We performed trans-thoracic echocardiography, but no segmental asynergy or right ventricular dilatation was observed. The operation was canceled, and subsequent blood test suggested anaphylaxis. Serum tryptase was elevated from baseline sample 4.1 μg/L to acute sample 7.8μg/L.

Conclusion: We experienced two cases of circulatory collapse suspected due to remimazolam anaphylaxis during anesthetic induction. As the prevalence of remimazoram anaphylaxis is unknown yet, further researches are needed.

References: 1. Tsurumi K, Takahashi S, Hiramoto Y, Nagumo K, Takazawa T, Kamiyama Y. Remimazolam anaphylaxis during anesthesia induction. J Anesth. 2021; 35: 571-5.

Perioperative Anesthesia - 5

Postoperative Vision Loss in a Young Patient with Ankylosing Spondylitis After Spine Surgery

Ashley V Chin[1], Ashley J So[1], Adam Dalia[1], Lucy T Li[1]

[1]Massachusetts General Hospital, Boston, MA

Introduction: Ankylosing spondylitis (AS) is an autoimmune disease characterized by systemic inflammation, primarily involving the axial skeleton. These patients commonly present for extensive corrective spine surgeries, which are associated with long operative times and high-volume estimated blood loss (EBL)[1]. Tranexamic acid (TXA) is a commonly employed antifibrinolytic to reduce intraoperative blood loss and need for postoperative transfusions[2]. However, a concern with usage of TXA is the highly debated risk of increased venous thromboembolism events (VTE), particularly in patients at risk of developing clots[3]. Additionally, patients with AS have been noted to have an increased risk of VTE and ischemic stroke[4,5].

Methods: A 41-year-old obese male (BMI 33) with AS and a 35 pack-year smoking history (quit 4 months prior to surgery) presented for T3-pelvis spinal fusion with pedicle subtraction osteotomy. Preoperative hemoglobin was 15.3, and coagulation labs were within normal limits. EBL was 3.2L and the patient was resuscitated with 2 units of RBCs, 1 unit of FFP, 5L crystalloid, and 1.75L colloid. Hemodynamic support was maintained with a phenylephrine infusion and no significant episodes of hypotension (MAP less than 60 for more than 5 mins) occurred. The patient received a TXA bolus (20mg/kg) followed by an infusion (1mg/kg/hr) for about 8 hours throughout the case. Anesthesia was maintained with propofol, remifentanil, and dexmedetomidine infusions. The patient received 250 mcg fentanyl, 1 mg hydromorphone, and 10 mg methadone for analgesia. Postop Hgb was 8.2. In the PACU, the patient reported painless, waxing and waning right-sided vision loss. A consistent neurologic exam was initially difficult to obtain due to residual anesthesia. Ophthalmology was consulted out of concern for posterior ischemic optic neuropathy and recommended a nonurgent MRI brain. Imaging revealed a left PCA territory infarction, and CTA head favored an embolic cause. Further workup during his admission revealed new bilateral axillo-subclavian deep vein thromboses on postoperative day (POD) 1. Patient was not started on VTE prophylaxis until POD3. On the evening of POD3, he had a brief desaturation and an episode of chest pain, with resultant CTPA revealing a new segmental pulmonary embolism and he was started on a heparin infusion. Initial and repeat transthoracic echocardiograms (TTE) were negative for patent foramen ovale (PFO) but were technically difficult studies. Hypercoagulability workup was remarkable for low protein S and elevated homocysteine levels, but complete workup is still pending at the time of this writing. By POD9, the patient reported improvement in his visual deficits and was discharged home on apixaban. He was scheduled for an outpatient transesophageal echocardiogram to further evaluate for presence of PFO and possible closure.

Conclusion: Although both TTEs were negative for a PFO (noting that TTE is not the gold standard for PFO detection), this patient likely suffered a paradoxical embolism. It was unlikely a watershed infarction given the lack of significant intraoperative hypotension or congruent findings on imaging. CTA was also unremarkable for significant atherosclerotic disease. Nonetheless, when caring for patients with AS in the perioperative period, it would be prudent for clinicians to consider systemic effects of this inflammatory disease - namely, the proclivity to VTE. TXA has become increasingly popular for intraoperative use when anticipating large EBL[5]. It has also been used successfully during hip surgeries in patients with AS, although these were small studies that reported using only peri-incisional TXA bolus (either 1g or 10mg/kg) without subsequent infusion and did not comment on post-operative clotting complications; one study started patients on VTE prophylaxis on POD0 [6,7]. Anesthesiologists may have to weigh the risks and benefits and dosing of TXA given the still unclear risks of VTE associated with this antifibrinolytic in patients at risk of thromboembolic events, such as those with AS and other risk factors for perioperative stroke.

References: 1. The Safety and Efficacy of Tranexamic Acid in Adult Spinal Deformity Surgery. Drugs. 2019 Oct;79(15):1679-1688. 2. The effectiveness of tranexamic acid on operative and perioperative blood loss in long-segment spinal fusions. J Neurosurg Spine. 2020 Jan 24:1-7. 3. Effect of tranexamic acid on surgical bleeding. BMJ. 2012;344:e3054. 4. Ankylosing spondylitis and risk of venous thromboembolism. Lung India. 2016;33(6):642-645. 5. Increased risk of stroke among patients with ankylosing spondylitis. Rheumatol Int. 2014 Feb;34(2):255-63. 6. Clinical curative effect of tranexamic acid on perioperative blood loss in primary total hip arthroplasty among patients with ankylosis spondylitis. International Journal of Surgery ; (12): 437-442, 2018. 7.Factors associated with blood loss in ankylosing spondylitis patients with hip involvement undergoing primary total hip arthroplasty: a cross-sectional retrospective study of 243 patients. J Orthop Surg Res 15, 541 (2020).

Perioperative Anesthesia - 6 An analysis of 4 cases of perioperative anaphylaxis: revisiting an old issue to improve perioperative care

Anna Budde[1], Ratan K. Banik[2]

[1]University of Minnesota, Minneapolis, MN, [2]university of minnesota, Minneapolis, United States of America

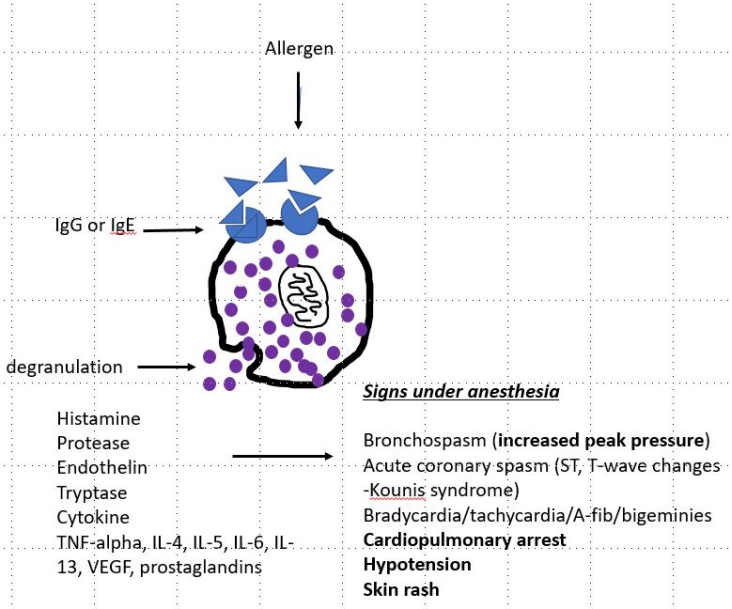
Introduction: Perioperative anaphylaxis is rare with a reported incidence of 1:6,000–1:20,000. A low number of complications makes it difficult to estimate the true incidence in a prospective study. Moreover, when it happens, anesthesiologists find it difficult to diagnose the condition due to its variable presentation or coexisting hypotension from anesthetics leading to suboptimal perioperative care. It is therefore imperative that all suspected cases of anaphylaxis are reported so that anesthesiologists can develop strategies to prevent and improve perioperative care of this rare complication.

Methods: Case series: Case 1: 71-year-old with a medical history of end-stage renal disease presented for deceased donor kidney transplantation. Induction of anesthesia was achieved with lidocaine, fentanyl, propofol, rocuronium, and sevoflurane. During arterial line placement, the patient became mildly hypotensive, which was briskly responsive to phenylephrine boluses. Next, a latex Foley was placed and cefuroxime given. At the time the patient became progressively hypotensive and unresponsive to both phenylephrine and ephedrine boluses. The patient's blood pressure dropped to 45/30 mm Hg and sevoflurane was turned off. The patient was then noted to have an enlarged tongue, raising concern for anaphylaxis. A tryptase level was elevated at 78 mcg/L (reference range <11mcg/L). Case 2: 53-year-old presented for left orbitotomy and underwent uneventful induction and intubation. A few minutes after administration of cefazolin, the patient's oxygen saturation gradually dropped to the low 90's with subsequent hypotension to the 60's. Albuterol, phenylephrine, ephedrine, and norepinephrine were administered and an arterial line was placed. Considering his rapid changes of hemodynamics after cefazolin administration, the presumptive diagnosis was anaphylaxis and therefore epinephrine, diphenhydramine were given. A tryptase level was elevated at 46 mcg/L. Case 3: 50 year old presented for posterior C6-7 laminectomy and placement of spinal cord stimulator. Postintubation the patient was hemodynamically stable but developed profound hypotension 5 minutes after administration of cefazolin and prone positioning requiring 1000mcg of phenylephrine and 30mg of ephedrine. Blood pressures stabilized with MAP>65, but the patient developed bronchospasm with peak pressures in the 70's requiring albuterol administration which resolved the bronchospasm. A second episode of bronchospasm occurred 10 minutes after the first episode and required position change to supine at which time a red rash over the patient's trunk was observed. Epinephrine was administered and the patient's rash and bronchospasm resolved. An arterial line and central line were placed, the procedure was aborted, and the patient was transferred to the ICU intubated with an epinephrine infusion for further care. A tryptase was not sent. Case 4: 73 year old with medical history of bladder cancer presenting for cystoscopy. After uneventful induction of anesthesia with placement of supraglottic airway, cefazolin was administered and immediately the patient developed hypoxia with oxygen saturation in the 70's and was impossible to ventilate through the supraglottic airway which had been previously well seated. The patient became bradycardic and hypotensive and epinephrine was administered for presumed anaphylaxis. The patient was intubated and transferred to ICU with an epinephrine infusion. A tryptase level was elevated at 21.5 mcg/L

Conclusion: An analysis of this case series show that despite the myriad of published cases, the diagnosis of anaphylaxis in all four cases was delayed. We hypothesize that the delay in diagnosis and treatment of periopherative anaphylaxis is due to variability in presentation and alternative explanations of symptoms under general anesthesia. Based on our analyses, we recommend consideration of this diagnosis in patients with refractory hypotension and bronchospasm which were common features in all four cases. We recommend epinephrine diluted to 10mcg/mL be available to treat anaphylaxis and tryptase levels be sent to confirm the diagnosis in suspected cases . Although perioperative anaphylaxis is rare, patients whose treatment is delayed are more likely to have life-

threatening complications and if this diagnosis is not made patients are at risk of exposure to the offending allergen in the future.

References: 1. Passia, E., & Jandus, P. (2020). Using baseline and peak serum tryptase levels to diagnose anaphylaxis: a review. Clinical reviews in allergy & immunology, 58(3), 366-376. 2. Takazawa, T., Yamaura, K., Hara, T., Yorozu, T., Mitsuhata, H., & Morimatsu, H. (2021). Practical guidelines for the response to perioperative anaphylaxis. Journal of Anesthesia, 1-16. 3. Hepner, D. L., & Castells, M. C. (2003). Anaphylaxis during the perioperative period. Anesthesia & Analgesia, 97(5), 1381-1395. 4. Brown, S. G. (2007). The pathophysiology of shock in anaphylaxis. Immunology and allergy clinics of North America, 27(2), 165-175. 5. Volcheck, G. W., & Hepner, D. L. (2019). Identification and management of perioperative anaphylaxis. The Journal of Allergy and Clinical Immunology: In Practice, 7(7), 2134-2142.



Perioperative Anesthesia - 7

Perioperative morbidity within the incubation period of Covid-19 infection

Alexa Christophides[1], Viktorya Poghosyan[1], Vahe Tateosian[1]

[1]Stony Brook University, Stony Brook, NY

Introduction: The outbreak of the 2019 SARS-CoV-2 lead to many issues in perioperative healthcare delivery due to its novelty and frequent changes in guidance. The most common symptoms of SARS-CoV-2 patients are fever, fatigue, cough, and expectoration (1). In the perioperative setting, providers were faced with optimum timing of accurate polymerase chain reaction (PCR) testing related to surgical procedures. Complications may arise from patients undergoing surgical intervention during the incubation period of the disease (2). The course of the SARS-CoV-2 disease is lengthy and highly contagious even during the incubation period (3). We present the case of a 58 year old man who underwent elective surgery during the incubation period of SARS-CoV-2 infection and the sequelae that arose postoperatively.

Methods: A 58 year old male presented for a left hemithyroidectomy; possible total thyroidectomy. He had a history of a multinodular goiter, left parotid mass, hypertension, obesity, and obstructive sleep apnea. He denied a history of respiratory or cardiac issues, and had no known history of prominent liver, kidney, or cerebrovascular disease. He had a 30 pack year history of smoking. He had a negative COVID-19 PCR test result 5 days prior to his scheduled surgery. Within the 5 days prior to surgery, he reported no fever, cough, or COVID exposure. Surgery was performed and anesthesia was maintained without notable complications. The surgery duration 203 minutes and emergence lasted 35 minutes. He was extubated with spontaneous ventilation and transported to PACU with a non-rebreather 10 L/min. In PACU, he was noted to be lethargic yet arousable to verbal stimulation. Upon auscultation, he had bibasilar, coarse breath sounds. A nebulizer treatment of Albuterol was administered with minimal change. Five hours postoperatively, he reported numbness, paresthesias, and had weakness in the right upper and lower extremities. Stroke team was mobilized and he was taken for brain imaging. Results showed no acute infarct or intracranial hemorrhage. He was admitted to post-surgical ward. On POD-1, the unilateral weakness persisted, and he was taken for a brain MRI. Findings included bilateral globus pallidus of flair hyperintensity with subtle DWI hyperintensity and ADC subtle hypointensity-isotensity. The findings were nonspecific and possibly represent carbon monoxide poisoning. After clinical correlation, it was determined the patient sustained an acute ischemic stroke. POD-3, the patient became febrile to 39.1 deg C; repeat COVID test was administered, and was positive. Chest x-ray was obtained; no evidence of pneumonia. On POD-5, chest X-ray showed evidence of diffuse airspace opacities compatible with multifocal pneumonia; The patient had increasing oxygen requirements and was receiving Remdesivir and Dexamethasone. POD-6; he had acute hypoxemic respiratory failure and was intubated. After a complicated course of multiple extubations and reintubations, he was discharged on POD-50 with persistent respiratory symptoms such as shortness of breath on minimal exertion and chronic renal insufficiency.

Conclusion: Patients undergoing surgery during the incubation period have developed sequelae of COVID-19, with many needing to be admitted to ICU (2). In addition to respiratory symptoms, there are alterations of consciousness, abnormal wakefulness after sedation, confusion, and agitation. There are many prominent neuroimaging findings with severe SARS-CoV-19 disease (4). Perioperative risks are increased in patients with persistent symptoms of COVID-19 compared with those who have been asymptomatic or those in whom symptoms have fully resolved at the time of surgery (5). The most common complications in non-survivors include ARDS, shock, arrhythmia, and acute cardiac injury (2). Timing of surgery is complex and must account for multifactorial considerations including urgency and SARS-CoV-2 status. Current data suggests that after SARS-CoV-02 infection, the majority of patients who have no symptoms or whose symptoms have resolved should have surgery scheduled at least 7 weeks after diagnosis, unless clinical urgency and risk of disease progression outweigh the risks of delayed procedures. For patients with persisting symptoms or who have more severe SARS-CoV-2, waiting beyond 7 weeks may be beneficial and a personalized multidisciplinary perioperative care plan is recommended (5).

