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Medically Challenging Cases

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Airway Management

Airway Management 1 - Anaesthesia management of a patient with unilateral absence of left pulmonary artery undergoing right lung cancer surgery: A case report

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Introduction: Unilateral absence of the pulmonary artery (UAPA) is a rare congenital malformation of the pulmonary vessels, with an incidence rate of 1/200000 – 1/300000[1, 2]. The occurrence of UAPA in patients with lung cancer is extremely rare. To date, 13 cases have been reported globally, and only four cases have been reported in patients with contralateral lung cancer. To our knowledge, this case is the fifth. This article provides insights into the important preoperative anaesthetic evaluation, airway management, and anaesthetic program formulation with a main focus on anaesthetic management of patients with UAPA with contralateral lung cancer.

This present case was a 72-year-old female who found a solid nodule in the posterior basal segment of the right lower lobe during incidental computed tomography (CT) screening. The chest CT revealed a lesion of approximately 10 mm x 9 mm, and the adjacent pleura was indented, suggesting lung cancer (Fig. 1A). In addition, the left lung volume was significantly reduced, and the lung exhibited honeycomb-like changes. The mediastinum was shifted to the left (Fig. 1B). The left pulmonary artery was noted to be absent through the CT mediastinal window in the chest (Fig. 1C) and in three-dimensional reconstruction of the coronary computed tomography angiography (CTA) (Fig. 1D).

In cases of malignant pulmonary nodules, right lower lobectomy is usually the chosen procedure; however, the calculated PPO-FEV1 and PPO-DLCO values were 39.9% and 40.3%, respectively. The two indices of our patient were at critical value, indicating that the risk of postoperative complications was significantly increased. In addition, one noteworthy report from 2016 described a case of a 60-year-old female with right UAPA and left lung cancer who underwent a left lower pulmonary lobectomy [3]. The day after surgery, the patient developed severe acute pulmonary hypertension and died of right heart failure. Thus, the selection of surgical methods requires careful consideration. These reports suggest that similar patients have a greater risk of adverse effects or death resulting from lobectomy and together with the physician should choose to undergo wedge resection to retain more lung tissue.

Methods: The patient gave written informed consent to publish this case.

Conclusions: Based on our thorough review of available cases, we conducted a complete preoperative evaluation and determined that the appropriate surgical method was wedge resection with thoracotomy as a secondary measure. Simultaneously, the anaesthesia protocol was determined, and the most important pulmonary isolation tool selected was the bronchial blocker. The plan for anaesthesia was to perform the right lower lobe occlusion first; the right lower lobe represents a relatively large proportion of the right lung. If intraoperative oxygenation cannot be maintained, more accurate pulmonary segment occlusion should be performed. If oxygenation cannot be maintained under the condition of pulmonary segment occlusion, the occlusion should be cancelled, and bilateral smaller tidal volume ventilation or conventional tidal volume ventilation should be used. If oxygenation still cannot be maintained, an emergency extracorporeal membrane oxygenation (ECMO) plan should be initiated as the last line of defense for the safety of the patient.

On the day of operation, a single-lumen endotracheal tube was inserted after a sufficient depth of anaesthesia was administered, and a bronchial blocker was placed under the guidance of a fiberoptic bronchoscope to block the right lower lobe. Mechanical ventilation (VT 4~6 mL/kg, f: 13~17 beats per minute, I: E= 1:2, positive end expiratory pressure (PEEP): 0 cmH2O, FiO2: 0.6~0.8) was performed on the other lobes. During surgery, a protective pulmonary ventilation strategy was adopted, and lung recruitment was performed intermittently. Propofol and remifentanil were pumped continuously to maintain anaesthesia. Regarding fluid management, restrictive infusion strategies were implemented, and Ringer's solution was infused at 1~2 mL/kg/h during surgery. Small doses of vasoactive drugs were administered when necessary to maintain stable circulation. The visual field for the thoracoscopic surgery was clear, and the surgery proceeded smoothly. The intercostal nerve block was performed through the thoracic cavity before the end of surgery. Intraoperative arterial blood gas analysis indicated that the pH, partial pressure of oxygen, and partial pressure of carbon dioxide were within normal ranges. Total operative time with corresponding anaesthesia was 2 hours. Five minutes after surgery, the tracheal catheter was smoothly removed.

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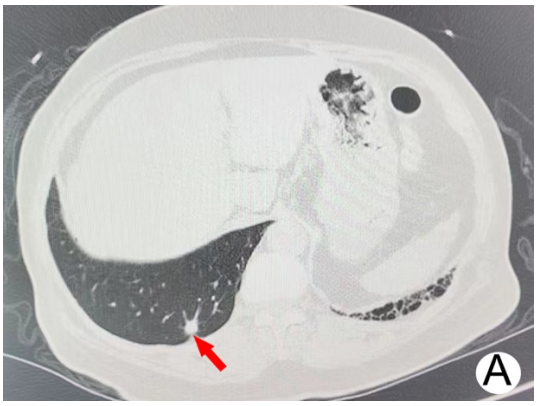
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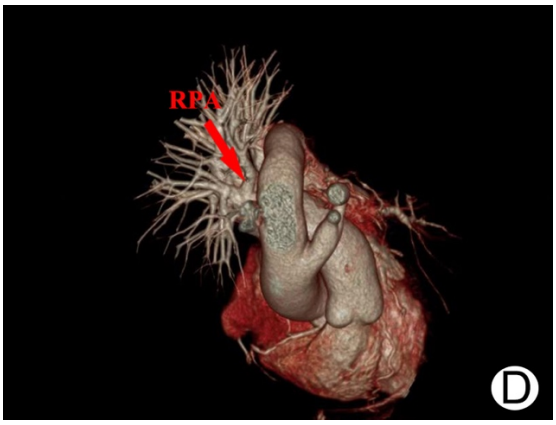
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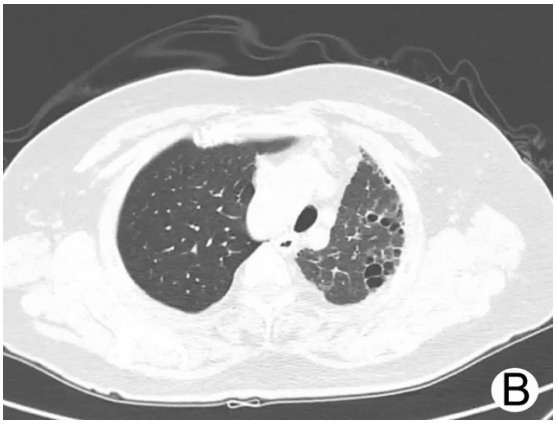
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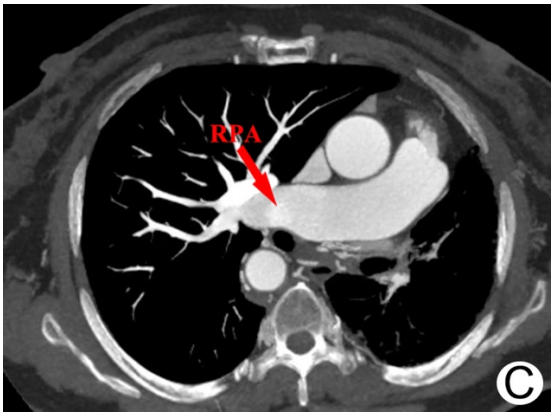
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Airway Management 2 - Difficult Airway Due to Inaccessible Face in A Child- Challenge to Anaesthesiologist

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Introduction: Mask ventilation is an essential component of airway management. Successful mask ventilation provides anaesthesiologist with a rescue technique during unsuccessful attempts at laryngoscopy and unanticipated difficult airway situations.[1] However, sometimes there may be situation when we do not have even access to face for mask ventilation. In such situations conscious sedation with preservation of spontaneous ventilation is of utmost importance and serves the purpose before we can achieve access to face.[2] We hereby present anaesthetic management of a 3-year-old child with no access to face.

Methods: A 3-year-old child with her face stuck into a steel box got admitted to emergency department Fig 1 (a). On examination child was crying. Heart rate was 150/min and heart sounds were normal. Chest was clear and oxygen saturation was normal. CT scan revealed frontal bone fracture along with dural rupture. Airway access was the biggest challenge as there was no access to face. Oxygen was administered via tubing through marginal gap between cheek & box Fig 1 (b). Difficult airway cart was kept ready. Surgeons were kept ready in case if tracheostomy was needed. Conscious sedation was given meticulously using ketamine 5 mg and then in increments of 2.5 mg with close watch on respiration, oxygen saturation and heart rate. After cutting the lower half of the box, face mask was placed very gently Fig 2 (a). Glycopyrrolate was given. Ketamine 10 mg was given and after assessing adequacy of ventilation, succinylcholine 15 mg & ketamine 10 mg were given, and intubation was done successfully Fig 2 (b). Fentanyl 30 µg and vecuronium were administered. The upper half of the box was removed and dural repair was done by neurosurgeon. At the end of surgery, trachea was extubated. Postoperatively the child was conscious, crying and reflexes were normal and was shifted to neuro ICU.

A difficult airway as defined by the ASA Task Force on management of the difficult airway is “the clinical situation in which a conventionally trained anaesthesiologist experiences difficulty with face mask ventilation, tracheal intubation or both. We encountered difficult airway as there was no access to face due to steel box stuck into the face.[3]

Anaesthetic management of such child was a big challenge to us as there was no access to face for airway management. We managed the child with conscious sedation using ketamine 5 mg and then in increments of 2.5 mg. Conscious sedation is safe because the patient remains conscious and can maintain a clear airway and adequate breathing. Conscious sedation drug technique has a margin of safety wide enough to make loss of consciousness unlikely.[4]

Conclusions: Careful and constant watch was kept on respiration, oxygen saturation and heart rate. After cutting the lower half of the steel box, access to face was achieved and we were able to mask ventilate & intubate. Preservation of spontaneous ventilation is key to successful anaesthetic management of such patients without airway access.

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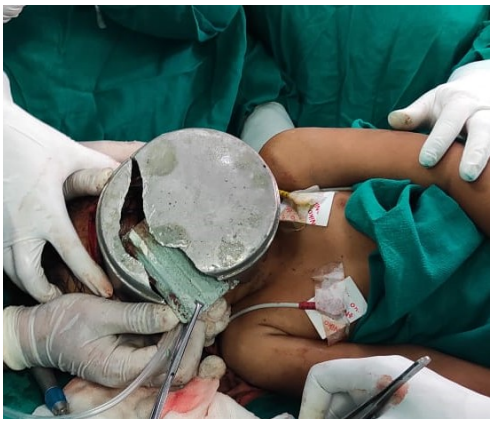

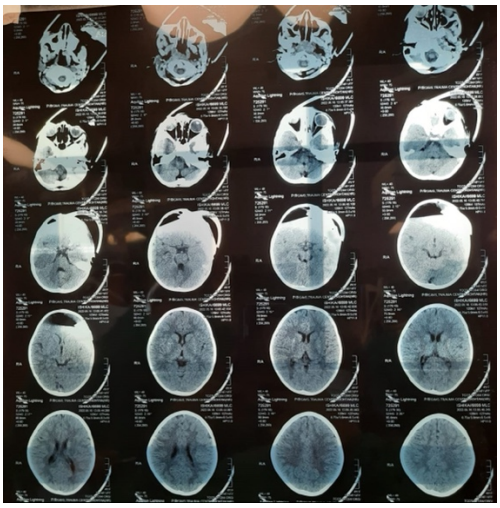

Airway Management 3 - Double Lumen Tubes: A Factor to Consider in Uvular Injury in the perioperative period

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Introduction: Uvular injury ranging from minimal abrasion and edema to severe ulcer and necrosis is an uncommon complication of airway manipulation. It can occur during intubation, maintenance, or even extubation. [1] Decreased blood perfusion to the uvula, due to pressure is the presumed mechanism for injury. [2] Double lumen tubes placed for one lung ventilation are typically more rigid and of wider diameter than single lumen tubes, making them more difficult to place and increasing the risk for potential traumatic injury. [3] We present a case of a patient with uvular ulceration after use of a double lumen tube for one lung ventilation. This specific cause for uvular injury is yet to be described in literature.

Methods: A 58-year-old male with left lower lobe lung adenocarcinoma presented for open thoracotomy and left lower lobectomy with mediastinal lymph node dissection. He had multiple previous general anesthetics with single lumen tubes that were uneventful. Preoperative assessment did not reveal any abnormalities with his airway on physician exam and he did not report any throat discomfort on review of systems. After adequate preoxygenation and bag mask ventilation (without an oral airway), he was induced with propofol, lidocaine, fentanyl and rocuronium. A left sided DLT size 41 Fr was introduced using CMAC-4 video-laryngoscope. The patient was positioned to the right lateral decubitus position and the position of the tube was again reconfirmed with the Storz, 4.0 mm Pediatric Fiberoptic bronchoscope (FOB). The procedure was uneventful, and he remained hemodynamically stable throughout the surgery. The extubation was performed in the supine position after suctioning and was also uneventful. Of note, there were minimal suction attempts needed prior to extubation, as his hypopharynx and oral cavity were quite dry. On postoperative day 1 he complained of pain, dysphagia, and a feeling of "an object dangling over his tongue." Head and neck surgery was consulted, and airway examination revealed necrosis and ulceration at the tip of his uvula. (Image 1)

Results: Emmett et al suggested that men are at a higher risk of developing uvular edema and/or necrosis owing to their bulkier tongues and palates, with significantly more non-fat tissue in the neck. [4] Uvular injury can present with wide varieties of symptoms ranging from persistent sore throat, dysphagia, foreign body sensation, and/or gagging. The tip of the uvula can be ischemic, inflamed, or may be covered with exudate in the post operative period. [5] A retrospective analysis conducted by Pamnani et al, reviewed 28,788 patients, showing higher incidence of uvular trauma amongst males who underwent general anesthesia. They noted that apart from the presence of an endotracheal tube or supraglottic device, the type of bite blocks (hard or soft) used, the length of the case, and the length of time that any gastric tubes were in-situ, were also considered significant factors. [6] Although studies have shown higher incidence of upper airway trauma, no study has clearly demonstrated the incidence of postoperative uvular trauma related to the use of single and DLT. [7] Patients undergoing thoracic surgery are often positioned in the lateral decubitus position. Chatterjee et al first described the possibility of increased incidence of uvular trauma following lateral decubitus position in patients who underwent bronchoscopies and endoscopies. [8] He mentioned the reason behind it to be the variation in the airway anatomy after assuming lateral position. The uvula getting caught between the hard palate and an airway device is yet another possibility.

Conclusions: Uvular trauma in the perioperative period is rare., but it can cause persistent discomfort. Anesthesia providers should be aware of potential causative factors, especially with regards to the use of DLTs. Being mindful of these factors and taking proper precautions can mitigate the chances of injury and lead to early recognition/intervention when injury occurs.

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Airway Management 4 - Emergent airway protection in patient with trismus, retrocollis, and airway obstruction

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Introduction: Stridor due to airway obstruction from inspissated nasopharyngeal secretions have been described in several case reports. Patients with depressed consciousness and poor oral hygiene are at particular risk of asphyxiation if these friable masses descend into the larynx. We present a case of a challenging emergent intubation for an elderly patient with trismus, severe kyphosis, and significant laryngeal mucoid impaction.

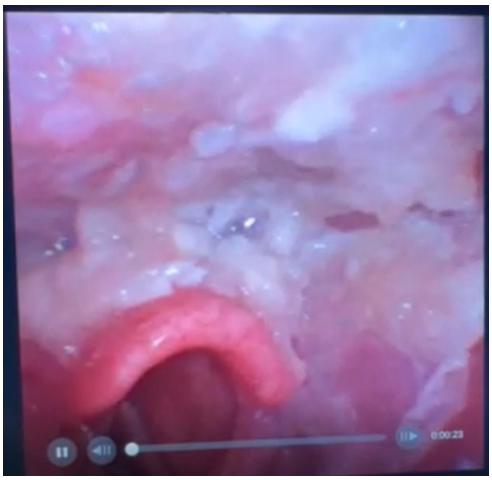
Methods: Our STAT airway team was paged to the floor unit to assist the ENT team with securing a protective airway for a stridulous patient who was undergoing a workup for progressive bulbar weakness, hypophonia, and quadriparesis. Upon arrival, ENT was at bedside performing a flexible fiberoptic laryngoscopy in an obtunded 65 y.o. man with marked retrocollis (height 178 cm, BMI 17.6 kg/m2, potassium 4.0mmol/L), generalized muscle rigidity including neck stiffness, and limited mouth opening from jaw clenching (interincisor gap 1.5cm). The patient was breathing spontaneously with SpO2 92-94% on nasal cannula, which we switched to humified oxygen via face tent. ENT found significant airway obstruction by a friable mass of inspissated crusts and secretions that abutted the epiglottis and limited glottal visualization. Oral access was limited, and the ENT team was unable to remove the obstruction transorally. A decision was made by the interdisciplinary teams present to intubate transnasally using fiberoptic bronchoscopy (FOB). Contingency plans were discussed. With the patient in semi-sitting position, the FOB scope was successfully maneuvered through the right naris past the mass, then an endotracheal tube (6.5mm ID) was threaded carefully and maximally past the cords with the beveled tip directed toward the luminal wall. FOB confirmed endotracheal intubation without evidence of mucosal dislodgement. After airway protection, sedation by propofol and pressure-support ventilation were initiated. Different extractive methods were attempted, including suctioning with hard- and soft-tipped suction devices, and dragging the mass from behind with an inflated FOB balloon. A 10mL syringe was wedged between the incisors to maximize oral access. Eventually a firm 3x3x7cm mucosal concretion was extracted transorally by Magills forceps. Remaining supraglottic crusting was removed by a combination of manual debridement and copious suctioning. The patient was then transferred to the neurological ICU.

Conclusions: Our patient had depressed consciousness, absolute trismus, and severe kyphosis that limited oropharyngeal access and neck mobility. Awake FOB intubation is an appropriate and accessible tool to manage such scenarios, even during emergent intubations. The patient remained spontaneously breathing and hemodynamically stable throughout the procedure, even without any narcotics or local anesthetic.

Extensive workup revealed the patient's clinical presentation to be consistent with progressive supranuclear palsy (PSP), a rare neurodegenerative disorder often involving postural instability, cognitive impairment, generalized hypertonia, dysphagia, trismus, and cervical dystonia. Though not always available, cervical MRI imaging can be helpful in detecting any airway stenosis. Literature on the anesthetic implications of this rare disease is limited, but most

agree that careful pre-anesthesia evaluation is critical.

The urgent nature of this case precluded extensive anesthetic preplanning. Clear communication and contingency planning with all team members (e.g., from anesthesia, pulmonary critical care, ENT, and nursing) were paramount for the safe and successful intubation. Expert opinion from non-anesthesia services can provide indispensable insight and assistance during difficult airway management. If our instrumentations had dislodged a mass into the trachea, the resolution was to drive it mechanically into one bronchus, thus permitting at least one-lung ventilation. We have demonstrated that a multidisciplinary approach in utilizing FOB is both and feasible for difficult and emergent airway management, including our case that involved significant airway obstruction, trismus, and retrocollis altogether.



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Airway Management 5 - Intraoperative Management of Right Lung Hemorrhage Post-Robotic Bronchoscopy

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Introduction: Flexible bronchoscopy visualizes the airway for diagnostic, therapeutic and palliative pulmonary interventions. Biopsy of pulmonary nodules is one such intervention. It aids in determining nodule pathology to guide treatment. Percutaneous image-guided and navigational biopsy with the help of endobronchial ultrasound or electromagnetic navigation were the primary options available for biopsy. More recently, robotic bronchoscopy systems have allowed pulmonologists to better access farther generations of airways and confirm the location of the targeted lesion. In general, the risk of significant morbidity and mortality associated with endoscopic techniques is low. Robotic bronchoscopy systems, in particular, have excellent safety profiles. Nevertheless, complications can occur and include trauma to the pharynx, vocal cords, and airway; atelectasis; pneumothorax; and airway hemorrhage. Here we discuss intraoperative management of airway hemorrhage during a diagnostic robotic bronchoscopy.
Methods: A 66-year-old female with a history of current smoking in the setting of chronic obstructive pulmonary disease on 2L oxygen at night, fibrocavitary lung disease secondary to M. kansasii, hypertension, cerebral vascular disease, and carotid endarterectomy presented for diagnostic robotic-assisted flexible bronchoscopy for biopsy of a right middle lung spiculated nodule. Induction of general anesthesia and intubation with an 8.5 mm endotracheal tube (ETT) occurred without complication. Bronchoscopy and tissue collection began without event. During the final planned biopsy, blood was noted in the ETT, and peak airway pressures were elevated. Further inspection by bronchoscopy discovered significant bleeding from the airways in the proximity of the biopsy site. Initial attempts to stop the bleeding with cold saline and suction were aborted when the patient's oxygen saturation (SpO2) fell below 90%. The patient was placed in the right lateral decubitus position and additional help was called. The anesthesiologist and pulmonologist decided to intentionally guide the endotracheal tube into the left mainstem bronchus to protect the left lung from the uncontrolled bleeding in the right lung. Isolation was completed under bronchoscopic guidance and placement was confirmed by fluoroscopy. After positioning the ETT in the left mainstem, a leak was noted, there was minimal end-tidal CO2 (ETCO2), and the patient's SpO2 continued to decrease to a nadir of 69%. An extracorporeal membrane oxygenation (ECMO) consultation was called. A second attempt at left mainstem positioning was successful and the patient's ETCO2 increased to more than 35mmHg and the SpO2 gradually started to increase. The ECMO consult team declined the consultation citing insufficient resources. The patient was placed on the ventilator and her SpO2 was sustained above 95%. Attempts were made to place a bronchial blocker into the right mainstem bronchus external to the ETT, but they were unsuccessful. At this time, the patient was hemodynamically stable, and bleeding from the right lung appeared stable on fluoroscopy. It was then determined that the patient should be transported to the medical intensive care unit (ICU) for further care with the ETT remaining positioned in the left mainstem bronchus. Prior to ICU transport, the anesthesia team gave additional neuromuscular blocking drugs given the concern that coughing or bucking could dislodge the blood clot leading to resumption of bleeding.

Results: Not applicable - Medically Challenging Case Report

Conclusions: While complications in bronchoscopy cannot be eliminated, when they do occur maintaining interdisciplinary communication is essential. One of the most challenging complications to manage is airway hemorrhage. Bleeding that occurs as a result of bronchoscopy is often self-resolving, however, it is critical that the anesthesiologist be aware of methods to control the extent of bleeding while maintaining adequate ventilation and oxygenation. When bleeding is refractory to techniques such as using cold saline or tamponading with a bronchial blocker, laying the patient in the lateral decubitus position so that the bleeding lung is in the dependent position is an important step in protecting the non-bleeding lung. Isolating the non-bleeding lung by intentional mainstem intubation or using a double-lumen tube or bronchial blockers provides further protection. By employing these methods, the bleeding was controlled, and the patient was safely transferred to the ICU for further management.

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Airway Management 6 - The Killian-Jamieson Diverticulum That Wasn't, And the Mass That Was: Obstructing the Airway!

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University of Florida[1]

Introduction: Killian-Jamieson diverticulum is a rare type of phrenoesophageal diverticulum that occurs in an area of anatomic weakness just inferior to the cricopharyngeus muscle: the Killian-Jamieson space. Similarly, to the more common Zenker's diverticulum, it can present with symptoms of dysphagia. We present the case of a 53-year-old male who presented for emergency esophagogastroduodenoscopy (EGD) to remove an acutely impacted food bolus. At video laryngoscopy, a large glottic mass was present, creating a challenging intubation and the need for rapid course correction by the anesthesia-endoscopy team in regard to the planned endoscopy and post-procedural care.

Methods: A 53-year-old male presented to UF with an acute history of food stuck in his throat after eating a sandwich. Past medical history was significant for: tobacco use and 6 months of dysphagia and dysphonia. Emergent EGD was recommended. Our chart review revealed the patient had a similar event 3 months before. On that visit a CT revealed a 2.9 x 1.4 x 6 cm mass at the level of the cricoid and adjacent to the carina. The official reading was "possible Killian-Jamieson diverticulum". The patient was then evaluated by ENT by laryngoscopy which showed: an esophageal mass that "fluctuated in size"; normal true vocal cords; normal vocal cord mobility; no epiglottic lesions. The patient was recommended follow up but had not done so. On the day of service, the patient had acute dysphagia, dysphonia and "inability to control his secretions". In the ED, plain neck film showed increased soft tissue density at the hypopharynx/piriform sinus and otherwise normal soft tissues. Chest X-ray was normal. He was given dexamethasone, sublingual nitroglycerin and glucagon to encourage passing the food bolus. In Endoscopy he was observed closely for two hours while awaiting an available proceduralist. He was able to swallow his saliva and had no respiratory abnormalities. He had stable vital signs, Mallampati 1 airway, >3 fingerbreadths mouth opening, >6 cm thyromental distance, no tracheal deviation, normal-appearing oropharynx, normal heart and lungs: all pointing to the appropriateness of RSI and general anesthesia. The endoscopist was asked about the pre-existing diagnosis of Killian-Jamieson diverticulum and said that the condition should not be of immediate concern. Pre-EGD, the patient received 2mg midazolam and 100% oxygen via face mask. Rapid sequence induction was with 100mg fentanyl, 80mg lidocaine, 150mg propofol, 100mg succinylcholine, and no bag-mask ventilation. With CMAC, we identified the epiglottis, a shiny mass that obscured other anatomy, and finally the glottis pushed far right. We managed to intubate before any desaturation <95% occurred. The endoscopist attempted EGD but was unable to get past the mass. The procedure was aborted. We kept the patient intubated post-EGD for airway protection. He was admitted directly to ICU. ENT was consulted and subsequently performed hot snare removal of a large pedunculated cystic lesion emanating from the apex of the left piriform sinus via a thin stalk-like projection. The patient was

extubated and did well.

Conclusions: This case report encapsulates the high stakes challenges that arise in GI NORA. Here we had a patient present for emergent EGD billed as acute food impaction plus incidental Killian-Jamieson diverticulum. We proceeded with RSI which created its own immediate challenge bordering on an airway emergency. This patient's cystic lesion, being pedunculated, most likely flipped upwards to produce significant glottic deviation not seen or appreciated on just-prior radiographic exams. In retrospect, we would opt instead for fiberoptic-assisted intubation under spontaneous ventilation rather than reliance on good fortune.

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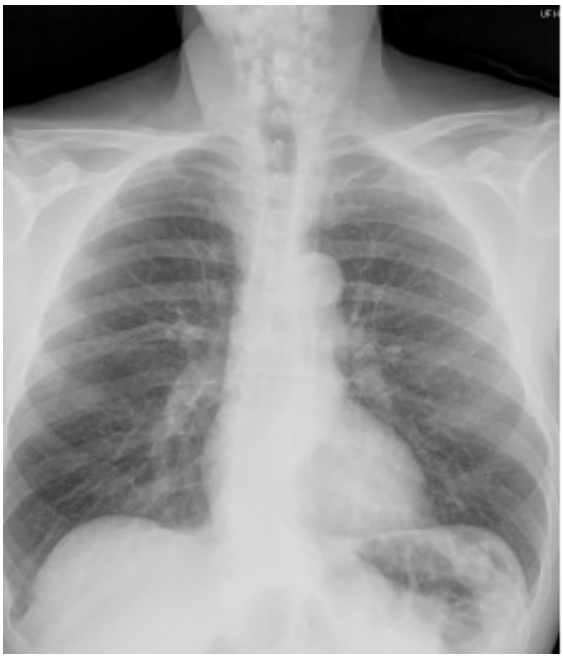

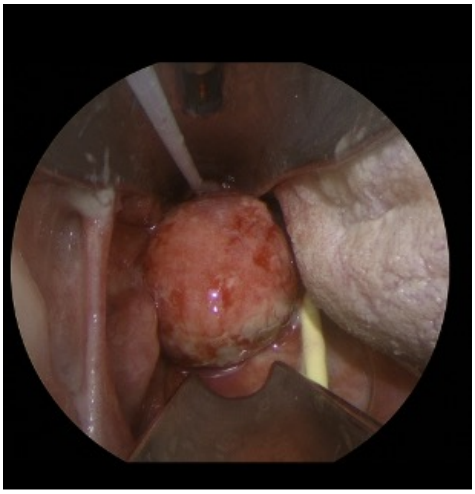

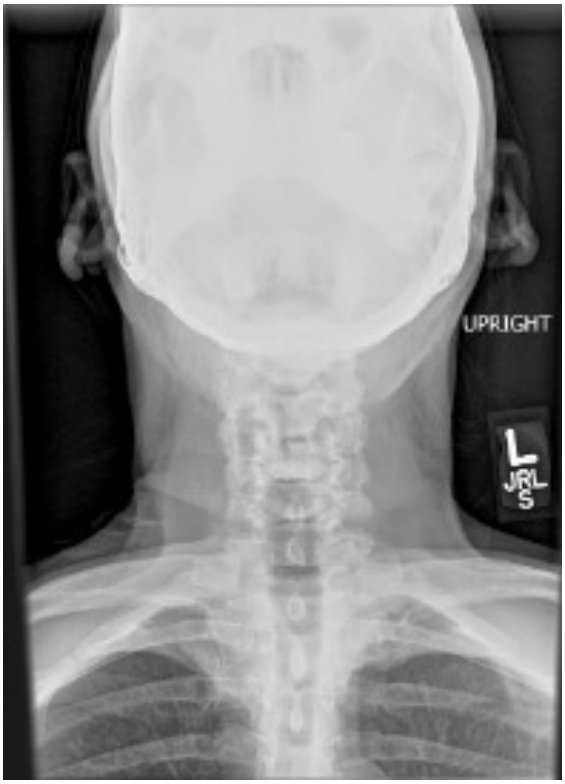

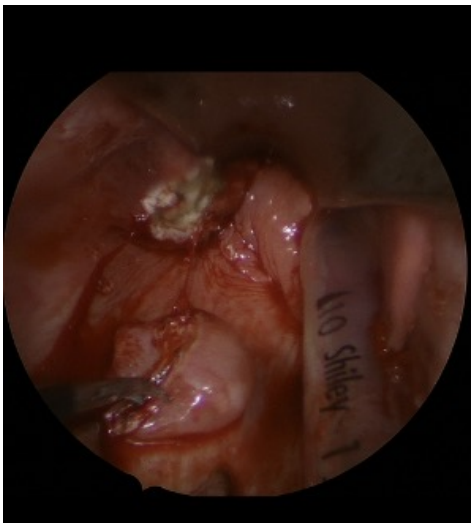

Airway Management 7 - Treatment of Severe Tracheal Stenosis by Advanced Bronchoscopic Interventions and Placement of Modified Montgomery T-Tube

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Introduction: A patient with tracheostomy dependence due to long-segment tracheal stenosis (LTS) secondary to prolonged intubation for severe COVID-19 infection presented with aphonia and dyspnea due to recurrence of complete tracheal stenosis. She was scheduled for operative treatment of her stenosis using combined flexible bronchoscopic, rigid bronchoscopic, and trans-cervical interventions.

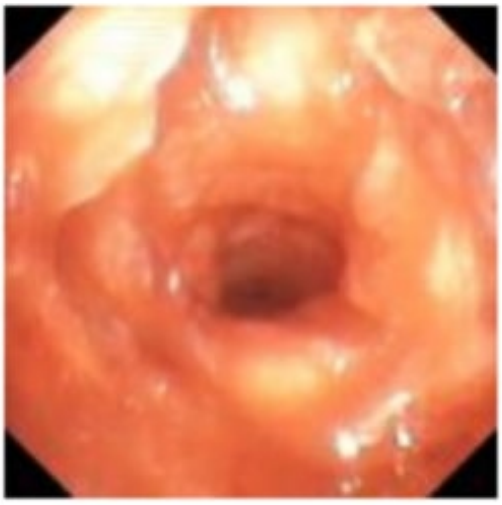
Methods: The patient was preoxygenated on 100% FiO2 through the existing 6 cuffless Shiley tracheostomy. Induction was performed with IV propofol, remifentanil, and rocuronium. The cuffless tracheostomy was then switched to an armored endotracheal tube placed through the well-formed tracheostoma, and end-tidal CO2 was confirmed.

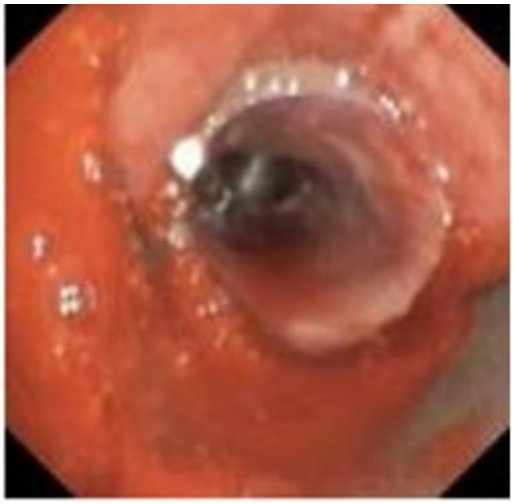
After exploring the distal airways, which were found to be unremarkable, a bronchoscope was placed through the oropharynx to the glottic level, revealing a 100% narrowing of the lumen of the proximal trachea above the tracheostoma (Figure 1). To improve airway patency, debridement and treatment of stenosis was with a combination of electrocautery, cryotherapy, and debridement with a rigid bronchoscope. Jet ventilation was utilized during periods where the armored endotracheal tube was removed and delivered through the rigid bronchoscope while occluding the tracheostoma. The suprastomal airway was found to still be significantly collapsed and required removal of granulation to allow for tracheal stent replacement (Figure 2). A T-tube was then modified by removal of the inferior limb and the bottom half of the horizontal limb to convert it into a suprastomal ‘J stent’ and placed via the tracheostoma in the proximal trachea (Figure 3). The stoma was then re-intubated with a 6 cuffless Shiley tracheostomy tube.

Conclusions: Laryngotracheal stenosis is a late complication after long-term intubation and is due to the result of mechanical trauma from the endotracheal tube or cuff leading to tissue necrosis and inflammation. The volume of patients requiring intubation and mechanical ventilation secondary to the COVID-19 pandemic has

led to an increased number of patients presenting with LTS as a sequale of their hospital stay[1]. The considerations and methods used in this case therefore become extremely important to characterize in this post-COVID era.

This patient had both a covered metallic tracheal stent and a Lymol Y-stent previously placed to attempt to recover her stenosis, both of which were complicated by stent migration and occlusion of her tracheostomy tube requiring emergent removal. The use of a J-stent offers the ability to maintain stent security by anchoring the horizontal limb through the tracheostoma and allows maintenance of a traditional tracheostomy tube below to reduce risk of mucus plugging. Rigid bronchoscopy is used in patients who have an obstruction of the upper airway and was an integral instrument in this procedure as well, allowing for the introduction of a variety of tools while also providing the means for ventilation.





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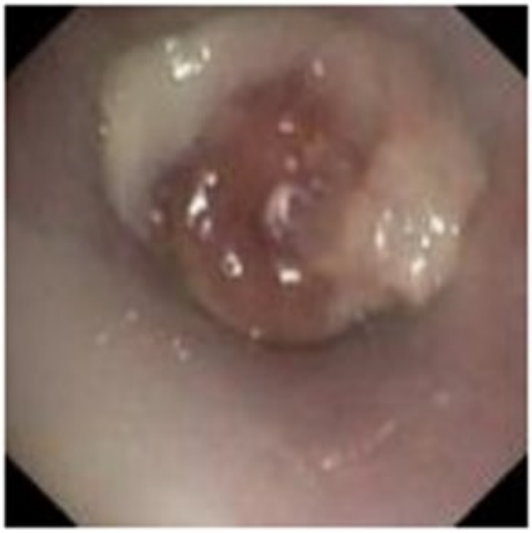

Airway Management 8 - A Case of the Mismanaged Difficult Airway

Shireen Abdel-Razzaq[1], Melodie Sarebanha[2]

Cleveland Clinic[1] Cleveland Clinic Foundation[2]

Introduction: Managing a difficult airway can be one of the most challenging scenarios faced by critical care physicians. The purpose of this case report is to explore the various challenges that can present in the care of patients labelled a "difficult airway" particularly during planned extubations. This case reiterates the importance of adequate planning, acquisition of patient history, review of prior anesthetic, surgical airway interventions and available imaging, along with a multidisciplinary approach to ensure safe management of "difficult airway" patients in critical condition.

Methods: In this case report we present the case of a 64-year-old woman admitted for recurrent incarcerated incisional hernia with documented prior "difficult airway" in 2018. At that time, there were 7 attempts at intubation prior to successful placement of 7.0 endotracheal tube with combination of fiberoptic bronchoscope and video laryngoscope. With that knowledge, the patient was taken to the OR for incisional hernia repair. Anesthesia successfully placed a 7.0 endotracheal tube on their 4th attempt with combination of video laryngoscope and bougie. She was treated for airway edema and remained intubated until post operative day 2. Following a successful spontaneous breathing trial and evaluation of cuff leak,

patient was extubated to 2L nasal cannula by the critical care physician in the evening just prior to shift change. Within 8 minutes of extubation wheezing and stridor were reported by the bedside nurse. The patient was treated with racemic epinephrine without improvement. It was 6 hours until there was true recognition of airway compromise and activation of the difficult airway response team with an additional hour of locating supplies and attempts to secure the patient's airway with ENT assisted awake fiberoptic and 5.0 MLT. Further history from the patient's family elucidated surgical history of two prior tracheostomies. Due to this history and the patient's clinical condition, a decision was made to proceed with tracheostomy. On post operative day 3 the patient underwent Bjork-flap tracheostomy with ENT due to discovered medialized right carotid artery during dissection. Patient was successfully weaned off mechanical ventilation and sent to the regular nursing floor post operative day 9.

Conclusions: Extubation of patients with known "difficult airway" in the ICU should always be considered an elective procedure and as such there should be sufficient planning and multidisciplinary teamwork involved to ensure the safety of these patients. This case exemplified how important review of prior airway interventions and surgical airway history are along with imaging, as emergent airway techniques may not be possible or appropriate for all patients. While it may seem obvious, extubation of these patients in particular should not be undertaken during hand-off of care or while airway experts (anesthesia and ENT) are not available for immediate guidance and hands on assistance.

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Airway Management 9 - Acquired Tracheal Abnormality Requiring Complex Airway Management in Critically Ill Burn Patient

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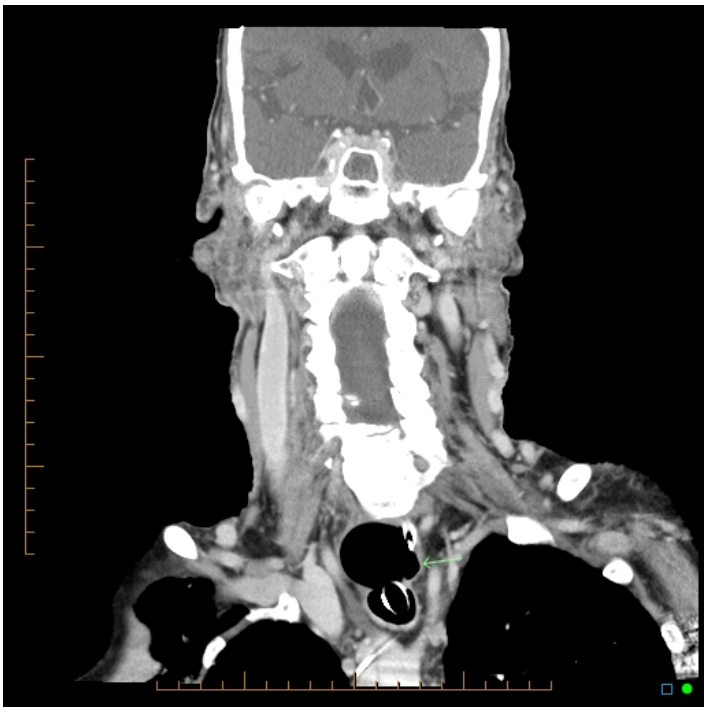
Loyola University Chicago Stritch School of Medicine[1] Loyola University Medical Center[2]

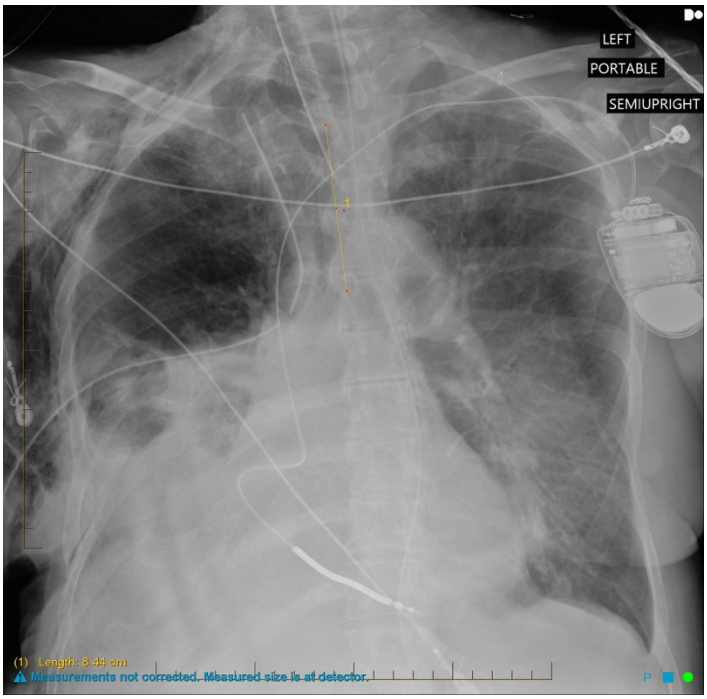
Introduction: Nearly 40,000 people in the United States suffer burn injuries that require hospitalization each year. For head and facial burns, inhalational injury is a concern and careful management of the airway is essential. Burn patients often have several risk factors for a difficult airway including facial swelling and airway edema. Many of these patients undergo numerous surgeries and require prolonged mechanical ventilation. Injuries to the trachea have been reported with prolonged mechanical ventilation, with dilatation as an early complication and stenosis as a late complication. In this case, we report a case of tracheal dilatation in a critically ill burn patient that led to complex airway management in the operating room.

Methods: A 61-year-old female with past medical history of hypertension, tobacco and alcohol abuse, and prior ventricular fibrillation was admitted for second- and third-degree flame burns to the head, face, and neck, comprising 5% total body surface area. Intubation was performed in the emergency department due to inhalational injury and acute hypoxic respiratory failure. The patient experienced recurrent episodes of respiratory decompensation in the intensive care unit which was associated with audible cuff leaks and loss of tidal volumes. Frequent repositioning and/or exchanging of the endotracheal tube (ETT) was performed. A CT neck performed 20 days after admission demonstrated a circular 4.1 cm dilatation at the proximal trachea. The recurrent air-leaks were attributed to migration of the ETT proximally into the dilated tracheal segment. The patient was weaned from mechanical ventilation as tolerated during hospitalization and was reintubated for airway management during operations. During intubation in preparation for a skin grafting operation, the team required three intubation attempts along with confirmation of ETT placement with a fiberoptic scope despite obtaining Grade 1 views on each attempt. On subsequent reintubations and repositioning, care was taken to seat the ETT cuff distal to the dilated segment of trachea, ensuring seal within the airway.



Results: Not Applicable – Medically Challenging Case Report

Conclusions: This case demonstrates how post-vocal cord airway pathology can cause a difficult airway. The case also highlights how interdisciplinary collaboration can be more important as hospital length of stay increases, and that accurate electronic medical record documentation of airway parameters is a key part of the airway assessment.



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Michelle Baker[1], Gyorgy Frendl[2]

Brigham and Women's Hospital[1] Brigham and Women's Hospital / Harvard Medical School[2]

Introduction: To review a medically challenging case.

Methods: We present a 67-year-old woman who presented with subglottic stenosis after a 1-day intubation for airway protection in the setting of status epilepticus. Patient was symptomatic with new hoarseness, wheezing, and stridor within 3 days of extubation. Flexible scope exam notable for edematous vocal folds but without upper airway obstruction. Chest CT is significant for narrowing of the subglottic trachea, with a 6mm opening at its narrowest. Initial concern for post-intubation tracheal edema causing airway compromise versus untreated bacterial tracheitis ultimatel resulting in subglottic stenosis. Patient started on IV steroids and racemic epinephrine without improvement, ultimately requiring ICU transfer for escalation of care including Heliox initiation to increase laminar flow. Bronchoscopy performed via LMA showing extensive mucosal sloughing and necrosis. Tracheal debridement performed utilizing cryotherapy with resolution of stenosis. Patient remained stable on room air after debridement.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: We concluded the patient likely had untreated bacterial tracheitis ultimately resulting in profound subglottic stenosis, although etiology remains unknown. Other possible causes are post-intubation stenosis and idiopathic subglottic stenosis, although less likely. Patient was given a full course of Augmentin along with bronchial debridement which provided full relief of her stenosis. Patient will return for a follow-up CT scan in 2 weeks' time to evaluate for recurrence.

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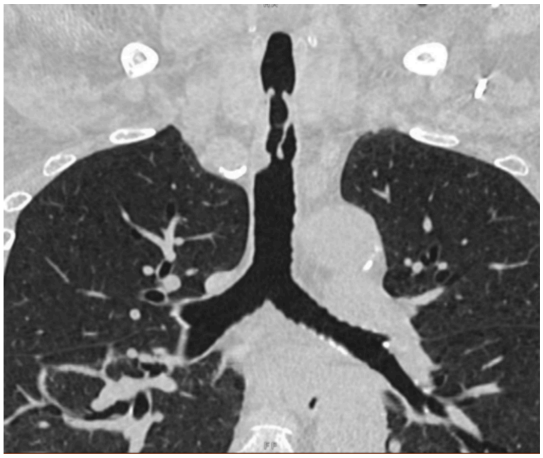

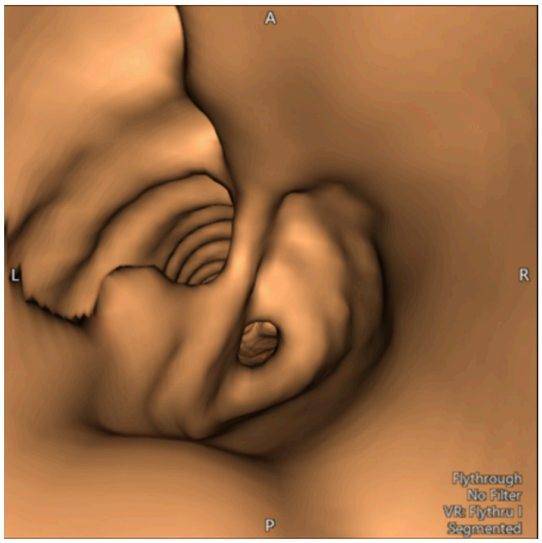

Airway Management 11 - Difficult airway management in an adult patient with end-stage achalasia

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Albany Medical Center[1]

Introduction: Achalasia is a failure of the lower esophageal sphincter (LES) to relax which is typically caused by the degeneration of inhibitory neurons within the esophageal wall. It is classified as either primary (idiopathic) or secondary to several conditions such as cancer, Chagas disease, amyloidosis, neurofibromatosis type 1, and sarcoidosis. Clinical features of achalasia include dysphagia to both solids and liquids, regurgitation, retrosternal pain, and later morbidities attributed to anorexia and weight loss; additionally, these patients also present a significant aspiration risk in the perioperative period.[1] Achalasia is a rare disorder, affecting roughly 1.6/100,000 individuals, with a mean age at diagnosis of 53 years.[1] In this case, we describe an adult patient with end-stage achalasia presenting for a three-field esophagectomy and gastric mobilization and the important of the difficult airway algorithm when architecting a safe airway plan.

Methods: Our patient is a 40-year-old female with a past medical history significant for several years of worsening achalasia that began 6 years ago initially treated with a Heller myotomy and fundoplication. She underwent a peroral endoscopic myotomy (POEM) three years ago which partially stabilized her symptoms. She notes daily regurgitation and dysphagia without chest pain and self-regulates her diet. She has difficulty lying down due to regurgitation of old food.

Patient underwent an esophagogastroduodenoscopy (EGD) 3 months prior to surgery that showed significant dilation and dysmotility of the esophagus filled with fluid and an open EG junction. A plan was initially made for RSI with cricoid pressure (easy mask, glidescope with a MAC 3 blade, grade 2 view, 7.5 ETT); during induction, two attempts to intubate with direct laryngoscopy were made due to uncertainty of initial tube placement. After the patient aspirated, a third attempt was made after thoroughly suctioning the oropharynx and utilizing video laryngoscopy; tube placement was confirmed with end tidal CO2 and bilateral chest rise.

When the patient presented for her recent esophagectomy, two attempts with video laryngoscopy with a MAC 3 blade (grade 1 view; albeit anterior airway) and a double lumen ETT were made. Patient aspirated despite thorough suctioning, change in anesthesiologist, and optimized positioning; a collective decision from the anesthesiology and surgery team was made to cancel the case and reschedule to optimize management of her airway.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Achalasia presents as a unique perioperative challenge to the anesthesiologist. In this patient with a known difficult airway, applying the 2022 difficult airway algorithm was necessary based on this patient's specific pathology in order to optimize the intubation.[2] In this scenario, thorough documentation of a prior difficult airway and ultimately canceling the case were key factors in optimizing patient safety.

Due to severe achalasia and significant risk for aspiration, the next step that will be considered for this patient is an awake intubation or placing a nasogastric tube while awake. The patient will be pretreated with midazolam and glycopyrrolate with multiple Yankauer suction devices on hand to manage her secretions. Her airway will be topicalized with a laryngeal tracheal atomizer (LTA with nebulized lidocaine) in addition to lidocaine-soaked pledges. Additionally, high-flow nasal cannula oxygen will be considered to prevent desaturation. Patient understood the complexity of her pathology and is agreeable with this plan.

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Airway Management 12 - Managing Difficult Pediatric Airways: An Early Success Story with the ASA 2022 Practice Guidelines

Michael Larkins[1], John Iasiello, Kevin Travia, Melisa Pasli, Sunny Cai[2], Adrienne Singleton

East Carolina University Brody School of Medicine[1] ECU Health[2]

Introduction: The 2022 American Society of Anesthesiologists (ASA) Practice Guidelines for Management of the Difficult Airway differ significantly from prior airway guidelines, particularly regarding pediatric patients.[1] Notable highlights of the new guidelines focus on alternative considerations for anticipated noninvasive airway management failure, including: emphasis on a time-out prior to procedure execution, careful selection of intubation technique, minimizing intubation attempts, and early evaluation for extracorporeal membrane oxygenation (ECMO) deployment. Our aim is to demonstrate the importance of the changes in the 2022 ASA Practice Guidelines for the Management of the Difficult Airway in pediatric patients with a case study of a patient with a rare adrenal neuroblastoma metastasized to the mandible.

Methods: A 16-month-old female presented to a rural tertiary care center with a rapidly growing right mandibular mass, suspected metastatic renal neuroblastoma, for meta-iodobenzylguanidine (MIBG) scan and bone marrow biopsy under sedation. Upon anesthesiologist preoperative examination, the mass was solid, dense, and hard, found to encompass 60% of the patient's oral cavity, cause copious secretions, and displaced the patient's teeth and tongue. The patient's parents also endorsed an inability to tolerate oral intake within the prior couple of days. Due to the size and location of the mass, the anesthesiology team determined the patient posed a difficult airway and subsequently implemented the 2022 ASA Practice Guidelines. A multidisciplinary team consisting of an otolaryngologist, pediatric surgeon, perfusionist (ECMO team), and anesthesiologists was assembled. Review of the patient's head and neck computed tomography (CT) scan indicated the mass did not extend or obstruct the patient's epiglottis and, when considering the possible need for nasal intubation, revealed an unimpeded nasopharynx. Given the technical challenges associated with the patient's airway, emergent tracheostomy was considered a contingency plan and ECMO circuit was available in the operating room on standby. Intraoperatively, the multidisciplinary team reviewed the imaging and clinical presentation, performed a time-out, and proceeded with induction. The patient tolerated inhalational induction and the anesthesiology team was able to successfully manually ventilate via a facemask. Intubation was achieved with video laryngoscopy and a C-Mac size 2 pediatric blade in one attempt.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: The importance of the 2022 ASA Practice Guidelines for Management of the Difficult Airway for pediatric patients cannot be understated. Emphasis on assembling a multidisciplinary team, performing a thorough airway exam with appropriate imaging, conducting a time out prior to procedure start, utilizing video laryngoscopy, and minimizing airway attempts is what likely yielded a successful and safe intubation in our case. Anesthesiologists should be familiar with and should be the driving force in implementing these new guidelines for potential pediatric difficult airways. Without the safeguards integrated into these new guidelines, the potential for adverse events is substantial.

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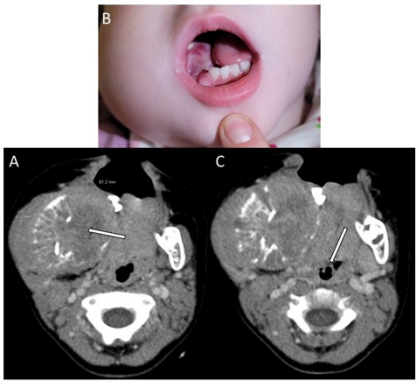

Figure 1 – A: Computed Tomography (CT) scan of our patient's head and neck, with arrow pointing to right mandibular metastasis at widest diameter in the jaw; B: Comparative pre-intubation photo of our patient's right mandibular mass; C: Another slice of our patient's CT, showing midline shift but no tumor involvement of our patient's epiglottis.

Airway Management 13 - Occult tracheobronchial injury Management following Lung Transplantation

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Cedars Sinai Medical Center[1]

Introduction: Tracheobronchial (TB) injury during intubation is a known risk that carries high morbidity and mortality(M&M). The use of double lumen tube (DLT) is associated with even increased risk, but sometimes the exposure required necessitates their use. We present a case of occult TB injury and its management.

Methods: A 65F with a history of rheumatoid arthritis (RA)-associated interstitial lung (IL) disease on chronic steroids presented for a bilateral lung transplant off pump via median sternotomy. Her intraoperative course was complicated by a left mainstem bronchus (LMB) laceration extending beyond the carina and ending roughly 1cm before the LMB anastomosis. This finding was noted when the DLT was exchanged for a single lumen (SL) endotracheal tube (ETT) at the end of operation. Photos and a video were taken, and a multidisciplinary team discussion was held to determine the best course. There was no air leak from either pleural cavity despite positive pressure ventilation, and it was decided to pursue conservative management. The patient was transferred to ICU with stable hemodynamic and respiratory function. With this known TB injury, the patient was managed judiciously to try and facilitate extubation to minimize positive pressure ventilation which could worsen the known defect. She was initially extubated on postoperative day (POD) 1 to nasal cannula but was reintubated on POD5 for acute hypoxemia during bedside bronchoscopy to monitor TB defect. After reintubation the patient underwent CT which demonstrated new significant pneumomediastinum, but the patient was otherwise hemodynamically stable with minimal ventilatory support, conservative management was continued. Given worsening subcutaneous emphysema now with air leak and known LMB laceration, the patient extubated on POD6 to minimize positive pressure ventilation. Subsequently, on POD8 patient again required intubation for acute hypoxemia and patient was deemed to have failed conservative management and interventional pulmonary was consulted for bronchial stenting. Given the location of the injury it was difficult to cover the defect and the patient required 2 stent exchanges before a successful Y-stent was placed that covered the trachea, the right mainstem bronchus, and the LMB on POD13. Patient was extubated successfully on POD15 and downgraded from the ICU.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: TB injury is a rare but serious complication of endobronchial intubation, with an estimated incidence of 0.001% to 0.005% (1) with single lumen tubes and 0.20% -0.25% with DLT placement (2). TB injury carries a marked increase in mortality; a case series of TB injury by Miñambres et. al demonstrated mortality of 44% (3). Despite the known impact on M&M, there is no official consensus on management of injury once it's been identified. Cardillo et. al offered a morphological classification system to help guide management. It stratified injuries into 4 possible groups based on depth of injury with Level 1 being superficial injury that can be observed to Level IIIB complete laceration with esophageal injury or evidence of mediastinitis (4). Any injury of level I or II could be initially managed conservatively, and any injury of Level III should be treated surgically or if the patient is a poor surgical candidate with endobronchial stenting (4). TB injury was identified during exchange of DLT to SL ETT, and as there was no evidence that the injury was causing issues with respiratory function the decision was made to observe. The patient progressed well until POD 5 when she required reintubation, at this time she should have been deemed failure of conservative therapy and definitive intervention should have been performed. Given her RA-ILD, age, short stature, and area of injury to the LMB re-operation would be difficult and as the patient would continue to require long standing immunosuppression decision was made to perform the less invasive procedure of endobronchial stenting. Ultimately, further studies are needed to best identify how to manage TB injury from intubation. How long should a patient be monitored if they continue to show issues with respiratory failure? As DLTs increase the risk even with perfect placement, should we be performing operations that typically require DLTs with SL-ETTs with bronchial blockers with patients identified at increased risk? Further studies should elucidate how to best manage TB injury.

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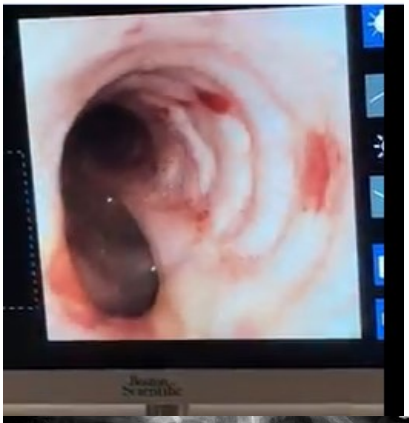

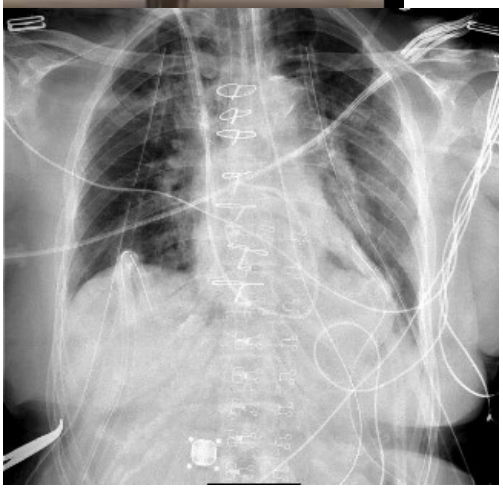

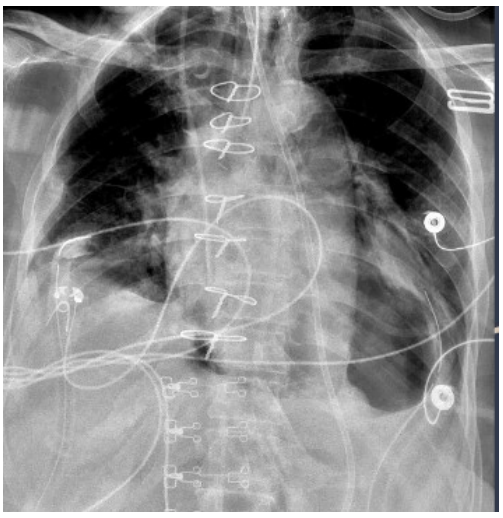

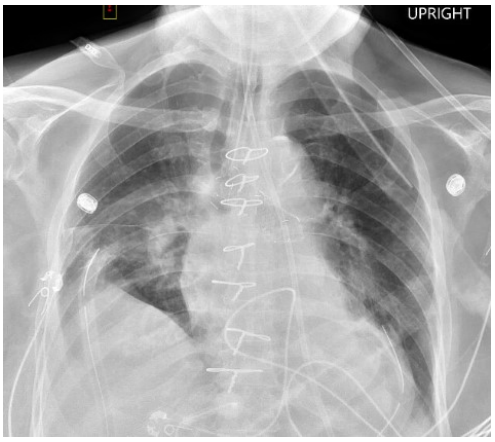

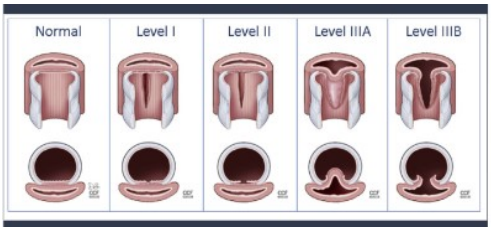

Airway Management 14 - Well what do I do now? Anesthetic Management in the setting of complete tracheal transection

Vyshakh Shibu[1], Clifford Song[2], Amit Prabhakar[2]

Emory department of Anesthesiology[1] Emory University School of Medicine[2]

Introduction: Tracheal injuries can be confounding situations for an anesthesiologist to manage as patients can frequently present with respiratory compromise. Complete tracheal transection is extremely rare. One study found that out of 1178 autopsies on patients with thoracic injuries, only 1 had complete tracheal transection. Generally, the mechanism of injury is blunt or sharp trauma to anterior or lateral aspects of the throat and hyperextension. Rupture at the thoracic trachea can be due to rapid acceleration and deceleration of the lungs along with sagittal compression of the trachea. We present a patient with complete transection of the trachea at the level of the carina and will review the anesthetic techniques used during the case.

Methods: A 26-year-old male with no prior medical history presents with complete tracheal transection at the level of the carina and bilateral tension pneumothoraces after a motorcycle accident. He was intubated, had bilateral chest tubes placed and was put on vasopressor support to stabilize his hemodynamic profile prior to presenting to the operating room (OR) emergently for repair. In the OR, the circuit was attached to the oral endotracheal tube (ETT) and bronchoscopy confirmed transection at the carina with a 5cm gap between the proximal and distal end. Appropriate tidal volumes were presumably maintained from mediastinal pressure enclosing the injury. Thoracotomy was performed with immediate loss of ventilation as the mediastinal pleura was opened. Subsequently, a reinforced ETT was advanced into the left mainstem bronchus under direct visualization and cross-field single lung ventilation was initiated. After lower airway structures were dissected, intermittent cross table ventilation was done to allow the surgeons to anastomose the trachea. After anastomosis, the oral ETT was advanced through the trachea, across the anastomosis and into the left mainstem bronchus. The cross-field tube was removed. An intercostal muscle flap was placed over the tracheal anastomosis. Afterwards, the ETT was withdrawn to above the anastomosis, thus ventilating both lungs. ABG showed improved oxygenation and ventilation. Bronchoscopy was performed and any remaining clot was removed. The patient was then transported to ICU intubated in guarded condition.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: In this case, anesthetic management focuses on maintaining oxygenation and ventilation while the trachea is repaired. Cross-field ventilation allows for maintenance of oxygenation and ventilation of one lung while the surgeon repairs the lesions. Once anastomosed, the cross-field tube can be removed, and the oral tube can be advanced past the anastomoses. The other lung can be ventilated while the surgeon completes the repair. PEEP comes with the risk of anastomotic rupture and must be used judiciously. During single lung ventilation, there is still perfusion to the non-ventilated lung resulting in an intrapulmonary shunt causing hypoxemia as shown here. Increasing FiO2 to 100% minimizes shunt, increases safety margins, and promotes vasodilation in the ventilated lung. However, high FiO2 increases risk of atelectasis and free radical damage. In addition, patient positioning also aided us during left lung ventilation secondary to gravity dependent pooling into the lung while in left lateral decubitus.

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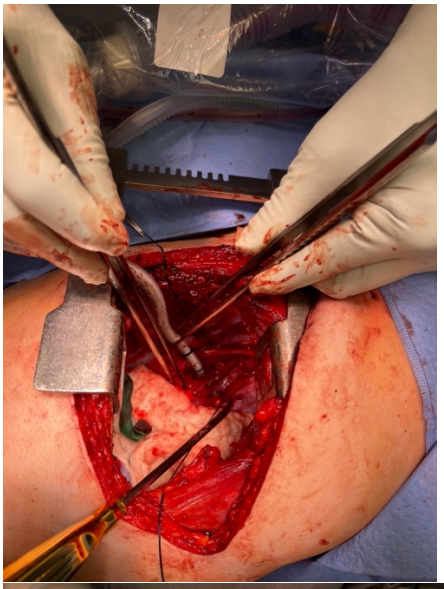

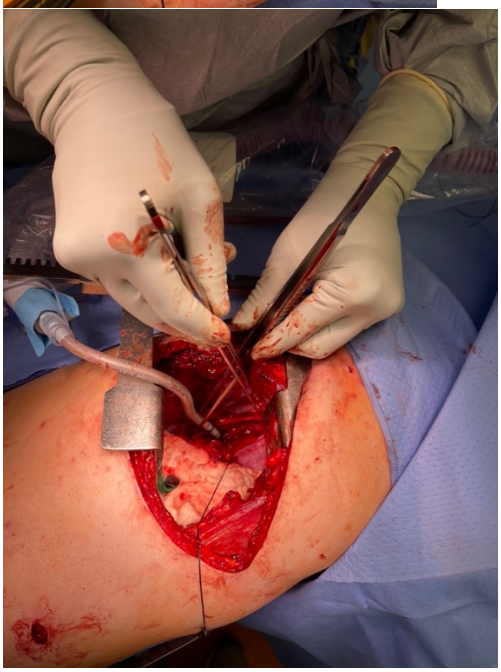

Ambulatory Anesthesia 1 - In NORA, Etomidate is Great!

Georgeanna Tsoumas[1], Angela Bott[1], Jeffrey White[1]

University of Florida[1]

Introduction: Etomidate is a trusted anesthetic induction agent with a 50-year history of use [1]. It is a preferred choice in the ED for emergency intubations [2], and it is often selected within the OR for patients with LVEF impairment or circulatory shock. Studies have suggested that the drug may be associated with increased morbidity and mortality [3] especially in septic patients [4] due to its blocking effect on 11-beta hydroxylase causing short-term adrenal insufficiency [1]. Other studies suggest that it is safe to use, even in sepsis [5]. We present a NORA case where an etomidate infusion monotherapy technique provided perfect deep sedation and cardiovascular stability without need for vasopressors or any changes in post-procedural acuity of care.

Methods: A 75-year-old 80kg male presented for screening colonoscopy in advance of planned LVAD placement. Past Medical History included: end-stage CAD and associated ischemic cardiomyopathy (ICM); s/p STEMI with 7 stents placed; Class 4 CHF; cardiac arrest with VT; AICD implantation; home inotropic support with milrinone. His cardiac workup included cardiac MRI, revealing LVEF 19% and minimal viability. Right heart catheterization showed severe post-capillary pulmonary hypertension (RVSP 65).

His pre-procedure vitals were noted as: BP 135/96, HR 72, RR 18, SAT 100%. On induction, he received etomidate 8mg (0.1 mg/kg), followed by infusion of etomidate (40mcg/kg/min). He maintained a satisfactory spontaneous respiratory rate with oxygen saturations of 95-99% on 4 LPMNC. Blood pressure was stable throughout the case without the use of vasopressors. Image one demonstrates the patient's stable vital signs during the etomidate infusion. He was awake and conversant on arrival to GI PACU, and discharge-ready 20 minutes thereafter.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: We present a case in GI NORA where an ASA Class IV outpatient with severe ICM needed to complete screening colonoscopy to qualify for destination LVAD therapy. Our hospital is in the midst of a bed crunch with long wait times for admission to inpatient wards and ICUs. By selecting etomidate for use in an infusion monotherapy technique (loading bolus of 0.1-0.15 mg/kg followed by a maintenance infusion (range 20-60 mcg/kg/min)), we were able to provide excellent conditions for deep sedation while maintaining the patient's cardiorespiratory stability. Due to etomidate's favorable context sensitive half-time, our patient recovered promptly and was able to be discharged home in a timely fashion.

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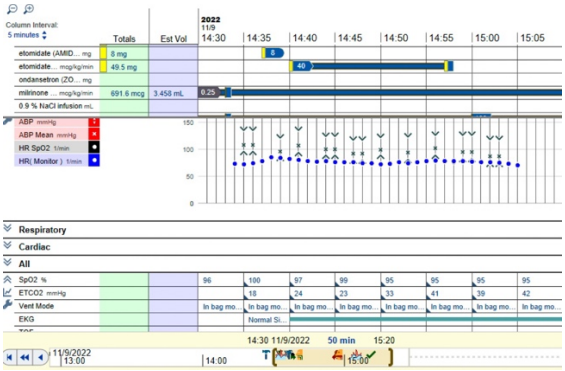

Anesthetic Pharmacology

Anesthetic Pharmacology 1 - Anesthetic considerations for patients with pheochromocytomas: clinical pearls

Tejas Kollu[1], Ashley Wicker[2], Arianna Cook[3], Emily Teeter

Albany Medical Center[1] University of North Carolina[2] The University of North Carolina Medical Center[3]

Introduction: Pheochromocytomas are rare neuroendocrine tumors that are typically found in the adrenal gland that secrete large amounts of catecholamines. These patients present with headaches, diaphoresis, hypertension, heart palpitations, and tachycardia. Management includes treatment of hypertension, surgical resection of the tumor, and possible chemotherapy for metastases. Pre-operative optimization is imperative due to the uncontrolled release of catecholamines that will occur during surgery. According to the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES), alpha-adrenergic receptor blockade should be considered in all patients prior to adrenalectomy to ensure less labile arterial blood pressure during surgical resection. Given the large hemodynamic shifts, intraoperative management of pheochromocytomas can be challenging.

Methods: Our patient is a 19-year-old male with a medical history of Von Hippel-Lindau disease with bilateral pheochromocytomas. On the day of surgery, a thorough preoperative evaluation was completed to assess his medication optimization. On exam, he was normotensive with a normal heart rate and rhythm; he had a reassuring airway exam. Prior to induction, an arterial line was placed for blood pressure monitoring due to the sympathetic surge that would occur with direct laryngoscopy. The patient subsequently underwent standard induction with fentanyl, propofol, and rocuronium. He was also preemptively given esmolol to blunt the stimulation of direct laryngoscopy.

The patient remained hemodynamically stable throughout induction, intubation, and monitor placement. Prior to surgical incision, the patient's anesthesia was deepened, and he was given a bolus of esmolol in anticipation of a catecholamine surge. We then slowly titrated clevidipine and esmolol infusions to stabilize his blood pressure and heart rate prior to manipulation of the tumor. Both of these medications were steadily up titrated once the tumor was manipulated. We chose to use these short-acting medications for easy titration before and after tumor resection. It is not uncommon for these patients to have refractory hypotension after the tumor is removed. Fortunately, our patient remained hemodynamically stable throughout the case without abrupt changes in his blood pressure. Clevidipine and esmolol were quickly titrated off following resection, and the patient did not require vasopressors.
He was successfully extubated at the end of the case, had an uneventful recovery, and was discharged home on postoperative day two.

Results: Not Applicable Medically Challenging Case Report

Conclusions: Patients with pheochromocytomas represent a unique hemodynamic challenge. Preoperatively, blood pressure control is imperative, with a target blood pressure of less than 130/80 mmHg per the Endocrine Society Clinical Practice Guidelines.[3] Antihypertensive medications should be initiated 14 days before surgery; surgery should be postponed if blood pressure control is not achieved. Alpha-adrenergic receptor blockers (such as terazosin or phenoxybenzamine) are the first-choice drug class to minimize perioperative complications. These drugs specifically block the overstimulation of α-adrenergic receptors by the high levels of circulating catecholamines, which can be released during surgery leading to hypertensive emergencies.

Intraoperatively, clevidipine was utilized for blood pressure control and esmolol for heart rate control. There are multiple vasoactive medications that can be used for blood pressure control intraoperatively, however, we believe clevidipine to be the superior choice. Clevidipine is a short-acting intravenous dihydropyridine calcium channel blocker that causes selective arterial vasodilation, leading to a predictable dose-dependent decrease in afterload and peripheral vascular resistance.[4] In these cases, it is important to use short-acting agents after resection of the pheochromocytoma because patients are susceptible to refractory hypotension due to the sudden withdrawal of catecholamines, reduced sympathetic tone, and decrease in arterial tone.[5] Clevidipine provides easy titration with precise hemodynamic control compared to other agents such as nitroprusside which non-selectively targets both arterial and venous smooth muscle and also has a side effect profile of cyanide toxicity, hypotension, and tachyphylaxis. As demonstrated in this patient case, clevidipine can be considered effective for use in patients with pheochromocytoma intraoperatively.

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Cardiovascular Anesthesiology

Cardiovascular Anesthesiology 1 - Iatrogenic Right Ventricular Rupture During Sternal Wound Debridement

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HCA Florida Kendall Hospital[1]

Introduction:
Post sternotomy sternal wound infection, also known as mediastinitis, is a disastrous and possibly life-threatening complication of cardiac surgery. Management of mediastinitis involves antibiotics and sometimes surgery for washout and drainage. Iatrogenic injury to the heart is an extremely rare occurrence that could happen during these procedures.

Methods:
A 40-year-old male with a past medical history of hypertension and type II diabetes mellitus presented to the hospital for purulent discharge from a sternal wound. The patient had undergone coronary artery bypass graft surgery (CABG) 6 weeks prior. On computed tomography (CT), retrosternal mediastinal fluid was located posterior to the sternum and xiphoid process, which abutted the pericardium. The plastic surgery service was consulted and recommended proceeding with the removal of implanted hardware, creation of bilateral pectoralis major muscle flap, and adjacent tissue rearrangement.

Upon debriding the inferior portion of the left hemi-sternum with the hydrosurgery tool, a laceration was created on the right ventricle. The surgeon placed towel clamps to control the bleeding. The patient was hemodynamically stable at this time. Cardiothoracic surgery service was then called. Once the cardiothoracic team arrived, the surgeons assessed the injury and determined the best approach is to have primary closure on cardiopulmonary bypass. Femoro-femoral cannulation was performed, and the patient was placed on cardiopulmonary bypass. A right-sided internal jugular cordis and a right-sided radial arterial line were placed. During the procedure, there was a noticeable amount of bleeding with an accompanying change in hemodynamics. The patient was then transfused with four units of packed red blood cells, two units of fresh frozen plasma, and one unit of plasma. The laceration was closed successfully and weaned off bypass. The plastic surgery team then continued on with their planned procedures. The patient was transferred to the critical care unit and continued IV antibiotics. He was extubated the next day and discharged thirteen days later.

Results: not applicable- medically challenging case report.

Conclusions: Mediastinitis is a complex and potentially life-threatening complication following median sternotomy in cardiac surgery. Rupture of the heart is a rare complication in the management of mediastinitis that requires immediate attention and timely management. Manual tamponading of the perforation is crucial. The anesthetic team should prepare by ensuring adequate access, invasive blood pressure monitoring, considering starting a massive transfusion protocol, and possibly preparing for cardiopulmonary bypass.

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Cardiovascular Anesthesiology 2 - Rare Iatrogenic Concomitant of Apical left ventricular Aneurysm and Ventricular Septal Defect

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Mayo Clinic[1]

Introduction: Concomitant left ventricular aneurysm (LVA) and ventricular septal defect (VSD) is rare. However, it has been described in case reports secondary to acute myocardial infarction[1,2]. We report a case of concomitant apical LVA and VSD as a late complication of ventricular tachycardia ablation.

Methods: A 66-year-old male presented for LVA and VSD surgical repair. He has a complicated past medical history includes chronic diastolic heart failure without coronary artery disease, cardiac arrest secondary to ventricular fibrillation (VF) requiring hypothermia therapy, ventricular tachycardia (VT) status post (s/p) automatic implantable cardioverter-defibrillator firing twice, valvular heart disease s/p robotic mitral valve annuloplasty and tricuspid valve band repair in 2015. Atrial fibrillation s/p ablation 2015, VF s/p ablation 2019.

During the VT ablation in 2019, he was found to have a small section of abnormality at the posterior papillary muscle towards the apex and significant disease adjacent to the mitral valve in the inferior lateral basilar segment. Both areas were ablated. After the ablation procedure, the patient felt well with no symptoms for a year.

A routine transthoracic echocardiogram (TTE) revealed an enlarged LV chamber with an ejection fraction of 45%. Basal inferolateral wall akinesis. An LV apical inferior septal pseudoaneurysm (1.6 cm x 1.4 cm) with associated apical VSD (7 millimeters (mm) width) (Figure 1). Color Doppler revealed a left-to-right shunt flow through the VSD (peak gradient 49 mmHg) (Figure 2). To better characterize the lesion, a computerized tomography cardiac angiogram (CTCA) with intravenous contrast was performed. It revealed a multilobulated pseudoaneurysm at the LV apex measuring 3.6 cm x 2.6 cm x 3.9 cm without thrombus. A 4 x 6 mm anteroseptal VSD was identified at the apex (Figure 3).

The patient proceeded with surgical intervention. Intraoperative transesophageal echocardiogram (TEE) revealed an LV apical pseudoaneurysm and an apical VSD with a left-to-right shunt on color doppler (Figure 4). Severe tricuspid regurgitation via perforated anterior leaflet (Figure 5).

After sternotomy, the surgeon had a direct view of the lesion. The patient was found to have perforated anterior leaflet and elongation of the free edge of the anterior leaflet due to lead coiling. A large apical LV defect and a smaller RV apex defect were observed through the pseudoaneurysm. Aneurysmectomy was performed. The VSD was repaired with a pericardial patch. Tricuspid valve anterior leaflet had plication and perforation repair. Post-by-pass TEE showed normal LV apex geometry. The patient was discharged on postoperation day 6. TTE before discharge showed the previously noted LV apical pseudoaneurysm, and VSD have been successfully repaired.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The combination presents of LVA and VSD are rare. To the best of our knowledge, this is the first reported case as a delayed complication of VT ablation. In terms of imaging, 2D TTE was the imaging modality of choice. However, the CTCA revealed more detailed anatomical information evidenced by identifying the anteroseptal location of the VSD. Therefore, multiple cardiac imaging modalities are essential to aid the appropriate course of surgical management.

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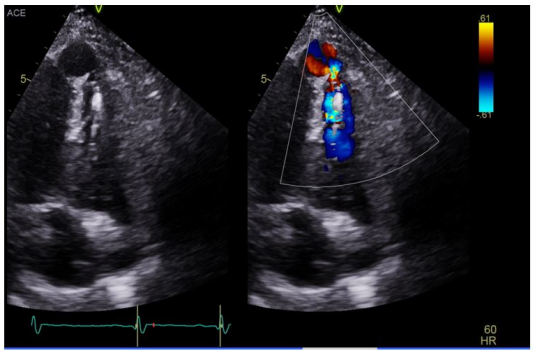

Figure 1: Transthoracic echocardiogram apical 4-chamber view revealed a left ventricular apical pseudoaneurysm (1.6 cm x 1.4 cm) with associated apical VSD (7 mm width).

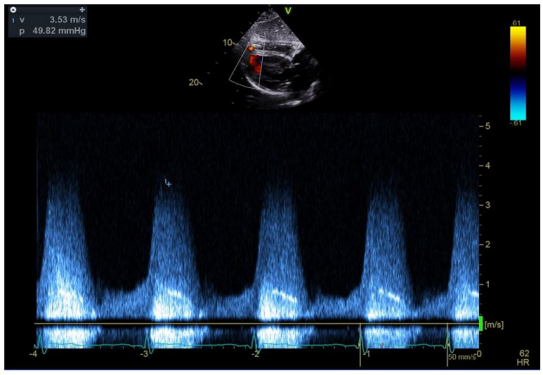

Figure 2: Transthoracic echocardiogram subcostal view revealed a left to right shunt with peak grad 49 mmHg.

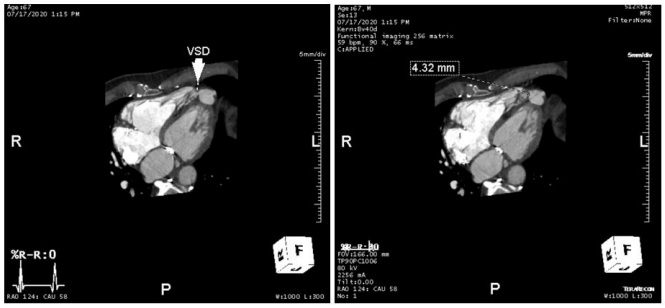

Figure 3: A computerized tomography cardiac angiogram with intravenous contrast showed a multilobulated pseudoaneurysm at the left ventricle apex measured 3.6 x 2.6 x 3.9 cm without thrombus, a 4 x 6 mm anteroseptal VSD was seen at the apex.

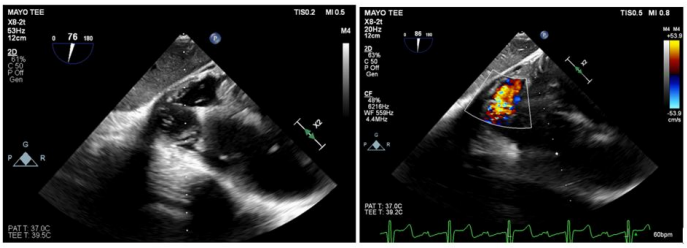

Figure 4: Intraoperative transesophageal echocardiogram showed LV apical pseudoaneurysm and an apical VSD with a left-to-right shunt on color doppler.

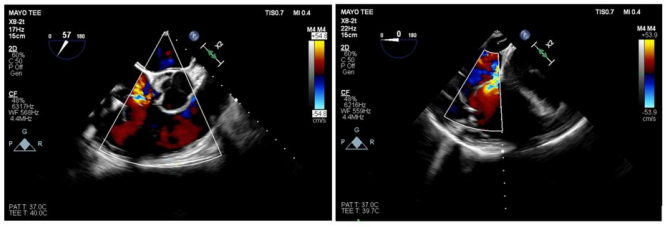

Figure 5: Intraoperative transesophageal echocardiogram showed severe tricuspid regurgitation via perforated anterior leaflet.

Cardiovascular Anesthesiology 3 - Accidental ECMO decannulation: Rapid Multidisciplinary Response

Tejas Kollu[1], Arianna Cook[2], Lavinia Kolarczyk

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Introduction: The ability to troubleshoot ECMO complications is imperative to providing safe perioperative management. Mechanical issues include pump thrombosis, failure of pump bearings, overheating of the circuit, and decannulation. Accidental decannulation can involve partial or complete removal of the access or return cannula leading to massive blood loss, hypoxemia, and hemodynamic collapse.[3,4] One of the main causes of accidental decannulation is excessive tension placed on the cannula, which can be prevented by anchoring and assigning a team member to ensure lines are free from entanglement.[4]

Methods: A 75 y.o. male with severe central cervical canal stenosis with significant myelopathy, hypertension, hyperlipidemia, prostate cancer, and DMII presented for elective C3-C6 laminectomy and fusion. After anesthetic induction, he became hypoxic and hypotensive requiring increasing vasopressor support. A rescue TEE revealed a significantly dilated right ventricle, severe TR, and a large thrombus in the proximal right PA. Cardiothoracic anesthesia, cardiothoracic surgery, and interventional cardiology decided to proceed with peripheral VA-ECMO as a bridge to mechanical thromboembolectomy given ongoing hemodynamic instability despite multiple vasopressor infusions.

After peripheral cannulation in the femoral artery and vein, the patient was taken to the cardiac catheterization lab where the interventional cardiology team performed pulmonary angiography prior to mechanical thromboembolectomy (Inari FlowTrieverÒ System). A large amount of clot was removed from the right and left PA. An attempt was made to place a left femoral distal reperfusion cannula, but his left superficial femoral artery (SFA) was injured requiring emergent vascular surgery intervention. While vascular surgery was attempting to repair the SFA, the left femoral artery ECMO cannula became dislodged during exposure with subsequent cardiac arrest. Sixteen minutes of CPR was performed. During this time, the anesthesiology team was running the code and managing the massive resuscitation effort while the surgeons worked to control the bleeding and insert a new femoral artery cannula. A distal reperfusion cannula was then successfully placed, and the patient was transported to the ICU. The patient's postoperative course was complicated by a large retroperitoneal hematoma, coagulopathy, respiratory failure, and delirium. Ultimately, the patient was weaned successfully from VA-ECMO support and suffered no neurologic sequelae.

Results: Medically Challenging Case Report

Conclusions: Although the incidence of accidental decannulation is low at 1.3%, it can have devastating consequences. ADDIN EN. CITE [5,6] Patient factors contributing to higher rates of accidental decannulation include poor access, obesity, and coagulopathies. If accidental decannulation occurs, there must be a coordinated response. These critical steps include emergent discontinuation of flow, clamping of lines or vessels both proximally and distally to the rupture site, calling for additional help (surgeon, perfusionist or ECMO consultant), and supporting the patient's circulation with high-quality ACLS. Rotating team members to avoid exhaustion from chest compressions is important, as it may take several minutes to re-establish ECMO support. In our patient, 16 minutes of CPR were required until successful arterial re-cannulization. In addition, massive blood loss (in the field and in the retroperitoneum) and subsequent profound coagulopathy further complicated the situation. The use of a massive transfusion device (i.e., BelmontÒ) and activation of the institution's massive transfusion protocol helped rapidly replace blood volume. As events like this are rare and potentially fatal, a multidisciplinary team-based simulation may play a role in training. The implementation of ECMO crisis management checklists to better equip our providers to handle ECMO challenges may also be beneficial. In these difficult and rare situations, the cardiothoracic anesthesiologist plays a vital role by coordinating multidisciplinary care for cannula reinsertion, managing severe hemodynamic instability, and mobilizing resources for massive transfusion as well as additional therapies.

