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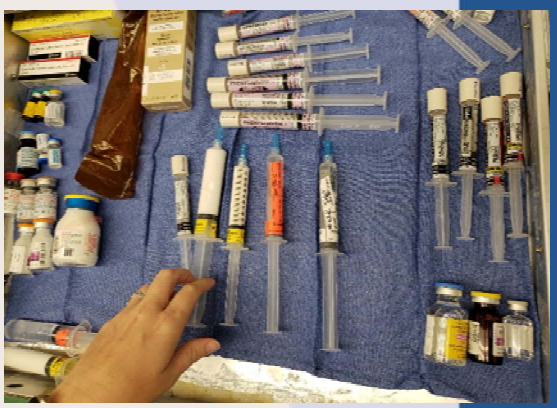
TOPICS IN THIS ISSUE



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Front Cover:
Yekaterina Shchapina, BSN, RN, a doctoral student enrolled in the University of Pennsylvania nurse anesthesia program prepares for a clinical practicum day in the operating room. Ms. Shchapina has a case report published in this issue of the ISJNA.

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Case Reports

Erector Spinae Plane Block for Mastectomies

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Keywords: Erector spinae plane block, mastectomies, postoperative pain, analgesia.

Mastectomies for breast cancer are known to cause chronic postoperative pain. Studies have suggested that using regional anesthesia and multimodal analgesia decreases stress and pain more effectively than the use of general anesthesia alone.[1] Therefore, methods to facilitate better regional techniques for these types of surgeries are encouraged for pain control. The erector spinae plane block (ESP) is a newer regional block that has shown promising results in controlling postoperative pain for mastectomies and minimizing the use of opioids.[2]

Case Report

A 87-year-old, 65 kg, 168 cm, Caucasian female presented with bilateral breast cancer scheduled for partial mastectomies. Past medical history included breast cancer, allergic rhinitis, insomnia, arthritis, and former smoker. Current medications included naproxen, zolpidem, and fluticasone. Preoperative laboratory tests were within normal limits. Pertinent results included: hemoglobin 13.1 g/dL, hematocrit 39.3%, platelets 371 K/uL, prothrombin time 13 seconds, international normalization ratio 1.2. An electrocardiogram showed sinus rhythm, and the chest x-ray was negative for any disease processes.

The anesthetic plan was to provide analgesia without the use of opioids, so it was agreed with surgeon, patient, and anesthesia practitioners that an erector spinae plane block would be performed and a propofol infusion used for intravenous sedation. As the patient entered the operating room, standard noninvasive monitors were placed, including a pulse oximeter, noninvasive blood pressure cuff, and three lead electrocardiogram. The patient was premedicated with midazolam 2 mg and dexamethasone 8 mg before being placed in a sitting position for the regional block.

A high-frequency linear array ultrasound transducer was positioned in the longitudinal plane, 2-3 cm lateral to the spinous process at the level of T5 as it was believed to adequately cover the T5 dermatome. Next, the trapezius, rhomboid major, and erector spinae muscles were identified with ultrasound as well as the hyperechoic transverse process. After local infiltration with 2% lidocaine 2 mL, a 4-inch insulated stimulating needle was inserted to perform the block. The needle was directed in-plane 1-2 cm cephalad to the transducer and advanced using a cephalad to caudad direction toward the transverse process until the needle was midline and touching the os. After negative aspiration, 1-2 ml of normal saline was injected to confirm needle tip placement between the erector spinae muscle and the transverse process. To mitigate intravascular injection of local anesthetic, 0.5% ropivacaine was administered 3-5 mL incremental doses for a total of 20 mL per side.