References: 1. Clinical characteristics of 3062 COVID-19 patients: A meta-analysis. Journal of medical virology, 92(10), 1902–1914 2. Clinical characteristics and outcomes of patients undergoing surgeries during the incubation period of COVID-19 infection. EClinicalMedicine, 21, 100331 3. A Familial Cluster of Infection Associated With the 2019 Novel Coronavirus Indicating Possible Person-to-Person Transmission During the Incubation Period, The Journal of Infectious Diseases, Volume 221, Issue 11, 1 June 2020, Pages 1757–1761 4. Brain MRI Findings in Severe COVID-19: A Retrospective Observational Study. Radiology, 297(2), E242–E251 5. SARS-CoV-2 infection, COVID-19 and timing of elective surgery: A multidisciplinary consensus statement on behalf of the Association of Anaesthetists, the Centre for Peri-operative Care, the Federation of Surgical Specialty Associations, the Royal College of Anaesthetists and the Royal College of Surgeons of England. Anaesthesia, 76(7), 940–946

Perioperative Anesthesia - 8 Anesthetic Management Of A Patient With Brugada Syndrome Who Underwent Shoulder Arthroscopic Surgery: A Case Report

Zhaoyi Tang[1], Roy Mongkolpradit[1], Jessie Lo[1], Henry Chou[1], Jun Tang[1]

[1]Cedars-Sinai Medical Center, Los Angeles, CA

Introduction: Brugada Syndrome (BrS) is a fatal cardiac channelopathy characterized by electrocardiographic (ECG) changes (ST elevations in V1-V3, right ventricular conduction delay, normal QT interval) in a structurally normal heart, predisposing patients to malignant ventricular arrhythmias and sudden cardiac death (1). BrS is a rare (0.05% prevalence) (2) inherited electrophysiological disorder, with approximately 20% of cases linked to mutations of the cardiac sodium channel gene (SCN5A) (3). Currently, ICD placement is the only available supportive therapy. Anesthetic and vasoactive drug administration along with the physiologic changes associated with anesthesia and surgery may interact with cardiac ion channels, increasing the risk of malignant ventricular arrhythmias. Recommendations have been made concerning drugs to avoid and preferably avoid (4,5). Due to its rare disease, controlled-trial study is not feasible and practicable. Here we present a case report of anesthesia management for a patient with BrS underwent shoulder arthroscopic procedure.

Methods: A 61-year-old male with BMI 26.2 (73.6 kg, 167.6 cm) was scheduled for left shoulder arthroscopic subacromial decompression secondary to left shoulder impingement syndrome. His past medical history included coronary atherosclerosis, Brugada Syndrome and Crohn disease. BrS was diagnosed through ECG 3 years ago (patient denied a history of syncope, palpitations, and family history of sudden premature cardiac death). Recent stress echo was normal, and no significant arrhythmias were detected on 1-month cardiac monitor. No ICD was placed. After discussion with the patient, surgeon and cardiologist, ASA standard monitors, A-line, 5-lead ECG, and a cardiac defibrillator pads were placed in the operating room. Isoproterenol drip was prepared prior to the case. Patient was premedicated with midazolam 2 mg IV and glycopyrrolate 0.2 mg IV. Induction consisted of sevoflurane (5-6%), fentanyl 150 mcg IV and rocuronium 50 mg IV. Airway was secured with ETT 7.0 mm assisted by video laryngoscopy. Anesthesia was maintained with sevoflurane (1.5-2.0%) in an air/oxygen mixture (50:50). A total dose of fentanyl 250 mcg IV and hydromorphone 1 mg IV was administered throughout the case. Surgical procedure (40 min) and anesthesia procedure (98 min) were uneventful. At the end of surgery, muscular reversal (sugammedex 200 mg IV) was administered. After extubation, patient was transferred to the postanesthesia care unit (PACU) where he was monitored for 4 hrs and 4 min before transfer to cardiac monitored floor bed. Patient was uneventfully discharged from hospital the next day.

Conclusion: As Brugada Syndrome is being increasingly recognized, more patients with this syndrome will present for ICD implantation and other noncardiac surgery. Even asymptomatic patients with abnormal ECG only induced by a drug challenge will have a more benign prognosis (6), the asymptomatic patients with the ECG findings of BrS at baseline (like our patient) carry the same rate of recurrent life-threatening arrhythmias as symptomatic BrS patients (30% within 3 years) (7-9). In conclusion, although there are still many unanswered questions regarding the best approach to these cases, understanding of potential triggers is vital to prevent serious complications or even death, such as (a) anesthetic considerations including the avoidance of excessive parasympathetic tone, and the avoidance of specific triggering agents (e.g., bupivacaine, beta-blocking agents, alpha-receptor agonists), (b) precautions in the operating room (e.g., external defibrillator, continuous ECG monitoring of right precordial ST segments, isoproterenol, surgical techniques) (10). In addition, close communication among the patient, surgeon, cardiologist and anesthesiologist is also very important to smoothly and successfully take care of BrS patient undergoing surgical procedure.

References: 1. Circ J 2012;76:1563–71. 2. Acta Cardiol Sin 2018;34:267–77. 3. Curr Opin Cardiol 2002;17:19–23. 4. Can J Anaesth 2011;58:824–36. 5. Heart Rhythm 2009;6:1335–41. 6. Circulation 2002;105:73–8. 7. Cardiovasc Res 1999;44:507–17. 8. Circulation 1998;97:457–60. 9. Cardiac Electrol Rev 2002;6:45–8. 10. J Clin Anesth 2017;36:168-73.

Perioperative Anesthesia – 9

Intraoperative Abdominal Compartment Syndrome as a Complication of Percutaneous Nephrolithotomy

Mark Abramowitz[1], Sahajahan Ashraf[1], Robert Zusman[2], Hector Davila[2]

[1]NSU-KPCOM, Davie, FL, [2]Mount Sinai Medical Center of Florida, Miami, FL

Introduction: Abdominal Compartment Syndrome (ACS) is defined as a sustained intraabdominal pressure (IAP) greater than 20 mmHg with or without an abdominal perfusion pressure of <60 mmHg, associated with new organ dysfunction or failure. The most common causes of ACS include abdominal surgery or trauma, severe hemorrhagic shock, severe burns, severe acute pancreatitis, large volume fluid resuscitation, ileus, and liver dysfunction. It is a condition that frequently requires early surgical or interventional radiological intervention to prevent end organ damage. Clinical signs and symptoms are more useful for diagnosing ACS than radiographs. Risk factors for the development of ACS include reduced abdominal wall compliance (obesity, ascites, fluid overload), increased intra-luminal or extra-luminal contents, capillary leak, and aggressive fluid resuscitation. ACS can cause ischemic damage to most organ systems leading to a variety of issues such as decreased cardiac output, increased peak airway pressures, hypoxemia, decreased cerebral perfusion pressure, and decreased blood flow to the gastric, hepatic, and renal systems. Percutaneous nephrolithotomy (PCNL) is a common treatment for large kidney and ureteral stones that cannot be passed. Complications most commonly present as fever and bleeding, but rarely can cause ACS due to intraperitoneal extravasation of irrigation fluid. It is important for anesthesiologists to recognize ACS as a cause of increased peak inspiratory pressure and possible hemodynamic compromise.

Methods: We present a 70-year-old male with a past medical history significant for hypertension, coronary artery disease, diabetes mellitus type 2, myelofibrosis, and recurrent nephrolithiasis that presented for percutaneous nephrolithotomy under general anesthesia. The patient was intubated and placed in the prone position. Approximately one hour into the procedure, the peak inspiratory pressures began to gradually rise, and we were unable to achieve the targeted tidal volumes. We checked the depth of the endotracheal tube and the pilot balloon, the breathing circuit, and suctioned the endotracheal tube without improvement. Auscultation revealed clear breath sounds bilaterally. Peak inspiratory pressures continued to rise to > 40 cm H2O. Oxygen saturation remained > 98% throughout. Arterial blood gas showed a respiratory acidosis with a pCO2 of 71 mmHg and a pH of 7.13. At this point, a multidisciplinary decision was made to abort the procedure. Upon repositioning of the patient to the supine position, physical exam revealed the abdomen was significantly distended and hard to palpation. Limited ultrasound was performed by radiology which showed a mixture of heterogenous fluid, but no specific fluid collection. General surgery performed exploratory laparotomy with drainage of approximately 3 liters of serous fluid. After drainage of fluid, there was an immediate improvement in peak inspiratory pressures. Subsequent arterial blood gas revealed resolution of respiratory acidosis. The patient remained intubated and was transported to the surgical intensive care unit in stable fashion.

Conclusion: Abdominal compartment syndrome is rarely described in the literature as an intraoperative complication of PCNL. However, prompt recognition of ACS is vital to prevent multiorgan failure. In the intraoperative setting, ACS can present with increased peak inspiratory pressures and difficulty in achieving adequate tidal volumes due to diaphragmatic elevation and increased intrathoracic pressure, subsequently leading to decreased venous return and cardiac output. It is important to have a high index of suspicion to promptly identify ACS. In this case, the procedure was quickly aborted, the patient was repositioned, and an urgent decompressive laparotomy was performed with immediate improvement. PCNL is commonly performed in the prone position, but studies have shown that it is safe with the patient in the supine position (5). ACS may be recognized more quickly in the supine patient who presents with a distended and firm abdomen.

References: 1) H. S. Ween and T. J. Florence, “The diagnosis of hydro-nephrosis by percutaneous renal punctures,” Journal ofUrology, vol. 72, no. 2, pp. 589–595, 1954. 2) Kimball EJ, Kim W, Cheatham ML,Malbrain ML (2007) Clinical awareness of intra-abdominal hypertension and abdominal compartment syndrome in 2007. Acta Clin Belg (Suppl): 66–73 3) Malbrain ML, Cheatham ML (2011) Definitions and pathophysiological implications of intra-abdominal hypertension and abdominal compartment syndrome. Am Surg 77(Suppl 1):6–11 4) Papavramidis T, Marinis A, Pliakos I, Kesisoglou I, Papavramidou N. Abdominal compartment syndrome–Intra-abdominal hypertension: defining, diagnosing, and managing. J Emergencies, Trauma Shock. 2011;4(2):279. 5) Giuseppe Giusti, Antonello De Lisa, "Supine Percutaneous Nephrolithotripsy in Double-S Position", Advances in Urology, vol. 2018, Article ID 7193843, 4 pages, 2018.

Perioperative Anesthesia - 10 Cefazolin Anaphylaxis In Previously Sensitized Patient

Lindsay D Nowak[1], Adam Wacher[1]

[1]University of Texas Medical Branch, Galveston, TX

Introduction: This case report is centered upon two objectives: - Recognizing how intraop anaphylaxis presents (including cases of allergy sensitization leading to anaphylaxis), how to confirm the diagnosis, and how to treat the anaphylactic reaction. - Learning about the crossreactivity of cefazolin and pencillins (antibiotics commonly given for surgical site infection prophylaxis) and what options there are when patients are allergic to either cefazolin or penicillin.

Methods: 43 year old male with a LUE AV graft infection and scheduled for LUE stent removal + possible L clavicle resection, who then pre-incision experienced cefazolin sensitization leading to anaphylaxis and intraop code. The presenting symptoms included bradycardia, hypotension, bronchospasm, and decreased cardiac output. He had received cefazolin without issue at least four times previously, including once this hospital admission for a different surgery. Tryptase (sent within 2 hours of the possible anaphylactic event) later came back very elevated, further supporting the anaphylaxis diagnosis, and the causative med was able to be identified given the intraop timeline.

Conclusion: This case reminds anesthesiologists to be aware of allergy sensitization--especially with common meds given every day in the OR--as well as to recognize anaphylaxis in an already intubated patient. Upon lit review, very few cases of cefazolin anaphylaxis in a previously sensitized patient have been published.

Perioperative Anesthesia - 11 Optimizing the Anesthetic Management of a Patient with Active DNR/DNI Orders through the Preoperative Discussion of Physician Orders for Life-Sustaining Treatment (POLST)

Tomás A Lazo

[1]Oregon Health & Sciences University, Portland, OR

Introduction: Do-not-resuscitate (DNR) and do-not-intubate (DNI) orders have been established in the clinical setting to provide a mechanism for withholding or limiting specific resuscitative therapies to be consistent with a patient's goals of care. However, for patients undergoing anesthesia for surgical procedures, the DNR/DNI status raises a perplexing ethical dilemma, as anesthetic care necessitates the provision of resuscitative measures such as intubation, mechanical ventilation, and potential administration of vasoactive medications. While approximately 15% of patients with an active DNR/DNI will present for surgery, anesthesiologists do not always feel comfortable addressing operative resuscitation requests during their pre-operative consent discussion. In this case, we describe the delivery of a non-standard anesthetic for ureteral stent placement in a patient with an active DNR/DNI order. We used this case to frame a discussion on the importance of patient self-determination and the importance of anesthesia providers adjusting perioperative plans for POLST and procedure-specific needs. This is especially relevant in terminally ill patients who may need palliative surgical interventions to relieve pain or facilitate care.

Methods: This is a case of an 80-year old woman with chronic diastolic heart failure, chronic lymphocytic leukemia, autoimmune hemolytic anemia, MDS, cryptogenic cirrhosis complicated by ascites, and hepatic encephalopathy, who was admitted to the ICU for septic shock secondary to a urinary tract infection. She was indicated for urgent ureteral stent placement for hydronephrosis of the left ureter. This patient was on multiple infusions, including vasopressin and norepinephrine for blood pressure support, sodium bicarbonate, as well as dexmedetomidine to facilitate sedation due to agitation and altered mental status. Prior to proceeding to the operating room, various team members met to discuss how to best proceed with this case. This included the attending anesthesiologist, CRNA, patient's healthcare surrogate decision-maker (spouse), and the surgeon. Since the standard of care for cystoscopy procedures is general anesthesia with airway management, this opposed the patient's active DNI order and POLST. Additionally, timely extubation post-operatively would be challenging, given her altered mental status and pressor requirements. Thus, the anesthesiologist spent time thoroughly going over a variety of options and scenarios of how to best proceed. Ultimately, the patient's spouse decided they wished to avoid intubation and agreed to the option of sedation in addition to local anesthesia. In addition, this discussion included additional clarifications for patient care, noting that the patient would not want intubation, chest compressions, or defibrillation as resuscitative measures in the operating room or postoperative period. The patient was transported to the operating room by the anesthesia team and the plan was clearly communicated during team timeout. We used standard ASA monitors and placed an arterial line prior to the procedure's start. Oxygen was provided via a face mask at 8 L/min. The patient was placed in a cystolithotomy position. The dexmedetomidine infusion was increased to to 0.8 mcg/kg/hr and remifentanil was added to enhance sedation at 0.05 mcg/kg/min. Local anesthesia was used by the surgical team in the form of a lidocaine-laden lubricant for passage of the cystoscope. The patient continued to required norepinephrine, up to 0.16 mcg/kg/min and vasopressin at 0.03 Units/min. Surgery proceeded uneventfully with successful placement of a left ureteral stent and drainage of purulent urine and the patient was brought back to the ICU for continued care.

Conclusion: We acknowledge that challenges may arise regarding patient autonomy versus standard of care practices. In addition, it is not necessary to suspend DNR/DNI orders in the perioperative period. Forcing patients or surrogate decision-makers to suspend DNR/DNI is coercive and disallows patient self-determination. Thus, we recommend having an open discussion with all involved members of the team and documenting specifics regarding resuscitation measures and goals of care. Each patient's unique circumstance warrants a customized approach to such discussion, and this case report demonstrates the

impact of a thorough goals-of-care discussion in a patient's care quality in the perioperative setting.

References: Waisel DB, Simon R, Truog RD, Baboolal H, Raemer DB. Anesthesiologist management of perioperative do-not-resuscitate orders: a simulation-based experiment. Simul Healthc. 2009 Summer;4(2):70-76. Burkle CM, Swetz KM, Armstrong MH, Keegan MT. Patient and doctor attitudes and beliefs concerning perioperative do not resuscitate orders: anesthesiologists' growing compliance with patient autonomy and self determination guidelines. BMC Anesthesiol. 2013 Jan 15;13:2. Cohen CB, Cohen PJ. Required reconsideration of "Do-Not-Resuscitate" orders in the operating room and certain other treatment settings. Law Med Health Care. 1992;20(4):354-363.

Perioperative Anesthesia - 12 Bad Case of NPPE or Some Other Acute Respiratory Pathology: A Peri Operative Complication During the Time of COVID

Matthew Cucino[1], Gaurav Budrani[1]

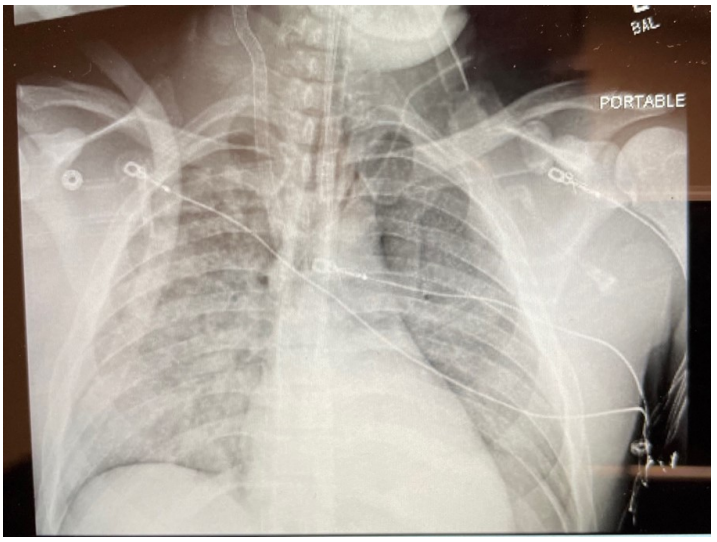
[1]Emory University, Atlanta, GA

Introduction: Pulmonary complications are one significant contributor to perioperative morbidity and mortality. The incidence of pulmonary complications varies depending upon the patient's risk factors and includes type of surgery and any other definition of complications. Postoperative respiratory failure comprised greater than 20% of all patients on ventilators pre-COVID (1, 2). Post-op respiratory failure requiring unplanned reintubation is associated with higher morbidity, longer hospital stays, and most importantly 30-day mortality (3-5). In the case presented today the primary diagnosis was negative pressure pulmonary edema (NPPE) as well as aspiration pneumonitis leading to ARDS. NPPE is a form of noncardiogenic pulmonary edema that results from the negative intrathoracic pressure generated by an attempted inhalation against an upper airway obstruction, acidic aspirate from the gastrointestinal tract, and uncontrolled inflammation of the pulmonary tissue from an immune response, respectively . Although each of these incidents may result in pulmonary failure, the combination of them lead to pulmonary failure refractory to traditional rescue therapies.