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Cardiovascular Anesthesiology 4 - Atrial septostomy to vent the left heart in a VA ECMO patient

Muna Oli[1], Nikolaus Gravenstein[1], Clifford Deerman

University of Florida[1]

Introduction: Internationally, over 5,000 heart transplants (HTx) are done annually (1). Reasons for HTx are varied but are often associated with heart failure which can be due to structural or functional abnormalities that are either inherited or acquired (2). When a patient develops end stage heart failure that can no longer be treated with goal directed medical therapy, devices or surgical management, cardiac transplant is the gold standard for treatment (3). While waiting for an appropriate donor, there are techniques that can be used to support the heart ("bridge to transplant"). These include a ventricular assist device (VAD), intra-aortic balloon pump (IABP), and veno-arterial extracorporeal membrane oxygenation (VA ECMO).

The retrograde aortic flow from VA ECMO can cause increased LV/LA filling pressures, LV dilation, pulmonary congestion, and even respiratory failure (4). Techniques have been developed to "vent" a patient's heart to help offload the LV. They include Impella and IABP devices which actively unload the left heart, or it can be done passively via percutaneous balloon atrial septostomy (4,6). Atrial septostomy provides effective LV unloading by creating a popoff left to right shunt but can be technically challenging to do (5). The case presented here demonstrates a successful use of atrial septostomy in a decompensating patient needing VA ECMO as a bridge to HTx.

Methods: 26-year-old male with PMH anxiety and ADHD who presented to OSH with shortness of breath and was admitted for acute biventricular heart failure secondary to unknown cardiomyopathy (possibly viral) and cardiogenic shock. At OSH, LHC/RHC showed EF 10-15% globally reduced systolic function/elevated biventricular filling pressures with clean coronary arteries. Patient also had bilateral pulmonary edema and concern for pneumonia. Patient was transferred to UF Health for management of cardiogenic shock with newly diagnosed HFrEF/NICM concerning for viral etiology.

An IABP was placed for bridging treatment to heart transplant, however, the patient continued to decompensate and had persistent NSVT and was therefore urgently scheduled for placement of VA ECMO as salvage procedure. At the time of ECMO cannulation, atrial septostomy was also performed for LV unloading prior to ECMO. TEE was utilized throughout the case to help visualize the wire and then balloon and penetration through the interatrial septum. TEE demonstrated global hypokinesis, dilated ventricles and an EF of 10%. After septoplasty was performed, TEE was used to visualize the 1.5x1cm atrial septal defect tht had been created at the level of the fossa ovalis with continuous left-to-right flow. LAP estimates 26mmHg. The patient received OHTx about a month after admission to hospital and was discharged to rehab a week later in stable condition.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: This case is an excellent example of the importance of understanding the anatomy and physiology of heart failure, along with bridge therapies to transplant and the use of TEE in procedures such as a septostomy. Each type of bridge therapy and venting technique has pros and cons. In this patient, an IABP had been attempted but was inadequately effective, thus VA ECMO was a last resort. Given that he already had biventricular dilation from cardiomyopathy and pulmonary edema, it was important to preemptively vent the heart to minimize any further damage that could be caused by ECMO. Passive atrial venting has mostly been done in children, with very little data evaluating its efficacy in adults (5). An obvious downside to any of the transeptal techniques is the permanent ASD created in patients who may never undergo a HTx. Ultimately, it is important to carefully evaluate the patient to determine if they are an appropriate candidate for VA ECMO and potential venting by atrial septostomy depending on the patient's cardiac anatomy and physiology. TEE is essential in guiding the surgeon or cardiologist when creating the septostomy to verify flow and caliber as well as to help the proceduralist avoid hitting the aorta or any other critical structures and identifying pericardial tamponade should the left atrial free wall be perforated.

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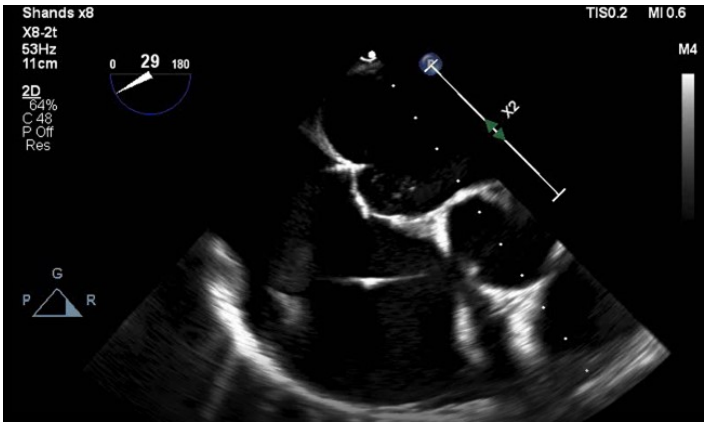
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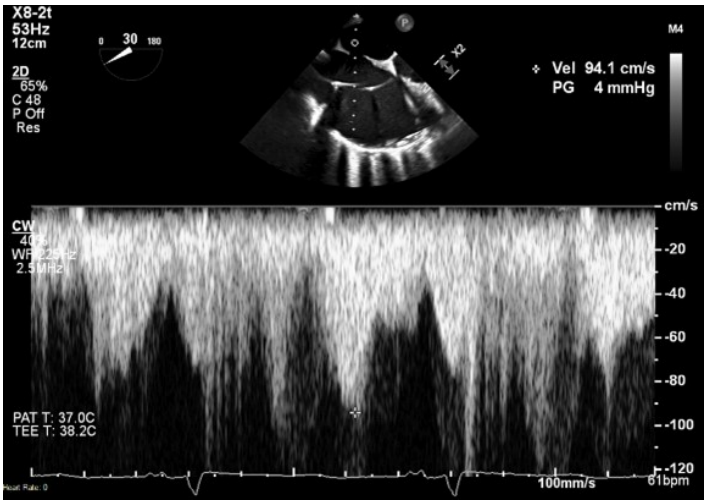
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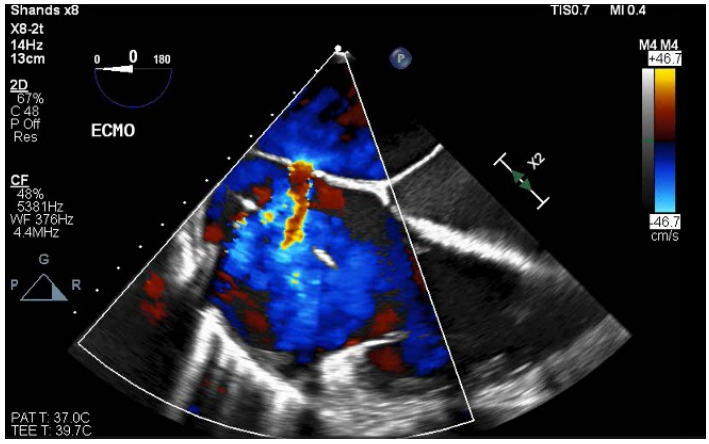
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Cardiovascular Anesthesiology 5 - Esophagopericardial Fistula: A Rare Complication of Atrial Fibrillation Ablation

Bonnie Yen[1], Kasmir Ramo, Andrew Greenwald[1], Allen Friedman[2], Teresa Mulaikal[2]

Columbia University Medical Center[1] Columbia University[2]

Introduction: Atrial fibrillation (AF) ablation is a treatment that utilizes radiofrequency, cryoablation, or laser energy to create lesions that disrupt the electrical pathways leading to dysrhythmia. Esophagopericardial fistula (EPF) formation is a rare but potentially lethal complication of this procedure, with reported incidence of 0.1% [1]. Here we present a case of EPF formation after AF ablation and its management.

Methods: A 68-year-old woman with hypertension, hyperlipidemia, and AF underwent radiofrequency ablation under general anesthesia with esophageal temperature monitoring. She was discharged in normal sinus rhythm on post-procedure day (PPD) 1 with pantoprazole. She developed heartburn the following week, and on PPD 13, she presented to the emergency department with severe chest pain, chills, and shortness of breath. Work-up revealed leukocytosis enlarged cardiac silhouette with pneumomediastinum on chest x-ray, and hydropneumopericardium on chest computed tomography (CT), raising high suspicion for EPF.
The patient was brought to the intensive care unit where radial arterial pressure monitoring showed exaggerated respiratory pulse pressure variation, and transthoracic echocardiography (TTE) revealed right atrial and ventricular compression. However, mean arterial pressure was maintained above 65 mmHg without vasopressor support. A repeat chest CT with oral contrast was performed, with no contrast extravasation beyond the esophagus. Because communication between the esophagus, pericardium, and heart could not be definitively excluded, the patient underwent emergent chest exploration.

In the operating room, a large-bore internal jugular catheter was placed for risk of large volume resuscitation and vasopressor requirement. She received 1.5L crystalloid, and low-dose norepinephrine and vasopressin were initiated before general anesthesia was induced with midazolam, fentanyl, and rocuronium. Endotracheal intubation was immediately followed by median sternotomy. Neither transesophageal echocardiography (TEE) nor pulmonary artery catheter was used due to risk of further injury. Surgeons opened a tense pericardium, releasing cloudy fluid. To better examine the left atrium and posterior pericardium, cardiopulmonary bypass was initiated. A 5mm defect was found in the posterior pericardium near the left inferior pulmonary vein, and upper endoscopy confirmed diagnosis of EPF. There was no injury to the left atrium. Pericardial and esophageal defects were repaired by primary closure.

The patient was weaned from vasopressor support, extubated on post-operative day (POD) 2, and discharged on POD 17. She completed a 2-week course of pantoprazole and metoclopramide, and she has since restarted a regular diet. TTE 9 weeks after surgery showed only trivial pericardial effusion, and an esophagram 10 weeks after surgery was negative for leaks.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Esophageal injury occurs in up to 18% of patients after AF ablation [1]. Although the exact mechanism of injury is unknown, proposed contributing factors include thermal injury to the esophageal wall and its arterial supply, gastroesophageal reflux, and damage to the vagal nerve plexus [2]. This case is an example of EPF formation after AF ablation, despite intra-procedural esophageal temperature monitoring and post-procedural use of a proton pump inhibitor. Challenges to the diagnosis of EPF include its rare occurrence and non-specific initial presentation [3]. Furthermore, while chest CT is an important component of evaluation, fistulous communication may not be definitively identified or excluded. High clinical suspicion and expeditious initiation of care aided in this patient's successful surgical intervention, highlighting the importance of vigilant monitoring and greater awareness for esophageal injury after AF ablation.

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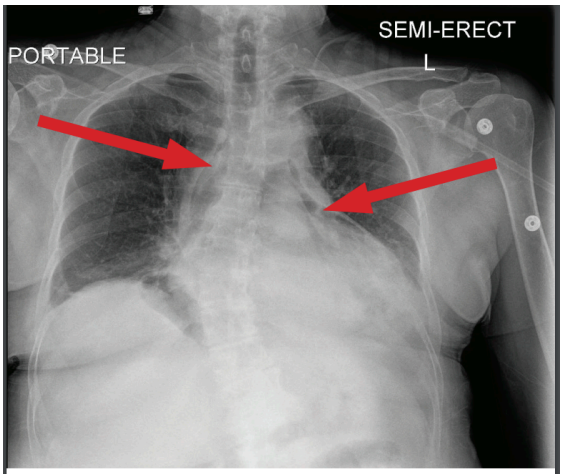
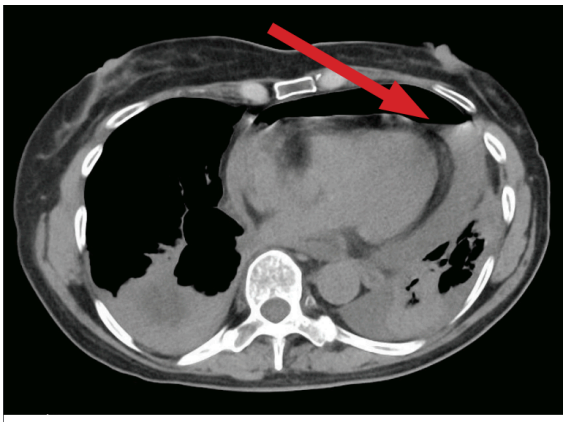


Chest X-Ray | Enlarged cardiac silhouette with pneumopericardium (red arrows)



Chest CT | Large hydropneumopericardium with dependent air-fluid level (red arrow)

Cardiovascular Anesthesiology 6 - Intraoperative Electrocardiograph Artifact Mimicking Ventricular Tachycardia During A Spine Surgery

Fei Wu[1], JP Wang[1], Shauna Williams

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Introduction: Ventricular tachycardia (VT) is a dangerous form of arrhythmia that often warrants immediate treatment. We report here a case of "intraoperative VT" as a result of electrocardiograph (EKG) artifacts during a spine surgery. Differentiating EKG artifacts from real VT can be challenging for clinicians. Awareness to rule out artifacts is important to avoid unnecessary interventions, especially in the acute care setting of the operating room for anesthesia providers.

Methods: A 66-year-old female with chronic neck pain, type 2 diabetes mellitus, sleep apnea, obesity presented for C3-C6 laminoplasty. She had no issues with her prior anesthetics. Her preoperative lab tests, including a complete blood count, basic metabolic panel, coagulation panel were unremarkable. A preoperative EKG shows normal sinus rhythm, with minor nonspecific ST-T change (Fig.1.a).

She had family history of sudden deaths of unknown reason at young ages (all<50, one<35) in second-degree maternal relatives. On day of surgery, preoperative evaluations, including physical examination and telemetry tracing at the preop bay showed no abnormalities. ASA standard monitor including a 5-lead EKG was applied before general anesthesia was induced with 150mg of propofol, 70mg of rocuronium and 100mcg of fentanyl, and endotracheal intubation was completed uneventfully. Anesthesia was maintained with sevoflurane at 0.9MAC. Phenylephrine drip was started at rate of 20-50mcg/min to maintain a mean arterial pressure of 75mmHg.

Patient was positioned prone with head placed in the Mayfield pins. About 70min after incision, abnormal EKG tracing was noted on the monitor, at that time displaying lead II and V1. It was noted to be a wide complex tachycardia with a rate of around 200 beats per minute, full tracing of which was later obtained from the control desk (Fig.1.c and d). Patient was hemodynamically stable and maintained good oxygen saturation during this episode. This rhythm lasted for about 30 seconds before it reverted to baseline normal sinus rhythm with a rate of 60. Patient tolerated the remainder of the procedure with no issues and was brought back to PACU for recovery. Blood work such as CBC, BMP, magnesium and phosphorous were obtained, and all within normal limits. A formal 12 lead EKG was obtained which showed normal sinus rhythm with nonspecific ST change, no significant change from prior (Fig.1.b). A Cardiology consult was called for "intraoperative ventricular tachycardia", and after evaluation deemed the intraoperative tracing as artifact with normal underlying sinus rhythm. The patient had an uneventful recovery and was discharged home on postoperative day 2.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Current surface EKG tracing is susceptible to various sources of artifacts, particularly in the operating room. Artifacts mimicking VT can be differentiated from real VT by evaluating tracings from all available EKG leads and looking for hidden normal QRS, as well as unchanged hemodynamics. Artifacts should be ruled out before actual treatment towards intraoperative arrhythmia is carried out, especially if the patient is hemodynamically stable.

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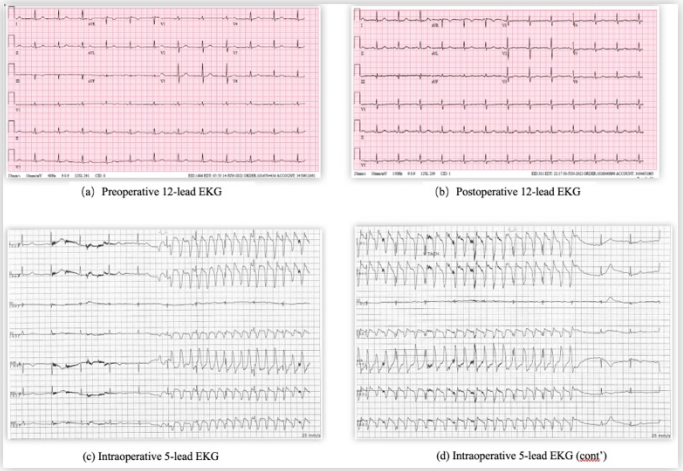

Cardiovascular Anesthesiology 7 - Management of Intramural Hematoma in Patients with Marfan Syndrome

Tejas Kollu[1], Nathan Leone[2], Jarrett Davis, Christopher Ma[3]

Albany Medical Center[1] Emory University[2] Emory University School of Medicine[3]

Introduction: Marfan syndrome (MFS) is an autosomal dominant connective tissue disorder that affects the microfibrils and elastin in connective tissues throughout the body with a reported incidence of 1 in 3000 to 5000 individuals.[1] MFS is associated with pathology of multiple organ systems and can include but is not limited to mitral valve prolapse, aortic disease, pectus excavatam and lens subluxation.

The incidence of aortic dissection in MFS syndrome patients is roughly 10% and is the primary cause of morbidity and mortality in up to 60% to 80% of patients.[2] Patients with MFS present a unique perioperative challenge to the anesthesiologist. We describe a case where the presence of MFS changed the aortic surgery plan and required anesthetic considerations to manage moderate hypothermic circulatory arrest (MHCA) and the use of TEE in diagnosing complications.

Our patient is a 29-year-old female with MFS, polysubstance abuse, and chronic DeBakey type 3B dissection who presented with chest pain and shortness of breath. She was found to have a penetrating aortic ulcer in the proximal descending aorta and aneurysmal degeneration of her thoracoabdominal aorta (7.8 cm) with compression of the heart, pulmonary veins, and left main bronchus resulting in post-obstructive pneumonia and hydrothorax. On imaging, dissection extended to her celiac trunk, mesenteric arteries, and left renal artery. Her preoperative echocardiogram showed a left ventricular ejection fraction of 30%, normal right ventricular function, no aortic valve pathology, and a 4.9 cm ascending aorta. She presented to us for open thoracoabdominal aortic aneurysm (TAAA) repair.

Methods: General anesthesia was induced, and the patient was intubated with a double lumen tube uneventfully. Central venous access, arterial line, and lumbar drain were all obtained in standard fashion. Transesophageal echocardiography (TEE) and neuromonitoring (motor and sensory) were used for the case. The patient underwent an open Extent II TAAA repair with intercostal, celiac, SMA, and bilateral renal artery reimplantation. Due to the poor quality of tissue in the proximal descending aorta, a crossclamp was not utilized and the proximal anastomosis was accomplished with cardiopulmonary bypass (CPB) and moderate hypothermic circulatory arrest (MHCA).

Following the repair, CPB was weaned uneventfully, and coagulopathy was corrected with allogenic blood transfusion. The patient was hemodynamically stable on minimal vasoactive medications, and all anastomotic sites were hemostatic. On TEE, biventricular function remained baseline, but a new circumferential intramural hematoma (IMH) extending from mid ascending to the distal aortic arch had developed. The decision was made to maintain stringent blood pressure control in order to maintain spinal cord perfusion but limit extension or evolution of the IMH. The patient's thoracotomy and abdominal incisions were closed, and she was brought to the ICU hemodynamically stable.

Postoperatively, the patient returned to the operating room on postoperative day (POD) 1 for concern of retroperitoneal bleeding and renal compression. All anastomoses were again verified as hemostatic with good flow to the celiac, SMA, bilateral renal, and reimplanted intercostal arteries on doppler. The patient was extubated POD 2 and ultimately discharged POD 17.

Results: Medically Challenging Case Report

Conclusions: Aortic complications in MFS patients receiving aortic surgery are high. TAAA repairs may require MHCA or DHCA depending upon arch involvement as well as tissue quality. The use of MHCA or DHCA has different implications in anesthetic management and morbidity (ie, higher incidence of stroke, coagulopathy, etc.) and should be considered depending on the surgical plan. Intraoperative TEE can be useful in diagnosing new aortic pathology such as intramural hematomas and helping monitor or expedite treatment paths for high-risk patients with connective tissue disease.

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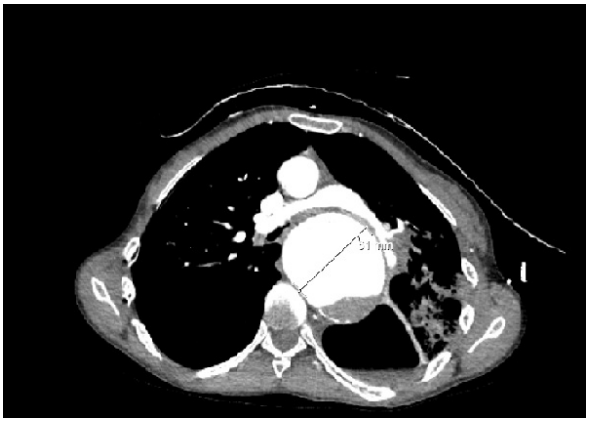

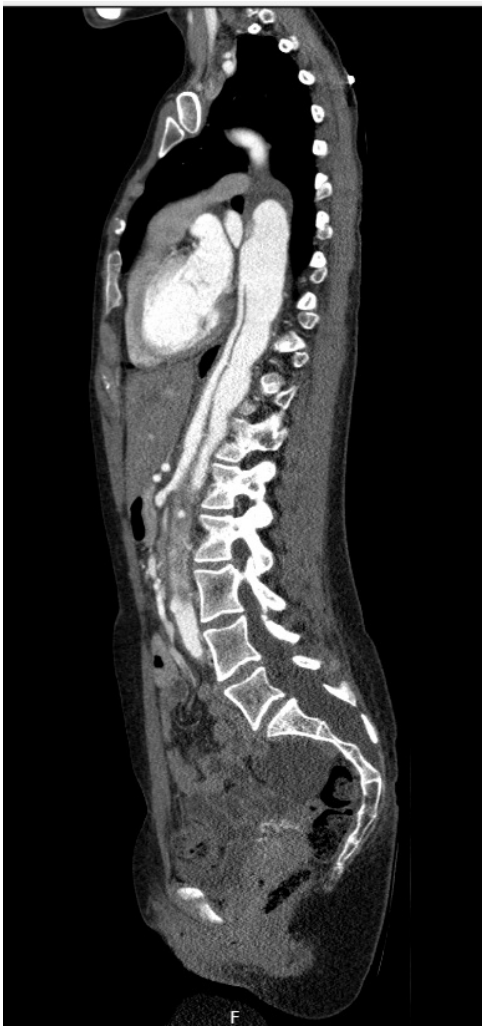

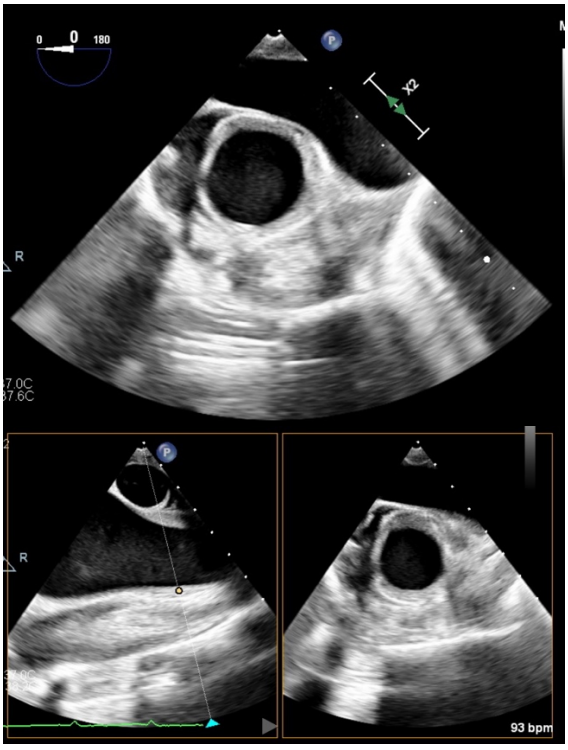
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Cardiovascular Anesthesiology 8 - Norepinephrine-secreting paraganglioma in a patient with cyanotic congenital heart disease post Fontan procedure with severe mitral regurgitation

Tara Ramaswamy[1], Fred Mihm, Ethan Jackson[2]

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Introduction: Pheochromocytomas/paragangliomas are catecholamine-secreting tumors with potentially lethal cardiac complications including arrythmias, cardiomyopathy, and catecholamine-myocarditis. Pathologic catecholamine secretion observed in these tumors can be particularly deleterious for Fontan patients[1-3].

Methods: A 29-year-old woman with a history of pulmonary atresia with intact ventricular septum (PA/IVS), staged univentricular lateral Fontan repair, Fontan-associated liver disease (FALD), chronic hypoxemia due to venovenous collaterals, chronic diastolic LV failure, and moderate-to-severe mitral regurgitation (MR) presented with a symptomatic norepinephrine-secreting paraganglioma. In a multidisciplinary preoperative meeting, the high-risk features of her anesthesia and surgery were discussed, including hypercarbia-related exacerbation of pulmonary vascular resistance (PVR), decreased venous return with a laparoscopic surgical approach, risk of malignant hypertensive crisis from endogenous norepinephrine (NE) release during tumor dissection, anticipatory vasoactive management to stabilize intraoperative rhythm, MR, PVR, and systemic vascular resistance (SVR), and vasoplegic crisis post-resection.

After treatment with phenoxybenzamine for two weeks there was near resolution of her presenting symptoms of dyspnea, palpitations, and near-syncope. Preoperatively, bilateral erector spinae plane (ESP) blocks were placed to avoid risk of bleeding and hypotension with a neuraxial technique. She underwent general anesthesia for open paraganglioma removal and left nephrectomy due to tumor involvement of the renal hilum. Transesophageal echocardiography (TEE) early in the case demonstrated improvement in MR from moderate to mild likely in setting of phenoxybenzamine and general anesthesia. Her intraoperative course was notable for hypertensive swings during tumor manipulation and post-resection profound mixed vasodilatory and cardiogenic shock requiring hemodynamic support with infusions of epinephrine, vasopressin, angiotensin II, and inhaled nitric oxide. Resuscitation included 2 liters of balanced crystalloid and 3 units packed red blood cells. Fontan pressures remained stable around 14 mmHg. The central venous oxygen saturation ($ScvO_2$) measured in the Fontan conduit remained stable between 68-83% except for a transient drop to 59% during an episode of bradycardia. She was extubated at the end of surgery to high-flow nasal cannula with in-line iNO. She received excellent pain control with the ESP catheters. Vasopressors and iNO were weaned throughout the following 6 postoperative days. She was discharged home on postoperative day 24.

Results: Not Applicable -- Medically Challenging Case Report.

Conclusions: A multidisciplinary approach is key in perioperative planning for pheochromocytoma resection in Fontan patients. This case highlights significant preoperative challenges including titration of alpha blockade and balancing the consequences of an open versus laparoscopic approach. Critical intraoperative issues included integrating Fontan pressures, TEE, and $ScvO_2$ to titrate vasoactive drugs and monitor SVR, PVR, and MR. Postoperative issues included providing adequate analgesia and optimizing fluid management given her nephrectomy and known heart failure.

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Figure 1: Left periaortic mass, at the level of the left renal artery, measuring 5.7 x 3.6 x 4.1 cm with max SUV 14.70

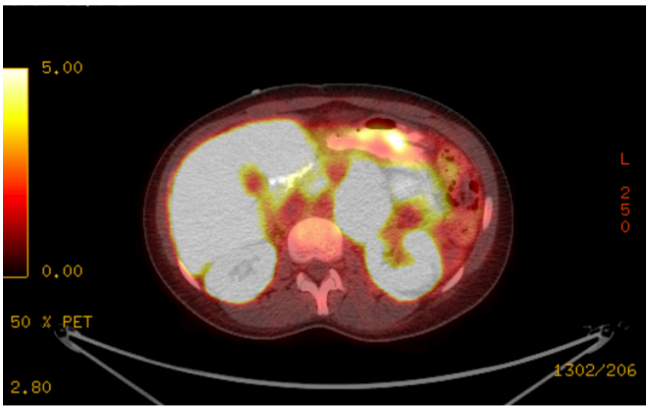


Figure 2: Preoperative TTE (still image)

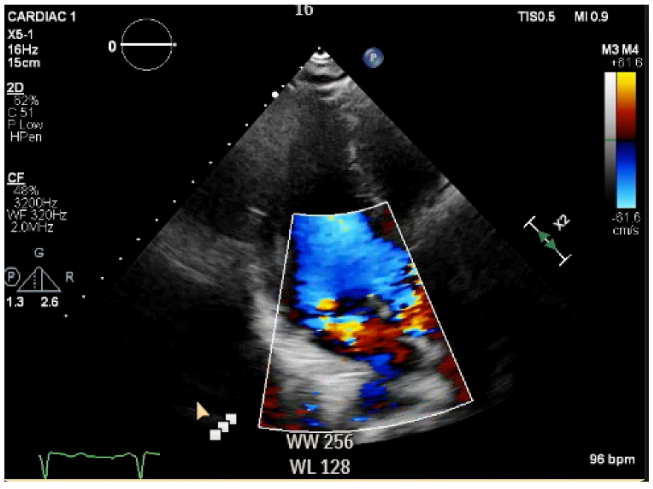


Figure 3: Surgical specimen

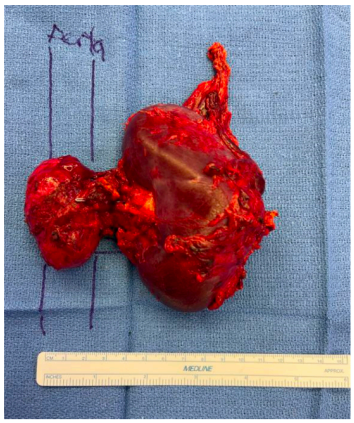
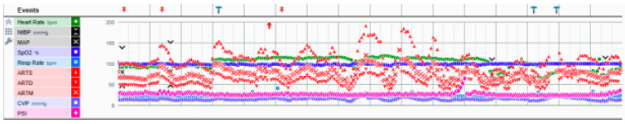


Figure 4: Portion of intraoperative record



Cardiovascular Anesthesiology 9 - Patient with sickle cell disease with complex congenital heart disease undergoing cardiopulmonary bypass: needle in the haystack!

Dr. Suhasinee Deokar, Dr. Kamlesh Tailor, Dr. Suresh Rao
Kokilaben Dhirubhai Ambani Hospital and MRC, Mumbai.

SUHASINEE DEOKAR[1]

Kokilaben Bhirubhai Ambani Hospital and MRC[1]

Introduction: Sickle cell disease is a rare genetic disorder. Birth prevalence of 115 cases per 100000 live births. Pathophysiology: replacement of valine for glutamic acid @ 6th position in chain due to point mutation in the gene which code it. Peri operative management is complicated due to increased propensity for sickling and further challenged by cardiopulmonary bypass (CPB).

Methods: 4.6 years boy weighing 11kg height 94 cm (BSA 0.5m2). Primary Diagnosis: Double outlet right ventricle with large subaortic VSD, S/P VSD Closure with subaortic membrane resection, diagnosed case of sickle cell disease. Clinical presentation: Dyspnea on exertion NYHA II/III.

Detailed echocardiography:

1. severe mitral inflow obstruction due to supra mitral ring, with small mitral annulus with single papillary muscle
2. Severe mitral stenosis with pressure gradient of 40/22. Moderate Tricuspid regurgitation
3. Severe pulmonary hypertension

Patient was planned for redo surgery for supra mitral ring excision as attempt for balloon mitral valvotomy was unsuccessful. CPB triggers profound sickling due to inflammatory cascade, hypothermia, hypoperfusion and acidosis which would lead to vaso-occulusive crisis. Exchange transfusion was done to avoid sickling considering HBS > 40% TIMING OF EXHNAGE TRANSFUSION. Preoperative exchange transfusion posing significant risk:

1. As in severe mitral inflow obstruction any volume shift is detrimental.
2. Being a redo case, it would be extremely difficult to initiate CPB if catastrophic changes in hemodynamics occur while doing exchange transfusion preoperatively or after induction of anesthesia but before CPB.
3. It was not advisable to expose the patient to the risk of general anesthesia only for preoperative exchange transfusion.

Results: After multidisciplinary team meeting decision was taken to perform intraoperative exchange transfusion, just before the initiation of CPB, with aortic and venous canulae in situ in quick and controlled fashion. Our Experience of Exchange Transfusion

- Removal of patients' blood volume gradually which was containing ample amount of HBS from venous canulae, and equal volume was replaced by oxygenated and pre washed prime fluid via aortic canulae.
- Prime fluid contained of packed red cells units (8U), fresh frozen plasm(3U), 5% Albumin 250 ML,100 ML Mannitol, Plasmalyte and adequate quantity of buffers.
- Patient had hypotension during the procedure which was taken care of by temporarily halting the procedure and gradual replacing volume through in situ aortic canulae till haemodynamics stabilized.
- Serial monitoring of HBS levels during the cardiopulmonary bypass dropped to 7.1% then 6.5% from baseline of 49.9%.
- Modification of circuit for exchange transfusion was done with large capacity venous reservoir, oxygenator with hemofilter and set gentle occlusion for pump.
  Optimization on Cardiopulmonary Bypass technique
  Mild hypothermia
  Avoided hypoperfusion, hypoxia and acidosis
  Continuous ultra filtration.
- We used Near Infrared Spectroscopy for continuous monitoring of cerebral regional perfusion, especially when patient had hypotension while performing exchange transfusion.

Conclusions: In patient with sickle cell anemia with HBS > 40 though exchange transfusion is mandatory it can safely differ in preoperative period and can be done intra operatively in quick and controlled fashion. Hypoxia, acidosis, and hypothermia are identified factors provoking sickling and hence must be avoided. Serial checking for HBS levels at multiple stages is essential. Patients family must be counselled for the risk associated with the surgery done under cardiopulmonary bypass with sickle cell disease.

Cardiovascular Anesthesiology 10 - Perioperative implications of rate drop response of pacemakers: Don't drop the ball!

Roopvir Kaur[1], Steven Kapeles

Medical college of Wisconsin[1]

Introduction: More than 200,000 people in the United States receive permanent pacemaker (PPM) implantation every year.[1] Advancements in PPM modalities have further expanded their use for a wide variety of disorders, including rate drop response (RDR) pacing to treat neurally mediated syncope. [2,3] This report presents a unique case of intraoperative hemodynamic collapse from RDR pacing in a patient with concomitant aortic stenosis (AS) and the subsequent management.

Methods: A 65-year-old man with a history of sick sinus syndrome and vasovagal syncope status-post dual chamber PPM implantation and moderate AS presented for robotic laparoscopic nephrectomy for renal cell carcinoma. On preoperative PPM interrogation, normal device function and baseline non-paced, sinus rhythm was confirmed. No changes to the PPM were made preoperatively. Following intraoperative abdominal insufflatio n, the patient became bradycardic with subsequent asystole. Reflex heart rate pacing to 100 BPM was seen on telemetry accompanied by loss of EtCO2, unmeasurable blood pressure cuff reading, and pulselessness (Figure 1). The abdomen was desufflated, and IV fluids and vasopressor administered resulting in hemodynamic stabilization. An arterial line and magnet over the PPM were also placed. On repeat insufflation attempt, a similar episode of bradycardia with RDR pacing and hemodynamic instability was observed leading to the decision to abort the surgery. PPM interrogation postoperatively confirmed multiple RDR events around the time of insufflation. Postoperative echocardiogram and regadenoson stress test were unchanged from previous exams. PPM reprogrammed from MVP_AAI_DDD to DDD at a lower rate with a longer AV delay and RDR was disabled before second attempt at surgery. During repeat attempt at nephrectomy, the patient was pretreated with atropine, phenylephrine, and IV fluids prior to abdominal insufflation resulting in uneventful intraoperative course.

Results: Not applicable - Medically Challenging Case Report

Conclusions: Rate drop response is an advanced pacemaker function that performs reflexive pacing in response to sensed bradycardia and is typically used to treat vasovagal syncope. Anesthetic agents, abdominal insufflation, surgical manipulation, and hyper vagotonia were all possible contributors to this patient's intraoperative bradycardia, and RDR pacing was poorly tolerated secondary to his known aortic stenosis, hypovolemia, and autonomically mediated hypotension. While unique patient and surgical factors require individual consideration, PPMRDR functions should preferably be suspended in the perioperative period to avoid undesired hemodynamic turbulence. Disabling RDR function with magnet application is less reliable and not always possible due to surgical positioning and device settings. A clear understanding of PPM indications and functions, including RDR, is essential for safe anesthetic management in patients with these devices.

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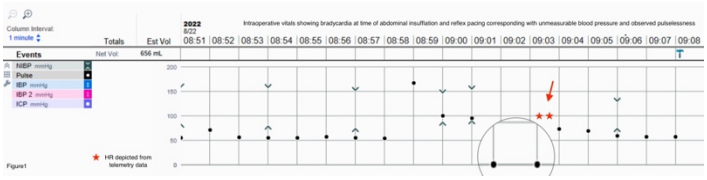

Cardiovascular Anesthesiology 11 - Successful Transjugular MitraClip™ Procedure in a Patient with an Intra-Aortic Balloon Pump

Genevieve Manahan[1], Connor O'Brien[2], Richard Cheng

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

Introduction: Transcatheter edge to edge mitral valve repair with the Abbott MitraClip™ system has become an increasingly popular treatment alternative for patients with severe primary mitral regurgitation but at high risk for open repair, as well as for symptomatic patients with severe mitral regurgitation and heart failure with reduced ejection fraction despite maximal goal directed medical therapy. The transcatheter clip mechanism is based on the surgical Alfieri stitch and essentially clips the anterior leaflet to the posterior leaflet. This creates a double orifice and a reduction of mitral regurgitation severity. As of 2019, the MitraClip™ procedure has been performed in over 100,000 patients, primarily through a transfemoral approach.[1] The EVEREST II trial, a randomized comparison of percutaneous mitral valve repair versus conventional surgery, showed that while the percutaneous repair group had a smaller reduction in mitral regurgitation severity, it had a significantly better safety profile with similar clinical outcomes.[2] Presence of a venous thrombus in the inferior vena cava or femoral veins has historically been a contraindication to MitraClip™ due to the inability to deploy the device. Alternative deployment sites such as the transjugular or transhepatic approach are rare due to increased technical challenges, but may be a lifesaving alternative for patients with hemodynamically significant severe mitral regurgitation.[3] We present a case of a successful transjugular MitraClip™ procedure in a patient presenting with cardiogenic shock requiring intra-aortic balloon pump support.

Methods: A 57-year-old male with a history of mechanical aortic valve replacement for aortic stenosis, biventricular heart failure (LVEF 15%), severe tricuspid regurgitation, and severe mitral regurgitation presented in cardiogenic shock requiring urgent IABP placement. The patient was not a candidate for LVAD or heart transplant due to severe myopathy and cachexia. The IABP was unable to be weaned in the setting of his severe mitral regurgitation, and he was at high risk for open mitral valve repair with a calculated STS risk of mortality of 21.46%. A TEE evaluation of his mitral valve showed severe functional mitral regurgitation with systolic flow reversal in the pulmonary vein, a regurgitant volume and regurgitant fraction of 125mL and 74%, and an effective regurgitant orifice area of 0.9 cm^2 (by PISA). The regurgitant jet was predominantly between A2/P2, and thus a percutaneous edge to edge mitral repair was attempted to facilitate IABP wean. However, procedural fluoroscopy revealed that the patient had a completely occluded IVC with numerous venous collaterals from the distal IVC to proximal IVC, and thus the procedure was aborted. A second procedure was attempted via the transjugular approach, with a standard Baylis transseptal sheath, and two XTW MitraClips™ were deployed, reducing his mitral regurgitation from severe to trace-to-mild. The IABP was subsequently successfully weaned and removed, and the patient was discharged home in stable condition.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The transjugular approach for percutaneous mitral repair has been documented previously in only a handful of case reports but can be a successful alternative for patients with IVC or femoral vein disease. Additional technical considerations starting with procedural positioning must be planned as unlike the femoral approach, interventionalists must stand above the patient's right shoulder with adequate table support for the MitraClip™ delivery system. The anesthesiologist and echocardiographer often have limited access to the patient's head. Other specific considerations include potential for carotid vessel injury during cannulation, venous air embolism, and possible post-operative neck hematoma with inadequate hemostasis.[4] We have no complications to report for this case, nor have there been complications reported for the case reports previously describing this technique; however, larger scale studies will need to be performed in order to gather stronger evidence to promote this approach as a viable alternative for the general population.

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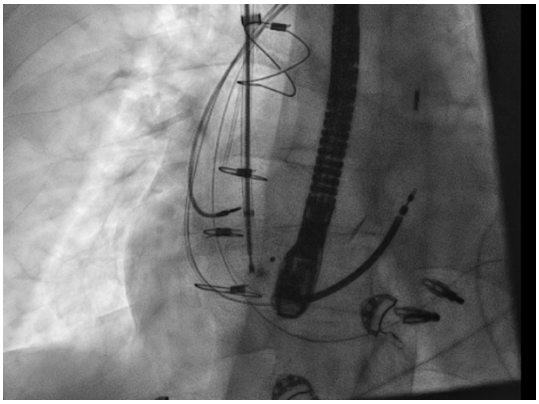

Cardiovascular Anesthesiology 12 - To ECMO or Not to ECMO? That is the question...

Caron Hong[1], Jaclyn Fleener[2], Siraj Meah[3], Sarah Ren, Lucas Martin[2], Keki Balsara[4]

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Introduction: What are the criteria for extracorporeal membrane oxygenation (ECMO)? In institutions that care for high acuity patients with resources to manage ECMO, it has been a growing option for survival. Conditions that have been associated with successful outcomes with ECMO include respiratory failure s/p lung transplant or ARDS and cardiac failure s/p primary cardiomyopathy, s/p heart/lung transplant, cardiogenic shock, pulmonary embolism, acutely after in-hospital cardiac arrest and s/p cardiac surgery with failure to wean from cardiopulmonary bypass. This medically challenging case describes a patient with severe tracheal stenosis with a diameter of less than 5 millimeters. Further the stenotic region was posterior to the sternum and there was high concern for inability to successfully perform an emergency/awake tracheostomy. Although this patient did not meet the traditional indications for ECMO with favorable outcome, her severe tracheal stenosis, and the position of stenosis with high likelihood of unsuccessful intubation and/or emergent/awake tracheostomy, the decision was made for ECMO support to secure an airway.

Methods: This is a 25-year-old female who presented to the emergency department with shortness of breath and stridor. After evaluation for pulmonary embolism and CTA of the chest, the patient was found to have severe tracheal stenosis measuring less than 5 millimeters posterior to the sternum and an enlarged thyroid. Vital signs were stable other than an SpO2 88-90 with audible stridor. Discussion with the anesthesiology and surgical teams resulted in concern for unsuccessful intubation and/or likelihood of unsuccessful awake/emergent tracheostomy secondary to severity of tracheal stenosis and site of narrowest stenotic region. Decision was to canulate the patient for ECMO prior to induction and surgical tracheostomy. On arrival to the operating room, the patient had stable vital signs with an SpO2 > 90% and audible stridor in the upright position. The patient was successfully cannulated for ECMO under local anesthesia. Patient was then induced and a single attempt, unsuccessful, was made to place a 4.0 ETT. Successful bag mask, resulting in adequate chest rise and tidal volume, resulted in the decision to place an LMA (Size 4) for the procedure. Ventilation during ECMO allows for static pulmonary circulation to receive some ventilation/oxygenation support as well as reducing atelectasis. A 7.0 Portex tracheostomy tube was placed between the third and fourth tracheal rings under general anesthesia and ECMO. The patient was decannulated from ECMO and the sites were closed immediately post procedure. The patient was weaned off the ventilator in the operating room to spontaneous ventilation and placed on oxygen mask. The patient was brought to PACU in good spirits with no post-operative complications. The patient was discharged on post-op day 4.

Conclusions: Assessing each case for immediate or pending heart and/or lung failure and the possible utility of ECMO could be lifesaving. In this case, under these circumstances, we were able to successfully save this 25-year-old patient.

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Cardiovascular Anesthesiology 13 - Undiagnosed Pheochromocytoma Resulting in Peripartum Acute Biventricular Heart Failure Requiring Veno-Arteriovenous Extracorporeal Membrane Oxygenation

Ryan Dodge[1], Catherine Jerman[1], Jeffrey Yu, Juan Ripoll[1], Jerome Crowley[1], Aranya Bagchi

Massachusetts General Hospital[1]

Introduction: Pheochromocytoma is a rare vasoactive neuroendocrine tumor characterized by episodic hypertension and tachyarrhythmias secondary to catecholamine secretion. Here, we describe the management of a pregnant patient who underwent emergent cesarean section (c-section) for uncontrolled hypertension and fetal bradycardia complicated by cardiac arrest requiring veno-arteriovenous extracorporeal membrane oxygenation (VAV-ECMO).

Methods: A 37-year-old woman with gestational hypertension and suspected neurofibromatosis type 1 (NF1) at 37 weeks gestation presented to labor and delivery with abdominal pain, nausea, and vomiting. Vital signs were notable for a heart rate of 129 and blood pressure of 183/122, and antihypertensive therapy was initiated for presumed pre-eclampsia. Due to concerns for fetal instability, the patient underwent emergent c-section under general anesthesia. Her intraoperative course was complicated by refractory hypoxemia and hemodynamic instability, and post-operatively she developed wide complex tachycardia that degenerated into PEA arrest. Following a brief round of ACLS with return of spontaneous circulation, she was emergently cannulated for VAV-ECMO with Impella placement due to ongoing refractory hypotension and hypoxia with acute biventricular heart failure.

Following a grossly negative cardiac workup, repeat echocardiography showed progressive improvement in cardiac function and the patient was decannulated from ECMO on post-operative day (POD) 4. Notably, during this time, the patient remained hypertensive despite adequate sedation and high dose vasodilator infusions, prompting a secondary workup with abdominal CT revealing a 7.3cm R adrenal mass. The diagnosis of pheochromocytoma was confirmed by elevated serum and urine metanephrines. Alpha blockade was initiated with doxazosin and clonidine, followed by beta blockade with metoprolol in anticipation of tumor resection.

Results: Pheochromocytomas are rare neuroendocrine tumors of the adrenal medulla that require thoughtful anesthetic preparation and perioperative optimization to mitigate life-threatening cardiovascular complications. Catecholamines secreted by these tumors produce episodic hypertension and tachycardia, leading to the classic symptom triad of diaphoresis, palpitations, and headache. Up to 40% of pheochromocytomas are associated with known genetic syndromes, including NF1 and MEN2B. The most commonly identified genes are the RET proto-oncogene, NF-1 gene, VHL gene, and genes encoding succinate dehydrogenase subunits. The diagnosis is established by measuring plasma and/or urinary fractionated metanephrines and catecholamine concentrations. Treatment usually involves tumor resection, with careful optimization of hemodynamic stability and euvolemia through sequential alpha and beta blockade.

Managing pheochromocytomas in obstetric patients presents a unique challenge due to the complex differential diagnosis of hypertensive disorders of pregnancy, the adjusted range of normal vital signs, and the temporal component of active labor requiring prompt intervention. Treatment varies based on symptomatology and gestational age, though successful surgical management has been described in all three trimesters. A well-developed multidisciplinary plan is key to the safe and appropriate management of these patients.

Though pheochromocytoma rarely presents with fulminant heart failure and even more rarely requires ECMO support, both have been described in peri-partum and non-pregnant populations. In our case, VAV ECMO was utilized for the management of hypoxemia and coexisting heart failure. VAV ECMO is a hybrid ECMO configuration that returns oxygenated blood to both the arterial and venous circulation following gas exchange through the oxygenator. This configuration avoids the ejection of deoxygenated blood by a recovering heart into the proximal arterial circulation. VAV ECMO is most useful in cases such as ours with concomitant pulmonary and cardiac dysfunction in a heart that is expected to regain inotropic ability prior to pulmonary recovery.

Conclusions: Pheochromocytoma is a rare condition that can result in acute heart failure requiring advanced cardiac life support. Clinicians should maintain a very high degree of suspicion in patients presenting with atypical, paroxysmal vital sign abnormalities. Thoughtful care should be given when providing advanced mechanical circulatory support.

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Critical Care

Critical Care 1 - Anesthetic Considerations for Supra-Systemic Pulmonary Artery Pressures

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Western University of Health Sciences[1] University of Utah Medical Center[2] University of Southern California, Keck School of Medicine[3] University of Utah[4]

Introduction: Pulmonary hypertension is defined as a mean pulmonary artery pressure of >25mmhg. This condition has an incidence of approximately 0.7% of patients undergoing noncardiac surgery and is of critical importance in the perioperative period (1,2). Undiagnosed pulmonary hypertension can dramatically increase perioperative morbidity and mortality and is by itself an independent risk factor for major adverse cardiovascular events. This condition is commonly divided into five distinct groups. The most common groups encountered by anesthesiologists in the operating room are groups 2 and 3 due to left sided heart disease and lung disease respectively. We describe a 30-year-old patient with group 1 pulmonary hypertension on maximal medication therapy who presented for bilateral orthotopic lung transplantation.

Methods: A 30-year-old patient with a past medical history of group 1 pulmonary hypertension presented for double lung transplantation. Preoperatively her right ventricular systolic pressure was estimated with transthoracic echocardiography by tricuspid regurgitation as 169mmHg. She was severely symptomatic with near syncopal events occurring multiple times throughout the day including when she laughed. She had been followed by her pulmonary team and was on "triple therapy" with macitentan 10 mg daily (an endothelium receptor antagonist), tadalafil 40 mg daily (a PDE5 inhibitor), and IV treprostinil (a synthetic analog of prostacyclin). She was also taking furosemide, aldactone, metolazone and diltiazem. After awake central venous access induction was completed with significant hemodynamic support with epinephrine and inhaled epoprostenol. The procedure was completed utilizing ECMO. The patient tolerated the procedure well and was able to be extubated a few hours after the completion of surgery.

Conclusions: Our case highlights the extreme intracardiac pressures that can be found in patients with pulmonary hypertension. Patients with pulmonary hypertension are at an elevated risk of right ventricular ischemia and dysfunction. Therefore, careful monitoring and induction strategies are critical to maintaining cardiac perfusion. Utilizing a combination of epinephrine and pulmonary vasodilators such as epoprostenol, has been shown to be an effective strategy to induce anesthesia in patients with acute and chronic pulmonary hypertension (3). Understanding the underlying physiologic principles of pulmonary hypertension can help guide successful management and intraoperative care for these patients during complex procedures.

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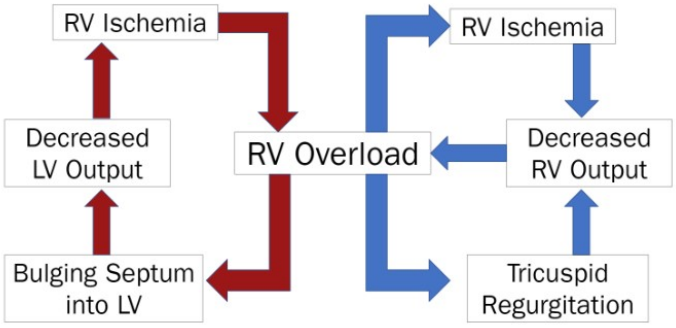

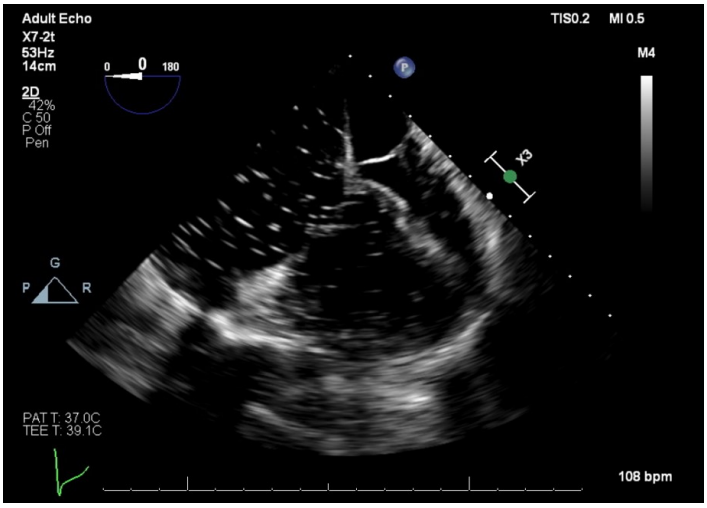

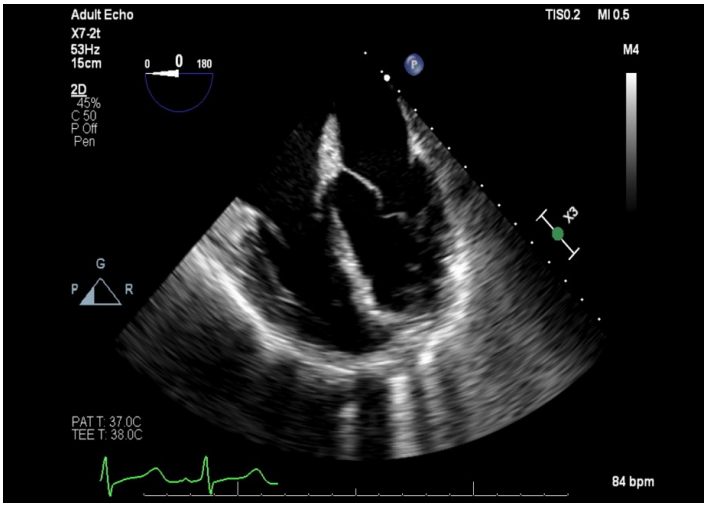

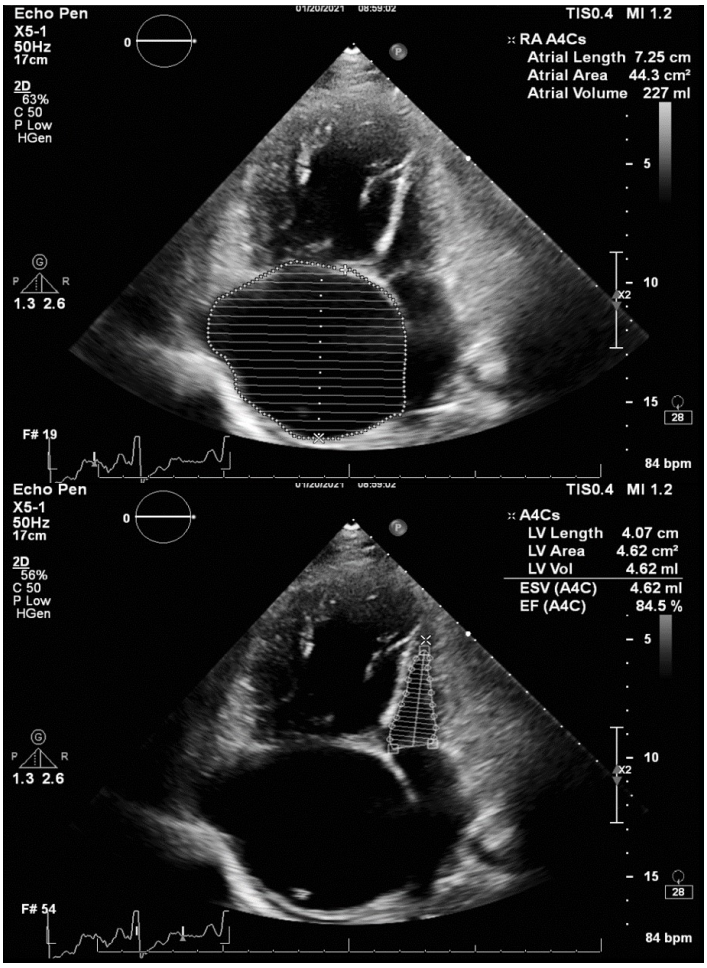
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Atrial Area 44.3 cm²
Atrial Volume 227 ml
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F# 19
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Echo Pen
X5-1
50Hz
17cm
2D
56%
C 50
P Low
HGen
TIS0.4 MI 1.2
:: A4Cs
LV Length 4.07 cm
LV Area 4.62 cm²
LV Vol 4.62 ml
ESV (A4C) 4.62 ml
EF (A4C) 84.5 %
1.3 2.6
F# 54
84 bpm

Critical Care 2 - Angiotensin II induced hypertension for management of refractory cerebral vasospasm following subarachnoid hemorrhage

Kelsei Keene[1], Chiamaka Amanchukwu[1], John Bryant, Christopher Hughes[1]

Vanderbilt University Medical Center[1]

Introduction: Cerebral vasospasm is a transient narrowing of cerebral vessels that causes decreased cerebral blood flow following subarachnoid hemorrhage (SAH), often leading to delayed cerebral ischemia (DCI) and poor outcomes after SAH. The American Heart Association Stroke Counsel recommends induced hypertension to improve cerebral perfusion as Class I level B evidence for the management of DCI with blood pressure titration to symptom resolution.[1] We report the successful use of Angiotensin II – a potent vasoconstrictor used in vasodilatory shock – to induce hypertension for the management of cerebral vasospasm refractory to conventional vasopressors.

Methods: A 37-year-old female with past medical history significant for hypertension and tobacco use presented with a two-day history of headache and neck pain following an unwitnessed fall. She also endorsed episodes of emesis, fatigue, fevers, and chills. Imaging was notable for a Hunt Hess 2, modified Fischer grade 4 subarachnoid hemorrhage in the setting of a ruptured 4mm aneurysm at the junction of the anterior communicating artery and the right A2 segment. The patient was admitted to the neurologic intensive care unit for strict blood pressure goals and serial neurologic examinations. She underwent urgent aneurysm embolization. Three days later, the patient had an episode of acute onset aphasia and right sided weakness. Imaging was concerning for vasospasm and induced hypertension was started with a systolic blood pressure (SBP) goal of >180 mmHg achieved with a norepinephrine infusion. The patient was taken to the operating room for intra-arterial verapamil (IAV), however, her neurologic exam worsened prompting increased SBP goal to ≥200 mmHg. While induced hypertension did improve her symptoms, norepinephrine, epinephrine, and vasopressin infusions, as well as corticosteroids, were required to meet this goal. Ongoing neurologic decline prompted repeat IAV as well as external ventricular drain (EVD) placement. The patient continued to have altered mental status, and SBP goals were increased to 240-260mmHg. An angiotensin II infusion was added to maintain this supranormal blood pressure goal and allowed weaning of epinephrine and vasopressin infusions. While there was some improvement in her neurologic exam, her neurologic lability ultimately led to intubation and multiple IAV procedures. She slowly progressed and all vasoactive medications and steroids were weaned over the next week. She was eventually discharged to a skilled nursing facility with tracheostomy and percutaneous gastrostomy tube.

Results: Not Applicable– Medically Challenging Case Report

Conclusions: Induced hypertension is often used when clinical signs of DCI present after SAH. Angiotensin II can be considered as an alternative to patients who are not responding to escalating doses of conventional vasopressors. While goals should still be titrated to symptoms, we present an example of a patient with a favorable outcome after treatment with angiotensin II for symptomatic cerebral vasospasm.

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Critical Care 3 - Aortic Dissection in the setting of HOCM - A Case Report

Michael Hollar[1], Hannah Bowe, Vikas Kumar[2], Vaibhav Bora, Robert Rice[3]

Augusta University, Medical College of Georgia[1] Augusta University[2] Augusta University Medical Center[3]

Introduction: In a type A aortic dissection, both pre and post operative medical management is critical. After symptom onset, mortality rate increases by 1 to 2 percent per hour and complications include cardiac tamponade from hemopericardium, aortic regurgitation, stroke, aortic rupture, myocardial infarction. Medical management centers around anti-impulse therapy: decreasing both the heart rate (HR) and afterload to reduce the velocity of left ventricular contraction and reduce shear stress on aorta. Beta blockers are typically used first and if needed an arterial vasodilator is added. As in the treatment of aortic dissection, lowering HR is one of the treatment goals in managing hypertrophic cardiomyopathy (HOCM); however, with different physiologic purpose. In a dissection the goal is to decrease shearing force; in HOCM this allows for an increased filling time which opens the left ventricle and prevents outflow track obstruction. In contrast to the treatment of a dissection, treatment of HOCM is dependent upon increased preload and afterload with avoidance of vasodilators. When managing both conditions in one patient, beta blockers should be maximized, and invasive monitoring can provide additional data to help with preload and afterload management.

Methods: A 55-year-old F with medical history significant for cerebral aneurysm s/p stent and VP shunt, seizure disorder, AAA s/p EVAR, and CAD s/p STEMI presented with chest pain and numbness/tingling sensation of bilateral lower extremities. CT showed an extensive Type A aortic dissection with extension to bilateral subclavian, innominate, and bilateral common carotid arteries. There was distal extension to the infrarenal aortoiliac stent graft causing occlusion. The celiac artery and SMA were supplied by the false lumen (with distal flow) and the renal arteries were supplied by the true lumen. A 6.2cm infrarenal AAA was also present with concerns of retrograde perfusion from the false lumen. The patient initially presented to a hospital without CT surgery, making medical management more critical. Patient was started on esmolol and nitroprusside, transferred, and immediately underwent emergent aortic repair upon arrival to our hospital. The TEE obtained in the OR showed a preserved ejection fraction and no involvement of the aortic root; however, an incidental finding of severe LVH was noted. Post operative patients were transferred to the ICU and managed by anesthesia critical care. Invasive hemodynamic monitoring with an arterial line and swan ganz catheter was in place to allow for tight blood pressure control using easily titratable IV drugs. On post op day one, CVP was noted to be 15. Bedside TTE on POD 2 showed EF 65-70%, severe basal septum hypertrophy, SAM, grade II diastolic dysfunction, and a LVOT gradient of 30mmhg. Advanced HF team consulted secondary to concern for HOCM in the setting of aortic dissection. On POD 17, patient underwent a left axillary to bifemoral bypass and was discharged home on POD 20.

Results: Not Applicable- Medically Challenging Case Report

Conclusions: Initially, her BP was controlled using a nicardipine infusion and transitioned to PO drugs after extubation. For anti-impulse control, patient was put on metoprolol which was titrated up to a max dose. Once the TTE was obtained, it became apparent that nicardipine should be discontinued as it is a pure afterload reducer (arterial vasodilator) which is contraindicated in HOCM. Patient was able to be weaned from the nicardipine quickly as metoprolol was up titrated. Eventually, verapamil was added which is the calcium channel blocker of choice in HOCM, since it is a non-dihydropyridine CCB, it does not drop afterload as the dihydropyridine class does and is relatively selective for the myocardium causing negative inotropy. Fluid balance in this patient was maintained by spot dosing Lasix with a goal fluid balance of net even and a CVP of 10-12 while in the ICU. Invasive monitoring was maintained throughout the ICU course. This allowed not only for closer monitoring of HR and BP but also allowed for monitoring of CVP to prevent over diuresis leading to worsening outflow obstruction and to prevent under diuresis which can lead to pulmonary edema.

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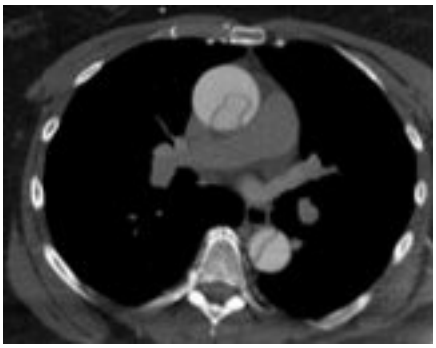

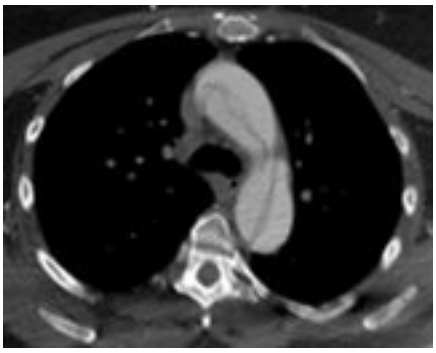

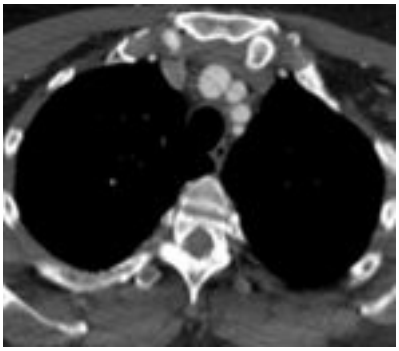

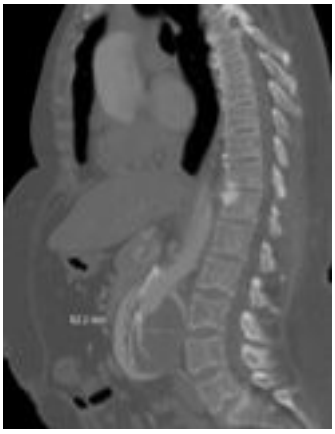

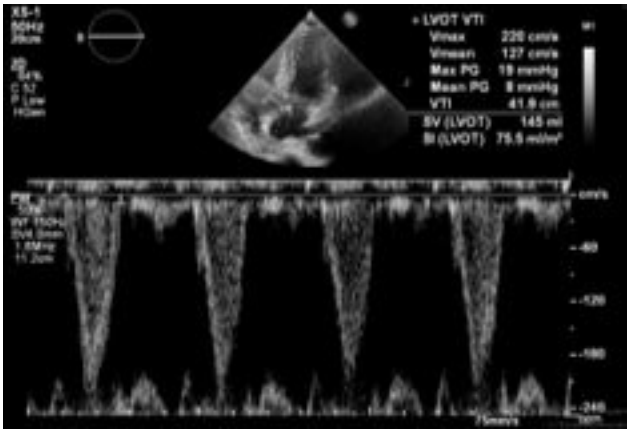
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Vmax 220 cm/s
Vmean 127 cm/s
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-60
-120
-180
-240

Critical Care 4 - Cardiogenic Shock in a Patient with Blunt and Penetrating Trauma: Blast Effect After Abdominal Gunshot Wound

Jonas Harley[1], Jenna Littmann[1], Toni Manougian[1]

Westchester Medical Center[1]

Introduction: This case describes an unusual presentation for blunt cardiac injury (BCI) from a gunshot wound and blast injury leading to cardiogenic shock. Blunt and penetrating cardiac injuries are seen in less than 10% of traumatic admissions. Most BCIs occur in the setting of motor vehicle accidents, pedestrians struck, motorcycle accidents, and falls from a substantial height - due to blunt force to the chest. In BCI, cardiac complications typically result from blood or fluid within the pericardial space with an intact pericardium. In our medically challenging case, the patient did not have an intact pericardium. Under direct visualization in the operating room, there was also no evidence of myocardial injury during the initial surgical repair of injuries.

In one review, pericardial tears were the most common cause of BCI on autopsy. When the mechanism of injury is suspicious for BCI, electrocardiogram, cardiac troponin levels, and echocardiographic evaluation is important. Access to bedside of point of care ultrasound (transthoracic and transesophageal) is increasing and especially useful. The six mechanisms for BCI are direct, indirect, bidirectional, deceleration, blast, crush, concussive, or combination. Further, the clinical presentation can vary between clinically silent injuries, transient arrhythmias, and fatal cardiac rupture.

Methods: The patient is a middle-aged male, and his only known history is for opioid use disorder and depression. He was found at home with a self-inflicted gunshot wound to his abdomen. The initial exploratory laparotomy found significant and extensive injuries to the large and small intestines, spleen, stomach, diaphragm, left kidney, and pericardium. The cardiac apex was directly visualized with the inferior aspect of the pericardium shredded and open. The heart itself was found to be completely intact without evidence of injury, and no blood was noted in the pericardial space. Twelve hours postoperatively, the patient was hemodynamically unstable, prompting ultrasound evaluation for cardiogenic shock. TTE showed global hypokinesia and inotropic support began with epinephrine. A Swan-Ganz catheter was placed. The patient was emergently taken to the OR for evaluation of any intra-abdominal cause of his decompensation, and the initial surgical repair was found to be intact without complications. Trans-diaphragmatic echocardiogram was performed intraoperatively through the surgical opening which clearly showed once again global hypokinesia that was improving with inotropic support. Initial troponins upon arrival at the emergency department were at 3 and peaked at 41. Two days later epinephrine was transitioned to dobutamine for pure inotropic support, which was eventually weaned off one day later. The patient has since made a significant recovery and is pending inpatient psychiatric care.

Results: Not applicable - Medically challenging case report

Conclusions: Significant research has been conducted for understanding causes of cardiogenic shock and its correlation with blunt cardiac injuries, and in our case report we find the development of shock without direct myocardial injury. This leads us to assume careful consideration of all trauma that could indirectly affect cardiac function. High suspicion of cardiac complications must be considered when there is trauma to the abdomen and chest with prompt use of echocardiography to confirm the diagnosis, as it is imperative for improved morbidity and mortality of the patient.

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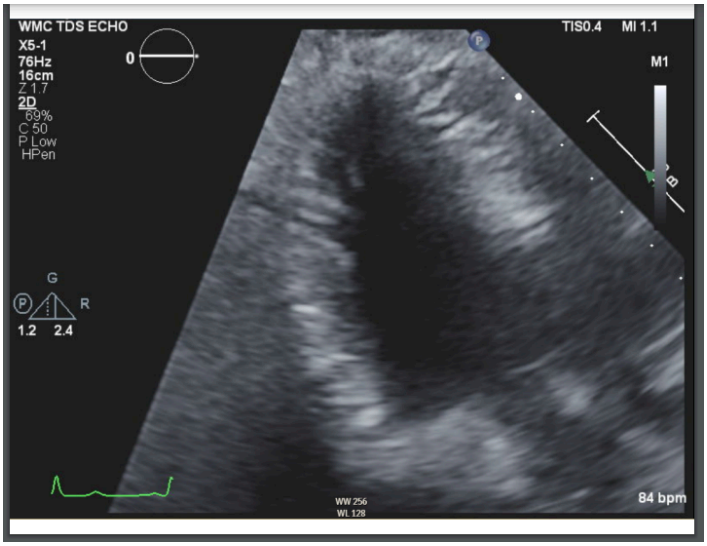

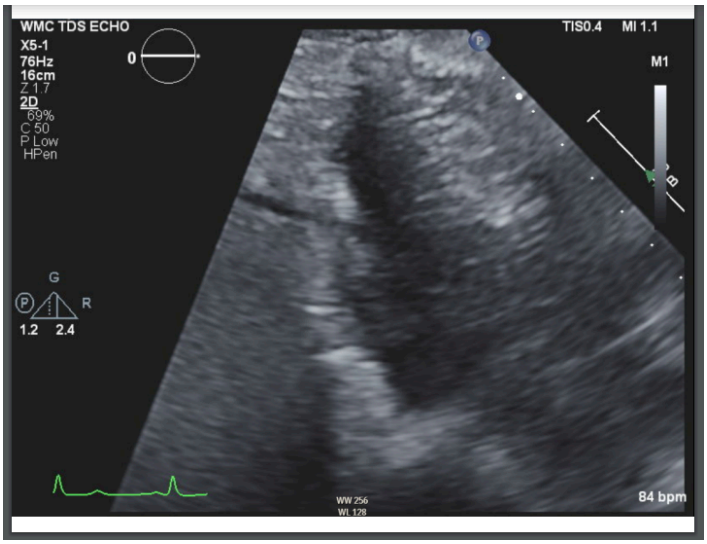
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1.2 2.4
WW 256
WL 128
84 bpm

Critical Care 5 - CATCH 22: DRESS Syndrome vs Aortic Pseudoaneurysm

Teuta Marsic[1], Brett Wakefield

Cleveland Clinic[1]

Introduction: DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms) syndrome is a rare, life-threatening drug reaction with many clinical features. With a mortality rate of up to 10%, this becomes further aggravated if co-existing with a secondary urgent surgical condition. We present a case of DRESS syndrome complicating an acute aortic pseudoaneurysm after cardiac surgery.

Methods: A 44-year-old male with Marfan's syndrome presented with severe paravalvular aortic regurgitation. His surgical history included a type A dissection s/p multiple repairs. He underwent a third sternotomy, for which he recovered uneventfully and was discharged home.

Two weeks post-operatively, he presented to the ED with clinical signs of sepsis and a CT showing an enlarging collection around the ascending aorta. He was started on broad-spectrum antibiotics, vancomycin and piperacillin-tazobactam, and taken to the OR for immediate mediastinal washout and debridement. Blood and mediastinal cultures were positive for methicillin-susceptible staph aureus (MSSA), with sensitivities guiding antibiotic de-escalation to oxacillin.

Three weeks into his hospitalization, he developed acute kidney injury, facial edema, and a morbilliform rash involving the face, neck, chest, upper arms, and abdomen developed. He had worsening agitation and hemodynamically instability, ultimately suffering a PEA arrest. Transthoracic echocardiogram (TTE) was unremarkable for a cardiogenic source. He was ultimately treated for distributive shock with high suspicion of anaphylaxis. Once stabilized, a repeat CT scan demonstrated a periaortic hematoma suggestive of an aortic root pseudoaneurysm.

Due to the morbilliform rash, worsening renal function, eosinophilia, fever and significant hemodynamic instability, DRESS syndrome was diagnosed with antibiotics deemed the culprit. High dose steroids were initiated and surgical repair of the pseudoaneurysm was delayed due to critical illness.

Results: Not applicable - Medically Challenging Case Report

Conclusions: DRESS has an estimated incidence of 1 in 100 to 1 in 10,000 drug exposures. It is a life-threatening syndrome that manifests 2-6 weeks after the triggering drug and typically includes a skin eruption, fever, eosinophilia, and multiorgan involvement. Prompt withdrawal of the drug is required. Treatment may involve immunosuppressive agents. In this case, an acute aortic pseudoaneurysm developed. A risk-benefit analysis is necessary when multiple life-threatening pathologies co-exist. Surgery was delayed due to the critical nature of DRESS, multiorgan involvement, and high-dose steroids. Impulse control was instituted while the patient recovered from this extensive systemic disease.

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Critical Care 6 - CMV Reactivation in Patients with COVID-19 on VV-ECMO: A Case Series

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The Ohio State University[1] The Ohio State University Wexner Medical[2]

Introduction: Reactivation of latent herpesviruses in immunocompetent, critically ill patients has a prevalence of 15-45% (1). Specifically, cytomegalovirus (CMV) is associated with severe pneumonia evolving towards acute respiratory distress syndrome (ARDS) with excess morbidity and mortality. However, clinicians do not routinely investigate CMV in previously immunocompetent, critically ill patients.

In patients with coronavirus disease 2019 (COVID-19), reactivation of herpesviruses has been reported. Proposed mechanisms include coronavirus-induced immunosuppression of T-cells that stimulates the lysogenic component of the herpes viral life cycle, and the use of immunomodulatory drugs, such as steroids or tocilizumab (1-3). However, it remains controversial if CMV is pathological or just a marker of severe illness since extrapolation of data from previous studies is confounded by the immune dysregulation caused by both COVID-19 and its treatments. Therefore, treatment of reactivated viremia remains debated regarding what viral levels warrant therapy, optimal dosing regimens for patients on veno-venous extracorporeal membrane oxygenation (VV-ECMO), and the need for prophylaxis (4,5).

Here, a case series of CMV reactivation is presented in patients with COVID-19 supported with VV-ECMO discovered during lung transplantation evaluation. The purpose of this case series is to further consider the prognostic impact and treatment strategies of CMV reactivation in this patient population.

Methods:

1. A 43-year-old male with hypertension presented with a severe cough and was found to have a positive COVID-19 polymerase chain reaction (PCR) assay, hypoxic hypercapnia, and bilateral pulmonary infiltrates. The patient developed ARDS requiring intubation, mechanical ventilation, treatment with 10 days of steroids, and VV-ECMO. When evaluated for lung transplantation, a positive CMV viral load of 1266 UI/mL by PCR was detected. The patient was treated for seven days with ganciclovir 5mg/kg, then switched to valganciclovir 450mg Q12H once tolerating PO until the viral load was undetectable. Thereafter, the patient continued to require ECMO support and ultimately had a successful double lung transplantation.

2. A 40-year-old male with asthma presented with shortness of breath and was found to have a positive COVID-19 PCR assay, hypoxic respiratory failure, and bilateral multifocal opacities. The patient required intubation, mechanical ventilation, treatment with 10 days of steroids, one dose of tocilizumab, and VV-ECMO. The patient was then found to have a positive CMV viral load of 10570 UI/mL when undergoing lung transplant evaluation. The patient was treated with ganciclovir 5mg/kg for a total course of fifteen days until levels were undetectable. Unfortunately, the patient expired due to multi-organ failure.

3. A 42-year-old female with migraines and polycystic ovarian syndrome presented with shortness of breath two months post-partum. Initial assessment revealed a positive COVID-19 PCR assay, hypoxia, and multifocal pneumonia. The patient evolved with ARDS requiring intubation and mechanical ventilation. Initial treatment included dexamethasone that was converted to hydrocortisone due to ongoing shock. The patient also received tocilizumab and required VV-ECMO. Lung transplantation evaluation revealed a positive CMV viral load of 610 UI/mL by PCR. The patient was not treated but had weekly viral monitoring to ensure resolution and was eventually discharged to rehab.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Management of COVID-19 patients on VV-ECMO with CMV reactivation is challenging, complex, and contentious. Multiple studies have investigated if suppressive therapy for CMV improves outcomes, but with varied results. While there is a trend in the literature to treat patients with positive CMV viremia defined as greater than 1000 copies/mL as done in this series, the primary outcome of decreased mortality has not reached consistent statistical significance (6). When considering CMV treatment or prophylaxis, any potential benefit to be gained must be weighed against potential drug toxicity. For ganciclovir and valganciclovir, this includes severe pancytopenia and decreased creatine clearance. Further investigations regarding CMV viremia in COVID-19 patients on VV-ECMO are needed to determine when treatment is appropriate and if potential benefits from early prophylaxis for patients being considered for a lung transplant outweigh the risks.

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Critical Care 7 - Competing Interests: Pulmonary Veno-Occlusive Disease and Right Ventricular Failure Following Septal Myectomy

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Introduction: Hypertrophic obstructive cardiomyopathy (HOCM) can have significant hemodynamic implications due to ischemia, arrhythmia, and valvular pathology.[1] Although HOCM's effects on the left ventricle are often cited, it can greatly impact the pulmonary venous circulation and the right ventricle (RV). Right ventricular hypertrophy (RVH) is associated with increased arrhythmias and worsening heart failure.[2] Pulmonary veno-occlusive disease (PVOD) has also been described in a subset of patients with HOCM.[3] This combination of conditions poses an exquisite challenge, particularly in the perioperative period. Here we discuss the management of a case of septal myectomy complicated by acute RV failure and PVOD.

Methods: A 41-year-old female with HOCM complicated by atrial fibrillation and ventricular tachycardia presented for septal myectomy. Echocardiography showed normal systolic biventricular function, severe left ventricular hypertrophy (LVH), RVH, diastolic dysfunction, and moderate mitral regurgitation with systolic anterior motion (SAM). Induction and initiation of bypass was uneventful. 5.8 g of muscle was resected. Echocardiography then illustrated mild RV dysfunction but no SAM; the rest of the exam was unchanged. Separation from the bypass was uneventful. She was transferred to the ICU.

She was extubated on postoperative day (POD) 0. Soon after, her cardiac index decreased, central venous pressures elevated, and pulmonary artery pressures (PAP) became suprasystemic. Epinephrine, norepinephrine, vasopressin, and inhaled epoprostenol (iEpo) were started. She was reintubated on POD 1 for respiratory failure. Despite this escalation in inotropic and respiratory support, point-of-care ultrasound suggested worsening cardiogenic shock and RV failure. Veno-arterial extracorporeal membrane oxygenation (VA ECMO) was deployed with hemodynamic improvement. After a standard weaning protocol, VA ECMO was decannulated on POD 5. Due to persistently high PAPs, inhaled nitric oxide was added to iEpo. Oxygenation and ventilation again deteriorated despite a trial of paralysis; on POD 9, veno-venous (VV) ECMO was cannulated as treatment of acute respiratory distress syndrome (ARDS). Unfortunately, protective lung ventilation was difficult to achieve while delivering inhaled pulmonary vasodilators for RV support. ECMO was reconfigured to veno-arterial-venous. Renal replacement therapy (RRT) was started for volume overload.

At this time, PVOD exacerbated by inhaled pulmonary vasodilators was suspected to have caused her hypoxia. This was further evidenced by absence of any identifiable etiology of ARDS. After pulmonary vasodilator discontinuation and aggressive volume removal, ECMO was de-escalated to VV and then weaned off days later. The rest of her course was notable for tracheostomy placement; she has since been decannulated and no longer requires RRT. She was discharged home safely on POD 64.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: HOCM with RV dysfunction is especially challenging due to competing hemodynamic goals: increased inotropy and decreased afterload improve RV dysfunction but worsen HOCM. In this case, PVOD leading to acute respiratory failure further complicated treatment. PVOD results from occlusion and fibrous remodeling of postcapillary venules. Despite being a subgroup of Group 1 pulmonary hypertension (Table 1), PVOD is often mistaken as pulmonary arterial hypertension.[4] Diagnosis is difficult and requires histologic examination, though high-resolution CT scan may show ground-glass opacities and interlobular septal thickening.[5] Hemosiderin-laden macrophages can be found on bronchoalveolar lavage.[6] Inhaled pulmonary vasodilators are helpful in RV dysfunction due to their selectivity for the pulmonary circulation. However, these same agents can paradoxically dilate arterial pulmonary vasculature without affecting the veins in PVOD. This leads to a relative decrease in venous outflow and can cause acute pulmonary edema and hypoxia.[7] Thus, there should be a high index of suspicion for PVOD in patients with worsening hypoxia after receiving inhaled pulmonary vasodilators.

Management of concomitant HOCM, RV failure and PVOD requires careful consideration of these conditions and potential escalation to mechanical circulatory support in the perioperative period. A multi-disciplinary approach involving cardiac surgery, pulmonary hypertension and critical care specialists is essential for the successful care of these challenging patients.

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Table 1. World Health Organization Classification of Pulmonary Hypertension[4]

Class 1	Pulmonary arterial hypertension
Class 1'	Pulmonary veno-occlusive disease and/or pulmonary capillary hemangiomatosis
Class 2	Pulmonary hypertension due to left heart disease
Class 3	Pulmonary hypertension due to lung disease and/or hypoxia
Class 4	Chronic thromboembolic pulmonary hypertension (CTEPH)
Class 5	Pulmonary hypertension from unclear multifactorial mechanisms

Critical Care 8 - Delayed Diagnosis of Medulloblastoma Complicated by CSF findings of HSV-1 in an Adult Patient

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Introduction: Medulloblastomas are a highly malignant intracranial tumor arising from the cerebellum.[1] While they are the most common primary intracranial neoplasm in children, they account for less than 1% of all intracranial tumors in adults.[1] Due to their location in the posterior fossa, medulloblastomas can produce an obstructive hydrocephalus with signs of increased ICP.[1] Additionally, due to their presence in the cerebellum, presenting symptoms are often related to ataxia, nystagmus, dizziness, and gait disturbances.[1] Medulloblastomas can disseminate throughout the neuraxial space with nearly one-third spreading via CSF as drop metastases.[1,2] Treatment generally consists of surgical resection followed by radiation therapy of both the brain and spinal cord, as medulloblastomas tend to be highly sensitive to radiation.[1,2] The role of chemotherapy in adult medulloblastoma patients remains under investigation. Recurrence of medulloblastoma is common in adults with a nearly 60% recurrence rate.[1]

Methods: Here, we present the case of a young adult found to have medulloblastoma after a complicated hospital course with confounding diagnoses. A 38-year-old Spanish-speaking male presented with weeks-long headache, dizziness, double vision, and emesis. Initial CT imaging revealed obstructive hydrocephalus with cerebellar tonsillar herniation, as well as marked papilledema on ophthalmologic workup. An MRI revealed changes suspicious for cerebellitis versus infarction in the cerebellum. Due to his symptomatic hydrocephalus, an EVD was placed with elevated opening pressure to 35. CSF studies were positive for HSV-1 on initial meningitis-encephalitis panel; however, repeat PCR was negative. Though his presentation was atypical, further workup was negative, thus he was treated for HSV cerebellitis with acyclovir. Despite antiviral treatment, repeat MRI demonstrated persistent enhancement in the cerebellum [Image 1a]. The patient had continued elevated ICPs, failing multiple EVD clamp-trials before undergoing ETV. A biopsy sample was taken during ETV but was inadequate for histologic diagnosis. After improvement in his neurologic status, the patient was discharged with plans for outpatient follow-up and repeat MRI. Due to social circumstances, the patient did not present for follow-up MRI. He represented after two months with worsening gait instability, dizziness, and blurry vision. MRI revealed a new left cerebellar mass [Image 1b] with severe, progressed mass effect, not clearly seen previously. He subsequently underwent craniectomy with debulking and cerebellar biopsy. His post-operative course was complicated by prolonged mechanical ventilation requiring tracheostomy after loss of cough and gag reflexes secondary to significant edema and mass effect. Intraoperative frozen biopsy was concerning for CNS lymphoma. Malignant hematology was consulted with plans to initiate methotrexate therapy. However, the final pathology was determined to be anaplastic medulloblastoma. An MRI spine was negative for metastatic disease. Due to the diffuse spread of the tumor throughout the cerebellum, surgical resection was not a viable option. Radiation was determined to be his only treatment option and radiation oncology was consulted. Because of his immigration status and lack of insurance, feasibility of radiation in the United States was limited. Ultimately, the patient was transferred to a hospital in Mexico, where his family lived, for radiation therapy and continued treatment.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: This case of medulloblastoma represents the challenges presented by confounding and preliminary diagnoses in efficient and appropriate treatment of disease processes. Additionally, it emphasizes the health disparities experienced by uninsured immigrants.