A propofol infusion was initiated at 60 mcg/kg/min and titrated for sedative effect. Initial surgical incision occurred thirty minutes after block placement. Throughout the procedure the

patient had minimal response to surgical stimulation with propofol infusion at a maximum rate of 120mcg/kg/min towards the termination of the surgery. The patient maintained a continuous sustained patent airway throughout the procedure with O_2 4 L/min and the aid of an oral pharyngeal airway. At the end of surgery, the patient was emerged and transported to recovery on O_2 4L/min. Upon arrival to the postoperative care unit, the patient had mild complaints of pain on a visual analog scale (VAS) of 3/10. The pain was described as achy and sore along the bilateral sternal borders. Ketorolac 30 mg and acetaminophen 1g IV were administered. After 45 minutes, the patient reported a VAS pain score of 0/10. The patient was ultimately discharged with minimal complications and pain.

Discussion

The combination of volatile anesthetics and opioids often display inadequate pain control in surgeries on or near the breasts.[1] Regional anesthesia, on the other hand, has been shown to control neuropathic pain as well as reduce cutaneous sensation of the breast derived from the intercostal nerves.[2] Also, anesthetics such as propofol and locoregional anesthesia, are known to increase natural-killer cell activity while decreasing surgery-induced neuroendocrine responses. The results are attenuated immunosuppression and recurrence of certain types of cancer compared to volatile anesthetics and opioids. Furthermore, lidocaine, ropivacaine, and bupivacaine are known to reduce mesenchymal stem cell (MSC) proliferation and have an innate ability to decrease metastatic spread in breast cancer cells.[3]

Currently the "gold standard" in breast surgeries is the paravertebral block (PVB), but the erector spinae block is beginning to be recognized as a comparable alternative as it is easy to perform.[4] Paravertebral blocks possess many critical drawbacks, including but not exclusive to increased risk of sympathetic blockade, and intrathecal or epidural spread. Block failure rates have been reported to be as high as 6.1%, and the incidence of inadvertent intravascular placement is 6.8%. The risk for pleural puncture and pneumothorax are reported to be 0.8% and 0.5% respectively.[5]

Pectoral nerve blocks (PECS) I and II have also been utilized for mastectomies. These fascial plane blocks provide significant anesthesia to the lateral mammary region, but lack internal mammary region coverage. In order to provide a greater field of pain relief, a combination of blocks should be considered. Unfortunately, as the ESP is a newer technique, there are limited studies that compare it to the PVB, PEC I and II blocks.

The erector spinae plane block works by interrupting transmission along the thoracic spinal nerves and sympathetic nerve fibers. The exact mechanisms are unknown, but it appears to be heavily dependent on the local anesthetic spreading to the paravertebral space. In cadaver studies conducted by Hershey and colleagues, fresh tissue was injected with methylene blue at the T5 level. This resulted in a spread from C7- T8 in a craniocaudal fashion and lateral spread extending to the tips of the transverse process and costotransverse junctions at the level of T3-T8.[1] Additionally, there was strong evidence that the dye penetrated the intercostal muscles near the ventral and dorsal spinal nerve roots, which would affect somatic and visceral pain signaling. Visceral pain has always been challenging to alleviate with pharmacotherapy alone; thus, leading to polypharmacy, chemical dependence, or addiction. The ESP, on the other hand, appears to

mitigate visceral pain at the central processing center, instead of ameliorating peripheral nociception - a critical aspect to consider amid the current opioid crisis.

The optimal plane for injecting local anesthetic for the ESP block is midline and deep into the erector spinae muscle, so more anesthetic deposits into the sheath near the ventral and dorsal rami.[1] With ultrasound assistance, the anatomy can be easily identified with minimal risks for needle injury as there are no structures in the vicinity that are subject to accidental penetration. Another advantage to this block is its suitability for the administration of liposomal bupivacaine which can impede the onset of pain up to 72 hours and the capability for the placement of indwelling catheters for the treatment of chronic neuropathic pain.[1]

Erector spinae plane block success has been proven in multiple cases where the administration of the block decreases pain for up to 9 hours.[6] In other research, the block has provided analgesic coverage until postoperative day 3. Gurkan and colleagues conducted a randomized control trial of 50 patients receiving ESP blocks and 24 hour morphine consumption monitoring. The results computed to 5.78 ± 3.8 mg of morphine administered in the group that received the ESP block and 16.6 ± 6.92 mg of morphine for the group without. These results demonstrated a 65% decrease in the consumption of morphine during the first 24 hours .[7] As these studies have shown, there are many advantages to the ESP block, and the pain control is significant. The ESP block can provide analgesia to a large area on the trunk of the body, so the potential for its use in thoracotomies, colectomies, hernia repairs, and even hip surgeries should be considered.