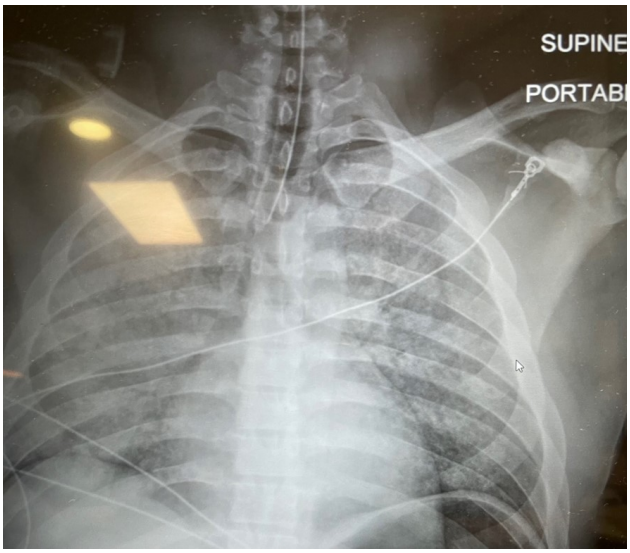
Methods: The patient is a 29 year old male with PMH of marijuana use and obesity who presented to a local hospital on the day for surgery for patellar tendon repair. The patient had a benign pre-operative assessment, with a reassuring airway exam. His ASA physical status was 2. The patient had a benign intra operative course with an uneventful induction of anesthesia. He was an easy mask and easy indirect laryngoscopy. Intraoperative medications included 250mcg of fentanyl in addition to 4mg of iv hydromorphone throughout the case. Chart review indicates that the patient met extubation criteria and he was brought to PACU (Post-anesthesia care unit) on facemask. Upon arrival, the patient was noted to be apneic. Naloxone 0.4mg IV push was administered. However, he continued to experience hypoxic respiratory failure and required emergent airway management. Both mask ventilation and endotracheal intubation were difficult. Opening the mouth resulted in a fountain of red pulmonary edema-like fluid releasing, splashing nearby anesthesia providers. After multiple attempts at intubation, the airway was secured by the attending anesthesiologist. However, oxygenation was challenging, with pulse oximeter readings as low as 30% for a prolonged period of time. Consults to the ICU were in place for further management of negative pressure pulmonary edema (NPPE) and suspected aspiration pneumonitis. It came to a point that advanced ventilator settings including IRV 2:1 with mean airway pressures in the 40s did not provide adequate oxygenation. CXR in PACU revealed bilateral patchy opacities, consistent with bilateral pulmonary edema and ARDS. One ABG result was 7.07/51/44 (pH/pO2/pCO2). This was on Inverse Ratio ventilation with 2:1 I:E. The ECMO team (extracorporeal membrane oxygenation) was mobilized and cannulation successful. The process however required hemodynamic support on 3 vasopressors with multiple near cardiac arrests. Post-cannulation CXR revealed more dense consolidations as the effects of aspiration set in. He was transferred to the academic center, started on angiotensin II for severe vasoplegia. He required a total of 4 days on VV ECMO and was discharged on POD 7.

Conclusion: •In the otherwise healthy patient ECMO should be considered a last line therapy for patients with severe NPPE, aspiration pneumonitis, ARDS or any combo of the above, with resulting hypoxia or hypercarbia refractory to maximal ventilatory support
•VV ECMO, although not first line should be considered a rescue therapy for those patients with an otherwise good prognosis that are suffering acutely from respiratory failure.
•In this case VV ECMO was successfully able to rescue a patient with both NPPE and ARDS secondary to a fulminant aspiration event leading to pneumonitis.
•As more centers begin to offer ECMO services, the role that ECMO plays in the post operative period may continue to grow for the specific substrate of healthy patients that suffer a devastating acute, but reversible pulmonary insult
•The time from consideration of ECMO to initiation of ECMO may be critical during these emergent

situations, so appropriate planning will be necessary. Early consultation with a critical care specialist and the ECMO team is recommended.



References: 1 Esteban A, Frutos-Vivar F, Muriel A, et al. Evolution of mortality over time in patients receiving mechanical ventilation. Am J Respir Crit Care Med 2013; 188:220. 2 Sun Z, Sessler DI, Dalton JE, et al. Postoperative Hypoxemia Is Common and Persistent: A Prospective Blinded Observational Study. Anesth Analg 2015; 121:709. 3 Nafiu OO, Ramachandran SK, Ackwerh R, et al. Factors associated with and consequences of unplanned post-operative intubation in elderly vascular and general surgery patients. Eur J Anaesthesiol 2011; 28:220. 4 Ramachandran SK, Nafiu OO, Ghaferi A, et al. Independent predictors and outcomes of unanticipated early postoperative tracheal intubation after nonemergent, noncardiac surgery. Anesthesiology 2011; 115:44. 5 Brueckmann B, Villa-Uribe JL, Bateman BT, et al. Development and validation of a score for prediction of postoperative respiratory complications. Anesthesiology 2013; 118:1276.



Perioperative Anesthesia - 13 The "Highs" and Lows of Chronic Marijuana Use: A Case of Impressive Intraoperative Hemodynamic Lability

Kelley Martin[1], Jeffrey D White[1]

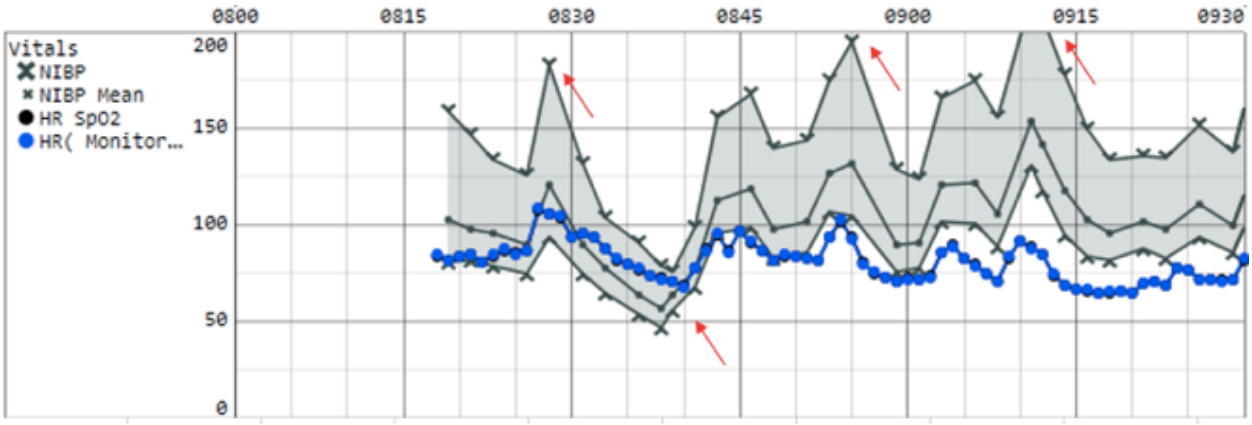
[1]University of Florida, Gainesville, FL

Introduction: Marijuana (MJ) is the most commonly used illicit drug in the United States [1]. Its use has been shown to affect significantly patient hemodynamics under anesthesia. This case report features the anesthetic course of a frequent MJ user.

Methods: A 56 yr old, 76 kg female patient presented to our ambulatory surgical center for elective ureteroscopy with stent placement. PMH included: recurrent nephrolithiasis, hypertension (controlled with losartan-HCTZ), COPD, DM2 and anxiety disorder. She endorsed regular MJ use, including that morning. During informed consent, she was educated on risks associated with general anesthesia for chronic MJ users, especially when not abstinent for > 72 hours. A mutual decision was made to proceed. The patient was hypertensive (168/72) in the pre-operative area and operating room prior to induction. Anesthesia induction was 2 mg/kg Propofol, 50 mcg Fentanyl and 50 mg Rocuronium, and after intubation she was maintained with Sevoflurane in 40% Oxygen. Post-induction and pre-incision her blood pressure (BP) declined to 76/56. She received phenylephrine followed by ephedrine while Sevoflurane was decreased. Her BP responded appropriately yet briefly. Fluctuating BPs continued, with maximum BP climbing to 218/131 without clear provocation. Fentanyl and labetalol were given along with frequent adjustments to Sevoflurane concentration. In PACU, the blood pressure remained labile for 45 minutes. Meanwhile her neurologic emergence from general anesthesia was smooth, and she was discharged home.

Conclusion: This patient illustrates the profound hemodynamic lability that can occur in MJ users. MJ use can cause multiple deleterious end-organ effects including bronchoconstriction, myocardial and cerebral ischemia, significant arrhythmias, coagulopathy, hypothermia, and even paradoxically a cannabinoid-related hyperemesis syndrome or increased postoperative pain [1,2]. BP and heart rate lability with orthostasis can occur due to MJ-induced alterations in the normal balance of sympathetic and parasympathetic tone. Norepinephrine levels are elevated for up to 2 hours after acute MJ use [1]. Cytochrome P450 enzymes, especially CYP3A4, can be inhibited by MJ components [2,3]. Fortunately for this patient, she only developed manageable BP lability that caused no ill effects. The increasing legalization and use of MJ raise the importance of recognizing its acute and long-term physiologic effects on the perioperative course. Anesthesia providers should ideally require >72 hours of MJ abstinence before proceeding with elective surgery [1,3].

References: Journal of Clinical Anesthesia; 57, 41–49, 2019 Cannabis and Cannabinoid Research; 4, 219–230, 2019 AANA journal; 87, 451-458, 2019


0800
0815
0830
0845
0900
0915
0930
Vitals
NIBP
NIBP Mean
HR SpO2
HR(Monitor…
200
150
100
50
0

Perioperative Anesthesia - 14 Chronic High-Dose Marijuana Use in a Patient Undergoing Cervical Spine Surgery with Neuromonitoring: Implications for Anesthetic Management

Tim L Morgan[1], Raphael A Bertasi[1], Tais G Bertasi[2], Chris W Fox[1], Shaun E Gruenbaum[3], Kevin T Riutort[1], Benjamin F Gruenbaum[1], Bruce J Leone[1]

[1]Mayo Clinic, Jacksonville, FL, [2]Mayo Clinic, Jacksonville , FL, [3]Mayo Clinic College of Medicine, Jacksonville, FL

Introduction: Marijuana use in the United States has recently increased in prevalence following its decriminalization, legalization for medical use, and legalization for recreational use in many states. With the current opioid crisis, patients and their providers are increasingly looking to alternative therapies such as marijuana to manage their chronic pain. As chronic and heavy marijuana users are more commonly presenting for elective surgery, its effects on anesthesia have recently been questioned. However, for a variety of reasons patients often fail to disclose their marijuana use to their anesthesia provider. Here, we describe the anesthetic management of a patient with chronic high-dose marijuana use who underwent cervical spine surgery under general anesthesia.

Methods: A 41-year-old man with an undisclosed history of heavy chronic marijuana presented for an elective C5-C6, C6-7 anterior cervical discectomy and fusion with intraoperative neuromonitoring (motor-evoked and somatosensory evoked potentials). The patient required unexpectedly high doses for induction and maintenance of general anesthesia and akinesis, prompting a urinary drug screen that was positive for tetrahydrocannabinol (THC), the main psychoactive compound in cannabis. In the post anesthesia care unit, the patient disclosed high-dose chronic cannabis use in the form of vaping and ingestion.

Conclusion: In this report, we review the current literature regarding the anesthetic implications of chronic and heavy marijuana use in patients presenting for surgery. With the current opioid crisis, patients and their prescribers are more commonly looking to non-opioid alternative therapies to manage chronic pain. In the United States, marijuana is being used with increasing prevalence to manage chronic pain, as well as for recreational use. In patients presenting for surgery, chronic and heavy marijuana use can significantly increase the anesthetic requirements, which can greatly impact the anesthetic management in patients in whom akinesis is required but muscle relaxation is contraindicated (e.g., when neuromonitoring with motor-evoked potentials is employed). We strongly recommend that the frequency and dose of marijuana use should be inquired from every patient presenting for elective surgery, especially when neuromonitoring is employed.

References: Not Applicable - Medically Challenging Case Report

Perioperative Anesthesia – 15

Undiagnosed Restrictive Cardiomyopathy Discovered Intraoperatively During Resection of Large Adrenal Tumor

Jessica Lowe[1], Jaffer Odeh[1], Ebin Neduvelil[1], Sagar Patel[1]

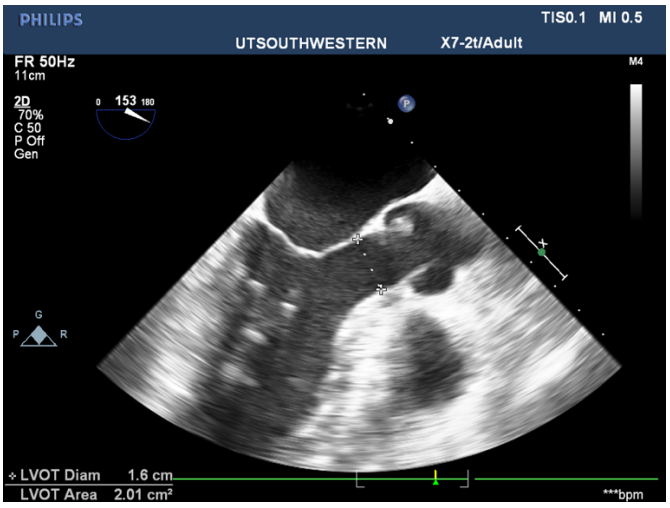
[1]University of Texas Southwestern Medical Center, Dallas, TX

Introduction: We report a case of a 45-year-old female with a history of Cushing syndrome and secondary hypertension who initially presented with generalized swelling. She was incidentally found to have a large right adrenal mass, later diagnosed as stage IV adrenocortical carcinoma with mass effect and invasion into the right hepatic lobe along with presence of an IVC thrombus. The oncological plan consisted of upfront surgical resection and thrombectomy prior to initiating chemotherapy. We report the anesthetic findings in this abstract.

Methods: The patient was intubated uneventfully and an internal jugular Multi-Lumen Access Catheter (Teleflex®), arterial catheter, and a rapid infusion catheter were placed. Intraoperative transesophageal echocardiogram (TEE) monitoring was performed to monitor the IVC thrombus and assess cardiac function.

TEE monitoring during the case demonstrated diastolic dysfunction with a restrictive filling pattern, which was not discovered on the preoperative transthoracic echocardiogram. Hemodynamic parameters were significant for low stroke volume and abnormally high systemic vascular resistance. This case necessitated massive resuscitation during the large en bloc resection (right hepatectomy, right adrenalectomy, right nephrectomy, and IVC thrombectomy and resection with primary reconstruction). The restrictive cardiomyopathy posed intraoperative concerns given the large amount of volume required to adequately resuscitate the patient. Given this finding along with a low-to-normal cardiac output, a low-dose infusion of epinephrine was started for inotropic support. Milrinone may have been a better option as it would provide lusitropy, but not chosen considering the large hemodynamic swings expected in this operation. The patient lost an estimated 7 L of blood, and was resuscitated with 2 L of crystalloid, 1.750 L of colloid, 13 units of packed red blood cells, 9 units of fresh frozen plasma, 1 unit of platelets, and 10 units of cryoprecipitate. At the end of the operation, the patient was extubated uneventfully and transferred to the surgical intensive care unit. The patient was discharged to home on postoperative day four.

Conclusion: The implications of the intraoperative TEE findings were significant as there was concern that the patient could not tolerate IVC clamping, and the volume resuscitation required for this surgery. Nevertheless, clear communication with the surgical team, close hemodynamic monitoring, and swift intervention prevented adverse outcomes. The restrictive cardiomyopathy could have been a result of long-standing hypertension and hypercortisolemia; however, there are other possible causes of restrictive cardiomyopathy. As such, a patient undergoing such an extensive resection could have warranted a more thorough preoperative assessment of the patient's cardiac function.



Perioperative Anesthesia - 16 NT-pro BNP and risk stratification pre-operatively in a patient presenting for lung resection.