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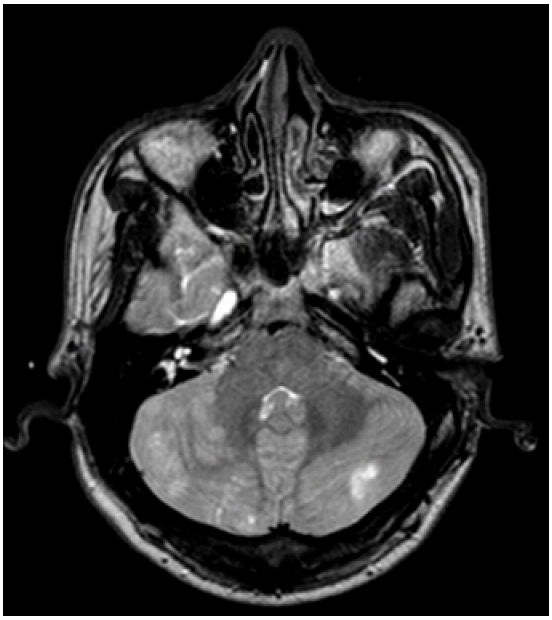

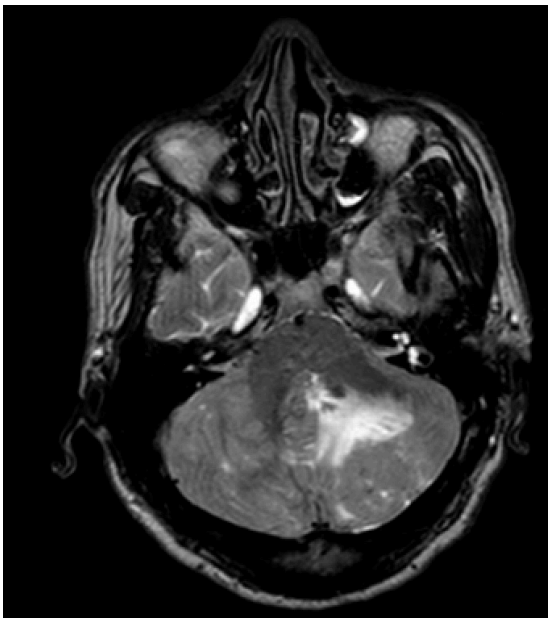

Critical Care 9 - Disaster averted: Phlegmasia Alba Dolens in a Post-Operative Patient

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Introduction: Deep vein thrombosis (DVT) is a common condition encountered amongst critical care patients. It is associated with substantial morbidity and mortality and is recognized as one of the most common preventable causes of hospital death.

A feared yet rare complication involves a massive DVT within the iliofemoral veins and/or inferior vena cava that can lead to phlegmasia alba dolens (PAD) or phlegmasia cerulea dolens (PCD). Severe venous flow obstruction, marked limb swelling, pain, acrocyanosis and arterial compromise to the lower extremity is possible, with development of gangrene and limb loss. Prompt recognition and diagnosis is crucial for limb savage. We present the management of a case of phlegmasia alba dolens in a post-operative patient with a complex surgical and intensive care course.

Methods: A 65-year-old female presented to our institution from an outside hospital for weakness and loss of consciousness with peritonitis, hemodynamic instability, and CT scan confirming free air and fluid consistent with perforated viscus. She had a significant past medical history for hypertension, diabetes mellitus, anxiety, and gastro-esophageal reflux disease with a complex surgical history including multiple abdominal surgeries and revisions for refractory ulcers. One week prior to her presentation, she had undergone an elective subtotal gastrectomy, resection of gastrojejunal (GJ) anastomosis, small bowel resection with takedown of prior jejunojejunal (JJ) anastomosis, with new GJ and new Roux-en-Y reconstruction.

Upon admission, she underwent exploratory laparotomy and was found to have necrotic dehiscence of her GJ anastomosis with gross spillage and was left in discontinuity with an open abdomen. On hospital day seven, it was noted that her left lower extremity had acutely become edematous and discolored. There was normal capillary return with diminished pulses. Assessment of pain and discomfort could not be completed due to heavy sedation. D-dimer was obtained with a result of >35,200 and duplex ultrasound of the lower extremities showed extensive clot involving the left proximal ileo-femoral veins extending distally through the popliteal, peroneal, and posterior tibial. The superficial veins were spared. Due to extensive concern for propagation to PE, an IVC filter was urgently placed. Therapeutic anti-coagulation was started with unfractionated intravenous heparin. Vascular medicine was consulted without immediate need for surgical intervention, and recommendations to closely monitor the limb for development of phlegmasia cerulea dolens.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: DVT/PE is a well-known cause of increased mortality in hospitalized patients. Virchow's triad describes the pathophysiology behind the development of thrombus, including venous stasis, hypercoagulability, and endothelial injury. Several risk factors specifically for our patient, included undergoing surgery (multiple times), a long intra-operative course, prolonged hospitalization, and immobilization.

Iliofemoral DVT, compared to the distal deep veins, have the potential to develop phlegmasia alba dolens (PAD). This represents about 25% of DVT diagnosed and is associated with a higher risk of adverse outcomes including severe leg pain, swelling, limb ischemia and gangrene, with ultimate limb loss, and death. PAD has been described as "milk leg" or "white leg" where there is complete occlusion of the deep venous system and sparing of the superficial, allowing drainage of the extremity. PCD is complete occlusion of the deep and superficial systems, for which arterial compromise is of critical concern.

Treatment for an iliofemoral DVT is aimed at lowering the risk of embolic complications. Systemic anti-coagulation is the treatment of choice. If anti-coagulation is contraindicated due to bleeding or need for surgery, an IVC filter should be placed to prevent propagation. Clot removal and reduction strategies need to be considered when life-threatening ischemia occurs, which includes endovascular thrombus removal or surgical thrombectomy.

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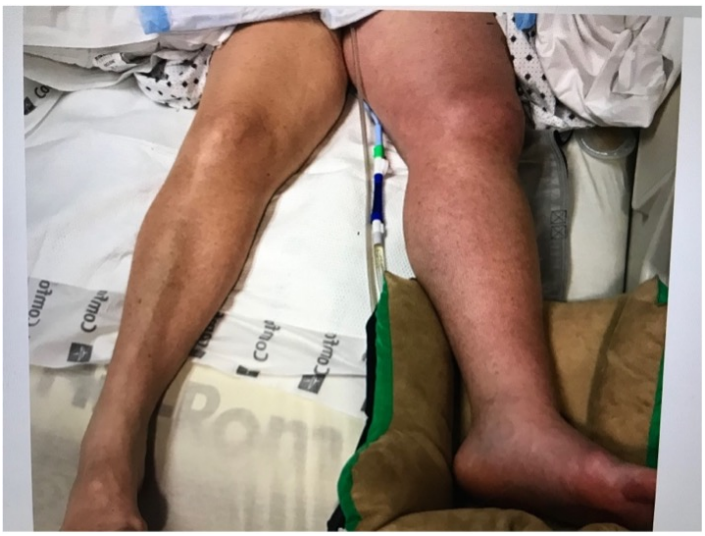

Critical Care 10 - Enigma wrapped in a conundrum: Possible Peripartum Hemophagocytic lymphohistiocytosis manifesting as acute respiratory failure requiring VV ECMO and LVAD support

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Cedars Sinai Medical Center[1]

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening disorder characterized by unopposed activation of one's immune system leading to rapid multi-organ failure. Oftentimes, HLH is mistaken for refractory sepsis and delayed or improper therapy results. We present a case of a peripartum patient who developed respiratory collapse requiring Venovenous (VV) ECMO and LVAD support whose work-up is ongoing for HLH.

Methods: 36 F G3P1 at 16 weeks gestation presented with fevers, chills, and dyspnea concerning for pneumonia complicated by acute hypoxic respiratory failure. Labs on admission notable for pancytopenia, coagulopathy, and elevated ferritin. TTE on admission found mildly depressed LV, normal RV with EF 45%. Given the constellation of symptoms and markedly elevated ferritin, highest on the differential was HLH so high dose dexamethasone was started concurrently with empiric antibiotics. Shortly after admission, the patient had spontaneous abortion with retained placenta requiring exploration. Subsequently, with worsening hypoxia despite maximal ventilatory support the decision was made to place the patient on VV ECMO. Despite ECMO support, the patient continued to have severe hemodynamic instability with repeat TTE now demonstrating EF < 20%, new wall motion abnormalities of LV, and moderately depressed right ventricular systolic function. Given the patient was requiring escalating inotropes with continued signs of multi-organ dysfunction of unclear source discussion with ECMO team for possibility of converting to VAV ECMO but given patient was still profoundly hypoxic with VV ECMO in place, left ventricular assist device (LVAD) was placed to maximally support the patient. During this time the patient developed oliguria and necessitated continuous renal replacement therapy for fluid optimization. After 24 hours of mechanical circulatory support and fluid optimization, the patient was able to be successfully decannulated from ECMO on hospital day (HD) 3, and on HD 5 LVAD was able to be removed. TTE showed normal biventricular function. The patient was able to be downgraded from the ICU on HD 10. The patient is still admitted in the hospital undergoing medical treatment for critical illness deconditioning with HLH studies still pending and work-up thus far inconclusive.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: HLH is characterized by uncontrolled proliferation of lymphocytes and macrophages secretes excessive inflammatory cytokines. Diagnosis of HLH requires a minimum of five of eight characteristic findings: fever, splenomegaly, at least two peripheral cytopenias, hypertriglyceridemia or hypofibrinogenemia, elevated ferritin levels, documented hemophagocytosis in bone marrow or lymphatic tissue biopsy, depressed natural killer cell function, and elevated CD25 levels 1. HLH is classified as primary or secondary depending on the patient's age of presentation and family history 1. As is the case of the patient described here, the secondary or acquired variant is seen typically in more adults without a clear family history of the disease. With treatment some case series report a 30-day mortality of 20–44% and overall mortality of 50–75% 1. Treatment of HLH is primarily supportive and includes immune-modulating therapies 2. HLH treatment protocols using single, or combination therapies include glucocorticoids, etoposide, and cyclosporine 2. However, the use of chemotherapeutics, steroids, or other immunosuppressants such as those mentioned may be extremely risky without the absolute exclusion of an infective process. The immunosuppression that ensues may result in an unchecked propagation of the underlying infection if that is the actual source, ECMO is a useful strategy aimed at providing temporary support for those with HLH, allowing them to recover the cardiac and pulmonary function compromised during the initial cytokine storm. There are few reports discussing the use of ECMO as a mechanism of supportive care in HLH, and the few that exist involve exclusively the pediatric population 3. This case proved even more unique in that the degree of hypoxia despite VV ECMO support and continued profound cytokine storm progressing to cardiogenic shock within less than 24 hours of admission lead to LVAD placement, where case reports using ECMO for patients with HLH typically undergo VAV ECMO. Ultimately, further investigations are necessary to elucidate those best supported with ECMO when affiliated with HLH.

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Critical Care 11 - High dose Hydroxocobalamin to Attenuate Vasopressor Requirement in Ischemic Bowel and Refractory Septic Shock

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Cleveland Clinic[1]

Introduction: Vasoplegia is described as hypotension with low systemic vascular resistance, normal cardiac function, and high dose vasopressor requirement despite adequate fluid resuscitation [1]. In critical illness, there is a depletion of catecholamines and vasopressors which leads to a vasodilatory state (Figure 1). In addition, nitric oxide synthase is stimulated by lactic acidosis, reactive oxygen species, and hypoxia. Inducible nitric oxide synthase generates large amounts of nitric oxide and causes further vasodilation (Figure 2). An imbalance of vasodilation and significant vasoconstriction from intravenous vasopressors may cause differential hypoxia and worsen vasoplegia (Figure 3). Hydroxocobalamin has been used to scavenge nitric oxide and attempt to reduce irregular vasodilation in vasoplegic syndrome [1].

Hydroxocobalamin has recently emerged in clinical use for refractory perioperative vasoplegia, particularly in the cardiac surgery and liver transplant patient population [2]. Its use in these populations and in septic shock is limited to case series [3]. Here, we present a case using high dose hydroxocobalamin to attenuate vasopressor requirement in ischemic bowel and septic shock.

Methods: This is a case of a 75-year-old male with a past medical history of coronary artery bypass graft surgery and Crohn's disease who presented with abdominal pain. He developed hypotension, renal dysfunction, and fevers with worsening clinical status consistent with a diagnosis of septic shock. He was taken to the operating room for exploratory laparotomy. He was found to have terminal ilietis and generalized patchy ischemia in the small bowel. He had a resection of the terminal ileum, was left in discontinuity, and was brought to the surgical intensive care unit for postoperative care.

On arrival to the intensive care unit, he was on norepinephrine 0.6 mcg/kg/min, vasopressin 0.04 Units/min. Bedside transthoracic echocardiogram revealed normal biventricular function and a plethoric IVC. He was given IV hydrocortisone, sodium bicarbonate, and calcium chloride. Despite this, his vasopressor requirement remained high at norepinephrine 0.4 mcg/kg/min and vasopressin 0.04 Units/min.

Given his profound vasoplegia, high pressor requirement, and presentation of ischemic bowel, he was administered IV hydroxocobalamin 5mg. Fifteen minutes after administration of hydroxocobalamin, he was completely off vasopressors. Two hours later, he experienced a rebound hypotension with an attenuated vasopressor requirement (norepinephrine 0.1 mcg/kg/min). Twenty-four hours after administration of hydroxocobalamin, he was off all vasoactive agents. Days later, he returned to the operating room for re-anastomosis, and was subsequently extubated and turned off all vasopressors.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: We presented a case of administering high dose hydroxocobalamin to attenuate vasopressor requirement in ischemic bowel and refractory septic shock. The use of hydroxocobalamin in this particular disease state may be of value in future clinical use. In our case, continuing to treat vasoplegia secondary to ischemic bowel with high dose vasopressors could have caused further injury to the small bowel. Allowing time for the bowel to rest and recover without significant splanchnic vasoconstriction may have changed this patient's clinical trajectory. Hydroxocobalamin should be studied in large clinical trials to evaluate its effectiveness in septic shock and should be considered for attenuating vasopressors in patients who present with ischemic bowel.

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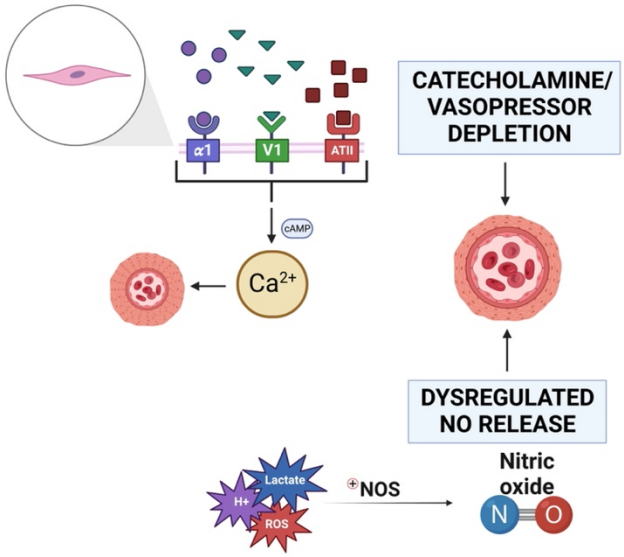

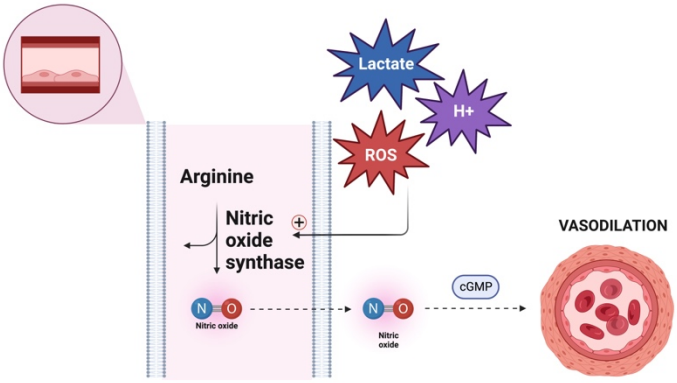
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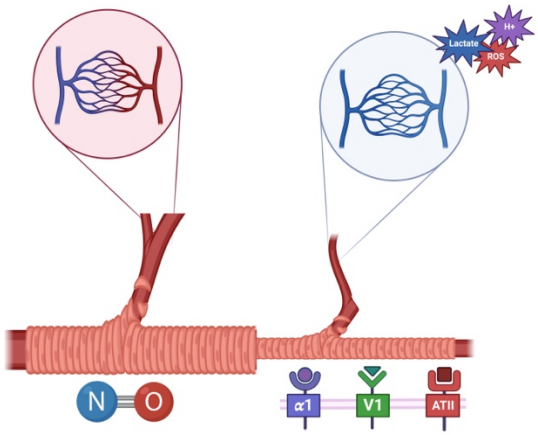
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Critical Care 12 - Incidental Discovery of an Isolated Levoatriocardinal Vein in a Post-Arrest CABG Patient

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Introduction: Levoatriocardinal veins (LACVs) are anomalous vessels connecting the pulmonary and systemic venous systems, originating from the left atrium or an upper pulmonary vein and draining most often into the superior vena cava or left innominate vein. While sometimes mistaken for a persistent left superior vena cava (PLSVC), they are embryologically distinct[1]. They are almost exclusively associated with left-sided obstructive anomalies; isolated LACVs are limited to under ten described cases[2,3]. LACVs typically drain cranially and result in a left-to-right shunt but have also been shown to have inducible flow reversal and right-to-left shunts with changes in intrathoracic pressure, as with Valsalva or coughing fits[2–4]. Given this variable shunt and the risk for paradoxical emboli, isolated LACVs have implications distinct from their more common mimics. Here we describe the case of an incidentally found, isolated LACV in a patient with unexplained cardiac arrest post-CABG and discuss the potential implications of this anomaly.

Methods: A 60-year-old male with a history of obesity, diabetes, hypertension, and tobacco use presented with anginal symptoms and was found to have an inferior STEMI. Cardiac catheterization showed severe multivessel disease including an occluded RCA and 80% proximal LAD stenosis. The RCA was revascularized with three stents; given his disease burden, intervention for other vessels would be staged. His post-PCI hospital course was notable for transient complete heart block that resolved into 1st degree heart block, mild acute kidney injury, mild ketoacidosis, and asymptomatic COVID. Eight days later he underwent an on-pump CABG with in-situ LIMA to distal LAD and SVG from aorta to OM1. Curiously, after aortic cross-clamping dark venous blood was seen draining from the aortic root vent. There were no intraoperative complications; post-pump cardiac function was normal. He received the institution's usual post-CABG care, though he remained intubated overnight for severe delirium. The next morning, he remained stable on low dose epinephrine and norepinephrine. With low dose dexmedetomidine, he followed commands and passed a breathing trial with an appropriate blood gas. He was extubated, but immediately afterwards became unresponsive with telemetry showing ventricular fibrillation. ACLS proceeded including reintubation and preparation for re-sternotomy. After compressions, epinephrine, amiodarone, and a third shock, he had ROSC, but remained markedly hypotensive and was cannulated for central VA ECMO bedside. After intubation he also remained hypoxemic with PaO2s in the 40s on 100% FiO2; bronchoscopy showed normal tube placement, and this did not resolve until on ECMO. No cause for the arrest was identified, as an angiogram showed patent stents and grafts, there were no gross lab derangements, and SpO2 pre-extubation was normal. During a takeback for ECMO decannulation two days later, a left IJ central line was attempted, and TEE showed a guidewire entering the left atrium via the left superior pulmonary vein. Review of a prior CT showed a previously unrecognized vessel connecting the left innominate vein and left superior pulmonary vein. Given concern for its possible contribution to his arrest, he then went back for ligation. He was extubated the next day without issue, though ultimately had a protracted, challenging hospital course.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: In this case there was no clear cause of the patient's arrest or residual hypoxemia after reintubation. Respiratory failure is high on the differential for a post-extubation arrest and with his comorbidities, but there are multiple inconsistencies. The onset was too acute, and this is also classically associated with PEA. Ventricular fibrillation is the most common post-cardiac surgery arrest and often a sign of graft failure, but coronary imaging was negative[5]. A pulmonary embolus causing ischemia could fit timing and hypoxemia but is unlikely to have resolved without treatment. The discovery of an isolated LACV, which to our knowledge has not been described in the adult cardiac surgical setting, raises the question of whether it contributed to this arrest via a right-to-left shunt induced by fluctuating intrathoracic and pulmonary venous pressures peri-extubation and peri-code. While it cannot be implicated with certainty, this case should prompt discussion whether LACVs should be addressed at the time of cardiac surgery.

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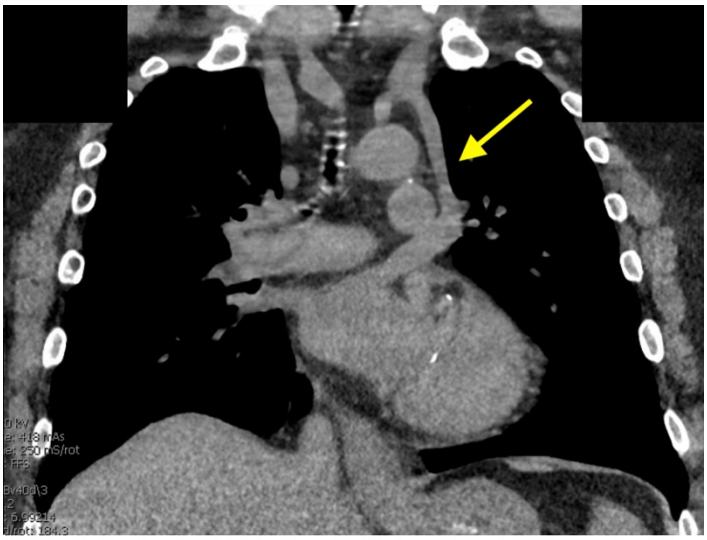
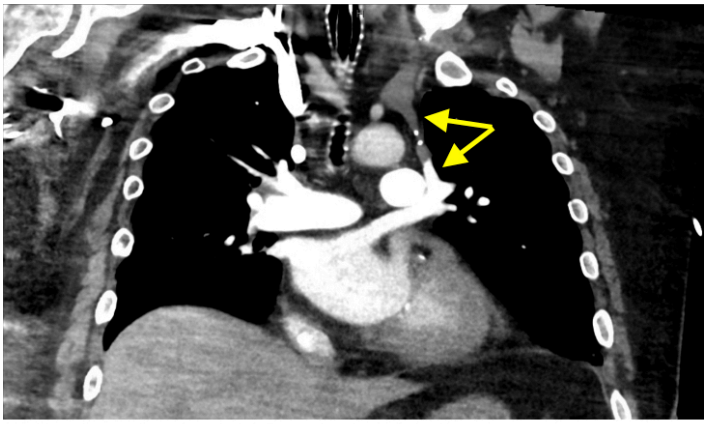
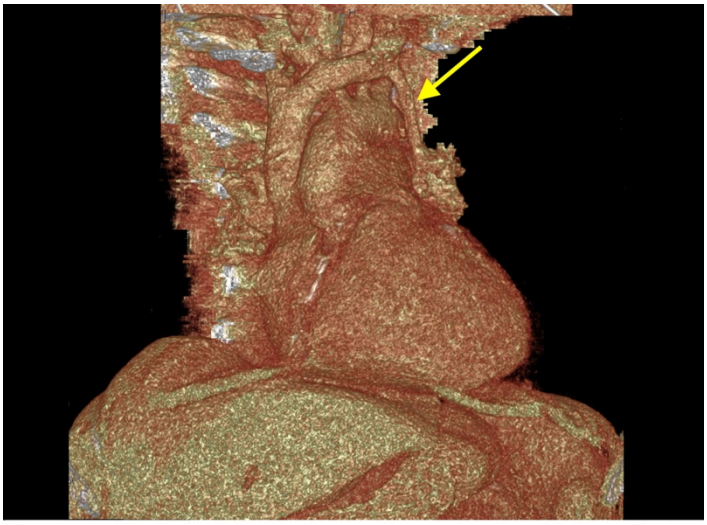


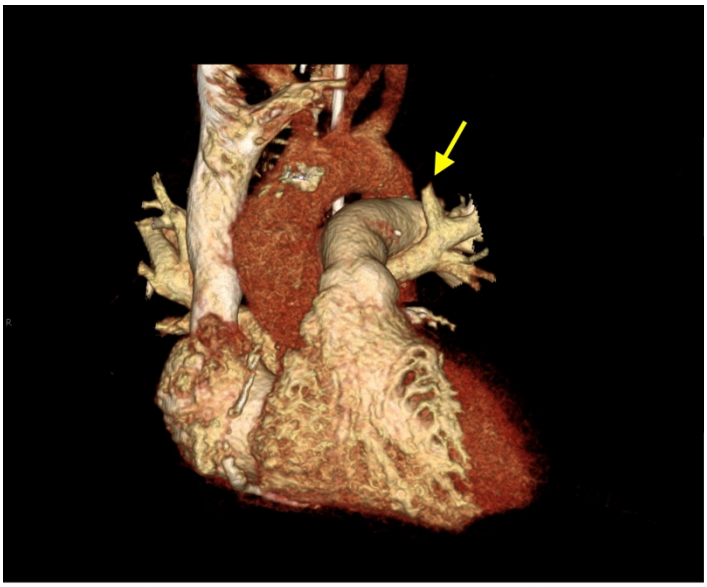
CT Chest without contrast obtained pre-CABG. Arrow shows LACV connecting left innominate vein and left superior pulmonary vein.



CT Chest with IV contrast obtained after LACV ligation. Arrows show vessel remnants connected to systemic and pulmonary venous systems. Non-contrast blood in lower segment comes from smaller venous connection (not shown).



3D reconstruction of patient's heart and surrounding vessels using a CT Chest without contrast obtained pre-CABG. Arrow shows LACV connecting left innominate vein and left superior pulmonary vein. Some structures omitted for better visualization.



3D reconstruction of patient's heart and surrounding vessels using a CT Chest with IV contrast obtained after LACV ligation. Arrow shows the remaining segment that connects to the left superior pulmonary vein and contains contrast. Some structures omitted.

V

Critical Care 13 - Malignant Mesothelioma Presenting as Constrictive Pericarditis and Hypoxemia

Sarvie Esmaeilzadeh[1], Louisa Palmer[2], Dirk Varelmann[2]

Brigham and Women's Hospital[1] Brigham and Women's Hospital[2]

Introduction: Malignant mesothelioma is a rare neoplasm arising from mesothelial cells of the body, most commonly from pleural surfaces, but also from the pericardium and peritoneum. Clinical presentation is often non-specific, and definitive diagnosis may require surgical biopsy.[1]

Methods: We describe a 75-year-old gentleman with recurrent pericardial effusion and dyspnea concerning for subacute constrictive pericarditis who presented for consideration of surgical intervention. He had recently undergone large volume pericardiocentesis at an outside hospital for newly diagnosed idiopathic pericardial effusion. He endorsed a 3-month history of dyspnea on exertion, fatigue, and unintentional weight loss, but denied recent infection or anginal equivalents.

Echocardiography revealed new biventricular systolic dysfunction, right ventricular dilation, severe functional tricuspid regurgitation. Clinical exam was concerning for acute heart failure. Computed tomography of the chest revealed moderate left pleural effusion and loculated pericardial effusion surrounding the right lateral heart border with compressed right atrium. Left thoracentesis revealed exudative effusion with 1193 nucleated cells/uL (82% lymph, 5% monos, 1% macros, and 4% mesothelial). Cytology noted reactive mesothelial cells and histiocytes. Cardiac magnetic resonance imaging was notable for post-contrast enhancement of the pericardium with extensive circumferential inflammation and marked thickening as well as evidence of pericardial constrictive physiology (paradoxical septal motion, dilatation of the inferior vena cava and bilateral atria).

Right heart catheterization was non-diagnostic but suggestive of constrictive pericarditis. It revealed relatively low cardiac pressures without evidence of ventricular interdependence but with an equivocal square root sign, consistent with restrictive pericarditis in setting of volume depletion. Given the posterolateral location of the pericardial effusion, placement of a pericardial drain was deemed infeasible. Despite maximal medical therapies, the patient's respiratory status deteriorated with increasing oxygen requirements, and he ultimately required intubation and emergent peripheral cannulation for veno-arterial extracorporeal membrane oxygenation given hypoxia and hypotension.

He was subsequently taken for emergent chest exploration and possible pericardiectomy. The intraoperative course was notable for what appeared to be grossly malignant tissue lacking structural integrity. Intraoperative pathology was consistent with mesothelioma. The patient was transported to the ICU in critical condition. Given the underlying diagnosis, the patient's family opted for comfort care measures, in line with the patient's prior stated wishes.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Mesothelioma is an often-insidious neoplasm with poor prognosis that can present with non-specific cardiorespiratory symptoms, signs and imaging. Exudative pleural effusions (especially unilateral) should always be regarded with skepticism. Pericardial involvement is possible. Pleural or pericardial fluid analysis and cytology are non-diagnostic in a large proportion of patients, and definitive diagnosis may require surgical biopsy.[2] Clinicians must maintain a high index of suspicion in order to facilitate appropriate diagnosis and allow for optimal treatment strategies, which in the late stage may consist of end-of-life care.

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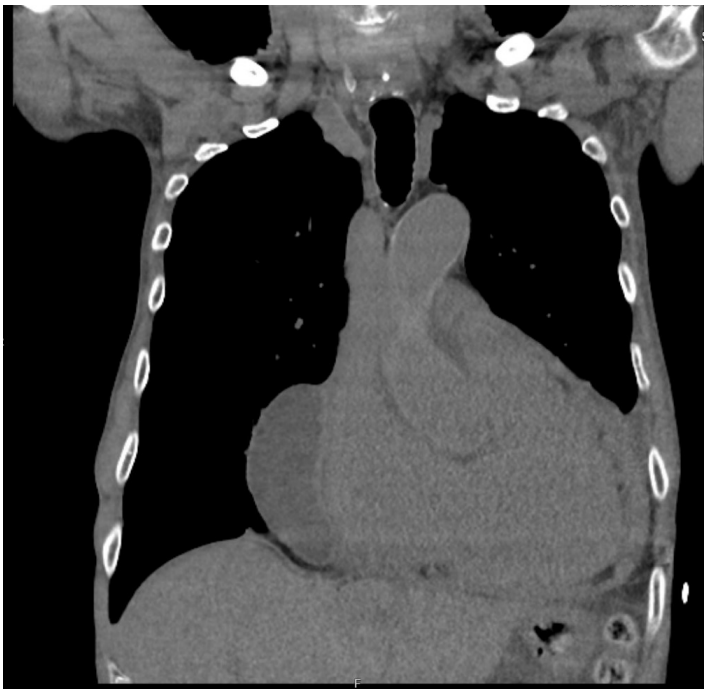

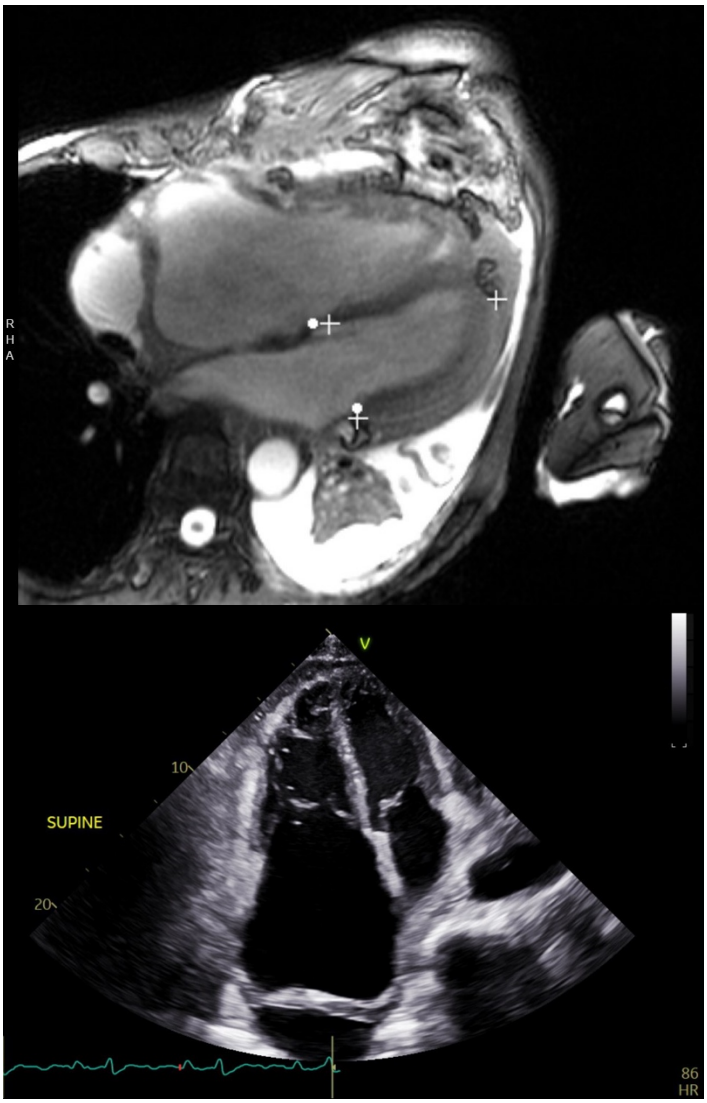
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Critical Care 14 - Migration of Temporary Percutaneous Dual-Lumen Right Ventricular Assist Device with Oxygenator (oxy-RVAD) with Subsequent Acute Liver Injury

Shannon Sparrow[1], James Barger[2], Sagar Dave

Emory University Hospital[1] Emory University[2]

Introduction: Acute right ventricular (RV) failure is a profoundly morbid condition, particularly in patients with left ventricular assist devices (LVADs) (1). Although there are several mechanical circulatory support options available for RV failure on the market, these treatment options are only FDA-approved for temporary use (2) and have significant complications, most commonly acute kidney injury (AKI), hemorrhage, intracerebral hemorrhage, sepsis, and thrombosis (2,3). Percutaneous right ventricular assist devices (RVADs) are also difficult to place properly, as the RV has a coarse trabecular network and thin, compliant walls that make device placement technically more challenging (4). Despite these challenges, little has been published about cannula migration as a complication of temporary RVAD support (5). Thus, we present a case of cannula migration in a dual-lumen cannula RVAD with a resultant rise in liver function tests (LFTs) as one of the key diagnostic clues.

Methods: We present a 34-year-old female with a history of seizure disorder, chronic pain, and post-partum cardiomyopathy complicated by recurrent VT s/p AICD and s/p LVAD x4, complicated by recurrent pump thromboses, CVA, and driveline infection. Her fourth LVAD (HM3) was implanted as destination therapy in 9/2022 due to worsening left ventricular (LV) failure and the presence of multiple antibodies preventing her from being a transplant candidate. She presented to the emergency department for VT and influenza A. Her VT became hemodynamically unstable after arrival and was refractory to defibrillation x3, amiodarone, and lidocaine. She required intubation and the ECMO team was consulted. The patient underwent placement of a dual-lumen right ventricular assist device cannula with an oxygenator (oxy-RVAD) for right heart failure and cardiogenic shock. Her baseline TEE was notable for laminar flow and proper position of her LVAD, with moderate MR, severely dilated RA and RV, with severe TR. Immediately postoperatively, she was on epinephrine at 0.1 mcg/kg/min, norepinephrine at 0.1 mcg/kg/min, and vasopressin at 0.08 units/hr. She had oxy-RVAD flows of 3.5 L/min with a 3 L/min sweep and HM3 flows of 4.5L/min. The next day, her LFTs were ALP 138, ALT 444, and AST 864, with a worsening lactic acidosis. Her nasogastric tube (NGT) dislodged and had to be advanced when her sedation was weaned, as she became agitated. The KUB for her NGT showed partial displacement of the oxy-RVAD cannula, although this was only seen in a retrospective review of the imaging. On POD 2 the patient's LFTs increased to ALT 6136 and AST 2290, despite improvement in lactic acidosis and AKI. Her CXR showed migration of the oxy-RVAD cannula with bedside US suggesting placement in the IVC. Flow on the oxy-RVAD was 3.6 L/min with a sweep of 1 L/min, though flow on the LVAD had decreased to 3.6 L/min from a prior 4.5 L/min on POD 0. The patient was off norepinephrine, epinephrine was at 0.02 mcg/kg/min, and vasopressin was at 0.02 units/hr. A CT chest confirmed cannula displacement into the IVC and occluding the hepatic veins. Given the patient's RV improvement without true support, the decision was made to decrease her flows, wean her sweep, and decannulate her at bedside. She was supported with vasopressors and inotropes and her LFTs improved.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: This unique patient suffered a rare cannula migration complication that presented as a simultaneous improvement in clinical hemodynamics with congestive hepatopathy due to her cannula occluding the hepatic veins. It is possible that the patient's agitation and her tricuspid regurgitation contributed to cannula migration and the improvement in hemodynamics may be due to the RV failure's etiology being quickly reversible once antiarrhythmics were at therapeutic levels. Her clinical improvement presented a challenge in diagnosing her cannula migration. The main clinical evidence of migration was the acute Budd-Chiari syndrome that developed from the cannula impeding liver venous drainage, which was described in V-V ECMO patients whose cannulas occluded hepatic veins (6). Additionally, one can see how an inexperienced provider may have believed the cannula on the KUB to still be in the proper position with a rotated X-ray. Thus, we recommend daily evaluation of cannula position and a high index of suspicion of migration in acute liver injury in patients with RVADs, even in the setting of hemodynamic stability.

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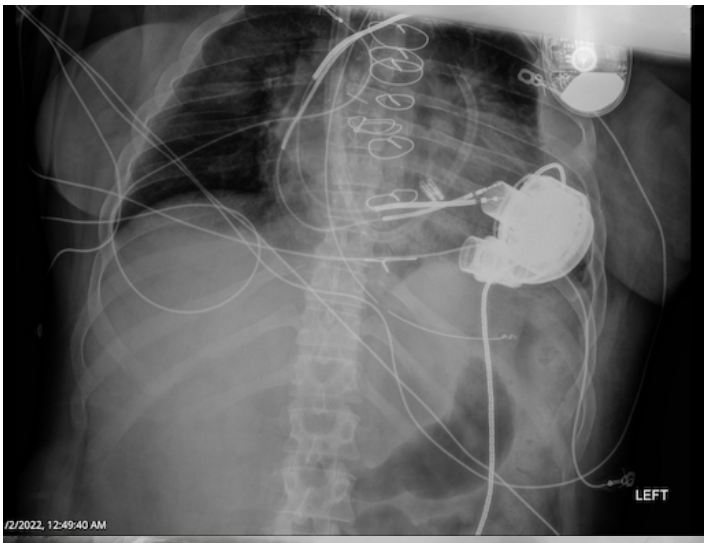
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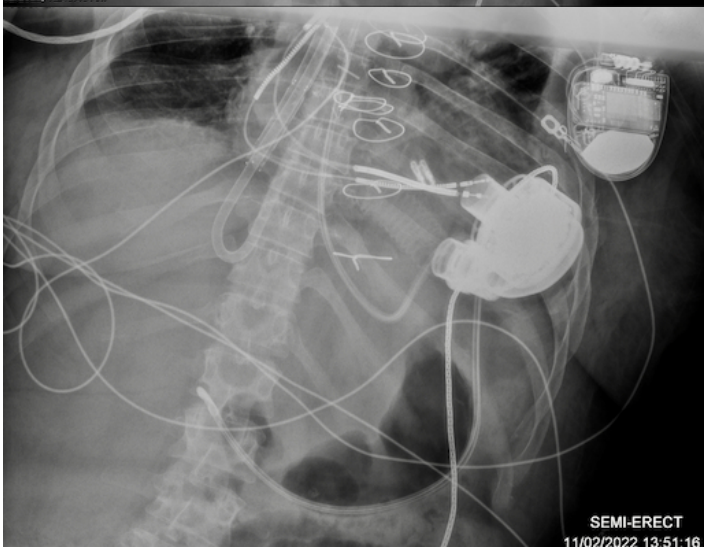
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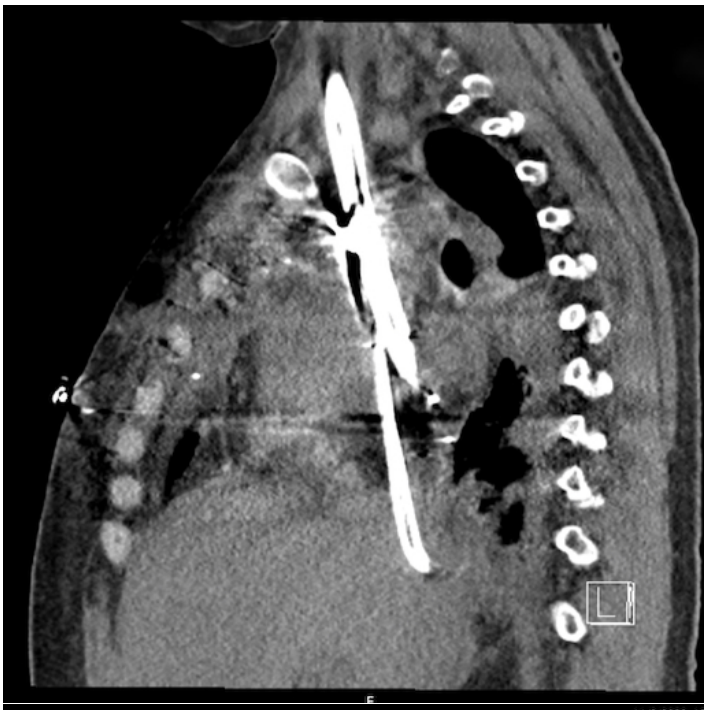
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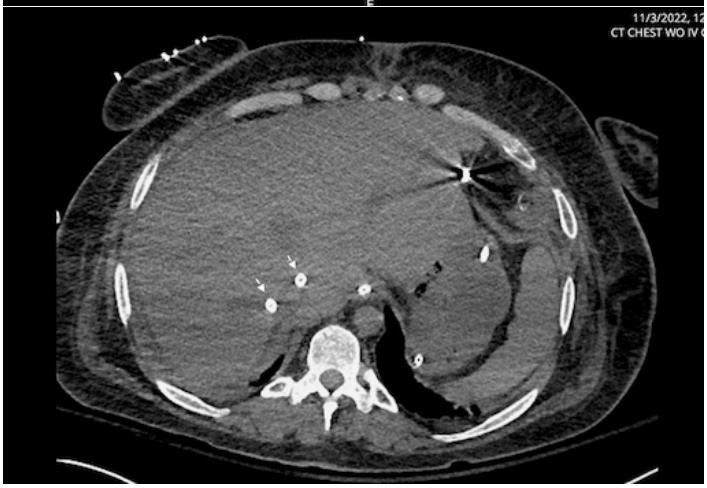

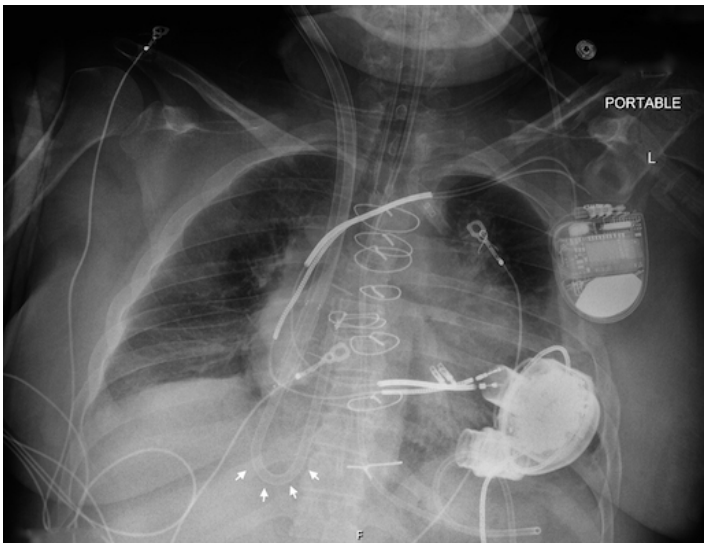
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Critical Care 15 - Peripartum Cardiomyopathy: Hoping for Recovery verses Speeding Towards Transplantation

Shamik Patel[1], Ankur Srivastava[2]

Weill Cornell New York Presbyterian[1] Weill Cornell Medical Center, New York Presbyterian Hospital[2]

Introduction: Peripartum cardiomyopathy is a rare yet deadly complication of pregnancy that affects 1 in 1000 to 4000 pregnancies a year in the United States with long-term mortality ranging between 7% to 20%. However, 72% of women will recover to an ejection fraction >50% by 12 months.[1] Though most women recover, many will need temporary mechanical circulatory support as a bridge to recovery. Some will need either long-term support or even heart transplant if there is no recovery in heart function. We present a difficult case of deciding between awaiting recovery with temporary mechanical circulatory support (MCS) versus expediting a heart transplantation.

Methods: A 34-year-old female G1P0 at 37 weeks and 5 days gestation with no past medical history presented to obstetric triage with worsening shortness of breath. An echocardiogram was performed which showed an ejection fraction of 17% concerning for peripartum cardiomyopathy. The patient was started on diuretics and a dobutamine infusion to assist her cardiac function. Unfortunately, the fetus developed non-reassuring fetal heart tones, and the patient was taken for emergent cesarean section under general anesthesia. Afterwards, an intra-aortic balloon pump (IABP) was placed to augment cardiac output. On post-operative day 2, the patient's condition worsened with increasing tachycardia and worsening biventricular failure on echocardiogram. The decision was made to intubate the patient and place her on peripheral veno-arterial extracorporeal membranous oxygenation (VA-ECMO) for hemodynamic support. The IABP was replaced with a percutaneous left ventricular assist device to decompress the left ventricle. Over the next 3 days, the patient was extubated, and the cardiac function appeared to have improved enough that there was consideration for decannulation of VA-ECMO while maintaining a ventricular assist device (VAD). Also under consideration was the patient's small vessel sizes which increased her risk of limb ischemia from peripherally placed MCS devices and the high risk of stroke from centrally placed VAD. The decision was made to urgently enlist the patient for heart transplant instead of awaiting return of cardiac function with a temporary VAD. While awaiting a transplant, the patient was placed on centrally cannulated VA-ECMO, however, she developed spinal cord infarct leading to paraplegia. The patient would subsequently receive a heart transplant on day 24 after presentation and was discharged to a spinal cord rehabilitation facility.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Peripartum cardiomyopathy is a rare indication for heart transplantation, accounting for approximately 1% of all heart transplantation, but is a viable alternative for patients who do not show signs of recovery.[2] The decision to proceed with urgent heart transplantation can be complicated as patients may recover from their cardiomyopathy but may be exposed to significant risk while waiting for recovery. It is reported that 10% of peripartum women who presented with acute or subacute heart failure requiring mechanical circulatory support had recovery of function.[3] In our patient, even though she may have had signs of recovery, the decision was made to urgently pursue a heart transplant as the risk of maintaining mechanical circulatory support was too high. Unfortunately, she did not receive a transplantation prior to a severe complication occurring but was discharged from the hospital with hopes of recovering her functional status.

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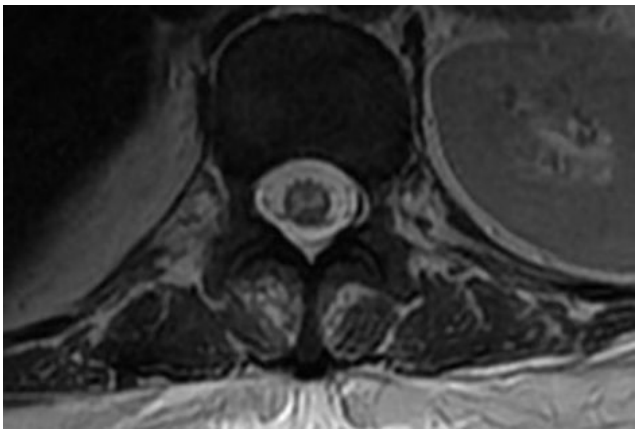

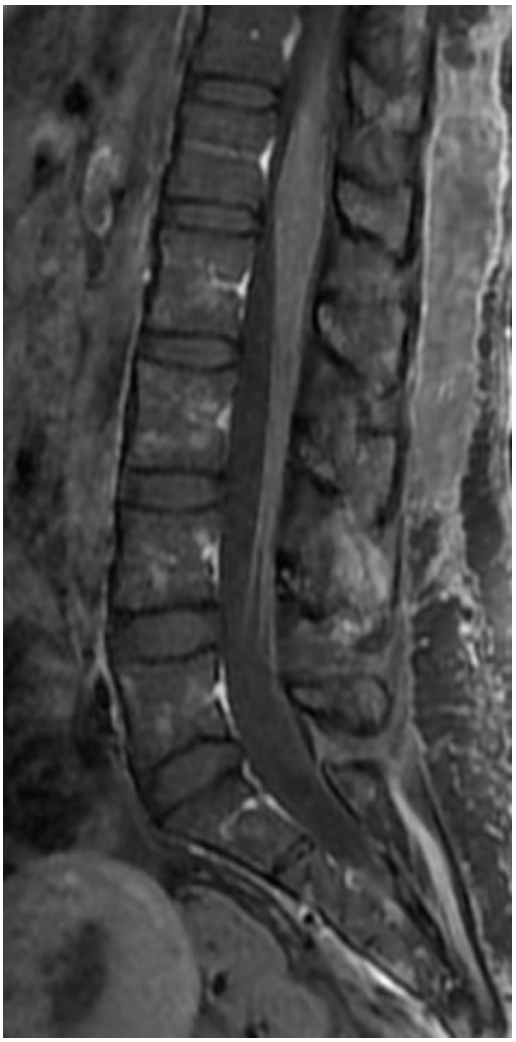

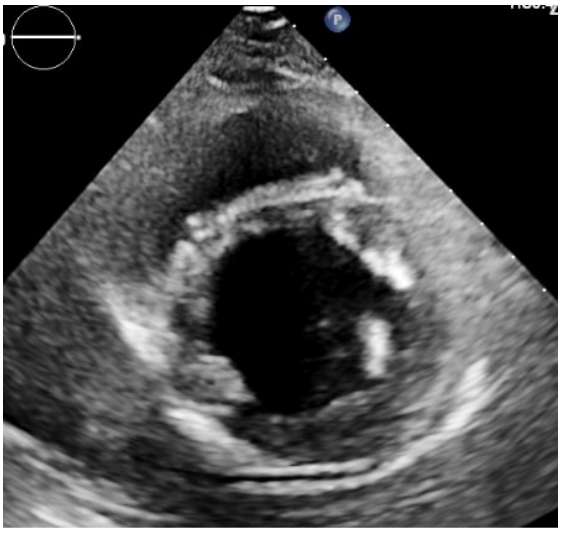

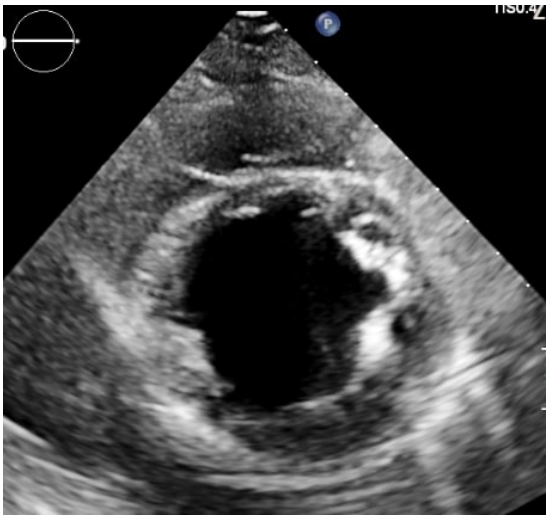

Critical Care 16 - Plexiform Neurofibroma of the Neck with Erosion of the Clavicle and Spontaneous Hemothorax

Omkaar Jaikaran[1], Ioannis Angelidis[2], Haviva Gsrrett

New York University Langone Medical Center[1] New York University Langone Medical Cent[2]

Introduction: Neurofibromatosis type 1 (NF1), also known as Von Reclinghausen's disease, is a multisystem genetic disorder which affects 1 in 3000. NF1 demonstrates an autosomal dominant inheritance pattern of the NF1 gene on chromosome 17 with half of the cases being sporadic [1,2]. NF1 gene encodes for the neurofibromin protein which functions as a tumor suppressor, regulating cell growth and division in nerve cells [1,3]. The signs and symptoms vary and include cafe-au-lait spots, neurofibromas, Lisch nodules, optic gliomas, hypertension, macrocephaly, scoliosis, learning disabilities, cardiovascular malformations, and less frequently vascular lesions [1,4]. While rare, these vascular lesions when located in the thorax can be associated with life threatening hemothorax [3].

Methods: A 22-year-old female with history of NF-1 with a plexiform neurofibroma of the left neck with extension to the left clavicle presented with an expanding palpable mass of the left clavicular region. An MRI of the chest identified a large destructive lesion of the medial left clavicle with suggestion of internal loculation (Fig 1). A subsequent ultrasound-guided fine needle aspiration identified neoplastic spindle cells with concern for malignant transformation. The patient underwent wide local excision of the neck mass with claviculectomy and sternectomy and was transferred to the ICU for further management. On postoperative day 5 the patient experienced a syncopal episode with tachycardia and hypotension. A CT scan of the chest revealed a large hemothorax with subsequent chest tube placement yielding 630 ml of sanguineous drainage. She underwent a thoracoscopy with evacuation of clots without evidence of active bleeding. Chest tubes were left in placement and were ultimately removed with no further evidence of recurrent bleeding.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Vascular involvement in patients with NF1 while rarely found in clinical practice can result in potentially fatal complications [3]. An incidence of 3.6% has been described with large vessel involvement usually resulting in either stenosis or aneurysm and small vessel involvement being responsible for the majority of bleeding sequelae [2,3]. Large vessel involvement has been described as secondary to direct invasion of the vessel wall by neurofibroma tissue, which compresses the vasovasorum and results in vessel weakness. Small vessel involvement, however, is proposed to be secondary to dysplasia of the wall itself with fibrohyaline thickening resulting in stenotic narrowing and weakening of the arterial wall [2]. Cardiothoracic hemorrhagic manifestations have also been proposed to be secondary to abnormalities of primary hemostasis secondary to intrinsic platelet and collagen abnormalities along with reductions of factor XII and von willebrand factor found in NF1 [5]. As in this case, cross sectional imaging and surgical exploration oftentimes does not yield an obvious source of bleeding [2]. Management of hemothorax depends on several factors, such as hemodynamic stability, coagulopathy, size of the possible identifiable vessel, and bleeding recurrence with options including thoracic drainage, endovascular embolization, video-assisted thoracic surgery, and thoracotomy [2,3].

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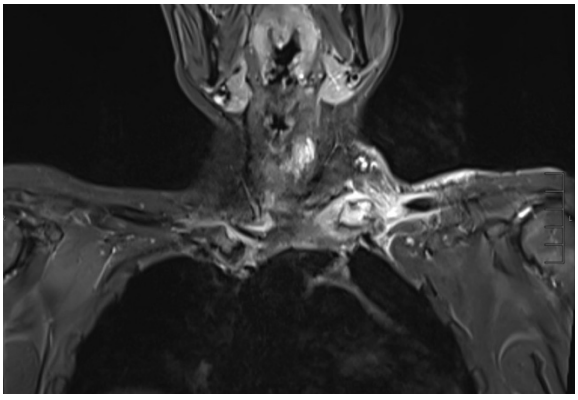

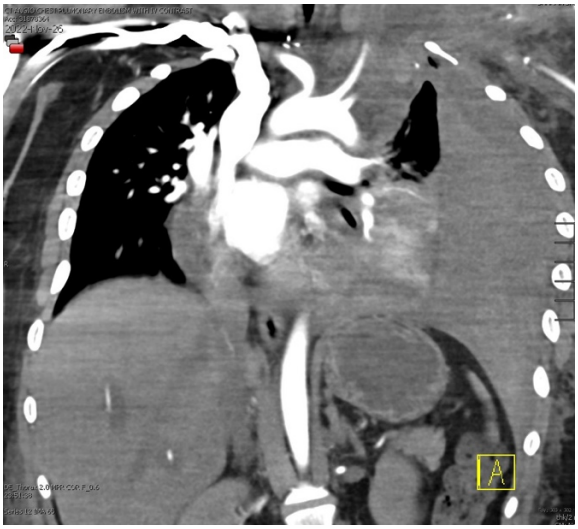

Critical Care 17 - Refractory Status Epilepticus as a Result of Hyperammonemia Secondary to Ureaplasma Parvum and Ureaplasma Urealyticum Infections after Bilateral Lung Transplant: A case report

Michael Hollar[1], Rosa Lopez Rincon[2], Vaibhav Bora, Vikas Kumar[3]

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Introduction: Ammonia is primarily produced within the gut via the breakdown of proteins. At supraphysiologic levels, there can be devastating consequences such as cerebral edema leading to herniation, intractable seizures, or death. We present a unique case of a patient who developed super refractory status epilepticus because of hyperammonemia secondary to Ureaplasma parvum and Ureaplasma urealyticum infections after a bilateral lung transplant.

Methods: A 69-year-old male with a history of severe pulmonary hypertension, COPD, chronic hypoxic respiratory failure, diabetes mellitus, and coronary artery disease was admitted to the ICU after a bilateral lung transplant. The patient's post-operative hospital course was complicated by pulmonary graft dysfunction which required VV-ECMO for three days. After ECMO decannulation there were concerns for stroke and seizure-like activity as the patient developed right-sided paresis and upper extremity rhythmic movements. CT, MRI, and MRA head were negative for acute abnormalities. Continuous EEG showed moderate to severe encephalopathy, no seizures. On postoperative day 25, the patient had a witnessed seizure; he was placed on cEEG revealing multiple continuous subclinical seizures. The patient continued with non-convulsive status epilepticus and was started on Propofol and Midazolam infusions, Levetiracetam q12 h, and Lacosamide 100 mg q12hs, but continued to seize. For refractory NCSE Ketamine infusion and Clobazam were started which were able to stop the patient's seizures. The patient was found to have a significantly elevated ammonia level, which maxed out at 412 µmol/L. Empirically, the patient was started on Doxycycline and Levofloxacin for suspected Mycoplasma/Ureaplasma pneumonia. Lactulose, sodium phenylbutyrate, L arginine, and L citrulline were initiated. Enteral nutrition was adjusted to a low-protein formula, and CRRT was initiated. Tacrolimus was discontinued as it can contribute to seizure onset. BAL revealed a positive infection with Ureaplasma Urealyticum and Ureaplasma Parvum. An MRI brain showed symmetric increased cortical T2/FLAIR hyperintensity involving the bilateral frontal, occipital and temporal lobes, as well as the signal abnormality involving the bilateral thalami, suspicious for acute hyperammonemia encephalopathy. After ammonia levels had normalized, attempts were made to slowly wean his ketamine infusion. After the second failed wean, he was placed on Phenobarbital, and he was started on Perampanel. During his third failed wean, the patient suffered 70 seizures in 24h, and then 10-20 per hour afterward. Two days later the family decided to pursue comfort care measures.

Results: Not Applicable- Medically Challenging Case Report

Conclusions: Ammonia is produced primarily within the gut and then transported to the liver where up to 90% is converted into urea. Accumulation of ammonia can cause disastrous consequences. Ammonia levels >200 µmol/L have been associated with severe brain damage. The initial symptoms may be attributed to ICU delirium, sedatives, or opiates and can often be missed. The most common cause of hyperammonemia in adults is hepatic dysfunction but it has been linked to lung transplantation, with an incidence of 1% to 4.1% carrying a 67% 30-day mortality rate. The etiology in transplant patients may be triggered by medical stressors, hepatic glutamine synthetase deficiencies, or partial urea cycle deficiencies but often systemic infections with urease-producing bacteria are the culprit. The first line treatments for hyperammonemia typically consist of lactulose and antibiotics such as rifaximin. These treatment regimens have shown no improvement in patient mortality in lung transplant patients, which is a reasonable assertation in patients with systemic ureaplasm infections as the source of the excessive ammonia production is not the gut. These treatments should not be withheld in transplant patients as often the definitive diagnosis of a ureaplasm infection can take up to ten days at many institutions as it typically is a send out lab. Supplementation to divert nitrogen from the urea cycle should be initated, but the initiation of early dialysis to reduce NH3 levels in the blood and empiric antibiotics for suspected disseminated urease-producing infections is paramount in the immunocompromised patient, as early treatment could be lifesaving.

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Critical Care 18 - Severe lactic acidosis of unknown etiology following open heart surgery

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Introduction: Lactic acidosis (LA) is a common metabolic disorder that occurs in patients who undergo cardiac surgery.[1] We present a case of a patient who experienced significant LA (peak lactic acid level of 18.2 mmol/L) following mitral valve replacement (MVR) and single vessel coronary artery bypass grafting (CABG).

Methods: A 63-year-old male (BMI 24 kg/m^2) with history of mitral regurgitation, CHF (LVEF 35%), CKD (stage III), HTH, HLD and prior full-thickness burn wounds (~30% TBSA) presented for MVR and single vessel CABG. The patient took holistic supplements and stopped one month before surgery. Home medications were furosemide 40 mg, metoprolol succinate 25 mg and aspirin 81 mg. Pre-operative transthoracic echocardiogram showed LVEF of 35%, a dilated LV, and a tethered posterior mitral valve leaflet. Left heart catheterization showed 70-80% stenosis of the diagonal artery and right heart catheterization showed pulmonary hypertension with a pressure of 74/34. Pre-operative labs were Hb 14.3 g/dL, Hct 45.1%, creatinine 1.5 g/dL, and INR of 1.1. Prior to induction, vital signs were blood pressure 154/109 mmHg, heart rate 98 beats/min, respiratory rate 12 breaths/min, and oxygen saturation of 100%. The patient was induced with fentanyl, lidocaine, ketamine, rocuronium**.** Following induction, the patient became hypotensive requiring norepinephrine (0.02-0.1 mcg/kg/min) and vasopressin infusions (0.04 units/min). Pre-bypass transesophageal echocardiography (TEE) showed dilated left ventricle with an EF of 30%, depressed right ventricular function, severe mitral regurgitation. While on CPB, cardiac flow rates ranged between 5.0-5.5 L/min, MAPs of 54-59 mmHg, SvO_2 of 78-86%, hematocrit of 30.7-33.6% and urine output of 250 ml. Total CPB time was 143 minutes. In the process of CPB separation, norepinephrine (0.02-0.08 mcg/kg/min), vasopressin (0.04 unit/min), epinephrine (0.04 mcg/kg/min) and milrinone infusions (0.375 mcg/kg/min) were started. Patient was in complete heart block (CHB) prior to separation from CPB and required ventricular pacing at 80 beats/min. Post-bypass TEE was unchanged. The new mitral valve was in an appropriate position. 700 ml of cell-saver blood, 500 ml of 5% albumin, and 940 ml of crystalloid solutions were given in the case. Total urine output was 950 ml with blood loss of 500 ml. In the ICU, lactic acid levels peaked at 18.2 mmol/L (Table 1). 8 ampules of sodium bicarbonate and 500 ml of 5% albumin were given overnight. Patient maintained cardiac output of 4.5-5.8 L/min, stroke volume of 50-72.5 ml, CVP of 12-17, MAPs of 66-94 mmHg, and mean PAP of 28-36 mmHg in the initial 15-hour post-operative period without escalation of vasopressors. The patient underwent pacemaker placement for CHB on post-operative day 5 and was discharged on post-operative day 7.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Causes of lactic acidosis following cardiac surgery are multifactorial.[2] Anemia, hypothermia, hypoglycemia, and hepatic dysfunction are potential causes, and were effectively ruled out. Patient specific comorbid conditions such as CHF, CKD, and pulmonary hypertension can be contributing factors. Other potential causes unique to our patient were the history of full thickness burn wounds (~30% TBSA). Interestingly, there is evidence in literature that show large burn wounds can induce a hypermetabolic stress response, which then can result in long-term mitochondrial dysfunction.[3-4] In the absence of other clear causes, we felt there could be a link between patient's history of burn wounds and the unusual presentation of LA that we observed. We otherwise reasoned CHB is an unlikely cause given patient was paced early on and pre-operative use of nutritional supplements containing calcium lactate was also unlikely given these were stopped long before surgery. In summary, it is imperative for clinicians to keep a wide range of differential diagnoses as LA of this severity is usually not compatible with life unless acutely recognized and corrected.

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Time	7:42	*	10:50	11:20	11:59	12:29	**	13:35	***	15:42	17:53	20:18	21:28	23:12	01:18	03:05	05:21
pH	7.42		7.376	7.325	7.319	7.375		7.308		7.193	7.293	7.345	7.342	7.461	7.506	7.460	7.460
Lactic Acid (mmol/L)	0.9		1.4	1.6	1.9	2.1		3.2		4.6	10.1	18.2	18.0	11.5	5.5	2.6	1.7
HCO3 (mmol/L)	23.6		24.3	25.5	24.8	20.7		18.8		18.9	18.9	17.2	18.1	24.6	28.6	29.8	30.2
Base Deficit	-0.5		-0.9	-0.8	-1.6	-4.0		-6.9		-9.2	-7.1	-7.6	-6.9	1.0	5.2	5.5	5.8
Hb (g/dL)	14.3		10.8	10.2	10.9	10.3		8.9		12.2	10.4	9.6	9.7	9.9	9.7	9.3	9.3
Hct (%)	44.8		33.5	31.3	33.6	31.7		25.7		36.3	31.3	28.9	28.9	28.4	26.8	25.6	25.4

Table 1. Trend of ABGs. **Start of CPB at 1041. **End of CPB at 1304. ***End of case at 1510.*

Critical Care 19 - Strongyloides Hyperinfection in an Immunocompetent Patient After Elective Cardiac Surgery

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Introduction: Strongyloides stercoralis hyperinfection has been reported in immunocompromised patients such as heart transplant recipients but has not been documented in immunocompetent patients who undergo elective cardiac surgery.[1] With the parasite persisting in the body for decades after initial infection, it can go unnoticed until an inciting stressor leads to an acceleration of the infection. We describe the case of a patient presenting for an elective cardiac procedure with a clinical course complicated by Strongyloides hyperinfection.

Methods: Our patient is a 65-year-old Vietnamese male with a history of type 2 diabetes, coronary artery disease, congestive heart failure with preserved EF and severe mitral regurgitation who underwent four vessel coronary artery bypass grafting, mechanical mitral valve replacement, left atrial appendage ligation and MAZE procedure. After an uneventful initial postoperative course, he was transferred to the stepdown unit.

On postoperative day six, he developed a high-grade fever with hypotension. With concern for septic shock, blood and respiratory cultures were obtained. He was started on broad spectrum antibiotics and was transferred to the ICU for further monitoring. His physical exam was unremarkable, and the hypotension responded to fluids without requiring vasopressors. Blood cultures eventually grew out enteric gram-negative bacteria (Serratia, Enterobacter cloacae, and E. coli). Further work up for the source of bacteremia included urinalysis, liver function tests, stool examination for parasites, hepatitis panel, EBV antibody, tick panel, T spot test, abdominal ultrasound, and CT abdomen. Apart from transaminitis, no obvious source of infection was identified. No valvular vegetations were seen on the transthoracic echocardiogram.

Given his polymicrobial gram-negative bacteremia with no clear inciting cause, the infectious disease team was consulted. He had a prior history of living in a small village in Vietnam with a high Stongyloides prevalence. He was then started on ivermectin for concern of strongyloides-associated gram-negative bacteremia. Strongyloides IgG antibodies resulted positive, making Strongyloides hyperinfection syndrome caused by reactivation due to the stress of cardiac surgery with cardiopulmonary bypass the most likely diagnosis. He completed a 14-day course in ivermectin and blood cultures cleared with appropriate antibiotic coverage.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Strongyloides stercoralis is a common parasitic disease that often goes undiagnosed in immunocompetent patients.[2] In patients presenting with polymicrobial gram negative bacteremia with no obvious source of infection, it should be considered as part of the differential diagnosis especially when patients have a history of living in a highly endemic area and are exposed to intense stressors such as cardiac surgery.[3]

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Critical Care 20 - Veno-Venous Extracorporeal Membrane Oxygenation to Facilitate Prolonged Inter-Facility Transport for Critical Airway Intervention

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Introduction: Veno-venous extracorporeal membrane oxygenation (VV ECMO) is used for patients with refractory hypoxemia despite maximal medical therapy. Its growing familiarity and safety have led to the application of VV ECMO to temporize pathophysiologic states culminating in severe acute respiratory failure. VV ECMO is also a recognized means to facilitate difficult airway management and can be planned in advance of such procedures to help facilitate gas exchange during periods of partial or complete airway obstruction.[1] Severe tracheal stenosis is one example that poses unique challenges to ventilation and oxygenation.

Methods: A 51-year-old man with a pertinent medical history of obesity and obstructive sleep apnea required orotracheal intubation for airway protection at a community hospital. He developed ventilator-associated pneumonia that required continued mechanical ventilation for acute hypoxemic respiratory failure. He was extubated six days later with an interval of self-extubation and reintubation. After discharge to an acute care floor, he developed acute hypoxemic respiratory failure due to pulmonary edema requiring orotracheal intubation, prolonged mechanical ventilation, and a tracheostomy. Mucus plugging, dyspnea, and challenging pulmonary mechanics persisted after tracheostomy. Bronchoscopy at that time demonstrated subglottic and distal tracheal stenosis that was partially bypassed by the tracheostomy tube.

Operative intervention was not feasible at the community hospital, and as a temporizing measure the tracheal tube was exchanged for one with a longer distal segment. This was dislodged ten days later, and the critical care team was unable to replace the tracheostomy tube due to obstruction. A pediatric endotracheal tube was placed via the stoma and sutured in place. A referral was made to our center for further evaluation and treatment by otolaryngology.

Interfacility transport carries substantial risks in a patient with such a tenuous airway, so VV ECMO was considered. After a multidisciplinary discussion and informed consent from the patient's family this was undertaken remotely by our ECMO team. The patient was cannulated via a femoral-femoral approach with a 25F multistage drainage cannula and a 23F single stage return cannula. VV ECMO was initiated with minimal hemodynamic changes. He was subsequently transported to our facility by ground for tracheostomy revision, which was accomplished uneventfully while on VV ECMO. He was then promptly decannulated without complication.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Laryngeal trauma from prolonged or repeated orotracheal intubation, and prior tracheostomy, can result in iatrogenic tracheal stenosis and pose significant airway management challenges in critical care settings. Tracheal blood supply is derived from the submucosa; mucosal compression from a tracheal tube or tube cuff can limit tracheal perfusion, resulting in ischemia and stenosis.[2] Patients with acquired tracheal stenosis develop symptoms of dyspnea, inspiratory stridor, and wheezing several weeks after prolonged intubation.

Management of tracheal stenosis includes bronchoscopy with dilation or surgical resection as definitive therapy. Severe tracheal stenosis complicates mechanical ventilation due to high airway resistance. VV ECMO in this setting can augment gas exchange or facilitate safer operative intervention.

The use of VV ECMO to aid in management of tracheal stenosis has been previously described, including in pediatric populations with congenital stenosis and malignant obstructions.[2] In this instance, relevant risk factors for tracheal stenosis included multiple orotracheal intubations, prolonged intubation, and a prior tracheostomy. Difficulties with airway management led to a temporized but unstable airway that required referral to our center for definitive intervention, which entailed a prolonged inter-facility transport. Loss of the airway during transport and difficulties with mechanical ventilation would have presented substantial challenges. As such, VV ECMO was employed to increase the margin of safety during transport for definitive airway management.

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Critical Care 21 - A Case Series Demonstrating Perioperative Use of Impella 5.5 For Hemodynamic Support in Patients with End-Stage Heart Failure Requiring Major Abdominopelvic Surgery

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Introduction: Patients with reduced cardiac function who require major noncardiac surgery are at high perioperative risk secondary to the primary cardiac dysfunction as well as related organ impairment impacted by anesthetic and surgical techniques. [1]. Temporary circulatory support with Impella (Abiomed Inc., Danvers, MA, USA) Heart Pumps has been widely used in high-risk PCI or as a bridge to advanced therapy in the US since their FDA approval in 2008. Here, we describe a series of three cases of the Impella 5.5 pump utilized for perioperative support in acute on chronic heart failure for major abdominal surgery.

Methods: Each patient was transferred from an outside hospital for higher level of care and whose transplant or durable VAD workup was notable for abdominal mass or concern for malignancy.

Case 1 (Laparoscopic Cholecystectomy)
A 62 yo woman with concern for porcelain gallbladder planned for laparoscopic cholecystectomy. Her preoperative left ventricular ejection fraction (LVEF) was 20% with moderate RV dysfunction and she required moderate doses of epinephrine, milrinone, and vasopressin for hemodynamic support. Impella 5.5 implantation was performed on the day of surgery. Drip support remained stable in the postoperative period, but post-Impella urine output was significantly improved. Following recovery from cholecystectomy and physical rehabilitation on Impella, she underwent an uncomplicated heart transplantation.

Case 2 (Open Retroperitoneal Mass Resection, Cecectomy, Appendectomy, Ileocolonic Anastomosis)
A 57-year-old man with retroperitoneal mass and likely metastatic disease to liver and spine. His preoperative LVEF was 10% with moderate right RV dysfunction and required high dose milrinone for support. Impella 5.5 implantation 48 hours prior to resection with recovery of AKI prior to surgery. After 3 weeks of recovery and rehabilitation with continued Impella support, he underwent durable LVAD placement.

Case 3 (Robotic Adrenalectomy)
A 49-year-old man whose durable VAD workup found an adrenal incidentaloma with elevated plasma normetanephrine and urine metanephrines, suspicious for pheochromocytoma or malignancy requiring surgical removal. His preoperative LVEF was 10% with mildly reduced RV function and he required high dose epinephrine, milrinone, and vasopressin for support. Patient underwent Impella 5.5 implantation two days prior to noncardiac surgery. He remained on inotropes at stable doses postoperatively. His urine output and creatinine improved significantly along with decreased diuretic requirement, reduced LV distension on TTE and down trending estimated LVEDP. Two days later he underwent uncomplicated robotic adrenalectomy under general anesthesia.

Results: N/A- Medically Challenging Case Report

Conclusions: This small case series suggests the feasibility of preoperative Impella implantation for cardiac support in major abdominopelvic surgery. While noncardiac surgery in patient with depressed cardiac function has been more frequently reported with Intra-Aortic Balloon Pump support, there are limited reports of Impella-assisted support, focused on the use of smaller percutaneously placed Impellas [2,3] that provide less support and associated with more complications with use for longer than 24-48 hours.

While both implantation and removal of the Impella 5.5 pump requires an operative technique, it offers several advantages. First is the added hemodynamic support (over 5L of flow), useful in patients with more significant cardiac dysfunction. The axillary approach additionally facilitates improved patient mobility and decreased hemolysis allowing for prolonged use [4]. These advantages allow us to use the device as a bridge to facilitate non cardiac surgery, minimize perioperative risks and then bridge to transplant or VAD in patients who would not tolerate device wean given their underlying cardiac dysfunction. We observed signs of improved renal function most uniformly. Not all patients demonstrated decreased inotropes, likely due to biventricular dysfunction. There was no significant bleeding despite the use of heparin purge solution. As Impella 5.5 use increases, more study is needed to demonstrate safety and utility in this setting.

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Critical Care 22 - A Delayed Tamponade Masquerading as a STEMI One Week Post-CABG

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Introduction: This is a case of a patient who presented for a multi-vessel CABG which was complicated by delayed tamponade that was presumed to be a STEMI.

Methods: This is a 61-year-old male with a history of AF, on apixaban, who presented to BWH with multivessel CAD. He had a four-vessel CABG and LAAL on September 21st, including LIMA to LAD as well as SV to D1, RI, and RPL. Prior to discharge, CT showed moderate circumferential pericardial effusion which was stable from intraoperative TEE. Otherwise, his clinical course was uneventful, and he was discharged home on postoperative day six.

Within 24 hours of discharge, he developed fatigue, nausea, and syncope, and presented to an outside hospital with ST elevations. He received ticagrelor and was taken for left heart catheterization, which demonstrated a patent LAD with additional grafts unable to be visualized. Due to concern for graft occlusion, he was transferred back to BWH on 7 ug/kg/min of dopamine. On admission he was noted to be in cardiogenic shock with a lactate of 8.7 and a new acute kidney injury with a creatinine of 2.7. On examination his heart rate was in the 50s, and on ECG there were ST elevations apparent in the inferior and lateral leads. A PA catheter showed PA pressures of 40/20 mmHg, a PCWP of 20 mmHg, and a CVP of 20 mmHg. Despite concern for cardiogenic shock from graft occlusion, bedside TTE demonstrated normal to hyperdynamic left ventricular function without evidence of RWMAs. However, a large posterior pericardial effusion was noted along with right atrial collapse indicating an elevated intrapericardial pressure. He was taken for pericardiocentesis with the cardiology team on the night of admission, postoperative day eight. An 8.3 Fr sheath was inserted subxiphoid, to an opening pressure of 18-20 mmHg and 510 mL of dark serosanguinous fluid, with a closing pressure of 1 mmHg. A drain was placed.

After drain placement there was resolution of the cardiogenic shock. The lactate and renal function trended towards normal within three days. The drain had a decreased output of less than 50 mL over 12 hours on postoperative day 13. It was accidentally self-removed on day 14, with repeat TTE finding a small circumferential pericardial effusion without hemodynamic compromise. The patient remained asymptomatic and stable. He was started on colchicine for pericarditis as well as amiodarone, aspirin, a statin, and a beta-blocker. He was discharged home once again on day 15.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: In one study of patients receiving cardiac surgery, effusions were present in 84%. The majority appeared by postoperative day two, reached maximum size by postoperative day ten, and resolved within one month (1). The reported incidence varies but has likely declined since this study.

In a larger and more recent retrospective survey, only 1% had moderate or large effusions, but of those, 75% met criteria for tamponade – especially in females, those who have had valvular surgery, and the pre-operatively anticoagulated (2).

Both echocardiography and hemodynamics play a key role in this surveillance. In a study of 16 patients undergoing pericardiocentesis, 12 had evidence of right ventricular and/or atrial collapse as well as hemodynamic evidence of tamponade. These patients' hemodynamic parameters improved as right ventricular diastolic collapse disappeared; cardiac output improved afterward, followed by right atrial collapse. Another 3 patients had neither collapse nor hemodynamic compromise, and these patients' hemodynamics did not improve. A single patient had hemodynamic compromise but no collapse. The sensitivity of right-sided collapse as a marker of tamponade was 92%, with 100% specificity, 94% accuracy, and 100% predictive value (3). However, more data is needed.

The European Society of Cardiology published a detailed triage strategy for the management of tamponade in 2014. Specifically, they state that drainage is indicated for all diagnosed tamponades. If the patient is hemodynamically stable, the procedure can be performed within 12-24 hours after a routine workup. On the other hand, immediate pericardiocentesis is recommended if there is instability or if their stepwise scoring system results greater than 5. This system adds a score for etiology, clinical presentation, and imaging. Further, urgent surgical treatment is indicated in the setting of type A aortic dissection, ventricular free wall rupture, trauma, purulence, and loculation.

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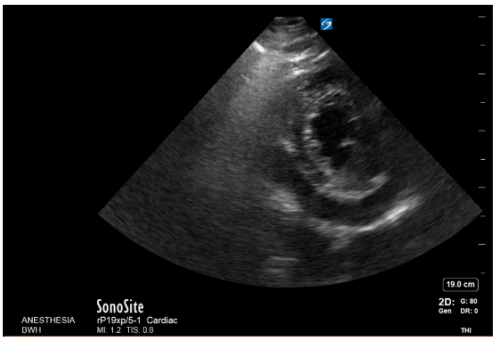

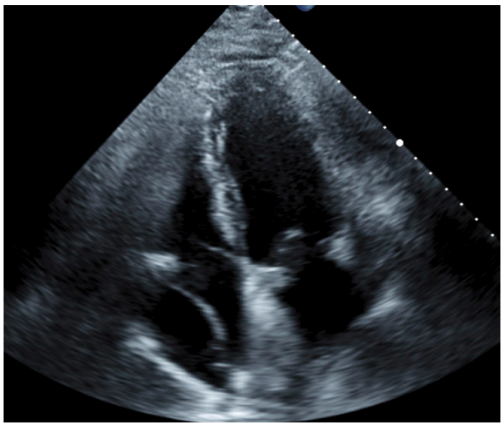

Critical Care 23 - A Severe Case of Acute Generalized Exanthematous Pustulosis Resembling Septic Shock

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Introduction: We present the case of a woman admitted to the ICU with concern for septic shock who developed an acute diffuse erythematous rash with pustules, who was found to have Acute Generalized Exanthematous Pustulosis (AGEP) as the underlying cause of her distributive shock.

Methods: A 75-year-old female resident of a skilled-nursing facility presented initially to the hospital with shortness of breath and was found to have hypercapnic respiratory failure which improved with non-invasive positive pressure ventilation. Her medical history was significant for obstructive sleep apnea, chronic kidney disease, and recurrent urinary tract infections.

On hospital day 5, she developed a fever and leukocytosis with elevated lactate and procalcitonin, prompting the initiation of vancomycin and meropenem for possible infection. The following day, she was transferred to the ICU with increasing oxygen requirements and hypotension concerning for septic shock. She was started on vasopressors and clindamycin was added. She developed worsening renal function and increasing vasopressor requirements. Serum cortisol was within normal limits. Her leukocytosis was notable for neutrophilic predominance, without peripheral eosinophilia. She subsequently developed a rash that began in the groin and spread to the trunk and extremities with diffuse erythematous plaques and pustules.

Dermatology was consulted, and a shave biopsy was performed given concern for a complex drug eruption. Imaging of the chest and abdomen, and cultures from sputum, blood, and urine remained negative for any infectious processes. With drug eruption increasingly higher on the differential, antibiotics were discontinued, and she was started on systemic corticosteroids. These interventions led to a dramatic improvement of her rash, renal dysfunction, and hemodynamics, and she was titrated off vasopressors within 36 hours. The shave biopsy demonstrated neutrophilic dermatitis, supporting a diagnosis of acute generalized exanthematous pustulosis.

Acute Generalized Exanthematous Pustulosis (AGEP) is an acute cutaneous reaction characterized by diffuse edematous erythema that evolves into areas with pinpoint subcorneal pustules and plaques, often affecting the flexural skin folds. It is typically triggered by medications (most commonly antimicrobials). AGEP is generally associated with fever, leukocytosis with neutrophilic predominance, and a milder acute hypersensitivity reaction that resolves after the cessation of the offending agent. In rare instances, such as in our case, AGEP can lead to a more severe shock state that can present similarly to septic shock.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Acute Generalized Exanthematous Pustulosis (AGEP) is an acute hypersensitivity reaction that is often triggered by medications such as antimicrobials (1). AGEP typically presents in a milder fashion that resolves with removal of the offending agent (2). In rare instances it can precipitate a distributive shock state, mimicking the presentation of septic shock. As such, it should be considered in cases where a patient in an unexplained distributive shock state develops a rash after antibiotic administration, and consideration should be given to cessation of such medications and the initiation of systemic steroid therapy (3).

