IARS & SOCCA
2021 Annual Meetings

Medically Challenging Case Reports

TABLE OF CONTENTS

Airway Management

Airway Management - 1 A Case Report of Oxygen Desaturation During a Routine Esophagoduodenoscopy in a Trangender Male; Initial Concerns and Recommendations To the Practice of Chest Binding

Eugene Kim[1], Shivali Mukerji[2], Deen Debryn[3], Ryan K Price[3], Ala Nozari[3]

[1]Boston University School of Medicine, Boston, United States of America, [2]Boston University, Boston, MA, [3]Boston University School of Medicine, Boston, MA

Introduction: Over 1.4 million U.S. adults identify as transgender when gender identity differs from the sex assigned at birth (1). Although transgender patients face adverse health outcomes, they remain an under-studied population (2). A 2017 study surveyed 411 practicing clinicians and found that 80% had been involved in treating a transgender patient, but 80.6% had never received training on transgender care (3). The purpose of this report is to describe prolonged desaturation in one case of a transgender patient who wore a chest binder intraoperatively due to a lack of preoperative recognition.

Methods: A 19-year-old transgender male with anxiety and class 3 obesity presented for an esophagogastroduodenoscopy to evaluate a 2 year history of upper abdominal pain unresponsive to proton pump inhibitor therapy, with a plan for monitored anesthesia care. His medications included sertraline, pantoprazole, zolpidem, ergocalciferol, leuprolide, and testosterone cypionate. Preoperatively, the patient was instructed to remove all clothing and to don a patient gown while in the bathroom. The attending anesthesiologist then conducted the interview and exam in the preoperative holding area. The patient was induced with 250 milligrams of propofol, and reassuring respirations were noted by capnography. Respirations and oxygen saturation (SaO2) remained stable upon insertion of the endoscope. Four minutes later, the patient's SaO2 rapidly decreased to 50% and end-tidal capnography was lost. The endoscope was removed, and the patient was given 200 mg of propofol and 20 mg succinylcholine. His SaO2 recovered to 80% and 100% after 2 and 5 minutes respectively of ventilation with 100% inspired oxygen. Endoscopy was resumed with no airway device. No further oxygen desaturation was noted throughout the procedure, and the patient was closely monitored for signs of respiratory difficulty during an uneventful postoperative course. After full emergence, the patient was informed of the oxygen desaturation event. At this time, it was revealed that the patient had been wearing a chest binder throughout the operative procedure. The patient was counseled on the necessity to communicate the presence of this accessory prior to all future procedures.

Conclusion: In the clinical narrative, a healthy patient was observed to have prolonged oxygen desaturation after induction of anesthesia. Laryngospasm was suspected clinically due to the sudden absence of end-tidal carbon dioxide. Prolonged oxygen desaturation despite appropriate interventions suggests the contribution of additional factors. We speculate that the presence of a chest binder intraoperatively predisposed the patient to more rapid oxygen desaturation less responsive to typical therapy. A chest binder would introduce mechanical restriction to the patient's breathing due to its inherent design to compress. A previous study showed an association between regular chest binding and an overall reduction in functional residual capacity,4 which would prevent effective recruitment of alveoli and prolong shunting and time to recovery in the setting of oxygen desaturation. Routine protocols were followed regarding clothing transition for perioperative care. Although the patient was asked to remove all clothing, specific instructions were not provided regarding the removal of a chest binder. The presence of chest binding was also absent in the electronic health record, despite the documented presence of the patient's preferred gender, hormonal therapy regimen, and medical history. Ultimately, this case reflects the gap between practitioner knowledge and hospital guidelines and the practices of transgender patients. A cross-sectional survey of transgender individuals who practice chest binding found that despite 89% reporting at least one negative symptom, only 15% sought care related to binding.5 This stresses the importance of education of clinicians to create an environment optimal for transgender therapeutic alliance and clinical care. At this time, there are no recommendations for patients who engage in chest binding for the perioperative period. In reviewing existing literature

and the potential for atelectasis with external compression, we would consider that patients refrain from chest binding for 12 to 24 hours before surgical procedures, resume no sooner than 24 hours after ambulation, and participate in diagnostic incentive spirometry pre-and postoperatively.

References: 1.Flores AR, Herman JL, Gates GJ, et al. How Many Adults Identify as Transgender in the United States? Los Angeles, California: The Williams Institute; 2016. Accessed September 22, 2020. 2.Reisner SL, Poteat T, Keatley J, et al. Global Health Burden and Needs of Transgender Populations: A Review. Lancet. 2016;388(10042):412-436. doi:10.1016/S0140-6736(16)00684-X 3.Dubin SN, Nolan IT, Streed CG, Greene RE, Radix AE, Morrison SD. Transgender health care: improving medical students' and residents' training and awareness. Adv Med Educ Pract. 2018;9:377-391. doi:10.2147/AMEP.S147183 4.Chung SA, Yuan H, Chung F. A systemic review of obstructive sleep apnea and its implications for anesthesiologists. Anesth Analg. 2008;107(5):1543-1563. doi:10.1213/ane.0b013e318187c83a 5.Payne K, Lipshultz LI, Hotaling JM, Pastuszak AW. Obstructive Sleep Apnea and Testosterone Therapy. Sex Med Rev. Published online July 4, 2020. doi:10.1016/j.sxmr.2020.04.004

Airway Management - 2 A patient with Duchenne Muscular Dystrophy with severe restrictive lung disease presents for dental extractions - a case report

Danielle M Desjardins[1], Leila Zuo[1]

[1]Oregon Health and Science University, Portland, OR

Introduction: Presentation: 22 year-old male with Duchenne Muscular Dystrophy (DMD) presenting for removal of impacted 3rd molars. Comorbidities included decreased mobility (wheelchair bound, s/p spinal fusion), restrictive lung disease (FVC 35% at baseline), adrenal insufficiency, and obstructive sleep apnea. The patient presented at his baseline.

Methods: Anesthetic challenge: General anesthetics (GA) are problematic in this population. The airway is typically compromised by macroglossia. Patients have reduced functional residual capacity so time to desaturation after induction is reduced; in patients with FVC<50% there is high risk of postoperative pulmonary complications, and in those with FVC<30% the risk is further increased. The use of depolarizing neuromuscular blockade is contraindicated due to the risk of hyperkalemia. Residual non-depolarizing neuromuscular blockade could compromise postoperative pulmonary status further. Controversy remains regarding use of inhaled anesthetics, but they are generally avoided given the risk of rhabdomyolysis. Additionally, DMD patients often have cardiomyopathy, further complicating management. The overall challenge was to avoid GA in this patient.

Results: How it was done: After a full discussion with the patient and family, we decided sedation with local anesthetic was the safest anesthetic plan. As a safeguard, inhaled anesthetics were removed, the machine was flushed, and filters were placed in the circuit; a Glidescope was available in case we needed to proceed with intubation. We proceeded with moderate sedation with propofol and dexmedetomidine. Given his reduced pulmonary function and need for oral access, the patient was placed on high-flow nasal cannula to provide supplemental oxygen and provide positive pressure. Opioids were kept to a minimum: 50mcg fentanyl given with local anesthetic injection. The anesthetic and dental teams talked the patient through his discomfort for much of the procedure- music of the patient's choosing was played to help maintain calm. Overall the patient did well with a combination of sedation, local anesthesia, and coaching by the surgery and anesthesia teams.

Conclusion: Conclusion: Preparation for this apparently 'simple' case in a complex patient was key to our success. By addressing the known pulmonary changes in DMD, we employed HFNC in a dental surgery - a new use for this device. By using all the tools available to us as anesthesiologists we were able to avoid an intubation and the possibility of a prolonged hospital stay for our patient.

References: Cureus. 2018;10(11):e3639. Published 2018 Nov 26. JAMA. 2016;315(13):1354-1361. Int J Pediatr Otorhinolaryngol. 2018;108:151-154. J Oral Maxillofac Surg. 2015;73(6):1058-1064. Complement Ther Clin Pract. 2018;31:158-163.

Airway Management - 3 Nasal mask-face tent provided oxygenation and reduced aerosol/droplet spread in an obese paraplegic patient with an expired COVID-19 test during suprapubic-catheter insertion

Devesh A Patel[1], Chandani Vekaria[1], James Tse[1]

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

Introduction: Patients under 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. A pediatric facemask has been shown to provide nasal CPAP oxygenation in deeply sedated obese patients. A nasal mask-face tent provided pre/apneic nasal oxygenation and reduced aerosol/droplet spread in a COVID-19 positive patient. We used it to provide nasal CPAP ventilation/oxygenation in an obese patient with an expired COVID test during urgent suprapubic catheter insertion.

Methods: A 65-year-old morbidly obese male, BMI 37.3 kg/m2, with NIDDM, HTN, atrial fibrillation, CHF, COPD, spinal stenosis, paraplegia, and severe urinary retention presented for urgent insertion of suprapubic catheter amid the COVID-19-19 pandemic. His COVID test was negative 10 days prior. An infant facemask was secured over his nose and connected to the anesthesia machine via an extended breathing circuit (Fig.1). HMEF/viral filters were placed between the nasal mask and the gas sampling catheter expiration circuit. The APL valve was adjusted to deliver 5 cm H2O CPAP with 4L/min O2. To reduce aerosol/droplet spread, his mouth was covered with a clear plastic sheet (face tent) and a suction catheter was placed under the plastic sheet for continuous suctioning (Fig.2). He was deeply sedated with a propofol infusion. He maintained spontaneous nasal ventilation and 99-100% SpO2 throughout.

Conclusion: This simple nasal mask-face tent maintained continuous CPAP ventilation/oxygenation in a morbidly obese paraplegic patient with COPD and an expired COVID test for emergent insertion of a suprapubic catheter. Combined with continuous suctioning, it reduced aerosol/droplet spread. It may optimize patient safety and provide additional provider protection amid the COVID-19 pandemic.

References: www.TSEmask.com; 2. SAMBA 28th AM, MCC, 2013; 3. ASA AM: (MC1280), 2020

Airway Management - 4 Orthotopic Liver Transplant in a Patient with Treacher Collins Syndrome and Known Difficult Airway

Cameron Sumner[1], Brittany Bunker[2], Edward Gillig[2], Amanda Kleiman[2]

[1]University of Virginia Department of Anesthesiology, Charlottesville, VA, [2]University of Virginia, Charlottesville, VA

Introduction: Treacher Collins Syndrome (TCS) is a rare genetic disorder affecting craniofacial development of the first and second branchial arches leading to maxillomandibular and malar hypoplasia, periorbital soft tissue anomalies, and ear anomalies [1]. This combination can result in a small oral aperture, a high arched palate, and temporomandibular joint abnormalities making mask ventilation, direct laryngoscopy, and endotracheal intubation challenging [1]. Staged surgical management of craniofacial abnormalities is common to protect the airway including mandibular distraction, zygomatic and chin augmentation, and even tracheostomy depending on severity of airway compromise [2]. TCS is associated with difficult airway management in children for which nasal fiberoptic intubation has been well described as a successful airway management method, but there is a dearth of literature on adult airway management [3]. While nasal fiberoptic intubation has been proven successful in patients with TCS, the coagulopathy and respiratory dysfunction present in end stage liver disease (ESLD) can further complicate airway instrumentation with little data to guide perioperative management [4]. In this case, we describe the perioperative airway management of a patient with TCS with ESLD undergoing orthotopic liver transplantation (OLT).

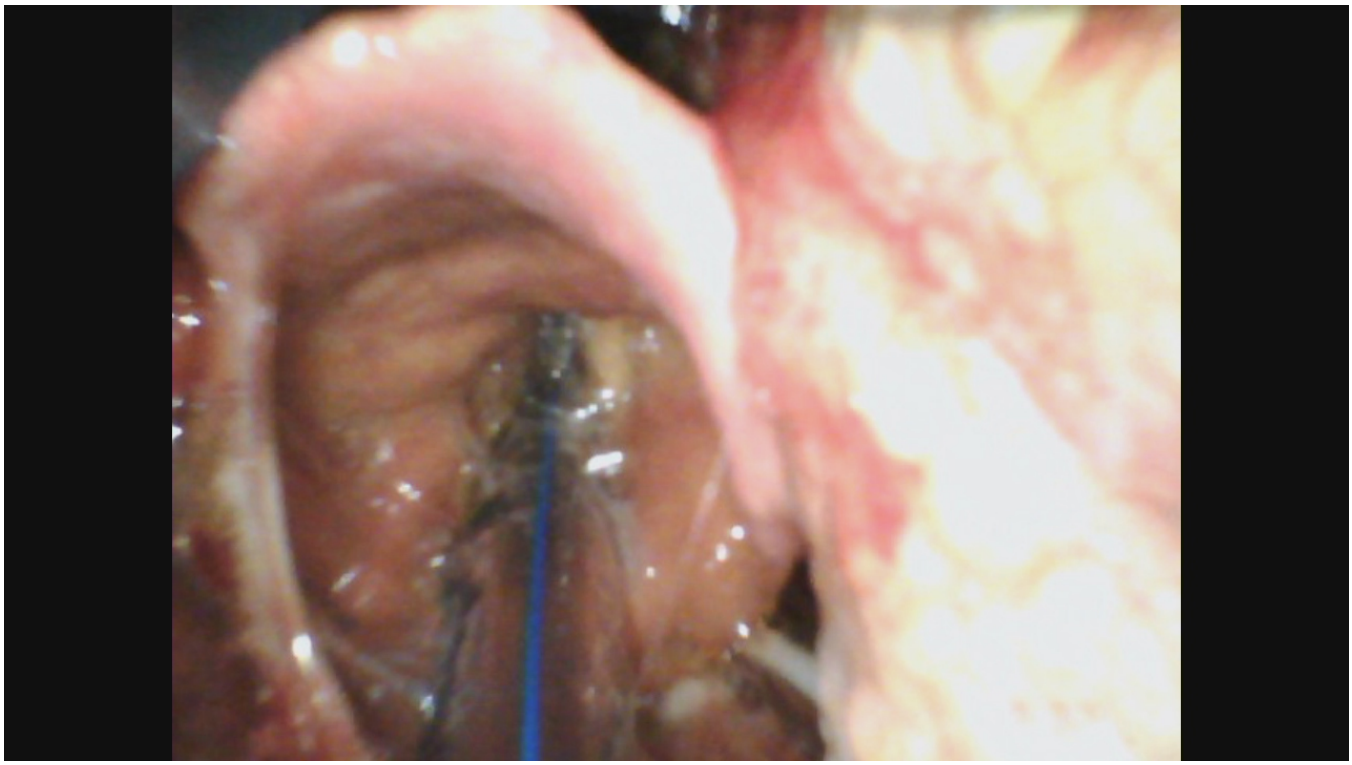
Methods: A 56-year-old man with history of TCS and ESLD secondary to primary sclerosing cholangitis and alcohol-related cirrhosis with a Model for End-Stage Liver Disease score of 37 who presented for OLT. Medical history was notable for partial complex seizures, obstructive sleep apnea, alcohol abuse, and hepatic encephalopathy. He had no history of paracenteses, spontaneous bacterial peritonitis, esophageal varices, or other cirrhotic stigmata. Surgical history was notable for prior facial reconstruction with cosmetic chin implants without jaw advancement surgery at which time he was told he was a difficult intubation. The patient further reported small nasal passages and a small glottic opening were noted on a flexible laryngoscopy by an otolaryngologist. Airway exam was notable for Mallampati IV, short thyromental distance, poor dentition, reduced neck extension, limited mouth opening, and a large beard. He was counseled preoperatively about his high-risk airway and the possibility of an awake intubation. When the patient presented to the OR, the difficult airway cart was present and a fiberoptic scope was prepared. The patient's beard was taped to aid masking prior to induction with 80mg Lidocaine and 250mg Propofol. After determining the patient could be mask ventilated with two providers, patient was paralyzed with 50mg of Rocuronium and successful fiberoptic oral intubation was performed. Abnormal airway anatomy was visualized with redundant tissue surrounding a small glottic opening. After successful intubation, video laryngoscopy was attempted to determine if it could be used in the future. Laryngoscopy with the Glidescope S4 blade yielded a Cormack-Lehane Grade 1 view. After airway management patient underwent uncomplicated OLT.

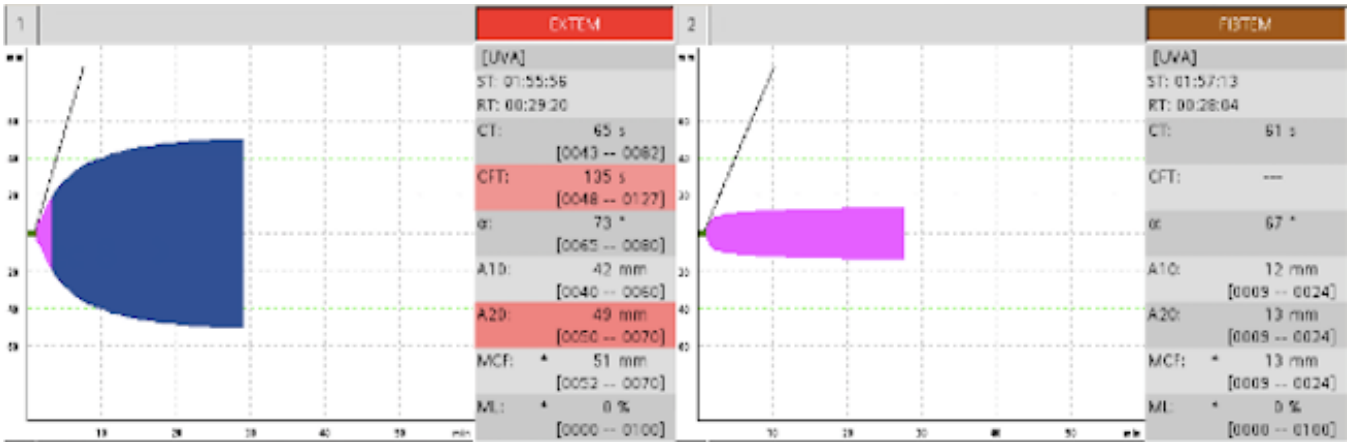
Conclusion: This case presented a unique combination of challenges including a known difficult airway in the setting of ESLD predisposing him to hypoxemia and bleeding [5]. Given TCS is a relatively rare disorder, the literature surrounding the management of the airway of a TCS patient is sparse with the vast majority of data found in the pediatric population [1]. Many patients with TCS will have undergone craniofacial reconstructive surgery to some capacity depending upon the severity of airway compromise as an infant [6,7]. Therefore, it is important to obtain an accurate surgical history to aid in airway management. There is also evidence to suggest that, unlike Pierre Robin sequence craniofacial abnormalities, the airway in TCS patients increases in difficulty as the patient ages [6]. Thus, a thorough airway evaluation and anesthetic history is vital prior to attempting endotracheal intubation and it is prudent to be prepared for a difficult intubation with advanced airway equipment and additional providers if necessary. In our case, an experienced anesthesiologist and senior level resident were able to navigate the patient's difficult airway and successfully

complete endotracheal intubation orally with a fiberoptic bronchoscope. This serves as an example of successful airway management in a patient with TCS in the setting of ESLD.

References: [1] Aljerian A, Gilardino MS. Treacher Collins Syndrome. Clin Plast Surg. 2019 Apr;46(2):197-205 [2] Cobb AR, et al., The surgical management of Treacher Collins syndrome. Br J Oral Maxillofac Surg. 2014 Sep;52(7):581-9 [3] Lin TC, et al., Perioperative airway management in a child with Treacher Collins syndrome. Acta Anaesthesiol Taiwan. 2009 Mar;47(1):44-7.

[4] Fauconnet P, et al., Hepatopulmonary syndrome: the anaesthetic considerations, European Journal of Anaesthesiology: December 2013 - Volume 30 - Issue 12 - p 721-730 [5] Naeije R, Melot C, Hallemans R, Mols P, Lejeune P., Pulmonary hemodynamics in liver cirrhosis. Semin Respir Med. 1985;7:164–71. [6] Hosking J, et al., 2012. Anesthesia for Treacher Collins syndrome: a review of airway management in 240 pediatric cases. Pediatric Surgery, 22(8), pp. 752-758. [7] Sinkueakunkit A, et al., 2013. Outcome of anesthetic management for children with craniofacial deformities. Pediatric International, 55(3), pp. 360-365.





Airway Management - 5 Post-COVID-19 Tracheal Stenosis requiring Tracheal Resection: A Case Report

James Beesley[1], Reena Parikh[1]

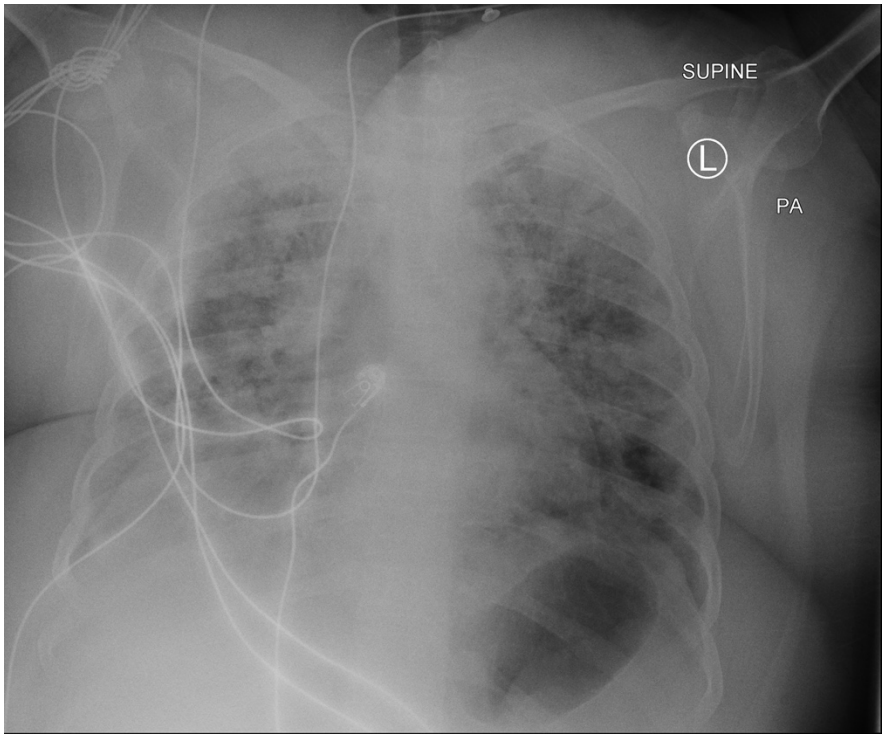
[1]Albany Medical Center, Albany, United States of America

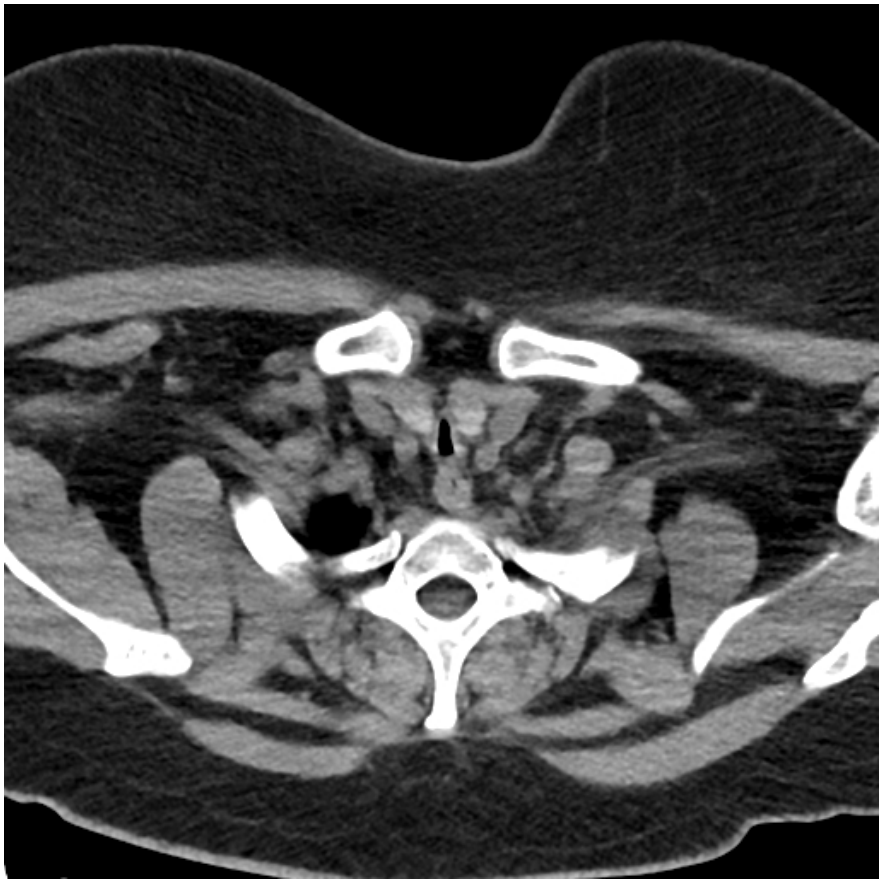
Introduction: Tracheal stenosis is a known complication of intubation, usually as a result of submucosal ischemia1, with an occurrence of 1-2%2, and increasing incidence as the duration of intubation increases. The impact of COVID-19 on the development of tracheal stenosis is unknown, but the incidence of COVID-19 patients who require intubation and mechanical ventilation is high - 9.8-15.2%3. There has been an anecdotal increase in tracheal stenosis in extubated COVID-19 patients even when intubated duration was short. This could have implications for anesthesia providers taking care of patients who were intubated in the past due to COVID -19, in terms of being prepared for potential tracheal stenosis.

Methods: A 39 year old female with a history of obesity-BMI 39.8, asthma, diabetes, relapsing remitting multiple sclerosis, hypertension, hyperlipidemia, and lumbosacral radiculopathy presented to the emergency department for shortness of breath in mid April 2020. She was diagnosed with acute hypoxic respiratory failure and was intubated in the emergency department. O2 saturation was between 55-70% prior to intubation. Chest X-ray showed bilateral patchy infiltrates consistent with pneumonia, and nasal swab was positive for COVID-19 via PCR. She was admitted to the medical ICU, and treated for respiratory failure. As part of a trial from Mayo Clinic, she also received convalescent plasma. The total length of intubation was 11 days. She recovered and was discharged from the hospital in early May. 3 months later, in August 2020, she presented with complaints of shortness of breath and stridor. She was found to have subglottic stenosis secondary to her intubation and was subsequently taken to the operating room for flexible bronchoscopy, laser ablation, and balloon dilation. Despite this, even a 6.0 ETT was not able to pass the stricture. Her stricture was 2.5 to 3cm in length, at a distance of 2.5 to 3cm from vocal cords, and the distal airways were noted to have thick secretions. She showed marked improvement after surgery for approximately 1 week, and then redeveloped identical symptoms. She returned for ablation and dilation approximately every two weeks for a total of 6 ablations and dilations, with a similar pattern of initial relief followed by quick restenosis. An LMA was the only way to secure her airway as an ETT could not pass the stricture. She elected to undergo tracheal resection of 3 tracheal rings with primary anastomosis in December 2020. This resection was successful and the patient continues to be symptom free.

Conclusion: This patient had multiple comorbidities that may have impacted her likelihood of developing tracheal stenosis after intubation, including diabetes, multiple sclerosis, and COVID-19. Because of the high rate of intubations in COVID-19 patients, and the possible increased risk of tracheal stenosis in these patients, there may be a significant increased incidence of tracheal stenosis in the near future. This is an evolving situation as we are starting to see more patients post intubation due to COVID-19. These patients may present unexpectedly with an element of tracheal stenosis if they are in the OR for other types of surgeries. This would imply that anesthesiologists should be prepared for tracheal stenosis and have difficult airway devices ready. As a result, we may also see more tracheal laser ablations, balloon dilations and perhaps tracheal resections.

References: 1. Seminars in Thoracic and Cardiovascular Surgery, 21:284-289, 2009 2. Acta Anaesthesiology Scandanavica, 63(7):905-912, 2019 3. The Lancet, 395:497-506, 2020


SUPINE
L
PA



Airway Management - 6 Tracheal Gunshot Injury: Airway Securement and One Lung Ventilation Considerations

Kenji Tanabe[1], Brian Keech[2]

[1]University of Colorado, Denver, CO, [2]University of Colorado School of Medicine, Denver, CO

Introduction: Traumatic tracheal lacerations are rare injuries with high morbidity and mortality that require unique airway considerations. This case report describes a tracheal laceration injury due to a gunshot wound and the subsequent airway management required for operative repair.

Methods: A 29 year-old male presented to the emergency department with a tension pneumothorax after sustaining a single gunshot wound to his right upper back. A surgical chest tube was placed and the patient was subsequently intubated for hypoxia using a video laryngoscope. Following transfer to the intensive care unit, the patient's pneumothorax worsened despite an adequately placed chest tube. Flexible bronchoscopy through the existing endotracheal tube demonstrated two full thickness tracheal lacerations two centimeters above the carina. The existing endotracheal tube tip was positioned superior to the tracheal lacerations. Given the inability to adequately oxygenate the patient and concern for worsening pneumothorax, the surgical team elected to pursue an emergent tracheal repair via right thoracotomy. A Seldinger fiberoptic technique was used to facilitate one lung ventilation in the operating room. A flexible bronchoscope was introduced through the existing endotracheal tube and carefully advanced into the left mainstem bronchus. The endotracheal tube was then advanced over the bronchoscope into the left mainstem bronchus. One lung ventilation was achieved and the surgical team repaired the two tracheal lesions. The patient was subsequently extubated in the operating room. Post-operatively, the patient underwent three unremarkable surveillance bronchoscopies and was discharged from the intensive care unit on post-operative day ten.

Conclusion: Tracheal lacerations are rare, taking place in an estimated 0.5-2% of all blunt traumas and 6% of penetrating neck traumas. The airway securement of tracheal lacerations involves unique considerations, including avoiding further iatrogenic tracheal damage and placing the endotracheal cuff distal to the tracheal disruption to avoid positive pressure across the injury. The Airway Disruption Algorithm published by the American Society of Anesthesiology Committee on Trauma and Emergency Preparedness (COTEP) recommends using fiberoptic intubation when allowable for tracheal lacerations. However, retrospective studies demonstrate that 60-73% of patients diagnosed with tracheal lacerations are intubated directly, without the aid of a fiberoptic scope, as was the case for this patient. Additionally, most patients with thoracic tracheal lacerations will require single lung ventilation for operative repair. While the COTEP algorithm recommends lung isolation with a double lumen tube or endobronchial blocker, we elected to guide the existing endotracheal tube into the left mainstem bronchus. A double lumen tube, with its increased width and stiffness, was felt to increase the risk of iatrogenic tracheal trauma. An endobronchial blocker would not have isolated the tracheal lacerations from positive pressure ventilation. As demonstrated by this case, mainstem intubation using the existing endotracheal tube can safely facilitate one lung ventilation.

References: 1. Kiser, Andy C., Sean M. O,ÄôBrien, and Frank C. Detterbeck. 'Blunt tracheobronchial injuries: treatment and outcomes.' The Annals of thoracic surgery 71.6 (2001): 2059-2065. 2. Johnson, Scott B. 'Tracheobronchial injury.' Seminars in thoracic and cardiovascular surgery. Vol. 20. No. 1. WB Saunders, 2008. 3. Prokakis, Christos, et al. 'Airway trauma: a review on epidemiology, mechanisms of injury, diagnosis and treatment.' Journal of cardiothoracic surgery 9.1 (2014): 117. 4. Hagberg, Carin A., and Olga Kaslow. 'Difficult airway management algorithm in trauma updated by COTEP.' ASA Newsletter 78.9 (2014): 56-60. 5. Rossbach, Mario M., et al. 'Management of major tracheobronchial injuries: a 28-year experience.' The Annals of thoracic surgery 65.1 (1998): 182-186.

Airway Management - 7 First Case of Missing Teeth after Bronchoscopy with a Laryngeal Mask Airway

Danielle Levin[1], Michael Schoor[1]

[1]St. Elizabeth's Medical Center, Brighton, MA

Introduction: About 1 in 4537 patients undergoing general anesthesia experience dental trauma. Patients most at risk for dental injury during anesthesia are those with preexisting poor dentition and those that have one or more risk factors for difficult laryngoscopy and tracheal intubation. Dental injury during general anesthesia with a laryngeal mask airway (LMA) is possible but quite rare. There are no formal recommendations for evaluating where missing teeth might be. The patient provided informed consent for the publication of this case report.

Methods: A 51-year-old female presented for flexible bronchoscopy, radial endobronchial ultrasound, endobronchial forcep biopsy, and endobronchial ultrasound-guided transbronchial needle aspiration to be performed under general anesthesia. She had a past medical history of lung carcinoma with brain metastasis, hypertension, and seizure disorder. Her medications included alprazolam, zolpidem, levetiracetam, oxycodone, bisacodyl, and polyethylene glycol. She had no known drug allergies. The patient drank 3-4 alcoholic beverages per day and was a former user of marijuana. She was 175.2 cm tall and weighed 54.4kg. During the preoperative evaluation on the day of surgery, the patient admitted. to having one chipped left upper molar and one chipped right upper molar, as well as several loose lower incisors. She denied any removable dental appliances. On physical exam, the patient had a Mallampati Class III airway, thyromental distance of 4 finger breadths, full range of motion, normal neck circumference, and ability to protrude mandible. After induction of anesthesia with lidocaine, propofol, and fentanyl, an I-GEL supraglottic airway size 4 was easily inserted and secured with tape around the mouth. Anesthesia was maintained with oxygen, propofol, and remifentanil infusion. The surgical procedure was uneventful despite a significant amount of manipulation of the supraglottic airway by the surgical team during the bronchoscopy. The patient was awakened following the procedure and the LMA was removed after meeting standard extubation criteria. Postoperatively, the patient's vitals were stable. She coughed several times and then appeared comfortable. In phase-2 post anesthetic recovery, the patient reported that her front incisor bridge was missing. All members of the anesthesiology and pulmonology team, as well as the Patient Care Advocate, came to the patient's bedside and offered empathetic listening to the concerns and distress of the patient and her family. A plan was made to find the missing teeth. The floor and trash of the bronchoscopy suite were searched entirely. During this time, a chest and abdominal x-ray were performed. The abdominal x-ray demonstrated a blunt-appearing 3-tooth crown in the area of the stomach. The gastroenterology team was consulted, who recommended for the patient to take senna glycoside and polyethylene glycol. Serial abdominal exams and x-rays were then to be performed. The patient had multiple bowel movements overnight, and the following morning, a repeat abdominal x-ray demonstrated the crown in the right lower quadrant of the abdomen, making progress through the gastrointestinal tract as expected, likely in the cecum or distal small bowel. Patient continued to be asymptomatic and was advised to restart a regular diet. Repeat imaging of the abdomen 3 weeks later did not show the crown. One month later, the patient continued to be asymptomatic, but the missing teeth were never found in the stool.

Conclusion: Although dental injuries during the perioperative period have been extensively reported in literature, the actual ingestion of dental prostheses during the perioperative period is rare. Only three cases of ingested dental prostheses have been reported in patients undergoing general anesthesia with an endotracheal tube. Only one case report was found of dental prostheses ingestion in a patient who had general anesthesia with a laryngeal mask airway. As far as we are aware, this is the first reported case of ingestion of a dislodged dental prosthesis during a bronchoscopy with a laryngeal mask airway. Through this case presentation, we hope to raise awareness of the importance of a detailed oral exam during the preoperative evaluation and remember to always have a patient-centered approach that involves empathy and strong teamwork to ensure patient comfort and safety.

References: 1. Perianesthetic dental injuries: frequency, outcomes, and risk factors. Anesthesiology. 1999;90(5):1302-1305. 2. Ingestion of Dental Prosthesis of A Patient with an LMA Under General Anesthesia: A case report. Arch Anesthesiol. 2019;2(1):25-27.

Figure 1: KUB on Post-operative Day 0

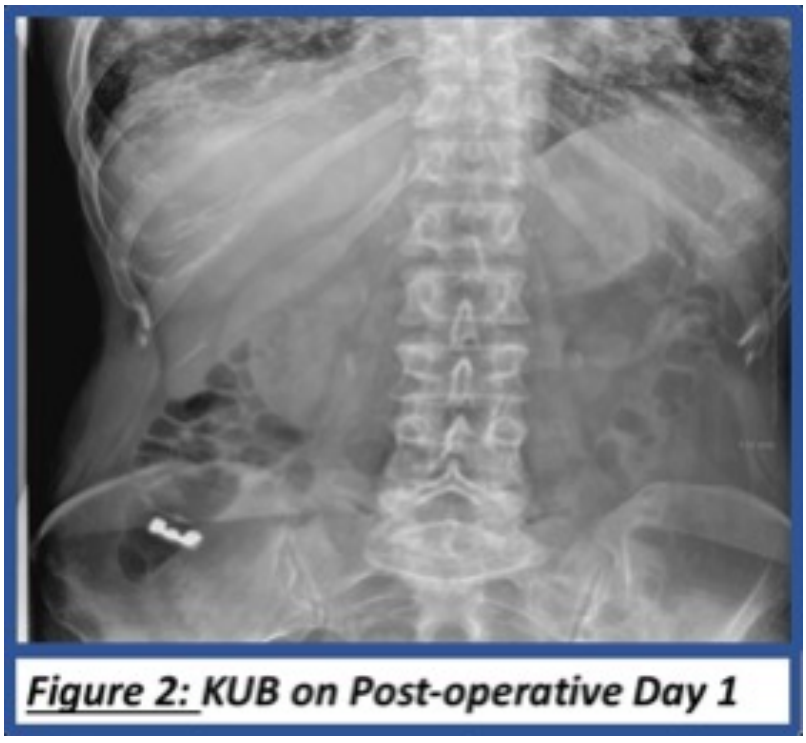


Figure 2: KUB on Post-operative Day 1

Airway Management - 8 Management of Lung Isolation in a Patient with Empyema Following Near-Drowning with an Incidental Finding of a Tracheal Bronchus

Daniel Rubinger[1], Stephanie Jankovic[1], Tichaendepi Mundangepfupfu[1]

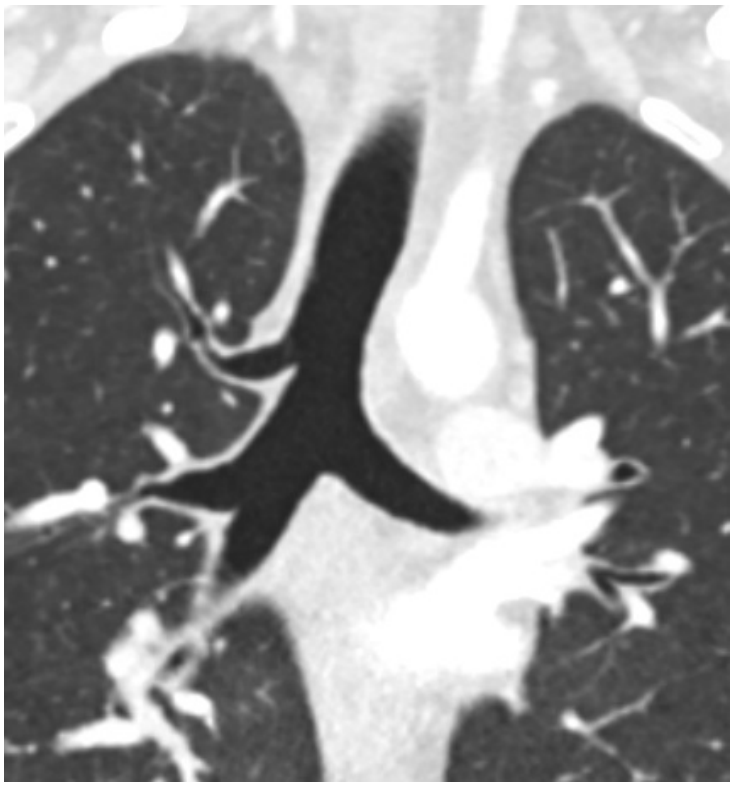
[1]University of Rochester, Rochester, NY

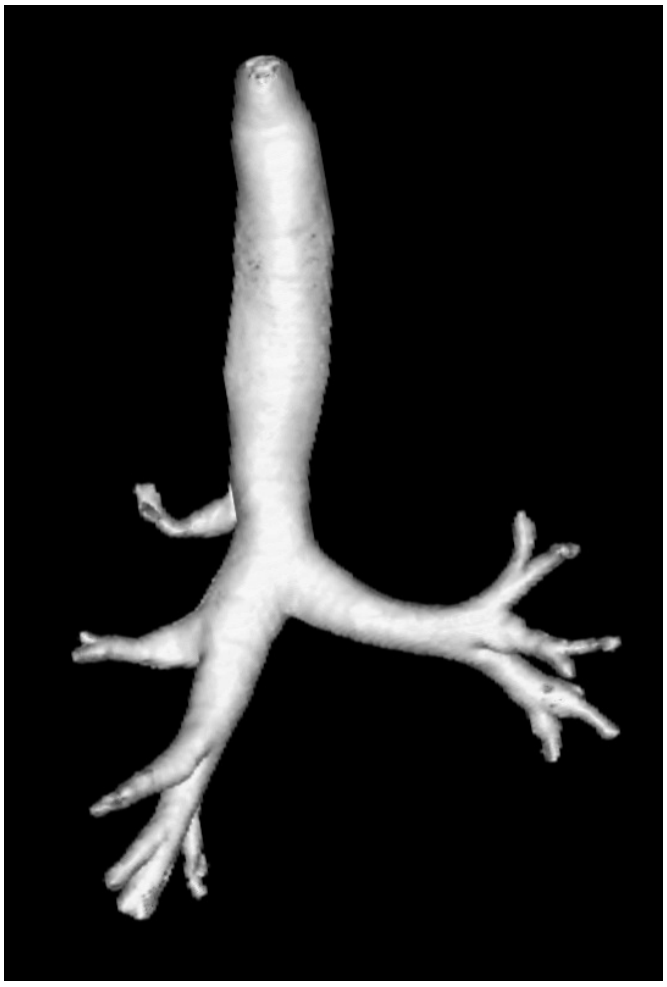
Introduction: A tracheal bronchus is a rare congenital anomaly where there is a tracheal outgrowth above the main carina directed towards the upper lobe. Tracheal bronchi are often asymptomatic and found incidentally on imaging or bronchoscopy. Here, we discuss lung isolation in a patient with an empyema following a near-drowning event. He was also incidentally found to have a tracheal bronchus and we address management of this finding.

Methods: A 28-year-old male with a past medical history of unknown cardiac surgery and TE fistula repair in childhood, presented to the emergency department with progressively worsening shortness of breath approximately 1 month after a near-drowning event. CT chest demonstrated significant left-sided parenchymal consolidation and several loculated fluid collections which did not improve after IR pigtail placement. He was admitted to the Medical ICU for management of acute hypoxemic respiratory failure with BiPAP. On hospital day 5, he was brought to the OR for VATS decortication of left-sided empyema. On preoperative assessment, he was tachypneic with an SpO2 93% on 4L nasal canula. His SpO2 did not increase when he was placed on non-rebreather mask. After induction of anesthesia, a 9.0 single lumen ETT was placed. Bronchoscopy was subsequently performed and a right-sided tracheal bronchus was visualized. A bronchial blocker was then placed in the left mainstem bronchus for lung isolation. The case progressed uneventfully and he was brought to the Surgical ICU intubated. He was successfully extubated on hospital day 7 and remained on the Medicine unit until being discharged on hospital day 11. Three months after the hospitalization, the patient presented for follow up with significant radiological improvement on repeat CT as well as minimal respiratory complaints.

Conclusion: Most patients with tracheal bronchi are asymptomatic. Estimates for the incidence of major anatomical variations of the tracheobronchial tree range between 0.1% - 5%. Initially described by Sandifort in 1785, a tracheal bronchus is an upper lobe bronchus arising from the trachea. The most common type of tracheal bronchus is a displaced tracheal bronchus, which occurs when one branch of the upper lobe is replaced by an aberrant bronchus originating from the trachea. When the entire upper lobe is supplied by the accessory bronchus, it is termed a 'pig bronchus' or 'bronchus suis' as this is the normal anatomy seen in pigs. [1] The vast majority of tracheobronchial tree anomalies are right-sided. Our patient was found to have an asymptomatic right-sided displaced tracheal bronchus. Anesthetic management of patients with a tracheal bronchus presents unique oxygenation and ventilation challenges. During intubation, the wall of the ETT may obstruct the tracheal bronchus. Additionally, the tracheal bronchus may be directly intubated. If lung isolation is required, the presence of a tracheal bronchus may also affect placement of a double lumen tube (DLT) or bronchial blocker. Our patient presented to the OR in acute hypoxemic respiratory failure. He had an empyema and had poor oxygen saturation on supplemental oxygen. While the initial anesthetic plan involved placing a DLT for lung isolation, there was concern that placing the DLT would delay securing the airway in a patient with possibly poor respiratory reserve. Therefore, it was decided to initially place a single lumen ETT (SLT) to improve his oxygenation, and once the patient was more stable, exchange the SLT for a DLT. After preoxygenation, rapid anesthetic induction and placement of the 9.0 SLT, the patient desaturated to SpO2 of 79%. Bronchoscopy was subsequently performed and a tracheal bronchus was identified distal to the end of the SLT. A bronchial blocker was then placed for lung isolation. In conclusion, managing the airway of a patient with a tracheal bronchus may require special considerations. While the presence of a tracheal bronchus is often an incidental imaging finding, it may affect airway management for procedures requiring lung isolation.

References: Ruchonnet-Metrailler, I, Abou Taam, R, De Blic, J. Presence of tracheal bronchus in children undergoing flexible bronchoscopy. Respir Med 2015; 109: 846–850.





Airway Management - 9 A Challenging Presentation of Late Onset Inspiratory Stridor Post Thyroidectomy Due to Delayed Recurrent Laryngeal Nerve Palsy

Tehmur Raja[1], Harsh Nathani[1], Gennadiy Voronov[1], Erica Spano[1]

[1]John H. Stroger, Jr. Hospital of Cook County, Chicago, IL

Introduction: Inspiratory stridor is an uncommon but potentially fatal postoperative complication of thyroid surgery indicating airway obstruction. It is produced by the rapid, turbulent flow of air through a narrowed or partially obstructed segment of the extrathoracic upper airway [1]. A thorough history and physical, with particular focus on the time of onset, can help substantiate a differential diagnosis and life saving therapy. Onset of stridor within the first 24-hours of thyroidectomy is predominantly due to laryngeal/vocal cord edema, compressing hematomas, or recurrent laryngeal nerve palsy/paralysis (RLNP). Although rare, hypocalcemic laryngospasm is typically suspected when onset of presentation exceeds the initial 24-hour period [2]. Despite the common causes attributed to the aforementioned timeframes, all possible differentials should still be considered. We report a 48-year-old patient presenting with respiratory distress and stridor more than 24-hours post-thyroidectomy due to delayed laryngeal nerve palsy.

Methods: A 48-year-old female with a 2-year history of T4bN1M0 papillary thyroid carcinoma (PTC) previously underwent a debulking procedure with an estimated 80% gross mass resection. The patient presented with complaints of progressive dysphagia, dysphonia, and frank hemoptysis and elected to undergo a secondary debulking procedure. Due to the extent of the carcinoma, the right RLN was compromised; resulting in a paretic right true vocal fold (TVF). Moreover, the patient was diagnosed with post-surgical hypothyroid and hypoparathyroid disease augmented with Levothyroxine and calcium. During the procedure, successful induction and intubation was achieved; the patient was intubated with an 8.0 mm ET tube. The surgeon elected to forgo any neuromuscular blocking agents. Exploratory assessment of the tumor size and location indicated that complete removal would likely pose serious risk; 30% of the remaining mass was successfully removed. The patient was revived and safely extubated. On POD 1, physical examination was notable for inspiratory stridor and respiratory distress; with saturations dropping to the mid-90's. Immediate evaluation by fiberoptic laryngoscopy visualized the right TVF fixed in the median position and the left TVF in the paramedian. No masses, edema, or signs of infection were noted. The need for urgent tracheostomy was discussed with her family and she promptly underwent the procedure with successful resolution of symptoms. Labs drawn in the afternoon revealed a blood calcium of 6.3 mg/dL. The patient was transferred to the SICU for central access, where a calcium gluconate drip was administered with the desired calcium goal of 8 mg/dL. The patient was then successfully discharged. A 6-week follow up with laryngoscopy illustrated slight improvement of left TVF movement.

Conclusion: Nearly 150,000 thyroidectomies are performed every year in the U.S by conservative estimates [3]. In light of advancing surgical techniques and medical technologies, it has evidently been established as a safe procedure. Yet, postoperative hypocalcemia and airway difficulties remain significant complications. Of these complications, bilateral RLNP is one of the most pernicious contributing factors; a leading cause of acute airway obstruction with an incidence of 0.39% [2]. Our unique case reveals that RLNP does not necessarily manifest within the 24-hours following thyroidectomy, as was previously assumed. It can have a delayed onset of presentation. Although epidemiological data is scarce, there are reports of similar incidents that have occurred as late as 5 weeks post-op. The mechanism of injury is unknown but is suspected to be provoked by mechanical trauma, leading to either devascularization or postoperative inflammatory processes [4]. Generally, severe hypocalcemia precipitating laryngospasm is thought to be the obvious cause of late-onset stridor, due to inadvertent parathyroidectomy [2]. Our patient was found to have a blood calcium of 6.3 mg/dL; when considering the time frame, hypocalcemic laryngospasm would typically be assumed as the underlying cause of airway obstruction. However, laryngoscopy demonstrated new findings of left TVF dysfunction, confirming the diagnosis of RLNP. When encountering late-onset stridor, it is vital to include delayed RLNP as a possible

differential. Premature exclusion could potentially lead to an untimely diagnosis and consequently fatal outcomes.

References: 1) Dezube, Rebecca. “Stridor - Pulmonary Disorders.” Merck Manuals Professional Edition, Merck Manuals, www.merckmanuals.com/professional/pulmonary-disorders/symptoms-of-pulmonary-disorders/stridor#:~:text=Stridor-,Pathophysiology,larynx%2C%20and%20the%20extrathoracic%20trachea. 2) “Thyroidectomy: Complications.” OpenAnesthesia, IARS, www.openanesthesia.org/thyroidectomy_complications/. 3) Al-Qurayshi Z, Robins R, Hauch A, Randolph GW, Kandil E. Association of Surgeon Volume With Outcomes and Cost Savings Following Thyroidectomy: A National Forecast. JAMA Otolaryngol Head Neck Surg. 2016;142(1):32–39. 4) Schulze, T., Heidecke, C., Maier, S. i Knigge, S. (2013). Late manifestation of bilateral laryngeal nerve palsy after thyroidectomy. Signa vitae, 8 (2), 56-57. 5) Zakaria HM, Al Awad NA, Al Kreedes AS, et al. Recurrent laryngeal nerve injury in thyroid surgery. Oman Med J. 2011;26(1):34-38.

Airway Management - 10 Urgent Retrograde-Guided Fiberoptic Intubation in a Patient with a History of Acromegaly and Ankylosing Spondylitis

Elisa C Walsh[1], Michele D Szabo[1]

[1]Massachusetts General Hospital, Boston, MA

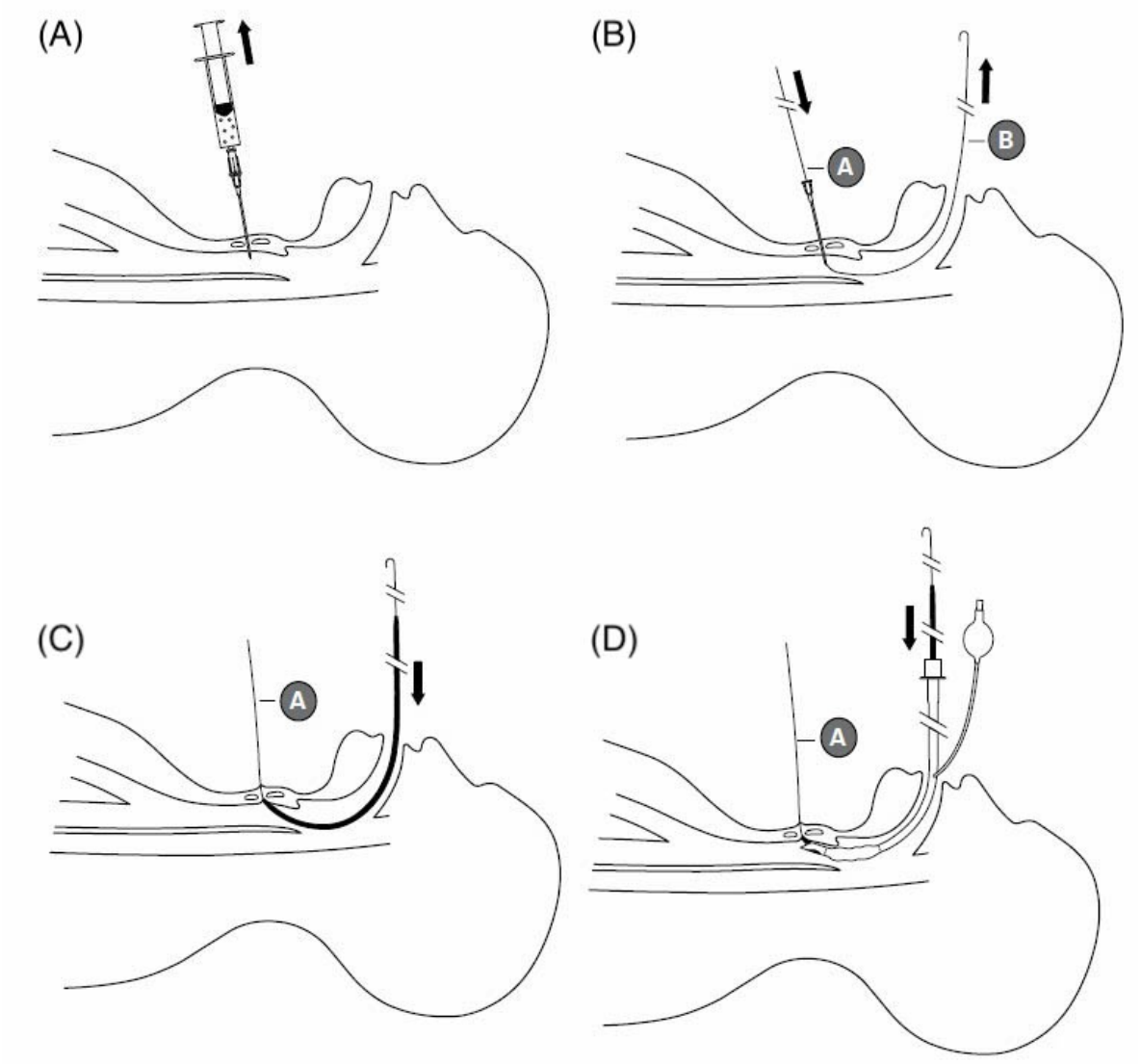
Introduction: Retrograde intubation was first reported in 1963 and modified to include the use of the fiberoptic bronchoscope in 1986 (1,2). While no longer routinely taught, retrograde intubation remains a useful technique in difficult airway cases where significant blood and secretions in the upper airway could obscure the view through a fiberoptic bronchoscope (3,4).

Methods: The patient was a 78-year-old man, with a history of acromegaly treated surgically 20 years ago, moderately severe ankylosing spondylitis with a history of a difficult airway, status-post coronary artery bypass grafting on aspirin, who presented to the emergency department with a recurrent episode of a spontaneous large volume epistaxis at home which had resolved. Labs were notable for a hematocrit drop to 29 from a previous baseline of 44. Magnetic resonance angiography demonstrated a 3mm pseudoaneurysm of the right internal carotid artery at the resection cavity and stenosis of the contralateral internal carotid artery. Since there was no active bleeding and evidence of complicated intracranial arterial pathology, it was decided to admit the patient to the ICU for an airway watch and plan for an elective interventional radiology treatment. However, during the first 24 hours of the ICU course, the patient began to bleed profusely from his nares and the decision was made to take him emergently to the IR suite. Upon arrival to the IR suite, it was noted that the nasal packing was providing reasonable control of the hemorrhage, the patient was cooperative, his vital signs were stable, and his oxygen saturation was normal on room air. He had the residual bony but not soft tissue features of acromegaly and a profound fixed kyphotic neck that was long enough to have access to his cricoid membrane. Given that there continued to be a small amount of blood in the airway and the history of a difficult intubation, along with the prediction that there would be no need for long term intubation, the decision was made to pursue an awake retrograde percutaneous wire-guided, fiberoptic-assisted endotracheal intubation. The team was prepared to do an awake tracheostomy if this technique failed. Under minimal sedation with fentanyl as well as local anesthesia, an 18-gauge introducer needle was inserted into the trachea approximately 1 centimeter caudal to the cricoid cartilage. A 145 cm Teflon-coated J wire, readily available in the IR suite, was introduced through the needle and directed upwards into the working port of a fiberoptic bronchoscope. This served as the guide to the antegrade placement of the bronchoscope which served as the guide for successful placement of a 7.0 Parker endotracheal tube. Subsequently, the patient underwent a pipeline flow-diverting stent to the right internal carotid artery.

Conclusion: Retrograde intubation remains a valuable tool for establishing an airway in difficult airway management. Because the retrograde guidewire permitted rapid passage of the bronchoscope through the pharynx and vocal cords with no need to visualize the anatomy, it was a particularly useful technique in our patient with a known difficult airway in an emergent situation where blood obscured direct bronchoscopic visualization of the vocal cords. After this event, our institution now stocks a standard retrograde intubation kit (Figure 1) with a 110cm length retrograde wire and 14.0 Fr Cook airway exchange catheter that can be used with endotracheal tubes greater than 5.0mm ID (5). Antegrade fiberoptic bronchoscopy may be used in place of the Cook catheter to directly visualize endotracheal tube placement, with the retrograde wire threaded through the working (suction) port of the bronchoscope. Ultimately, we recommend that trainees learn and practice retrograde intubation as an essential component of emergency airway management.

References: 1. Waters D. Guided blind endotracheal intubation. For patients with deformities of the upper airway. Anaesthesia. 1963 Apr;18:158-62. 2. Lechman MJ, Donahoo JS, and Macvaugh H. Endotracheal intubation using percutaneous retrograde guidewire insertion followed by antegrade fiberoptic bronchoscopy. Crit Care Med. 1986 Jun;14(6):589-590. 3. Dey S, Ninu M, Yunus M, and Syiemiong N. 2016. Fiberoptic Guided Retrograde Intubation in an Anticipated Difficult Airway: Revival of

an Antiquated Technique. J Clin Diagn Res. 2016 Oct; 10(10): UD06-UD07. 4. Dhanyee AS, Pillai R, and Sahajanandan R. Wire guided fibreoptic retrograde intubation in a case of glottic mass. Indian J Anaesth. 2016 Mar;60(3):219-21. 5. “Cook Retrograde Intubation Kit.” Cook Medical. https://www.ciamedical.com/cook-medical-g10554-each-c-retro-14-0-70-38j-110-cae. Accessed 1/12/2021.



Airway Management - 11 Face-to-Face Awake Video Laryngoscopy: When Time is Rigid, not Flexible

Derek West[1], Micah Long[2]

[1]University of Wisconsin-Madison UW Health, Madison, WI, [2]University of Wisconsin, Madison, United States of America

Introduction: Awake intubation in the spontaneous ventilating patient remains a mainstay in safely securing the airway in patients who have a known or potentially challenging airway, or when medication induction would not be tolerated or safe. In the case of a self-positioned, leaned forward, stridulous and hypoxic patient, awake intubation in the seated position is indicated.

Methods: Review of Case Report A 76-year-old woman with a past medical history of obesity (BMI 35) presented to the hospital after a motor vehicle crash with resultant single right rib fracture, gluteal hematoma, left neck hematoma and fractures of the left radius and ulna. She required massive transfusion for a gluteal arterial hemorrhage, complicated by expansion of her neck hematoma, with imaging showing a 5 cm x 8 cm hematoma with 2 cm of tracheal deviation. Dexamethasone was initiated for airway swelling (10 mg administered intravenously every 8 hours for 2 days). She was medically stable with excellent lung compliance and oxygenation, but had no cuff leak and minimal improvement in her neck examination for 9-days. On post-injury day 10 she developed a stable cuff leak, and she was extubated over a wire the following day. After initial stability, the wire was removed. Approximately 2-hours after extubation she developed progressive, severe dyspnea with increased work of breathing and a preferred upright-forward-leaning position that progressed rapidly to florid biphasic stridor with hypoxia, with oxygen saturations in the mid 80%'s. She was not responsive to racemic epinephrine nebulized therapies. The emergency airway team was called and an awake intubation strategy planned with the patient in the seated-upright position, facing the intubating team. Topicalization of the airway was performed with directed and atomized 1% lidocaine. As bronchoscopy equipment was not yet set up, a video laryngoscopy was utilized and inserted, 'upside down' to typical orientation (Figure 1). Additional atomized lidocaine was administered to the vocal cords, guided by VL. Next, a 6.0 mm internal diameter endotracheal tube was inserted into the trachea, using a stylet. Placement was confirmed and anesthesia was induced.

Conclusion: Desaturation due to severe upper airway obstruction is a critical medical emergency that warrants prompt intervention. If orotracheal intubation is unable to be completed quickly, emergent tracheostomy is indicated. For the patient with stridor, the seated position facilitates several advantages over the supine position. First, in a sitting position, gravity facilitates downward displacement of the soft tissues of the neck, helping to align the oro-pharyngeal-tracheal axis and improve secretion management (1). Respiratory mechanics and ventilation are also improved in the sitting position, where FRC and tidal volume are increased and atelectasis is decreased, all contributing to a decreased risk of hypoxia (2). Head elevation along with head-forward position in emergent airways has been shown to decrease intubation related adverse events, such as aspiration, hypoxia and esophageal intubation (3). Classical training emphasizes use of the flexible fiberoptic bronchoscope to facilitate awake tracheal intubation. Unfortunately, the advent of rigid video laryngoscopy has decreased anesthesiology resident experience in this important procedure. Improving education, including simulation, is critical to maintain this skillset. When experience is lacking, video laryngoscopy is a well-tolerated, readily available alternative approach to awake intubation of the trachea. In fact, a recent meta-analysis found that awake video laryngoscopy guided tracheal intubation has equal first-pass and overall success to flexible fiberoptic bronchoscopy and is on average 40-seconds faster,(4) not accounting for equipment delays common to bronchoscopy. In a time-critical emergency, providers should utilize readily available tools and play to their skillsets. In many cases, indirect video laryngoscopy should be considered first-line for the emergent, awake intubation, particularly when experience in flexible fiberoptic technique is lacking or when equipment delays prevent its rapid deployment. It is easily adapted to the upright, forward-facing position and benefits from increased operator familiarity and availability.

References: 1. NEJM JW Emerg Med Mar 15 2016; and Anesth Analg 2016; 122:1101). 2. Katz, S., Arish, N., Rokach, A. et al. The effect of body position on pulmonary function: a systematic review. BMC Pulm Med 18, 159 (2018). 3. Khandelwal N et al. Head-elevated patient positioning decreases complications of emergent tracheal intubation in the ward and intensive care unit. Anesth Analg 2016 Feb 10.

4. Alhomary M, Ramadan E, Curran E, Walsh SR. Videolaryngoscopy vs. Fiberoptic bronchoscopy for awake tracheal intubation: a systematic review and meta-analysis. Anaesthesia. 2018 Sep;73(9):1151-1161.

Figure 1: Upright, Face-to-Face Video Laryngoscopy. Video laryngoscopy is easily adapted to this unique position, is readily available in an emergency, and benefits from provider comfort and experience.

Airway Management - 12 Extracorporeal Membrane Oxygenation for Complex Intrathoracic Tracheal Resection

Angelica M Jaramillo[1], Pankaj Jain[1]

[1]University of Miami, Miami, FL

Introduction: Anesthetic and airway management of a patient undergoing repair for tracheal stenosis can be challenging. Surgical repair for distal stenosis near the carina by conventional airway management can deleteriously affect patient oxygenation and ventilation, rendering such procedures infeasible. We describe the intraoperative use veno-venous extracorporeal membrane oxygenation (VV-ECMO) to successfully facilitate tracheal resection and anastomosis.

Methods: A 42-year-old female, with past medical history of diabetes mellitus and asthma, developed tracheal stenosis following prolonged intubation in the setting of COVID-19 pneumonia. She had recurrent episodes of respiratory distress despite medical management and multiple attempts at bronchoscpic balloon dilation. Imaging demonstrated a 3.7 cm length of distal tracheal stenosis of (less than 6 mm), starting 6 cm from the glottic inlet and extending to the vicinity of the carina (Figure 1)

After uneventful anesthetic induction and careful endotracheal intubation with a 7.0 mm ID endotracheal tube, radial arterial and wide bore peripheral venous access was obtained. Supra-stenotic placement of the ETT was confirmed bronchoscopically. Cervical mediastinoscopy was then performed to release the trachea anteriorly. Right lung isolation, to allow a right robotic approach to the tracheal resection, was then easily achieved with the Rusch EZ Blocker TM . Peripheral femoral and internal jugular cannulae were inserted and VV-ECMO was initiated to prevent stasis. As robotic approach did not provide adequate exposure, an upper sternotomy was performed. Prolonged apnea, under full VV-ECMO support, enabled the technically challenging tracheal segment resection and anastomosis (Figure 1). After chest closure, Grillo sutures were placed to keep the neck in flexed position. This was followed by a smooth emergence and uneventful extubation in the OR, and the patient was transferred to the ICU for close monitoring.

Conclusion: Tracheal stenosis may complicate prolonged intubation. Cross table ventilation with periods of short apnea has been the coventional approach for major airway surgery. VV-ECMO is an alternative approach that can be advantageous in the setting complex intrathoracic tracheal resections, especially when minimally invasive robotic or thoracoscopic surgery is planned. Surgical complexity, availability of expertise, and need for anticoagulation are important considerations for this approach.

Extracorporeal Membrane Oxygenation for Complex Intrathoracic Tracheal Resection

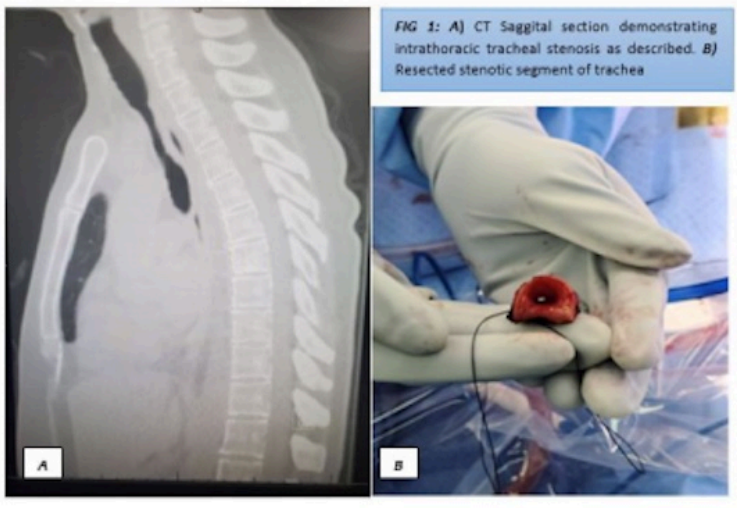


FIG 1: A) CT Saggital section demonstrating intrathoracic tracheal stenosis as described. *B)* Resected stenotic segment of trachea

Airway Management - 13 Nasal Mask-Face Tent and Oral Suctioning Maintained Nasal Ventilation/Oxygenation and Lowered Aerosol/Droplet Spread during Extubation/Recovery in a COVID Positive Patient undergoing EGD/Necrosectomy

James Tse[1], Mariah McGrory[1], Supreet Grewal[1]

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

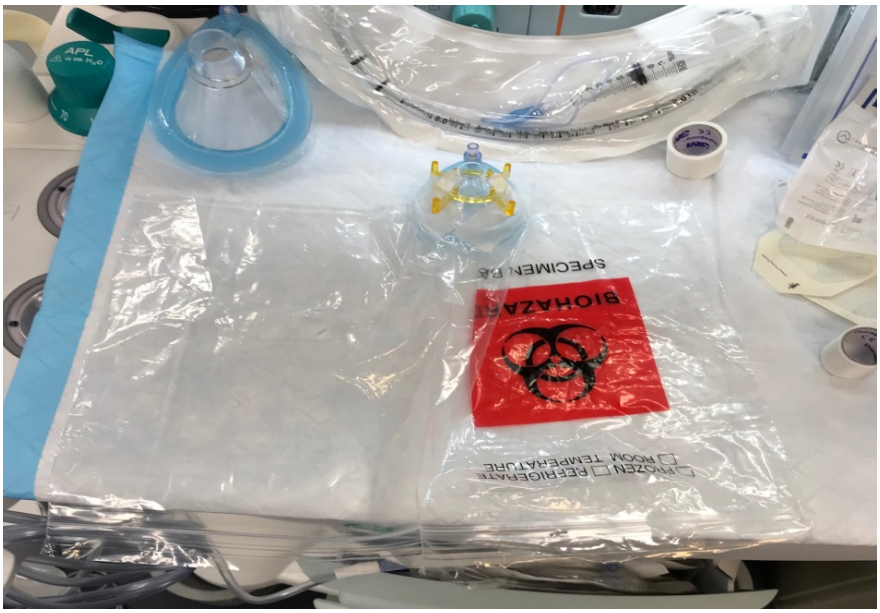
Introduction: Endotracheal intubation/extubation and upper GI endoscopy are aerosol/droplet-generating procedures that increase the risk of transmission of infection. This is of particular concern during the COVID-19 pandemic. A simple combined nasal mask-face tent provided pre/apneic nasal oxygenation and reduced aerosol/droplet spread during RSI, intubation and extubation in a COVID-19 positive patient undergoing exploratory laparotomy (Fig. 1-3).(1,2) We combined this simple technique and oral suctioning to provide continuous nasal oxygenation and lower aerosol/droplet spread in a COVID-19 positive patient during EGD/necrosectomy and extubation.

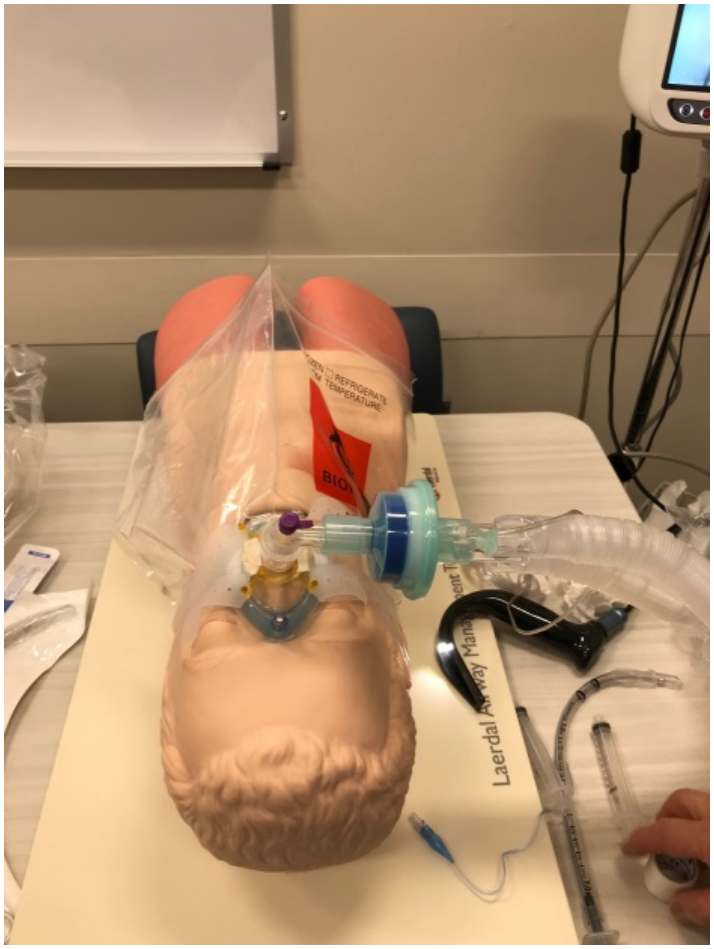
Methods: Case Report A 66-year-old COVID-19 positive male with NIDDM, HTN, atrial fibrillation/atrial flutter, biliary sepsis, pericardial effusion, ATN on hemodialysis, biliary fluid collection presented for EGD/necrosectomy.

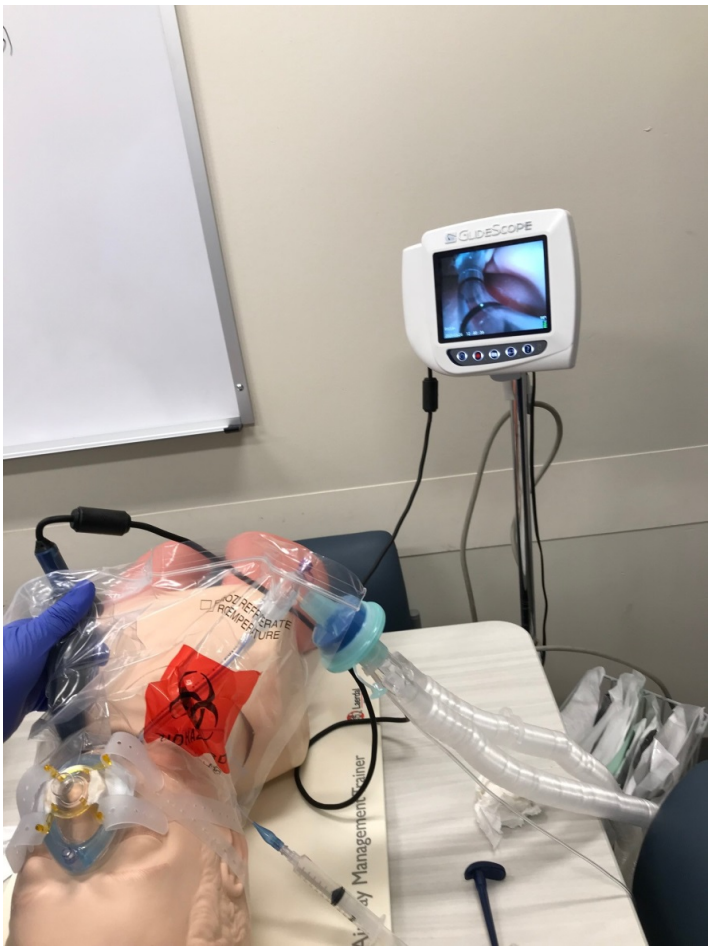
The patient was intubated by the COVID Intubation Team with full PPEs in a negative room. During the procedure, his mouth was covered with a clean clear plastic sheet (face tent). Frequent oral suctioning was performed under the face tent to reduce aerosol/droplet spread. He tolerated the difficult procedure well. Following removing the endoscope, a modified infant facemask was secured over his nose with elastic head-straps and a large plastic sheet covered his mouth (Fig. 4). After he resumed spontaneous ventilation, his mouth was suctioned clear of any secretion (Fig. 4). He was then extubated smoothly. The breathing circuit was immediately connected to the nasal mask. The mouth was closed under the plastic sheet to reduce aerosol/droplet spread. He maintained spontaneous nasal ventilation and 99-100%SpO2 during extubation/ recovery in the negative pressure room (Fig. 5). He was transported back to the COVID-19 unit with a N95 face mask without any complications.

Conclusion: This simple nasal mask-face tent maintained spontaneous nasal ventilation and oxygenation post-extubation in a COVID-19 positive patient undergoing EGD/necrosectomy. Combining with suctioning, the face tent also helped lower aerosol/droplet spread during the procedure, extubation and recovery. Amid the COVID-19 pandemic, this technique may improve patient safety and provide additional provider protection at a low cost.

References: 1. www.TSEmask.com; 2. ASA virtual Annual Meeting: (MC1280), Oct, 2020




Laerdal Airway Management Trainer


GLIDESCOPE


SIEMENS

Airway Management - 14 An asymptomatic adult patient of tracheal narrowing due to the double aortic arch: A case report

Keisuke Kawata[1], Tomoko Hiraga[1], Naohiko Funatsu[1]

[1]National Hospital Organization Yamaguchi-Ube Medical Center, Ube, Yamaguchi, Japan

Introduction: Central airway stenosis is a critical issue in the context of airway management in anesthesia. Herein we describe an adult case of tracheal narrowing due to an extremely rare congenital vascular malformation:an asymptomatic double aortic arch.

Methods: A 66-year-old Japanese man (weight, 89 kg; height, 182 cm; body mass index, 27 kg/m^2; and American Society of Anesthesiologists physical status class 2) with lung cancer was referred for a right upper lobectomy under video-assisted thoracoscopic surgery. The space-occupying lesion on the dorsal side of the trachea and esophagus was found incidentally during a preoperative examination, and the patient had no complaints related to tracheal narrowing.

Preoperative radiological examination revealed tracheal narrowing (Figure 1). Contrast-enhanced computed tomography (CT) images showed that the intrathoracic trachea was pinched by unusual vessels (Figure 2). Furthermore, a three-dimensional CT reconstruction clarified the overall structure of the vascular ring and compressed trachea (Figure 3). Therefore, the patient was diagnosed with central airway stenosis due to asymptomatic double aortic arch during the preoperative assessment.

General anesthesia was induced with propofol (1.5 mg/kg, body weight), followed by administration of sevoflurane (2-5%), remifentanil (0.5 ug/kg, body weight/minute), and rocuronium (0.6 mg/kg, body weight). After muscle relaxation, the glottis was identified with a video laryngoscope (McGRATHTM Mac, Aircraft Medical, UK). Then, orotracheal intubation was performed with a left-sided double-lumen endotracheal tube (Broncho-Cath^{TM}, 35-Fr, Mallinckrodt Medical, Ireland) under the guidance of a fiberoptic bronchoscope (LF-GP, OLYMPUS, Japan), passed through the bronchial lumen.

Postoperatively, the patient awakened normally and was extubated in the operating room as planned. Occurrence of postoperative pulmonary complications was not examined. He was then transported to the post anesthetic care unit under spontaneous respiration. The total operation time was 3 hours and 8 minutes; one lung ventilation time was 3 hours and 3 minutes; and total anesthetic time was 4 hours and 20 minutes.

Conclusion: 1. We encountered a case of tracheal narrowing due to rare, asymptomatic congenital vascular anomaly, double aortic arch.[1]
2. Appearance of tracheal deformation resembled "saber-sheath trachea".[2,3]
3. The double aortic arch might be recognized as one of the differential diagnosis causing saber-sheath trachea.
4. To our knowledge,this is the first report to confirm that the double aortic arch might cause the tracheal narrowing in an adult patient.

References: 1. Radlology 138:399, 1981 2. AJR, 174:1315, 2000. 3. AJR, 196:W240, 2011.

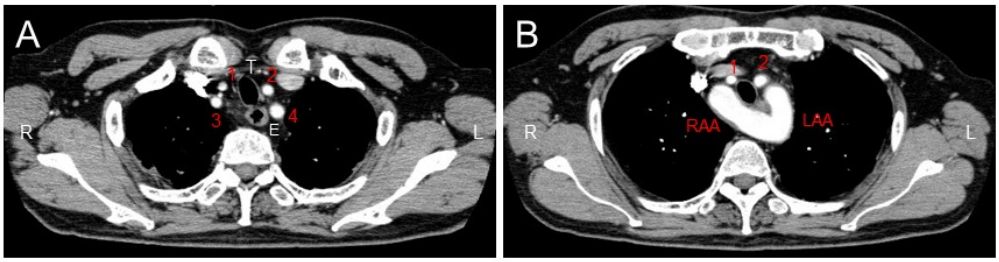


Figure 2. Contrast-enhanced computed tomography image, horizontal planes.
(A) Four vessels surround and pinch the trachea and the esophagus on the cephalic plane above aorta.
(B) Right aortic arch (RAA) and left aortic arch (LAA) compose vascular ring that compress the trachea decreased in the coronal diameter (13 mm) associated with an increase in the sagittal diameter (20 mm).
1, right common carotid artery; 2, left common carotid artery; 3, right subclavian artery;
4, left subclavian artery; E, esophagus; L, left; R, right; T, trachea.

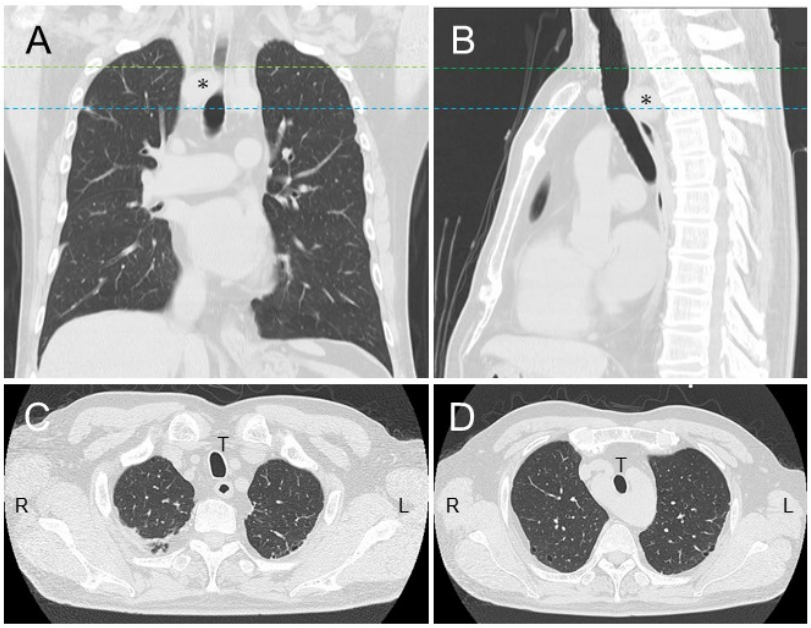


Figure 1. (A) Computed tomography (CT) image, coronal view. Unusual shadow (＊) overlaps trachea. (B) Sagittal view. Space occupied lesion (＊) compresses trachea anteriorly. (C) and (D) are transverse planes corresponding to levels of green and blue dotted lines respectively in the coronal (A) and sagittal (B) views. Narrowing trachea decreased in the coronal diameter. L, left side; R, right side; T, trachea.

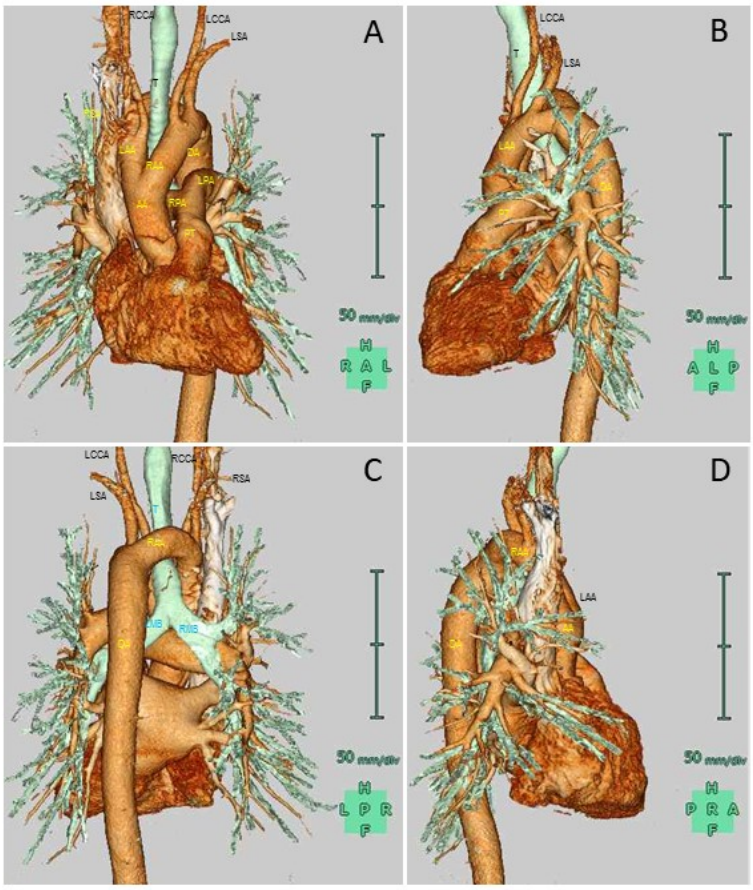


Figure 3. Three-dimensional reconstructed images (A, anterior; B, Left; C, posterior; and D; right view). These pictures represent that trachea is encircled by the vascular ring composed of the double aortic arch (RAA and LAA). AA, ascending aorta; DA, descending aorta; LAA, left aortic arch; LCCA, left common carotid artery; LMB, left main bronchus; LPA, left pulmonary artery; LSA, left subclavian artery; PT, pulmonary trunk; RAA, right aortic arch; RCCA, right common carotid artery; RMB, right main bronchus; RPA, right pulmonary artery; RSA, right subclavian artery; T, trachea

Ambulatory Anesthesia

Ambulatory Anesthesia - 1 Pediatric computed tomography guided biopsies for lung nodules: Ambulatory procedure or Intensive care unit disposition?

Claude Abdallah[1], Renee J Roberts[2]

[1]Children's National Health System, Washington, DC, [2]Children's National Medical Center, Washington, DC

Introduction: Pediatric computed tomography (CT) guided biopsies for lung nodules or masses suspected to be malignant is achieved under anesthesia. In different situations, this procedure is preferred, to avoid injury to normal lung tissue from an alternative lobectomy. These procedures are usually scheduled as ambulatory cases; however, complications may arise requiring hospital admission and intensive care treatment and monitoring. We present two cases of pulmonary hemorrhage and a pneumothorax in pediatric outpatients following CT guided biopsy requiring transfer to the intensive care unit for treatment and observation.

Methods: The first patient is a 13-year-old with refractory acute myeloid leukemia, a previous history of viridans-group streptococcal sepsis, receiving chemotherapy who presented for CT guided transthoracic lung biopsy for persistent lung nodules. The concern was for undertreated fungal versus leukemic infiltrates. The patient came from home, afebrile, on room air, with a previous acceptable complete blood count. Toward the end of the procedure, blood was noticed and suctioned from the trachea. Post biopsy images demonstrated significant increase in the amount of consolidation involving the biopsied lung. Patient was transported with an endotracheal tube to the intensive care unit (PICU) in stable condition. Post procedure care included imaging showing near complete consolidation involving the biopsied lung secondary to pulmonary hemorrhage. Due to the intermittent coagulopathy from relapsed AML, the patient was transfused over the next two days (total 280cc PRBC and 200cc platelets). Over three days after the bleeding stopped, the patient's trachea was extubated to BiPAP, then weaned to room air. Patient was discharged home one week following the biopsy. The second patient is a 6-year-old patient with a history of hepatoblastoma with metastases to the lungs, status post chemotherapy and resection two years ago with a residual pulmonary nodule. The decision was made for needle biopsy with possible cryoablation of the nodule with interventional radiology. After the procedure, the patient had a pneumothorax and blood was suctioned from the endotracheal tube with around 100 mL of pulmonary hemorrhage. The patient was transferred to the PICU, sedated and ventilated after chest tube placement. Hemoglobin level during the procedure was stable, and a type and screen was sent. Pneumothorax resolved and the patient was discharged after two days stay in the intensive care unit.

Conclusion: Transthoracic lung biopsy are often scheduled as ambulatory procedures, however these procedures carry the risk of severe complications, especially in most fragile pediatric patients. Described factors associated with post-biopsy pulmonary hemorrhage were longer needle path length between pleura and the lesion, smaller target lesions, non-pleural contact lesions, non-restrictive lung function test, and patients in supine positions. (1,2) A discussion of these factors may facilitate pre-procedure planning. Specific anesthetic concerns include communicating of potential risks to the patient and family comprehensively, endotracheal intubation from the outset with frequent vigilance for bleeding from the endotracheal tube, availability of PICU bed, recent lab work prior to the procedure to estimate the risks of transfusion and preparation to initiate a blood transfusion if necessary.

References: 1. Radiology. 2016; 279(1): 287-96. 2. Diagn Interv Radiol. 2017; 23(5): 347-353

Anesthetic Pharmacology

Anesthetic Pharmacology - 1

Administration of Sugammadex Induced Anaphylaxis (ASIA): An Updated Clinical Review

Irene Kimlee[1], Mirsad Dupanovic[2], Kassatihun Gebre-Amlak[3]

[1]VCU School of Medicine, Richmond, VA, [2]University of Kansas Medical Center, Kansas City, MO, [3]Abbott Northwest Hospital, Minneapolis, MN

Introduction: Sugammadex (Bridion®; Merck & Co.) is currently the only available drug capable of selective reversal of neuromuscular blockade. The reported incidence of hypersensitivity to sugammadex is 0.22% and the reported incidence of administration of sugammadex induced anaphylaxis (ASIA) is 0.059%.[1] This review examines publications reporting ASIA for the most common presentation, treatment, outcome, and diagnostic tests.