Carly Porat[1], James Waris[1], Tichaendepi Mundangepfupfu[2]

[1]University of Rochester Medical Center, Rochester, NY, [2]University of Rochester, Rochester, NY

Introduction: -type natriuretic peptide (BNP), and its biologically inactive yet more stable in vivo precursor NT-Pro BNP, are proteins released from the heart when it is stretched due to pressure or volume overload. NT-pro BNP can be used as both a diagnostic biomarker for heart failure (HF) and also as a functional biomarker for HF. NT-Pro BNP levels greater than 900 pg/mL have been used reliably in the diagnosis of HF¬1. We describe application of using these biomarkers to improve peri-operative management of a patient presenting for lung resection surgery.

Methods: Case: A 60-year-old female with HF, type 2 diabetes mellitus, hypertension, morbid obesity and a prior lung resection for a right middle lobe carcinoid tumor was found to have a left upper lobe lung nodule. She presented for a left VATS upper lobe wedge resection. EKG showed normal sinus rhythm. Pulmonary Function Tests were within normal limits and similar to prior studies. TTE obtained 10 days prior to her planned surgery was consistent with HF, revealing LVEF 73%, left ventricular hypertrophy, and diastolic dysfunction, and was unchanged from study obtained 1 year earlier. On the day of surgery, the patient appeared to have shortness of breath with minimal exertion and her degree of dyspnea appeared worse than baseline along with bilateral lower extremity edema. On discussion with the patient, she denied any acute worsening of her functional condition and did not want her surgery to be postponed. Based on the preoperative history and exam we continued to be concerned for an acute HF exacerbation and so sent an NT-pro BNP. We discussed our concerns with the Surgeon and agreed we would postpone the case if the NT-pro BNP >1000 pg/ml to allow for further preoperative optimization. NT-pro BNP levels were 1463 pg/ml. We called the patient's cardiologist and discussed our concerns. The patient underwent an evaluation with cardiology the next day and was started on a more aggressive diuretic regimen with increased doses of torsemide and spironolactone. On day 8 after her case was postponed, the patient developed slurred speech and tingling of her upper lip and was found to have new ischemic infarcts which were highly suggestive of a cardioembolic process. Following treatment her stroke symptoms improved although she had some residual deficits. Her heart failure was optimized and her repeat NT-pro BNP 353 pg/mL. Once fully optimized, the patient successfully underwent her lung resection 5 months after her initial case was postponed without any adverse events.

Conclusion: NT-pro BNP already has a well-established role in diagnosis and monitoring response to medical therapy in patients with HF. Even in patient's without an established diagnosis of HF there is literature supporting the role of NT-pro BNP as a functional biomarker in patients undergoing lung resection to identify those at increased risk of developing post-operative complications such as a-fib, postoperative deterioration of functional status, and higher mortality2,3. With our patient, good physical exam, a high index of concern and the decision to obtain an NT-pro BNP on the day of surgery helped to identify an opportunity for optimization of her health status. While trying to achieve this optimization, and despite efficient multidisciplinary communication regarding her care, our patient did experience morbidity. We believe her outcomes would have likely been worse if she instead suffered a stroke post-operatively.

References: 1. Januzzi JL, van Kimmenade R, Lainchbury J, et al. NT-proBNP testing for diagnosis and short-term prognosis in acute destabilized heart failure: an international pooled analysis of 1256 patients The International Collaborative of NT-proBNP Study. Eur Heart J.2006;27:330-337. 2. Cardinale D, Colombo A, Sandri MT, et al. Increased perioperative N-terminal pro-B-type natriuretic peptide levels predict atrial fibrillation after thoracic surgery for lung cancer. Circulation. 2007; 20;115(11):1339-44. 3. Nojiri, T., Inoue, M., Shintani, Y., et al. B-type natriuretic peptide-guided risk assessment for postoperative

complications in lung cancer surgery. World J. of surgery, 2015;39(5), 1092-1098.

Perioperative Anesthesia - 17 A Tailored Approach to Anesthetic Management of Patient with Multiple Sclerosis

Nishita N Patel[1], Ashleigh Menhadji[2]

[1]Boston University School of Medicine, boston, MA, [2]Boston University School of Medicine, Boston, MA

Introduction: Multiple sclerosis is an autoimmune mediated demyelinating disease of the central nervous system, characterized by variable course and severity from relapsing and remitting to chronic progressive. Globally, it is one of the most common causes of neurological disability among young adults. Symptoms vary depending on the type of nerve fibers that are affected, with visual disturbances (such as vision loss, optic neuritis), sensory deficits (numbness, tingling, spasms) and motor deficits (paraparesis or paraplegia). Anesthetic choices and perioperative management must be carefully considered as it can increase the risk of exacerbation of disease following anesthesia and surgery.

Methods: This is a 35 years old female with a history of relapsing remitting multiple sclerosis, anxiety, mandibular hyperplasia and maxillary hypoplasia presented for Osteotomy LEFORT. Patient reported the last MS flare 2 years ago. Currently on natalizumab, with no neurological deficits on preoperative physical exam. Patient underwent a General Anesthesia with induction using Lidocaine, Propofol and Remifentanil 1mcg/kg. General Anesthesia was maintained with Propofol and Remifentanil infusions. Paralytics were avoided. Normothermia was maintained during perioperative and postoperative periods. In our case, patient had an uneventful Emergence and recovery in PACU. Patient was found to have no weakness or neurological deficits on the repeat physical exam prior to discharge.

Conclusion: Multiple sclerosis is a demyelinating disease of the central nervous system that is more prevalent in females than males [1]. As neuronal reserve is decreased in multiple sclerosis, recovery from relapses become incomplete, and neurological deficits follow leading to sustained disability [4]. Main concern for patients with multiple sclerosis is triggering exacerbation following stressful situations such as surgery, anesthesia, infection, inflammation, fever, emotional stress, vaginal delivery and pain. MS patients are noted to have upregulation of nicotinic acetylcholine receptors of skeletal muscles which increases the risk of producing life threatening hyperkalemia with use of Succinylcholine, thus should be avoided. Increased number of post junctional receptors could also lead to an unpredictable increased response to nondepolarizing neuromuscular blocking agents so it is advised to carefully titrate the dose due to increased risk of residual muscular weakness. Continuous body temperature monitoring and control is very important in MS due to increased risk of exacerbation with elevated temperature of as much as 1 degree C from baseline [3]. Therefore, it is important to have neurological examination and careful perioperative management of patients with MS, with attention to anesthetic agents, perioperative stress and hyperthermia.

References: 1. Makris A, Piperopoulos A, Karmaniolou I. Multiple sclerosis: basic knowledge and new insights in perioperative management. J Anesth. 2014;28(2):267-278. 2. Oh J, Vidal-Jordana A, Montalban X. Multiple sclerosis: clinical aspects. Curr Opin Neurol. 2018;31(6):752-759. 3. Ohshita N, Gamoh S, Kanazumi M, Nakajima M, Momota Y, Tsutsumi YM. Anesthetic Management of a Patient With Multiple Sclerosis. Anesth Prog. 2017;64(2):97-101 4. Dobson R, Giovannoni G. Multiple sclerosis - a review. Eur J Neurol. 2019;26(1):27-40

Perioperative Anesthesia - 18 A case of pheochromocytoma pre-operative workup requiring drug-eluting stent just before resection

Jocelyn Wang[1], Fredrick Mihm[1], Larissa Kiwakyou[1]

[1]Stanford, Palo Alto, CA

Introduction: Pheochromocytomas (pheo) are rare catecholamine-secreting tumors with potentially lethal cardiac complications (arrythmia, cardiomyopathy, and myocarditis). Pre-operative pheo workup literature places emphasis on electrocardiogram (ECG) and sometimes echocardiogram (echo) (1, 2).

Methods: A 76yo female with a history of CVA, MI, stable angina, carotid stenosis, and T2DM, was found to have bilateral retroperitoneal 10cm masses concerning for pheos, and presented to the pre-anesthesia clinic the day before bilateral adrenalectomies with normal blood pressure (BP) and heart rate (HR) on doxazosin 2mg TID and an echo from 2.5 months prior with a left ventricular ejection fraction (LVEF) of 60%. ECG was unchanged, yet routine troponin (TP) was 0.244 and echo showed an LVEF 52%. Repeat TP increased to 0.271, prompting surgical delay for inpatient cardiac workup. Coronary CTA showed severe 3-vessel disease and catheterization showed 95% left anterior descending stenosis (fig 1). A 2.75x15mm Resolute Onyx Drug-Eluting Stent (RO-DES) was placed and she was continued on dual-antiplatelet therapy (DAPT) for 1 month. Clopidogrel was held 5 days before surgery, 5 weeks after stent placement. TP and ECG were unremarkable pre-operatively. Pre-induction pulmonary artery pressures (PAP) were 30/17, PAOP 12, and cardiac output (CO) 4 L/min. Intraoperative course was notable for unexpected difficult airway and BP/HR lability. Clevidipine, nitroprusside, and esmolol infusions and IV pushes were used for hemodynamic control with limited success. Despite high-dose esmolol (300 mcg/kg/min plus IV pushes), HR stayed in the 130s for about 30 minutes (fig 2). After tumor removal, vasopressin 0.04 units/min was required for vasoplegia. She was taken to the ICU intubated, given the difficult airway, where vasopressin was quickly weaned with stable CO. TP peaked to 0.498, without ECG changes, and quickly decreased. On post-operative day (POD) 1, only aspirin was resumed for the stent and she was extubated and following commands. On POD 2, TP peaked to 1.3 and CO decreased to 3. Echo showed a newly reduced LVEF of 26%, global hypokinesis, but no wall motion abnormalities or ECG changes. She was given stress-dose steroids and put back on vasopressin, norepinephrine, and epinephrine, with return of CO to baseline. Post-operative course notable for fluctuating TP levels, but no ECG changes or regional wall motion abnormalities to raise concern about the LAD stent function. Subsequent postoperative course was complicated by klebsiella pneumonia and multiple cerebral infarcts with clinical deterioration. The family requested transition to comfort measures on POD 5.

Conclusion: This case highlights the value of a thorough/continuous cardiac workup in pheo patients, particularly those with risk factors. Literature discusses ECG +/- echo, but rarely troponin testing (1, 2). At clinic, our patient had a recent normal TTE and unchanged ECG, but the TP check prompted the cardiac interventions that followed. TP elevations in pheos can be due to acute myocarditis, but also CAD, as in our patient. Thrombotic events, including spontaneous coronary thrombosis, have been associated in pheo crisis (3). Most cases had negative catheterizations suggesting myocarditis, with only one case thus far showing angiographic evidence (4). Given the intra-operative hemodynamic lability, it is unlikely the patient would have survived surgery with 95% LAD stenosis. Furthermore, this patient underwent pheo resection 5 weeks after DES placement. To our knowledge, it is the first known case of DES placement prior to a pheo resection. Given the size/nature of the masses, surgery was considered urgent. The RO-DES was chosen given its lower rate of stent thrombosis allowing DAPT to be stopped 4 weeks after placement, instead of the traditional 3 months (5). In summary, this case demonstrates the necessity of vigilant pre-operative cardiac workup as well as newer cardiac interventions, which may change the timing for surgery in urgent cases.

References: 1. J Clin Endocrinol Metab. 2007;92(11):4069-4079. 2. Journal of Cardiothoracic and Vascular Anesthesia. 2017;31:1427-1439. 3. Journal of Medical Case Reports. 2011;S310. 4. BMJ. 2010;340:1038. 5. Catheter Cardiovascular Interventions. 2021;98:E188-E196.

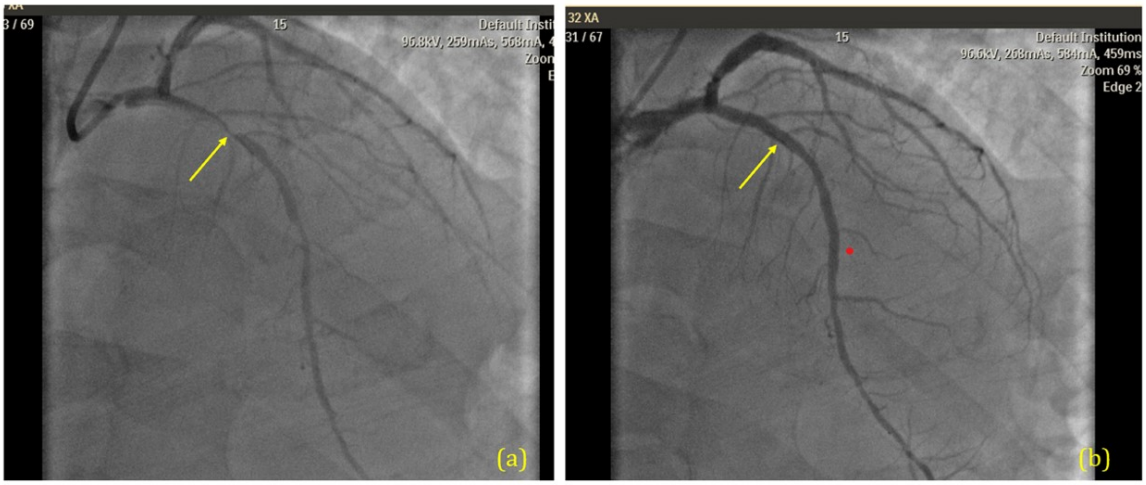


Figure 1 Cardiac catheterization demonstrating (a) 95% occlusion of LAD pre-stent placement (b) Cardiac catheterization post-stent placement.

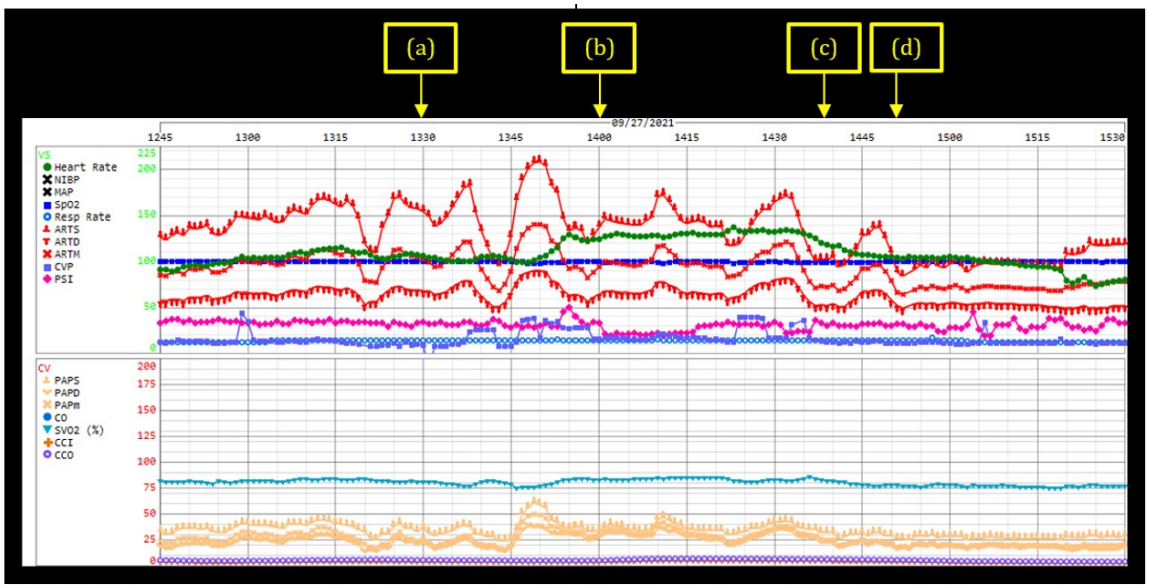


Figure 2 Intra-operative vital signs showing when (a) adrenal vein clamped, (b) adrenal artery was clamped, (c) last feeder vein clamped, and (d) removal of the adrenal tumor.

Perioperative Anesthesia - 19 Why is it problematic to diagnose anaphylaxis during anaesthesia process? Anaphylaxis to latex. Case report.

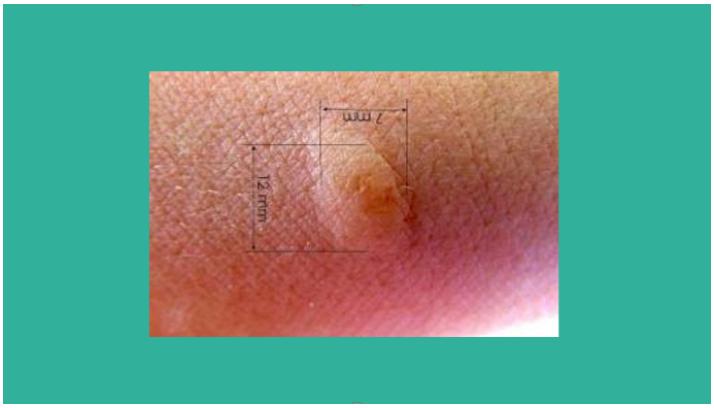
Grażyna Michalska - Krzanowska1

1Samodzielny Publiczny Specjalistyczny Zakład Opieki Zdrowotnej "Zdroje", Szczecin, Poland

Introduction: Anaphylactic reactions during anesthesia present unique diagnostic concerns. In this setting, signs and symptoms that alert the provider to a potential problem and that are apparent under other circumstances are often missing, as a consequence of the absence of an alert and communicative patient. It is difficult to diagnose anaphylactic reaction during anaesthesia because its signs and symptoms can be masked by the anesthetized status and sterile draping. In most cases, a large number of drugs will be administered during anaesthesia, and it is not possible to identify the exact allergen. The rate of latex sensitization has been increasing since its first recognition in 1979. In this paper we present a case of unpredictable adverse latex reaction which developed shortly after the induction of anaesthesia. The patient suffered a profound anaphylactic shock.