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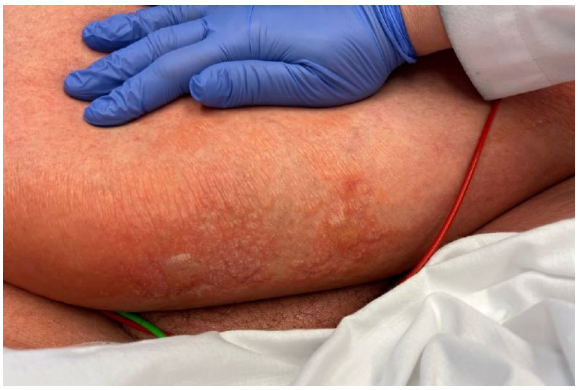

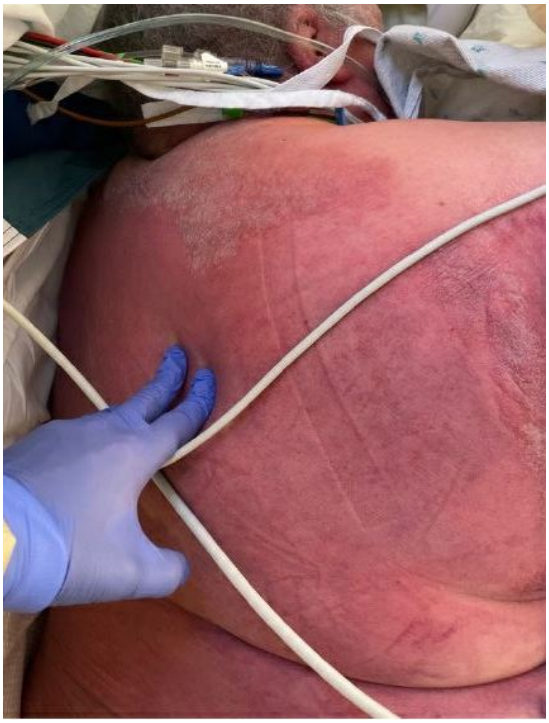

Critical Care 24 - Accidental ingestion of drain cleaner in an elderly patient

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Introduction: Caustic ingestion is a medical emergency that has the potential to cause life-threatening conditions such as airway compromise and gastrointestinal perforation. It poses a serious medical problem that is associated with high morbidity. In 2020, over 2.1 million toxic exposures were reported in the United States, with 8% attributed to household cleaning products.[1] The majority of pediatric cases are accidental, whereas most adult cases are intentional.[2] Here, we present a case of accidental caustic ingestion in an elderly male.

Methods: An 81-year-old man arrived in the emergency department with a 1-day history of vomiting, inability to tolerate secretions, voice changes, and difficulty breathing. His medical history included hypertension on enalapril, coronary artery disease, diabetes, and alcohol use disorder on monthly naltrexone injections.
On arrival, he was noted to have copious secretions requiring frequent suctioning. Flexible laryngoscopy performed by the otolaryngologist revealed significant edema with mucosal sloughing throughout the hypopharynx. Given the patient's tenuous respiratory status, inability to manage secretions, and edematous oropharynx, he was nasally intubated with a 6.5mm ETT under fiberoptic guidance. The nasal ETT was secured with a silk suture to the left naris, and the patient was subsequently transferred to the ICU for further management.

Initial care in the ICU was focused on airway edema with a 24-hour steroid course. The differential diagnosis at the time of admission included angioedema, a dermatologic process, or severe fungal infection. The patient was noted to have skin darkening in the pattern of drip marks on his chin and right thigh, prompting high suspicion of exposure to a caustic substance. Further information gathered from the patient's wife revealed recent alcohol relapse; she identified a black bottle of drain cleaner upon closer inspection of his alcohol bottles, thereby confirming the diagnosis of unintentional caustic ingestion.

The gastroenterology service was emergently consulted for further management. CT imaging with IV contrast of the chest was obtained with no evidence of esophageal perforation. A bedside esophagogastroduodenoscopy (EGD) was performed, revealing an edematous uvula, clots and mucosal sloughing in the posterior oropharynx, and grade 1 caustic injury to the proximal esophagus. Although the nasal ETT was sutured to the naris, it became dislodged on hospital day 5 in the setting of copious secretions and mucosal saponification. An urgent bedside tracheostomy was performed, thereby establishing a definitive airway that would not be compromised by the sequelae of caustic injury to the oropharynx. The remainder of the patient's hospital course was uneventful, marked by the introduction of a speaking valve, gastric tube placement, and discharge to a long-term acute care facility on hospital day 26.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The ingestion of a caustic substance can cause damage to the aerodigestive tract. The degree of injury is determined by the substance type, amount, and duration of exposure. Injury following the ingestion of a strong alkali, such as drain cleaner, begins shortly after exposure. It is characterized by liquefactive necrosis, and transmural injury can lead to perforation.[2]

Airway management is critically important, and initial assessment should begin with X-ray imaging of the chest and abdomen to assess for evidence of perforation. EGD is the gold standard procedure to assess the extent and severity of caustic injury and should be performed within the first 24 hours, ideally within 6 to 12 hours following ingestion.[2] In the absence of perforation, which would require surgical intervention, supportive care involves respiratory support, fluid resuscitation, and pain control. Development of esophageal strictures is the most common long-term sequelae, with risk increasing with higher grade of injury.[2]

Our elderly patient's clinical presentation was not initially concerning for caustic injury. A high index of suspicion is required in the setting of an unclear clinical picture with signs and symptoms that are suggestive of caustic ingestion. Respiratory complications following ingestion may result in upper airway edema, and the potential for extensive oropharyngeal damage may ultimately require a tracheostomy. Early recognition of a caustic ingestion in patients of any age is imperative in decreasing morbidity.

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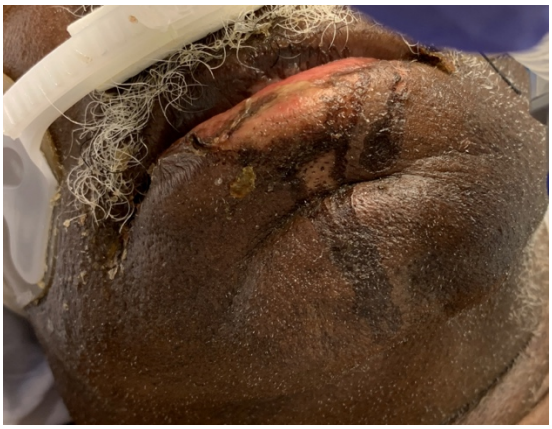

Critical Care 25 - Acute liver failure after Ross Procedure complicated by RV failure and SAM physiology

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Introduction: Acute liver failure after cardiac surgery is associated with high mortality. Ischemic liver injury is the most common etiology. Early identification is critical as progression to fulminant liver failure is often lethal. We present a case of severe early ischemic liver injury (SEILI) after Ross procedure complicated by postoperative RV dysfunction and concurrent systolic anterior motion of the mitral valve (SAM).

Methods: 46M s/p TAVR presented with prosthetic valve stenosis and underwent a Ross procedure. Calcific scar was observed encasing the subvalvular mitral valve apparatus, without apparent LVOT post bypass. TEE is otherwise notable for normal biventricular function on low dose inotropes. Upon ICU admission, the patient was hypoxic and hypotensive with elevated filling pressures. Bedside TTE showed moderately reduced RV function requiring escalation of inotropes. The patient was extubated, however requiring HFNC. That evening, an acute rise of LFTs to the 1000s was observed (Table 1). Liver ultrasound was unremarkable except for hepatic steatosis. Hepatitis serologies were negative. TTE now showed severe SAM with an LVOT gradient of 47mmHg. RV function remained moderately reduced with severe TR and high RA pressures. The cause of SAM is thought to relate to RV dysfunction (leading to underfilling of LV) and relative immobility of the subvalvular mitral apparatus causing dynamic LVOT obstruction. The coexistence of RV failure and SAM is a clinically challenging situation with competing hemodynamic goals. In an effort to lessen SAM, inotropes were discontinued. Diuresis was started to offload the dilated and hypokinetic RV while the LVOT gradient was carefully monitored by repeated bedside ultrasound. Despite improving hemodynamics, LFTs continued to worsen. The presumed etiology of acute liver failure was thought to be ischemic hepatitis, in addition to RV failure and concomitant LVOT obstruction. On POD 5, ALT and AST began down trending after peaking at 4,383 U/L and 6,219 U/L, respectively. The patient was discharged from ICU and eventually home on POD 16 with complete resolution of liver dysfunction.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Mild transaminitis following CPB is common, typically peaking at POD 3 1. The incidence of severe early ischemic liver injury after cardiac surgery is rare with limited mention in the literature2. SEILI is described as a rise in ALT above 500 IU/l within 48 hours of insult with a concomitant reduction in liver synthesis and a mortality of 65% 3. The liver is sensitive to hemodynamic perturbations, and this was highlighted in our patient who's dynamic LVOT obstruction/SAM with concurrent RV failure precipitated repeated ischemic injuries on a background of hepatic steatosis. Management is purely supportive and minimizes additional stress from ischemia, hemorrhage, or sepsis. In our case, judicious hemodynamic management with repeated TTE assessment avoided further complications leading to resolution of liver failure.

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Table 1

Labs	POD 0	POD 1	POD 2	POD 3	POD 4	POD 5	POD6	POD 7
PT	15.8	15.4	20.1	22.8	25.6	25.0	26.8	24.6
INR	1.3	1.2	1.7	1.9	2.3	2.2	2.4	2.1
ALT	23	35	1220	2962	4383	3243	2494	1802
AST	88	153	1828	4110	6219	2641	1485	915
Dbili	0.3	0.3	0.6	1.2	2.1	2.4	2.3	2.3
IBilli	0.4	0.4	0.4	0.6	1.2	1.3	1.4	1.5
TBili	0.7	0.7	1.0	1.8	3.3	3.7	3.7	3.8
Lactate	12.7	6.0	5.6	3.3	3.4	3.1	3.1	2.2
NH3					206	153	147	72

Critical Care 26 - Acute Promyelocytic Leukemia Presenting as Subdural Hematoma Treated with Middle Meningeal Artery Embolization

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Introduction: Patients with Acute Promyelocytic Leukemia (APL) present with bleeding diatheses due to leukocytosis, thrombocytopenia, and hypofibrinogenemia. The most common cause of treatment failure is early death from bleeding-related complications during the induction of chemotherapy. [i] [ii] While the ability to induce remission in these patients has improved, the initial management of bleeding-related complications continues to present a clinical challenge, especially in cases where bleeding requires procedural control and comes with additional risks. There has been a growing interest in middle meningeal artery embolization (MMAE) as an interventional technique for treatment of chronic subdural hematoma (SDH). It seems an attractive option compared with open surgical techniques in patients with a high risk of bleeding and is currently being evaluated in clinical trials. [iii] [iv] We present a case here of a patient who presented with subdural hematomas, was diagnosed with APL, then developed worsening subdural hemorrhage during treatment induction and was successfully treated with bilateral MMAE.

Methods: A 23-year-old woman without any history of trauma presented to a community hospital with the worst headache of her life accompanied by dizziness, muffled hearing, and blurry vision. Workup including a CT scan showed a left 4.7 mm mixed density subdural hematoma and a right 1 cm mixed density subdural hematoma without midline shift, both thought to be acute on chronic. Bloodwork showed a platelet count of 30,000, leukocytes of 10,300 with 15% blasts, and a hemoglobin of 7.4, prompting transfer to our medical center. Repeat scan after transfer showed stable findings, and the patient was admitted for further management and hematology consultation. Treatment was initiated with ATRA induction with subsequent improvement in platelet count. Arsenic was started thereafter. On day 3, a worsening headache prompted repeat imaging which showed worsening subdural hematoma and new midline shift. These findings lead to urgent neurosurgical evaluation and transfer to the ICU. While initially managed with multiple transfusions, the patient subsequently underwent successful bilateral MMAE and was able to undergo the remainder of induction without incident.

Conclusions: Intensivists are often faced with complex clinical scenarios requiring rapid risk-benefit analysis and timely treatment. APL is a readily treatable malignancy with a high complication rate early in treatment including risk of life-threatening bleeding. In patients with intracranial hemorrhage (ICH), close monitoring for neurologic deterioration as well as prompt intervention is key. Further, in these patients, starting treatment for APL poses an additional risk. Prior described cases have included the management of bleeds following treatment of APL-related central venous thrombosis. [v] Here we recount a case where bilateral MMAE was used to manage subdural hematoma in the setting of APL. This case highlights the importance of multidisciplinary care of critically ill patients. Intensivists should be familiar with unusual presentations of APL including ICH, be facile with the management of bleeding complications that arise from induction therapy and should be familiar with rapidly expanding use of emerging interventional techniques.

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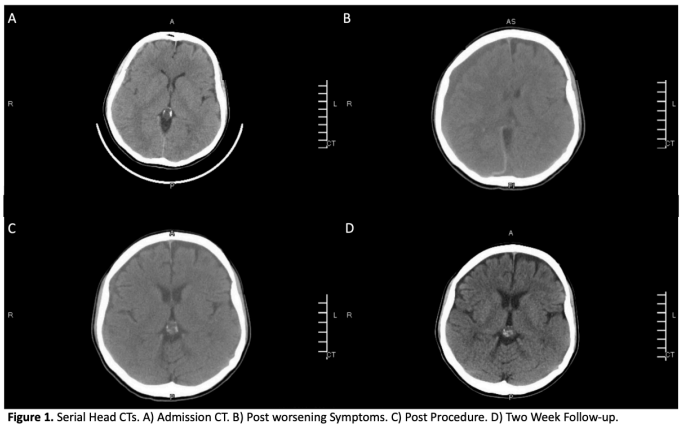

Figure 1. Serial Head CTs. A) Admission CT. B) Post worsening Symptoms. C) Post Procedure. D) Two Week Follow-up.

Critical Care 27 - Airway Management in Cardiac Tamponade

Ashley Lipscomb[1], Habib Srour[1], Jason Huston

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Introduction: The development of cardiac tamponade in the critically ill patient is often confounded by the presence of multiple comorbid conditions and multifactorial shock. The management of concomitant respiratory failure under such circumstances presents an interesting challenge, as the initiation of positive pressure ventilation can quickly result in total hemodynamic collapse and cardiac arrest. Thus arises the challenge of the physiologically difficult airway, for which Srour et al. previously proposed an algorithmic approach, emphasizing spontaneous, awake ventilation, initiation of inotropes, and optimization of respiratory physiology: referred to as the SAVIOR algorithm.[1] Herein, we present the utility of the SAVIOR algorithm in the management of a critically ill patient presenting with comorbid hypervolemia, hypoxic respiratory failure, and cardiac tamponade.

Methods: A 55 year old male patient with decompensated cirrhosis underwent liver transplantation complicated by intra-operative cardiac arrest requiring cardiac massage via a left hemidiaphragm incision. He was ultimately stabilized without further complication and discharged home following routine recovery. Eight weeks later, the patient was readmitted with progressive hypervolemia and dyspnea. On admission, he was found to be hypoxic and hemodynamically unstable. Diagnostic evaluation was remarkable for large bilateral pleural effusions on CT chest and echocardiographic signs of tamponade including right and left ventricular diastolic collapse. Pleural effusions were treated with bilateral thoracostomy drains, which were clamped following drainage output of 1500mL to prevent re-expansion pulmonary edema. Multiple attempts at pericardial drain placement were unsuccessful due to the patient's worsening hypoxia with supine positioning. With continued hemodynamic deterioration requiring inotropic support and worsening respiratory failure, intubation was deemed necessary to facilitate fluoroscopy-guided pericardiocentesis. With inotropic support in place, a dexmedetomidine infusion was initiated for patient comfort and increased to a rate of 1.4mcg/kg/hr. Following lidocaine topicalization of the upper airway, intubation was achieved using a 7.5mm flexible, midline tip endotracheal tube placed over a fiberoptic bronchoscope. Spontaneous, awake ventilation was maintained throughout with the patient positioned upright. Proper positioning of the endotracheal tube was confirmed under direct fiberoptic visualization. Following securement of the endotracheal tube, spontaneous ventilation was maintained with initial ventilator settings of pressure support 0mmHg and PEEP 0mmgHg. Pressure support was slowly titrated up to 8mmHg. The patient's clinical status remained stable on inotropic support, and he proceeded to the cardiac catheterization lab for successful placement of a pericardial drain. Post-procedure, plural and pericardial effusions were adequately drained, and the patient's clinical status improved. He was ultimately discharged home in stable condition.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: It is well established that positive pressure ventilation may be poorly tolerated in cardiac tamponade, however when complicated by respiratory failure and challenges in definitive pericardial drainage, intubation may become inevitable. Prompt recognition of this clinically challenging course can allow for optimization of cardiovascular and respiratory mechanics, and ultimately a favorable outcome. The SAVIOR algorithm provides a standardized approach to the physiologically difficult airway, where optimization of cardiac preload, afterload, and respiratory mechanics is of critical importance.

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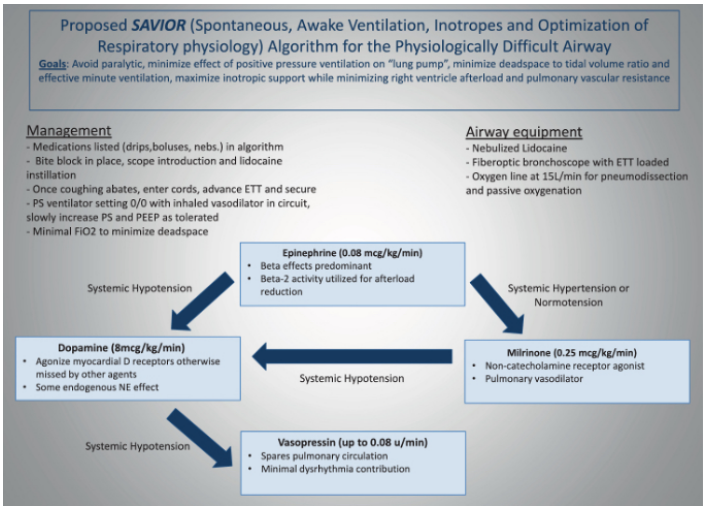

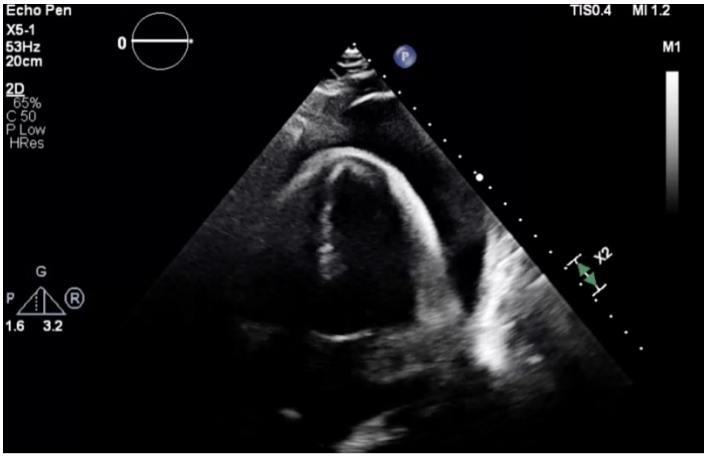

Critical Care 28 - Altered Mental Status from Dietary Supplements

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Introduction: A 48-year-old Spanish speaking married female with no psychiatric history was admitted to the ICU after emergent surgery for a right ureteral stent placement. Over the next few days, she becomes agitated and delirious without an obvious cause.

History of Presenting Illness: The patient presented to the Emergency department with a chief complaint of right flank pain and leukocytosis of 22. Other labs on admission are WBC 22.2; H/H: 13.9/43.4; MCV 81.7; Plt 220. The patient states that she got home from working in the kitchen and noticed a sharp Right -sided flank pain that radiated to her Right ovary. She took a shower and acetaminophen and tried to sleep but the pain was too great. She states the pain never goes away, and occasionally swells. She denies hematuria, dysuria, fevers, nausea, vomiting, diarrhea. She was found to have a 6mm obstructing stone in the Right ureter. She was admitted to hospital and started medical management with medical expulsive therapy with Intravenous hydration and Tamsulosin. However, within hours of admission she decompensated and became hypotensive and tachycardic. Thus, the decision was made to take her to the operating room for emergent ureteral stent placement.

Past Medical History: No significant past medical history.

Methods: Course in Hospital: On Admission to the ICU the patient remained hypotensive requiring vasopressors and was diagnosed with Septic Shock. She also started on Antibiotics. Her urine culture grew Proteus, and initially she was started on Vancomycin and Cefepime, but was switched to Meropenem. The patient appeared to respond to IV antibiotics with decreasing vasopressor requirements and a normalizing lactate level. On Day 2 of admission to the ICU the patient began to be confused. Oriented only to person. Our differential diagnosis included sepsis, AKI, or ICU delirium. Over the next 24 hours her confusion progressed to agitation, and she started pulling out IV lines and her NG tube. She required Haldol and Seroquel but there seemed to be little effect. We initiated a workup for Altered Mental status including an Ammonia level, TSH and a CT head all of which were negative. Upon further questioning of her family, we found out that prior to admission the patient had been taking a medication known as Reumofan at home. We therefore began to suspect that the patient may have adrenal insufficiency due to the abrupt cessation of glucocorticoids that are contained in Reumofan. We started her on Hydrocortisone 100mg then 50mg Q6 hours. Within 48 hours her confusion, agitation and disorientation resolved. She was given a steroid taper over the course of 7 days. She was discharged home one week later.

Results: Not Applicable- Medically Challenging Case Report

Conclusions: What is Reumofan? Reumofan also known as Reumofan Plus and Reumofan Premium are products which are marketed as natural dietary supplements. They contain several potential harmful active pharmaceutical ingredients[1]. FDA laboratory analysis revealed that Reumafan contains the undeclared and potentially harmful prescription drug ingredients dexamethasone, diclofenac sodium and methocarbamol.

What is the nature of Neuropsychiatric Presentation of Adrenal Insufficiency? It is probably still not commonly appreciated that patients with Adrenal insufficiency may present with psychiatric symptoms[3,4]. There are three possible theories for the neuropsychiatric symptoms seen with adrenal insufficiency.

1. Electrolyte and metabolic abnormalities. Hyponatremia occurs commonly in patients and may contribute to cognitive changes and encephalopathy. Our patient did have hyponatremia. [5]

2. Glucocorticoid deficiency. Glucocorticoids have been shown to be essential for maintain prefrontal cortical cognitive function.[6]

3. Increased Endorphins. In response to decreased glucocorticoids the anterior pituitary synthesizes endorphins. Increased endorphin level and psychosis has been suggested based on the findings of elevated Cerebral spinal fluid endorphins in schizophrenia patients.[7]

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Critical Care 29 - An Unusual Case of Acute Heart Failure Following Hyponatremic Seizure

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Introduction: Takotsubo cardiomyopathy (TCM), coined by Sato et al in 1990, is a syndrome characterized by transient left ventricular (LV) dysfunction, EKG abnormalities, and elevations in cardiac enzymes that may mimic acute coronary syndrome.[1] Thought to be caused by catecholamine toxicity on cardiac myocytes, TCM classically affects post-menopausal women after significant emotional or physiologic stress.[2] While an association between acute neurological disease and TCM has been established, the development of TCM after seizure is uncommon.[3, 4] We describe an unusual case of TCM following hyponatremic seizure in a patient without typical risk factors.

Methods: A 22-year-old male with ADHD presented to the emergency department after he reported feeling acutely unwell and experiencing a possible seizure. The patient's parents reported that he was previously healthy and denied illicit drug use, seizures, or family history of cardiac disease. He was adhering to a low sodium diet to enhance athletic performance. He was ill appearing and cyanotic, with frothy secretions and vomitus in his oropharynx. He was intubated for airway protection. Laboratory workup revealed severe metabolic derangements (pH 7.04, pCO_2 68, bicarb 18, Na 112, K 3.3, Cl 82, lactate 15.1) and an elevated CK and troponin; CBC and urine toxicology screen were unremarkable. CXR showed diffuse pulmonary opacities, and EKG showed sinus tachycardia with frequent PVCs, wide QRS, and ST elevations in the inferior, septal, and lateral leads. He became progressively hypoxic despite prone positioning, and he was transferred to a quaternary care center with extracorporeal membrane oxygenation (ECMO) capabilities. On arrival at the intensive care unit (ICU) a transthoracic echocardiogram (TTE) demonstrated severe LV systolic dysfunction (LVEF 20-24%) with global hypokinesis and akinesis of the basal to mid inferior and septal segments. CT head showed diffuse edema. He was started on broad spectrum antibiotics. A lumbar puncture did not reveal CNS infection. He initially required high dose vasopressors. Left and right heart catheterization showed normal coronary arteries, pulmonary capillary wedge pressure of 21 mmHg, and a cardiac index of 2.75 l/min/m^2. His severe hyponatremia was treated with 3% saline. His mental status gradually improved. His shock and respiratory failure resolved without the need for inotropes, mechanical circulatory support, or ECMO. He was extubated on day 3. A repeat head CT showed interval resolution of the diffuse edema; MRI brain showed no significant abnormalities. On day 5 his electrolytes had normalized and repeat TTE showed interval improvement with mild LV systolic dysfunction; he was discharged home. Several months later he reported a return to baseline health, and cardiac MRI showed no abnormalities.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: This case describes a chain of events by which a low-sodium diet resulted in severe hyponatremia causing cerebral edema and a grand mal seizure. We suspect that this seizure led to an inverted type Takotsubo cardiomyopathy with cardiogenic pulmonary edema and acute respiratory failure. Hyponatremia is a well-established cause of seizures, however it is frequently associated with malignancy, renal disease, medication side-effects, neurohormonal diseases, or primary polydipsia or sodium avoidance due to psychiatric disorders- none of which applies to this patient.[5, 6] The majority of TCM caused by acute neurological disease is due to subarachnoid hemorrhage with seizures being a less common cause.[2, 7] While the classical subtype of TCM is most commonly reported in cases of seizure induced TCM, the inverted subtype, as seen with this patient, has been described.[8] This patient lacked the typical risk factors associated with development of TCM including female gender (80-90%), age 50-70, coronary artery disease, peripheral vascular disease, and chronic psychiatric or neurologic disorders.[1, 4, 9, 10, 11] There are no proven therapies for TCM, and patients may require aggressive therapies including mechanical ventilation, vasopressor/inotrope support, mechanical circulatory support, or ECMO; despite this, most patients with TCM will make a full recovery.[1, 8, 12] This case adds to a growing body of literature describing seizure-triggered TCM, and the intensivist should be cognizant of this rare but reversible complication.

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Critical Care 30 - Case Report: Direct Lung Injury, Acute Respiratory Distress Syndrome, and Myocardial Dysfunction Resulting from a Lightning Strike

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Introduction: A lightning strike is a rare injury with multi-system involvement. We present a case of a 52-year-old female who sustained burns from a lightning strike and developed cardiovascular and respiratory sequelae. We discuss the diagnosis, treatment, and progress of the patient.

Methods: Our patient was struck by lightning while crossing a field during a thunderstorm. She was found unresponsive and in ventricular fibrillation. Spontaneous circulation returned after cardiopulmonary resuscitation and one cycle of defibrillation. Superficial partial thickness burns were seen on the anterior chest wall (4%), abdomen (8%), right palm (0.5%) and right posterior leg (5%). Computed tomography of the thorax (Figure 1A and B) and chest radiographs (Figure 2A) showed ground glass opacities in the left upper lobe with patchy consolidation and septal thickening, reported as mild lung contusion and hemorrhage.

The patient was admitted to the burns intensive care unit after endotracheal intubation for a low Glasgow Coma Score. She required inotropic support to maintain a mean arterial pressure of 65 mmHg. She developed Acute Respiratory Distress Syndrome (PaO_2/FiO_2 ratio of 67) within 24 hours. This was managed with lung-protective ventilatory strategies, prone positioning, and paralysis. Antibiotics were escalated from intravenous co-amoxiclav to piperacillin and tazobactam.

Supportive management included maintaining euvolemia and adequate urine output, early enteral feeding, continuous cardiac monitoring with telemetry, and targeted temperature management (35-36ºC). Operative treatment involved scrub-down of burn wounds and dressing on day 5 of injury followed by interval burns excision and skin grafting on day 9.

On day 4, her ventilator settings were weaned down, and she no longer needed inotropic support.

On day 6, a trans-thoracic echocardiogram showed a left ventricular ejection fraction (LVEF) of 35-40%, left ventricular diastolic dysfunction, akinetic apical segments and hyperkinetic basal segments. Impression was likely stress-induced cardiomyopathy.

On day 7, extubation was attempted but failed due to acute pulmonary edema. Blood and sputum cultures yielded no growth. Bronchoscopy with alveolar lavage on day 8 showed patchy erythema with no mucosal sloughing (Figure 3), suggestive of airway injury secondary to lightning strike. Lavage fluid was clear, and cytology was unremarkable. She was extubated to non-invasive ventilation on day 10 of injury after her fluid status was optimized.

On day 11, she was discharged to the ward, and back home on day 58 after intensive rehabilitation.

A repeat echocardiogram done 4 months post-injury showed a normalised LVEF (65%), with subtle remnant hypokinesia of the apex; thus, no coronary evaluation was done.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Reports of lung injury resulting from electrical shocks, especially lightning strikes, are scarce. Direct lung injury to the parenchyma by electricity results in infarction and coagulation necrosis - theorised mechanisms include vascular spasm, electroporation of cell membranes, and Joule heating causing irreversible denaturation of cell macromolecules. Indirect lung injury described in the context of lightning includes barotrauma and penetrating injury from shrapnel. Lung consolidation or contusions, hemorrhage, and effusions are seen in direct lung injury from lightning. Upper lung lobe involvement, as here, has been described in previous case reports on lightning strikes.

Cardiac dysfunction from lightning injury which is well described may contribute to resulting pulmonary edema, although ARDS has been attributed to non-lightning direct high voltage lung injuries. As with our case, such cardiac dysfunction has been noted to be transient, where a repeat echocardiogram was performed 4 months post-injury.

The diagnosis of direct lung injury from electricity should be one of exclusion given its rarity, whether as a result of lightning or otherwise. Our case is one such instance resulting from lightning, with radiological features of lung contusion and haemorrhage, and associated pulmonary oedema attributed to cardiogenic and non-cardiogenic causes. While such injury is severe, as here, it may be adequately treated with supportive measures under close monitoring in a critical care setting.

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Figure 1: Computed tomography of the thorax (Figure 1A: Axial plane; Figure 1B: Sagittal plane), showing patchy consolidation and septal thickening in the left upper lobe, reported as mild lung contusion and hemorrhage.

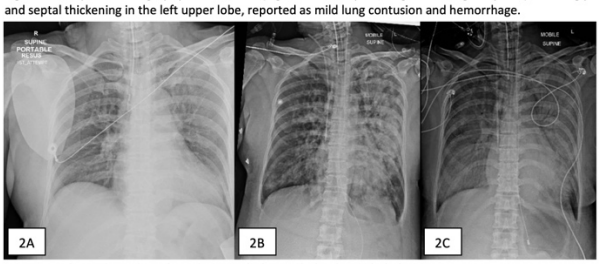

Figure 2A-C: Chest radiographs performed on Day 1 (2A), Day 2 (2B) and Day 3 (2C) of injury.

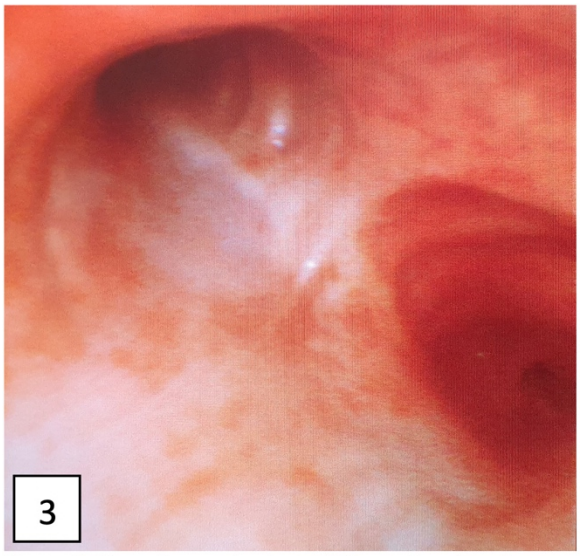

Figure 3: Bronchoscopic view of the carina showing patchy erythema of the large airways.

Critical Care 31 - Change the Tide with Octreotide: A Case of Sulfonylurea Overdose

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Introduction: Sulfonylurea toxicity is rare but can be life threatening, often leading to severe hypoglycemia refractory to treatment with dextrose. Sulfonylureas are insulin secretagogues, stimulating insulin secretion from the beta cells of the pancreas (1). We report a case of an acute sulfonylurea (Glimepiride) overdose and its successful management.

Methods: A 58-year-old male with a past medical history of depression, type II diabetes mellitus, and hyperlipidemia presented to the emergency department after an intentional overdose of glimepiride. Patient reported an ingestion of forty tablets of 2 milligrams (mg) glimepiride with suicidal ideation. Patient received 100 mg of activated charcoal and 25 grams of 50% dextrose in the ED. On the initial complete metabolic panel, the patient's glucose was 139 mg/dl. However, within an hour, his glucose on next accucheck decreased to 70 mg/dl. He was subsequently admitted to ICU for further management and frequent glucose monitoring. Dextrose infusion was continued and 75 micrograms of subcutaneous octreotide was administered every 8 hours. Patient resumed a normal diet after 6 hours and dextrose infusion was discontinued. Octreotide was discontinued after 3 doses and the patient was safely discharged to the inpatient psychiatric unit for management of major depression.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Symptoms of sulfonylurea overdose and hypoglycemia include but are not limited to weakness, diaphoresis, palpitations, confusion, seizure, altered mental status, and death. Dextrose administration alone in patients with sulfonylurea overdose can be detrimental since dextrose stimulates the pancreas to release more insulin, further worsening hypoglycemia (2). This case highlights that while patients with sulfonylurea overdose may initially appear well there is a high risk of delayed presentation with hypoglycemia. Also, timely administration of octreotide is very important in patients presenting with sulfonylurea overdose because octreotide decreases the incidence of recurrent hypoglycemia by preventing insulin secretion. With an increase in the use of sulfonylurea for glycemic management in diabetes, it is imperative for the intensivist to recognize the danger of delayed hypoglycemia and role of octreotide in management of sulfonylurea overdose.

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Critical Care 32 - Clevidipine Associated Refractory Hypoxemia in a Cardiac Surgery Patient

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Introduction: Afterload reduction plays an essential role in the management of acute decompensated heart failure. Dihydropyridine calcium-channel blockers (DHP-CCB) such as nicardipine and clevidipine are frequently used to accomplish this goal. These medications work by decreasing the entry of calcium into cells and preventing smooth muscle contraction. This results in the peripheral vasodilation that effectively reduces SVR. At lower doses these agents have minimal effect on the cardiac myocytes, making them attractive agents for afterload reduction. However, at higher dose, these agents lose their peripheral selectivity and can cause myocardial depression. Additionally, these agents can lead to refractory hypoxemia secondary to a blunting of the hypoxic pulmonary vasoconstrictive (HPV) response. We present a case of refractory hypoxemia in an ICU patient being treated with clevidipine, which resolved after transitioning to a different class of afterload-reducing agent.

Methods: A 68-year-old gentleman with a past medical history of hypertension, hyperlipidemia, coronary artery disease, and severe aortic insufficiency presented to our hospital for scheduled cardiac surgery. The patient underwent coronary artery bypass grafting and an aortic valve replacement. He arrived in the ICU on low-dose epinephrine and low-dose clevidipine. He was extubated on post-operative day (POD) one to high flow nasal canula (HFNC), flowing at 50 liters with a fraction of inspired oxygen of 70%. Over the next few days, the patient required a significant amount of HFNC support, greater than what was expected given his uncomplicated operative course and unrevealing work up of this hypoxemia in the ICU. Notably, our patient's post-operative chest x-ray demonstrated small bilateral pleural effusions and on POD two, he underwent thoracentesis, removing 300cc of blood from his right pleural space, without any improvement in oxygenation. Additionally, he underwent significant diuresis for a net negative of five liters over the first two post-operative days, also without any improvement in his oxygenation. He was also receiving aggressive chest physiotherapy. A transthoracic echocardiogram was performed on POD two demonstrating mild diastolic dysfunction without any intracardiac or transpulmonary shunt. The decision was made to transition him from clevidipine to a combination of scheduled intravenous hydralazine and labetalol in the morning of POD three and he was able to be weaned from 60L/90% of HFNC to 5L nasal cannula by the afternoon. The patient had no further issues with hypoxemia and had an unremarkable remaining ICU course. He was transferred to the floor on POD five and discharged from the hospital on the morning of POD eight.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Clevidipine is commonly used for afterload reduction in patients with cardiac dysfunction. However, our case highlights its potential to cause refractory hypoxemia. Normally, the pulmonary circulatory system responds to a low partial pressure of oxygen (PO2) by constricting vascular smooth muscle to shunt perfusion toward better oxygenated regions of the lung. This mechanism is known as hypoxic pulmonary vasoconstriction. Hypoxic vasoconstriction creates a favorable ventilation to perfusion ratio, maximizing pulmonary oxygen exchange. Age, pH, and the partial pressure of carbon dioxide (PCO2) are known factors affecting the pulmonary circuit's ability to vasoconstrict in response to low PO2. The mechanism of clevidipine provides a physiologic rationale for the refractory hypoxemia demonstrated in our case: a blunting of the hypoxic vasoconstrictive response. Rat models have demonstrated that extracellular and intracellular calcium flow is essential to the excitation-contraction coupling of vascular smooth muscle. Acute hypoxemia has been shown to increase both intracellular calcium and calcium entry through channels in the smooth muscle of distal pulmonary arteries. These distal pulmonary arteries are the predominant location of vasoconstriction in response to regional hypoxemia. Animal models have shown that calcium channel blockers may have a dose-dependent reduction in the hypoxic vasoconstrictive response. A case report of a patient after CABG demonstrated hypoxemia that resolved after discontinuing clevidipine. Additionally, another case report of a patient following a neurosurgical procedure demonstrated an association with clevidipine and refractory hypoxemia. Patients taking DHP-CCB were also found to have increased risk of intubation following COVID-19 infection, hypothesized to be secondary to a disruption of the HPV response. Our patient had a thorough investigation of his hypoxemia, including several interventions that did not improve his oxygenation. However, after discontinuing clevidipine we were able to rapidly wean his high-flow nasal cannula to a low-flow nasal cannula, and ultimately off oxygen support. Our patient's clinical course illustrates the need for further study of DHP-CCB-associated hypoxemia as improved understanding of this phenomenon could avoid invasive and expensive interventions, potentially reducing ICU length of stay for affected patients.

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Critical Care 33 - Diabetic Ketoalkalosis: A Case Report

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Introduction: Diabetic ketoacidosis remains the deadliest complication of type 1 diabetes mellitus. Typically, patients present with a plasma glucose > 250, serum bicarbonate < 18, blood pH <7.3, and an elevated anion gap metabolic acidosis with ketosis. Here, we present a rare case of 47-year-old female with hyperglycemia, ketosis, elevated anion gap, and profound *alkalemia.* The term diabetic ketoalkalosis has been given to patients presenting with these findings as a rare complication of diabetic ketoacidosis. As of 2014, only 30 cases have been presented in the literature.

Methods: The patient had a past medical history significant for T1DM, gastropareses, hypertension, noncompliance, and polysubstance use. She presented to the ED with a two-day history of intractable vomiting, intractable diarrhea, and altered mental status. She was combative and had dry mucosa, and kussmual respirations. Vitals: afebrile, MAP 58, HR 133 bpm, respirations >30 breaths/min and 99% on room air. Initial labs: blood glucose 354mg/dL, Na 130mEq/dL, K+ 2.7mEq/dL, Cl 62mEq/dL, HCO3 38mEq/dL, anion gap 30, Cr 2.74 mEq/dL, BUN 56mg/dL, lactic acid 7.1mmol/L, phosphorus<1.0 mEq/dL, albumin 4.2g/dL, beta-hydroxybutyric acid > 1.6. Urinalysis with high ketones and glucose. VBG pH 7.66, PCO2 36, PO2 40, and HCO3 40. ABG pH 7.93, PCO2 < 13, and PO2 116. CT chest revealed pneumonia and CT abdomen/pelvis showed dilation of the right ureter and collecting system. She was intubated after she became lethargic with continued emesis. Post intubation, she developed polymorphic ventricular tachycardia and required IV Mg. She was then placed on the hospital's standard DKA protocol and her anion gap closed within 16 hours. She was transitioned to subcutaneous insulin and extubated without complication. Repeat labs: Na 140 mEq/dL, K+ 4.0mEq/dL, Cl 97mEq/dL, HCO3 34mEq/dL, ABG: pH 7.49, PaCO2 42, HCO3 32.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: This patient had severe primary metabolic alkalosis, a secondary high anion gap metabolic acidosis unmasked by the significantly elevated lactate and ketones, and compensatory respiratory alkalosis from her Kussmaul respirations. It is likely she also had a non-anion gap metabolic acidosis from her intractable diarrhea; however, blood gases and chemistries were too deranged to calculate. The patient's severe primary metabolic alkalosis resulted from uncontrolled vomiting which led to direct loss of HCl in excess of duodenal bicarbonate loss. Her high glucose led to osmotic diuresis which caused activation of RAAS. The activation of RAAS increased sodium reabsorption, bicarbonate reabsorption, and bicarbonate production by secreting H+ ions into the renal tubules. It also caused a depletion of potassium by shifting H+ ions from the extracellular fluid into the intracellular fluid at the expense of potassium ions leading to alkalemia. This patient's kidney function was also impaired thus effecting bicarbonate clearance. Multiple other factors could have led to her profound hyporchloremic metabolic alkalosis. High anion gap acidosis causes an internal shift of chloride from the extracellular space to the interstitial fluids or into erythrocytes via the hamburger phenomenon. Chloride is thought of as a passive participant by simply replacing the negative charged ion of the outward moving bicarbonate thus exaggerating this patients alkalemia. It has also been suggested that the greater the patient's ketonemia, the more severe the chloride deficit is. Up to 30% of DKA patients will present with mixed acid base disorders. Usually, the factors that lower HCO3 levels, such as elevated lactate and ketoacid production, have a greater impact on the overall acid base equilibrium. With this patient, the typical presentation was reversed because of her emesis. The treatment of ketoalkalosis is the same as the treatment for ketoacidosis; however, potassium supplementation is needed even more judiciously. It has been stated that the patients with a pH greater than 7.65 have upwards to 80% mortality and that there are highly regulated protective transport mechanisms regulating the pH of the cerebral spinal fluid which can be overwhelmed at extremes in pH leading to death. If not for this patient's concurrent metabolic acidosis, this patient may have not survived her acid-base disequilibrium.

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Critical Care 34 - Diagnosis and Management of Submassive Pulmonary Embolism in the Postoperative Period

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Introduction: Pulmonary embolism (PE) is a significant cause of perioperative mortality. The incidence in surgical patients is five times as high as the general population[1], in part due to tissue trauma, prolonged immobility and venous stasis. Recognition and management of perioperative PE depends on a high clinical index of suspicion and its severity.

Methods: A 73-year-old male with BMI 30.8 and a history of prostate cancer and hypertension was admitted to the ICU after an abdominal wall reconstruction. On POD2, he became tachypneic and experienced O_2 desaturation to 60% requiring oxygen through a non-rebreather mask with subsequent improvement to 96%. He was found to be in supraventricular tachycardia (SVT) with rate in the 190s, which resolved with 5mg of IV Metoprolol. Although a lower extremity DVT scan was negative, CT-PE demonstrated submassive pulmonary emboli in the left upper and lower lobe, and right middle and lower lobe, and dilated inferior vena cava without signs of right ventricular dilation. Echocardiogram showed right ventricular strain, ejection fraction of 55%, and aortic dilation to 5cm. The patient was started on a heparin infusion. Given the extensive PE, he was closely monitored for the need for an embolectomy or ECMO. On POD3, he experienced confusion signifying worsening perfusion, and underwent bilateral pulmonary aspiration thrombectomy, with the removal of large bibasilar clots. Although he developed hemoptysis on POD5, requiring intubation, he was uneventfully extubated on POD7. On POD9, bridge to Warfarin was initiated, planned for a minimum duration of 6 months. Direct oral anticoagulant use was avoided due to the elevated risk of bleeding in the immediate postoperative period.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: The recognition and management of perioperative PE can present a challenge to multidisciplinary teams as the presenting signs can be confounded by multiple factors. The presentation of a submassive PE with SVT highlights the importance of a high index of suspicion and vigilant monitoring in high-risk postoperative patients. Appropriate and rapid intervention is warranted to prevent progression of disease and poor outcomes.

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Critical Care 35 - Diagnosis of Antiphospholipid Antibody Syndrome After LVAD Placement

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Introduction: Antiphospholipid antibody syndrome (AAS) is a rare autoimmune disease associated with the production of auto antibodies which promote arterial and venous thrombosis. The implantation of mechanical assist devices promotes host immune system activation and a hypercoagulable state, thus placement in a patient with a pre-existing increased risk of thrombosis can result in markedly increased morbidity and mortality if not diagnosed prior. Given potentially catastrophic outcomes with undiagnosed AAS, it's imperative to be aware of clinical and laboratory findings to keep this rare diagnosis on the differential. Here we present a patient who presented after LVAD implantation and was found to have AAS given multiple venous thromboses and a persistently elevated PTT.

Methods: A 54-year-old M with extensive past cardiac history including ICM with EF 30%, CAD with MI at 37yrs old, recurrent VT/SVT s/p BiV ICD, HTN/HLD presented to the ED following a syncopal event and increasing SOB. He remained hospitalized, during which he had several occurrences of symptomatic VF/VT with activation of his ICD, a submassive PE, and an incidentally found RIJ DVT. After IABP placement, the decision was made to place a HeartMate 3 as a bridge to transplant. His operative course was unremarkable, but post-operatively his PTT was persistently elevated and the decision was made to hold systemic anticoagulation necessary for the LVAD. Hematology noted the chronicity of the elevated PTT and that the patient had tolerated numerous interventions without significant bleeding, it was postulated an inhibitor was resulting in the falsely elevated PTT. He then developed numereous acute LE DVTs and an absence of right radial pulse with acrocyanosis. Workup revealed low Factor Xa, further validating the suspicion for the presence of a factor inhibitor. The recommendation for systemic a/c was made for the LVAD and as treatment of the numerous VTEs. Titration of the heparin drip was based on anti-factor Xa levels given falsely elevated PTT. AAS workup was carried out and the diagnosis was confirmed with markedly elevated beta 2 glycoprotein.

Results: Not Applicable- Medically Challenging Case Report

Conclusions: AAS is an autoimmune disease with factor inhibitors, including anti-beta 2 glycoprotein 1 Ab, anticardiolipin Ab and lupus anticoagulant which can cause thrombosis in any blood vessel. The development of AAS is believed to be by a 1st hit of circulating antiphospholipid Abs with underlying endothelial dysfunction combined with a 2nd hit of upregulation of beta-2 GP receptors which can be initiated by a variety of clinical stressors. Resultant impairment of NO-dependent endothelial function with activation of complement system, platelet activation and accelerated atherosclerosis causes morbidity including venous and arterial thrombosis. These thromboses can lead to increased risk of cardiac events, stroke, OB complications and valvular heart disease. To prevent these sequelae, identification of the syndrome is necessary, but often doesn't occur until after a thrombogenic event occurs. Increased thrombogenic risk occurs after LVAD placement and can result in catastrophic events, including pump thrombosis, which are unlikely to be tolerated in the acute post-cardiac surgery period. Even in patients without a preexisting hypercoagulable state like AAS it has been shown that the implanted device results in further defects in cellular immunity which could increase one's propensity to develop not only an infection but also allosensitization which is detrimental in a bridge to transplant patient. The prevalence of AAS is estimated as 40–50 cases per 100,000 individuals and thus is relatively rare, although it is likely underreported and under recognized. Our case shows the importance for timely consideration for further investigation into abnormalities in coagulation to prevent catastrophic outcomes. Given our patient's PTT was chronically elevated before his most recent admission, an earlier diagnosis of AAS could have been identified, potentially avoiding numerous post operative venous and arterial thromboses. Fortunately, post-operative identification of a falsely elevated PTT allowed the reinstatement of systemic anticoagulation prior to the development of a potentially fatal pump clot. It is known that patients with LVADs are at an increased risk of a hypercoagulable state and may result in increased morbidity. Further studies may help identify patients with risk of having potential factor inhibitors prior to assist device implantation.

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Critical Care 36 - Diagnostic and therapeutic limitations in a Jehovah's Witness receiving a Left Ventricular Assist Device

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Introduction: While previous data have suggested higher rates of morbidity and mortality amongst Jehovah's Witness patients as compared to blood product accepting patients (1), more recent data has emerged suggesting this disparity may be reduced as surgical techniques have evolved (2,3). Nevertheless, there are many unique challenges associated with the care of patients undergoing cardiac surgery or receiving mechanical circulatory support who do not accept blood products. These include both diagnostic and therapeutic limitations that preclude clinicians from making care decisions with the standard data that is typically relied on. This case details the obstacles faced in the work up of a Jehovah's Witness patient who presented with an Impella as a bridge to 'bloodless' LVAD and the neurologic and hemodynamic sequela of these therapeutic and diagnostic decisions.

Methods: A 74 year old Jehovah's Witness female with past medical history significant for CVA with left upper extremity deficits, OSA, atrial fibrillation complicated by amiodarone induced thyrotoxicosis, chronic normocytic anemia with baseline hemoglobin of 8 and COVID-19 related NICM with new LVEF of 15-20% presented from an outside hospital for bloodless LVAD placement due to progressive cardiogenic shock despite maximal medical therapy, vasopressin, milrinone and right subclavian Impella placement. In the days leading up to LVAD placement, the patient experienced mild ICU delirium but otherwise did not have neurologic changes below baseline. Intraoperatively, cardiac surgery noted that the previous Impella had developed significant clot burden in all 4 chambers of the heart, which were subsequently removed. Post LVAD insertion, intraoperative TEE noted severe right ventricular dysfunction with complete hypokinesis at the base of the right ventricle. Upon returning from the operating room, the patient was hemodynamically supported with dobutamine, vasopressin and inhaled nitric oxide. Additionally, intrinsic rhythm was noted to have frequent runs of VT and atrial fibrillation with RVR, so the patient was paced with epicardial pacing wires and started on a lidocaine infusion, as amiodarone was contraindicated given her history of amiodarone induced thyrotoxicosis. Labratory studies revealed the patient had a hemoglobin of 5.1. On post op day 1, the patient was noted to have new anisocoria, loss of all brainstem reflexes besides a cough and was in status epilepticus. Patient was taken for a CTA of the head and neck which did not show evidence of ischemia or intracranial hemorrhage, however, the patient became anuric after the contrast load and was initiated on CVVHD. The patient was then noted to have a pulseless right upper extremity and was taken for a subclavian arterial embolectomy with vascular surgery. The patient continued to require vasoactive support now with the addition of epinephrine, norepinephrine, and angiotensin 2. She became anasarcic and her hemoglobin continued to drop to a nadir of 4 despite maximal doses of IV iron and epoetin alfa. Unfortunately, due to profound hypovolemic and cardiogenic shock LVAD flows dropped and patient expired.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Managing Jehovah's witness patients in the ICU requires intentional consideration of a scarce resource– the patient's own blood. Routine blood draws are not justifiable in this population and as such, clinicians are unable to rely on this constant stream of data to make decisions. In this patient's case, inability to draw PTTs for heparin drip monitoring to prevent clotting of the CVVHD machine, routine BMPs to assure electrolyte disturbances were not attributing to the patient's VT, lactic acid and ABGs to determine if the patient's ischemic limb was overcoming the ability of CVVHD to maintain physiologic pH, and lidocaine levels to prevent lidocaine toxicity were not at our disposal. Additionally, the futility of further procedures should be weighed as these can contribute to additional blood loss. Particularly for this patient, such procedures included– an arterial embolectomy, placement of a femoral trialysis line for CVVHD and consideration for VA ECMO or placement of an RVAD. This case highlights the niche clinical limitations when caring for a Jehovah's witness and should further illustrate the importance of communication between the medical team and the patient's family in order to facilitate an environment that does no harm and honors their religious beliefs.

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Critical Care 37 - Dialyzer Membrane Reaction in a Patient with a Ventricular Assist Device

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Introduction: Intensive care units (ICUs) frequently have patients with severe acute kidney injury (AKI) necessitating renal replacement therapies (RRT), whether via intermittent hemodialysis (iHD) or continuous renal replacement therapies (CRRT). Fortunately, with appropriate management, adverse events related to RRT are rare. A potentially lethal complication of RRT are hemodialysis membrane reactions. Rapid recognition, diagnosis, and management are imperative to prevent catastrophic complications in critically ill patients. Here, we present a case of a dialyzer membrane reaction in a patient with severe AKI after left ventricular assist device (LVAD) placement.

Methods: TP is a 70-year-old male with a medical history significant for dilated valvular cardiomyopathy who was admitted to the ICU after an uncomplicated HeartMate 3 LVAD placement. On arrival to the ICU, TP was continued on dobutamine and epinephrine infusions for right heart support. On postoperative day (POD) 1, TP was extubated and hemodynamically stable; however, he was volume overloaded and anuric, so nephrology service was consulted for initiation of CRRT.

Within 30 minutes after initiation of CRRT, the patient became profoundly hypotensive requiring initiation of norepinephrine and vasopressin to maintain adequate blood pressure. He also became dyspneic with accessory muscle use. POCUS exam showed right ventricular (RV) enlargement with mild RV dysfunction, unchanged EF, and no evidence of tamponade. A chest X-ray showed mild bilateral pulmonary edema. At this time, CRRT was disconnected with the blood returned, and treatment with hydrocortisone, antihistamines, and acetaminophen was administered intravenously. Shortly after, there was a rapid improvement in vasopressor requirements and the patient was again without hypotension.

Four hours after the resolution of hypotension, CRRT was restarted without complication and was continued for 72 hours. On POD5, due to poor communication, the patient became again profoundly hypotensive, tachypneic, and hypoxic at the time of a CRRT filter exchange. After treatment with steroids, antihistamines, and high-dose epinephrine, this episode resolved after five hours. On POD9, the patient was transitioned to iHD after pre-treatment with diphenhydramine and famotidine and tolerated a four-hour session without complication.

Results: NA - Medically Challenging Case

Conclusions: Dialyzer membrane reactions are fortunately quite rare. These reactions have been traditionally classified into Type A or anaphylactic reactions and Type B or nonspecific reactions. Type A dialyzer membrane reactions are typically quite severe reactions with rapid onset within 20-30 minutes of starting dialysis. These reactions can be variable in severity ranging from mild symptoms of urticaria, rhinorrhea, abdominal cramping to severe hypotension, dyspnea, and even cardiac arrest. Unfortunately, this is a diagnosis of exclusion, and the case did not fit neatly into either the Type A or Type B category.

Several different filters have been implicated in Type A reactions, some with associations with angiotensin-converting enzyme inhibitors (ACEi) or angiotensin II receptor blockers (ARBs). This case of a membrane reaction was using a NxStage machine with a PUREMA polyethersulfone filter sterilized by gamma radiation. There has been at least one prior published case report of an anaphylactoid reaction to this specific filter[1] and multiple published case reports of reactions to other polyethersulfone filters.

Management of severe dialysis membrane reactions include immediately stopping treatment. Notably, the blood in the circuit should not be returned to the patient. Supportive care should be administered with epinephrine, bronchodilators, vasopressors, antihistamines, and steroids. Prevention is key in these patients and includes proper rinsing of the dialyzer or if possible, transitioning to another synthetic dialysis membrane. It may be necessary to pre-treat patients with antihistamines and/or steroids as well. Communication is key between critical care providers and dialysis teams to mitigate the errors in repeat exposure that we experienced.

Although dialysis membrane reactions are becoming less frequent as membranes evolve, this is still a severe life-threatening reaction in our ICU patient population. All intensivists should be aware of this potentially fatal complication and be familiar with its management. We hope to raise awareness of this reaction and prevent future fatalities.

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Critical Care 38 - ECMO Consult Team Used to Rapidly Cannulate for ECMO Cardiopulmonary Resuscitation (ECPR)

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Introduction: ECMO Cardiopulmonary resuscitation (ECPR) has been shown to have survival benefits for in-hospital cardiac arrest. We describe a case of activating an ECMO team to perform ECPR for a patient with an intra-operative arrest.

Methods: 58-year-old female presented for an elective hysterectomy with bilateral salpingectomy. Surgical incision was made at 09:51, procedure advanced uneventfully until 11:15, when the uterine artery was clamped for the removal of the uterus. Patient became acutely hypotensive and desaturated, leading to circulatory arrest. A code was called and ACLS with high-quality CPR was immediately initiated, the airway was already secured, and the adequacy of the compressions was confirmed by both end tidal CO2 and arterial line. The emergent ECMO consult team was mobilized and by approximately 11:45 TEE was being performed as cardiothoracic surgery prepped for ECMO canulation. TEE showed a dilated right ventricle (RV) with reduced function, an underfilled hyperdynamic left ventricle (LV) and an embolism in the pulmonary artery. The patient was successfully cannulated and flowing on VA ECMO by 12:09. After the initiation of venous arterial ECMO (VA ECMO), the RV was decompressed. The hysterectomy was completed, with initial pathology significant for leiomyosarcoma. Post operatively, patient was taken to the cath lab, with findings of no evidence of occlusive coronary disease. Patient was brought to the CTICU where ECMO was weaned, and she was ultimately decannulated post op day 5. On post op day 7, patient had an MRI that showed diffuse anoxic brain injury. Patient was ultimately placed on comfort care.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The use of ECMO Cardio-Pulmonary Resuscitation (ECPR) has been shown to increase survival for in-hospital arrest when compared to CPR alone. Multicenter registry cohort studies have predicted the survival of in-hospital cardiac arrest to be 27-30%.[1,2] This is compared to the in-hospital arrest with CPR only survival rate of 10 - 20%.[1] The morbidity and mortality are even greater when considering out-of-hospital arrests. When calculating this patient's "survival after veno-arterial-ECMO (SAVE)-score", it was in the range of 30-40% chance of surviving to hospital discharge.[3]
This patient was in the optimal situation from ECPR. She had a witnessed arrest surrounded by medical staff and ACLS was initiated immediately. As soon as the arrest occurred, the hospital shock/ECMO team was consulted. The consult automatically started a secure group chat among the consulting group, cardiac and critical care anesthesia, cardiothoracic surgery, pulmonary critical care and perfusion teams. This allowed for rapid communication amongst the groups to determine if the patient was a candidate, get appropriate staff and equipment mobilized to the operating room. The ECMO consult enabled teams to quickly get a TEE machine, ECMO pump, cannulating equipment along with all the required staff in under an hour, which was a large success from a systems prospective Although the ACLS and the ECPR was enacted rapidly and effectively, the patient ultimately had the negative outcome of global anoxic brain injury. The outcome was likely secondary to the mechanism of the arrest, obstructive shock secondary to an embolism, possibly tumor or thrombus. Obstructive shock corresponded with the TEE finding of a dilated poorly functioning RV with a hyperdynamic underfilled LV and a clot seen in the pulmonary artery. With an obstructed RV outflow, chest compressions were not able fill the LV, thus leading to inadequate pre-load, stroke volume and cardiac output, resulting in malperfusion of the brain. VA ECMO did the work of the heart and lungs, unloading the RV and circumventing the obstruction.
The use of ECPR in this case provided the greatest likelihood of survival. Although the quick initiation of ECPR prevented mortality, due to the degree of obstruction the patient suffered anoxic injury. The different teams all did an amazing job of providing this patient with the best chance, utilizing all resources including ECPR.

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Critical Care 39 - Emergency Use of Veno-Arterial Extracorporeal Membrane Oxygenation (VA ECMO) for Resuscitation from Flecainide Toxicity: A Case Report

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Introduction: Flecainide overdose can be a problem for critical care physicians, as there is no reversal agent and all care is supportive only. An overdose can lead to fatal arrhythmias that may require the use of mechanical support devices such as VA ECMO in order to save the patient's life and to allow time for metabolism and elimination. Flecainide overdose is a rare but potentially lethal condition with reported mortality of around 10%.[1,2] The typical treatment to remedy arrhythmias and temporize heart rate as in transvenous pacing and chronotropic medications are ineffective in flecainide overdose.[3-5]

Standard of care is hypertonic sodium bicarbonate infusion which competes with flecainide at the sodium channels in myocardial cells.[4,6-8] If this treatment is ineffective, lipid emulsion infusions, lidocaine, and amiodarone have shown effectiveness in reversing bradyarrhythmias.[4,9-13] If treatments above fail or patient suffers cardiogenic shock or cardiac arrest, VA ECMO has been described as a last line of resuscitation.[5] In this case report, we highlight the use of VA ECMO to rescue a patient with flecainide toxicity refractory to therapeutic interventions.

Methods: A 33 year-old female with history of PVCs treated with verapamil and flecainide presented after a suicide attempt. The patient ingested 60 pills of flecainide (total dose of 6 grams). Symptoms at presentation included shortness of breath and palpitations. Initial EKG revealed atrial fibrillation with rapid ventricular response and prolonged QRS and QTc. The patient was started on a sodium bicarbonate infusion and admitted to the intensive care unit. Over 6 hours, the patient's heart rate continuously slowed and QRS duration prolonged further. To prevent cardiovascular collapse, a lipid emulsion infusion was initiated after a loading dose. The use of chronotropic agents or transvenous pacing were not attempted.

The heart rate continued to deteriorate resulting in cardiac arrest. ACLS was initiated and emergent use of VA ECMO was employed for concerns of prolonged and irreversible cardiac failure from flecainide toxicity. ROSC was achieved after VA ECMO was established. An epinephrine infusion was started for vasoactive and inotropic support. The infusion had no effect on the heart rate. Lidocaine boluses were given without effect. While on VA ECMO support, the sodium bicarbonate infusion was discontinued as no benefit was shown. Day 1 of VA ECMO, the patient experienced refractory ventricular fibrillation with decreased pulsatility on arterial waveform and no cardiac ejection on transthoracic ultrasound. A defibrillation shock was administered with return of sinus bradycardia. After 3 days on VA-ECMO, the patient's clinical status improved and was decannulated. The patient was hemodynamically stable post decannulation and discharged on hospital day 9.

Conclusions: Flecainide is a Class Ic antiarrhythmic with effects on the cardiac sodium channels resulting in a slowing of phase 0 depolarization of the fast sodium channels.[14] This reduces conduction of action potentials in the atria, AV node, and His-Purkinje leading to control of supraventricular tachyarrhthmias.[2,4,10,14] Due to its narrow therapeutic index, the chances of inadvertent toxicity is higher and reports of toxicity have been reported.[6,14] After cannulation onto VA ECMO, a flecainide level was drawn and found to be 5.16 mcg/ml (normal: 0.2-1.0 mcg/ml)

There are no antidotes or therapies such as dialysis that can offset the effects of flecainide. Standard of care is support measures and sodium bicarbonate infusion, which acts as a competitive antagonist.[4,6] The use of a temporary venous pacemaker was not attempted as pacing may not be attainable with flecainide overdose.[3,15] Intravenous lipid emulsion was given to help with absorption and elimination of flecainide.[11-13] Without treatment success and cardiac arrest, the patient was emergently placed on VA-ECMO for rescue therapy.

VA ECMO is a documented rescue strategy for flecainide toxicity in case reports with long term survival of 25-30%.[4,5] It is an ideal treatment modality in situations with cardiogenic shock as it allows time for flecainide to be metabolized and eliminated.[4,5] Possible future studies include determining a flecainide dose that could argue for pre-emptive VA ECMO placement to avoid cardiac arrest and the pharmacokinetics of lipophilic flecainide absorption by the membrane lung of VA ECMO as a potential mean of elimination of the drug.

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Critical Care 40 - Emergent intubation in a liver transplant recipient with hypoxic respiratory failure, acute RV failure, severe left-to-right atrial shunt, moderate mitral stenosis, septic shock and atrial fibrillation with RVR

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Introduction: We present a medically challenging case of a critically ill patient with altered mental status and acute hypoxic respiratory failure requiring intubation in the setting of severe mixed cardiogenic, obstructive and septic shock.

Methods: A 67-year-old woman with alcoholic cirrhosis and subsequent orthotropic liver transplant in January 2022, a large ASD with left-to-right shunt, and atrial fibrillation for which she underwent a left atrial appendage ligation via left groin access in September. Following this intervention, she developed a common femoral artery pseudoaneurysm that was repaired in October. Her postoperative course was further complicated by a left groin abscess with graft compromise, requiring repeat intervention. She was eventually discharged to a rehabilitation facility until re-presenting for chest pain and dyspnea.

On arrival, the patient was minimally responsive, hypotensive requiring norepinephrine and vasopressin infusions, in atrial fibrillation with rapid ventricular response and physical exam features consistent with volume overload with hypoxemic respiratory failure. Her shock picture appeared to be cardiogenic with possible underlying sepsis given her recent infection and immunocompromised status. CT chest, abdomen, and pelvis revealed a dilated pulmonary artery, ASD with bi-atrial enlargement, and evidence of moderate pulmonary edema with bilateral pleural effusions. TTE confirmed a large ASD, severe left-to-right shunt, depressed RV function, and moderate mitral stenosis. There were no wall motion abnormalities and the LVEF was normal. Her labs revealed a metabolic acidosis with a pH of 7.1, PaO2 of 80 mmHg on 15L nonrebreather mask, PaCO2 of 50 mmHg, lactate of 5.5 mEq/L, potassium of 5.8 mmol/L, and troponin of 0.15 ng/mL.

Defibrillator pads were placed on the patient and a code team was assembled should cardiac arrest follow induction. We proceeded with intubation via a rapid sequence induction with midazolam, etomidate, and rocuronium. She tolerated induction well and remained hemodynamically stable with bicarbonate, calcium chloride, vasopressin and epinephrine boluses. Following induction, CRRT for volume removal and inotropic infusions were initiated to continue to support her cardiovascular function.

Results: NA – Medically Challenging Case Report

Conclusions: During induction, we attempted to balance the hemodynamic goals for each of the patient's independent pathologies. In septic shock, the first line pressor is norepinephrine in order to maintain MAP >65 mmHg [1]. However, she was grossly hypervolemic, and while norepinephrine was added as an infusion to maintain her systemic vascular resistance, it was not administered in large doses to avoid worsening her RV dysfunction. Avoidance of factors that will precipitate pulmonary hypertension such as hypoxemia, hypercarbia, pain, hypervolemia, and excessive use of vasopressors are key tenants in management of RV failure [2]. To support her RV, we preoxygenated via nonrebreather and expeditiously secured her airway. RV inotropy was maintained with boluses of dilute epinephrine. Calcium and bicarbonate boluses were also given prior to induction to temporarily ameliorate the effects of her metabolic acidosis on her RV function.

Ideal hemodynamic goals of mitral stenosis include maintaining slow heart rate, sinus rhythm, normal inotropy and preload and cautiously maintaining afterload without precipitating pulmonary hypertension [3]. Given her long-standing atrial fibrillation with rapid ventricular response and global hypervolemia, we were prepared for electrical cardioversion following induction if indicated. However, we did not prioritize this intervention prior to induction as she would likely not sustain normal sinus rhythm. Therefore, during induction, we opted to maintain LV afterload with boluses of vasopressin and calcium.

When considering atrial septal defects and left-to-right shunt, it is crucial to maintain adequate preload, normal sinus rhythm, and adequate contractility throughout induction while also avoiding extremes of afterload [4]. Excessive increases in SVR may worsen the left-to-right shunt and further strain a failing RV. Conversely, hypotension following induction could precipitate shunt reversal, which would worsen hypoxemia and risk paradoxical embolism. Therefore, SVR was maintained at pre-anesthetic levels throughout induction with norepinephrine and vasopressin infusions and boluses of vasopressor and inotropes as indicated.

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Critical Care 41 - Encephalopathy Associated with Management of Type B Aortic Dissection

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Introduction: Aortic dissection is described as the tear between the intima and the adventitia layer of the aorta leading to a true and false lumen within the aorta wall. This disruption in the integrity of the aorta wall can cause compromise of branch vessels along the aorta as well as the coronary vessels depending on the extent of the dissection. Aortic dissections that occur distal to the left subclavian artery are classified as type B according to the Stanford Classification. We will present a patient presenting with acute type B dissection with a hospital course complicated by prolonged encephalopathy with eventual recovery.

Methods: The patient was a 47 year old male with a medical history significant for hypertension and hyperlipidemia who initially presented to a hospital with crushing chest pain with radiation to the back. CT imaging revealed type B aortic dissection with flap extending to the distal abdominal aorta. There was partial nonstenotic extension into the left renal artery with sparing of the right renal artery, mesenteric, and iliac vessels. The patient's vitals were significant for hypertensive emergency requiring medical stabilization with esmolol and nicardipine. He was subsequently transferred to our institution for further evaluation and management. On admission to the cardiothoracic intensive care unit (ICU), the patient was alert and oriented and continued on IV antihypertensives for anti-impulse therapy (SBP < 120 mmHg and HR < 80). Initial labs were grossly within normal limits except for a slightly elevated creatinine and lactate level. Transthoracic echocardiography (TTE) was remarkable for concentric left ventricular hypertrophy (LVH) with normal biventricular function and a mildly dilated ascending aorta. A CT head study was obtained on hospital day 3 in the setting of altered mental status (AMS) that began as waxing and waning states of delirium and agitation with progression to comatose periods. The CTH was remarkable for cerebral edema and loss of gray-white differential in bilateral occipital lobes as well as loss of cerebral sulci. Due to concerns for cerebral ischemia secondary to impaired cerebral autoregulation, the blood pressure goals were liberalized to SBP < 140 mmHg. This was followed by interval improvement in the patient's mental status with resolution of the encephalopathy by day 7.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Our patient had long-standing uncontrolled hypertension and this is the most likely cause of his type B aortic dissection. In regards to the encephalopathy that complicated his hospital course, differential diagnoses include hypertensive encephalopathy, posterior reversible encephalopathy syndrome (PRES), cerebral ischemia, and ICU delirium. The timeline of the onset of encephalopathy best supports cerebral ischemia. In the setting of long standing hypertension, the dramatic reduction in SBP impaired the native cerebral autoregulation mechanism leading to cerebral malperfusion. Cerebral ischemia as a result of an embolic etiology should be considered, but is less likely with Type B dissections (1). The diagnosis of PRES associated with HTN was considered due to cerebral edema in the occipital lobes on CTH, but became less likely once a MRI head study revealed ischemic findings as opposed to vasogenic edema that would be seen in PRES (2).

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Critical Care 42 - Esophageal Injury Associated with Thoracic Aortic Aneurysm Repair – A Case Series

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Introduction: Information regarding the frequency and cause of esophageal perforation (EP) post aortic surgery is limited to case reports and series.[1–3] Further, since the advent of modern CT, endoscopy, and surgical techniques the clinical and imaging features of these injuries have not been well described.[3] Rarity and lack of clinical criteria for early identification makes this diagnosis difficult for clinicians in the perioperative period. After EP occurs, the diagnosis carries significant mortality, approaching 100% in the majority of cases secondary to severe mediastinitis, prolonged intensive care unit (ICU) admission, and multiple surgical repair attempts. [1–5] In the most extreme cases, aortoenteric fistulation leads to acute hemorrhagic shock.[2] Here we present a case series of four EPs associated with thoracic aortic aneurysms. This series highlights several clinical and radiographic features that may aid clinicians in early recognition of impending esophageal complication.

Methods: During 2022, four patients who were treated for thoracic aortic aneurysm at our large tertiary care center had courses complicated by EP. Median age was 67 (range, 55-79) years old, two patients were male, all hypertensive smokers, and three were also diabetic. Two patients had prior thoracic endovascular aortic repair (TEVAR).

Patient one was found to have an endoleak involving prior TEVAR. During open total arch a transesophageal echocardiography (TEE) exam was attempted but aborted due to resistance. Postoperatively an esophogram showed esophageal compression and repeat computed tomography (CT) scan was unremarkable. The patient returned one month later with hematemesis. CT scan demonstrating an aortoenteric fistula. The patient ultimately died two months later.

Patient two was diagnosed with a large thoracic aortic aneurysm. After Zone 2 TEVAR, due to a persistent Type 1 endoleak, they underwent open total arch. They returned one month later with cough, fever, and dysphagia. CT scan showed a fistula between the esophagus and the excluded portion of the aneurysm. The patient was discharged with hospice care.

Patient three presented with a ruptured thoracic aortic aneurysm requiring emergency open total arch. Post operatively, esophageal injury was suspected due to a re-accumulating unilateral, purulent, pleural effusion. CT scan showed no signs of esophageal injury, but endoscopy demonstrated transmural necrosis with EP. The patient died shortly after diagnosis.

Patient four underwent a planned TEVAR. They returned one month later with fever, weakness, dysphagia, and hiccups. CT demonstrated increasing aneurysmal size and open total arch was performed. There was difficulty passing both an enteral feeding tube as well as the TEE probe. Endoscopy demonstrated a small esophageal tear, but CT scan was normal. Due to ongoing dysphagia, the patient underwent endoscopy for percutaneous gastric tube placement and a large area of necrosis and EP was identified. Operative repair of the lesion was performed, and patient was transferred to long term care facility.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: While EP associated with thoracic aortic aneurysm is rare, our case series demonstrates that there are several clinical features to be aware of. Prior literature describes several mechanisms that may contribute to EP. (1) Direct erosion of the stent into the esophagus, (2) pressure necrosis due to external compression, (3) esophageal ischemia due to disruption of arterial supply, and (4) infection of the graft leading to erosion.[1,6] Clinical history and imaging in our cases is suggestive of a similar pattern amongst each patient. Three patients complained of dysphagia prior to aneurysm repair suggesting direct esophageal compression, likely causing impaired vascular supply. This combined risk factors for microvascular disease are likely the most relevant features in these cases. Given the significant morality associated with EP, it is imperative that physicians maintain a high clinical suspicion in the perioperative period. Unfortunately, even modern CT scan is unlikely to detect early esophageal injury.[6] Therefore, it is important to consider esophageal compromise in the perioperative period among patients with dysphagia, risk factors for impaired microvascular supply, and thoracic aortic aneurysm. While there is no consensus on how to prevent EP, early esophageal decompression and careful monitoring of clinical symptoms are likely highly beneficial.[6]

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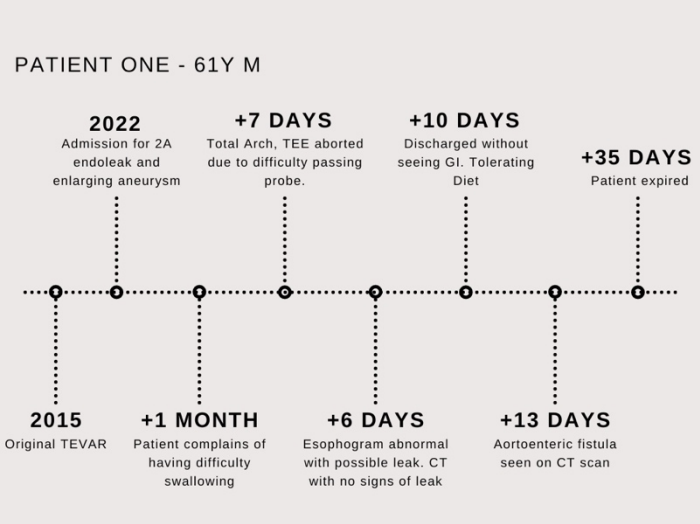
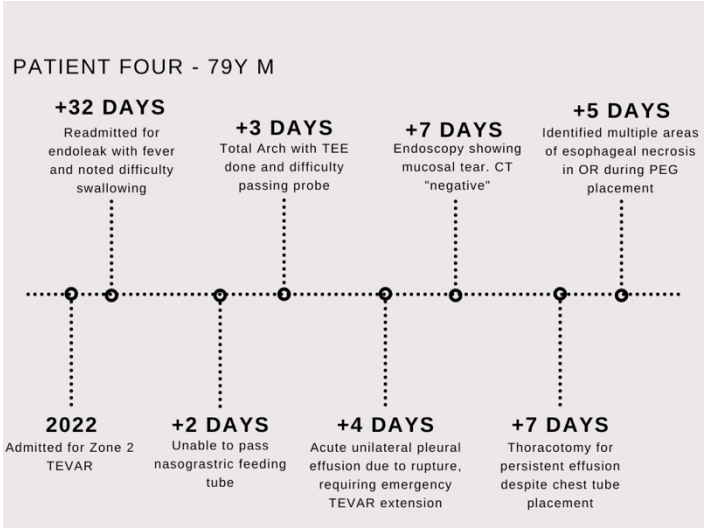
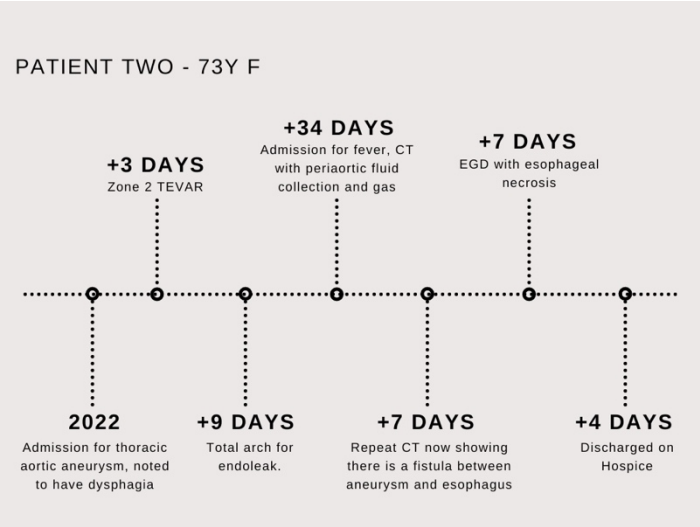
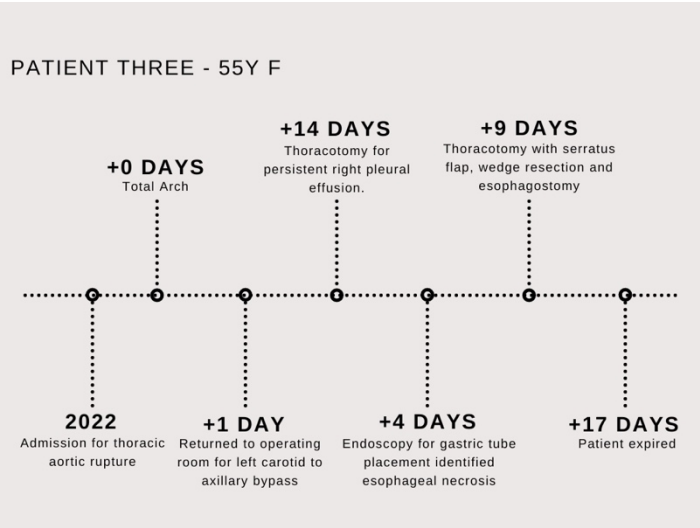

PATIENT ONE - 61Y M
2015
Original TEVAR
2022
Admission for 2A endoleak and enlarging aneurysm
+1 MONTH
Patient complains of having difficulty swallowing
+7 DAYS
Total Arch, TEE aborted due to difficulty passing probe.
+6 DAYS
Esophogram abnormal with possible leak. CT with no signs of leak
+10 DAYS
Discharged without seeing GI. Tolerating Diet
+13 DAYS
Aortoenteric fistula seen on CT scan
+35 DAYS
Patient expired


PATIENT FOUR - 79Y M
2022
Admitted for Zone 2 TEVAR
+32 DAYS
Readmitted for endoleak with fever and noted difficulty swallowing
+2 DAYS
Unable to pass nasograstric feeding tube
+3 DAYS
Total Arch with TEE done and difficulty passing probe
+4 DAYS
Acute unilateral pleural effusion due to rupture, requiring emergency TEVAR extension
+7 DAYS
Endoscopy showing mucosal tear. CT "negative"
+7 DAYS
Thoracotomy for persistent effusion despite chest tube placement
+5 DAYS
Identified multiple areas of esophageal necrosis in OR during PEG placement


PATIENT TWO - 73Y F
2022
Admission for thoracic aortic aneurysm, noted to have dysphagia
+3 DAYS
Zone 2 TEVAR
+9 DAYS
Total arch for endoleak.
+34 DAYS
Admission for fever, CT with periaortic fluid collection and gas
+7 DAYS
Repeat CT now showing there is a fistula between aneurysm and esophagus
+7 DAYS
EGD with esophageal necrosis
+4 DAYS
Discharged on Hospice


PATIENT THREE - 55Y F
2022
Admission for thoracic aortic rupture
+0 DAYS
Total Arch
+1 DAY
Returned to operating room for left carotid to axillary bypass
+14 DAYS
Thoracotomy for persistent right pleural effusion.
+4 DAYS
Endoscopy for gastric tube placement identified esophageal necrosis
+9 DAYS
Thoracotomy with serratus flap, wedge resection and esophagostomy
+17 DAYS
Patient expired

Critical Care 43 - Ethical Dilemmas in Cardiac Surgery and Critical Care: A Type-A Aortic Dissection Repair Without Consent

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Introduction: The Cardio-Thoracic Intensive Care Unit is often full of complex, challenging patients. The aberrant physiology and high-risk procedures create an environment ripe for learning and the advancement of the sciences. This environment also creates novel ethical dilemmas. Despite extensive training during residency and fellowship, clinicians are often faced with challenges that advance beyond the clinical realm. Ethical conundrums bring concepts native to the practices of philosophy, religion, and social sciences to the otherwise sterile field of intensive care medicine. This case-report details a patient at high-risk for significant morbidity and mortality due to her pathology, race/ethnic and socio-economic status who's clinical course was further complicated by ethical dilemmas associated with capacity and informed-consent.

Methods: This case presents a not-uncommon clinical scenario. The patient presented with classic signs and symptoms of an aortic dissection and there was no delay in attempting to proceed with the appropriate, timely surgical management. This situation was complicated by the balance of respect for individual autonomy and the determination of capacity.

This patient presented with a type A aortic dissection. After arriving in the operating room, she refused operative repair. After admission to the CTICU, the intensivist ensured the appropriate parties were consulted, namely, Psychiatry and the Medical Ethics Consultation Service (MECS). Additionally, attempts were being made to contact surrogate decision makers or at least family or acquaintances that were more familiar with the patient's history. As these efforts were made concurrently, a path forward began to emerge. Based upon the evaluation of the ICU team, the Psychiatrists and the MECS, the patient did not have capacity to make decisions for herself. Furthermore, her risk of mortality increased as much as 0.5% for each hour of delay[1]. In this setting, it was the responsibility of the medical team to make decisions in her stead based upon the "reasonable person statute"[2]. A reasonable person would likely wish to continue with life, therefore it was appropriate to re-approach the surgical team with this new determination. Shortly thereafter, family members were contacted and able to provide substituted judgement, which aligned with the wishes of a reasonable person.

This patient underwent surgery and had a protracted post-operative course, not uncommon with such an extensive operation. Her course was complicated by her history of Opioid Use Disorder which led to significant withdrawal symptoms, presenting as agitation, hypertension and altered mental status. Ultimately, the patient survived to discharge and through the initial thirty-day post-operative course. Her survival was a testament to collaboration among several different medical specialties as well as an intensivist passionate about delivering ethical, patient-centered care.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: In addition to complex physiology and medical decision-making, an intensivist must be trained and equipped to manage complex ethical decision-making as well. This case report details a patient with an extremely high-risk pathology. Initially her decision to refuse treament were respected. However, upon further evaluation, the patient was found to not have capacity to make medical decisions. The intensivist relied heavily on consultation with the Psychiatry and Medical Ethics Consultation Services to plan a way forward in providing the appropriate care. Ultimately, a surrogate decision-maker was identified and using substituted judgement, the patient underwent operative repair of her type A aortic dissection.

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Critical Care 44 - Euglycemic Diabetic Ketoacidosis in CABG Patient Secondary to SGLT-2 Inhibitor Use Despite Preoperative Cessation for Three Days

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UTHSCSA[1]

Introduction: We present the case of a 72-year-old male with a past medical history of type II diabetes on empagliflozin, hypertension, hyperlipidemia, chronic kidney disease, history of gastric bypass surgery, and coronary artery disease who presented to the intensive care unit (ICU) after coronary artery bypass graft (CABG) who developed euglycemic diabetic ketoacidosis (DKA) secondary to sodium/glucose cotransport-2 (SGLT-2) inhibitor use despite preoperative cessation for three days.