Methods: PubMed, Web of Science, and Cochrane Library were searched for cases reporting anaphylaxis associated with sugammadex administration using the keywords: sugammadex, allergy, anaphylaxis, hypersensitivity, reaction, anaphylactoid, anaphylactic and hypotension. Authors independently screened abstracts before determining a consensus for case selection for inclusion in this analysis. Full reports had been published in journals in either English or in Japanese. An additional four reports were published on the Japanese Society of Anesthesia website. A total of 57 cases of ASIA were included in the analysis which was done in Microsoft Excel.

Results: The mean sugammadex dose administered was 2.6 mg/kg (range 0.7-4.9 mg/kg). ASIA occurred typically within 1-5 minutes (average 2.9 min) after administration, but symptoms developed much later in 4 reports (8-10 min). No patients had prior exposure to sugammadex. Symptoms were profound hypotension (87.7%), skin findings (71.9%), tachycardia (54.4%), hypoxia (42.1%) and bronchospasm (31.6%). Angioedema was reported in 21% of cases and 6 patients (13.6%) required (re)intubation. Intravenous epinephrine was the most commonly used vasoactive drug (73.7%). Intermittent boluses of 10-200 mcg of epinephrine was often successful, while epinephrine infusion was needed in 22.8% of cases. Other common treatments were steroids (68.4%), H1 blockers (40.4%), H2 blockers (22.3%) and beta agonists (15.8%). More than one third of affected patients remained intubated (41%) while 58% of reported patients were transferred to the ICU. Intradermal test and skin pin-prick test were the most commonly used confirmatory diagnostic tests and were positive in 88% and 77.7% of tested patients respectively. Tryptase was positive in 76.5% of tested patients while IgE was positive in 100% of patients (4/4). Signs of ASIA generally resolved within a few hours, and there was one reported mortality.

Conclusion: The above dose findings challenge the information from premarketing studies in which ASIA occurred only with a dose of 16 mg/kg. Patients should be closely observed for 10 minutes following intravenous administration of sugammadex for presenting symptoms of ASIA: deterioration of vital signs, development of skin rash, and increased peak airway pressure. Re-intubation may be required if tracheal extubation was performed before onset of ASIA symptoms. Anesthesia providers should be cognizant that hemodynamically unstable patients, those with a history of asthma, or patients difficult to intubate and/or ventilate may be particularly vulnerable for severe symptoms in case of this complication. Immediate availability of an epinephrine syringe in the anesthetizing location is necessary for timely treatment of ASIA.

References: Anesth Analg. 2018 May;126(5):1505-1508

Anesthetic Pharmacology - 2

Pseudocholinesterase Deficiency in a Patient with Prolonged Recovery of Neuromuscular Function after Succinylcholine for Electroconvulsive Therapy

Hamelmal Kassahun[1], Katherine Forkin[2], Jessica Sheeran[3]

[1]University of Virginia, Charlottesville, VA, [2]University of Virginia, Charlottesville, VA, [3]University of Virginia, Charlottesville, United States of America

Introduction: Neuromuscular blockade is administered during anesthesia for patients undergoing electroconvulsive therapy (ECT) treatment in order to reduce the risk of musculoskeletal complications by reducing motor activity during the seizure. Succinylcholine is the preferred neuromuscular blocking agent for this procedure due to its rapid onset and short duration of action. Succinylcholine is quickly hydrolyzed by pseudocholinesterase (also known as plasma cholinesterase or butyrylcholinesterase) as it diffuses away from the motor end plate(1). Pseudocholinesterase deficiency results in a low activity level of the enzyme, thus reducing the ability to hydrolyze ester bonds in succinylcholine. Pseudocholinesterase deficiency is a rare, acquired or inherited defect of the pseudocholinesterase enzyme produced by the liver that metabolizes succinylcholine. Pseudocholinesterase deficiency leads to prolonged duration of action of succinylcholine, and patients can experience prolonged muscle paralysis from standard doses. We present a case report of a 30-year-old woman with no known history of anesthetic complications who experienced prolonged recovery of neuromuscular function after ECT.

Methods: A 30-year-old woman with psychiatric medical history of obsessive-compulsive disorder and severe bipolar disorder with psychotic features presented for initial ECT treatment. The patient had been admitted to the psychiatric ward after transfer from an outside hospital following a suicide attempt (intentional acetaminophen overdose). The patient was referred for ECT due to continued severe depression refractory to medication with two suicide attempts within a few months prior to this admission. The patient's medical history also included tobacco use and cannabis use disorder. Five months prior, she had experienced a possible seizure versus syncopal episode. Head CT scan was unremarkable and she was not started on antiepileptic medication nor had she experienced a recurrent episode. Her home medications included quetiapine 350 mg nightly, lamotrigine 50 mg daily, trazodone 100 mg PRN nightly, clonazepam 0.5 mg BID PRN, and hydroxyzine 25 mg PRN. The patient denied problems with anesthesia in the past. Surgical history included breast implant surgery at an outside hospital with no records immediately available. The patient was brought to the procedure room, standard American Society of Anesthesiologists monitors were placed, and she was preoxygenated at 10 L/min. She received intravenous (IV) methohexital 40 mg for sedation followed by IV succinylcholine 40 mg IV for muscle relaxation. ECT was administered without any complications and the patient was bag-mask ventilated throughout. After completion of the seizure, she received ketorolac 30mg and midazolam 1mg IV. Post-ECT course was complicated by continued apnea for 30 minutes after the ECT treatment. Electroencephalogram leads were replaced by the psychiatrist and demonstrated no evidence of ongoing seizure. A twitch monitor was obtained to assess prolonged recovery of neuromuscular blockade concerning for potential pseudocholinesterase deficiency and demonstrated a train-of-four count of 1/4. The train-of-four count eventually improved to 4/4 with sustained tetanus over the next 15 minutes. The patient began breathing spontaneously during this time. Forty-five minutes after the procedure, she was awake and alert and had no recall of the prolonged recovery of neuromuscular function. During one other ECT treatment approximately 3 weeks after the initial treatment, the patient received a smaller dose of succinylcholine (20 mg) and again had a prolonged recovery of neuromuscular function. She underwent subsequent ECT treatments with administration of low-dose rocuronium and reversal with sugammadex without complications.

Conclusion: Patients with pseudocholinesterase deficiency are often only diagnosed after experiencing prolonged neuromuscular paralysis with associated respiratory failure following standard doses of

succinylcholine. Continued respiratory support, which may include intubation and mechanical ventilation, is required until spontaneous return of neuromuscular function (2,3) This case report adds evidence that low-dose aminosteroid neuromuscular blocking agents for neuromuscular blockade and reversal with sugammadex is effective and efficient for management of neuromuscular blockade in a patient with pseudocholinesterase deficiency for ECT.

References: 1 Naguib M, Lein CA, Meistelman C. Pharmacology of Neuromuscular Blocking Drugs. In: Miller RD, ed. Miller's Anesthesia. Philadelphia, PA: Elsevier, 2015; 958-62. 2 Andersson ML, Møller AM, Wildgaard K. Butyrylcholinesterase deficiency and its clinical importance in anaesthesia: a systematic review. Anaesthesia 2019; 74: 518-28. 3 Lee S, Han JW, Kim ES. Butyrylcholinesterase deficiency identified by preoperative patient interview. Korean J Anesthesiol 2013; 65: S1-3.

Blood Management

Blood Management - 1 Perioperative optimization and management of anemia in a patient of the Jehovah's Witness faith

Caleb Kennon[1], Nadia Romero[2], Annette Rebel[3], Thomas McLarney[3], Bjorn T Olsen[3], Eric A Wetherington[2]

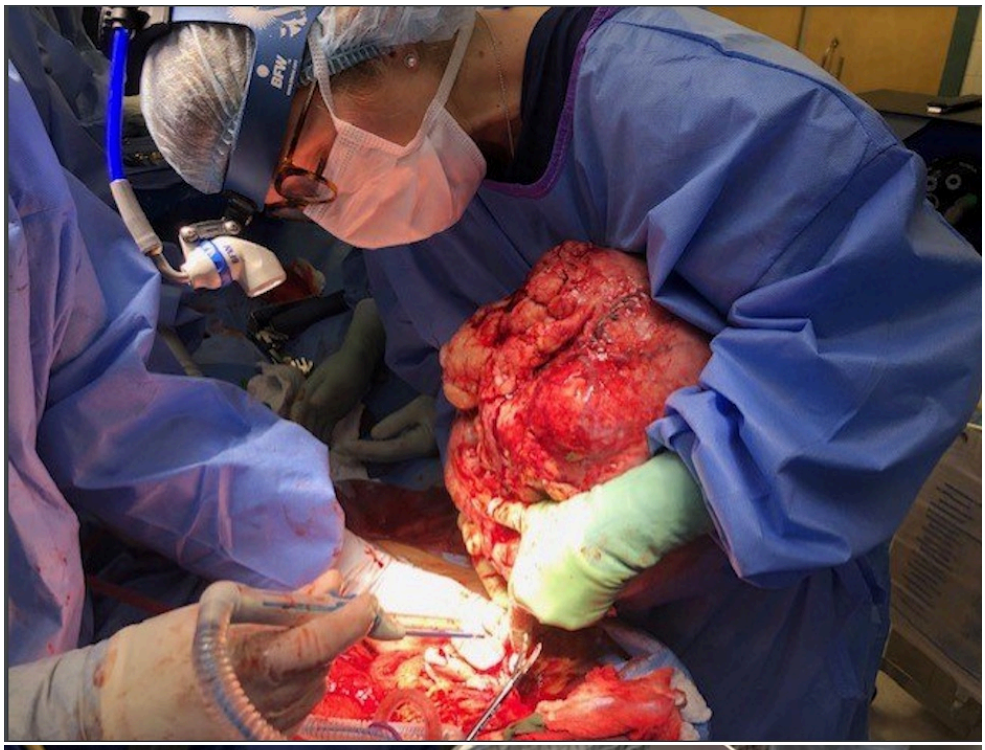
[1]University of Kentucky, Lexington, United States of America, [2]University of Kentucky Medical Center, Lexington, KY, [3]University of Kentucky, Lexington, KY

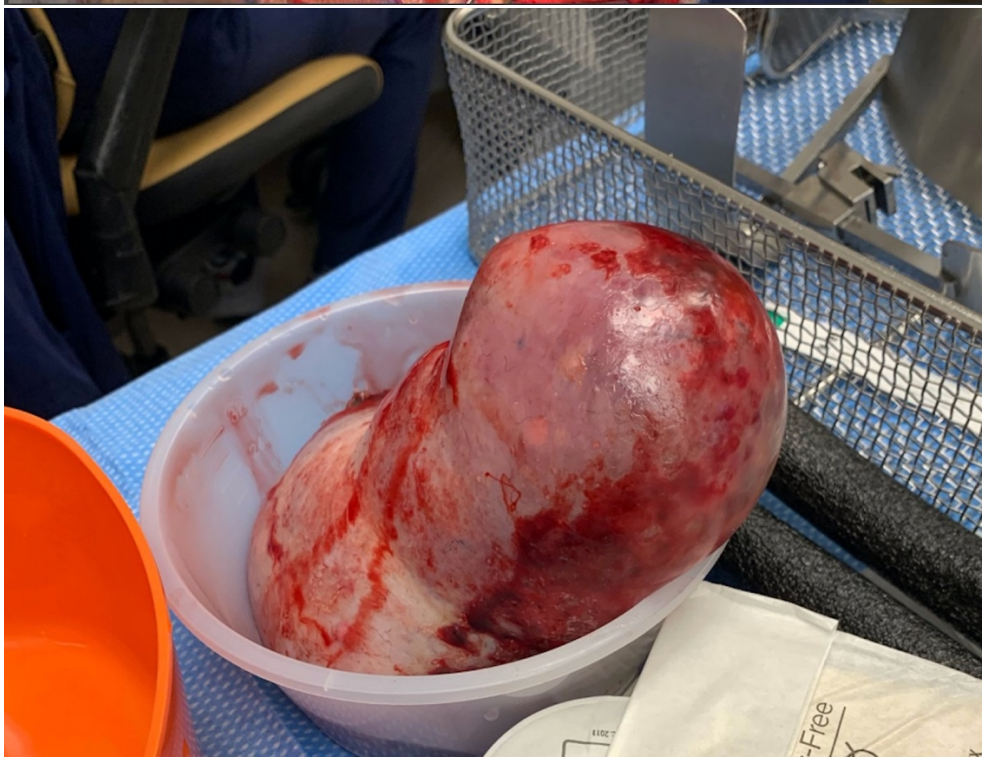
Introduction: The perioperative management of patients with the Jehovah's Witness faith poses many legal, ethical and medical challenges for medical professionals. With more than an estimated 8 million congregation members worldwide, medical professionals in all settings can expect to care for this population. Medical teams must be familiar with the common challenges and methods available to resuscitate this population while respecting the patient's faith and autonomy. Preoperative severe anemia has been identified as predictive of postoperative complications. The combination of refusal of autologous blood as part of the JW faith, severe anemia and anticipated significant surgical blood loss create a very challenging environment for perioperative care providers. This case demonstrates the challenges of preoperative communication, planning and optimization, followed by utilization of system-based resources for optimization of the care for an anemic JW patient.

Methods: The 37 year old female patient presented to the HEME/ONC service with severe anemia and shortness of breath, caused by massive ascites, and 30 lb weight loss within the last 6 months. Workup indicated large bilateral ovarian masses (27x23x26cm right, 10x10x16cm left), elevated tumor markers [CA125 of 5813] and a Hgb concentration of 5.2g/dl. She denied any previous health history, but revealed her JW faith, therefore declining blood products even if required for life saving measures. She was referred to the GYN/ONC service with the preoperative anesthesia clinic consulted for preoperative optimization. Continued discussions with the patient indicated that she refused autologous blood transfusions but she was amenable to receiving Iron, TXA, FFP, Cryoprecipitate, Albumin, Cell Saver, and Autologous Normovolemic Hemodilution (ANH). She was started on IV iron therapy 6 weeks pre-surgical intervention, in combination with PO iron supplementation. Interventional radiology was consulted for preoperative ovarian tumor embolization. Four days prior to resection, the left ovarian mass was successfully embolized with embolization of the right ovarian mass only partially successful. On the day of surgery, in effort to minimize iatrogenic blood loss, no labs were drawn with her most recent Hgb at 6.4g/dl post embolization. OR accommodations included Point-of-care (PoC) measurements in the OR (GEM 5000), Cell Saver and ANH supplies. A low thoracic epidural catheter was placed for postoperative analgesia. Operative monitoring included cerebral oximetry, invasive arterial blood pressure monitoring in addition to standard ASA monitoring. After general anesthesia induction (RSI), a central venous catheter (12 Fr) was placed as ANH (350 mL of blood in exchange with 500ml 5%Albumin) was performed through a peripheral large bore intravenous catheter. To minimize surgical blood loss related to tumor related fibrinolysis, the patient was started on TXA loading dose and infusion. Normothermia was maintained throughout the procedure. PoC ABG and VGB were obtained pre-incision and post tumor resection. Notable intraoperative events included removal of 7 liters of ascites without any appreciable hemodynamic swings and adherence of the right mass to approximately 15 cm of jejunum. ANH collected blood remained in physical connection to the patient and was re-transfused after the tumor mass was removed. Despite normovolemic conditions, a norepinephrine infusion was needed post tumor removal to maintain MAP and cerebral oximetry goals. The patient was extubated 2 hours postoperatively in the ICU with a Hgb of 4.2g/dl and with adequate analgesia via Epidural PCA. Despite significant anemia (lowest Hb 4.5g/dl on POD2), the patient did not indicate any anemia related complications. The patient was transferred to the progressive care POD 3 and discharged home on POD 7.

Conclusion: Caring for patients of the Jehovah's Witness faith poses significant challenges to the medical team. Not the least of which is being familiar with and finding compromise in available methods that allow the clinician to circumvent preventable morbidity and mortality. Although ANH in anemic patients is seen

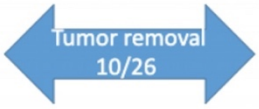
as relatively contraindicated, this case report demonstrates the necessity of discussing the pro and cons of this technique in conjunction to preoperative optimization, addressing pre-existing anemia, and minimizing perioperative blood loss.





Time course and Hb/Creatinine

Report Time	Result Type	Value	Units
2020-11-23 08:01:00	Hemoglobin	10.9	g/dL
2020-11-11 09:09:00	Hemoglobin	8.2	g/dL
2020-11-02 04:19:00	Hemoglobin	5.9*	g/dL
2020-11-01 04:51:00	Hemoglobin	5.7*	g/dL
2020-10-31 03:40:00	Hemoglobin	5.5*	g/dL
2020-10-30 02:07:00	Hemoglobin	5.1*	g/dL
2020-10-29 01:50:00	Hemoglobin	4.5*	g/dL
2020-10-28 13:13:00	Hemoglobin	4.5*	g/dL
2020-10-28 01:32:00	Hemoglobin	4.3*	g/dL
2020-10-26 17:23:00	Hemoglobin	5.0*	g/dL
2020-10-23 05:13:00	Hemoglobin	6.5	g/dL
2020-10-22 16:24:00	Hemoglobin	6.4*	g/dL
2020-10-15 13:59:00	Hemoglobin	7.3	g/dL
2020-10-01 10:03:00	Hemoglobin	7.8	g/dL
2020-09-17 09:55:00	Hemoglobin	6.9	g/dL
2020-09-11 10:47:00	Hemoglobin	6.0	g/dL
2020-09-03 05:23:00	Hemoglobin	4.3	g/dL
2020-09-02 04:30:00	Hemoglobin	4.7*	g/dL
2020-09-01 10:39:00	Hemoglobin	5.2	g/dL



Report Time	Result Type	Value	Units
2020-11-23 08:01:00	Creatinine Level	0.31	mg/dL
2020-11-11 09:09:00	Creatinine Level	0.22	mg/dL
2020-11-02 04:19:00	Creatinine Level	0.23	mg/dL
2020-11-01 04:51:00	Creatinine Level	0.22	mg/dL
2020-10-31 03:40:00	Creatinine Level	0.21	mg/dL
2020-10-30 02:07:00	Creatinine Level	0.17	mg/dL
2020-10-29 01:50:00	Creatinine Level	0.19	mg/dL
2020-10-28 01:32:00	Creatinine Level	0.22	mg/dL
2020-10-27 09:06:00	Creatinine Level	0.29	mg/dL
2020-10-26 17:23:00	Creatinine Level	0.31	mg/dL
2020-10-23 05:13:00	Creatinine Level	0.37	mg/dL
2020-10-22 16:24:00	Creatinine Level	0.33	mg/dL
2020-10-15 14:00:00	Creatinine Level	0.41	mg/dL
2020-10-01 10:03:00	Creatinine Level	0.48	mg/dL
2020-09-17 09:55:00	Creatinine Level	0.44	mg/dL
2020-09-03 05:23:00	Creatinine Level	0.38	mg/dL
2020-09-02 04:00:00	Creatinine Level	0.41	mg/dL
2020-09-01 10:40:00	Creatinine Level	0.49	mg/dL

Cardiovascular Anesthesiology

Cardiovascular Anesthesiology - 1

Electroconvulsive Therapy in a Patient with a Left Ventricular Assist Device

Daniel Fullin[1], Geoffrey Hayward[1]

[1]Brown University, Providence, RI

Introduction: Left ventricular assist devices (LVADs) provide mechanical circulatory support in patients with severe heart failure. LVAD implantation has been associated with improved survival compared to medical therapy alone. There has been a rapid expansion in the number of LVADs being implanted with more than 18,500 continuous flow LVADs reported in the Intermacs database. As LVAD implantation numbers have increased, patient outcome measures have also been improving (1). LVAD support is increasingly being used not just as a bridge to transplantation but also as destination therapy. As the number of patients with LVADs increases, anesthesiologists are more frequently being consulted to care for these patients during non-cardiac surgery. In this report we discuss a patient with a HeartMate II LVAD with medically refractory depression who presents for electroconvulsive therapy (ECT).

Methods: 75 year old male with a past medical history which includes ischemic cardiomyopathy with a left ventricular ejection fraction of 15%, coronary artery disease status post coronary artery bypass grafting, atrial fibrillation status post maze, left ventricular aneurysm repair, HeartMate II LVAD placement 3 years ago, spontaneous ventricular tachycardia status post ablation and implanted cardiac defibrillator placement, hypertension, chronic kidney disease, gastroesophageal reflux disease, and generalized anxiety disorder who presents for treatment of severe major depressive disorder with ECT. The patient reported substantial impairment in his quality of life with daily feelings of worthlessness, hopelessness, and suicidal thoughts all of which were refractory to medical therapy. After multidisciplinary discussions with the heart failure team, psychiatry, anesthesiology, and the patient, it was determined that ECT was likely to provide him with the safest and most effective treatment for his symptoms. The anesthetic for the patient's ECT treatment included 6mg of etomidate and 40mg of succinylcholine. The patient remained stable during the procedure with a slight increase in blood pressure and decrease in heart rate. He was given 5 mg of labetalol after the procedure for hypertension. The patient continued to remain stable after the procedure and was discharged home. Following this initial anesthetic, the patient continued his series of ECT treatments. They were well tolerated, without any adverse events. The patient has reported an improvement in his depressive symptoms.

Conclusion: ECT is a generally safe and effective procedure for rapid treatment of major depressive disorder. However, the procedure is associated with significant hemodynamic changes (2). Between the stimulus and the onset of the seizure there is increased vagal tone that results in bradycardia or even asystole in some cases. This is followed by catecholamine surges during and after the seizure that lead to an increase in heart rate and blood pressure. In this report we demonstrate that the hemodynamic changes were well tolerated in our patient. To the best of our knowledge, this is only the second report of successful ECT in a patient with a LVAD (3). Further research is necessary to determine the optimal anesthetic management of patients with a LVAD who present for ECT.

References: 1. Kormos, Robert L., et al. “The Society of Thoracic Surgeons Intermacs Database Annual Report: Evolving Indications, Outcomes, and Scientific Partnerships.” The Annals of Thoracic Surgery, vol. 107, no. 2, 2019, pp. 341–353., doi:10.1016/j.athoracsur.2018.11.011. 2. Tess, Anjala V., and Gerald W. Smetana. “Medical Evaluation of Patients Undergoing Electroconvulsive Therapy.” New England Journal of Medicine, vol. 360, no. 14, 2009, pp. 1437–1444., doi:10.1056/nejmra0707755. 3. Augustin, M., et al. “Electroconvulsive Therapy in a Patient with Left Ventricular Assist Device Following Deliberate Disconnection of the Device Driveline.” Brain Stimulation, vol. 10, no. 4, 2017, pp. 843–844., doi:10.1016/j.brs.2017.02.002.

Cardiovascular Anesthesiology - 2

Traumatic Atrial Septal Defect in Blunt Force Trauma

Manila Singh[1], Jason Altman[2]

[1]UConn Health, Farmington, CT, [2]Hartford Hospital, Department of Anesthesiology
University of Connecticut School of Medicine, Farmington, United States of America

Introduction: The incidence of traumatic atrial septal defects (ASD) after blunt force trauma is unknown. Only a few cases of isolated atrial septal injury have been reported in the literature. We report a case of traumatic ASD in a 24 year old, previously healthy female, which was discovered incidentally on transesophageal echocardiogram (TEE) during placement of veno-venous extracorporeal membrane oxygenation (ECMO) cannulas.

Methods: A 24-year-old female with no significant past medical history presented emergently to our level one trauma center for resuscitation and treatment. Massive transfusion protocol was initiated for multiple injuries and hemorrhagic shock. She underwent STAT exploratory laparotomy and right thoracotomy during which, pericardium was noted to be ecchymotic. A pericardial window was done leading to frank blood aspiration. Midline sternotomy was then performed and right atrial laceration was identified as the source of hemorrhage. This was repaired without the use of cardiopulmonary bypass. ECMO team was later consulted for hypoxia after right upper and middle lobectomy during part of the bronchus intermedius had to be oversewn albeit without difficulty in ventilation. TEE for V-V ECMO cannulae insertion, revealed an ejection fraction of 45%, moderate to severe right ventricular dysfunction and large 2.5cm ASD (Figure 4) with bidirectional shunt which was left alone given the coagulopathy and head injury. Over the subsequent days the coagulopathy and rhabdomyolysis resolved and the abdominal wound was successfully closed. Her mental status, however did not show any improvement with only bilateral lower extremity movement to painful stimuli. ECMO could not be weaned due to the bidirectional shunt from the traumatic ASD, seen on attempts at clamping the ECMO cannula. The pulmonology team eventually stented the obstructed bronchus to prevent post-obstructive pneumonia. After multidisciplinary discussion, the patient underwent surgical ASD closure with a Bovine pericardial patch on day 17 of hospitalization to enable weaning from ECMO. ECMO was weaned over the next two days with successful decannulation. MRI head done thereafter, showed diffuse axonal injury. She also remained dyssynchronous on the ventilator. Discussion about eventual right lung pneumonectomy to divert the pulmonary blood flow to the good left lung was underway on day 20 when she had an apneic episode with bradycardia which stabilized after bag masking and suctioning. Her family, having been counseled already about the neurological insult, decided to withdraw supportive care at that point. She unfortunately succumbed on day 21.

Conclusion: Isolated atrial septal rupture presenting acutely after blunt force trauma is extremely rare, with only a few cases reported in literature . The likely mechanism of injury is suggested in the drawings by the authors (Figure 1-3) Although an unfortunate conclusion to the medical course of the patient took over at the very end, there were several valuable lessons from her care and course of treatment. Isolated right atrial injuries can occur with blunt force trauma and should always be suspected especially in patients with hemothorax and extensive chest wall injuries. The absence of injury to other structures of the heart or great vessels does not exclude a traumatic ASD. The right atria is a low pressure chamber and is prone to injury and eventual transmission of force to the interatrial septum, especially during systole. Early repair is dictated by comorbid conditions and anatomy of defect. This injury can easily be missed intraoperatively if not included as part of the differential diagnosis for hypoxia in trauma. We suspect the true incidence is likely more common than reported in literature due to the low utilization of TEE in multi trauma situations with facial and vertebral injuries on the backdrop of initial panscan ruling out major cardiovascular injuries. Here, this injury was discovered incidentally because TEE was requested by Cardiothoracic Surgery to assist with ECMO cannula placement. Although, traumatic ASD discovery and eventual repair did not change the ultimate course of her hospitalization, it did allow her family to spend additional time with her at the end of her life. Recovery from this injury could be possible in

patients without serious injuries to other organ systems.

References: 1. Baumgartel, E.D. Cardiac rupture from blunt trauma with atrial septal defect. Arch Surg. 1992;127:347–348 2. MA Huis, CA Craft, RE Hood. Blunt cardiac trauma review. Cardiology clinics, 2018; cardiology.theclinics.com 3. J Menaker, RB Tesoriero, M Hyder et.al. Traumatic atrial septal defect and papillary muscle rupture requiring mitral valve replacement after blunt injury J Trauma. 2009 Nov;67(5):1126.

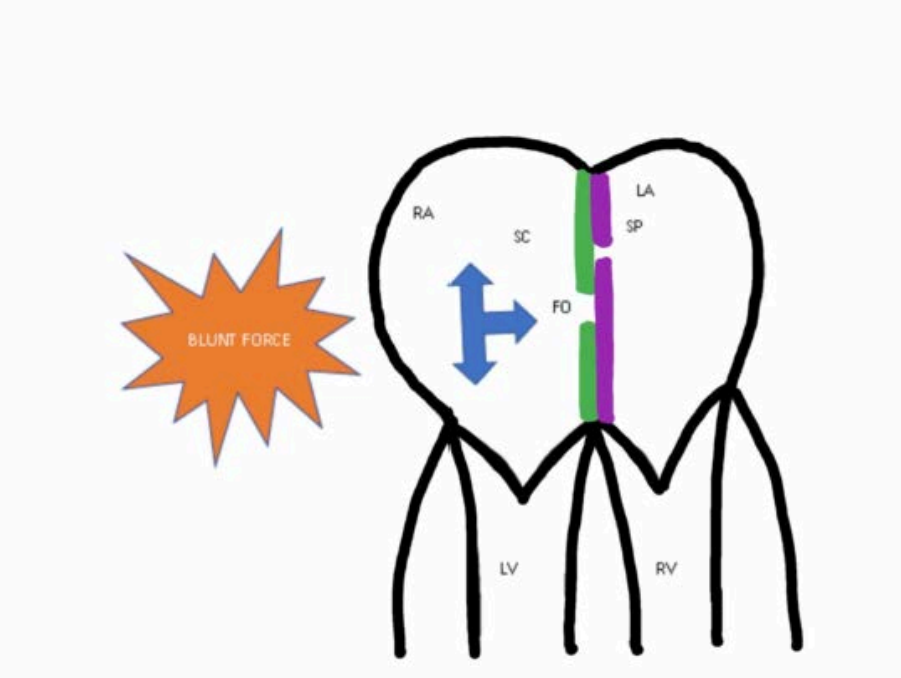


Fig 1: Drawing depicting the transmission of blunt force concentrated to the right atrial wall and the inter atrial septum as in our patient who incurred RA wall tear as well as a tear of the IAS. RA Right atrium, LA left atrium, SC Septum secundum, SP Septum primum, FO Foramen ovals, RV Right ventricle, LV Left ventricle.

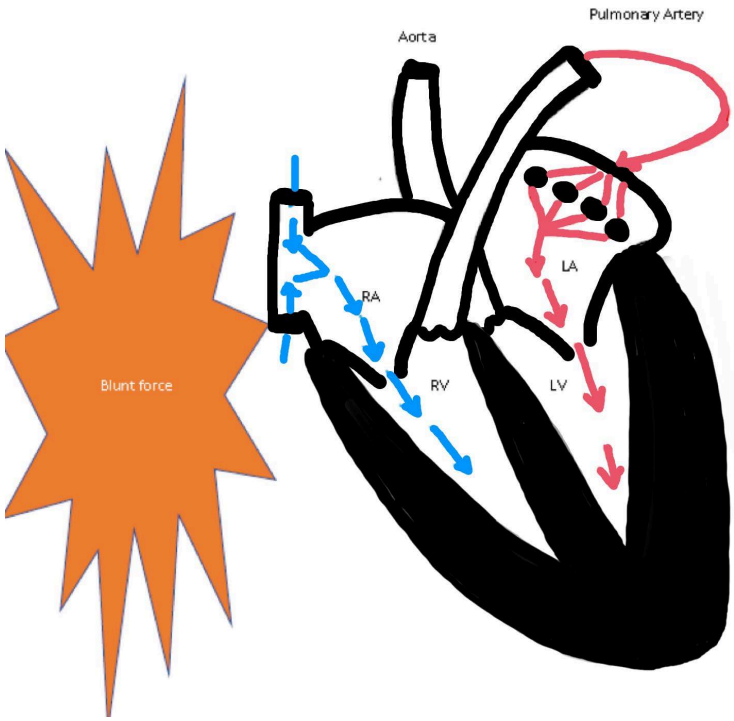


Fig 2: Blunt force trauma during cardiac diastole. Drawing demonstrates that owing to open atrioventricular valves and closed ventricular outflow tracts, the force will most likely get concentrated over the ventricles and inter ventricular septum and the great vessels at the ligamentous attachments.

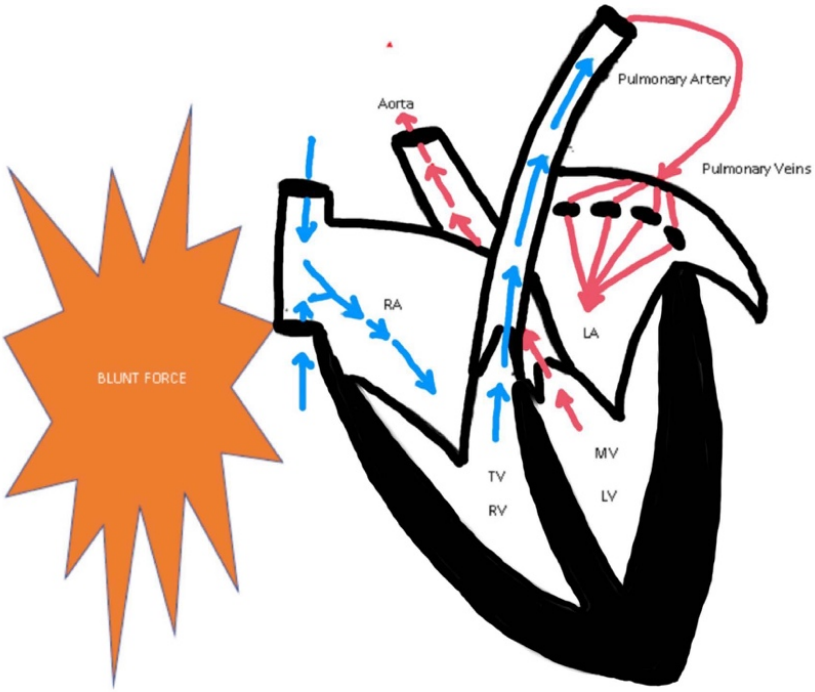


Fig 3: Blunt force during systole. Drawing demonstrates that blunt force during systole is likely to be concentrated at the right atrium and the inter atrial septum as the force over the ventricles gets dissipated by the forward flow through the great vessels.

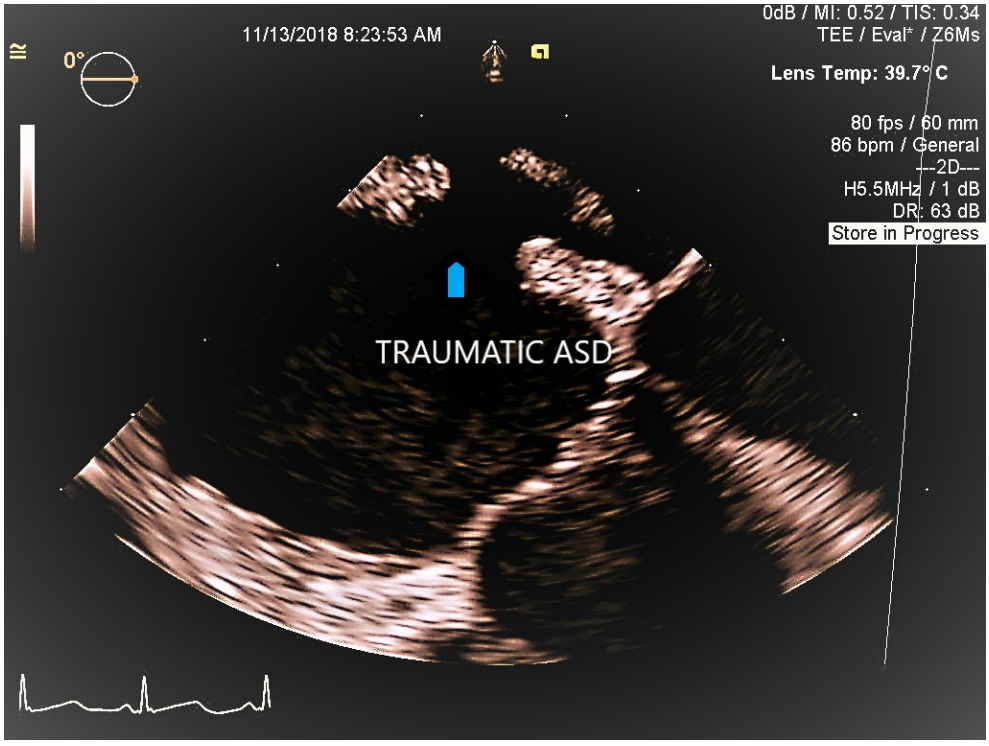


Fig 4. TEE depicting ASD

Cardiovascular Anesthesiology - 3

Inferior Basal Ventricular Septal Rupture: A Devastating Complication Of Myocardial Infarction

Yvonne Fetterman[1], Benu Makkad[2]

[1]University of Cincinnati Medical Center, Cincinnati, OH, [2]UCMC, cincinnati, OH

Introduction: Post myocardial infarction (MI) ventricular septal rupture (VSR) is extremely rare with poor survival. Anatomic location, morphology, and timing of presentation may vary (Figure 1) (1, 2). Medical management is typically futile (1). Surgery is the treatment of choice, but operative mortality is 42.9%, the highest of any cardiac surgery (1). When cardiogenic shock (CS) is present, in-hospital mortality climbs to 87.3% with surgical repair (3). Percutaneous closure (PC) of VSR has been used as primary repair and as a temporizing measure, but outcomes are equally poor with a 30-day mortality of 88% in patients with CS (4). There is lack of consensus on the optimal timing of intervention. We describe a patient in CS who was found to have VSR and multi-vessel coronary artery disease (CAD) requiring a multidisciplinary approach to coronary revascularization and PC of his VSR.

Methods: A 68-year-old male with a past medical history of hypertension presented with dyspnea and acute hypotension. Electrocardiogram showed a subacute ST segment elevation MI with inferior lead Q waves, and left heart catheterization (HC) revealed multi-vessel CAD. A step-up in oxygen saturation in the pulmonary artery was noted on right HC suggesting VSR. Transesophageal echocardiography (TEE) confirmed the presence of a 1.5 cm inferior basal VSR and severely reduced biventricular function (Figure 2). A percutaneous ventricular assist device was placed for refractory hypotension. Due to continued hemodynamic instability, venous arterial extracorporeal membrane oxygenation (VA ECMO) was also implemented. A week later, the patient underwent coronary revascularization and trans-atrial closure of the VSR with a double disk occlusion device under TEE guidance (Figure 3). Intraoperative TEE showed mild-to-moderate residual shunt due to lack of a superior muscular rim and new moderate-to-severe tricuspid regurgitation (Figures 4, 5). Post-procedure efforts to wean VA ECMO were unsuccessful, and care was withdrawn by the family.

Conclusion: VSR complicating acute MI is rare, with a recent reported incidence of 0.17 to 0.31% (1). Despite advances in surgical and percutaneous approaches, the mortality rate from VSR is dismal. In addition, posterior septal rupture has been cited as a risk factor for operative death and poses challenges for PC due to proximity of the tricuspid valve (1, 2, 4, 5). Timing of VSR repair is controversial. Contrary to U.S. guidelines, which recommend immediate operative intervention regardless of hemodynamic status due to high risk of acute decompensation, recent European guidelines suggest that delayed repair may benefit those responding to aggressive management to allow stabilization of infarcted tissue (2, 4, 6). For our patient, PC of the VSR and surgical coronary revascularization were performed with the hope of recovering biventricular function to allow future definitive surgical repair. Sadly, our patient's VSR was complicated by CS, large size, and inferior location. Despite an individualized multidisciplinary team approach, his ventricular function never recovered.

References: 1. Jones BM, Kapadia SR, Smedira NG, et al. European Heart Journal, vol. 35, no. 31, pp. 2060-2068, 2014. 2. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. New England Journal of Medicine, vol. 347, no. 18, pp.1426-1432, 2002. 3. Menon V, Webb JG, Hillis LD, et al. Journal of the American College of Cardiology, vol. 36, no. 3, pp. 1110-1116, 2000. 4. Thiele H, Kaulfersch C, Daehnert I, et al. European Heart Journal, vol. 30, no. 1, pp. 81-88, 2009. 5. Arnaoutakis GJ, MD, Zhao Y, PhD, George TJ, MD, Sciortino, Christopher M., MD, PhD, McCarthy PM,MD, Conte JV, MD. The Annals of Thoracic Surgery, vol. 94, no. 2, pp. 436-444, 2012. 6. Ibanez B, James S, Agewall S, et al. European Heart Journal, vol. 39, no. 2, pp. 119-177, 2018.

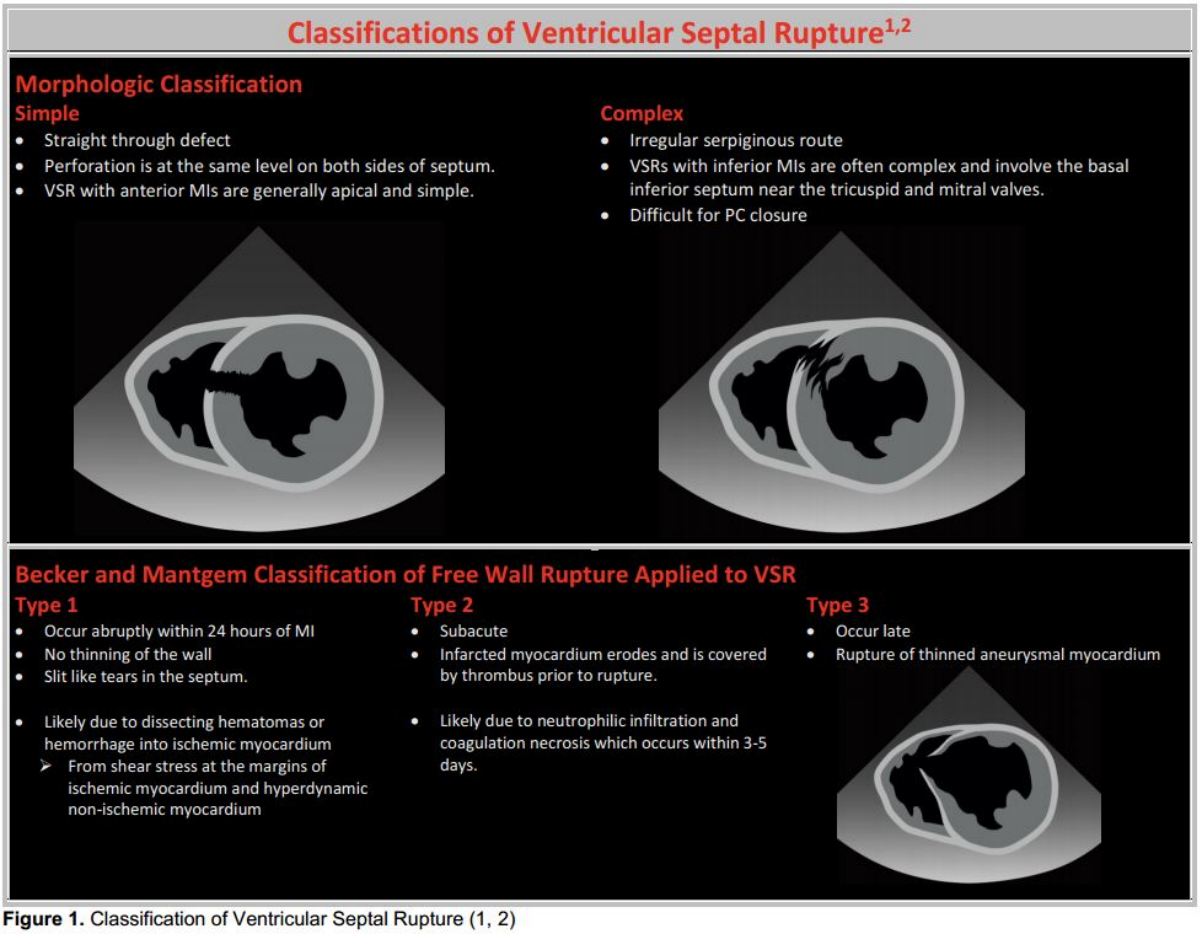


Figure 1. Classification of Ventricular Septal Rupture (1, 2)

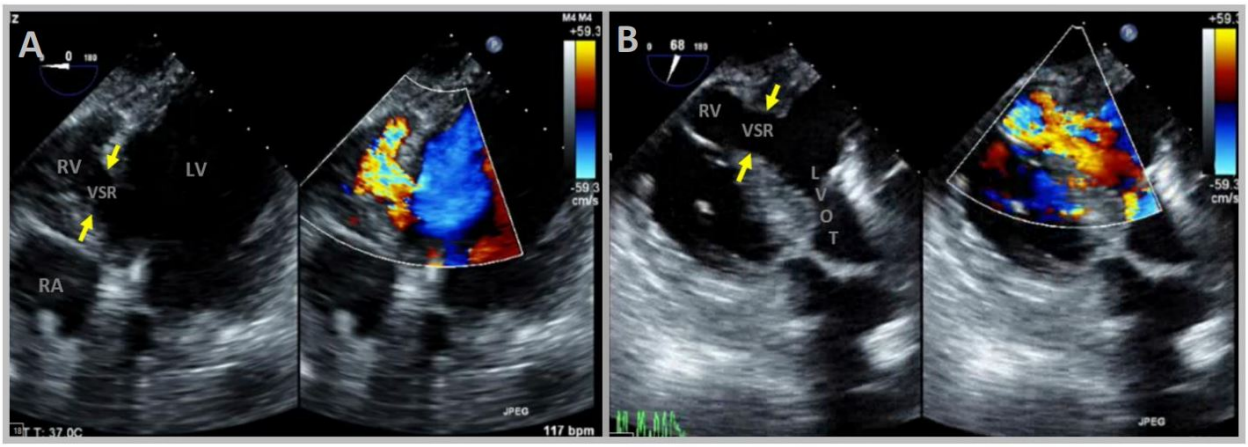


Figure 2: (A) Deep Transgastric view shows large VSR (yellow arrows) with left to right shunt. (B) Deep transgastric view shows the left ventricular outflow tract sharing flow with the VSR (yellow arrows). RV indicates right ventricle; LV, left ventricle; RA, right atrium; LVOT, left ventriclar outflow tract; VSR, ventricular septal rupture.

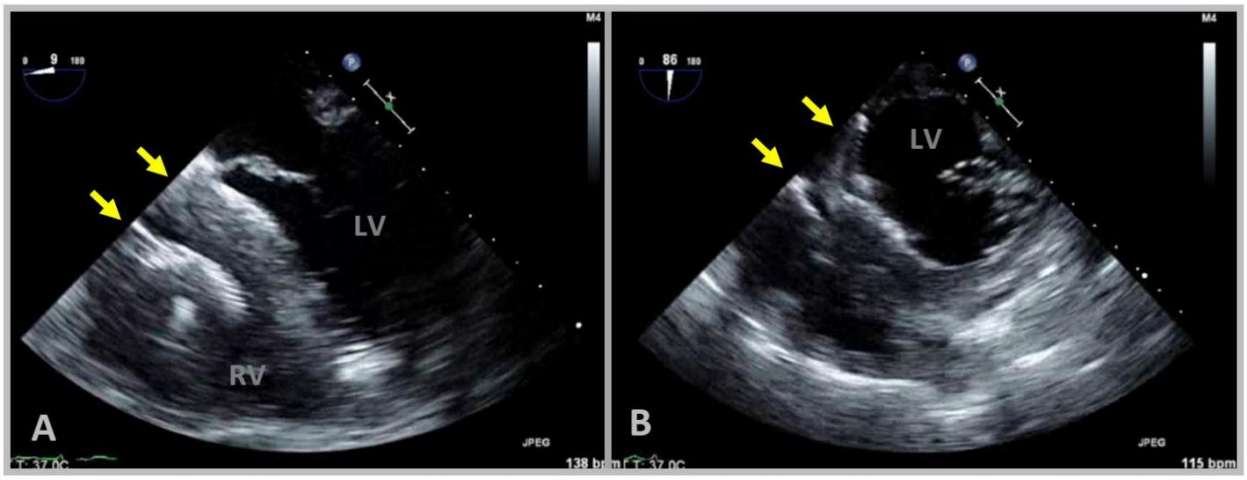


Figure 3: (A) Mid-esophageal 4-chamber view and (B) Transgastric short axis view showing closure of the VSR with a double disk device (double yellow arrows). RV indicates right ventricle; LV, left ventricle.

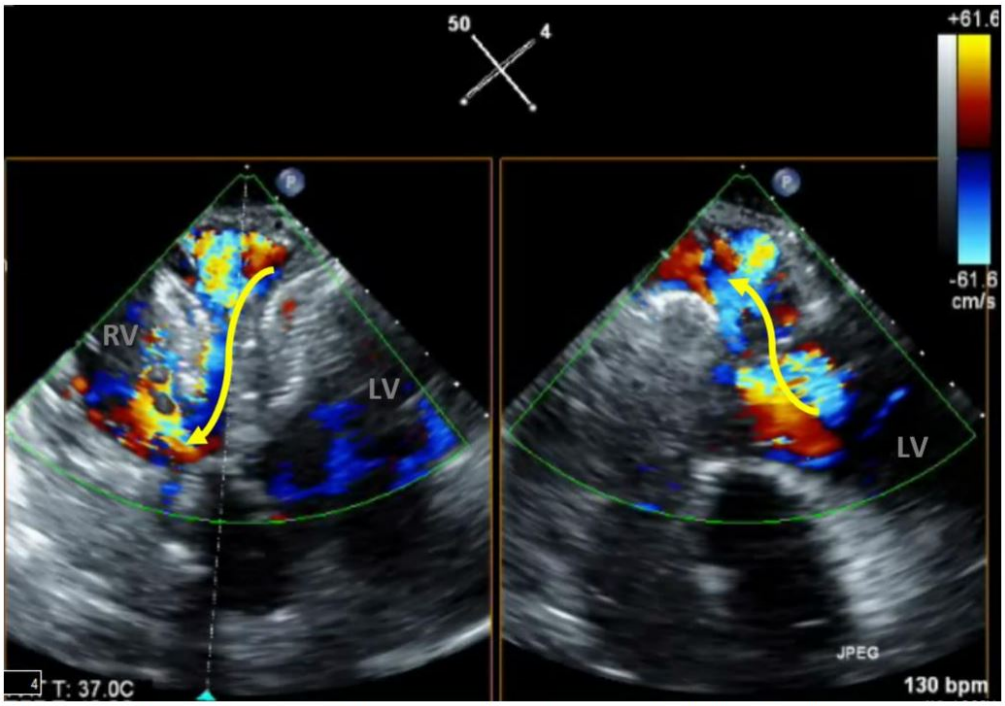


Figure 4: Deep transgastric views showing residual flow (yellow arrows) around double disk occlusion device. RV indicates right ventricle; LV, left ventricle.

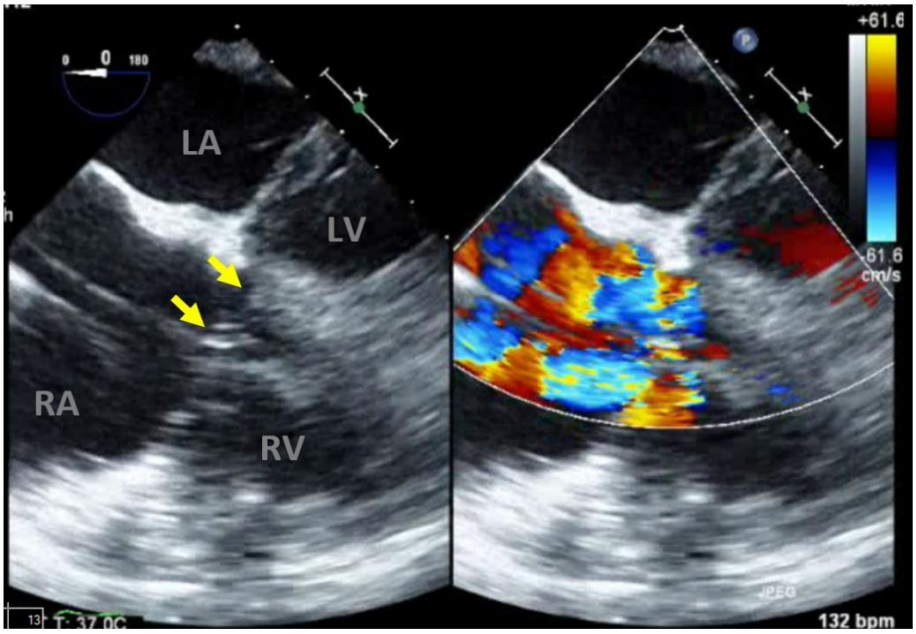


Figure 5. Mid-esophageal short axis views showing moderate-severe tricuspid regurgitation and disk interfering with tricuspid valve (double yellow arrows). RV indicates right ventricle; LV, left ventricle; RA, right atrium; RA, right atrium.

Cardiovascular Anesthesiology - 4

Managing Right Ventricular Failure (RVF) in Massive Pulmonary Embolism with Percutaneous Right Ventricular Assist Device (RVAD)

Annandita Kumar[1], Jayanta Mukherji[2]

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

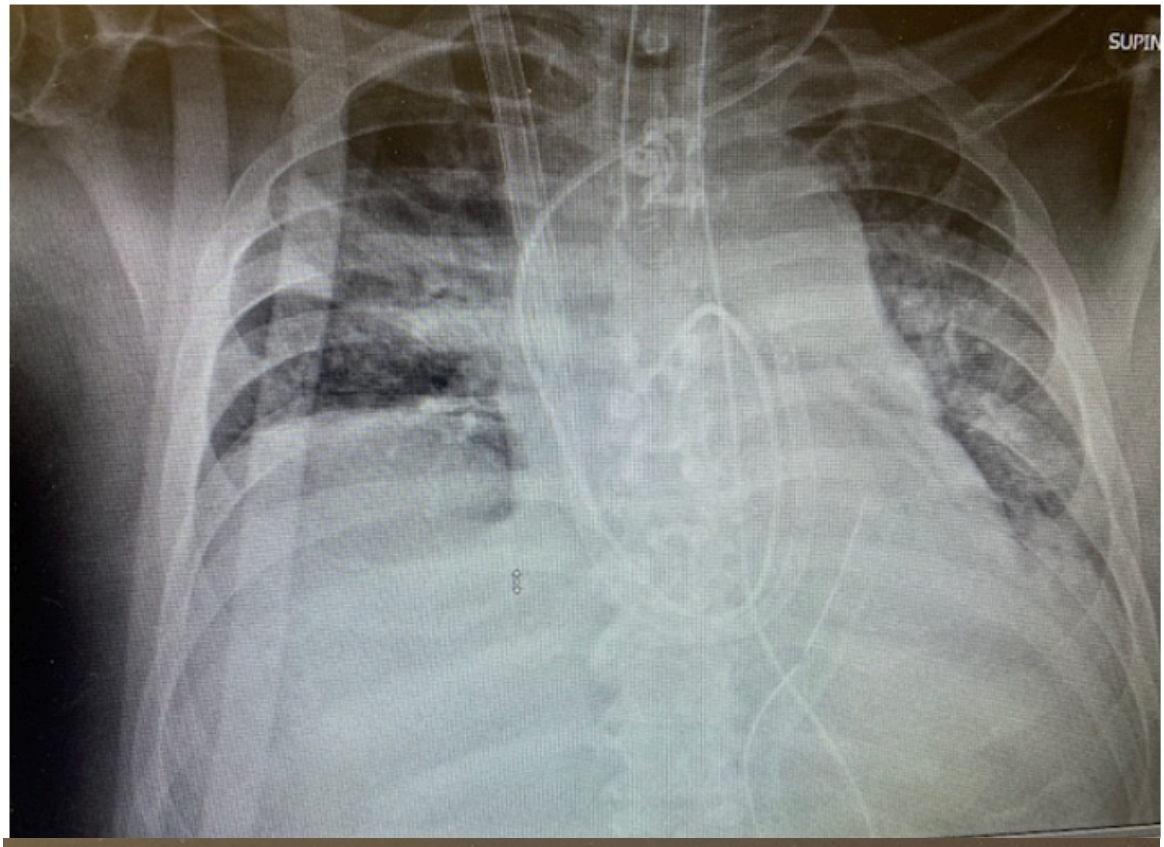
Introduction: Acute right ventricular failure remains the leading cause of mortality associated with acute pulmonary embolism (PE). Management is predicated on an understanding of the physiology of the RV in order to carefully support its cardiac output. When medical therapies are insufficient, mechanical circulatory support (MCS) is needed to maintain systemic and RV perfusion. Acute RV failure is a life-threatening condition with a poor prognosis, sometimes necessitating the use of mechanical circulatory support (MCS). Most MCS device placement for RV support has been for RV failure in the context of left ventricular assist device (LVAD) placement (1). We describe a patient with massive pulmonary embolism s/p pulmonary embolectomy who was initially stabilized post CPB with VA ECMO. However, with ongoing RV failure and normal LV function a temporary percutaneous Protek Dual-Lumen RV assist device (RVAD) was used to selectively support the RV.

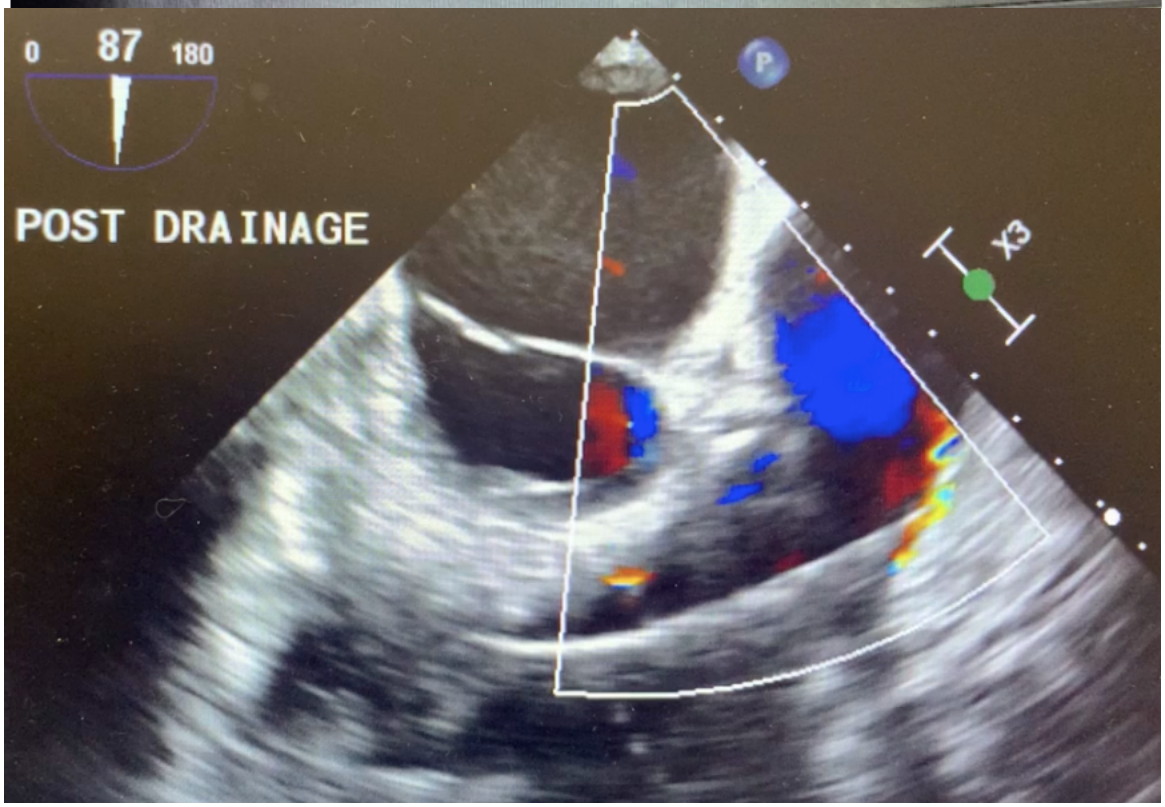
Methods: A previously healthy 58y male developed acute shortness of breath after a plane ride. TTE showed an in-transit clot in the RA and RV and a CT scan that showed submassive emboli in his lungs. Pulmonary embolectomy was planned for clot in-transit and concern for abrupt hemodynamic deterioration. The patient was somewhat unstable on induction. Intraoperatively large organized PA clots were extracted and the PA catheter was advanced into the main PA. His deteriorating RV function with elevated CVP and PA pressures prevented weaning. Post-CPB he was supported with inotropes, VA ECMO (IVC-Femoral artery), and intra-aortic balloon pump (IABP). TEE showed underfilled LV with normal contractility, dilated hypokinetic RV, and moderate TR with mild improvement on VA ECMO off-loading. Hypoxemic respiratory failure was the result of V/Q mismatch and massive pulmonary embolic burden. Hypoxemia and RV failure persisted and he was converted to VV ECMO on the 7th post-operative day, using a Protec Duo RA/PA ECMO, with the removal of VA (IVC-Fem artery) ECMO. Cannula in PA was positioned just above the pulmonary valve and confirmed on TEE. ECMO flows adjusted to maintain pO2 over 50-55 mm Hg. The patient developed a pro-thrombotic state with multiple DVTs and catheter thromboses and required Bivalirudin infusion for suspected HIT. With clinical improvement, he was decannulated from ECMO on the 19th post-operative day. Complications ensued with AKI from cardiogenic shock requiring CVVH and sepsis from C. Difficile and fungemia. RV function rapidly deteriorated with severe pulmonary artery hypertension, likely from ongoing pulmonary embolic burden. Severe hypoxemia despite optimal medical therapy and mechanical ventilation necessitated the withdrawal of care after 28 days of hospitalization.

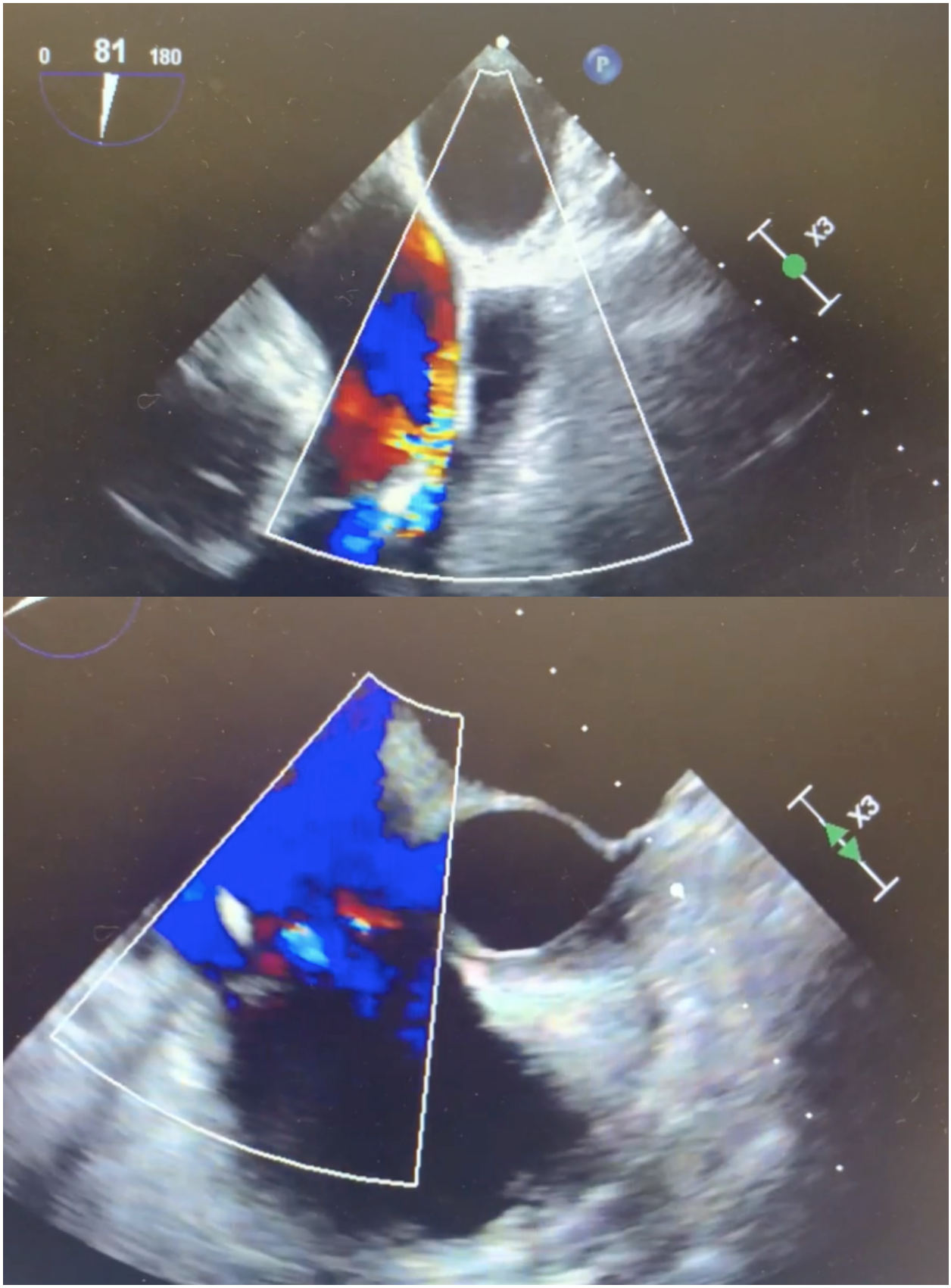
Conclusion: Strategies for managing RV failure in massive PE include immediate clot reduction via systemic thrombolytics, catheter-based procedures, or surgery if stable. While waiting to mobilize these resources it often becomes necessary to support the RV with vasoactive medications or RVAD. An acute PE causes a sudden increase in RV afterload, both through direct increases in PVR from clot burden as well as through neurohormonal and hypoxia-mediated mechanisms. In this case, massive pulmonary embolism with an in-transit clot prompted pulmonary embolectomy, bypassing fibrinolytic therapy. Cardiogenic shock from RV failure was managed with inotropes, inhaled epoprostenol while VA ECMO was used as a bridge to recovery. The Protek Duo (TPD) is a temporary RVAD that is capable of providing up to 4.5 L of flow. In this case, it was placed percutaneously via the right internal jugular vein with the proximal inflow lumen positioned in the RA and distal outflow lumen in the main PA trunk (2). This system provides full RV support and oxygenation. The data on MCS for isolated RV failure are limited and most case reports have described their use for transient support of RV after LVAD implantation (3). Our case report suggests that Protek Duo may be used as a safe alternative for support in isolated refractory RV failure in the context of massive PE. The ease of insertion, weaning and explantation of this percutaneous device limits

complications and assists inpatient mobility justifying a liberal use of this device.

References: 1. Management of Right Ventricular Failure in Pulmonary Embolism. Crit Care Clin. 2020 Jul;36(3):505-515. doi: 10.1016/j.ccc.2020.02.006. Epub 2020 May 1. PMID: 32473695. 2. Outcomes with the Tandem Protek Duo Dual-Lumen Percutaneous Right Ventricular Assist Device. ASAIO J. 2018 Jul/Aug;64(4):570-572. doi: 10.1097/MAT.0000000000000709. PMID: 29095736. 3. Novel percutaneous dual-lumen cannula-based right ventricular assist device provides effective support for refractory right ventricular failure after left ventricular assist device implantation. Interact Cardiovasc Thorac Surg. 2020 Apr 1;30(4):499-506. doi: 10.1093/icvts/ivz322. PMID: 31986207.


SUPIN


0
87
180
POST DRAINAGE
X3


0
81
180
P
X3
X3

Cardiovascular Anesthesiology - 5

Resection and repair of the descending thoracic aorta utilizing left heart bypass

Marcus Bruce[1], Bradford Smith[2]

[1]Mayo Clinic Arizona, Phoenix, AZ, [2]Mayo Clinic, Scottsdale, AZ

Introduction: Left heart bypass (LHB) is a surgical technique often utilized for open surgical repair of the thoracoabdominal aorta. Left heart bypass is accomplished by cannulation of the left atrial appendage, left pulmonary vein, left ventricle apex, or the aorta itself. They oxygenated blood then passes through a centrifugal pump with the return cannula placed distal to the aortic cross clamp. LHB requires close hemodynamic monitoring as blood is commonly shunted away from the native circulation to the upper body and brain toward the lower extremities via the LHB return cannula. Slowing LHB pump flow provides more blood for upper body and brain perfusion while high flow rates cause increased shunting. Invasive blood pressure monitoring is typically implored above and below the aortic cross clamp to assist in strict hemodynamic management (1). Blood pressure augmentation should be undertaken to maximize spinal cord perfusion given the risk of ischemia due to aortic cross clamping.

Methods: We present a case of a 49-year-old male with a past medical history of testicular, papillary thyroid, and esophageal adenocarcinoma with vascular invasion of the descending thoracic aorta. The patient underwent left thoracotomy for resection and repair of the thoracic aorta with LHB. The left superior pulmonary vein and distal thoracic aorta were cannulated. Femoral and radial arterial lines were used for hemodynamic monitoring with mean arterial pressure goals of 60-70 and 80-90 mmHg respectively. Clevidipine, phenylephrine and pump flow rates were used to augment hemodynamics. Posterior thoracic aortic bleeding complicated the surgical intervention and the patient was transfused 1 unit packed red blood cells and volume resuscitated. The patient tolerated the procedure well and was extubated in the operating room and transferred to the intensive care unit neurologically intact. The patient then underwent successful minimally invasive esophagectomy two days later. His postoperative course was uncomplicated and he was ultimately discharged home on postoperative day 11.

Conclusion: As demonstrated in our case, LHB is a technique that can be implemented to minimize distal ischemia when aortic cross clamping is required. LHB has also been shown to reduce the risk of postoperative paraplegia and paraparesis in patients undergoing thoracoabdominal surgical repair (2). While distal aortic perfusion techniques are commonly used for repair of aortic aneurysms, other pathologies such as those seen in our patient can lead to aortic compromise and LHB can facilitate surgical intervention. Success of LHB depends on open communication with the surgeon, anesthesiologist, and perfusionist to ensure adequate perfusion of the patient (3). This open communication should also include discussion of the location of aortic cross clamping and area of surgical resection due to the possibility of compromise of the artery of Adamkiewitz resulting in spinal cord ischemia.

References: 1. Schepens MA. Left heart bypass for thoracoabdominal aortic aneurysm repair: technical aspects. Multimed Man Cardiothorac Surg. 2016 Jan 28;2016 2. Coselli JS. The use of left heart bypass in the repair of thoracoabdominal aortic aneurysms: current techniques and results. Semin Thorac Cardiovasc Surg. 2003 Oct;15(4):326-32. 3. Ailawadi G, Zacour RK. Cardiopulmonary bypass/extracorporeal membrane oxygenation/left heart bypass: indications, techniques, and complications. Surg Clin North Am. 2009 Aug;89(4):781-96

Cardiovascular Anesthesiology - 6

Perioperative management of the adult on VA ECMO requiring PE mechanical thrombectomy

Jin Min[1], Mon Briones[1], Luke Landolt[1], Alison Alpert[1]

[1]Saint Louis University, St. Louis, MO

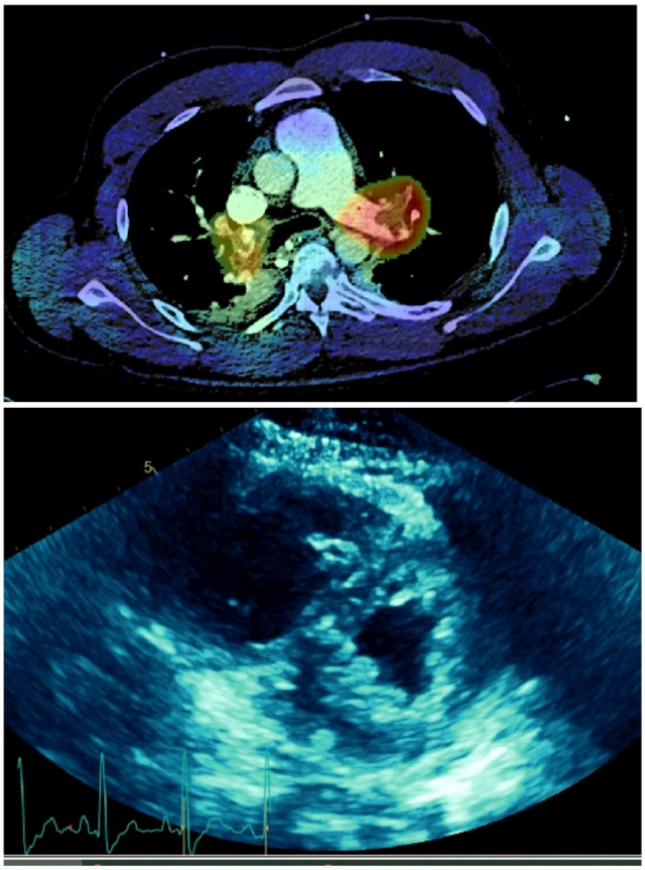
Introduction: The role of veno-arterial (VA) ECMO has been utilized in patients with massive pulmonary embolisms with significant right ventricular strain as a salvage therapy (1,2,3). In the past several years, it has shifted towards a destination or initial therapy as studies have shown lower morbidity, morality and improved patient outcomes. This article will address the fundamentals of VA ECMO and it's anesthetic considerations for pulmonary embolism.

Methods: The patient is a 31 year-old African-American male with a history of a prior pulmonary embolism with no identifiable cause and negative coagulation work up who presented with shortness of breath and chest pain. CT angiography revealed a massive saddle pulmonary embolism. As he was hemodynamically stable he was started on a heparin infusion, instead of tPA. Shortly afterwards, he had a syncopal episode and went into PEA arrest. After 40 minutes of CPR, ROSC was achieved. The patient had 3 subsequent episodes of cardiac arrest over the next hour in which he was able to be fully resuscitated. Cardiology emergently cannulated the patient for V-A ECMO with flow rates of 6L/min and a sweep speed of 4L/min. Bedside TTE revealed LVEF 40% (decreased from 65%), and severe pulmonary artery systolic pressures of 71 mmHg. His RV:LV ratio was 1.1. Interventional radiology then performed an emergent PE mechanical thrombectomy with anesthesia. Intraoperatively, the patient was anesthetized with 0.5 mac of sevoflurane. Due to severe pulmonary hypertension, milrinone and vasopressin infusions were started. An epinephrine infusion was initiated when arterial line waveforms were lost, indicating he was completely ECMO dependent, requiring further inotropic support. After successful thrombectomy, end-tidal carbon dioxide improved from 0-5 mmHg to 20 mmHg. Postoperatively, the patient exhibited rapid clinical improvement and he was decannulated successfully two days later, extubated the following day and then discharged home.

Conclusion: Risk stratification of pulmonary embolism can be classified as massive, sub-massive or low risk. Massive is defined as having hemodynamic instability, while submassive presents with hemodynamic stability but with signs of RV dysfunction. ECMO has become a newer option for treatment of patients with massive PE due to the significant right heart strain that can be caused by clot burden. Intraoperative anesthetic considerations must be made for the obstructive shock. Milrinone provides positive inotropy and lusitropic effects without further increasing pulmonary vascular resistance (PVR), but can cause hypotension. Vasopressin is often used as an adjunct as it can raise systemic vascular resistance without affecting PVR unlike other vasopressors (4). With the rapid growth of ECMO utilization, it is important to understand that anesthesiologists and their vigilance improve patient outcomes (4). By utilizing ECMO as a first line therapy rather than tPa for this patient, he was able to have a short hospital stay without any complications. By working closely with perfusionists, we worked closely together to assess hypoxemia and hypercarbia and to allow for adjustment of sweep speeds. The patient's Pao2/paCo2 is primarily dictated by blood flow through the ECMO, sweep speeds which will assist with decarboxylation, cardiac output and ventilator assistance. ETCo2 was grossly inaccurate and a swan catheter or intra-op TEE may have provided more information. Lastly, it is crucial to keep patients normothermic during the procedure as hypo/hyperthermia has shown increased risks of bleeding, myocardial injury and high rates of renal damage (5).

References: Michael A. Fierro, Mani A. Daneshmand, Raquel R. Bartz; Perioperative Management of the Adult Patient on Venovenous Extracorporeal Membrane Oxygenation Requiring Noncardiac Surgery. Anesthesiology 2018; 128:181–201 doi: https://doi.org/10.1097/ALN.0000000000001887’
Maxwell A. Hockstein, et. al. Institutional Experience with Veno-Arterial Extracorporeal Membrane Oxygenation for Massive Pulmonary Embolism: A Retrospective Case Series. Journal of Cardiothoracic

and Vascular Anesthesia January, 2020.12.045
Monica I. Lupei, Benjamin Kloesel, Lida Trillos, Ioanna Apostolidou; Survival of intraoperative massive pulmonary embolism using alteplase and VA-ECMO, Journal of Clinical Anesthesia, Volume 57, 2019, Page 112, ISSN 0952-8180, https://doi.org/10.1016/j.jclinane.2019.03.007.
Alibrahim OS, Heard CMB. Extracorporeal Life Support: Four Decades and Counting. Curr Anesthesiol Rep. 2017;7(2):168-182. doi:10.1007/s40140-017-0210-0 Bergan HA, Halvorsen PS, Skulstad H, Fosse E, Bugge JF. Doe



Cardiovascular Anesthesiology - 7

Postoperative Takotsubo Cardiomyopathy Manifesting as Nausea and Agitation Upon Emergence from Anesthesia Following Robotic Cholecystectomy

Nicole Massucci[1], Marc S Kanchuger[1]

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

Introduction: Takotsubo cardiomyopathy (TTCM) is being reported with increasing frequency in hospitalized patients, including perioperatively. It is thought to be precipitated by a medical condition, surgery or procedure, and possibly anesthesia itself. This secondary type of TTCM is what is encountered by anesthesiologists. TTCM has been described in the preoperative, intraoperative, and postoperative periods with a variety of presentations, durations, and associated complications. It has been seen with both cardiac and noncardiac surgery, open versus laparoscopic surgery, as well as with different types of anesthesia. There is no consensus on the optimal treatment and prevention of this condition, nor the optimal anesthesia for subsequent surgeries in such patients. This is a case of a postmenopausal woman with a significant cardiac history who had undergone multiple previous uncomplicated surgeries, that developed TTCM immediately after an elective robotic cholecystectomy. The subsequent hospitalization was complicated by cardiogenic shock and persistent atrial fibrillation with rapid response following a brief dobutamine infusion.

Methods: 81 year old female former smoker with a history of two cardiac stents placed twelve years prior, bilateral moderate carotid stenosis, hypertension, hyperlipidemia with statin intolerance, and chronic pain presented for elective robotic cholecystectomy due to chronic cholecystitis. She had undergone various surgical procedures without complication both before and after her cardiac stents. Preoperatively, the patient was not anxious and vital signs were within normal limits. Intraoperatively, induction of general anesthesia and intubation was uneventful followed by uncomplicated surgery. Upon emergence, the patient immediately began to complain of nausea, denied chest pain but was agitated and inconsolable. Upon arrival to the post-anesthesia care unit, the patient remained uncomfortable and supraventricular tachycardia was observed on the monitor. An EKG demonstrated ST-elevations in the lateral leads with reciprocal changes inferiorly and QTc prolongation. She was taken immediately for coronary angiography where no coronary obstruction was found and apical ballooning was appreciated on left ventriculogram. Her ejection fraction was severely decreased and a formal echocardiogram that was ordered to rule out obstruction found the ejection fraction to be twenty percent. She was started on a dobutamine infusion due to laboratory evidence of end organ dysfunction and shortly thereafter developed non-sustained ventricular tachycardia and atrial fibrillation with rapid ventricular response to 200. She was started on anticoagulation and multiple antiarrhythmic drugs before eventually converting to normal sinus rhythm on postoperative day two. She was discharged on postoperative day four with a beta blocker, ace inhibitor, and oral anticoagulation.

Conclusion: While there are many reported cases of perioperative TTCM, this may be one of the first reported cases to involve a robotic surgery. Although this patient fits the commonly seen demographic of a post-menopausal woman with hypertension and hyperlipidemia, many observational studies involving stress cardiomyopathy excluded patients with a history of cardiac stents and thus it is possible that this was an additional risk factor. While anti-adrenergic medications and ace inhibitors have been recommended for treatment and prevention, there is little evidence to support any specific treatment. Our patient was already taking a moderate dose of beta blocker as well as ace inhibitor prior to surgery. She received additional beta blockade prior to the coronary angiography and it is likely that this may have worsened her myocardial function and cardiogenic shock before it was identified. The short period of inotropic support that preceded the arrhythmias might have been a casual factor, and there is controversy surrounding the use of adrenergic drugs versus mechanical support devices such as intraaortic balloon pumps during low output states. Lastly, our patient had a history of chronic pain and there is likely an interplay between pain transmission, catecholamine response, and the development of TTCM. More research is needed on TTCM in the perioperative period regarding

risk factors, diagnosis and presentation, type of anesthesia, as well as optimal treatment and prevention.

References: Journal of Cardiothoracic and Vascular Anesthesia, Volume 22, Issue 3, June 2008, Pages 438-441 American Heart Journal, Volume 164, Issue 1, July 2012, Pages 66-71.e1 Eur Heart J Acute Cardiovasc Care, Volume 6, Issue 3, February 2016, Pages 280-286 N Engl J Med 2015; 373:929-938 Eur Heart J Acute Cardiovasc Care, 2016 Aug;5(4):308-16. Canadian Journal of Anesthesia, 2016, Volume 63, pages1059-1074. Anesthesia & Analgesia: March 2010 - Volume 110 - Issue 3 - p 712-715 Anesthesia & Analgesia: March 2010 - Volume 110 - Issue 3 - p 674-679 Int J Cardiol. 2014 Jul 1;174(3):696-701 Ann Card Anaesth. 2019 Jul-Sep; 22(3): 309-315

Cardiovascular Anesthesiology - 8

Mobile Pledget Masquerading as Late Endocarditis in Severe Bioprosthetic Aortic Regurgitation

Mohammad Sadat[1], Mark Heath[2], Craig Smith[2], Jessica Spellman[3]

[1]Columbia University, New York, United States of America, [2]Columbia University Medical Center, New York, United States of America, [3]Columbia University Medical Center, New York, NY

Introduction: In patients with bioprosthetic aortic valves, aortic regurgitation typically occurs through the mechanisms of transvalvular regurgitation or paravalvular leakage. Transvalvular regurgitation may be secondary to structural valve deterioration or non-structural valve dysfunction such as with endocarditis or thrombus.[1] In patients with bioprosthesis, 10 to 30 percent had significant structural valvular disease at 10 years, and 20 to 50 percent at 15 years.[2] Furthermore, the pathogenesis of late onset bioprosthetic valve endocarditis is thought to resemble native valve endocarditis and affect the valve leaflets.[3] We will discuss a case of severe bioprosthetic aortic regurgitation secondary valve deterioration that masqueraded as endocarditis due to findings on transesophageal echocardiography (TEE).

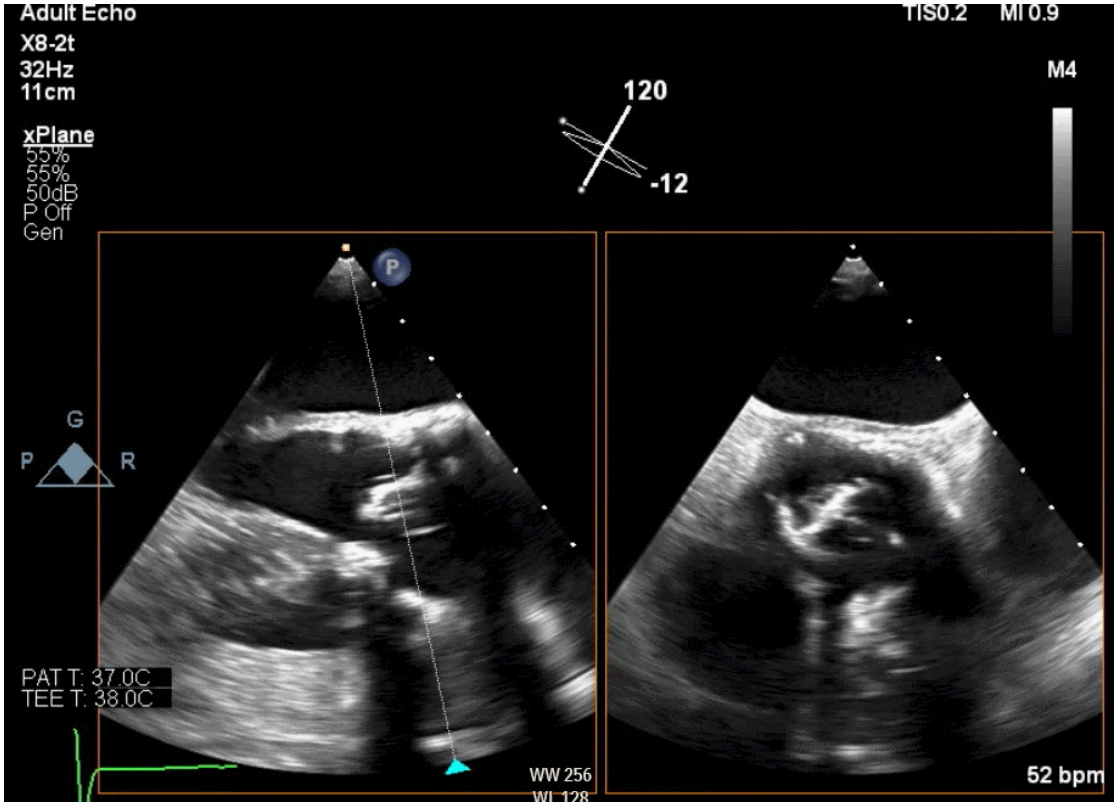
Methods: A 67 year-old woman presented for a reoperative aortic valve replacement for severe prosthetic valve aortic regurgitation (AR).

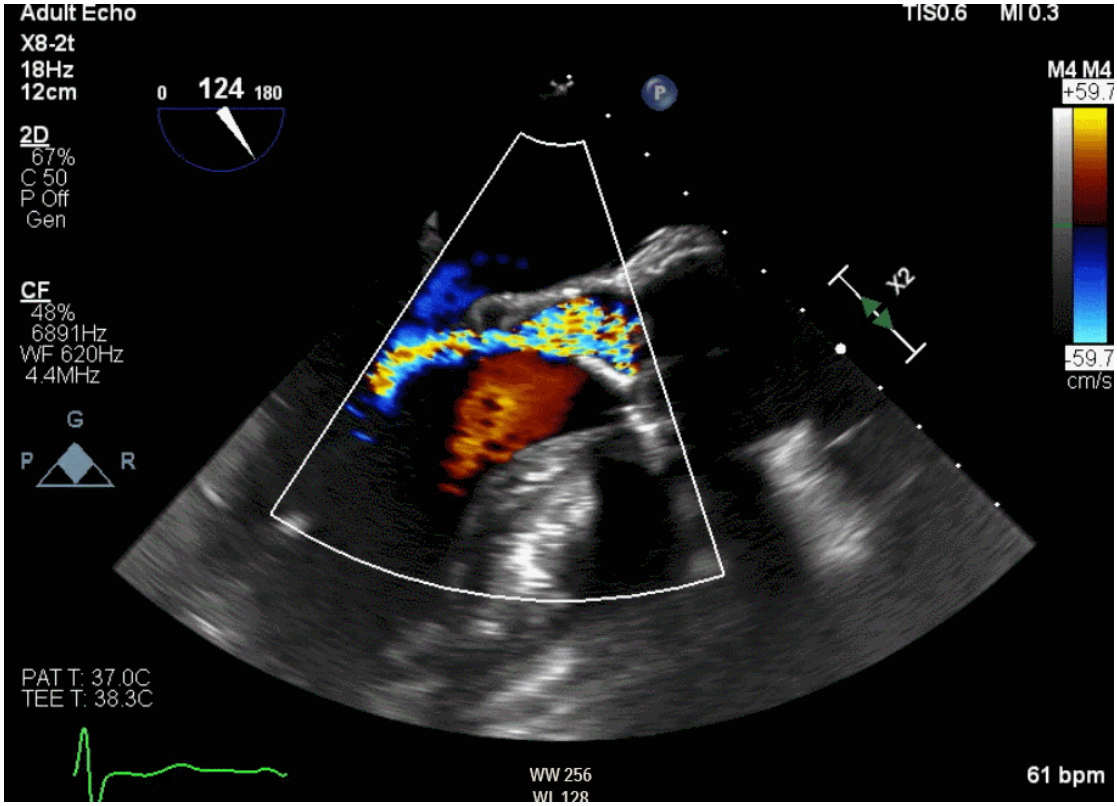
Her past medical history included diabetes mellitus type 2, asthma, hypertension, and obstructive sleep apnea. She had rheumatic fever as a child developed rheumatic heart disease, for which she underwent bioprosthetic aortic valve replacement seven years ago. She reported 4 weeks of worsening shortness of breath and was found to have newly depressed ejection fraction of 35% with severe AR. Transeophageal echocardiography (TEE) revealed a 2.2 cm mobile echodensity attached to the right coronary cusp of the bioprosthetic aortic valve which flailed into the left ventricular outflow tract during diastole causing a large area of leaflet malcoaptation. She reported recent dental cleaning with pre-procedure antibiotics though blood cultures were negative. She was then scheduled for reoperative aortic valve replacement.