Methods: A 23-year-old man with no previous history of atopy or any known allergies was admitted to hospital with reflux nephropathy in order to undergo nephrectomy. He had a history of many general and spinal anaesthetics with good results. In induction multiple anaesthetics were used [including cisatracurium (nimbex), thiopentone, fentanyl, midazolam. The patient was monitored as usual. Approximately 30 minutes following the induction and the endotracheal intubation, and soon after the intra-abdominal manipulation, the patients respiratory status quickly deteriorated with bronchospasm with oxygen desaturation. Auscultation of breathing sounds revealed diffuse wheezing bilaterally. Within further 30 seconds he was grossly hypotensive. An intravenous fluids was started and the patient was given prednizolon , epinephrine and salbutamol nebulised. Arterial pressure was stable within 15 min. Over the following 20 minutes, the patient developed flushing, diffuse erythema, cyanosis, and his systolic arterial pressure fell back, tachykardia and oxygen desaturation. Continuous infusion of Dopamine was started. After the nephrectomy the patient was admitted to the critical care unit. Haemodynamic parameters had significantly improved and he was extubated uneventfully. Allergological investigation was carried out only 20 months after this incident. All the tests for anaesthetic drugs were negative. A positive skin test and RAST revealed a latex allergy. It was suggested to the patient and his parents that he should: 1) avoid contact with rubber products 2) wear a MedicAlert bracelet indicating the allergy to latex, 3) purchase a supply of non-latex gloves that could be given to a dentist, to emergency room staff, etc. should they need them urgently, 4) be prepared to treat an allergic reaction to latex. Antihistamines and injectable adrenalin must be available. Written informed consent was obtained from the patient's parents for publication of this Case Report

Conclusion: Our experience demonstrates that allergic reactions to latex may have life-threatening consequences. Establishing standardized operating protocols in the surgical process guarantees a structured reaction in case of perioperative anaphylaxis.



Regional Anesthesia

Regional Anesthesia - 1 Implementation of the Erector Spinae Plane Block with Liposomal Bupivacaine within an Enhanced Recovery after Surgery Program for the Nuss Procedure: A Case Series

Henry DeYoung[1], Travis Morioka[1], Maureen Higgs[1], Jacob Cole[1]

[1]Naval Medical Center Portsmouth, Portsmouth, VA

Introduction: The ultrasound-guided erector spinae plane (ESP) block is an effective technique for perioperative pain management for many abdominal and thoracic procedures. Both single shot and catheter-based techniques have been described. Prior reports have described the use of the single shot and catheter-based ESP blocks for the Nuss Procedure, a technique for surgical correction of pectus excavatum. Patients in both cases achieved successful pain control without complication, though no prior studies have described the use of liposomal bupivacaine. Liposomal bupivacaine is a potentially advantageous approach to long lasting perioperative pain control for the Nuss procedure when in-dwelling catheters are not desired or are unavailable. Here we describe the successful implementation of an ERAS protocol utilizing ESP blocks with liposomal bupivacaine in a case series of five patients.

Methods: As part of an ongoing quality improvement initiative to implement Enhanced Recovery After Surgery (ERAS) protocols, ESP blocks were performed with liposomal bupivacaine for all Nuss Procedures unless otherwise contraindicated. Single-shot, bilateral ESP blocks were performed preoperatively in a series of 5 patients undergoing Nuss procedures for surgical correction of pectus excavatum. The blocks were performed under direct ultrasound guidance by injecting 20mL of a solution of 1.3% liposomal bupivacaine (10mL) and 0.25% bupivacaine (10mL) into the fascial plane between the deep surface of the erector spinae muscle and the transverse processes of the thoracic vertebrae. The patients were followed in the immediate postoperative period as inpatients and two weeks post-operation by telephone.

Conclusion: In a series of 5 patients, the ESP block was safely performed using liposomal bupivacaine, resulting in sustained analgesia throughout admission and up to a maximum of 4 days after surgery. Patients who received the ESP block reported postoperative analgesia with visual analog scale pain scores less than 4 out of 10, and all patients reporting tolerable pain. Patients required limited postoperative opioids for pain control through hospital admission and upon discharge for the first 72 hours postoperatively. Early and rapid patient mobilization was seen, as all patients were able to participate in physical therapy on postoperative day one. Furthermore, all patients were discharged no later than three days after surgery.

The results observed in this case series indicate that the performance single-shot ESP block with liposomal bupivacaine may be a safe and effective technique when added to ERAS protocols for the Nuss Procedure. The patients in this series had prolonged analgesia, in some cases persisting after hospital discharge. This technique was particularly beneficial in our community hospital because the resources required for the placement and follow-up evaluation for ambulatory indwelling catheters were not available.

References: Ultrasound-guided erector spinae plane block for postoperative analgesia: a meta-analysis of randomized controlled trials. 14;20(1):83. 2020. The effect of ultrasound-guided erector spinae plane block on postsurgical pain: a meta-analysis of randomized controlled trials. 1;20(1):99. 2020. Ultrasound-Guided Continuous Thoracic Erector Spinae Plane Block Within an Enhanced Recovery Program Is Associated with Decreased Opioid Consumption and Improved Patient Postoperative Rehabilitation After Open Cardiac Surgery-A Patient-Matched, Controlled Before-and-After Study. 33(6):1659-1667. 2019. The erector spinae plane block: a narrative review. 72(3):209-220. 2019. Bilateral erector spinae plane block using a programmed intermittent bolus technique for pain management after Nuss procedure. 57:51-52. 2019. Bilateral single shot erector spinae plane block for pectus excavatum and pectus carinatum surgery in 2 pediatric patients. 65(9):530-533. 2018.

Regional Anesthesia - 2 Transversus Thoracic Muscle Plane Block for Acute Pain Management after Sternal Wire Removal: A Case Series

Robert Suriani[1], Wolf Vogel[2], Nomita Bhawal[1], Vlad Frenk[1], Michael Rubin[1], David Maduram[3]

[1]St. Vincent's Hospital, Bridgeport, CT, [2]Integrated Anesthesia Partners, Bridgeport, CT, [3]St. Vincent's Medical Center, Bridgeport, CT

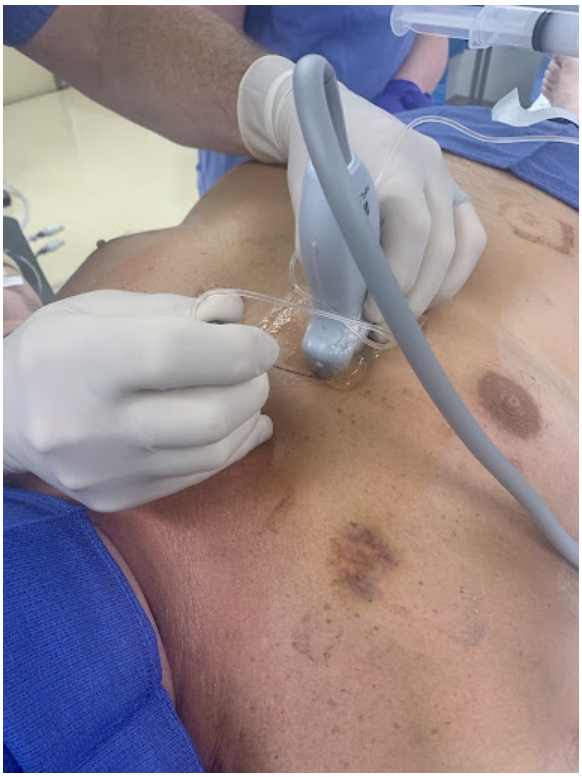
Introduction: Secure sternal closure after median sternotomy is important to facilitate sternal healing, optimize ventilatory mechanics, and minimize discomfort in the early postoperative period. Unfortunately, persistent anterior chest wall pain after sternotomy and wire closure can occur in as many as 28% of patients after median sternotomy. Although the underlying primary etiology of this pain is often unclear, secondary causes include sternal irritation, scar entrapment neuralgia, chondritis, wire hypersensitivity reaction, and protruding wires. Perioperative pain management after cardiac surgery has traditionally been accomplished with liberal use of potent narcotics. Fascial plane blocks can help to decrease opioid consumption, improve patient pain scores, decrease duration of hospitalization, and thus may be useful in minimizing pain. We present two cases in which transversus thoracic plane blocks (TTPB) were used to minimize opioid requirements as part of enhanced recovery after a cardiac surgery for sternal wire removal. Written consent for publication of non-identifying medical information was obtained from both patients.

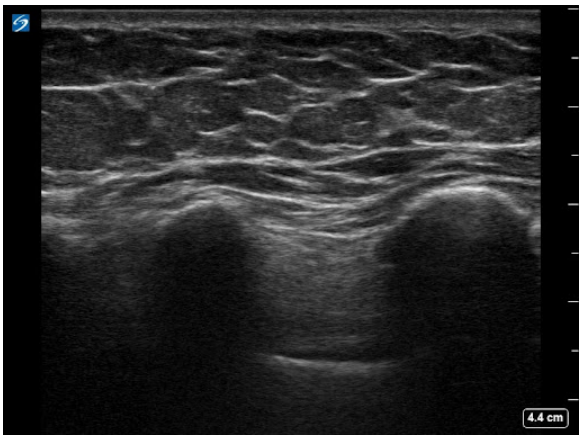
Methods: Case 1: An 84-year old 82-kg male with significant past medical history including hypertension, chronic atrial fibrillation, CHF, renal insufficiency, aortic valve replacement (2005), ascending aortic aneurysm repair (2002) and mitral valve repair (1989), presented for the removal of sternal wires protruding from his upper sternum that were causing him sternal pain. Of note, the patient related significant recent weight loss and his physical examination was notable for a lean body habitus. Case 2: a 77-year old 88 kg male with significant past medical history including hypertension, CAD, DM type II, and coronary artery bypass grafting two years prior, presented for removal of sternal wires due to persistent midsternal discomfort with movement thought to be secondary to his sternal wires. After a thorough discussion, both patients consented to bilateral transversus thoracic plane blocks (TTPB) as part of an enhanced recovery after cardiac surgery (ERAS-C) multimodal pain management plan. In both patients, general anesthesia was induced with propofol, midazolam, and rocuronium. Bilateral TTP bocks were then performed at the T4 level using a combination of fifteen milliliters of 0.25% bupivacaine with 10 ml of liposomal bupivicaine was injected in 5 ml aliquots. Both patients received 1000 mg intravenous acetaminophen, 15 mg ketoralac, and 4 mg dexamethasone prior to surgical incision. The patient in Case one had two sternal wire removed and the patient in Case two had six sternal wires removed. After completion of surgery, both patients were taken to the postanesthesia care unit (PACU). Demographic and pain information on each case is illustrated in the table below: Both patients made an unremarkable recovery without narcotics and were discharged home on the day of surgery. Both patients note complete resolution of their preoperative pain during their postoperative visit with their surgeon two weeks after surgery.

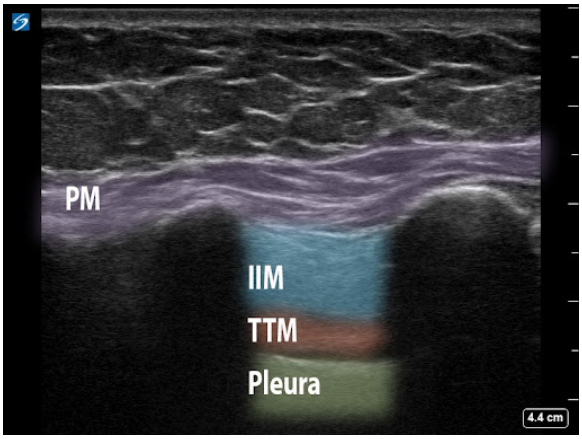
Conclusion: In these case reports, a single-shot injection provided analgesia at multiple intercostal levels due to the cranial-caudal spread of local anesthetic. Liposomal bupivacaine was utilized to provide extended analgesia at the site of surgical incision. While the potential risk of pneumothorax and internal mammary artery injury does exist during TTPB, we did not observe this adverse event clinically. Limitations of this case series include small sample size, non-homogenous distribution of patients, as well as the fact that the sensory-cutaneous blockage could not be evaluated because the blocks were administered after the induction of general anesthesia. Combined regional anesthesia techniques may be underutilized in the population of patients with acute post-sternotomy pain, and increased usage may provide significant benefits for both patient morbidity and satisfaction. In these particular patients, the combination of intraoperative conduction blockade with TTPB, administration of anti-inflammatory agents, and modulation of nociceptive processing may explain the robust analgesic effect observed in these case reports. The potential that this multimodal anesthetic approach

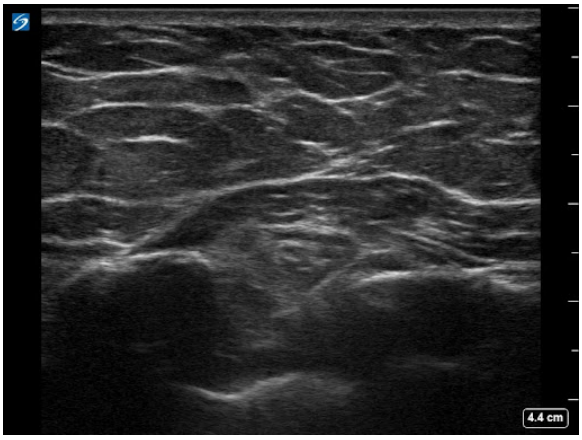
may hold for significantly improving postoperative pain control in this patient population as part of an enhanced recovery after cardiac surgery warrants further investigation.

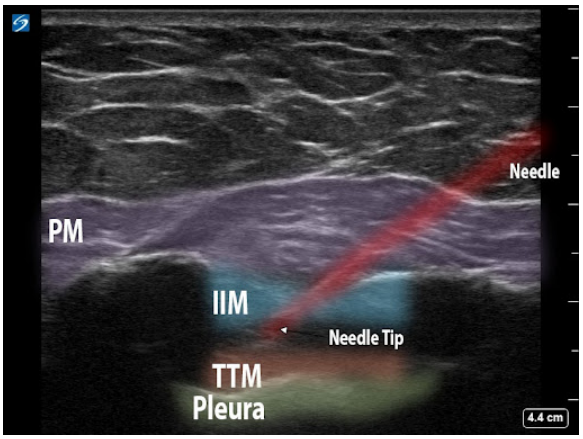
References: Norgaard MA, Andersen TC, Lavrsen MJ, Borgeskov, S. The outcome of sternal wire removal on persistent anterior chest wall pain after median sternotomy. European Journal of Cardio-Thoracic Surgery, 29, (6), 920–924 Abo El Nasa, M.M.,Abdelhady, T. (2017). Persistent post sternotomy chest pain: Does sternal wire removal have a role? Journal of the Egyptian Society of Cardio-Thoracic Surgery 25 (2), 142-146 Zhang, Y., Li, X., & Chen, S. (2021). Bilateral transversus thoracis muscle plane block provides effective analgesia and enhances recovery after open cardiac surgery. Journal of cardiac surgery, 36(8), 2818–2823










Needle
PM
IIM
Needle Tip
TTM
Pleura
4.4 cm

Regional Anesthesia - 3 Multimodal opioid-sparing analgesia for EVAR procedures using ilioinguinal blocks: A Case Series

Robert Suriani[1], Tori Pacquadio[2], Theresa Bowling[1], David Maduram[3], Kevin Jon[3]

[1]St. Vincent's Hospital, Bridgeport, CT, [2]St Vincent's Medical Center, Bridgeport, CT, [3]St. Vincent's Medical Center, Bridgeport, CT

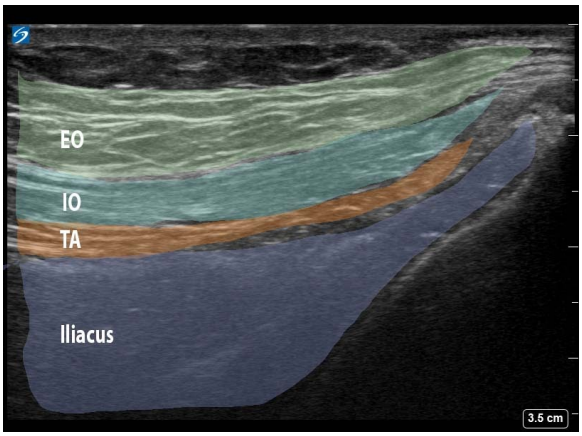
Introduction: Endovascular aneurysm repair (EVAR) is a minimally invasive procedure commonly performed for abdominal aortic aneurysm (AAA). Patients undergoing EVAR tend to be older and have a greater incidence of major comorbidities that increase the risk of adverse drug events, including opioid analgesics1,2. Given the ongoing opioid epidemic and its associated morbidity and mortality, it is important to consider opioid-sparing modalities to optimize patient care. We report the results of a case series of three patients undergoing EVAR in which a multimodal anesthetic regimen including ultrasound-guided ilioinguinal/iliohypogastric blocks were used to provide robust opioid-sparing postoperative analgesia. Our main question was how this multimodal regimen would impact postoperative pain and opioid requirements, and length of hospital stay in patients undergoing EVAR.