Methods: A 72-year-old male had undergone uneventful two-vessel CABG and arrived to the surgical ICU intubated and sedated with minimal vasopressor support. Intraoperatively, the patient maintained a normal pH and the blood glucose never exceeded 130 mg/dL. On arrival to the ICU, routine labs were drawn demonstrating a respiratory acidosis, anion gap of 12, and a normal blood glucose. The ventilator was adjusted to correct the respiratory acidosis, and after normalization of the PaCO2 a spontaneous breathing trial was initiated. During this time, urine output was 50-100 cc/hr, cardiac index 3.3 and SvO2 76, on a minimal dose of norepinephrine. The patient met extubation criteria and to confirm stable acid-base balance, one last arterial blood gas was drawn that showed a worsening metabolic acidosis. The spontaneous breathing trial was terminated, and a basic metabolic panel showed an anion gap of 20 with continued downtrend of HCO3- and pH while maintaining normal levels of blood glucose and lactic acid. After broadening the differential diagnosis of a gap metabolic acidosis and reviewing his medication list, which included an SGLT-2 inhibitor, serum ketones were ordered and found to be elevated at 87.6 mg/dL (normal: 0.2 - 2.8 mg/dL). The patient was diagnosed with euglycemic DKA and an insulin infusion was initiated at 2 units/hr. Shortly after, the patient's HCO3- continued to downtrend and 50 mEq of sodium bicarbonate were administered. After a few hours, the HCO3- level and acidosis began to normalize and the patient was safely extubated.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Euglycemic DKA is not an uncommon diagnosis in the ICU postoperatively due to increased SGLT-2 inhibitor use. cases have been reported in patients undergoing gastric bypass surgery after holding the medication for seven days prior to day of surgery [1]. Reports in patients undergoing CABG have occurred despite appropriate discontinuation of the drug [2]. More commonly [3], patients that undergo emergent surgery and are unable to hold the medication in a timely manner have developed DKA. In this case, the patient developed DKA despite holding the medication for three days prior to his day of surgery. This may be due to his chronic kidney disease and history of bariatric surgery in addition to factors such as decreased carbohydrate intake and surgical stress. Current recommendations for preoperative SGLT-2 use include discontinuation three days prior to the day of surgery [4]. In postoperative patients who chronically use these medications, euglycemic DKA remains an important consideration in the setting of an anion gap metabolic acidosis. From this case and other case reports, holding this medication for longer than three days may be warranted in patients having high-risk surgeries.

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Time	1530	1609	1629	1752	2104	2125	2232	2352	0122
NA	144					146			
K	5.2					4.6			
CL	112					111			
CO2	20					15			
BUN	30					31			
Cr	1.1					1.4			
GLU	136		131	152	185	172	191	189	186
AG	12					20			
pH		7.19	7.20	7.32	7.23		7.24	7.22	7.28
pCO2		53.8	50.8	37.3	37.2		33.4	34.3	37.9
pO2		50	107.4	157	135		144	138	143
HCO3		20	19.2	18.8	15.2		14	13.6	17.4
Lactate			0.8	0.8	1.1		1.0	0.9	0.9

Critical Care 45 - Hemodynamic Management of Simultaneous Heart-Liver Transplant

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Introduction: Congestive hepatopathy is a severe complication of heart failure that occurs due to venous congestion impairing hepatic perfusion[1,2]. This condition has been seen in patients with congenital heart defects who have undergone surgical repair, such as with the Fontan procedure. Combined heart-liver transplant (CHLT) is a definitive treatment for cardiac cirrhosis secondary to a congenital heart defect but requires careful management in the intensive care unit to support the function of both grafts. We describe a CHLT case in which significant extracorporeal support was necessary to preserve graft function and subsequent hemodynamic goals to maintain heart and liver graft function were at odds, with diastolic dysfunction requiring an elevated central venous pressure (CVP) that was concerning for liver graft congestion.

Methods: A 51-year-old man with a history of double inlet left ventricle and left transposition of the great arteries status post Fontan procedure, Fontan-associated cardiac cirrhosis, chronic kidney disease, and obstructive sleep apnea presented for CHLT. The intraoperative course was notable for primary graft dysfunction of the heart requiring veno-arterial extracorporeal membrane oxygenation (VA-ECMO) for support. The patient was decannulated on postoperative day (POD) 7 but developed worsening acute lung injury with metabolic acidosis requiring initiation of veno-venous ECMO (VV-ECMO) on POD8. He was subsequently found to have hepatic graft congestion related to cardiac primary graft dysfunction, for which an arterial limb was added to the ECMO circuit for veno-arterio-venous ECMO (VAV-ECMO). Cardiac function improved with additional support, liver function tests (LFTs) improved, and the patient's arterial ECMO limb was removed on POD17 without issue.

Upon reversion to VV-ECMO, the patient's heart was noted to exhibit diastolic dysfunction. Therefore, CVP goals were elevated (~15-18 mmHg) to ensure adequate cardiac output despite normal CVP goals for liver transplantation being <12mmHg. On POD19, the patient's LFTs increased significantly, causing concern for hepatic congestion and liver graft failure. A CT scan of the abdomen showed a narrowing of the hepatic artery near the anastomosis, which was likely due to postoperative edema or a donor-recipient hepatic artery size mismatch. The differential diagnosis for this included congestion secondary to elevated CVP and vasoconstriction of the donor and recipient hepatic arteries in the setting of a previously known hepatic artery size mismatch. However, it was also noted that LFT elevation occurred after removal of the arterial ECMO cannula, which may have been providing sufficient hepatic blood flow to overcome the artery size mismatch at flows of 3.4L/min. Given this likely cause, it was decided to target a goal CVP of 15-18mmHg to optimize diastolic filling of the transplanted heart and thus optimize liver and other end-organ perfusion. Moreover, to minimize vasoconstriction in the hepatic artery, the patient was weaned off epinephrine and vasopressin while maintaining inotropy and vasomotor tone with dopamine, dobutamine, and norepinephrine. LFTs subsequently normalized with this treatment plan.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The Fontan operation for congenital cardiac abnormalities with single-ventricle physiology results in the right-sided circulation bypassing the heart, improving systemic perfusion while also causing venous hypertension and possible congestive hepatopathy among other complications often requiring eventual transplant[7–9]

We present a case of conflicting aims related to CHLT in an adult post-Fontan patient. Our patient's diastolic primary graft dysfunction made it imperative to maintain a high CVP with judicious management of fluids and vasopressors, contrary to the usual recommendation for low CVP in liver transplant patients to avoid hepatic congestion. The difficulty of the decision to optimize heart function despite the risk to the liver showcases the importance of multidisciplinary priority-setting for the care of CHLT patients.

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Critical Care 46 - Hemopericardium with Tamponade Following Cardiopulmonary Resuscitation in a Patient Receiving Dual Antiplatelet Therapy

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Introduction: Closed-chest cardiopulmonary resuscitation (CPR) is a validated form of maintaining end-organ perfusion following cardiovascular collapse, however it carries an inherent but necessary risk to the patient due to the violent nature that chest compressions require to achieve proper blood circulation. Rib and sternal fractures, pneumothoracies, and skin injuries are commonplace. Rare but serious complications include cardiac, liver and gastric injuries[1]. A 2014 review reported pericardial injury occurs in 8.9% and hemopericardium occurs in 7.5% of CPR-treated patients[2]. Despite these percentages, relatively few case reports of hemopericardium progressing to cardiac tamponade have been reported[3]. Below we report a case of echocardiographic-confirmed cardiac tamponade following CPR in a patient receiving dual antiplatelet therapy.

Methods: An 84-year-old male with a history of hypertension and hyperlipidemia was admitted to our Neurosciences ICU following right lower extremity weakness and a subsequent diagnosis of a left MCA acute ischemic stroke. He received intracranial mechanical thrombectomy with excellent reperfusion and had resolution of his stroke symptoms. Antiplatelet therapy with Aspirin 81mg and clopidogrel 75mg daily was initiated for secondary prophylaxis. The day prior to planned discharged the patient was hemodynamically stable and without complaint.That evening he developed pulseless electrical activity. Advanced Cardiac Life Support (ACLS) was initiated and continued for 45 minutes before spontaneous circulation was achieved. Following return of circulation, the patient was electrically cardioverted from ventricular tachycardia to sinus tachycardia with marked ST-depressions. Vasopressor infusions were initiated and balanced cyrstalloid resuscitation was continued. Bedside echocardiography revealed severe LV failure and a 1cm thick pericardial effusion juxtaposed to the RV free wall with RV diastolic collapse consistent with tamponade physiology. Provisions were made to perform bedside pericardiocentesis. Repeat echocardiographic imaging for ultrasound-guided pericardiocentesis revealed the effusion was rapidly expanding. Pericardial access was obtained and 150cc of blood was evaucated from the pericardial space with resolution of RV diastolic collapse. Despite tamponade resolution, the patient continued to have escalated inotropic requirements and ongoing ST-segment depressions. Due to his worsening clinical status despite maximal medical support and not meeting criteria for mechanical circulatory support, the decision was made to withdraw life sustaining measures due to futility. The patient expired quickly following removal of vasopressor support.

Conclusions: Cardiac tamponade is a life-threatening cardiovascular emergency that may be determined clinically with the associated findings of hypotension, JVD, and diminished heart sounds on auscultation—known as Beck's triad[4]. Continuous hemodynamic monitoring may reveal pulsus paradoxus--a greater than 10mmHg drop in systolic blood pressure during inspiration with spontaneous respiration. This pattern is reversed in mechanically ventilated patients. Studies have found both Beck's triad and a pulsus paradoxus of 10mmHg to be poorly to moderately sensitive to diagnose tamponade[5,6]. Echocardiography, conversely, has been shown to be extremely sensitive and specific for diagnosing tamponade with the American Society of Echocardiography reporting that right atrial collapse exceeding one-third of the cardiac cycle is nearly 100% sensitive and specific for tamponade[7].

It is possible this patient developed hemopericardium and tamponade that resulted in cardiac arrest, but the rapid expansion of the effusion visualized on bedside echocardiogram suggests it was most likely iatrogenic--especially in the setting of dual antiplatelet therapy. Whatever the cause, this case report highlights the facts that clinicians should be mindful that CPR is an inherently dangerous life-saving technique that may cause serious complications, and that the use of echocardiography is a useful, if not essential, tool for aiding in the diagnosis and management of patients with ongoing hemodynamic instability in critical situations.

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Critical Care 47 - Heparin Induced Thrombocytopenia Type II: When anticoagulation causes life threatening clotting

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Introduction: Heparin induced thrombocytopenia type II (HIT), a life threatening autoantibody response to platelet factor 4 (PF4), is a sequela of great concern in the cardiac intensive care unit. While HIT type 1 is more common, with literature reporting rates of approximately 10-20% of those exposed to heparin, it is typically a benign course recovering within 4 days with no increased risk of thrombosis. On the contrary, HIT type II occurs in 1-3% of those exposed, generating a hypercoagulable state that creates significant morbidity and a early mortality rate of up to 50% (Hogan M, 2020). This case presents an alternative treatment approach with Intravenous immunoglobulin (IVIG) when expectant management and supportive therapy do not show signs of improvement in the setting of advancing thrombosis.

Methods: A 62 year old ASA 4 with a history of HTN, HLD, T2DM, OSA, Anxiety, chronic left posterior tibial DVT, and BMI of 53 underwent a 3 vessel coronary artery bypass graft (CABG) and was admitted to the cardiac ICU for postoperative care with concern for severe HIT type II. Before operation he received an inpatient workup and optimization where he was initiated on a heparin drip nine days prior to his CABG for a chronic posterior tibial DVT. His platelets had been stable at a mean of 250, then precipitously dropped to 200 two days pre op, and 37 the day prior to surgery. This went unnoticed, and the patient proceeded to have the operation where he would have a heparin load to facilitate cardiopulmonary bypass. A HIT panel and coagulation study were ordered on admission to the ICU. His platelets nadired at 6K post op day 0. Traditionally with HIT type II it is expected to see an increase in platelets over up to 8 days post op. Discontinuing the offending agent, alternative anticoagulation and supportive therapy are the mainstay of treatment during this period. During this interval his PF4 antibody resulted as positive. He proceeded to suffer new acute DVT in bilateral lower extremities, including the right femoral, popliteal, and gastroc veins, and left common femoral vein, superficial femoral vein, popliteal and posterial tibial vein. Arterial duplex imaging did not show any apparent perfusion defects. Clinically distal profusion was strained, resulting in distal necrosis of his right toes, even in the setting of bilvarudin anticoagulation. The patient reported pain in these toes unilaterally, and bilateral calf pain. The patient's severe obesity with a BMI>50 limited anticoagulation options. On day 8 his platelets started to downtrend from a high of 37 on day 6.

At this time a multidisciplinary decision was made to initiate an uncommon treatment using high dose IVIG in light of worsening thrombosis and further decline in platelet count. This decision was directed by an article in the expert review of hematology (Warkentine TE, 2019) that pooled data from rare cases and their outcomes with administration of IVIG. Over the next week, the platelet count rose by 100k. During this time he did not show any clinical signs of worsening thrombotic burden. He was continued on Bilvarudin therapy until he reached a therapeutic goal on PO warfarin therapy. The patient was ultimately discharged to a local rehab facility with frequent coagulation and platelet studies.

Results: Not Applicable – Medically Challenging Case Report
Conclusions: While traditional philosophy points to the inherent danger of thrombosis with IVIG as a contraindication to the treatment of HIT, we present this case as supporting evidence to a newer body of literature suggesting the contrary. This patient provides an example of possible treatments when the traditional therapies fail to control worsening symptomology. It is also important to note, not only does HIT type II carry increased morbidity and mortality risk, but this patient was also required a prolonged stay of 18 days in the intensive care unit.

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Critical Care 48 - Hyperammonemia Secondary to Mycoplasma and Ureaplasma Co-Infection Following Heart and Kidney Transplant

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Emory University[1]

Introduction: Hyperammonemia is a metabolic condition that may be liife threatening if untreated. Hepatic dysfunction is a common etiology and therefore a focus of initial workup. Normal hepatic function prompts a broader workup. Mycoplasma and Ureaplasma infections have been identified as a rare cause of hyperammonemia. Regardless of the etiology, early treatment is critical to prevent cerebral edema and increased intracranial pressure that may be fatal. In immunocompromised patients with hyperammonemia, direct testing for Mycoplasma and Ureaplasma species should be obtained, and empiric treatment should be initiated.

Methods: A 55-year-old male whose past medical history included advanced heart failure owing to ischemic cardiomyopathy (ICM) and chronic kidney disease underwent an orthotopic heart transplant (OHT) followed by a kidney transplant on postoperative day (POD) 1. Concern for bleeding prompted a return to the operating room for a chest washout on post operative day POD 2. On POD 7 he was extubated, and his clinical status progressively improved. On POD 11, he had intermittent hypotension and was becoming more encephalopathic. Given his immunosuppression and increased risk for infection, blood and urine cultures were obtained, and broad spectrum antimicrobial therapy with vancomycin and ceftazidime was initiated. His urine cultures ultimately revealed Hafnia alvei infection, and his antibiotics were changed to Meropenem due to an Amp C resistance pattern with plans for a 7-day treatment course. Throughout the next few days his encephalopathy worsened and was initially attributed to his uremia and infection that was being treated; however, further lab testing revealed an unexpected hyperammonemia to 186µmol/L. Further workup followed, including a CT of the head, RUQ ultrasound, liver function tests, repeat cultures, urea cycle disorder workup, and PCR testing for Mycoplasma and Ureaplasma. A liver biopsy done prior to his transplant showed congestive disease; however, his liver function tests were normal. His CT did not show any cerebral edema, and the RUQ ultrasound was unrevealing. His encephalopathy continued to worsen, prompting intubation. Treatment for his hyperammonemia was initiated including rifaximin, metronidazole, thiamine, levocarnitine, Lactulose, and zinc. Continuous renal replacement therapy was initiated with a dialysate rate of 4500ml/hr, and empiric doxycycline was started. Over the next few days his encephalopathy improved as his ammonia levels decreased. Five days after collection of the specimens, PCR test results returned positive for Ureaplasma urealyticum and Mycoplasma hominis. Treatment with doxycycline continued for a total of 7 days with resolution of his encephalopathy and hyperammonemia.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Hyperammonemia can lead to life-threatening complications, including cerebral edema and brain herniation if left unrecognized and untreated. Hepatic dysfunction is one of the most common causes for hyperammonemia, but other causes that must be considered include drugs, congenital urea cycle defects, renal disease, GI bleeding, Reye syndrome, shock, and infection with urease-producing organisms. Mycoplasma and/or Ureaplasma infections must be considered as a potential cause of hyperammonemia in immunocompromised patients. Diagnosis is often difficult as these organisms are not typically revealed by routine microbiologic testing, such as gram stain and basic cultures. Specialized cultures or nucleic acid amplification testing can be used for detection; however, these specimens must often be sent to a reference lab for testing with varying turnaround times. Empiric treatment against these organisms must be initiated early as delaying treatment until labs result may lead to poor clinical outcomes. Mycoplasma and Ureaplasma species are usually susceptible to tetracyclines making doxycycline a good first-line option.

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Critical Care 49 - Hypocapnia diagnoses cardiopulmonary stasis in patient on peripheral VA ECMO

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Brigham and Women's Hospital[1]

Introduction: This case exemplifies how a severely abnormal blood gas was used to identify cardiopulmonary stasis due to inadequate left ventricular unloading, despite pristine ECMO flow conditions and a pulsatile arterial waveform with IABP in place. TTE confirmed there was a lack of cardiac ejection and no forward flow across the aortic valve. Close cardiac evaluation including serial blood gases and TTE may be required in addition to a pulsatile arterial waveform to optimize mechanical support conditions when an IABP is used as a venting device for the LV during VA ECMO.

Methods: A 51-year-old gentleman presented to the hospital for increased shortness of breath and initial workup revealed decompensation of known heart failure. His medical history was significant for nonischemic cardiomyopathy with baseline ejection fraction 10% s/p ICD placement, monomorphic ventricular tachycardia, type 2 diabetes mellitus, and stage 3 chronic kidney disease. Despite medical management he had worsening cardiogenic shock and decision was made to use venous-arterial extracorporeal membrane oxygenation (VA ECMO) as a bridge to left ventricular assist device (LVAD). He was cannulated onto VA ECMO with left femoral arterial cannulation (17 Fr) and right femoral vein access (25 Fr) with right axillary intra-aortic balloon pump (IABP).

After initiation of VA ECMO, ECMO flows were stable, mean arterial pressure (MAP) >70 with apparent pulsatility on the arterial line. The patient was noted to have a severely abnormal venous blood gas obtained from pulmonary artery catheter (Figure 1). $PvCO_2$ (9-14) was markedly decreased and PvO_2 (208-254) was increased concerning for stasis in cardiopulmonary tree. Bedside transthoracic echocardiography (TTE) confirmed dilated left ventricle (LV) with minimal contractility, color and continuous wave doppler revealed reversal of flow in the left ventricular outflow tract (LVOT) (Figure 2).

Patient returned to the operating room (OR) for conversion of IABP to Impella® device to improve LV unloading and forward flow across the aortic valve. On return from OR blood gases normalized, central venous pressure and pulmonary capillary wedge pressure decreased, and patient had increased urine output confirming stabilization of hemodynamics and improvement in perfusion. On ECMO day 5 the patient underwent successful placement of left ventricular assist device (LVAD) followed by placement of right ventricular assist device (RVAD) and was transitioned off ECMO. Right ventricular function improved allowing removal of the RVAD, and the patient was ultimately discharged to a rehabilitation center with LVAD as destination therapy.

Results: Not Applicable- Medically Challenging Case Report

Conclusions: VA ECMO allows full circulatory support for a patient in severe cardiogenic shock providing both blood flow and gas exchange acting as a partial cardiopulmonary bypass.[1,2] However, continuous blood flow through peripherally cannulated VA ECMO can increase LV afterload directly opposing ejection of blood flow across the aortic valve.[3] Stasis of blood flow within the LV leads to ventricular dilation, thrombus formation, pulmonary edema and can further limit cardiac recovery.[4]

Mechanical support modalities can be used as adjuncts to VA ECMO to assist left ventricular unloading and prevent stasis. In this case, a right axillary intra-aortic balloon pump (IABP) was in place at time of cannulation and utilized as a left ventricular venting strategy.[3,5] Inflation and deflation of the IABP gave an arterial wave form on the radial a line that suggested ventricular ejection. However, the severe venous blood gas abnormalities including $PvCO_2$ (9-14) and venous PvO_2 (208-254) inferred repeated ventilation and oxygenation of standstill blood in the cardiopulmonary tree. TTE depicting a dilated left ventricle, minimal contractility, increased viscosity of blood in the left ventricle, and reversal of flow in the LVOT confirmed the concern for stasis and inadequate unloading of the left ventricle.

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4 Hrs:	07/09 20-00	07/10 00-04	07/10 04-08	07/10 08-12	07/10 12-16	07/10 16-20	07/10 20-00	07/10 00-04
BP (a-line 2)	93/61	94/51	110/67	106/63+	116/107	95/92	84/77	76/70
MAP (a-line 2)	71+	70+	86+	82+	111	94+	79+	72+
Blood Gas								
PH Arterial	7.52	7.32+	7.35+	7.41	7.60	7.62	7.57	7.30
PCO2 Arterial	28	54	48	37	18	21	26	54
PO2 Arterial	358	369	409	373	188	269	217	130
SO2 Arterial	100.0	99.7	99.8	99.5	100.0	100.0	99.8	98.3
FiO2		30+	30+					100
PH Venous	7.49	>7.80	7.74	7.68	7.50			7.27+
PCO2 Venous	30	<9	12	14	32			63
PO2 Venous	44+	208	245	254	34			51
SO2 Venous	80.1+	100.0	100.0	100.0	61.7			77.4+

Figure 1: Severely abnormal venous blood gases outlined in black. Patient placed on ECMO at 20 00. Repeat venous blood gases noted to have PvCO2 <9, 12, 14. After exchange IABP to Impella device repeat gases normalized.

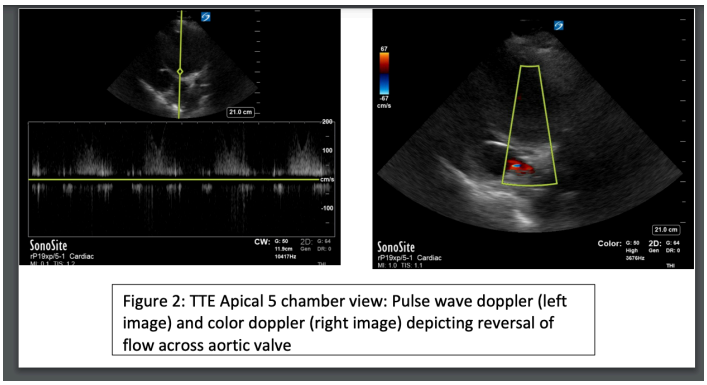

Figure 2: TTE Apical 5 chamber view: Pulse wave doppler (left image) and color doppler (right image) depicting reversal of flow across aortic valve

Critical Care 50 - Illicit Substance Intoxication Causing Rare CHANTER Syndrome

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Washington University St. Louis[1]

Introduction: Cerebellar hippocampal and basal nuclei transient edema with restricted diffusion (CHANTER) syndrome is a rare syndrome with a specific pattern identified on the MRI imaging of some patients who presented with altered mental status in the context of substance intoxication.
These patients are at risk of developing obstructive hydrocephalus that may require therapy with osmotic agents and/or surgical interventions (i.e., drains and decompressive craniectomy). If left without treatment it may lead to herniation and irreversible brain damage.

Methods: In our case we had a 23-year-old Male with PMH of polysubstance abuse, suicidal ideation, bipolar disorder presented to ED with altered mental status after being found down by his fiancé. He was given intranasal Narcan by EMS.

In ED, patient was alert and oriented to person, time and place but was quite somnolent, with labs being notable for Cr 2.47, Anion Gap 19, K 5.8, VBG 7.15/74/39/27, WBC 27, lactate 3.1, troponin 377. His Urine Drugs Screening was positive for amphetamine, benzodiazepine, cannabinoids, fentanyl.

On the floor, patient had acute LLE weakness and sensory loss and Code stroke team activated. CT head showed foci of hypoattenuation within both basal ganglia and a diffuse hypoattenuation involving both cerebellar hemispheres. Brain MRI demonstrated symmetric foci of diffusion restriction involving bilateral globus pallidus and cerebellar hemispheres as well as vermis suspicious for opioid neurotoxicity/CHANTER syndrome.

Patient was admitted to the ICU with plans for frequent neuro checks, serial brain imaging. Neurosurgery was aware of the patient with plans for early intervention at the signs of any clinical deterioration or signs of developing hydrocephalus on his imaging. Patient was also found to have elevated CK of 160K concerning for possible rhabdomyolysis with his left leg being more tense compared to his right leg. He was started on normal saline fluid infusion for his rhabdomyolysis.

And he also received hypertonic saline boluses twice during the course of his hospitalization to avoid hyponatremia <140
Fortunately, the patient's mental status improved gradually with his serial imaging being stable and his CK level gradually dropped. He was discharged from the ICU after 5 days

Results: Not Applicable – Medically Challenging Case Report

Conclusions: CHANTER syndrome is a rare condition affecting patients with drug overdose that may have severe repercussions on the patients but early diagnosis with close monitoring and appropriate interventions can lead to positive outcomes
But data are still lacking about the factors that could impact disease outcome, including variables such as the substance of use, the duration and quantity of use, and the chronic medical conditions for each patient.

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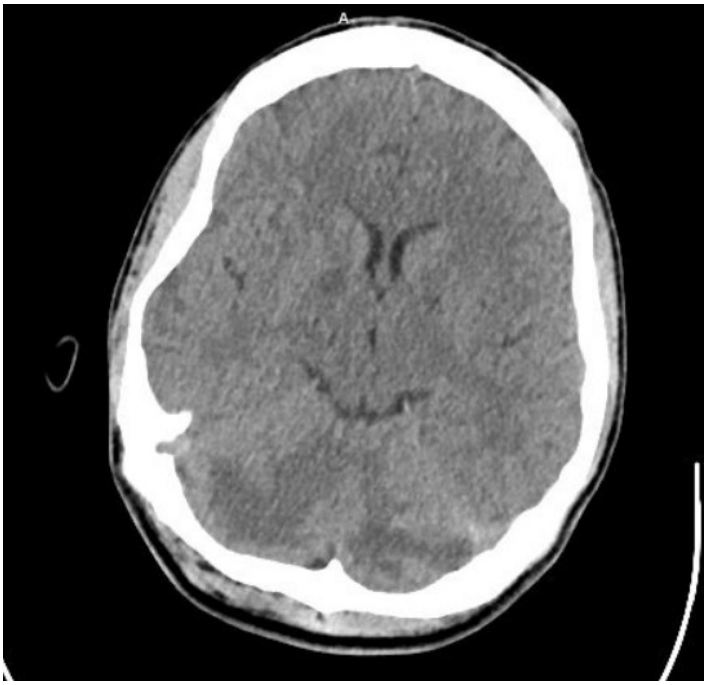

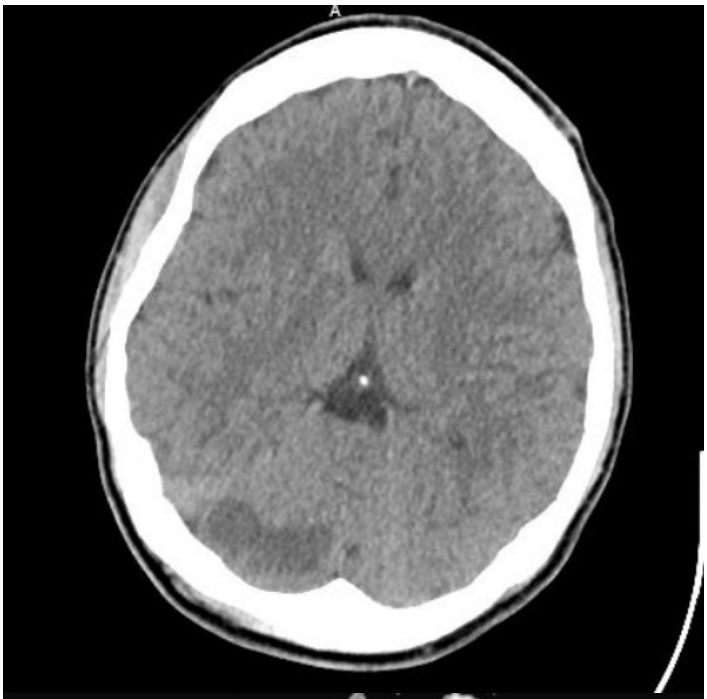

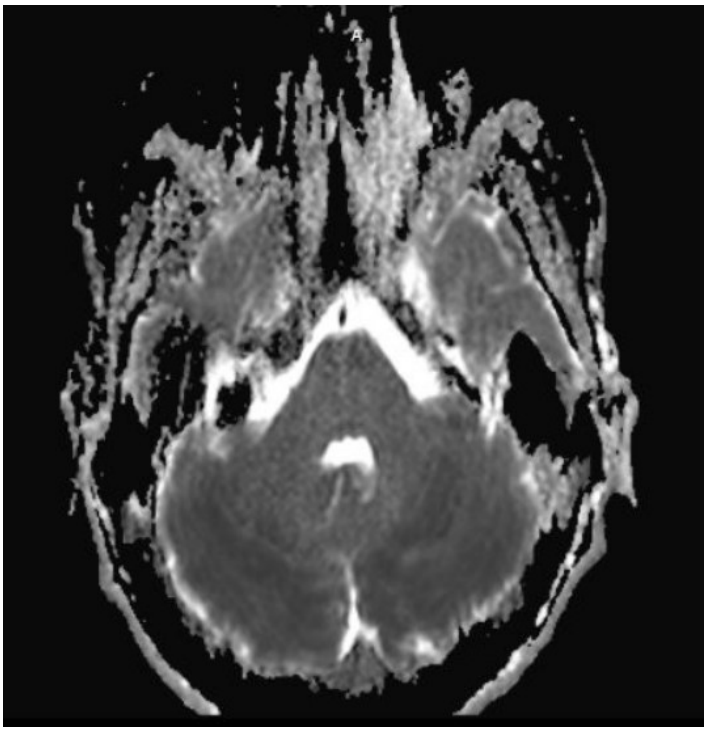

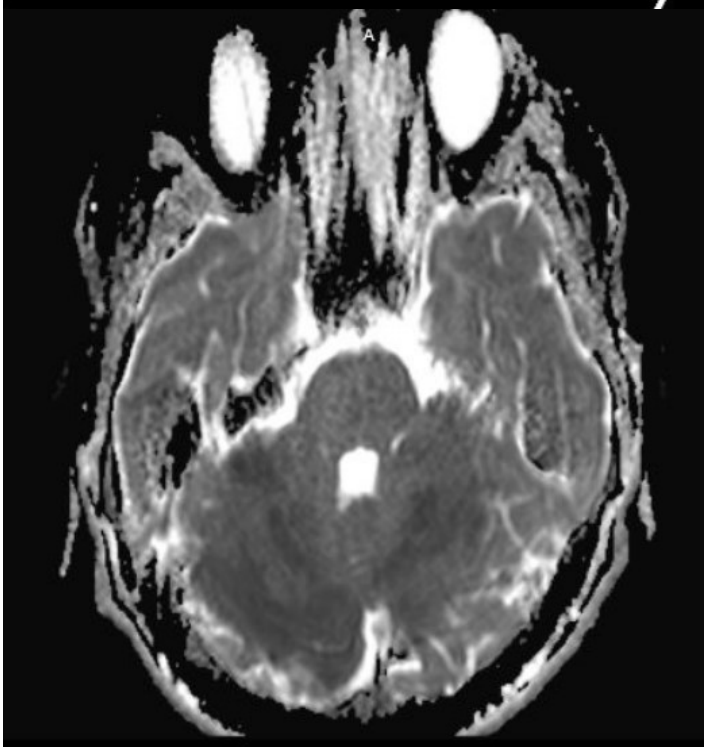

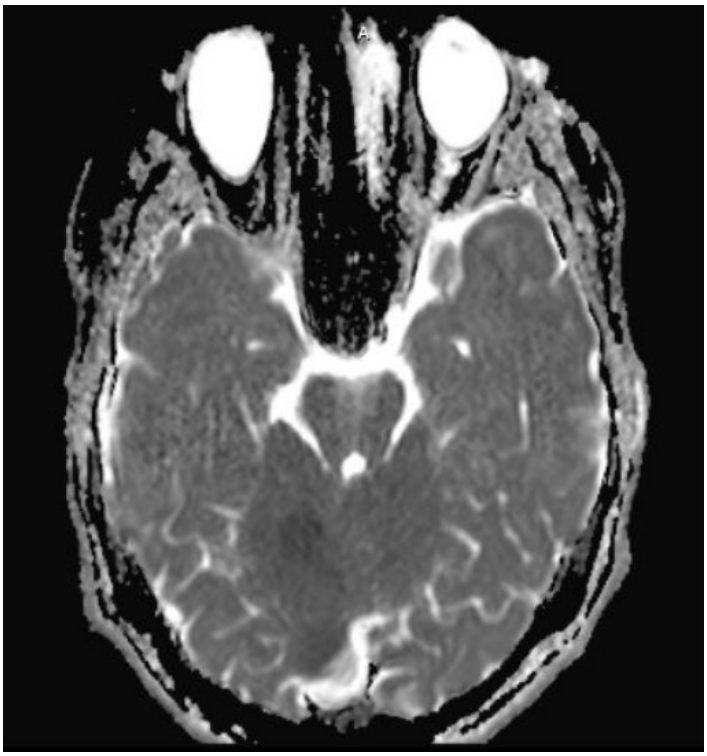

Critical Care 51 - Lance-Adams Syndrome After Cardiac Arrest And VA-ECMO Cannulation

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Introduction: Lance-Adams syndrome (LAS), or chronic post-hypoxic myoclonus, is a rare neurological complication following cardiac arrest with successful cardiopulmonary resuscitation (CPR)[1]. It is characterized by action myoclonus that develops and persists days to weeks after regaining consciousness [2]. Diagnosis is primarily clinical, as there are no specific consistent findings on either electroencephalogram (EEG) or brain imaging [3]. Recent studies suggest the pathophysiological basis of LAS may stem from hypoperfusion of the basal ganglia and thalamic regions, which then disrupts circuit transmission to other brain regions [4]. To date, less than 150 cases have been reported worldwide, and there have been no reported cases in patients who underwent extracorporeal CPR. Here we report a case of a patient who was placed on venoarterial extracorporeal membrane oxygenation (VA-ECMO) after CPR and subsequently developed LAS.

Methods: A 53-year-old male with unknown past medical history was brought in by Emergency Medical Services in cardiac arrest. He was initially found on the sidewalk, disoriented with agonal breathing and sluggish, dilated pupils. Upon arrival to the emergency department, his rhythm degenerated into pulseless electrical activity and CPR was initiated. Return of spontaneous circulation was achieved but ventricular fibrillation was then observed, with electrocardiogram showing ST elevations in the lateral leads. He received one round of defibrillation before being cannulated for peripheral VA-ECMO. Time from initial arrest to ECMO initiation was twenty minutes. He was transported emergently to the cardiac catheterization lab where he underwent placement of two drug-eluting stents in the proximal and mid-left anterior descending coronary artery. He returned for stenting of the right coronary artery two days later. His cardiac function recovered and he was successfully decannulated from VA-ECMO five days after admission.

During his initial intubation, the patient was noted to have violent shaking movements and was placed on continuous EEG upon arrival to the intensive care unit. Initial head imaging showed no acute findings. He continued to display episodes of myoclonus while intubated; however, his EEG demonstrated burst suppression with no corresponding seizure activity. The day after admission, he woke up neurologically intact, followed commands and was extubated the day after his second stent procedure. He continued to have significant intention tremors in bilateral upper and lower extremities as well as significant dysmetria where he was unable to even localize his head with his hand. He was started on Levetiracetam with moderate improvement in symptoms. Neurology was consulted and magnetic resonance imaging of the brain was obtained which showed mildly reduced diffusion involving the basal ganglia suggestive of either hypoxic ischemic injury or acute encephalitis. Based on the patient's constitution of symptoms, EEG, and imaging, a diagnosis of LAS was made. Given his difficulty even with feeding and walking due to tremors, he was discharged to a skilled nursing facility on Levetiracetam 1250mg twice daily, with arrangements for rehabilitation and outpatient Neurology follow-up.

Results: Not Applicable -- Medically Challenging Case Report
Conclusions: LAS remains a rare and difficult diagnosis that can severely impact a patient's quality of life. Early identification and management of symptoms as well as aggressive rehabilitation are key to preventing long-term disability. Currently, Levetiracetam, Piracetam, Clonazepam, and Valproate are recommended as treatment options [5]. As this appears to be the first case of LAS associated with ECMO, further research is needed regarding any association between extracorporeal CPR and the development or prevention of this disorder.

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Critical Care 52 - Management Considerations in a Patient with Patent Foramen Ovale with Thrombus Requiring Urgent Abdominal Surgery

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Introduction: The management of Patent Foramen Ovale (PFO) in patients with cryptogenic stroke requires a personalized approach, and the perioperative care of these patients requires multiple considerations. The numerous perioperative challenges in the management of these patients are highlighted by our case of a patient with recurrent cryptogenic stroke with a PFO, saddle pulmonary embolism, and large deep venous thrombosis (DVT) who developed acute gastrointestinal hemorrhage requiring urgent colectomy.

Methods: A 59-year-old male with ulcerative colitis, recent stroke in the setting of COVID-19 infection, and a patent PFO on dual anti-platelet therapy, presented with new upper and lower extremity weakness. Head CT revealed new areas of infarct, and the patient was admitted. Subsequently, after endorsing shortness of breath and leg swelling, he was found to have a large pulmonary embolus (PE) involving bilateral upper and lower lobes extending to the level of the sub- segmental arteries with evidence of right heart strain on echocardiogram. This prompted the patient's admission to the ICU. Ultrasound at the time showed deep vein thrombus from common femoral vein past the knee. Interventional cardiology and radiology teams were consulted for consideration of thrombectomy, but due to risk of further thromboembolism from the procedure via PFO, the care teams opted for a conservative approach with anticoagulation and delayed PFO closure. The patient was placed on a high-dose heparin drip. 24- hours after the initiation of heparin therapy the patient experienced an acute episode of hematochezia. Colonoscopy at the time revealed deep ulcers, friable mucosa, and clots throughout the rectum secondary to underlying ulcerative colitis. Due to the extent of the lesions and ongoing transfusion requirements, acute care surgery was consulted, and determined that a surgical colectomy was indicated. Creation of a surgical plan was complicated, however, due to the risk of major perioperative complications secondary to the presence of the patient's saddle PE and PFO. Given this, the patient was taken the next day to the interventional suite where interventional cardiology first performed a percutaneous PFO closure with a septal-occluding device under echocardiogram guidance. A clot-retrieving catheter was then advanced and thrombectomy performed, yielding significant improvement in pulmonary blood flows. Finally, an IVC filter was placed, and the patient was returned to the ICU. The rate of heparin infusion was decreased as he continued to have large volume hematochezia requiring blood transfusions. 72-hours following PFO closure and thrombectomy, the patient was taken to the operating room where he successfully underwent total abdominal colectomy. The patient's postoperative course was complicated by recurrent episodes of intraabdominal bleeding over the next few weeks due to anticoagulation, but he recovered and was eventually discharged to a rehabilitation center.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: PFO closure is not without complications, including stroke, and the decision to undertake this procedure is made on a case-by-case basis (Gonzalez, 2021). Surgery of any kind increases the risk of stroke in the setting of PFO, and risk is mitigated by antiplatelet therapy (Friedrich, 2019). This case details a patient with recurrent thromboembolic stroke due to a recurrence of venous thrombus, likely due to underlying inflammatory disease, with a PFO, who failed outpatient dual anti-platelet therapy. In this case, the confluence of competing surgical needs required careful coordination and timing of multiple procedures: thrombectomy of a massive PE causing right heart strain, PFO closure, and urgent abdominal colectomy, all in the setting of subacute thromboembolic stroke. PFO closure preceding thrombectomy prevented further systemic embolic insult, while thrombectomy stabilized the patient for general anesthesia for a subsequent colectomy.

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Critical Care 53 - Management of acute pulmonary hypertension due to aortic stenosis and an iatrogenic aorto-atrial fistula

Caroline Ruminski[1], Philip Leisy[1]

Vanderbilt University Medical Center[1]

Introduction: Pulmonary hypertension (PH) is defined as a mean pulmonary artery (PA) pressure > 20 mmHg.[1] It can be further classified into pre-capillary or post-capillary, based on pulmonary capillary wedge pressure ≤15 or >15 and pulmonary vascular resistance (PVR) ≤3 or >3 Wood's units, respectively (Figure 1).[1] WHO Group 2 PH, PH due to elevated left heart pressures, is the only mechanism that results in post-capillary hypertension. Etiologies encompassing WHO Group 2 PH include left sided valvular disease and heart failure with reduced or preserved ejection fraction.[2] Longstanding WHO group 2 patients can develop pre-capillary PH due to dysregulation of vascular smooth muscle tone and pulmonary vascular remodeling. Mislabeling patients as pre-capillary PH may result in potentially harmful and costly treatments.[2]

Methods: A 47-year-old male with a bicuspid aortic valve and severe aortic stenosis, hypertension, and prior alcohol abuse with hepatic inflammation, presented as a transfer from an outside hospital after an aborted aortic valve replacement. An intraoperative transesophageal echocardiogram (TEE) at the outside hospital showed near-systemic pulmonary artery (PA) pressures, severe aortic insufficiency, severe mitral regurgitation, and an aorto-left atrial fistula. One month prior, he had undergone an aortic balloon valvuloplasty, which reduced his mean transvalvular gradient from 85 mmHg to 26 mmHg. He arrived supported on 3 L/min nasal cannula (NC) and a milrinone infusion. Bedside transthoracic echocardiography demonstrated normal biventricular function, severely dilated and hypertrophied right ventricle with evidence of volume overload, severe aortic insufficiency, and mild mitral regurgitation. At that time, his systemic blood pressure was 129/53, PA pressure was 106/46, and CVP was 9 mmHg. Milrinone was increased to 0.5 mcg/kg/min and cardiac index was adequate. Pulmonary vasodilators were avoided due to presumed left sided pressures causing his pulmonary hypertension. He was diuresed on a furosemide infusion and PA pressures improved to 60s/30s with systemic pressures of 120s/50s. Pulmonary capillary wedge pressure on hospital day 2 was 57 mmHg. He underwent tissue aortic valve replacement with aortic root patch on hospital day 8. Post-procedural TEE was notable for aortic valve mean gradient of 13 mmHg with no communication between the aorta and left atrium. He was supported on milrinone, epinephrine, and inhaled epoprostenol post-operatively. He was extubated to 4L/min NC and epinephrine was weaned off on post-operative day (POD) 0. Epoprostenol was weaned off on POD1 and diuresis was initiated, facilitated by milrinone, which was weaned off on POD2. The remainder of his course was uncomplicated, and he was transferred to the ward on POD3 and discharged to home on POD7.

Results: Not Applicable-Medically Challenging Case Report

Conclusions: This patient presented with acutely elevated PA pressures in the setting of an iatrogenic aorto-left atrial fistula, superimposed on likely chronic group 2 PH in the setting of longstanding aortic stenosis. Despite near systemic PA pressures, he was well compensated on arrival, as indicated by a PA pulsatility index (PAPI) of 6.7. He was further optimized with diuresis to offload the right heart and inodilators to improve contractility and reduce left ventricular afterload. In this situation, it can be enticing to start pulmonary vasodilators; however, in the setting of severely elevated left sided filling pressures, the risk of inducing congestive pulmonary edema outweighs the unlikely benefit of improving right heart afterload when the PVR is normal. In patients presenting with undifferentiated acute or chronic pulmonary hypertension, it is imperative to gather left-sided filling pressure and PVR data to determine whether the mechanism of pulmonary hypertension is a pre-capillary or post-capillary etiology, as this can greatly influence the type of therapy that is chosen. This patient ultimately required definitive surgical repair of aortic stenosis, aortic insufficiency, and an aorto-left atrial fistula that were worsening his pulmonary hypertension.[3] His postoperative recovery was optimized in the perioperative setting using information gathered from transthoracic echocardiography, pulmonary artery pressures, and pulmonary capillary wedge pressure.

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Classification	Mean pulmonary artery pressure	Pulmonary capillary wedge pressure	Pulmonary vascular resistance
Isolated pre-capillary PH	>20 mm Hg	<15 mm Hg	>3 WU
Combined pre- and post-capillary PH		>15 mm Hg	>3 WU
Isolated post-capillary PH		>15 mm Hg	<3 WU

The 6th World Symposium on Pulmonary Hypertension defined three hemodynamic profiles of pulmonary hypertension (PH): isolated pre-capillary PH, combined pre- and post-capillary PH, and isolated post-capillary PH. WU, Wood units.

Figure 1: Hemodynamic profiles of pulmonary hypertension.[4]

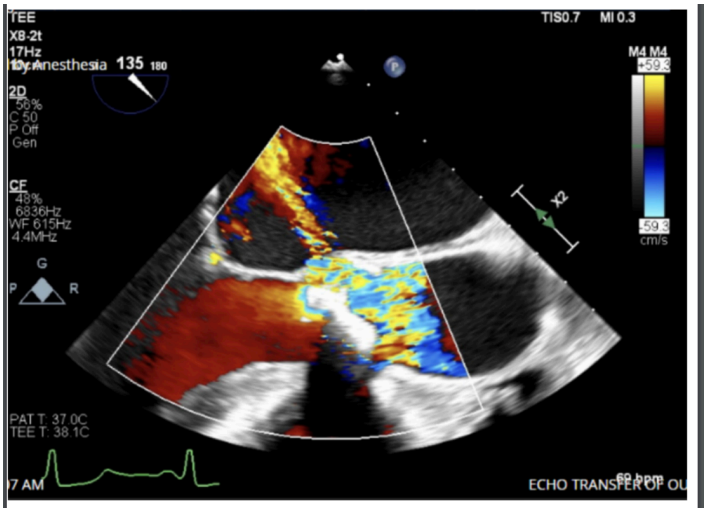


Figure 2: Transesophageal aortic valve long axis view demonstrating high velocity color flow Doppler jet from the aortic root into the left atrium.

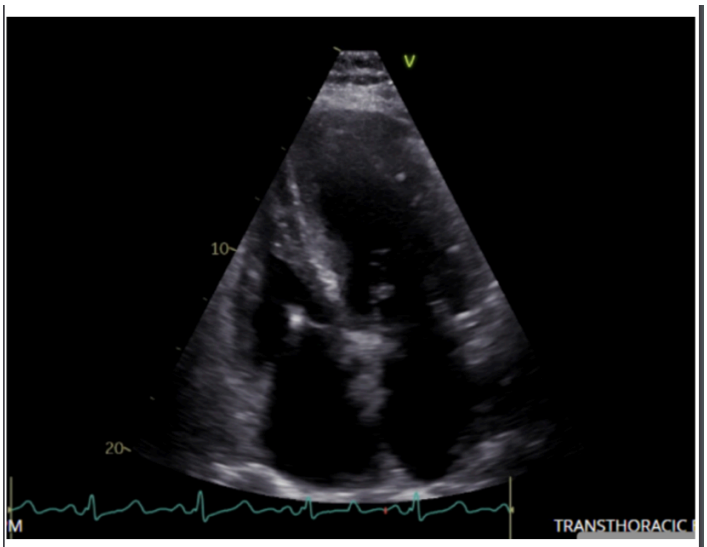


Figure 3: Transthoracic apical four chamber view demonstrating biventricular hypertrophy.

Critical Care 54 - Management of Large Mediastinal Mass in Intravenous Drug User: Role of Extracorporeal Membrane Oxygenation (ECMO)

Brian Sou[1], *Joshua Finkel*[1]

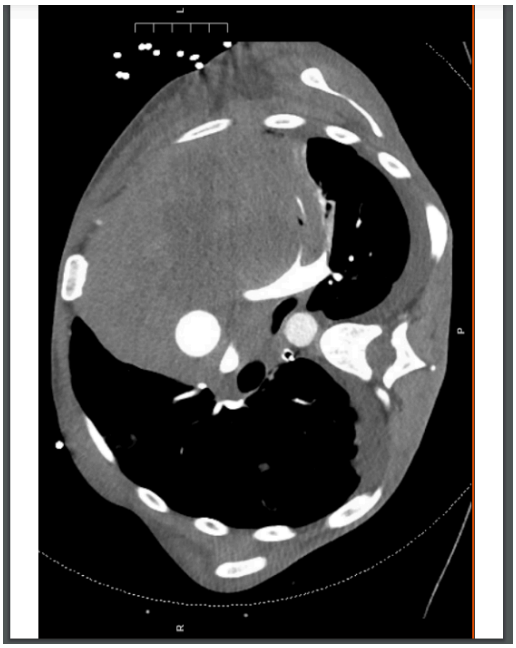
Brigham and Women's Hospital[1]

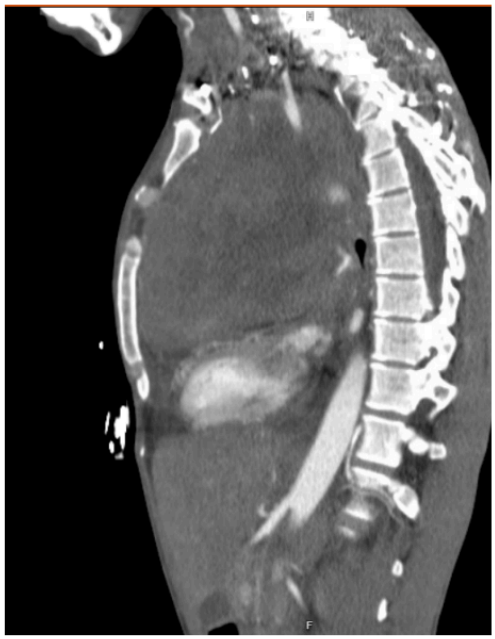
Introduction: Anterior mediastinal masses are uncommon pathologies that carry the risk of fatal consequences in the perioperative setting, particularly acute central airway collapse in the setting of general anesthesia. Avoiding cardiopulmonary compromise requires multidisciplinary assessment of the patient and meticulous planning. High-risk features, such as opioid use disorder, with respiratory symptoms may necessitate admission to the intensive care unit for close monitoring while perioperative planning is underway.

Methods: We present a 31-year-old female with opioid use disorder admitted for worsening dyspnea and was found to have a new large mediastinal mass compressing the great vessels and right ventricle. Initial attempts at intraoperative biopsy were aborted due to hemodynamic instability. She was admitted to the intensive care unit and femoral arterial and venous catheters were strategically placed in order to smoothly facilitate ECMO cannulation should she develop acute cardiopulmonary collapse. Several challenges were faced, including the patient's high opioid tolerance, low pain threshold, and balance of ICU sedation techniques without compromising her airway dynamics.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: For patients with concerning mediastinal masses, careful planning is required perioperatively in order to minimize the risk of a catastrophic event. Perioperative ECMO cannulation should be considered for high-risk patients requiring non-elective surgery. Traditionally, maintaining spontaneous, negative-pressure ventilation and avoiding neuromuscular blockade are prioritized. However, recent data suggests muscle paralysis may not lead to clinically significant airway collapse during anesthetic induction. Depending on surgical acuity, type of surgery, and patient symptoms, several anesthetic and advanced ventilation modalities should be considered to maximize patient safety.





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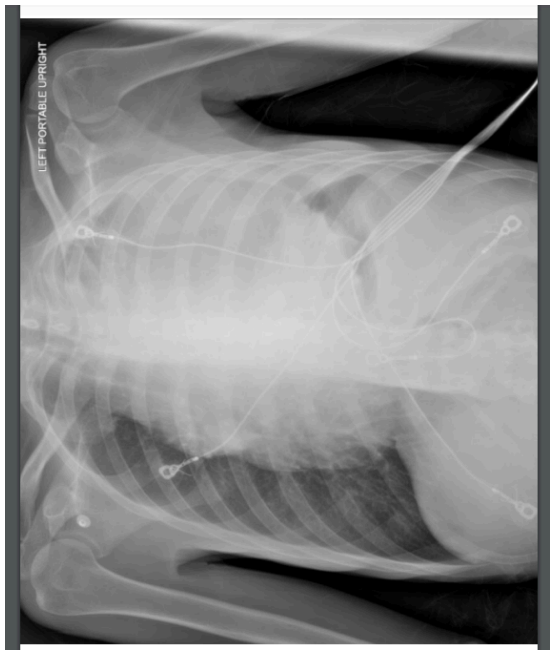
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Critical Care 55 - Managing Hemoptysis and Diffuse Thromboses Following CABG & Aortic Valve Replacement

Samuel Rackley[1], Abram Feldman[1]

Brigham and Women's Hospital[1]

Introduction: The purpose of this case report is to inform colleagues of the unique causes of hemoptysis, thrombocytopenia, and coagulopathy following cardiac surgery. This case of hemoptysis and diffuse thromboses followingn aortic valve replacement required the ICU team to manage a bleeding airway, diagnose coagulopathy, and manage therapeutic anticoagulation while preventing re-emergence of life-threatening hemoptysis.

Methods: This report highlights the case of an 83-year-old man who underwent CABG x 5 and biological Aortic valveimplantation with a post-operative course complicated by hemoptysis, diffuse thromboses, and multisystem organ failure. This case highlights frequent causes and management of hemoptysis following cardiopulmonary bypass. This case also highlights the causes and management of diffuse thromboses, including heparin-induced thrombotic thrombocytopenia. Finally, this case addresses the causes of post-operative thrombocytopenia following cardiopulmonary bypass with biologic valve implantation.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Following a review of available data, it is sensible to use a non-systemic, non-surgical management approach (endo-bronchial blocker) for focal hemoptysis in the immediate post-operative state following cardiac surgery. In our case, we benefitted from the swift identification of the source of hemoptysis, and the rapid implementation of endobronchial blockade to the site of bleeding. Hemoptysis following cardiopulmonary bypass can be paroxysmal and massive, requiring a swift response by either the intra-operative or intensive care team. The management of hemoptysis requires the ability to evacuate blood rapidly to locate the source, maintaining ventilation if possible, or venovenous ecmo if not. Ultimately, management of hemoptysis requires addressing the bleeding airway, ventilation, and maintaining circulatory volume in a hemorrhagic patient. Multiple data sources support the suspicion of heparin-induced thrombotic thrombocytopenia in our case. Despite unusual timing, HITT was suspected due to the absence of lab markers consistent with DIC, presence of arterial and venous thrombosis, and skin necrosis on approximately post-op day 5 with thrombocytopenia. Our case reviews the management of HITT by the American Society of Hematology, which demonstrates the counter intuitive conclusion heparin should not have been stopped despite our suspicions of HITT.

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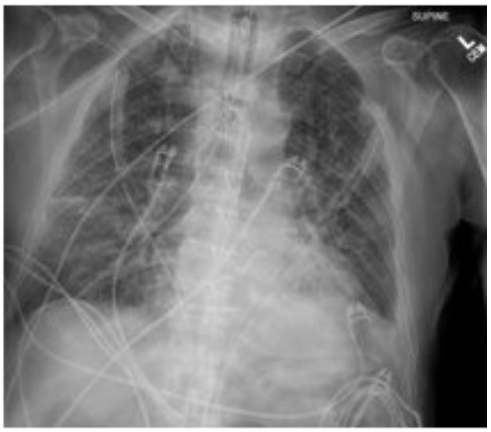
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Critical Care 56 - Massive Gastrointestinal Bleed: the Minnesota Tube

Petra Elias[1], Amanda Fields[1]

Mayo Clinic[1]

Introduction: Treatment of upper gastrointestinal (GI) tract hemorrhage secondary to esophageal or gastric variceal rupture has been well-described. Common interventions include pharmacologic, endoscopic, surgical, and vascular therapies[1]. When these options are either unavailable or fail, tamponade may be a viable modality to treat uncontrolled hemorrhage[2]. The Minnesota tube is a 4-lumen device that offers the ability to tamponade bleeding from both the esophagus and stomach via esophageal and gastric balloons while also providing esophageal and gastric suction[1]. We describe the application of the Minnesota tube in a patient whose uncontrolled upper GI hemorrhage was secondary to non-steroidal anti-inflammatory drug overdose rather than varices.

Methods: A 72-year-old male presented to the emergency department with hypotension and a two-day history of melena followed by acute onset dyspnea, angina, and hematemesis. His symptoms were preceded by the ingestion of 20,000 mg of aspirin to treat right leg pain. He was resuscitated and admitted but decompensated shortly after arrival to the ICU, where he required emergent intubation for airway protection and on-going massive transfusion. After stabilization, he underwent computerized tomography imaging of the chest, abdomen, and pelvis, which identified active hemorrhage from the left gastric artery. He was emergently transferred to interventional radiology for embolization, where he continued to have large volume hematemesis. Due to the brisk bleeding, the Minnesota tube was placed, which allowed for tamponade and successful embolization of the left gastric artery. However, hematemesis continued after deflation of the tube. Persistent hemorrhage from the splenic artery was identified, which was subsequently embolized. Resolution of bleeding was confirmed via deflation of the Minnesota tube under fluoroscopic visualization. The patient received 34 units of blood products throughout this process. After a brief period of hospitalization, he was successfully discharged.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Minnesota tube placement is indicated as a temporizing measure for uncontrolled gastric or esophageal hemorrhage. Caution should be undertaken in patients with esophageal stricture or recent esophageal/gastric surgery given potential for esophageal rupture and necrosis.

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Critical Care 57 - Mediastinitis and Pneumomediastinum secondary to Left Subclavian Central Venous Catheter Erosion and Perforation of the Superior Vena Cava

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Mayo Clinic Department of Anesthesiology[1] Mayo Clinic[2]

Introduction: Central venous catheter (CVC) placement is a commonly performed procedure that is associated with significant risks. This case highlights the rare late complication of CVC erosion with vascular perforation through the superior vena cava resulting in mediastinitis.

Methods: 81 yo female with past medical history notable for CAD s/p 3 vessel CABG, ischemic cardiomyopathy (LVEF 56%), paroxysmal atrial fibrillation, HTN, PAD s/p left CEA, severe COPD (FEV1 43% predicted), CKD, s/p left total hip replacement c/b infection on lifelong antibiotic suppression, rectal cancer s/p proctocolectomy presented to the Emergency Department with bilateral lower extremity pain. Workup was remarkable for critical limb ischemia secondary to extensive atherosclerotic disease affecting the aortoiliac and common femoral arteries bilaterally. She underwent bilateral common femoral endarterectomy and retrograde iliac artery stent placement. A left subclavian clip in triple lumen CVC was placed without complication prior to this procedure under ultrasound guidance with confirmatory chest radiography. Intraoperative course was notable for hemorrhagic shock due to right external iliac artery rupture requiring massive transfusion protocol (9 u pRBCs, 5 u FFP, 2 u plt, 4 u cryoprecipitate), stent placement, exploratory laparotomy, and ligation of the right internal iliac artery. Ten days after this operation the patient developed hypotension and altered mentation. ABG was remarkable for severe respiratory acidosis. The patient was re-intubated and underwent bronchoscopy which demonstrated diffuse white exudates. Further workup with CT imaging (**A**) demonstrated that the subclavian CVC had eroded through the later wall of the SVC and migrated into the mediastinum with resultant mediastinitis, as depicted by the dark-appearing air and surrounding fluid collection (shaded orange). Purulent fluid was easily aspirated from the triple lumen CVC at bedside. Perforation through the right lateral SVC wall was confirmed by central venogram (**B,** arrow), followed by fluoroscopically guided catheter removal and balloon tamponade for control of extravasation. Due to extensive vascular access issues a right EJ triple lumen non-tunneled CVC was placed. Thoracentesis yielded 200 mL of straw colored exudative fluid. Bronchial washings grew multi-drug resistant Klebsiella Pneumonia and E. coli. No growth was noted from her mediastinal cultures. Incrementally she improved and was discharged home in stable condition.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Central venous access is a commonly performed procedure in intensive care units that facilities numerous venous interventions as well as device insertions. The site and indication is dependent on indications and patient related factors. Numerous complications are associated with CVC placement with the majority recognized at the time of insertion. CVC erosion with vascular perforation is a rare complication of central line placement, with catheters introduced via the left subclavian vein most often involved.[1,2] This is likely related to the acute angle at which the left brachiocephalic vein meets the SVC,[3] which may be exacerbated by prolonged maintenance of catheters terminating at this high-risk anatomic site. Prevention strategies include avoiding left subclavian insertions when alternate sites are available, ensuring catheter tip termination in the "safe zone" (**B,** shaded blue) parallel to the venous wall in the lower SVC, avoiding catheter tip abutment against the SVC wall, and removing catheters when clinical necessity has resolved. In the event of erosion into the mediastinal cavity prompt recognition and safe catheter removal are essential for the optimization of outcomes. Catheter removal should be performed under real-time fluoroscopic guidance with expert consultation, as active extravasation may require balloon tamponade or other interventions for hemostatic control.

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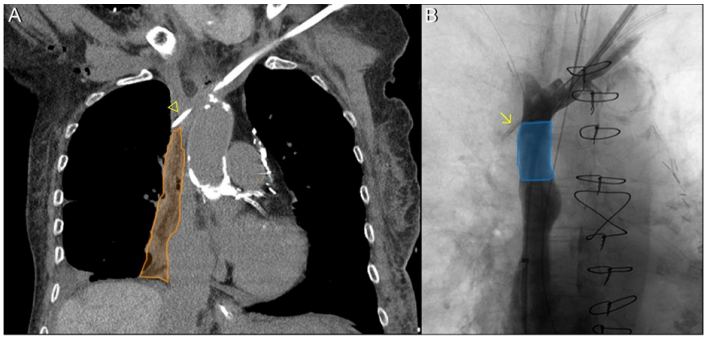

Critical Care 58 - Methylene blue to the rescue? A complicated picture of serotonin syndrome

Alexis Keyworth[1], Caitlin Blaine, Hans Tregear[2]

University of Colorado[1] University of Colorado - Anschutz Medical Campus[2]

Introduction: Serotonin syndrome is a potentially lethal syndrome caused by excess serotonin leading to characteristic clinical findings of altered mental status, neuromuscular changes, and autonomic instability. It can be caused by use of selective serotonin reuptake inhibitors (SSRIs) or drug interactions, particularly concomitant use of SSRIs with monoamine oxidase inhibitors (MAOIs). Methylene blue is a potent MAOI frequently used to treat vasoplegic syndrome in cardiac surgery patients due to its effects on the nitric oxide pathway.[1] We present a case of a patient who suffered from serotonin syndrome after receiving methylene blue while undergoing heart transplantation.

Methods: A 61-year-old male with a history of ischemic cardiomyopathy status-post left ventricular assist device presented to our institution in summer 2022 with cardiogenic shock secondary to pump thrombosis and underwent a heart transplant shortly after his admission. Of note, at the time of admission, he was taking sertraline for depression. His intra-operative course was remarkable for significant vasoplegia and moderate biventricular dysfunction requiring high-dose vasopressors, inotropes, and administration of methylene blue. He was admitted to our cardiothoracic intensive care unit for ongoing management following his operation. On postoperative day zero he developed a new onset of nystagmus, hyperreflexia, lower extremity clonus, severe upper and lower extremity hypertonia associated with a fever as high as 38.9° Celsius and a new lactic acidosis. Neurology and toxicology were consulted and determined that the constellation of symptoms was most consistent with serotonin syndrome, likely caused by the interaction between the patient's chronic SSRI use and methylene blue administration. Treatment was started immediately with a midazolam infusion that was titrated to the cessation of neuromuscular symptoms and cooling pads. After 24 hours, the midazolam infusion was weaned off without recurrence of symptoms. The patient's course progressed as expected and he was discharged to rehab without symptoms of serotonin syndrome approximately two weeks after his operation. The patient's SSRI was not resumed for the duration of his hospital stay.

Results: Not Applicable -- Medically Challenging Case Report

Conclusions: Cardiovascular disease is associated with higher rates of depression and use of SSRIs.[2-3] Many of these patients may need to undergo cardiac surgery to improve their quality of life. Vasoplegic syndrome is a frequent complication of cardiopulmonary bypass with increasing use of methylene blue as a treatment. It is important to be aware of the potential for serotonin toxicity with the use of this medication in patients taking SSRIs[1] and alternative treatment strategies for vasoplegic syndrome should be considered in this patient population.

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Critical Care 59 - Mirror Syndrome - A Rare Cause of Acute Respiratory Distress Syndrome in Pregnant Women

Sonja Foo[1], Kate Frost[1], Michael Furdyna[1], Daniela Lazea[2]

Brigham and Women's Hospital[1] BWH[2]

Introduction: Mirror Syndrome (MS), also known as Ballantyne Syndrome, pseudotoxemia, acute second trimester gestosis, maternal hydrops syndrome, or triple edema, is a rare obstetric condition with a reported incidence of 1 in 3000 pregnancies [1]. First described by Scottish obstetrician Dr. John William Ballantyne in 1892, it is characterized by fetal hydrops with mirrored placental and maternal edema [2]. The etiology of fetal hydrops in MS varies, with reports describing fetal anemia from Parvovirus B19, high fetal cardiac output from twin-to-twin transfusion syndrome and fetal anomalies like a cystic hygroma [3]. Maternal effects range from weight gain, hypertension, and proteinuria to severe pulmonary edema [4]. The pathophysiology of this syndrome, however, is poorly understood and is thought to emulate preeclampsia in its association with defective placental angiogenic factors [5]. Here we describe MS that progressed to acute respiratory distress syndrome (ARDS) in a 33-year-old woman following pregnancy termination.

Methods: A 33-year-old female with a history of a pituitary adenoma and exercise-induced asthma presented to our intensive care unit for acute hypoxic respiratory failure that required intubation following a surgical abortion. Her pregnancy, conceived by in vitro fertilization, was complicated by chronic hypertension and an enlarging fetal sacrococcygeal teratoma (SCT), causing fetal hydrops and placentomegaly, which was noted at 21 weeks and 6 days. Despite an attempted intrauterine transfusion and SCT vessel embolization, the decision was made to terminate her pregnancy at 22 weeks and 5 days given worsening maternal anemia, hypoalbuminemia and transaminitis, as well as a deterioration in fetal status with absent umbilical Dopplers. A dilation and evacuation were initially performed under monitored anesthesia care but was converted to general anesthesia with intubation when she was noted to have increased secretions and reflux of gastric contents. She remained hemodynamically stable and was adequately oxygenated and ventilated. Estimated blood loss was 500 mL, for which she was transfused 1 unit of packed red blood cells. At the conclusion of the case, she was easily extubated to room air. One hour later, she became hypoxic and dyspneic, with oxygen saturations in the low 90s. Her chest radiograph showed bilateral infiltrates despite a normal bedside echocardiogram. She was given supplemental oxygen, nebulizers, and diuretics to treat potential aspiration pneumonitis, asthma exacerbation, transfusion related lung injury and volume overload. Despite this, her oxygenation worsened, and she ultimately required initiation of paralysis per ARDS net protocol after transfer to the intensive care unit. On arrival, her initial P/F ratio was 130 and she required paralysis for ventilator dyssynchrony. Broad spectrum antibiotics were started for a new leukocytosis, which is inconsistent with MS, but no pathogen was ever identified. A formal echocardiogram did not reveal any cardiac dysfunction although she briefly required vasopressors for hypotension in the setting of heavy sedation. After 2 days of diuresis with a furosemide infusion and intermittent albumin boluses, her P/F ratio increased to 310 and she was successfully extubated.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Our case demonstrates an unusual etiology for ARDS in the pregnant or postpartum patient. While multiple causes of our patient's acute respiratory decompensation were considered, the timing of her deterioration and rapid improvement with supportive therapy appeared consistent with her known MS. An awareness of the presenting symptoms and signs of MS can aid in the diagnosis and treatment of maternal respiratory distress.

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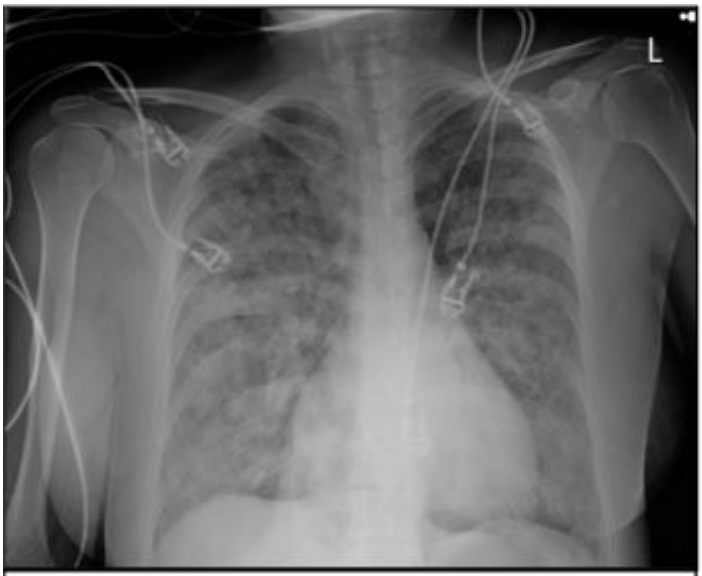
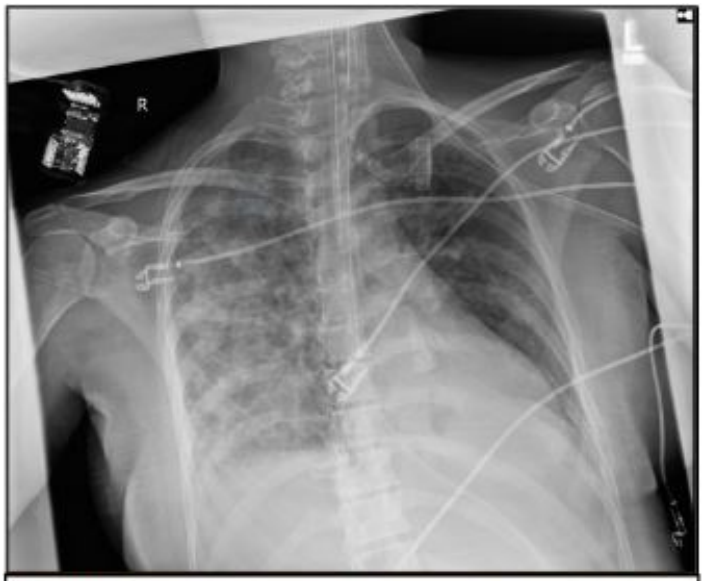


Chest radiograph post-dilation and evacuation and prior to intubation



Chest radiograph 2 days post intubation for acute respiratory distress syndrome and day of extubation

Critical Care 60 - Myocardial Ischemia Immediately Following Coronary Artery Bypass Graft Surgery Due to External Graft Compression.

Rebecca Piland[1], Kunal Karamchandani

University of Texas Southwestern[1]

Introduction:

1) Recognize acute myocardial ischemia immediately after CABG surgery

2) Enumerate causes of graft dysfunction post-CABG surgery

3) Summarize management of myocardial ischemia post-CABG surgery

Methods: A 73year-old female with no significant past medical history presented with chest pain and was found to have elevated serum troponin levels. A trans-thoracic echocardiogram (TTE) showed left ventricular ejection fraction (LVEF) of 66% with no regional wall motion abnormalities (RWMA), however, a nuclear stress test revealed, inducible inferolateral perfusion defect. Left heart catheterization demonstrated 90% distal left main coronary and severe three vessel coronary artery disease (CAD). She underwent intra-aortic balloon pump (IABP) placement followed by an uneventful three vessel coronary artery bypass graft (CABG) surgery on cardio-pulmonary bypass. Her post-pump transesophageal echocardiogram (TEE) did not reveal any RWMA, and she was admitted to the intensive care unit (ICU) after surgery. On admission to the ICU, she remained intubated, and required inotropic support with 0.02mcg/kg/min of epinephrine and 1:1 augmentation with IABP. Immediately after surgery, she had rising creatine kinase-myoglobin binding (CKMB) levels and decreasing mixed venous O2, requiring continued inotropic support. IABP was removed 24 hours after surgery and she was extubated early morning on postoperative day (POD) 2. Shortly after extubation, her EKG showed ST segment elevation in leads V1-3 (picture 1) and repeat TTE showed LVEF ~51% with new RWMAs. Though the patient denied any chest pain, she did endorse nausea and symptoms of gastro-esophageal reflux. Given these findings, along with significant baseline CAD and small size of native coronary vessels, she underwent left heart catheterization where it was discovered that the proximal LIMA graft was severely obstructed from external impingement by the tip of the left sided chest tube. (picture 2) The chest tube was removed, and the proximal obstruction entirely resolved. Additionally, the distal LIMA graft was found to have two, serial 80-90% lesions proximal to the anastomosis to the LAD and required percutaneous transluminal coronary angioplasty and DES placement. Immediately post-procedure, ST elevations resolved (picture 3), CKMB levels began to normalize, and she was weaned from inotropic support.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Post-CABG myocardial ischemia is an uncommon, yet lethal complication associated with high morbidity and mortality. We present a case which describes an unusual cause of iatrogenic acute myocardial ischemia, that is potentially preventable.

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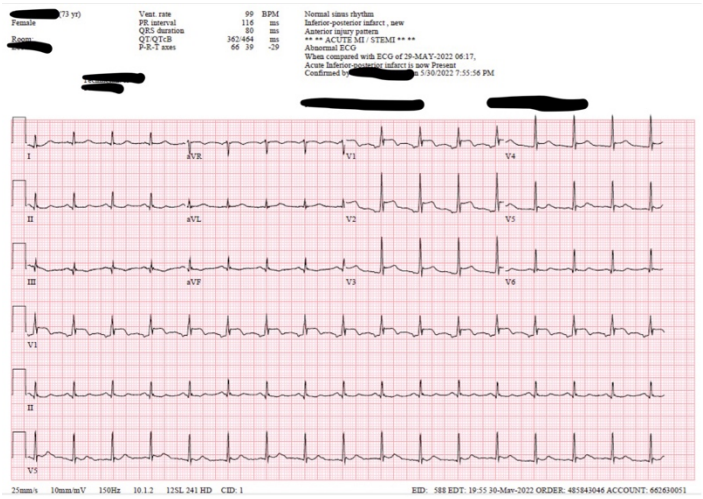

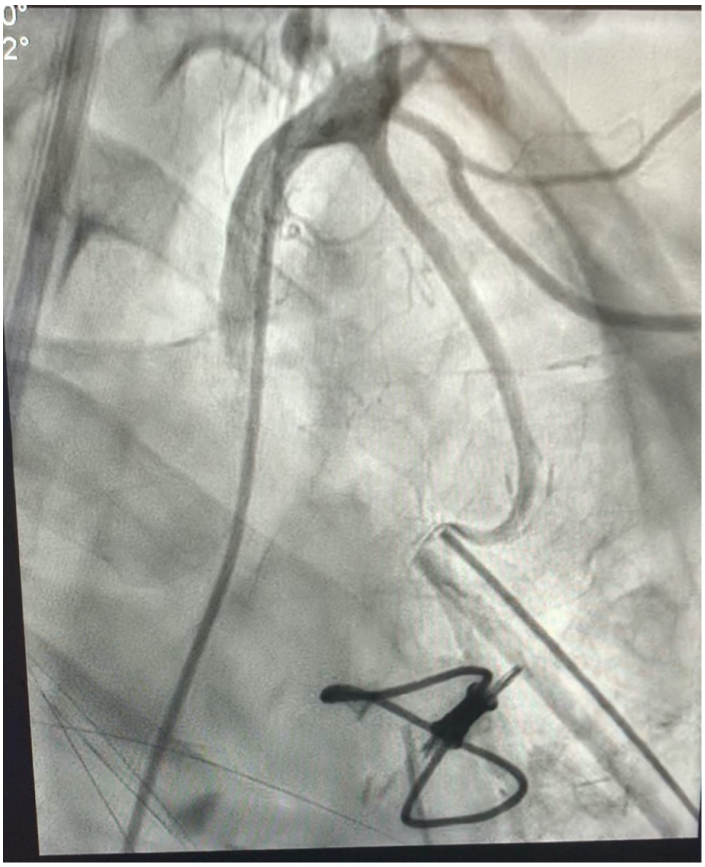
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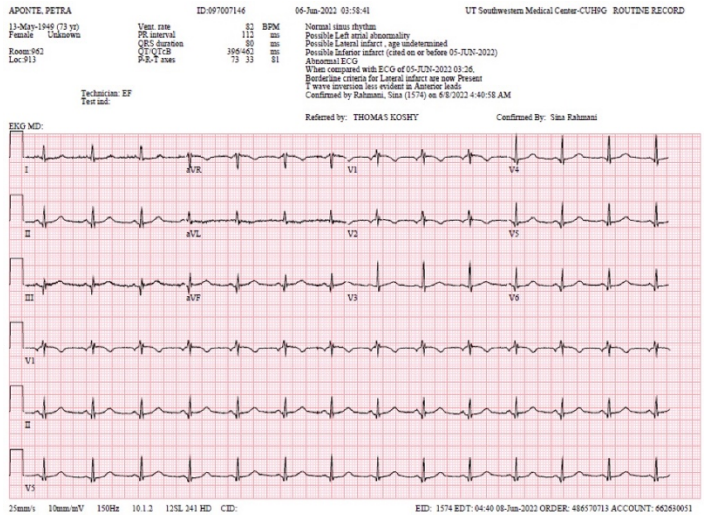
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Normal sinus rhythm
Possible Left atrial abnormality
Possible Lateral infarct , age undetermined
Possible Inferior infarct (cited on or before 05-JUN-2022)
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Critical Care 61 - New Onset Cardiomyopathy in a Teenager

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Introduction: There are many sequalae of COVID-19 infection beyond the known pulmonary implications, including myocarditis and hypercoagulability.[1-3] As an increasing number of patients are admitted to the hospital with a history of COVID-19 infection(s), the non-pulmonary sequelae may become more apparent, and COVID-related complications are increasingly being considered as possible etiologies of presenting diagnoses. We present a case of newly diagnosed severe cardiomyopathy one month after COVID-19 infection with additional complications during his hospital course where sequelae of COVID-19 infection were considered.

Methods: A previously healthy 16-year-old male presented with vomiting and syncope one month after mildly symptomatic COVID-19 infection. He was unvaccinated against COVID-19 and reported COVID-19 infection one year prior also with mild symptoms. Family history was notable for a brother who was recently hospitalized with COVID-19 pneumonia. During his workup for vomiting and syncope, he was diagnosed with severe dilated cardiomyopathy with ejection fraction of 24%. Initially COVID-19 myocarditis was suggested as the etiology; however, cardiac MRI showed epicardial scar throughout the left ventricle without edema, most consistent with chronic myocarditis or genetic cardiomyopathy rather than an infectious etiology. Despite oral heart failure therapy, his symptoms worsened and he underwent placement of a temporary percutaneous transvalvular left ventricular assist device (PLVAD, Impella 5.5) as bridge to heart transplant. Unfortunately, the PLVAD tip became malpositioned resulting in frequent ventricular ectopy and repositioning the device led to unstable ventricular tachycardia requiring cardioversion. The decision was made to place an emergency durable LVAD due to continued hemodynamic instability. Intraoperatively, the PLVAD tip was found to have extensive thrombus (Figure 1). He was extubated within a day of surgery; however, his postoperative course was complicated by acute kidney injury and hypoxemic respiratory failure. He was re-intubated, given nitric oxide, and started on empiric antibiotics. Despite adequate ventilator settings for ARDS, pO_2 remained less that 90 mmHg on an FiO_2=1. CT scan showed significant bilateral ground glass opacities thought to be more consistent with pulmonary edema, especially since all cultures for infectious etiologies were negative (Figure 2a). His clinical condition deteriorated and VV ECMO was considered to treat his hypoxemia and hypercarbia; however, pharmacologic paralysis and diuresis demonstrated remarkable improvement in ventilation over two days. He continued to make clinical improvement, remaining off dialysis, and ultimately received a heart transplant three weeks later. CT scan around the time of transplant showed marked improvement in lung aeration (Figure 2b). He stayed in the hospital for 68 days and was discharged on post-op day 13 after heart transplant. He continues to have good cardiac function without need for readmission and is currently enrolled in college.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: More patients are being admitted to the hospital with a history of COVID-19 infection, whether symptomatic or incidentally found. When treating these patients, clinicians need to consider sequalae of COVID-19 infection in the differential diagnoses; however, care must be taken not to narrow the differential too quickly. In this case, the initial presentation of myocarditis in an otherwise healthy teenager was initially considered to be related to COVID-19, but upon further investigation, the etiology was more likely chronic myocarditis or a genetic cardiomyopathy. The blood clot at the PLVAD tip was unusual. Although it could be related to COVID-19 hypercoagulability, it could also be attributed to the malposition in the left ventricle or acquired during removal through the axillary graft. Furthermore, the patient's pulmonary decline after durable LVAD implantation could also be related to pulmonary complications from COVID-19 or pulmonary embolism related to hypercoagulability. This was ultimately thought to be pulmonary edema, although he received empiric antibiotics to rule out superimposed infection. Sequelae from COVID-19 infection may not have been the cause for each of these issues that arose during his prolonged hospital course, but they remained high on the differential diagnosis throughout his hospitalization.

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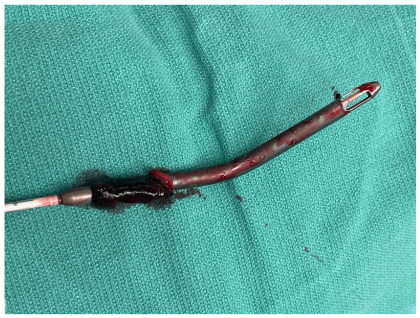

Figure 1. Picture of temporary percutaneous transvalvular left ventricular assist device (PLVAD, Impella 5.5) tip after removal from patient.

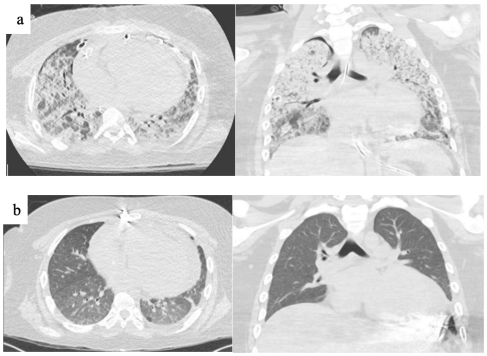

Figure 2: a) Computed tomography (CT) of chest showing significant bilateral ground glass opacities postoperatively from LVAD placement. b) CT of chest prior to heart transplantation one month later showing marked improvement in aeration of both lungs with residual bibasilar ground glass opacities.

Critical Care 62 - Novel use of rituximab for acquired hemophilia A in a patient with persistent post-tonsillectomy hemorrhage

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UT Southwestern Medical Center[1] University of Texas Southwestern Medical Center[2]

Introduction: Acquired hemophilia A (AHA) is a rare bleeding disorder characterized by autoantibody-mediated depletion of coagulation factor VIII (FVIII). FVIII is an essential component of the intrinsic pathway of coagulation, and its depletion can lead to life-threatening bleeding. Patients with AHA will most often present with severe subcutaneous or soft tissue bleeding, although muscular, vaginal, gastrointestinal, and joint bleeding can be seen as well. Prompt diagnosis and treatment is paramount, as AHA is associated with significant morbidity and mortality. This case reports highlights the novel use of rituximab to treat AHA in a post-tonsillectomy patient with persistent hemorrhage despite undergoing multiple surgical procedures to establish permanent hemostasis.