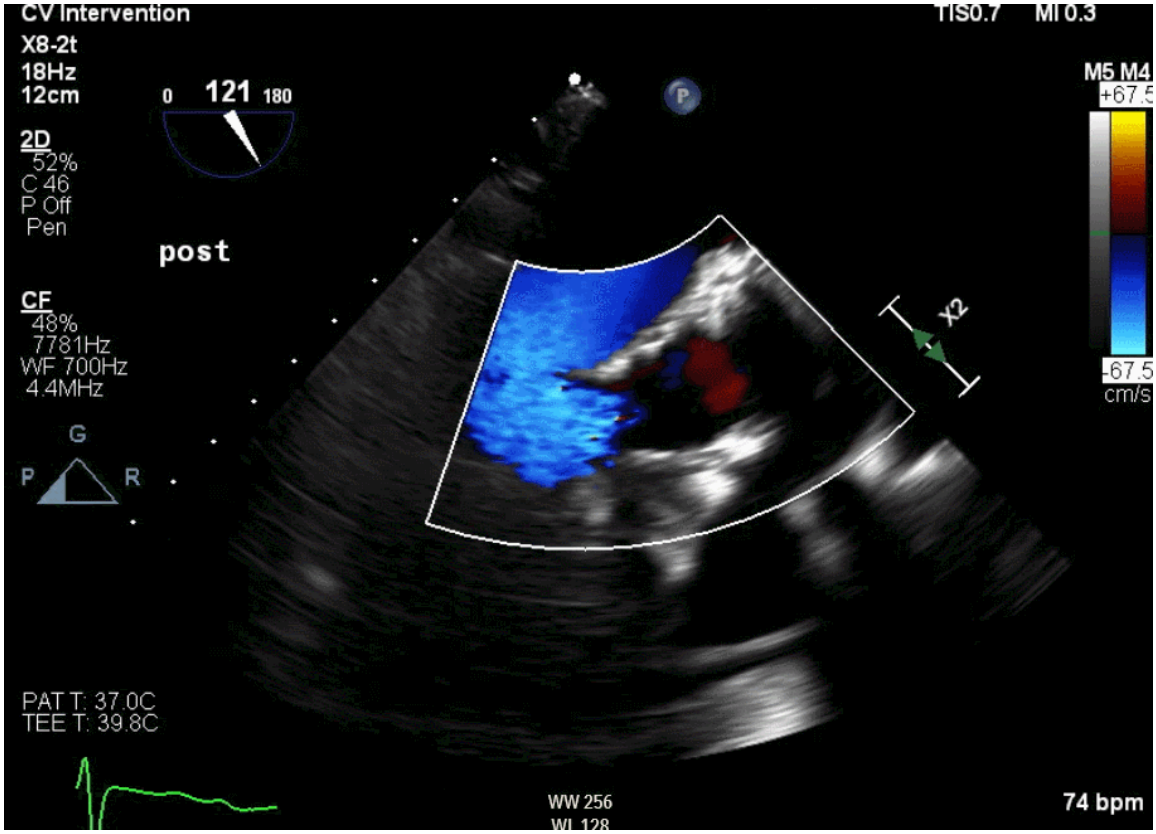
Intraoperatively, there was no suggestion of an infection. On inspection of the valve prosthesis, the mobile structure seen on TEE was noted to resemble a suture pledget that detached from the bioprosthesis and prolapsed into the left ventricle. A new aortic valve bioprosthesis was inserted and the patient had an uneventful surgical course.

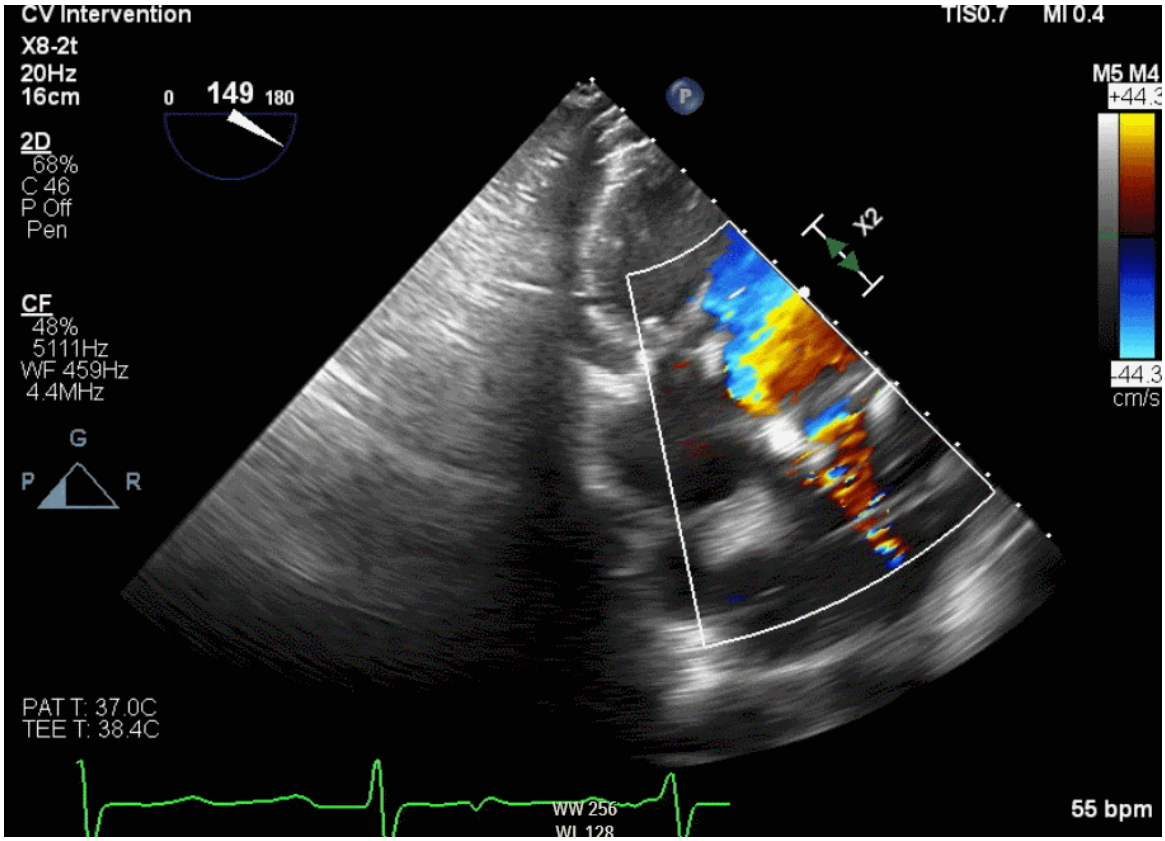
Conclusion: The above demonstrates a rare mechanism leading to acute aortic regurgitation many years following aortic valve replacement: structural deterioration resulting in mobility of pledgeted suture. Bioprosthetic valves are commonly sewed into the aortic root through the means of pledget-armed sutures. This rare and unique pathology may be mistaken for endocarditis based on TEE images and valvular deterioration, though clinical correlation is always the gold standard in diagnosis of endocarditis. While structural causes of bioprosthetic aortic valve regurgitation are generally more common for valves that are older than 10 years[4], structural failure occurred sooner in this patient.

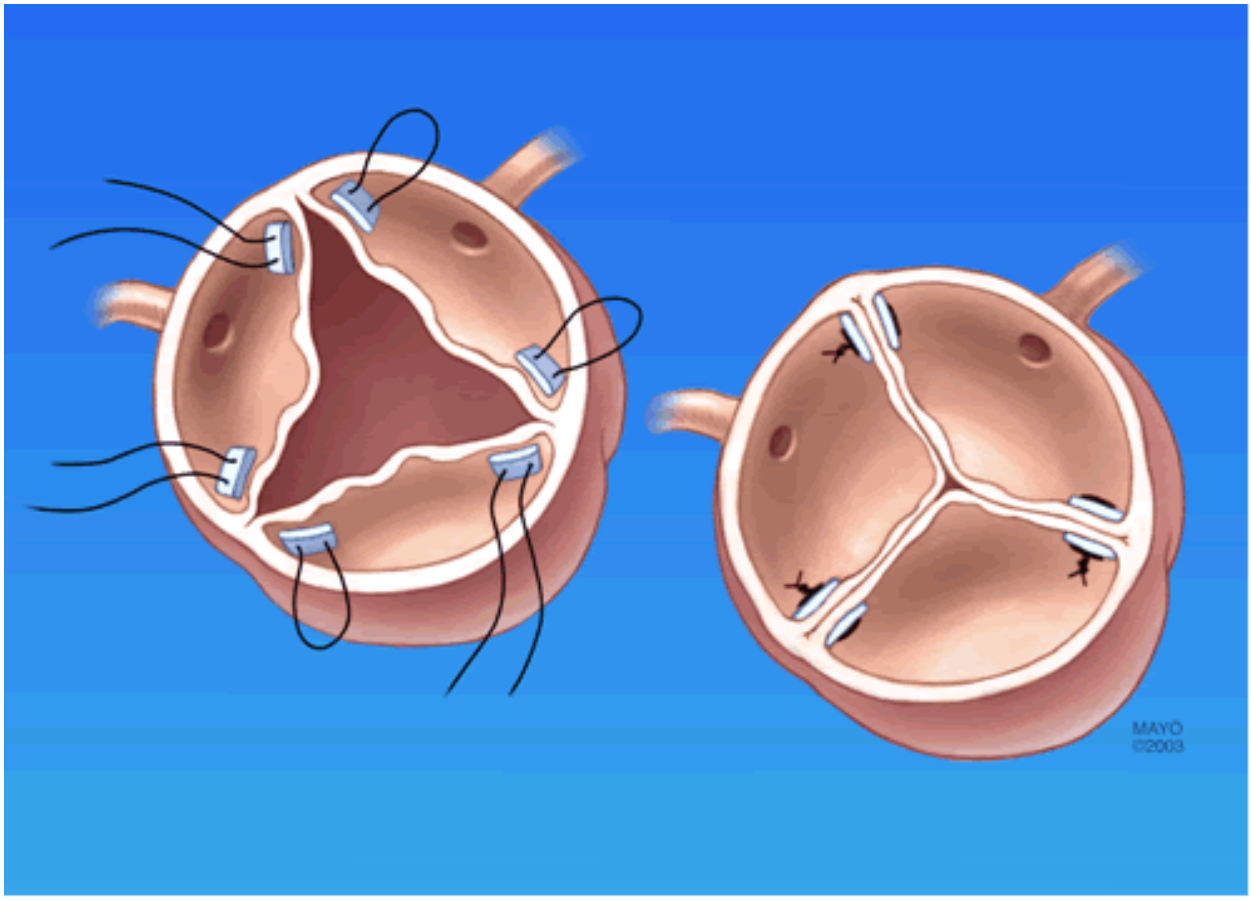
References: 1) Ionescu A, Fraser AG, Butchart EG. Prevalence and clinical significance of incidental paraprosthetic valvar regurgitation: a prospective study using transoesophageal echocardiography. Heart 2003; 89:1316. 2) Hammermeister K, Sethi GK, Henderson WG, et al. Outcomes 15 years after valve replacement with a mechanical versus a bioprosthetic valve: final report of the Veterans Affairs randomized trial. J Am Coll Cardiol 2000; 36:1152. 3) Wang A, Athan E, Pappas PA, et al. Contemporary clinical profile and outcome of prosthetic valve endocarditis. JAMA 2007; 297:1354. 4) De Cicco G, Lorusso R, Colli A, et al. Aortic valve periprosthetic leakage: anatomic observations and surgical results. Ann Thorac Surg 2005; 79:1480. Figure 1 - McKellar S, Zehr K. Aortic Valve Repair for Aortic Insufficiency. The Cardiothoracic Surgery Network. Dec 2010.


Adult Echo
X8-2t
32Hz
11cm
xPlane
55%
55%
50dB
P Off
Gen
TIS0.2
MI 0.9
M4
120
-12
G
P
R
PAT T: 37.0C
TEE T: 38.0C
WW 256
WL 128
52 bpm


Adult Echo
X8-2t
18Hz
12cm
2D
67%
C 50
P Off
Gen
CF
48%
6891Hz
WF 620Hz
4.4MHz
0
124
180
TIS0.6
MI 0.3
M4 M4
+59.7
-59.7
cm/s
X2
G
P
R
PAT T: 37.0C
TEE T: 38.3C
WW 256
WL 128
61 bpm


CV Intervention
TIS0.7 MI 0.3
X8-2t
18Hz
12cm
0 121 180
2D
52%
C 46
P Off
Pen
post
CF
48%
7781Hz
WF 700Hz
4.4MHz
G
P R
M5 M4
+67.5
-67.5
cm/s
X2
PAT T: 37.0C
TEE T: 39.8C
WW 256
WL 128
74 bpm


CV Intervention
TIS0.7 MI 0.4
X8-2t
20Hz
16cm
0 149 180
2D
68%
C 46
P Off
Pen
CF
48%
5111Hz
WF 459Hz
4.4MHz
G
P R
M5 M4
+44.3
-44.3
cm/s
X2
PAT T: 37.0C
TEE T: 38.4C
WW 256
WL 128
55 bpm


MAYO
©2003

Cardiovascular Anesthesiology - 9 A Case Presentation - Pulmonary Hypertension

Joshua Hubert[1], Miguel A Yaport[2]

[1]Tufts Medical Center, BOSTON, MA, [2]Brigham and Women's Hospital, Boston, MA

Introduction: Pulmonary hypertension (pHTN) is defined as mean Pulmonary Artery(PA) Pressure ,â• 25. Numerically, the chamber pressure alone does not convey functional information regarding ventricular compliance, coupling with pulmonary vasculature or etiology. Measures of static baseline functional status may not resemble function during induction and emergence, when transpulmonary pressures and left ventricular function may be affected, limiting their usefulness in risk stratification during the perioperative period. Quantitative measures of ventriculo-arterial coupling are relatively new and not commonplace in standard clinical practice. Meanwhile, pHTN is associated with severe morbidity and mortality in all surgical cases. Median survival rate of 1 year, 3 year, and 5 year mortality rate is 68%, 48%, and 34% respectively. Ultimately the anesthesiologist must obtain whatever workup informs the anesthetic management enough to proceed safely understanding there is a certain level of non-modifiable risk, all while using tools with limited sensitivity and specificity. Here we present a patient who underwent multiple anesthetics while simultaneously undergoing workup for pulmonary hypertension. Highlighting gaps in preoperative risk stratification.

Methods: A 77 year-old female with pulmonary hypertension after bilateral lung transplantation two decades prior, interstitial lung disease, and scleroderma, presented for urgent irrigation and staged liner exchange of an infected knee prosthesis. She was WHO functional class IV with shortness of breath at with activities of daily life. She was a poor historian and additional records were unavailable. Physical exam revealed a chachetic, woman appearing older than stated age, BMI of 17, diffuse crackles throughout with bibasilar prominence, and tight glossy skin over her digits. Frailty score was determined to be 'moderately frail'. Labs showed a ProBNP of 16,125pg/mL, Bicarb 36 and INR 2.7. Limited Transthoracic Echocardiography (TTE) revealed a left ventricular (LV) ejection fraction (EF) of 55%, with inability to obtain right ventricular (RV) windows. First stage surgery was safely performed under a dural puncture epidural (DPE) with boluses of 2% Lidocaine, and dexmedetomidine sedation. Inotropic and vasopressor medications were prepared but not required. Repeat TTE revealed stable LVEF, severe RV dilation, with low-normal function, RV systolic pressure 30-40mmHg, moderate tricuspid regurgitation, and moderate mitral regurgitation. Second stage surgery proceeded safely with a spinal anesthetic, propofol drip 15-20mcg/kg/min and phenylephrine drip of 10-20 mcg/min. One month later the patient was encountered in the MICU in hypoxic respiratory failure requiring emergent intubation. Induction was carried out with 80mg of propofol, 50mg of rocuronium, two 120mcg boluses of phenylephrine. She was started on a norepinephrine infusion and quickly weaned off. She was extubated within 24 hours after a toilet bronchoscopy.

Conclusion: Our case further highlights that in pHTN, clinical disease severity and numeric measures do not always closely correlate. Our patient was most likely a combination of WHO Group 2 and Group 3 pulmonary hypertension given her relatively preserved LV function but moderate valvular disease. She had severe respiratory symptomatology and signs of RV overload (severe RV dilation, jugular venous distension, lower extremity edema and elevated NT-ProBNP). We felt a DPE would be the safest way to proceed by maintaining spontaneous ventilation, analgesia, rapid onset but relatively hemodynamically stable. Ultimately, despite strong clinical evidence of advanced disease and mixed TTE findings she was able to tolerate general anesthesia safely while in respiratory extremis. Given her initial presentation, had her case been elective, it would not be unreasonable to have requested further workup. RV-PA coupling has emerged as the gold standard in patients with pulmonary hypertension, however most of the work has been limited to Group 2 pHTN, and evaluation of RV-PA coupling requires cath lab expertise that is not yet commonplace. Without established guidance, most anesthesiologists are left to fend for themselves, weighing patient safety, care delays, production pressure and healthcare costs. We feel that our case highlights the need for pragmatic evidence-based guidelines, given the increasing prevalence of cardiovascular and pulmonary disease.

References: Perioperative Management of Pulmonary Hypertension and Right Ventricular Failure During Noncardiac Surgery. Adv Anesth. 2018 Dec;36(1):201-230 Dural puncture epidural technique improves labor analgesia quality with fewer side effects compared with epidural and combined spinal epidural techniques: a randomized clinical trial. Anesth Analg. 2017;124:560-569 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, European Heart Journal, Volume 27, Issue 3, February 2006, Pages 330-337 Impact of pulmonary hypertension on the outcomes of noncardiac surgery: predictors of perioperative morbidity and mortality. J Am Coll Cardiol. 2005 May;45(10):1691-1699. doi: 10.1016/j.jacc.2005.02.055 Exercise physiology and pulmonary arterial hypertension. Prog Cardiovasc Dis. 2012 Sep-Oct;55(2):172-9

Cardiovascular Anesthesiology - 10

Acute mesenteric vein thrombosis causing mesenteric ischemia after open aortic valve replacement surgery in a patient with MGUS: a case report

Jeanette Chin[1], Joy Chen[1]

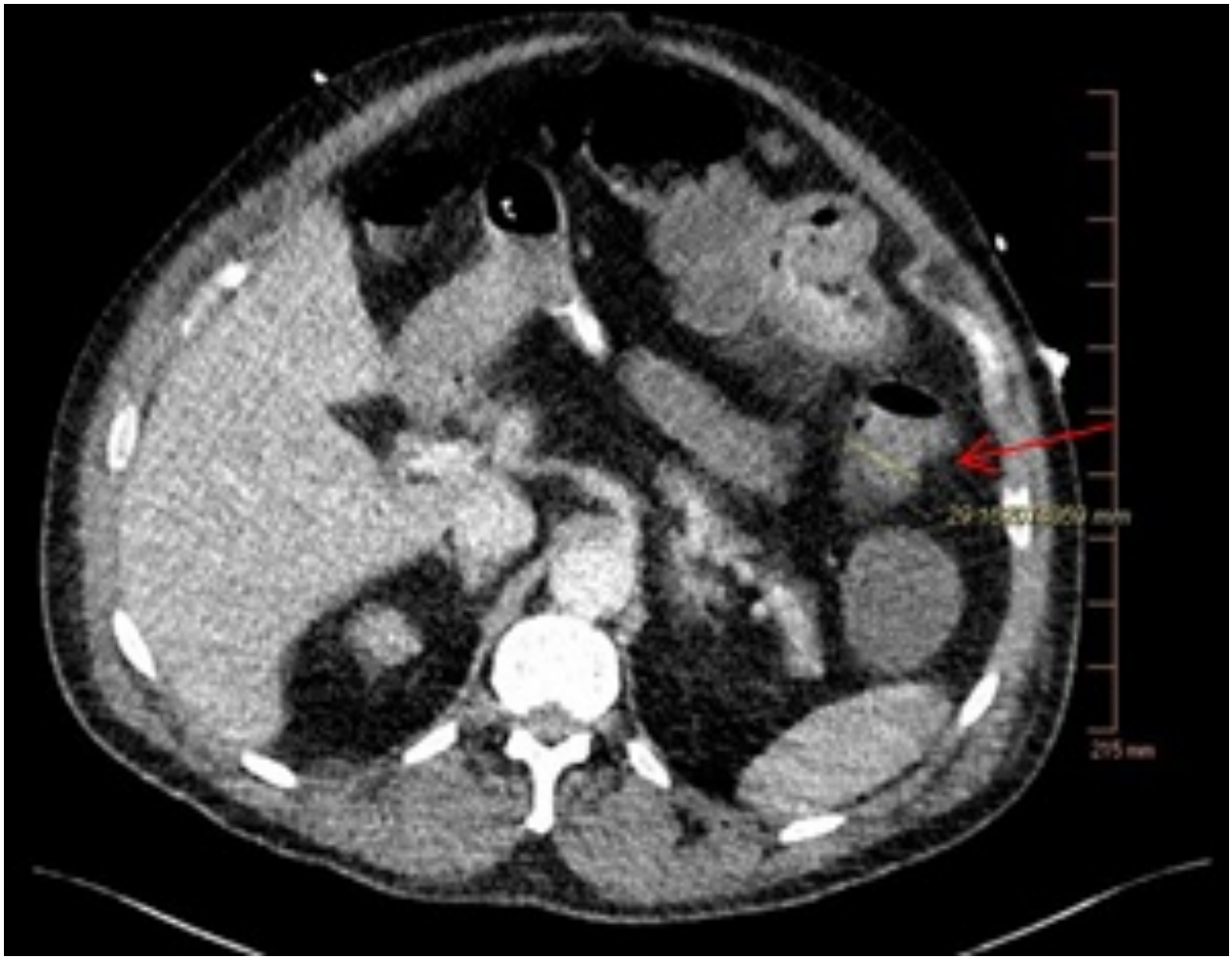
[1]The University of Texas Southwestern Medical Center, Dallas, United States of America

Introduction: Mesenteric vein thrombosis (MVT) accounts for 2-10% of all cases of acute mesenteric ischemia (AMI) [1]. While non-occlusive AMI due to hypoperfusion is a known gastrointestinal complication following cardiac surgery, there have been no known reports of MVT causing AMI after open heart surgery to date. Approximately half of patients who develop MVT already have preexisting hypercoagulable disorders or positive family history of venous thromboses [2]. Monoclonal gammopathy of undetermined significance (MGUS) is a premalignant clonal proliferative disorder of plasma cells. It is one of the precursor states to multiple myeloma. Patients with MGUS are known to be at increased risk for developing fractures, thromboembolic events, and additional cancers. There are no established perioperative management guidelines specific to MGUS patients undergoing surgery.

Methods: A 71 year-old male with a past medical history of nonobstructive coronary artery disease, hypertension, hyperlipidemia, type II diabetes mellitus, stage 3 chronic kidney disease, gout, and IgG kappa MGUS underwent an uncomplicated open aortic valve replacement (AVR) surgery with placement of a 23 mm Magna Ease valve for severe aortic stenosis. Intraoperative transesophageal echocardiography showed no evidence of thrombi with normal ejection fraction after weaning from cardiopulmonary bypass. He was started on warfarin postoperatively according to the American College of Cardiology recommendations after AVR surgery. The patient developed persistent vague abdominal pain and distention despite tolerating a diet and passing flatus. On postoperative day 5, computed tomography imaging showed small bowel wall thickening in his left abdomen with adjacent mesenteric fluid concerning for bowel ischemia (Figure). He underwent emergent exploratory laparotomy, and examination of the bowel in the operating room identified inflamed jejunum with scattered patches of ischemia throughout the mesentery. The patient ultimately recovered from his ischemic event without the need for bowel resection. The patient's clinical presentation and operating room findings were consistent with a diagnosis of MVT. The jejunum and distal small intestine is the site most often affected by MVT, as ischemia induced by organ hypoperfusion predominantly affects watershed areas [3]. In MVT-induced AMI, abdominal pain is dull and nonspecific, in contrast to the pain out of proportion to physical exam often seen in AMI caused by arterial thrombi or hypoperfusion. The patient was also in a hypercoagulable state at baseline due to his history of MGUS in addition to being at risk for venous stasis postoperatively. Studies have shown that the incidence of venous thromboembolism among MGUS patients is 23 times higher than that of the general population [4]. Furthermore, initiating warfarin without bridging decreases Protein C and S levels in warfarin-naive patients and may have further increased his risk of thrombi formation. Other etiologies including postoperative ileus-induced abdominal compartment syndrome and hypoperfusion-induced non-occlusive ischemia were thus considered possible, but less likely, causes of the patient's symptoms and findings.

Conclusion: The reported case of AMI after surgical AVR was attributed to an acute MVT. Due to the more indolent nature of MVT, the diagnosis may be easily missed until severe ischemia has already developed. One must therefore maintain a high suspicion in those with significant risk factors so as to avoid potential major complications that may lead to increased morbidity and mortality. Additionally, it may be prudent to implement perioperative anticoagulation guidelines in patients with MGUS or other hematological malignancies in order to decrease the risk of thromboembolic complications. For postoperative AVR patients specifically, it may be necessary to be more selective when initiating warfarin or at least consider bridging therapy in patients with increased risk profiles.

References: 1. Eur J Trauma Emerg Surg. 2016 Apr;42(2):253-70. 2. J Vasc Surg. 1989 Feb;9(2):328-33. 3. J Gastrointest Surg. 2009 Jul;13(7):1245-50. 4. Clin Adv Hematol Oncol. 2009;7(12):827-832.



Critical Care

Critical Care - 1 Is Multisystem Inflammatory Syndrome in Children also a Phenomenon in the Adult Population?

Katelyn Devine[1]

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

Introduction: Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-Cov-2) has been devastating globally with most cases affecting adults and only a subset of adult cases progressing unpredictably to severe hyperinflammatory disease during acute infection1. Notably, there is a large amount of heterogeneity in the immune response between individuals and children are relatively spared from symptoms of acute infection2. In the pediatric population, multisystem inflammatory syndrome in children (MIS-C, also called 'pediatric inflammatory multisystem syndrome' in Europe) is a newly described condition in literature that occurs weeks to months after asymptomatic or mild SARS-Cov-2 infection, sharing elements of Kawasaki disease, toxic shock syndrome, and macrophage activating syndrome3,4,5. Its presentation is characterized by prominent gastrointestinal symptoms and cardiac myocardial damage with the disease course highly responsive to immunomodulatory agents like corticosteroids and immunoglobulin therapy. Since June 2020, MIS-C has also been seen in adults (known as multisystem inflammatory syndrome in adults, MIS-A) with less than 30 cases reported so far. Like MIS-C, MIS-A is highly responsive to immunomodulatory agents. Hallmarks of MIS-A include cardiac failure (arrhythmias, troponinemia, left ventricular failure) as well as gastrointestinal, neurologic, and dermatologic symptoms without pulmonary involvement6,7. We will highlight a case of presumed MIS-A to bring awareness to clinicians of this rare sequela of COVID-19 as its incidence will likely increase with spread of SARS-Cov-2.

Methods: A 54-year-old female presented from an outside hospital for management of ischemic hepatitis and consideration for transplantation. The etiology of liver failure was presumed to be ischemic in the setting of urosepsis. At the time of admission to the outside hospital, the patient's chief complaint was dysuria and right upper quadrant pain with vital signs only significant for fever. Physical exam was unremarkable. Of note, the patient had recent COVID-19 infection with mild respiratory symptoms about 4 weeks prior to presentation. At the time of admission she had new onset atrial fibrillation with rapid ventricular rate and was also found to have acute kidney injury. The patient's condition continued to deteriorate so she was intubated, maintained on triple vasopressor therapy for progressive hypotension, and was transferred to my institution for liver transplantation workup. During transfer, stress dose steroids were begun. On arrival at Hospital of the University of Pennsylvania, the patient's pressor requirement was weaned quickly and stress dose steroids were stopped. Two days later, the patient spontaneously went into a pulseless tachyarrhythmia requiring initiation of venoarterial extracorporeal membrane oxygenation (VA ECMO). Echocardiography demonstrated ejection fraction of 15% with near akinesis globally despite being normal hours earlier. Ultimately, stress steroids were restarted and as a result, the patient was able to be decannulated from ECMO and ejection fraction returned to normal. The patient underwent a month long taper from high dose steroid therapy that prevented further symptoms.

Conclusion: After careful multidisciplinary review of the case presented above, we noted as a team that the patient's improvement in symptoms through her episodes of critical illness were temporally related to immunomodulation with steroid therapy and that her presentation was similar to MIS-C in the pediatric population. To date, this remains a rare phenomenon in the pediatric population and exceedingly rare in adults6. Much is unknown about long term implications of COVID-19 and it is possible that MIS-A will increase in frequency as more people recover from acute infection so clinicians should be aware of this possible post infective syndrome, particularly as steroids are being used increasingly in primary treatment of the disease. It is still unclear what immune complex is responsible, however, a pattern of symptoms has emerged in the pediatric population8 that could help identify the characteristics of the syndrome in adults (uncontrollable arrhythmias, cardiac failure, and gastrointestinal symptoms are most common)6,7. Further, more research is necessary to determine necessary health maintenance and surveillance in those who have recovered from COVID-19 and MIS-C/A.

Critical Care - 2 Retained Endovascular Balloon After FEVAR

Jennifer Meeker[1], Peter Roffey[2]

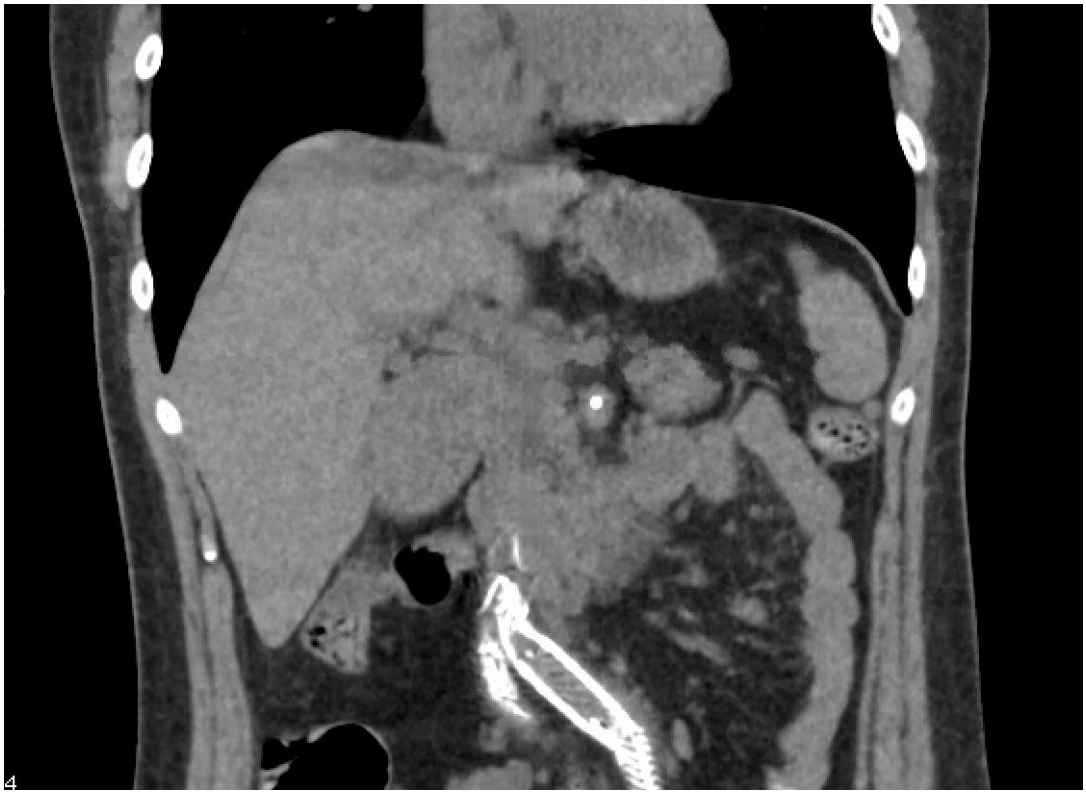
[1]LAC+USC Medical Center, Los Angeles, CA, [2]Keck Medical Center of USC, Los Angeles, CA

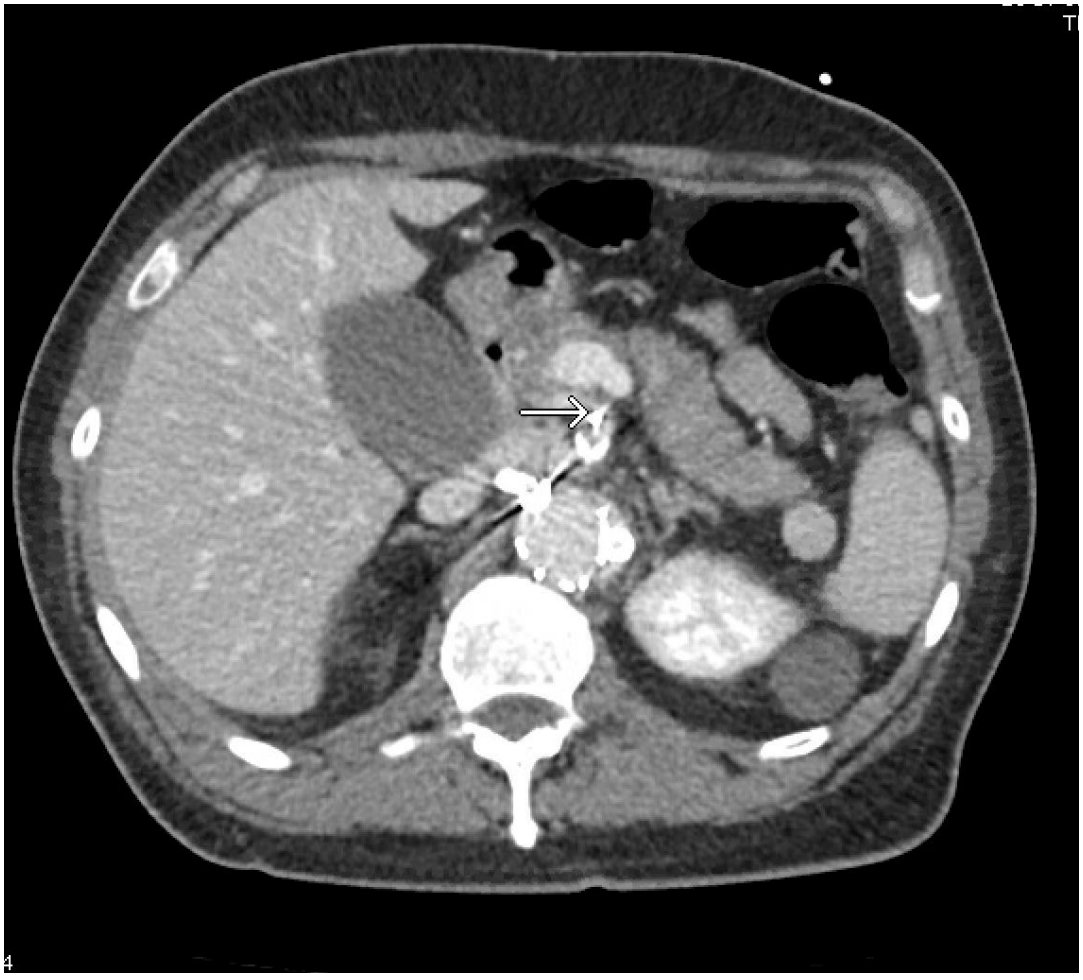
Introduction: Background: FEVAR techniques are commonly used to treat abdominal aortic aneurysm and dissections. Utilization of endovascular aortic repair has rapidly expanded, though its advantage over open repair is still equivocal (1-4) and is associated with a unique set of perioperative complications. This case report presents an unusual etiology of postoperative nausea and vomiting after FEVAR.

Results: Case Report: BC is a 59 yo male with PMH of DeBakey I aortic dissection status post multiple repairs including total arch replacement with TEVAR extension and endoleak repairs. BC had continued enlargement of a false lumen in the descending thoracoabdominal aneurysm with retrograde endoleak treated with 4 vessel FEVAR which was uncomplicated with completion angiography showing widely open branch perfusion without significant endoleak. After FEVAR, BC was transferred to SICU for further care. Starting POD1 patient experienced intermittent nausea and vomiting (N/V). Initially BC thought his N/V was related to movement. Abdominal x-ray and abdominal exam were unremarkable; lactate was normal. N/V symptoms continued through POD3 despite maximal anti-emetic therapy and unremarkable repeat abdominal x-ray, abdominal exams and lactates, after which time his symptoms improved. An attempt to advance BC to a clear liquid diet resulted in recurrence of N/V. CTA, obtained on POD3 for further evaluation, revealed a new filling defect in in the superior mesenteric artery and possible retained balloon in the SMA just distal to the SMA stent without evidence of bowel ischemia. BC was taken for exploratory laparotomy and SMA exploration on POD3 during which a retained balloon catheter tip (part of the disposable stent delivery system) was extracted. POD4/1 patient without N/V; diet advanced POD6/3 without issue. Patient discharged on POD8/5.

Conclusion: Discussion: Endovascular aortic repair comes with a unique set of complications, most commonly endoleaks, stent graft thrombosis, migration, kinking and infection (4). This case report presents a rare complication of retained endovascualr equipment causing uncontrolled N/V in the absence of concerning abdominal exam. Upon literature review, no other published report described inadvertent intravascular retained balloon after FEVAR. Though this is a very rare cause of N/V after FEVER and a rare complication of FEVAR in general, retained endovascular surgical equipment should be included in the differential of a patient experiencing ischemic or ischemic-like signs or symptoms in the distribution of aortic branches stented during FEVAR and associated organ systems.

References: 1. Bulder RMA, Bastiaannet E, Hamming JF, Lindeman JHN. Meta-analysis of long-term survival after elective endovascular or open repair of abdominal aortic aneurysm. Br J Surg. 2019 Apr;106(5):523-533. 2. Li B, Khan S, Salata K, Hussain MA, de Mestral C, Greco E, Aljabri BA, Forbes TL, Verma S, Al-Omran M. A systematic review and meta-analysis of the long-term outcomes of endovascular versus open repair of abdominal aortic aneurysm. J Vasc Surg. 2019 Sep;70(3):954-969.e30. 3. Powell JT, Sweeting MJ, Ulug P, Blankensteijn JD, Lederle FA, Becquemin JP, Greenhalgh RM; EVAR-1, DREAM, OVER and ACE Trialists. Meta-analysis of individual-patient data from EVAR-1, DREAM, OVER and ACE trials comparing outcomes of endovascular or open repair for abdominal aortic aneurysm over 5 years. Br J Surg. 2017 Feb;104(3):166-178. 4. Valentine EA, Ochroch EA. 2017. 'Anesthesia for Vascular and Endovascular Surgery'. p1137-1138 in Clinical Anesthesia. Philadelphia: Wolters Kluwer.





Critical Care - 3 Right Coronary Artery Vasospasm Following Anastomotic Leak Status Post Esophagectomy

Yogita Mosalpuria[1], Anoop Chhina[1]

[1]Henry Ford hospital, Detroit, MI

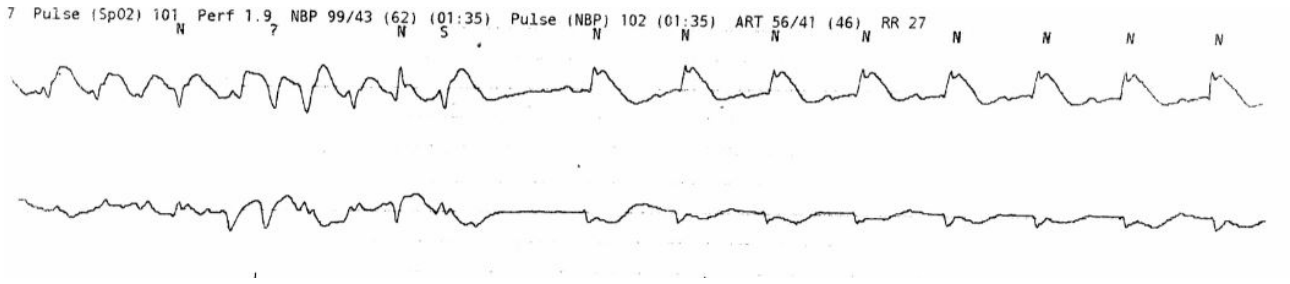
Introduction: MINOCA (Myocardial Infarction with Non-Obstructive Coronary Arteries) is a syndrome defined as MI with normal or near normal coronary arteries on angiography. Although the prevalence is estimated to be1-14%, it's incidence in patients undergoing non-cardiac surgery, is not established. Etiologies such as coronary artery vasospasm, acute thrombosis at site of a non-obstructive eccentric plaque thrombus, takotsubo cardiomyopathy, coronary microvascular dysfunction, viral myocarditis, thrombophilia, and coronary artery embolism, have been identified as culprits for MINOCA. We present here a case of a 65-year-old male, with past medical history significant for esophageal adenocarcinoma, COPD, 45-year pack tobacco history, and obesity, who developed MINOCA secondary to coronary artery vasospasm postoperatively.

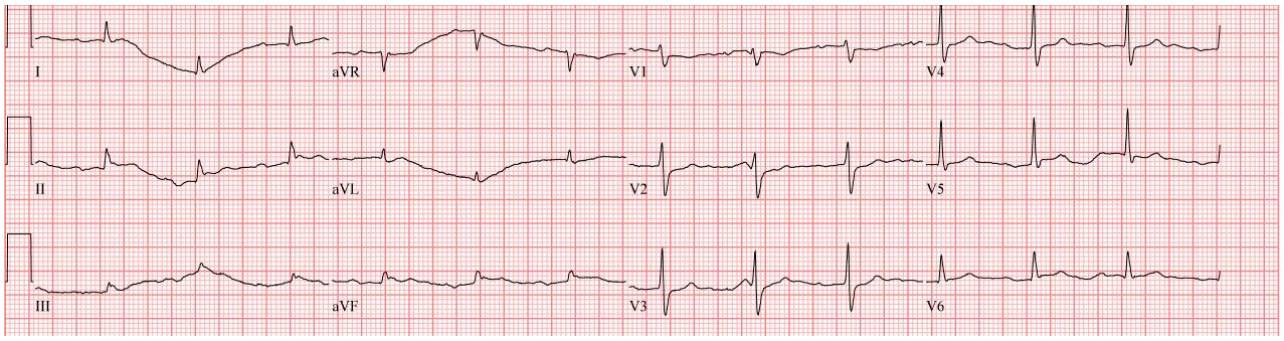
Methods: Patient underwent esophagectomy at an outside hospital. After multiple post-op interventions and complications (atrial fibrillation, delirium), patient was transferred to our facility for recurrent anastomotic leaks. At our institution patient underwent a redo thoracotomy with pulmonary decortication, complete gastrectomy and creation of esophagostomy. Postoperative day 0, patient had transient ST elevations with ventricular tachycardia. Differential diagnosis included acute coronary syndrome, coronary vasospasm, coronary plaque rupture and recanalization secondary to pericarditis. Echocardiogram performed after the event showed normal LV systolic function globally with ejection fraction (EF) 70%. Postoperative day 5, patient developed transient bradycardia without hypotension or changes in mental status. EKG was significant for ST elevations in the inferior leads with reciprocal ST depressions, transient AV Type II block with 1:2 and 1:3 conduction variance. Within few minutes, rhythm converted to atrial fibrillation with RVR without ST elevations. Patient underwent a left heart catherization, which was negative for obstructive coronary artery disease, but significant for moderate hypokinesis of the apical and periapical wall with reduced EF 35-40%.

Results: In view of continued suspicion for RCA vasospasm as etiology for these episodes, patient was started on diltiazem, amiodarone, and low dose ASA 81mg. Unfortunately, patient was unable to tolerate diltiazem secondary to hypotension and was switched to nitroglycerin infusion. He was eventually transitioned to isordil without further issues. He continued to remain in atrial fibrillation, rate controlled, without any further episodes of coronary vasospasm.

Conclusion: Pericarditis and cardiac tamponade are rare complications of esophagectomy. Pericarditis, in this setting can present as MINOCA, which can be an easily missed diagnosis. As troponins are not helpful in a post surgical setting, high vigilance for cardiac complications due to less common causes is warranted.

References: Mizuguchi Y et al. A case of cardiac tamponade following esophageal resection. J Anesth - January 1, 2005; 19 (3); 249-51 • Yinghao Lim et al. MULTIVESSEL CORONARY ARTERY SPASM IN PERICARDITIS Singapore Med J 2018; 59(11): 611-613 • Tamis-Holland et al . Contemporary Diagnosis and Management of Patients With Myocardial Infarction in the Absence of Obstructive Coronary Artery Disease. Circulation. 2019;139:e891–e908


7 Pulse (SpO2) 101 Perf 1.9 NBP 99/43 (62) (01:35) Pulse (NBP) 102 (01:35) ART 56/41 (46) RR 27


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Critical Care - 4 Diagnosing Delayed-Onset Pericardial Tamponade after HeartMate III Left Ventricular Assist Device (LVAD) Placement: A Clinical Challenge

Shannon Sparrow[1], Jayanta Mukherji[1], Annandita Kumar[2]

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

Introduction: Delayed-onset pericardial tamponade in patients on mechanical circulatory support is an infrequent complication that must be considered when hemodynamics begin to decline in the late postoperative period. Active bleeding may not be found with late tamponade, as it can be caused by clots. Patients with an LVAD who have tamponade physiology will lack many of the traditional signs of tamponade, such as pulsus paradoxus, JVD, and distant heart sounds. They may instead exhibit a decreased pulsatility index (PI), decreased flows, hypotension, or low urine output (1,2). Our case illustrates the challenges of diagnosing delayed-onset cardiac tamponade several weeks after HeartMate3 (HMIII) placement in a patient presenting with variable flows, low MAPs, intermittent low PI, and elevated CVP.

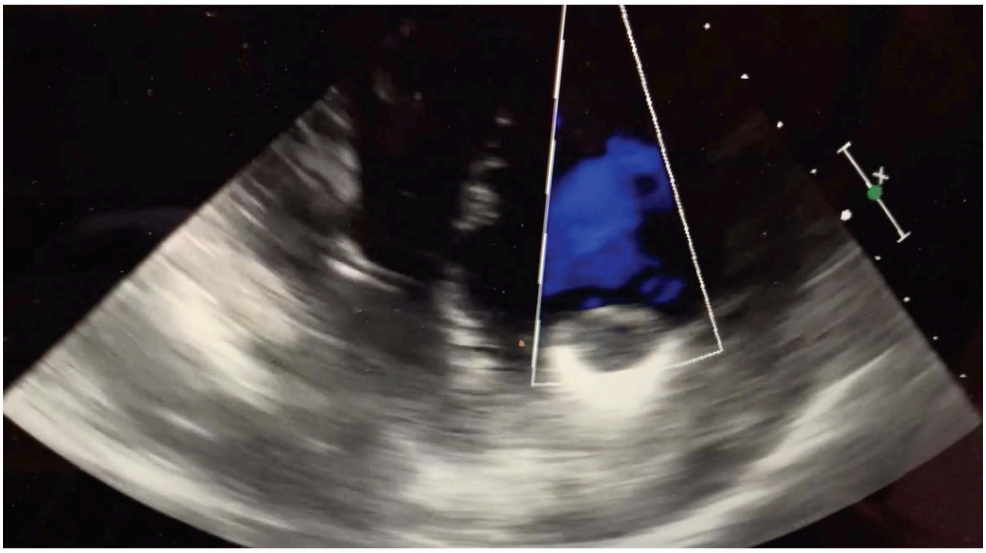
Methods: A 65-year-old male with a past medical history of acute myeloid leukemia s/p bone marrow transplant, CKD stage III, ventricular arrhythmias s/p radiofrequency ablation and nonischemic cardiomyopathy (LVEF 15%) underwent HMIII implantation. His hemodynamic profile during the early postoperative period revealed low PI, low CVP, (<10 mmHg), and low cardiac index, which was responsive to fluid challenge. Transthoracic echocardiography (TTE) imaging was suboptimal and did not show signs of pericardial collection or worsening RV dysfunction. Anticoagulation (AC) with heparin infusion was initiated on the 3rd POD. He continued to require ventilatory support and presented with non-sustained stable ventricular tachycardia. A bedside TEE showed RV systolic dysfunction and a thin rim (<10mm) of fluid around the heart. His hemodynamic profile by the 10th POD showed signs of persistent low flow state and oliguric renal failure. Fluid boluses and diuretics were transiently helpful and he required renal replacement therapy. He had persistent leukocytosis and CT imaging on POD 10 showed a moderate pericardial effusion with bilateral pleural effusions. His hemodynamic status deteriorated by POD#14, requiring epinephrine and vasopressin for MAPs >60 mmHg. A diagnosis of pericardial tamponade was suggested with an insidious rise of CVP to 17-22 mm Hg. Heparin infusion was held and additional fluid boluses and PRBC transfusions were provided. A bedside TEE showed a localized hyperechoic collection compressing the RV with diastolic collapse and decreased LV filling. The LVAD speed was decreased to prevent suction events and arrhythmias. He underwent tracheostomy, pleural fluid drainage, and pericardial window, resulting in improved hemodynamics, (CVP 60 mmHg) and reduced pressor requirements.

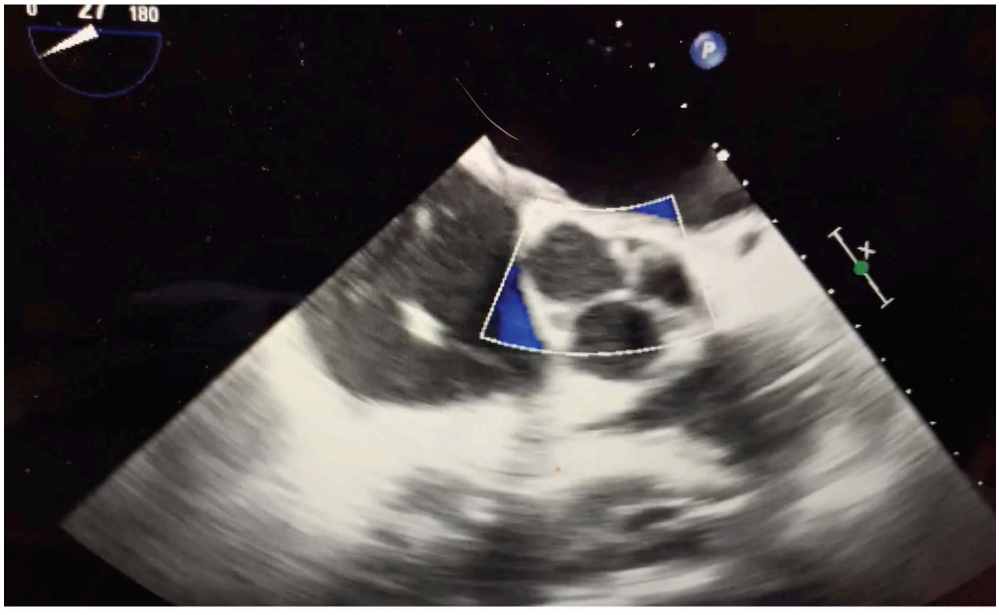
Conclusion: Patients receiving an LVAD are at higher risk of bleeding and cardiac tamponade, as they require early postoperative AC and antiplatelet therapy. An isolated RA/RV tamponade from a localized collection can occur following device placement, especially in patients with multiple comorbidities. A localized clot may be large enough to limit ventricular filling in the diastolic phase, causing tamponade. In critically ill postoperative patients, it may be difficult to recognize a hematoma adjoining RA/RV in the retrosternal space using TTE, as it has limited imaging windows especially in the presence of mediastinal thoracostomy drains (3). A high index of suspicion for tamponade should remain and a diagnosis of delayed pericardial tamponade considered when a patient's hemodynamic status changes, even in the late postoperative period. In mechanically ventilated patients, TEE may be favorable for posterior fluid collections. Paradoxical flow across the tricuspid and mitral valves, as classically seen with cardiac tamponade, may not be reliably present in patients with an LVAD (4). Although cardiac tamponade is a clinical diagnosis, CT imaging may be helpful in assessment, and CT findings associated with cardiac tamponade include moderate to large pericardial collection, distention of SVC/IVC, and deformity of the cardiac chambers. If the clinical manifestations are consistent with cardiac tamponade, early consideration for mediastinal drainage should be considered. Familiarity with the clinical and

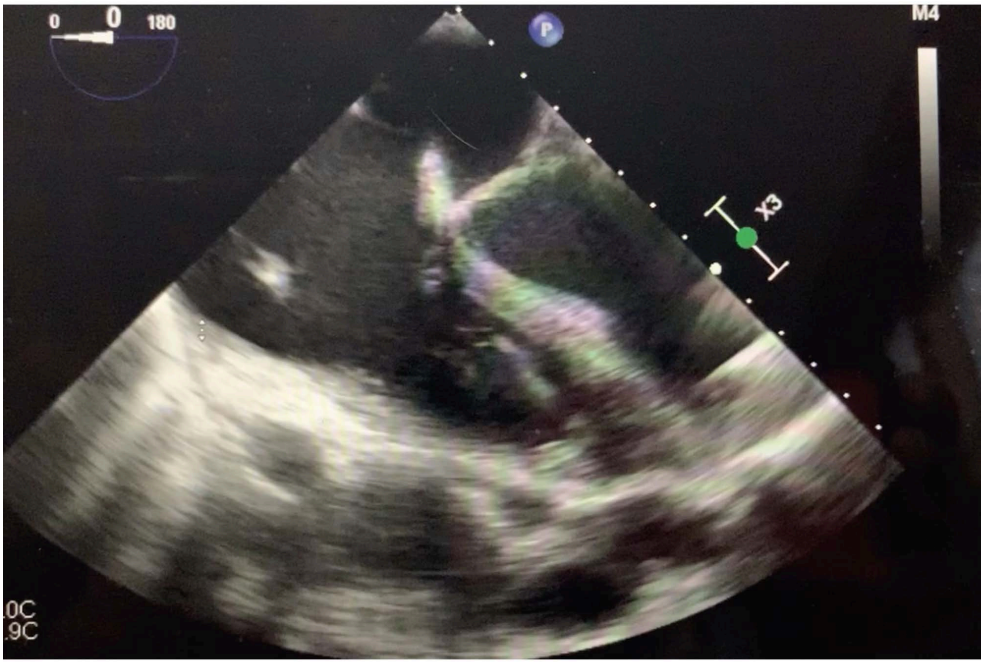
pathophysiologic features of cardiac tamponade are essential for early and accurate diagnosis.

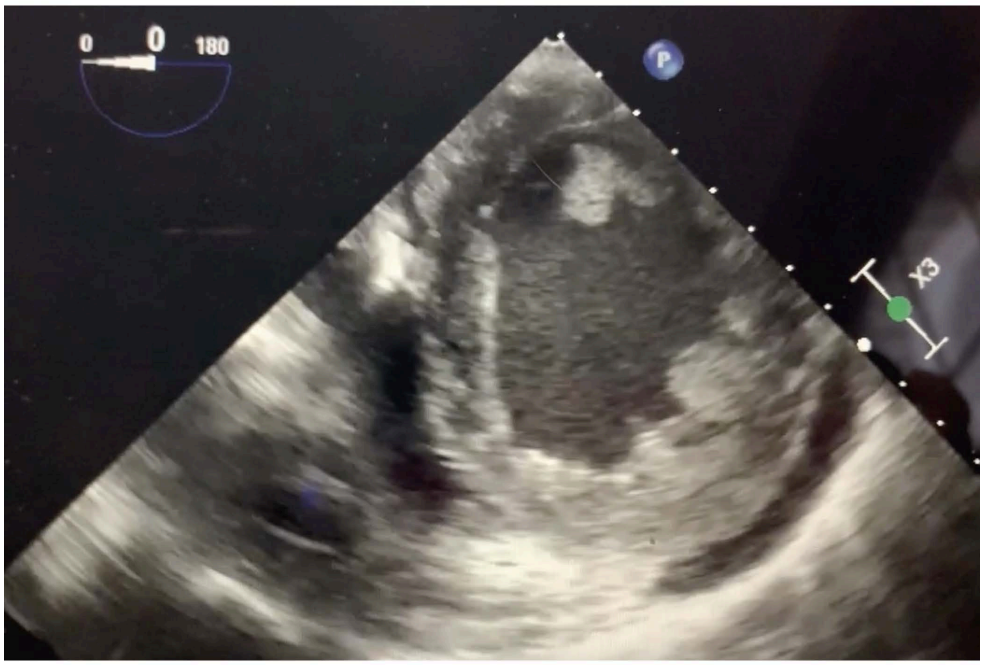
References: 1)Sen, Ayan, et al. “Mechanical Circulatory Assist Devices: A Primer for Critical Care and Emergency Physicians.” Critical Care, vol. 20, 2016, doi:10.1186/s13054-016-1328-z. 2) Araj FG, Sasaki JD Pulseless Paradoxus: A unique sign of Cardiac tamponade in the era of Continuous flow left ventricular devices. Mayo Clin Proc vol 95(1), 2020; 199-202 3) Lesicka, Agnieszka, et al. “Echocardiographic Artifact Induced by HeartWare Left Ventricular Assist Device.” Anesthesia & Analgesia, vol. 120, no. 6, June 2015, pp. 1208–1211, doi:10.1213/ANE.0000000000000664.

4)Flores AS, Essandoh M, Yerington GC, Bhatt AM, Iyer MH, Perez W, Davila VR, Tripathi RS, Turner K, Dimitrova G, Andritsos MJ. Echocardiographic assessment for ventricular assist device placement. J Thorac Dis. 2015 Dec;7(12):2139-50. doi: 10.3978/j.issn.2072-1439.2015.10.69. PMID: 26793334.






0
0
180
P
M4
X3
0C
.9C


0
0
180
P
X3

Critical Care - 5 COVID-19 pneumonia and thoracic aorta aneurysm: can probiotics lead to a mycotic aneurysm? A case report

Alberto Furzan[1], Natalia Martinez-Acero[2]

[1]Stanford University, Palo Alto, CA, [2]Stanford Healthcare, Stanford, CA

Introduction: Lactobacillus are common components of probiotics, and although low virulence, they have been reported in serious infections such as bacteremia and endocarditis[1]. Recently there has been an increased use of probiotics in patients with severe Coronavirus disease 2019 (COVID-19), that often have an excessive immune response, increasing gut permeability and bacterial translocation. Probiotics are thought to strengthen the gut epithelial integrity against pathogens and to increase immunoscence[2]. Mycotic aneurysm of the thoracic aorta is a rare condition caused by a preexisting aneurysm that becomes secondarily infected or by aneurysmal degeneration of the arterial wall due to bacteremia or septic embolization. The most common pathogens are Staphylococcus spp and Salmonella spp[3], but cases of mycotic aneurysm due to Lactobacillus have not yet been reported. Treatment consists of antibiotics and aggressive surgical debridement with vascular reconstruction, but even in specialized centers, it carries high mortality and morbidity[4]. Here we present a successful repair of a thoracic aorta mycotic aneurysm in a patient with ARDS due to COVID-19 complicated with aorto-bronchial fistula.

Methods: A 62-year-old man with recent COVID-19 five months prior to his referral to our center. At that time his symptoms included hemoptysis and respiratory distress requiring intubation. Also he received broad-spectrum antibiotics for ventilator associated pneumonia. On evaluation with chest CTA there was a mycotic aneurysm at the aortic arch, adjacent to a left upper lobe consolidation (LUL). He declined surgery and was discharged with antibiotics and probiotics (Lactobacillus Rhamnosus). Two weeks prior to his referral, he presented with acute chest pain, fever, hemoptysis, and concerns for aspiration pneumonia. Repeat CTA chest demonstrated interval increase of the thoracic aortic aneurysm from 7.0x4.4x5.5 cm to 8.0x7.0x7.0 cm in less than 3 months. (image 1). He was transferred to our center for surgical repair. Preoperatively, lumbar drain was placed for spinal cord protection and a bronchoscopy showed bloody secretions from left lung and a collapsed subsegment of the LUL. He underwent repair of a large saccular aneurysm beginning distal to the left subclavian artery with homograft. Surgery was uneventful, and there was no obvious signs of infection, but the LUL was adherent to the entire aneurysm, highly concerning for bronchoaortic fistula. Postoperatively, his condition continued to improve. Surgical material from the aortic tissue showed presence of L. Rhamnosus, resistant to vancomycin and penicillin, and he was discharged to a SNF with 6 weeks of Ceftriaxone and Clindamycin.

Conclusion: COVID-19 presentation varies from asymptomatic carriers to severe respiratory distress. Hemoptysis is an unusual presentation and the few cases described are associated with pulmonary embolus and diffuse alveolar hemorrhage[5]; therefore, presence of hemoptysis should raise concerns for other serious pathologies. Such as, our patient was found to have aorto-bronchial fistula, which is a rare yet fatal complication of mycotic thoracic aortic aneurysm that could have resulted from lung damage caused by COVID-19 pneumonia. Although there are case reports of serious endovascular infection (endocarditis)[6], to our knowledge there have not been any reported cases of L. Rhamnosus associated with mycotic aneurysm of the aorta. We favor a primary lung process that was likely contiguous to the aortic area and was subsequently complicated with a mycotic aneurysm. We presume that his initial antibiotic coverage (included vancomycin, ceftriaxone and metronidazole) was chosen to target a polymicrobial lung infection, and also partially treated the multi-organ mycotic aneurysm, and L. Rhamnosus persisted since it had drug resistance. Hence, we gave clinical significance to our patient culture because he had multiple risk factors for L. Rhamnosus bacteremia such as poor dentition, aspiration pneumonia, invasive procedures (bronchoscopy), along with his aorto-bronchial fistula and relatively stable course on prior antimicrobials[7]. We conclude that although probiotics are being used for immunomodulation in patients with severe COVID-19 infection, there is little evidence about its liberal use, and it may not be indicated for

every patient, as it can potentially lead to Lactobacillus bacteremia and other serious infections.

References: 1.Doron, S. & Snydman, D. R. Risk and Safety of Probiotics. Clin. Infect. Dis. 60, S129-S134 (2015). 2.Zeng, J. et al. Effect of probiotics on the incidence of ventilator-associated pneumonia in critically ill patients: a randomized controlled multicenter trial. Intensive Care Med. 42, 1018-1028 (2016). 3.Huang, Y.-K. et al. Clinical, microbiologic, and outcome analysis of mycotic aortic aneurysm: the role of endovascular repair. Surg. Infect. 15, 290-298 (2014). 4.Aoki, C. et al. Surgical Management of Mycotic Aortic Aneurysms. Ann. Vasc. Dis. 10, 29-35 (2017). 5.Thevarajan, I. et al. Clinical presentation and management of COVID-19. Med. J. Aust. 213, 134-139 (2020). 6.Campagne, J. et al. Lactobacillus endocarditis: a case report in France and literature review. IDCases 21, e00811 (2020). 7.Salminen, M. K. et al. Lactobacillus bacteremia, clinical significance, and patient outcome, with special

Image 1A. Computed tomography angiogram of chest demonstrating thoracic aortic aneurysm measuring 8.0x7.0x7.0 cm concerning for mycotic aneurysm and adjacent left upper lung consolidation.

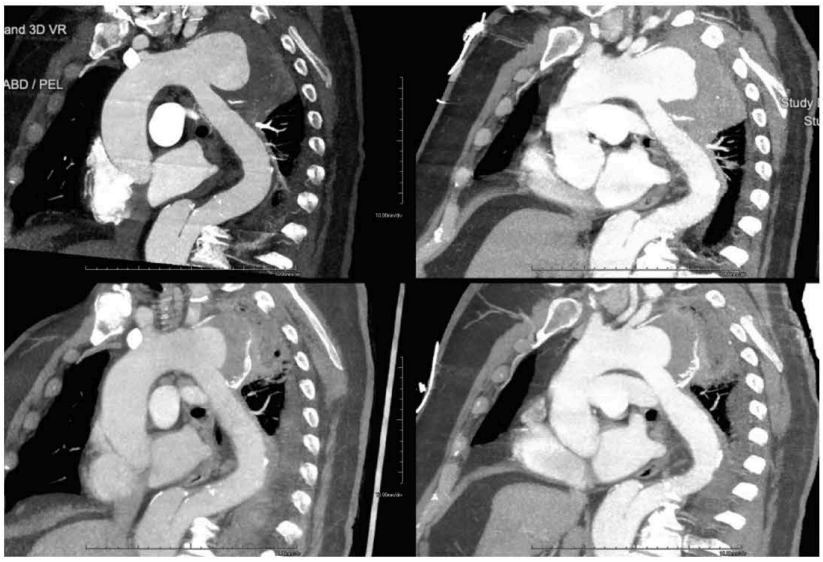
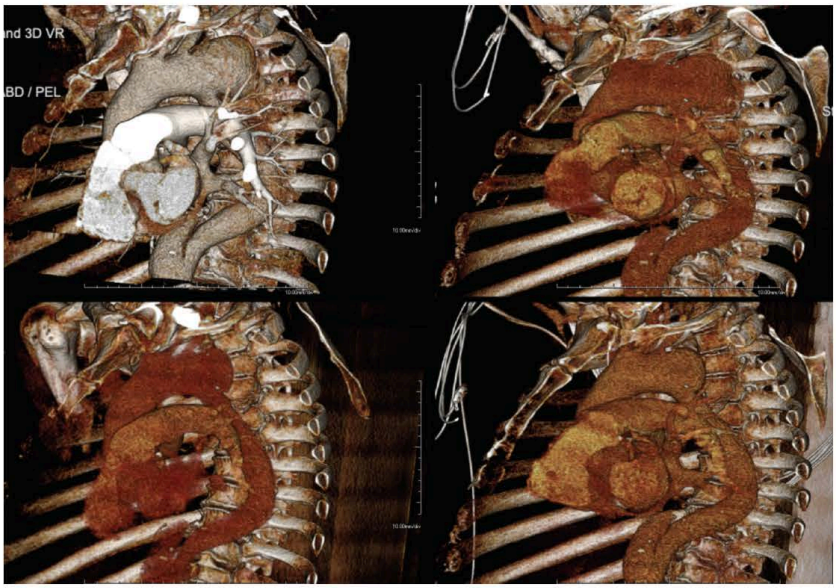


Image 1B. Computed tomography angiogram with 3D reconstruction of aorta and mycotic aneurysm



Critical Care - 6 Inhaled tranexamic acid for the management of hemoptysis in a patient on extracorporeal membrane oxygenator: a case report.

Shamik Patel[1], Ioannis (Yanni) Angelidis[2], Ashley Budd[3]

[1]McGaw Medical Center at Northwestern University, Chicago, IL, [2]Northwestern University, Feinberg school of medicine, Chicago, United States of America, [3]Northwestern Memorial Hospital, Chicago, United States of America

Introduction: Mechanical circulatory support (MCS) devices include a wide range of new technology including extracorporeal membrane oxygenation (ECMO) and ventricular assist devices that are being increasingly used to manage critically ill patients. Bleeding continues to be a significant complication of these devices due to disruption of the coagulation cascade and need for anti-coagulation to prevent thromboses.[1] Pulmonary hemorrhage can be a life-threatening complication that can be particularly difficult to treat in patients with MCS devices. Inhaled tranexamic acid (TXA) has been used in the management of pulmonary hemorrhage with a recent clinical trial showing 96% resolution within five days.[2] Though there is not enough literature on the use of inhaled TXA in patients on MCS devices, it may be an effective treatment for pulmonary hemorrhage in such patients due to its minimal systemic side-effects.[2,3,4]

Methods: We present a case of a 28-year-old male with a history of non-compaction cardiomyopathy admitted with cardiogenic shock. He was additionally found to have pulmonary emboli requiring anti-coagulation and eventually required placement of a left ventricular assist device (LVAD) due to persistent shock. Following LVAD placement, he developed severe right ventricular failure initially requiring veno-arterial (VA) ECMO, and then transitioned to a right ventricular assist device (RVAD). His hospital course was further complicated by massive hemoptysis resulting in an inability to both oxygenate and ventilate. He also developed severe pulmonary hypertension and cardiopulmonary collapse requiring blood products, vasopressors, resuscitation, and cessation of his anti-coagulation. An oxygenator was then placed in the RVAD and the patient started to stabilize. Bronchoscopy showed occlusion of the entire tracheobrochial tree with both active bleeding and clots. The patient underwent bedside clot extraction, cryotherapy, and saline lavage resulting with modest improvement in tidal volumes. He was subsequently started on inhaled tranexamic acid, every eight hours for a total of fifteen doses. After the initiation of inhaled tranexamic acid, the patient had no further episodes of major hemoptysis. Repeat bronchoscopies showed improvement in hemoptysis, and the patient was restarted on systemic bivalirudin. His oxygenation and ventilatory parameters continued to improve, and he was soon extubated. No evidence of any adverse events were noted after initiation of inhaled tranexamic acid.

Conclusion: Data for the use of antifibrinolytic agents in actively bleeding patients is lacking. Leo et. al demonstrated that intravenous aminocaproic acid may be a viable option in bleeding patients on ECMO.[5] Inhaled tranexamic acid may also be an efficacious and safe option in the treatment of pulmonary hemorrhage in patients with MCS devices without systemic reversal of anticoagulation and with a low side-effect profile.

References: 1. Mechanical circulatory support: balancing bleeding and clotting in high-risk patients. Hematology Am Soc Hematol Educ Program. 2015;2015:61-8 2. Tranexamic Acid for Hemoptysis Treatment: A Randomized Controlled Trial. Chest. 2018;154(6):1379-1384 3. Inhaled Tranexamic Acid As a Novel Treatment for Pulmonary Hemorrhage in Critically Ill Pediatric Patients: An Observational Study. Crit Care Explor. 2020;29;2(1): e0075 4. Pulmonary hemorrhage: A novel mode of therapy. Respir Med. 2009;103(8):1196-1200 5. Aminocaproic acid for the management of bleeding in patients on extracorporeal membrane oxygenation: Four adult case reports and a review of the literature. Heart Lung. 2016;45(3):232-6

Critical Care - 7 Peripartum ECMO for COVID-19

Kasey Grewe[1], James Madrian[2], Wolf Kratzert[1]

[1]UCLA, Los Angeles, CA, [2]UCLA, Los Angeles, United States of America

Introduction: Here we present four cases demonstrating the utility and challenges of ECMO support for severe ARDS due to COVID-19 in the peripartum period.

Methods: Case 1: A 27 yo G3P0020 at 30w5d with obesity and COVID-19 ARDS was transferred to our center and placed on VV ECMO. She received supportive care per ARDSnet, anticoagulation, and steroids for COVID-19 and fetal lung maturity. Her ECMO course was complicated by flow issues secondary to contractions and caval compression. She was persistently hypertensive with elevated UPC, and on ECMO day 8 a cesarean delivery was performed due to worsening thrombocytopenia concerning for PEC and DIC. Her postoperative course was complicated by DIC and AKI. She was removed from ECMO POD2, and following tracheostomy-assisted ventilator wean, was discharged 1 month after admission. The baby was discharged following a stay in NICU. Case 2: A 42 yo G2P0101 at 26w5d with chronic HTN, gDM, and COVID-19 ARDS was transferred to our center and placed on VV ECMO. On ECMO day 1 she went into preterm labor requiring cesarean delivery. Despite initiation of heparin infusion on POD1, a DVT was discovered on POD2. She was weaned from VV ECMO on POD7 and extubated POD8. Her post-ICU course was complicated by PE, but she was ultimately discharged 16 days after admission while her baby remained in the NICU. Case 3: A 33 yo G3P3003 presented postpartum day 2 following full term NSVD of a healthy infant for severe dyspnea due to COVID-19. Following a cardiac arrest in the ambulance with subsequent ROSC and seizure-like activity she was placed on VV-ECMO, but transitioned to VV-A ECMO due to hemodynamic instability. Initial studies were negative for PE and demonstrated an LVEF 20-25% that subsequently recovered. She experienced severe anoxic brain injury that did not improve. Her course was otherwise complicated by pneumothoraces, renal failure, and sepsis. With support of the ethics committee, care was not escalated for continued decompensation, and the patient passed away 33 days after initiation of ECMO. Case 4: A 31 y.o. G4P1122 at 33w3d was admitted with COVID-19 pneumonia requiring HFNC and BiPAP, and had NSVD at 35 weeks following induction of labor. On postpartum day 2 she was intubated, transferred to our center, and placed on VV ECMO. Following 16 days of supportive care she was successfully weaned from ECMO and discharged to home 9 days later.

Conclusion: The peripartum period presents unique hemodynamic and ventilatory challenges in ARDS and for ECMO management, some previously described[1,2]. Given the logistical challenges of proning with a gravid uterus, ECMO allowed us to achieve a higher SpO2 goal of >92% for fetal oxygen delivery[3] while maintaining lung protective ventilation supine. We took care to minimize sweep to avoid hypocapnia as pCO2 is a potent driver of placental perfusion[4]. As with other hyperdynamic states, these patients were prone to hypoxemia with increases in cardiac output due to increased shunt fraction on ECMO. We experienced challenges with device flow due to caval compression and contractions that were responsive to fluid resuscitation and left lateral positioning. In discussion with our obstetric colleagues, we learned that preterm delivery has not been shown to improve maternal oxygenation in ARDS[5] and thus planned for delivery in the OR only for fetal indications, or bedside cesarean for maternal arrest. In two cases we saw marked improvement in lung compliance following delivery, however in two others, respiratory failure worsened within 48 hours postpartum. Steroids provided dual benefit in COVID-19 pneumonia and fetal lung maturity[6,7]. While anticoagulation was necessary due to thrombotic risk of ECMO, pregnancy, and COVID-19, at times it was contraindicated due to postoperative state and DIC, requiring careful attention to bleeding and thrombosis. We encountered ethical dilemmas unique to the peripartum population. In one case a conscious decision was made to forgo continuous fetal monitoring to prioritize maternal outcomes. In another case, peri-arrest hypoperfusion led to severe anoxic brain injury, but the emotional weight of facing a motherless newborn led to a prolonged ECMO course and an end-of-life ethical standoff. These experiences at our center highlighted the ability of ECMO to enhance peripartum supportive

care in COVID-19, and the importance of multidisciplinary teamwork and careful patient selection.

References: 1. Lapinsky SE et al. Am J Respir Crit Care Med. 1995;152(2):427-455. 2. Moore SA et al. J Thorac Cardiovasc Surg. 2016;151(4):1154-1160. 3. Ashokka B et al. Am J Obstet Gynecol. 2020;223(1):66-74.e3. 4. Hanka R et al. J Physiol. 1975;247(2):447-460. 5. Tomlinson MW et al. Obstetrics & Gynecology. 1998;91(1):108-111. 6. RECOVERY Collaborative Group. N Engl J Med. 2020 Jul 17:NEJMoa2021436. 7. Roberts D et al. Cochrane Database Syst Rev. 2017 Mar 21;3(3):CD004454

Critical Care - 8 Necrotizing Pneumonia Leading to Arterial Air Embolism Causing Acute Ischemic Stroke

Brandon Layton[1], James L Walker[1]

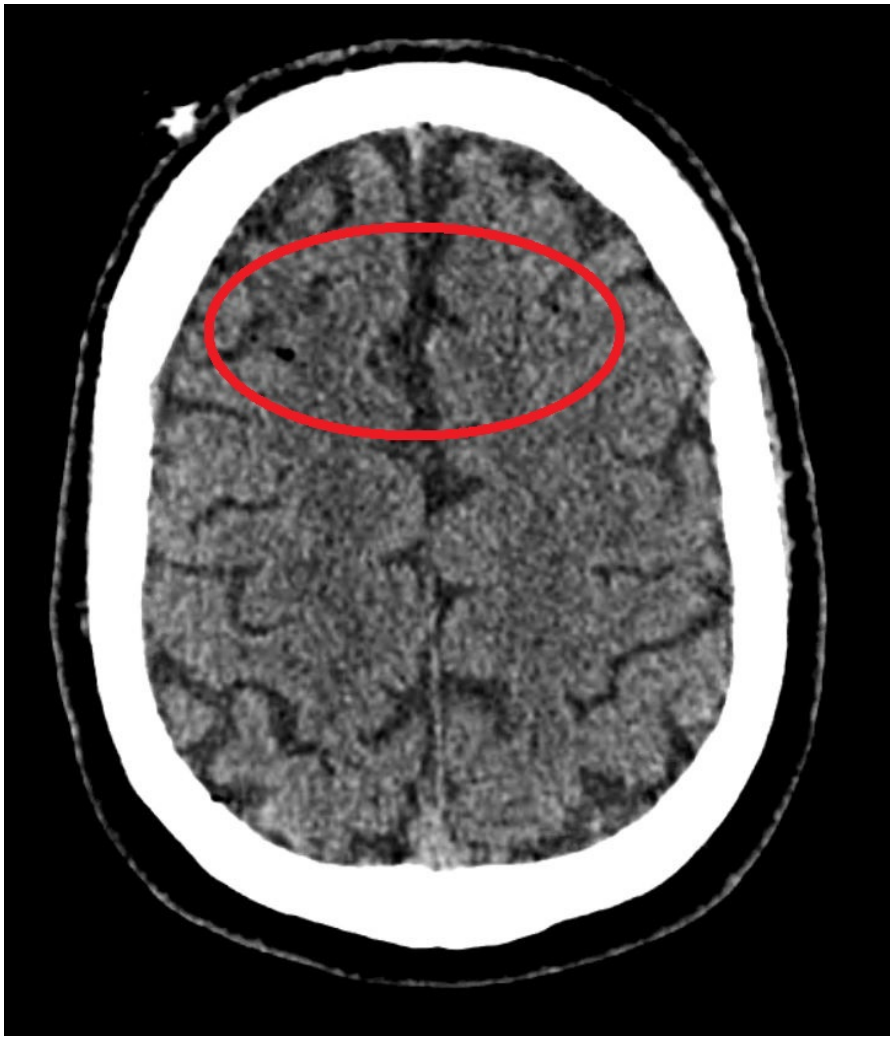
[1]University of Kansas School of Medicine-Wichita, Wichita, KS

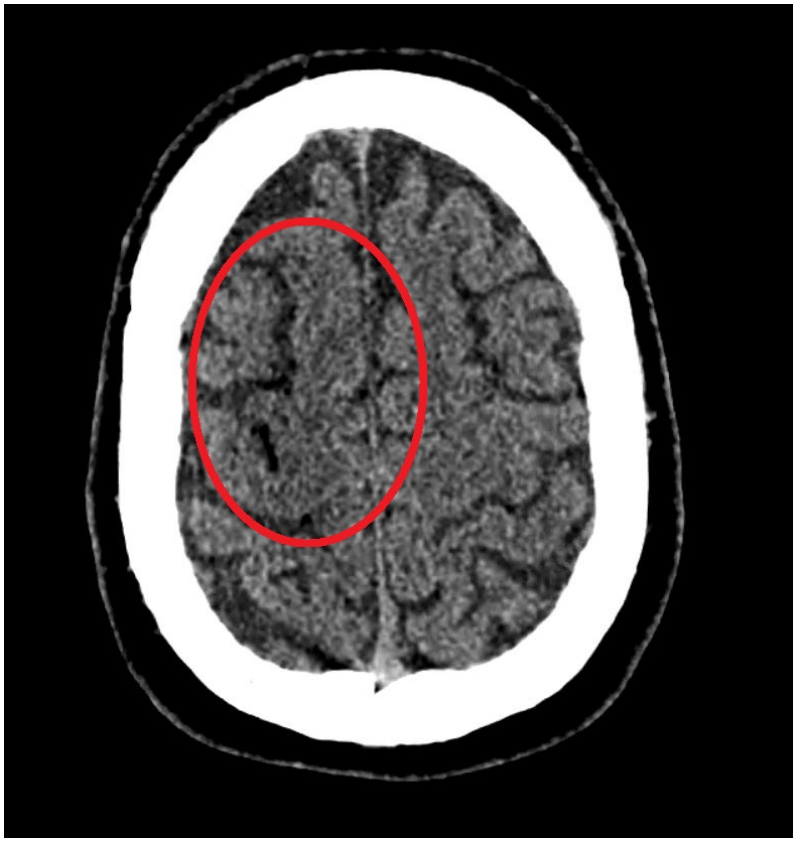
Introduction: Air embolism (AE) is a rare but established cause of acute ischemic stroke (AIS). While quantitative epidemiological data is not available, the majority of reported cases are from iatrogenic causes such as accidental IV AE in the presence of a PFO or vascular injury during upper endoscopy. This is a case report of an AIS caused by an arterial AE after an intense coughing fit in a patient with necrotizing pneumonia.

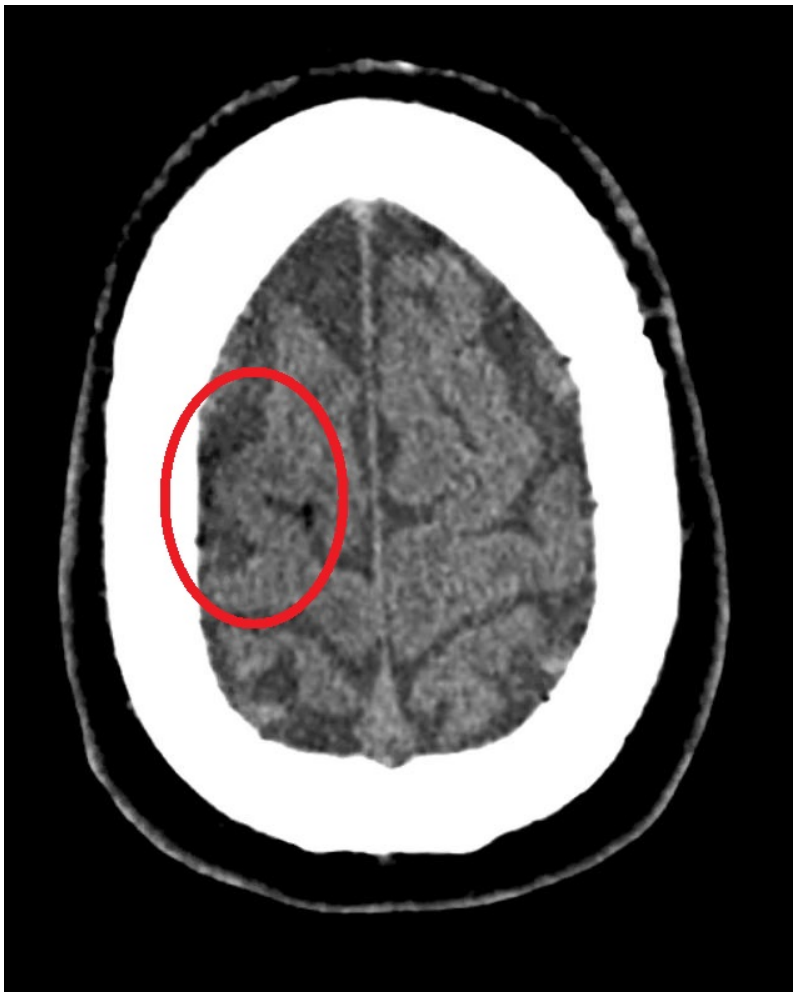
Methods: A 65-year-old male was admitted for worsening fevers, chills, and shortness of air. CT thorax showed a 7x7cm necrotic consolidation in the right lower lobe with central foci of air. The patient was diagnosed with necrotizing pneumonia and treated with broad-spectrum antibiotics. The day after admission he had a violent coughing fit in front of his wife and suddenly became mute with altered mental status and severe weakness in all four extremities. CT head and CTA head and neck were obtained and initially read as normal. The patient rapidly regained his ability to speak but significant weakness persisted, worse on the left side. Given the initial negative imaging findings, the patient qualified for and received IV tPA, and was transferred to our comprehensive stroke center for further care. MRI of the brain revealed patchy areas of acute/subacute bilateral parasagittal cerebral infarcts in a watershed distribution. TTE revealed normal systolic function and no right-to-left shunt including no patent foramen ovale. The lack of hypotensive episode or intracranial vascular abnormality combined with normal cardiac function and anatomy left little to account for the MRI imaging findings. However retrospective review of the initial CT and CTA revealed the presence of small but diffuse bilateral air emboli, most significant in the right hemisphere. Throughout his stay, the patient continued to make modest neurologic improvements and was given a prolonged course of antibiotics and pulmonary rehabilitation before discharge to a skilled nursing facility. He experienced no other acute neurological events or complications associated with his necrotizing pneumonia and did not require surgery or other intervention.

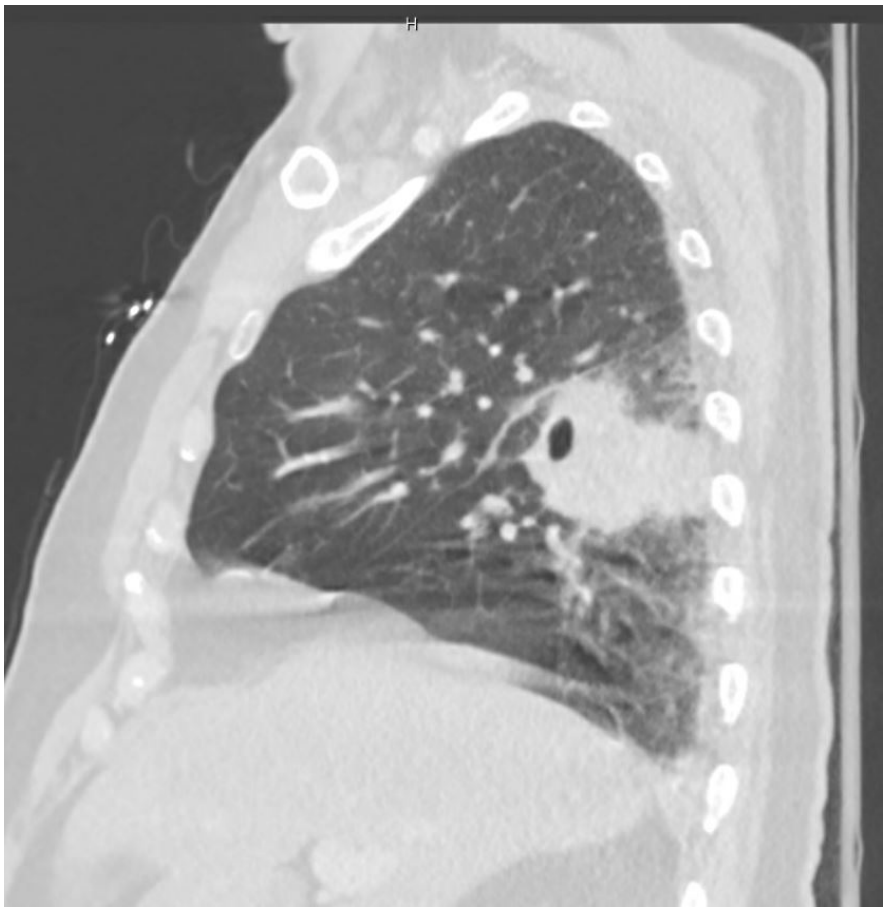
Conclusion: Several aspects of this case are relevant for clinical consideration. Given the rarity of air-embolic strokes, more common etiologies were considered. A low-flow state-such as from systemic hypotension or severe systolic heart failure-is the most likely cause of watershed-distribution ischemia. But careful review of the patient's vitals revealed no significant hypotension preceding the neurological symptoms and echocardiogram showed no evidence of heart failure. A paradoxical AE was also considered as the patient did have an IV at the time, but no irregularities were suspected with his IV infusions and he had no evidence of PFO that could have led to arterial distribution watershed stroke. Both the patient and his wife stressed how violent the coughing fit was, and how temporally related this coughing episode was to the onset of his neurological symptoms. It is plausible that a violent cough increased intrathoracic pressure enough to force trapped air in the necrotic lung into the pulmonary veins with subsequent transit to the left side of the heart and into the systemic arterial circulation. Two previous cases of severe coughing fits leading to AIS have been reported, one associated with a necrotizing pneumonia (1) and the other with tuberculosis(2) . While high-quality evidence is sparse, hyperbaric oxygen therapy (HBOT) is considered the most effective treatment of cerebral AE. The increased partial pressure of oxygen facilitates the nitrogen washout and absorption of the trapped air. While it is most advantageous in the acute phase, there are reports of its effectiveness even when delayed by as much as 39 hours (3). While the air burden on the head CT was low, this patient may have benefited from HBOT. This would not have been without risks given the patient's concomitant lung disease. HBOT has been shown, however, to be well-tolerated even in patients with significant pulmonary disease including large bullae (1,4) . This unique case demonstrates the possibility of an air embolus causing an acute ischemic stroke in the setting of a necrotizing pneumonia. This should be included in the differential when acute neurological symptoms are observed in a patient with severe pulmonary disease accompanied by a severe cough.