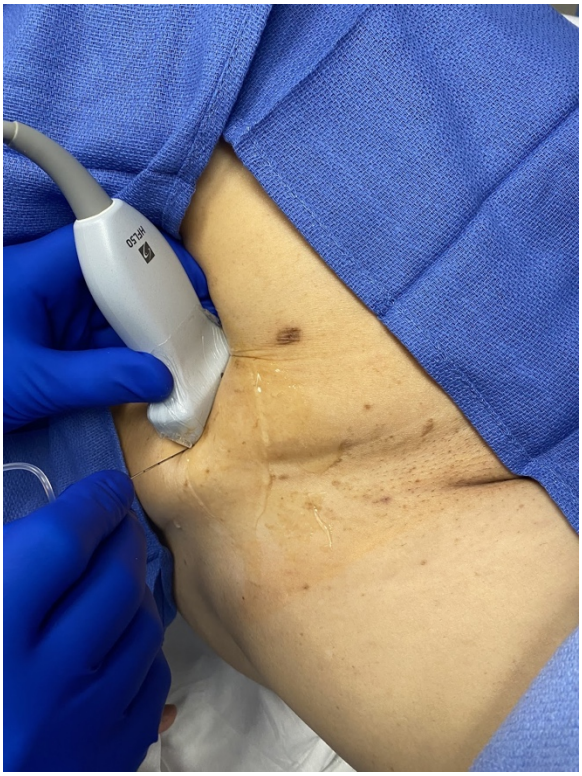
Methods: We reviewed medical records of three patients who underwent elective EVAR at St. Vincent's Medical Center. Case 1: 87 year old male with a past medical history of CAD, Hyperlipidemia, COPD, and lymphoma presented for repair of a 6 cm abdominal aneurysm. Case 2: 70 year old male with a past medical history of anemia, ulcerative colitis, and psoriasis presented for repair of a 6 cm abdominal aneurysm. Case 3: 92 year old male with a past medical history of hypertension, atrial fibrillation and hyperlipidemia presented for repair of a 6 cm abdominal aneurysm. After a thorough discussion, all patient consented to bilateral ilioinguinal/iliohypogastric blocks (II/IH) as part of an enhanced recovery after surgery multimodal pain management plan. General anesthesia was induced with propofol, sevoflurane and rocuronium, and the trachea intubated after LTA spray with 4% lidocaine. As part of a multimodal anesthetic regimen, all patients received intraoperative dexmedetomidine 0.4-0.6 mcg/kg/hr and remifentanil 0.1 mcg/kg/min for the duration of the procedure. Bilateral II/IH blocks were then performed. After wide chlorhexidine 4% prep, an ultrasound transducer probe (SonoSite PX, SonoSite Inc, Bothell, WA) was positioned in a longitudinal orientation on a line joining the anterior superior iliac spine and the umbilicus (Figure 1 and 2). A hyperechoic 22-gauge needle (B-Braun, Melsungen, Germany) was inserted in a lateral-to-medial direction using the in-plane technique (Figure 3). The correct tip position was confirmed by the visualization of linear fluid spreading in the myofascial plane between the internal oblique and the transverse abdominus muscles (Figure 4). After confirming needle tip position, a total of 15 milliliters of 0.25% bupivacaine with 5mg dexamethasone was injected in 5cc aliquots. This procedure was repeated on the opposite side for a total of 30 ml of local anesthetic. No other local anesthetics were used. After completion of surgery, all patients were taken to the postanesthesia care unit (PACU) and then to cardiac stepdown unit. No patients required additional narcotics in the intraoperative or postoperative periods. All were discharged to home the next day.

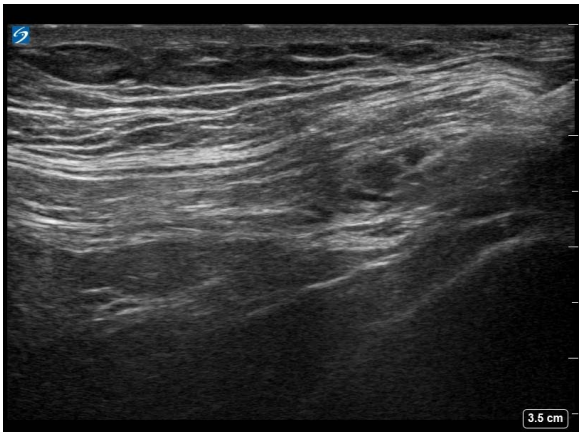
Conclusion: No published case series to date describes II/IH block use in EVAR procedure. In this retrospective case series, a single-shot injection provided analgesia at multiple cervical levels due to the extensive cranial-caudal spreading of local anesthetic in the musculofascial plane deep into the erector spinae muscle. This procedure is safe, because the needle remains distant from the neuraxis, pleura, major vessels, and nerves at all times. While recent cadaveric studies have described the potential risk of phrenic nerve paralysis, we did not observe this adverse event clinically. Our findings on efficacy of II/IH blocks for EVAR posterior cervical spine fusion are consistent with other studies of erector spinae blocks for posterior thoracolumbar surgery. The combination of intraoperative conduction blockade with ESP blocks, administration of anti-inflammatory agents, and modulation of nociceptive processing may also explain the robust analgesic effect observed in this case series. The potential that this multimodal anesthetic approach may hold for significantly

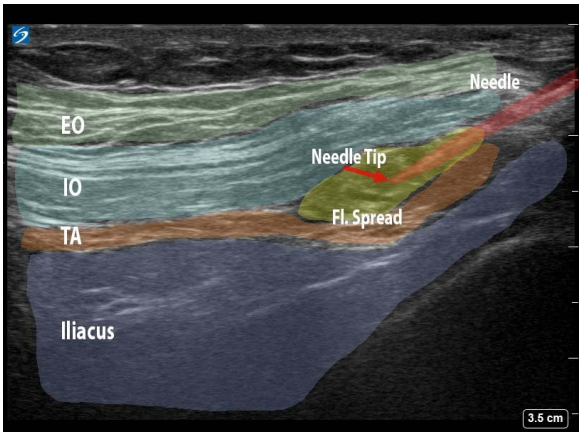
improving postoperative pain control warrants further investigation.

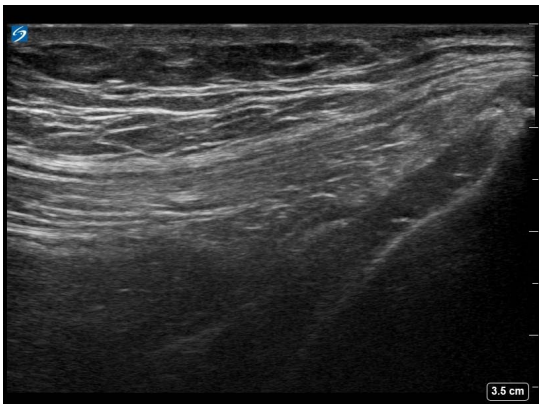
References: Kim HO, Yim NY, Kim JK, Kang YJ, Lee BC. Endovascular Aneurysm Repair for Abdominal Aortic Aneurysm: A Comprehensive Review. Korean J Radiol. 2019;20(8):1247-1265. doi:10.3348/kjr.2018.0927 Müller V, Miszczuk M, Althoff CE, et al. Comorbidities Associated with Large Abdominal Aortic Aneurysms. Aorta (Stamford). 2019;7(4):108-114. doi:10.1055/s-0039-1692456











Regional Anesthesia - 4 Analgesic efficacy of ultrasound-guided bilateral ESPB and II/IH blocks for ablation of atrial fibrillation via the Convergent Procedure: A case series

Robert Suriani[1], Wolf Vogel[2], Nomita Bhawal[1], Vlad Frenk[1], Michael Rubin[1], David Maduram[3]

[1]St. Vincent's Hospital, Bridgeport, CT, [2]Integrated Anesthesia Partners, Bridgeport, CT, [3]St. Vincent's Medical Center, Bridgeport, CT

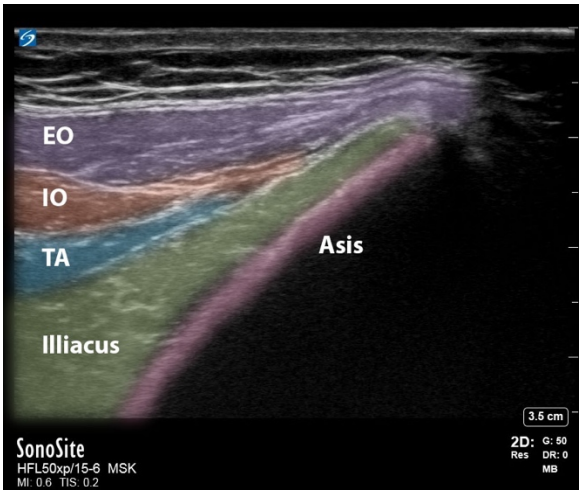
Introduction: Convergent surgery is an emerging surgery option for treatment of atrial fibrillation, which consists of (1) surgical ablation of the posterior left atrium with monopolar radiofrequency through a minimally invasive approach, followed by (2) endocardial catheter ablation to perform pulmonary vein dilation and completion of any additional ablation lines. Although this procedure is designed to be minimally invasive, opening of the chest wall, entering the pericardium, and ablating the left atrium can cause significant pain. Treatment of this pain with opioids can cause significant adverse effects including nausea and respiratory compromise. We present a retrospective case series of patients undergoing a convergent procedure with a multimodal anesthetic regimen including an erector spinae plane and ilioinguinal/iliohypogastric blocks to provide a robust opioid-sparing perioperative analgesia. Our main outcome was the patient's postoperative pain scores and opioid requirements at 24, 48 and 72 hours after the end of surgery. As secondary endpoints, we looked at length of hospital stay.

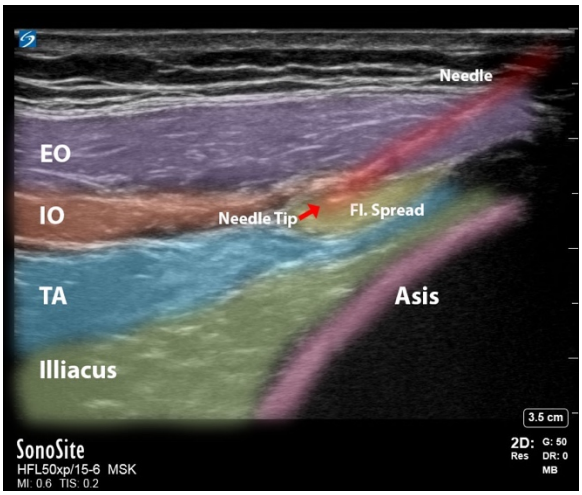
Methods: We reviewed medical records of three patients who underwent convergent surgery between March 11, 2021 to June 3, 2021 at St. Vincent's Medical Center, Bridgeport, Connecticut. The demographic data of all patients in this sample were recorded. In the postoperative period, pain scores on movement were measured via visual analogue scale(VAS) at 24, 48, and 72 hours postoperatively. Postoperative morphine equivalent requirements were noted and recorded. All patients in this series received general anesthesia with intraoperative erector spinae plane and ilioinguinal / iliohypogastric blocks. As part of a multimodal regimen, all patients received acetaminophen and a gabapentinoid preoperatively. Anesthesia was induced with propofol, midazolam, rocuronium, LTA spray, and ketamine 0.5 mg/kg and maintained intraoperatively with dexmedetomidine 0.6 mcg/kg/hr, ketamine 0.25 mg/kg/hr and sevoflurane. Post-operative pain was treated with scheduled oral acetaminophen and opioids as needed for breakthrough pain.

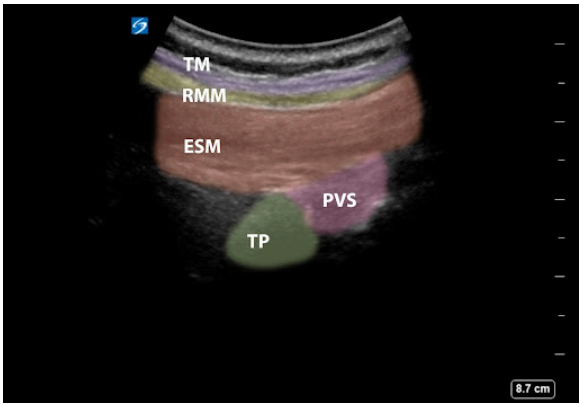
Conclusion: The main findings of the current case series demonstrate that combined ESP and ilioinguinal / iliohypogastric blocks in patients slated for convergent procedures are associated with low postoperative opioid requirements, decreased pain scores, and high patient satisfaction. Although formal assessment of time to first rescue analgesia, length of ICU stay, and hemodynamic stability were not performed, our initial results, coupled with the absence of any notable complications, make ESP/ILI/IH blocks a promising perioperative addition to any cardiac ERAS / opioid-sparing regimen. It is important to note that other important elements of the anesthetic regimen included preemptive analgesic administration of dexmedetomidine, as well as liposomal bupivacaine and dose ketamine. Our findings on efficacy and safety of ESP/ILI/IH blocks for convergent procedures are consistent with other studies of fascial plane blocks for cardiac surgery. Although other fascial plane blocks have been shown to be effective for cardiac surgery, ESP/ILI/IH blocks blocks may provide an unique advantage in terms of ease of performance and reduced risk of complications such as pneumothorax, chest wall hematoma, and local anesthetic toxicity. Further research is warranted.

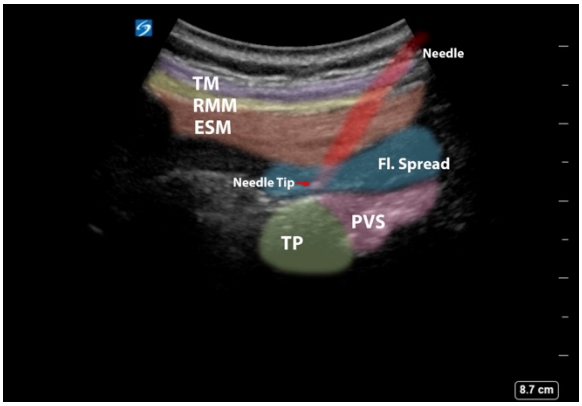
References: Cox, J. L., Churyla, A., Malaisrie, S. C., Pham, D. T., Kruse, J., Kislitsina, O. N., & McCarthy, P. M. (2019). A hybrid maze procedure for long-standing persistent atrial fibrillation. The Annals of thoracic surgery, 107(2), 610-618. Varzaly, J. A., Lau, D. H., Chapman, D., Edwards, J., Worthington, M., & Sanders, P. (2021). Hybrid Ablation for Atrial Fibrillation: A Systematic Review and Meta-analysis. JTCVS Open. Trzcinka, A., Lee, L. S., Madias, C., Homoud, M. K., Rastegar, H., Couper, G. S., & Cobey, F. C. (2021). The convergent procedure: A unique multidisciplinary hybrid treatment of atrial

fibrillation. Journal of Cardiothoracic and Vascular Anesthesia, 35(2), 631-643. Li, X., Li, M., Shao, Y., Gu, W., Ni, B., Gu, J., & Chen, M. (2021). Thoracoscopic ablation delays progression from paroxysmal to persistent atrial fibrillation. The Journal of Thoracic and Cardiovascular Surgery.