Methods: A 65-year-old male on aspirin and clopidogrel presented as a transfer due to persistent bleeding after undergoing a bilateral tonsillectomy, partial uvulectomy, right myringotomy and tube placement due to right sided peritonsillar abscess. During this initial procedure, he required multiple blood transfusions and remained intubated postoperatively for airway protection due to persistent oral bleeding. After arrival, despite undergoing left pharyngeal artery embolization, he continued to have persistent bleeding from the oral mucosa resulting in multiple blood transfusions and ultimately two additional trips to the operating room with ENT in order to establish hemostasis. He remained intubated due to continued slowing oozing from the oral mucosa despite these interventions so hematology was consulted for possible clotting disorder. He was noted to have had a PTT of 104.6 seconds prior to transfer and on the day of consultation, his PTT was 53.4 seconds. That, in combination with a Factor VIII activity of 10%, ultimately led to diagnosis of acquired hemophilia with autoimmune antibody to Factor VIII. Under the supervision of hematology, the patient was started on a combination therapy of recombinant factor VIIa and anti-inhibitor coagulant complex with close monitoring of hemostasis as well as daily PTT and Factor VIII levels. He was eventually transitioned from recombinant factor VIIa N and anti-inhibitor coagulant complex to antihemophilic factor (recombinant), porcine sequence. Shortly before discharge, emicizumab-kxwh was added to his regiment and he was transitioned to a regimen of daily prednisone and rituximab weekly for eradication of the autoimmune antibody.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: In this case, the off-labeled use of rituximab was utilized per the recommendation of the hematology team for the treatment of acquired hemophilia A. Rituximab is a recombinant chimeric murine/human antibody directed against the CD20 antigen, a hydrophobic transmembrane protein located on normal pre-B and mature B lymphocytes. Following binding, rituximab triggers a host cytotoxic immune response against CD20-positive cells. Typical labeled uses for Rituximab include the following: chronic lymphocytic leukemia, non-Hodgkin lymphomas, pemphigus vulgaris, rheumatoid arthritis, antineutrophilic cytoplasmic antibody (ANCA)-associated vasculitis. However, it has been used off-label for a multitude of indications, including as treatment for acquired hemophilia conditions such as described above.

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Critical Care 63 - Pace Maker Mediated Supraventricular Tachycardia mimicking Ventricular Tachycardia postoperatively

Ricky Hoang[1], Ticha Munda

University of Rochester Medical Center[1]

Introduction: Cardiac arrythmias are a common problem in intensive care units and are a major source of morbidity. Tachyarrhythmias are of particular concern. In the case of tachyarrhythmias, there is an immediate need to differentiate between supraventricular tachycardia (SVT) and ventricular tachycardia (VT). We present a case in of a patient with a pacemaker developing what appeared on telemetry to be ventricular tachycardia, but later investigation indicated the possibility of pace maker mediated SVT was more likely.

Methods: A 69-year-old male with paroxysmal atrial fibrillation, right bundle branch block, hypertension, hyperlipidemia, sick sinus syndrome, dual chamber pacemaker and Crohn's colitis presented to the hospital with a leak following an open ileocectomy. He underwent an exploratory laparotomy, small bowel resection, repair of enterotomy and washout. His bowel was left in discontinuity and he was admitted to the Surgical Intensive Care Unit. Postoperatively the patient experienced two episodes of atrial fibrillation with rapid ventricular response for which he was given metoprolol and started on amiodarone. The amiodarone was transitioned to digoxin and the patients home medications for atrial fibrillation apixaban, metoprolol and dofetilide were held. The patient required multiple trips to the OR for washout before abdominal fascial closure. A week after patient's abdominal closure, the patient developed what appeared to be VT on telemetry with heart rate (HR) around 130. The patient was hemodynamically stable and alert at the time. Vagal maneuvers were unsuccessful. The patient was given 6mg adenosine and went back into normal sinus rhythm after 10 minutes. External cardioversion was not needed. Cardiology was consulted after the event. Interrogation of patient's pacemaker afterwards showed a run of SVT.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Wide complex tachycardias can either be VT or SVT with aberrancy. It is sometimes difficult to distinguish between the two, especially when time is of the essence. In our patient, the initial concern was for VT especially with the failure of vagal maneuvers. However, the arrhythmia resolved after the use of adenosine, which lends some credence to the arrhythmia being SVT. During interrogation of the pacemaker afterwards, a run of SVT was noted but no events were noted on the date of this tachyarrhythmia event. This run of SVT could have been stimulated by the Sudden Brady Response (SBR) of the pacemaker, which will pace the heart at an elevated rate when a sudden drop in HR is sensed. The patient had a history of the SBR being triggered when his HR dropped to less than 70. Interestingly, the max paced HR under the SBR algorithm should be 130 but the patient's HR went above 130 during this event. The SBR would only be in effect for a short duration of time before automatically dropping back to the regular set pacemaker rate. The other possible explanation would be pacemaker mediated tachycardia (PMT. The SVT caused by PMT would be terminated using adenosine as well, or it could be terminated by the pacemaker's native PMT termination feature. We present an interesting case of a paroxysmal VT-mimicking SVT that may have been pacemaker mediated in a stable ICU patient that resolved with the use of adenosine.

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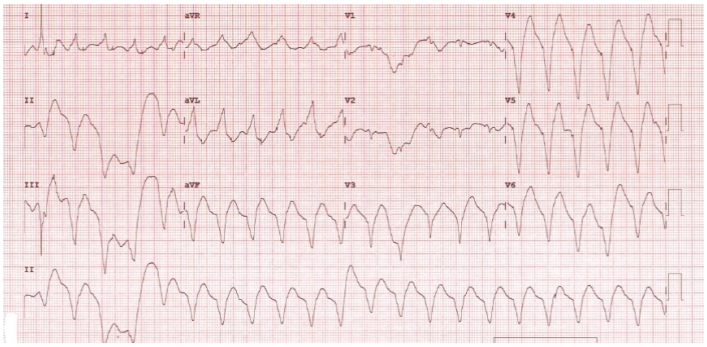

Critical Care 64 - Percutaneous RVAD Motor Failure during Cardiogenic Shock secondary to STEMI

Brian Hanst, Scott Erwin[1], balbino lopez

University of California, Irvine[1]

Introduction: The Impella® RP is a commonly used right ventricular assist device (RVAD) indicated for mechanical support of the right ventricle in patients with RV failure secondary to myocardial infarction (1). It is placed through femoral vein access through the inferior vena cava with the outflow cage of the device sitting in the pulmonary artery just before the pulmonary artery bifurcation. This RVAD is approved by the FDA to provide RV support for up to 14 days. There is currently limited safety data for this device and similar types of devices. We present the case of a patient in cardiogenic shock who required mechanical support with percutaneous RVAD, with the unfortunate complication of motor failure requiring removal prior to scheduled coronary artery bypass graft. We aim to foster discussion surrounding next steps in the face of RVAD failure and to promote diligent correspondence with manufacturers to optimize the safety of these devices for all patients who need them.

Methods: The patient is 69-year-old male with coronary artery disease with a stent placed ten years ago, peripheral vascular disease complicated by right foot amputation, and type II diabetes who presented to our facility from an outside hospital for higher level of care for cardiogenic shock in setting of coronary artery disease. Patient had presented to an outside facility for left foot osteomyelitis leading to left fourth and fifth ray amputation with subsequent left superficial femoral/popliteal artery angioplasty and stenting. This was complicated by postprocedural thrombosis requiring bypass to the anterior tibial artery that had to be aborted due to extensive saphenous vein insufficiency. Two days later, patient had a STEMI. On left heart catheterization, patient had 80% distal LM stenosis, 90% ostial LAD stenosis, and 100% RCA stenosis. An Impella® was inserted during the left heart catheterization due to a drop in the blood pressure during the procedure with poor response to vasopressors. He arrived at our facility with norepinephrine and heparin infusions. On arrival, patient had a white cell count of 22.9 mg/dL, hemoglobin of 7.9 mg/dL, PT 17.2, INR 1.46. On the second day of hospital course, the patient had an episode of ventricular tachycardia to the 170s requiring defibrillation at 200J. The RVAD settings were increased to P7. The next day, the patient developed atrial fibrillation and went into ventricular tachycardia requiring another shock at 200J. The RVAD could not be increased to P8 due to suction event, a lidocaine infusion was started for rhythm control. The patient was set to undergo CABG the following day. Overnight, the medical team was called by the bedside RN because the Impella® motor stopped working. The Impella® was removed after speaking with the speaking with the device representative. Repeat Impella® was not placed due to compromised femoral access from extensive vascular disease. Two hours later, the patient went into pulseless ventricular tachycardia and code blue was called. He received 3 defibrillation shocks and was intubated. The patient was emergently placed on right sided veno-arterial extracorporeal membranous oxygenation, with infusions of dobutamine, epinephrine, norepinephrine started. Over the course of the nexttwo days, the patient went into renal failure and his family decided to pursue comfort care. The patient expired three days after RVAD failure.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: This case demonstrates the catastrophic consequence of RVAD motor failure in patients with cardiogenic shock. In a query of a database of medical device reports that are submitted to the FDA, between 2009 and 2018 it was found that out of 436 device reports for the Impella® RP, 20 cases reported to have injury, 10 reported malfunction, and 5 reported death (2). Most common modes of failure were fracture of the device elements, thrombus, or clot in the system, and problems with device attachment. This outside study noted a limitation of low compliance with returning the device to its corresponding company for investigation. Due to the lack of information on mechanical complications of RVADs, we hope that discussing this case will prompt greater discussion and promote data gathering and greater adherence to device investigation for malfunction as consequences can be dire.

Figure 1. Chest X-Ray with Cardiomegaly.

Figure 2. Echo with severe diffuse hypokinesis.

Figure 3. EKG with STEMI V2-V3.

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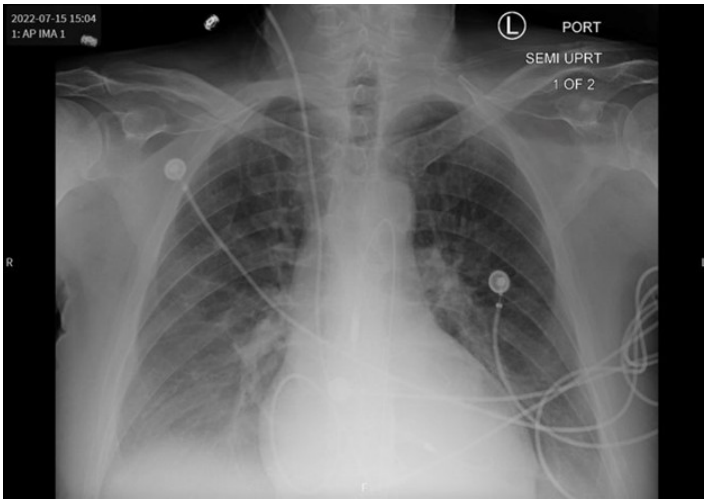

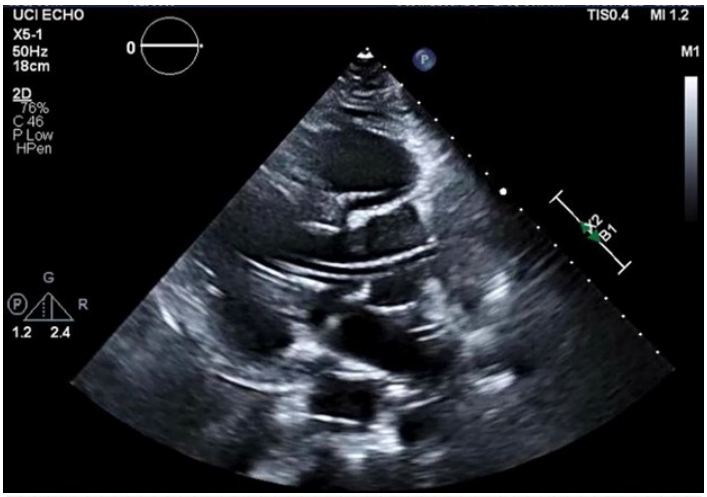
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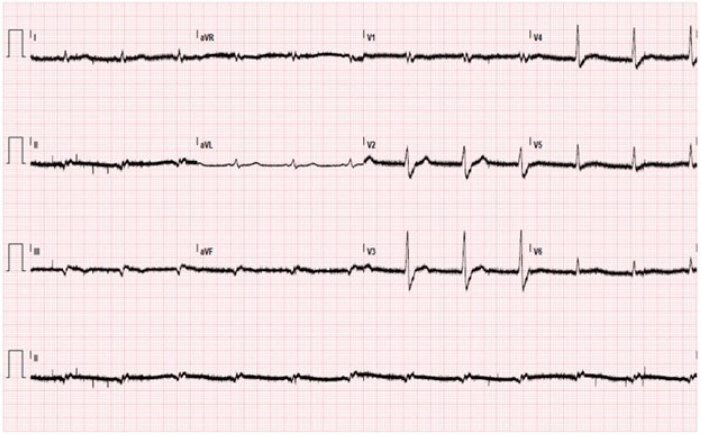

Critical Care 65 - Peroral endoscopic myotomy (POEM) procedure complicated by severe subcutaneous emphysema and pneumothorax

Yerin Koh[1], Avneep Aggarwal[2]

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Introduction: EGJOO is a rare disease diagnosed by an elevated relaxation pressure of the lower esophageal sphincter (LES) with preserved esophageal peristalsis on manometry. Patients commonly present with dysphagia, regurgitation and heartburn. Although many improve without further interventions, some may require oral calcium channel blockers and botulinum injections. If refractory, POEM is the next step in management.[1,2] Endoscopic procedures present with unique complications due to limited access and visualization of the surgical site. The most common complication during POEM is subcutaneous emphysema from CO2 insufflation but esophageal perforation, pneumothorax and pneumomediastinum can also occur as well. We present a case of pneumothorax following a POEM procedure.

Methods: 51-year-old female with history of heterozygous Factor V Leiden, provoked DVT (In the setting of traveling), morbid obesity post mini-gastric bypass surgery, hypothyroidism and esophagogastric junction outflow obstruction (EGJOO) with persistent dysphagia refractory to EGD with dilations and botulinum toxin injections was admitted for peroral endoscopic myotomy (POEM).

Preoperative physical exam was remarkable for a mallampati score of 1 and full neck range of motion. The patient was intubated with video laryngoscopy and a 7.0 ETT without difficulty. The surgery was uncomplicated. Postoperatively, the patient was noted to have significant subcutaneous emphysema but was successfully extubated in the OR and taken to the post-anesthesia recovery unit (PACU). 15 minutes in the PACU, the patient became tachypneic and began having increased work of breathing requiring re-intubation. She was admitted to the surgical ICU. Chest X-ray was remarkable for a significant right pneumomediastinum and extensive subcutaneous emphysema in the lower neck and bilateral chest walls. A right pigtail catheter was placed for the right pneumothorax. The following day, the patient was weaned off ventilatory support and extubated, requiring only 2 L/min nasal cannula. The following day, the patient was transferred to the regular nursing floor and discharged without further complications.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: POEM is an endoscopic procedure that starts by making an incision in the esophageal mucosa. Methylene blue is used to identify the layers of the esophagus by staining the submucosal but not the muscular layers. A submucosal tunnel is created by insufflating CO2 between the mucosal and muscular layers towards the stomach. During POEM, the inner circular (muscular) layer is resected at the level of the stomach and removed to relieve the symptoms from EGJOO. Without the circular layer, the longitudinal layer is vulnerable to injury. If this layer is accidentally injured, air can enter the chest cavity and cause a pneumothorax as seen in our patient.[3,4] Pneumothorax can be diagnosed clinically by auscultating the lungs and confirmed with chest x-ray. Intraoperatively, high airway pressures may be indicative of the presence of a pneumothorax. Our patient did not present with high airway peak pressures intraoperatively, was stable and successfully extubated at the end of the surgery. Due to low suspicion for pneumothorax, the decision to obtain a chest X ray intraoperatively was deferred. Small pneumothoraxes may resolve without interventions, but large pneumothoraxes can cause pulmonary and cardiovascular instability that warrants chest tube placement to evacuate intrapleural air.

Another common complication of POEM is subcutaneous emphysema due to rapid absorption of CO2 that is used to dissect the esophageal layers. Hypercarbia can be treated with hyperventilation, but once subcutaneous emphysema is present, it may be difficult to achieve normocarbia which can lead to respiratory acidosis, prolonged mechanical ventilatory support and emergence.[3,4] With an increase in the number of peroral endoscopic myotomy (POEM) procedures performed annually, it is imperative for the anesthesiologists to recognize and manage associated perioperative complications.

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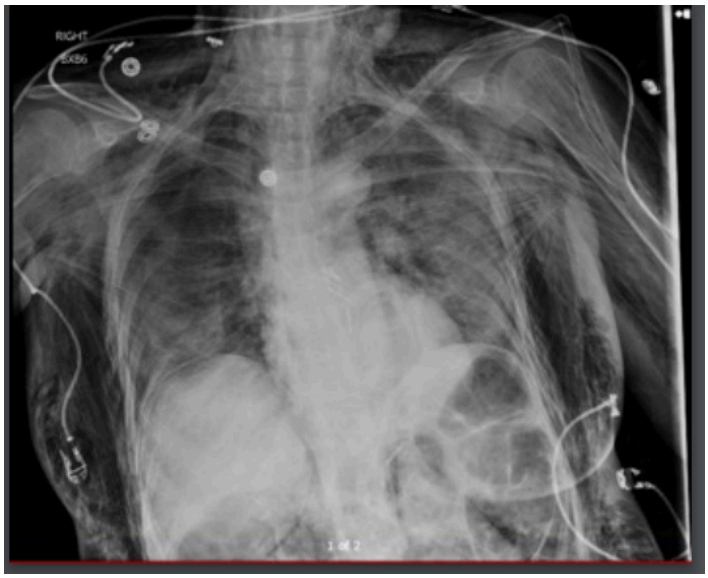

Critical Care 66 - Possible Severe Ovarian Hyperstimulation Syndrome Presenting as NSTEMI and VT Storm Requiring VA ECMO

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Introduction: To describe a rare presentation of possible severe ovarian hyperstimulation syndrome (OHSS) in a patient without history of cardiac disease who ultimately required veno-arterial extracorporeal membrane oxygenation (VA ECMO) and impella placement. Ovarian hyperstimulation syndrome is thought to arise from supraphysiological levels of luteinizing hormone (LH) and estrogen which can cause vascular remodeling, hypovolemia and electrolyte abnormalities. The hormone changes seen during IVF, namely peaks in estradiol, have been shown to cause QT prolongation, making these women more susceptible to cardiac arrhythmias.1, 2 Additionally, fatal arrhythmias in the absence of structural heart disease are more likely in women than men at baseline.2 Once OHSS arises, management is largely supportive, namely correcting and preventing any further hypotension or electrolyte abnormalities and their sequelae. OHSS is considered self limiting and the expected time course of OHSS is one to two weeks; longer if fertilization occurred.3

Methods: We present a 33-year-old female with a history of generalized anxiety disorder (GAD) and infertility secondary to a complicated appendiceal rupture status post repair now undergoing in vitro fertilization (IVF). She presented with chest discomfort, nausea, malaise and was found to have an inferior NSTEMI; no lesions were found on the emergent left heart cath. Initial bedside transthoracic echocardiogram (TTE) showed an EF of 40% and biventricular hypokinesis. She became progressively hypotensive and tachycardic and was found to have depressed biventricular function on TTE with a left ventricular ejection fraction (EF) of 40%. The next day the patient experienced increasing ectopy and bigeminy before deteriorating into ventricular tachycardia (VT) and VT storm despite three unsuccessful cardioversions. This led to cardiac arrest with return of spontaneous circulation (ROSC) after two rounds of advanced cardiac life support (ACLS). The patient was in three pressor shock and the decision was made to emergently cannulate for peripheral veno-arterial extracorporeal membrane oxygenation (VA ECMO). A bedside point of care ultrasound (POCUS) revealed severely depressed left ventricular function and a dilated right ventricle (RV). She was taken to the operating room (OR) for conversion of peripheral to central VA ECMO and for placement of a right axillary impella. The next day, the patient's hemodynamic support was weaned to just Epinephrine and she was continued on Amiodarone. Chest closure occurred two days after ECMO cannulation and the next day, the Epinephrine was weaned off. Intraoperative transesophageal echocardiogram (TEE) showed an EF of 25% and moderately depressed RV function. The patient completed a six day run of ECMO as her EF continued to improve, intermittently needing Epinephrine to augment her cardiac function. Her axillary impella was removed six days after decannulation and the remainder of her hospital course was uneventful. Cardiac MRI prior to discharge showed a recovered EF and normal global LV function, diffusely thickened pericardium and overall resolving myocarditis. She was hospitalized for a total of twenty days, and she has since been discharged from the cardiac surgery clinic.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: In an otherwise healthy female who is undergoing ovarian stimulation for in vitro fertilization who presents with hypotension and an unstable cardiac arrhythmia, ovarian hyperstimulation syndrome should be included on the differential. The QTc prolonging effects of estrogen can make this population particularly susceptible to life threatening arrhythmias.

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Critical Care 67 - Post-Operative Complications and Critical Care Management of an Aortic Valve Replacement Patient

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Introduction: Aortic valve stenosis is a common condition among the elderly population. It typically starts off mild and gradually becomes more severe, causing symptoms such as fainting, chest pain, shortness of breath, and heart failure. Severe aortic stenosis can be fatal, with a 50% mortality rate within two years of diagnosis. Due to its high mortality rate and prevalence, aortic valve replacement surgery is one of the most common valvular replacement surgeries performed.

Aortic valve replacement surgery is commonly performed at many medical centers due to the high demand for the procedure. Most patients tolerate the procedure and are able to be discharged home a few days after surgery. However, even though the surgery is considered routine, complications can still occur. In this case report, we present the case of a young and healthy patient who underwent aortic valve replacement but developed multiple post-operative complications requiring intensive critical care management.

Methods: A 60F with history significant for a bicuspid aortic valve, hypertension, and severe aortic stenosis presented for elective aortic valve replacement. She underwent minimally invasive aortic valve replacement with a 19 mm On-X valve under cardiopulmonary bypass and was admitted to the ICU intubated and sedated upon completion.

On arrival, the patient was relatively hemodynamically stable, but shortly after went into cardiac arrest due to ventricular fibrillation. Cardiac life support was initiated with chest compressions, and she eventually regained ROSC following the administration of multiple medications and defibrillation. There were no underlying causes for the arrest such as hypotension or hypoxia. The cardiac surgeon was notified, and the decision was made to return the patient to the operating room. On arrival into the OR, the patient suffered VFib arrest again, so she was immediately placed on cardiopulmonary bypass. Upon re-exploration, it was found that the patient had an improperly seated artificial aortic valve that was blocking the right coronary artery along with right ventricular hypokinesis. She underwent aortic valve repair and a CABG x1 with a saphenous vein graft to the right coronary artery. Following closure, the patient was brought back to the ICU.

She remained hemodynamically stable upon returning to the ICU from the re-exploration, then was successfully extubated the following night. Her course was uncomplicated for the next two days until signs of multiorgan decompensation arose. Her urine output decreased, her serum creatinine elevated, she developed shock liver with elevated liver enzymes, and developed altered mental status with encephalopathy. The decision to reintubate her was made, and a Swan-Ganz catheter was placed showing a cardiac index of 1.8. Trans-esophageal echocardiography was performed, and a massive perivalvular leak around the artificial aortic valve was appreciated along with right-ventricular hypokinesis. Her cardiogenic shock was managed with epinephrine and milrinone, and she required continuous atrial pacing to maintain her heart rate. She slowly improved and her cardiac index was able to be maintained above 2.1. Weaning her from the ventilator was particularly challenging, as she required a long course of diuresis to reduce her pulmonary edema. However, with careful and persistent management, she was eventually able to be extubated and her oxygen requirements were gradually weaned to low-flow nasal cannula.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Despite them being a very common procedure, aortic valve replacements can have serious and potentially life-threatening complications. These include perivalvular leaks and malpositioned valves, which can lead to hemodynamic instability and organ damage. In the critical care unit, healthcare providers must be vigilant for these complications and be prepared to manage them promptly and effectively to prevent further harm to the patient. This may require advanced hemodynamic monitoring, surgical interventions, and close collaboration with other specialists to ensure the best possible outcomes for the patient.

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Critical Care 68 - Postoperative thrombosis of a bioprosthetic mitral valve following massive transfusion and venoarterial ECMO in an adult patient with congenital heart disease salvaged by CentriMag LVAD as a bridge to decision

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Introduction: Thrombosis of a prosthetic heart valve is a serious complication following valve replacement and is associated with major morbidity and mortality. Bioprosthetic valves have a significantly lower risk of thrombosis than mechanical valves (1), however they can still serve as a nidus for thrombus formation particularly in the setting of low flow and coagulopathy (2). Here we present a case of complete thrombosis of a bioprosthetic valve following mitral valve replacement in a patient who required both massive transfusion protocol and venoarterial ECMO postoperatively. The situation was temporized with a non-standard cannulation for CentriMag LVAD support to allow time for the patient and her family to make decisions regarding goals of care and future intervention.

Methods: The patient is a 37-year-old woman with a history of d-TGA/VSD/PS with a prior classic right BT shunt and Rastelli procedure with RV-PA conduit. She presented with worsening fatigue secondary to RV-PA conduit stenosis as well as mitral regurgitation and aortic insufficiency. Her anatomy was complicated by a single coronary artery with an LAD that coursed underneath the conduit graft. Her past medical history included noncompliance with medication. For this reason, she was scheduled for RV-PA conduit replacement as well as attempted mitral valve repair. Intraoperative findings included severe mitral regurgitation and severe aortic insufficiency requiring replacement of both valves with bioprosthetics in addition to replacement of the RV-PA conduit. Separation from bypass was complicated by compression of the LAD requiring reheparinization to repair, as well as significant bleeding requiring massive transfusion. The patient was unable to wean from bypass a second time. She was placed on central venoarterial ECMO and transported to the CTICU where she continued to require massive transfusion.

Given her ongoing coagulopathy, anticoagulation was held upon arrival to the ICU. She remained in biventricular failure and the ECMO circuit was left on full flows. A heparin infusion was eventually started on post-operative day 3 and was changed to a bivalirudin on day 4.

On post-operative day 7 the patient was brought to the operating room for ECMO revision, but TEE demonstrated no flow across the mitral valve due to thrombosis of the bioprosthetic. Given her ongoing poor left ventricular function, she underwent conversion to CentriMag BiVAD on day 8 as a bridge to future decision. The RVAD (with oxygenator) was configured with the inflow cannula in the right atrium and the outflow cannula in the RV-PA graft. To bypass the dysfunctional mitral valve, the LVAD inflow cannula was placed in the left atrium via the right upper pulmonary vein with the outflow cannula in the aorta.

The patient's right side heart function improved, and the oxygenator was removed on day 15 with subsequent removal of the RVAD on day 20. TEE and TTE continued to show no motion of the bioprosthetic mitral leaflets. Her course was further complicated by embolic stroke and encephalopathy as well as unrecovered acute kidney injury. The patient was maintained on full LVAD support pending further discussion with her family about goals of care and possible further intervention.

Results: Not applicable - Medically Challenging Case Report

Conclusions: This case posed multiple challenges to the critical care team, the largest of which was managing the patient's post-operative resuscitation and coagulopathy in the setting of recently implanted bioprosthetic valves and venoarterial ECMO. All three factors of Virchow's triad were likely present in the local environment surrounding the newly implanted bioprosthetic mitral valve: 1. A low flow state secondary to severe LV dysfunction and venoarterial ECMO, 2. Coagulopathy secondary to large volume blood loss and ensuing massive transfusion, and 3. Simulated endothelial dysfunction with the artificial surface of the bioprosthetic valve serving as a site for fibrinogen deposition and eventually thrombus formation (1). The thrombosed mitral valve was eventually bypassed by a CentriMag LVAD using a non-standard cannulation with the inflow cannula introduced into the left atrium via the right upper pulmonary vein. Furthermore, the patient's history of medical noncompliance, persistent renal failure, and embolic stroke with encephalopathy presented multiple ethical dilemmas, and precluded a listing for heart transplant or durable LVAD despite her hemodynamic stability with CentriMag LVAD support.

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Critical Care 69 - Profound hypoxemia following LVAD implantation

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Introduction: Congestive heart failure is a major cause of morbidity and mortality, affecting over 6 million adults with an estimated cost of over $30 billion annually.[1] In patients with disease refractory to guideline-directed medical therapy, the left ventricular assist device (LVAD) is an advanced therapy option. LVAD implantation is associated with improved patient outcomes and is used as a bridge to transplantation or as destination therapy. Here, we present a case of hypoxemia due to right ventricular (RV) failure and shunt across a patent foramen ovale (PFO) after LVAD implantation.

Methods: A 72-year-old man with hypertension, diabetes, nonischemic cardiomyopathy with a left ventricular (LV) ejection fraction of 15%, coronary artery disease, severe mitral regurgitation status post MitraClip, and prior COVID pneumonia presented to the emergency department with three days of chest pain, dyspnea, and worsening bilateral lower extremity edema. In the last year, he had required multiple admissions for heart failure exacerbation, and he underwent a MitraClip implantation six months prior.
On this admission, he presented with signs and symptoms of acute decompensated heart failure that progressed to cardiogenic shock. He was admitted to the coronary care unit for further management with diuresis and inotropic support with expedited LVAD workup. On hospital day 7, he was taken to the OR for implantation of a HeartMate 3. Intraoperative transesophageal echocardiography (TEE) prior to initiation of cardiopulmonary bypass (CPB) confirmed severely depressed LV function. When the interatrial septum was interrogated, no ASD was demonstrated, and a bubble study with Valsalva maneuvers was negative for an intracardiac shunt. As a result, a dual-stage venous cannulation strategy was chosen rather than a bicaval approach. Implantation of the HeartMate 3 was uneventful; however, a PFO was newly diagnosed during the post-bypass echocardiographic assessment. It demonstrated left-to-right flow that subsequently resolved, and P_aO_2 was above 300mmHg on 100% F_iO_2. PFO closure would have required reinitiation of CPB and re-cannulation with a bicaval approach; it was considered but not pursued given lack of hypoxemia and resolution of visualized shunting. The patient was brought to the intensive care unit intubated on vasopressor and inotropic support as well as inhaled epoprostenol.
Overnight on postoperative day 0, he developed profound hypoxemia despite provision of 100% F_iO_2, recruitment maneuvers, and a normal-appearing chest X-ray. Bedside TEE revealed new continuous right-to-left shunt across the PFO along with leftward interatrial septal bowing, and severe RV hypokinesis. The decision was made to perform percutaneous closure of the PFO, and despite worsening of RV function after PFO closure, there was immediate improvement in oxygenation. The remainder of the hospital course was notable for persistent atrial fibrillation requiring cardioversion and RV failure requiring slow inotropic wean; the patient was discharged to an extended care facility on hospital day 41.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The presence of a PFO in LVAD patients can lead to hypoxemia and paradoxical embolization. Bubble contrast TEE is the gold standard test for diagnosing a PFO; however, in the setting of elevated left atrial pressure prior to LVAD implantation, interatrial shunting may not be visualized even with provocative maneuvers. Unloading of the LV by LVAD leads to decreased left atrial pressure while increased venous return to a poorly functioning RV can increase right atrial pressure. This combination of effects can markedly increase right-to-left intracardiac shunting through a PFO.[2]

In the setting of delayed symptomatology of an intracardiac shunt, temporizing medical management should be aimed at equalizing atrial pressures. This can be achieved by maximizing RV inotropic support and minimizing afterload using pulmonary vasodilators and careful titration of mechanical ventilation settings. Treatment with percutaneous PFO closure in the early postoperative days is a safe option for definitive management.[2]

Profound hypoxemia in a patient with a newly implanted LVAD and prior transseptal access or known PFO should carry a high index of suspicion for right-to-left intracardiac shunting.

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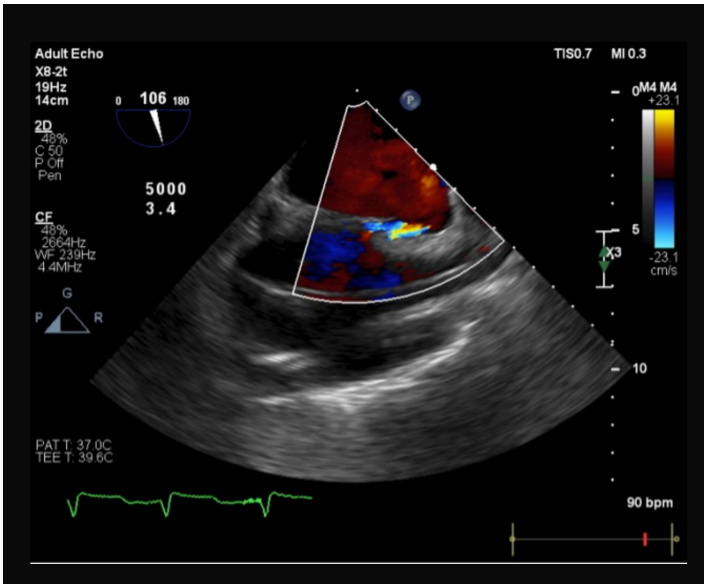

Critical Care 70 - Prolonged Neuromuscular Blockade Following Orthotopic Liver Transplant

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Beth Israel Deaconess Medical Center[1]

Introduction: Patients with liver disease have an increased risk for intraoperative and post-operative morbidity and mortality due to abnormalities in both hepatic synthetic function and drug metabolism. The time to onset and duration of action of some commonly used neuromuscular blocking agents can be impacted by hepatic disease. While cisatracurium spontaneously degrades by Hoffman elimination, an organ-independent process, vecuronium and rocuronium are primarily metabolized by the liver; drug elimination and the duration of neuromuscular block are extended in patients with advanced liver disease. Decreased rocuronium clearance has further been associated with extended warm ischemic times. The choice of preferred intraoperative neuromuscular blocking agent is institution dependent, however, anecdotally in all types of operative cases there recently has been an increased preference for the use of liberal dosing of rocuronium following the introduction of sugammadex, a potent reversal agent.

Methods: We report extended neuromuscular blockade beyond anticipated times in two patients administered rocuronium for intraoperative neuromuscular blockade requiring sugammadex administration for extubation in the ICU.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Liver transplant patients receiving a graft without an extended warm ischemic time or concurrent significant renal dysfunction, should still be carefully evaluated for prolonged neuromuscular blockade if they received intraoperative rocuronium.

Critical Care 71 - Pulmonary complications from Neostigmine use for management of ileus in Lung Transplant recipient

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Introduction: Neostigmine is a reversible acetylcholinesterase inhibitor that functions as a parasympathomimetic drug. It is indicated in the off-label treatment of acute colonic pseudo-obstruction and critical illness associated ileus. Neostigmine is associated with side effects such as bradycardia, asystole, bronchospasm and increased pulmonary secretions[1,2]. There are no studies or case reports describing the use of neostigmine in lung transplant patients. Presented is the case of a 68-year-old comorbid male who underwent bilateral lung transplantation with a post operative course complicated by ileus that was treated with a series of IV neostigmine boluses resulting in increased pulmonary secretions and oxygen requirements.

Methods: Case presented is a 68-year-old male with a past medical history of hypertension (HTN), asthma, obstructive sleep apnea (OSA), history of prostate cancer (s/p prostatectomy), atrial fibrillation, and progressive interstitial pneumonia with autoimmune features who presented for bilateral lung transplantation.

After an uneventful intraoperative course, he was extubated on postoperative day (POD) #1 to continuous positive airway pressure given his OSA. This was weaned to low flow nasal canula within few hours. Patient was kept nil per oral (NPO) and a postoperative bowel regiment was initiated through his post pyloric small bore feeding access. POD #2 was eventful due to the recurrence of his known Atrial Fibrillation with rapid response rate (RVR). Despite beta-blockers and amiodarone boluses, patient underwent cardioversion with subsequent amiodarone infusion.

On POD #3, patient had increased abdominal distension and pain with preliminary diagnosis of ileus. Patient had placement of nasogastric (NG) tube for gastric & bowel decompression. Patient's abdominal discomfort extended into POD #4. A computerized tomography (CT) of abdomen and pelvis without contrast displayed significant air with stool burden and without evidence of any mechanical obstruction. Due to ongoing clinical concern for ileus and failure to respond to conservative treatments with existent bowel regimen, decision was made to treat patient with Neostigmine.

Patient was given a cumulative dose of 7.5 mg of neostigmine in 2.5 mg increments over 30 minutes. Patient had copious amounts of flatus and liquid bowel movements that started during the 30 minute period of neostigmine administration resulting in improvement of the abdominal distension. Several hours after treatment with neostigmine, patient started having copious pulmonary secretions and associated dyspnea in effort to clear his airways. Patient was switched from NC to HFNC to improve dyspnea. Aggressive pulmonary toilet was provided by Respiratory Therapists. Secretions and dyspnea slowly improved with adequate pulmonary toilet and HFNC approximately 12 hours after neostigmine administration.

Conclusions: Neostigmine is an effective method to treat ileus in the ICU but can be accompanied by several side effects. In the presented case, the patient did have a successful decompression of his bowels from Neostigmine administration, but it was accompanied by significant pulmonary side effects. Several different dosing strategies have been shown in the literature in the ICU of neostigmine administration for the treatment of ileus vary from 0.4-0.8 mg/hr for 8 hours[3] to a bolus of 2.5 mg over an hour[4] to 2 mg over 3-5 minutes[5]. The safety profile and efficacy of bolus and infusion dosing of neostigmine are similar[6].

In the presented case, patient's copious airway secretions as well as hypoxemia was not an immediate side effect and worsened with peak effect at 4-6 hours after Neostigmine administration. Differential diagnosis included evolving primary graft dysfunction, ileus related regurgitation, hospital acquired pneumonia, and iatrogenic side effects of neostigmine.

In summary the presented case, the post-surgical lung transplant patient did have successful decompression of his bowels with neostigmine treatment but at the cost of having increased secretions that caused an increased oxygen requirement and work of breathing. In future cases, glycopyrrolate could be utilized as a pretreatment to help mitigate neostigmine's cardiovascular and pulmonary side effects. Careful selection of patient population should be utilized in the treatment of ileus with neostigmine to ensure that patients can tolerate the side effect profile associated with neostigmine.

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Critical Care 72 - Pupillary Responses to High-Dose Inhaled Sevoflurane as a Rescue Therapy for Severe Asthma Exacerbation: A Case Report

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Introduction: Volatile anesthetics have been proposed as rescue therapies for severe refractory acute asthma exacerbations, given their known bronchodilator properties. This practice has not been studied systematically and currently is not part of clinical practice guidelines. Absence of systematic data has limited understanding of immediate effects and side-effects associated to these agents repurposed for this indication.

We present a case of a severe acute asthma exacerbation, refractory to maximal medical treatment, with high invasive ventilatory support needs that received inhaled Sevoflurane as a rescue therapy. We highlight significant pupillary changes that accompany high dose Sevoflurane therapy which can potentially mimic other catastrophic complications in critically ill patients.

Methods: A 52 male with a history of NSAID allergy presented in acute hypercarbic respiratory failure after accidental exposure to over-counter medication containing aspirin. Despite maximal medical therapy (inhaled beta-2 agonists, anticholinergics, corticosteroids and intravenous magnesium sulfate) invasive mechanical ventilatory support was needed. Because of progressive hypercarbia with severe respiratory acidosis a trial of inhaled sevoflurane as a rescue therapy was initiated.

The patient was transitioned from the ICU ventilator to a Dräger Fabius® (Draeger Inc., Germany) anesthesia machine, employed to ventilate and deliver inhaled anesthetic simultaneously. Volume control mode was set, altering I:E ratios to facilitate prolonged expiratory times. Sevoflurane was dosed by adjusting the variable bypass vaporizer to an achieve end-tidal concentration of 2 MAC (approximately 4% by volume).

After approximately 6 hours of therapy, patient pupils were noted to be mydriatic and fixed, without any reactivity to light. Although this was an acute change, imaging studies of the CNS were deferred given the tenuous clinical condition and likelihood of clinical presentation being secondary to a pharmacologic intervention. An automatic pupilometer (NPi-300 Pupillometer® Neuroptics, Irvine, CA, USA) was used to perform serial pupillary assessments and reactivity, as determined by the proprietary algorithm.

High dose sevoflurane was continued for the next 36 hours which led to improvement in pulmonary mechanics and normalization of acid base status. Pupillary exam remained unchanged throughout. Concomitant with improvement in pulmonary mechanics, volatile anesthetic was weaned off, while carefully monitoring respiratory parameters. Follow-up assessment of pupillary reflexes demonstrated return to baseline diameter (2-3mm), with recovery of conjugate function and reactivity to light. The remainder of therapies were weaned off and finally the patient was extubated. No neurological sequelae were identified.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Pupillary size is determined by the opposing interplay between the sympathetic and parasympathetic nervous system.[1] To date, the exact mechanism through which mydriasis occurs in patients receiving inhaled anesthesia is unknown. Nonetheless, bilateral mydriasis in critically ill patients sedated with volatile anesthetics has been reported, especially when using increased concentrations.[2-4] A proposed mechanism of mydriasis is via transient systemic sympathetic stimulation.[5] However, the use of both topical alpha- (dapiprazole) and beta-blockers (esmolol and timolol) during general anesthesia doesn't prevent dilation in response to desflurane, contradicting this hypothesis.[4,6] Instead, it is believed that inhibition of the Edinger-Westphal (pupilloconstrictor) nuclei in the midbrain is what facilitates mydriasis.[4,7]

Increasing attention must be paid to the effects of volatile anesthetics on pupillary dilatation. In general, high dose halogenated agents can lead to significant pupillary changes and should be considered in the differential while other potentially devastating or reversible causes are addressed. This is particularly important given the increasing role of volatile anesthetics used for sedation in critically ill patients.[8]

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Zoom out
Right Left Diff
Size [mm] 6.68 < 6.91 0.23
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NPi 0 0
Size [mm] 7.09 < 7.30 0.21
Right Left Diff
NPi 3.9 3.9
Size [mm] 2.21 < 2.31 0.10
Right Left Diff
NPi 4.2 < 4.3 0.1
Size [mm] 2.24 > 2.22 0.02
End tidal Sevoflurane concentration
18 hours
36 hours
42 hours
62 hours

Critical Care 73 - Reversal of severe vasoplegia following single stress-dose of steroid in a patient on longstanding antipsychotic medication therapy

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Introduction: Hemodynamic instability requiring vasoactive medications (pressors) is a common occurrence in the surgical intensive care unit (SICU). The differential for hypotension requiring pressors is broad, though sepsis remains one of the most frequent etiologies. In patients who present with bowel obstruction, the abdomen is often implicated as a source of sepsis and shock that might explain postoperative hypotension[1]. Adrenal insufficiency remains a less frequently encountered etiology for hypotension, but should always be considered, particularly in select patient populations. Current evidence suggests that long-term antipsychotic use can result in adrenal insufficiency[2,3]. We describe a case of severe hemodynamic instability and refractory vasoplegia in a patient with significant psychiatric history who received a single dose of hydrocortisone with immediate and profound reversal of hypotension.

Methods: A 71-year-old man with history of schizoaffective disorder was admitted for management of agitation and psychosis. His outpatient medication regimen was notable multiple psychotropic medications: clozapine, benztropine, divalproex, paroxetine, and olanzapine.

Three days into his psychiatric admission he was transferred to the surgical service for fever, hypotension, and concern for sepsis. Workup revealed colonic dilation (~10.5cm) and large stool burden. He underwent a subtotal colectomy, disimpaction, and delayed abdominal wall closure. Upon admission to SICU, he was acidotic and on pressors, but his hemodynamics improved with antibiotics and pressors were quickly weaned off. On postoperative day (POD) 2 he returned to the operating room for ileosigmoid anastomosis and abdominal closure. He was extubated, and psychiatric medications were resumed stepwise over three days (valproate, then olanzapine, then clozapine.) A dexmedetomidine infusion was used for agitation. On POD 5/7, the patient became hypotensive and developed hypoxic respiratory failure. CT contrast imaging did not demonstrate pulmonary embolism nor overt infectious source, but due to presumed septic shock he was intubated, given broad spectrum antibiotics, and underwent surgical re-exploration. The surgeons noted healthy appearing bowel and intact anastomoses.

Postoperatively, he continued to have worsening hypotension and escalating pressors to very high doses (norepinephrine 0.4mcg/kg/min, vasopressin 0.06units/kg/hr), refractory to 2 pRBC transfusions. Bedside ultrasound was limited and unable to visualize myocardium, but velocity-time-integral (VTI) of the carotid artery and arterial line waveform analyses suggested severe vasoplegia and lack of fluid-responsiveness by high estimated cardiac index and low stroke volume variation. Bronchoscopy was performed and cultures repeated. The instability seemed out of proportion to the laparotomy findings, so alternative diagnoses to sepsis were considered, including adrenal insufficiency due to long-term antipsychotic usage. A stress-dose of hydrocortisone 100mg IV was administered and, dramatically, the hypotension resolved. Both pressors were off within 2 hours.

Subsequently, the patient was treated with cefepime and caspofungin for Citrobacter pneumonia and fungemia. He received 3 days of intravenous steroids. He was ultimately discharged back to a skilled nursing facility.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Clinical studies suggest that antipsychotics have an association with adrenal insufficiency, though the relationship is poorly understood. Several molecular mechanisms by which antipsychotic drugs may influence steroid stress hormones have been proposed[4]. Newer data also suggests steroids may reduce pressor requirements in septic shock[5]. The above case demonstrates a profound reversal of shock in a patient without known history of adrenal insufficiency, but on longstanding and high dose antipsychotics, including clozapine. As borne out by the blood and respiratory cultures, this patient was undoubtedly suffering from sepsis. However, the immediate and dramatic reversal of his hypotension and pressor requirement after a single stress-dose of steroids indicates a degree of adrenal insufficiency as well.
This case is a reminder to always entertain a broad differential in the face of acutely worsening hemodynamic instability in the ICU. In patients on longstanding, high-dose antipsychotic therapy, adrenal insufficiency must be considered in the differential for severe life-threatening hypotension and shock.

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Outpatient Medication List

Clozapine 200mg PO BID
Benztropine 1mg PO BID
Divalproex sodium 500mg PO BID
Olanzapine 15mg PO QHS
Paroxetine 40mg PO daily

Docusate sodium 250mg PO daily
Aspirin 81mg PO daily
Atorvastatin 20mg PO QHS
Ferrous sulfate 325mg PO BID
Multivitamin

Acetaminophen 325mg Q6H PO PRN
Lorazepam 2mg IM BID PRN
Diphenhydramine 50mg IM BID PRN

Critical Care 74 - RV failure after MVR requiring IABP, ECMO, and RVAD in a younger adult female

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Introduction: Although many are familiar with the common adage, "the most common cause of right heart failure is left heart failure," this is not true in all cases, especially in post-operative cardiac surgery patients. Greatly feared (and with good reason), right heart failure can devastate not only the output of the heart but also the working of multiple other organs (such as the lungs, liver, and kidneys). With the inability to compensate acutely to strain as much as the left ventricle, the right ventricle fails much more quickly and can do much more damage to the body. In these cases, inotropes may not be enough--mechanical support such as intra-aortic balloon pump (IABP), veno-arterial extracorporeal membrane oxygenation (VA-ECMO), and right ventricular assist device (RVAD) may be warranted. We present a case of acute right ventricular failure after elective mitral valve repair requiring each of these support devices and resulting in a prolonged hospital stay for a younger adult female patient.

Methods: A female under 45 years of age presented initially with symptomatic severe mitral regurgitation that was first noticed during pregnancy six years prior. Her palpitations, fatigue, and shortness of breath gradually progressed to the point of being extremely limiting for her career and day-to-day life, and she was scheduled for elective mitral valve repair. Right heart dysfunction was initially noticed upon chest closure, prompting chest reopening before leaving the OR and intra-aortic balloon pump (IABP) placement. During the first few post-op days, she was initially extubated, the IABP removed, and pressors and inotropes weaned. However, she became hypotensive within 12 hours after final inotrope removal, and again required pressors and inotropes. Her acute right heart failure quickly progressed to acute kidney failure requiring CRRT, shock liver, respiratory failure requiring re-intubation, and VA-ECMO to temporalize her cardiac function. Within a week she was decannulated from VA-ECMO and a percutaneous RVAD was placed via the RIJ. She was extubated soon after this. Her RV was slow to recover and required multiple weeks before inotropes were stopped and the RVAD was removed. The patient was eventually discharged and in the subsequent months has had gradual recovery of her RV function and overall bodily functional status.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: We present a case of severe right ventricular heart failure after MVR that required IABP, VA-ECMO, and RVAD placement--and significantly extended a younger patient's hospital course. Although her pre-operative echo showed normal right ventricular function, it is possible that the severity of her mitral regurgitation and the gradual increase in her symptoms masked worse RV function--which suddenly became an issue when her cardiovascular physiology changed so drastically post-cardiopulmonary bypass. It is also possible that the post-operative inotropes were masking the severity of her RV failure, and weaning them too quickly lead to the subsequent drastic decompensation in multiple organ systems. Regardless, a look at the literature indicates that mitral valve repair has been known to induce a change in RV mechanical pattern, not-infrequently causing RV systolic dysfunction and a decrease in RVEF post-op. Use of IABP (<2%) and ECMO (<0.5%) is rare after mitral valve repair surgery, but both should be kept in mind for acute cardiogenic shock after mitral valve repair, especially when pharmacologic support fails. However, both lead to high mortality rates when used in this setting (~20% and ~50%) and in-hospital mortality has been seen to be significantly higher after mechanical support is used in mitral valve surgery compared to other cardiac procedures--indicating the severity of heart failure in post-MV surgical patients. These patients should be closely monitored, and their care quickly escalated, especially when right ventricular dysfunction occurs.

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Critical Care 75 - Secondary Arterioenteric Fistula Formation in a Patient with Giant Cell Aortitis and Extensive Abdominal Vascular Reconstruction

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Introduction: The patient is a 66-year-old female with Giant Cell Aortitis status post Elephant Trunk repair, TEVAR, and Innominate-SMA grafting complicated by cardiac arrest, necrotizing pancreatitis, graft pseudoaneurysm, and arterioenteric fistula requiring massive transfusion.

Methods: The patient is a 66-year-old female with an extensive PMH including ulcerative colitis, chronically occluded celiac artery, giant cell aortitis, ascending aortic aneurysm (Image 1) status post aortic valve repair, ascending aorta replacement, and first stage elephant trunk in 2015 followed by second stage elephant trunk and TEVAR in 2017. She was initially scheduled for a redo thoracoabdominal aortic aneurysm repair of an enlarging suprarenal abdominal aortic aneurysm seen on follow up imaging. However, her preoperative workup revealed 3+ aortic insufficiency and 2+ tricuspid insufficiency and multivessel coronary artery disease so she underwent a redo aortic valve replacement, coronary artery bypass grafting, tricuspid valvular repair, and innominate-SMA interposition graft instead.On postop day 9 she had abdominal pain and was found to have pancreatitis. She left the CVICU on postop day 12. The next day her abdominal pain worsened. A CT of her abdomen showed hemorrhagic pancreatic fluid collections adjacent to the innominate-SMA graft anastomosis (Image 2) and she was transferred back to the CVICU. On postop day 16 she had bloody nasogastric tube output and hematemesis requiring intubation. An EGD the next day showed extrinsic compression of the stomach with pale mucosa and concern for gastric pneumatosis (Image 3). Blood cultures were positive for Enterobacter cloacae. Two days later she was transferred to the SICU where she improved and was extubated on postop day 22. A CT on postop day 27 showed a pseudoaneurysm of the innominate-SMA graft at the anastomosis with the SMA (Image 4). 3 days later she had another profuse upper GI bleed which led to cardiac arrest. She was intubated and ROSC was obtained after 20 minutes of CPR and ongoing massive transfusion. Emergent EGD showed gastric perforation and intraluminal blood. CTA showed large extravasation from Innominate graft/SMA intra abdominal anastomosis (Image 5). The patient was emergently taken to the OR for ex-lap, and stent angioplasty of innominate-SMA graft. She was readmitted to CVICU postoperatively. two days later she again had hematemesis requiring massive transfusion. Her lactate continued to rise despite resuscitation efforts and she was made DNR-Comfort Care. She died shortly thereafter.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Arterioenteric fistula (AEF) formation is a rare and potentially devastating complication that can afflict individuals with vascular pathology or who undergo surgical or endovascular correction of vascular pathology. AEF are divided into two groups: primary (PAEF) and secondary (SAEF). PAEF describes an abnormal connection between a native artery and the gut. SAEF describes the same phenomenon in a surgically repaired or grafted artery [1]. Though both are rare, SAEF is more common than PAEF with an incidence of 0.36%-1.6% in patients who undergo repair of the aorta [2]. The mechanism of SAEF appears to be direct contact between the artery or graft and the GI tract with erosion occurring due to pulsatile and peristaltic rubbing between the two structures [1]. The median age of onset is 61 years and is 3 times as likely to occur in males [3]. AEF can be difficult to diagnose expeditiously due to its rarity and variability in presentation. Initial presentations include abdominal pain, back pain, fever, sepsis, shock, drop in hemoglobin, pulsatile abdominal mass, melena, and hematemesis. The most common presentations are melena, hematemesis, or shock [1,3]. Diagnostic modalities include direct visualization via laparotomy or endoscopy as well as radiologic studies with CTA being the preferred method [1,3]. Treatments are surgical or endovascular in nature [1,3]. Our case describes a classic presentation of SAEF with the patient experiencing abdominal pain, bacteremia with GI organisms, and a herald GI bleed prior to the catastrophic bleed that ultimately led to her demise. Our case demonstrates the importance of considering SAEF in a patient who has undergone abdominal vascular surgery and experiences a GI bleed with no apparent source.

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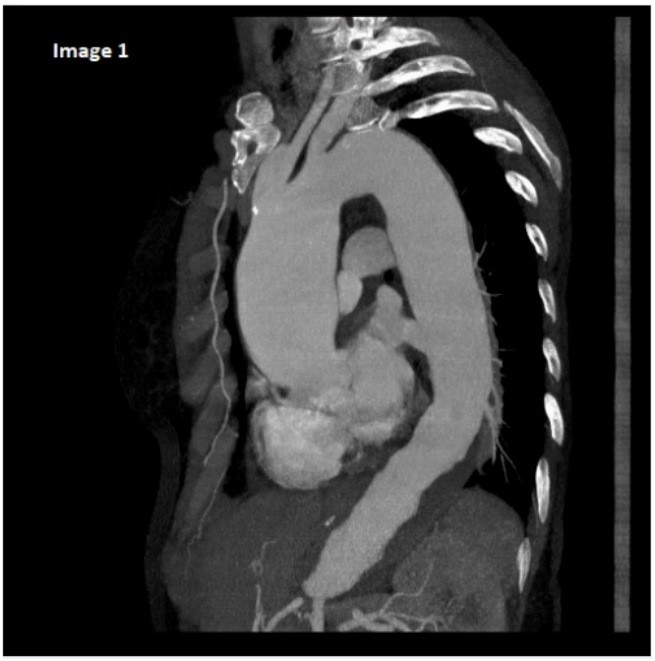


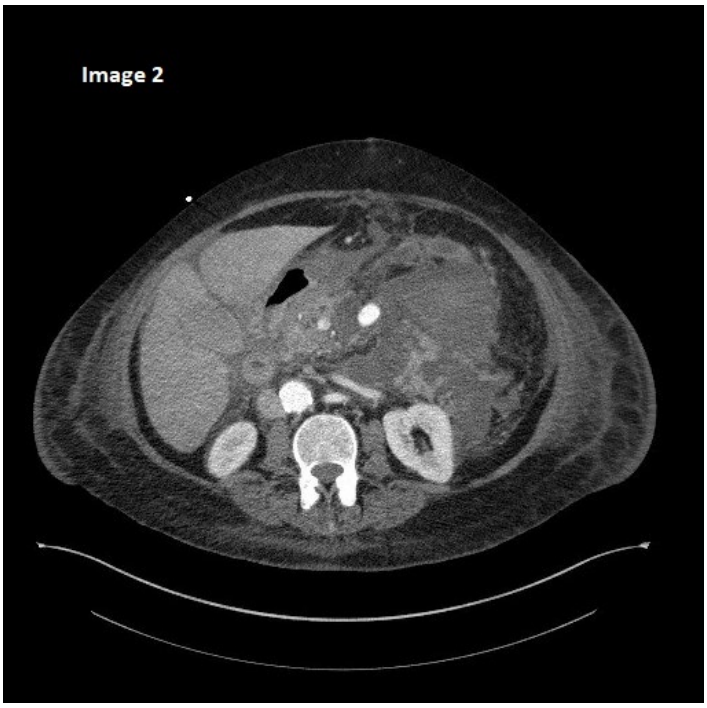
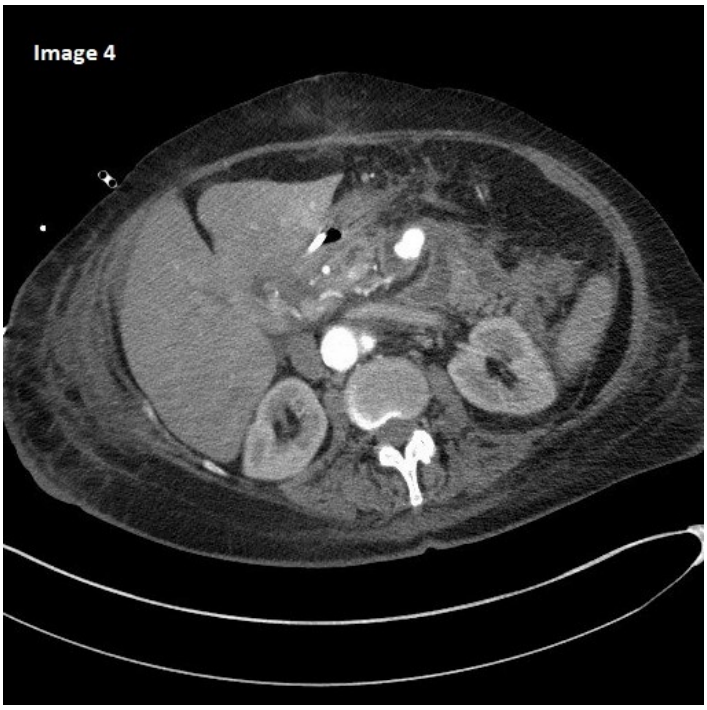


Image 2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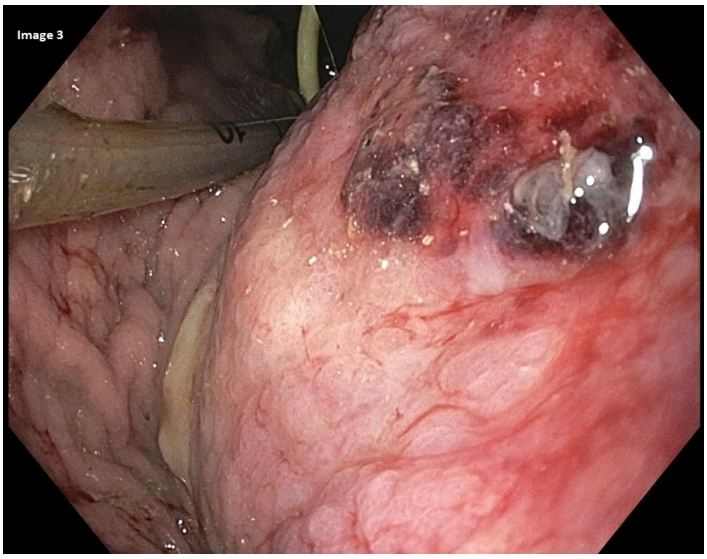
Image 3

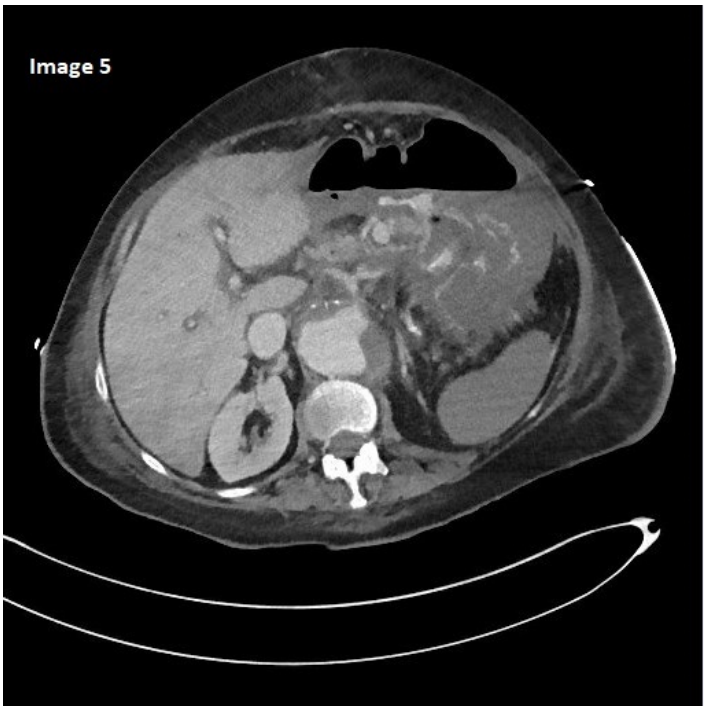
Image 5

Critical Care 76 - Severe nausea and vomiting - an unexpected adverse effect of bivalirudin for anticoagulation.

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Brigham and Women's Hospital[1]

Introduction: Heparin induced thrombocytopenia (HIT) is a common adverse effect of heparin. This paradoxical phenomena causes a prothrombotic state. In HIT, antibodies form against heparin and platelet factor 4 immune complexes. This leads to the destruction of platelets ultimately causing the formation of new blood clots. In the hospital setting, HIT is first detected by a fall in platelet count one to two weeks after starting heparin. The diagnosis is made by evaluating platelet count trend and testing for heparin PF-4 antibodies. After confirming the diagnosis of HIT, all forms of heparin must be stopped.

Patients who develop HIT can receive bivalirudin as an alternative anticoagulant. Bivalirudin is a direct thrombin inhibitor. Unlike heparin, bivalirudin does not bind to any other protein and is metabolized via proteolysis. Manufacturer's package insert listed contraindications are known hypersensitivity to bivalirudin and active bleeding. Thrombosis, hemorrhage, and anaphylaxis are listed as most common side effects and nausea is listed as a rare adverse reaction. Nevertheless, here, we present a patient who developed severe nausea and intractable projectile vomiting after bivalirudin administration.

Methods: 38-year-old male (172.7 cm, BMI 29) with a history of HTN and NIDDM presented with acute onset chest pain. He was afebrile, normotensive, in normal sinus rhythm, and not hypoxic. Labs: K 2.9, Cr 1.10, Hematocrit 41.1, platelets 332, AST 16, ALT 16, lipase 35, lactate 8.4. CTA notable for severe aortic insufficiency and acute type A dissection extending to the left distal renal artery. He was brought emergently to the OR for total arch repair and mechanical valve replacement. The patient had no immediate post-operative complications such as bleeding, hypoxia, nausea/vomiting (n/v) and was extubated on POD 1.

He was anticoagulated with a heparin infusion. His platelet count had a notable drop from 194 to 92 on POD7. A PF4 resulted positive at 2.320. Bivalirudin infusion was started at 0.15 mg/kg/hr on POD 9. All other medications remained unchanged: acetaminophen, amlodipine, aspirin, atorvastatin, captopril, esomeprazole, ipratropium, metoprolol, and valproate. Within an hour the patient developed severe nausea with intractable projectile vomiting with altered mental status and stable hemodynamics. His abdomen remined soft, nontender, and nondistended. AST 17, ALT 23, lipase 153, lactate 0.8. KUB demonstrated nonobstructive gas and no significant gastric dilation. Zofran (5HT3), Compazine (D2), Haldol (D2), and Reglan (D2) were attempted treatments without success. Bivalirudin was held and the patient was brought for a CT head to rule out a bleed. N/v transiently improved. CT was negative and infusion was restarted with recurrence of symptoms. The drug was then discontinued and anticoagulation was changed to Argatroban infusion on POD10. Patient's n/v resolved almost immediately without any further episodes in his admission.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Bivalirudin has been studied as a first-line anticoagulant. The literature describes that it is as effective as heparin at reaching target measures of anticoagulation. In addition, retrospective studies have shown bivalirudin to be safer with regards to bleeding and thromboembolic complications.
Bivalirudin is a specific and reversible direct thrombin inhibitor with immediate onset of action that binds to the catalytic site of thrombin preventing thrombin-mediated cleavage of fibrinogen to fibrin monomers, and activation of factors V, VIII, and XIII.
In our patient, severe n/v was seen immediately after the bivalirudin infusion was initiated. Several antiemetics were administered without relief. The patient's intractable projectile vomiting subsided once bivalirudin was discontinued. Extensive review of the literature has returned zero reports of a similar adverse reaction.
N/v is an intricate balance between signaling pathways of the area postrema, the nucleus tractus solitarius, and the gastrointestinal system. Neurotransmitter receptors that mediate vomiting include muscarinic (M1), dopamine (D2), histamine (H1), serotonin (5HT-3), and substance P (NK1). The pharmacology and chemical structure of bivalirudin do not point to any mechanism or reason why this drug should exhibit this n/v effect.
Given the suggestion of bivalirudin as a first line anticoagulant, further prospective randomized studies elucidating the adverse effects of bivalirudin are necessary.

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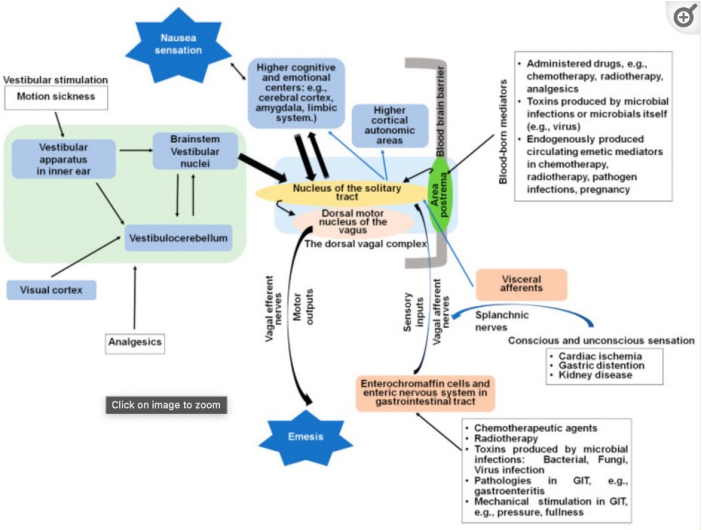

Critical Care 77 - Spinal Decerebrate-Like Posturing: An Uncommon Reflex in a Brain-Dead Patient

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University of Kentucky[1] Univ. of Kentucky College of Medicine Dept of Anesthesiology[2]

Introduction: Brain death is defined as a permanent, irreversible loss of whole brain function, including the brainstem. Typically, these intubated patients lack spontaneous or reflexive movement, resulting in a Glasgow Coma Scale (GCS) score of 3T (1-1-1T) – but not always. Reflexes often attributed to the spinal cord, such as "triple flexion" (flexion of the thigh and leg, and dorsiflexion of the foot) and “Lazarus sign” (when a patient briefly raises their arms and drops them across their chest), are well-documented and have been observed in patients that meet clinical criteria for brain death and lack cerebral blood flow on subsequent testing. As a result, while these well-defined reflexes can be unsettling, especially to an untrained observer, their appearance does not generally require confirmatory testing. A lesser-observed spinal reflex after brain death results in decerebrate-like posturing, and has been called "spinal decerebrate-like posturing". This reflex has been observed both spontaneously and after patient stimulation. It can be problematic, as true decerebrate posturing is believed to originate from the brainstem, and results in a GCS score of 4T (1-2-1T). It can be associated with reversible metabolic disturbances and disqualifies a patient from meeting clinical criteria for brain death. We present a case of man diagnosed with brain death by nuclear medicine scan who exhibited spinal decerebrate-like posturing during his brain death examination.

Methods: Our patient was a 45-year-old man with a PMH of HTN and HLD who was brought to our hospital by EMS after being found unresponsive at work. Upon arrival, vital signs were remarkable for blood pressure 260/160. He demonstrated intermittent decerebrate posturing, but was otherwise unresponsive. He was found to have a massive basal ganglia hemorrhage with uncal herniation. He was intubated for airway protection and admitted to the ICU. On DOH2, he became bradycardic and hypotensive, lost gag reflex, stopped over-breathing the ventilator, and pupils were noted to be fixed and dilated. At the family’s request, supportive care was continued for several days, at which point formal brain death testing was performed. Upon painful stimulus to the patient’s lower extremities, the triple flexion reflex was noted. He also exhibited extension-pronation movements consistent with decerebrate posturing in the upper extremities in response to nailbed pressure of his fingers. An apnea test was performed, but aborted due to desaturation and hypotension. A nuclear medicine scan was performed due to the inability to complete an apnea test and concern for retained brain stem function due to the observed extensor posturing. The nuclear flow study demonstrated a complete absence of cerebral and cerebellar perfusion, consistent with brain death. After discussing the results with the patient’s family, withdrawal of life sustaining therapy occurred.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: True spinal reflexes that occur after brain death have been hypothesized to result from disinhibition of cervical spinal cord motor generators. In some instances, observed reflexes are not adequately mechanistically explained to be of “spinal” origin, yet are attributed as such nonetheless. Further, some movements may occur only when provoked by stimulus and others may occur spontaneously. Clearly, these complex movements have the potential to lead to uncertainty and confusion to both families and trained physicians. This is particularly true of the spinal decerebrate-like posturing reflex, as it is infrequently seen and mimics a movement consistent with preservation of some brainstem function. Very few case reports have been published describing this reflex, with a recent 2016 case report and literature review by Kumar et al finding only four such examples. The scant description of and lack of familiarity with this reflex can delay the diagnosis of brain death leading to prolonged distress of family members, loss of organ donation in those wishing to donate, and lack of trust in the physician diagnosis of brain death. In our case, confirmatory ancillary testing was required due to the inability to complete an apnea test. Still, even for cases in which the exam otherwise meets clinical criteria for brain death, we propose ancillary testing should be performed, as spinal-mediated decerebrate-like posturing cannot be reliably distinguished from the brainstem-mediated, true decerebrate posturing.

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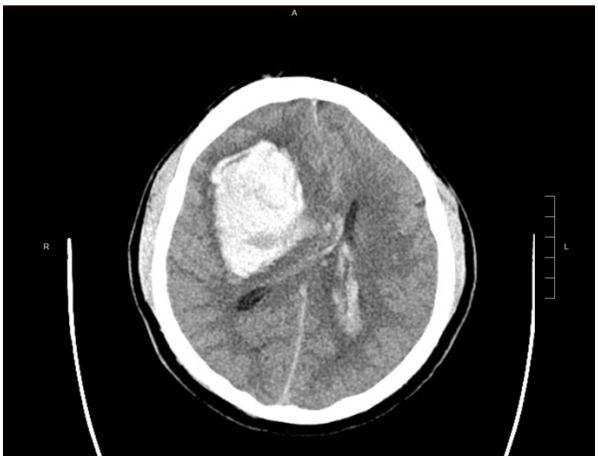

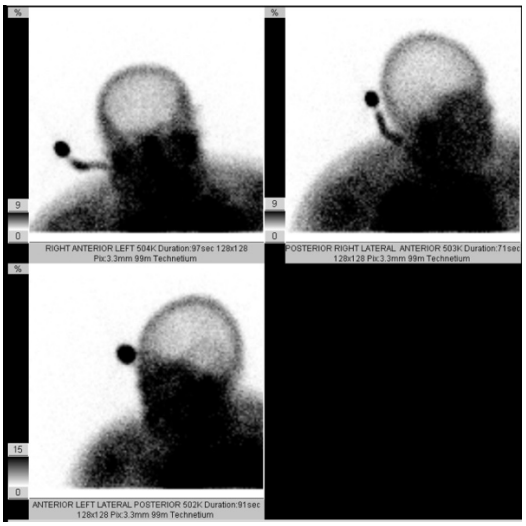
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Critical Care 78 - Stellate Ganglion Block for Cerebral Perfusion in a Patient with Moyamoya Disease - A Case Report

Christine Orser[1], Brendan Wanta[1], Luke Matzek[1]

Mayo Clinic[1]

Introduction: Stellate ganglion block (SGB) has been utilized as an effective modality for the treatment of refractory cerebral vasospasm in the setting of subarachnoid hemorrhage. Its efficacy in treating severe symptomatic vasoocclusive disease is not known.

Methods: Methods: A 67-year-old male presented with stroke symptoms including dysarthria and facial droop. On first neurologic exam, he was disoriented with mild dysarthria, right facial droop, and right pronator drift. He was hypertensive with systolic pressures above 200. A stroke code was activated and IV vasodilators were initiated for blood pressure control. His symptoms had fully resolved at the time neurology first evaluated him and thus chemical thrombolysis was not pursued. However, his symptoms recurred despite adequate control of his hemodynamics. CTA revealed bilateral MCA occlusions and he was admitted to the ICU for further management where upon arrival his NIHSS was 4. Permissive hypertension was implemented with improvement in his symptoms. Further workup revealed scattered bilateral hemispheric ischemic infarcts, severe occlusive disease with demonstration of hypoperfusion dependent collateralization consistent with Moyamoya disease.

Interestingly, the patient's symptoms were absent when supine with permissive hypertension, however they recurred with postural ischemia to the left hemisphere when elevated beyond 10 degrees or when the systolic blood pressure was lowered to 180 mmHg. After discussion with the multidisciplinary team, a left-sided stellate ganglion block was performed in an attempt to enhance cerebral perfusion. Using ultrasound guidance, 10 cc of 0.25% Bupivacaine was injected between the left carotid artery and longus colli with adequate spread visualized. The patient developed ipsilateral ptosis and miosis as expected but failed to have resolution of his positional neurologic deficits. Ultimately, the patient underwent craniotomy with left temporal artery to temporal M4 bypass 2 days later. Postoperatively, his blood pressure was gradually lowered without recurrence of his symptoms.

Conclusions: Stellate ganglion block has been utilized as an effective modality for the treatment of refractory cerebral vasospasm in the setting of subarachnoid hemorrhage. It has been demonstrated that an effective SGB reduces cerebral blood flow velocity, pulsatility index and improves neurologic exam in the setting of severe cerebral vasospasm [source 1]. Monitoring modalities such as transcranial doppler ultrasonography have further demonstrated improvement in cerebral blood flow and perfusion following SGB [source 2]. However, the direct effect of SGB on the intracranial cerebral vasculature is unclear [source 3]. In the case described above, the patient was unable to sit up past 10 degrees without recurrence of significant neurologic deficits. Furthermore, the eventual surgical procedure required detailed anatomical planning that was unable to be performed urgently. Thus, we performed a SGB to enhance cerebral perfusion with a goal of allowing the patient to liberalize his positional and activity restrictions while residing in the intensive care unit awaiting his surgical procedure. Unfortunately, despite a satisfactory blockade with local anesthetic as demonstrated by his ptosis and miosis, the patient did not experience any improvement in his positional stroke symptoms. Thus, his positional and activity restrictions remained intact until he was able to undergo a successful surgical procedure. Importantly, the patient did not suffer any complications or hemodynamic derangements from the SGB. This suggests that the intricacies of cerebral perfusion autoregulation and the effects of SGB are not as simple as vasodilatory relaxation. The patient's underlying pathophysiology of severe vasoocclusive disease with Moyamoya may have also contributed.

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Critical Care 79 - Systemic absorption of normal saline irrigation during laser TURP and cystolitholapaxy causing severe metabolic acidosis

Steven Morozowich[1], Alexander Roesler[2], Matthew Hammer[3]

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Introduction: Monopolar transurethral resection of the prostate (TURP) requires electrolyte-free irrigation fluid where excessive absorption of hypoosmolar irrigation fluid is known to cause hypervolemia, hyponatremia, and TURP syndrome in severe cases (1). However, normal saline (NS) can be used as the irrigation solution in bipolar and laser TURP, and the risk of TURP syndrome and severe electrolyte disturbances are thought to be minimized. Further, bipolar laser TURP results in superior field hemostasis, and the use of NS reduces the risk of irrigating fluid absorption, hypervolemia, and electrolyte abnormalities (2). However, despite this, the use of NS is known to cause acid-base imbalances and electrolyte disturbances and anesthesia providers must remain vigilant during these cases (2,3).

Methods: A 79-year-old male with benign prostatic hypertrophy and multiple bladder stones (> 4 cm) presented for elective laser TURP and cystolitholapaxy. His past medical history was significant for hypertension, mitral valve regurgitation, and nonischemic cardiomyopathy that was well-managed (left ventricular ejection fraction 45-65%) on sacubitril, valsartan, spironolactone, metoprolol, and amiodarone.

The patient underwent general anesthesia with an uncomplicated induction and tracheal intubation. Intraoperative anesthesia was maintained with sevoflurane, fentanyl, and rocuronium. The patient's anatomy and nodular massive hyperplasia made the procedure technically challenging due to difficulties mobilizing and identifying anatomical structures. The cystolitholapaxy was also complicated by recurrent instrument outflow clogging that required clearing with NS irrigation boluses and caused bladder dilations. Continuous bladder irrigation with NS was used throughout the procedure to improve field visualization and although the total volume of irrigation fluids used was unknown it was acknowledged to be more than routine.

After four hours of surgery the patient developed hypotension with mean aterial blood pressure of approximately 50 mmHg and hypoxemia (pulse oximetry 65%) that were refractory to initial interventions. Arterial blood gas analysis demonstrated a profound metabolic acidemia (pH 7.0) with HCO3 12.6 mEq/L and base deficit -17.5 mmol/L, PaO2 60 mmHg and PaCO2 40 mmHg. Labs otherwise showed Na 140 mEq/L, Cl 129 mEq/L, Hgb 9.4 g/dL, PT/PTT 19.4/48 sec, and lactate 3.1 mmol/L. Intraoperative transesophageal echocardiography showed hyperdynamic function with LVEF 70% and LVOT VTI 11.2 cm and small LV/RV sizes, consistent with either hypovolemia and vasoplegia. Based on these findings, bladder perforation with acute blood loss anemia were considered and an exploratory laparotomy was performed but was unrevealing. The procedure was completed but due to the on-going hemodynamic instability requiring vasoactive support the patient was transferred to the intensive care unit (ICU) intubated for further management with the working diagnosis of systemic absorption of excessive NS irrigation. Subsequently, the patient rapidly improved with discontinuation of bladder irrigation and diuresis.

Results: Not applicable – medically challenging case report

Conclusions: We present a case of systemic absorption of NS irrigation during laser TURP causing severe metabolic acidosis resulting in profound metabolic acidosis with secondary vasodilation/vasoplegia and pulmonary edema and hypoxemia requiring postoperative ICU transfer. Bipolar cautery and laser TURP is now the standard of care in prostate surgery and allows for irrigation with electrolyte-containing fluids such as NS to avoid classic TURP syndrome. However, although TURP syndrome is avoided, systemic absorption of NS may still occur and may result in complications. Risk factors for the systemic absorption of irrigation in this case included increased prostate size, prostatic vascularity, total surgical time, and the acknowledged high volume of irrigation fluid used. Retrospective acknowledgement by the surgeon also noted that irrigation boluses to unclog the resectoscope were required and caused repeated bladder dilations, which are known to accelerate fluid absorption (2). In conclusion, when perioperative risk factors for systemic irrigation fluid absorption present intraoperatively during laser TURP, particularly in the setting of hypotension and/or hypoxemia, prompt evaluation must be made to initiate early management to mitigate complications.

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Critical Care 80 - Tachyarrhythmias following large volume blood loss orthotopic liver transplant: pulmonary artery catheter irritation vs critical illness etiology

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Introduction: Tachyarrhythmias are commonly associated with increasing age, coronary artery disease, heart failure, recent surgery, myocardial infarctions, and thyroid disease. Arrhythmias are also commonly associated with pulmonary artery catheter (PAC) placement (37%) and removal (25%). (1) There is a case report of postoperative life-threatening arrhythmias related to severe angulation of PAC (2), but the presence of a correctly placed PAC does not seem to increase the incidence of tachyarrhythmia. We present the case of a 40-year-old male who developed atrial bigeminy and supraventricular tachycardia with hemodynamic instability in the presence of a verified, correctly placed PAC, refractory to both electrical and chemical cardioverting agents.

Methods: This patient was admitted to the Surgical Intensive Care unit following an orthotopic liver transplant for a history of alcoholic cirrhosis with ascites, hepatic encephalopathy, pancytopenia, and hepatorenal syndrome necessitating continuous renal replacement therapy (CRRT). A pulmonary artery catheter was placed to optimize fluid management. Intraoperative course was significant for 12L of estimated blood loss and a lactate of 6.0. Bedside echocardiography showed evidence of stress cardiomyopathy, with evidence of worsening left ventricular function.

Upon recognition of arrhythmias on formal electrocardiogram [Image 1,2], management included: lidocaine (100 mg); Magnesium (2 g); and amiodarone bolus (150 mg/kg) with infusion (1 mg/min). CRRT was resumed with an ultrafiltration rate of 50 mL/hr. No significant laboratory abnormalities were considered to be the cause of the tachyarrhythmia, with a glucose of 276; potassium of 5.4 mg/dL; pH of 7.4; and a down-trending lactate of 5.1 in the immediate postoperative period. The patient remained hemodynamically unstable, necessitating infusions of epinephrine and norepinephrine. Chest X-ray confirmed PAC placement in pulmonary artery [Image 3]. The PAC was readjusted on multiple occasions, but arrhythmias did not resolve. Electrical cardioversion with 200 Joules was performed four times with no resolution of arrhythmia. Cardiovascular medicine suspected the arrhythmias were related to his critical illness status.

On Postoperative Day (POD) 3, his PA catheter was removed with complete resolution of his arrhythmias. No defect or angulation was noted in the PAC upon removal. He was transitioned from an amiodarone infusion to twice daily oral dosing and his trachea was extubated on POD 6. CRRT was transitioned to intermittent hemodialysis on POD 10.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: PACs allow for advanced and accurate data regarding cardiac output, SvO2, left and right ventricular filling pressure and right ventricular afterload. (3) This data can then be interpreted and help guide resuscitative efforts both in the operating room and intensive care unit. There is conflicting data whether PACs improve mortality - despite having additional hemodynamic monitoring there are multiple studies showing no benefit, or even worse outcomes with PAC guided resuscitation. (4) Proponents of PAC usage argue that the data obtained is superior other methods; this is especially true for right ventricular failure. (5)

Ventricular arrhythmias and even complete heart block can occuring during placement and removal of PACs. This occurs when the catheter is passing through the right ventricle. There are no reported case studies reporting supraventricular arrhythmias related to PACs. This patient's arrhythmias are likely multifactorial in nature, with new stress cardiomyopathy, large volume shifts, vasopressors and PAC agitation of his right heart all likely contributing. With complete resolution occurring immediately following the removal of his PAC is likely that, even without an intrinsic defect in the PAC, its position caused a degree of myocardial irritation.

The timing of PAC removal typically coincides with improving clinical status and hemodynamic stability. In the setting of hemodynamic instability in the postoperative period following high volume blood loss, it is very helpful to have invasive monitoring techniques to help guide resuscitation. In the setting of frequent arrhythmias contributing to said instability, iatrogenic induced arrhythmias related to PAC irritation of the myocardium or PAC intrinsic defect leading to myocardial irritation should be on the differential and the timing of removal should warrant active discussion.

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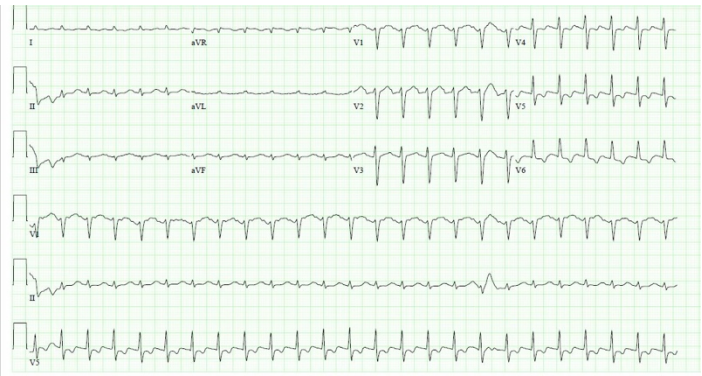

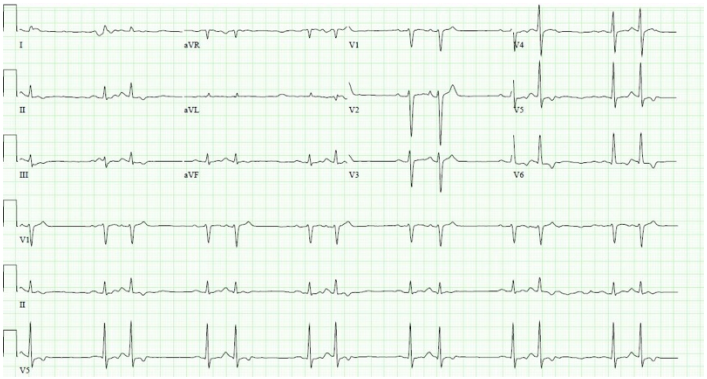
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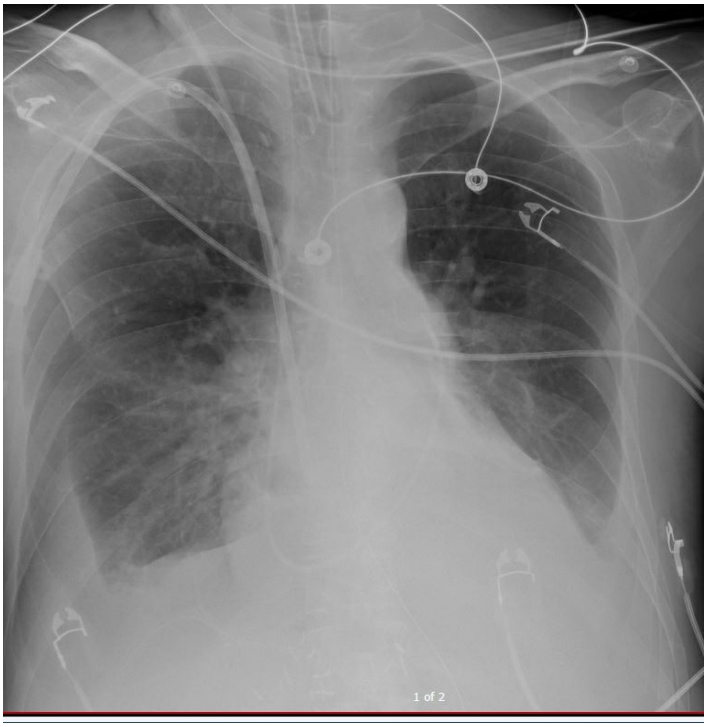
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Critical Care 81 - Tension Capnothorax During Laparoscopic Robotic Assisted Esophagogastric Resection

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Introduction: Tension capnothorax is a known complication of laparoscopic esophageal surgery that can lead to significant intraoperative morbidity and mortality.1 In this case report, we describe the diagnosis and management of a tension capnothorax in a patient undergoing esophageal resection for gastroesophageal junction adenocarcinoma. Specifically, we highlight the use of intraoperative transthoracic ultrasonography as a diagnostic tool. Additionally, we highlight a conservative management strategy, which allowed for successful completion of the procedure without the need for chest tube placement or conversion to an open surgical approach.