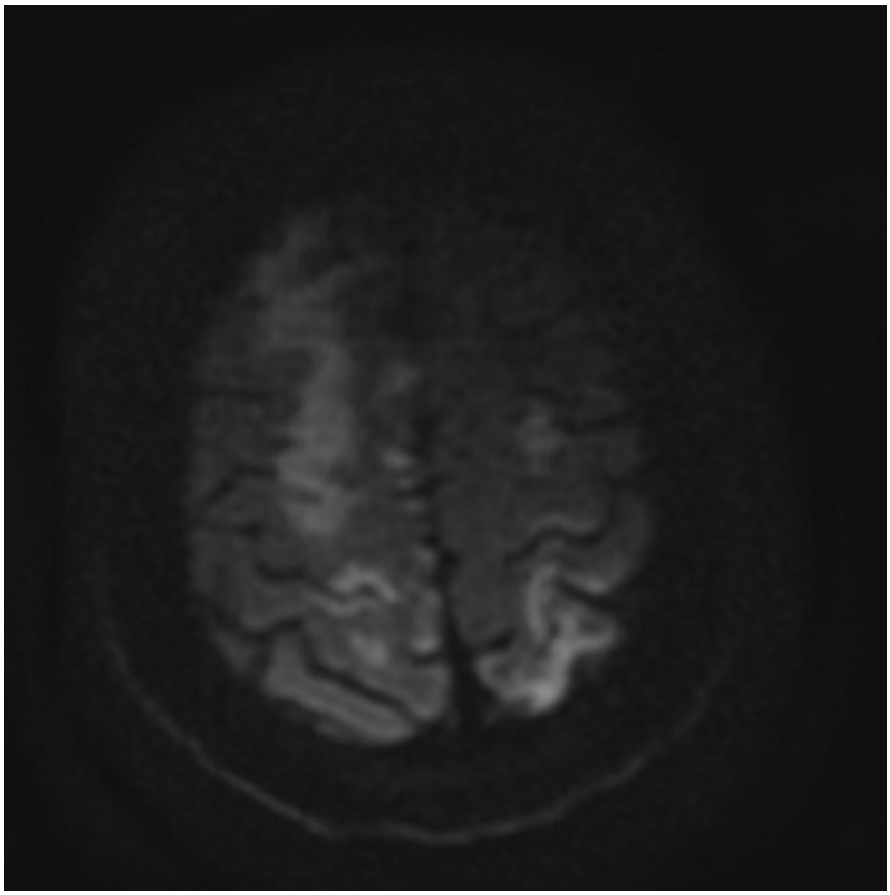
References: Non-dysbaric arterial gas embolism associated with chronic necrotizing pneumonia, bullae and coughing: a case report. 2017; 44:73-77. Cerebral gas embolism after a cough fit. 2005; 24:64-7. A favorable outcome despite a 39-hour treatment delay for arterial gas embolism: case report. 2016; 43:457-61. Massive Ischemic Stroke Due to Pulmonary Barotrauma and Cerebral Artery Air Embolism During Commercial Air Travel. 2017; 13:660-4.








H



Critical Care - 9 Venoarterial Extracorporeal Life Support in COVID-19 Myocarditis

Kyle Ferguson[1], Brendon Hart[1], Cory Roth[2], Amar M Bhatt[1]

[1]Wexner Medical Center at The Ohio State University, Columbus, OH, [2]The Ohio State University College of Medicine, Columbus, OH

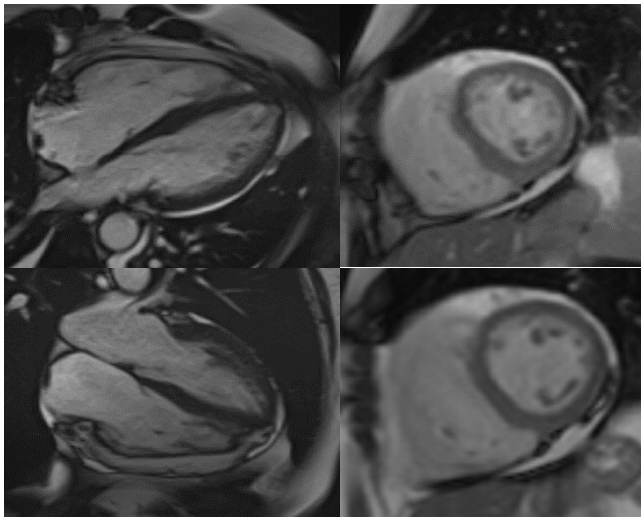
Introduction: In late 2019 a novel coronavirus was identified named coronavirus disease 19 (COVID-19). Complications include acute respiratory distress syndrome (ARDS), secondary infections, and myocardial dysfunction1. Here we describe a case of fulminate myocarditis (FMC) secondary to COVID-19 managed with venoarterial extracorporeal life-support (VA-ECLS).

Methods: A 60 year old female presented to the emergency department with palpitations and malaise 9 days after a positive COVID-19 PCR test. She was found to have diffuse ST depressions with an elevated troponin and pro-BNP. Transthoracic echocardiogram (TTE) showed biventricular systolic dysfunction with an LVEF of 30%. Left heart catheterization showed no obstructive cardiac disease. The patient became hemodynamically unstable requiring intubation as well as inotropic and vasopressor support. With continued clinical deterioration, peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) was initiated via the left femoral artery and vein for full cardiac support. She was treated with high-dose steroid therapy as well as cytokine hemofiltration therapy. Repeat echocardiogram following initiation of VA-ECMO showed an EF of 10-15% with severe biventricular dysfunction. Her hospital course was further complicated by arrhythmias requiring pharmacologic and electrical cardioversion.

Results: Mechanical and pharmacologic support was weaned with improvement in cardiovascular function, and on hospital day (HD) 11 the patient was decannulated from VA-ECMO. Intraoperative transesophageal echocardiogram (TEE) showed an LVEF of approximately 25%. The patient was extubated on HD 13 and transferred out of the ICU on HD 19. Cardiac MRI showed findings consistent with acute myocarditis and subsequent myocardial biopsy was positive for diffuse inflammatory cell infiltrates and myocyte injury consistent with acute myocarditis. Immunohistochemical stain of the endomyocardial biopsy was negative for other viruses. The patient continued to improve with follow up echocardiography and cardiac MRI showing significant improvement in her cardiac function.

Conclusion: The primary use of ECLS for COVID-19 has been due to hypoxemia rather than circulatory failure. This case highlights a unique application of ECLS as primary circulatory support for FMC due to COVID-19. Various studies have demonstrated successful weaning rates of approximately 80% and survival rates of 72% in patients with FMC supported with VA-ECLS2. Myocardial hypoxia leads to a substantial inflammatory response, cellular injury and death. In this case, the circulatory support provided by VA-ECLS may have helped to minimize hypoxic injury and the subsequent inflammatory cascade. Additionally, this patient's presentation of COVID-19 FMC is unique due to exclusive myocardial involvement with sparing of respiratory function. Previous reports of COVID-19 FMC were complicated by concomitant fibro-proliferative ARDS3. Lastly, ECLS exit strategies were limited in this case. Due to multi-organ failure in the setting of active infection, this patient was not a candidate for heart transplantation or advanced circulatory support devices. In this case ECLS was instituted as a bridge to recovery with a good outcome.

References: 1. Cardiovascular disease and COVID-19. Diabetes Metab Syndr. 2020; 14(3):247-250. 2. Favourable clinical outcome in patients with cardiogenic shock due to fulminant myocarditis supported by percutaneous extracorporeal membrane oxygenation. Eur Heart J. 2005; 26(20):2185-92. 3. Extracorporeal membrane oxygenation (ECMO) in patients with COVID-19: a rapid systematic review of case studies. Eur Rev Med Pharmacol Sci. 2020;24(22).



Cardiac MRI with gadolinium contrast enhancement from 9/4/2020 (Top) versus 12/31/2020 (Bottom) demonstrating significant improvement in biventricular function as well as resolution of myocardial edema and inflammation secondary to COVID-19 myocarditis.

Critical Care - 10 Orthotopic Liver Transplantation Associated Graft-Versus-Host Disease: A Report on Post-Operative Intensive Care Unit Management

Jonathan M Weimer[1], Blair Bigham[2], Spencer J Craven[1], Jeffrey Jopling[1], Tiffany Lee[1], Deshka S Foster[1], Ara Ko[1]

[1]Stanford Hospitals, Stanford, CA, [2]Stanford U, Palo Alto, CA

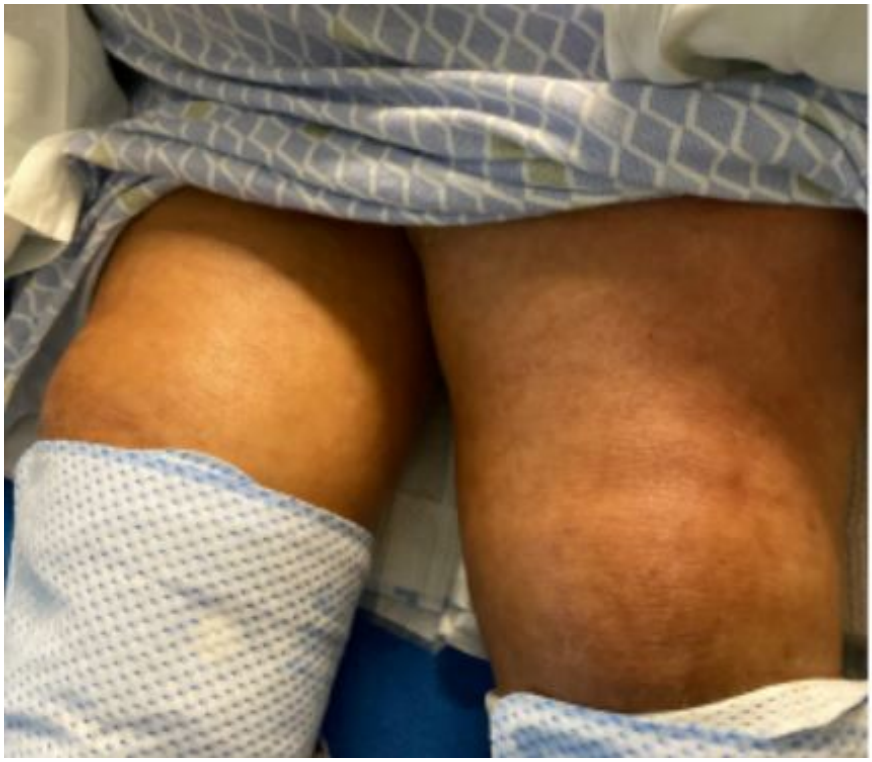
Introduction: Graft-versus-host disease (GVHD) is a rare complication following orthotopic liver transplantation (OLT) and is associated with a disturbingly high mortality rate of nearly 75% at 6 months [1]. Diagnosis can be challenging due to the nonspecific clinical findings at presentation and broad differential for the post-operative OLT patient. Management is also complex due to variable results and limited data. Here we describe a fatal course of OLT associated GVHD, discussing the presenting signs, diagnostic workup, and attempted therapeutic interventions prior to patient death.

Methods: A 68-year-old female with history of obesity, type 2 diabetes, renal cell carcinoma status post nephrectomy, and NASH/alcoholic cirrhosis (initial MELD-Na 23) complicated by refractory ascites, was admitted to the hospital following TIPS complicated by development of hepatorenal syndrome. On hospital day 7, she underwent OLT with veno-veno bypass. Intraoperative course was uneventful, but pathology later revealed a 4 cm cholangiocarcinoma without lymphatic or vascular invasion. Both donor and recipient were CMV and EBV positive matches. The patient received intraoperative methylprednisolone, as well as postoperative tacrolimus and anti-thymocyte globulin. She was extubated POD 1, transitioned to intermittent hemodialysis POD 3, and transferred to the floor POD 4. Prophylaxis was maintained with trimethoprim/sulfamethoxazole, valganciclovir, and caspofungin. She remained thrombocytopenic with platelet counts in the 20 to 40 K/¬µL range despite several transfusions, and anemic with hemoglobin < 10 g/dL. Beginning POD 14, the patient experienced a slow decline in her leukocyte count from 8.3 to <0.1 K/¬µL over 12 days. Valganciclovir and trimethoprim/sulfamethoxazole were discontinued as leukopenia worsened. On POD 22, she returned to the intensive care unit for hypotension and worsening pancytopenia. There was no evidence of ongoing hemorrhage or hemolysis. Empiric antibiotics were started with linezolid and piperacillin-tazobactam, transitioned to meropenem, but initial microbiology data was negative. Plasma CMV and EBV were identified but below the lower limit of detection. Erythropoietin and granulocyte colony-stimulating factor were started without effect on cell counts. The patient required re-intubation on POD 25. Tacrolimus was discontinued and acyclovir started. The patient had increasing stool output, and a CT scan revealed distal small bowel and colonic thickening, as well as lytic bone lesions concerning for metastasis. Oral vancomycin was started, but Clostridium difficile testing was negative. A diffuse morbilliform rash of chest and extremities (Figure 1) appeared at this time and was biopsied, demonstrating an interface dermatitis. On POD 26, repeat blood cultures grew Enterococcus faecium, while on POD 28 human herpesvirus 6 (HHV-6) viremia was discovered with 21,956 copies/mL. Given the clinical progression, a chimerism assay was sent, revealing 78% donor CD3 cells. Due to concern for underlying GVHD, the patient was started on methylprednisolone and photopheresis on POD 31. Basiliximab was started on POD 34. Despite continued antibiotic, immunosuppressive, and marrow stimulant therapies, the patient's vasopressor requirement escalated and cell counts did not recover. Candidemia was discovered on POD 36 despite antifungal therapy. The patient expired POD 37.

Conclusion: In summary, a 68-year-old female with NASH/alcoholic cirrhosis underwent OLT with course complicated by new diagnosis of cholangiocarcinoma, renal failure, respiratory failure, septic shock, neutropenic enterocolitis, pancytopenia, and diffuse morbilliform rash. Multidisciplinary discussions were held regarding the etiology of her pancytopenia and rash, with early focus on infectious (bacterial and viral) and drug causes. Though non-specific findings, concern was raised for OLT associated GVHD, which was screened using a blood chimerism assay. This assay revealed a majority of donor derived CD3 cells, suggesting that donor T lymphocytes accompanying the liver persisted and mediated marrow hypoplasia. The findings of dyskeratotic cells in the follicular

infundibulum also favored GVHD. Given the above, our patient was started on an immunosuppressive regimen of steroids, IL2 receptor antibody, and photopheresis in an attempt to suppress the donor derived T cells. Unfortunately, the patient succumbed to overwhelming sepsis.

References: [1] Transplantation. 2016 Dec; 100(12): 2661-2670.



Critical Care - 11 Perioperative Challenges and Management of Status Dystonicus: A Case Report

Derek W Scott[1], Steven T Morozowich[2]

[1]Mayo Clinic Alix School of Medicine, Scottsdale, AZ, [2]Mayo Clinic, Phoenix, AZ

Introduction: Dystonia is a state of involuntary sustained muscle contractions causing abnormal twisting movements or postures that impair normal movement (1,2). Historically, it has been classified as primary, secondary, or psychogenic/functional (1,3). Severe dystonia manifesting as continuous generalized contractions is referred to as status dystonicus and is considered rare (2,4), but represents a medical emergency associated with significant morbidity and mortality due to the potential to develop rhabdomyolysis and/or progressive respiratory failure (2). Some patients are prone to recurrent episodes and status dystonicus is often a triggered event (2). Although infection, dehydration, and medication adjustments are the most common triggers, trauma/injury, pain, surgery, anesthesia, and psychological stress have also been reported (2). Thus, anesthesiologists must be prepared for critical care management throughout the perioperative period when caring for patients with a history of dystonia. We present a case of an adult female with a history of dystonia following prior general anesthesia (GA) who developed status dystonicus following a minor surgical procedure under monitored anesthesia care (MAC) targeting minimal sedation.

Methods: A 41-year-old female presented for a routine pacemaker generator change as an outpatient. Her past medical history was significant for a history of dystonia following prior surgery under GA at an outside hospital that required postoperative intensive care management, but her diagnosis of dystonia was not specified further during her medical evaluation at the time. She denied any recent changes to her health or medications and her vital signs, physical exam, and preoperative laboratory values were unremarkable. The procedure was performed under MAC targeting minimal sedation in an attempt to prevent the development of dystonia that followed her prior GA. Sedation with a propofol infusion of 25mcg/kg/min and analgesia with infiltration of 15 mL of 1% lidocaine was administered. During the procedure the patient verbally noted that she was beginning to develop generalized contractions of her extremities. The procedure was completed successfully and the patient was taken to the postanesthesia care unit where she developed progressive worsening generalized tonic flexion of all extremities that remained fixed. Neurology consultation established the working diagnosis of status dystonicus. Supportive treatment including initial sedation with benzodiazepines was recommended with consideration of intubation and sedation with a dexmeditomidine infusion as successful treatment with other apha-2 adrenergic receptor agonists, such as clonidine, have been reported in the literature (2). After a period of observation following a total midazolam dose of 2 mg intravenously her condition did not improve and an arterial blood gas demonstrated a respiratory acidosis indicating progressive impairment of respiratory function (2). This prompted intubation and sedation with dexmedetomidine. Routine laboratory values obtained perioperatively were otherwise unremarkable, there was no evidence of a metabolic acidosis, and a creatine kinase level was normal. The following day her dexmedetomidine was weaned, she was following commands, was no longer exhibiting dystonia, and was successfully extubated. Upon subsequent questioning, her prior episode of dystonia following GA was very similar and thus likely represented status dystonicus.

Conclusion: Dystonia has been classified as primary, secondary, or psychogenic/functional (1,3). Status dystonicus is rare but may require critical care management perioperatively. Anesthesia is a reported trigger (2) and considering our patient's history of dystonia following prior GA we attempted to perform her procedure under MAC targeting minimal sedation. However, she ultimately developed status dystonicus likely due to procedural related trauma/injury, pain, and/or psychological stress, all of which are reported triggers (2). Although noteworthy, the significance of our patient's rapid onset and resolution of dystonia remains unclear (5). This case illustrates the substantial perioperative challenges that patients with a history of dystonia can present. Based on the use of clonidine for the management of dystonia in the literature, consideration should be given to the use of dexmedetomidine as part of the anesthetic and

perioperative management when caring for patients with a history of dystonia.

References: 1. Albanese A, et al. Phenomenology and classification of dystonia: a consensus update. Movement disorders: official journal of the Movement Disorder Society 2013;28:863-73. 2. Allen NM, et al. Status dystonicus: a practice guide. Dev Med Child Neurol 2014;56:105-12. 3. Ganos C, et al. The Phenomenology of Functional (Psychogenic) Dystonia. Movement Disorders Clinical Practice 2014;1:36-44. 4. Manji H, et al. Status dystonicus: the syndrome and its management. Brain: a journal of neurology 1998;121 (Pt 2):243-52. 5. Termsarasab P, et al. Dystonic storm: a practical clinical and video review. Journal of Clinical Movement Disorders 2017;4:10.

Critical Care - 12 Complexities of Anticoagulation: A Trauma Critical Care Dilemma Involving Pulmonary Emboli, Atrial Septal Defect, and Traumatic Brain Injury

Dylan Masters[1], Julin F Tang[2]

[1]UCSF, San Francisco, CA, [2]UCSF/ZSFGH, San Francisco, CA

Introduction: In the management of a trauma critical care patient, there are often directly opposed medical factors that create decision-making complexity. In this case of a 79 year old trauma patient, after control of initial injuries, competing priorities of bleeding risk and clotting events create a therapeutic dilemma that highlights the challenges of anticoagulation in the trauma critical care population. A new ischemic stroke of unknown origin is often secondary to atrial fibrillation, or carotid atherosclerosis, yet paradoxical embolus across an atrial septal defect (ASD) remains a rare but significant potential cause1,2. In the case of known deep vein thrombosis and pulmonary emboli, to prevent further embolization, inferior vena cava (IVC) filters remain a commonly employed therapy when anticoagulation is contraindicated3,4. Progression of embolic events in the setting of widespread deep vein thrombosis (DVT) can necessitate reconsideration of therapeutic anticoagulation5. Much of the research on anticoagulation in traumatic brain injury focuses on prophylactic anticoagulation6. The relevant studies on therapeutic anticoagulation mainly focus on pulmonary emboli, but the same principles may apply for paradoxical emboli across an ASD7. In this rare case of a trauma patient developing multiple embolic ischemic insults from an ASD despite an IVC filter, we discuss paradoxical emboli and anticoagulation in the setting of traumatic brain injury.

Methods: A 79 year old man with history of diabetes mellitus complicated by bilateral lower extremity diabetic neuropathy, and chronic hypertension, presented after motor vehicle accident as a restrained driver hitting concrete at 50-60 miles per hour. He was found to have a 5mm subdural hematoma (SDH) without mass effect or herniation, and also had bedside abdominal ultrasound positive for free fluid. He was taken emergently to surgery and found to have small bowel and superior mesenteric artery injuries, which were controlled. He was stabilized in the intensive care unit (ICU), and had postoperative imaging showing stable SDH, new right middle cerebral artery (MCA) ischemic stroke, left upper lobe pulmonary embolus (PE), and multiple limb deep vein thrombosis (DVT). He had a second operative session including endoscopic retrograde cholangiopancreatography (ERCP), inferior vena cava (IVC) filter placement, and a second-look laparotomy. After surgery, he was noted to have new ST elevations in leads II, II and aVF, with new ST depressions and old T wave inversions in leads V1 and V2. Serial high sensitivity troponins were elevated, and peaked at 4768. He was considered for urgent cardiac catheterization, as well as anticoagulation for acute coronary syndrome. Repeat neurological imaging revealed hemorrhagic conversion of his MCA stroke, and anticoagulation was deferred. He subsequently was found to have bilateral pulmonary emboli, and non-obstructive coronary arteries on CT angiography. Echocardiography revealed an ostium secundum atrial septal defect (ASD), without depressed cardiac function or wall motion abnormality. Further embolic injuries then developed with multiple new brain infarcts, as well as new splenic and renal infarcts. He started therapeutic heparin anticoagulation, and fortunately did not progress to have any further cerebral hemorrhage. He had a prolonged ICU course but ultimately discharged to a rehabilitation facility after five weeks, with plan for outpatient ASD closure.

Conclusion: In this case, a patient with severe injuries from trauma had extensive serial imaging, which chronicled the unusual progression of DVTs leading to both PEs, and paradoxical emboli across an ASD. Although management with an IVC filter was employed, continued embolic events necessitated consideration of systemic anticoagulation. However, his traumatic brain injuries created a dichotomy of conflicting priorities in regards to his further management. Ultimately, he was successfully treated with systemic anticoagulation, and planned for future correction of his symptomatic ASD. This rare case illustrates the unique pathophysiology of a bi-directional ASD, prompts discussion of conflicting priorities related to systemic anticoagulation, and highlights the complexities of trauma critical care.

References: 1. Homma et al. Patent foramen ovale and stroke. J Cardiol. 2010 Sep;56(2):134-41. 2. Nakanishi et al. Patent foramen ovale and cryptogenic stroke. Trends Cardiovasc Med. 2017 Nov;27(8):575-581. 3. Bikdeli et al. Inferior Vena Cava Filters to Prevent Pulmonary Embolism: Systematic Review and Meta-Analysis. J Am Coll Cardiol. 2017 Sep 26;70(13):1587-1597. 4. Marshall et al. Diagnosis and management of life-threatening pulmonary embolism. J Intensive Care Med. Sep-Oct 2011;26(5):275-94. 5. Akinseye et al. Simultaneous acute cardio-cerebral infarction: is there a consensus for management. Ann Transl Med. 2018 Jan;6(1):7. 6. Lu et al. Clinical outcomes following early versus late pharmacologic thromboprophylaxis in patients with traumatic intracranial hemorrhage. Neurosurg Rev. 2020 Jun;43(3):861-872. 7. Chipman et al. Therapeutic anticoagulation in patients with traumatic brain injuries and pulmonary emboli. J Trauma Acute Care Surg. 2020 Sep;89(3):529-535.

Critical Care - 13 A large Malignant Peripheral Nerve Sheath Tumor of the Mediastinum in a Patient with Neurofibromatosis Type 1

Ioannis (Yanni) Angelidis[1], Ashley Budd[2]

[1]Northwestern University, Feinberg school of medicine, Chicago, United States of America, [2]Northwestern Memorial Hospital, Chicago, United States of America

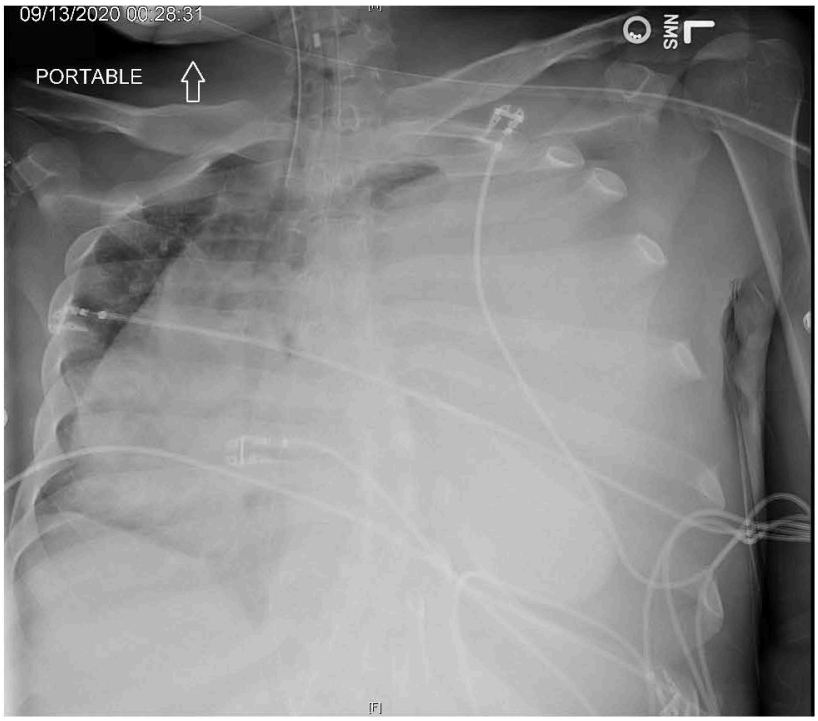
Introduction: Neurofibromatosis type 1 (NF1) is an autosomal dominant disease with a worldwide incidence of 1 per 2500 to 3000 individuals1. It is caused by a mutation in the NF1 gene on chromosome 17 and is associated with increased morbidity and mortality. Malignant peripheral nerve sheath tumors (MPNSTs) are usually associated with NF1. They can be found in any part of the body, however intrathoracic MPNSTs are rare with only a few reported adult cases2. Even though not common, cardiovascular malformations (CVMs) have been reported in these unique patients3,4,5.

Methods: A 24-year-old male with history of NF1 and developmental delay presented with acute hypercapnic respiratory failure in the setting of a large mass in the left posterior mediastinum and hemithorax. Imaging showed a 26 cm vascular mass in the left hemithorax causing near collapse of the left lung with mediastinal shift leading to partial compression of the right lung (fig. 1). Biopsy was performed and pathology revealed sarcoma with necrosis, consistent with malignancy in the peripheral nerve sheath. Interventional radiology performed embolization of the large collateral vessels feeding the tumor prior to surgical intervention. The patient then underwent clamshell thoracotomy with mediastinal mass excision. He did well immediately post-operatively; however, on post-operative day seven he suffered a cardiac arrest of unknown etiology. Return of spontaneous circulation was achieved, yet multiple hemodynamically unstable aberrant rhythms persisted requiring Veno-Arterial (VA) Extracorporeal Membrane Oxygenation (ECMO). Transthoracic echocardiography (TTE) revealed an ejection fraction (EF) of 28% and the patient was diagnosed with a new acute cardiomyopathy thought to be secondary to acute illness. Over days, the patient had recovery of his heart function and hemodynamics, and he was transitioned to Veno-Venous (VV) ECMO due to persistent hypoxemia and hypercapnia. He was subsequently decannulated three days after. A follow-up TTE showed EF 59% and a membranous ventricular septal defect (VSD) with moderate concentric left ventricular hypertrophy.

Conclusion: The prognosis of patients with NF1 and a malignant nerve sheath tumor is generally poor, with a 5-year survival rate of about 40%5. CVMs can be another source of morbidity or even mortality for these patients. Lin et al. found that pulmonary stenosis was the most frequent CVM, but cases of VSD, tetralogy of Fallot, and cardiomyopathies were also reported4. Hamilton et al. presented a case of sudden death of a young adult with NF15. Autopsy revealed cardiomyopathy, fibrotic coronary arteries and a 'floppy' mitral valve. Data about the frequency of CVMs in young NF1 adults are scant4. Treatment can vary from lifestyle changes and pharmacological management to more aggressive measures such as surgical correction and ECMO placement. As depicted in this case, the potential for CVMs and its associated consequences must be considered in this patient population.

References: 1. Neurofibromatosis Type 1 Revisited, 2009;123:124-133 2. Huge Intrathoracic Malignant Peripheral Nerve Sheath Tumor in an Adolescent with Neurofibromatosis Type 1, 2014;2014:951252 3. Cardiovascular Malformations and Other Cardiovascular Abnormalities in Neurofibromatosis 1, 2000, 95:108-117 4. Cardiac Findings in an Individual with Neurofibromatosis 1 and Sudden Death, 2001, 100:95-99 5. A Malignant Peripheral Nerve Sheath Tumor of the Mediastinum in a Patient with Neurofibromatosis Type 1: Report of a Case, 2008, 38:945-947

Fig. 1



Critical Care - 14 Amlodipine overdose complicated by non-cardiogenic pulmonary edema and diffuse alveolar hemorrhage

Will Bringgold[1], Micah Long[2]

[1]UW Hospital and Clinics, Madison, United States of America, [2]University of Wisconsin, Madison, United States of America

Introduction: Amlodipine, a calcium channel blocker (CCB), is a commonly prescribed antihypertensive agent.1 Overdose with it is relatively rare, < 1 in 200 cases,2 but can lead to bradycardia, hypotension, non-cardiogenic pulmonary edema (NCPE) and death if not identified and treated aggressively.3-6 Management involves judicious intravenous (IV) crystalloid resuscitation and vasopressor infusions to counter amlodipine-induced vasodilation, glucagon to promote cardiac contractility, and IV calcium to overcome blocked calcium channels to improve cardiac contractility and vascular tone.7 In addition, intravenous lipid emulsion therapy binds excess CCB and promotes clearance, while a hyperinsulinemia-euglycemia infusion strategy counters CCB induced insulin resistance thereby increasing glucose uptake and promoting improved peripheral vascular resistance and cardiac contractility.8 Finally, while not targeting and reversing specific amlodipine effects, extracorporeal membrane oxygenation (ECMO) can be used in cases of refractory cardiogenic shock.7

Methods: A 39-year-old female with a history of hypertension, migraine headaches and depression was admitted to the intensive care unit (ICU) of an outside hospital after an attempted suicide by amlodipine overdose. She was initially seen after ingesting 300 mg of amlodipine. Her blood alcohol content was 210 mg/dL but the remainder of her drug screen was negative. Her vital signs on arrival were: heart rate 110, blood pressure 140/100 with a normal respiratory rate and oxygen saturation on room air. Her initial treatment included three grams of calcium gluconate and 8 liters of intravenous crystalloid. Unfortunately, 24 hours after arrival to the referring hospital, she developed tachypnea and hypotension unresponsive to three additional liters of fluid resuscitation. She was started on a norepinephrine (NE) infusion. Chest radiograph revealed patchy infiltrates and bilateral pulmonary edema. Due to increased work of breathing, she was intubated prior to transfer to our ICU. On arrival to our hospital, NE was quickly weaned and discontinued. Labs revealed a creatinine of 1.35 mg/dL (KDIGO Stage 2 AKI) and a leukocytosis of 25.5 K/uL. Point-of-care echocardiography exhibited normal biventricular function without valvular abnormalities or pericardial effusion. A contrast-enhanced chest computed tomography was negative for pulmonary embolism but demonstrated diffuse ground glass opacities 'most consistent with lung edema.' Furosemide and empiric antibiotics were initiated empirically to treat volume overload and aspiration pneumonia. Serum calcium was replenished with additional intermittent IV calcium injections and a high-dose insulin and dextrose infusion was started. Over the next 24 hours, the patient responded well to IV diuresis and was extubated. Afterwards, she remained tachypneic requiring intermittent high-flow nasal cannula and non-invasive positive pressure ventilatory support. Her respiratory status continued to decline and she required reintubation three days after ICU admission. Surprisingly, a bronchoscopy after her reintubation demonstrated progressive bloody aliquots consistent with diffuse alveolar hemorrhage (DAH). Her subsequent infectious, connective tissue and autoimmune workups were negative. Dexamethasone 20 mg IV daily was started and a negative fluid balance was maintained without further diuretic administration. Within 24 hours, she was extubated and transferred to general care. She completed five days of steroid therapy and was discharged home 48 hours after leaving the ICU.

Conclusion: Non-cardiogenic pulmonary edema in CCB overdose is rare but well documented in case reports. It is thought to be secondary to precapillary vasodilation and heavy fluid resuscitation in the setting of shock.6 This patient's course had the additional complication of DAH, which has not been previously reported in CCB overdose. We hypothesize that her symptoms were caused by precapillary pulmonary vasodilation and subsequent increased pulmonary capillary pressure, with further insult from crystalloid-administration induced loss of the glycocalyx.9, 10 This case adds to the literature by documenting an amlodipine overdose resulting in NCPE complicated by diffuse alveolar hemorrhage. We recommend

circumspect fluid management in amlodipine overdose.

References: 1. James P, Oparil S, Carte B, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014;311:507-20. 2. Bronstein A, Spyker D, Cantilena LJ, Green J, Rumack B, Giffin S. 2008 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 26th Annual Report. Clinical Toxicology (Philadelphia). 2009;47(10):911-1084. 3. Hasson R, Mulcahy V, Tahir H. Amlodipine poisoning complicated with acute non-cariogenic pulmonary oedema. BMJ Case Reports. 2011;4. Siddiqi TA, Hill J, Huckleberry Y, Parthasarathy S. Non-cariogenic Pulmonary Edema and Life-Threatening Shock Due to Calcium Channel Blocker Overdose: A Case Report and Clinical Review. Respiratory Care. 2014;59(2):15-21. 5. Hedaiaty M, Eizadi-Mood N, Sabzghabaee A. Noncardiogenic Pulmonary Edema after Amlodipine Overdose without Refractory Hypotension and Bradycardia. Case Reports in Emergency Medicine. 2015;2015:1-4. 6. Humbert VJ, Munn N, Hawkins R. Noncardiogenic pulmonary edema complicating massive diltiazem overdose. Chest. 1991;99(1):258-9. 7. St-Onge M, Dubé P, Gosselin S, et al. Treatment for calcium channel blocker poisoning: a systematic review. Clin Toxicol (Phila). 2014;52(9):926-44. 8. Engebretsen K, Kaczmarek K, Morgan J, Holger J. High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning. Clin Toxicol (Phila). 2011;49(4):277-83. 9.Schmidt E, Yang Y, Janssen W, et al. The pulmonary endothelial glycocalyx regulates neutrophil adhesion and lung injury during experimental sepsis. Nat Med. 2012;18(8):1217-23. 10. Chappell D, Jacob M. Role of the glycocalyx in fluid management: Small things matter. Best Pract Res Clin Anaesthesiol. 2014;28(3):227-34.

Critical Care - 15 VA-ECMO for Life-threatening Drug Overdose Complicated by Episodic Pulmonary Edema

Mikita Fuchita[1], Sarah Alber[1], Karsten Bartels[2], Samuel Gilliland[1], Martin Krause[1]

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

Introduction: Pulmonary edema and North-South syndrome are common complications associated with veno-arterial extracorporeal membrane oxygenation (VA-ECMO). We experienced a case of fluctuating cardiac function during the management of VA-ECMO for refractory circulatory shock secondary to drug overdose.

Methods: A 57 year-old woman with past medical history of insomnia, anxiety, and depression presented after ingesting approximately 60 tablets of 75 mg clomipramine and an amount of atenolol 25 mg tablets. Her initial vital signs were notable for a blood pressure of 77/49 mmHg and a heart rate of 45/min. Transthoracic echocardiography (TTE) suggestive of vasodilatory shock and reduced left ventricular (LV) ejection fraction. Norepinephrine infusion was started and titrated up to 2.0 mcg/kg/min. Following a seizure, she was emergently intubated. Upon toxicology consultation, the patient was started on epinephrine infusion, high-dose insulin therapy (11 units/kg/hr), and continuous veno-venous hemodialysis (CVVHD) for clearance of atenolol. Despite these therapies, the patient remained in refractory shock and was placed on peripheral VA-ECMO (femoro-femoral configuration) 24 hours after hospital arrival. VA-ECMO flow of 3.2 L/min ensured adequate organ perfusion. Overnight, insulin, vasopressors, and epinephrine infusions were successfully weaned off. On ECMO day 2, a chest x-ray (CXR) showed worsening pulmonary edema. TTE confirmed improved LV function and the flow was weaned to 2.0 L/min. A few hours later, the patient became acutely hypotensive requiring re-initiation of vasopressors. Concomitantly, upper body oxygenation declined precipitously. After ECMO flow was increased on ECMO day 3, pressor requirements improved dramatically but pulsatility declined. A repeat TTE revealed severe global hypokinetic LV with worsening pulmonary edema on CXR. Positive end-expiratory pressure on the ventilator was increased from 12 to 18 cmH2O, and fluid was removed aggressively via CVVHD. A dobutamine infusion was able to regain pulsatility. CXR on ECMO day 4 showed significant improvement. On ECMO day 5, CXR, again, showed complete opacification of bilateral lung fields in conjunction with vasopressor therapy for acute hypotension. Following continuous fluid removal and weaning of vasopressors, the pulmonary edema resolved. The patient remained stable, displayed improved biventricular function on bedside TTE, and was subsequently decannulated after a total duration of 7 days on VA-ECMO.

Conclusion: We describe a case of severe episodic pulmonary edema while on VA-ECMO. The concurrent use of LV unloading (venting) is known to be effective in preventing and treating pulmonary edema on VA-ECMO, but its use is not ubiquitous. Our case highlights the need to continuously re-assess the balance of inotropic and vasopressor therapy in the context of recovery from cardiogenic and distributive shock. Peripherally-cannulated VA-ECMO patients without a concurrent LV vent in place are at high risk for pulmonary edema and increased myocardial wall tension during the dynamic phase of recovery.

References: Roumy A, et al. Pulmonary complications associated with veno-arterial extra-corporeal membrane oxygenation: a comprehensive review. Crit Care 2020;24:212. Guglin M, et al. Pulmonary congestion (white lungs) on VA ECMO. The VAD Journal 2016;2(4)

Critical Care - 16 Euglycemic diabetic ketoacidosis

Caitlin Burke[1], Louisa Palmer[2]

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

Introduction: Euglycemic diabetic ketoacidosis is rare and under-reported secondary to normoglycemia and non-specific symptoms, representing only 2.6-3.2% of DKA admissions. This case report highlights the subtlety with which euglycemic DKA can present and reviews the pathophysiology and treatment of both diabetic and starvation ketoacidosis. Additionally, the case reviews patient-related risk factors for euglycemic DKA including that of sodium-glucose cotransporter-2 (SGLT-2) inhibition.

Methods: A 66 year-old male with a history of coronary artery disease, type 2 diabetes, hypertension, and hyperlipidemia presented with chest pain and dyspnea. The patient underwent coronary artery bypass grafting, LAD endarterectomy, and intra-aortic balloon pump placement. On postoperative day 5, the patient developed a new anion gap metabolic acidosis. The anion gap peaked at 22, beta-hydroxybutyrate was elevated at 4.8, and glucose remained below 250, consistent with euglycemic diabetic ketoacidosis. The patient responded to D5W and insulin therapy resulting in anion gap closure within 48 hours.

Conclusion: This case and related information inspire more effective anticipation of ketoacidosis in hospitalized patients and timely initiation of correct treatment for euglycemic diabetic ketoacidosis when it is diagnosed. Contribution from SGLT-2 inhibitors to euglycemic DKA should be considered if relevant, with particular attention to higher risk populations such as low body mass index and pregnant patients.

References: Clinical Practice and Cases in Emergency Medicine. Volume IV, no 2: May 2020 Diabetes Care 2015;38:1687-1693 Diabetes Metab Res Rev 1999; 15: 412-426 Intensive Care Soc. 2017 Aug; 18(3): 265 Treasure Island (FL): StatPearls Publishing; Jan 2020

Critical Care - 17 Clozapine-induced bowel obstruction and post-operative sedation challenges

Benjamin Stix[1], Ross Blank[1]

[1]University of Michigan, Ann Arbor, MI

Introduction: This medically challenging case report highlights that commonly used first and second-generation antipsychotics can cause a paralytic ileus resulting in bowel obstruction, due to their anticholinergic side effects. Since these drugs are commonly prescribed for delirium in the intensive care unit, further awareness of this rare, but clinically significant side effect, is warranted.

Methods: A 50-year-old man with a history of schizophrenia on high-dose clozapine presented to the Surgical Intensive Care Unit after an emergent bowel resection. His OR course was complicated by an intraoperative pulseless electrical activity cardiac arrest on induction of general anesthesia, as well as aspiration of gastric contents on rapid sequence induction, resulting in acute respiratory distress syndrome postoperatively. During his intensive care stay, he was made strict nil per os (NPO) for bowel rest after his resection and placed on total parenteral nutrition. Concurrently, the patient experienced acute agitated delirium in the setting of underlying psychosis, making his sedation while on ventilatory support difficult. Of note, the patient was allergic to haloperidol and had been trialed on multiple antipsychotics prior to initiation of clozapine as an outpatient. Psychiatry was consulted for management of his agitated delirium, and the etiology of his bowel obstruction was postulated to be related to high-dose clozapine that he received as an outpatient. Clozapine, an atypical second-generation antipsychotic, with high efficacy in treatment-resistant schizophrenia, has many side effects, including agranulocytosis, which is monitored for with regular surveillance as an outpatient. But clozapine has other lesser-known side effects, including chronic constipation, paralytic ileus, and intestinal obstruction. These are thought to be due to the anticholinergic effect of clozapine. [1,2] This case study is significant because intensive care physicians often use antipsychotics for agitated delirium and should be aware that clinically significant constipation could result in severe ileus. Ultimately, the patient recovered from his acute respiratory distress syndrome after a tracheostomy and a prolonged ventilator wean, and at the time of this case report is recovering in the hospital after a several month intensive care stay.

Conclusion: Clinically significant constipation can have life threatening complications, including bowel obstruction and even ischemia. Many of the drugs we use in the intensive care unit have anticholinergic side effects that contribute to ileus. This medically challenging case highlights a lesser-known complication of antipsychotics, a commonly used medication in the intensive care unit for delirium. Clinicians should be aware of this side effect and monitor patients closely for ileus, while they are receiving antibiotics.

References: 1. Cam B, Tanircan MZ. Clozapine-induced paralytic ileus: a case report. Ther Adv Psychopharmacol. 2014;4(4):170-172. 2. Nielsen J., Meyer J. (2012) Risk factors for ileus in patients with schizophrenia. Schizophr Bull 38: 592-598.

Critical Care - 18 A Case of Heparin-Induced Thrombocytopenia with Thrombosis in a Patient with a Left Ventricular Assist Device

Johnny Wei[1], Brent Kidd[1]

[1]University of Kansas Medical Center, Kansas City, KS

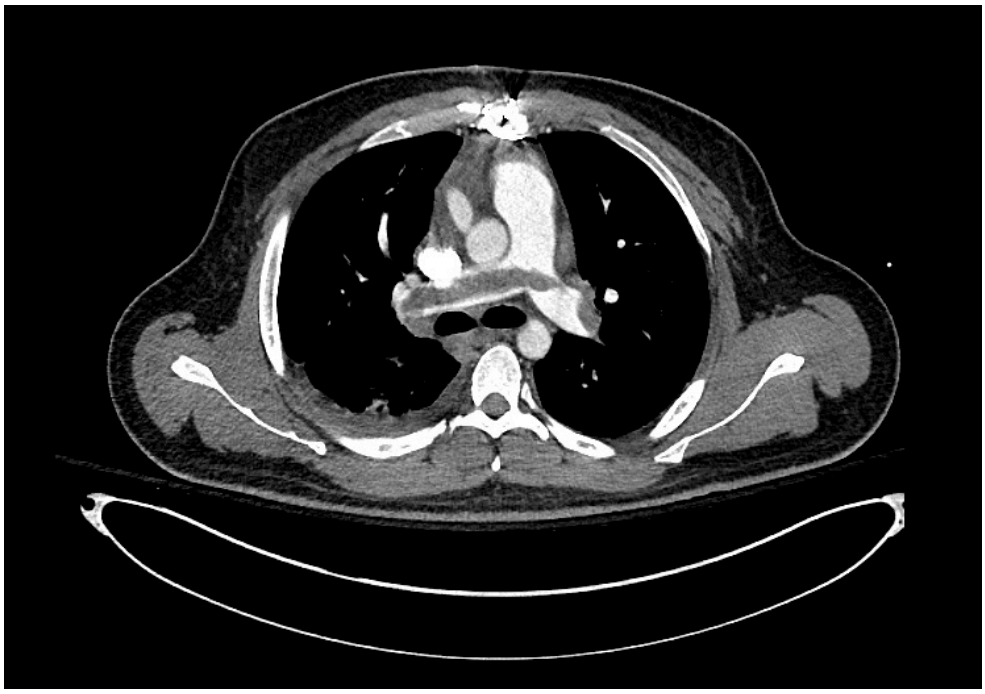
Introduction: Heparin-induced thrombocytopenia (HIT) is a rare but serious immune-mediated complication of heparin exposure and can develop in 1-3% of cardiac surgical patients. Approximately half of patients with confirmed HIT can develop thromboembolic complications, which can present serious implications and unique challenges for anticoagulation in this patient population. We present the case of a patient who underwent left ventricular assist device (LVAD) implantation and subsequently developed a saddle pulmonary embolus (PE) secondary to HIT with thrombosis (HITT).

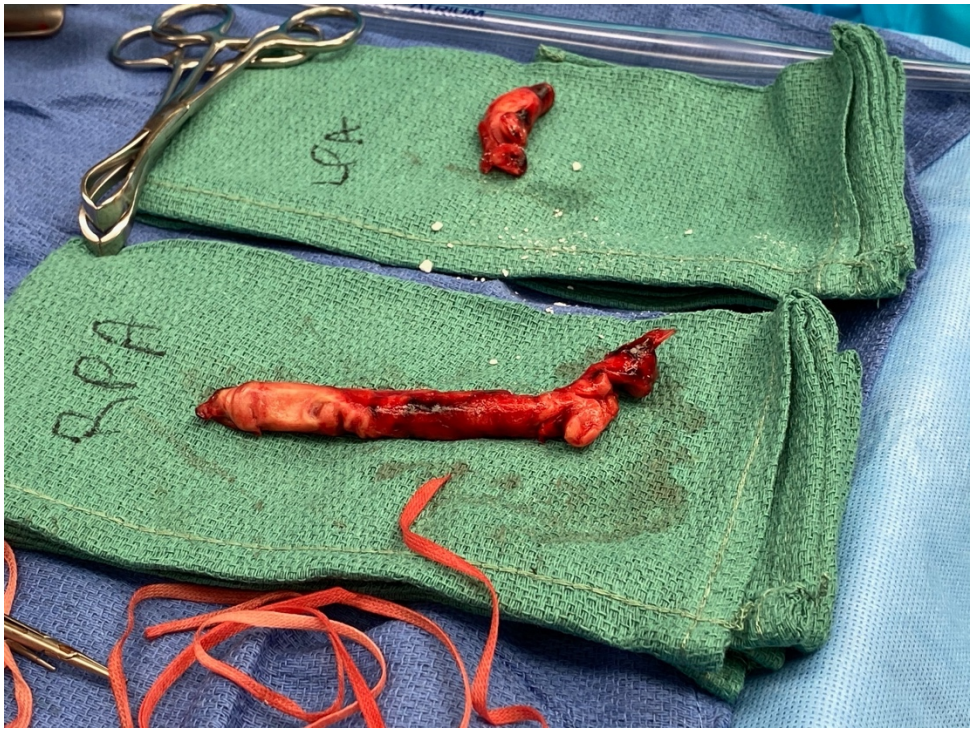
Methods: A 29-year-old previously healthy male presented to our institution with cardiogenic shock due to non-ischemic cardiomyopathy. Initial evaluation was significant for an ejection fraction of less than 20% and a cardiac index of 1.5 while on dobutamine and norepinephrine infusions. He was categorized as INTERMACS Level 1 and underwent Heartware LVAD placement on hospital day 4. He was initially anticoagulated with heparin, and subsequently bridged to warfarin. He progressed appropriately postoperatively and was transferred from the intensive care unit to a stepdown unit on postoperative day 5. On postoperative day 12, the patient was found collapsed and unresponsive with agonal respirations and low flow alarms from his LVAD. Once stabilized hemodynamically, a CT scan of the chest showed a large saddle PE and he underwent successful operative embolectomy. He developed newfound acute thrombocytopenia to a nadir of 37,000 and was evaluated for HITT given venous thromboembolism in the setting of therapeutic anticoagulation. He was found to be HIT antibody positive with a strongly positive optical density of 2.32. His postoperative course was complicated by both acute liver injury due to shock liver and acute renal failure requiring renal replacement therapy. Bivalirudin was ultimately chosen for anticoagulation after careful consideration of his multi organ dysfunction. A serotonin release assay was obtained and ultimately positive, confirming the diagnosis of HITT. The patient was eventually weaned off of RRT and transitioned back to anticoagulation with warfarin following the recovery of his thrombocytopenia and was subsequently discharged home on postoperative day 46.

Conclusion: HIT is an immune-mediated phenomenon in which an antibody targets the complex of platelet factor 4 (PF4) and heparin. This results in platelet activation and aggregation and subsequently thrombin generation, which predisposes patients to higher risk of thrombosis rather than bleeding. The platelet-rich clot that develops often appears white on visual inspection, which was the origin of the original moniker of HITT as the 'white clot syndrome'. The diagnosis of HIT involves clinical scoring systems such as the 4T score, and confirmatory laboratory testing with anti-PF4-heparin enzyme immunoassays and functional platelet activation tests. Treatment of HIT involves discontinuation of heparin (and heparin-like medications) and administration of alternative anticoagulants, particularly direct thrombin inhibitors (DTI). The two most commonly used DTI in the United States are argatroban and bivalirudin, although bivalirudin is currently not formally approved specifically for medical treatment of HIT. Argatroban is hepatically metabolized while bivalirudin is renally cleared, which can make choice of anticoagulation difficult in critically ill patients with injury to both organ systems. Although dosing adjustments can be made for either DTI to account for decreased drug elimination, we considered bivalirudin to be a more appealing and safe choice given the initial concern with the severity and acuity of our patient's liver injury. Patients are typically transitioned to long-term anticoagulation with warfarin, although there is growing evidence supporting use of direct oral anticoagulants.

References: Heparin-Induced Thrombocytopenia. N Engl J Med 2015 Jul 16;373(3):252-61 Heparin-Induced Thrombocytopenia and Cardiac Surgery. Semin Thorac Cardiovasc Surg. 2019 Autumn;31(3):335-344. Heparin-induced thrombocytopenia (HIT): Review of incidence, diagnosis, and management. Vasc Med. 2020

Apr;25(2):160-173. Heparin-induced thrombocytopenia: natural history, diagnosis, and management. Vasc Med. 2001;6(2):113-9. When heparin causes thrombosis: significance, recognition, and management of heparin-induced thrombocytopenia in dialysis patients. Semin Dial. Jul-Aug 2006;19(4):297-304





Critical Care - 19 Perioperative Management Of Paraganglioma

Valerie Verdun[1], James A Onigkeit[1]

[1]Mayo Clinic, Rochester, MN

Introduction: Paragangliomas are rare neuroendocrine tumors with a variable presentation. Patients can develop lethal cardiovascular complications and appropriate perioperative care can minimize catecholamine-related adverse events. We present a case of a newly diagnosed paraganglioma, with highly labile hemodynamics both pre- and post-operatively requiring ICU admission for hemodynamic stabilization.

Methods: A 69 year old female with past medical history of breast cancer and type 2 diabetes, presented to the emergency department with abdominal pain. Of note, she had newly diagnosed hypertension and 100 pound unintentional weight loss for the past one year. CT abdomen pelvis showed an 8.7 cm left, periaortic, retroperitoneal paraganglioma. She was hospitalized for hypertensive urgency; however, due to significant nausea, vomiting, she was unable to take oral antihypertensives, resulting in significant hypertension with systolic blood pressure greater than 200 mm Hg. Thus, she was admitted to the ICU for anti-hypertensive infusions. Calcium channel blockade with clevidipine and alpha blockade with phentolamine infusions were initiated. Gradual oral metyrosine daily up-titration was accomplished via gastric tube. After five days of medical optimization, she underwent surgical resection of the paraganglioma. A combined spinal-epidural was placed pre-operatively with intrathecal hydromorphone. Blood pressure was labile throughout the case. Hypertension was treated with clevidipine infusion as well as boluses as needed during surgical manipulation of the tumor. After tumor removal, the patient required phenylephrine, norepinephrine, epinephrine, and vasopressin infusions. Blood loss was 950 mL. She received 2 units of blood and 4.5 L of intravenous crystalloid solution. She remained in the ICU for 3 days post-operatively to wean off vasopressors and was discharged home on post-operative day 6.

Conclusion: The classic presentation of a paraganglioma consists of episodic hypertension, tachycardia, diaphoresis, and headache, but may also present with cardiomyopathy and hypertensive crisis. Mortality in these patients is high (24%), particularly in those with cardiogenic shock.2 Pre-operative management consists of alpha blockade followed by beta blockade and volume expansion to reverse catecholamine-induced blood volume contraction to prevent severe hypotension after tumor removal.4 Metyrosine inhibits the rate-limiting enzyme tyrosine hydroxylase, decreasing catecholamine synthesis by 50-80%. When used in combination with phenoxybenzamine, it facilitates intraoperative hemodynamic management.3 Intraoperative hypertension is best treated with direct vasodilators, Œ≤-adrenergic antagonists, or calcium channel blockers prior to tumor removal, followed by volume resuscitation and vasopressors after tumor removal.1 Ultimately, definitive treatment is with surgical resection of the tumor.

References: 1. Kinney, M. et. al (2002) Journal of Cardiothoracic and Vascular Anesthesia 16:359-369. 2. Sauneuf, B. et al. (2017) Critical Care Medicine 45:657-665. 3. Stoelting's Anesthesia and Co-Existing Disease, 7th Edition: 466. 4. Lenders, J. et. al. (2014) J. Clinical Endocrine Metabolism 2014; 99(6):1915-1942.

Critical Care - 20 Kratom Induced Liver Injury in 37-year-old female: A Case Report

Courtney James[1], Faith N Factora[2], Jaime Freiburger[3], Kyle McGrath[3]

[1]Cleveland Clinic Foundation, Columbus, OH, [2]Cleveland Clinic Foundation, Cleveland, OH, [3]Ohio University Heritage College of Medicine, Warrensville Heights, OH

Introduction: Kratom is an herbal supplement native to Malaysia and Thailand that has become increasingly popular among Americans in the last 5 years. It is now available via online markets and used for its stimulant, pain killing, and sedative properties with varying doses. It has become an increasingly popular choice for patients looking to taper opiate use, for stimulant effects, and as a party drug. While it is loosely known what dosage one can achieve the desired effects, research is limited on how toxic levels of Kratom can affect the body, or the doses at which this occurs. Per case reports in the literature, Kratom in toxic levels can produce a large variety of symptoms including respiratory suppression and pulmonary edema (1,2) seizures, psychosis and hallucinations(3), cardiotoxicity(4), markedly elevated bilirubin with jaundice(5), hepatomegaly(6), nephrotoxicity(7), and fulminant liver failure(8,9,10). In this report, we explore a case of long term Kratom use for stimulant use and pain control, resulting in subacute drug induced liver failure.

Methods: Our case is a 37-year-old female who initially presented to the emergency department with abdominal pain, nausea, and vomiting. Her admission labs were significant for elevated liver enzymes, alkaline phosphatase, bilirubin, BUN, and serum creatinine. This patient had a past medical history of hypertension and opiate dependence secondary to chronic pain. She was also a former smoker. The patient underwent an exploratory laparotomy with subtotal colectomy and end ileostomy for pancolitis with ischemic colon, complicated by acute liver failure. The patient had an extensive and complicated 73-day hospital stay, finally discharged with elevated but stable liver enzymes. At the time, the presentation was consistent with intrahepatic cholestasis or hepatitis, and a history with Kratom supplementation had not been determined. She was discharged home with outpatient monitoring. The patient presented to the emergency department 21 days later with a colocutaneous fistula. Colorectal surgery determined there was no intervention needed for the fistula, but it was noted that her previously stable transaminitis and elevated bilirubin had become acutely worse. This prompted urgent liver transplant consideration. The patient's MELD score was calculated to be 33 and she was listed for liver transplant and approved quickly. Two days after approval, the patient underwent an orthotopic liver transplant given her subacute liver failure secondary to drug-induced Liver Injury (DILI). The etiology of the patient's liver injury was originally unknown and thought to be associated with undiagnosed inflammatory bowel disease, but through history taking it was found that the patient regularly ingested Kratom powder in her tea for the past 2 years. Given the systemic effects, the variety of toxicities reported in the literature, DILI via Kratom supplementation was diagnosed by exclusion. The patient stopped ingesting Kratom during her first hospital admission, but given the lack of suspicion for Kratom toxicity during that stay, blood samples for Kratom metabolites were not obtained. After transplant, liver function tests and overall liver function continued to improve significantly over the course of several weeks. The ex-planted liver biopsy was consistent with DILI, and the liver transplant did not show evidence of rejection. She ultimately discharged to a rehabilitation facility.

Conclusion: Kratom is an herbal substance which poses a threat to individuals seeking over the counter remedies for pain, lethargy, and mental illness. These pharmacological effects can cause dependence and withdrawal in cases of chronic use. As a commercially available supplement, it is imperative that clinicians become familiar with Kratom use. Significant questions still have yet to be answered in regard to Kratom supplementation including the toxic and lethal doses of Kratom in humans, the dosage at which organs begin to experience tissue damage, and how chronic use changes each of these variables. From case reports, Kratom induced cholestasis can be self-limiting and patients can go on to have a full-recovery, although some patients, like the present case, may require a liver transplant(5). This case demonstrates the unique clinical presentation seen in long term Kratom use,

further emphasizing the need for research and regulation on Kratom supplementation.

References: 1. Fatality of 33-Year-Old Man Involving Kratom Toxicity, Vol. 64, No. 6, 1933-1936, 2019. 2. Deaths in Colorado Attributed to Kratom, 380:1, 97-98, 2019. 3. Characteristics of deaths associated with kratom use, 33:9, 1102-1123, 2019 4. Intrahepatic Cholestasis Following Abuse of Powdered Kratom (Mitragyna speciosa), 7, 227-231, 2011. 5. Clinical outcomes after Kratom exposures: A poison center case series Kratom, 36, 134-168, 2018. 6. Possible kratom-induced hepatomegaly: A case report, 58, 561-563, 2018. 7. The pharmacology and toxicology of kratom: from traditional herb to drug of abuse, 130, 127-138, 2016. 8. Characteristics of deaths associated with kratom use, 33:9, 1102-1123, 2019. 9. Cholestatic Hepatitis From Prolonged Kratom Use: A Case Report, 61:3, 1086-1087, 2015. 10. Histologic Characterization of Kratom Use-Associated Liver Injury, 11:1, 79-82, 2018.

Figure 1: Kratom plant

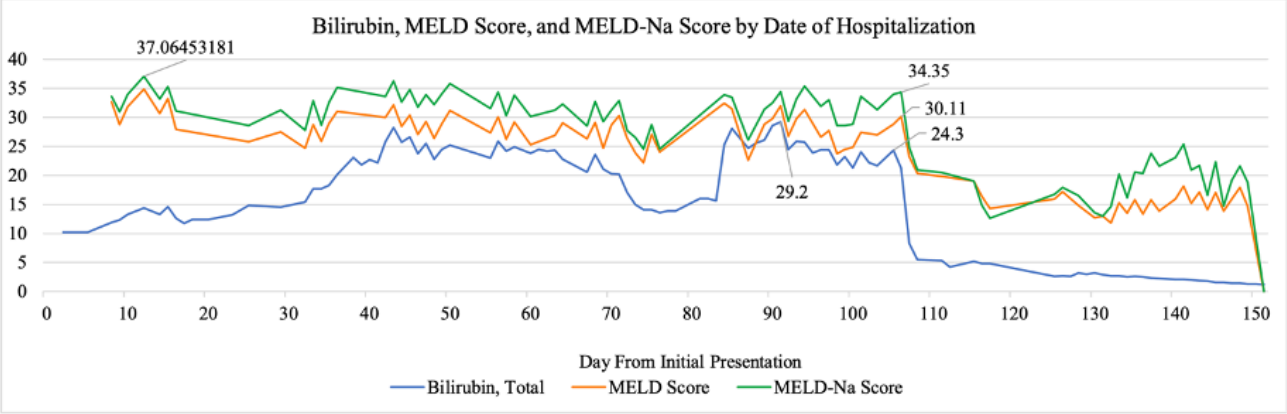
Table 1: Significant Values for Patient Throughout Hospitalization

Table 1. Laboratory Values at the Time of the First and Second Episodes of Jaundice + Post Transplant

		Laboratory Test					
Preexposure		ALT (U/L) value	ALP (U/L) value	Bilirubin (mg/dL) value	INR value	Creatinine value	Comment Routine Testing
First hospitalization (day 0)	Jun-20	325	608	10.2	2.6	4.61	Initial presentation with subsequent colectomy
Second hospitalization (day 99)	Sep-20	61	1001	28.1	1.7	3.26	Second presentation for rectal stump blowout
Immediately Post Liver Transplant (day 110)	Oct-20	338	190	5.5	1.5	1.97	Post liver transplant
1-month Post Liver Transplant (day 130)	Oct-20	6	184	1.2	1	2.51	
Normal Values		17-63	42.113	0.3-1.1	<1.2	0.84-1.21	

ALT = Alanine Transaminase
ALP = Alkaline Phosphatase
INR = International Normalized Ratio

Figure 2: Bilirubin, MELD Score, and MELD-Na Score by Date from Original Hospital Presentation



Critical Care - 21 Right Ventricular Failure During Venovenous Extracorporeal Membrane Oxygenation for COVID-19 Induced Acute Respiratory Distress Syndrome

Elizabeth Champion[1], Karen Katrivesis[2], Jennifer Elia[3]

[1]UCI, ORANGE, CA, [2]University of California Irvine, Orange, CA, [3]University of California, Irvine, Costa Mesa, CA

Introduction: Right ventricular (RV) failure is a well-documented complication of severe ARDS and results in increased mortality (1, 2). It is unknown if the rate of pulmonary hypertension (HTN), RV failure and acute cor pulmonale are higher in COVID-19 patients with severe ARDS and specifically those on VV-ECMO compared to the general ARDS population. We describe three COVID-19 patients with severe ARDS who developed pulmonary hypertension and right ventricular failure after several days on VV-ECMO. Our aim is to discuss the etiology and alert intensivists caring for this unique patient population to this complication.

Methods: The first patient was a 57-year-old man admitted for COVID-19 pneumonia. He failed all conventional therapies and was placed on VV-ECMO 10 days after intubation. Transthoracic echocardiogram (TTE) the day of cannulation was unremarkable. On ECMO day 16, repeat TTE revealed severely elevated pulmonary artery systolic pressure and severely reduced RV systolic function. CT exam ruled out PE. The patient was treated with inhaled nitric oxide and milrinone, was decannulated on ECMO day 61, and discharged to a long-term acute care facility. The second patient was a 66-year-old man with COVID-19 diagnosed six weeks prior who developed severe ARDS and was placed on VV-ECMO 35 days after intubation. TTE the day of cannulation was unremarkable. On ECMO day 14, repeat TTE revealed severely enlarged RV, severely reduced RV systolic function, moderately dilated right atria and pulmonary HTN. His course was complicated by sepsis, encephalopathy, failure to wean ECMO, and he expired on ECMO day 36. The third patient was a 48-year-old woman admitted for treatment of acute promyelocytic leukemia who developed COVID-19 pneumonia. She was intubated for severe ARDS 12 days after symptoms developed and cannulated for VV ECMO six days later. TTE after cannulation was unremarkable. On ECMO day 23, chest CT ruled out PE but revealed dilated pulmonary trunk suggestive of pulmonary HTN. TTE confirmed severely enlarged RV with severely reduced RV function. RV failure continued to worsen requiring transition to VA-ECMO. She expired on ECMO day 39.

Conclusion: We present three COVID-19 patients with severe ARDS supported on VV-ECMO who had a normal TTE at the time of cannulation but developed pulmonary HTN and RV failure after initiation of VV-ECMO. They all had severe ARDS for weeks prior to onset of RV failure. In addition to ARDS specific causes of acute RV failure, ECMO related causes must also be explored due to the delayed onset. Development of pulmonary HTN and RV failure in patients with severe ARDS is multifactorial and has profound clinical implications. Mechanisms of increased pulmonary vascular resistance and RV stress related to ARDS are well described. Abnormal echocardiography findings typically manifest by 48-72 hours of disease onset in non-ECMO severe ARDS patients (3). The etiology of RV failure several weeks into the disease process is unclear. One possible explanation is VV-ECMO support delays or prolongs the ARDS stages due to ultra-lung rest and improved gas exchange. Another explanation is VV-ECMO itself may cause increased pulmonary vascular resistance eventually causing RV failure. One case series published in 2018 presents three patients who developed acute cor pulmonale weeks after initiation of VV-ECMO for severe ARDS (4). Due to the delayed onset compared to non-ECMO ARDS patients, the authors highlight potential VV-ECMO related causes including non-physiologic flow across the tricuspid valve which may increase PVR and cause RV failure. In 2015, a similar case report was published describing RV failure during VV-ECMO for H1N1 induced ARDS (5). Delayed onset of RV failure was noted on day 20 of ECMO. They proposed non-pulsatile ECMO flow sustained RV overloading despite normal RV contraction, inducing decreased RV recoil function. In addition to the non-physiologic flow mechanism, the possibility that the circuit components induce a systemic inflammatory reaction, hemolysis, bleeding, or thromboembolism resulting in pulmonary artery vasoconstriction. Our case series highlights a

potentially fatal complication associated with COVID-19 induced ARDS patients placed on VV-ECMO. Further data and studies are needed to describe the exact mechanism and help guide prevention strategies.

References: References 1) Guérin, C. and M.A. Matthay, Acute cor pulmonale and the acute respiratory distress syndrome. Intensive Care Med, 2016: p. 1-3. 2) Repessé, X., C. Charron, and A. Vieillard-Baron, Acute respiratory distress syndrome: the heart side of the moon. Curr Opin Crit Care, 2016. 22(1): p. 38-44. 3) Repessé X, Charron C, Vieillard-Baron A. Acute cor pulmonale in ARDS: rationale for protecting the right ventricle. Chest 2015.147: 25926 4) Dong, Erik R, Ng, David G, Ramzy, Danny, Chung, Joshua S, et al. Acute cor-pulmonale in veno-venous extracorporeal membrane oxygenation: Three Case Reports. ASAIO Journal, 2018 5) Lee S, Jung J, Chung J, et al. Right heart failure during veno-venous extracorporeal membrane oxygenation for H1N1 induced acute respiratory distress syndrome. Korean J Thorac Cardiovasc Surg 2015; 48(4): 289293.

Critical Care - 22 Severe Hyperammonemia In The Absence Of Liver Disease

Faina Kotova[1], Daniela Lazea[1]

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

Introduction: Medically challenging case. The purpose of the case is to highlight the danger of undiagnosed hyperammonemia in ICU patients, and to discuss the etiology, prevalence, symptoms, diagnosis and treatment of hyperammonemia in critically ill.

Methods: Hyperammonemia caused by disorders of the urea cycle is a rare cause of metabolic encephalopathy that may be underdiagnosed by intensivists because of its rarity(1). Timely diagnosis as well as immediate initiation of aggressive treatment are crucial for preservation of patients' neurological function and survival. This case highlights the danger of late diagnosis of hyperammonemia in the ICU in a patient admitted with an unrelated surgical pathology. A 72 year old man with past medical history of hypertension, hyperlipidemia and worsening paroxysmal atrial fibrillation due to long-standing mitral regurgitation presented for an elective mitral valve repair. His surgery was technically complicated, and a mitral valve replacement was performed instead. The patient was transferred to the ICU in stable condition. Upon awakening trial he was noted to have no response to painful stimuli despite sufficient time and administration of naloxone. CT head was performed and revealed no acute intracranial process. The patient started to slowly regain neurological function and was able to get extubated. MRI of the brain on post-operative day 3 revealed multiple scattered watershed infarcts that were thought to be causing the neurological symptoms. The patient has slowly recovered his neurological function, however developed post-operative pericardial effusion and a urinary tract infection that kept him in the ICU. He was improving and nearing discharge from the intensive care unit. On post-operative day 12 the patient was suddenly found unresponsive with shallow breathing. He was emergently intubated and head imaging was obtained, however it failed to reveal a new intracranial process. His EEG showed focal status epilepticus, and among his laboratory examinations a very high level of ammonia (>600 mmol/L) was noted. Remarkably, this elevation manifested in the absence of liver disease. Emergent continuous renal replacement therapy was initiated in an attempt to clear ammonia, however it kept rising and peaked at 1,000 mmol/L. The patient eventually developed persistent status epilepticus, severe metabolic acidosis and succumbed to the illness. As a part of clinical investigation, genetic workup was done and revealed that the patient was a carrier of two autosomal recessive illnesses that can lead to hyperammonemia. Hyperammonemia in the absence of liver disease is uncommon, but may carry disastrous consequences for patients. Of particular concern are unsuspected inherited defects of the urea cycle and fatty acid oxidation presenting with catastrophic illness in previously normal individuals. Early identification of the problem is the challenge (2). Patient survival depends on immediate treatment of intracerebral hypertension and reduction of ammonia levels. Lactulose, a mainstay of treatment in chronic hyperammonemia, has not been shown to affect mortality in acute cases. Hemodialysis and continuous renal replacement therapy can remove ammonia rapidly.

Conclusion: A variety of genetic and environmental triggers can precipitate acute late onset hyperammonemia in adults. In critically ill population the picture is often clouded by patient acuity and disease burden, thus making hyperammonemia unlikely to be diagnosed on time. This case highlights the importance of rapid diagnosis and aggressive treatment to avoid devastating consequences. Intensivists should be familiar with pathophysiology of diseases of the urea cycle, as well as clinical signs and best treatment modalities for acute hyperammonemia in the ICU population.

References: 1. Redant S, Empain A, Mugisha A, Kamgang P, Attou R, Honor√© PM, De Bels D. Management of late onset urea cycle disorders-a remaining challenge for the intensivist? Ann Intensive Care. 2021 Jan 6;11(1):2. doi: 10.1186/s13613-020-00797-y. PMID: 33409766. 2. Walker V. Severe hyperammonaemia in adults not explained by liver disease. Ann Clin Biochem. 2012 May;49(Pt 3):214-28. doi: 10.1258/acb.2011.011206. Epub 2012 Feb 20. PMID: 22349554.

Critical Care - 23 Sudden Carbon Dioxide Embolism during Diagnostic Laparoscopy

Shara S Azad[1], John Fox[2], Pavan Sekhar[1], Claudia L Sotillo[3]

[1]Tufts Medical Center, Boston, MA, [2]Tufts Medical Center, Boston, United States of America, [3]Brigham & Women's Hospital, Boston, MA

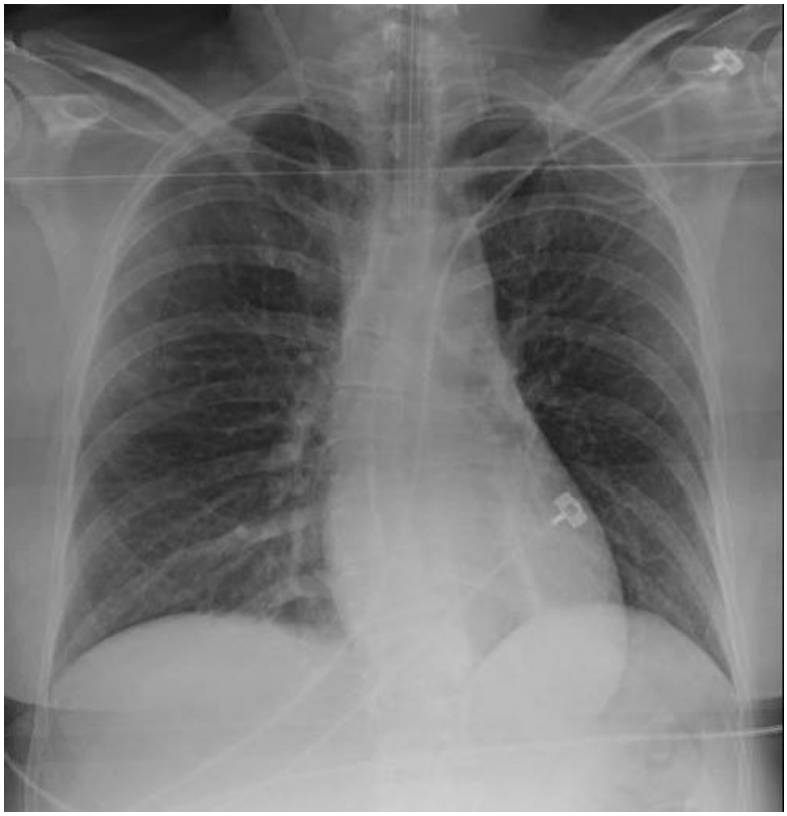
Introduction: The incidence of venous air embolism (VAE) during laparoscopic procedures is between 11% to 97% based on documentation from case reports (1,2). Subclinical VAE is a more frequent phenomenon, but because of the relative risk, it is well within the realm of possibility that an air embolism causing major hemodynamic shifts may occur during an anesthesiologist's clinical career. In this case report, we document a carbon dioxide embolism leading to major hemodynamic shifts during a routine laparoscopic evaluation of a right endometrioma.

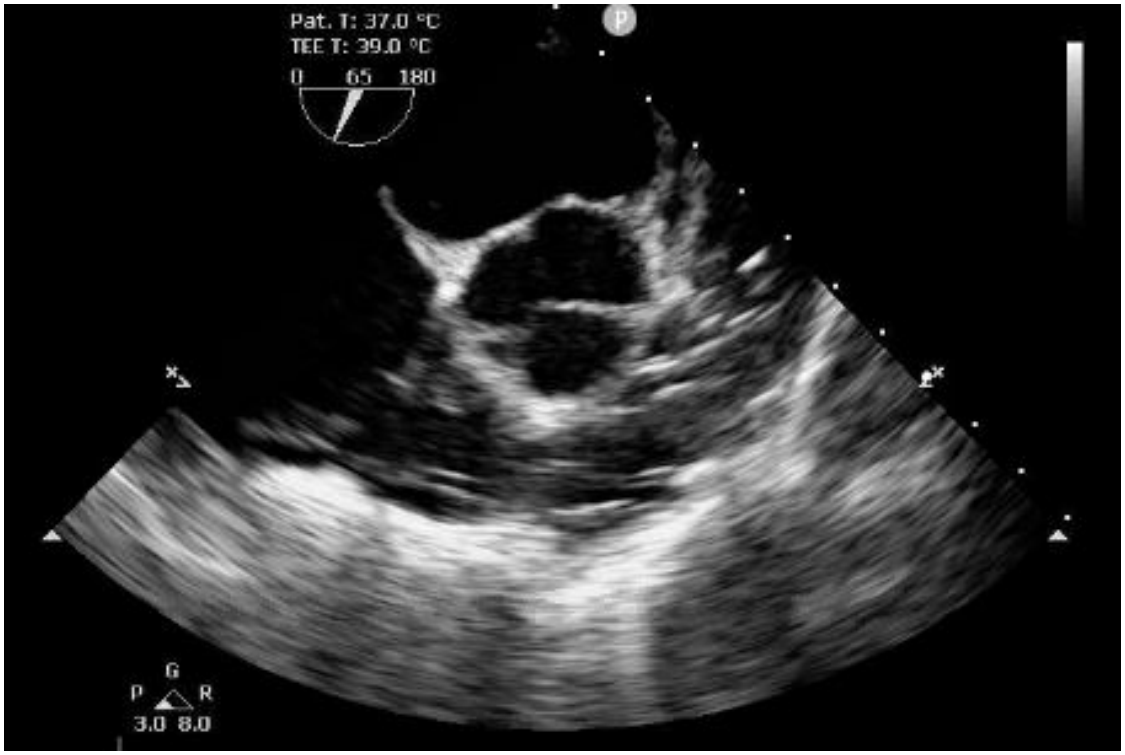
Methods: A 48 year-old, 71.6kg ASA II woman, with past surgical history significant for previous cesarean section and bilateral tubal ligation, presented for an elective salpingo-oophorectomy and diagnostic laparoscopy for a possible right endometrioma. Anesthesia was induced with 2 mg midazolam, 200 mg propofol. 100 mcg fentanyl, and 50 mg of rocuronium, and maintained with a combination of sevoflurane and propofol infusion at 30 mcg/kg/min. After full muscle relaxation was achieved, the patient was intubated easily and without complication. The surgical team attempted to obtain pneumoperitoneum using the Veress needle technique through the umbilicus. Insufflation was initiated but discontinued within seconds as insufflation pressures increased to 12mmhg. The Veress needle was removed and replaced and insufflation attempted again with elevated insufflation pressures. Direct trochar entry was then attempted, however, the attempt was immediately aborted, due to acute decrease in end tidal CO2 from 33mmHg to 8mmHg and profound hypotension, with drop in systolic blood pressure from 100 mmHg to 70mmHg. The hypotension was immediately treated with 10mg of ephedrine and phenylephrine infusion at 100mcg/min. Additional anesthesiology staff was called to the room, including a cardiac anesthesiologist for evaluation of suspected CO2 embolism vs pulmonary embolism. The patient was immediately positioned in a modified Trendelenburg, left lateral recumbent position. A left radial arterial line was placed as a transesophageal echo (TEE) was performed. TEE showed a large volume of gas bubbles (likely carbon dioxide) in the pulmonary artery and right ventricle. Emergent placement of a right internal jugular MAC line was performed as a vehicle for aspiration of gas bubbles as well as manual chest physical therapy/thumping in an attempt to break up CO2 gas through external force. After one hour of the aforementioned interventions, the gas burden in the patient's right ventricle and pulmonary artery were noted to decrease on TEE. Given the patient's overall hemodynamic improvement, but continued presence of right ventricular CO2, she was left intubated and transferred to the intensive care unit and extubated without incident within 6 hours. Subcutaneous emphysema was noted in the left anterior abdominal wall. X-ray imaging and subsequent computed tomography showed locules of gas in the right atrium, pulmonary artery, abdominal cavity and left rectus muscle. This patient was diagnosed with suspected carbon dioxide embolism after unintentional venous puncture during attempt at insufflation.

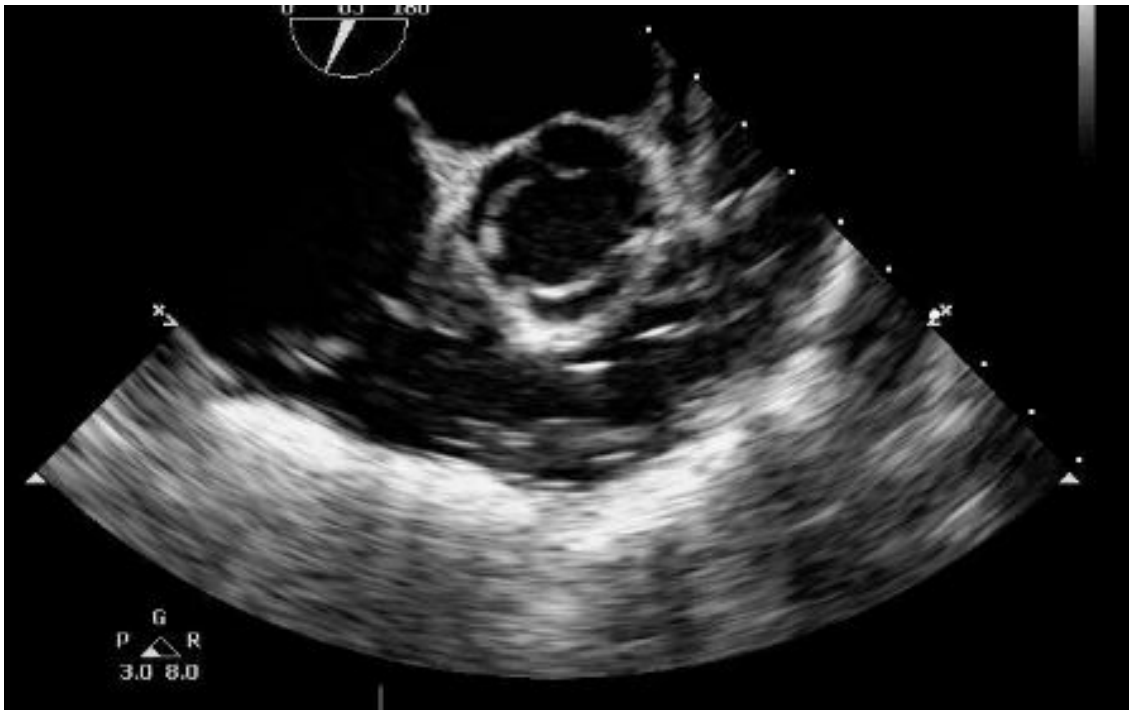
Conclusion: Cases of carbon dioxide embolism during laparoscopic procedures are well-documented, and thus the perioperative anesthesiology team must remain vigilant. While interventions such as central-line aspiration of gas bubbles, chest physical therapy and left-sided table tilt may be beneficial, transesophageal echo remains the most sensitive modality for diagnosis and monitoring gas embolisms (3,4).

References: Shaikh, N., & Ummunisa, F. (2009). Acute management of vascular air embolism. Journal of emergencies, trauma, and shock, 2(3), 180-185. https://doi.org/10.4103/0974-2700.55330 Kim, C., Kim, J., Kwon, J., Choi, S., Na, S., An, J., & Kim, K. (2009). Venous Air Embolism during Total Laparoscopic Hysterectomy. Anesthesiology, 111(1), 50-54. doi:10.1097/aln.0b013e3181a05ac7 Surendranath, V., Radhakrishna, P., & Raghavendran, K. (2020). Early Detection is Vital in Successful Resuscitation after Mixed-Air and Carbon Dioxide Venous Embolism during Laparoscopic Surgery. Journal of Anaesthesia and Critical Care

Case Reports, 6(2), 14-16. McCarthy, C. J., Behravesh, S., Naidu, S. G., & Oklu, R. (2016). Air Embolism: Practical Tips for Prevention and Treatment. Journal of clinical medicine, 5(11), 93. https://doi.org/10.3390/jcm5110093






G
P R
3.0 8.0

Critical Care - 24 What Caused This Rapid Response? Aspiration, Tension Pneumothorax, Large Mucous Plug or All of the Above?

Anesah Elhaddi[1], Mada F Helou[2]

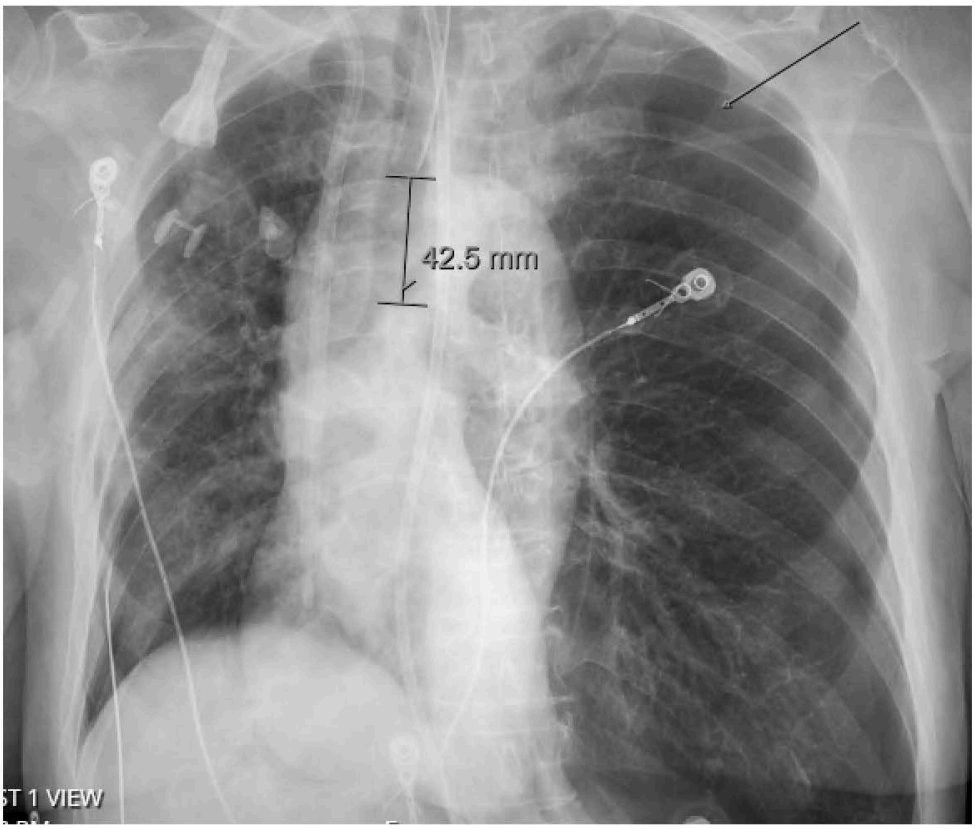
[1]CWRU/University Hospitals, Cleveland, OH, [2]University Hospitals at Case Medical Center, Cleveland, OH

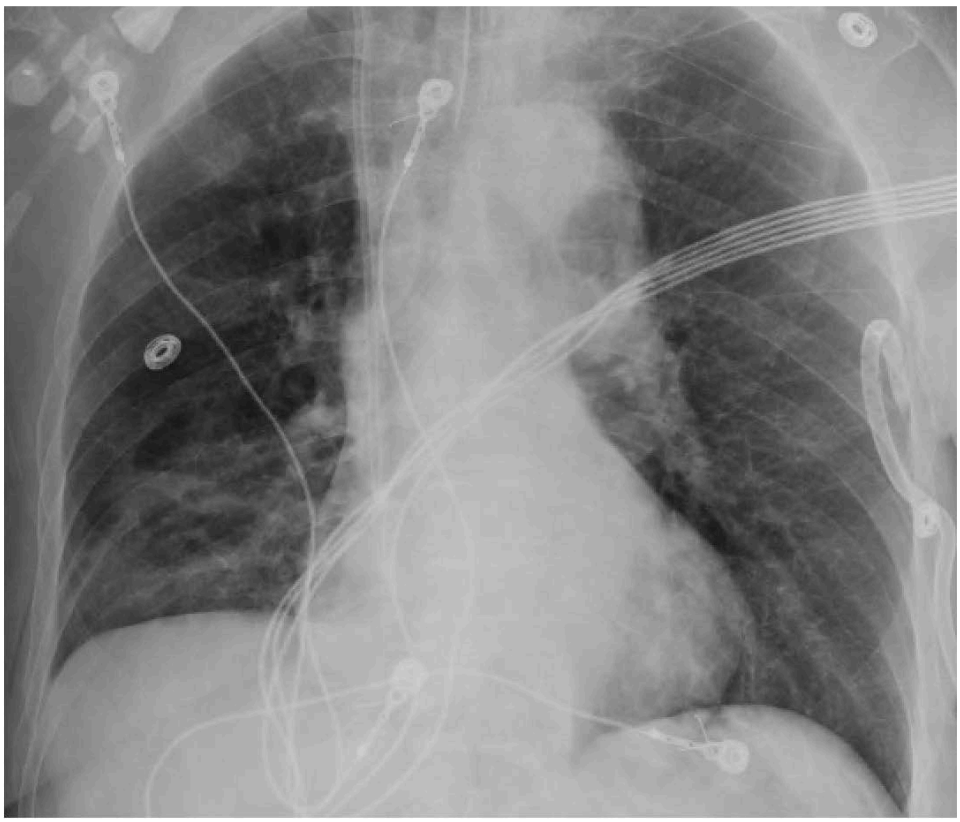
Introduction: Acute respiratory failure is an emergent situation that can lead to hypoxia and subsequent cardiac arrest.¬π It accounts for 10% of all ICU admissions.¬π The purpose of this case report is to highlight the importance of the immediate recognition of the underlying cause of acute respiratory failure in patients with altered mental status. The use of multiple diagnostic and therapeutic techniques, timely diagnosis and appropriate management are all modalities crucial for successful patient outcomes within the critical care setting.