Regional Anesthesia - 5 ICD Placement in a Critically ill Patient Using Transthoracic Plane Block and Pectoral Nerve Blocks

Li Lin[1], Miguel A Yaport[1], Pavan Sekhar[1]

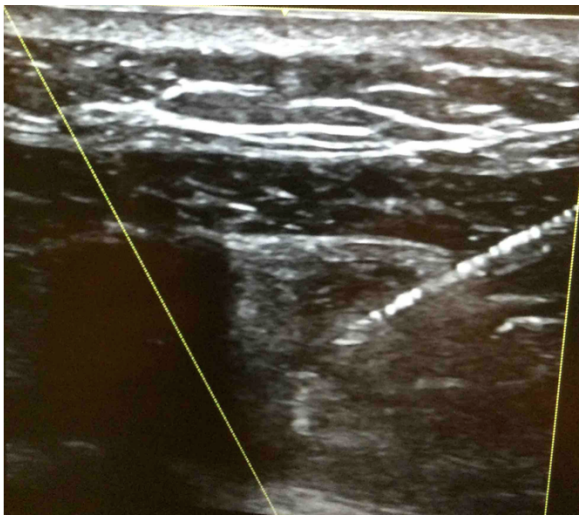
[1]Tufts Medical Center, Boston, MA

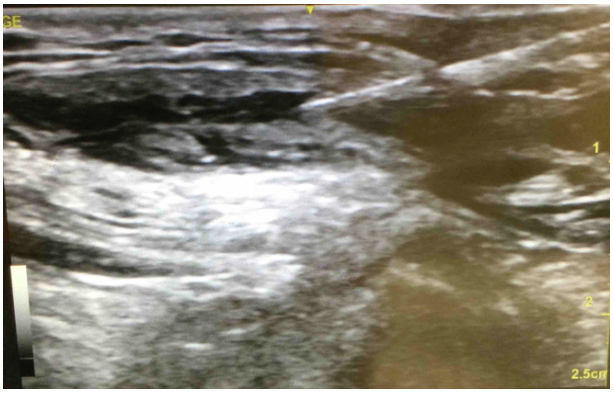
Introduction: In our institution, a large proportion of patients undergoing Implantable Cardioverter Defibrillator (ICD) implantation are critically ill. ICD devices can be implanted via the transvenous (TV-ICD) or subcutaneous (S-ICD) routes and are typically implanted under general anesthesia (GA) due to pain and discomfort associated with tunneling and dissection. GA can be associated with hemodynamic instability and perioperative respiratory adverse events (PRAE). Relative to regional anesthesia (RA), GA may have longer recovery time and is associated with increased overall cost. TV-ICD may be placed under Monitored Anesthesia Care (MAC) with surgical infiltration, however, Infiltration techniques by the interventionalist could have unreliable efficacy. Regional anesthesia is an attractive alternative to GA for both sets of patients. Recently there has been increasing interest in using combined Pectoral Nerve blocks, or Serratus Anterior Plane blocks, with and without the Transvers Thoracic Plane (TTP) block for ICD placement.

Methods: 53yo man with PMH of severe ischemic cardiomyopathy status post coronary artery bypass graft (CABG) x 4, chronic kidney disease (CKD) 3, pulmonary embolism (PE) on rivaroxaban, was admitted for acute on chronic systolic heart failure, mixed cardiogenic and septic shock, large right pleural effusion requiring therapeutic thoracentesis, diuresis and inotropic support. 12-lead EKG showed sinus tachycardia with LBBB, associated ST elevation in anterior leads. Transthoracic Echocardiogram (TTE) showed a reduced Ejection Fraction of 10-15%, and moderately reduced Right Ventricular function. Once hemodynamically stable he underwent ICD placement for primary prevention. Given the patient's comorbidities, his case proceeded with MAC and a surgical block with combined left TTP and PEC I and II. Prior to the block, the patient received 50mcg Fentanyl and 20 mg of Propofol for anxiolytic purposes. A total of 30cc of a mixture containing 28cc of 0.25% bupivacaine 4mg Dexamethasone, and 100 mcg Clonidine were used. 10cc were injected in each of the three blockade sites. Intra-operatively the patient received 5-20 mcg/kg/min propofol, 24mcg Dexmedetomidine. An additional 25 mcg of fentanyl were given at the time of incision as an adjunct for sedation and anxiolysis. The patient required a low dose Norepinephrine infusion at 2-3 mcg/kg/min. Post operatively, the patient returned to baseline hemodynamics. Patient reported a 0-2 out of 10 on the pain scale. 24 hr postoperative Morphine Milligram Equivalents were 0.

Conclusion: Currently no standard anesthetic management exists for ICD placement, GA remains the most common anesthetic method. In sicker patients, regional anesthesia has an unparalleled advantage over GA, including but not limited to, a better hemodynamic profile, lower risk of PRAE, shorter recovery time, and possibly lower cost. Bhatt et al demonstrated successful S-ICD placement with the use of TTP and Serratus Plane Block with a reduction in opioid use. In the pediatric literature, PEC blocks have been shown to reduce postoperative pain and opioid use after ICD placement. In this case report, we demonstrate successful use of TTP with Pectoral Nerve blocks for ICD placement in a critically-ill adult patient. Regional techniques for ICD placement have many potential benefits, especially in this population. Future goals include larger clinical studies comparing the use of regional techniques to local infiltration by surgeon, comparing TTP with PEC to PEC alone, total MME perioperatively, patient satisfaction level, and surgical, anesthesia and recovery times.

References: 1. J Cardiovasc Electrophysiol. 2019 Jan;30(1):141-148. 2. J Cardiothorac Vasc Anesth. 2018 Feb;32(1):e22-e23. 3. Pacing Clin Electrophysiol. 2018 Jul;41(7):807-816. 4. J Cardiothorac Vasc Anesth. 2021 Nov;35(11):3294-3298 5. Heart Rhythm. 2020 Aug;17(8):1346-1353.




1
2
2.5cm

Regional Anesthesia - 6 Combined Ultrasound-Guided Anterior Suprascapular and Infraclavicular Brachial Plexus Block for Shoulder Arthroscopy and Rotator Cuff Repair in a Patient with Severe Cardiopulmonary Disease

Sherry Liao[1], Robert Maniker[2]

[1]New York Presbyterian Hospital - Columbia University Irving Medical Center, New York, NY, [2]New York Presbyterian - Columbia University, New York, NY

Introduction: Regional anesthesia is a mainstay of post-operative analgesia for shoulder surgery and often utilized as a primary anesthetic. Interscalene block (ISB), while reliable and widely utilized for this purpose, blocks the phrenic nerve and is therefore contraindicated in many pulmonary conditions. Other blocks have been utilized for shoulder surgery but confer either partial coverage or a high incidence of phrenic block [1-3]. We report a combination of ultrasound-guided anterior suprascapular nerve block (SSNB) and infraclavicular brachial plexus block (ICB) as the primary anesthetic for shoulder arthroscopy and rotator cuff repair in a patient with end-stage pulmonary disease and review the anatomy of brachial plexus block for shoulder surgery.

Methods: A 77-year-old female with interstitial lung disease due to hypersensitivity pneumonitis on home oxygen and cardiomyopathy with ejection fraction of 15% presented for elective shoulder arthroscopy and rotator cuff repair. After extensive counseling regarding the risks of anesthesia and surgery, the patient wished to proceed as she did not want to live without better arm function. A plan was made for a primary regional anesthetic with attempt to avoid phrenic nerve block. She underwent ultrasound-guided anterior SSNB and ICB targeting the posterior and lateral cords, followed by sedation with low-dose propofol and dexmedetomidine. Surgery was performed uneventfully, and the patient was discharged home on the same day.

Conclusion: Over the past two decades, alterations to ISB using small volume or dilute local anesthetic concentrations, injection lateral to the brachial plexus, and targeted C7 nerve root block have not been successful in avoiding phrenic block while maintaining complete shoulder analgesia [1]. Combined SSNB and axillary nerve block is also described but results in incomplete block since subscapular, musculocutaneous and lateral pectoral nerves are spared [2]. Furthermore, supraclavicular block and superior trunk block have been shown to provide similar analgesia compared with ISB but may not completely spare the phrenic nerve [3]. A recent case series demonstrated that combined SSNB and ICB are effective for postoperative analgesia with low risk for hemidiaphragmatic paralysis in patients undergoing total shoulder arthroplasty [4]. Similarly, a recent meta-analysis showed that SSNB may produce similar analgesia to ISB with decreased risk of Horner syndrome, numbness, dyspnea, and hoarseness [5]. We describe the case of a patient with significant pulmonary and cardiac disease who safely underwent shoulder arthroscopy under light sedation utilizing combined SSNB and ICB to provide coverage of suprascapular, axillary, subscapular, lateral pectoral and musculocutaneous nerves while sparing phrenic block. This combination may warrant further study as an alternative to ISB as the gold standard block for shoulder surgery.

References: 1. Reg Anesth Pain Med 42:32-38. 2017 2. Arthroscopy 24:689-96. 2008 3. Reg Anesth Pain Med 45:73-78. 2020 4. Acta Anaesthesiol Scand 65:675-680. 2021 5. J Orthop Surg Res 16:376. 2021

Regional Anesthesia - 7 Liposomal Bupivacaine Stellate Ganglion Block in Neuromodulation-resistant Electrical Storm

Georges Abi Roustom[1], Miguel A Yaport[1]

[1]Tufts Medical Center, Boston, MA

Introduction: Ventricular arrhythmias (VA); Ventricular tachycardia (VT), ventricular fibrillation (VF) and Electrical storm (>= 3 episodes VT or VF in 24h) are life-threatening phenomena that are often difficult to treat and refractory to medical therapy. Stellate ganglion block (SGB) has been shown to be an efficacious treatment modality. While still relatively uncommon and transient in nature, SGB has become the intervention of choice in our institution for patients on whom it would be infeasible or unsafe to perform definitive management. Oftentimes the goal is to use SGB to provide a bridge to definitive therapy. While SGB has been shown to reduce arrhythmia burden for 24-48h, end-stage heart failure patients may be supported by extracorporeal membrane oxygenation (ECMO), or mechanical circulatory support (MCS) for days to weeks, with waitlist averages of approximately 123 days. We report the case of a patient who had multiple returns of VT several hours following SGB. Prolonged symptom-free therapy was only obtained following the use of liposomal bupivacaine.

Methods: A 48-year-old female with a medical history of type 2 diabetes mellitus, hypertension, coronary artery disease and ischemic cardiomyopathy with ICD, was transferred from an outside hospital after a witnessed VT arrest requiring defibrillation x3 prior to achieving ROSC. Subsequently she underwent placement of a cardiac resynchronization therapy device (CRT-D) which was complicated by cardiogenic shock and initiation of Venoarterial ECMO (VA-ECMO) with an IMPELLA and infusions of norepinephrine, vasopressin and dobutamine. 8 days after admission, she began experiencing episodes of VT. The first episode abated following 2 discharges of her CRT-D. She was then started on amiodarone and lidocaine infusions. Two days later, in the setting of lidocaine infusion weaning she experienced another episode of persistent VT requiring multiple shocks, amiodarone and lidocaine boluses and lidocaine infusion uptitration. A SGB was performed on the left side with a mixture of 5ml of 2% lidocaine, 3ml of 0.5% bupivicaine, 4mg Decadron and 50mcg clonidine. Needle stimulation of the stellate ganglion induced VT, which ceased after administration of the local anesthetic. Her tachytherapies were deactivated. 30 hours later she experienced sustained VT lasting over one hour. She was defibrillated 11 times, boluses and infusions of both amiodarone and lidocaine were ineffective. Medication infusions were maintained and an urgent SGB was done on the left side with 10ml 0.5% Bupivicaine, without any additives. Subsequently, she remained in a stable rhythm until approximately 12h later, when she had a single episode of VT that resolved after defibrillation and amiodarone and lidocaine boluses. At this time, she had a bilateral SGB, with 10ml of 1.3% liposomal bupivacaine per side. Following this, the patient went to the operating room for placement of Biventricular Assist Device and remained arrhythmia free for 13 days, upon which she experienced a return of VT followed by degeneration into VF.

Conclusion: MCS as a bridge to therapy is often complicated by ventricular arrhythmias, a phenomenon that will likely continue to increase since UNOS changed their Heart Transplant allocation algorithm in 2018. As catheter based ablation or surgical sympathectomy can be impractical in critically ill patients, autonomic modulation via stellate ganglion block (SGB) has emerged as the best alternative, however it remains a short-lived intervention. Given most of the patient population experiencing the burden of VA is on anticoagulant agents, repeat interventions may put them at risk of bleeding complications, thus, reducing the number of interventions is in this population's best interest. Other methods of achieving long-lasting SBG have been done such as placement of stellate ganglion catheters, which can be associated with a risk of malposition, infection and bleeding. While we know that the half-life of Liposomal Bupivacaine (13 to 34 hours) is longer than that of Bupivacaine (around 8 hours), this alone does not explain the 13-day duration of therapy and warrants further investigation evaluating multiple options for long term management of electrical storm using SGB.

References: 1.“Treating Electrical Storm: Sympathetic Blockade Versus Advanced Cardiac Life Support.” Circulation. 2000; 102:742-747 2. Circulation. 2000; Vol 102:742-747 3. Auton Neurosci, 205 (2017), pp. 1-11 4. J Cardiovasc Electrophysiol. 2017; 28: 1460– 1467 5. The Journal of Thoracic and Cardiovascular Surgery, Volume 155, 2018, p.1607-1618 6. Circ Arrhythm Electrophysiol, 12 (2019), Article e007118 7. Curr Hypertens Rep 2020; 22: pp. 100. 8. J Heart Lung Transplant. 2020 Aug 26:S1053-2498(20)31701-0

Respiration

Respiration - 1 A Low-Flow Nasal Mask-Face Tent Maintained Spontaneous CPAP Ventilation/Oxygenation and Reduced Aerosol/Droplet Spread in an Elderly Patient with h/o Severe COVID-19 Infection during AICD Insertion

James Tse[1], Kimberly Trojak[1], Julie Wise[1], Vincent Reformato[1]

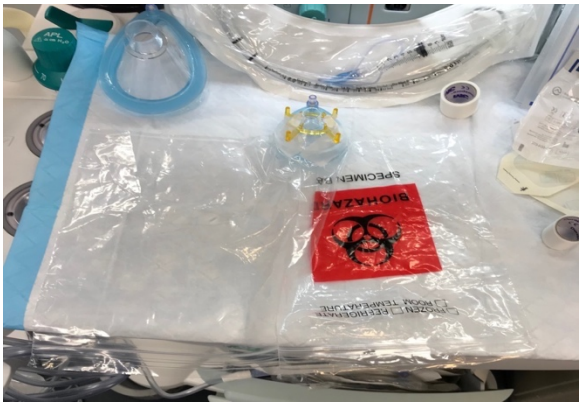
[1]Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ

Introduction: Patients under IV sedation often receive oxygenation via a nasal cannula. Over-sedation and/or airway obstruction may result in severe desaturation, especially in obese patients with obstructive sleep apnea (Ossa). A pediatric facemask has been shown to provide spontaneous nasal CPAP ventilation and improve oxygen delivery in deeply sedated OSA patients.(1-6) A simple combined nasal mask-face tent provided pre/apneic nasal oxygenation and reduced aerosol/droplet spread during RSI, intubation and extubation in a COVID-19 patient (7) and during peroral endoscopic myotomy (POEM) and extubation in a COVID-19 patient (Fig. 1).(8) We used this technique to provide nasal CPAP oxygenation and reduce aerosol/droplet spread in a high-risk patient with previous severe COVID-19 infection during AICD insertion.

Methods: Case Report A 83-year-old female, 5'1', 144 lbs, with NIDDM, HTN, former smoker (quitted 6 months prior), atrial fibrillation, severe cardiomyopathy presented for AICD insertion. The previously healthy patient suffered severe COVID-19 infection 6 months prior and was on ventilator support for 10 days, and developed paroxysmal atrial fibrillation and severe cardiomyopathy (LVEF 15-20%). She was admitted for fatique/dyspnea on exertion and her baseline SpO2 was 91%. Her COVID test was negative 2 days prior. She was fitted with a modified infant facemask and gave her consent for photography and case report. The infant facemask was secured over her nose with elastic head-straps and connected to the anesthesia machine via a long breathing circuit (Fig. 2). The APL valve was adjusted to deliver 3-5 cm H2O CPAP with 4L/min O2. To reduce aerosol/droplet spread, a clear plastic sheet (face tent) was taped along her lower jaw and covered her mouth (Fig. 2-3). Following nasal CPAP pre-oxygenation, her SpO2 improved from 91 to 99%. Deep sedation was titrated with dexmedetomidine and propofol infusion (75 mcg/kg/min). She maintained spontaneous nasal ventilation and 97- 99% SpO2 throughout (Fig. 4-5). She tolerated the procedure well without the need for airway manipulation. In PACU, she maintained 96% SpO2 with 4 LO2/min via a nasal cannula and a face tent covering her mouth to reduce aerosol/droplet spread.1

Conclusion: This simple combined nasal mask-face tent maintained spontaneous CPAP ventilation/oxygenation in a high risk patient with previous severe COVID infection during AICD insertion. It also reduced aerosol/droplet spread and oxygen level near the surgical site during the procedure. Amid the ongoing COVID-19 pandemic, this technique may improve patient safety and provide additional provider protection at no extra cost.

References: 1. www.TSEmask.com; 2. SAMBA 28th AM, MCC, 2013; 3. SASM MC's, 2013; 4. ASA AM (MC536 & 1100), 2013; 5. NYSSA 67th PGA (MCC 7094, 7115, 7120, 7129, 7189, 7199 & 7203), 2013; 6. SAM AM MCC's, 2014; 7. ASA virtual AM (MC1280), 2020: 8, IARS virtual AM (MCC 1306), 2021




VITAL SIGNS
14-APR-2021 11:27
DPROD-20334
96
RATE 84
135/
70
88
Dash 5000


VITAL SIGNS
14-APR-2021 11:28
DPROD-20334
96
RATE 82
135/
70
88
Dash 5000

Technology, Computing and Simulation, Equipment Monitoring

Technology, Computing and Simulation, Equipment Monitoring - 1 Combined Recurrent Laryngeal Nerve Monitoring and Non One-lung Ventilation Using the Artificial Pneumothorax and an Electromyographic Endotracheal Tube

Wataru Danjo[1], Shingo Nisida[1], Tomohiro Kiya[1], Yoshitada Ito[1], Mio Terasaka[1]

[1]Sapporo City General Hospital, Sapporo, Japan

Introduction: Intraoperative neuromuscular monitoring (IONM) is a widespread procedure to identify and protect the recurrent laryngeal nerve (RLN) during thyroid surgery. However, utilization of IONM during thoracic surgery has not become common [1-3]. We describe a case of mediastinal schwannoma of the vagus nerve that we removed using the IONM technique with artificial pneumothorax in video-assisted thoracoscopic surgery (VATS) [4,5].