Methods: A 39-year-old female with a past medical history of morbid obesity presented for a laparoscopic robotic assisted esophagogastric resection for adenocarcinoma. Her past surgical history was significant for a sleeve gastrectomy complicated by severe gastroesophageal reflux disease, ultimately requiring laparoscopic hiatal hernia repair and Roux-en-Y gastric bypass. On the day of surgery, the patient underwent uneventful rapid sequence induction of anesthesia and tolerated initial incision, peritoneal insufflation, and robotic assisted laparoscopy well. After 45 minutes, however, the pulse pressure variation of the arterial waveform began to increase rapidly, and the patient experienced an abrupt PEA arrest. The heart rate remained in normal sinus rhythm at approximately 80 bpm, but EtCO2 dropped significantly and arterial and plethysmographic waveforms were lost. Prior to this, mechanical ventilatory parameters and arterial blood gas analysis, including electrolytes, were normal and unchanged. A brief physical exam revealed no carotid pulse and no obvious crepitus over the upper chest, neck, or arms. After administration of epinephrine and rapid peritoneal desufflation the patient regained of spontaneous circulation. Given the pulse pressure variation and rapid improvement after peritoneal desufflation, tension capnothorax was suspected.

After undocking the robot, point of care ultrasonography revealed the absence of lung sliding in both 2-D and M-mode to the level of the left 5th intercostal space at the anterior axillary line. Ultrasonography of the right hemithorax revealed normal lung sliding. Based upon these findings, the surgical team was able to perform transabdominal pleural decompression under direct visualization, and upon inspection of the left diaphragm, a small defect was identified and repaired. Afterwards, the decision to proceed with laparoscopy at low insufflation pressures with increased tidal volumes and positive end-expiratory pressure allowed for successful esophageal resection without the need to convert to an open approach. After additional transabdominal pleural decompression and abdominal closure, lung sliding was present in the left hemithorax; the patient was hemodynamically stable; and mechanical ventilatory parameters were at baseline. The patient was extubated with an intact neurologic exam and recovered uneventfully in the ICU.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Our patient's complex surgical history led to an increased risk for CO2 entry into the thorax through the pleuroperitoneal hiatus, diaphragm, mediastinum and pleuroperitoneal connections that likely existed as a result of previous procedures.1,2 Interestingly, increased pulse pressure variation and PEA arrest without significant changes to mechanical ventilatory parameters were the presenting symptoms of a tension capnothorax. This may be due to a combination of the ability for CO2 to accumulate rapidly in the thoracic cavity and diminished preload from peritoneal insufflation, positive pressure ventilation, and reverse Trendelenburg positioning.3,4 We also demonstrated the utility of point of care ultrasound to promptly diagnose tension capnothorax in the intraoperative setting. This provided diagnostic certainty for a complex differential diagnosis which included cardiac tamponade, tension pneumomediastinum, massive pulmonary embolism, anaphylaxis, profound vagal response, hemorrhage, and hypovolemia.2,4 Based on the ultrasound findings we urgently treated the tension capnothorax, employed a conservative perioperative management strategy, and ultimately facilitated safe and successful laparoscopic esophageal resection for the patient.

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Critical Care 82 - The Use of VV-ECMO in a Patient with COVID-19 and Human Immunodeficiency Virus co-infection who developed Pneumocystis jirovecii Pneumonia

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Introduction: The use of veno-venous extracorporeal membrane oxygenation (VV-ECMO) in immunocompromised patients had historically resulted in poor outcomes. However, there is more recent evidence to suggest otherwise. There is a paucity of data on the use of VV-ECMO for respiratory failure in immunocompromised patients who are co-infected with both human immunodeficiency virus (HIV) and SARS-CoV-2 virus.
We report a patient with previously undiagnosed HIV infection and acquired immune deficiency syndrome (AIDS) who also had concurrent COVID-19 infection. The patient subsequently developed acute respiratory distress syndrome (ARDS) requiring VV-ECMO.

Methods: A 48-year-old female with only a past medical history of obesity presented to a community hospital with dyspnea and hypoxia. Of note, she had received the complete primary series of the COVID-19 vaccine and had a positive COVID test a month prior with mild symptoms not requiring hospitalization at that time. At the time of her presentation, the Omicron strain of the SARS-CoV-2 virus was predominant in the country.

Her symptoms resolved within days but returned a week later eventually resulting in presentation to the emergency department. She was hypoxic in the ED. On chest CT, she was noted to have diffuse ground-glass opacities throughout the lungs, with no cavitary lesions, masses, effusions, or pneumothorax. COVID test was performed and found to be positive again. During this initial admission, she was treated with a course of steroids and discharged home days later without oxygen.

A week after discharge, she presented again to the hospital with significantly worsening respiratory status and high oxygen requirements. She again tested positive for COVID. Her respiratory status precipitously declined and met criteria for the diagnosis of ARDS.

She was intubated and required harmful ventilator settings and paralytic infusion. Extensive infectious workup was initiated, and broad-spectrum antimicrobials were administered. The etiology of her ARDS was presumed to be from COVID-19. After showing no improvement, she was deemed to be a good candidate for ECMO support. Our patient was transferred to our academic medical center where she was immediately cannulated for VV-ECMO.

The day after cannulation, a test for PJP previously sent by the transferring facility resulted as positive. A rapid HIV test was then immediately sent, which resulted positive and subsequent workup revealed an absolute CD4+ count of 11.4 cell/mm^3 (normal: 500 to 1500 cell/mm^3). This met criteria for a diagnosis of AIDS. She began antiretroviral therapy, as well as steroids and sulfamethoxazole / trimethoprim as treatment for PJP pneumonia. The patient's hospital course was complicated by hypotension requiring pressors, need for renal replacement therapy, secondary bacterial and fungal infection, and right ventricular failure. She went into cardiac arrest on hospital day 34 and was unable to be resuscitated.
Results: Not Applicable – Medically Challenging Case Report
Conclusions: This was a challenging case due to a lack of information regarding the patient's immunocompromised status prior to cannulation. Knowledge of our patient's undiagnosed HIV/AIDS status may have altered our decision to initiate VV-ECMO. Outcomes in this population are mixed. It is often difficult to identify patients that would benefit from ECMO because of the pathophysiologic heterogeneity across various etiologies of immunosuppression.

In the ECMO to Rescue Lung Injury in Severe ARDS (EOLIA) trial, immunocompromised patients made up 22% of the patients, and the 60-day mortality rates for the ECMO and control groups were 56% and 78%, respectively[1]. Additionally, studies have shown acceptable outcomes, particularly in patients with HIV/AIDS, with the conclusion that HIV shouldn't be viewed as a contraindication to ECMO[2-5].

The subgroup of patients with HIV/AIDS is of particular interest, given the impressive improvement of therapeutic options over the last two decades.

In conclusion, albeit based on very limited data, there is a growing consensus in the literature that immunocompromised status, including a diagnosis of HIV infection or AIDS, should not be considered a contraindication to ECMO, but that this becomes a highly individualized decision[6]. Our case report adds to this small but growing volume of data that calls for more research to fully understand when to utilize ECMO treatment in patients with severe immunosuppression.

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Critical Care 83 - Veno-venous ECMO Membrane Oxygenator Exchange in an Extubated Patient

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Introduction: Veno-venous extracorporeal membrane oxygenation (VV ECMO) serves as a bridge to recovery for patients with acute respiratory distress syndrome (ARDS). While often used in tandem with mechanical ventilation, VV ECMO can alternatively facilitate early extubation, ventilator independence and a reduction in sedation requirements, a strategy termed “awake” ECMO. Benefits of this strategy include elimination of ventilator-associated risks and early initiation of physical therapy, but it is not without potential hazards. In an awake ECMO strategy, extracorporeal support becomes the sole guarantor of oxygenation and ventilation, and the threat of mechanical failure and need for component exchange presents without the safety of a definitive airway, a vulnerability highlighted by lengthy ECMO runs and aggressive attempts at early extubation during the COVID-19 pandemic.

We present a case of successful oxygenator exchange in an awake ECMO patient with ARDS secondary to SARS-CoV-2 pneumonia. A procedure checklist and recommendations were created by a multi-disciplinary team to further increase safety during awake ECMO oxygenator exchange (Figure 2), a clinical scenario currently without universal recommendations to guide management.

VV ECMO can be used to facilitate recovery from ARDS by temporarily assuming the role of the blood gas interface when the native lungs are injured. Frequent monitoring and technical evaluation are required, including pre and post membrane oxygenator arterial blood gas analysis (ABG) to assess oxygenator function (Figure 1). In the event of a damaged or ineffective oxygenator, prompt replacement is necessary. Oxygenator exchange requires a systematic approach (Figure 2) by our team, as even brief interruptions of extracorporeal support can result in complete cardiovascular collapse and death, particularly in awake ECMO patients. At present, there are no universal recommendations to guide the management of oxygenator replacement in this patient population.

Methods: A 23-year-old patient with ARDS secondary to SARS-CoV-2 pneumonia on VV ECMO was transferred to our facility for continued management. Under our care, the patient was extubated to high-flow nasal cannula (HFNC), and their circuit was reconfigured to oxy-RVAD for RV failure and hypoxemia. Four weeks of clinical stability ensued until routine pre and post oxygenator blood gas analysis displayed reduced oxygenator function. A collective decision was made by the patient and our team to attempt “awake” oxygenator exchange. A checklist and recommendations were created by the team to ensure safe oxygenator exchange (Figure 2), which was performed on HFNC without major complications. Bag masking was used to assist spontaneous respirations for transient hypoxemia and tachypnea that fully resolved upon re-initiation of extracorporeal support.

A summary of our team's recommendations follows. Once oxygenator failure has been identified, pre-procedural transfusion need should be evaluated, along with sedation and analgesia, and vasopressor and right heart support requirements. Next, an anticipatory increase in ECMO support should be made, an airway asessment performed and pre-oxygenation begun. After emergency resuscitation equipment has been arranged at the bedside and a safety time-out conducted, a pre-procedural clamp trial should be performed to assess the patient's pulmomary reserve. A re-assessment of sedation, analgesia and hemodynamic support requirements should then follow based on the results of the clamp trial. Respiratory support may be provided during the procedure by bag masking in addition to the continuation of pre-procedural, non-invasive oxygenation methods. Should intubation become necessary, a hemodynamically stable induction should be made, with a return to spontaneous breathing especially in those patients with right heart dysfunction. Immediately following oxygenator exchange, ECMO support requirement and tranfusion need should be re-evaluated.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: The management of patients requiring VV ECMO for ARDS secondary to SARS-CoV-2 continues to evolve, including an “awake” ECMO strategy. At present, there are no universal recommendations to help guide oxygenator replacement in these patients. Our multi-disciplinary team successfully exchanged an oxygenator in an awake ECMO patient and developed a checklist and recommendations to further enhance the safety of the process in this patient population.

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Figure 1: *VV ECMO Oxygenator Exchange Criteria*
Post oxygenator ABG pO2 <150 mmHg **or** <200 mmHg with increasing FiO2
Increased Delta P 2x baseline with reduced flow despite increased RPMs
Post-oxygenator outflow cannula clot with possible embolization
Tubing or oxygenator fracture with possible air entrainment

Figure 2: *VV ECMO Oxygenator Exchange Procedure Checklist & Recommendations*
Early Identification of Oxygenator Failure
Determine Pre-procedural Transfusion Need based on Pre-oxygenator PaO2 & Hematocrit
100% ECMO Circuit **AND** Increase Sweep 1-2L
Airway Assessment
Pre-oxygenation: NIPPV, HFNC, 100% NRB
Assess Right Heart & Need for Pharmacologic Support
Assess Need for Vasopressor Support
Assess Need for Sedation & Analgesia
Place Defibrillator Pads
Ensure Emergency Resuscitation Medications & Equipment is Present
Perform Safety Time-out: Intensivist, ECMO Specialist, Perfusionist, Respiratory Therapist, ICU Nurse Present
Perform Pre-procedural Clamp Trial
Re-assess Sedation, Analgesia & Hemodynamic Support Needs based on Clamp Trial Observations
Determine Post-procedural Transfusion Need and/or ECMO Settings Adjustment based on CBC & ABG

Critical Care 84 - Venoarteriovenous ECMO in Cardiogenic Shock and Severe ARDS associated with COVID-19 Infection

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Introduction: Cardiomyopathy has been reported in up to 20% of patients with COVID-19, and severe disease can lead to cardiopulmonary failure. There were no pre-specified criteria for extracorporeal membrane oxygenation (ECMO) placement. However, retrospective studies have showed improved outcomes with early initiation of ECMO in setting of cardiopulmonary failure. Here we present a case that demonstrates the role of venoarteriovenous (VAV) ECMO in the case of severe ARDS and advanced biventricular systolic failure in the setting of COVID-19.

Methods: 46-year-old-male with a history of class 2 obesity, paroxysmal atrial fibrillation, and heart failure with recovered ejection fraction was admitted to ICU for acute hypoxic respiratory failure, 3 days after COVID-19 pneumonia diagnosis. He was initiated on BIPAP but soon required intubation and paralysis for worsening ARDS on hospital day (HD) 1, however circulatory shock continued to worsen. Transthoracic Echocardiogram (TTE) on HD2 revealed left ventricular ejection fraction (LVEF) of 8% with new biventricular systolic failure. On HD3, decision was made to initiate temporary mechanical circulatory support (tMCS) with VAV-ECMO and intra-aortic balloon pump (IABP) for left ventricular (LV) venting with goal of bridge to recovery. Drainage site was the right common femoral vein and the return site was the left subclavian vein and right common femoral artery. After initiating ECMO therapy, his clinical condition gradually improved. On ECMO day 5, LVEF improved to 20% and right ventricular systolic function recovered, thus he was transitioned to veno-venous (VV) ECMO with IABP support. Eventually, IABP was weaned and removed on ECMO day 7, and VV-ECMO weaned over the subsequent 2 days. Our patient still required ventilatory support and underwent tracheostomy prior to discharge to long term care facility.

Conclusions: In the setting of COVID-induced cardiomyopathy, circulatory failure, and severe ARDS, the application of VAV-ECMO provides optimal circulatory support and pulmonary protection while avoiding differential hypoxia, which is a severe complication of VA-ECMO. In our case, we utilized VAV-ECMO and IABP to optimize oxygenation, hemodynamic support and left ventricular unloading, which served as a bridge to our patient's recovery.

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Liver

Liver 1 - Early onset invasive pulmonary aspergillosis in a liver transplant patient: a case report

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Introduction: Over 9,000 liver transplantations (LTs) are performed annually in the United States.[1] Invasive pulmonary aspergillosis (IPA) is rare in LT patients with an incidence rate between 1.2-5.8%, but the mortality rate is as high as 93%. Median time of onset of IPA after LT is 18-126 days.[2] Earlier onset of IPA is rare in non-high-risk patients. We herein report a case of early onset IPA after LT.

Methods: A 63-year-old man with a medical history of non-alcoholic steatohepatitis (NASH) cirrhosis underwent uncomplicated deceased donor LT and intensive care unit (ICU) recovery. On postoperative day (POD) 4, he developed dyspnea, cough, and transient hypoxemia. A chest x-ray was concerning for unilateral pneumonia and empiric broad-spectrum antibiotic coverage was started. Three days later, he developed hypotension and shortness of breath with bloody drain output. A CT abdomen noted a liver hematoma and bibasilar lung infiltrates. He was re-admitted to the ICU, where antibiotics were broadened and micafungin was added. He was managed with nasal cannula oxygen and nightly home continuous positive airway pressure (CPAP) until two nights later when he was intubated for increasing respiratory distress. A chest CT scan (figure1) revealed widespread bronchopneumonia, and a bronchoscopy (figure 2) demonstrated yellow-white plaques, thick white secretions, and uneven bronchial surfaces. Endobronchial biopsies and bronchoalveolar lavage were performed, and amphotericin B was started. *Aspergillus fumigatus* was subsequently isolated from the bronchoalveolar lavage samples, and voriconazole therapy was initiated. The biopsy revealed necrosis and rare hyphae. Over the next 5 days, the patient's condition deteriorated to severe septic shock and multiorgan failure. The patient's family decided to move toward comfort-focused measures only, and he expired on POD 15. Post-mortem lung tissue cultures were notable for *A. fumigatus*.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: IPA is rare but highly deadly in LT patients. Therefore, prediction and early identification is critical. Risk factors include a Model for End-Stage Liver Disease (MELD) score > 20, bacterial or cytomegalovirus (CMV) infection, renal insufficiency, re-transplantation, CMV donor positive recipient negative serology, bilirubin > 1.5 during LT, previous ICU stay, renal replacement therapy (RRT) after LT, and *Aspergillus* antigenemia.[3] Among these factors, our case was only notable for RRT. Although prophylaxis with voriconazole is effective in high-risk LT recipients, our patient did not meet criteria for IPA prophylaxis.[4]

Diagnosis of IPA typically includes laboratory, radiology, and histopathology data. However, current diagnostic tools prevent timely, accurate diagnosis and therapy. Serum Galactomannan (GM) and beta-D-glucan (BG) have low diagnostic accuracy in LT.[5] Of note, he had a positive BG and GM before transplantation. Radiologic findings of IPA include nodules, masses, and patchy consolidations.[2] CT evidence of a multifocal process was evident in this case on POD 6, and strongly suggestive of fungal pneumonia on POD 9. Endobronchial biopsy is required to make a formal diagnosis but is invasive and has a long turnaround time. Histopathologic evidence of invasive hyphae and bronchial wall necrosis are typical of IPA.

Voriconazole is the first-line therapy for IPA. As it inhibits the CYP450 3A4 isoenzyme, second-line therapies such as amphotericin B may be preferred to avoid immunosuppressant toxicity. In this case amphotericin was initially used while awaiting fungal speciation, which did not occur until POD 14. These diagnostic challenges, the rarity of early IPA in non-high-risk LT patients, and the initial presentation of hypotension and bleeding led to a delay in starting first-line therapy for IPA.

It is possible that prior colonization may have accelerated the onset time to infection. Six months before his transplant, the patient was treated for COVID pneumonia with dexamethasone, placing him at higher risk of subsequent *Aspergillus* infection.[6] Additionally, a CPAP device that was used before and after LT could have been the source of colonization. Nonetheless, laboratory cultures of the CPAP tubing reported the growth of yeast, not mold.

Early onset IPA is rare in non-high risk LT patients, but mortality is very high. Timely diagnosis and therapy can be critical to preventing disseminated disease and death.

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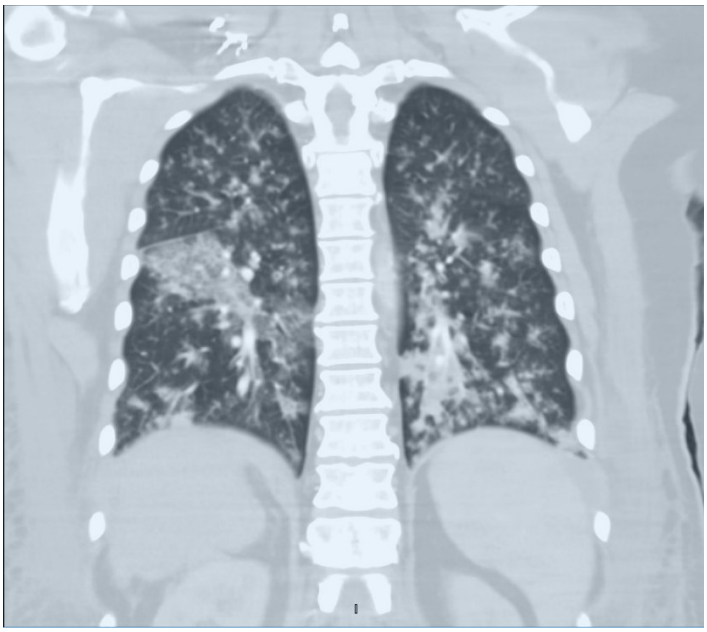

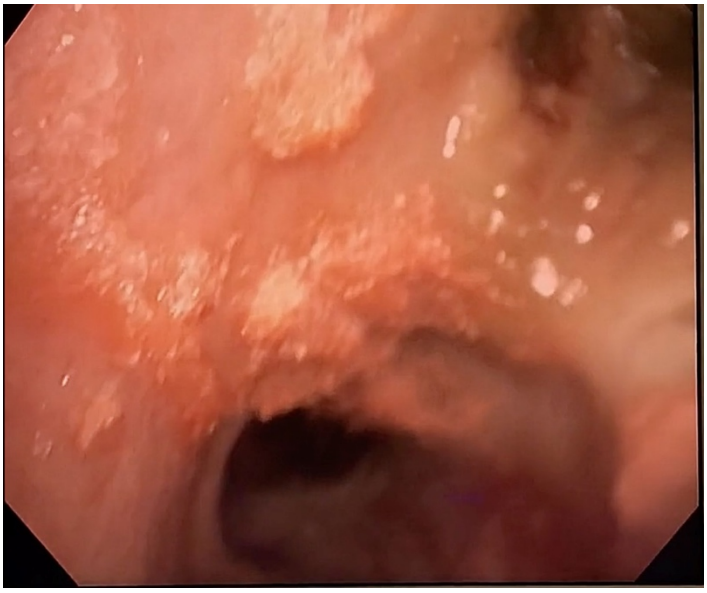

Liver 2 - Multifactorial Pulmonary Hypertension After Liver Transplantation

Krishnan Ramanujan[1], Pratik Pandharipande

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Introduction: Patients with liver disease are at increased risk of pulmonary disease, including pulmonary hypertension (pHTN). Although liver transplantation can be a treatment for pHTN from liver disease, preoperative pHTN is associated with increased morbidity and mortality after transplant.[1] Therefore, significantly elevated pulmonary artery pressures, with a mean pulmonary artery pressure (mPAP) of 50mmHg, are an absolute contraindication to transplant.[2] Here, we describe a patient who underwent liver transplantation with severe intraoperative pHTN and subsequent complex postoperative management.

Methods: A 51-year-old man with end-stage liver disease secondary to NASH cirrhosis presented for orthotopic liver transplantation. Preoperative transthoracic echocardiogram (TTE) showed normal biventricular function with a right ventricular systolic pressure (RVSP) of 71mmHg. He had no prior history of pHTN. A pulmonary artery (PA) catheter was placed with an initial PA pressure of 61/26 and mPAP 44. Intraoperatively the patient was noted to have increasing PA pressures with a peak of 93/44 with mPAP 62. Postoperative TTE showed LVEF of >75% with normal RV function and RVSP 59mmHg. Cardiac output (CO) was initially 11.1L/min and ranged from 7-15L/min. Initial differential diagnosis was that the patient's pHTN was secondary to his high CO state. Therefore, an epoprostenol wean was attempted on postoperative day (POD) 1 with resultant increases in PA pressures without right ventricular strain and slight decrease in blood pressure, suggesting a component of pulmonary arterial hypertension (PAH). The pHTN service was consulted on POD2 to assist with further management. After assessment of the patient's hemodynamic measurements and interdisciplinary conversations between all involved teams, it was concluded that the patient's pHTN was likely related to high CO and hypervolemia without PAH. As a result, diuresis was initiated and subsequently epoprostenol was slowly weaned off on POD2. PA pressures remained stable but persistently elevated with epoprostenol wean, and after discussion with the pHTN service about this, the PA catheter was removed on POD3. The remaining postoperative course was uncomplicated, and the patient was discharged from the hospital on POD7. Right heart catheterization one month after discharge was notable for a mPAP 71mmHg, pulmonary vascular resistance of 6.84 Woods units, and pulmonary capillary wedge pressure of 27mmHg. Given these findings, it was deemed that the patient likely had pHTN both from portopulmonary hypertension (PoPH) and diastolic dysfunction which had been previously masked by low systemic vascular resistance from end-stage liver disease.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Pulmonary hypertension in liver disease can be secondary to PoPH, high cardiac output, or intravascular overload, amongst other inherent causes such as primary pulmonary disease or left ventricular dysfunction. Although elevated mPAP in liver disease was initially universally associated with increased morbidity and mortality after transplant, recent retrospective and single-center studies have demonstrated that outcomes may be different for patients based on the specific etiology of pHTN.[1,2] Therefore, it may be appropriate to pursue further testing, such as right heart catheterization, in patients undergoing liver transplant workup who show signs or symptoms of pHTN, including those found on TTE such as elevated RVSP. This may help not only with assessment of appropriateness for transplant, but also with risk stratification in the ICU after successful transplant from a pulmonary standpoint.

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Liver 3 - Two-Hit Hypothesis – Massive Air Embolus followed by Intracardiac Thrombus during Liver Transplant

Zachary Fleissner[1], Valerie Smart[2], Ryan Chadha[1], Justin Burns

Mayo Clinic Florida[1] US Navy[2]

Introduction:

1. Identification of air emboli and intracardiac thrombi using standard as well as advanced intraoperative monitoring

2. Identification of air emboli and intracardiac thrombi with transesophageal echocardiography

3. Explain the treatment modalities during impending right-sided cardiac failure with perioperative air emboli and intracardiac thrombi

Methods: Intraoperative air embolus during liver transplantation is a common event with potential for devastating perioperative complications, particularly cardiopulmonary and neurologic in the presence of right-to-left intracardiac or intrapulmonary shunting (1). Intraoperative cardiac thrombus is rare, but is associated with high perioperative mortality (2). We present a case of intraoperative massive air embolus followed by intracardiac thrombus during the anhepatic phase of liver transplantation. To our knowledge, there are no other cases describing a massive air embolus followed by massive intracardiac thrombus during liver transplant.
A 60-year-old female with decompensated liver cirrhosis complicated by ascites, jaundice and hyponatremia secondary to alcohol and hepatocellular carcinoma (HCC) s/p Y-90 underwent an orthotopic liver transplant. Pretransplant cardiac testing showed normal biventricular function with trivial intrapulmonary shunting and negative stress testing.

Induction was uneventful. Two 9Fr introducers were placed in the right internal jugular vein and connected to a rapid transfuser. Standard monitors, bilateral radial arterial lines, a pulmonary arterial catheter with real-time continuous cardiac output monitoring, and transesophageal echocardiogram (TEE) were placed. Initial TEE examination redemonstrated normal biventricular function with trivial intrapulmonary shunt. An IVC tear occurred during liver mobilization. Mean pulmonary artery pressures (mPAP) rose from 11mmHg to 50mmHg, end tidal carbon dioxide (ETCO2) dropped from 40 cmH20 to 20 cmH20, with systemic hemodynamic instability (Figure 1). TEE showed air in all four chambers. Hemodynamic instability was supported with epinephrine boluses followed by vasopressor and inotropic infusions. Cerebral oximetry was placed. The patient then developed right atrium (RA) and right ventricle (RV) thrombus (Image 1). The thrombus was treated with heparin bolus and low dose tissue plasminogen activator (tPA). The remainder of the case proceeded uneventfully, and patient was transferred to the ICU with minimal inotropic support. She was discharged from the hospital on postoperative day 8 without neurologic or cardiac complication.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Large air emboli and intracardiac thrombi can have devastating intraoperative consequences if not diagnosed quickly. Transesophageal echocardiogram and pulmonary arterial catheters with continuous cardiac output are monitors that allow for nearly instantaneous recognition and real-time monitoring of resolution of intracardiac outflow tract obstruction. Judicious treatment of the cause of acute right heart failure with real-time monitoring are keys to successul perioperative management in these cases.

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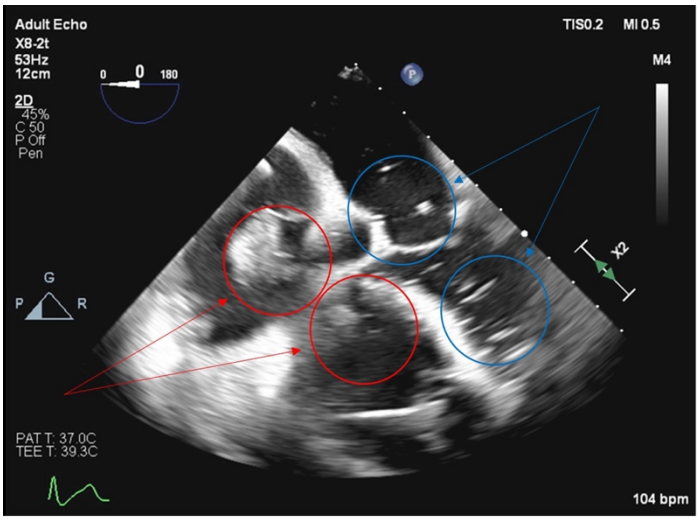

Image 1: Midesophageal 4 Chamber view. Red Arrow with circles: right-sided intracardiac thrombus. Blue Arrows with circles: left-sided air from massive air embolus

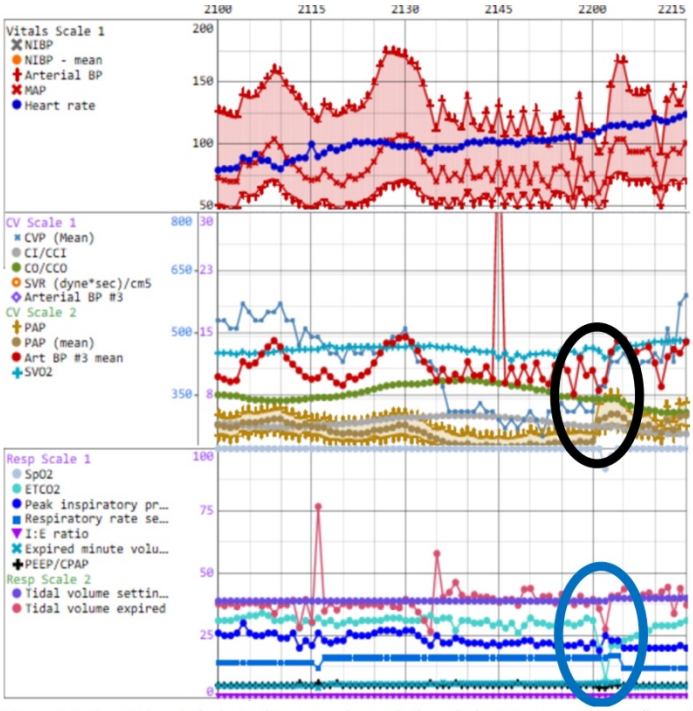

Figure 1: Perioperative vitals during liver transplant. Black oval: sharp increase in mPAP (brown) with decrease in MAP (red) followed by increase in CVP (dark blue). Blue oval: precipitous

Neuroscience in Anesthesiology and Perioperative Medicine

Neuroscience in Anesthesiology and Perioperative Medicine 1 - A challenging case of severe hypoxia due to respiratory distress syndrome after subarachnoid bleeding

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Introduction: Acute lung injury, including respiratory distress syndrome after subarachnoid bleeding causes worse outcomes in terms of postoperative complications and mortality. Although there are several causes of intraoperative hypoxia, we experienced severe intraoperative hypoxia during arterial clipping of subarachnoid bleeding that was diagnosed as respiratory distress syndrome due to neurogenic lung edema(1,2). A multimodal approach, including ventilatory management with high PEEP, pharmacological injection of steroids and inhaled nitric oxide enabled continuation of the surgical procedure.

Methods: A 71-year old woman (157 cm, 56 kg, BMI: 24 kg/m^2) experienced deterioration of consciousness due to subarachnoid bleedings from the middle cerebral artery. She had convulsions and aspiration pneumonia requiring tracheal intubation. Emergency craniotomy for surgical clipping of the artery and decompression of brain edema were deemed necessary. At her entry to the operating room, her oxygenation was relatively low (P/F ratio: 110), although her oxygenation gradually deteriorated before craniotomy. We increased the inhaled oxygen concentration to 100%, but the P/F ratio further decreased to 50-60. Hence, we discussed with the surgeon about discontinuation of the surgery. Since evaluation of her chest X-ray showed respiratory distress syndrome, we administered 1 g methylprednisolone and increased PEEP from 5 to 12 cmH_2O. However, since her SpO_2 still remained at 75-80%, we started nitric oxide inhalation at 100 ppm. With this, her SpO_2 gradually increased within a few minutes. Further, her systolic blood pressure was maintained at 80-90 mmHg using inotropic support with phenylephrine. We finally decided to continue her surgery. Intraoperatively, her SpO_2 improved after craniotomy, and the surgery was completed without any other complications.

Results: Not Applicable – Medically Challenging Case Report
Conclusions: Assuming that the cause of respiratory distress syndrome in our patient was aspiration pneumonia, acute heart failure and neurogenic lung edema, we introduced nitric oxide inhalation in order to improve the ventilation-perfusion mismatch, which significantly improved her oxygenation. In cases such as ours with respiratory distress and subarachnoid bleeding, it is essential to suspect neurogenic pulmonary edema and promptly administer aggressive supportive respiratory therapy. Additionally, inhaled nitric oxygen should be introduced for patients with severe hypoxia.

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Neuroscience in Anesthesiology and Perioperative Medicine 2 - Propofol-Induced Myoclonus During Maintenance of Anesthesia: A Case Report

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

Introduction: Propofol, a widely used sedative-hypnotic, is a short-acting gamma-aminobutyric acid receptor ($GABA_A$) agonist commonly used as a general anesthetic during surgeries requiring trans-cranial motor-evoked potential neuromonitoring. An infrequent side effect of propofol is seizure-like phenomena, such as myoclonus. When they occur, these phenomena are rarely seen during the maintenance phase of an anesthetic. Here we report a case of propofol-induced myoclonus during the maintenance phase of anesthesia.

Methods: A 23-year-old 66-kg male patient underwent an L4-S1 posterior spinal fusion for resection of an osteoblastoma under general anesthesia. On the morning of surgery, the patient was anxious and complained of lower back pain that was associated with bilateral radiculopathy. He was otherwise healthy with no allergies, family history of seizures, or family history of anesthetic complications.

The patient was pre-medicated with acetaminophen and midazolam. He then received a balanced induction and an endotracheal airway was secured. An arterial line and additional IV access were obtained. A Sedline Brain Function Monitor (Masimo, Inc. Irvine, CA) was applied, and a neurophysiologist placed leads for motor-evoked (MEP) and somatosensory-evoked potentials (SSEP). Maintenance of anesthesia was performed using a total intravenous anesthetic consisting of propofol, lidocaine, magnesium, ketamine, and fentanyl infusions.

90 minutes after incision the patient developed myoclonic jerks in his bilateral upper extremities. Each episode lasted for 5-10 seconds before spontaneously resolving and then recurred within a few minutes. These episodes did not correlate with any surgical or MEP stimulus. During these episodes, the patient remained hemodynamically stable and normothermic. Electroencephalography (EEG) was consistent with an appropriate depth of anesthesia and did not demonstrate any evidence of seizure. Furthermore, no lower extremity EMG activity correlated with the upper extremity myoclonic movements. This pattern of recurrent myoclonus continued for approximately 30 minutes.

We suspected that propofol was the likely culprit and significantly decreased the infusion. To compensate for this, we added 0.3 MAC of sevoflurane and increased the ketamine infusion to 10 mcg/kg/min. To ensure amnesia and an adequate depth of anesthesia, we also administered a 2 mg bolus of midazolam and 2 mg bolus of hydromorphone. After these interventions, the patient did not have any additional episodes of myoclonus. He was extubated at the end of the procedure and recovered from anesthesia without incident. The patient denied having any complications from anesthesia including intraoperative awareness, unpleasant postoperative sensory perceptions, or postoperative seizures.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: A rare side effect of propofol is seizure-like activity such as myoclonus or generalized tonic-clonic seizures. A systematic review demonstrated that these phenomena most often occur during induction, emergence, or the immediate post-operative period. There are very few published cases during anesthetic maintenance.

Our institution has seen intraoperative myoclonus during the maintenance phase of an anesthetic several times. In our experience, it is most common in pediatric patients undergoing correction for neuromuscular scoliosis. In adults, it can occur in patients with pre-existing grade 3 or 4 hyperreflexia secondary to brain or spinal cord pathology. The myoclonus is preceded by a stimulus, usually electrical cautery stimulation, and tends to develop approximately 60-90 minutes after surgical incision. The distribution of myoclonus is variable and can be confined to the upper or lower extremities or involve the whole body in various patterns. Because of the way in which it presents, we hypothesize that the mechanism is cortical reflex myoclonus.

The myoclonus is successfully treated 80% of the time by increasing the propofol infusion rate. Rarely, the myoclonus is resistant to higher levels of propofol, and various alternative anesthetic techniques need to be employed. In our patient's case, we were able to suppress the myoclonus and maintain a general anesthetic compatible with MEP and SSEP neuromonitoring using a predominantly ketamine-based anesthetic supplemented with sevoflurane, lidocaine, and a low infusion rate of propofol.

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Obesity

Obesity 1 - Repeated Use of a Simple Low-Flow Nasal Mask-Face Tent to Provide Spontaneous CPAP Ventilation/Oxygenation and Reduce Droplet/Aerosol Spread in a High-Risk Super-Obese Patient with Previous COVID-19 Infection during TEE under MAC at NORA

Sabina Musovic[1], Benjamin Landgraf, Carly LaRue-Beach, Mariah McGrory, Christine Fratzola, James Tse[1]

Rutgers Robert Wood Johnson Medical School[1]

Introduction: It is always very challenging to provide MAC for patients at non-OR anesthesia (NORA) locations. Over-sedation and/or airway obstruction may result in severe desaturation, especially in obese patients with obstructive sleep apnea (OSA). A pediatric facemask has been shown to provide spontaneous nasal CPAP ventilation and improve O_2 delivery in deeply sedated OSA patients.[1]

A simple low-flow nasal mask-face tent provided pre/apneic nasal oxygenation and reduced aerosol/droplet spread during RSI, intubation and extubation in a COVID-19 patient [2] and during extubation in a COVID-19 patient undergoing peroral endoscopic myotomy.[3]

We used this simple technique to provide nasal CPAP oxygenation and reduce aerosol/droplet spread in a super obese patient during TEE under MAC amid the ongoing COVID-19 pandemic.

Methods: A 41-y/o male 5'11", 486 lbs, BMI 67.8 kg/m^2, with anxiety/depression, bronchitis, severe OSA requiring daytime CPAP, anemia, polycystic kidney disease, CKD lV, HTN, non-ischemic cardiomyopathy, HFrEF (EF 20-25%), PAF s/p recent AICD insertion with pocket infection and Staph bacteremia, presented for TEE to rule out endocarditis in the Cardiac Catheterization Lab. He had COVID-19 infection one year prior and received home monoclonal antibody therapy. He was tested COVID-19 negative upon admission.
However, he required facial CPAP support while sitting up in a chair in the SICU and was transported to the Cath Lab with portable facial CPAP.

He had a Mallampati Class III airway. An infant facemask for delivering nasal CPAP was shown to the patient and he gave his consent for photography and case report.

The nasal mask-face tent was secured over his nose with elastic head-straps and connected to the anesthesia circuit/machine (Fig. 1). Pads were placed over his nasal bridge and under the head-straps. He mouth was covered by a face tent to reduce aerosol/droplet spread. His oropharynx was then pretreated with local anesthetic gargle and spray.

The APL valve was adjusted to deliver 8-10 cm H_2O CPAP with fresh O_2 flow of 4 L/min. A nasal cannula with air sampling tubing was taped below his lower lip underneath the face tent to continuously evacuate oral droplet/aerosol and monitor CO_2 exhaled from the mouth. Following nasal CPAP pre-oxygenation, his SpO_2 improved from 94% to 100%. Deep sedation was then titrated with lidocaine (100 mg), propofol boluses (2 x 50 mg) and propofol infusion (100 mcg/kg/min). He maintained spontaneous nasal ventilation and 100% SpO_2.

Subsequently, his airway was partially obstructed during manipulation of the TEE probe. Bilateral jaw thrust was immediately applied (Fig. 2) and nasal CPAP was increased to 16-18 cm H_2O. He maintained spontaneous nasal ventilation and 99-100% SpO_2 throughout the procedure (Fig. 3).

He was awake, and alert soon following withdrawing of the TEE probe and was transported back to the SICU without any complications.

Six months later, the patient presented for follow-up TEE under deep propofol sedation. The same anesthesia team was surprised that he had lost 58 lbs and was able to maintain patent airway and spontaneous ventilation and 100% SpO_2 with nasal mask-face tent (5-8 cm H_2O CPAP) and without the need of jaw thrust (Fig. 4-5).

Results: Not Applicable - Medically Challenging Case Report

Conclusions: This simple low-flow nasal mask-face tent maintained spontaneous CPAP ventilation/oxygenation in a high risk super obese patient with severe OSA during TEE under MAC at NORA. It also reduced aerosol/droplet spread during the aerosol generating procedure.

Amid the ongoing COVID-19 pandemic, this technique may improve patient safety and provide additional provider protection at no extra cost.

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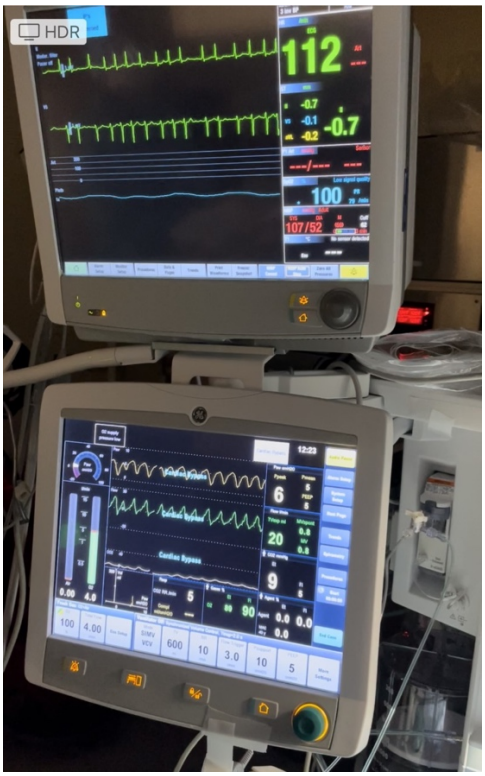
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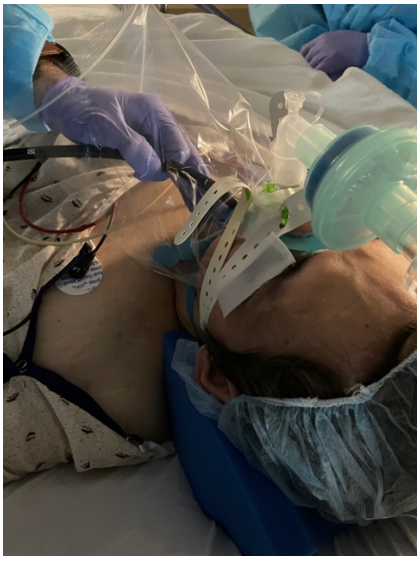

Obstetric Anesthesiology

Obstetric Anesthesiology 1 - Amniotic Fluid Embolism: Post-Operative Perils

Jessica Richelieu[1]

Rush University Medical Center[1]

Introduction: Amniotic fluid embolism remains one of the most dangerous complications of delivery with the potential for devastating sequalae generally characterized by either cardiopulmonary collapse, DIC, or a combination of the two. These cases bring an unusual patient demographic to ICUs and the service of the critical care anesthesiologist.

Methods: A 33-year-old patient G7p3124 at 29w0d with placenta previa presented to OB triage with her third episode of vaginal bleeding. Her history was significant for a c-section during her first pregnancy secondary to eclampsia with three subsequent successful VBACs. The decision was made to proceed with repeat c-section for delivery. Patient underwent low transverse c-section and after placental delivery began having signs of altered mental status, episodes of ventricular tachycardia and hypotension to 80s/40s. Patient was subsequently maxed out on a phenylephrine infusion at 250 mcg/kg/min and ultimately required IV epinephrine to maintain adequate blood pressures. Throughout this time, she became hypoxic and increasingly unresponsive requiring transition from neuraxial to general anesthesia for airway protection and further stabilization. No signs of bleeding were noted during the case. Post-procedure she was transferred to the surgical ICU for suspected amniotic fluid embolism. Echo obtained in the unit demonstrated right heart strain consistent with this diagnosis and CTPE was negative. Patient's course was further complicated when labs drawn upon SICU admission were consistent with DIC – Hgb 11.6, platelets 94 (from 235), fibrinogen 54 (from 514). and newly prolonged PT, PTT, INR 1.76 (from 0.93). Patient's lactic acid was 5.9 and LDH 684. Labs two hours later demonstrated Hgb 8.1, platelets of 142 post-transfusion, INR 3.36, and fibrinogen < 50. She received a total of 9 units PRBCs, 5 units of cryoprecipitate, 4 units of platelets, and 6 units of FFP for treatment along with 24 hours of Pitocin, two doses of TXA and foley balloon tamponade applied to the uterus. Patient's labs subsequently normalized, she was extubated on post-op day 1 and transferred back to the labor and delivery floor on post-op day 2.

Results: Not applicable - medically challenging case report

Conclusions: This case illustrates the complications following amniotic fluid embolisms and the necessity of ICU level of care for these patients. In this case, the patient recovered unusually quickly in the setting of such rapid decline seen both intraoperatively and upon ICU admission. In the event of suspected AFE it is imperative to escalate to ICU level care as quickly as possible and initiate a thorough workup to evaluate for further complications including coagulopathy and change in cardiac function to optimize outcomes.

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Obstetric Anesthesiology 2 - Emergent Cesarean Delivery in a Patient with Sickle Cell Disease; Anesthetic Considerations

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Introduction: Sickle cell disease (SCD) is a hereditary hematologic disorder in which hypoxic stress leads to sickling of red blood cells[1]. Further hemolysis and vaso-occlusion lead to repeated microvascular occlusion, which can result in acute pain crises and acute chest syndrome[1]. Pregnancy in women with SCD is associated with increased maternal and fetal complications including preeclampsia and preterm delivery[1-2].

Methods: A 42-year-old female with a history of SCD and preeclampsia with severe features presented emergently at 33w1d with acute chest syndrome and underwent a cesarean delivery due to recurrent late fetal decelerations. Her preoperative hemoglobin (Hb) was 7.2 g/dL. The patient also had pneumonia requiring supplemental oxygen. General anesthesia (GA) with rapid sequence induction was performed. Following uterine incision, the patient became hemodynamically unstable, and blood transfusion was initiated. A radial arterial line was obtained for continuous blood pressure monitoring and frequent laboratory drawings. Intraoperative Hb and lactate were 6.6 g/dL and 4.3 mmol/L respectively. Transthoracic echocardiography of the heart showed an underfilled left ventricle (fig.1). The patient received multiple blood products which improved her intraoperative laboratory findings. Given the patient's increased preoperative oxygen requirements, the considerable amount of blood products and fluids she received, and the known physiologic airway changes that occur with pregnancy, the decision was made to keep the patient intubated. The patient was transferred to the intensive care unit where she was optimized and extubated.

Prophylactic transfusion of packed red blood cells has been controversial. However, there are various trials showing that preoperative transfusion decreases perioperative complications, including acute chest syndrome in pregnant patients with SCD[2-3]. An early neuraxial block should always be considered as pain can also trigger acute chest syndrome. GA is discouraged due to the increased risk of blood loss and sickling complications[2]. In the event of GA, total intravenous anesthesia with propofol can minimize the risk for uterine atony. When making the decision to extubate in the operating room, multiple factors must be considered such as preoperative supplemental oxygen requirements, the volume of fluid and products received, and the physiologic changes of pregnancy such as increased vascularity and edema of the upper airway, especially in the setting of preeclampsia.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Physicians should be familiar with anesthetic management of parturients with SCD, as maternal death occurs six times more frequently and fetal death is more than double that of women without SCD[2]. Preoperative optimization of any condition that could lead to hypoxia, hypercarbia, hypothermia, acidemia, and pain is imperative to lower the risk of sickling and reduce perioperative complications.

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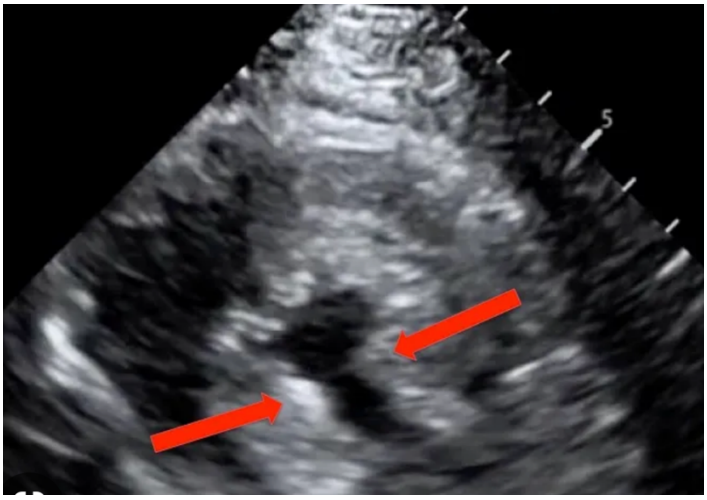

Obstetric Anesthesiology 3 - Flurona during the third trimester! Challenging care during the SARS-CoV-2 pandemic

Arjun Alva, Robert Premkumar, Amrutha Haridasan, Kumar Belani[1]

University Of Minnesota[1]

Introduction: During pregnancy infection with the SARS-CoV-2 virus is high risk and associated with an increase in maternal morbidity, preterm birth and venous thromboembolism[1]. It is expected that a dual concomitant additional viral infection (flurona)[2] will certainly be challenging for both the patient and caregivers. In this report, we describe our unique experience with a 38-year-old, BMI 30.1 kg/m2, gravida 4, para 1, who presented with severe respiratory illness at 27 weeks of gestation
o.

Methods: A 38-year-old female, BMI 30.1 kg/m2, gravida 4, para 1 had received the second dose of COVID 19 vaccine 3 months prior to this illness. She presented to our hospital with severe respiratory illness at 27 weeks of gestation. Non-invasive ventilation was initiated because of hypoxemia with significant respiratory distress. There was bilateral perihilar and basal consolidation by chest CT and a transthoracic echo (TTE) revealed a TAPSE of 17 mm, EF of 55%, pulmonary systolic pressure of 64 mmHg and IVC of 17 mm (<50% collapsible). The point of care ultrasound exam (POCUS) revealed bilateral basal consolidation with > 3 b lines. Viral studies were positive for both COVID-19 and H1N1 infections. Because of her pregnancy, flurona, and serious systemic illness a multidisciplinary team (MDT) approach to care was implemented. The obstetricians tracked fetal wellness (fetal heart rates ranged between 110-160 bpm, and no fetal distress was evident). The figure displays her progress during her care at our institution. A combination of pharmacotherapy, invasive respiratory and cardiac support with prone and supine positioning along with appropriate uterine displacement was required for her management. The MDT entertained the need for VV ECMO if necessary. At week 28 of her gestation, she developed a generalized clonic-tonic seizure suggesting eclampsia, and emergency lower segment cesarean section (LSCS) was done while she was receiving significant respiratory and pharmacodynamic support. A live 1.3 kg female was delivered. Post-delivery, her condition improved but a tracheostomy was necessary for ongoing respiratory support. On day 28 she was decannulated and a few days later she was discharged home with a live and active baby.

Conclusions: This occurrence of flurona during the third trimester is a rare event and has not been previously reported. Aggressive care with an MDT was necessary and resulted in a successful outcome for both the mother and the baby. In our case and as has been reported earlier the baby was protected from both viral infections because of a lack of maternal-fetal transmission [3].

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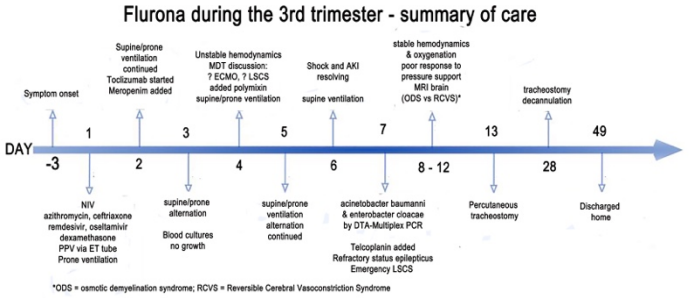

Obstetric Anesthesiology 4 - Perioperative considerations of postural orthostatic tachycardia syndrome in a parturient with implanted spinal cord stimulator device undergoing obstetric surgery

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Introduction: Postural orthostatic tachycardia syndrome (POTS) is an autonomic condition characterized by a marked increase in heart rate of at least 30 beats per minute or a rate that exceeds 120 beats per minute that occurs within 10 minutes of standing or head-up tilt without accompanied orthostatic hypotension (1). The clinical course of POTS in pregnancy is variable and has not been shown to contribute to perinatal morbidity or mortality (1). The etiology remains unclear, although proposed mechanisms involve failure of peripheral vasculature to vasoconstrict in response to a decline in blood pressure, resulting in compensatory tachycardia (2, 3). During the peripartum period there is potential to trigger tachycardia, syncope and other dysautonomic symptoms due to sympathetic activation from the pain and stress of labour. This can be worsened by peripheral vasodilation and hypotension resulting from neuraxial anesthesia. Our patient's history was further complicated by the presence of a sacral neuromodulation system implanted at S3 for the treatment of refractory overactive bladder. We present a case report discussing the anesthetic implications of these conditions on the perioperative management of a parturient undergoing an elective cesarean section (CS).

Methods: A 23-year-old G1P0 parturient presented at 38 weeks gestational age for an elective CS. Her past medical history was significant for obesity, a pre-existing diagnosis of POTS based on recurrent episodes of orthostatic syncope, and refractory neurogenic bladder which was managed with a sacral spinal cord stimulator (SCS). During the early stages of pregnancy, she presented to the Emergency Department several times for worsening syncope and tachycardia which were treated with intravenous fluids and beta blockers. Initial therapies included midodrine and fludrocortisone which were discontinued at the onset of pregnancy. She was then started on propranolol 10mg three times a day and 1L normal saline infusions daily, which were self-administered via a peripherally inserted central catheter (PICC). The stimulator was inactivated at the onset of pregnancy due to its unknown potential risks to the developing fetus (4). Operative reports and old radiographs were reviewed to determine the anatomy of lead placement, wires, and pulse generator. After discussion with Obstetrics, Cardiology and Anesthesia, the patient elected to proceed with an elective CS to avoid worsening of symptoms during labour. On the day of surgery, the patient was brought to the operating room and standard monitors applied. An arterial line was inserted for invasive blood pressure monitoring. After volume loading with 1L of lactated Ringer's solution, an epidural catheter was placed at the L1-2 interspace in the left lateral position and gradually bolused with fentanyl 75mcg followed by 15ml of lidocaine 2% with 1:200000 epinephrine in 5ml increments. The patient remained hemodynamically stable, and the case proceeded without any complications. She remained comfortable in the recovery room with no evidence of tachyarrhythmias, though she did experience episodes of hypotension which resolved with fluid boluses. The rest of her postoperative course was uneventful, and she was discharged home on post-operative day 4. Her SCS remained inactivated at time of discharge, and she was advised to follow up with her urologist for interrogation postpartum.
Results: Not Applicable – Medically Challenging Case Report

Conclusions: This case demonstrates the importance of meticulous preoperative planning and optimization. An elective CS was performed to attenuate the stress of labour and associated exacerbation of symptoms. We describe the successful implementation of neuraxial anesthesia with a slowly titrated epidural and adequate fluid preloading to diminish the sympathetic surge associated with neuraxial induced hypotension. Early anesthetic assessment was crucial and provided adequate time for review of prior records to identify the location of SCS leads and wires. This allowed for safe establishment of an epidural above the level of the implant, minimizing the chances of lead migration or damage (5, 6). An understanding of the anesthetic implications of these conditions is imperative for successful peripartum management.

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Obstetric Anesthesiology 5 - Postpartum Aortic Dissection

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Introduction: A 44-year-old woman at 38 weeks gestation was admitted for repeat cesarean delivery after presenting with vaginal bleeding and contractions. She had a history of migraines, hypertension, and one prior cesarean delivery for umbilical cord prolapse. Her current pregnancy was notable for vaginal bleeding in the first trimester. Ten days prior to admission she presented with chest pain, palpitations, and blurry vision. Her digital watch recorded her heart rate in the 160s prior to arrival and she had mild hypertension. Her EKG was unremarkable, and labs were reassuring against pre-eclampsia or cardiac ischemia. Her symptoms gradually resolved, and she was discharged home.

At the time of her labor presentation, her vitals were notable for blood pressure in the 150/60s. She denied a headache, epigastric pain, or vision changes. Combined spinal and epidural anesthesia was administered and she delivered a healthy infant after uncomplicated repeat low-transverse cesarean section. Post-operatively, she had shortness of breath, mild hypoxemia with saturations of 93-94% on 2-3L by nasal cannula and ongoing hypertension. She denied chest pain or palpitations. Labs demonstrated a NT-proBNP of 3100 with an unremarkable CBC, BMP, EKG, and chest x-ray. Cardiology consultants noted an elevated JVP and 3/4 diastolic murmur at the left lower sternal border. Bedside TTE noted an aortic root dissection and CTA demonstrated a type A aortic dissection originating at the proximal right coronary artery ostium and extending to the aortic arch. Her scan also demonstrated a dilated left ventricle, pulmonary edema with right greater than left pleural effusions and visceral evidence of arteriopathy. Her interview with cardiology revealed that she had an uncle with an “aortic problem” that was ultimately managed surgically.

She underwent emergent aortic arch replacement and aortic valve repair, where operative findings included a large serous pericardial effusion and small, thin arteries. She was extubated four hours after her surgery and left the ICU the next morning. Notably, she did not have postpartum bleeding despite full heparinization for cardiopulmonary bypass 12 hours after her cesarean delivery. Her post-operative course was notable for episodes of rapid atrial flutter that were medically managed. She is recovering well and further work-up of an arteriopathy is pending.

Methods: Cardiovascular disease contributes to 26.5% of maternal deaths in the United States (1, 2). Aortic dissection occurs in only 0.4 per 100,000 pregnancies but is nonetheless significant given a pre-hospital mortality rate as high as 53% (3, 4, 5). The majority occur in the third trimester and early post-partum period, with Stanford type A dissections more common than type B (5, 6). While risk factors include age, hypertension, pre-eclampsia, congenital heart disease and most significantly, connective tissue disease (OR 4,960) (7), half of cases have no predisposing risk factor other than pregnancy (OR 25 for women under 40) (5). The aorta becomes larger and more compliant throughout normal pregnancy, and hormonal changes induce histological changes in the structure of the aortic wall (8, 9). Additionally, the contribution of hemodynamic stress may explain why dissection occurs most frequently near the end of pregnancy, when cardiac output and blood volumes are highest (1).

Prompt investigation of acute chest or back pain should include aortic imaging by CT, MRI or echocardiography. For our patient, bedside echocardiography, prompted by mild post-partum hypoxemia without chest or back pain, demonstrated dissection at the aortic root and aided in timely mobilization of resources. Whether her episode of chest pain ten days prior to labor onset was related to her aortic pathology remains unknown. Patients with type A dissection require urgent surgery, with concurrent cesarean delivery if the fetus is viable and has not yet been delivered. Our patient underwent aortic arch repair approximately twelve hours after her cesarian section and has fortunately recovered well.

Conclusions: Pregnancy is increasingly recognized as an independent risk factor for aortic dissection (7) and mortality is associated with a delay in diagnosis (10). We present this case to emphasize the importance of prompt diagnosis and to demonstrate an example of full heparinization for aortic repair shortly after cesarean delivery without significant postpartum bleeding.

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Pain Medicine

Pain Medicine 1 - Botulinum Toxin Injections into Scalene Muscles Provide Prolonged Relief from Thoracic Outlet Syndrome Stefanie Pena, Konstantinos Sarantopoulos, Mitzy Torres-Loaisiga

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Introduction: Between the anterior and middle scalene muscles above the first rib, there is an anatomical area in the lower neck identified as the thoracic outlet. Several causes including injury and/or spasm of scalene muscles can lead to compression of neurovascular structures that supply the upper limb leading to what is known as Thoracic Outlet Syndrome (TOS). Thoracic outlet syndrome manifests with complex intractable symptoms affecting upper extremities including pain and motor dysfunction. Symptoms can be debilitating and frequently require extensive surgery for relief which is risky. We report the successful management of a patient with chronic TOS affecting his right upper extremity with repeated Botulinum Toxin injections into his scalene muscles as an alternative for surgery.

Methods: A 41-year-old male suffered electrocution injury twice within 24 hours about seven years ago with resultant neurogenic TOS and complained of pain radiating through the anterior portion of his arm to his right hand. He complained of accompanied burning pain radiating all the way to his right-hand including stiffness, dysmotility and discoloration with subjective weakness. Patient also complained of pain and stiffness in right sided pectoralis muscles and result in restriction of the abduction movements of his right arm. The patient initially received right anterior and middle scalene muscle local anesthetic injections under ultrasound and fluoroscopic guidance for diagnostic evaluation regarding the TOS on 8/24/2020 and had transient but significant relief of his symptoms consistent with a diagnosis of neurogenic TOS. Risks associated with surgery that included nerve damage, vessel injury, pneumothorax and persistent pain were discussed with the patient with vascular surgery. Patient wished to attempt Botulinum Toxin injections prior to deciding on surgery at that time. He was then treated with a series of repeated Botulinum Toxin injections into his right anterior and middle scalene muscles and trigger point injections (TPI) into tight pectoralis muscles with excellent results that lasted for four months each time. We have been scheduling the patient to repeat both Botulinum Toxin and TPI every four months with excellent pain relief.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: TOS is a rare condition caused by compression of the neurovascular structures of the thoracic outlet. Symptoms can sometimes be incited by a neck injury, especially in patients with abnormal cervical anatomy, or, in this case, electrical injury. This case report highlights a unique patient with a history of electrocution injury while welding with resultant neurogenic TOS who presented with complicated symptoms who required a series of surgical interventions to improve. The patient wanted to try alternative treatment options prior to attempting surgery and attempted Botulinum Toxin and Trigger point therapy every three to four months with excellent results and no need for surgery.
The surgery complications for TOS may be severe, risks including nerve damage, vessel injury, pneumothorax, and persistent pain. Due to potential risks associated with undergoing surgery, conservatism in the treatment has been encouraged in recent years. According to recent critical studies, good results after surgery for TOS are achieved in less than 40% of all cases.2 Even the use of firm, stated operational criteria yields no more than 28% of symptom-free patients and the reported recurrence rate can be as high as 20%, with cases often ending in litigation.2 On the other hand, botulinum toxin offers an attractive alternative by alleviating the symptoms as an alternative to surgery. Apparently, Botulinum Toxin produces muscle relaxation and decreases spasticity and dystonia by blocking presynaptic acetylcholine release.3 Our results are consistent with other published studies.1

In conclusion, TOS is a rare condition caused by compression of the neurovascular structures of the thoracic outlet. Once identified, treatment thrives on a multidisciplinary approach to help manage symptoms. A series of surgical interventions including surgical decompression is usually recommended for these patients in conjunction with the help of a supportive physical therapist to reinforce strengthening and the importance of a professionally constructed home exercise regimen. Alternatively, due to potential surgical risks, in our pain medicine practice, we have seen excellent success with scalene muscle blocks.

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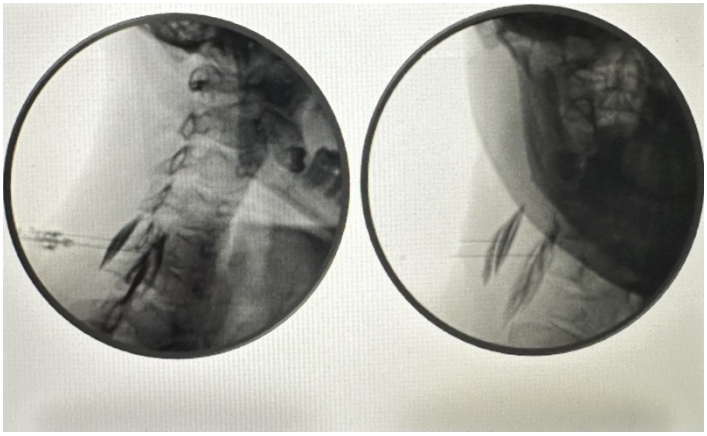

Pain Medicine 2 - New Minimally Invasive Cervical Discectomy Relieves Chronic Neck Pain

Wael Elkholy, Danielle Levin[1], Mouhammad Ayoubi, Salma Elkholy, Michael Morales, Ashraf Sakr

St. Elizabeth's Medical Center[1]

Introduction: Cervical disc herniation is a common pathology that may cause significant disability to individuals. When this condition provokes disabling root compression symptoms unresponsive to conservative treatment, patients have the option to undergo operative decompression of the root via various surgical techniques. Over the last several decades, cervical discectomy with fusion has become one of the most common cervical spine operations. Although it may help a number of patients, this surgery is typically performed through about a five-centimeter incision and results in permanent limitation of normal neck motion postoperatively, which can lead to difficulties, such as pseudoarthrosis and adjacent segment disease [1-2]. We would like to share a novel and successful minimally invasive technique for anterior cervical discectomy without fusion performed by pain management physicians.

Methods: A 33-year-old female presented with severe neck pain that started two years prior to presentation after a trauma – patient came into her home, slipped on wet floor, and lost consciousness. At the hospital, the patient was evaluated and discharged the same day. However, since then, the patient had been experiencing this severe neck pain, which she rated 7-8 out of 10 on the Numeric Rating Scale. Associated with the neck pain, the patient had pain radiating down her upper extremities, which she described as tingling and numbness in her fingers. On examination, Spurling test was positive bilaterally, decreased upper extremity reflexes were present bilaterally, and there was decreased sensation over the right C6 and C7 dermatomal distribution. MRI demonstrated slight reversal of normal cervical lordosis, annular bulging with thecal sac compression at C2-3 and C3-4 levels, and central subligamentous disc herniation with thecal sac compression at C4-5, C5-6, C6-7 levels. EMG demonstrated bilateral C7 radiculopathy. Pain management with meloxicam, cyclobenzaprine, acetaminophen, home exercises, and chiropractor therapy, provided the patient minimal relief. Repeat cervical epidural steroid injections provided 80% radicular pain relief, but the pain gradually returned with each injection.

After failed conservative treatment and failed cervical epidural steroid injections, which provided only partial and short period of time relief, the patient was offered a novel minimally invasive anterior cervical discectomy approach without fusion at the C6-C7 level.

Under general anesthesia with endotracheal tube and orgogastric tube, with guidance of fluoroscopy and EMG monitoring, approximately 1gm of nucleus pulposus was removed and decompression of the affected disc in 4 different positions was performed, with just a one-centimeter incision. At the three-month follow-up, the patient reported significant neck pain relief. She rated the pain as 2 out of 10 on the Numeric Rating Scale and described it as mild and occasionally stiff in nature. Furthermore, she experienced 70% radicular pain relief as well. As a result of this procedure, the patient had significant improvement in her mood and activities of daily life.

Results: Not Applicable – Medically Challenging Case Report
Conclusions: Cervical discectomy with fusion is a common surgery, but it is not void of complications. This case report demonstrates a novel, minimally invasive surgery without cervical fusion that could be an effective option for those afflicted with debilitating cervical disc herniation.

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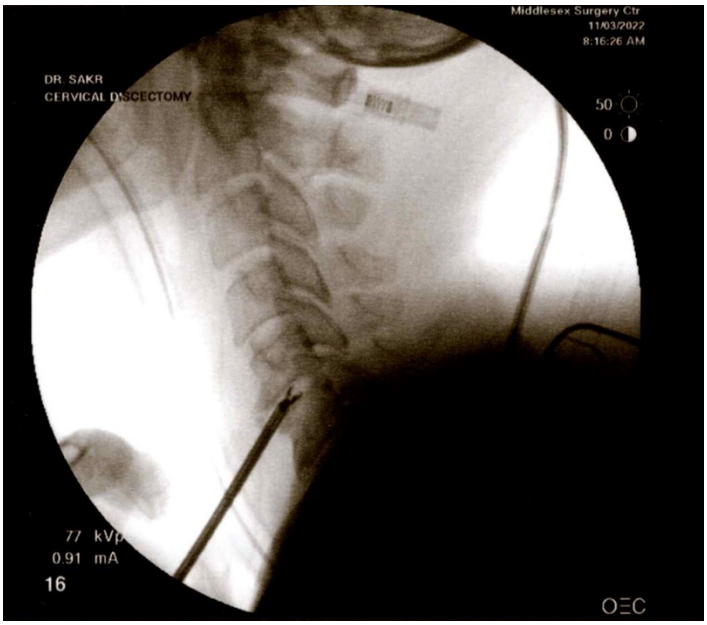

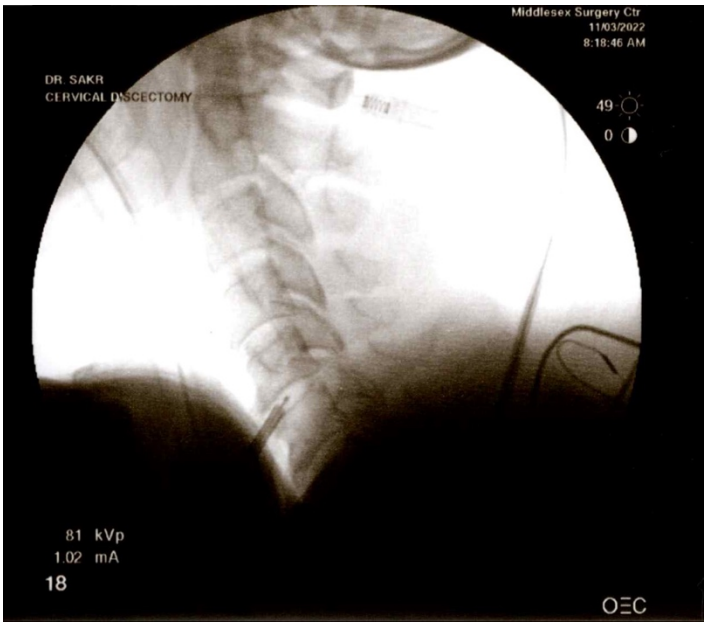
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Pain Medicine 3 - Pain Service Using Analgesic of Dose Ketamine for Treatment of Refractory Seizures

Vraj Patel[1]

George Washington University[1]

Introduction: Objective: Discuss an uncommon use of Ketamine

Background: Ketamine is a unique IV anesthetic that has been in clinical use since 1970 and is able to provide a wide range of pharmacologic effects including bronchodilation, sympathetic nervous system stimulation, sedation, analgesia. Although the drug has been known to the scientific community for more than half-century, there are new applications of the drug that are continuously being discovered. Erdogen et. al published a paper in 2012 showing ketamine can be used as a strategy to enhance seizure quality and clinical efficiency of ECT. However, there are also some instances where high doses of ketamine (2.5mg/kg/hr) have been used to control refractory seizures (Ayham et al 2020). We present a case where we use a much lower dose of ketamine that is often used for analgesia to decrease refractory seizures in a patient who was experiences up to 20 seizures an hour.

Methods: Case Report: Patient was a 43-year-old male with medically refractory bilateral temporal lobe epilepsy with neuropacer placement. He reported having subsequent long term memory impairment with increased frequency of seizures in the few days prior to arrival to the hospital. He noted have approximately 20 seizures per hour that were recorded by his neuropacer. He described having focal seizures involving blank stares despite being compliant on home medications. His regimen of medications for home included phenobarbital 64.8 (one in the morning and two at nighttime), cenobamate 400 mg, levetiracetam 750 mg (two in the morning and two in the nighttime). He was diagnosed with refractory status epilepticus in 2012 after viral encephalitis. At the time, patient had an MRI/PET scan which revealed bilateral temporal lobe vasculitis.

Patient presented to the hospital with increased number of seizures and mentioned that he had obtained reprieve from his seizures in the past by receiving a high dose of ketamine at 1 mg/kg/hr. The primary team consulted the acute pain service to evaluate for intervention with ketamine. After thorough discussion with patient, he was started on a low dose ketamine infusion of 15 mg/hr which decreased the patient's hourly focal seizures to 0-5 per hour. The infusion was discontinued after 12 hours when patient was able to go six hours without any seizures.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: There are multiple case reports of patients receiving high dose ketamine in the magnitude of 2 mg/kg/hr for several hours in treatment of refractory status epilepticus. In this patient, we were able to reduce the number of focal seizures while using a significantly lower dose of ketamine. Using a dose of ketamine that is significantly lower than full anesthesia reduces the risk of complications such as transient respiratory depression, increases tracheal secretions, laryngospasms, and need for intubation. Further studies are warranted to fully explore the effect of low dose ketamine on refractory seizures.

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Pain Medicine 4 - The Use of Radiofrequency Ablations (RFA) in The Treatment of Upper Extremity Pain and Spasticity

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Introduction: Radiofrequency Ablations (RFAs) are non-surgical, minimally invasive procedures that use heat to reduce or stop the transmission of pain. RFAs are commonly used to treat patients with chronic pain and conditions such as spondylosis and sacroiliitis.[1]

The use of RFAs for pain is based on the premise that the transmission of currents near nociceptive pathways interrupt pain impulses.[1]The thermal energy associated with the procedures leads to tissue destruction targeted at the nerves responsible for transmitting the sensation of pain.
RFAs are generally targeted at strictly sensory nerves so motor function can be preserved. After a literature search, we did not see many examples of RFAs being used for the intentional ablation of motor nerves. Thus, we present a case of the use of a Brachial Plexus RFA, targeted at the interscalene level, to alleviate the symptoms of spasticity in the upper extremity.

Methods: Patient is a 29M who was born with Cerebral Palsy and is in full care for ADL's along with a tracheostomy and PEG tube. Patient initially presented with concerns of left shoulder pain and spasticity of the upper extremity that led to left arm abduction and lateral extension. Parents were concerned about the patient accidentally pulling out his GJ tube; prior to presentation he had multiple ED/hospital visits for dislodged GJ tubes. He previously had extensive treatments to address the issue including surgical neurotomy and Botox injections.

On 8/16/22 the patient underwent a Brachial Plexus RFA, targeted at the interscalene level, to help alleviate his symptoms. We utilized the RF needle to help identify the specific motor response we were looking to eliminate. The brachial plexus was identified in the supraclavicular fossa and traced up to the interscalene level. The RFA needle was directed towards the brachial plexus and once it was placed in the proper location; the generator was used to provide motor stimulation to identify the trunk of the plexus responsible for the deltoid (C5). We targeted this trunk to prevent shoulder abduction via the deltoid muscle. Once the proper motor response was identified, the ablation was performed. In his 4 week follow up, the patient's parents endorsed that he had a significant reduction in spastic episodes of his extremity along with significant pain improvement, thus indicating that the procedure was successful
Prior to this we had attempted a Left Axillary Nerve RFA which provided symptomatic relief for only one week. We then attempted a combined Left Axillary Nerve and Suprascapular RFA in hopes of targeting some motor nerves as well. However, the spasticity returned after 2 weeks. It became apparent that the movement at the shoulder (abduction), elbow (flexion and extension) along with the use of the biceps and triceps was leading to this pulling behavior. At that time, we decided to proceed with another RFA of the brachial plexus, this time at the interscalene level to find a more proximal target for the procedure.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: The brachial plexus arises from the C5-C8 and T1 cervical nerve roots. Two nerves affected in this block that stick out are the Musculocutaneous (C5-C7) and Radial (C5-T1) nerves. These nerves innervate the flexor and extensor muscles of the arm and forearm respectively.[2] The axillary nerve block is also reliably blocked, and it innervates the deltoid and teres minor muscles along with the skin of the shoulder. Given our patient's presenting symptoms of spasticity of the upper extremity, notably at the elbow, and shoulder pain, we made the decision to do an RFA at this level.

Brachial Plexus blocks have been used in a variety of procedures involving the upper limb and shoulder to help alleviate acute pain. In that sense, radiofrequency ablations of the brachial plexus at various levels can be a useful method to treat conditions such as spasticity and chronic pain as well. However, their actual clinical significance is still somewhat unclear. This warrants further studies and trials to see if these results can be applicable to a larger patient population.

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Patient Blood Management

Patient Blood Management 1 - Perioperative Management of Pediatric Patients Undergoing Juvenile Angiofibroma Resection. An Educational Review Highlighting Patient Blood Management

Susan Goobie[1], Mckenna Longacre[2], Samir Seshadri[2]

Boston Children's Hospital, Harvard Medical School[1] Boston Children's Hospital[2]

Introduction: Juvenile nasopharyngeal angiofibromas (JNA) are rare, benign, highly vascular tumors with a propensity towards aggressive local invasion (Figure). Surgical resection was typically associated with massive blood loss; historically as high as 3500 mL (mean 1100 mL, range 300-3500 mL)[1]. Management employing multimodal blood conservation strategies is now an essential standard for perioperative care. These surgeries, once synonymous with massive transfusion, may potentially be performed without autologous transfusion, or deliberate hypotension. Described is a contemporary approach in the management of 3 patients with high grade JNA tumors performed without autologous transfusion employing patient blood management (PBM) techniques[2,3].

Methods: Three otherwise healthy patients with JNA had robust preoperative hematocrits and normal coagulation profile. Surgical and anesthesia discussion included blood loss risk, PBM techniques (antifibrinolytics and acute normovolemic hemodilution (ANH)) and possible blood transfusion. Informed consent for research dissemination and de-identified publication was obtained from families and assent from each teenage patient. Local IRB approval was waived per standard policy.

JNA resection was preceded by successful interventional radiology trans arterial embolization of the tumor's accessible vascular supply. Surgical resection was the following day. Patients were induced with propofol and rocuronium, orally intubated, and maintained with combined narcotic infusion and inhalational technique. Large bore intravenous lines and an arterial line were placed. ANH was performed and modified for pediatric patients based on AABB guidelines.[4] Autologous whole blood was removed (volume of <20% total blood volume with a target post Hb of 9 g/dL), replaced 1:1 with 5% albumin, and stored in a designated collection bag containing the anticoagulant Citrate Dextrose solution. Tranexamic acid (TXA); loading dose of 30 mg/kg and infusion of 10 mg/kg/h was given. Hemodynamic stability was maintained. Autologous whole blood was returned after surgical resection. Two patients were transported extubated to the PACU and one to the ICU due to length and complexity of surgery. Ondansetron and decadron were given as prophylaxis for postoperative nausea, vomiting and swelling. Pain and/or agitation was managed with acetaminophen, sufentanil, dexmedetomidine and hydromorphone/morphine (see Table).

Conclusions: Patients presenting for JNA surgeries present a unique opportunity to harness PBM, as they tend to present as semi-elective procedures (with time for perioperative planning) in otherwise robust patients (healthy adolescent males). Over the past decade, pediatric PBM has evolved to include additional evidence-based tools, including preoperative anemia management, antifibrinolytics, cell salvage, and physiologically driven individualized goal-directed transfusion algorithms. In addition, further recognition of the risks, costs, and stewardship associated with allogeneic blood transfusion has led to a call for action for healthcare providers and entities to widely implement PBM[5].

We describe the successful use of normal hemodynamic goals, antifibrinolytic therapy, ANH, and early extubation in the care of adolescent males with highly invasive tumors. New surgical and anesthetic strategies have yielded a significant decrease in blood loss and eliminated the need for transfusion of autologous red blood cells, which lead to improved outcomes.

Other PBM strategies that will likely further improve care of patients at high risk for blood loss include preoperative preparation optimizing preoperative hemoglobin with iron and erythropoietin therapy and intraoperatively using viscoelastic testing for hemostatic management. Other evolving tools for continuous PBM monitoring include noninvasive hemoglobin monitoring, somatic end organ near infrared spectrometry (to monitor end organ oxygen supply, demand, and saturation) and continuous base deficit and lacate monitors.[6] Going forward, the standard of care should include a pediatric health care team and multidisciplinary patient-centered approach incorporating multimodal perioperative PBM strategies. "Bloodless surgery", or at least minimizing blood transfusion for patients undergoing JNA resection and other procedures at risk for significant blood loss, can improve overall patient blood health and may improve pediatric perioperative outcomes.[5]

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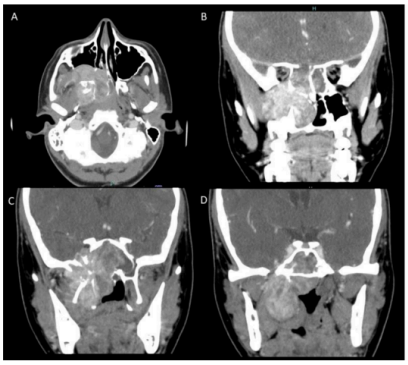
Table. Summary of Intraoperative Management for Juvenile Angiofibroma Resection.

	Procedure	Starting preoperative Hematocrit (Hct) (%)	Autologous Whole Blood Removed via NVH	NVH Replacement	Post Hemodilution Hct	Estimated Blood Loss (EBL)	Final Hct (%)	Surgery Time (hours, minutes)	Weight (kg)	Calculated RBC Conservation*
Case 1 UPMC Stage V	IR Embolization	44.8	n/a	n/a	n/a	25-50 mL	40.6	3h 19m	73.5	n/a
	Extracranial Resection	43.5	750 mL	750mL 5% albumin	38.8	300mL	35.6	9h 2m	73.5	12.6
	Second Stage Resection (Intracranial)	37.6	350 mL	500mL 5% albumin	34.7	600mL	31.8	9h 48m	70.8	18
Case 2 UPMC Stage V	IR Embolization #1	38.5	n/a	n/a	n/a	25-50 mL	39.6	5h 44m	67.8	n/a
	Extracranial Resection	39.6	900mL	900mL 5% albumin	31.8	600 mL	29.5	12h 37m	67.8	46.8
	IR Embolization #2	38	n/a	n/a	n/a	25-50 mL	33.6	3h 20m	65.7	n/a
	Second Stage Resection (Intracranial)	33.6	1,200mL	1,000mL 5% albumin	29	500mL	27.6	8h. 38m	66.0	61
Case 3 UPMC Stage III	IR Embolization	39.2	n/a	n/a	n/a	~25-50 mL	34.6	4h 48m	72.7	n/a
	Extracranial and Intracranial Resection	39.2	300mL	250mL 5% albumin	34.6	100mL	N/A	6 hr. 16 min.	72.7	3.7

*Red Blood Cell (RBC) Conservation Calculation: EBL * (Starting Hct/100) – EBL * (Final Hct/100) = RBC Conservation factor. (If the patient loses blood while their Hct is lower, they therefore lose a lower proportion of RBCs.)

Figure 1. CT Preoperative Imaging of a JNA Patient.



Select images from maxillofacial computerized tomography (CT) scan with contrast (JNA Case 2):

A) Axial image depicting the classic Holman-Miller sign (anterior displacement of the posterior maxillary wall). B) Dumbell appearance to the anterior component of the tumor with large medial component obstructing the right nasal passage and lateral component extending into the inferior orbital fissure and infratemporal fossa. C) Extension of the avidly enhancing tumor through the superior orbital fissure and lateral right sphenoid wall into the middle cranial fossa. D) Large inferior component of tumor extending into the parapharyngeal space and abutting the right tonsil.