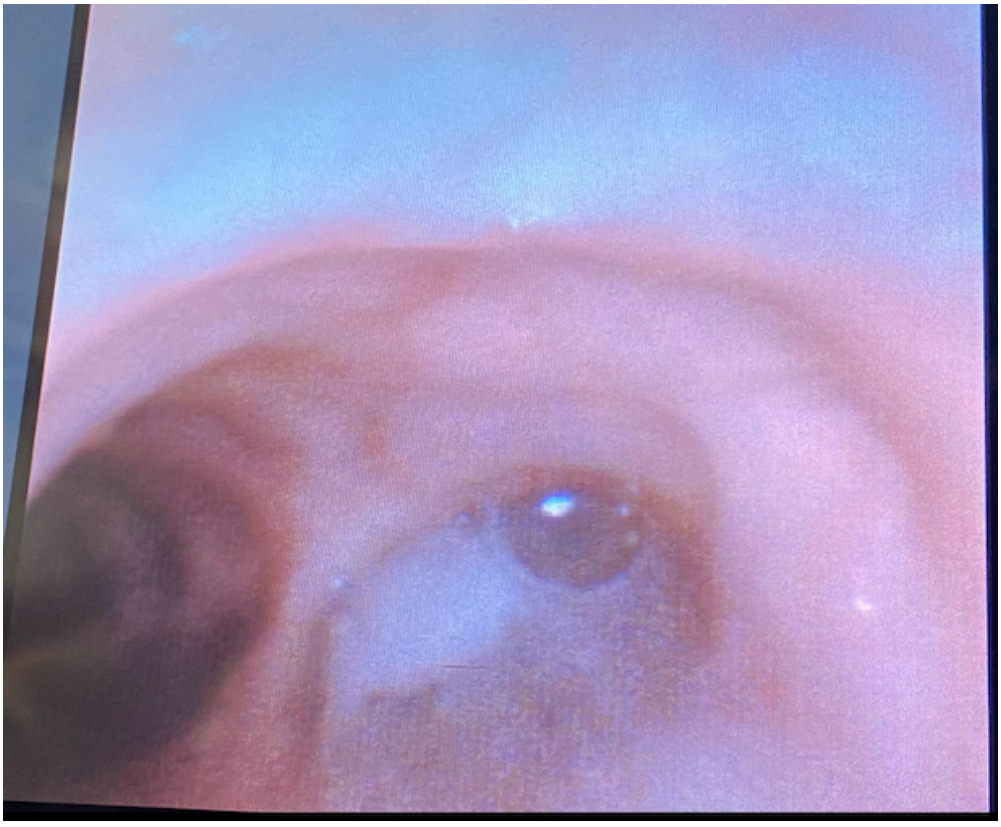
Methods: We report a 75 year-old recently COVID recovered female with multifactorial encephalopathy, seizures and altered mental status in acute hypoxic respiratory failure. A rapid response was called and successful emergent endotracheal intubation on 100% FiO2 with appropriate ventilation settings ensued. The patient briefly improved and aspiration was ruled out. After transferring to the ICU, peak airway pressures were elevated and she rapidly became hemodynamically unstable; asymmetric chest rise was observed despite bilateral breath sounds. CXR identified a left sided tension pneumothorax with mediastinal shift. Small right lung volumes were observed, however this was present on admission. A left pigtail catheter was placed with immediate improvement in oxygenation and hemodynamics. However, this was transient and in ten minutes the patient again deteriorated hemodynamically with low oxygen saturations and high peak airway pressures despite resolution of the pneumothorax. Emergent bronchoscopy showed diffuse, thick mucous plugging in the right upper, middle and lower bronchioles. Upon adequate suctioning and retrieval of the mucous plugs, oxygenation and hemodynamics stabilized thereafter.

Conclusion: In this critically ill patient, the differential diagnosis continuously evolved as the patient displayed transient improvements and multiple decompensations. It is important to evaluate recurrent respiratory and cardiovascular collapse by employing timely diagnostics techniques and implementing emergent interventions. Identifying a tension pneumothorax with immediate decompression of the pleura by either needle decompression or a thoracostomy tube, is critical and life-saving.¬≤ In retrospect, initial hypoxic respiratory failure in this case was likely due to mucous plugging of the right lung due to the chronicity of this patient's clinical history. Subsequent manual ventilation with elevated peak pressures by bag-valve likely led to rupture of a bleb on the left lung, thereby leading to a pneumothorax. Successful bronchoscopic suctioning of the diffuse airway obstruction led to improvement in the patient's clinical status. In summary, evidence of multifactorial causes of respiratory failure are uncommon but may occur. This highlights the important role of expert opinions to assess the underlying causes and initiate complex diagnostic and therapeutic modalities to achieve successful patient outcomes.¬≥

References: 1. Chest, volume 152, 867-879, 2017 2. Annals of Surgery, volume 261, 1068-78, 2015 3. Resuscitation, volume 198, 191-197, 2018


42.5 mm
T 1 VIEW





Critical Care - 25 Extracorporeal Ventricular Assist Device (EcVAD) for Refractory Cardiogenic Shock

Se Fum Wong[1], Tao Shen[2], albert yen[3], Gregory Peterfreund[3], Anahat Dhillon[4], Raymond Lee[1]

[1]University of Southern California, Los Angeles, United States of America, [2]Cedars-Sinai Medical Center, Los Angeles, CA, [3]University of Southern California, Los Angeles, CA, [4]N/A, Los Angeles, CA

Introduction: Temporary mechanical circulatory support restores perfusion in refractory cardiogenic shock to reverse and prevent multi-organ dysfunction. Peripheral extracorporeal membrane oxygenation (ECMO) has been the principal modality of cardiopulmonary support due to its relative ease in bedside implementation. However, unfavorable hemodynamic alterations such as increased myocardial afterload can increase myocardial oxygen consumption, especially when the left ventricle is not decompressed. The combination of additional support modalities including percutaneous and extracorporeal ventricular assist devices (VAD) facilitate left ventricular unloading to mitigate the undesired hemodynamic compromise from increased left ventricular afterload with ECMO. The purpose of this report is to review an alternative strategy using a minimally invasive apicoaxillary external VAD as a short-term MCS device.

Methods: A 44 year old male with ischemic cardiomyopathy EF <10% presented with decompensated heart failure INTERMACS I intubated on multiple inotropic infusions, continuous renal replacement therapy and femoral IABP support. A recent angiogram demonstrated patent coronary stents. He underwent a right axillary cut down for placement of an Impella 5.5 device, removal of the IABP and peripheral ECMO cannulation for ECPELLA configuration. His hemodynamics improved in the following week such that he was decannulated from ECMO and maintained on full Impella support. However, he was unable to wean from inotropic support with worsening renal function, moderate RV dysfunction, persistent LV dilation with poor function, and severe mitral regurgitation. He subsequently underwent a left mini-thoracotomy for an ECMO external LVAD (Ec-VAD) configuration, femoral venous and left ventricular apex drainage and right axillary artery outflow, in an effort to unload the LV and recover organ function while avoiding sternotomy. The patient remained extubated and hemodynamically stable with minimal inotropic requirements on Ec-VAD support and was able to actively engage in physical therapy until he received a combined heart-kidney transplant three weeks later. After an extended ICU admission, the patient recovered well and was discharged home.

Conclusion: Advantages of LV venting include reducing hydrostatic pulmonary edema, lowering LV distension and filling pressure and preventing stagnation of blood in the hypokinetic LV. The ECPELLA dual MCS system creates a favorable hemodynamic profile for myocardial recovery by decreasing CVP, PAP and inotropic medications required which in turn facilitate ECMO weaning and bridging to durable therapies. A secondary device however increases risk of hemolysis, and in limited studies the ECPELLA strategy is associated with higher need for transfusion and acute kidney injury. The Ec-VAD configuration employs a single device that allows for biventricular support with partial volume unloading of the RV with the venous limb cannula and direct unloading of the LV with the apical cannula and return of flow via an axillary artery graft. This strategy is non-inferior to conventional BiVAD via sternotomy approach by providing similar flow support with perfusion and hemodynamic improvement while sparing the sternum for a potential transplant candidate. Additionally, the Ec-VAD can be easily converted to an external VAD by eliminating the venous limb when the patient's condition has stabilized which enhances patient mobility to participate in physical therapy and rehabilitation. In conclusion, appropriate selection of MCS strategies is an individualized process which optimizes organ recovery in refractory cardiogenic shock and facilitates transition to destination therapy.

References: HT Massey et al. Temporary support strategies for cardiogenic shock: extracorporeal membrane oxygenation, percutaneous ventricular assist devices and surgically placed extracorporeal ventricular assist devices. Ann Cardiothorac Surg. 2019. 8(1): 32-43. ER Silvestri et al. Impella to

unload the left ventricle in patients undergoing venoarterial extracorporeal membrane oxygenation for cardiogenic shock: a systemic review and meta-analysis. J Card Surg. 2020. 35: 1237-42. K Takeda et al. Novel minimally invasive surgical approach using an external ventricular assist device and extracorporeal membrane oxygenation in refractory cardiogenic shock. Eur J Cardiothorac Surg. 2017. 51: 591-6. K Takeda et al. Minimally invasive centrimag ventricular assist device support integrated with extracorporeal membrane oxygenation in cardiogenic shock patients: a comparison with conventional centrimag biventricular support configuration. Eur J Cardiothorac Surg. 2017. 52: 1055-61.

Critical Care - 26 Extracorporeal Membrane Oxygenation in a Patient with Bronchopleural Fistula and Concurrent ARDS: A Tale of Two Lungs

Fernando R Santos[1], Jennifer Elia[2], Sonali Rao[3]

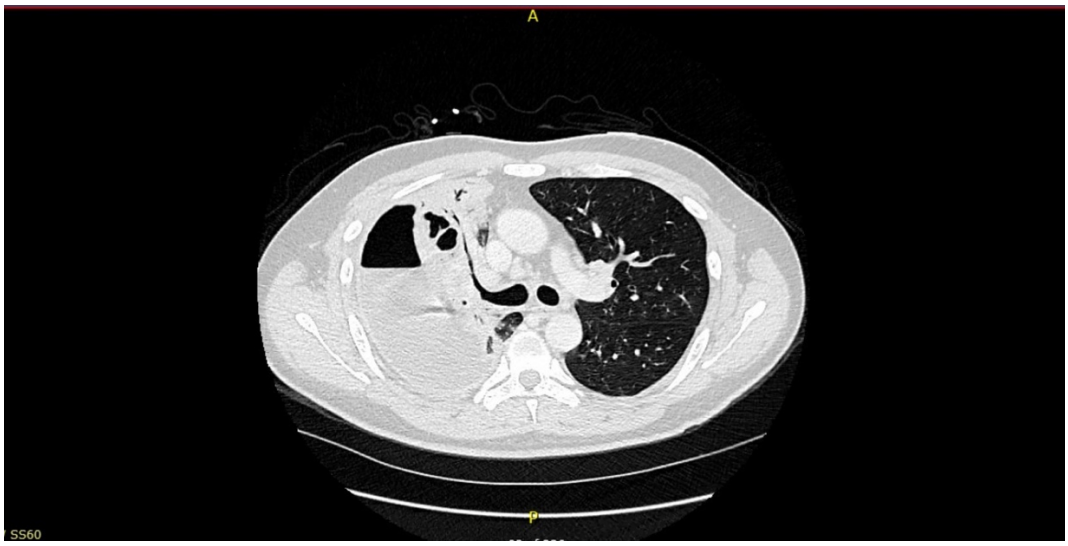
[1]University of California Irvine, Orange, CA, [2]University of California, Irvine, Costa Mesa, CA, [3]UC-Irvine, orange, United States of America

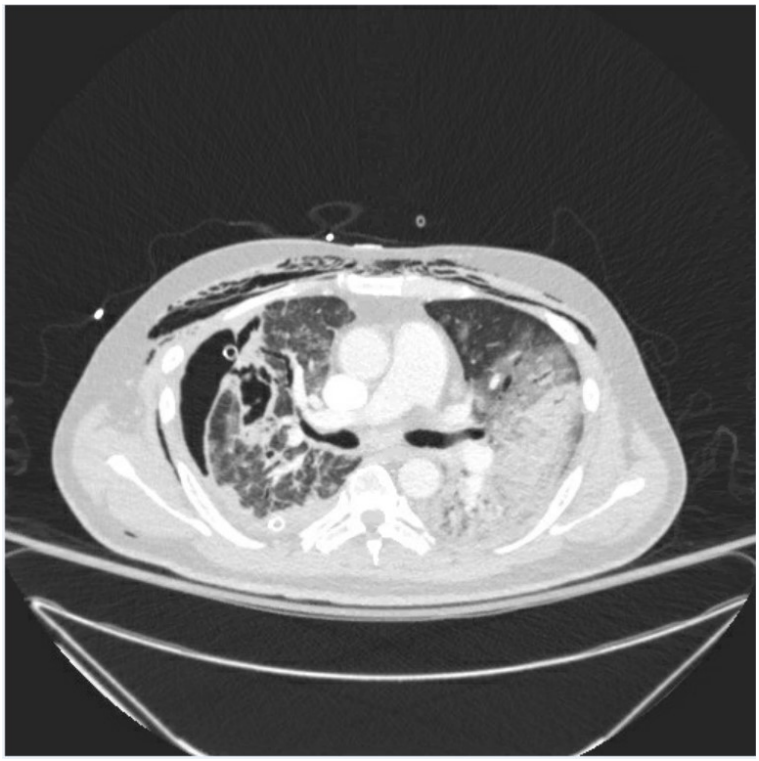
Introduction: Bronchopleural fistula (BPF) is a potentially fatal complication that can occur after lung surgery, but also may result from infection, malignancy, chemotherapy, and radiation (1,2). A variety of surgical, endoscopic, and medical treatments are used for BPF since they rarely close spontaneously (1,3). In severe cases, extracorporeal membrane oxygenation (ECMO) can provide a therapeutic bridge to allow bronchial fistula healing (4). We present a patient with refractory BPF and concurrent adult respiratory distress syndrome (ARDS) of the unaffected lung, posing a significant challenge due to competing management strategies .

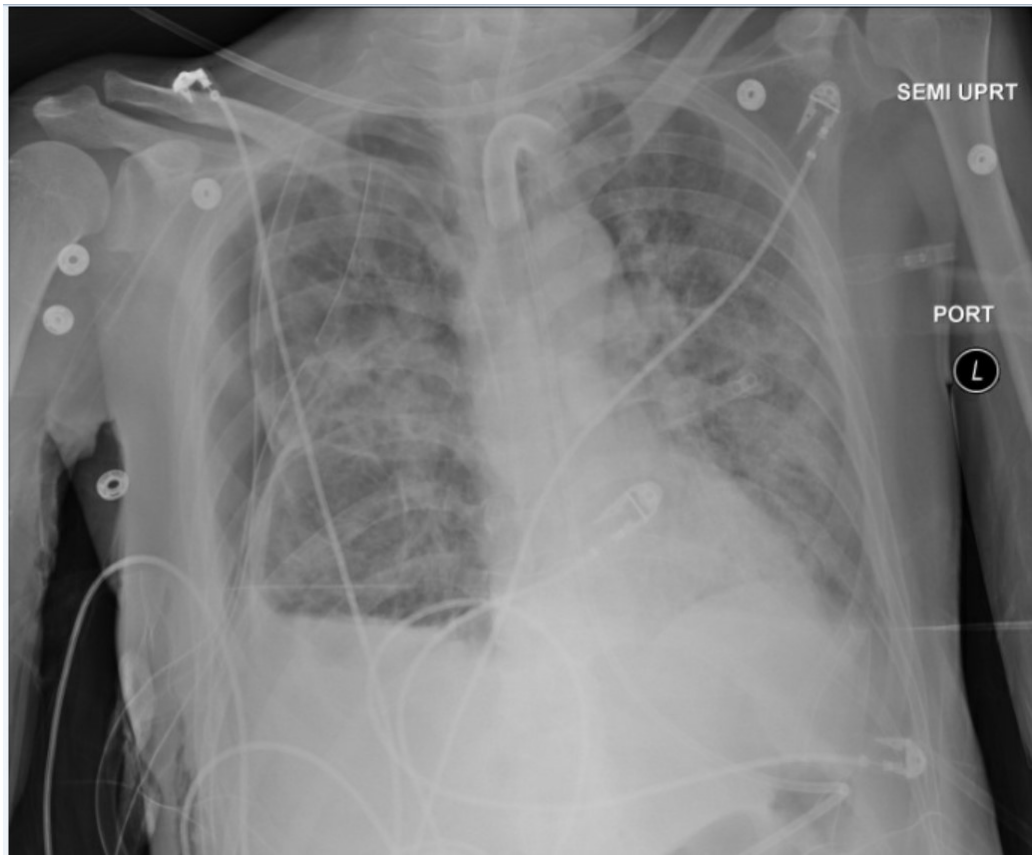
Methods: A 43 year-old male with no past medical history presented with right-sided pleuritic chest pain, shortness of breath, and cough. CT scan revealed right upper lobe (RUL) and right middle lobe pneumonia with cavitary lesion and concern for BPF (Fig. 1). He subsequently developed septic shock and hypoxic respiratory failure, requiring emergent video-assisted thoracopic surgery (VATS). Post-operatively, his respiratory status worsened due to large air leaks despite multiple chest tubes, concerning for persistent BPF. Imaging also revealed developing ARDS of the left lung (Fig. 2). He required increasing ventilator support, and inhaled nitric oxide. Lung protective ventilation and paralysis was attempted to aid in BPF healing, but resulted in de-recruitment and worsening of his ARDS. Lung isolation was contraindicated due to ARDS of the left lung. On hospital day 5, he was placed on veno-venous (VV) ECMO to rest his left lung and allow further intervention of the BPF. Bronchoscopy revealed multiple BPF in the RUL so oxidized cellulose polymer hemostatic agents were used two separate times to pack all RUL segments. This significantly improved his air leak, allowed his right lung to expand, and improved his left lung ARDS (Fig. 3). This facilitated ventilator weaning and he was decannulated on ECMO day 12. Given how critically ill he was, surgery was deferred and conservative management was continued. Unfortunately, due to continued need for mechanical ventilation, his BPF recurred and he again developed hypoxic respiratory failure with left-sided ARDS. He was re-cannulated on VV-ECMO just a week after being weaned. This forced the team to pursue surgical correction of his BPF while on ECMO. He underwent a right thoracotomy with right upper lobectomy. His perioperative course was complicated by coagulopathy and hemorrhagic shock, requiring massive transfusion and several surgical re-explorations, however, his air leak significantly improved. After nearly a month of ECMO support, his ARDS resolved and the patient was successfully decannulated (Fig. 4). He was weaned from all ventilator support weeks later and discharged to a long-term care facility.

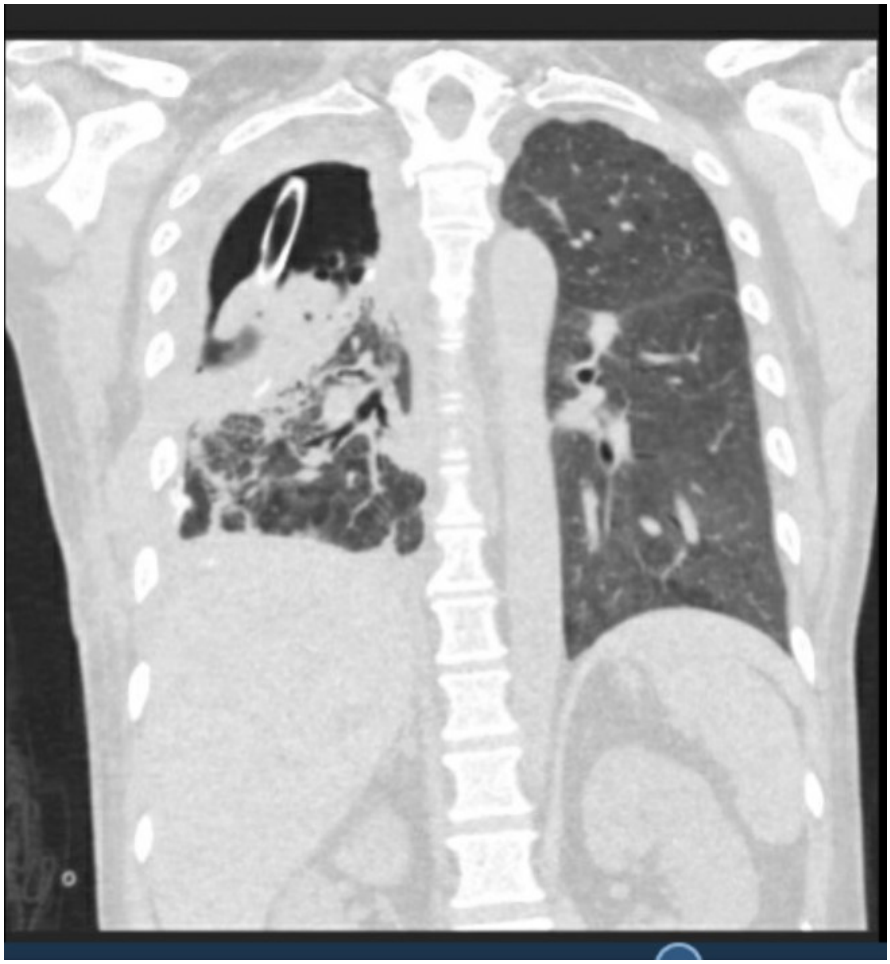
Conclusion: To the authors' knowledge, this is one of the few patients to have required multiple ECMO runs for BPF and survived. BPF alone is associated with significant morbidity and mortality that is further exacerbated for patients requiring ECMO support (1,5). The management of BPF with contralateral ARDS raises several management challenges. After the initial phases of lung rest, higher ventilation pressures are necessary for lung recruitment but detrimental for fistula healing and adequate recruitment is prevented by the large air leak (3). Furthermore, contralateral ARDS is a contraindication for lung isolation, a frequently utilized technique to manage BPF (1,3). Endoscopic interventions are indicated for infectious causes or patients deemed too high-risk for surgery (1). Should these fail, surgical correction can be attempted (2). Patients undergoing surgery while on ECMO, however, have a significantly increased perioperative risk (5,6). Our case highlights the challenges associated with treatment of BPF, even when ECMO is utilized. The presence of ARDS of the unaffected lung made it difficult to treat the BPF with standard management strategies, necessitating a high-risk surgical procedure. Further cases should be identified to help guide management of these complex clinical scenarios.

References: 1. Bronchopleural fistula: an update for intensivists. 2010. 25(1):47-55. 2. Risk factor of bronchopleural fistula after general thoracic surgery: review article. 2017; 65(12):679-685. 3.Management of pneumothorax and prolonged air leak. 2014; 35(6):706-14. 4. Refractory traumatic bronchopleural fistula: Is extracorporeal membrane oxygenation the new gold standard? 2020;35(1):242-5. 5.Outcomes of Multiple Runs of Extracorporeal Membrane Oxygenation: An analysis of the Extracorporeal Life Support Registry. 2020. doi: 10.1177/0885066620981903. 6. Perioperative Management of the Adult Patient on Venovenous Extracorporeal Membrane Oxygenation Requiring Noncardiac Surgery. 2018; 128(1):181-201






SEMI UPRT
PORT
L



Critical Care - 27 A case of bilateral forearm amputation resulting from DIC with purpura fulminans following subtotal colectomy for perforated ulcerative colitis

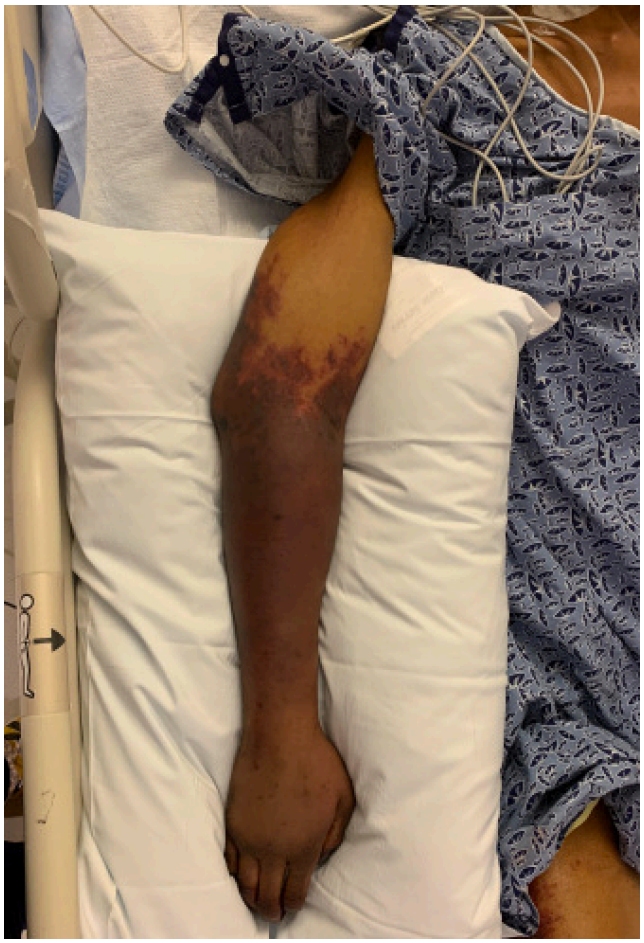
Jialing Zhu[1], Ala Nozari[2]

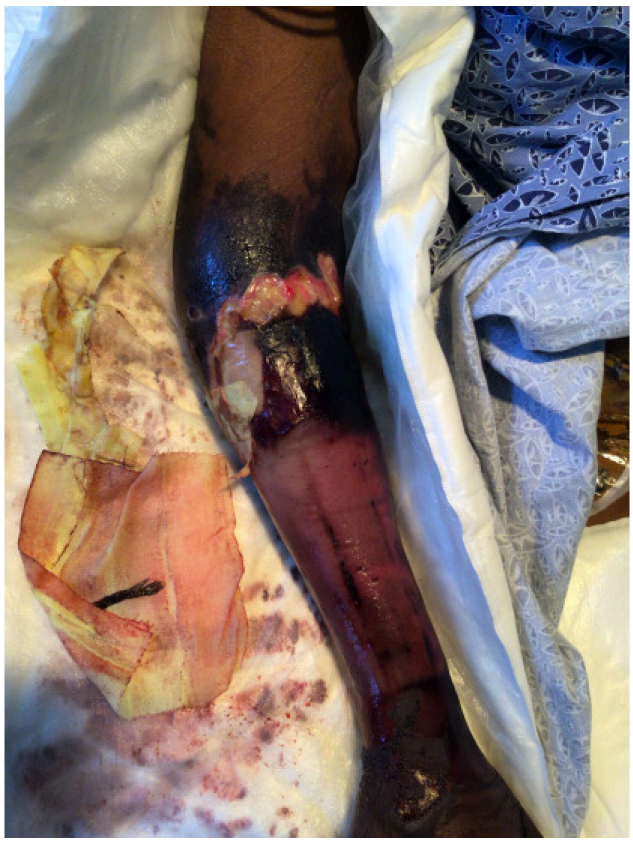
[1]Boston Medical Center, Boston, MA, [2]Boston University School of Medicine, Boston, MA

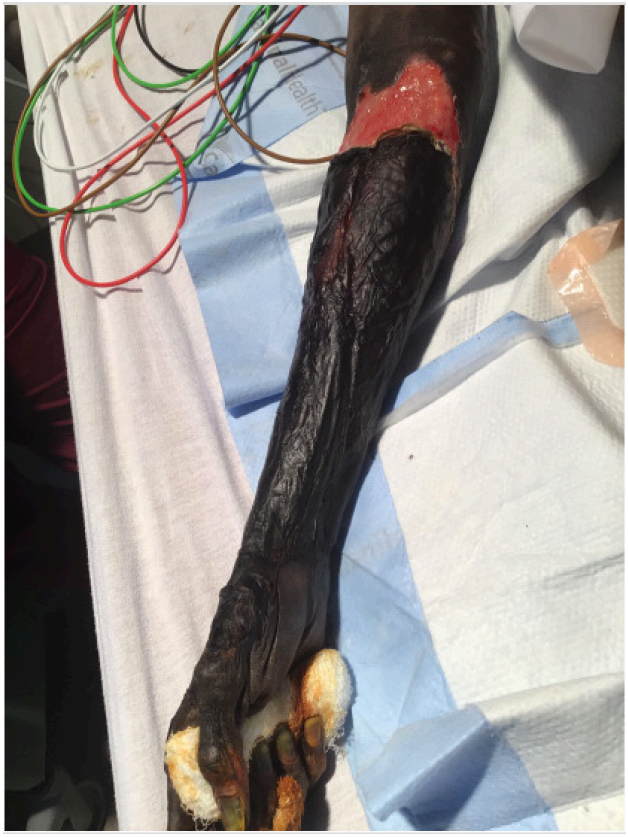
Introduction: Purpura fulminans is a rare complication of severe sepsis. Few studies have reported on the coagulopathy and limb ischemia associated with acute purpura fulminans. The aim of this case report is to highlight the clinical presentation and management of this devastating complication.

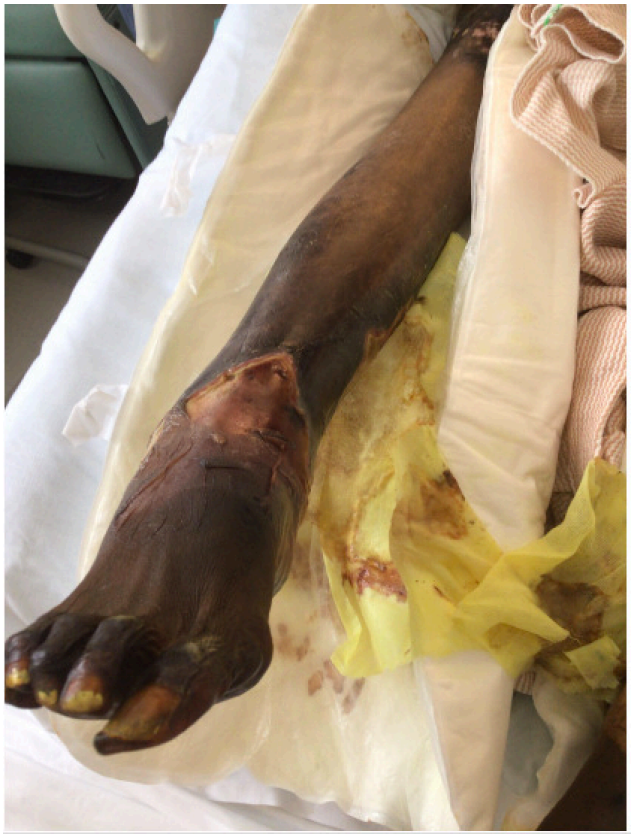
Methods: A 49-year-old female was admitted due to hematochezia. She underwent a colonoscopy with diagnostic findings of ulcerative colitis and was discharged to her home with prednisone taper for four weeks. The following week she presented with a transverse colonic perforation, requiring subtotal colectomy, abdominal washout and ileostomy. Her postoperative course was complicated by polymicrobial intra-abdominal infection causing septic shock. Treatment with broad-spectrum antibiotic and vasopressor agents was initiated. On post operative day 3, patient developed non-blanching, angulated, purpuric smooth skin patches and thin plaques with areas of hemorrhagic bullae over her distal extremities, sparing the trunk. Over the next 24 hours she developed dry gangrene of bilateral upper and lower extremities. Laboratory results revealed significant thrombocytopenia and elevated PT, PTT and INR, as well as elevated D-dimer and decreased fibrinogen, consistent with disseminated intravascular coagulation. Skin biopsy revealed thrombotic vasculitis and findings consistent with purpura fulminans. Bilateral upper extremities doppler ultrasound showed deep vein thrombosis. Patient received platelet transfusion when platelet count decreased to 18. Anticoagulation with heparin infusion was initiated as thrombocytopenia started to resolve. Treatment with fresh frozen plasma was also started. Due to the short half-life of protein C in the plasma, two to three units of fresh frozen plasma approximately every six hours was recommended. However, due to patient's stress cardiomyopathy with significant reduced ejection fraction and worsening pulmonary edema, administration of large volume of fresh frozen plasma was not feasible. Dry gangrene of bilateral upper extremities continued progress despite resolution of her septic shock. Patient's hemodynamic and respiratory status gradually improved and she underwent bilateral forearm amputations 72 days after her subtotal colectomy.

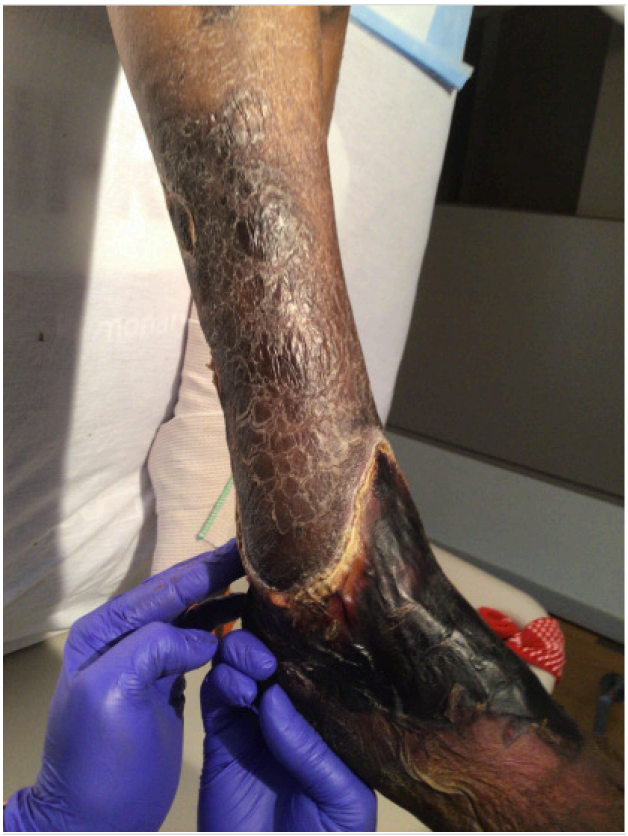
Conclusion: Purpura fulminans is a rare but disabling complication of severe sepsis and can be associated with tissue necrosis, DIC with small vessel thrombosis and limb ischemia. Supportive care and treatment of underlying infection and coagulopathy and novel therapies including administration of protein C concentrate and plasma exchange can decrease the mortality and overall outcome.











Critical Care - 28 Veno-venous extracorporeal membranous oxygenation for the treatment of near-fatal asthma

Kenji Tanabe[1], Mark Robitaille[2], Martin Krause[3], Samuel Gilliland[3], Eric R Leiendecker[4], Breandan Sullivan[5]

[1]University of Colorado, Denver, CO, [2]Beth Israel Deaconess Medical Center, Brookline, MA, MA, [3]University of Colorado, Aurora, CO, [4]University of Wisconsin Hospital and Clinics, Madison, WI, [5]University of Colorado School of Medicine, Aurora, CO

Introduction: The World Health Organization estimates that 250,000 people die prematurely each year from asthma. We present a case of a 25-year-old female with poorly controlled asthma who failed conventional treatment and required veno-venous extracorporeal membranous oxygenation (VV-ECMO) for severe respiratory failure secondary to a near-fatal asthma exacerbation.

Methods: A 25-year-old woman with poorly controlled asthma presented with shortness of breath and hypoxemia to the emergency department. She was diagnosed with a severe asthma exacerbation. Initially, albuterol nebulizers and intravenous magnesium were given and non-invasive ventilation was initiated, but the patient rapidly deteriorated and required intubation, paralysis, deep sedation, and ketamine. Despite maximal asthma-directed therapy, the patient's condition worsened overnight. She developed severe bronchospasm and progressed to severe hypercapnic respiratory failure (PaCO2 >100 mmHg) despite driving pressures greater than 20 cm H2O. Cardiothoracic surgery was urgently consulted, and the patient was cannulated at the bedside in a femoral-femoral fashion for VV-ECMO. She was transferred to the cardiothoracic intensive care unit for further management. Full ECMO support allowed for lung protective ventilator settings. High-dose steroids were continued for the remainder of her ECMO course with gradual improvements in her lung compliance and bronchospasm severity. Her paralysis was weaned off on the third day of ECMO, followed by her ketamine and sedation in a stepwise fashion. She was extubated on the sixth day of ECMO and decannulated the following day. Her only complications from ECMO were multiple deep venous thrombi at the sites of her cannulation. She was discharged from the hospital one week after decannulation with close pulmonary follow up.

Conclusion: The potential benefit of VV-ECMO in near-fatal asthma was clear in this case, as the patient would have likely either died from fulminant respiratory failure or sustained a significant ventilator-induced lung injury without ECMO support. Given that most asthma exacerbations eventually respond to high-dose steroids, including in near-fatal asthma, VV-ECMO is a logical supportive treatment option as a bridge to lung recovery. Accordingly, VV-ECMO is growing in prevalence as a treatment modality for near-fatal asthma. Over 500 patients were cannulated for asthma in a recent 25-year period based on data from the Extracorporeal Life Support Organization (ELSO) Registry. Additionally, initial outcome data for patients with near-fatal asthma on ECMO is promising. According to the ELSO Registry, the ECMO weaning success rate for this population was 86.7%, and the mean duration of support was seven days. Thus, as evidenced by this case and existing outcome data, VV-ECMO is a reasonable consideration for treating near-fatal asthma.

References: 1. Multidisciplinary Respiratory Medicine, Volume 11 (37), 2016 2. Journal of Intensive Care Medicine, Volume 33(9), 491-501, 2018 3. Critical Care, Volume 21(1), 1886-1888, 2017

Critical Care - 29 Octreotide Infusion Associated with Severe Hyperkalemia after Living Donor Liver Transplantation

Moheb A Gohar[1], Gohar Dar[1], Roshni Sreedharan[1]

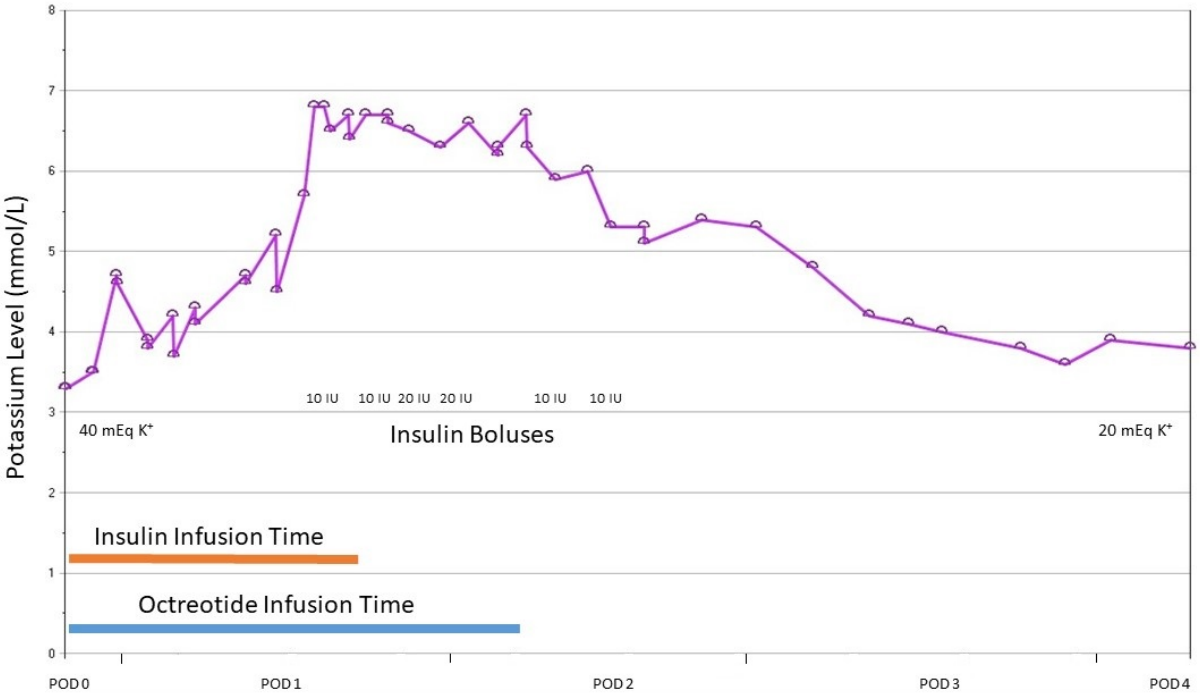
[1]Cleveland Clinic Foundation, Cleveland, OH

Introduction: Octreotide is a somatostatin analogue that inhibits the release of insulin from pancreatic beta cells through a G-protein coupled receptor(1). It is used in the treatment of severe hypoglycemia in the setting of sulfonylurea poisoning. It also reduces intestinal blood flow and portal venous pressure which makes it useful in the treatment of active upper gastrointestinal hemorrhage. It has been used as a modality to reduce the risk of 'Small for Size' syndrome in split liver transplantation. Here we describe severe hyperkalemia as a rare side effect of Octreotide infusion.

Methods: A 30-year-old female patient with a history of alcohol abuse developed liver cirrhosis and end stage liver disease complicated by portal hypertension and moderately severe tricuspid regurgitation. She underwent a tricuspid valve repair and a living donor liver transplantation. Upon arrival to the intensive care unit, Octreotide infusion was started to reduce the risk of 'Small for size' syndrome. On post-operative day 1, She developed severe hyperkalemia with levels persisting between 6-7 mmol/L. The patient was not acidotic and maintained normal blood glucose levels and urine output. Multiple attempts to induce intracellular shift of potassium using intravenous insulin and dextrose injections were marginally effective with potassium levels remaining above 6 mmol/L. No EKG changes were noted during the period of hyperkalemia. A decision was made to stop the Octreotide infusion on post-operative day 2 and later on that day potassium levels receded to previous normal levels.

Conclusion: Octreotide is a synthetic octapeptide that binds to somatostatin receptors (mainly SSTR2) and inhibits the release of Insulin, Glucagon and Growth hormone among others. It has been used in the treatment of acromegaly, sulfonylurea poisoning and neuroendocrine tumors. It also reduces intestinal motility and blood flow reducing portal venous pressure. This effect makes it a useful modality in the treatment of bleeding esophageal varices and in liver transplant patients to reduce excessive blood flow to small grafts. Hyperkalemia is a rare adverse effect of octreotide and is thought to be mediated by insulin inhibition and reduced intracellular shift of potassium. Abadala et al report a case of severe hyperkalemia after subcutaneous octreotide administration to treat sulfonylurea poisoning in a patient with chronic renal failure. In that patient, stopping octreotide in addition to hemodialysis led to the resolution of the hyperkalemia. In our case, severe hyperkalemia with octreotide infusion developed in the absence of chronic renal failure and resolved without the need for hemodialysis. In conclusion, severe hyperkalemia is a rare but serious adverse effect of Octreotide that can be managed by cessation of the Octreotide infusion in non-dialysis dependent patients.

References: (1) Glatstein M, Scolnik D, Bentur Y. Octreotide for the treatment of sulfonylurea poisoning. Clin Toxicol (Phila). 2012;50(9):795-804. (2) Adabala M, Jhaveri KD, Gitman M. Severe hyperkalaemia resulting from octreotide use in a haemodialysis patient. Nephrol Dial Transplant. 2010;25(10):3439-3442.


Potassium Level (mmol/L)
8
7
6
5
4
3
2
1
0
10 IU
10 IU
20 IU
20 IU
10 IU
10 IU
40 mEq K+
Insulin Boluses
20 mEq K+
Insulin Infusion Time
Octreotide Infusion Time
POD 0
POD 1
POD 2
POD 3
POD 4

Critical Care - 30 Massive Pulmonary Embolism During Stereotactic Laser Ablation for Temporal Epilepsy

Levent Sahin[1], Jared Staab[2]

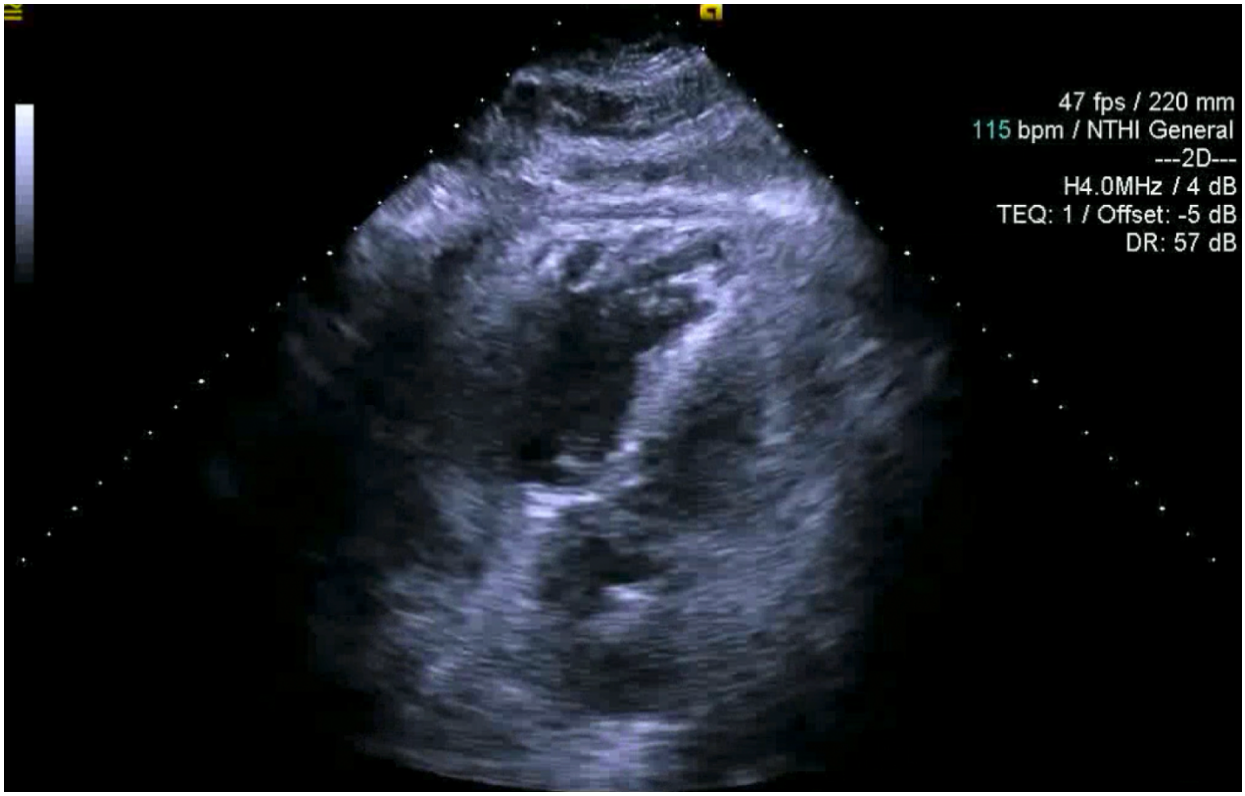
[1]University of Kansas Department of Anesthesiology, Kansas City, KS, [2]University of Kansas Medical Center, Kansas City, KS

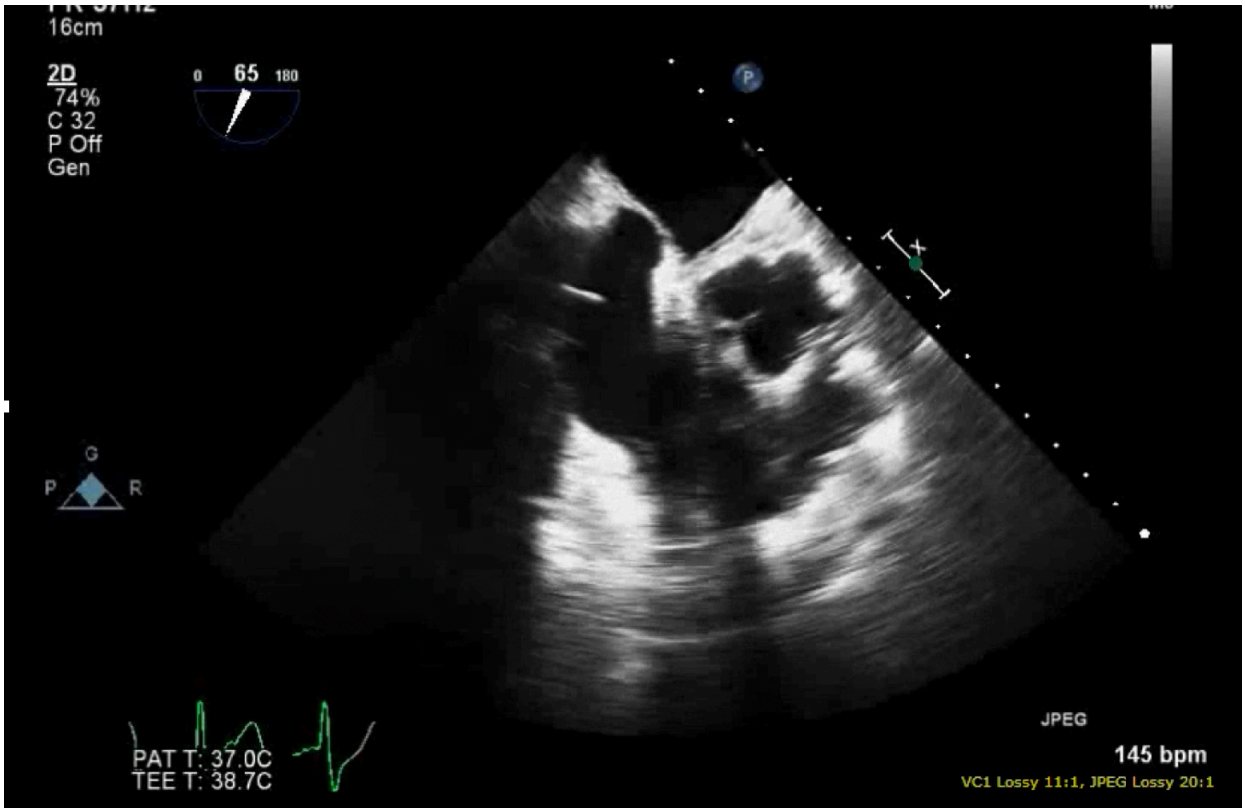
Introduction: Stereotactic laser ablation (SLA) is a minimally-invasive technique for eliminating epileptogenic area, deep intracranial tumors, and recurrent metastases. The duration of invasive MRI procedures, such as laser ablation, is lengthy due to extended image acquisition time. A prolonged procedure under general anesthesia as well as history of thromboembolic disease, heart disease, cancer, obesity, and serious medical illness potentially increases the risk of venous thromboembolism (VTE). Complications of SLA are related to neurocognitive deficits in general; however, in this case we present the first complication in the literature to our knowledge of massive pulmonary embolism (PE) over the perioperative period.

Methods: Case Report: A 72-year-old Caucasian female with a history of drug resistant epilepsy beginning 45 years ago and occurring approximately every 3 weeks was scheduled to undergo a magnetic resonance image-guided laser ablation of the hippocampus and amygdala due to right temporal mesial sclerosis. She was admitted on the morning of operation with ASA Physical Status 3 and BMI 38.5. General anesthesia was performed and the patient was turned to the prone position onto the MRI table with the head affixed in pins. Total anesthesia duration was 13 hours and laser ablation was total 1.5 hours. At the end of the procedure, the patient was removed from the MRI scanner. Once she was turned to supine position, she quickly decompensated, became severely hypotensive, and progressed to cardiac arrest. CPR was performed and medication administered. Once she stabilized, she was transported to the ICU where she continued to display hemodynamic instability and needed additional CPR twice. Bedside TTE demonstrated acute decompensated right heart failure likely secondary to large PE (Image 1). Finally, the patient was transferred to the OR for TEE and arterial thrombectomy. The TEE showed failing and massively dilated RV without visualized thrombus (Image 2). Open sternotomy was performed, and a large amount of clot was removed from a branch of the pulmonary artery. In the postoperative period, the patient was intubated and mechanically ventilated. She experienced AKI, transaminitis, SIRS, lactic acidosis, respiratory acidosis, vasogenic shock and cardiogenic shock. All necessary management was performed. Despite all measurements, her situation deteriorated and, on day 6 of admission, her status changed to CMO and she died.

Conclusion: Discussion: SLA is becoming an increasingly popular method for resistant epilepsy. The most common reported complications of SLA are transient neurologic deficit (13%), new progressive or permanent neurologic symptoms (3%), intracranial hemorrhage (2.5%), and deep venous thrombosis (2.5%). Life-threatening complications include intracranial hemorrhage, ventriculitis, meningitis, and refractory intracranial HT. Only two deaths have been reported from intracranial hemorrhage and meningitis in patients with brain tumor (1). Another study reported similar neurologic complications without VTE or death (2). Our patient had some risk factors for VTE such as obesity, long procedure time, and multiple serious diseases. Additionally, the effect of the laser and gadolinium on the coagulation system is not yet fully understood. Anticoagulation should be withheld prior to certain image-guided interventions due to intracranial hemorrhage risk (3); however, perioperative mechanical VTE prophylaxis using a lower extremity sequential compressive device is needed for high-risk patients undergoing any MRI-guided intervention (4).

References: 1. Am J Neuroradiol. 2015; 36: 1998-2006. 2. Neurosurg Clin N Am. 2016; 27: 37-50. 3. J Vasc Interv Radiol. 2012; 23: 727-36. 4. Eur J Radiol. 2015; 84: 668-670.


47 fps / 220 mm
115 bpm / NTHI General
---2D---
H4.0MHz / 4 dB
TEQ: 1 / Offset: -5 dB
DR: 57 dB


16cm
2D
74%
C 32
P Off
Gen
0
65
180
G
P
R
PAT T: 37.0C
TEE T: 38.7C
JPEG
145 bpm
VC1 Lossy 11:1, JPEG Lossy 20:1

Critical Care - 31 Acute Pericardial Tamponade After Pericardiocentesis

Levent Sahin[1], Elizabeth Cotter[2], Joel Grigsby[3]

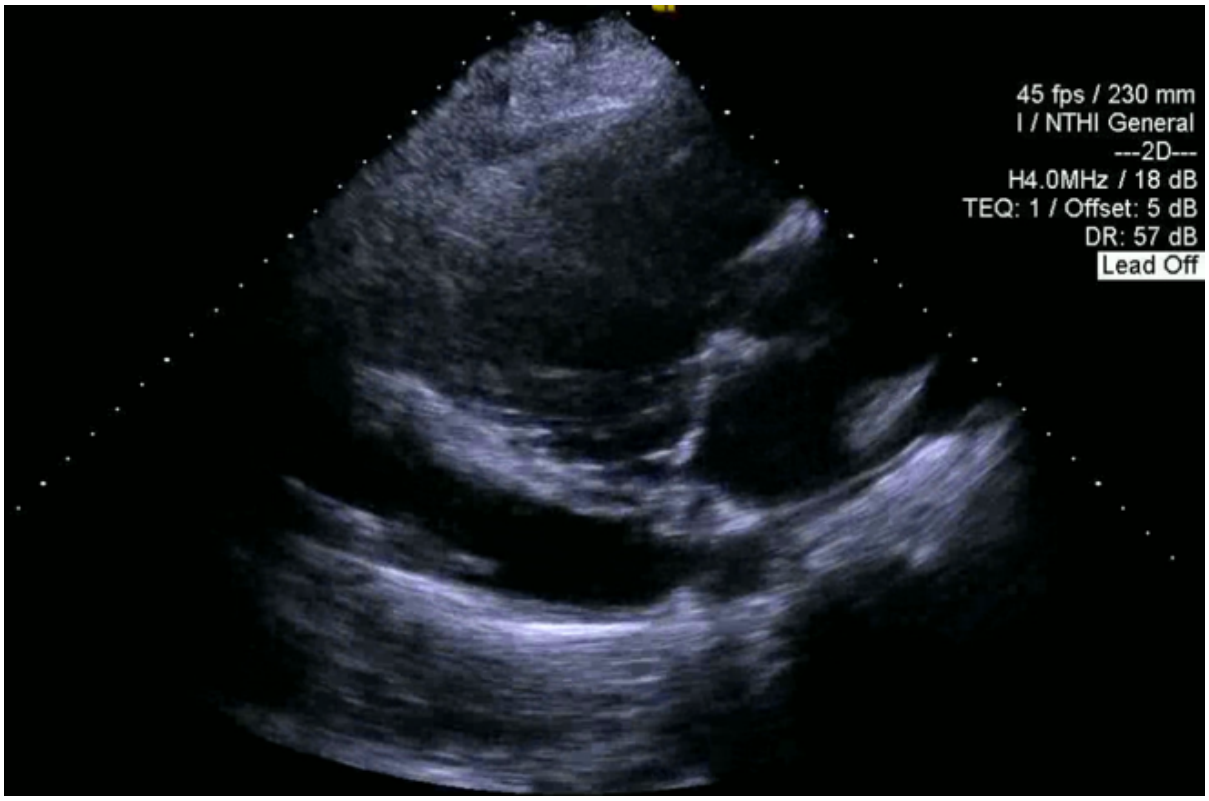
[1]University of Kansas Department of Anesthesiology, Kansas City, KS, [2]University of Kansas Medical Center, Kansas City, KS, [3]University of Kansas, Kansas City, KS

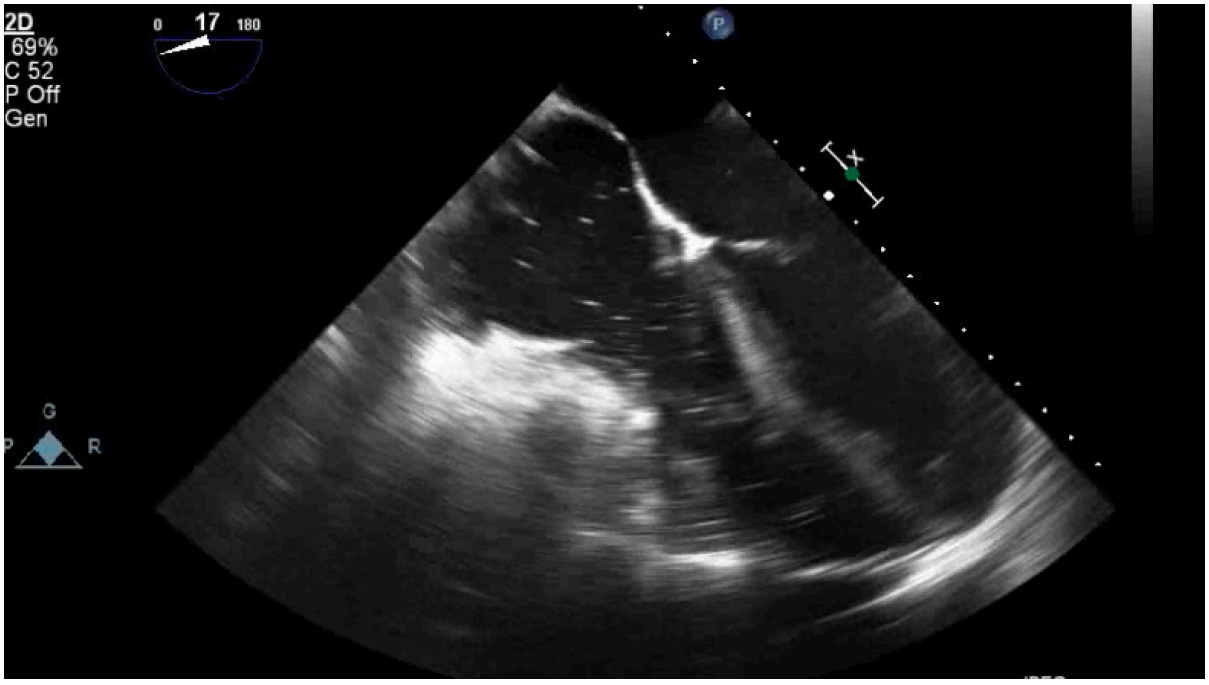
Introduction: Pericardial effusion may develop acute or gradually with wide variation in clinical presentation (1). While all significant pericardial effusions are of clinical importance, emergency drainage is needed only for patients with hemodynamic compromise. Cardiac tamponade with hemodynamic collapse is an absolute indication for emergent pericardial drainage via pericardiocentesis or surgical pericardiotomy. Here, we present a case of acute pericardiac tamponade after large pericardiac effusion drainage.

Methods: Case Report: A 44-year-old morbidly obese female was admitted with worsening dyspnea and weight gain related to acute exacerbation of heart failure and cardiogenic shock like picture. She had known medical history of heart failure with reduced ejection fraction (30%), ischemic cardiomyopathy, status post CAD, percutaneous coronary intervention to LAD and Cx, pulmonary hypertension, uncontrolled DM Type 2, OSA, and chronic kidney disease stage III. Right heart catheterization showed increased filling pressure with a RA pressure of 22 mmHg and PA pressure of 100/35 mmHg, with a mean PA of 63 mmHg and PCWP of 50 mmHg. Cardiac indexes were 2.2-2.4 L/min/m2. TTE also revealed large pericardial effusion, which was chronic but gradually increasing in size (Image 1). Her treatment plan included aggressive diuresis and inotrope (Milrinone) therapy and planned pericardial drain placement to relieve the restrictive pathophysiology. During the pericardiocentesis, 1L of serosanguinous fluid was evacuated and a drain was placed. Over the first post-procedure hours, the patient looked relieved and comfortable; however, lethargy, dyspnea, hypotension, and increased CVP slowly emerged after the sixth hour with minimal pericardial drain output. While initiating vasopressors, bedside TTE showed an amount of pericardial effusion similar to that seen before pericardiocentesis. The drain was then flushed and 250 cc sanguineous output was manually withdrawn. Hemodynamics improved immediately over the next hour with intermittent drainage but, within a short period, the patient became more unstable and the drain no longer functioned. She immediately underwent open heart surgery. Before sternotomy, the patient experienced cardiac arrest, needing 10 min of CPR. After ROSC was achieved, a 2L hematoma was evacuated from the pericardial space without active extravasation or bleeding and a pericardial window was performed (Image 2). After operation, hemodynamic instability continued for three days, necessitating inotropes and vasopressors.

Conclusion: Discussion: Pericardiocentesis is a life-saving procedure that quickly decreases pericardial pressure and relieves cardiac function for symptomatic large pericardial effusion or tamponade. Unfortunately, some complications have been reported at a rate of 10% such as cardiac puncture, coronary artery injuries, arrhythmias, pneumothorax, hemothorax, pneumopericardium, and hepatic injury (2). According to the reports, pericardiocentesis seems to be less efficacious in terms of preventing fluid re-accumulation in the long-term compared with pericardial window. The risk for pericardial re-accumulation after pericardiocentesis is as high as 76.9% and 15.4% after pericardial window (3). Theoretically, the reason of re-accumulation after drainage is pericardial stretching due to chronic large pericardial effusion may account for a higher negative pressure in the pericardial cavity, creating a suction effect toward pericardial cavity. It should be considered as a differential diagnosis Pericardial Decompression Syndrome, that manifests as right ventricle, left ventricle, or biventricular failure and/or pulmonary edema paradoxically occurring after successful pericardial fluid drainage (4). In this case, acute pericardial tamponade occurred after pericardiocentesis although there was a pericardial catheter for drainage. In conclusion, invasive pericardial drainage should not be recommended in patients with an asymptomatic large idiopathic pericardial effusion. If pericardial effusion drainage has to be done, anesthesiologists and intensivists should be mindful of the possibility of re-accumulation and tamponade even if there is a pericardial catheter.

References: 1. N Engl J Med 2003; 349:684. 2. Eur Heart J. 2015; 36: 2921-64. 3. Am J Cardiol. 2020; 126: 89-93. 4. BMJ Case Rep. 2016. pii: bcr2015211550.





Critical Care - 32 Complications of multiple thromboembolic events after surgical application of a thrombin hemostatic matrix

Danielle M Williamson[1], Matthew Dudley[2]

[1]University of California San Francisco, San Francico, CA, [2]University of California San Francisco, San Francisco, CA- CALIFORNIA

Introduction: Use of topical fibrin and thrombin containing products to achieve surgical hemostasis is commonplace. Here, we report the case of a patient undergoing posterior spine decompression and fusion with intraoperative course complicated by multiple thromboembolic events causing acute right heart failure, cardiac arrest, cerebral infarction, and disseminated intravascular coagulation, as a result of presumed intravascular hemostatic agent-induced emboli.

Methods: A 65 year old woman with chronic pain and radiculopathy due to lumbar spine lordosis, with prior L4-S1 fusion, presented for revision posterior spine decompression and L1-S1 fusion with bilateral pelvic fixation. During closure of the osteotomy, venous bleeding was identified. To ensure hemostasis, after cauterizing the bleeding vessel, thrombin gel foam was applied to the area. Within minutes, the patients blood pressure dropped precipitously, followed by PEA arrest. ROSC was achieved after approximately 10 minutes of resuscitation; immediately post-ROSC, transesophageal echo demonstrated acute cor pulmonale, with spontaneous echogenicities visualized in transit through the right and left heart, and right-to-left shunt across a patent foramen ovale. In addition, the patient was hypoxemic with alveolar hemorrhage, and found to be in DIC. Urgent cannulation for ECMO was discussed with cardiothoracic surgery, however given improvement in the patients hemodynamics, this was not felt to be necessary. After further stabilization, the patient was taken for CT/CTA of the head and chest. Chest imaging was notable for multiple bilateral pulmonary emboli from the main pulmonary artery to lobar and subsegmental branches, as well as right ventricular enlargement, consistent with elevated right heart pressures. Initial CT head did show a possible MCA stroke, and areas of delayed perfusion suggestive of ischemia in multiple vascular territories, however there was no evidence of large vessel occlusion, intracranial hemorrhage, or herniation. The patient was then taken to the ICU, and was further stabilized, with targeted temperature management, correction of her coagulopathy, and was initiated on a heparin drip. Approximately 5 hours post-operatively, the patient became acutely hemodynamically unstable with hypertension followed by hypotension, at which time pupils were noted to be fixed and dilated; repeat head CT revealed worsening cerebral edema resulting in transtentorial and tonsillar herniation as well as diffuse anoxic brain injury. Despite aggressive management of ICP with mannitol and hypertonic saline, the progression of her neurologic injury was felt to be unrecoverable, at which point the family opted to transition to comfort care, and the patient passed peacefully.

Conclusion: We believe the etiology of the multiple thromboembolic events and coagulopathy described above was inadvertent introduction of the hemostatic matrix into the spinal vasculature. Such agents work by both activation of platelets and the intrinsic clotting cascade, as well as swelling to provide physical tamponade of the wound. The timing of the application, followed by acute cardiopulmonary compromise and arrest, along with the visualized clot on both TEE and CT imaging suggests that small particles of matrix provided nidus for clot formation as well as activation of the clotting cascade, triggering DIC. Plausibility for this is lent by consistent large showering of injected matrix into the right ventricular outflow tract in a porcine model (1), as well as similar reports in the literature, wherein several hemostatic agents have been implicated in cases of thromboembolism after their use in spine surgeries (2,3,4). The case described above was further complicated by right-to-left shunting via a previously asymptomatic PFO, which allowed transit of clot into the systemic circulation, and showering of clot in multiple vascular territories, ultimately leading to catastrophic cerebral edema and herniation. The use of topical hemostatic agents is widely used intraoperatively; while clinically significant complications associated with their use are rare, it is possible that subclinical embolization is more common than recognized. It is therefore critical that providers are aware of this possibility, especially in patients who have known compromised cardiopulmonary reserve or increased risk of right-to-left shunting.

References: 1. Kuhns, C. A. et al. Spine (Phila. Pa. 1976). 40, 218-223 (2015). 2. Ji, P., Jiang, Y., Hou, W., Li, Q. & Kang, Y. World Neurosurg. 137, 183-186 (2020). 3. Wei, Z., Elder, B. D., Goodwin, C. R. & Witham, T. F. J. Clin. Neurosci. 22, 1502-1505 (2015). 4. Ferschl, M. B. & Rollins, M. D. Anesth. Analg. 108, 434-436 (2009).

Critical Care - 33 Use of lidocaine infusion as an adjunct to sedation in two patients requiring venovenous ECMO for COVID 19 related ARDS with refractory agitation

Christofer D Barth[1], Ruth Musselman[2]

[1]Aurora St. Luke's Medical Center, Wauwatosa, WI, [2]Aurora St. Luke's Medical Center, Milwaukee, United States of America

Introduction: The purpose of this report is to describe a novel approach to sedation for patients while on ECMO with COVID 19 related ARDS in whom refractory agitation creates risk for patient disability and death. Lidocaine infusion is commonly used for ventricular arrhythmia and acute pain conditions(1); lidocaine blood levels can be obtained by clinical labs. Lidocaine infusion may cause sedation and has been described as an adjunct to anesthetics (2,3). COVID 19 related delirium is a condition being reported with increasing clarity (4). ECMO patients with ARDS may require high levels of sedation for prolonged periods; this seems pronounced for COVID 19(5). ECMO circuits disrupt normal pharmacokinetics and pharmacodynamics(6). These clinical factors present a significant clinical dilemma that risk patient disability and harm. We describe our clinical experience of using lidocaine as an adjunct to sedation in two COVID 19 patients with refractory agitation who were not tolerating multimodal sedation and analgesia while on ECMO.

Methods: In the first case, the patient required ECMO with separate right jugular and femoral venous canulation after approximately ten days of COVID-19 symptoms and approximately two days of intubation. The patient had acute hypoxemic respiratory failure refractory to inhaled nitric oxide, neuromuscular blockade, and prone positioning; ECMO canulation was elected. For weeks this patient required variable sedation including intravenous propofol, midazolam, ketamine, as well as other oral adjuncts to maintain ventilator synchrony. Due to the patient's history of ventricular tachycardia and ongoing issues with ventilator asynchrony, lidocaine infusion was elected as an adjunct; the infusion began at 0.5 mg/min for a day and was titrated up to 1 mg/min to obtain lidocaine blood levels of 2-4 mcg/ml. The results to ventilator synchrony seemed beneficial and the sedative regimen was simplified to the following intravenous medications: midazolam, fentanyl, and lidocaine. The patient's ventilator asynchrony was maintained and the patient began to be interactive again. Unfortunately, the patient succumbed to complications associated with COVID-19 ARDS with spontaneous pneumothoraces and bleeding. In the second case the patient experienced ECMO canulation via right internal jugular dual stage pulmonary artery ECMO canulation approximately twenty days after the onset of COVID-19 symptoms and four days of intubation for COVID-19 related ARDS. The patient had been trialed with prone positioning, neuromuscular blockade, and inhaled nitric oxide. At about fifteen days of ECMO the patient continued to have ventilator asynchrony requiring simultaneous midazolam, propofol, ketamine, opioids infusions and a number of oral sedative agents. Again we elected to add lidocaine to the regimen. The patient experienced similar benefits as the first patient; with an infusion rate of 1 mg/min we obtained fairly steady stage lidocaine blood levels between 2 - 4 mcg/ml for more than three weeks and we able to withdraw other intravenous and oral sedatives so that the patient could again become interactive yet synchronous with the ventilator. Unfortunately, at forty-five days the patient decompensated, and it was elected to transition the patient to comfort care.

Conclusion: Intravenous lidocaine has been explored as an adjunct to acute pain management(1) and as an adjunct to anesthesia (2). Also due to clinical laboratories' ability to monitor blood levels, lidocaine should provide a uniquely safe adjunct in sedation of agitated mechanically ventilated patients. Reports of COVID-19 ARDS requiring high levels of sedation to maintain ventilator synchrony are a significant feature of the disease(4,5). Patients on ECMO requiring higher levels of sedation due to variable pharmacokinetics and pharmacodynamics is a known problem(6). In this case reports we describe the use of intravenous lidocaine as an adjunct to sedation that permitted significant reduction in standard infused sedatives. The patients re-emerge from deep sedation into a state of wakefulness with reasonable ventilator synchrony. Lidocaine levels were readily monitored for weeks. Lidocaine infusion may represent a safe and effective adjunct to sedation for patients on ECMO. Its

use in critically ill patients requiring high levels of intravenous sedation due to refractory agitation should be further explored.

References: 1. Cochrane Database Syst Rev, 6: CD009642, 2018 2.Libyan J Med 13:1433418, 2018 3. Br J Anaesth 121:1059-1064, 2018 4.Critical Care, 24:491, 2020 5. Anesth Analg, 10:1213, 2020 6. J Thorac Dis 10:S629-S641, 2018

Critical Care - 34 Spontaneous massive hemothorax in a patient with VV-ECMO with COVID-19 after spontaneous right pulmonary artery rupture

Ronny Munoz-Acuna[1], Annette Ilg[1], Daniel Walsh[1], Somnath Bose[1]

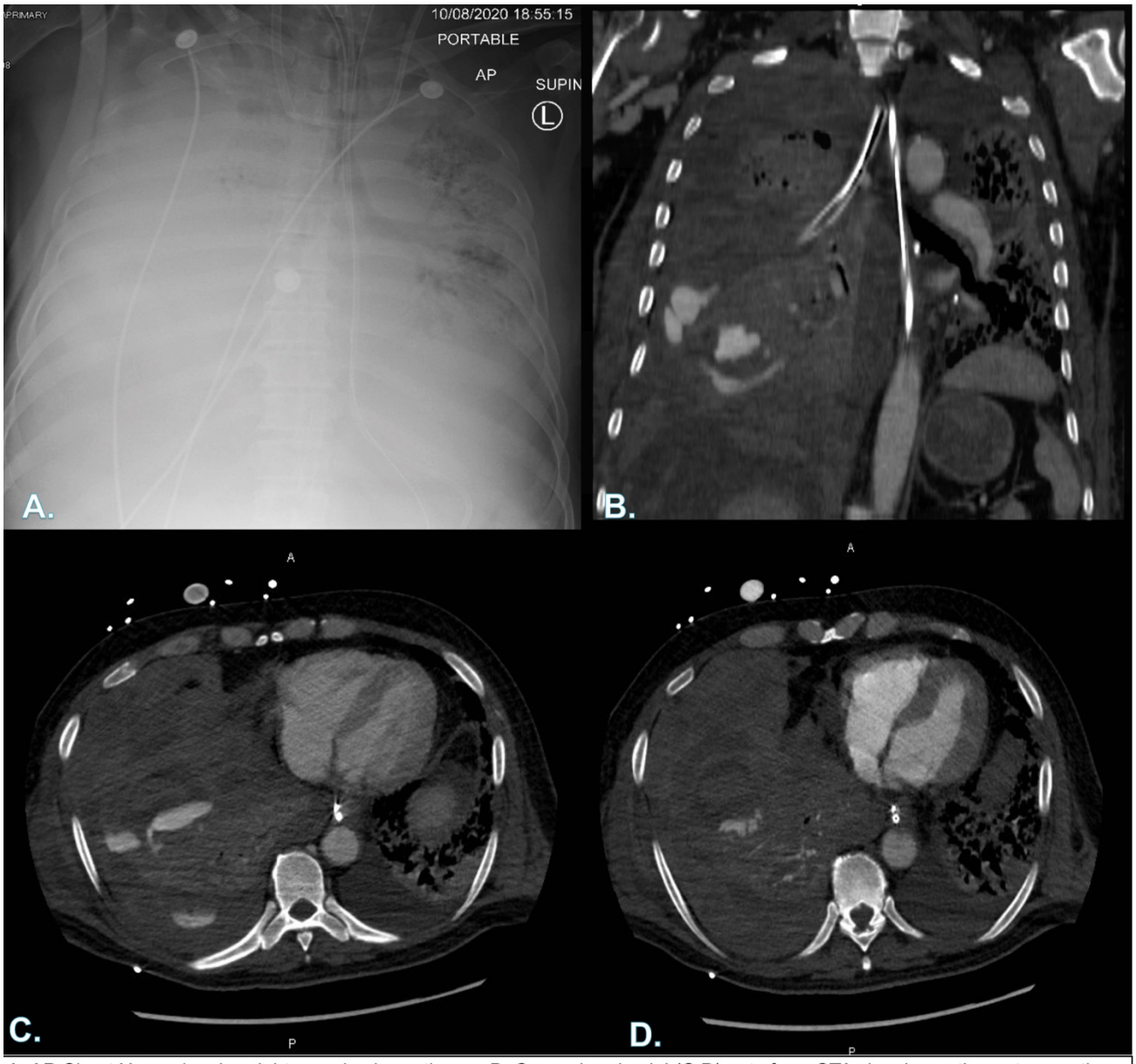
[1]Beth Israel Deaconess Medical Center, Boston, MA

Introduction: The occurrence of massive hemothorax in patients on extracorporeal membrane oxygenation (ECMO) is an unusual and life-threatening complication(1). ECMO-related massive hemothorax usually occurs unilaterally and presents as a hemorrhagic or obstructive shock. Among several etiologies, the two most common are rupture of a pseudoaneurysm or erosion of the main pulmonary artery (PA) or one of its branches(2). It remains a medical challenge to diagnose it promptly. Appropriate management consists of cardiovascular support and transfusion therapy while at the same time providing definitive endovascular or surgical treatment(3). Published literature shows that even with intensive treatment, only nearly two-thirds of the patients can be saved. The most significant risk factor for mortality is the presence of comorbidities, such as sepsis, diabetes mellitus, or immunocompromised status(3).

Methods: A 61-year-old Spanish-speaking male with a history of HTN, HLD presented with ten days of shortness of breath, after which he was diagnosed with COVID-19 pneumonia. He had a slow but steady decline culminating in initiation of VV-ECMO 17 days after hospitalization using a 25 Fr drainage right femoral vein cannula and a 17 Fr return right IJ cannula. On VV-ECMO day 12, a tracheostomy was performed under direct bronchoscopic guidance with no issues. On VV-ECMO day 13, the tidal volume suddenly deteriorated, with suctioning of copious amounts of blood and bloody secretions through the tracheostomy tube. An emergent bronchoscopy showed extensive clots in the airway from the trachea to the bilateral mainstem airways deeming a tracheostomy site bleeding unlikely. Heparin was stopped, and the patient was aggressively resuscitated with crystalloids and blood products. Point-of-care ultrasound was suggestive of hypovolemia. A chest X-ray noted a large right-sided pleural effusion, with a contralateral shift of the trachea. After insertion of a right chest tube, there was an immediate return of 1500 cc of blood. Blood output continued with another 1500 cc over the next hour, prompting an emergent CTA, which showed that along the medial aspect of the right lower lobe, there was a focus of active extravasation consistent with massive bleeding erupting into the adjacent pleural space, coming from the right main pulmonary artery. It was found that the patient had a cavitation secondary to aggressive pneumonia caused by Pseudomonas aeruginosa, which had persisted despite multiple courses of targeted therapy. It was deemed that the arterial rupture was most likely secondary to erosion from the contiguous cavitation, which had debilitated the vessel wall. It was decided not to pursue salvage radioembolization, which would compromise most blood flow to the right lung, further limiting the gas exchange capabilities of an already debilitated respiratory system. Given not only the high mortality of this condition in COVID-19 patients but the already poor prognosis given his pulmonary compliance of less than 10 cc/cmH2O, and the family wishes regarding goals of care, no further intervention was sought, and the patient passed away in the following hours.

Conclusion: Massive hemothorax secondary to spontaneous pulmonary artery rupture is very rare and carries high morbidity and mortality. Prompt recognition and aggressive supportive care are imperative. A high level of vigilance is fundamental in patients with multiple risk factors, including sepsis, diabetic mellitus, or immunocompromised status. Physicians taking care of VV-ECMO patients should be aware of this unusual complication's occurrence and potentially fatal outcomes.

References: 1. Crit Care. 2020 Nov 6;24(1):638. 2. Circulation. 2015 Jan 20;131(3):310-6. 3. Asian J Surg. 2012 Jan;35(1):16-22.



A. AP Chest X-ray showing right massive hemothorax. B. Coronal and axial (C,D) cuts from CTA showing active extravasation of IV contrast from the right main pulmonary artery, in relation to a pulmonary cavitation caused by P. aeruginosa pneumonia.

Critical Care - 35 Culture Negative Bilateral Lung Abscess in a Re-Transplanted Kidney Recipient: What Could It Be?

Laurent Del Angel Diaz[1], Mada F Helou[1]

[1]University Hospitals at Case Medical Center, Cleveland, OH

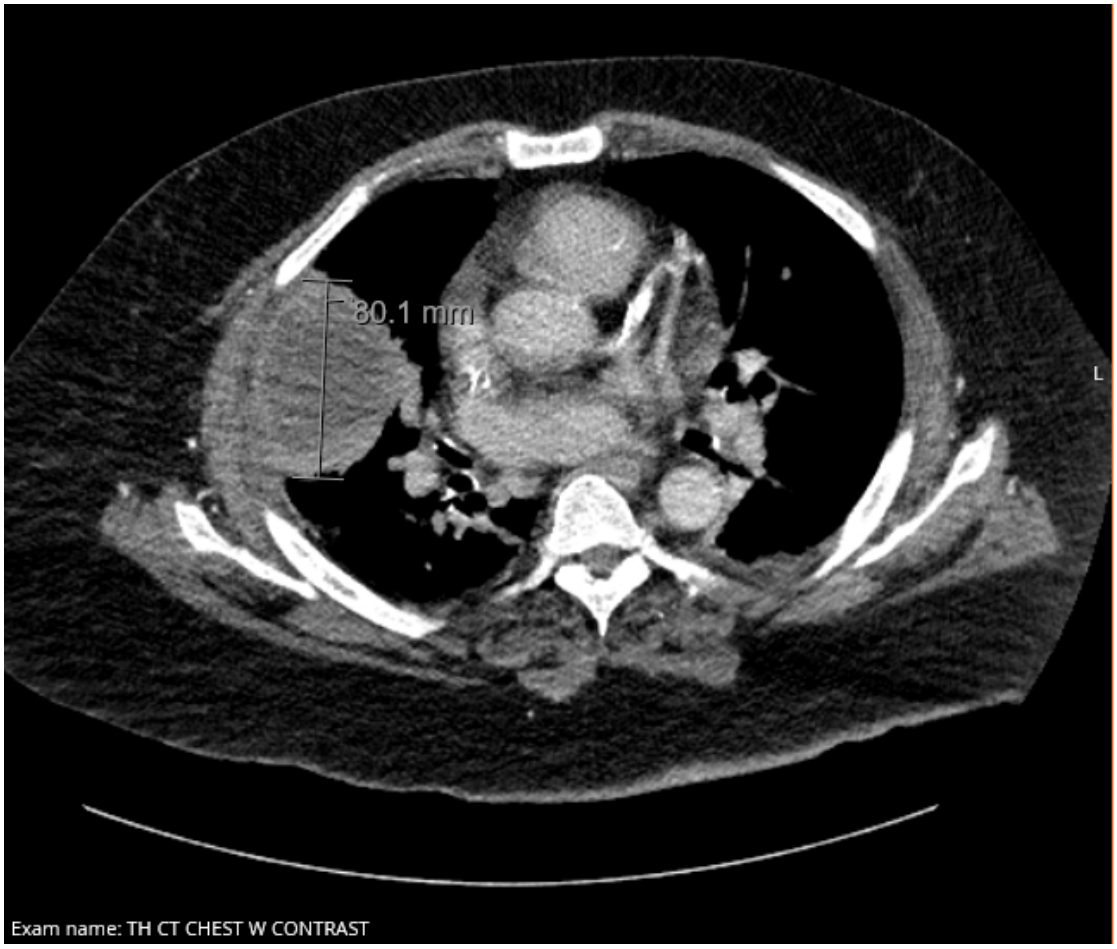
Introduction: Mucormycosis (previously called zygomycosis) encompasses a group of opportunistic fungal infections caused by a group of mold of the order Mucorales (1,2). Infections tend to be aggressive in nature and can have a mortality up to 90% in disseminated disease (3). Overall, rhino-cerebral mucormycosis is the most common form of disease (33.3%), followed by pulmonary (25.9%), disseminated (14.4%), transplanted kidney (11.5%), cutaneous (7.5%), gastrointestinal (5.7%), peritoneal (1.1%) and artery stent (0.6%) (4). Immunocompromised patients, especially those with hematological malignancies, hematopoietic stem cell transplantation, solid organ transplantation, diabetes and long term corticosteroid or voriconazole prophylaxis are most affected(1). Rhizopus spp. is usually the most common culprit, but more than 5 other species including Mucor spp. and Rhizomuchor spp. can also cause disease (5,6). We present a case report of isolated pulmonary mucormycosis (IPM) in a re-transplanted kidney recipient presenting as non-resolving pneumonia.

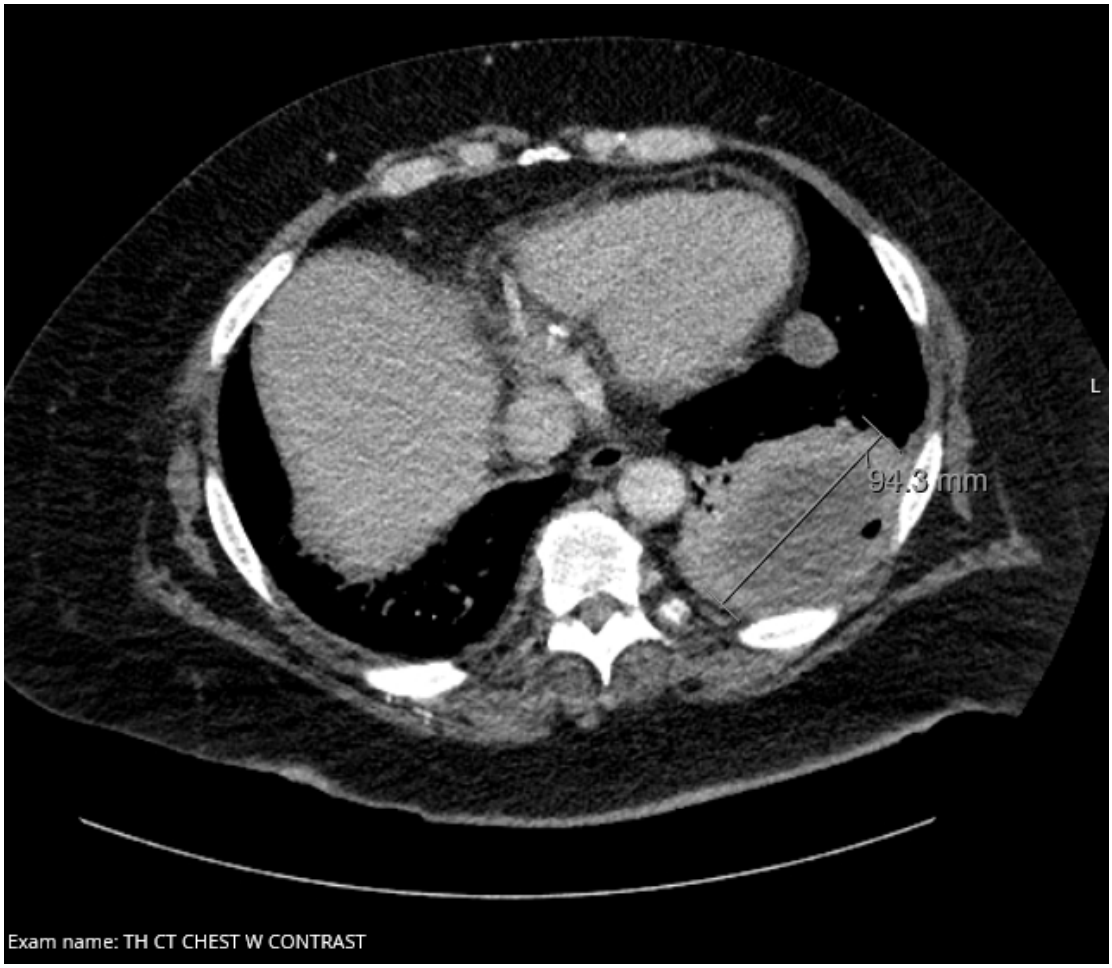
Methods: 66 y/o AA male with history of ESRD, HTN and SLE, DDKT twice (2015, 6/2020) who presented to the ED with a chief complaint of chest pain and shortness of breath. Patient was admitted and evaluated for possible pneumonia vs PE vs cardiac etiology. He was discharged home with oral antibiotics, Ampicillin/sulbactam, and a diagnosis of left lower lobe pneumonia. The patient continued to deteriorate over the next 2 weeks experiencing shortness of breath, cough, diarrhea and weight loss. Covid-19 infection was ruled out in the ED. He was admitted and underwent bronchoscopy and chest CT. CT demonstrated bilateral pulmonary abscesses concerning for bacterial vs fungal etiology. Bronchoscopy didn't show any endobronchial lesions, samples from BAL, brushing & needle aspiration were negative for any clear pathological process He was discharged home with Ampicillin/Sulbactam IV for 6 weeks. Patient required readmission for increasing O2 needs and transfer to ICU for respiratory distress. A chest CT scan was ordered, the findings demonstrated worsening bilateral abscesses. Interventional radiology was consulted, bilateral pigtail drains were placed with minimal output. Patient suffered hypoxic respiratory failure after the procedure that required emergent intubation and ICU management. Two days after pigtail placement, fluid culture started growing molds. Repeated cultures grew Rhizopus species with subsequent determination of Microsporus subspecies. Patient was taken to the OR where he underwent right VATS, washout and pleural biopsy. Patient was started on lipophilic amphotericin B daily and will continue for a total of 8-12 weeks.