Methods: A 22-year old female suffering mediastinal schwannoma originating in the left intrathoracic vagus nerve, was referred for VATS. Surgery was performed under epidural and general anesthesia using propofol and remifentanil. The patient was intubated with an NIM TriVantage EMG tube (Medtronic Inc., USA) [Figs.1,2]. After the patient had been placed in right decubitus position, the correct placement of endotracheal tube (ET) using McGRATH MAC (Aircraft Medical Ltd., UK) video laryngoscope. We managed the patient undergoing single-lumen ET ventilation, and the thoracic cavity was inflated with 6-8mmHg of CO2 insufflation pressure [4,5]. The patients underwent IONM by the NIM-response system 3.0 (Medtronic Inc., USA). The tumor was resected using VATS with artificial pneumothorax [Figs.3-5]. After the surgery, the patient presented with only slight hoarseness.

Conclusion: We describe our case, focusing on a mediastinal schwannoma resection along the RLN utilizing the IONM undergoing non one-lung ventilation with artificial pneumothorax. The combination of IONM and artificial pneumothorax have substantial advantages in safe mediastinal tumor resection during VATS.

References: 1. Int J Surg, 56, 301-6, 2018 2. J Cardiothorac Surg, 14, 111, 2019 Published online 3. Ann Thorac Surg, 108, e287-8, 2019 Published online 4. J Thorc Dis, 9, 903-6, 2017 5. Surg Endosc, 34, 5501-7, 2020

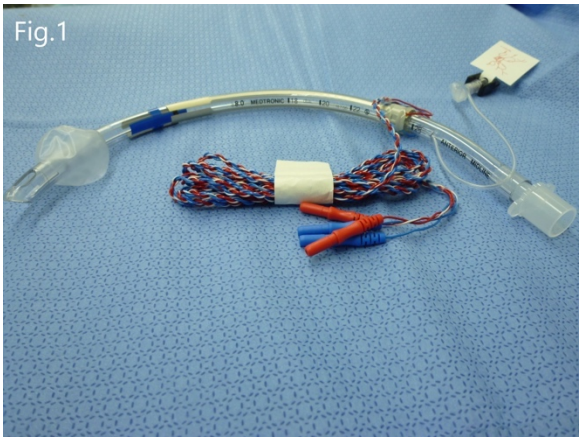


Fig.1

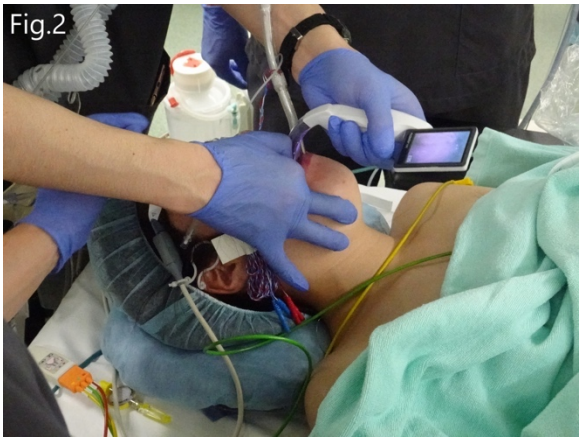


Fig.2

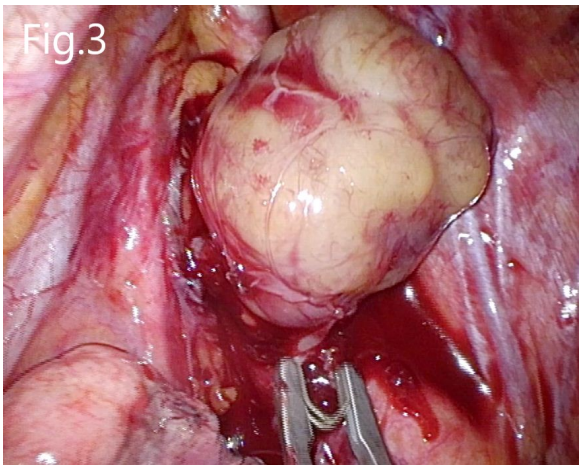


Fig.3

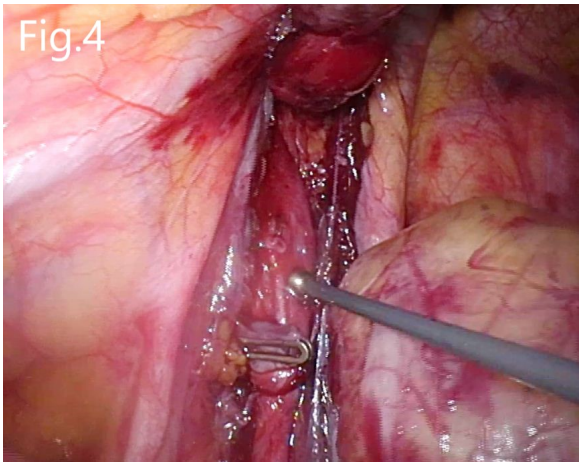


Fig.4

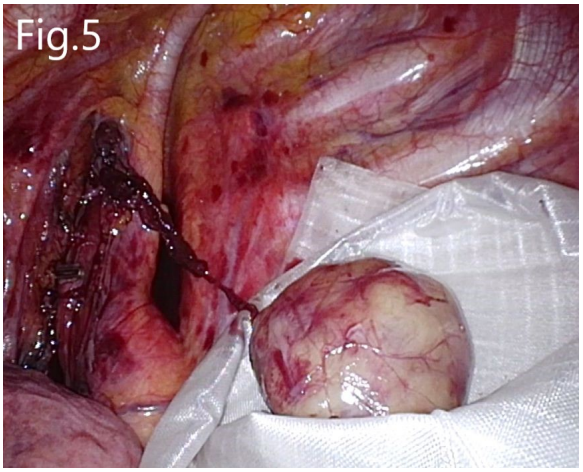


Fig.5

Trauma

Trauma - 1 Suspected tracheal laceration presenting as isolated pneumomediastinum after laparoscopic hemicolectomy: A Case Report

Bryan J Lynn[1], Jonathan Cohen[1], Hector Meija[1]

[1]Tulane, New Orleans, LA

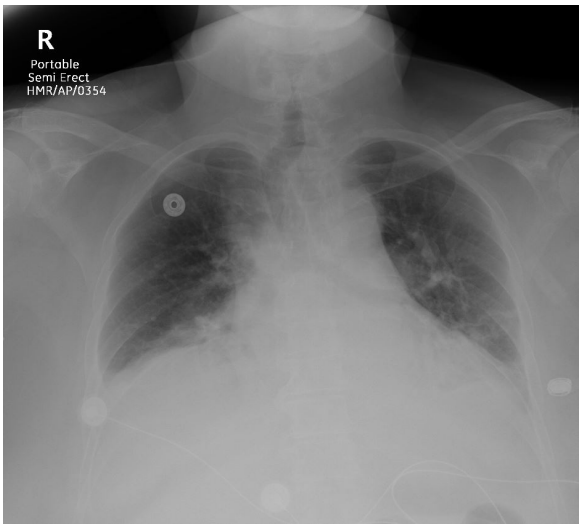
Introduction: Upon intubation, damage to the trachea can range from minor laceration to tracheal rupture. Subsequent management from tracheal disruption ranges from observation to immediate surgical repair depending on the extent of damage. We present a case of suspected tracheal laceration in a patient who underwent laparoscopic hemicolectomy for malignancy. Hypoxia, hypotension, and tachycardia arose on induction and sporadically occurred throughout, until the conclusion of the case. Post-operatively, an isolated pneumomediastinum was found; patient was discharged home after four days of observation. We highlight the importance in consideration of iatrogenic tracheal injury and the utility of bronchoscopy as the diagnostic modality of choice for tracheal disruption.

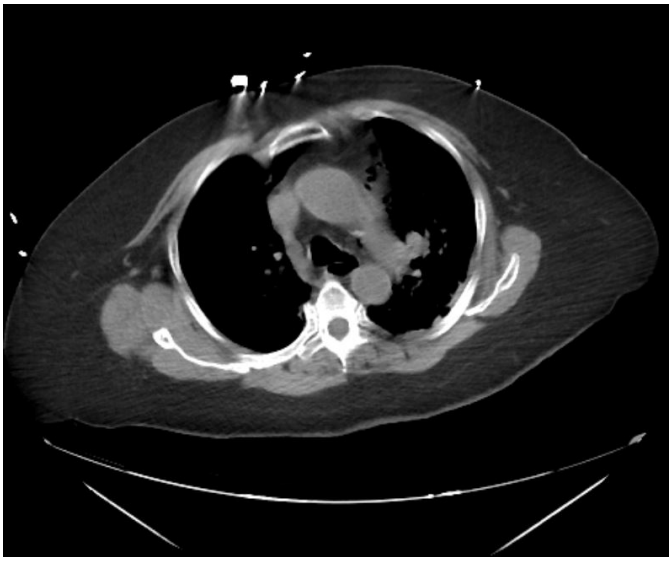
Methods: A 59 year old female with history of HTN, ESRD status-post kidney transplant on chronic cyclosporine and prednisone, OSA, and obesity who found to have biopsy confirmed hepatic flexure colonic adenocarcinoma; presented for a laparoscopic right hemicolectomy. Pre-operative laboratory findings two weeks prior to surgery were significant for a hemoglobin and hematocrit of 10.3 and 32.8, respectively. Physical exam including Mallampati score and pre-operative vitals were unremarkable. Upon arrival to the OR, ASA standard monitors were applied, and the patient was pre-oxygenated to an end-tidal oxygen concentration >80%. Pre-induction vitals were a BP 115/60, pulse of 75 sinus rhythm, Pulse oximeter 100%. Induction of anesthesia was uneventful and the patient was intubated in a single attempt with a 3 glidescope with a grade I view. The first set of vital signs after intubation were BP 80/60, pulse 100, and pulse oximeter 73%. Hypotension was treated with phenylephrine and ephedrine. Hypoxia was treated with hand-bag ventilation and 100% FiO2. Invasive blood pressure monitoring was obtained. After stabilization of vital signs, the case proceeded after discussion with the surgical team. Hypoxia, tachycardia, and hypotension continued intermittently throughout the case. Hypoxia was treated with repeated recruitment maneuvers. Hypotension and tachycarida were treated with fluids, beta blockers, and vasopressors. ABG obtained intra-operatively showed hypoxia and respiratory acidosis. Ventilator settings were adjusted accordingly. EBL was 250cc with 2 liters crystalloids and 1 liter colloids transfused. After completion of surgery, the patient was reversed and extubated to BiPAP. Upon arrival to the PACU, labs were drawn that showed H/H of 5.8/18.2. In PACU, CXR was obtained that showed rightward tracheal deviation, bovine arch aortic anatomy and pneumomediastinum. ECG showed normal sinus rhythm without ST changes or evidence of right heart strain. During this time the patient remained intermittently hypotensive and was treated with vasopressin and epinephrine pushes. Throughout the time in PACU, the patient's mental status did not change; she denied dyspnea or chest pain. Vital signs stabilized after transfusion of 2 units pRBCs. Patient was observed in ICU for 4 more days and ultimately discharged to home.

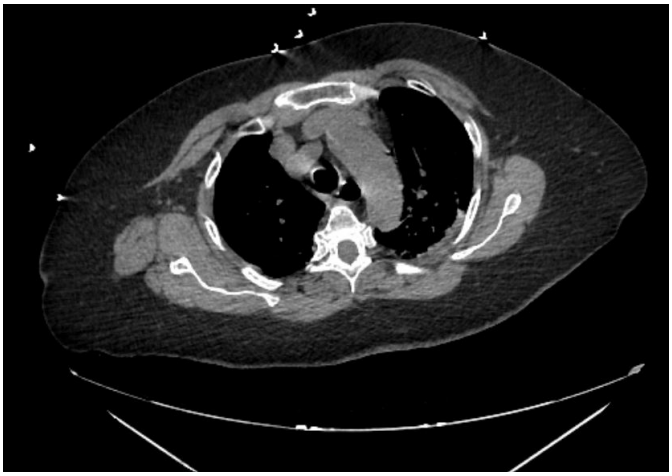
Conclusion: Iatrogenic tracheal injury is a rare, potentially fatal complication of intubation. The onset of non-specific symptoms has a wide range in timing from immediately after the injury to days later. In order to diagnose and begin management of tracheal disruption, a high clinical suspicion is required. The most common risk factor in tracheal disruption is emergency intubation. Other risk factors include variant tracheal anatomy, chronic corticosteroid use, and an inexperienced laryngoscopist. The most common clinical findings in spontaneous tracheal rupture is subcutaneous emphysema and pneumomediastinum. Direct visualization of the trachea via bronchoscopy is the diagnostic modality of choice when disruption is suspected; moreover endoscopic findings aid in guiding management. Patients who are spontaneously respirating with stable vital signs and a lesion length of <2 cm should managed conservatively. Whereas patients requiring mechanical ventilation, evidence of esophageal or mediastinal communication, and a lesion length of >2cm with transmural damaged should be managed more aggressively with surgery or fibrin glue. To date, the only statistically significant difference in mortality

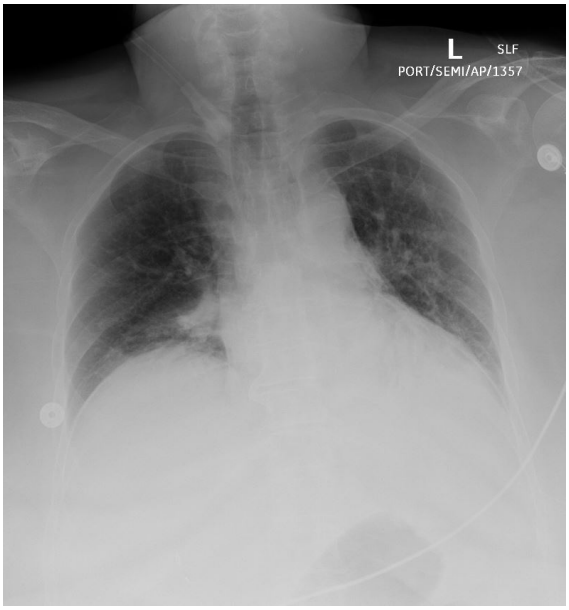
rates of patients going for surgical repair is the presence of mediastinitis.

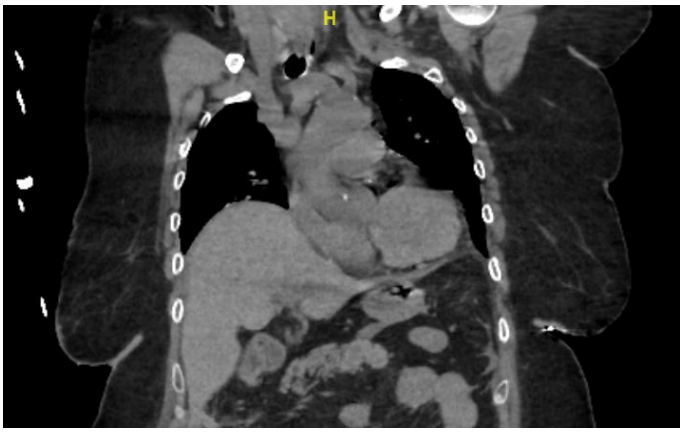
References: 1. Minambres E, Buro J, Ballesteros MA, et al. Tracheal rupture after endotracheal intubation: a literature systemic review. European Journal of Cardio-thoracic Surgery. 2009;35;1056-62 2. Paraschiv M. Iatrogenic tracheobronchial rupture. Journal of Medicine and Life. 2014;7;343-48 3. Kouritas V, Papgiannopoulos K, Lazaridis G. Pneumomediastinum. Journal of Thoracic Disease. 2015;7;1-1 4. Herrmann D, Volmerig J, Ahmad A, et al. Does less surgical trauma result in better outcome in management of iatrogenic tracheobronchial laceration? Journal of Thoracic Disease. 2019;11;4772-81 5. Gil T, Warmus J, Wlodarczyk J, Grochowski Z, et al. Iatrogenic injuries to the trachea and main bronchi. Polish Journal of Thoracic and Cardiovascular Surgery. 2016. 2;113-16 6. Mantovani S, Poggi C, Massimiliano B, et al. Surgical or conservative management of post intubation tracheal injury: when and how? Journal of Visualized surgery. 2020;6;4-4










H