Patient Blood Management 2 - Severe hypotension during intraoperative transfusion of autologous cell-salvaged blood in revision lumbar spinal fusion

Alexander Metzger[1], Kamilla Esfahani

University of Virginia[1]

Introduction: Autologous cell salvaged blood transfusion is a commonly used blood conservation strategy in major spine surgery, which is often well-tolerated and reduces the need for perioperative allogenic blood product transfusion (1, 2). In this report we describe an incident of transient, but profound hypotension after initiation of transfusion of cell salvaged blood during an elective revision lumbar fusion in an otherwise healthy 21-year-old female. In this report we describe the details of this intraoperative event, how the causative factor was recognized, and treatment methods used to restore hemodynamic stability. We also discuss hypothesized mechanisms for this reaction and considerations for effective treatment based on review of prior case reports.

Methods: A 21-year-old female with a history of congenital scoliosis and T2-L4 posterior fusion 10 years prior underwent exploration and revision of the previous fusion to the ilium, with intraoperative neuromonitoring. The patient received general anesthesia with an endotracheal tube, standard ASA monitors, additional 16-gauge peripheral IV access and a radial arterial line. Intraoperative cell salvage was used throughout the surgery. Four hours into the operation, we transfused 256ml of autologous cell salvaged RBCs through a fluid warmer. Within five minutes of initiating transfusion, a profound decrease in arterial blood pressure to a nadir of 41/31 (MAP 35) and tachycardia with heart rate up to 118 were recorded. No significant changes in esophageal temperature, ETCO2, SpO2, or peak airway pressures were noted. There were no visible skin changes. The surgeons did not endorse visible acute hemorrhage, and there were no significant changes in neuromonitoring per the surgical neurophysiologist. Blood pressure recovered promptly and remained stable after administering boluses of IV vasopressors, which included 24mcg norepinephrine and 200mcg epinephrine. Arterial blood gas following this event was notable only for a slight uptrend in lactate (1.76 to 2.74 mmol/L). Otherwise, all other lab values including hemoglobin, platelet count, electrolytes, and point of care thromboelastometry results, were within normal limits. Two hours later another episode of precipitous hypotension occurred after an additional 25ml of cell salvaged blood was transfused. At this time the transfusion was discontinued and disconnected from the patient, and the patient's blood pressure improved quickly following an additional 50mcg IV epinephrine bolus. Following this event, the patient had an unremarkable completion of her surgery, emergence, extubation, and postoperative recovery with no further hypotensive events.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Our initial differential diagnosis included hypovolemia/hemorrhagic shock, anaphylaxis, neurogenic shock, DVT/PE, tension pneumothorax, and cardiac tamponade. We suspected the causative factor after the second hypotensive episode given the close temporal association with its use and prompt and sustained recovery after stopping the transfusion. Descriptions from prior case reports closely match the findings in this patient's case, with similar treatment strategies (3, 4). Though data are limited due to the rare incidence of this reaction, some hypotheses for the mechanism of this response include the release of bradykinin through an interaction between platelets and negatively charged leukocyte reduction filters in cell salvage systems, citrate toxicity, or contamination with bacteria or amniotic fluid (5, 6). In summary, acute hypotension from intraoperative transfusion of autologous cell salvaged blood is an exceedingly rare event which requires rapid recognition, removal of the offending agent, and temporary vasopressor support to avoid end-organ injury.

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Patient Safety

Patient Safety 1 - Novel Approach to Catheter Securement for a patient with Epidermolysis Bullosa

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Children's Hospital Colorado[1] University of Colorado/Children's Hospital Colorado[2]

Introduction: Epidermolysis bullosa (EB) is a rare, often genetic, skin disorder and a challenge to the anesthesiologist. In EB patients, blisters and bullae form in response to minor shear forces or friction applied to the skin.[1,2] The injury occurs due to an abnormality in collagen that leads to a loss of intracellular bridges in the dermo-epidermal junction or the basal layer of the epidermis. These patients are often seen by the anesthesiologist for esophageal dilations due to strictures, dental restoration, orthopedic surgery (syndactyly release and amputation), or gastrostomy tube placement due to chronic malnutrition and progressive dysphagia.[1,2] Securement of intravenous (IV) catheters (peripheral and central) and peripheral nerve catheters has historically been challenging. Traditionally adhesive dressings are made with acrylic, and removal of these dressings causes substantial injury to this patient population.

Methods: A 40-year-old male with recessive dystrophic epidermolysis bullosa presented to the operating room for revision amputation of his right arm due to squamous cell carcinoma. A 22-gauge peripheral intravenous line was placed in his left forearm and secured with a Covalon^{R} IV Clear dressing. This dressing is clear, transparent, and occlusive. Most importantly, it is made with silicone-based adhesive, which releases from the skin with water, saline, or spray adhesive remover without causing damage. It is impregnated with chlorhexidine and silver for a dual-antimicrobial effect. The dressing was outlined with Mepitac^{R} tape, which is also made of a silicone-based adhesive, though is not transparent.

The patient was induced with propofol, fentanyl, and lidocaine with maintenance of spontaneous ventilation, and supplemental oxygen was provided. A 6.0 nasotracheal tube was placed in the left nare via fiberoptic bronchoscopy. Additional IV access was obtained by placing a 20-gauge peripheral intravenous line in the left leg. This catheter was also secured with Covalon^{R} IV Clear dressing and outlined with Mepitac^{R} tape as described above. A right infraclavicular catheter was then placed with ultrasound guidance and also secured with Covalon^{R} IV Clear dressing. Anesthesia was maintained with inhaled sevoflurane and a ketamine infusion. There were no noted intraoperative complications, and the patient was extubated awake, recovered in the post anesthesia care unit, and went to the general care floor.

Results: Not applicable – Medically Challenging Case Report

Conclusions: The patient had excellent analgesia while an inpatient and was discharged from the hospital on postoperative day 2. The peripheral intravenous lines were removed prior to hospital discharge. The patient experienced no skin damage from the intravenous lines securement dressings. The patient went home with the interscalene pain catheter in place and successfully removed this catheter at home on postoperative day 3. The acute pain service followed the patient via telephone while at home with the pain catheter in situ. Upon removing the pain catheter, the patient noted no skin damage. This novel use of Covalon^{R} IV Clear dressing in epidermolysis bullosa patients has improved the safety of indwelling catheter placement, decreased chances of dislodgement, infiltration, and infection, and most importantly not caused any additional skin damage.

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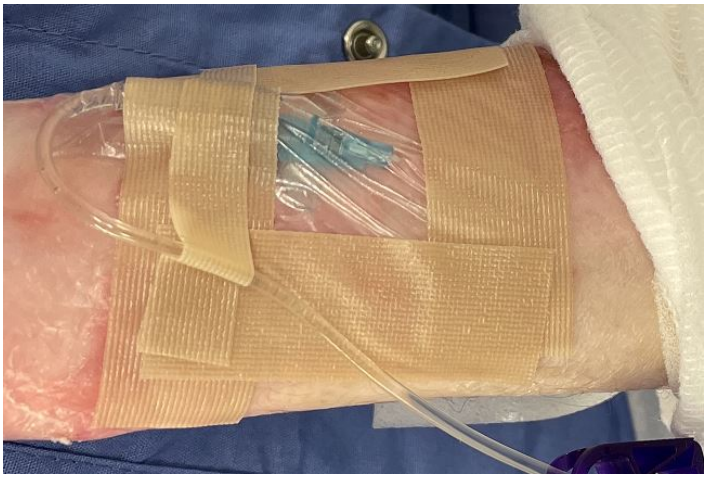

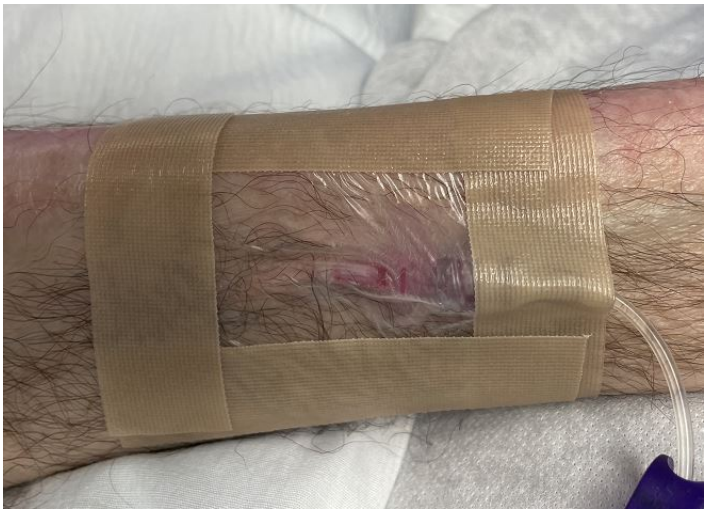

Pediatric Anesthesiology

Pediatric Anesthesiology 1 - Undiagnosed Pediatric Congenital Diaphragmatic Hernia in The Gastroenterology Suite

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University of Chicago[1]

Introduction: Congenital diaphragmatic hernia (CDH) is typically diagnosed in utero or soon after birth. We report a case of CDH, discovered in the perioperative period by the anesthesia care team, in a 10-month-old infant presenting for endoscopy in the gastroenterology suite for severe failure to thrive. With this case, we aim to describe an atypical presentation of CDH that revealed itself through unexpected intraoperative respiratory complications.

Methods: A 10-month-old, ex-full-term boy presented to the gastroenterology suite for esophagogastroduodenoscopy (EGD). He weighed 6.8kg (<1st percentile) and was admitted two days prior for failure to thrive. He was seen a month ago as an outpatient for weight loss, with a history of poor feeding and near daily emesis. He was diagnosed with oromotor dysfunction and had a strong family history of many food allergies.

The patient's preoperative exam was unremarkable other than his small size. He had a peripheral IV in situ and was induced with propofol, dexmedetomidine, and rocuronium. He was intubated easily by direct laryngoscopy with a Miller 1 blade and a 3.5 cuffed endotracheal tube (ETT). Bilateral breath sounds were confirmed, and the cuff was inflated to a minimal occlusion pressure of 20cm H2O. The patient was maintained at 0.8-1.0 minimum alveolar concentration on sevoflurane/air/oxygen and pressure control ventilation (PIP 20cm H2O, RR 22 bpm, PEEP 5cm H2O, FiO2 40%, Vt 60mL). It was unexpectedly challenging for the attending pediatric gastroenterologist, an experienced endoscopist, to manipulate the scope to obtain the necessary biopsies. Fifteen minutes into the case, tidal volume dropped to 20-30mL with little change in EtCO2. The ETT position was confirmed, and the tube was suctioned without evidence of obstruction. It was difficult to auscultate breath sounds with the scope in place. We increased PIP to 22cm H2O with little change in tidal volume. Oxygen saturation remained >97% and the case was completed 10 minutes later. Tidal volumes improved somewhat after the scope was removed. The patient had poor breath sounds during emergence and was given albuterol empirically. Oxygen saturation was 100%. Neuromuscular blockade was fully reversed with sugammadex and he was extubated uneventfully. In the postanesthesia care unit, the patient was tachypneic (RR 43-52 bpm) with room air saturation 96-98% and poor breath sounds, worse on the left. A chest Xray was obtained which showed bowel loops in the left hemithorax. We diagnosed the patient with a left CDH and recommended pediatric surgical consultation.

The patient underwent primary repair of his CDH 4 days later. He was left intubated postoperatively and extubated on POD#2. His EGD biopsies were normal. He had a slowly resolving left pneumothorax, as well as a postoperative fever with a negative infectious workup. He began tolerating feeds with rapid weight gain and was discharged home on POD #10.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The anesthesia care team plays an integral role in the overall course of a patient's clinical care. We report a case of CDH in a 10-month-old infant boy that was diagnosed in the perioperative period. He had intraoperative difficulty with positive pressure ventilation and postoperative respiratory distress. Gastric insufflation during the EGD exacerbated the patient's underlying CDH, resulting in worsened respiratory symptoms. Our initial differential diagnosis for intraoperative low tidal volume included obstructive pathology but diagnostic and therapeutic maneuvers during the procedure were unrevealing, leading us to pursue alternative diagnoses and further testing. It is critically important to have a wide differential for common problems encountered in the perioperative period.

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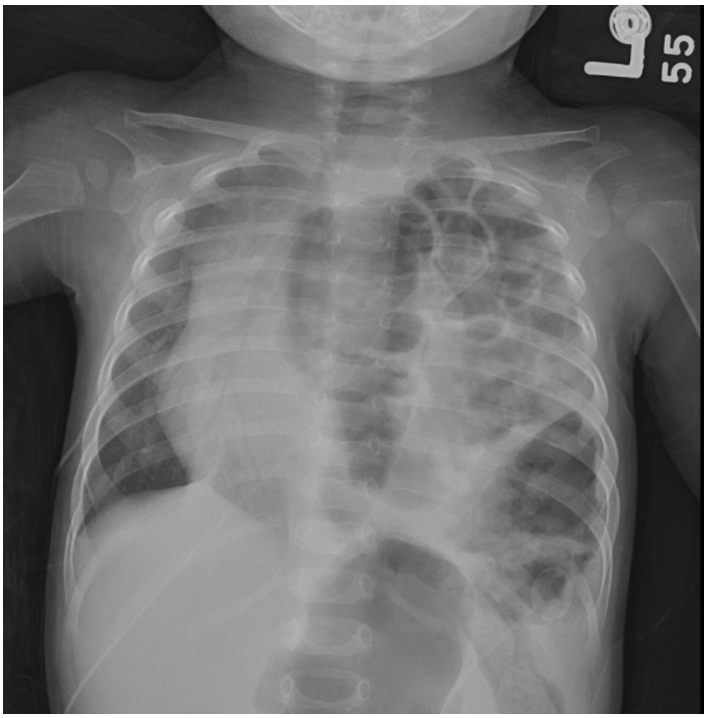

Pediatric Anesthesiology 2 - ECMO and its Role in The Management Of Giant Cervical Teratoma In A Neonate

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University of Arkansas for Medical Sciences[1] Arkansas Children's Hospital[2]

Introduction: We discuss the many challenges and preparations taken to secure an airway in a neonate with a giant congenital cervical teratoma.

Methods: Congenital teratomas are rare germ cell neoplasms and while sacrococcygeal teratomas are more common (1,2), a giant cervical teratoma can potentially be an airway nightmare. While there have been case reports where fetuses with prenatally diagnosed cervical teratomas have been managed with ex-utero intrapartum treatment (EXIT) procedures (1,2), we present a case of giant cervical teratoma that did not cause immediate airway compromise following birth.

A 2-week-old infant with a prenatally diagnosed cervical teratoma presents for a planned excision of the cervical neck mass. A fetal ultrasound demonstrated a left sided neck mass with no airway narrowing. A fetal MRI demonstrated the bounds of tumor without evidence of polyhydramnios. The absence of polyhydramnios was reassuring that obstruction was not present (3). Upon delivery the infant did not experience respiratory distress. A neonatal MRI demonstrated the neck mass in relation to surrounding structures, including a narrowed airway. A multidisciplinary approach was taken to help secure the airway in a controlled fashion to facilitate excision of the cervical mass. This included:

1. Pre-operative ultrasound of the right side of the neck to evaluate size and flow neck vessels, allowing for ECMO planning.

2. ECMO team consult.

3. General Surgery consult for emergent cannulation for ECMO.

4. Blood products available in case of crash ecmo.

General anesthesia was induced with inhaled sevoflurane. There was difficulty with bag mask ventilation initially, which improved with manual displacement of the mass anteriorly and to the left. Laryngoscopy was performed with continuous manual anterior and lateral displacement of the tumor with the neonate breathing spontaneously. Bronchoscopy was performed using a Hopkins rod sheathed with a 3.0 cuffed endotracheal tube, which revealed the subglottis to be compressed eternally from the left with significant narrowing. The trachea was also found to be compressed externally from the left with significant narrowing of the superior half of the trachea. The endotracheal tube was then passed over the rod into the trachea beyond the external compression. After confirming bilateral breath sounds, the endotracheal tube was secured for the remainder of the case. The procedure resulted in successful resection of the tumor; the patient was left intubated and transported to the NICU for further management.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Neonates with congenital cervical teratomas present an airway management challenge to both ENT surgeons and anesthesiologists. Early diagnosis in the antenatal period is possible with fetal ultrasound and MRI, and this is paramount for adequate planning and coordination between management teams. Management options following diagnosis depend on the size of the tumor and the patency of the airway. Larger tumors causing airway compression in utero may require securing an airway with EXIT procedure or operation on placental support (OOPS) procedure (4,5). In our patient, airway was not compromised at birth and having the patient positioned in left lateral position helped keep the airway patent.

The possibility of airway collapse cannot be overstated when taking care of a neonate with a giant cervical teratoma. This is where having an all-hands-on deck approach is important. A multidisciplinary plan was developed in conjunction with the experienced pediatric anesthesiologist, pediatric otolaryngologist, ECMO team and the OR staff. In our case, if intubation were not possible, a surgical airway would also be near impossible due to the location of the cervicofacial teratoma. The OR staff and ECMO team were prepared and on standby if ECMO cannulation was required due to airway compromise and an inability to intubate and/or ventilate. Fortunately for us, we were able to pass the ETT beyond the compressed trachea, and surgical resection was completed without any complications.

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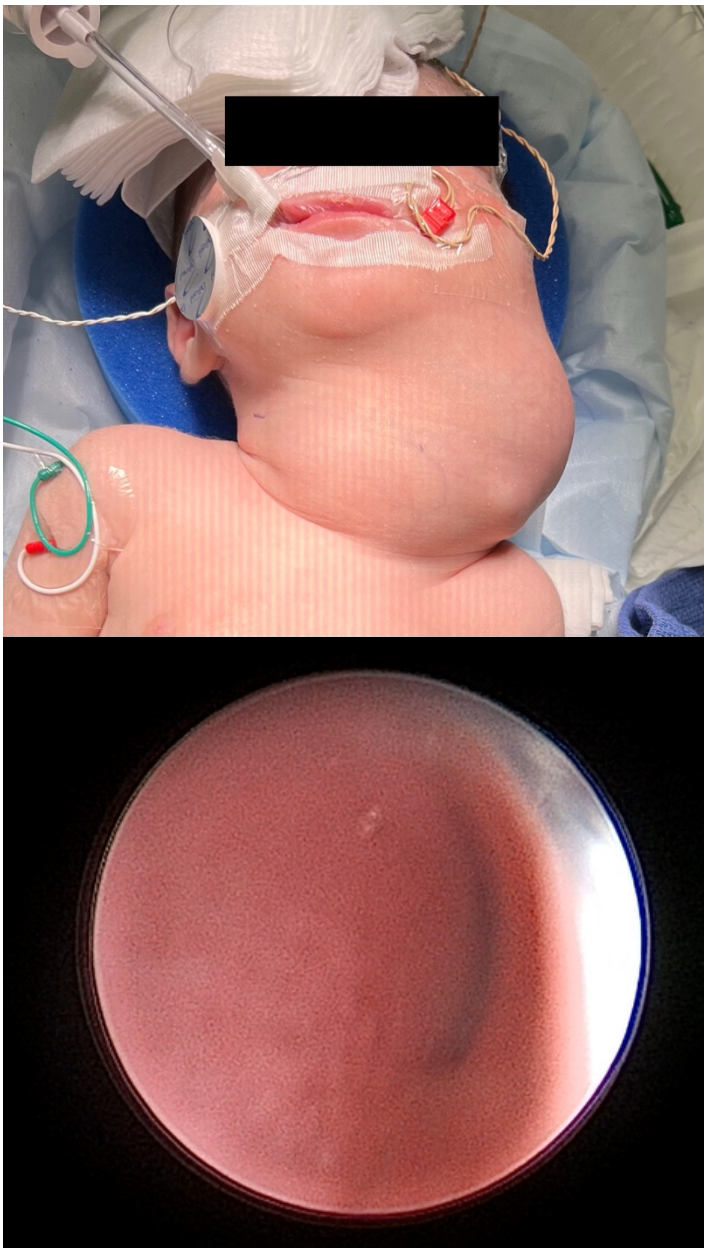

Pediatric Anesthesiology 3 - Underutilized Technique of Fiberoptic Intubation Through Nasopharyngeal Airway

Sam Postma[1], Fernando Alquicira-Macedo[2]

University of Texas Medical Branch[1] UTMB[2]

Introduction: Awake fiberoptic intubation is a technique utilized in patients with known or predicted difficult airways that have a severely limited mouth opening and are predicted to be challenging to ventilate by mask (1). In the setting of upper airway obstruction, it can be difficult to identify relevant anatomy for successful intubation. This case highlights a potentially valuable technique that providers can employ when managing difficult airways using a nasopharyngeal airway (NPA) as a conduit for the flexible fiberoptic bronchoscope.

Methods: We present a 4-year-old male who was admitted to the pediatric inpatient floor for Epstein-Barr virus tonsilitis, fever, coughing, and respiratory distress. CT head and neck obtained in the ER was notable for bilateral tonsil (orange arrows) and adenoid (red arrows) hypertrophy without evidence of retropharyngeal or peritonsillar abscess. ER labs were notable for positive Epstein-Barr virus mononucleosis screen and complete blood count without leukocytosis; COVID-19, Influenza A/B, streptococcus antigen testing, and throat culture were negative. Upon admission to the floor, he was given one dose of clindamycin due to concern for a developing peritonsillar abscess and started on dexamethasone every 8 hours for airway edema. His clinical status markedly improved by the evening of admission. However, he was found with sleep-disordered breathing, stridor, and severe retractions with adequate oxygen saturation while asleep that night. The patient was then placed on high-flow nasal cannula overnight with little benefit. ENT and Anesthesia were consulted for airway management the following morning. After multidisciplinary consultation with family and staff, it was decided to proceed with elective awake fiberoptic nasal intubation in the operating room and transfer to the Pediatric Intensive Care Unit (PICU) for a higher level of care, as there was a concern for airway compromise and the patient's mouth opening was severely limited.

With ENT available in the operating room, a safety timeout was performed, the patient was given midazolam, and brought to the operating table. Oxymetazoline spray was used in the bilateral nostrils for decongestion and hemostasis, and sedation was provided with ketamine during intubation. The left nostril was serially dilated using NPA's coated in viscous lidocaine. After which, the patient remained with significant adenoid hypertrophy, making it difficult to pass the bronchoscope beyond this point. It was then decided to use the NPA as a conduit for the bronchoscope, as the NPA readily passed beyond the obstruction. The bronchoscope was passed through the NPA, excellent visualization of the cords was achieved, and the NPA was removed with scissors. The bilateral true vocal cords were anesthetized with 2 ccs of 4% lidocaine, and a 5.0 cuffed endotracheal (ET) tube was inserted into the airway and secured. ET tube placement was again confirmed with repeat bronchoscopy and a chest x-ray. The patient was then transported to the PICU.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Awake fiberoptic nasotracheal intubation was deemed the safest option as the patient's mouth opening was markedly limited and there were signs of significant upper airway obstruction before intubation. Although it likely would have been possible to force the camera beyond the obstruction, it was deemed safer and easier to use the NPA as a conduit. The NPA not only provides a direct route for the bronchoscope to the true vocal cords but also acts as a barrier against secretions. Future considerations include appropriate sizing of the NPA, creating a longitudinal slit before inserting the NPA to facilitate its removal, and attaching a suture to the proximal end of the NPA to avoid dislodgment into the oropharynx upon insertion of the bronchoscope. This technique is not routinely described, and only a few publications document a similar use of the nasopharyngeal airway (2,3,4). This case highlights a useful technique that providers can add to their toolbox when managing difficult airways.

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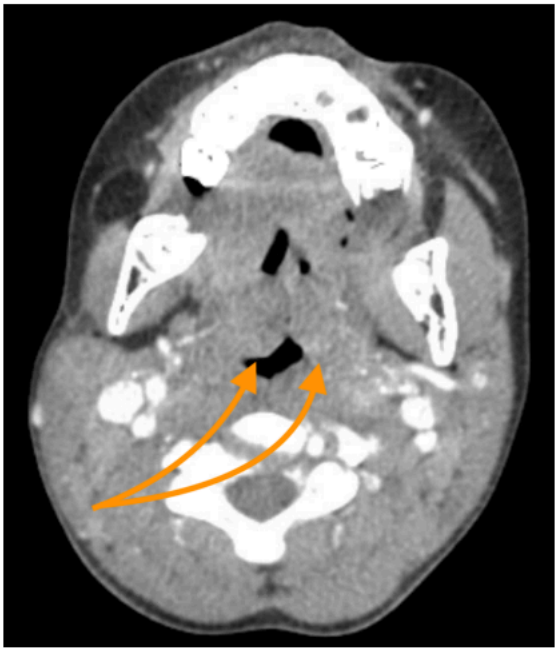

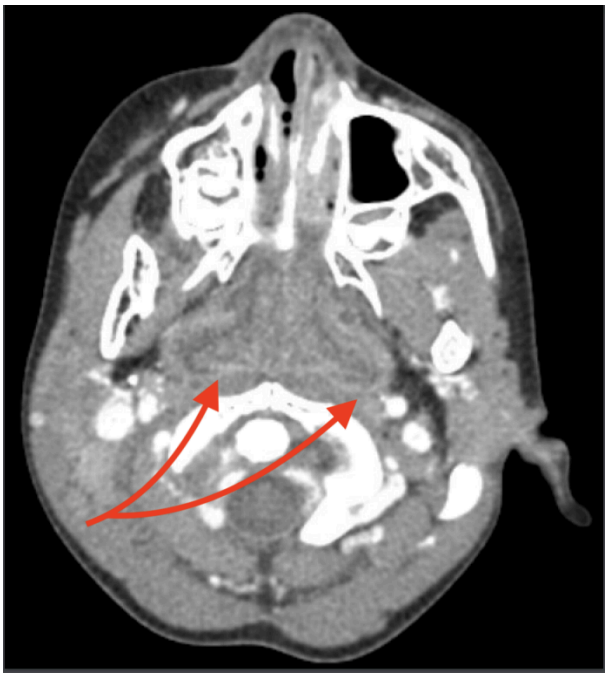

Pediatric Anesthesiology 4 - Use of inhaled tranexamic acid for control of acute intra-procedure pulmonary hemorrhage in a pediatric patient undergoing cardiac catheterization

Fanny Yeung[1], An Kim, Riva Ko[2]

Columbia University Medical Center[1] Columbia University, College of Physicians & Surgeons[2]

Introduction: Pulmonary hemorrhage is an uncommon but life-threatening condition in the pediatric population. [1,2] Inadequate control of bleeding into the airway can lead to airway obstruction, respiratory arrest, neurological damage, hemorrhage shock, and death. [3] Tranexamic acid reduces bleeding by blocking the conversion of plasminogen to plasmin and inhibiting binding of plasmin to fibrin. [2] While the use of systemic tranexamic acid in the pediatric population is well-documented, there is limited data for the use of inhaled tranexamic acid. [2] We describe a case of a 17-month-old male with acute pulmonary hemorrhage successfully treated with inhaled tranexamic acid.

Methods: A 17-month-old male with a history of hypoplastic left heart syndrome status post Norwood/Sano followed by bidirectional Glenn, complicated by severe acute on chronic systolic heart failure requiring insertion of a ventricular assist device placement (VAD), presented to the pediatric cardiac catheterization lab for evaluation of Glenn pressures. Increased work of breathing was noted on pre-operative evaluation.

Induction and intubation went smoothly; patient was maintained on FiO2 50-60% to maintain SpO2 >70. Cangrelor and bivalirudin infusions were continued in setting of VAD.

Shortly after obtaining hemodynamic data, there was a sudden increase in peak inspiratory pressures, with concomitant decreases in tidal volumes and SpO2. Gentle suctioning of the endotracheal tube revealed copious bright red secretions. Manual bag ventilation with 100% FiO2 resulted in minimal improvement. Cangrelor and bivalirudin infusions were discontinued. Fluoroscopy showed diffuse capillary blush in multiple segments of the right lung, concerning for bleeding but with no obvious single source of bleeding or vessel injury (Figure 1). Multiple repeat images demonstrated worsening opacification.

Balloon inflation in the right interlobar pulmonary artery (RIPA) was initiated to occlude flow and promote hemostasis. Simultaneously, oxygenator was added to the VAD circuit and 250mg of TXA was administered through the endotracheal tube via nebulizer. Oxygenation and acid-base status quickly improved. Balloon was deflated after forty-three minutes with subsequent fluoroscopy demonstrating improved aeration of the right lung. Repeat fluoroscopy after fifteen minutes showed no reaccumulating blood in the right lung. Scant blood was noted upon suctioning of the endotracheal tube. Patient was transported back to the intensive care unit uneventfully.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: Acute pulmonary hemorrhage is a rare but life-threatening complication of pediatric cardiac catheterization procedures. Quick recognition and treatment are essential for effective control. Nebulized tranexamic acid may play a role, in conjunction with other treatment modalities. This case describes the safe and successful use of inhaled tranexamic acid in control of acute pulmonary hemorrhage.

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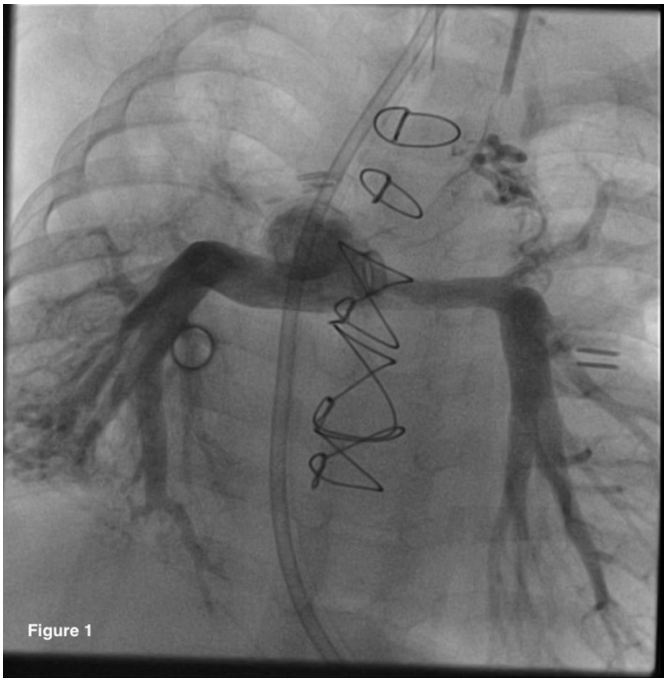
Figure 1

Perioperative Anesthesia

Perioperative Anesthesia 1 - Perioperative Diagnosis of Euglycemic Diabetic Ketoacidosis

Katherine Durant[1], Monica Paganessi, Michael Block[1]

Hackensack University Medical Center[1]

Introduction: Euglycemic diabetic ketoacidosis (EDKA) is a life-threatening medical emergency that can be challenging to diagnose in the setting of an additional acute illness. Clinically, it is defined by relative euglycemia with a serum glucose <250 mg/dL, metabolic acidosis with serum pH <7.3, serum bicarbonate <18mEq/L, high anion gap and ketosis.[1] The pathophysiology of EDKA is related to carbohydrate deficit from excretion of glucose causing an increased glucagon/insulin ratio and increased production of ketone bodies.[2] Cases of EDKA are more prevalent since the introduction of SGLT2 inhibitors for treatment of type two diabetes mellitus; however the true incidence is unknown.[3]

Methods: A 45-year-old 68kg woman with a history of type 2 diabetes mellitus on metformin and empagliflozin presented with right upper quadrant pain, poor appetite, nausea and vomiting for 4 days. The patient's last dose of metformin and empagliflozin was two days prior to arrival. On admission she had a leukocytosis to 14.7, anion gap metabolic acidosis (Na+ 136 mmol/L; K+ 3.9 mmol/L; Cl- 107 mmol/L; HCO3- 15 mmol/L, anion gap 17.9), blood glucose 151 mg/dL, Hgb A1c 7.2%. She was diagnosed with acute cholecystitis by right upper quadrant ultrasound and was kept NPO for laparoscopic cholecystectomy.

On the morning of surgery, the patient was lethargic with an anion gap metabolic acidosis (Na+ 137 mmol/L; K+ 4.3 mmol/L; Cl- 117 mmol/L; HCO3- 7 mmol/L, anion gap 17.3), blood glucose 121 mg/dL, lactic acid 0.9 mmol/L. The patient was brought to the operating room, routine monitors were applied, and she was induced with midazolam 2mg, fentanyl 100mg, lidocaine 60mg, propofol 150mg and rocuronium 40mg. Following intubation, arterial blood gas revealed metabolic acidosis (pH 6.827; PCO2 34.2 mmHg; PO2 265.6 mmHg; HCO3- 5.7 mmol/L; base excess -28.0 mmol/L). The fingerstick glucose was 127 mmol/L. The patient was given two intravenous doses of sodium bicarbonate 8.4% 50mL each. The patient was explored laparoscopically for possible etiologies of metabolic acidosis, such as necrotic gallbladder or bowel; however, findings were unremarkable.

Following the surgical procedure, the patient remained intubated and was admitted to the surgical intensive care unit for postoperative management. She continued to have persistent metabolic acidosis with normal blood glucose and lactic acid levels. Beta-hydroxybutyrate was elevated to 5.7 mmol/L. The patient was started on an insulin infusion at 2 units per hour with dextrose containing fluid infusion. After 12 hours, her metabolic acidosis resolved and beta-hydroxybutyrate decreased to 1.6 mmol/L. The patient was transitioned to subcutaneous insulin, was extubated, and discharged a few days later.

Results: Not Applicable- Medically Challenging Case Report

Conclusions: EDKA can be triggered by starvation, infection, chronic alcohol use, pregnancy and more recently recognized, SGLT2 inhibitor use.[1] SGLT2 inhibitors lower blood sugar by inhibiting glucose reabsorption in the proximal convoluted tubule in the kidney, leading to increased urinary glucose excretion.[4] The symptoms of EDKA include lethargy, malaise, nausea and vomiting. EDKA is a diagnosis of exclusion; the differential diagnosis in these patients must include and rule out other causes of high anion gap metabolic acidosis, such as lactic acidosis, alcohol ingestion, salicylate overdose, and pregnancy. Treatment of EDKA requires continuous insulin infusion at 0.05-0.1 U/kg/hr combined with dextrose containing fluids until the acidosis is resolved. [2]
This case illustrates the importance of considering EDKA in the setting of acute illness. This patient's risk factors included underlying infection, poor oral intake, and management with SGLT2 inhibitors. Although this patient did not take the SGLT2 inhibitor in 2 days, it is important to recognize that the pharmacologic effects of these drugs can last 10-14 days after the medication is discontinued.[4] Additionally, SGLT2 inhibitors increase the risk of EDKA regardless of duration of exposure.[2] Had this patient's metabolic acidosis been evaluated by measuring beta-hydroxybutyrate earlier, an insulin and dextrose infusions could have been initiated earlier to avoid perioperative complications.

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Perioperative Anesthesia 2 - Intraoperative Transient Central Diabetes Insipidus Status Post Cerebellopontine Meningioma Resection: A Case Report

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University of Texas Medical Branch[1] University of Texas Medical Branch Galveston[2]

Introduction: Central Diabetes Insipidus (CDI) is a pathological condition in which vasopressin synthesis has been compromised, leading to symptoms such as polydipsia and polyuria (Arima et al., 2016). The causes of CDI include neoplastic etiologies such as craniopharyngiomas, meningiomas, germinomas, or metastatic lesions. Traumatic events or surgeries involving regions near the neurohypophysis can also lead to central DI with varying presentations. CDI can also be secondary to autoimmune conditions. A new potential CDI cause involves IgG4-producing cells and is subsequently named IgG4-related infundibula-hypophysitis (IgG4-RH) (Shimatsu et al., 2009). Rarely, CDI may also be caused by infectious etiologies such as tuberculosis and toxoplasmosis.

Detecting CDI secondary to surgical events is distinctive from other etiologies, despite similar presentations. The classic signs that prompt a CDI evaluation are excessive thirst and hypotonic polyuria (<300mOsmol), and importantly, the volume of fluid intake and urine output is critical to making the diagnosis (Almalki et al., 2021). A consensus for adult patients is to suspect CDI if documented urine output exceeds 250mL per hour with a urine osmolarity <300 mOsm/kg (Almalki et al., 2021). Postoperatively, the management of CDI is critical, as hypernatremia can lead to multiple complications, including blood vessel rupture, coma, hypotension, headaches, and kidney damage (Hui, 2022). In cases where the patient's thirst mechanism is intact, management of CDI post-operatively primarily involves increasing fluid intake. In cases where the thirst mechanism is impaired, the preferred therapy is Desmopressin, which can be given as an intranasal spray, intranasal liquid, or tablet (Arima et al., 2016). Due to the possibility of causing hyponatremia in patients receiving DDAVP for CDI, it is crucial to begin treatment with low-dose DDAVP and titrate as needed (Hossain et al., 2018).

Methods: A 52-year-old African American male with a past surgical history of frontal lobe dural mass resection in 2008 presented to his primary care physician for evaluation of worsening occipital headache, abnormal gait and difficulty speaking. Computed tomography (CT) head scan was performed and results showed a 3.5 cm mass in the right cerebellopontine angle (CPA) with mass effect on the brainstem and obstructive hydrocephalus. Additionally, a right inferior frontal lobe dural lesion was seen on scan, with differentials including postsurgical changes from prior craniotomy vs. mass recurrence. Patient was eventually admitted by neurosurgery and otolaryngology for a CPA mass excision and interior auditory canal (IAC) drill-out. Prior to surgery, anesthesia evaluation showed no neurological deficits. Renal evaluation was unremarkable with patient exhibiting normal urine output without any urinary complaints. No electrolyte abnormalities were seen on pre-procedure labs and patient was cleared by the anesthesia team for surgery. Intraoperative course began with successful induction using intravenous Propofol and Sufentanil. Within the first three hours of surgery, the patient had produced more than 750ml of urine, and by hour 8, had produced a total of 5000ml urine. He was then started on 0.02 units/min of Vasopressin, and this was subsequently increased to 0.04 units/min about 20 minutes later to account for the large volume depletion. At hour 13, the patient was additionally started on 4mcg of intravenous Desmopressin (DDAVP), and over the next two hours, received two supplemental rounds of 4mcg DDAVP. Despite the co-administration of DDAVP and Vasopressin, the patient continued to produce large amounts of urine, and by the end of the surgery had a total intraoperative urine amount of 8650ml. In total, the patient received 16.68 units of Vasopressin and 12 mcg of DDAVP intraoperatively. Surgery was successful and the patient was admitted to the ICU for post-surgical care.

Results: Not Applicable – Medically Challenging Case Report.

Conclusions: This patient case was unique in the unknown etiology and presentation of intraoperative diabetes insipidus. There are numerous reports that recommend administering desmopressin over vasopressin for CDI, especially for neurosurgery cases that normally warrant a longer operative period. Intraoperative nephrogenic diabetes insipidus could present similarly with an increased urine output but could require a different treatment for hemodynamics.

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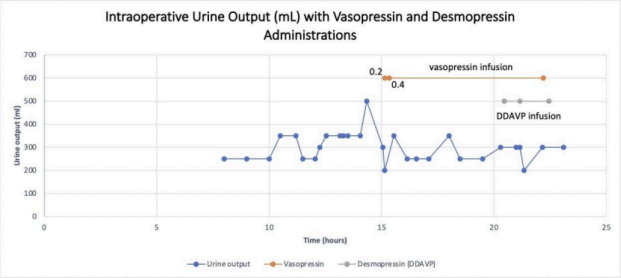
Intraoperative Urine Output (mL) with Vasopressin and Desmopressin Administrations
Urine output (ml)
700
600
500
400
300
200
100
0
0
5
10
15
20
25
Time (hours)
0.2
0.4
vasopressin infusion
DDAVP infusion
Urine output
Vasopressin
Desmopressin (DDAVP)

Perioperative Anesthesia 3 - Perioperative Considerations of A Parturient Patient With Polyethylene Glycol Allergy Undergoing Obstetric And Gynecologic Surgery Under Neuraxial Anesthesia

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University of Ottawa[1]

Introduction: Polyethylene glycol (PEG) is a common additive to medications (tablet surface coating, pill binder, intravenous liquid stabilizer) and materials (wound dressings, lubricants, bone cement) used in the perioperative period (1, 2). Concerningly, there have been increasing reports of hypersensitivity to PEG, ranging from local allergic reactions to anaphylaxis (1-4). Minimizing exposure to PEG containing substances is challenging but imperative in the perioperative setting for patients with known or suspected PEG allergies. We present a case report of the perioperative anesthetic management of a patient with confirmed PEG allergy, who underwent an elective cesarean section (C/S) and concurrent total abdominal hysterectomy (TAH) and bilateral salpingectomy.

Methods: A 25-year-old female patient underwent an elective C/S at term, with concomitant TAH and bilateral salpingectomy for treatment of chronic pelvic pain. The patient's medical history was significant for multiple drug allergies across several medication classes. Skin prick test confirmed an allergy to PEG. The patient was assessed in the preoperative anesthesia clinic. She was advised to continue prophylactic loratadine on the day of surgery and to bring her supply of PEG-free liquid acetaminophen to hospital. In collaboration with surgery, anesthesia, pharmacy and nursing, a list of medications and materials required for the perioperative care of the patient was generated. Product monographs were then reviewed for PEG, according to route of administration, concentration, and manufacturer. Medications and materials where PEG status could not be confirmed, were not used. On the day of surgery, the patient was vitally stable with no clinical allergic manifestations. Combined spinal epidural was chosen as the primary anesthetic. The patient received a fentanyl and hyperbaric 0.75% bupivacaine spinal and neuraxial block was maintained with an epidural infusion of lidocaine 2% with 1:200000 epinephrine. Close communication with the patient and vigilant monitoring of vital signs, skin and mucosa ensured early detection of allergic reactions. The patient remained hemodynamically stable, and no respiratory or mucosal changes were identified. The surgery proceeded without complications. Following surgical closure, bilateral transversus abdominis plane blocks were performed under ultrasound guidance using 0.25% ropivacaine with 1:200000 epinephrine for postoperative pain. On postoperative day (POD) 0, multimodal analgesia was achieved with scheduled acetaminophen IV, fentanyl IV as needed and a titrated epidural infusion of 0.1% bupivacaine with 2 mcg/mL of fentanyl. On POD1, acetaminophen IV was transitioned to the patient's PEG-free supply of liquid acetaminophen as needed. Epidural infusion was weaned and transdermal fentanyl at 6 mcg every 3 days via a PEG-free fentanyl patch was initiated. The patient was discharged home on POD 2 with a 6-day course of transdermal fentanyl. No allergic reactions were observed or reported in the perioperative period.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Patients with a history of multiple medication allergies to structurally distinct drugs and patients with severe hypersensitivity to medications where the active ingredient has been ruled out should be considered for investigation of PEG allergy. We detail the importance of meticulous preoperative planning with multi-disciplinary involvement and the feasibility of a combined spinal-epidural technique. Neuraxial anesthesia, without sedation, allowed for direct communication with the patient in the intraoperative period, conferring the advantage of self-reported detection of allergic reactions. The case also demonstrates a potential postoperative pain management strategy for patients with PEG allergy in the post-partum period. Overall, we propose perioperative recommendations for patients with PEG allergy.

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Perioperative Anesthesia 4 - Perioperative Management of a Patient with Boerhaave Syndrome

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Creighton University School of Medicine[1] CHI Creighton University Medical Center[2]

Introduction: Boerhaave syndrome is a full-thickness tear of the esophagus caused by a sudden increase in intraesophageal pressure, such as from vomiting. Rupture of the intrathoracic esophagus leads to contamination of the mediastinum with food particles and gastric contents, resulting in increased risk of septic shock. Prompt diagnosis and management of Boerhaave syndrome is crucial as the mortality rate increases with increased delay in treatment.

Surgery is the preferred method of treatment for Boerhaave syndrome. Given the risk of severe infection and hemodynamic instability, anesthesia providers are central to the survival of these patients. These patients are at increased risk for aspiration, so care must be taken to prevent coughing or straining during induction. Rapid sequence induction is indicated due to aspiration risk. Additionally, the fasciculations from succinylcholine may increase intraabdominal pressure and risk further damage to the esophagus, so a defasciculating dose of rocuronium may be considered.

Postoperatively, patients need close monitoring for signs of sepsis in an intensive care unit. Due to the use of a rib spreader and the manipulation of the chest cavity, these patients also require a multimodal pain management approach.

Methods: A previously healthy 52-year-old man presented with two hours of severe left-sided back pain which began after an episode of emesis. On arrival, he was tachycardic, tachypneic, and hypertensive. He had diminished breath sounds in the left lower lung. CT chest with IV contrast demonstrated a left hydropneumothorax with fluid and free air in the posterior mediastinum tracking along the distal esophagus, concerning for esophageal perforation. Diagnosis was confirmed with CT chest with oral contrast which showed extravasation of contrast into the left pleural space. Decision was made to bring him emergently to the OR, approximately 10 hours after symptom onset.

Given that this patient was at high risk of aspiration, rapid sequence induction was done with 80 mg lidocaine, 100 mg propofol, 20 mg ketamine, and 150 mg succinylcholine. An endotracheal tube was placed using a glidescope to avoid esophageal intubation. A right internal jugular central venous catheter and a right radial arterial line were placed. His lactate level was 4.4, so the respiratory rate was set to 20 bpm to mediate his acidosis. General anesthesia was maintained with fentanyl, sevoflurane, and rocuronium.

Surgery began with a laparoscopic approach, but the perforation was not visualized, so decision was made to transition to left thoracotomy. An airway exchange catheter was used to place a double lumen endotracheal tube, and placement was confirmed with fiberoptic bronchoscopy. The patient was placed under one-lung ventilation to allow for a left thoracic approach. The patient underwent successful repair of the esophagus with no adverse intraoperative events.

Postoperatively, an ultrasound-guided left-sided erector spinae catheter was placed. He also received ketamine, lidocaine, acetaminophen, and hydromorphone. He was extubated on post-op day 1, and the erector spinae catheter was removed on post-op day 10. He was discharged on post-op day 13.

Results: Not Applicable - Medically Challenging Case Report

Conclusions: While Boerhaave syndrome is rare and infrequently described in the literature, it is a life-threatening surgical emergency that all anesthesia providers should be prepared to manage. Delayed diagnosis is common because it presents with non-specific symptoms, and prompt treatment is critical to survival. It can be rapidly fatal due to septic shock from a mediastinal or lung infection. Given this, anesthesiologists play a critical role in maintaining the hemodynamic stability of these patients.

The survival of this patient was in part due to careful and efficient anesthetic management. He was intubated without significant increase in intraabdominal pressure or further esophageal injury. A defasciculating dose of rocuronium was not used in this case, but we would consider using this in future instances to decrease the risk of increasing intraabdominal pressure from fasciculations due to succinylcholine. Otherwise, his operative course was managed successfully with the use of single lung ventilation and close hemodynamic monitoring. Additionally, his postoperative pain was adequately controlled with the use of an erector spinae catheter and multimodal pain management. We hope this report helps other anesthesia providers act quickly and attentively if they encounter a similar case.

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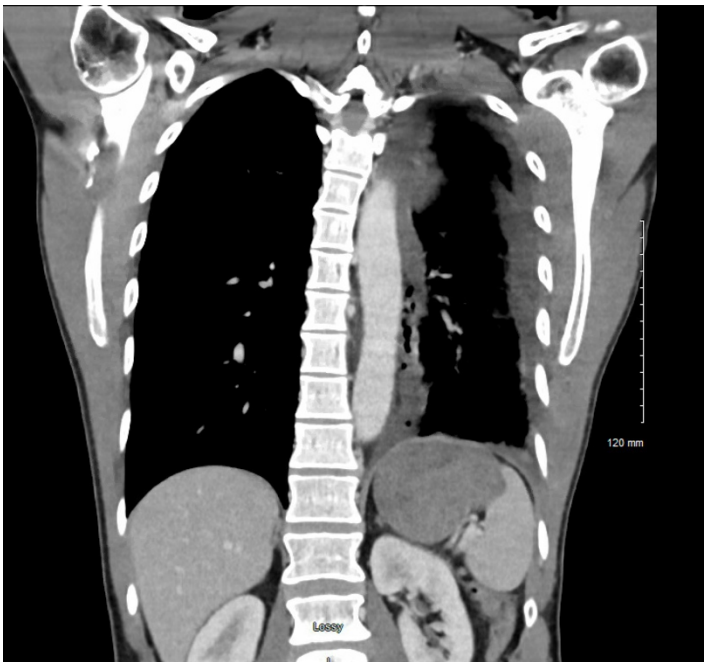
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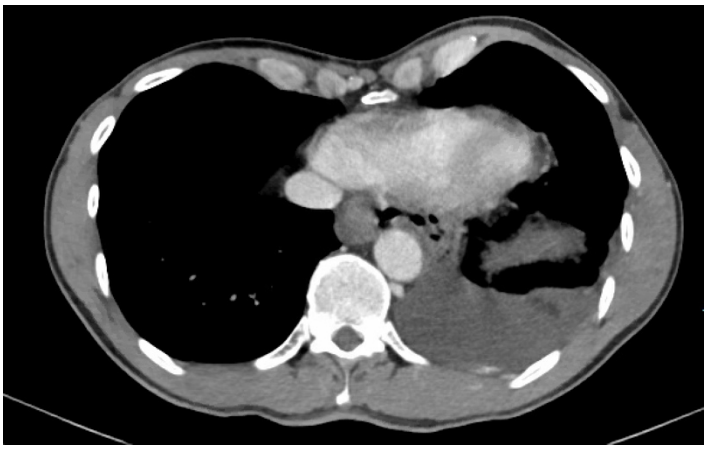

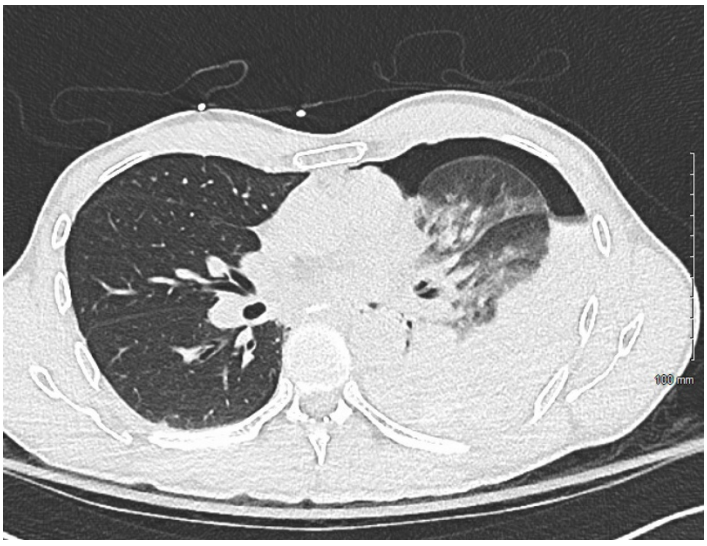
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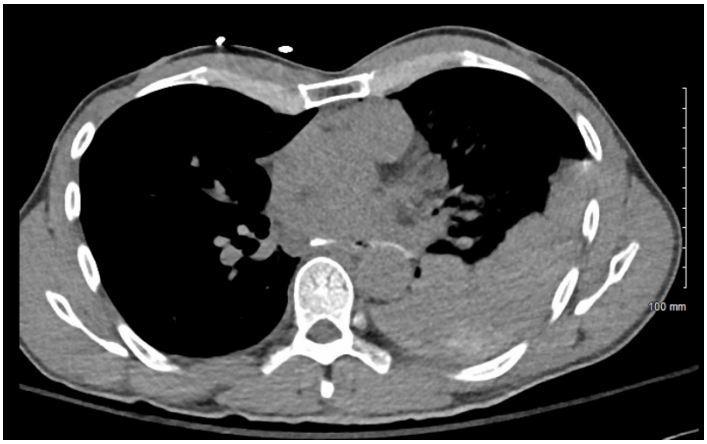
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Perioperative Anesthesia 5 - Reuse of liver allograft from a recipient declared dead by neurological criteria shortly after transplantation

Alessandro Galli[1], Nicholas Mendez[2], Helge Eilers[1], Micheal Bokoch, Dieter Adelmann[2], Kate Kronish

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

Introduction: Despite efforts to expand the pool of available liver grafts, there is a severe shortage of organs to treat individuals suffering from end stage liver disease. Efforts to safely expand the pool of transplantable grafts include use of living donors, partial grafts, donation after circulatory death programs, and machine perfusion techniques. (1) Due to this organ shortage, it is necessary to evaluate each patient's suitability for transplantation, including the assessment of whether a patient is too sick to transplant. For example, patients with severe, irreversible cardiac, pulmonary, or neurologic injury are likely to have very poor outcomes despite transplantation. Such patients may be considered too sick to transplant and should not be offered this extremely scarce resource. (2)

We present a case of liver transplantation (LT) in a recipient with poor pre-operative neurologic status who was declared brain dead on postoperative day (POD) 1 after transplantation due to brain hemorrhage and herniation. The liver allograft was then successfully explanted and re-transplanted into a second recipient.

Methods: A 53-year-old female with history of alcohol-related cirrhosis complicated by spontaneous bacterial peritonitis, hepatorenal syndrome type 1 and hepatic encephalopathy was admitted to our hospital with acute-on-chronic liver failure. Her pre-transplant workup was completed, and she was considered an acceptable transplant candidate by the hospital's liver transplant selection committee - a committee comprised of hepatologists, transplant surgeons, transplant anesthesiologists and social workers. She was listed for LT with a MELD-Na+ score of 40 (creatinine 3 mg/dl, total bilirubin 27 mg/dl, INR 2.7, Na+ 125 mmol/l).
Her clinical status deteriorated, and she was soon transferred to the intensive care unit (ICU) for hypovolemic shock, with hematochezia and bleeding from a paracentesis site, and for initiation of CRRT. Over the next 24 hours, her neurologic status rapidly worsened, believed to be due to worsening hepatic encephalopathy, now requiring intubation for airway protection. A head CT was not obtained.

That same day, a donor liver offer was accepted, and the patient underwent a successful LT. Estimated intraoperative blood loss was 14 L, with 14 units of pRBC, 22 units of FFP, and 3 units of platelets transfused. The patient returned to the ICU intubated, on propofol and norepinephrine at 0.12 mcg/kg/min.

Within a few hours of completion of the LT, the patient was noted to have non-reactive pupils, with no movement to noxious stimuli while off sedation. The patient underwent a stroke workup, and the head CT revealed a "[..] large intraparenchymal hemorrhage centered in the left basal ganglia with intraventricular extension [..], diffuse cerebral edema with loss of sulci and basilar cisterns [..] and diffuse loss of gray-white differentiation."

The neurosurgery service determined that any intervention would be unlikely to confer any meaningful benefit. The family was notified, and the patient was declared brain dead less than 8 hours later, two days after LT.
Given signs of good graft function in this brain-dead recipient (ALT/AST 105/89 U/L, total bilirubin 5 mg/dL, INR 1.1), consent for organ donation was obtained from the patient's family. On POD 3, the graft was explanted for donation and subsequently re-transplanted into another recipient. Currently the second recipient of the graft is alive, maintained on standard immunosuppression therapy, with good liver function.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The perioperative management of this case was discussed at the Morbidity and Mortality meeting of the multidisciplinary liver transplant team. Importantly, it is unknown whether the cerebral hemorrhage developed before, during or after the LT. Acute extensive cerebral hemorrhage is a contraindication to liver transplantation. The patient's neurologic decline requiring tracheal intubation was a missed opportunity to assess the patient with a head CT. We subsequently updated our practice to obtain a head CT scan in cirrhotic patients with worsening neurological status requiring intubation before undergoing LT. Although we were able to successfully retransplant the liver graft into another recipient, this case highlights the need for vigilance in assessing critically ill patients for their suitability for transplant prior to LT.

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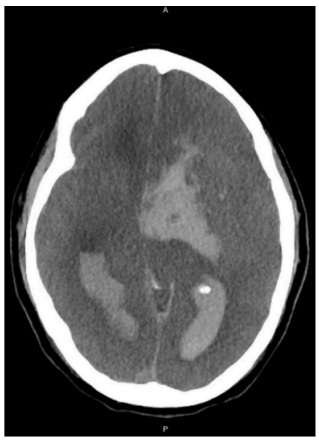


Figure 1. Head CT scan on POD 1 showing large intraparenchymal hemorrhage centered in the left basal ganglia with intraventricular extension, diffuse cerebral edema with loss of sulci and basilar cisterns and diffuse loss of gray-white differentiation.

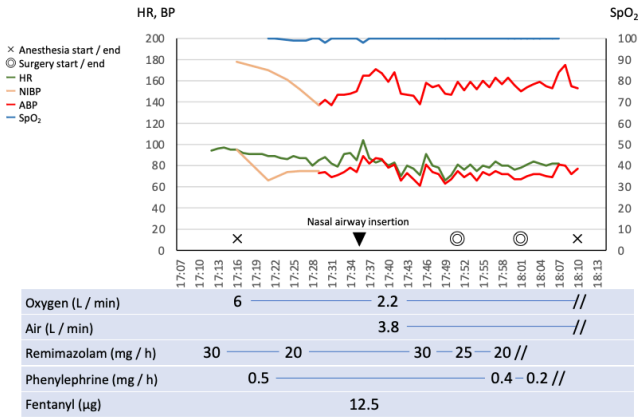
Perioperative Anesthesia 6 - Successful Anesthetic Management with Remimazolam For Non-Cardiac Surgery In An Elderly Patient With Severe Aortic Stenosis: A Case Report

Riho Hagihara[1], Hiroyuki Seki[2], Tomoko Yorozu[3]

Kyorin University[1] Kyorin University School of Medicine[2] Kyorin University School of Med[3]

Introduction: Anesthetic management in patients with severe aortic stenosis (AS) is challenging because even small doses of anesthetics can easily cause hemodynamic deterioration. Here, we report a case of successful a nesthetic management with remimazolam (RMZ) for non-cardiac surgery in an elderly patient with severe AS under monitored anesthesia care (MAC).

Methods: A 92-year-old woman (150 cm, 30 kg) underwent emergency ureteral stent insertion for calculous pyelonephritis under local anesthesia in our hospital. She had been institutionalized with dementia and followed-up in our hospital for rheumatoid arthritis, severe AS, and chronic heart failure. Three months after discharge from the hospital, stent replacement was scheduled. However, it failed because of lack of patient's cooperation due to dementia. The replacement was re-scheduled under anesthesiology supervision. Preoperative echocardiogram revealed a paradoxical low-flow low-pressure-gradient severe AS with an aortic valve area of 0.42 cm2 and worsening heart failure. Cardiologists recommended prior heart failure control; however, considering the urgency of the surgery, stent replacement under MAC with RMZ was scheduled. On admission, the patient's blood pressure was 178/95 mmHg, heart rate was 95 beats/min, and SpO2 was unmeasurable due to peripheral circulatory failure and patient's severe movement (Figure). As the patient refused to wear an oxygen mask, RMZ (1 mg/kg/h) was started without administering oxygen. Six minutes later, oxygen was initiated via closed anesthetic circuit owing to reduction in her movements. Two minutes later, the patient fell asleep. Because of upper airway obstruction due to glossoptosis, a nasal airway was inserted after administering 12.5 μg of fentanyl, and surgery was started. During the surgical procedure, RMZ was administered at 0.7-1 mg/kg/h and phenylephrine was administered continuously (0.4-1 mg/h). Although jaw thrust maneuver was temporarily required to secure the airway, stable hemodynamics were maintained. The surgical procedure was completed uneventfully, and RMZ administration was stopped. The patient opened her eyes 10 minutes later. She was transferred to the intensive care unit and discharged from the hospital on postoperative day 7 uneventfully.



Results: Not Applicable – Medically Challenging Case Report

Conclusions: This case suggests that RMZ could be an anesthetic option for non-cardiac surgery in patients with severe AS.

Regional Anesthesia

Regional Anesthesia 1 - Examining the Role of the Fascia Iliaca Nerve Block on Perioperative Hip Fracture Pain Management

Tejas Kollu[1], Jacqueline Quinn, Elizabeth Willis[2], Jahnavi Trivedi[3], David Creighton[4]

Albany Medical Center[1] University of North Carolina[2] UNC[3] UNC Chapel Hill[4]

Introduction: In the United States, more than a quarter of a million people suffer from a hip fracture every year, with more than 75% occurring in women over the age of 65.[1] The most common fractures occur at the femoral neck and intertrochanter, which is an area that can be partially numbed by a fascia iliaca nerve block.[8] During a fascia iliaca nerve block, local anesthetic is injected inferior or superior to the inguinal ligament to create a motor and sensory block of the femoral, genitofemoral, lateral femoral cutaneous, and obturator nerves. This block can be performed either pre- or post-operatively to improve hip fracture pain management.

Our patient is a 92-year-old female (ideal weight 52.5 kg) with a past medical history of hypertension, osteoporosis, lumbar disc disease with spinal cord stimulator implanted, aortic stenosis status-post TAVR, and LV systolic dysfunction (EF 55%) brought to the emergency department for evaluation of left hip pain after a non-syncopal, mechanical fall. The patient was not able to ambulate since the time of injury due to isolated left hip pain.

Methods: On physical exam, the left lower extremity showed a hip deformity with swelling and tenderness laterally and pain with log roll test. Skin was intact without tenting or impending open fracture. Sensation intact to light touch in deep peroneal, saphenous, sural, and tibial distributions. Ligamentous exam was stable. Compartments were soft and compressible, with no pain on passive stretch. X-Ray of left hip demonstrated a transcervical femoral neck fracture.

In the ED, the patient's pain was self-reported as 10/10 on the VAS scale on arrival. She received IV fentanyl (50 mcg) with minimal pain relief. She then received a single-shot supra-inguinal fascia iliaca nerve block by the regional anesthesia team in the ED. Skin was prepped with 2% chlorhexidine. Local infiltration with 5 ml of 2% lidocaine was made at the needle insertion site. The sonographic anatomical landmarks of the ASIS, iliacus muscle, sartorius muscle, internal oblique muscle, and deep circumflex artery, were identified using a linear ultrasound probe (Figure 1). A 21-gauge, 100-mm short bevel needle pierced the skin at the base of the probe and was advanced in an "in-plane" fashion towards the target. The needle was advanced from lateral to medial, toward the fascial layers separating the internal oblique and iliacus muscles, ultimately piercing through the fascia iliaca, with a goal of visualizing the needle tip in between the iliacus muscle fibers and their overlying fascia, and seeing local anesthetic spread medially below the deep circumflex artery. Following initial negative aspiration, 30 ml of 0.25% bupivacaine was administered using fractionated injection. Local anesthetic was visualized spreading in the desired fascial plane.

Over the next 24 hours, the patient received a total of 20mg oxycodone PO and 2mg morphine IV. She received medical clearance for surgery and was brought to the operating room 24 hours after initial presentation in the ED. Given that 22 hours had elapsed since her initial nerve block, and given a half-life of bupivacaine of 3.1 hours, the anesthesia team decided to perform a second single shot fascia iliaca block immediately preoperatively in order to provide ongoing regional anesthesia coverage through the initial post-operative period.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: The fascia iliaca nerve block can reduce femoral neck fracture pain when executed successfully. Prior research has demonstrated that the fascia iliaca block can reduce pain when assessed by a 10 point verbal ranking scale as well as morphine consumption following a hip fracture.[3,4] Steenberg et al found that preoperative fascia iliaca compartment block resulted in superior analgesia during movement compared to opioids (p=.02) and resulted in decreased consumption of preoperative opioid medications (p=.03).[5]

One of the most concerning adverse events of peripheral nerve blocks includes local anesthetic toxicity, especially when considering administration of repeat nerve blocks in a single patient. Bupivacaine has an elimination half-life of 3.1 hours (assuming normal liver metabolism and renal clearance) with a measurable nerve blockade for 8 to 12 hours. Our 52.5 kg patient initially received a dose of 75 mg which is 71.43% of her estimated safe dose (2 mg/kg, or 105 mg). When she re-presented for surgery, 7 elimination half-lives had elapsed since the initial nerve block at which point we expected more than 97% of the drug to be eliminated.[6] Therefore, a repeat nerve block was considered safe. Our patient tolerated the initial block as well as the re-dosed block very well and had significant improvement of her hip pain.

The fascia iliaca nerve block plays an important role in hip fracture management. Most interventions consist of a single injection in the ED, a single injection pre-operatively, or a continuous nerve catheter placed at some time after admission but before surgery. This patient case is unique in that we repeated a single shot injection on hospital day 2. Our recommendation is that at institutions that lack the infrastructure for peripheral nerve catheter placement, providers caring for elderly and frail hip fracture patients should consider repeating single shot fascia iliaca blocks as needed, in order to maximize the ongoing benefits of regional anesthesia, taking into account the local anesthetic half-life, the initial dose given, and any modifying factors such as hepatic and renal function.

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Regional Anesthesia 2 - Thoracic Epidural Anesthesia In A Patient With Achondroplasia On Buprenorphine: A Case Report

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Introduction: Achondroplasia is the most common bone dysplasia disorder, with a frequency of roughly 1 in 20,000 live births.[1] Individuals with achondroplasia have distinctive features, such as short stature, craniofacial abnormalities, and spinal deformities. Variations in anatomy of the spine can make neuraxial access difficult and the spread of local anesthetic unpredictable. Though multiple reports have documented the use of neuraxial anesthesia in achondroplasia, these are typically limited to lumbar epidurals, often for cesarean delivery.[2-4] There is a paucity of reports describing the successful use of thoracic epidurals for perioperative pain management in these patients. Yet, in individuals undergoing major abdominal surgery with preexisting chronic pain or opioid tolerance, thoracic epidurals can provide invaluable benefit for managing postoperative pain, reduce medication-related complications, and improve patient satisfaction.

Methods: Our team cared for a 38-year-old female with achondroplasia and a past medical history that included hypertension, obstructive sleep apnea, endometriosis, peptic ulcer disease complicated by multiple prior perforations, and chronic abdominal pain on buprenorphine/naloxone and oxycodone. She presented to the emergency department with worsening abdominal pain, hematemesis, and hypotension with concern for a gastric ulcer perforation, and was taken to the operating room for urgent exploratory laparotomy. She was hemodynamically stable upon presentation to the preoperative area, was consented for general anesthesia, and following discussion about pain management options, agreed to receive a preoperative epidural. The patient had received lumbar epidurals with prior Cesarean sections without complications but had no prior thoracic epidurals. Review of radiographic images showed mild scoliosis of her thoracic spine with otherwise normal vertebral spaces.

A thoracic epidural was placed preoperatively at the T8-9 level by a paramedian approach without apparent complications. Intraoperatively, a large midline abdominal incision was performed, and repeated small doses of 0.125% bupivacaine were given for a total of 8 milliliters. Postoperatively, in the recovery room she was found to have narrow dermatomes not fully covering her incision, and she endorsed severe pain. Additional epidural bolus doses of 0.125% bupivacaine totaling 10 milliliters were given, resulting in significant reduction in pain and improved dermatomal coverage of her entire surgical incision.

During her hospital course, patient controlled epidural analgesia was titrated to eventual dosages of bupivacaine 0.1% with hydromorphone 12 micrograms/milliliter at 6 milliliters per hour with a 3-milliliter demand dose every 30 minutes. She was additionally given her home doses of buprenorphine/naloxone, oxycodone, venlafaxine, and gabapentin. Although the patient reported severe pain, she described this as typical of her chronic abdominal pain, and the additional surgical pain was felt to be tolerable to the patient. She continued to endorse satisfactory pain levels during her postoperative hospital course, appearing comfortable with appropriate participation in post-operative recovery milestones, including ambulation. She had no apparent epidural-related complications. She was successfully transitioned to an oral pain regimen and her epidural was removed on postoperative day 5.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: Achondroplasia is a common condition that warrants special anesthetic considerations. Although there is a lack of data regarding the use of thoracic epidurals in this population, this technique can be especially useful in individuals with achondroplasia who undergo invasive abdominal procedures, particularly in patients with preexisting chronic pain. This report describes the effective and safe use of thoracic patient controlled epidural analgesia for postoperative pain in a medically complex patient with achondroplasia. Further studies are needed to help provide recommendations and guide management for this specific patient population.

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Trauma

Trauma 1 - Acute Contralateral Epidural Hematoma After Evacuation of Traumatic Subdural Hematoma: A Case Report and Review of Literature

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Introduction:
Acute contralateral epidural hematoma after evacuation of a traumatic subdural hematoma is a rare but potentially devastating complication in traumatic head injury patients. There have been few case reports documenting this phenomenon and the underlying pathophysiological mechanism remains a mystery. In this case report, we describe a 37-year-old male who presented to our facility after being involved in a motorcycle accident, resulting in an acute right sided traumatic subdural hematoma. He underwent an emergency right craniectomy with subdural evacuation; approximately 12 hours later, the patient developed an acute contralateral epidural hematoma which was not present on initial diagnostic imaging. He was then emergently returned to the operating room for a left craniectomy with epidural hematoma evacuation.

Traumatic brain injuries (TBIs) have contributed to approximately one million deaths in the United States over the last 2 decades; during 2016–2018, a total of 181,227 TBI-related deaths (17.3 per 100,000 population per year) occurred in the United States[1]. Acute subdural hematomas after trauma remain one of the most life-threatening outcomes of traumatic head injuries, with a reported estimated mortality of greater than 50%[2]. Prognostic indicators include (but are not limited to) initial GCS, admission to a trauma facility, age, injury severity score (ISS), initial intracranial pressure (ICP), and timing of surgical intervention.

Methods: Case Presentation
The patient is a 37-year-old male with no significant medical history who presented to our facility after being involved in a motorcycle collision. He was evaluated by the trauma surgery service upon arrival, was found to have a GCS of 3 and was intubated in the trauma bay for airway protection. After intubation, he was taken to the computed tomography (CT) scanner which demonstrated a large right sided subdural hematoma with a 10mm midline shift, as well as a left temporal bone fracture, right L1-3 transverse process fractures, and a left temporal subarachnoid hemorrhage. He was taken to the operating room emergently by the neurosurgery service where he underwent a right craniectomy with hematoma evacuation.

There were no reported intraoperative complications. Patient was then transported to the trauma surgical intensive care unit (TSICU) for further care. Approximately 12 hours later, the patient had a repeat CT scan performed which showed a new left sided epidural hematoma as well as a left parietal intraparenchymal hemorrhage. The patient was then taken back to the operating room for a left sided craniectomy with hematoma evacuation.

There were no intraoperative complications. The patient was again taken to the trauma surgical intensive care unit for continued care and treatment. He underwent a tracheostomy and percutaneous gastrostomy tube placement procedure as well as a temporary external ventricular drain (EVD) while in the TSICU. Repeated imaging after anticoagulation did not show any additional intracranial hemorrhage. The patient was then deemed stable for hospital discharge and was transferred to a rehabilitation facility.

Results: not applicable - medically challenging case report

Conclusions: Traumatic brain injury (TBI) remains one of the leading causes of mortality in the United States, with higher incidences reported in young male patients. Approximately 33% of patients with severe TBIs are diagnosed with acute subdural hematomas (ASDH). For significant subdural hematomas, the mainstay of treatment is decompressive craniotomy, especially in cases with significant intracranial shift or potential for herniation[3].

Review of literature suggests that acute, delayed epidural hematoma following evacuation of a subdural hematoma is a rare but life-threatening event; there are fewer than 40 documented cases. There is only one documented case report of a delayed epidural after subdural occurring twice in the same patient[4]. Although, due to the rarity of this pathology, no clear guidelines are established, there are some risk factors and warning signs of potential epidural hematoma after subdural evacuation; these include skull fracture, intraoperative brain swelling, pupillary dilation, unmanageable elevations in intracranial pressure, seizures, large volume intraoperative blood loss, long duration of craniotomy, and a large craniotomy area[4].

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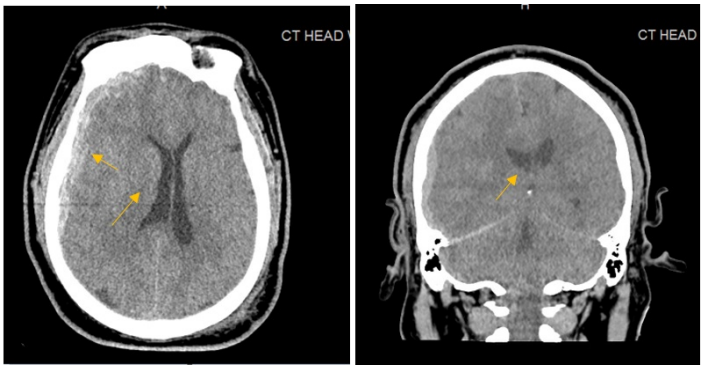

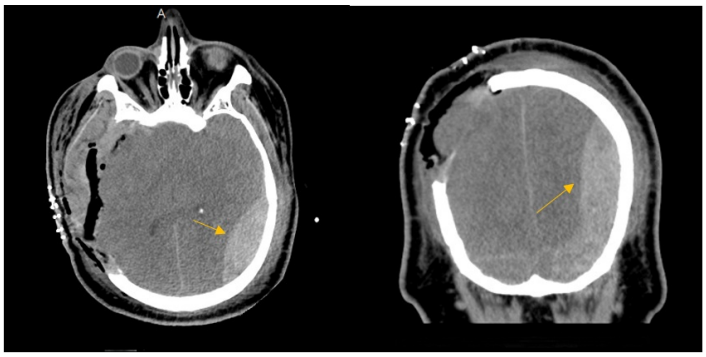

Trauma 2 - A Lesson Of Anesthesia Management of A Patient With Severe Trauma Who Underwent Nine Post-Trauma Surgeries Within One Month: A Case Report

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Introduction: A patient undergoing multiple surgeries in a short duration confronts the heterogeneity in the management of each operation anesthesia performed by a different anesthesiologist. Differences in anesthesia choice, induction protocols, anesthesiologists' procedures, intraoperative management, and postoperative analgesic strategies of several times anesthesia on the patient can result in potential negative impacts on postoperative recovery. This report presents a case of a severe bilateral lower extremity trauma due to a traffic accident, underwent one bilateral lower extremity amputation and other eight times post-amputation surgeries within one month. This case report is to propose an issue of how to minimize potential risks resulting from the heterogeneity of nine anesthesia performed by eight different anesthesiologists. (1)

Methods: A 57-year-old healthy man had severe lower extremity damages from a traffic accident and underwent bilateral above-knee amputation in a tertiary hospital. The patient was in a coma on arrival at the Emergency Room (ER). Preliminary evaluations showed that he was in a severe hypovolemic shock, with blood pressure (BP) of 40/29 mmHg and heart rate at 122 beats per minute (BPM). The patient accepted emergency amputation surgery. Considering a hypovolemic shock and extreme low BP, general anesthesia (GA) and intubation was performed, not a combined spinal epidural anesthesia (CSEA). A central venous access and an invasive blood pressure of the radial artery were established. After the surgery, the patient was admitted to the intensive care unit (ICU).

Due to severe soft tissue necrosis and further wound bleeding, the patient underwent eight subsequent surgical procedures (Table 1) after amputation with a significant decrease in hemoglobin (Figure 1). On the 6th day of admission to ICU, the patient regained his consciousness, was extubated, and transferred from ICU to the ward of the Department of Orthopedics. The patient then underwent five limb soft tissue debridement procedures under CSEA, performed by four different anesthesiologists. Five anesthesia sessions were performed with 0.5 % bupivacaine (volume 2.0ml to 2.4 ml), and the duration of the procedure ranged from 95 to 240 minutes. On the 24th and 26th days of the admission, the patient underwent two operations under GA due to progressive decline in hemoglobin and RBC count (Figure 1). Both surgeries were performed with the same GA induction anesthetics at different doses, by different two anesthesiologists. The use of postoperative self-controlled analgesia was not consistent among different anesthesiologists. During each procedure, the patient received unequal amounts of concentrated red blood cells and plasma. Finally, the patient was discharged safely on admission day 65.

Results: Not Applicable – Medically Challenging Case Report

Conclusions: For a patient who underwent repeated surgical treatments, the heterogeneity in perioperative anesthesia managements performed by different surgeons and anesthesiologists are ignored usually. In consideration of a series of repeated debridement surgeries, a serial of anesthesia management should be planed as a whole, including preoperative preparation, intraoperative anesthesia choice, the choice and dosage of anesthetics, intraoperative monitoring, and postoperative analgesia strategies. Obviously, the case shows a lesson that no multidisciplinary team (MDT) made detailed treatment and anesthesia plans on the patient, in advance. Therefore, an MDT including surgeons, anesthesiologists, ICU physicians, transfusion specialists, and nurses, is necessary to plan the entire treatment. Furthermore, the patient had CSEA performed by different anesthesiologists several times in a short period of time. Repeated lumbar puncture injury would lead to an increased risk of inflammatory infections, scar tissue formation and adhesion, (2) which may cause difficulties when the patient needs to undergo regional spinal anesthesia again in the next anesthesia.

Based on this case of a patient who underwent repeated surgical treatments, an efficient MDT should participate in planning the whole treatment, especially in perioperative anesthesia management, which finally improves patient anesthesia safety and contribute to the Enhanced Recovery After Surgery (ERAS).

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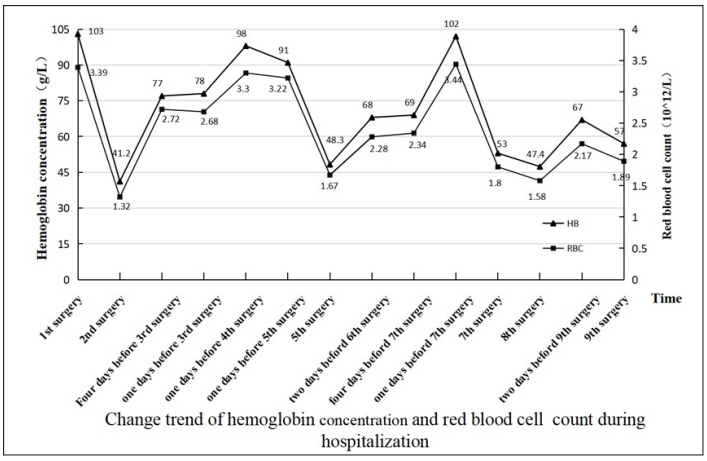
Table1. Operation and anesthesia information of the case

Surgery Times	Performed Surgery	Post-trauma days	Operation duration (minute)	Anesthesia	Induction / combined spinal epidural anesthesia drugs	Postoperative analgesia	Anesthesiologist*
1	Bilateral lower limb amputation	0	40	GA	Midazolam 1mg, Fentanyl 0.2 mg, Etomidate 4mg, Cis-atracurium 10 mg	/	Dr A
2	Soft tissue debridement /VSD negative pressure suction	2	110	GA	Midazolam 3mg, Fentanyl 0.4 mg, Cis-atracurium 20 mg	/	Dr B Dr C
3	Bilateral osteotomy stump repair / free flap reconstruction /debridement /VSD negative pressure suction	9	240	CSEA	Local anesthesia of 1%Lidocaine, 0.5% Bupivacaine 2.4 ml	Morphine 5 mg, Dezocine 2.5 mg, Ropivacaine 170 mg 100 ml NS	Dr D
4	Right lower limb soft tissue debridement / VSD negative pressure suction	12	95	CSEA	Local anesthesia of 1%Lidocaine, 0.5% Bupivacaine 2.4 ml	/	Dr E*
5	Right lower limb soft tissue debridement	17	95	CSEA	Local anesthesia of 1%Lidocaine 0.5% Bupivacaine 2 ml	/	Dr F
6	Debridement of bilateral lower limbs+ hematoma removal / free-flap reconstruction/ negative pressure suction	20	90	CSEA	Local anesthesia of 1%Lidocaine, 0.5% Bupivacaine 2.4 ml	/	Dr E*
7	Detection of hematoma after bilateral thigh amputation/ negative pressure suction/ flap reconstruction	23	140	GA	Midazolam 2.5 mg, Sufentanil 25 ug, Etomidate 20 mg, Cis-atracurium 20 mg	Hydromorphone 4 mg, Flurbiprofen Axetil 100 mg, Dezocine 20 mg, Palonosetron hydrochloride, 0.25 mg 100 ml NS	Dr G
8	Debridement after eight amputation/ right thigh osteotomy/ hematoma removal/ flap reconstruction	25	65	GA	Midazolam3 mg, Sufentanil 20 ug, Etomidate 16 mg, Cis-atracurium 20 mg	/	Dr H
9	Re-amputation of right thigh stump / flap reconstruction	31	240	CSEA	Local anesthesia of 1%Lidocaine, 0.5% Bupivacaine 2.5 ml	Hydromorphone 4 mg, Flurbiprofen Axetil 100 mg, Dezocine 20 mg, Palonosetron hydrochloride, 0.25 mg 100 ml NS	Dr G*

* The same anesthesiologist was involved in both procedures.



Change trend of hemoglobin concentration and red blood cell count during hospitalization

Trauma 3 - Case Report: Exogenous Vasopressin Induced Hyponatremia in The Setting of Traumatic Penetrating Spinal Cord Injury

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Introduction: Maintenance of mean arterial pressure (MAP) in a certain goal range has been the standard of care at many institutions for traumatic spinal cord injuries, independent of operative versus non-operative management[1]. Currently MAP >85 mmHg is the standard that is best supported by data[2]. The duration of MAP goals required for a statistically significant benefit in partial recovery following traumatic spinal cord injury has yet to be fully elucidated by literature. Most papers show a likely benefit of a targeted MAP range in the first 48-72 hrs, with no improvement in recovery outcomes beyond that[2,3]. Further still, much of the data relies on patients with blunt traumatic injuries rather than penetrating trauma[4]. Despite this, many of the principles requiring MAP goals for patients with blunt spinal cord injuries are applied to the treatment of penetrating spinal cord injuries[5]. Generally, the patient population most susceptible to penetrating traumas, such as missile and stab wounds, are younger and unlikely to have medical comorbidities[6]. As such, they often require the use of vasopressors to maintain MAP goals in the immediate post-injury setting. The use of vasoactive medications, like any medication we use, is not fully benign and often has side effects that we must weigh against the benefit they provide to patient outcomes.

Vasopressin is often used as an adjunct to other vasopressors in the ICU for maintenance of blood pressure goals, mainly in the setting of septic shock. Vasoconstriction is mediated by V1 receptors that cause vasoconstriction of smooth muscle[7]. Vasopressin additionally works on renal V2 receptors via insertion of aquaporin 2 channels and a subsequent increase in free water reabsorption[7]. While it is beneficial to reabsorb free water in times of dehydration, exogenous vasopressin administration can be associated with hyponatremia. There have been case reports of patients with septic shock who have become hyponatremic after vasopressin administration, with resolution of symptoms following cessation of the offending agent[6,7,8]. Case reports in the trauma population with vasopressin and the side effect of hyponatremia are scarce.

Methods: A 40-year-old male presented to the trauma bay after sustaining a single gunshot wound with a single missile wound seen on his left upper back. Imaging was significant for left pulmonary contusion with associated hemopneumothorax, left first rib fracture, T2 incomplete spinal cord transection, T2 posterior vertebral body fracture, and a retained bullet in the right paraspinal muscles. The patient was brought to the surgical trauma ICU (STICU) immediately after initial trauma scans were complete.
Upon arrival at the STICU, the patient reported no significant medical history, no home medications, and social history significant for five beers a day. Exam showed intact sensation and motor function above the level of T4. Below T4, deep pressure to the perirectal and left posterior thigh region was intact. The neuro-spine service was consulted, and they recommended non-operative treatment with MAP goals over 90 mmHg for 72 hours after presentation.

The patient was initiated on a norepinephrine drip to maintain blood pressure goals. In addition, steroid therapy with dexamethasone 10mg every eight hours for 48 hours was initiated. The night after presentation, the patient's norepinephrine rate was climbing, and a vasopressin drip was added to decrease single pressor burden. 24 hours after addition of the vasopressin drip, the patient was found to be hyponatremic with a steep decrease from 135 to 127. The patient was worked up for other causes of hyponatremia including adrenal suppression from the stress dose of steroids, fluid status, and intrinsic renal causes. Urine osmolarity was found to be 783 and the overall clinical picture was consistent with vasopressin induced hyponatremia.

The vasopressin was stopped and the patient immediately started to autodiurese at a rate of iver 1L per hour if urine output for several hours. His hyponatremia subsequently resolved over the next 18 hours.

Results: N/a - Medically Challenging Case Report

Conclusions: Exogenous vasopressin administration can lead to hyponatremia. While case reports exist of vasopressin induced hyponatremia in the setting of vasodilatory shock, there are few case reports in the trauma patient population.

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