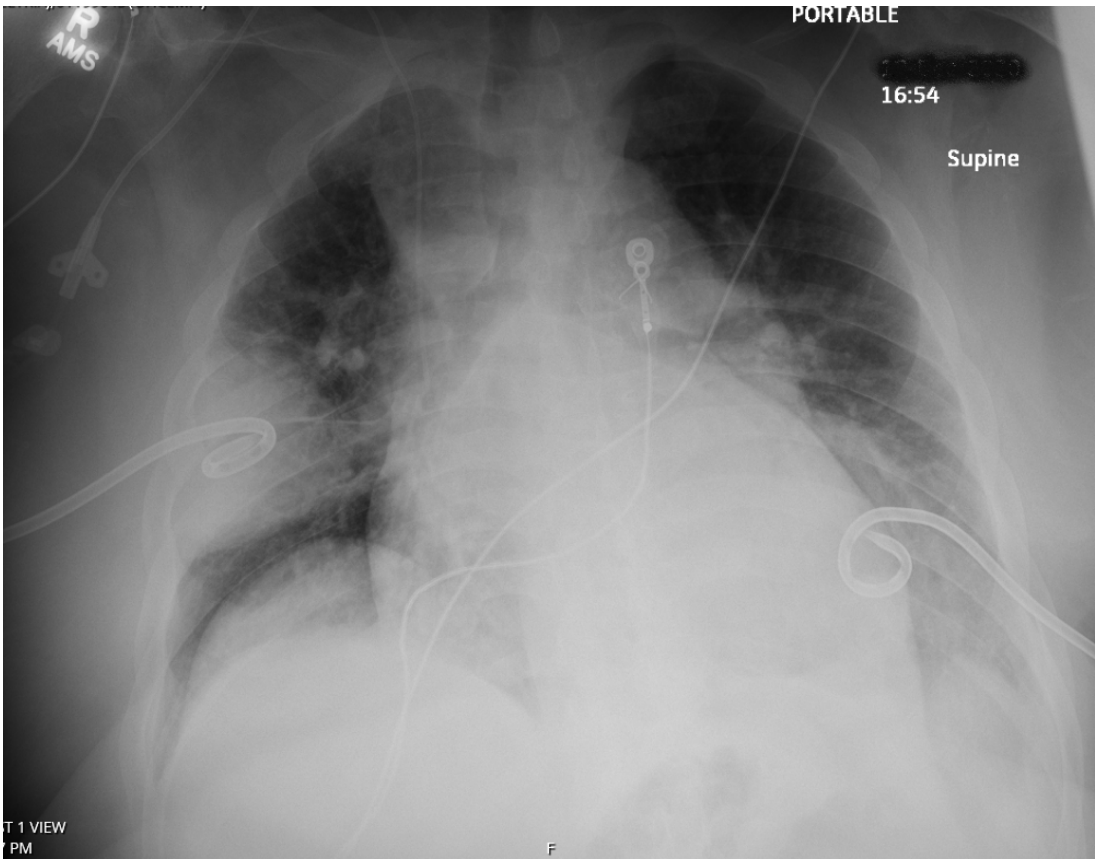
Results: After 3 weeks of daily treatment with lipophilic amphotericin B, the abscesses remain stable without progression, and with no increase in O2 requirements. Patient remained in 2 L nasal canula. The patient was discharged home with continuation of lipophilic amphotericin B and follow up CT scan every 2 weeks. Thoracic service will ultimately be consulted for debridement of necrotic lung tissue.

Conclusion: Isolated Pulmonary Mucormycosis (IPM) is an exceedingly rare and potentially lethal disease. IPM carries a worse prognosis when compared to the classic rhino-orbital-cerebral disease (9). IPM is believed to be caused by spore inhalation leading to necrotizing pneumonia (10). Surgical debridement continues to be the cornerstone of treatment of invasive Mucormycosis. When in combination with antifungal therapy, outcomes may improve for localized disease, but becomes extremely challenging with bilateral lung involvement (11-13). Surgical resection of IPM is reserved for localized disease (11), and only very few cases have been reported of surgical intervention with multifocal pneumonia (10). In our experience, low threshold should exist to investigate IPM in selected populations to prevent delay in treatment and improve mortality.

References: Pulmonary mucormycosis presenting as nonresolving pneumonia in a patient with diabetes mellitus. 59 (12) e201-e205. 2014. Global epidemiology of mucormycosis. 5(1):26 2019. Mucormycosis. 099-114. 2020. Mucormycosis in renal transplant recipients: Review of 174 reported cases. 17(1) 283. 2017. Epidemiology and clinical manifestations of Mucormycosis. 2012. Invasive non-Aspergillus mold infections in transplant recipients, 17(10):1855-1864. 2011. Early identification of rhinocerebral mucormycosis. 34(3):166-71. 2005 Chronic rhinocerebral mucormycosis:.57(11):699-702. 2014 Isolated pulmonary mucormycosis. 219342. 2017 Surgical Treatment of Multifocal Pulmonary Mucormycosis. 106(2):e93-e95. 2018 Pulmonary Mucormycosis Treated With Lobectomy. 103(6):e531-e533. 2017 ESCMID and ECMM joint clinical guidelines for the diagnosis and management of mucormycosis. Suppl 3:5-26. 2012 Pulmonary mucormycosis: The last 30 years. 159(12):1301-1309.1999




L
94.3 mm
Exam name: TH CT CHEST W CONTRAST


R
AMS
PORTABLE
16:54
Supine

Critical Care - 36 Sevoflurane as a lifesaving therapy in Status Asthmaticus: A case report.

Adriana S Martini[1], Omar Elnaggar[1], Shravan Gupta[1], Andres F Ocampo-Salazar[2]

[1]University of Miami, Miami, FL, [2]N/A, -, United States of America

Introduction: Asthma is a chronic disease of airway inflammation and hyperreactivity and one of the most common respiratory pathologies in the United States. Status asthmaticus (SA) is a severe exacerbation characterized by hypoxemia, hypercarbia, and respiratory failure refractory to traditional therapies with bronchodilators and corticosteroids. Mortality for patients with SA requiring ICU admission is as high as 10%1,2. Patients unresponsive to traditional therapies may require intubation and mechanical ventilation (MV). In refractory disease, inhalational anesthetics (IA) have shown promise in multiple case studies3,4. In this case report we demonstrate the successful use of sevoflurane in a patient with severe SA.

Methods: A 40-year-old female presented to our emergency department with malaise and worsening dyspnea. The patient had history of asthma with previous ICU admissions, systemic lupus erythematosus, hypertension and insulin dependent diabetes mellitus. Upon admission, patient required BiPAP and was initiated on albuterol-ipratropium nebulizer, IV methylprednisolone, montelukast, and IV magnesium. Arterial blood gas showed pH 7.34/PaCO2 42/PaO2 102/HCO3 22/O2Sat 97.7. Her worsening respiratory status prompted intubation and transfer to ICU. IV ketamine was added for sedation and bronchodilation. On hospital day 2, hypercarbia and acidosis continued to worsen. After anesthesiology consultation, 1.5% sevoflurane was started. Epinephrine infusion was initiated to provide further bronchodilation and hemodynamic support. After 48 hours of sevoflurane, ABG showed pH 7.33/PaCO2 47/PaO2 210/HCO3 24/O2Sat 99.2; sevoflurane and epinephrine were discontinued, and patient was transitioned to heliox. On hospital day 6, ECCOR was started due persistent hypercarbia. By hospital day 12, the patient's condition continued to improve, and she was weaned off heliox and ECCOR. On hospital day 19, the patient was extubated to BiPAP.

Conclusion: SA is a life-threatening condition that requires immediate recognition and aggressive treatment. In refractory cases, intubation and MV are undertaken to provide oxygenation and ventilation until bronchospasm resolves. Rescue therapies include muscle relaxation, infusion of ketamine, heliox, and general anesthesia with IA 2,5. IA have shown to decrease airway resistance, dynamic hyperinflation, and intrinsic PEEP5. They work via direct relaxation of bronchial smooth muscle, inhibiting the release of inflammatory mediators, Œ≤-adrenergic receptor stimulation, reduction of vagal tone and vagal-mediated reflexes, and antagonism of the effects of histamine and methacholine 5,6. Sevoflurane was used successfully in this case as a rescue treatment for SA in a patient failing standard medical management. Current literature supporting the use of IA in SA is only available at the case report level and further research is necessary to determine optimal agent, dosing and duration of treatment.

References: 1-Asthma Exacerbations: Pathogenesis, Prevention, and Treatment. J Allergy Clinical Immunol Pract. Jul-Aug 2017;5(4):918-927. 2-Adjunctive extracorporeal carbon dioxide removal in refractory status asthmaticus. BMJ Case Rep. 2017 3-Acute Severe Asthma in Adolescent and Adult Patients: Current Perspectives on Assessment and Management. J Clin Med. 2019 Sep; 8(9): 1283. 4Refractory Status Asthmaticus: A Case for Unconventional Therapies. Indian J Crit Care Med. 2018 Oct; 22(10): 749-752. 5-Refractory Status Asthmaticus: Treatment with Sevoflurane. Fed Pract. 2019;36(10):476-479. 6-Volatile anaesthetic for treatment of respiratory failure from status asthmaticus requiring extracorporeal membrane oxygenation. BMJ Case Reports CP 2020;13: e231507.

Critical Care - 37 A Case of Clotting and Bleeding: When Differentiating the Zebra from Horses became a Ticking Time Bomb in the Critical Care Unit

Louise Gliga[1], Arnav Kumar[1], Maan Jokhadar[1]

[1]Emory University, Atlanta, GA

Introduction: We present a unique case of heparin-induced thrombocytopenia (HIT), notable for life threatening thrombotic event prior to platelet decline, followed by significant bleeding complication that required complex critical care deduction and therapeutic interventions.

Methods: A 49 year old male with a history of hypertension, atrial fibrillation, and smoking presented to our critical care unit with hypotension, supraventricular tachycardia (SVT) and ileus. He had initially presented to an outside hospital for syncope. He underwent a left heart catheterization (LHC), notable for acute right coronary artery thrombus during the procedure. Incidentally, angiographic images showed an abdominal aortic aneurysm (AAA). Subsequently, he was transferred to our institution for an open AAA repair. On post-operative day 2, he was noted to be hypoxic and hypotensive, and was found to have new large bilateral pulmonary emboli (PEs) (Fig 1). Echocardiogram demonstrated severe right ventricular strain. Upper and lower extremity venous dopplers were negative. Given the proximity to surgery, he was taken first for mechanical thrombectomy, and then started on a heparin drip. Over the next 3 days, his platelets slowly declined. He was also noted to have hypotension and persistent SVT despite anti-arrhythmics. Upon reviewing the acute RCA thrombus with initial heparin exposure during LHC, followed by acute post-operative PE, plus subsequent decline in platelets, suspicion for HIT was raised. Argatroban was started and HIT panel was sent. Unfortunately, patient's abdominal pain worsened. Abdominal CT demonstrated a new, large retroperitoneal hematoma with no active extravasation (Fig 2). To balance the PEs, bleed, and platelet drop, the patient was trialed on a lower dose of argatroban. After a multidisciplinary meeting, it was deemed not beneficial to pursue arterial embolization or inferior vena cava filter. Fortunately, hemoglobin and platelets began to stabilize, and abdominal pain and arrhythmias improved. He was resumed on full dose argatroban. HIT antibody came back positive at 2.3, and serotonin release assay confirmed the diagnosis of HIT. Platelets recovered, and he was discharged on apixaban, with education about heparin allergy.

Conclusion: Heparin-induced thrombocytopenia is a rare but life-threatening disorder. This case illustrates an unusual presentation in which thrombosis took place prior to thrombocytopenia. In addition, significant bleeding occurred, which is exceedingly rare in HIT(1,2). Prior heparin also confounded the case, as did arrhythmia, syncope and, ileus. HIT remains an important differential for thrombocytopenia and concurrent thrombosis. Heparin exposures are important to be aware of, and recognition of possible HIT can be life-saving.

References: 1. Levy JH, Tanaka KA, Hursting MJ. Reducing thrombotic complications in the perioperative setting: an update on heparin-induced thrombocytopenia. Anesth Analg. 2007 Sep;105(3):570-82. 2. Kelton JG. Heparin-induced thrombocytopenia: an overview. Blood Rev. 2002 Mar;16(1):77-80.

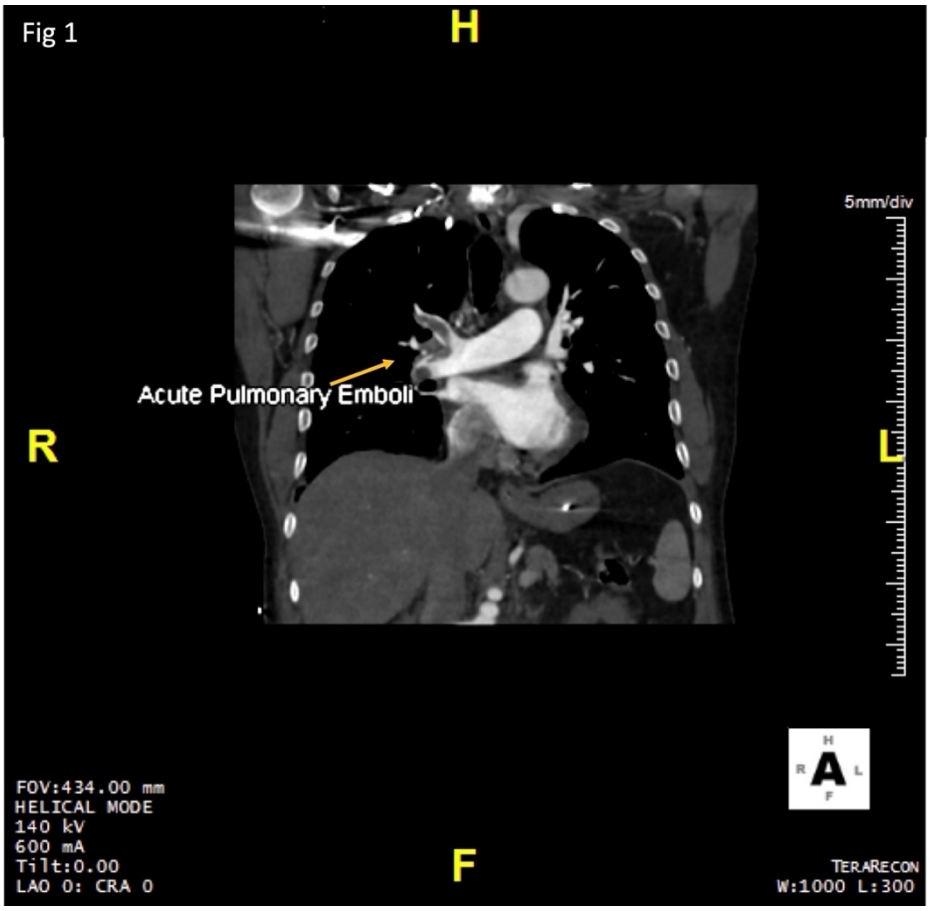


Fig 1
H
Acute Pulmonary Emboli
R
L
5mm/div
FOV:434.00 mm
HELICAL MODE
140 kV
600 mA
Tilt:0.00
LAO 0: CRA 0
F
TERARECON
W:1000 L:300

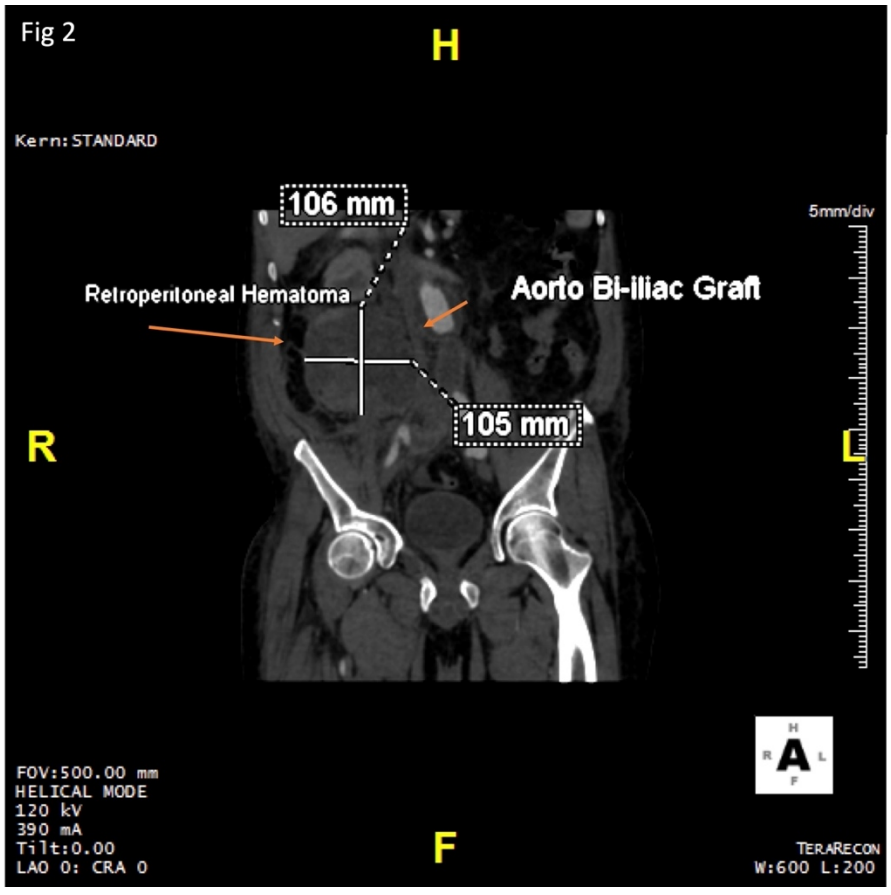


Fig 2
H
Kern: STANDARD
106 mm
Retroperitoneal Hematoma
Aorto Bi-iliac Graft
105 mm
R
L
5mm/div
FOV:500.00 mm
HELICAL MODE
120 kV
390 mA
Tilt:0.00
LAO 0: CRA 0
F
TERARECON
W:600 L:200

Geriatric Anesthesia

Geriatric Anesthesia - 1 Teamwork Makes the Dream Work: The Importance of Multidisciplinary Management of a Previously Undiagnosed Uretero-Iliac Artery Fistula During Routine Cystoscopy in a Geriatric Patient

Alexande Skojec[1], Jessica Sheeran[2]

[1]University of Virginia Health, Charlottesville, VA, [2]University of Virginia, Charlottesville, United States of America

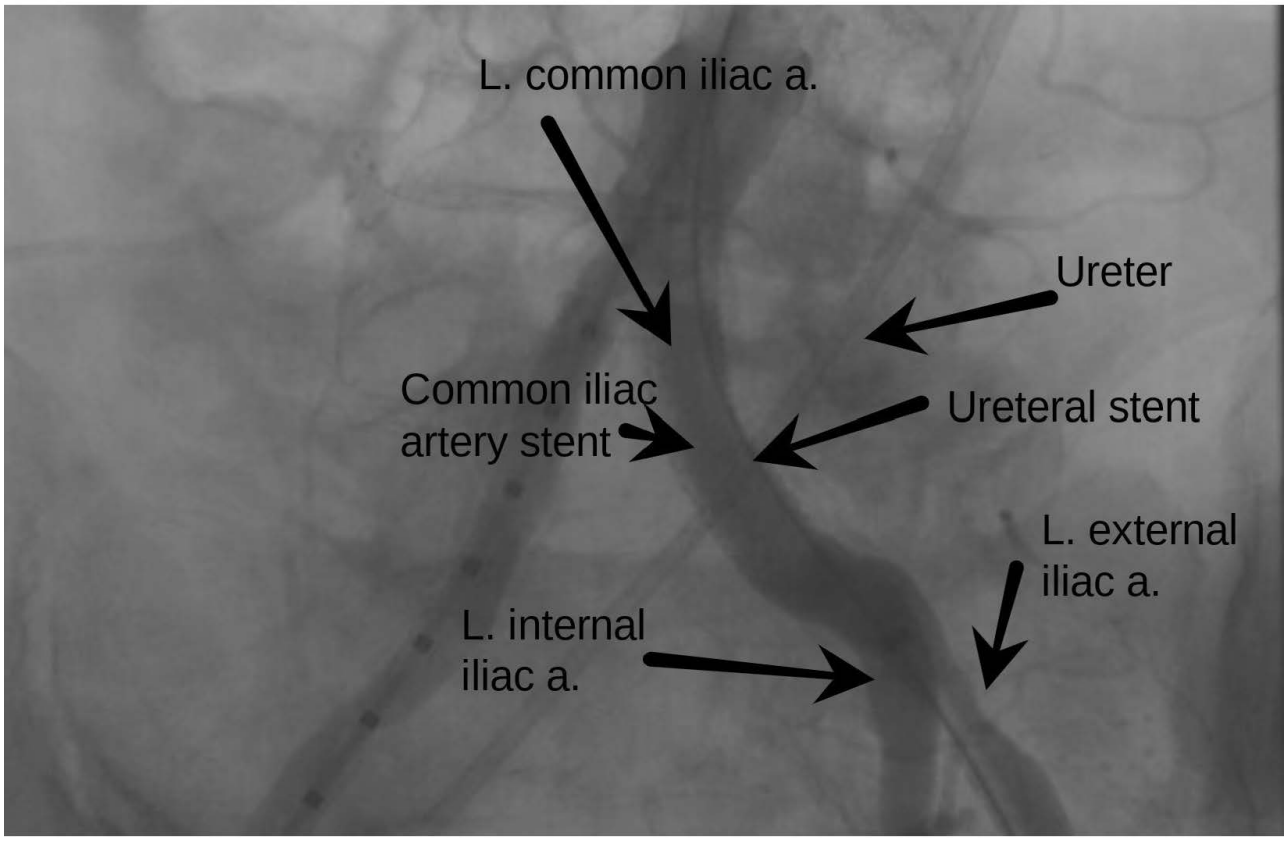
Introduction: Anesthesiologists will invariably encounter rare, undiagnosed pathologies during otherwise routine general procedures, especially in geriatric oncologic populations 1. Utero artery fistulas (UAFs) are known but exceptionally rare complications of ureteral manipulation that can cause rapid and profound hemodynamic instability 2. More common in elderly patients with ureteral or vascular stenting, patients commonly have a history of abdomino-pelvic cancer and/or pelvic radiation 3. UAFs are difficult to diagnose due to the prolonged intermittent hematuria (3-21 days) before hemodynamically threatening hemorrhage develops 4. UAFs are better described in non-anesthesiology medical literature (urology, vascular surgery, and interventional radiology), but they are life-threatening emergencies requiring prompt diagnosis, clinical stabilization, and management. In addition to recognizing clinical deterioration, anesthesiologists play an integral role within the multidisciplinary care of these events, often leading the initial stabilization and resuscitation while coordinating more definitive procedures with other specialists. Here, we present the case of a previously undiagnosed UAF in a geriatric oncologic patient presenting for routine retrograde ureteral stent exchange. We seek to emphasize the instrumental role anesthesiologists play during unanticipated, rare, and hyperacute intraoperative events requiring a multidisciplinary care team for treatment.

Methods: AZ, a 74 year-old patient with past medical history notable for cervical cancer status-post chemoradiation and total pelvic supralevator exenteration complicated by persistent left hydronephrosis requiring ureteral stenting, presented to the emergency department with excruciating flank pain and gross hematuria from her ileal conduit. AZ had no prior episodes of hematuria. AZ was admitted and underwent ileal conduit cystoscopy under light sedation in the main operating room. Nearing the end of the case, gross hematuria was again appreciated while attempting to remove the ureteral stent. AZ became profoundly hemodynamically unstable with associated acute mental status deterioration, necessitating conversion to general anesthesia. Aggressive resuscitation was initiated with crystalloid fluids at first because the patient did not have an active 'type and screen' for this low risk procedure. Norepinephrine and vasopressin infusions were required to maintain AZ's mean arterial blood pressure above 60 mmHg. During AZ's stabilization and resuscitation, extensive discussion between the anesthesiology team and our urology and interventional radiology colleagues culminated in emergently transporting the patient from the main operating room to interventional radiology for angiogram and stent removal/replacement. While awaiting room readiness, additional large bore IV access and a radial arterial line were placed. After transport, AZ's angiogram revealed an active ureteral-iliac fistula. A 9x38 iCast stent was placed in AZ's left common iliac artery with appropriate angiographic result. AZ required continued high dose vasoactive infusions in addition to 4.5L of plasmalyte and multiple units of blood products during the procedure. Her vasoactive agents were weaned on post-operative day 1, and she was successfully extubated on postoperative day 2 after aggressive fluid diuresis. She underwent successful percutaneous nephrostomy placement on postoperative day 6, and she was ambulating at her baseline on room air on the day of discharge on post-operative day 7.

Conclusion: Anesthesiologists are often the first to recognize and treat acute hemodynamic collapse. Though impossible to predict every possible intraoperative specialty-specific event, the anesthesiologist should be able to stabilize, resuscitate, and recruit other specialties for definitive management. The anesthesiologist should always be vigilant for hyperacute, rare, and unanticipated intraoperative decompensation, even during routine cases under minimal sedation. Lastly, the anesthesiologist should be aware that geriatric patients, particularly geriatric patients with oncological

histories and those who have undergone radiation, may be especially predisposed to these types of life-threatening and extraordinarily rare pathologies, and they're likelier to suffer serious medical complications should they result.

References: 1. Geriatr. Oncol (2012), 3(2), 147-162. 2. Eur J Vasc Endovasc Surg (2001), 22, 191-196. 3. Curr Urol (2018), 12, 54-56. 4. J Vasc Surg (1986), 3(1), 155-158.



Global Health

Global Health - 1 Clonality analysis of clinical isolates of Pseudomonas aeruginosa: the outbreak of high-risk clone ST357

Toshihito Mihara[1], Atsushi Kainuma[2], Koichi Akiyama[3], Junya Ohara[1], Keita Inoue[4], Mao Kinoshita[2], Masaru Shimizu[5], Teiji Sawa[2]

[1]Kyoto Prefectural University of Medicine, Kyoto, Japan, [2]Kyoto Prefectural University of medicine, Kyoto city, Kyoto prefecture, [3]Yodogawa Christian Hospital, Osaka, Osaka, [4]Kyoto Prefectural University of Medicine, Kyoto city, Kyoto prefecture, [5]Uji-Tokushukai Medical, Kyoto city, Kyoto prefecture

Introduction: Multi-drug resistant Pseudomonas aeruginosa cause severe infections, especially in the elder and immunocompromised patients. It is critical to characterize the epidemiological trends of high-risk P. aeruginosa isolates by continuous monitoring in hospitals. In this study, we performed two genetic analyses, Multi-Locus Sequence Typing (MLST) and Multiple-Locus Variable-number tandem repeat Analysis (MLVA), to check the clonality of clinically isolated antimicrobial-resistant P. aeruginosa strains. Then, we investigated the association between the characteristics of the isolates and the patient background.

Methods: This study was approved by the Medicine Ethics Committee of Kyoto Prefectural University. We investigated the antimicrobial susceptibility profiles of clinically isolated drug-resistant P. aeruginosa and the medical records of drug-resistant infected. Then, we performed clonality tests, MLST and MLVA, for 53 antimicrobial-resistant strains out of 2,119 P. aeruginosa isolated in the hospital between the years 2005 and 2018. In MLST, the DNA sequences of seven housekeeping genes were analyzed to determine the sequence type (ST). In MLVA, 15 genomic regions possessing variable-number tandem repeats (VNTR) were analyzed to determine the MLVA type (MT).

Results: Among 53 isolates, 36 (67.9%) were genotyped as ST357 in MLST and identified to have the same clonality type in MLVA. The most of these ST357 isolates possessed two class one integrons associated resistance to Œ≤-lactams and aminoglycosides. All 53 isolates were fluoroquinolone-resistant, 39 isolates (73.6%) were gentamicin-resistant, and 18 isolates (34.0%) were carbapenem-resistant. None of the isolates satisfied the clinical criteria for MDR P. aeruginosa. Comparison of ST357 and other STs revealed that a significantly higher proportion of ST357 isolates was resistant to gentamicin ($p = 0.006$). Among 53 patients, 13 patients (24.5%) died during the period of hospitalization. The mortality rates of the patients who harbored with ST357 and other ST isolates were 19.4% (n = 7/36) and 35.3% (n = 6/17), respectively (not significant).

Conclusion: In the P. aeruginosa outbreak survey, prevalent STs such as ST357 and ST235 were associated with fluoroquinolone resistance, class 1 integron-associated resistance to Œ≤-lactams and aminoglycosides, and cytotoxic exoU (+) genotypes. It is crucial to evaluate epidemiological trends for high-risk P. aeruginosa isolates by continuous hospital monitoring.

Neuroscience in Anesthesiology and Perioperative Medicine

Neuroscience in Anesthesiology and Perioperative Medicine - 1 Case Report of suspected diabetes insipidus during pineal tumor resection

Philip Lin[1], Maya Mikami[2]

[1]Columbia University Irving Medical Center, New York, NY, [2]Columbia University, New York, NY

Introduction: Intracranial germinomas are a relatively rare cancer, commonly arising from pineal and suprasellar regions1. Pineal lesions generally present with symptoms of increased intracranial pressure2. Central diabetes insipidus (DI) is characterized by symptoms of polyuria, plasma hyperosmolality and urine hypoosmolality due to vasopressin deficiency caused by a lesion at hypothalamic-hypophyseal tract3. However, central DI has even been observed in patients with pineal region germinomas4. Here we present a case of suspected intraoperative DI during pineal tumor resection.

Methods: A 15-year-old male with known pineal germinoma presented with acutely worsening neurologic symptoms including lid lag, vomiting and headache with interval worsening of the tumor's localized mass effect on the midbrain. He was scheduled for parietal craniotomy with interhemispheric transtentorial approach. After standard monitors were applied, he was induced for general anesthesia. A foley catheter was placed and 2.1 Liters of urine were collected within initial hour. Also noted was an elevated serum lactate level of 2.34 mmol/L, which increased intraoperatively to a peak of 6.36 without acidemia. Urine output (UOP) continued at a rate of approximately 300 mL/15 min, with a concurrent rise in serum sodium to 149 mEq/L from a preoperative baseline of 130s. Initial intraoperative urine osmolality was low at 214 mOsm/kg, compared to 660 prior to surgery. Suspected DI was discussed with the neurosurgery team, but treatment with ddAVP was deferred given hemodynamic stability and a below 150 mEq/L serum sodium level. As resection of the pineal tumor progressed, UOP appeared to decrease and urine osmolality improved to 560 mOsm/kg. Serum lactate and sodium stabilized around 5 mmol/L and low 140s mEq/L respectively. Lactated ringer's solution was used throughout the case to replete UOP 1:1 and blood loss was minimal (50 mL).

Conclusion: There are 2 phenomena that occurred during this case that are interesting and warrant further discussion: a hyperlactatemia and a suspected DI of unconfirmed etiology acutely developed and resolved intraoperatively. Pineal tumors could cause hypothalamic dysfunction by mass effect, loss of pineal inhibitory influences on the hypothalamus, or metastatic extension to the hypothalamus5. Per chart review, the patient did have remote history of transient DI after a brain biopsy. In retrospect, it may be prudent to anticipate intraoperative DI in patients with known pineal tumor who have both previous history of DI and interval worsening of mass effect. Regarding intraoperative hyperlactatemia, brain tumor metabolism and association with tumor malignancy to hyperlactatemia has been reported. Malignancy could explain the elevated lactate given his acute increase in tumor size and final pathology report.

References: 1: Journal of Neurosurgery. 63, 155-167, 1985. 2: International Journal of Radiation Oncology, Biology, Physics. 39, 419-426, 1997. 3: Journal of Clinical Endocrinology and Metabolism. 82, 1362-1367, 1997. 4: Neuro Oncology. 6(2): 127-133, 2004. 5. Clinical Pediatrics. 39, 693-695, 1998.

Neuroscience in Anesthesiology and Perioperative Medicine - 2 Now that's a Clot: Anesthetic Management of COVID-19 Recovered Stroke for Carotid Endarterectomy

Shashidhar Manchegowda[1], Michael A Chyfetz[2]

[1]Jackson Memorial Hospital, Miami, FL, [2]Montefiore Medical Center; Albert Einstein College of Medicine, Bronx, NY

Introduction: COVID-19 is a systemic disease characterized by an innate immune response predisposing to systemic inflammation, hypercoagulability and localized endotheliitis. A high degree of intraluminal carotid thrombosis resulting in large clot and subsequent stroke burden was commonly seen in our institution as a COVID-19 sequala. A 57-year-old female past medical history of hypertension and hyperlipidemia presented with encephalopathy and altered mental status approximately two weeks after diagnosis with COVID-19. She initially presented with generalized malaise without respiratory symptoms and underwent supportive treatment at home. Imaging demonstrated left MCA hypodensities with angiographically evidence of approximately 2 mm length of complete ICA occlusion. Patient was admitted to stroke neurology with weakness and aphasia and despite dual antiplatelet management symptoms failed to improve. Given the risk of ongoing embolic events, the patient was scheduled for carotid endarterectomy with intraoperative neuromonitoring and consented for ligation of Internal Carotid Artery if clot was unamendable to surgical removal.

Methods: Patient was brought to operating room and standard ASA monitors applied. Prior to induction, systolic blood pressure noted to range from 220-240 mmHg. After uneventful induction and intubation, arterial line was placed for hemodynamic monitoring and anesthesia maintained with EEG targeted Remifentanil and Propofol infusion and phenylephrine infusion for MAP control. During surgical dissection patient was noted to have brief episode of asystole resolving with cessation of surgical stimulation. Surgeon infiltrated carotid sinus with lidocaine and patient tolerated cross clamping without any major hemodynamic, EEG or SSEP changes. Intraoperatively, a long segment of plaque with near occlusion at the carotid bifurcation was removed. The patient was weaned from anesthesia, extubated, and taken to recovery room in stable condition.

Conclusion: The COVID-19 pandemic demonstrated that underlying vascular risk factors predispose to the development of cerebrovascular strokes with larger clot burden and worse clinical outcomes. This case highlighted the development of a complete carotid occlusion in a COVID-19 recovered patient. Given the risk of embolic propagation of clot and intracranial reconstitution of distal flow, consent was obtained for carotid ligation without a balloon occlusion test. This case demonstrates the unique pathophysiology of COVID-19. Future research is required to fully understand the end-organ effects of this devastating disease and the utility of prophylactic anticoagulation in high-risk patients.

References: Esenwa, C., et al. (2020). 'COVID-19-Associated Carotid Atherothrombosis and Stroke.' AJNR Am J Neuroradiol 41(11): 1993-1995. Indes, J. E., et al. (2020). 'Early experience with arterial thromboembolic complications in patents with COVID-19.' J Vasc Surg.

Neuroscience in Anesthesiology and Perioperative Medicine - 3 IARS General Abstract Submission - 0587

Casey T Carr[1], Carolina B Maciel[2], Katharina Busl[2], Marc Babi[2]

[1]University of Florida, Gainesville, FL, [2]University of Florida, Gainesville, United States of America

Introduction: Intracranial pressure (ICP) monitoring and management are cornerstones of severe traumatic brain injury (TBI). Cerebral edema and intracranial hypertension can also occur after cardiac arrest due to reperfusion injury and associated vasogenic edema. In patients with concomitant hypoxic ischemic brain injury and severe TBI, elevated ICP can be challenging to interpret, but management would differ significantly depending on etiology. We present a case of concomitant severe TBI and hypoxic brain injury, with occurrence of compartmentalized intracranial hypertension.

Methods: A 35-year-old male presented to the emergency department after a fall from a roof after a lightning strike. On initial assessment, he was in pulseless electrical activity. Return of spontaneous circulation was obtained after approximately 8 minutes and the patient was intubated. Trauma evaluation revealed C3 and C7 spinous process fractures, complex T3 - T5 chance fractures with an associated T4 spinal cord transection, skull base fracture, and bilateral pulmonary contusions. Head CT on admission showed of the gray white matter differentiation, subarachnoid hemorrhage in the interpeduncular cistern, and no findings suggestive of anoxic changes. He had an episode of tonic seizure activity and was started on levetiracetam and continuous EEG. An intraparenchymal ICP monitor was placed, and his ICP was 14 mm Hg. On hospital day 3, his ICP suddenly increased to >150 mm Hg, with a consistent waveform. He was treated with hyperosmolar therapy, additional sedation, and neuromuscular blockade, but had continued elevated ICP, consistently above 100 mm Hg. During this time, he had no change in neurologic examination with maintained pupillary reflexes. Repeat head CT showed new hypodensity involving the occipital lobes - pattern more consistent with hypoxic ischemic injury. His ICP monitor was positioned in the right frontal lobe. His lack of response to ICP lowering medications, presence of ischemia on head CT, lack of neurologic examination change were all examples of intracranial hypertension isolated to the area of brain parenchyma surrounding the ICP monitor. His EEG continued to show reactivity and a pattern consistent with viable cortex, despite his elevated ICP. These data prove intracranial compartmentalization.

Conclusion: This case illustrates the challenge of ICP management in patients with combined hypoxic and traumatic brain injury. Management guided by ICP measurements must also be understood in the context of limitations and complexities of ICP monitoring systems and neurophysiology. Under normal circumstances, cerebral blood flow (CBF) - and therefore ICP - is inversely proportional to arterial blood pressure. However, during a state of impaired cerebral autoregulation, increases in arterial blood pressure lead to cerebral blood flow. This results in hyperemia, vasogenic edema, and a rise in ICP. This leads to a critical question when a rise in ICP occurs - is this loss of autoregulation or is this intra cranial hypertension or compartment syndrome from another cause? In patients such as the one presented, the knowledge of this limitation is critical, as interventions for either problem are different and oftentimes significantly dichotomous. While interventions to lower ICP are necessary and time critical, acknowledging the limitations of ICP monitoring and the possibility of compartmentalized intracranial hypertension and neuronal cell death is equally important.

Neuroscience in Anesthesiology and Perioperative Medicine - 4 Attempted Neuro-Interventional Radiology Thrombectomy Immediately after Hemiarch Replacement Surgery

Kenji Tanabe[1], Kelly Aunkst[2], Claudia Clavijo[3], Maung Hlaing[1]

[1]University of Colorado, Denver, CO, [2]University of Colorado School of Medicine, Aurora, CO, [3]University of Colorado, Aurora, CO

Introduction: Thoracic aortic surgery is associated with high neurologic morbidity related to the risk of stroke. In this report, we present a case of major embolic stroke during aortic arch surgery and review the neuromonitoring findings that prompted an early diagnosis and workup.

Methods: An eighty year-old female with prior cerebellar stroke without residual deficits, severe aortic stenosis, and ascending aortic aneurysm was scheduled to undergo an aortic valve replacement and hemiarch procedure. Following a routine induction, baseline values for multi-channel electroencephalography (EEG) and somatosensory evoked potentials (SSEP's) were established. During aortic cannulation, subtle changes were noted on the EEG: smaller amplitudes on the right as compared to the left without any correlating SSEP changes. Following the initiation of cardiopulmonary bypass and patient cooling, the left lower extremity SSEP exhibited a more pronounced increase in latency, decrease in amplitude, and signal attenuation as compared to other extremities. The aortic valve and hemiarch were then replaced during a two-hour bypass period with eleven minutes of circulatory arrest. During rewarming, the left lower extremity SSEP signal did not return, the left upper extremity SSEP signal demonstrated increased latency and decreased amplitude compared to baseline, and the EEG remained asymmetric with smaller amplitudes on the right side. Due to these concerning changes, the patient was urgently transported to neuro-interventional radiology (IR) prior to chest closure. Cerebral arteriography demonstrated multiple right middle cerebral artery occlusions in the M3 segment, all too distal for embolectomy. The patient then returned to the operating room for chest closure. Brain MRI on post-operative day two demonstrated extensive multi-focal subacute infarctions in both cerebral hemispheres. The patient's neurologic exam remained limited to withdrawal from pain on her right side. The family elected to palliatively extubate the patient on post-operative day five.

Conclusion: As demonstrated by this case, the combination of neuromonitoring modalities is more effective than one modality alone during aortic arch surgery. After identifying a pattern of neuromonitoring changes concerning for right sided stroke, the anesthetic and surgical teams worked to facilitate a rapid neurologic workup. After separating from cardiopulmonary bypass, the cardiac surgery team worked to expeditiously decannulate the patient. Additionally, the decision was made to proceed directly to neuro-IR with an open chest. With these actions, only seventy-five minutes elapsed between separating from cardiopulmonary bypass and the cerebral arteriogram. This placed the patient well within the six-hour window for mechanical thrombectomy and minimized time to potential reperfusion, a known prognostic factor for stroke recovery. Unfortunately, the diffuse nature of this patient's emboli precluded thrombectomy.

References: 1. Goldstein LJ, Davies RR, Rizzo JA, et al. Stroke in surgery of the thoracic aorta: incidence, impact, etiology, and prevention. J Thorac Cardiovasc Surg. 2001;122(5):935-45. 2. Liu H, Giorgio AMD, Williams ES, Evans W, Russell MJ. Protocol for electrophysiological monitoring of carotid endarterectomies. Journal of Biomedical Research. 2010;24(6):460-466. 3. Jahan R, Saver JL, Schwamm LH, et al. Association Between Time to Treatment With Endovascular Reperfusion Therapy and Outcomes in Patients With Acute Ischemic Stroke Treated in Clinical Practice. JAMA. 2019;322(3):252-263. 4. Shreve L, Kaur A, Vo C, et al. Electroencephalography Measures are Useful for Identifying Large Acute Ischemic Stroke in the Emergency Department. Journal of Stroke and Cerebrovascular Diseases. 2019;28(8):2280-2286.

Obstetric Anesthesiology

Obstetric Anesthesiology - 1 Anesthetic Management of Placenta Percreta with Bladder Invasion: A Case Report

Anthony Alexander[1], Bobby Houston[1], Ned Nasr[1], Gennadiy Voronov[1]

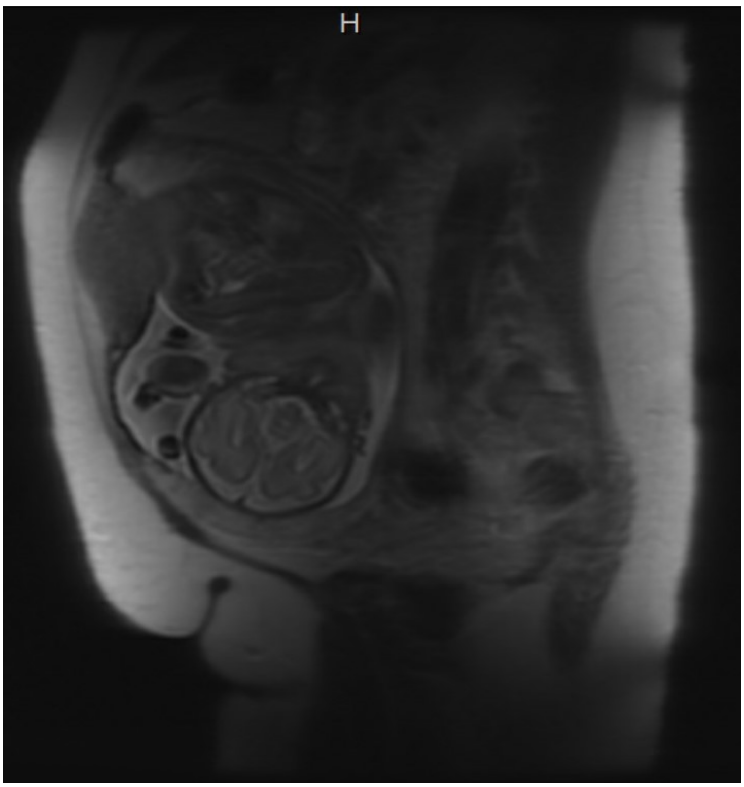
[1]J. Stroger Hospital of Cook County, Chicago, IL

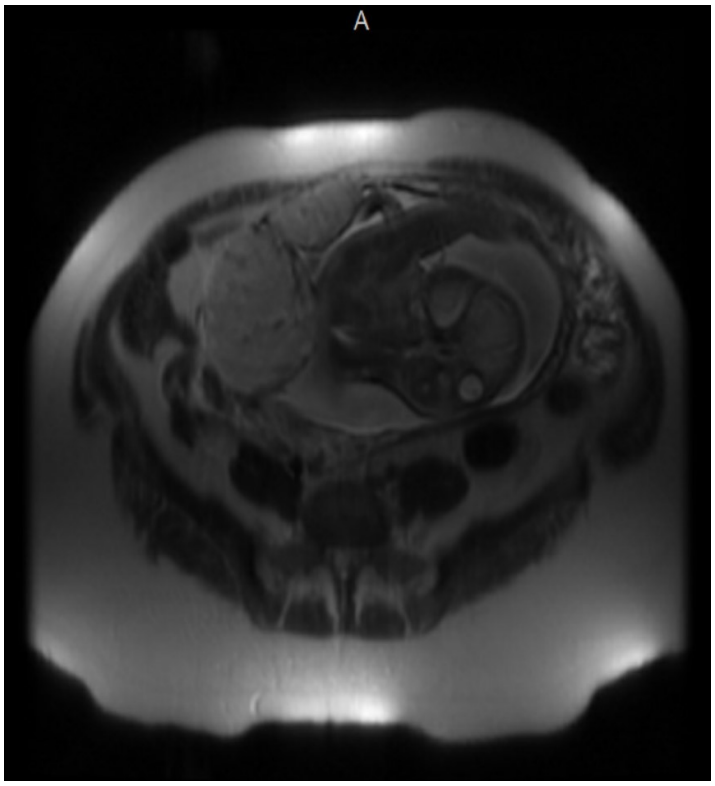
Introduction: Morbidly adherent placenta (MAP) is the term used to describe the spectrum disorder in which the placenta abnormally attaches to and penetrates the uterine wall during pregnancy [1]. This spectrum includes placenta accreta (chorionic villi adhere to myometrium), increta (chorionic villi penetrate the myometrium), and percreta (chorionic villi invade through the myometrium and potentially attach to surrounding organs) [2]. This disorder occurs at a rate of 1:500 cases and is a leading cause of life-threatening hemorrhage during childbirth [3,4]. Within the spectrum, percreta has the lowest incidence, but highest mortality rate ranging from 5-10% [4-6]. Optimal management requires a multidisciplinary approach, with anesthesia playing a central role in coordinating care throughout the perioperative period. In this case report, we present a 34-year-old woman with placenta percreta complicated by invasion of the bladder dome. We will discuss the importance of having a multidisciplinary treatment approach to this disorder, emphasizing anesthetic management.

Methods: A 34-year-old female, gravida 7 with a history of 4 previous cesarean sections presented for routine prenatal care. Ultrasound done at 16 weeks gestation was suspicious for placenta accreta. The degree of abnormal placentation was further confirmed at 31 weeks 4 days via an MRI of the pelvis demonstrating placenta percreta with bladder dome invasion. A preoperative cystoscopy demonstrated enhanced vasculature in the dome of the bladder. She was scheduled for elective cesarean hysterectomy at 33 weeks gestation. Anticipating potential massive blood loss, a type and screen was done, and blood products were delivered to the room prior to induction. Intravenous access was secured with two large-bore peripheral catheters. An arterial line was placed for invasive hemodynamic monitoring and serial blood draws. Cesarean delivery was achieved via a combined spinal- epidural anesthetic technique. A female neonate with APGAR 9/9 and weight 1970g was delivered and resuscitated by the attending neonatologist. An oxytocin infusion was started to ensure adequate uterine tone. The hysterotomy was then sutured closed and general anesthesia was induced for the hysterectomy portion of the procedure. Rapid sequence induction with propofol and succinylcholine was done, and the airway was secured uneventfully with GlideScope laryngoscopy. Placental invasion into the muscularis of the bladder was confirmed during surgical dissection. A 5 x 7 cm portion of the bladder dome was resected due to abnormal invasion of the placenta resulting in significant blood loss requiring immediate transfusion. Blood loss for the procedure was estimated at 3.3L. The patient received 4L crystalloids, 4U PRBC, 3U FFP, one pooled unit of platelets, 260 ml cell saver and 500 ml albumin. 1 g of tranexamic acid (TXA) was given to assist with hemostatic control. A phenylephrine infusion was titrated to a maximum rate of 70 mcg/min and weaned and discontinued by the end of the procedure. Due to the extent of blood loss and fluid resuscitation, the patient was taken to the SICU intubated to monitor for post-operative respiratory and hemodynamic compromise. She was extubated uneventfully on post-operative day 0 in the SICU.

Conclusion: A multidisciplinary approach with contributions from obstetrician, neonatologist, urologist, radiologist, blood bank physician and anesthesiologist is key to improving patient outcome. Diagnosis in the antenatal period using ultrasound or MRI and performing a cesarean hysterectomy are standard of care [6-9]. With percreta, the placental bed is fed with an extrauterine blood supply and there is invasion of surrounding organs, making efforts to mitigate blood loss more difficult than accreta and increta [7,8]. In anticipation of hemorrhage superimposed on pregnancy-associated anemia, it is essential to monitor preoperative hemoglobin and optimize when appropriate [7]. Good vascular access and placement of an arterial line are vital for adequate resuscitation and hemodynamic monitoring. A rapid blood infuser, cell salvager, and blood products readily available in the room prior to surgery are critical if a massive transfusion protocol is initiated [7,10-11]. With the high likelihood of hemorrhage and longer surgical duration with a cesarean hysterectomy compared to routine cesarean, general anesthesia may be more ideal than regional [8].

References: 1. Ultrasound Obstet Gynecol. 2018;51(2):165-166. 2. Rev Urol. 2009;11(3):173-6. 3. Saudi J Anaesth. 2016 Jul; 10(3):332-4. 4. Cent Asian J Glob Health. 2019 ;8(1):345. 5. JA Clinical Reports. 2019; volume 5, Article 9. 6 Int J Womens Health. 2012;4:451-4. 7. Anesthesiology. 2011;115(4):852-7. 8. Journal of Obstetric Anaesthesia and Critical Care. 2018;8(2), 99. 9. Am J Obstet Gynecol. 2018;219(6):B2-B16. 10. Indian J Anaesth. 2014 Sep;58(5):590-5. 11.Product Replacement: Obstetric Hemorrhage. California maternal quality care collaborative. 2015.





Obstetric Anesthesiology - 2 Neuraxial Anesthesia for C-section in a Morbidly Obese Parturient with Severe Dilated Cardiomyopathy

Seung S Choi[1], Michael Wong[2], Shobana Bharadwaj[1], Jessica Galey[3], Ashanpreet Grewal[1]

[1]University of Maryland Medical Center, Baltimore, MD, [2]University of Maryland, Baltimore, United States of America, [3]University of Maryland School of Medicine, Baltimore, MD

Introduction: With an incidence of 5-8 per 100,000 live births, dilated cardiomyopathy (DCM) in pregnancy is rare with potentially disastrous fetal and maternal complications.1, 2 Caring for such patients requires careful planning with a multidisciplinary approach. We present a case of cesarean section under regional anesthesia in setting of severe DCM complicated by acute exacerbation.

Methods: A 31-year-old African American female G5P2114 with history of chronic hypertension, type 2 diabetes, DCM, and morbid obesity (BMI of 62) presented with dyspnea due to volume overload at 37 weeks estimated gestational age. Her previous c-section for twin pregnancy was complicated by preeclampsia and DCM diagnosed nine months postpartum. On admission, transthoracic echocardiogram (TTE) showed severely dilated LV with global hypokinesis (LVEF 20%), moderate MR, mild to moderate TR, and mild pulmonary hypertension (RVSP 45 mmHg). A multi-disciplinary team decision was made to optimize maternal hemodynamics with diuretics, deliver via c-section after resolution of maternal symptoms, and recover in cardiac intensive care unit (CCU) when obstetrically stable post-delivery. Anesthetic management included pre-induction intra-arterial and central venous access, combined spinal epidural with Intrathecal opioids and incremental dosing of local anesthetic via epidural catheter. Low dose phenylephrine infusion started during epidural dosing was weaned off after delivery. Intravenous furosemide was administered soon after delivery when there was no concern for on-going hemorrhage to offload volume secondary to auto-transfusion from uterine contraction. Intraoperatively, patient remained hemodynamically stable with no change in cardiac output. However, in the recovery room, patient became hypotensive and hypoxemic with low PaO2 and SvO2, and worsening LV function on repeat TTE. Dobutamine infusion was started, and with additional diuresis the acute exacerbation resolved over 24 hours in the CCU. Patient was discharged with her healthy neonate on postoperative day 5.

Conclusion: The timing of this patient's DCM diagnosis was not consistent with peripartum cardiomyopathy. DCM is more prevalent in African Americans, and 50% of cases are idiopathic.1 Both maternal and fetal morbidity and mortality correlate with the severity of the disease.2 Perioperatively, TTE, arterial line, and cardiac output monitoring may be helpful.3 With close control of hemodynamics, neuraxial anesthesia can be utilized in patients with DCM, avoiding the risk of general anesthesia and positive pressure ventilation.1, 3 Risk of decompensation is elevated through the third trimester to postpartum, as shown in our patient in whom auto-transfusion and increased preload were poorly tolerated.3 This case demonstrates the challenges of severe cardiomyopathy in pregnancy. Such patients deserve comprehensive multidisciplinary planning and vigilant anesthetic management to ensure optimal outcomes.

References: 1. Lewey J, Haythe J. Cardiomyopathy in pregnancy. Semin Perinatol. 2014;38(5):309-317. doi:10.1053/j.semperi.2014.04.021 2. Luthra A, Bajaj R, Jafra A, Jangra K, Arya VK. Anesthesia in pregnancy with heart disease. Saudi J Anaesth. 2017;11(4):454-471. doi:10.4103/sja.SJA_277_17 3. Arendt KW, Lindley KJ. Obstetric anesthesia management of the patient with cardiac disease. Int J Obstet Anesth. 2019;37:73-85. doi:10.1016/j.ijoa.2018.09.011

Obstetric Anesthesiology - 3 Post Cesarean Section Respiratory Failure

Naveen Shetty[1]

[1]*NYU, New York, NY*

Introduction: In this medically challenging case, we describe a patient who suffered from acute respiratory failure in the PACU after a cesarean section done under general anesthesia. We emphasize unique considerations when taking care of an obstetrical patient, and describe our institutional response to make this less likely to occur in the future.

Methods: A 40 years old female, G4P0121, presented to L&D at 37w3d gestation. Her history included a prior cesarean section, and pre-eclampsia (PEC) with severe features. She was noted to be hypertensive (152/101) with a platelet count of 73,000. Induction of labor was begun. After a lengthy discussion of the risks and benefits of neuraxial anesthesia, it was ultimately decided to forego epidural placement, given the thrombocytopenia. Our team placed the patient on an intravenous fentanyl PCA for labor analgesia. A magnesium sulfate infusion was started as well, with serial checks for toxicity. Subsequently, the decision was made to proceed with immediate delivery via cesarean section given persistent category 2 fetal heart tracings. The patient was brought to the operating room, and underwent an uncomplicated cesarean section under general anesthesia with endotracheal intubation (GETA). RSI with succinylcholine was performed uneventfully, followed by a single dose of rocuronium. The patient received a total of 1.4mg of hydromorphone intraoperatively. After reversing neuromuscular blockade with neostigmine and glycopyrrolate, the patient was extubated, and brought back to her labor room for recovery. She was noted to be somewhat sleepy, but easily arousable to voice. She was placed on an oxygen facemask at 5LPM, and was signed out to the labor recovery RN. Approximately 30 minutes later, the patient was found to be apneic, with an oxygen saturation of 60%. The anesthesia team arrived at bedside, and the patient was mask ventilated with improvement in oxygen saturation. 400 mcg of naloxone was given, without clinical improvement. The patient was reintubated and IV calcium gluconate given for possible magnesium toxicity. The patient was immediately taken for a CT scan of the brain, which was negative for acute pathology. An ABG was obtained: pH: 6.8

Conclusion: The presumed diagnosis was acute hypercarbic respiratory failure, resulting from perioperative opiates, magnesium, residual neuromuscular blockade, or a combination. It is likely that her extreme hypercarbia depressed her mental status, making her clinically unresponsive to naloxone. This patient was on an IV PCA fentanyl regimen for labor pain, due to a contraindication to epidural placement. Given fentanyl's long context-sensitive half-life, care should be taken when dosing intraoperative opiates, should such a patient require a caesarean section under general anesthesia. Due to reduced FRC and increased oxygen consumption, pregnant patients are exquisitely sensitive to systemic opioids. Remifentanil may be a better alternative in these situations. Furthermore, this patient's hypoventilation in the PACU eventually led to hypercapnic respiratory failure, lost of consciousnesses, and eventually hypoxia. This occurred over a 30 minute period. Had the patient been stimulated by staff to take breaths, this situation could have been avoided. Of note, pulse oximetry is a lagging indicator of respiratory depression, particularly when supplemental oxygen is used. Our institution implemented mandatory EtCO2 monitoring, with an alarm, for all patients recovering after a cesarean section performed with a general anesthetic. Many institutions recover these patients on the L&D floor; however, it may be prudent to transport higher risk patients to a traditional PACU environment, where the staff is more accustomed to caring for general anesthesia patients, and the environmental stimulation is higher. Finally, it is critical to remember that magnesium can potentiate the effects of non-depolarizing muscle relaxants. This occurs even without toxic doses of magnesium. These factors, which are unique to the obstetrical patient, underscore the risks of GETA in this population, and should be weighed appropriately when making a risk vs. benefit analysis compared to neuraxial techniques.

References: McGlennan A, Mustafa A.¬†General anaesthesia for caesarean section. Contin Educ Anaesth Crit Care Pain.¬†2009;9:148-151

Obstetric Anesthesiology - 4 Unexpected High Spinal Anesthesia From Tetracaine Combined Spinal And Epidural Anesthesia For Cesarean Section, A Case Report.

Akiko Komura[1], Samuel Dejoy[2]

[1]Case Western Reserve University/Metrohealth Medical Center, Eastlake, OH, [2]Case Western Reserve University/Metrohealth Medical Center, Cleveland, OH

Introduction: A 28-year-old primagravida with 35 weeks gestation, height of 163 cms weight of 171 kg (BMI65), presents for an urgent primary lower segment cesarean section in view of dichorionic-diamniotic twin intrauterine pregnancy, twin A with malpresentation and intrauterine growth restriction (less than 3rd percentile), short cervix, premature preterm rupture of membrane, preterm labor with 4-5cm cervical dilation. She was nil per oz for more than 8 hours. Patient has a history of intermittent mild asthma, and hip osteoarthritis. Examination of pulmonary, cardiovascular, central nervous system revealed no abnormalities, and laboratory testing revealed mild anemia, Hg 10.6, Hct 32.4, with other values within normal limits.

Methods: After taking informed consent, spinal anesthesia was planned for this patient. Monitors were connected, and baseline vitals were recorded. Due to body habitus, we encountered difficulty with spinal anesthesia. Combined spinal epidural anesthesia was planned. In a sitting position, a dose of tetracaine (isobaric, 1%) 10 mg, fentanyl 20 mcg and morphine 0.5 mg/ml 200mcg was administered intrathecally. The patient was then immediately helped to supine position with left side tilt. Within 1 minute of spinal anesthesia dosing, she immediately developed dyspnea, as well as weakness and parasthesia of bilateral upper extremities. She was able to speak coherently at this time. For a suspected high spinal anesthesia, oxygen was administered, and the decision was made to proceed with general anesthesia. Rapid sequence induction with propofol and succinylcholine was administered for induction. The babies were delivered immediately. Intraoperative hypotension was treated with IV fluid bolus, phenylephrine bolus and infusions. After closing the wound, she was extubated without any respiratory or hemodynamic compromise, and transferred to post anesthetics care unit.

Conclusion: Epidural, spinal, combined spinal and epidural anesthesia, and general anesthesia are the anesthetic techniques commonly utilized for cesarean deliveries. The decision to choose one technique over others should be individualized based on anesthetic, obstetric, and fetal risk factors as well as patient's preference and anesthesiologists' clinical judgment. The current practice guideline for obstetric anesthesia from ASA task force and SOAP recommends neuraxial techniques in preference to general anesthesia for most cesarean sections1. High spinal or complete spinal block is a known complication of spinal or epidural anesthesia. The SOAP SCORE project reported the incidence in the obstetric population in the United States was 1 in 4336 blocks2. Parturients demonstrate increased sensitivity to both regional and general anesthetics3. Among parturients, associated characteristics for high spinal include obesity, spinal technique after failed epidural anesthesia2. High spinal/regional block is defined as a spread of local anesthetics affecting the spinal nerves above T4 level. The effects are of variable severity depending upon the maximum level that is involved. Blocking T1-4 levels of cardiac sympathetic fibers leads to hypotension and bradycardia. At C6-8 levels, high spinal leads to parasthesia or numbness and weakness of hands and arms. At C3-5 levels, it results in shoulder weakness, respiratory compromise with hypoventilation, desaturation, and it may lead to respiratory arrest. Intracranial spreads into brainstem leads to slurred speech, sedation, and loss of consciousness. The high spinal block in this case report is likely due to an effect of local anesthetic cephalic spread related to multitude of factors affecting baricity of the anesthetics. First, density varies inversely with temperature. Lui et al studied effect of body temperature making hypobaric. The reported densities of opioids are as follows: fentanyl 50 mcg/ml is 0.9932. Morphine EPD preservatives free 1mg/ml is 0.9933. Both of these opioids were hypobaric. Densities of commonly used local anesthetics lidocaine, bupivacaine with and without opioids of fentanyl sufentanil, morphine, and meperidine were all hypobaric at 37C, and this was lower than that at 22C. Interestingly, they also demonstrated that density of CSF in pregnant patients

were slightly lower than mean density of all patients of age ranged from 18 to 96 yr (1.00033+0.00010 and 1.00059 + 0.00020 respectively) 4.

References: 1. American Society of Anesthesiologists task force and SOAP: Practice guidelines for obstetric anesthesia. Anesthesiology 2016;124:00-00/ 2. D'Angelo R. Smiley R. Riley E, Segal S: Serious complications related to obstetric anesthesia." Anesthesiology 2014;120, 1505-12. 3. Miller RD, Eriksson LD, Fleisher LA, Weiner-Kronish JP, Young WL, Miller's Anesthesia. 7th ed. Churchull Livingstone Elsevier; 2010. 4. Liu ACP, Polis TZ, Cicutti NJ; Densities of cerebrospinal fluid and pinal anaesthetic solutions at body temperature. Can J Anesth 1998 45:4:297-303 5. Kakazu CZ, Lee C, McDonald JS; Addition of fentanyl alters the baricity of hyperbaric spinal bupivacaine. Anesthesiology 2004; 101:A1071.

Obstetric Anesthesiology - 5 Neuraxial Anesthesia For Cesarean Section With Unrepaired Tetralogy Of Fallot

Karishma Batra[1], Suniti Kale[1]

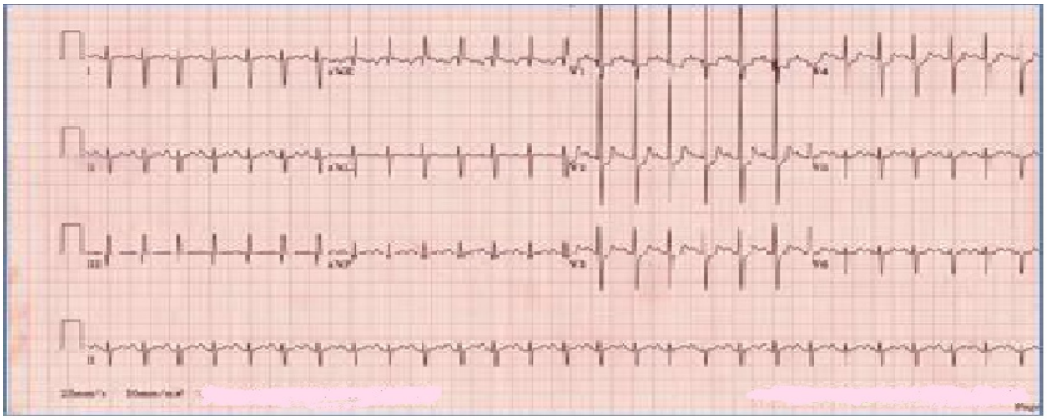
[1]VMMC & Safdarjung Hospital, New Delhi, New Delhi

Introduction: Uncorrected Tetralogy of Fallot (TOF) in pregnancy poses a significant risk to the mother and baby. Due to the additional demands inflicted by physiological changes of pregnancy, symptoms of TOF worsen. General anesthesia was traditionally considered as the anesthetic technique of choice for such patients undergoing cesarean section[1] , however, has been associated with significant feto-maternal morbidity. Although subarachnoid block remains relatively contraindicated[2] , with emerging trends, combined spinal-epidural techniques provide an effective anesthesia for cesarean section, with adequate hemodynamic control. We report the case of a 23 year old parturient with uncorrected TOF managed under combined spinal-epidural anesthesia for cesarean section.

Methods: A 23 year old female, 5th gravida presented at 35 weeks of gestation with chief complaints of dyspnea (grade II) and cyanosis. TOF was diagnosed and tablet Propranolol 20mg was started. There was no chest pain, palpitations, cough, orthopnea/ paroxysmal nocturnal dyspnea.
On examination, she had central and peripheral cyanosis along with grade II clubbing. Pulse was 92/min, Blood Pressure was 116/70 mm Hg, airway-Modified Mallampati Grade II. Peripheral oxygen saturation (SpO_2) was 84% on room air and 88% on a Fraction of Inspired Oxygen Concentration (FiO_2) of 50%. Cardiovascular examination revealed a pansystolic murmur (grade III) in pulmonary and aortic areas. Hemoglobin was 14.1 g%, Hematocrit 41.7%, room air ABG revealed pH 7.440, pO_2 58.4 mmHg, pCO_2 21.4 mmHg, HCO_3 14.3 mmol/L, SaO_2 88.3%, all other hematological and biochemical investigations were within normal limits. ECG revealed features suggestive of Right Ventricular Hypertrophy with sinus tachycardia and poor progression of R wave. Cardiac Echocardiography confirmed the presence of a large subaortic Ventricular Septal Defect with a predominant right to left shunt, aortic override of <50%, severe infundibular pulmonary stenosis with a 85 mmHg gradient, normal biventricular function.
Patient presented for emergency cesarean section in view of fetal growth retardation and non progress of labour. Antiaspiration and infective endocarditis prophylaxis were administered. All intravenous lines were de-aired. Preloading was done with 500 ml Ringer's Lactate while radial artery cannula was being placed on the left side. Combined Spinal-Epidural anesthesia was placed at L3-4 level and 5mg 0.5% hyperbaric bupivacaine with 25 mcg fentanyl administered intrathecally. 8ml of 2% lignocaine was administered in titrated aliquots of 4ml each, every 10 minutes through the epidural catheter till a sensory level of T4 was achieved and surgery was started. After delivery of the baby, injection oxytocin 3U was administered slow intravenously over a period of 30 minutes. The remaining intraoperative period remained uneventful, hemodynamics were maintained with a Mean Arterial Pressure ≥70mmhg and SpO_2 maintained at >92-93% on a FiO_2 of 100%. Blood loss of 800 ml was replaced with 1.5L Ringer's Lactate. Urine Output was 200ml. Patient was shifted to the High Dependency Unit in the postoperative period. Postoperative analgesia was maintained on epidural Morphine 2mg every 24 hours with intravenous Paracetamol 1g 8 hourly and the epidural catheter was removed after 48 hours. Both maternal and fetal outcomes were uneventful.

Conclusion: By choosing a regional technique, we avoided all the cardiorespiratory changes associated with general anesthesia drugs, laryngoscopy and tracheal intubation. Also, we were able to give a higher concentration of oxygen, which is essential, both in obstetrics as well as in a case of cyanotic heart disease. Moreover, the fall in systemic vascular resistance was minimized by decreasing the dose of local anesthetic drug and having a higher dose of opioid, keeping the total volume of drug for subarachnoid block low. Further titration was done with epidural aliquots to achieve an adequate level of block for surgery, thus preventing the fall in blood pressure. Therefore, we felt that Combined Spinal-Epidural Anesthesia provides a relatively safe anesthetic technique for unrepaired TOF undergoing cesarean section. This development in managing unrepaired TOF in pregnancy could significantly reduce the anesthetic risks involved to both mother and the baby.

References: 1. Anesthesia for Cesarean Section. In: Chestnut D.H, editor. Obstetric Anesthesia Principles and Practice. 3rd ed. Philadelphia: Elsevier Mosby; 2004:429-39. 2. Neuraxial anesthesia in obstetrical patients with cardiac disease. Curr Opin Anaesthesiol. 2005;18:507-12.



Obstetric Anesthesiology - 6 Severe Progressive Peripartum Cardiomyopathy and Pulmonary Hypertension During Urgent Pre-Term Cesarean Delivery

Taimoor A Khan[1], Rebecca Anderson[2], George Semien[3], Jean Miles[4], Benjamin Houseman[5]

[1]Memorial Healthcare System, Pembroke Pines, FL,
[2]Memorial Healthcare System, Plantation, FL,
[3]Envision Healthcare Services, Hollywood, FL,
[4]Memorial Healthcare System, Hollywood, FL,
[5]Envision Physician Services, Pembroke Pines, FL

Introduction: Peripartum cardiomyopathy (PPCM) is a significant anesthetic challenge in C-section deliveries. PPCM typically develops in late pregnancy and in the peripartum period, and is diagnosed based on heart failure appearing towards the end of pregnancy, left ventricular systolic dysfunction, and having no other identifiable cause [1]. It is frequently associated with and can worsen underlying pulmonary hypertension, which itself can cause elevated risk of major adverse cardiac event [2].

Methods: A 42-year-old Hispanic female presented with a past medical history of metastatic osteosarcoma treated with left arm amputation, left lobectomy, and chemotherapy, as well as pulmonary embolism, left bundle branch block, and hypertension. She was G3P1011, with a previous C-section performed for pre-eclampsia and one first trimester spontaneous abortion several years prior. Her echocardiogram at approximately 26 weeks gestational age showed an ejection fraction of 40%. Later imaging at 34 weeks GA showed deterioration to an EF of 19% with moderate tricuspid and mitral regurgitation and elevated filling pressures. She had also had interval worsening of her pulmonary hypertension (WHO Group 2) with a pulmonary artery systolic pressure of 60 mmHg. There were elements of restrictive lung disease secondary to her history of chemoradiation, lobectomy, and PE. She was urgently admitted for preoperative optimization with initiation of heparin, diuresis, and beta blocker therapy. She was transferred to the cardiovascular ICU for placement of a pulmonary artery catheter, arterial line, and preparations for possible ECMO, which were challenging due to her lack of peripheral vascular access. The patient was initiated on continuous milrinone to support cardiac contractility without worsening her pulmonary HTN. Anesthesia was induced with etomidate, lidocaine, and norepinephrine, and endotracheal intubation was achieved using succinylcholine and a video-assisted laryngoscopy. The intubation was difficult due to airway edema, which was attributed to peripartum changes. The induction was also complicated by severe hypertension, peaking at 194/130 but which responded well to nitroglycerin. A TEE conducted during the case revealed an EF of 17% with global hypokinesis. Delivery, bilateral tubal ligation, and closure were only marked by persistent tachycardia and moderate hypertension. The patient was transported back to the CVICU where she was extubated several hours later. After 8 days of postoperative and postpartum management, the patient and her baby were safely discharged home.

Conclusion: Having adequate vascular access, invasive monitoring, and an ECMO reserve option are part of a safe strategy in severe cases of PPCM and pulmonary HTN. Preload and afterload must be maintained in a narrow range to prevent cardiovascular decompensation. Adequate diuresis and conservative fluid management can ensure that autotransfusion after delivery does not cause circulatory overload - particularly in a patient with less lung volume. Pulmonary hypertension and restrictive lung disease also worsen the risk of rapidly developing hypoxia and hypercarbia, and thus require careful ventilator management.

References: Eur J Heart Fail. 2010 Aug;12(8):767-78. J Am Heart Assoc. 2017;6(10):e006144. Published 2017 Oct 24

Obstetric Anesthesiology - 7 Acute Fatty Liver of Pregnancy Necessitating an Emergent Liver Transplantation: A Case Report

Patriot Yang[1], Rutuja R Sikachi[2], Madina Gerasimov[3], Greg Palleschi[3]

[1]Zucker School of Medicine at Hofstra/Northwell Program, Manhasset, United States of America, [2]Zucker School of Medicine at Hofstra/Northwell Program, Manhasset, NY, [3]Zucker School of Medicine at Hofstra/Northwell, Manhasset, NY

Introduction: Acute Fatty Liver of Pregnancy (AFLP) is a potentially fatal metabolic disorder in pregnant patients that requires urgent delivery and aggressive medical and anesthetic management of maternal complications associated with acute liver failure

Methods: A 41yo-F G1P0 at 31 weeks gestation presented with nausea, vomiting, pruritus and jaundice. Laboratory findings demonstrated severe transaminitis and signs of acute liver failure Platelet count was 289,000, making HELLP Syndrome less likely. Patient was diagnosed with AFLP. We proceeded with urgent delivery via caesarean section under general anesthesia. On day 19 post caesarean section patient progressed to fulminant liver failure with transjugular liver biopsy demonstrating persistent AFLP. With Meld Na 37 score she was listed as UNOS status 1A and underwent deceased donor orthotopic liver transplantation. General anesthesia was induced with propofol and succinylcholine and maintained with propofol, fentanyl and rocuronium. In anticipation of intraoperative veno-venous bypass, 16Fr venous cannulas were inserted in the right internal jugular and left femoral veins. Intravenous mannitol was administered to reduce intracerebral pressure and risk of cerebral edema. During the operative course patient received 2800 ml of crystalloids, 750 ml of 5% albumin, 8 units pRBC, blood from cell saver unit, 8 units FFPs, 3 units platelets and 4 units cryoprecipitate with intraoperative estimated blood loss of 3L. She was successfully extubated the following morning and eventually discharged home.

Conclusion: This case report highlights the need for close observation and continuous vigilance when taking care of patients with AFLP. Our patients developed hepatic encephalopathy and profound coagulopathy 3 weeks following an urgent delivery, necessitating liver transplantation.

References: 1. Acute Fatty Liver of Pregnancy: Pathophysiology, Anesthetic Implications, and Obstetrical Management. Anesthesiology 2019;130(3):446-461. 2. Anaesthesia for liver transplantation, BJA Education, Volume 17, Issue 1, January 2017, Pages 35–40 3. Orthotopic liver transplantation for acute liver failure resulting from “acute fatty liver of pregnancy.” Ann Transplant 2003;8:8-11. 4. Acute fatty liver of pregnancy: A clinicopathologic study of 35 cases. Hepatology 1985; 5:1149–58 5. Outcomes of severe pregnancy-related liver disease: Refining the role of transplantation. Am J Transplant 2010;10:2520-6. 6. Idiopathic fatty liver of pregnancy: findings in ten cases. Medicine (Baltimore). 1984 Jan;63(1):1-11. 7. Auxiliary or orthotopic liver transplantation for acute fatty liver of pregnancy: case series and review of the literature. BJOG 2016; 123: 1394– 1398. 8. Anesthesia for Liver Transplantation. Anesthesiol Clin. 2017;35(3):491-508. doi:10.1016/j.anclin.2017.04.006

Obstetric Anesthesiology - 8

Management of Acute Hypoxemic Respiratory Failure Secondary to COVID-19 in Two Pregnant Patients in the ICU

Rashad Sirafi[1], Erica Nicasio[1], Cassian Horoszczak[1], Tichaendepi Mundangepfupfu[1]

[1]University of Rochester, Rochester, NY

Introduction: Pregnancy has been identified as a high-risk condition for COVID-19 infection. Recent studies have identified increased rates of developing severe disease, requiring ICU admission, invasive ventilation, ECMO and mortality in this population [1]. Due to the multiple physiologic changes in the gravid patient and considerations affecting both the mother and the fetus, decisions throughout the hospital course are best made by a multidisciplinary team to optimize outcomes. The decision to proceed with delivery prior to 32 weeks is complex, but delivery in the setting of worsening critical illness should be considered [2], and in our cases, resulted in significant clinical improvement. In our case series, we describe the challenges of treating two parturient patients with acute hypoxemic respiratory failure secondary to COVID-19.

Methods: Case 1: A 30-year-old G2P1 at 31 weeks and 3 days gestational age, without significant medical history, presented with dyspnea and was COVID-19 positive. She was admitted to the ICU for acute hypoxemic respiratory failure. An arterial line was placed, and she was managed on high-flow nasal cannula (HFNC) and self-prone positioning was trialed. On hospital day (HD) 2 her respiratory status worsened requiring intubation. The patient was given phenylephrine prior to induction with rocuronium and propofol and vitals remained stable without fetal distress. On HD 4, concern for additional maternal stress and worsening respiratory failure led the OB, ICU, and anesthesia teams to proceed with a bedside c-section (C/S) in the ICU. Communication between two teams, inside and outside the room, was managed by two nurses with walkie-talkies to relay needs for equipment and/or medications. The patient's anesthetic consisted of infusion of hydromorphone and propofol and one dose of rocuronium. Her oxygenation improved within minutes after an uneventful delivery. She was extubated the next day and discharged home on HD 11. Case 2: A 42-year-old G7P2042 28 at weeks and 2 days gestational age, with a medical history of BMI 56 and type II diabetes mellitus, presented with dyspnea and was COVID-19 positive. Initially, she was admitted to the OB service with acute pulmonary insufficiency and managed with nasal cannula 6lpm. On HD 4 her respiratory status worsened requiring ICU admission and intubation. She was induced with midazolam, etomidate, and succinylcholine; vitals remained stable without fetal distress. On HD 5 fetal monitoring showed category II fetal heart tracing and minimal variability prompting the OB team to proceed with bedside C/S. The anesthetic was a continuation of her infusions of cis-atracurium, fentanyl, and midazolam with propofol boluses. Communication was managed similarly to the first case. There were no adverse outcomes in an uneventful delivery, leading to an improved respiratory status. On HD 9 she was extubated and discharged home on HD 12.

Conclusion: In our institution patients with COVID-19 in the ICU are cared for in negative pressure rooms. Careful monitoring and good multidisciplinary communication led to successful deliveries in a controlled, non-emergent setting. Cautious induction was necessary to avoid drastic hemodynamic changes that may compromise placental blood flow and lead to fetal distress. Thus, it is necessary to plan for possible adverse events during both the induction and C/S in a non-OR setting with regards to anticipated medications/blood products, equipment, and procedures. This can only be achieved with timely multidisciplinary communication during critical events [3].

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.” 69.44 (2020):1641-7. 2.) Society for Maternal-Fetal Medicine. Management Considerations for Pregnant Patients With COVID-19. SMFM COVID Task Force. (2020) 3.) “Recommendations for Endotracheal Intubation of COVID-19 Patients.” 130.5 (2020): 1109–1110.

Pain Mechanisms

Pain Mechanisms - 1 Intraoperative Methadone: Proceed with Care

Nicholas Flores-Conner[1], Sindhu Krishnan[2], Abdul Kader Tabbara[3], Donald Lambert[4], Tadeh Setaghian[4], Edward A Vaynberg[5], Nicole Spence[4]

[1]Boston Medical Center, Boston, MA, [2]Brigham and Womens Hospital, Boston, MA, [3]Henry Ford Hospital, Detroit, MI, [4]Boston Medical Center, Boston, United States of America, [5]Boston University Medical Center, Boston, MA

Introduction: There is interest in perioperative methadone's lasting analgesia and its ability to reduce postoperative opioid consumption due to its unique pharmacodynamics and pharmacokinetics.1 However, a significant concern is the potential for methadone's postoperative RD due to its long duration of action.2 Two publications offer opposing views on the incidence of perioperative RD with methadone use. Murphy and Szokol perceive that the risk of RD is low based on their findings. Conversely, a retrospective study reported RD in approximately one-third of the patients, and hypoxemia in roughly 80%.3 Owing to the high prevalence of opioid misuse in our patient population, and the apparent safety of perioperative methadone use predicted by Murphy and Szokol,1 our department Increased the utilization of methadone to improve perioperative analgesia and reduce opioid use.4 We witnessed three patients who required intravenous naloxone to counter postoperative RD. Consequently, we question Murphy and Szokol's1 suggestion that perioperative methadone use is devoid of RD. Our clinical experience with perioperative methadone use leading to RD and subsequent naloxone use in 8% of patients in conjunction with the report by Dunn, et al3 tells a different story. Therefore, the clinical use of methadone requires considerably more study than currently exists in the literature.

Methods: A 49-year-old male (1.67 m, 86 kg, BMI 30.6), underwent a C6-C7 ACDF for cervical stenosis and myelopathy. He did not have prior opioid use. Following induction of general anesthesia with propofol, intravenous 10 mg of methadone (0.12 mg/kg) was administered. Anesthesia was maintained with propofol 150 ¬µg kg-1¬∑min-1. Immediately prior to emergence, he received fentanyl 100 ¬µg intravenously. He did not satisfy extubation criteria due to RD, thus a single 80 ¬µg dose of naloxone was administered, which led to RD reversal and extubation. An 18-year-old male (1.72 m, 66.5 kg, BMI 22.29), underwent a thoracic (T2-T11) PSF for scoliosis correction. He received midazolam 2 mg IV (0.03 mg/kg) for anxiolysis preoperatively. Anesthesia was induced with propofol and fentanyl 250 ¬µg. After induction, he received methadone 20 mg (0.3 mg/kg) intravenously. Anesthesia was maintained with propofol 110-150 ¬µg¬∑kg-1¬∑min-1 and remifentanil 0.2-0.4 ¬µg¬∑kg-1¬∑min-1. Two hours after the administration of methadone, he received intravenous hydromorphone 2 mg (0.03 mg/kg). At the end of the operation, his respiratory rate was 8 breaths per minute with tidal volumes of 10-12 mL/kg. He was uneventfully extubated. In the PACU, he was somnolent and hypercarbic (paCO2 = 79). The PACU physician administered two 80 ¬µg doses of intravenous naloxone. A healthy 12-year-old female (1.62 m, 54.1 kg, BMI 20.5) underwent a thoracic to sacral (T2-S1) PSF for scoliosis correction. She received midazolam 2 mg (0.04 mg/kg) prior to induction. At induction, she received propofol and 10 mg of methadone (0.2 mg/kg). Anesthesia was maintained with propofol 150-200 ¬µg kg-1¬∑min-1, ketamine 15-75 ¬µg kg-1¬∑min-1, and no additional opioids. Upon completion of the operation, she was successfully extubated and transferred to the pediatric intensive care unit (PICU). In the PICU, she was obtunded with pinpoint pupils, unresponsive to sternal rub, and with shallow breathing (RASS of -5). The staff administered two intravenous doses of naloxone (40 ¬µg each). The patient then became alert and oriented with improved respirations.

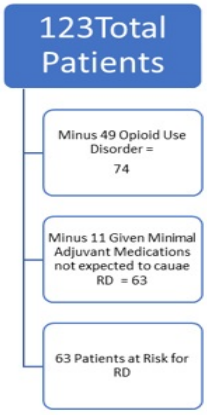
Conclusion: A recent meta-analysis reinforces low RD rates but accounts for a possibility of studies being underpowered.5 We queried our EMR for patients who received methadone between 1/1/19 and 8/30/20 (Table 1). Excluding patients with opioid use disorder and those undergoing cesarean section or tubal ligation, we identified 5 patients who received naloxone for RD out of 63 included patients (7.9%). Whereas the reports by Murphy and Szokol control for polypharmacy, contemporary anesthesia practice does not. We postulate that our patients who were given adjuvant medications in addition to methadone likely rendered them more susceptible to RD. Methadone has a brief redistribution phase and prolonged analgesia (Figures 1 and 2), its MEC differs between

patients (Figure 3).6 Guidance regarding safety of perioperative methadone is scarce. We suggest minimizing adjuvant medications and titrating intraoperative and postoperative opioids to prevent RD.

References: 1. Intraoperative Methadone in Surgical Patients: A Review of Clinical Investigations. Anesthesiology 2019;131:678-692. 2. Methadone produces prolonged postoperative analgesia. Br Med J (Clin Res Ed) 1982;284:630-631. 3. Safety profile of intraoperative methadone for analgesia after major spine surgery: An observational study of 1,478 patients. J Opioid Manag 2018;14:83-87. 4. Intraoperative methadone: rediscovery, reappraisal, and reinvigoration? Anesth Analg 2011;112:13-16. 5. Intraoperative methadone administration and postoperative pain control: a systematic review and meta-analysis. Pain 2020;161:237-243. 6. Postoperative pain control with methadone: influence of supplementary methadone doses and blood concentration--response relationships. Anesthesiology 1984;61:19-26.

Table 1 Number of Patients Detected by EMR Review Who Received Methadone for the Period January 1, 2019 – August 30, 2020

Methadone Dose (mg)	Number
7.5	2
10	95
15	4
20	18
30	3
40	1
Total	123



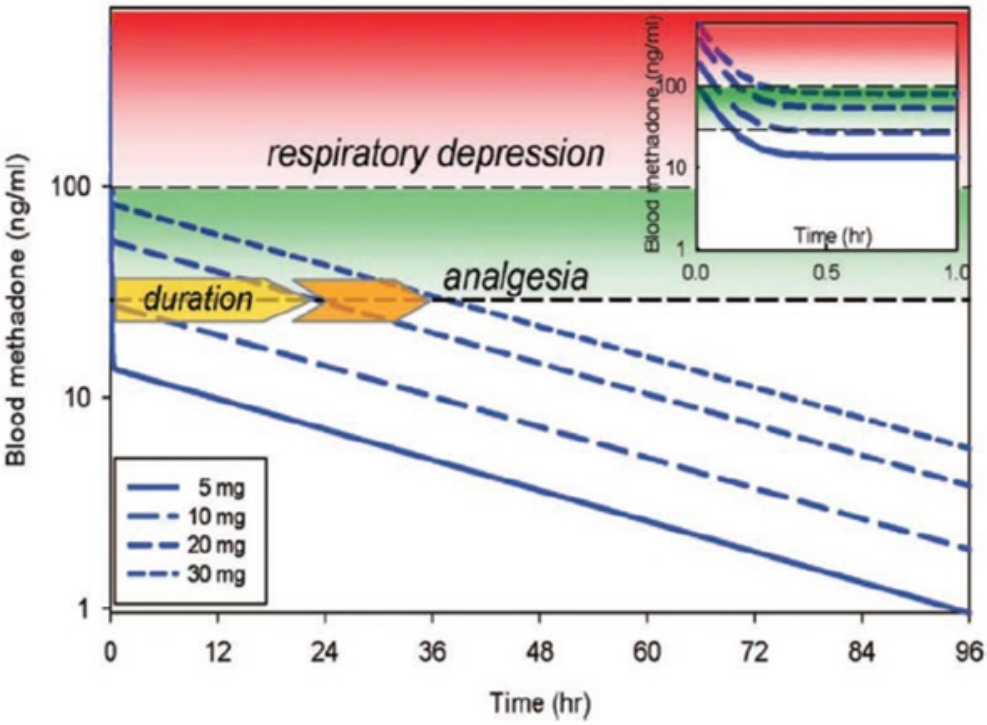


Figure 1. Relationship between methadone dose and duration of effect. Simulated methadone blood concentrations versus time based on pharmacokinetic parameters, the minimal effective analgesic concentration (approximately 30 nanograms (ng) per ml), and the threshold for significant respiratory depression (approximately 100 ng/ml), as determined by Gourlay et al., [2, 6] Data are shown for bolus methadone doses of 5, 10, 20, and 30 mg, with the estimated duration of analgesia of less than 0.5, less than 0.5, approximately 24, and approximately 36 h, respectively. The inset shows plasma concentrations for the first hour after dosing. Because of rapid redistribution, anticipated respiratory depression would be less than 30 to 45 min, even at the higher single bolus doses. (Reproduced with permission, Murphy and Szokol.) [1]

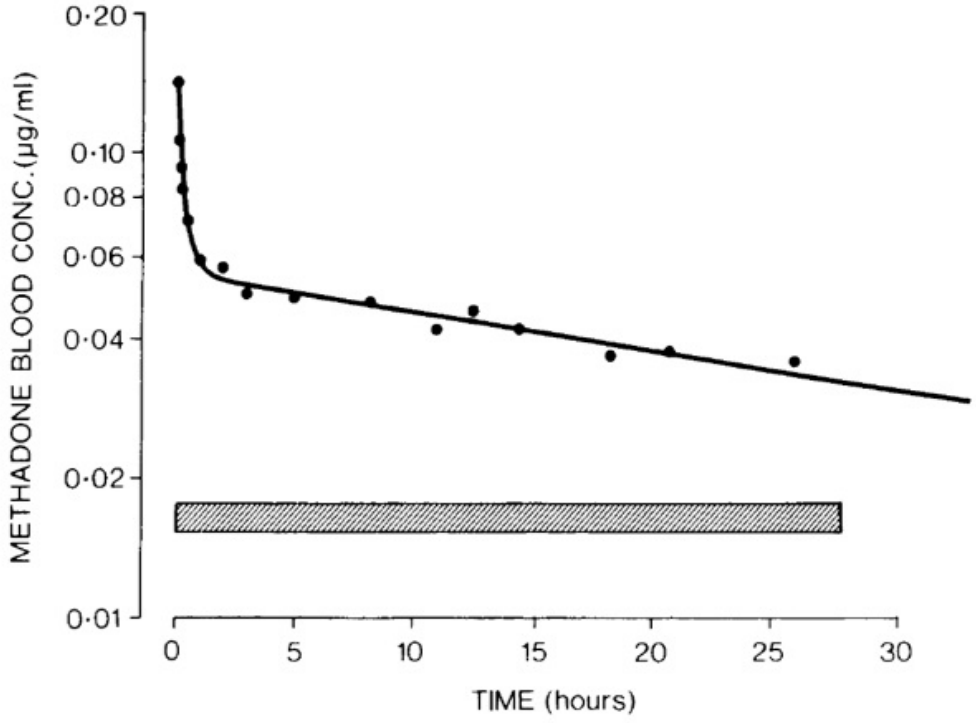


Figure 2. Blood methadone concentration (µg/mL) as a function of time (hours) in a patient undergoing repair of hiatus hernia. The 49-year-old man was administered 20 mg of methadone as an IV bolus. The black dots represent the measured blood methadone concentration while the curve represents the computer -generated line of best fit of a biexponential equation to the data. The shaded area represents the time period that the patient was continuously free of pain (28 hours). This patient had a methadone clearance of 113 mL min^{-1} and a terminal half-life of 36.5 hours. (Reproduced with permission, Gourlay, et al) [4]

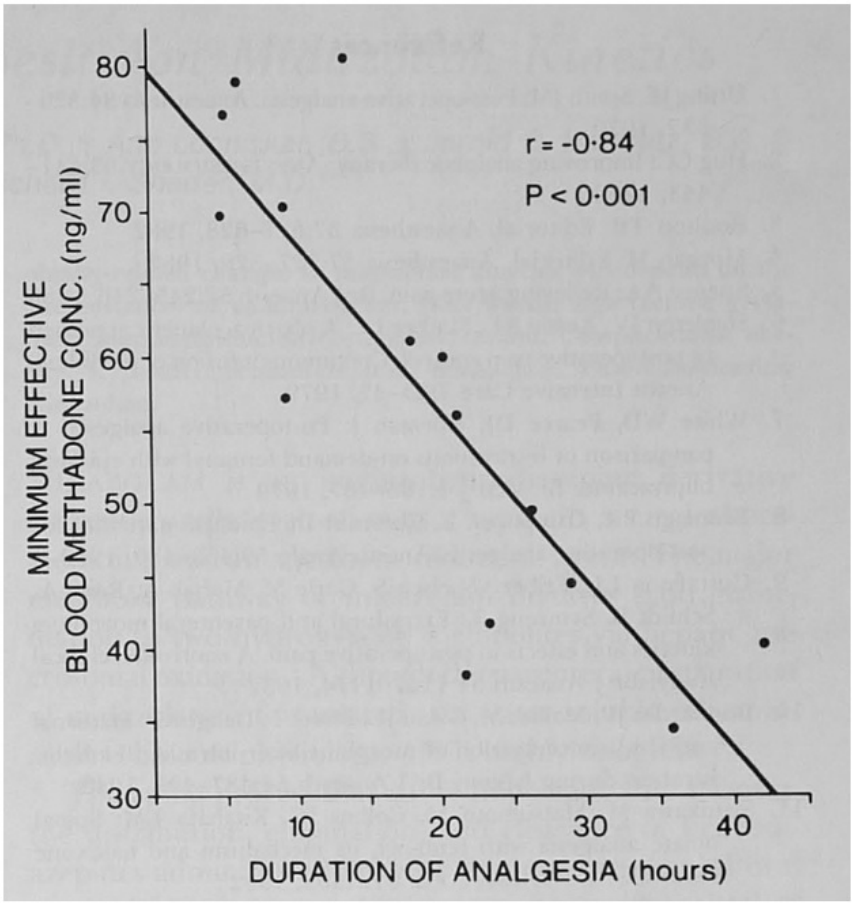


Figure 3. Correlation of minimum effective blood methadone concentration (ng/mL) with duration of analgesia (h), r = -0.84. P < 0.001 (Reproduced, with permission from Gourlay, et al.) [5]

Pain Medicine

Pain Medicine - 1 Intercostal Muscle Trigger Point Injection for Chronic Chest Wall Pain and Associated Dyspnea, Diaphoresis, and Tachycardia Secondary to Trauma: A Case Report

Mila Pastrak[1], Ognjen Visnjevac[2]

[1]McMaster University, Toronto, Ontario, [2]McMaster Faculty of Health Sciences, Dept. of Anesthesiology, Toronto, Ontario

Introduction: Myofascial pain syndrome is one of the common causes of chronic pain encountered in clinical practice. Trigger points, which are commonly present in patients with myofascial pain can cause localized pain in the affected muscles and referred pain patterns resulting in impaired muscle coordination and reduced range of motion. Here, we describe a 55-year-old male patient who developed myofascial pain syndrome involving the left posterolateral intercostal muscles with sudden onset after acute chest trauma 14 years ago (the patient's son jumped on him by surprise). Although tachypneic with shallow breathing to avoid pain, obese, and diaphoretic on presentation, the patient and his family members reported that this was a constant state of existence for him for 14 years. Cardiopulmonary etiologies were previously ruled out and the patient was referred for chest wall interventions. Following examination and suspicion of intercostal muscle trigger points as an etiology, the patient was treated with an ultrasound-guided intercostal muscle trigger point injection involving the 7th to 10th intercostal spaces on the left in an outpatient anesthesia-interventional pain management clinic.

Methods: A 55-year-old obese male patient presented with a history of shortness of breath, diaphoresis, conversational dyspnea and chronic pain in the left lateral chest wall with radiation from the posterior chest wall following the direction of the T7-10 involved intercostal spaces. On physical examination he had focal posterolateral tenderness between T7-10 exacerbated by deep breathing and worsened by deep palpation, even more so with concurrent deep breathing, suggestive of a pleuritic or chest wall pathology. Consent was obtained and ultrasound-guided intercostal muscle trigger point injections (12 cc of 0.25% bupivacaine mixed in equal parts 0.9% normal saline) were performed with patient feedback to localize spastic myofascial elements, starting from the distal perceived border of his pain moving proximally to avoid intercostal nerve analgesia as a false sign of successful myofascial release. Post procedure, the patient's pain resolved completely, the respiratory rate and subjective assessment of the patient's respiratory comfort normalized, and conversational dyspnea was no longer present. There were no complications and ultrasound was used to identify normal pleural signs post-procedure to rule out pneumothorax. The effect of trigger point injection on pain relief and other symptoms persisted at 1-month follow-up.

Conclusion: Intercostal muscle trigger point injection with ultrasound guidance are an effective and safe treatment option for non-cardiac chest pain, which can easily and widely be used to treat intercostal myofascial pain in an outpatient management setting.

Pain Medicine - 2 Intercostal Nerve Block and Decompression for Post-Surgical Pain

Stephen Hannaford[1], Mia Castiglione[2], Nicole Brand[2], Andrew Kauffman[3]

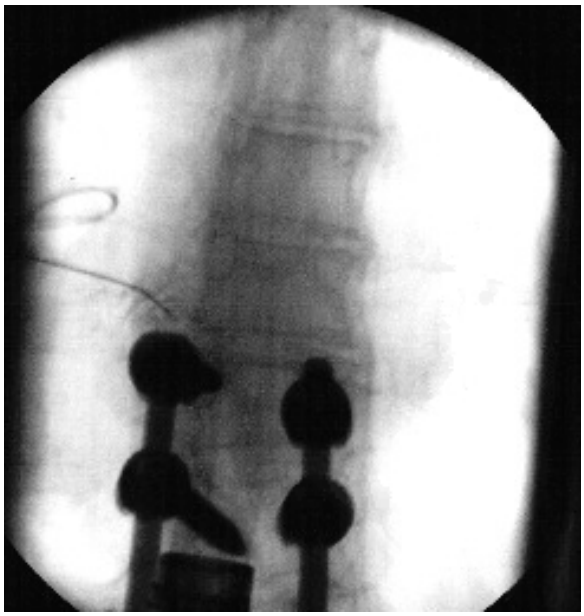
[1]New Jersey Medical School, Maplewood, NJ,
[2]Rutgers New Jersey Medical School, Newark, NJ,
[3]NJMS Rutgers, Newark, NJ

Introduction: The intercostal nerve block (INB) procedure is a useful intervention for various painful conditions affecting the torso. These include, but are not limited to post-thoracotomy pain, post-rib fracture pain, cancer pain, post-mastectomy pain, costochondritis, and intercostal neuralgia.[1] INB is considered an alternative to epidural anesthesia for analgesia of the torso, with advantages including less technical skill required, and less risk of complications including hypotension, urinary retention, and injury to nerve roots or the spinal cord.[1] Although it has been shown to provide comparable relief of pain with potentially better outcomes than epidural anesthesia, 2-4 INB in the paravertebral space has not been described as a long-term intervention for intercostal neuralgia. Painful neuropathies that arise from nerve compression by scar formation after surgery have been approached with various treatments, including percutaneous neurolysis.[5] INB can be used as a diagnostic tool to identify the offending nerve for potential ablation. Here we report successful, lasting treatment of post-surgical intercostal neuralgia with INB alone.

Methods: A 58-year-old woman with history of osteoporosis, diabetes, hypertension, and chronic back pain after motor vehicle accident 9 years ago presented to the clinic with left-sided back pain. She underwent T8 to L2 posterior fusion 3.5 years ago with some improvement in her pain; however, has experienced constant back pain of varying intensity and character since her accident. In particular, after surgery she describes the onset of 'burning, sharp' left-sided back pain, with range in intensity from 5 to 10/10 on a numerical rating scale. This pain recently worsened, prompting her to seek our consultation. She indicated the left T7-10 dermatomes as the location of her pain, and denied any radiation, accompanying weakness or numbness. She noted the pain worsened with tasks such as bending over, and with prolonged sitting. She could not cite any alleviating factors. The patient reported she had tried various pharmacological treatments, which aided with other aspects of her back pain, but did not help with the current symptom. Her medications were significant for metaxalone 800mg TID, cyclobenzaprine 5mg TID PRN, venlafaxine 75mg daily, and gabapentin 300mg TID. Due to the severity of this pain, she had recently started oxycodone-acetaminophen 5-325mg q4h PRN. Physical examination was significant for tenderness to palpation in the left T8 dermatome from spine to mid-abdomen. There were no accompanying sensory deficits, allodynia, or hyperalgesia. Given her history of extensive spinal surgery, along with the character and distribution of her pain, the diagnosis of T7-T9 intercostal neuralgia was made, and she was scheduled for diagnostic INB. The T8 rib/transverse process complex was visualized under fluoroscopic guidance with the patient in prone position on the procedure table. A 22 gauge 3.5 inch Chiba needle was advanced through the skin, and with some difficulty into the area just adjacent to the neural foramen. With placement of the needle she reported reproduction of the vast majority of her pain. After negative aspiration, 1cc of contrast was injected. At this point, there appeared to be visualization of a scar type formation, as there was appropriate but not true free flow along the nerve. There was no flow into the epidural space. Subsequently, 1cc of 0.5% bupivacaine was injected. Initially during the injection there was significant resistance, and then a release was felt, followed by free flow. After ten minutes, and again one month later in clinic, the patient reported 80% improvement in her pain in the entire affected area. As a result, no additional injections were performed at the adjacent levels, nor was the previously-discussed radiofrequency ablation (RFA).

Conclusion: Here we present the successful use of INB for treatment of intercostal neuralgia after posterior fusion surgery. Interestingly, our patient's pain seemed to stem from compression of the nerve within scar tissue. This was evidenced by the pattern of contrast visualization along the nerve during fluoroscopy, palpable release during injection, and lasting relief without RFA. Thus, in cases such as ours, the diagnostic INB can itself be a therapeutic intervention through decompression of the nerve.

References: 1. Fraifeld IM. Intercostal nerve blocks. In Deer TR, Leong MS, Buvanendran A, Kim PS, Panchal SJ, eds. Treatment of Chronic Pain by Interventional Approaches. New York, NY: Springer; 2015;173-206. 2. Davies RG, Myles PS, Graham JM. A comparison of the analgesic efficacy and side effects of paravertebral vs epidural blockade for thoracotomy - a systematic review and meta-analysis of randomized trials. Br J Anaesth. 2006;96(4):418-426. 3. Am K, et al. Prospective, randomized comparison of extrapleural versus epidural analgesia for postthoracotomy pain. Ann Thorac Surg. 1998;66:367-372. 4. Luketich JD, Sullivan EA, Ward J, Buenaventura PO, et al. Thoracic epidural versus intercostal nerve catheter plus patient-controlled analgesia: a randomized study. Ann Thorac Surg. 2005;79:1845-1850. 5. Ulloa M, Banda MC. Scar tissue causing saphenous nerve entrapment: percutaneous scar release and fat grafting. Plast Reconstr Surg Glob Open. 2017 Sep;5(9):e1495.



Pain Medicine - 3 Novel Treatment of Bell's Palsy by Percutaneous Decompression (Trigger Point Injections) of the Masseter Muscle: A Report of 2 Cases

Mila Pastrak[1], Ognjen Visnjevac[2], Frederick Ma[3]

[1]McMaster University, Toronto, Ontario, [2]McMaster Faculty of Health Sciences, Toronto, Ontario, [3]Bloor Pain Specialists, Toronto, Ontario

Introduction: Bell's palsy is a debilitating neurological condition caused by the neuropathy of the seventh cranial nerve as a result of traumatic, infective, compressive, inflammatory, or idiopathic etiologies. Bell's palsy clinically presents as an acute unilateral paralysis or weakness of the facial muscles. Current treatment options depend on the etiology and may include: antiviral medication, corticosteroids, biofeedback training, physical therapy, and surgical interventions. We postulated that acute-onset facial nerve paresis could arise from compressive pathology akin to that of piriformis syndrome. Piriformis injections can induce decompression of the sciatic nerve by the release of the piriformis muscle in piriformis syndrome, thereby alleviating the symptoms of sciatic nerve compression. Herein, we describe 2 patient cases with acute-onset Bell's palsy that were treated with trigger point injections of the deep and superficial heads of the masseter muscle to induce percutaneous decompression of the facial nerve by releasing myofascial spasm-induced TMJ and mandibular asymmetry and suspected traction on the traversing facial nerve.

Methods: Case 1: An otherwise healthy 34-year old male anesthesiologist presented as an outpatient in anesthesia-interventional pain management clinic with right acute facial nerve paresis of 4 hours onset without infectious, traumatic, or inflammatory history. Physical exam findings were significant for hemifacial paresis consistent with Bell's palsy and asymmetric ipsilateral myofascial spasm of the masseter muscles. Case 2: A 54-year old male presented as an outpatient in anesthesia-interventional pain management clinic with left acute facial nerve paresis of 2 days duration with no infectious, traumatic, or inflammatory history and similar asymmetric myofascial spasm of the jaw. Neither patient presented with clinical signs of CNS lesions or cerebrovascular accident. Consent was obtained with emphasis of the experimental nature of this treatment and percutaneous decompression of the muscle was performed by palpation-guided trigger point injections of the deep and superficial heads of the masseter using approximately 8mL of 0.25% bupivacaine in 1cc increments in each patient. There were no complications throughout the procedure and facial nerve function and facial muscle movements normalized by the next day, as evidenced by return of symmetric ocular muscle function, normalization of eyebrow lift, and return of symmetry of smile. Both patients showed complete recovery with no signs of recurrence at 2 and 1 year follow-up, respectively.

Conclusion: Two patients with unilateral Bell's palsy, both with associated asymmetric masseter ipsilateral myofascial spasm and suspected compressive etiology, were successfully treated with trigger point injections for myofascial release of the masseter muscles with the aim of releasing mechanical traction on the traversing facial nerve. To our knowledge, there have not been previous attempts to perform a release of the masseter muscle resulting in decompression of the facial nerve in patients with Bell's palsy. As such, this therapeutic approach may be considered as an alternative to surgical intervention for compressive facial paralysis in Bell's palsy.

Pain Medicine - 4 Opioid-Free Perioperative Pain Management In A Patient On Continued Buprenorphine Therapy For Opioid Use Disorder (Oud).

Kate Zipperer[1], Sal Salavat Yulaman[2], Courtney Williams[3]

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

Introduction: The management of acute pain for patients on chronic buprenorphine therapy for opioid dependency is a challenging process. According to the CDC, opioid addiction rates are at an all-time high. Buprenorphine is a partial agonist of the Œº-opioid receptor (MOR) and an antagonist of the kappa opioid receptor (KOR). MOR agonism is related to analgesia and sedation while KOR agonism has hallucinogenic or dissociative effects. Buprenorphine reaches a plateau at higher doses and is approved by the FDA to treat opioid dependency disorder as part of a treatment plan called medication-assisted treatment (MAT) (1). Today there is no consensus regarding optimal perioperative management of patients undergoing buprenorphine treatment for opioid use disorder (OUD), so most strategies include a multimodal pain control approach. These approaches range from continuing buprenorphine and supplementing with full opioid agonists, to discontinuing buprenorphine preoperatively to instead utilize opioid agonists (2).

Methods: A 59-year-old female, BMI 20.4, with a history of OUD, attention deficit disorder, chronic pain, irritable bowel syndrome, gastroesophageal reflux disease, and hypertension, presented for elective inpatient fundoplication. The patient was taking buprenorphine-naloxone for OUD 8-2 mg twice daily. The anesthesia plan was discussed with the patient and her expectations were evaluated. Multimodal opioid-free pain management was emphasized. The patient was premedicated with midazolam 2 mg IV, and famotidine 20 mg IV. The patient was intubated using rocuronium 0.6 mg/kg IV, propofol 2.5 mg/kg IV, and lidocaine 2%-5 ml. Anesthesia was maintained with sevoflurane, ketamine IV boluses for pain, and 0.3 mg/kg per bolus of ketamine with a total dose of 50 mg was used. In addition, dexmedetomidine infusion at 0.5 mcg/kg/hr was used throughout the case. The patient also received dexamethasone 4,Äâmg IV, and ondansetron 4 mg IV. During the case, the patient's blood pressure and heart rate remained within 15% of baseline. At the end of the case, the wound was infiltrated with 0.25% bupivacaine mixed with liposomal bupivacaine solution. The rocuronium was reversed and the patient was extubated in the operating room. In the postoperative period, patient's reported pain was below 3 on the numerical pain scale. The patient's PACU stay was uneventful and the patient remained overnight for observation. During the hospital stay, the patient received ketorolac 30,Äâmg IV, and acetaminophen 1 g PO.

Conclusion: Multimodal pain control is a pain management technique centered around the idea that multiple medications with different mechanism of action used together for pain control are more effective than one method alone (3). Despite some recommendations to taper buprenorphine prior to surgery, buprenorphine may be continued in some circumstances by utilizing multimodal pain control. The most significant benefit of this approach is the decreased risk of relapse in patients with OUD. It has been shown that patients are much more likely to relapse after tapering off buprenorphine than when they are taking it daily, regardless of whether they are undergoing concurrent addiction counseling (4). Patients on continued buprenorphine therapy are more likely to use opioids when they experience increased feelings of craving (5). Pain and anxiety, both of which are associated with surgical procedures, may increase feelings of cravings in patients with a history of OUD (6). Since buprenorphine can treat feelings of craving in people with a history of opioid use, it is likely that buprenorphine discontinuation may lead to increased feelings of craving perioperatively. For these reasons, it may be beneficial to continue buprenorphine perioperatively while maximizing the use of other non-opioid options such as acetaminophen, gabapentin, or dexmedetomidine. If buprenorphine use absolutely must be discontinued, substance use disorder specialists should be consulted.

References: 1. 'Medication treatment of opioid use disorder.' Biological psychiatry 87.1 (2020): 82-88. 2. 'To Stop or Not, That Is the Question: Acute Pain Management for the Patient on Chronic Buprenorphine.' Anesthesiology: The Journal of the American Society of Anesthesiologists 126.6 (2017): 1180-1186 3. Carpenter RL. Optimizing postoperative pain management. Am Fam Physician. 1997 Sep 1;56(3):835-44, 847-50. Erratum in: Am Fam Physician 1997 Dec;56(9):2192. 4. Counseling During Brief and Extended Buprenorphine-Naloxone Treatment for Prescription Opioid Dependence: A 2-Phase Randomized Controlled Trial. Arch Gen Psychiatry. 2011;68(12):1238-1246. doi:10.1001/ Arch Gen Psychiatry 2011.121 5. Craving predicts opioid use in opioid-dependent patients initiating buprenorphine treatment: a longitudinal study. Am J Drug Alcoh Abuse. 2014;40(2):163-169 6. Chronic pain, craving, and illicit opioid use among patients receiving opioid agonist therapy. Drug Alcohol Depend. 2016;166:26-31

Pain Medicine - 5 Transversus Thoracic Muscle Plane Block Following Sternotomy In Cardiac Surgery May Decrease The Need For Opioid Use.

Kate Zipperer[1], Sal Salavat Yulaman[2], Courtney Williams[3], Noel M Giesecke[4]

[1]UTMB, Galveston, United States of America, [2]University of Texas Medical Branch, Houston, United States of America, [3]University of Texas Medical Branch, Galveston, TX, [4]Houston Methodist, Houston, United States of America

Introduction: According to recent data 9.8% of patients developed new persistent opioid use after cardiac surgery [1]. Sternotomy and the nature of cardiac surgery itself is associated with high levels of postoperative pain [2]. An adequate technique of post-operative analgesia enables early extubation, early rehabilitation and discharge from the intensive care unit, and decreases patient exposure to airborne pathogens. Opioids have been the mainstay of analgesic therapy for perioperative pain management in cardiac surgery. Even so, this approach has many drawbacks, including pulmonary complications. Increasing availability of ultrasound machines and increasing expertise in ultrasound-guided blocks will lead to increased utilization of transversus thoracic muscle plane blocks (TTP block) for pain control following sternotomies. TTP block provides effective analgesia for cardiac surgeries and acute and chronic pain management [3]

Methods: A 61-year-old male weighing 135kg, with a history of diabetes mellitus, hypertension, chronic renal insufficiency, and obesity, was admitted to the hospital for elective coronary bypass grafting (CABG) surgery. After the completion of the CABG surgery, including the placement of sternal wires and wound closure, a bilateral bilevel transversus thoracic plane (TTP) block was performed in the operating room. The patient was not repositioned for the procedure. A high-frequency linear probe was utilized to scan in a parasagittal fashion medial to the mid-clavicular line over ribs 3 and 4. The pectoralis major muscle was appreciated beneath subcutaneous tissue and overlying the ribs. Next, the intercostal muscles were appreciated between the ribs and superficial to the transversus thoracic muscle (TTM), which usually appears as a hypoechoic band that overlies the pleura. The TTP was identified between the internal intercostal muscle and the TTM. In plane technique was used to place the tip of the needle to the TTP. A 10cm needle was utilized for this patient due to body habitus. With constant tip visualization, after intermittent negative pressure aspiration to avoid intravascular injection of the internal thoracic vessels, the needle tip was placed between the internal intercostal muscle and the TTM. Once the needle was placed, 10 ml bupivacaine 0.25% was injected with intermittent aspiration bilaterally under the guidance of ultrasound. Following this block, the patient was transported to the cardiovascular ICU for recovery. The patient was extubated within 1 hour and remained pain free for 16 hours. After that, IV hydromorphone had to be used for pain control since the effects of the block had worn off. This patient received substantially lower amounts of opioids than patient would under normal circumstances without this type of block.

Conclusion: Sensory innervation of the chest wall is supplied by branches of intercostal nerves, which are formed by the ventral ramus of the T1-T11 spinal nerves. The anterior chest wall is primarily supplied by the anterior division of the second through sixth thoracic intercostal nerves, which are collectively the target for anesthesia by a TTP block [4]. Since the TTP block targets the anterior division of the second through sixth thoracic intercostal nerves, it can be an appropriate method of analgesia for a wide range of procedures involving the anterior chest wall. It has been successfully used in mastectomies, sternotomies, pericardiocenteses, and has more recently been used for cardiac surgeries [5]. Since the TTP block target is less invasive than thoracic epidural or intrathecal morphine administration, it poses less risk of heparinization-related bleeding. Ideally, the TTP block for pain control in cardiac surgery could reduce the need for postoperative opioids. This is a huge benefit considering the high rate of opioid dependence in post-operative patients. In addition to common side effects such as nausea, constipation, and respiratory depression, this risk of new-onset addiction is a significant concern. A systematic review conducted in 2017 found that regional anesthesia was seldom used for cardiac surgery patients, and that opiates were widely used to treat postoperative pain [6]. By avoiding the coagulopathy-related risks of other regional

anesthesia methods, as well as avoiding the dependence-related consequences of opioid use, the TTP block is a promising method of regional anesthesia for cardiac surgery.

References: 1. Brown, Chase R., et al. 'Development of persistent opioid use after cardiac surgery.' JAMA cardiology 5.8 (2020): 889-896. 2. Silva, Juliana Fernandes, et al. 'Minimally invasive cardiac surgery versus sternotomy-Pain investigation.' International Journal of Cardiovascular Sciences 33.1 (2020): 24-33. 3. Ueshima, H., and H. Otake. 'Addition of transversus thoracic muscle plane block to pectoral nerves block provides more effective perioperative pain relief than pectoral nerves block alone for breast cancer surgery.' BJA: British Journal of Anaesthesia 118.3 (2017): 439-443. 4. Ueshima H., Takeda Y., Ishikawa S., Otake H. Ultrasound-guided transversus thoracic muscle plane block: a cadaveric study of the spread of injectate. Journal of Clinical Anesthesia. 2015;27(8):p. 696. doi: 10.1016/j.jclinane.2015.05.013. 5. Fujii S, Bairagi R, Roche M, Zhou JR. Transversus Thoracis Muscle Plane Block. Biomed Res Int. 2019;2019:1716365. Published 2019 Jul 7. doi:10.1155/2019/17163

Patient Safety

Patient Safety - 1 Endovascular Thrombectomy And Systemic Thrombolysis In A Pediatric Patient: Anesthetic Management Considerations And Perioperative Complications

Andres Bacigalupo Landa[1], Premal M Trivedi[2]

[1]Baylor College of Medicine, Hosuton, TX, [2]Baylor College of Medicine, Houston, TX

Introduction: Endovascular thrombectomy and systemic thrombolysis are established therapies in managing life- or limb-threatening vascular occlusion.1 Their associated complications, however, warrant vigilance in the peri-anesthetic period. Attempts at thrombectomy can lead to embolization, anemia, and hyperkalemia depending on the device(s) used. Thrombolysis, on the other hand, can cause significant bleeding at access sites, and can present a challenge in the child with developmental delay who may be at risk of pulling invasive catheters post-procedure. Beyond these risks, the patient's underlying disease state(s) and altered physiology that led to the thrombosis must also be managed. To illustrate these points and their consequences, we present the case of an 8-year-old child with acute occlusion of an infrarenal aortic stent who underwent endovascular thrombectomy followed by systemic administration of tissue plasminogen activator (tPA).

Methods: An 8-year-old (27 kg) male with history of mid-aortic syndrome, renal artery stenosis and chronic kidney disease presented for angioplasty of the stenotic aorta and renal arteries in the setting of hypertensive urgency and worsening claudication. The average blood pressure pre-procedure was as high as 220/125 mmHg in the upper extremities and 140/95 mmHg in the lower extremities. Aortic angiogram showed a gradient of 70 mmHg across the infrarenal aorta that decreased to 7 mmHg following angioplasty and stenting. Anticoagulation was maintained with heparin during the case, and transitioned to clopidogrel and aspirin postoperatively for stent prophylaxis. On postoperative day (POD) # 1, complete occlusion of the aortic stent was observed on routine ultrasound and further confirmed with computed tomography (CT) scan (Figure 1). Heparin was given and the patient was subsequently returned to the catheterization suite for thrombectomy. General anesthesia was induced and a right median arterial line was attempted to allow more accurate blood pressure monitoring (the left radial artery which had been previously accessed was now thrombosed and no right radial artery was present). After an unsuccessful attempt, blood pressure was monitored via a non-invasive cuff on the same arm. Thrombectomy was performed through the right femoral artery using both mechanical extraction and the Penumbra Aspiration System¬Æ (Penumbra Inc, California). Approximately 400 cc of blood was removed during aspiration with a concurrent decrease in hematocrit (40 to 29 %) and on the systemic blood pressure. Finally, the aortic stent was successfully re-canalized. To ensure stent patency, a low-dose tPA infusion was started (transitioned to enoxaparin and aspirin after 48 hours). Given the patient's developmental delay, he was kept intubated while tPA was infusing. The postoperative course was complicated by right superficial femoral artery thrombus, likely from distal embolization of clot, and bleeding from the right femoral artery access site and right forearm. The latter resulted in a compartment syndrome (deep volar compartment) requiring fasciotomy on POD # 13.

Conclusion: Endovascular thrombectomy and systemic thrombolysis are relatively infrequent procedures in children that can lead to complications both during and after intervention. Specific to thrombectomy, questions to address include the location and size of the thrombus, end-vessels or -organs at risk should embolization occur, and which device will be used. The latter can give insight into the severity of blood loss and metabolic derangements to anticipate. In this patient, nearly 20 % of his blood volume was extracted along with thrombus. Blood should therefore be available during aspiration. Further, hemolysis leading to hyperkalemia may occur during thrombectomy. Attention to the EKG and frequent blood gases can help identify derangements early. Bleeding is a known risk associated with the use of tPA, but particular caution should be used when attempting to access deep arterial structures. While a hematoma at the radial or femoral artery is readily apparent, one occurring in a deep compartment may be more difficult to discern. By attempting to access this patient's medial artery, we likely provided the nidus for subsequent bleeding and a compartment syndrome

while on tPA.2 The need for invasive blood pressure monitoring in pediatric patients must be weighed against this risk if such a site is the only option.

References: 1. Monagle P, et al. Antithrombotic therapy in neonates and children: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012 Feb;141:e737S-e801S. 2. Freyer M, et al. Compartment syndrome following thrombolysis: clinical features and associated conditions. J Thromb Thrombolysis. 2014;38(2):201-7.

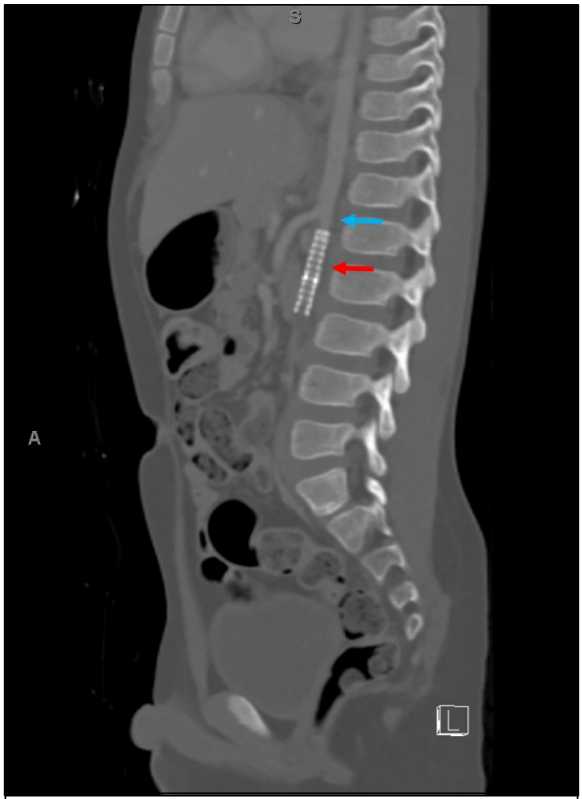


Figure 1. Abdominal CT Angiography (sagittal plane) showing contrast in the descending aorta (blue arrow), and absence of flow through the aortic stent (red arrow).

Patient Safety - 2 Ethical Challenges And Considerations In Surgical Decision Making For A Covid Positive Patient Undergoing Laparoscopic Appendectomy For Acute Appendicitis

Courtney Chow[1], Piyush Mathur[2], Clayton Petro[3]

[1]Cleveland Clinic Foundation, Cleveland, OH, [2]Cleveland Clinic, Cleveland, OH, [3]Cleveland Clinic Foundation, Cleveland, United States of America

Introduction: As the number of people testing positive for COVID-19 continues to rise within the population, so does the number of COVID-19 positive patients require hospital admission. These patients present ethical challenges in how to provide them the best care while protecting healthcare workers from potential exposure and conserving resources. Here we present a case of a COVID-19 positive patient presenting with acute appendicitis with the plan to undergo urgent appendectomy.

Methods: A 32yo male with past medical history of Tetralogy of Fallot status post repair as a child, pulmonic stenosis status post pulmonic valve repair six years prior, and hypertension presented with abdominal pain and was found to have acute appendicitis confirmed with an abdominal CT scan. A COVID test obtained upon admission to the regular nursing floor was positive, although the patient was asymptomatic. The patient agreed to be enrolled in a study that randomized COVID-19 positive patients with acute appendicitis to medical management versus appendectomy. The patient was randomized to the surgical group. Cardiology and Infectious Disease consults were obtained. The patient underwent an uncomplicated laparoscopic appendectomy on hospital day number two, and was discharged home the very next day. There are four key principles of ethics in healthcare: autonomy, beneficence, non-maleficence and justice[1]. COVID-19 positive patients with acute appendicitis pose unique ethical challenges along principles of non-maleficence and justice, which are not commonly encountered during provision of care in the US. Non-maleficence is intended to be the end goal for all of a practitioner's decisions, and means that medical providers must consider whether other people or society could be harmed by a decision made, even if it is made for the benefit of an individual patient. The principle of justice supports fairness in decisions, equal distribution of scarce resources and new treatments, and for medical practitioners to uphold applicable laws and legislation when making choices. Patients such as ours can be managed surgically and potentially be discharged home within 24 hours, or managed medically while remaining hospitalized and potentially exposing other caregivers over a longer time while consuming limited hospital resources. Perioperative anesthesia care requires special precautions including use of personal protective equipment (gowns, double gloves, N95 mask, eye shield) and minimization of duration of exposure to airway particles by utilizing a rapid sequence induction and video-laryngoscopy for intubation amongst others during perioperative care of a COVID-19 disease patient[2].

Conclusion: In conclusion, this pandemic has highlighted the importance of ethical principles such as non-maleficence and justice in perioperative decision making during the pandemic which are equally important as autonomy and beneficence [3].

References: 1. Beauchamp T.L., Childress J. Oxford University Press; New-York/Oxford: 2001. Principles of Biomedical Ethics. 5√®me Edition 2. Mather P, Cywinski JB, Khanna S, Trombetta C. Perioperative anesthesia care for patients with confirmed or suspected COVID-19. Cleve Clin J Med. 2020 May 20. doi: 10.3949/ccjm.87a.ccc035. Epub ahead of print. PMID: 32434805. 3. Kramer JB, Brown DE, Kopar PK. Ethics in the Time of Coronavirus: Recommendations in the COVID-19 Pandemic. J Am Coll Surg. 2020 Jun;230(6):1114-1118. doi: 10.1016/j.jamcollsurg.2020.04.004. Epub 2020 Apr 9. PMID: 32278728; PMCID: PMC7194670.

Pediatric Anesthesiology

Pediatric Anesthesiology - 1 Anesthetic Challenges in a Patient with TANGO2 Gene Deletion and Coexistent DiGeorge Syndrome and Tetralogy of Fallot: A Medically Challenging Case

Ivana Wrobleski[1], Richard Hubbard[2], Nischal K gautam[3]

[1]University of Texas at Houston/McGovern Medical School, Houston, TX, [2]UT Health Science Center Houston, Houston, TX, [3]McGovern Medical School, Houston, TX

Introduction: MECRCN Syndrome (Metabolic Crises, Recurrent, with Rhabdomyolysis, Cardiac Arrythmias, and Neurodegeneration) is a recently described disorder involving autosomal recessive deletions of the TANGO2 gene.[1,2] This gene is involved in vesicular transport between the Golgi and endoplasmic reticulum, with deletions causing stress on both organelles.[2] The resultant disease is characterized by long-term neurological decline, interspersed with acute metabolic crises during times of stress (Table 1&2).[1-6] Reported triggers for crises include fasting, dehydration, infection, fever, and general anesthesia.[1,3,6] Metabolic crises are often a late finding, though they may occur as young as infancy.[2,7] As DiGeorge Syndrome is caused by a deletion of 22Q11 (including the TANGO2 gene) on one allele, it is possible for the two conditions to coexist if a TANGO2 mutation exists on the other allele. The following report describes the anesthetic challenges of a patient with simultaneous MECRCN and DiGeorge Syndromes.

Methods: Case Report: A 2-year-old patient with a history of Tetralogy of Fallot, as well as MECRCN and DiGeorge Syndromes presented for outpatient brain MRI and cardiac CT. The patient had undergone cardiac surgery as a neonate and at one year of age. In both cases, frequent intraoperative blood gases were drawn to monitor for signs of metabolic distress and he had close observation in the ICU postoperatively. For this case, anesthetic goals included avoidance of metabolic crisis with safe and expeditious same-day discharge. A smooth inhaled induction was achieved, IV access was established, and he was intubated. Venous blood gases were drawn hourly to monitor lactate and glucose levels, both of which remained normal. To maintain euvolemia and avoid hypoglycemia, 12mL/kg of dextrose-containing crystalloid was given over the two-hour case. Deep tracheal extubation was performed, with dexmedetomidine used to prevent emergence agitation. In the PACU, the patient was closely monitored for signs of acute crisis and to ensure adequate oral intake. He was discharged home without complication.

Conclusion: MECRCN syndrome is a previously unknown genetic condition which places patients at significant risk for perioperative metabolic crises. This risk was further elevated in this patient with a history of congenital heart disease and the well-documented anesthetic risks associated with DiGeorge Syndrome. Preventing crisis events requires careful anesthetic planning to ensure adequate preoperative and intraoperative hydration, stress reduction (adequate anxiolysis, deep extubation when appropriate, avoidance of hemodynamic swings, etc) and careful monitoring for laboratory and clinical signs of metabolic derangement. If metabolic crises do occur, it can be difficult to differentiate from other crises such as malignant hyperthermia. Treatment includes hydration, treatment of metabolic acidosis, and management of end organ complications (Table 3).[2-4,6]

References: 1. Journal of Inherited Metabolic Disease. 1– 11. 2020. 2. American Journal of Human Genetics. Vol 98: 347- 357. 2016. 3. GeneReviews. Seattle (WA): University of Washington, Seattle; 1993- 2020. 4. Heart Rhythm Case Reports. Vol 6: 256- 260. 2020. 5. Frontiers in Pediatrics. Vol 8. 539. 2020. 6. University of Toledo Journal of Medical Sciences. Vol 7. 24-27. 2020. 7. Genetics in Medicine. Vol 21, 3. 601-607. 2019.

Table 1: Acute and Chronic Manifestions of MECRCN Syndrome						
	Neurologic	**Cardiac**	**Endocrine**	**Metabolic**	**Musculoskeletal**	**Hepatic**
Chronic	Developmental Delay	Hypertrophic Cardiomyopathy	Hypothyroidism		Motor impairment	
	Epilepsy		Adrenal Insufficiency		Spasticity	
	Ataxia				Musclar Atropy	
Acute	Encephalopathy	Long-QT Syndrome	Hypoglycemia	Lactic Acidosis	Muscle Weakness	Hepatopathy
	Seizures	Torsades/VT		Ketoacidosis	Rhabdomyolysis	

Table 2: Laboratory Findings in MECRCN Syndrome Crises
Hypoglycemia
Myoglobinuria
Elevated Creatinine Phophokinase (CPK)
Transaminitis
Elevated Aldolase
Lactic Acidosis
Acetylcarnitine Elevation
Ketonuria
Hyperammonemia

Table 3: Clinical Pearls in Management of MECRCN Crises
Adequate Hydration
Correction of metabolic acidosis
Treatment of underlying cause (infection, fever, etc)
Laboratory Assessment: Blood Gas, Glucose, Lactate, CPK, Ammonia, Urine Myoblobin & Ketones, TSH & Free T4, Cortisol, Transaminases, Aldolase, Acetylcarnitine Panel, Complete Metabolic
Neurologic Evaluation (especially for altered mentation/seizures)
Cardiac Evaluation including close rhythm monitoring, echocardiogram, and electrophysiology consultation
Rapid management of malignant arrhythmias
Postoperative PICU admission if not previously planned

Pediatric Anesthesiology - 2 Anesthetic Management Of A Child With Trisomy 21, Osteogenesis Imperfecta, Moya Moya Disease, And Uncontrolled Hyperthyroidism Undergoing Urgent Surgery.

Claude Abdallah[1], Connie Lin[2], Giannina Robalino[2]

[1]Children's National Health System, Washington, DC, [2]Children's National Hospital, Washington, DC

Introduction: Trisomy 21 may be associated with different comorbidities. Down's Syndrome and osteogenesis imperfecta (OI) have been associated with thyroid dysfunction. The combination of uncontrolled hyperthyroidism,, OI, Moya Moya disease and Trisomy 21 in a child requiring urgent surgery, presents a challenging anesthesia management with increased risk of complications.

Methods: A 7-year-old girl (21.8 kg) with a history of OI Type I, trisomy 21, stroke with residual left hemiparesis secondary to Moya Moya disease, asthma and hyperthyroidism sustained a femur fracture after a misstep without a fall over a Holiday weekend. Preoperative evaluation revealed poorly controlled hyperthyroidism (TSH 0.01 mcunit/ml, T3 321 ng/dl, T4 6.09 ng/dl). Free T4 was found to be high (7.74 ng/dl). Medications included Methimazole 1.25 mg daily, Baclofen 3ml TID, Aspirin 81mg daily, Beclamethasone Diproprionate HFA 2 puffs BID, Albuterol PRN, and Morphine PRN. Surgery was deferred. Aspirin was discontinued prior to surgery. After increasing dose of methimazole to 4mg PO BID and adding potassium iodide 65mg, PO QD, patient was assessed to be clinically euthyroid with occasional mild tachycardia probably consistent with pain from fracture although the patient was receiving intermittent morphine and diazepam doses. After a multidisciplinary discussion with the surgeon and endocrinologist, a decision was made to proceed with the surgery. One unit of platelet was given prior to incision. The patient was positioned very carefully on the operation table, and pressure points were padded adequately. Intravenous induction was titrated to achieve and maintain hemodynamic stability with the availability of vasoactive infusions, red blood cells and platelets. After securing the airway, adequate intravenous access and an arterial line was placed. The case proceeded and at the end of surgery, the patient's trachea was extubated, and neurological status verified. The patient was transferred to the intensive care unit in stable condition for postoperative monitoring and pain control.

Conclusion: Uncontrolled hyperthyroidism is challenging to manage in an urgent situation. It predisposes to hemodynamic instability and perioperative complications including the risk of thyroid storm. This patient's anesthetic management was further complicated by Moya Moya disease, the use of aspirin, and OI. Urgency of surgery should be weighed against the increased risk of anesthesia including cardiovascular, hemodynamic and neurological complications. Anesthetic concerns after preoperative evaluation include optimization of homeostasis, normalization of thyroid hormone levels prior to surgical intervention, and when not feasible in the case of urgent surgery, maximizing hemodynamic stability and preventing decompensation. This patient's multiple co-morbidities emphasizes the importance of communication and multidisciplinary discussions regarding the risks and benefits.

References: 1. Neurol Med Chir 2018;58(3):116-123
2. Orthopedics 2007;30(12):1043-9

Pediatric Anesthesiology - 3 Anesthetic Management of a Patient with S-Adenosylhomocysteine Hydrolase (AHCY) Deficiency

Cori Van Gorkom[1], Taylor Mastin[1], Emily Black[1], Joelle Karlik[1]

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

Introduction: S-Adenosylhomocysteine hydrolase (AHCY) deficiency is a rare congenital disorder in methionine metabolism that was first reported in 2004. Patients with AHCY deficiency may develop white matter atrophy, delayed myelination, progressive myopathy, hypotonia, developmental delay, hepatitis, and coagulation abnormalities. One case of cardiomyopathy associated with AHCY deficiency has also been reported. There are currently less than ten cases of ACHY deficiency reported in the literature and there are no formally recommended or established guidelines regarding genetic or anesthetic management in this population.

Methods: We present the anesthetic management of the first patient with AHCY deficiency at our institution.

Results: This case report describes a 19-year-old male presenting for liver biopsy in interventional radiology due to a history of transaminitis and elevated creatinine kinase. His presentation included psychomotor delay and progressive weakness, and he initially was thought to have Becker's muscular dystrophy. He was then correctly diagnosed with ACHY deficiency which was confirmed by Baylor Genetics (Houston, TX) when he was 19-years-old. He had limited recent history or lab work but had transaminitis (AST 174, ALT 348) and a CPK ranging in the 10,000's unit/L which was elevated from a year ago when it was in the 8,000's. He received an awake pre-induction IV and was placed on dextrose containing maintenance intravenous fluids. Preoperatively echocardiography and coagulation studies showed no abnormalities. He received an IV induction, and there were no difficulties in airway management. General anesthesia was maintained with a propofol infusion and opioids. Halogenated inhaled anesthetics and muscle relaxants were avoided due to concern for further rhabdomyolysis and/or hyperkalemia. His CK decreased from 10,065 to 8,608 units/L after anesthesia.

Conclusion: Patients with AHCY deficiency can safety receive general anesthesia. Developmental delay may require premedication. Pre-operative coagulation studies and echography should be considered given previously reported complications in this population. We avoided halogenated inhaled anesthetics and muscle relaxants including succinylcholine to prevent worsening rhabdomyolysis or hyperkalemia. Our patient experienced a postoperative decrease in his creatinine kinase level thought secondary to fluid hydration. In conclusion, anesthetic goals for patients with AHCY deficiency should focus on avoiding rhabdomyolysis, monitoring for potential coagulopathy and cardiomyopathy, and anxiolysis.

References: Baric I, Fumic K, Glenn B, et al. S-adenosylhomocysteine hydrolase deficiency in a human: a genetic disorder of methionine metabolism. Proc Natl Acad Sci U S A. 2004 Mar 23;101(12):4234-9. doi: 10.1073/pnas.0400658101. Epub 2004 Mar 15. Honzík T, Magner M, Krijt J, et al. Clinical picture of S-adenosylhomocysteine hydrolase deficiency resembles phosphomannomutase 2 deficiency. Mol Genet Metab. 2012 Nov;107(3):611-3.

Pediatric Anesthesiology - 4 Anesthetic Management of an Infant with Loeys-Dietz Syndrome Undergoing Major Abdominal Surgery

Sushan Gupta[1], Mark Teen[2]

[1]Montefiore Medical Center/Albert Einstein College of Medicine, New York, NY, [2]Montefiore Medical Center, Bronx, NY

Introduction: Loeys-Dietz syndrome (LDS) is a rare autosomal dominant vascular disorder characterized by aggressive arterial involvement with arterial tortuosity, high risk of aortic aneurysm, and aortic dissection.[1] Patients may also present with cervical spine instability, joint laxity, and scoliosis.[2] We herein report the anesthetic management of a patient with Loeys-Dietz Syndrome with congenital cardiac defects and VACTERL for Kasai Procedure.

Methods: Our patient was an 8-week old boy, ex-full term, with congenital biliary atresia. He was positive for SMAD 3 mutation, diagnostic of LDS type 3 syndrome. An echocardiography revealed severely dilated aortic root, measuring 1.8cm, and dilated ascending aorta measuring 1.4cm. Other cardiac defects on echocardiography included moderate tricuspid valve hypoplasia leading to stenosis and mild hypoplastic right ventricle. The patient also had a patent foramen ovale, a large atrial septal defect (ASD) with bidirectional flow, and patent ductus arteriosus with predominantly left to right shunt. Given the tricuspid hypoplasia and hypoplastic right ventricle and a large ASD, any increase in pulmonary vascular resistance can cause right to left shunting leading to oxygen desaturation. At birth, the patient was also diagnosed with VACTERL disorder with bilateral thumb hypoplasia and a single right kidney. Intraoperatively, the patient was anesthetized using titrated sevoflurane induction. We had multiple failed attempts at arterial and venous access due to difficulty in threading the cannula despite a flash of blood in the hub and ultrasound guidance. In the absence of an X-ray, we avoided excess head extension and ensured careful positioning throughout the procedure. Also, we planned for a caudal catheter for perioperative analgesia, but due to multiple failed attempts to thread the caudal catheter, the procedure was abandoned. We maintained a tight blood pressure control throughout the procedure. The patient's intraoperative saturation was maintained between 93-95% on FiO2 of 30-40%. At the end of surgery, the patient was transferred to the pediatric ICU, intubated for further monitoring.

Conclusion: This is the only case report, to the best of our knowledge, discussing about anesthetic management plan in an infant with both VACTERL and LDS undergoing major abdominal surgeries. Patients with LDS are at high risk of aortic dissection, even at a very young age.[3] Cervical instability is common and makes patients with LDS a potentially difficult airway.[2,4] The presence of scoliosis and other vertebral anomalies can make the caudal catheter placement difficult in these patients.[5] Also, vascular tortuosity can lead to difficult arterial and venous access.[1] When associated with other congenital cardiac defects, careful use of balanced anesthesia, normovolemia, and adequate analgesia are critical in managing these patients to avoid complications in the perioperative period.

References: 1. MacCarrick G, Black JH, 3rd, Bowdin S, et al. Loeys-Dietz syndrome: a primer for diagnosis and management. Genet Med 2014; 16: 576-87. 2. Kalra VB, Gilbert JW, Malhotra A. Loeys-Dietz syndrome: cardiovascular, neuroradiological and musculoskeletal imaging findings. Pediatr Radiol 2011; 41: 1495-504; quiz 616. 3. Malhotra A, Westesson PL. Loeys-Dietz syndrome. Pediatr Radiol 2009; 39: 1015. 4. Fuhrhop SK, McElroy MJ, Dietz HC, 3rd, MacCarrick GL, Sponseller PD. High prevalence of cervical deformity and instability requires surveillance in Loeys-Dietz syndrome. J Bone Joint Surg Am 2015; 97: 411-9. 5. Bressner JA, MacCarrick GL, Dietz HC, Sponseller PD. Management of Scoliosis in Patients With Loeys-Dietz Syndrome. J Pediatr Orthop 2017; 37: e492-e9.

Pediatric Anesthesiology - 5 Difficult Intubation due to Severe Post-Radiation Laryngeal Edema in a Pediatric Patient

Noel Shaheen[1], Chyongjy Liu[2], Titilopemi Aina[3]

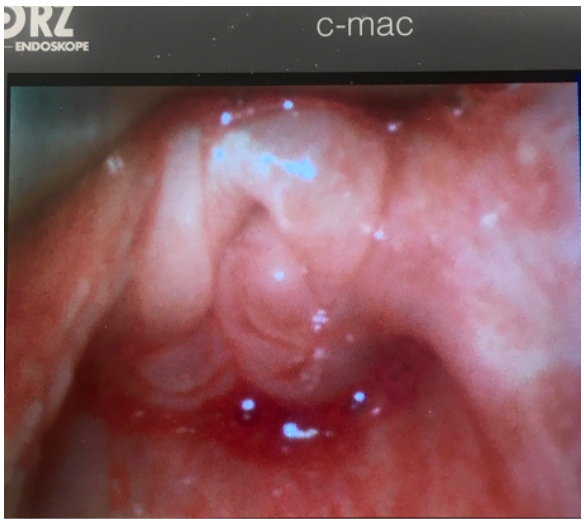
[1]Baylor College of Medicine, Houston, TX, [2]Baylor College of Medicine, Texas Children's Hospital, Houston, TX, [3]Texas Children's Hospital/Baylor College of Medicine, Houston, TX

Introduction: Radiotherapy (RT) is a treatment modality for head and neck cancers. However, RT-induced lymphatic disruption, local tissue inflammation, and successive fibrosis can lead to problems with swallowing, phonation, and breathing (1). Laryngeal edema (LE) is a notable sequela of RT to the head and neck. RT-induced LE may occur after the first few weeks of RT initiation. It tends to regress over time, however, it may persist for more than three months post-treatment in 15% of patients (2).

Methods: Case Description: A 3-year-old, 15 kg, male presented for complete oral rehabilitation at the ambulatory surgical center. Past medical history significant for right neck liposarcoma status post resection 10 months ago, and completion of proton XRT 2 months ago. Preoperatively, the patient showed no signs of difficulty breathing, had normal phonation and no stridor. Following mask induction, direct laryngoscopy proved to be challenging due to edematous airway structures. Video laryngoscopy was then utilized to intubate the patient, with a grade III Cormack-Lehane view obtained (Figure 1). A size 3.0 cuffed ETT was placed, which was 1.5mm smaller in internal diameter than the tube that was originally intended to prevent further airway trauma. IV dexamethasone was given for airway swelling. A leak test was performed before extubation. The Post Anesthesia Care Unit (PACU) had racemic epinephrine ready at the bedside for immediate administration upon arrival. After extubation, the patient had significant stridor. Otorhinolaryngology examined the patient in the PACU and admitted him to the hospital for overnight observation.

Conclusion: RT-induced LE commonly presents as hoarseness in adults, but symptoms do not typically manifest in children until significant airway compromise occurs. Thus, LE may not be recognized until the time of intubation. Anesthesia providers must be vigilant about obtaining RT history and remember that the lack of symptoms is not an exclusion of disease. Prevention of acute-on-chronic LE may be accomplished by downsizing the ETT and administering steroids3. Airway patency prior to extubation can be assessed by implementing a cuff leak test (3). Also, a rescue plan should be developed prior to induction, such as racemic epinephrine use (2,3). Finally, providers should have a low threshold for postoperative ventilation and admission.

References: 1. Fung K, Yoo J, et al. Vocal Function following radiation for non-laryngeal versus laryngeal tumors of the head and neck. Laryngoscope 2001; 111:1920-4. 2. Fu KK, et al. The significance of laryngeal edema following radiotherapy of carcinoma of the vocal cord. Cancer 1982;49(4):655-658. 3. Pluijms WA, et al. Postextubation laryngeal edema and stridor resulting in respiratory failure in critically ill adult patients: updated review. Critical Care 2015;19:295.



Pediatric Anesthesiology - 6 Old Meets New-Is It Time To Change The Approach To The Difficult Pediatric Airway?

Hemanth Baboolal[1], Dylan J Hunter[2], Aaron Low[2]

[1]UNC Chapel Hill, Chapel hill, NC, [2]UNC Chapel Hill, Chapel Hill, NC

Introduction: Historically, mask induction with maintenance of spontaneous ventilation has formed the cornerstone of difficult pediatric airway management. However, this technique does not preclude relaxation of upper airway tone, resultant obstruction and lost airway. We posit that a paradigm shift in thinking, utilizing an IV induction with an old drug - Ketamine and a new drug - dexmedetomidine is a safer alternative. Both medications not only maintain spontaneous ventilation but also upper airway tone. We present 3 such cases and contrast them with a fourth case where an inhaled induction almost led to a lost airway.

Methods: 1. 3 yo female w/ venous malformation of the tongue requiring sclerotherapy demonstrating severe macroglossia and limited mouth opening secondary to AVM. She was premedicated with PO midazolam and LMX was placed on her thigh. IM Ketamine was then administered to facilitate IV placement. She was then deepened with IV ketamine and glycopyrrolate. A nasal trumpet with ETT connector was then placed in the left nare and she was placed on pressure support via the ventilator. Nasal fiberoptic intubation via the contralateral nare was then performed. Spontaneous ventilation, hemodynamic stability and oxygenation were maintained throughout the induction. 2. 9 yo female with a sewing needle lodged in the tonsillar fossa with 4 inches protruding from the mouth required emergent laryngoscopy. IM glycopyrrolate and ketamine were administered to facilitate IV placement. She was then deepened with IV midazolam, dexmedetomidine and a low dose propofol infusion. Nasal fiberoptic intubation was achieved in the setting of spontaneous ventilation without desaturation. 3. 14 yo male w/ Hunters Syndrome and prior failed airway presented for tracheostomy in the setting of worsening upper airway obstruction. IM glycopyrrolate, ketamine, and dexmedetomidine were administered to facilitate IV placement with subsequent titrated induction with IV ketamine and dexmedetomidine. A two handed mask with oral airway provided adequate ventilation and significantly better airway control than prior inductions. A nasal airway connected to the ventilator via ETT connector was placed for continuous oxygenation, and oral fiberoptic intubation was achieved in the setting of spontaneous ventilation without desaturation. 4. 3 yo female w/ sinusitis and OSA due to a rapidly growing nasal mass scheduled for endoscopic excision, underwent mask induction. Mask ventilation was achieved, but became inadequate following Rocuronium administration for intubation. She required video laryngoscopy for emergent intubation.

Conclusion: We believe that successful management of the difficult pediatric airway requires both maintenance of upper airway tone as well as spontaneous ventilation. Use of volatile agents can result in loss of one or both. When administered judiciously, ketamine and dexmedetomidine possess both qualities and can achieve safer intubating conditions. We believe this approach should form the cornerstone of inducing difficult pediatric airways, and a rethinking of historic inhalational induction.

References: PMID 18038146, PMID 30375140, PMID 18613935

Pediatric Anesthesiology - 7 Surgical Cardiac Denervation For Recalcitrant Arrhythmia In A Pediatric Patient

Adaora Chima[1], Erica Lin[1]

[1]Cincinnati Children's Hospital Medical Center, Cincinnati, OH

Introduction: Ventricular arrhythmias are typically uncommon events in the pediatric population. Primary arrhythmias occurring in children without structural heart disease are thought to be caused by cardiac ion channelopathies. Secondary arrhythmias are due to congenital malformations, electrolyte derangements, surgical scarring, inflammation and medications. Management of recalcitrant arrhythmias includes antiarrhythmics, implantable cardioverter-defibrillator (ICD) devices, and catheter-based ablation techniques. Surgical thoracic sympathectomy has also been successfully used in managing cardiac arrhythmias that have proved intractable to other treatment modalities. We describe the multidisciplinary approach to, and perioperative course of a pediatric patient with intractable ventricular arrhythmia, who was treated with left thoracoscopic sympathectomy.

Methods: A 14 year old male with a history of catecholamine polymorphic ventricular tachycardia (CPVT) secondary to ryanodine receptor gene mutation was admitted on account of multiple ICD shocks for ventricular tachycardia (VT), after missing a scheduled dose of nadolol and flecainide. Of note, this patient's autism disorder contributes to medical non-compliance and difficulty with activity restriction. Eighteen months earlier, the patient had an automatic cardioverter-defibrillator device placed following aborted ventricular fibrillation cardiac arrest. Following admission, VT was controlled with medication. However, on graded exercise stress test, the patient had multiple runs of nonsustained VT and was therefore scheduled for thoracoscopy and cervical sympathectomy. Perioperative course: Preoperatively, a multidisciplinary plan was made. The general surgeons were the primary surgeons, while Cardiology-Electrophysiology and Cardiac Surgery teams were on standby to manage complications if they arose. Patient was premedicated with intravenous (IV) dexmedetomidine. General anesthesia was induced with IV lidocaine, fentanyl, propofol and rocuronium. The airway was secured with a 35 French double-lumen endotracheal tube to provide lung isolation. External defibrillation pads were placed, and the ICD was turned off. On obtaining additional vascular access, the patient's lines included 2 peripheral IVs (18g and 22g), 22g arterial line, and 7 Fr triple-lumen internal jugular venous access. General anesthesia was maintained with inhaled sevoflurane and IV fentanyl, and rocuronium. Patient was positioned in the right lateral decubitus position. In the event of ventricular arrhythmia, the external defibrillator was available; additionally, the electrophysiologist was present to restart the implanted defibrillator if needed. Infusions of epinephrine and lidocaine, and bolus esmolol dosing were immediately available as medical therapeutics. In the event of circulatory collapse from intractable arrhythmias, the plan was to turn supine for femoral cannulation for ECMO initiation by the cardiac surgeons. The intraoperative course was uneventful. After thoracoscopic access to the left pleural cavity was obtained, the sympathetic chain was identified, injected with bupivacaine and the stellate ganglion excised. Cardiac rhythm remained in sinus bradycardia and normal sinus rhythms throughout and did not require pharmacologic or electrophysiological intervention. Postoperative course: Postoperative episodes of premature ventricular contractions, bigeminy, trigeminy and couplets were controlled with increased beta blockade. The patient subsequently developed a hydropneumothorax which resolved with chest tube placement. He was discharged home on postoperative day 4, on rate control medication, with no further reported events at the time of this review (5 weeks post operatively).

Conclusion: Surgical sympathectomy can be performed safely and effectively in the pediatric population. It can be used as an adjunct strategy alongside other well established modalities and approaches to intractable arrhythmia treatment. A collaborative team approach is necessary for effective and efficient execution of contingency plans, in the event of perioperative complications.

References: 1. Roggen A, Pavlovic M, Pfammatter JP. Frequency of spontaneous ventricular tachycardia in a pediatric population. Am J Cardiol. 2008;101(6):852-854 2. Ackerman M.J. Cardiac channelopathies: it's in the genes. Nat Med.

2004;10:463-464. 3. Brugada, J., Blom, N., Sarquella-Brugada, G., Blomstrom-Lundqvist, C., Deanfield, J., Janousek, J., Rosenthal, E. (2013). Pharmacological and non-pharmacological therapy for arrhythmias in the pediatric population: EHRA and AEPC Arrhythmia Working Group joint consensus statement. Europace, 15(9), 1337-1382. doi:10.1093/europace/eut082 4. Hofferberth SC, Cecchin F, Loberman D, Fynn-Thompson F. Left thoracoscopic sympathectomy for cardiac denervation in patients with life-threatening ventricular arrhythmias. J Thorac Cardiovasc Surg. 2014 Jan;147(1):404-9. doi: 10.1016/j.jtcvs.2013.07.064. Epub 2013 Oct 24. PMID: 24268954.

Pediatric Anesthesiology - 8 Use of Minimally Invasvie Anesthesia in Sutureless Circumcision

Seung Mi Oh[1], Galila Flatow[1], Daniel Ramirez Parga[2], Mark Teen[2], Roshan Patel[3]

[1]Albert Einstein College of Medicine, Bronx, NY, [2]Montefiore Medical Center, Bronx, NY, [3]Montefiore Medical Center, Bronx , NY

Introduction: Pediatric circumcision using 2-octyl cyanoacrylate (2-OCA) for skin closure, also known as sutureless circumcision, has become increasingly popular over the last decade as a safe, efficient, and cost-effective alternative to traditional circumcision using sutured closure [1, 2]. Several studies have demonstrated reduced surgery duration, reduced cost, fewer complications and better cosmesis compared to sutured circumcision, with operating time less than 15 minutes [3-5]. Despite the changing landscape, there is little information regarding the best anesthetic management to accommodate the shortened procedure. Unlike neonatal circumcisions, which are performed with local anesthesia, pediatric circumcisions typically require general anesthesia. Traditionally, this can involve perioperative analgesic medication and inhalation induction with insertion of an intravenous line (IV), which can be difficult and time consuming in pediatrics. It might also involve a caudal, pudendal, or penile nerve block, and airway management with supraglottic airway device (SGA) or endotracheal tube [6]. Comparatively short procedures such as the bilateral myringotomy and tympanostomy (BMT) are often performed without IV, and typically with the patient spontaneously breathing [7]. Fittingly, authors believe the same anesthetic protocol could be applied to the shortened circumcision protocol with no significant risks. To date, there is no literature on the use of IV-less, spontaneous ventilation general anesthesia in pediatric sutureless circumcision. In this case series, we describe five post-neonatal circumcision cases with a rapid anesthesia protocol, and report the outcomes, complications, and post-anesthesia care unit/discharge times. Adaptation of anesthetic management for the surgical procedure may be able to enhance the efficiency and cost-effectiveness of circumcision without compromising quality of care.

Methods: In this report, we explored different anesthesia technique to improve operating room efficiency. Five cases of pediatric circumcision under rapid anesthesia were reviewed using the electronic medical record. Excluded from the series were patients categorized as ASA class III-VI, those with a known allergy to fentanyl, a history of postoperative vomiting (POV), known airway problems (obstructive sleep apnea, craniofacial syndromes), or known cardiac, pulmonary, or multisystem disease. The rapid anesthesia protocol involved the following: 1) pre-operative oral acetaminophen 2) general anesthesia with inhaled induction 3) intranasal fentanyl 4) dorsal penile nerve block 5) surgery, and 6) post-operative oral ibuprofen for pain scores 6/10 an above on a numerical pain scale. The following elements were examined, in a qualitative manner: in-room anesthesia-related complications, post-operative anesthesia related complications including pain and POV, anesthesia start time to discharge time, in-post-anesthesia care unit (PACU) time to discharge time, and other notable anesthesia-related events. In our series, the average anesthesia to discharge time was 150 min, and the average in-PACU time was 95 minutes. Only one of the five cases had an intraoperative complication during anesthesia induction that required airway management.

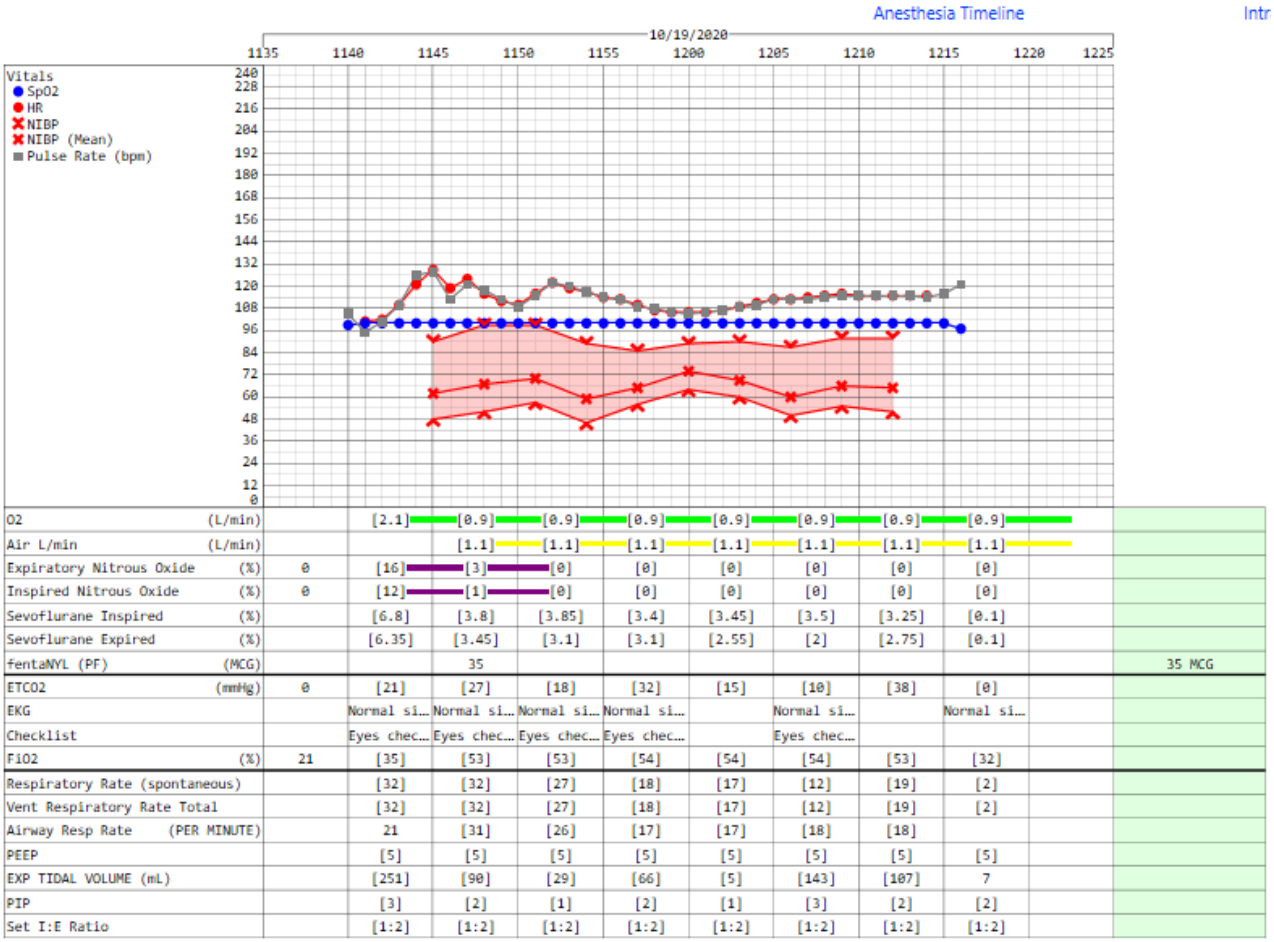
Conclusion: Given an increase in circumcisions using 2-OCA with operation times less than 15 minutes [3 ,4], the development of a protocol based on other short surgical procedures can lead to increased efficiency and decreased costs while minimizing complications [6]. In this case series, the average anesthesia start to discharge time among five cases was 150 min, and the average in-PACU to discharge time was 95 min. In comparison, among five randomly selected circumcision cases using the traditional anesthetic method, the average anesthesia start to discharge time was 184 min, with in-PACU to discharge time of 129 min. Overall, minimally invasive anesthesia showed about 18.5% and 26.4% reduction from the total time and in-PACU time, respectively. While our aim is not to compare superiority of either method, we report that this anesthesia protocol was shorter than our usual average, without compromising effectiveness.We report this adaptation of anesthetic may be able to enhance the efficiency and cost-effectiveness of circumcisions without compromising patient health, and would benefit from future studies.

References: Sutureless circumcision using 2-octyl cyanoacrylate (Dermabond): appraisal after 18-month experience. Urology 2007, 70(4):803-806. Sutureless and scalpel-free circumcision--more rapid, less expensive and better? J Urol 2010, 184(4 Suppl):1758-1762. The advantages of cyanoacrylate wound closure in circumcision. Pediatr Surg Int 2011, 27(8):879-883. Sutureless Circumcision - An Indian Experience. Indian J Urol. 2011 Oct-Dec;27(4) 475-478 Sutureless versus sutured circumcision: A comparative study. Urol Ann 2019, 11(1):87-90. Anesthesia methods used by anesthetic specialists for circumcision cases. National survey study for Turkey. Saudi Med J 2017, 38(1):75-81. Use of intranasal fentanyl in children undergoing myringotomy and tube placement during halothane and sevoflurane anesthesia. Anesthesiology 2000, 93(6):1378-1383.

Table 1. Comparison of average times spent in the OR and PACU between fast anesthesia protocol vs. traditional anesthesia protocol

	Minimally Invasive Anesthesia (N = 5)	Traditional Anesthesia (IV, SGA) (N = 5)
Average Anesthesia Start to Discharge time (min)	150	184
Average In-PACU to Discharge time (min)	95	129

Figure 1. Anesthesia Record for Case 3



O2	(L/min)		[2.1]	[0.9]	[0.9]	[0.9]	[0.9]	[0.9]	[0.9]	[0.9]	
Air L/min	(L/min)			[1.1]	[1.1]	[1.1]	[1.1]	[1.1]	[1.1]	[1.1]	
Expiratory Nitrous Oxide	(%)	0	[16]	[3]	[0]	[0]	[0]	[0]	[0]	[0]	
Inspired Nitrous Oxide	(%)	0	[12]	[1]	[0]	[0]	[0]	[0]	[0]	[0]	
Sevoflurane Inspired	(%)		[6.8]	[3.8]	[3.85]	[3.4]	[3.45]	[3.5]	[3.25]	[0.1]	
Sevoflurane Expired	(%)		[6.35]	[3.45]	[3.1]	[3.1]	[2.55]	[2]	[2.75]	[0.1]	
fentaNYL (PF)	(MCG)			35							35 MCG
ETCO2	(mmHg)	0	[21]	[27]	[18]	[32]	[15]	[10]	[38]	[0]	
EKG			Normal si...	Normal si...	Normal si...	Normal si...		Normal si...		Normal si...	
Checklist			Eyes chec...	Eyes chec...	Eyes chec...	Eyes chec...		Eyes chec...			
FiO2	(%)	21	[35]	[53]	[53]	[54]	[54]	[54]	[53]	[32]	
Respiratory Rate (spontaneous)			[32]	[32]	[27]	[18]	[17]	[12]	[19]	[2]	
Vent Respiratory Rate Total			[32]	[32]	[27]	[18]	[17]	[12]	[19]	[2]	
Airway Resp Rate	(PER MINUTE)		21	[31]	[26]	[17]	[17]	[18]	[18]		
PEEP			[5]	[5]	[5]	[5]	[5]	[5]	[5]	[5]	
EXP TIDAL VOLUME (mL)			[251]	[90]	[29]	[66]	[5]	[143]	[107]	7	
PIP			[3]	[2]	[1]	[2]	[1]	[3]	[2]	[2]	
Set I:E Ratio			[1:2]	[1:2]	[1:2]	[1:2]	[1:2]	[1:2]	[1:2]	[1:2]	

Pediatric Anesthesiology - 9 Two Novel Mutations in the CHD7 Gene in Patients with Typical and Mild Signs of CHARGE Syndrome Occurring with Esophageal Atresia

Devon M Evanovich[1], Jue T Wang[2], Anne H O'Donnell-Luria[3], Russell W Jennings[2], Dusica Bajic[2]

[1]Tufts University School of Medicine, Boston, MA, [2]Boston Children's Hospital, Boston, MA, [3]Boston Children,Äôs Hospital, Boston, MA

Introduction: CHARGE syndrome (Coloboma, Heart defect, Atresia of the choanae, Retarded growth and development, Genital abnormalities, Ear anomalies/deafness) is a rare multiorgan genetic autosomal dominant disease with a prevalence of about 1/10000 live births [1]. It is associated with mutations in the chromodomain-helicase-DNA-binding protein 7 (CHD7) gene [2]. As outlined in its acronym, CHARGE syndrome has a wide clinical symptom variability that continues to be investigated. Although CHARGE syndrome is known to be associated with esophageal atresia (EA) and tracheoesophageal fistulas in 15-20% of cases [3], there are only a few clinical case descriptions of CHARGE syndrome genetics in the context of EA. We present two patients with novel CHD7 variants with variable clinical expression of CHARGE syndrome occurring with EA. As such, it is a novel contribution to our knowledge of the phenotypic spectrum of CHD7 mutations.

Methods: CASE REPORTS. Patient 1 is a male infant born at 40 weeks of gestation to non-consanguineous parents. According to the Sanlaville and Verloes diagnostic criteria [1], Patient 1 presented with typical CHARGE syndrome with two out of three major CHARGE anomalies and four out of five minor CHARGE anomalies (Table 1). In addition, Patient 1 had a rare type of EA - type D, occurring <1% of EA cases [4], which was repaired by primary anastomosis. Genetic testing revealed a novel likely pathogenic essential splice site variant in the CHD7 gene which the parents had not been tested for at the time of this report. Patient 2 is a male infant born at 38 weeks after an in vitro fertilization pregnancy to non-consanguineous parents. Patient 2 did not meet diagnostic criteria for typical, partial, or atypical CHARGE syndrome. Instead, he had what is described as a mild CHARGE phenotype [5, 6] since he had none of the major CHARGE anomalies and presented with only one out of five minor CHARGE anomalies (Table 1). Patient 2 also presented with a rare type of esophageal atresia - type A, occurring in 7% of EA cases [4] that required complex repair via the Foker process [7]. Genetic testing revealed a novel paternally inherited CHD7 missense variant of uncertain significance. The father's previous medical history was only significant for cryptorchidism and varicocele. Table 1 lists the patients' novel variants and a summary of their anomalies associated with CHARGE syndrome.

Conclusion: The described two novel variants in the CHD7 gene expand the current knowledge of typical and mild expression of CHARGE syndrome occurring with EA. Patient 1 had typical CHARGE syndrome, and a rare type D EA that required direct anastomosis repair. In contrast, Patient 2 had a mild CHARGE phenotype and a rare type A EA that required complex repair with Foker process. The variable expression of mild CHARGE phenotypes with the same CHD7 variant in the case of patient 2 and his father calls for early genetic testing even in extremely mild anomalies known to be associated with CHARGE. Future studies should also look into the clinical complexity of CHARGE syndrome in relation to EA severity.

References: [1] Sanlaville, D., European Journal of Human Genetics, 2007. 15(4): p. 389-99. [2] Vissers, L.E., et al., Nat Genet, 2004. 36(9): p. 955-7. [3] Lee, K.D., et al., Pediatr Surg Int, 2008. 24(10): p. 1153-6. [4] Spitz, L., Orphanet journal of rare diseases, 2007. 2: p. 24-24. [5] Hale, C.L., et al., American journal of medical genetics, 2016. 170A(2): p. 344-354. [6] Bergman, J.E.H., et al., Journal of Medical Genetics, 2011. 48(5): p. 334-342. [7] Bairdain, S., et al., J Pediatr Surg, 2015. 50(6): p. 933-7.

Table 1

	Patient 1	Patient 2
Genetic Report		
Point mutation	c.4644+1G>T	c.4613C>T p.Ala1538Val
Genetic mutation description	Nucleotide change from Guanine to Thymine at a splice site that likely conferred loss of function	Nucleotide change from Cytosine to Thymine resulting in a missense change of Alanine to Valine at amino acid 1538
Developmental delay	**Yes**	**No**
Congenital Anomalies		
CHARGE Major Anomaly		
Coloboma	Bilateral chorioretinal Colobomas	None
Choanal atresia	None	None
Hypoplasia of semicircular canals	Absent semicircular canals	None
CHARGE Minor Anomaly		
Rhombencephalic dysfunction	Bilateral sensorineural hearing loss Hypoplastic left cranial nerve VIII	None
Hypothalamo-hypophyseal dysfunction	None	None
Malformation of the ear	Eustachian tube dysfunction Left cochlear canal atresia Dysplastic cochlea and vestibules	None
Malformation of mediastinal organs	1. Two atrial septal defects 2. Ventricular septal defect 3. Coarctation of the aorta 4. **EA type D**	1. Patent ductus arteriosus 2. **EA type A**
EA repair	Primary anastomosis	Foker process

Table 1. Summary of Genetic and Clinical Presentation of Two Infants with Novel CHD7 Mutations Occurring with Esophageal Atresia (EA). According to Sanlaville and Verloes diagnostic criteria [1], **typical** CHARGE has: three major OR two major + two minor anomalies; **partial** CHARGE has: two major + one minor anomalies; and **atypical** CHARGE has: two major but no minors OR one major + two minor anomalies. Patients that have only minor CHARGE anomalies have been described as having a mild phenotype [5, 6].

Pediatric Anesthesiology - 10 Multiple Procedures In Different Hospital Locations Under One Anesthetic In Acute Trauma Pediatric Patient

Alyssa K Streff[1], Alexandra Baumgarten[1], Hannah Lovejoy[1], Camila Walters[1]

[1]Vanderbilt University, Nashville, TN

Introduction: Background: Trauma is a major cause of pediatric morbidity and mortality (1) . Motor vehicle collisions (MVC) account for 59% of mortality in ages 5-14 (1). Many of these require complex care that involves multiple care teams during imaging and emergent surgery under general and regional anesthesia, along with pediatric intensive care (PICU) admissions, Multiple procedures may expose them to several anesthetics and care transitions. This can lead to parental and child distress and loss of information, which can negatively affect care (2). We present a case of a patient involved in an MVC that required exploratory laparoscopy, an MRI to assess spinal injuries, and posterior spinal fusion under one anesthetic coordinated by one anesthesiology team, multiple surgery teams, and imaging proceduralists.

Methods: Clinical Course: A 3-year-old previously healthy female presented to the ED as a lap belt restrained passenger in an MVC with lumbar deformity and inability to move lower extremities. Labs showed elevated transaminases and lipase. Imaging showed hepatic contusion, mesenteric hematoma, small bowel injury, and a L2 chance fracture with retroperitoneal hematoma. She was transferred to the OR for an exploratory laparoscopy where large hematomas were noted with viable bowel. Rapid sequence induction with cervical spine precautions was performed. Arterial and subclavian central line were placed to maintain mean arterial pressures (MAP) in the setting of spinal cord injury. Maintenance was achieved with sevoflurane. She received 350ml RBCs and norepinephrine to maintain MAPs. She was transferred to the MRI suite and orthopedic spine was consulted due to L2 flexion distraction and spinal cord injury. She returned to the OR for an L1-L3 posterior spinal fusion. A total intravenous anesthetic was necessary for neuro monitoring. She transferred to the PICU extubated. On post-operative day 1, she was sitting up playing and.on day 12, she was transferred to rehab.

Results: Lessons Learned:
- Under one anesthetic, multiple injuries can be assessed and quickly treated.
- Abdominal and spinal injuries were stabilized prior PICU admission, eliminating multiple transfers to OR and imaging suites.
- Length of stay was potentially reduced by eliminating coordination of multiple surgeries.

Conclusion: Discussion: Our Institution offers coordination of care for elective surgeries in children undergoing multiple procedures in one day organized by the anesthesiology team, surgeons, and nurse practitioners (3). We have seen benefit in easing anxiety of patients and parents and also increasing the speed at which a patient is able to undergo treatment. This method may be employed in trauma care as well. There is paucity of data for pediatric trauma coordination of care and more data will help us understand the impact of coordinated care events.

References: 1. Ivashkov Y, Bhananker SM. Perioperative management of pediatric trauma patients. Int J Crit Illn Inj Sci. 2012;2(3):143-148. 2. Hoonakker PLT, et al. Information flow during pediatric trauma care transitions: things falling through the cracks. Intern Emerg Med. 2019 Aug;14(5):797-805. 3. VUMC Reporter. Care coordination program eases children's stress. https://news.vumc.org/2013/06/06/care-coordination-program-eases-children-stress/ on 10.15.2020.

Pediatric Anesthesiology - 11 Anesthetic Management of Pediatric Patient with Multiple Complex Comorbidities

Alexandra Baumgarten[1], Hannah Lovejoy[1], Alyssa K Streff[1], Camila Walters[1]

[1]Vanderbilt University, Nashville, TN

Introduction: At our tertiary children's hospital, patients often present for surgery while concurrently undergoing work-up for other complex medical issues that have the potential to impact anesthetic care. With an incomplete workup, the anesthetic plan must account for the worst potential forms of each pathology. We present a child with three medical conditions of indeterminate etiology leading to a challenge in optimal anesthetic selection.

Methods: A 15-kilogram 3-year-old former 24-week premature male with multiple medical conditions was undergoing evaluation for congenital myasthenic-like abnormality with hypotonia and ataxia, an undiagnosed inborn error of metabolism and increased ferritin, when he presented for esophagogastroduodenoscopy and colonoscopy to workup his persistent dysphagia and reflux.

Results: Preoperatively, an intravenous catheter was placed, and glucose-containing fluids were started. Due to a history of frequent vomiting, dysphagia, and aspiration pneumonias, a rapid sequence induction was performed with 5mg/kg propofol and 1.5 mcg/kg fentanyl, while paralytic was avoided given his unknown metabolic derangements and myasthenia. An endotracheal tube was placed, and lung protective ventilation was initiated. Maintenance anesthesia was achieved with both volatile and a propofol infusion to allow for low concentrations of each. At the conclusion of the case, the patient was successfully extubated and taken to recovery. He had no perioperative complications and was discharged on postoperative day 1.

Conclusion: As seen with our patient, the anesthetic plan for children with multiple complex medical conditions must be adaptable and tailored to a patient's unique circumstance. Patients with myasthenic like abnormalities tend to have a higher propensity for apneic spells and depressed postoperative respiratory muscle strength (1). They are sensitive to nondepolarizing neuromuscular blockers and require higher doses of depolarizing agents. Given our patient's hypotonia, we avoided paralytics to prevent a hyperkalemic response or residual weakness. Children with inborn errors of metabolism must have vigilant perioperative management to minimize stress on their bodies. These patients are at increased risk for aspiration, electrolyte and glucose abnormalities, coagulopathies and end-organ damage. Perioperative management must focus on acid-base control, and avoidance of hypoxia, hyperthermia and prolonged fasting (3). While the metabolic derangement in our patient was undiagnosed, we initiated dextrose containing fluids preoperatively, ensured normothermia, and euvolemia to maintain stability. Lastly, increased ferritin is indicative of iron overload which can lead to impaired function of multiple organ systems (4). Thorough preoperative screening for cardiac, hepatic and endocrine organ impairment is critical. Our child presented with a normal echocardiogram and reassuring laboratory values. While all anesthetics must be tailored to the individual patient, we were faced with the challenge of managing a child with incomplete medical workup for conditions that had the potential for anesthetic complications. We chose to take a conservative approach while considering the potential anesthetic implications of each.

References: 1. McBeth C, Watkins TG. Isoflurane for sedation in a case of congenital myasthenia gravis. Br J Anaesth 1996; 77:672–674. 2. Baum, Victor C., and Jennifer E. O'Flaherty. Anesthesia for Genetic, Metabolic, and Dysmorphic Syndromes of Childhood / Victor C. Baum, Jennifer E. O'Flaherty. Philadelphia: Lippincott Williams & Wilkins, 1999. Print. 3. Kloesel, Benjamin, and Robert S Holzman. "Anesthetic Management of Patients With Inborn Errors of Metabolism." Anesthesia and analgesia 125.3 (2017): 822–836. Web. 4. Shander, Aryeh et al. "Iron Overload and Toxicity: Implications for Anesthesiologists." Journal of Clinical Anesthesia 24.5 (2012): 419–425. Web.

Perioperative Anesthesia

Perioperative Anesthesia - 1 Anaesthesia For Thalidomide Embryopathy Survivors 60 Years On - A Case Report And Discussion

Shyrana A Siriwardhana[1], Dee Morrison[2], Simon Choong[1], Fergus Cairns[3]

[1]Wellington Hospital (HCA), London, UK, [2]The Thalidomide Trust, Saint Neots, Cambridgeshire UK, [3]NAM Publications, London, UK

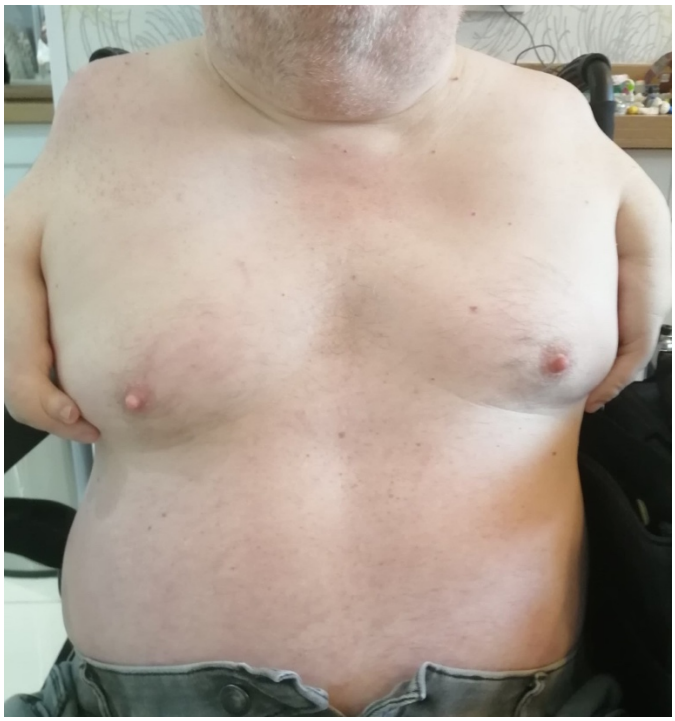
Introduction: The deaths and birth defects caused by the drug thalidomide were one of the worst medical accidents in history. Thalidomide Embryopathy (TE) presents as shortened or absent limbs and other external defects, but also causes profound disorders in the peripheral vascular system, heart defects, skeletal abnormalities and other multi-organ disorders. TE survivors present specific difficulties for anaesthesia, especially in the placement of lines, in airway management, in regional anaesthesia, in monitoring, and in anaesthetic dosing. Survivors of TE, now in their 50s to 60s, may need surgery and anaesthesia for conditions related both to their specific disabilities and to age-associated conditions. However the literature on anaesthesia for these patients has been sparse and in recent years almost non-existent. We present a case report on providing anaesthesia for a male TE survivor in his late 50s who needed a procedure to remove renal calculi, and where several previous attempts at anaesthesia had failed.

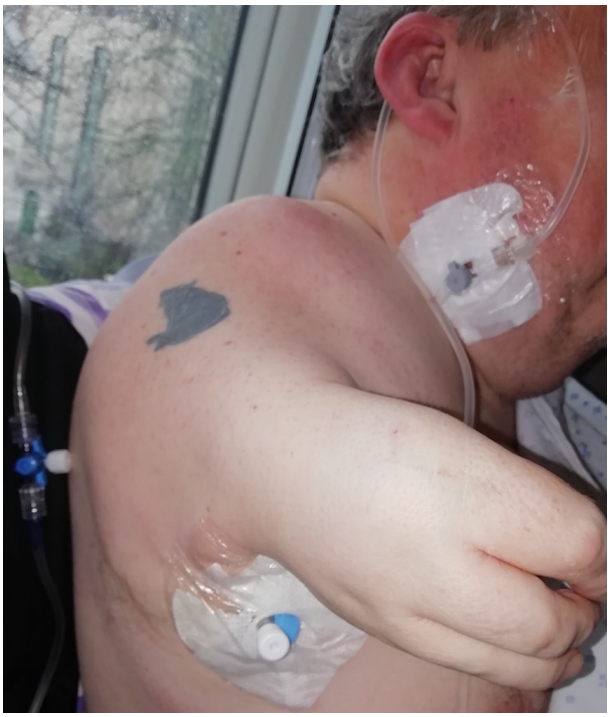
Methods: The local teaching hospital had attempted surgery, but no venous access could be obtained despite multiple attempts. Living 200 miles from our hospital, the patient was admitted the day before the operation. Important parts of his medical history were missing, and had to be supplemented by the patient. The patient had very short deformed limbs, a large head and a short and thick neck (see figure 1). He had a Mallampati 2 score and ASA III. There were small but visible veins in the anterior axillary wall, and the right external jugular was visible when lying down. A small cannula was inserted into the former and a larger one into the latter (see figure 2). Blood pressure and O2 saturation was detected by attaching, with difficulty, a small cuff on one of the small upper limbs; vital signs were normal. Plan A was a sub-arachnoid block, but after several unsuccessful attempts was abandoned. X-rays showed marked fusion of the lumbar vertebrae, revealing the reason for its failure. Following pre-oxygenation, GA was commenced using midazolam, fentanyl, ketamine and ondansetron, with the smallest recommended doses; sevoflurane was given with oxygen. When the patient was deeply anaesthetised and stable, a size 5 IGEL was inserted easily. Anaesthesia was maintained with 50% air/oxygen with sevoflurane, and with intermittent ketamine and fentanyl boluses. Throughout the operation vital signs were stable. Surgery was short and the patient was fully awake and pain-free after only 30 minutes. Surgery unblocked the main ureteric calculus; however, pus was released from a renal calyx and a decision was made to end the procedure to prevent urosepsis. A further operation was arranged for as soon as possible to ensure consistency in the team. This time surgery allowed complete removal of the staghorn calculus. The patient was discharged the following day fully satisfied with his care and treatment.

Conclusion: Anaesthesia for these patients should be undertaken by an experienced multidisciplinary team. Facilities to cope with complications must be available. Skeletal abnormalities may prohibit regional anaesthesia, and may lead to difficult intubation. MRI scanning to establish skull, neck, spinal and pelvic abnormalities is invaluable. Minimal anaesthetic doses should be used; lack of peripheral muscle may cause faster absorption and potential toxicity. Lower blood volume implies lower dilution. In addition, haemorrhage is a critical matter in people with reduced blood volume. Regional anaesthesia, if possible, needs to be short-acting; patients need to rapidly recover the use of lower limbs needed for routine activities. Lack of easily accessible blood vessels can pose challenging problems. Central venous or peripherally inserted lines with ultrasound guidance should be considered and, in elective surgery, performed in advance. Where this cannot be done, trans-oesophageal cardiac monitoring should be considered for intraoperative haemodynamic management. Patients should carry copies of, or have instant access to, their surgical, anaesthetic and investigatory history. In our case, spinal anaesthesia would not have been attempted had we a prior X-ray of the spine. The patient's previous experience of vascular access is invaluable. Patients with TE may have previous traumatic medical and

surgical experiences and should be treated with consideration and dignity.

References: Mansour S, Baple E and Hall CM. J Hand Surg Eur Vol, 44 (1): 96-108. 2019. Weinrich JM et al. Circulation Journal, 82(9):2364-2371. 2018. Mukhtar K, Jayaseelan S, Allsop E. European Journal of Anaesthesiology 2012, 29:353-354.





Perioperative Anesthesia - 2 Did 1,4-Butanediol Abuse Tear Open Our Patient's Stomach? It Certainly Caused His Near Fatal Withdrawal.

Samuel Jensen[1], Steven Bourland[2]

[1]University of Colorado Department of Anesthesiology, Aurora, CO, [2]University of Colorado Department of Anesthesiology, Denver Health Hospital, Denver, CO

Introduction: We document and discuss a potential side effect of chronic 1,4-butanediol abuse, a perforated viscus. We also present the challenges of managing post-operative acute GHB withdrawal.

Methods: A 37-year-old, 70kg male with past medical history of chronic polysubstance abuse presents to the ED complaining of abdominal pain. He admits to years of daily oral 1,4-butanediol (BD) every 3 hours for anxiolysis. A CT scan showed pneumoperitoneum concerning for a perforated ulcer. Urgent surgical intervention was planned. In the preoperative arena, about 3 hours after arrival to the ED, the patient appeared anxious but also seemed to exhibit signs of acute withdrawal with agitation, pressured speech, elevated mood, and inability to follow commands. He received 6 mg of IV midazolam without any effect on his acute status. Induction was uneventful and included 100 mcg of fentanyl, 290 mg of propofol, and 60 mg of succinylcholine. He was maintained on 1.7% sevoflurane and 1 mcg/kg/hr of dexmedetomidine (total of 70 mcg) infusion during the case. The patient was given 2 L of lactated ringers and 2mg of hydromorphone. His emergence was uneventful and he was transferred in a stable condition to the post-anesthesia care unit. As our patient emerged from anesthesia, he exhibited agitation, hypertension, disorganized speech, inability to follow commands, hyperthermia, hyperreflexia and tachycardia. Blood analysis revealed normal creatine kinase and a normal metabolic panel. The patient was given 12 mg of intravenous lorazepam during the course of his PACU stay, around 60 minutes. Upon arrival to the surgical ICU and over the next two days he received a total of 2480 mg IV phenobarbital (28 mg/kg) then was transitioned to a dexmedetomidine infusion ranging from 0.4 - 1.4 mcg/kg/hr and a ketamine infusion at 0.3 mg/kg/hr, these were maintained until POD 5 when he began to develop bradycardia likely from the dexmedetomidine. At this point his acute withdrawal was treated with 60-100 mg IV diazepam daily along with IV haloperidol 5-15 mg prn. Throughout his eight-day hospitalization, intravenous hydromorphone was used for analgesia until acetaminophen and oxycodone could be consumed. His formal EEGs were negative for seizure activity. And an H.pylori infection was treated with quadruple therapy. Our patient was discharged on a baclofen taper of 10 mg BID PO for 7 days, then 10 mg QD PO x 7 days then stop.

Conclusion: This case report highlights a potential side effect of BD leading to perforated viscus. Although he was H. pylori positive, chronic BD ingestion should be considered. Additionally, it highlights the importance in management of substance abuse in the perioperative setting. BD is a compound that is converted to gamma-hydroxybutyric acid (GHB) in vivo by alcohol and aldehyde dehydrogenase (1). After ingestion, BD is rapidly metabolized into GHB (2). GHB exhibits its pharmacological effects by acting as a GABA agonist, specifically the GABA-B receptor (3). Acute intoxication may present as central nervous system depression, agitation, bradycardia, respiratory depression, rhabdomyolysis, and vomiting (4,5,6). Clinical features of GHB withdrawal are similar to alcohol or benzodiazepine withdrawal given similar mechanisms of action. The withdrawal symptoms occur rapidly, in some studies within 6 hours of the last dose. These may progress to severe within 24 hours and include hallucinations, rhabdomyolysis, acute kidney injury, and even death (7). Duration of withdrawal ranges from 2 to 15 days and it is suggested that patients who use GHB in higher doses may experience more severe symptoms and have a longer withdrawal period (7). It has been hypothesized that resistance to benzodiazepine therapy might occur since these medications work at the GABA-A receptor rather than the GABA-B receptors that GHB utilizes; which also explains why baclofen tapers may benefit due to their GABA-B agonism (7,9). With phenobarbital, a loading dose of 250 mg and then increments of 125-250 mg IV every 15-30 minutes has been accepted to help control withdrawal symptoms (8). The research is lacking on how to effectively manage acute GHB withdrawal however this case study sheds light on benzodiazepine-resistant treatment. It emphasizes phenobarbital as an effective therapy to control the agitation and dexmedetomidine

as an adjuvant to consider when dosing of phenobarbital reaches near-toxic limits.

References: 1. Eur. J. Pharmacol., 473 (2–3): 149–52. 2003. 2. Clin Pharmacol Ther 2007; 81:178. 3. Drug Test Anal 2011; 3:417. 4. Emerg Med J 2011; 28:462. 5. Int J Drug Policy 2018; 58:137. 6. Clin Toxicol 2017; 55:432. 7. CNS Drugs 2017; 31:51. 8. Texas Commission on Alcohol and Drug Abuse. Austin, TX 2001. Available online at: http://www.dshs.state.tx.us/WorkArea/DownloadAsset.aspx?id=10417. 9. Addiction 2011; 106:442.

Perioperative Anesthesia - 3

Perioperative Management Of Presumed Carcinoid Syndrome In A Patient With Primary Bronchial Carcinoid Tumor

Elisa C Walsh[1], Daniel Ankeny[1]

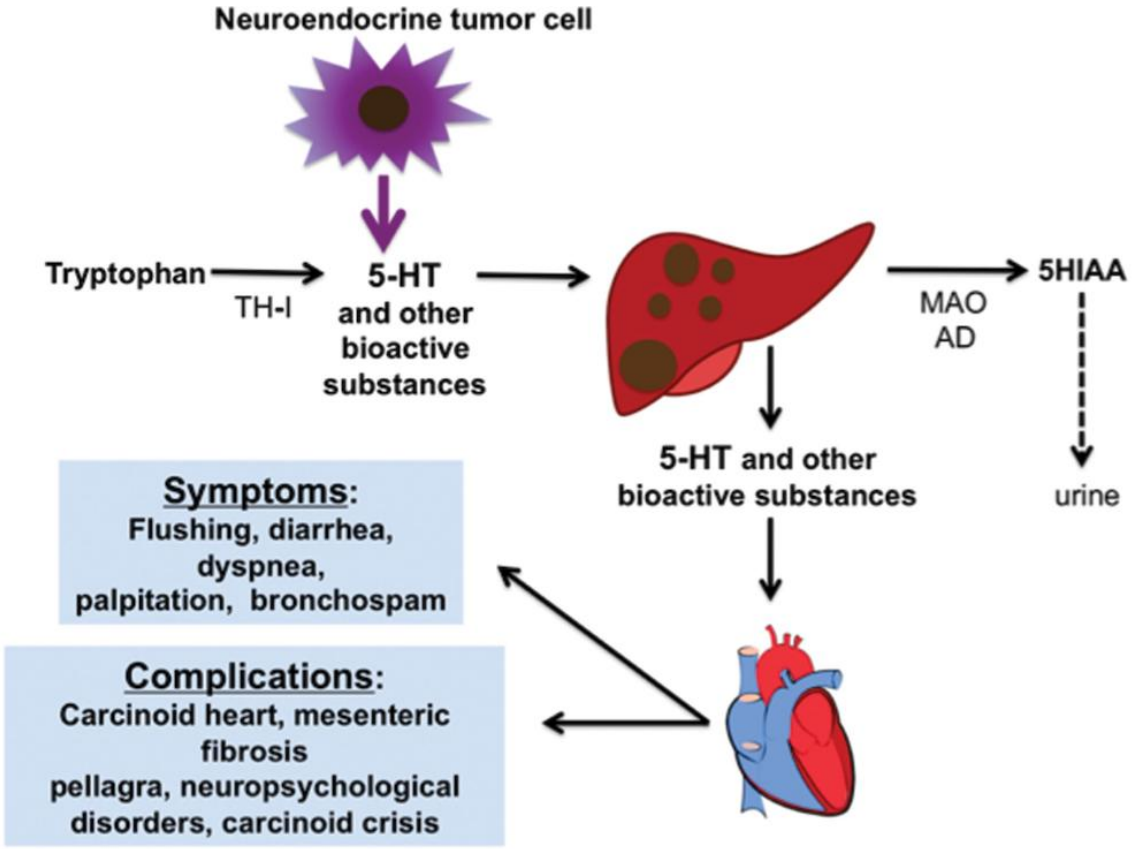
[1]Massachusetts General Hospital, Boston, MA

Introduction: Carcinoid syndrome is a potentially life-threatening disorder in which 5-hydroxytryptaine release from neuroendocrine tumors causes vasoactive symptoms such as flushing, diarrhea, tachycardia, and bronchospasm. Perioperative management relies on inhibition of tumor activity, avoidance of triggering stimuli and vigilance for carcinoid crisis manifesting as refractory hypotension, hypertension, arrhythmias, and/or bronchospasm (1, 2).

Methods: The patient was a 23-year-old woman with a past medical history significant for depression/anxiety and a newly diagnosed bronchial carcinoid tumor who presented for right middle lobectomy. She reported a 2-year history of progressive exertional dyspnea and recurrent episodes of dizziness, nausea, and facial flushing that ultimately led her to present to the emergency room. Computed tomographic angiography of the chest was performed due to concern for pulmonary embolism and revealed a 2.7cm right middle lobe bronchus tumor. She subsequently underwent a bronchoscopy and endobronchial ultrasound that demonstrated a vascular endobronchial lesion in the right middle medial segmental bronchus with complete obstruction of the bronchial lumen. Histopathology was consistent with carcinoid tumor. Transthoracic echography and pulmonary function tests were grossly normal. Given evidence of recurrent vasoactive symptoms, the patient was premedicated with intravenous diphenhydramine 25mg and octreotide 50mcg to prevent vasoactive peptide release, with additional doses and a continuous infusion of octreotide immediately available. Given low likelihood of a bronchial sleeve lobectomy, we deferred thoracic epidural placement and provided analgesia with intravenous fentanyl 100mcg and lidocaine 100mg at induction and hydromorphone 2mg in divided doses throughout the case. Depth of anesthesia was maintained between 0.7 and 1 MAC with sevoflurane. Both one-lung and two-lung ventilation were uncomplicated. An arterial line was placed post-induction for invasive hemodynamic monitoring but she did not have significant episodes of hypotension. She extubated uneventfully and no issues in the immediate recovery period.

Conclusion: In patients with suspected carcinoid tumor, it is essential to perform a thorough preoperative assessment of vasoactive symptoms, fluid and electrolyte abnormalities due to diarrhea, and potential right heart pathology. The literature recommends premedication with octreotide 100mcg TID for 2 weeks prior to surgery, but alternatively, octreotide 50-200mcg boluses and 50mcg/hr infusion can be used to rapidly reverse severe hypotension or bronchospasm. Intraoperatively, it is essential to maintain adequate anesthetic depth and analgesia, including regional or neuraxial techniques, to avoid carcinoid mediator release leading to potential carcinoid crisis.

References: 1. Antibes Consensus Conferenc. ENETS Consensus Guidelines for the Standards of Care in Neuroendocrine Tumors: Pre- and Perioperative Therapy in Patients with Neuroendocrine Tumors. Neuroendocrinology. 2017 Sep; 105(3): 245-254. 2. Mancuso K, Kaye AD, Boudreaux JP, Fox CJ, Lang P, Kalarickal PL, Gomez S, and Primeaux PJ. Carcinoid syndrome and perioperative anesthetic considerations. JClin Anesthesia. 2011 (23):329-341. 3. Rubin de Celis Ferrari AC, Glasberg J, Riechelmann RP. Carcinoid syndrome: update on the pathophysiology and treatment. Clinics. 2018 (73, 1).



Abbreviations: 5-HT: serotonin; TH-I: tryptophan hydroxilase I; MAO: monoamine oxidase; AD: aldehyde dehydrogenase

Perioperative Anesthesia - 4 Anesthetic Management Of Laparoscopic Adrenalectomy For Patient With Pheochromocytoma And Bilateral Carotid Artery Stenosis

Kristina L Michaud[1], Robert H Thiele[1], Katherine Forkin[2]

[1]University of Virginia Health System, Charlottesville, VA, [2]University of Virginia, Charlottesville, VA

Introduction: Resection of pheochromocytoma was historically associated with up to a 50% mortality rate. Preoperative alpha and beta blockade and titratable vasoactive agents intraoperatively are used in an attempt to both maintain adequate perfusion pressure to vital end organs and avoid significant hypertensive events that could lead to myocardial ischemia or stroke (1.) The potential risk of stroke is magnified in a patient with concomitant carotid artery stenosis. We present the following medically-challenging case involving a patient with both carotid artery stenosis and pheochromocytoma and discuss the multidisciplinary decision-making involved to optimize care.

Methods: 46-year-old, 86 kg male with PMH of DM2, HLD, hypothyroidism, gout, history of Hodgkin's lymphoma and recent diagnosis of bilateral carotid artery stenosis and pheochromocytoma presented for laparoscopic adrenalectomy. Six months prior, he experienced acute slurred speech, numbness and left facial twitching. Bilateral carotid artery duplex showed severe right common carotid artery stenosis and moderate left common carotid artery stenosis. During this same time, he was also being evaluated for a growing adrenal mass. Labs showed a 24-hour urine normetanephrines of 1400 pg/mL and free normetanephrine of 3.9 pg/mL. Laparoscopic adrenalectomy was scheduled for December 2020 for suspected pheochromocytoma. Following appropriate alpha and beta blockade and adequate rehydration, the patient presented for surgery with well-controlled blood pressure. The anesthesiologist, vascular surgeon and general surgeon agreed it was appropriate to proceed. He was pre-medicated with IV midazolam and pre-induction arterial line was placed. General anesthesia was induced with lidocaine and propofol with rocuronium for muscle paralysis. Intubation was performed on the first attempt with direct laryngoscopy. Sevoflurane was used for maintenance of anesthesia. Intraoperative hypotension was treated with volume resuscitation, norepinephrine and vasopressin. Insulin was administered for blood glucose elevations in the 300s. Cerebral oximetry remained within 20% of baseline. The operation was performed without complication. The patient was extubated and brought to PACU in stable condition. Post-op course was notable only for hyperglycemia. On post-op day 1, vitals were stable, pain well-controlled, ambulating with very little assistance, tolerating a regular diet and voiding independently; he was discharged with scheduled follow-up.

Conclusion: This patient had concurrent diagnoses of pheochromocytoma and carotid artery stenosis both requiring invasive intervention. Adrenalectomy and carotid endarterectomy have significant potential risks with delays in intervention increasing morbidity and mortality. Pheochromocytoma, a rare neuroendocrine tumor of the adrenal medulla, produces, stores and releases catecholamines into circulation causing headache, poorly controlled hypertension and palpitations (1.) Preop management involves antihypertensive treatment with alpha blockade then beta blockade, as well as sodium and fluid hydration (2.) Hypertensive crises during resection of pheochromocytoma or any surgical procedure can be provoked by noxious stimuli, positional changes and manipulation of the abdomen or tumor. Additionally, following the resection of the pheochromocytoma, significant hypotension may occur due to reduction in catecholamines, often resolving within 24 hours (1.) In our patient with symptomatic bilateral carotid artery stenosis, maintaining adequate cerebral perfusion pressure is particularly critical. With symptomatic carotid stenosis, revascularization is recommended as optimal treatment in stroke prevention with ideal timing of endarterectomy occurring within 14 days of symptoms (3.) Our patient was concomitantly undergoing workup for pheochromocytoma making it inappropriate to proceed with carotid endarterectomy before that workup was complete. It was decided that proceeding with pheochromocytoma resection with adequate preop alpha and beta blockade and hydration would be appropriate to perform first. Cerebral oximetry during the adrenalectomy allowed

for additional monitoring to identify cerebral hypoperfusion that could potentially be resolved with vasopressor administration or fluid resuscitation. This challenging case highlights the importance of multidisciplinary discussion and planning for complex patients to optimize outcomes.

References: 1. Perioperative Management of Pheochromocytoma. J Cardiothorac Vasc Anesth. 2017 Aug;31(4):1427-1439. 2. Pheochromocytoma and paraganglioma: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2014 Jun;99(6):1915-42. 3. Timing of Carotid Endarterectomy for Symptomatic Carotid Stenosis: A Snapshot of Current Trends and Systematic Review of Literature on Changing Paradigm towards Early Surgery. Neurosurgery. 2019 Aug 1;85(2):E214-E225.

Perioperative Anesthesia - 5 Atypical Hypoxia and Fever After Cardiopulmonary Bypass in a Healthy Young Man with Remote SARS-CoV-2 Infection

Linjia Jia[1], Marguerite Hoyler[1], Natalia S Ivascu[2], James Littlejohn[3]

[1]New York Presbyterian Hospital - Weill Cornell Medical Center, New York, NY, [2]Weill Cornell Medical College, New York, NY, [3]Weill Cornell Medicine, New York, NY

Introduction: More than one year into the coronavirus 2019 pandemic, recovered patients from severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection are increasingly presenting to operating rooms. Sparse existing literature describes uneventful perioperative courses of patients with active infections, including those undergoing cardiopulmonary bypass (CPB) surgeries.[1,2] Chronic SARS-CoV-2 sequelae remain largely uncharacterized and reports of surgical patients with remote infections are limited. We describe a case of a young man with known prior SARS-CoV-2 infection who presented for cardiac surgery, and who postoperatively experienced an atypical fever and profound hypoxia distinct from post-CPB fluid mobilization.

Methods: A 27 year old man with congenital bicuspid aortic valve (bAV) and worsening symptomatic aortic insufficiency (AI) presented for surgical intervention. He had a SARS-CoV-2 infection 9 months prior with mild symptoms that did not require hospitalization. He had no other medical history and was a non-smoker.

Preoperative transesophageal echocardiography (TEE) demonstrated normal right ventricular (RV) size and function, left ventricular (LV) eccentric hypertrophy, 46% LV ejection fraction, bAV with severe AI, and moderate-severe mitral regurgitation (MR). Intraoperative course was unremarkable, and he required low-dose inotropic support and no blood products coming off CPB. Postoperative TEE showed resolution of AI, trace MR, and unchanged RV and LV functions.

He was admitted to an intensive care unit (ICU) where he was weaned off inotropic infusions and extubated 16 hours later to nasal oxygen at 4 liters per minute (LPM). 14 hours post-extubation, he became febrile to 38.5°C, and developed acute hypoxia with an oxygen saturation (SaO2) in 70%s and arterial partial oxygen pressure of 44 mm Hg on 6 LPM nasal oxygen. He was placed on 60 LPM and 100% fraction of inspired oxygen via high flow nasal cannula (HFNC) with improvement to 80%s SaO2, but eventually required 4 hours of bilevel positive airway pressure support to achieve 95% SaO2. IV diuresis was initiated at this time. Chest x-ray (CXR) showed no pulmonary edema, small-moderate bilateral pleural effusions, and lower-lobe atelectasis. Mean pulmonary arterial systolic pressures (PASP) were 14-18 mm Hg, mean central venous pressures (CVP) were 6-8 mm Hg, and he remained normotensive. Tidal volumes on incentive spirometry were ≥1 liter. A repeat SARS-CoV-2 test was negative. Awake fiberoptic bronchoscopy showed scant mucoid secretions in the right-sided airways only. The patient was successfully weaned off HFNC on early postoperative day 3 (POD3), and to room air shortly thereafter. The rest of his course was uneventful; he was transferred out of ICU on POD3 and discharged on POD5.

Conclusion: This patient without pulmonary disease underwent CPB cardiac surgery 9 months after a SARS-CoV-2 infection, with sudden and profound hypoxia during the fluid-mobilization phase post-CPB not otherwise explained by fluid overload or infectious processes. Findings on CXR and bronchoscopy did not correlate with the degree of desaturation, mean PASPs and CVPs were not suggestive of acute cardiogenic pulmonary edema, and he improved quickly with routine diuresis and without antibiotics. In absence of other explanations, we consider the possibility of an underlying mechanism related to his prior SARS-CoV-2 infection.

Emerging data suggest that SARS-CoV-2 may cause chronic organ dysfunction and lung injury even in patients who have only mild illness.[3,4] A series of 13 patients had decreased pulmonary function tests and persistent ground glass opacities on chest imaging at time of clinical recovery.[5] A large study of 1733 patients showed that up to 22% of patients who did not require supplemental oxygen during acute illness continued to have impairment in pulmonary diffusion capabilities and abnormal imaging findings up to 6 months later.[6] These data are consistent with literature describing ongoing small-airway dysfunction and new-onset restrictive lung disease in SARS-CoV-2

survivors, independent of severity of acute pneumonia.[6,7]

This case of atypical hypoxia and fever after CPB surgery illustrates an example of possible long-lasting pulmonary injury after SARS-CoV-2 infection. Critical care anesthesiologists should have heightened clinical suspicion in patients with remote infections.

References: 1. Journal of Cardiac Surgery. 2020 Sep;35(9):2361-2363. 2. Journal of Cardiac Surgery. 2020 Jun;35(6):1351-1353. 3. Journal of the American Medical Association. 2020 Aug 11;324(6):603-605. 4. Infection. 2020 Jul 28:1-5. 5. Lancet. 2021 Jan 8:S0140-6736(20)32656-8. 6. Journal of Infection. 2020;81(2):e150-e152. 7. Pulmonology. 2020;S2531-0437(20)30245-2.

Perioperative Anesthesia - 6 Severe Pulmonary Mucormycosis in a Young Diabetic Adult Requiring Pneumonectomy.

Liliana Z Goelkel-Garcia[1], Kumar G Belani[2], Andrew Wilkey[3]

[1]University of Minnesota, Minneapolis, MN, [2]m Health Fairview Masonic Children's Hospital, Edina, MN, [3]University Of Minnesota, Minneapolis, MN

Introduction: Mucormycosis is a rare but serious disease that progresses rapidly requiring urgent intervention and life-saving care[1]. Immunocompromised patients and uncontrolled diabetics are prone to this uncommon and difficult to diagnose invasive Mucorales fungal infection[2, 3]. We describe the care of a critically ill 21-year-old female admitted with diabetic ketoacidosis (DKA) and concomitant invasive pulmonary mucormycosis requiring surgical care and intense medical therapy.

Methods: Report of the case: Our patient was admitted with type 2 diabetes mellitus (T2DM) and severe DKA. Her hemoglobin A1C was 14%. During her care for this problem there was radiological evidence of a right hilar mass. After gaining control of her DKA, she underwent a biopsy of the mass by interventional radiology. This revealed mucormycosis. She was negative for the SARS-CoV2 virus and the Aspergillus Galactomannan Antigen test was negative. She was transferred to a high-intensity unit to receive aggressive antifungal therapy with IV amphotericin. Despite this, reimaging after 48 hours of antifungal treatment there was rapid disease progression to the entire right lung including the posterior mediastinum and subcarinal space. There was no evidence of any extrapulmonary spread. She was prepared for urgent right extra-pulmonary pneumonectomy. In addition to standard ASA monitors, she received an arterial line, a thoracic epidural catheter, and a right Fem-fem cannula insertion for urgent cardiopulmonary bypass if required. All this was done after she received general anesthesia including a double lumen endotracheal tube for lung isolation. Stage 1 of the operation was done in the supine position. Median sternotomy revealed severe inflammation surrounding the right pulmonary artery, the anterior pericardium, the subcarinal space and a small pericardial effusion and extensive adenopathies. Uneventful intrapericardial division of right pulmonary artery, right superior pulmonary vein and right inferior pulmonary vein and trans pericardial mediastinal lymphadenectomy was achieved. A 19-French Blake drain was left in the pericardium. After this, the need for cardiopulmonary bypass was deemed unlikely and the femoral cannulas removed to complete the pneumonectomy in the left lateral decubitus position. This included a carinal bronchoplasty and latissimus dorsi flap buttress. Patency of the left main bronchus was confirmed by fiberscope bronchoscopy. Hemodynamic stability was ensured, and she was successfully extubated prior to transfer to the ICU. Postoperatively she developed right sided empyema and a bronchopleural fistula requiring drainage and repair with an Eloesser flap on postoperative day 13 and an omental flap on the bronchial stump along with a tracheostomy for respiratory support on day 18. She remains stable breathing effortlessly via the tracheostomy while receiving ongoing antifungal treatment and antibiotics for Prevotella isolated from her lung abscess.

Conclusion: We describe the care of a patient with extensive mucormycosis in a poorly controlled young diabetic requiring extended perioperative anesthesia, surgical and intensive care. This often fatal infection requires a quick and accurate diagnosis with implementation of aggressive care as demonstrated to allow survival[1-3]. After control of this infection the patient will need close monitoring, care for her T2DM and life-style changes to prevent similar serious recurrences.

References: 1. Cornely, O.A., et al., Global guideline for the diagnosis and management of mucormycosis: an initiative of the European Confederation of Medical Mycology in cooperation with the Mycoses Study Group Education and Research Consortium. Lancet Infect Dis, 2019. 19(12): p. e405-e421. 2. Santo, M., et al., Pneumonectomy in pulmonary mucormycosis complicating Behcet's disease. Postgrad Med J, 1986. 62(728): p. 485-6. 3. Vercillo, M.S., M.J. Liptay, and C.W. Seder, Early pneumonectomy for pulmonary mucormycosis. Ann Thorac Surg, 2015. 99(3): p. e67-8.

Regional Anesthesia

Regional Anesthesia - 1 Combined Ilio-Inguinal Block And Fascia Illicia Block For Balloon Aortic Valvuloplasty In An Elderly Patient With A Hip Fracture: A Case Report

Robert Suriani[1], Juliet Jackson[1], David Maduram[1]

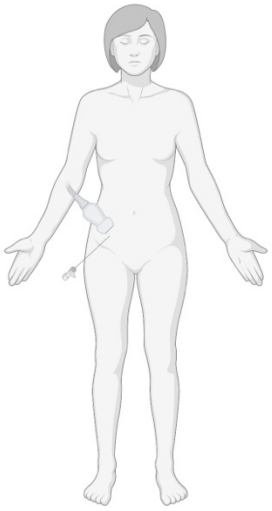
[1]St. Vincent's Medical Center, Bridgeport, CT

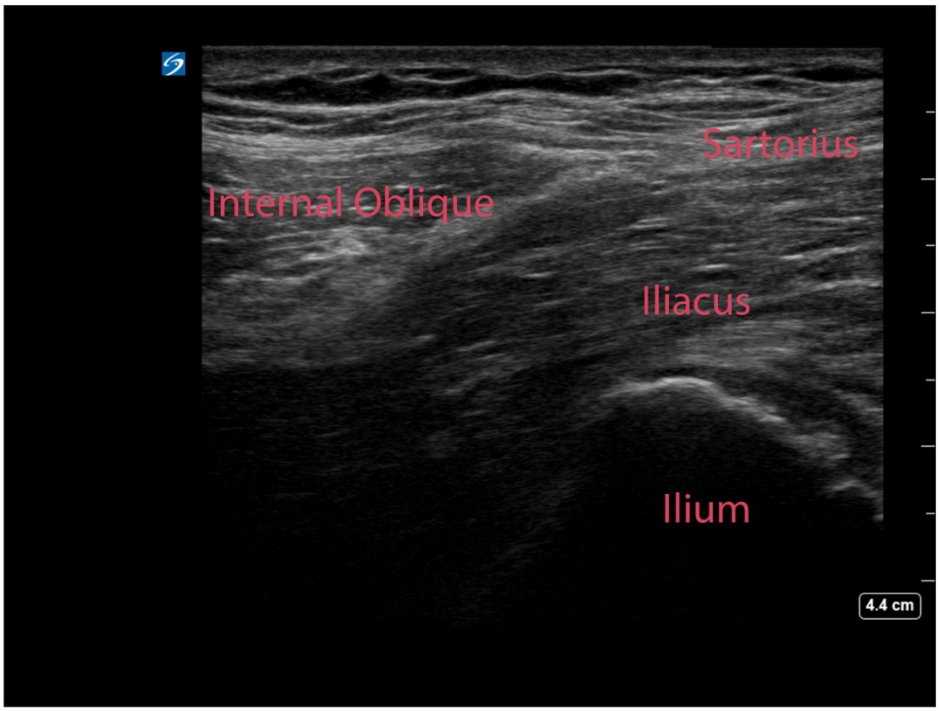
Introduction: Acute pain after a hip fracture can be associated with impaired respiratory function, prolonged intubation and intensive care unit (ICU) admission, as well as the potential development of chronic pain. Aggressive acute pain management not only alleviates pain and increases patient satisfaction, but also accelerates patient recovery and mobilization. Regional pain management can be useful in minimizing pain after a hip fracture, and there is strong evidence that fascia iliaca blocks decrease opioid consumption, improve patient pain scores, and decrease length of stay. However, these nerve blocks are typically administered immediately prior to hip fracture surgery. In this medically challenging case, we present the multimodal opioid-sparing management of a patient with a hip fracture and severe aortic stenosis who underwent balloon aortic valvuloplasty prior to hip fracture surgery. Although hip fractures in patients with aortic stenosis are not uncommon, this is the first described case of a balloon aortic valvuloplasty conducted using a combined ilio-inguinal blocks and fascia iliaca block. This case report was approved by the St Vincent's Medical Center Institutional Review Board (Bridgeport, CT). Written consent for publication of non-identifying medical information and Health Insurance Portability and Accountability Act authorization was obtained from the patient in this study.

Methods: An 88-year old 53-kg woman with robust past medical history including critical aortic stenosis (valve area 0.69 cm2) and recent sustained left intertrochanteric hip fracture presented for a balloon aortic valvuloplasty. Her other comorbid conditions included a history of coronary artery disease with inferior wall myocardial infarction complicated by cardiogenic shock status post multiple cardiac stents, as well as severe bilateral carotid disease. Her physical examination was notable for a frail body habitus, as well as severe pain from her hip fracture. After thorough discussion, the patient agreed to both an ilioinguinal block as well as fascia iliaca block. After connecting the patient to appropriate monitors, an ilio-inguinal block (10 mL) and fascia iliaca block (20 mL) was planned using bupivacaine 0.25%. Ultrasound-guided blocks were performed using a linear array ultrasonography 8-13 Hz probe (HFL38x, M-Turbo; SonoSite, Bothwell, WA) and 50-mm 22-gauge Stimulplex needle (B-Braun, Melsungen, Germany). The level was assessed using pin-prick sensation. The surgeon refrained from making an incision until the patient appeared comfortable. After establishing patient comfort, the patient underwent general endotracheal anesthesia in order to facilitate intraoperative transesophageal echocardiogram. General anesthesia was induced with etomidate 20 mg and rocuronium 50mg. Anesthesia was maintained with sevoflurane, as well as intravenous remifentanil 0.05 mcg/kg/min and dexmedetomine 0.2 mcg/kg/hr. The patient was comfortable in the post-anesthesia care unit, and reported no pain. She was given scheduled acetaminophen surgical protocol, during her hospital course. She received no opioid medications on postoperative day 1 and 2. After receiving cardiac monitoring for the postoperative day 1 and 2, the patient was transferred to the orthopedic unit for open reduction and internal fixation of left hip.

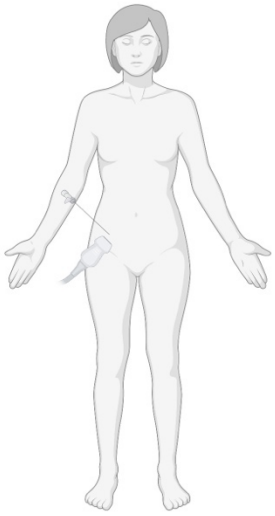
Conclusion: Hip fractures are one of the primary causes of admission in a trauma department. In 2016, its incidence was estimated at 120/100,000 inhabitants in the USA and in Europe with an increase of almost 30% between 2000 and 2009. Aortic stenosis in this population is not uncommon given its age distribution, and recent epidemiological studies have shown that the incidence of degenerative AS can reach 10% in people over 80 years of age. As one can see in our case report, individuals with both AS and a hip fractures can also have robust comorbid conditions that increase the risk of patient management both in the operating room and on the floor. We believe that combined regional anesthesia techniques may be underutilized in this patient population, and propose that increased usage can provide robust benefits for both patient morbidity and satisfaction.

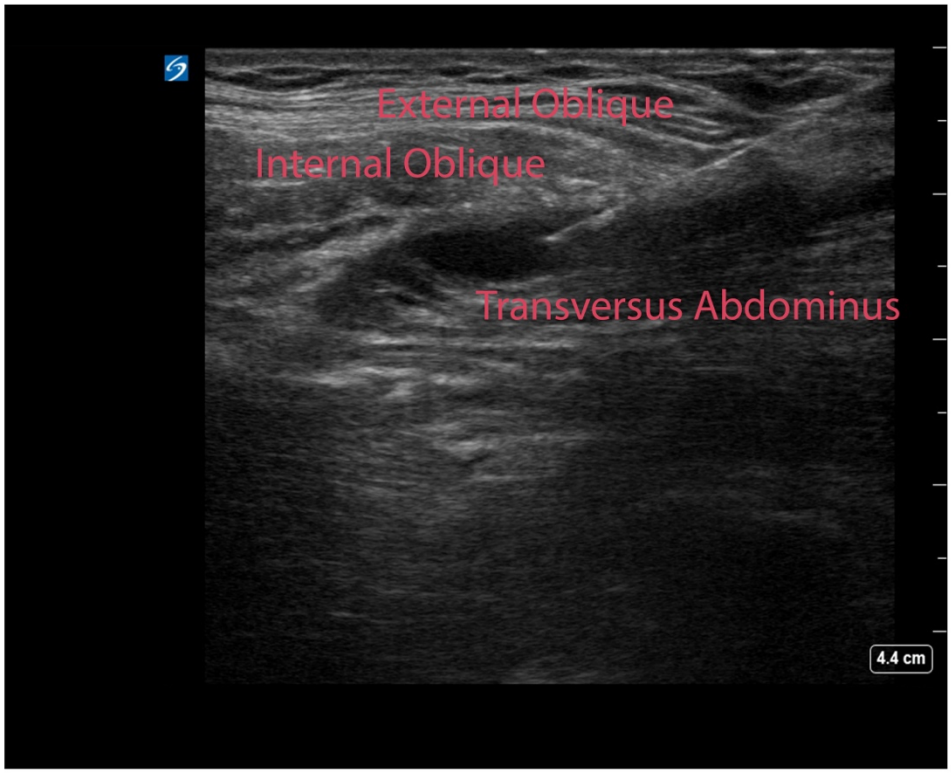
References: 1. Shah A, Hayes CJ, Martin BC. Characteristics of Initial Prescription Episodes and Likelihood of Long-Term Opioid Use - United States, 2006-2015. MMWR Morb Mortal Wkly Rep. 2017;66(10):265-269. 2. Angst MS, Clark JD. Opioid-induced hyperalgesia: a qualitative systematic review. The Journal of the American Society of Anesthesiologists. 2006;104(3):570-587. 3. Shafi S, Collinsworth AW, Copeland LA, et al. Association of opioid-related adverse drug events with clinical and cost outcomes among surgical patients in a large integrated health care delivery system. JAMA surgery. 2018;153(8):757-763.





Fascia Illiaca Block





Ilioinguinal Block

Regional Anesthesia - 2 Novel and Targeted Approach to End Stage Cancer Pain: Mechanical Neurolysis of the Brachial Plexus

William Wang[1], Sachin Jha[2], Jack M Berger[3], David Liang[1]

[1]LAC+USC Medical Center, Los Angeles, CA,
[2]University of Southern California, Los Angeles, CA,
[3]Keck School of Medicine of USC, Los Angeles, CA

Introduction: Neoplastic brachial plexopathy can occur due to primary and secondary tumors. Metastasis from breast and lung cancers are most common. It has been reported that 0.4% of patients with cancer have brachial plexus metastasis [1]. Metastatic brachial plexopathy is commonly accompanied with functional weakness and pain [2]. Over time, pain can become unremitting, severely diminishing the patients' quality of life, and therefore it is important to adequately manage [3]. We present a case of a patient with advanced breast cancer that had metastasized to the brachial plexus, who received a palliative neurolysis procedure.

Methods: This is a 51-year-old female with history of triple negative oligometastatic left breast cancer cT3N2M1 metastasis to lung, left iliac bone, and left brachial plexus, who presented as an inpatient Anesthesia consult for 10/10 left upper extremity pain. She was first diagnosed with breast cancer in 2016. She underwent left breast mastectomy and subsequent chemotherapy. Over the last year, she had recurrent emergency room visits for left upper extremity pain. She was followed by pain medicine, who had subsequently increased her narcotic pain regimen over time. On this admission, she presented with 10/10 sharp, burning pain that accompanied any movement of her left arm. Her pain had previously been controlled on a heavy regimen of opioids. While inpatient, her opioid regimen was uptitrated with no effect. Anesthesia was consulted after failed medical management of pain control. After reviewing her MRI Brachial Plexus, it was discovered that she had a large tumor burden surrounding the entirety of the right brachial plexus. Therefore, the decision was made to help treat her pain with a brachial plexus nerve block. A diagnostic interscalene block was performed with 20 mL of 0.5% bupivacaine injected in 5 mL aliquots with negative aspiration between injections, which improved her pain from 10/10 to 1/10. A standard 20 gauge peripheral nerve catheter was then placed next to the plexus and a continuous infusion of 0.2% ropivacaine at 6 mL/hr from an on-Q infusion pump was set. The following day, the patient's pain was very much improved. However, the nerve catheter was not a permanent solution because of the likelihood of catheter migration over time and possible infection risk. After discussion with the patient and her family, the patient was evaluated for potential neurolytic procedure. Due to limitations at our facility, alcohol or radiofrequency ablation were not options. Neurosurgery was also consulted but deemed the brachial plexus inoperable. At this point, the decision was made to physically ablate the nerves with a direct injection of the brachial plexus nerves with local anesthetic using ultrasound guidance. Her pre-neurolysis vital signs were BP 148/76, HR 114, RR 22, O2 saturation 98% on room air. With sterile precautions, a 25G Quincke needle was used under ultrasound guidance. The C5-C7 nerve roots were identified, and intentional intraneural injections of 3-5 mL 0.5% bupivacaine were performed after negative aspiration at each level. The patient experienced significant pain during this process even with midazolam and fentanyl for sedation, and analgesia for the procedure. However, she had significant continuous pain relief after the procedure, although she had no mobility of her left arm.

Conclusion: Percutaneous peripheral nerve block catheter or neurolysis can be an effective treatment for severe unremitting pain stemming from a neoplastic brachial plexus lesion. This case also demonstrates the utility of advanced regional anesthesia skills outside the operating room and the importance of access to interventional pain procedures in palliative care and end of life medicine. Through better collaboration with our palliative care colleagues, we can provide improved quality of life for these patients through significant pain relief without the need for large amounts of opioids.

References: 1. Neurology 1981; 31:45-50 2. Supportive Care in Cancer 2020 July; 28:1913-1918 3. American Society of Regional Anesthesia meeting,

Las Vegas, NV, May 8, 2011 4. The Korean Journal
of Pain 2016 July; 29(3): 179-84

	1 Day Prior to Catheter placement	Post Catheter Day 1	Post Neurolysis Day 1
Worst Pain Score (1-10)	10	5	2
Opioid requirements	- 75 mcg fentanyl patch - 4 mg dilaudid - 50 mg oxycodone	- 50 mcg fentanyl patch - 0 mg dilaudid - 20 mg oxycodone	- 50 mcg fentanyl patch - 0 mg dilaudid - 10 mg oxycodone
MME	335	150	135

Regional Anesthesia - 3 The Identification and Management of Acute Respiratory Failure Following Interscalene Catheter in a Patient with Limited Pulmonary Reserve

Alexa Lean[1], Jiang Wu[1]

[1]University of Washington, Seattle, WA

Introduction: Per American Pain Society recommendations, continuous interscalene nerve block (CISB) is the preferred method of perioperative analgesia for upper extremity procedures, improving postoperative analgesia and patient satisfaction. With the known side effect of ipsilateral phrenic nerve paralysis in up to 100% of patients, it has been proven that most healthy people can tolerate unilateral diaphragm paralysis without any clinical symptoms or significance (1-3); however, risk stratifying patients with preexisting lung disease remains elusive, without clear function-based or objective criteria.

We present the following case to discuss the clinical assessment and management of ambulatory CISB in patients with respiratory insufficiency. Objective clinical data, for example, incentive spirometry (IS), may help guide CISB management safely and reliably.

Methods: The patient was a 56-year-old female, morbidly obese, ASA 3, with a history of asthma, pulmonary sarcoidosis, obstructive sleep apnea, smoking, chronic pain on opiates, and left rotator cuff tear presenting for an outpatient rotator cuff arthroplasty. She underwent a left interscalene nerve block with peripheral nerve catheter (PNC) placement for postoperative pain management. The regional anesthesia team infused the PNC preoperatively with a single 10mL bolus of 0.5% ropivacaine, appropriately demonstrating complete sensory and motor blockade, as well as hemodynamic stability without signs or symptoms of respiratory distress. The PNC was infused with 0.2% ropivacaine at 6cc/h after her procedure. In the post-anesthesia care unit, the patient reported uncontrolled surgical site pain and PNC infusion was increased to 10cc/h. At this time she was able to pull 1600mL on IS with maximal effort. In preparation for discharge, the regional anesthesia team initiated an ambulatory PNC auto-infusing pump. Within 3 hours the patient experienced shortness of breath unresolved by albuterol, inability to take deep breaths, and increased work of breathing. We urgently stopped the PNC infusion. The auto-infusion pump rate was set to 14cc/h, rather than the intended rate, 10cc/hr. On physical assessment the patient was hemodynamically stable on 2L oxygen, appeared anxious, and showed ipsilateral lacrimation, mild facial droop, and miosis, consistent with Horner's Syndrome. She was able to pull only 1250mL on IS, with increased effort and discomfort. We ordered inspiratory and expiratory plain x-ray films to rule out other etiologies of perioperative respiratory distress. The films showed 'excellent right hemidiaphragm excursion without definite left hemidiaphragm excursion' (Figure 2). Within 10 hours her facial droop and lacrimation had resolved and sensation returned to the left side of her face. Objectively, her IS results improved to 1550mL. Repeat x-rays demonstrated adequate bilateral diaphragm contraction. The patient was hemodynamically stable on room air and discharged without regional anesthesia.

Conclusion: We present this case to discuss: the clinical assessment and decision making of CISB placement in patients with underlying respiratory insufficiency, the utility of baseline pulmonary function tests (PFT), and IS as an accessible and objective measurement of lung function pre- and post-PNC placement. Thorough risk-benefit analysis with the patient and perioperative team should occur when selecting patients appropriate for ambulatory CISB. The literature lacks objective data to provide clear contraindications to this intervention, but there are patients more at risk for respiratory compromise from phrenic nerve paralysis. PFTs may be helpful in some cases but must be considered with all other clinical risk factors like ASA status, weight, and smoking history (Figure 1). Although it has been demonstrated that ISB reduces FEV1 26%, there is no clinical correlation to respiratory compromise (4). Our patient was unable to tolerate a denser motor block, but received adequate analgesia with minimal opioid use on the lower infusion rate. Further randomized control trials to evaluate objective criteria to risk stratify and manage patients with pulmonary disease may prove useful in utilizing this preferred method of postoperative analgesia. IS is accessible bedside and a simple tool to help assess

patients' respiratory function while managing CISB infusions in patients with limited pulmonary reserve.

References: 1. Management of Postoperative Pain: A Clinical Practice Guideline From the American Pain Society. Pain, 2016; 17(2). 2. The indications and applications of interscalene brachial plexus block for surgery about the shoulder. Acute Pain, 2004; 6(2). 3. Perioperative interscalene blockade: an overview of its history and current clinical use. Journal of Clinical Anesthesia, 2002; 14(7). 4. Hemidiaphragmatic paresis during interscalene brachial plexus block: effects on pulmonary function and chest wall mechanics. Anesthesia and Analgesia, 1992; 74(3). 5. Clinical significance of severe isolated diaphragm weakness. The American Reserve of Respiratory Disease, 1988; 138. 6. The effect of initial local anesthetic dose with continuous interscalene analgesia on postoperative pain and diaphragmatic function in patients undergoing arthroscopic shoulder surgery: a double-blind, randomized controlled trial. BMC Anesthesiology, 2012; 12(6).

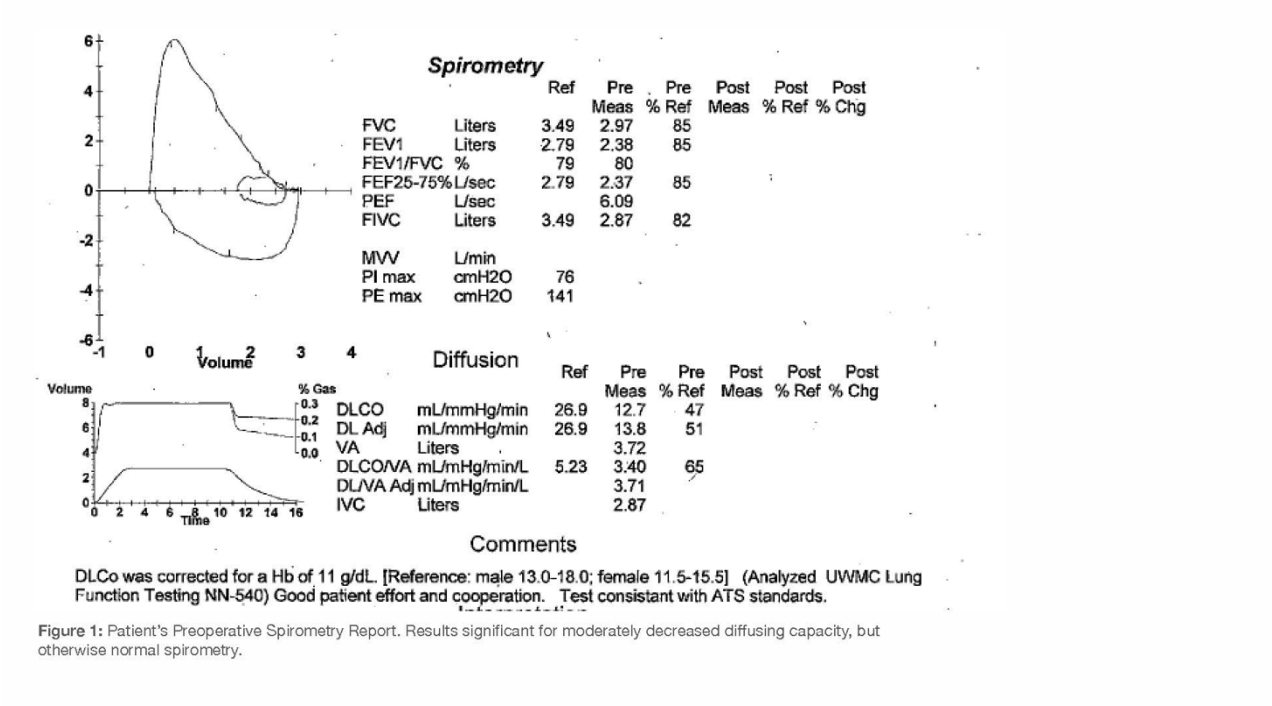


Figure 1: Patient's Preoperative Spirometry Report. Results significant for moderately decreased diffusing capacity, but otherwise normal spirometry.

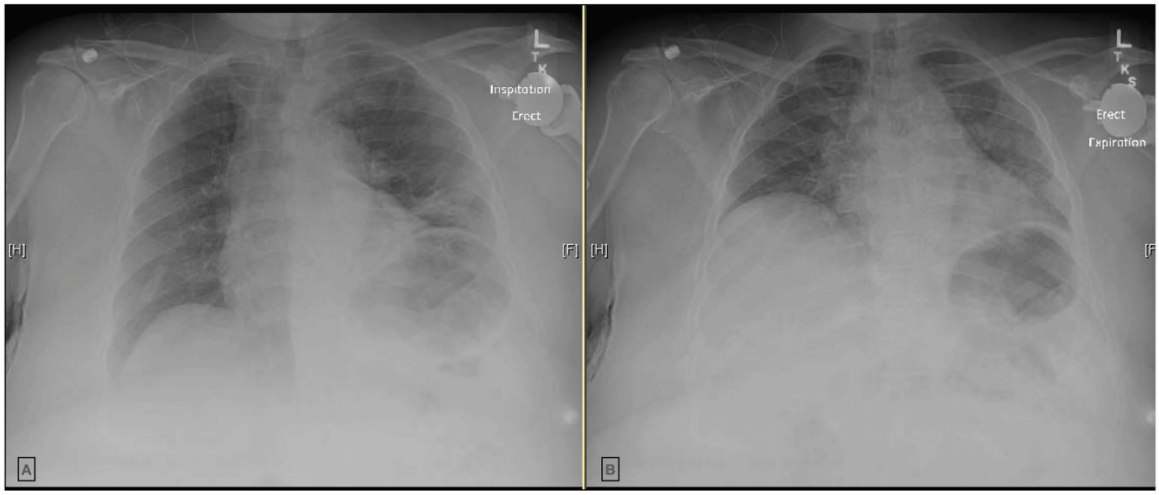


Figure 2: Patient's inspiratory (A) and Expiratory (B) plain x-ray films demonstrating clear right lung, no pneumothorax or pleural effusion. Inspiratory and expiratory exams show excellent right hemidiaphragm excursion. No definite left hemidiaphragm excursion. Per radiology report, "possible phrenic nerve injury or diaphragm paralysis can be further evaluated with fluoroscopic sniff test clinical discretion."

Respiration

Respiration - 1 A Bullet with High Aspirations

Shivani Patel[1], Raymond J Malapero[2], James Schiffenhaus[2]

[1]New Jersey Medical School, Newark, NJ, [2]Rutgers New Jersey Medical School, Newark, NJ

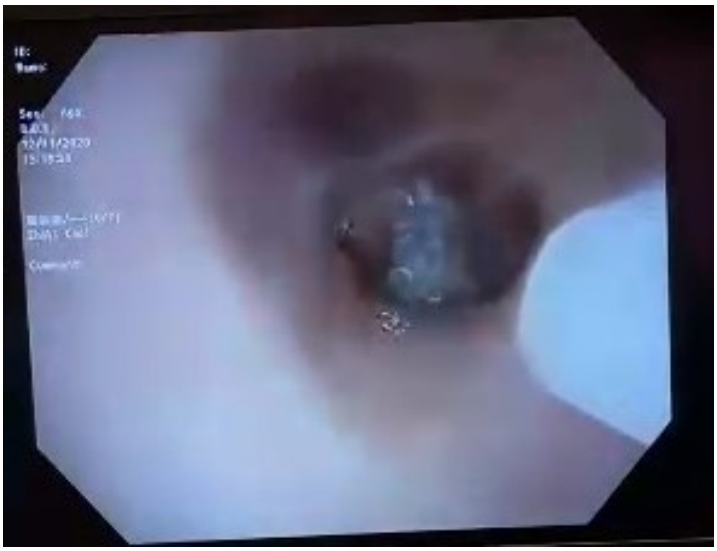
Introduction: Airway management in facial trauma poses an anesthetic challenge with often times a difficult airway due to bleeding, distorted anatomy, and unreliable mask ventilation. Following the acute trauma, patients post repair may still present with reduced range of motion and fibrotic anatomic changes. The addition of a foreign body in the lung can further complicate management. Although intrabronchial foreign objects are not unheard of and can be asymptomatic, the risk for chronic inflammatory changes, infection, and atelectasis can warrant removal. In this case, a bullet was aspirated into the lung following penetrating trauma to the face. Although the bullet was in a position to be removed without open thoracotomy, it was too large to pass through an endotracheal tube (ETT) posing an interesting intraoperative airway management scenario.

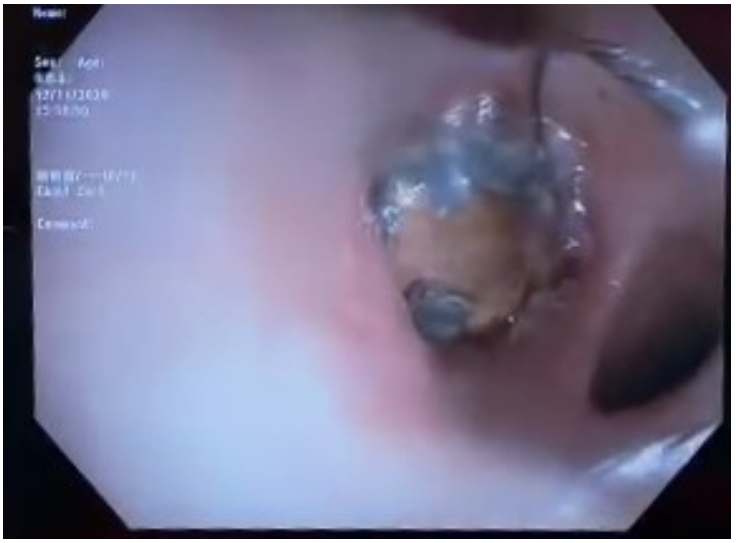
Methods: This is the case of a 36-year-old male with no significant past medical history who presented following an altercation which included a gunshot wound to the face. The bullet had passed through the jaw into the mouth and was found on imaging to have been aspirated into the right bronchus intermedius. At the time of presentation, the patient remained stable from a hemodynamic and respiratory perspective and was taken emergently by oromaxillofacial surgery for open reduction and fixation of the mandible. Patient was subsequently discharged from the hospital and returned several weeks later electively for removal of the aspirated bullet. Removal of the bullet was performed via flexible bronchoscopy with possible open thoracotomy planned, if necessary. A balloon was passed distal to the bullet and inflated. The bullet was then pulled back to the secondary carina. Subsequently the bullet was grasped with a snare type device. As the bullet was too large to be removed through the ETT, the patient needed to be extubated with the bronchoscope and ensnared bullet in one step. Anesthetic management involved maintenance of spontaneous ventilation throughout the procedure. Patient was induced with midazolam 1 mg, fentanyl 100 Œºg, lidocaine 60 mg, propofol 50 mg, followed by sevoflurane mask induction at 1.5 minimum alveolar concentration (MAC). Video laryngoscopy was performed secondary to the patient's limited mouth opening. Patient was maintained with the inhaled agent and kept spontaneously ventilating during flexible bronchoscopy and placement of endobronchial balloon. The ETT was removed under general anesthesia, during the bronchoscope and bullet removal, and patient was subsequently reintubated for final bronchoscopy and suctioning. Patient was extubated at the end of the case and recovered without complication or desaturation.

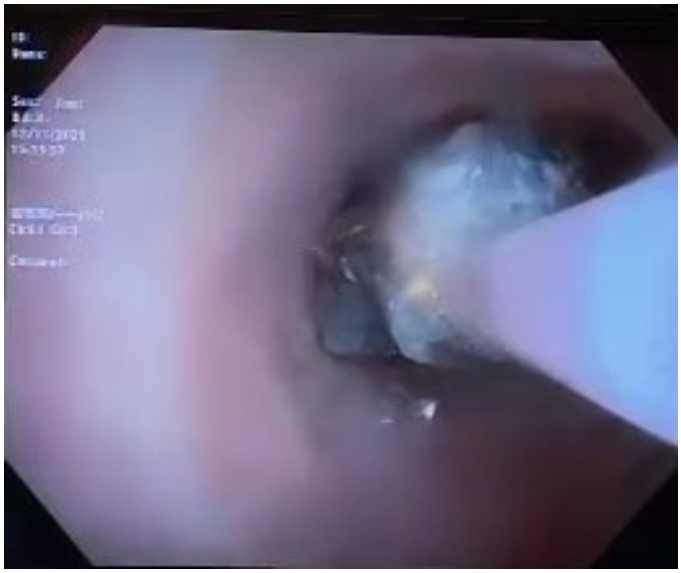
Conclusion: Foreign objects in the tracheobronchial tree are not uncommon and frequently asymptomatic. However, given the potential for long term complications, removal is indicated. In one previous case of an aspirated bullet, two years had gone by before the patient became symptomatic - removal was then performed via thoracotomy and wedge resection due to localized bronchiectasis caused by association of the bullet [1]. In most other cases, bronchoscopy remained the primary method of removal [2]. In this case, as the patient was stable from a respiratory perspective, he did not require urgent removal and management of the broken mandible took precedence. Given the object size and location, the need for flexible bronchoscopy, intraoperative extubation, and reintubation were anticipated and prepared for during extensive planning prior to the procedure. Accordingly, recovery from the initial surgery was particularly important to maintain a controlled airway and minimize risk of complications. Extubation during surgery is more commonly performed during functional neurosurgery requiring awakening of the patient for localization of seizure foci. Avoidance of prolonged paralysis and maintenance of spontaneous ventilation are frequently employed to prevent respiratory decompensation in these cases and was used in this case as well. General anesthesia was maintained with inspired oxygen and inhaled anesthetic as the time to reintubation was relatively quick, though intravenous propofol boluses were kept readily available. With proper communication between the anesthesiology and surgical teams and appropriate preparedness, removal of the bullet was performed without

complications and the patient was discharged after observation postoperatively.

References: 1. Anesthetic management in removal of neglected tracheobronchial foreign body. J Anaesthesiol Clin Pharmacol. 2013;29(1):127-128. 2. Aspiration of a bullet after a gunshot wound to the face. Journal of Pediatric Surgery Case Reports.2020;61.101616.







Respiration - 2 Combination of Nasal Mask-Face Tent and Suctioning Maintained Spontaneous Ventilation/Oxygenation and Reduced Aerosol/Droplet Spread in a Morbidly Obese COPD Patient during Difficult Colonoscopy/EGD

James Tse[1], Christine H Fratzola[1]

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

Introduction: Patients routinely receive IV sedation and nasal cannula (NC) O2 during GI endoscopy. Over-sedation and/or airway obstruction may cause severe desaturation, especially in obese patients. A simple plastic sheet has been used to convert ineffective NC to a face tent which delivers >0.6 FiO2.(1, 2) A pediatric facemask was used to deliver nasal CPAP to maintain spontaneous ventilation and improve oxygenation in sedated obese patients.(1,3) A novel nasal mask-face tent was used to provide continuous oxygenation and reduce aerosol/droplet spread during RSI, intubation and extubation in a COVID-19 positive patient.(1,4) We used it to provide nasal CPAP and reduce aerosol/droplet spread during difficult GI endoscopy.

Methods: Case Report: A 65-year-old morbidly obese female, BMI 38.4 kg/m2, with HTN, NIDDM, CKD, severe CAD s/p recent MI, CABGx2v (5 weeks prior) and PCI (one day prior), severe COPD O2 dependent, anemia and melena presented for colonoscopy and EGD with capsule endoscopy camera insertion. The patient had a Mallampati Class IV airway and 93% SpO2 on NC O2 6 L/min with a surgical facemask. She had shallow breathing and complained of dyspnea that was improved after receiving the missing morning inhalation treatment. Following removing the surgical facemask, her SpO2 quickly dropped to 86% while on NC O2 4L/min. Her SpO2 improved to 95% with a face tent quickly secured over her nose. An infant facemask-face tent was then secured over her nose with elastic head-straps and connected to the anesthesia circuit/machine delivering 5 cm H2O CPAP with 4 LO2/min (Fig.1-2). After her SpO2 improved to 98%, moderate-deep sedation was titrated with lidocaine/propofol and propofol infusion (50-75 mcg/kg/min). A suctioning catheter was placed under the face tent to reduce aerosol/droplet spread. She maintained spontaneous nasal ventilation and 98-99% SpO2 throughout the difficult procedures abdominal compressions and position changes (1 hr 22 min) (Fig. 3-4). She tolerated the procedure well with stable hemodynamics. She was transported to PACU with a face tent and NC O2 6L/min and maintained 96% SpO2 (Fig.5). A facial BiPAP (IPAP 12, EPAP 5, 0.6 FiO2) provided further respiratory support until she fully recovered from sedation.

Conclusion: This simple nasal mask-face tent provided nasal CPAP and maintained spontaneous ventilation and oxygenation in a morbidly obese patient with severe cardiopulmonary diseases during difficult GI endoscopic procedures. Combining with suctioning under the face tent, it reduced aerosol/droplet spread. This simple technique should be used as universal precaution amid the COVID-19 pandemic. It may improve patient safety and provide additional protection to providers at a low cost.

References: 1.www.TSEmask.com; 2. Anesthesiology 102:484, 2005; 3. SAMBA 28th AM, 2013; 4. ASA AM: (MC1280), 2020


Dash 5000
Cardiac Bypass


Dash 5000
Cardiac Bypass


Dash 5000
Cardiac Bypass


Dash 5000
Cardiac Bypass

Sleep Medicine

Sleep Medicine - 1 Perioperative Considerations For A Patient With Kleine Levine Syndrome Undergoing A Neurosurgical Procedure Under General Anesthesia.

Wesley Rajaleelan[1], Mandeep Singh[2], Tumul Chowdhury[3]

[1]Toronto Western Hospital, Toronto, Ontario, [2]University of Toronto, Toronto, Ontario, [3]Toronto Western Hospital, Toronto, Canada

Introduction: Kleine Levin syndrome (KLS) is a rare central disorder of daytime hypersomnolence, often characterized by a relapsing-remitting course, characterized by recurrent episodes of excessive sleep lasting between 12-20 hours, associated with varied symptoms such as hyperphagia, hallucinations, derealization, disorientation and hypersexuality KL syndrome may present multiple perioperative challenges including varied requirement to anesthetics, weaning difficulties, postoperative sleep disorders and delirium; however due to rare occurrence, its anesthetic considerations remain poorly described. This case report outlines the anesthetic considerations and management plan for a young female patient with known KL syndrome undergoing trans nasal excision of a trigeminal schwannoma under general anesthesia

Methods: We present the case of a 27-year-old female patient (weight 54 Kg, height 166 cm, body mass index 19.,6 Kg/m2) with a known history of KL syndrome undergoing endoscopic skull base surgery under general anesthesia

Results: General anesthesia (GA) was then induced with Fentanyl 2mg/kg, propofol (2.5 mg/kg), and remifentanil (1 ¬µg/kg) and paralyzed with Rocuronuium 0.9mg/kg. General anesthesia was maintained with air-oxygen mixture (FiO2 0.5), 0.8 MAC of sevoflurane and remifentanil (0.25 ¬µg/kg/min) infusion. The depth of anesthesia was monitored using Entropy (GE Datex-Ohmeda Entropy, USA) targeted to values between 40-60. The intraoperative period was unremarkable. Intra-arterial blood pressure (BP) monitoring was done to maintain a mean arterial BP within 20% baseline using short acting direct vasopressors (phenylephrine). The total duration of the procedure was 7 hours with estimated blood loss and urine output of 600mls. No neuromuscular reversal agent was given as it was longer than 7 hours since first dose, and complete recovery was noted on neuromuscular monitoring indicated (train-of-four: 4 twitches, with no fade. Extubation was performed while continuing remifentanil infusion at 0.03/mcg/kg/min, and when fully awake, obeying verbal commands and no evidence of residual neuromuscular blockade. Kleine Levine (KL) syndrome is a rare, relapsing-remitting disease of unknown origin.1,2 It is characterized by recurrent episodes of hypersomnia (more than 18 hours of sleep/day), lasting one to several weeks, associated with cognitive impairment, derealization, apathy, and behavioral changes. Less frequently, episodes include disinhibition with hyperphagia and hypersexuality, or depression, hallucinations, and delusions 1,2 The condition is noted to occur in adolescents, with male predominance, with a history of birth of developmental problems.3 Between episodes, patients have normal sleep, mood, cognition, and behavior for one to several months. Episodes may be triggered by infection, alcohol intake, or sleep deprivation. Episodes usually become less frequent and less severe by middle age.1,2,3 During an episode, the patient may exhibit abnormal behavior such as excessive sleep cycle, hallucinations, hyperphagia, derealization (a striking feeling of unreality), irritability and hypersexuality. These symptoms tend to decrease as age progresses For our patient, the overnight PSG was normal for any sleep-related breathing disorder, and the MSLT indicated severely reduced sleep onset latency, indicating severe daytime hypersomnolence. PSG studies are often difficult to interpret and results are dependent on the duration of recording as well as the timing.

Conclusion: This is the first case report to focus on the anesthetic considerations for patients with KL syndrome undergoing a neurosurgical procedure. We found a lack of literature in this syndrome undergoing procedures under GA. Effective preoperative work up, continuation of preoperative medications, depth of anesthesia monitoring, use of multimodal analgesia

using short-acting agents, and regional anesthetic techniques are considered to be a safer option.

References: 1. Arnulf I. Kleine Levin Syndrome. In: Kryger M, Roth T, Dement W, eds. Principles and Practice of Sleep Medicine,. 6th ed. Elsevier Inc.; 2016:96 976.e4 2. Rosenow F, Kotagal P, Cohen BH, et. al.: Multiple sleep latency test and polysomnography in diagnosing Kleine-Levin syndrome and periodic hypersomnia. J Clin Neurophysiol 2000; 17: pp. 519-522 3. Aflaki S, Hu S, Kamel RA, Chung F, Singh M. Case Report of a Patient With Idiopathic Hypersomnia and a Family History of Malignant Hyperthermia Undergoing General Anesthesia: An Overview of the Anesthetic Considerations. A A Case Rep. 2017 May 1;8(9):238–41. 4. Itokawa K, Fukui M, Ninomiya M, et. al.: Gabapentin for Kleine-Levin syndrome. Intern Med 2010; 48: pp. 1183-1185. 5. Kellett J: Lithium prophylaxis of periodic hypersomnia. Br J Psychiatry 1977; 130: pp. 312-316

Figure 1: Preoperative Magnetic Resonance Imaging delineating the intracranial lesion.

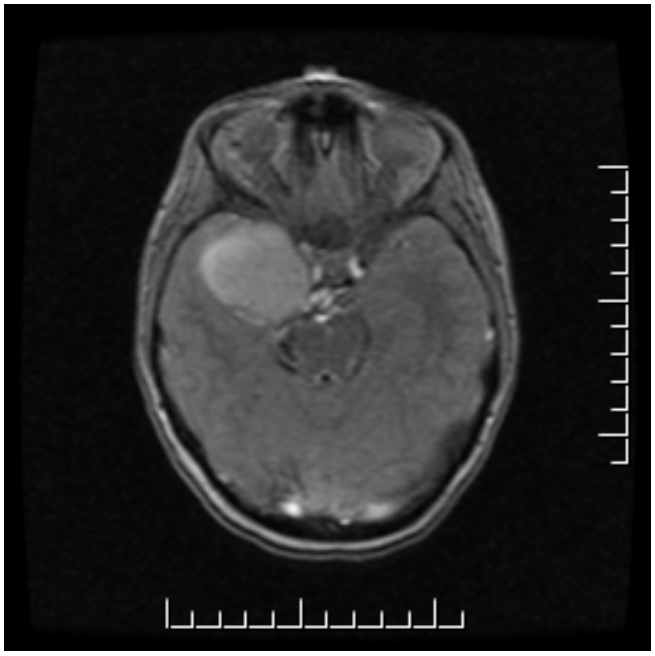


Figure 1: MRI brain demonstrates large extra-axial cystic and peripherally enhancing mass in the right middle cranial fossa, unchanged in morphology and size with stable degree of mass effect on the surrounding structures and bony remodeling of the right maxillary sinus and sphenoid sinus. There was no hydrocephalus, midline shift, restricted diffusion or any other intracranial lesions. Peripherally enhancing cystic structure along the trajectory of the right trigeminal nerve most likely representing a cystic schwannoma with stable mass effect on the surrounding structures and chronic osseous remodeling of the maxillary and sphenoid sinuses. There was no involvement of the hypothalamus, or the third or fourth ventricle.

Trauma

Trauma - 1 Successful Operative Removal Of A Chest Tube Placed Into The Subclavian Vein Using Sedation

Artem Emple[1], Preshita Date[1], Eileen Wang[2], Hasan Aldailami[2], Jay Berger[3]

[1]Montefiore Medical Center, Bronx, NY, [2]Montefiore Medical Center, Bronx, United States of America, [3]Montefiore - Albert Einstein School of Medicine, Bronx, NY

Introduction: Tube thoracostomy is frequently employed in order to evacuate the pleural space of air, fluid, or infection, among other pathologies. Insertion can either be performed via lateral or anterior approach. The procedure is relatively benign but carries several risks including pain, hemopneumothorax, infection, obstruction of the tube, and damage to the heart and great vessels. The overall complication rate varies widely in the literature, but cardiovascular complications are rare[1], commonly quoted as only 0-3%[2]. We report a case of inadvertent chest tube insertion into the subclavian vein, through the right atrium, and into the inferior vena cava (IVC), and resultant successful removal under monitored anesthesia care (MAC).

Methods: An 88-year-old female with a history of atrial fibrillation not on anticoagulation, SSS s/p pacemaker, CKD III, COPD, and PAD presented to the emergency room with undifferentiated sepsis. A large right sided pleural effusion was drained with a pigtail chest tube, but complicated by a small contralateral pneumothorax. Worsening hypoxemia after removal of the chest tube revealed an enlarged pneumothorax, which prompted placement of a chest tube at the bedside. The pleural space was accessed in the usual manner and a 14F catheter was inserted. However, after chest tube placement, brisk dark red blood was noted within the lumen of the catheter. The chest tube was immediately occluded with a Kelly clamp to prevent further bleeding. CTA showed the chest tube entering the right subclavian vein and terminating within the right atrium (Fig 1, 2). The patient remained hemodynamically stable with the chest tube clamped and was monitored in the ICU overnight. The next day, the patient was brought to the OR for removal of the chest tube under angiographic guidance by vascular surgery, with cardiothoracic surgery and perfusion on standby. The anesthetic plan was to proceed with moderate sedation and determine the precise course of the chest tube. Angiography and intravascular ultrasound were used to confirm that the chest tube was entirely intraluminal (Fig 3). The chest tube was removed while deploying a covered stent in order to minimize bleeding. Angiography confirmed successful removal of the tube with no active bleeding and appropriate positioning of the pacemaker leads. The patient was hemodynamically stable throughout the procedure, requiring intermittent vasopressor support. She was transferred to the ICU for further monitoring and was later discharged home in stable condition.

Conclusion: Tube thoracostomy is routinely used as a treatment for pleural pathology. However, this procedure carries risk, most commonly including incorrect positioning or infection[3]. Rare complications include direct injury to the lung parenchyma or intercostal vessels, and injury to the long thoracic or phrenic nerves[3]. Serious and potentially fatal complications include esophageal injury, trauma to the heart or great vessels, and intraperitoneal chest tubes[3]. Several cases of misplaced chest tubes have been described in the literature, including a chest tube placed into the left atrium via the pulmonary vein[4], into the right ventricle via the hepatic vein[5], into the posterior pleural space by coursing in between the lung lobes[6], and even a chest tube fatally injuring the subclavian vein[7].

In this case, our patient had a 14F chest tube placed into her subclavian vein, through the right atrium, terminating in the inferior vena cava. We thereby demonstrate the safety of angiographic removal of an intravascular chest tube using sedation. This required close communication with the surgical team to ensure that the chest tube was entirely intravascular. An additional consideration for this patient included dislocation of the pacemaker. Though it was placed several years prior, we were concerned the leads may dislodge during removal of the chest tube. Confirmation that the chest tube was intraluminal without perforation of the great vessels or lodged within the myocardium allowed for an endovascular approach to removal and repair. Diligent preparation and strategic planning for various adverse outcomes contributed to success in this case. Fortunately, we were able to approach a potentially morbid surgery in

a minimally invasive manner, while ensuring patient safety and surgeon ease.

References: Thoracostomy tubes: A comprehensive review of complications and related topics. Int J Crit Illn Inj Sci. 2014;4(2):143-155. Chest tube complications: how well are we training our residents? Can J Surg. 2007;50(6):450-458. Chest tubes. Indications, technique, management and complications. Chest. 1987;91(2):258-264. Misadventures of a Pigtail: Case Report of Accidental Insertion of a Chest Tube Into the Left Atrium During Interventional Radiology-Guided Placement. A & A Practice. 2018;11(10):273-275 A chest tube misplaced in the heart? Eur J Cardiothorac Surg. 2018;53(5):1091-1092. An unusual complication of pigtail catheter insertion. J Thorac Dis. 2018;10(10):5964-5967

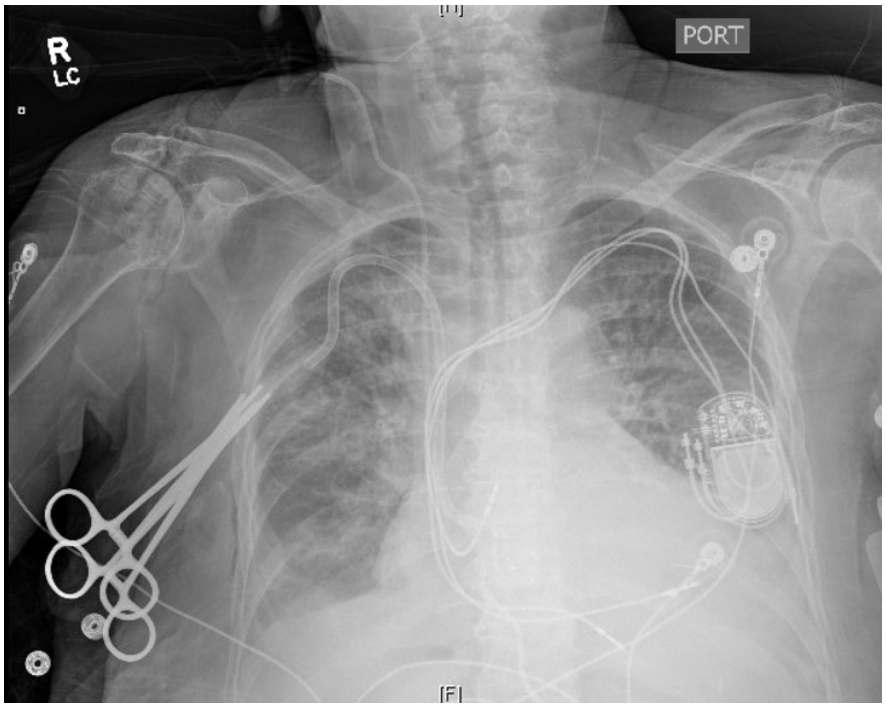


Figure 1. Pre-op portable chest x-ray

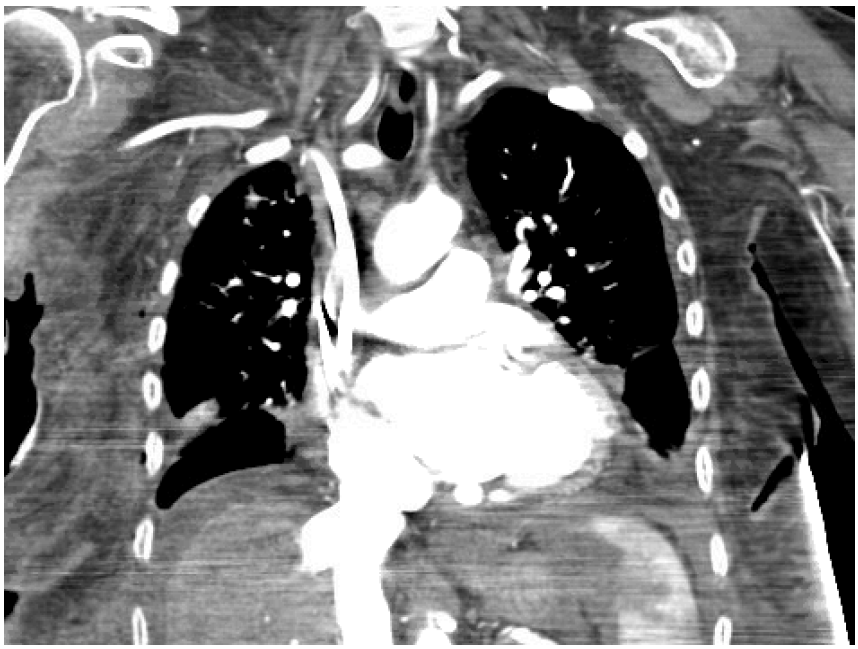


Figure 2. Coronal view of CT Chest/Abdomen/Pelvis with contrast after initial chest tube placement.

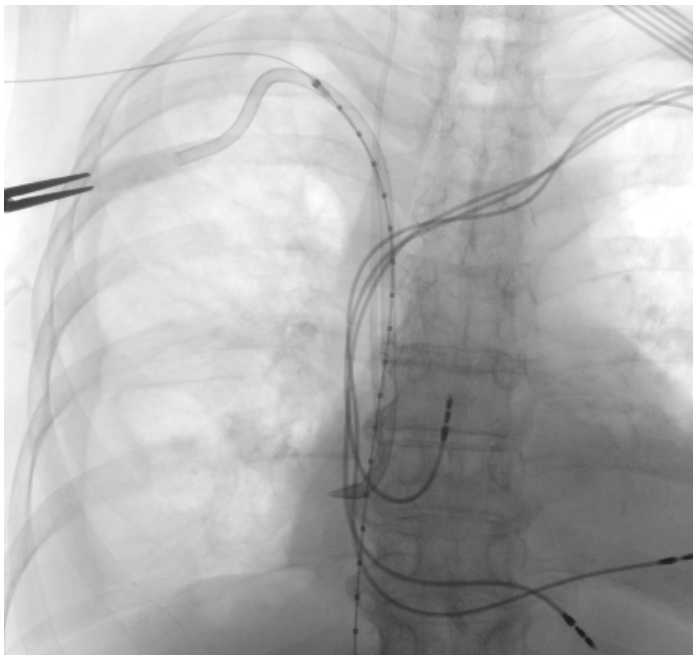


Figure 3. Intraoperative image of wire passing through chest tube from basilic vein to femoral vein

Trauma - 2 A Case Of Acute Refractory Hypoxemia In A Patient With Severe Acute Respiratory Distress Syndrome Requiring Intraoperative Extracorporeal Membrane Oxygenation

Cassian M Horoszczak[1], Tichaendepi Mundangepfupfu[1]

[1]University of Rochester, Rochester, NY

Introduction: Acute respiratory distress syndrome (ARDS) has been defined by the Berlin Criteria as hypoxic respiratory failure associated with bilateral pulmonary opacification occurring within a week of initial insult [1]. There are various forms of ARDS with differences in their management. The origin of edema in ARDS should not fully be explained by cardiac failure or fluid overload [2], but fluid as seen in transfusion-associated circulatory overload (TACO) can complicate existing ARDS.

Methods: A 44 year old male without significant PMH presented to the ED with multiple stab wounds to the left lower thorax, left flank and left lower abdomen. His initial GCS was 15. He was noted to be in hemorrhagic shock and massive transfusion protocol was initiated. He was intubated for hypoxic respiratory failure and a left chest tube was emergently placed. The patient was taken to the OR for exploratory laparotomy. Intraoperatively, a resuscitative thoracotomy with aortic cross clamping was performed. His small bowel was left in discontinuity and the left iliac artery was repaired. He received 14 units of packed red blood cells, 14 units of fresh frozen plasma, 2 units of platelets, and 3 units of cryoprecipitate. He was brought to the trauma ICU on a norepinephrine infusion. That same day, the patient went back to the OR for re-exploration. Small bowel was resected and and large bowel was repaired. At the end of the operation, he developed profound hypoxemia. PEEP was titrated without improvement. The patient had adequate neuromuscular blockade and inhaled epoprostenol was administered. Intraoperative ECMO was requested. A chest x-ray in the OR demonstrated prominent bilateral interstitial markings. A transesophageal echo revealed good cardiac function. He underwent VV ECMO cannulation with a right internal jugular vein inflow catheter and right femoral vein outflow catheter with an improvement in oxygenation. He was transferred to the cardiothoracic surgery ICU postoperatively for ECMO management. The patient returned to the OR a third time on POD 0 and underwent re-exploration for hypotension and decreased ECMO flows. The retroperitoneum was explored and active arterial bleeding was controlled. In total, he received >100 units of blood products during the course of his resuscitation on POD 0. The patient's abdomen was closed on POD 2 then re-explored and closed on POD 4. VV ECMO was explanted on HOD 5. Mechanical ventilation was weaned and he was extubated on HOD 12. He was discharged home with nursing services on HOD 23.

Conclusion: Severe ARDS was the most likely diagnosis when the patient developed profound hypoxemia at the end of the second OR case. To manage this, PEEP was titrated but oxygenation did not improve. Tidal volumes were titrated to maintain plateau pressure under 30cmH20. Neuromuscular blockade was continued. Prone position for oxygenation was deemed not feasible due to an open abdomen. In the setting of refractory hypoxemia with SpO2 values in the range of 73-89% and PaO2 values in the range of 34-43 mmHg, inhaled epoprostenol was started and an early consult for ECMO was placed intraoperatively. Ultimately, good communication between the surgical and anesthesia team and prompt intraoperative ECMO contributed to survival with a good clinical outcome. It is important to characterize the mechanism of lung injury because the specific etiology of ARDS often specifies the management that is indicated. ARDS can be complicated by other mechanisms of hypoxemic respiratory failure which may further complicate management [1,2]. In our case, the initial etiology of ARDS was related to penetrating chest trauma and hypoxic respiratory failure was further complicated by TACO in the setting of massive transfusion. A formal diagnosis of transfusion-related acute lung injury (TRALI) was not possible because of the temporal relationship of lung trauma with ARDS. Blood products were administered in a 1:1:1 ration of plasma to platelets to red blood cells. In the PROPPR Trial [3] a 1:1:1 ratio compared to a 1:1:2 ratio did not result in significant differences in mortality at 24 hours or 30 days. More patients achieved hemostasis and fewer patients experienced death from exsanguination by 24 hours in the 1:1:1 group. Despite concerns that

the 1:1:1 group would experience higher rates of ARDS, no differences were detected between the 2 treatment groups.

References: 1. Intensive Care Med. 2016 May;42(5):674-685. 2. JAMA. 2012 Jun 20;307(23):2526-33. 3. JAMA. 2015 Feb 3;313(5):471-82.