The ESP block has been recognized as a viable alternative to PVBs for breast surgeries. However, there is a lack of research regarding the optimal dosage and its efficacy compared with other regional methods. In this case study, it would have been optimal to follow up with the patient on postoperative days 2-3 following surgery to assess the severity of the pain and further evaluate the success of the block. For future research, studies should focus on the reproducibility of the block, optimal dose, and concentration to prevent local anesthetic toxicity, as well as its use in analgesic relief for other truncal surgeries. For this particular case study, as the patient still experienced bilateral sternal border pain, a combination of PECS and ESP may have provided a more complete and thorough anesthetic as well as for postoperative analgesia.

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2018.06.033.

Mentor: Nathan Adams, DNP, CRNA

Hay Bale Tractor Trauma

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Keywords: impaled trauma, exploratory laparotomy, hypothermia, lethal triad

Injury from trauma is one of the leading causes of death and disability in adults.[1] In-hospital hemorrhage is most likely to occur within six hours of admission for a trauma patient[1]. Mortality is greatly increased in patients with coagulopathy, which can be worsened by hypothermia and acidosis. Together, they create the "lethal triad of trauma."[1,2] The following report demonstrates the anesthetic management of a trauma patient impaled by a large metal spear.

Case Report

A 43-year-old male arrived via helicopter to the emergency department (ED) for an emergency exploratory laparotomy after being impaled by a large steel spear from a hay bale tractor fork. The object penetrated the posterior flank near midline and exited the anterior left lower quadrant of the abdomen. The patient's height was 170 cm and weight was 88 kg, with a calculated body mass index (BMI) of 30 kg/m.[2] Past medical history included being a former smoker, gastroesophageal reflux disease, benign prostatic hyperplasia, and a history of spinal meningitis 11 years ago. He had no known drug allergies. Surgical history included nasal surgery and tonsillectomy. The patient's history was obtained from his wife.

On arrival to the ED, the patient stayed on the stretcher in a right lateral decubitus position. The steel spear protruding through the lower left flank was held in place posteriorly by a registered nurse. The patient arrived to the hospital intubated with a stable SpO_2. He had two 18 gauge peripheral intravenous (IV) lines. The patient was sedated with midazolam at this time and attached to standard monitors which showed sinus tachycardia and blood pressures 170s/90s. Equal bilateral breath sounds and palpable dorsalis pedis pulses were present. Initial laboratory results were as follows: white blood cell count 16,000/uL, hemoglobin 11.2 g/dL, hematocrit

32.3g/dL, platelets 233,000/uLA. All other electrolytes were normal, except for calcium which was 6.8 mg/dL. Glucose was elevated at 135mg/dL.

The patient was taken to the operating room (OR) on the stretcher he arrived on and noninvasive monitors were reapplied. A left subclavian triple lumen intravenous line was established concurrently with a right radial arterial line before transferring the patient slowly to the OR table in the right lateral position.

The patient's endotracheal tube was connected to the anesthesia machine circuit and general anesthesia was maintained with sevoflurane in O_2 2.5 L/min. Expired sevoflurane concentration was 1.2 to 2% throughout the surgery. Upon urinary catheter insertion, temperature showed 34.8ºC. Antibiotics administered included: clindamycin 900mg, metronidazole 500mg, and vancomycin 1g. A tetanus vaccine was also given. Packed red blood cells (PRBCs), fresh frozen plasma, and platelets were in the OR ready to be transfused if needed.

A midline laparotomy incision was made by the surgeon. The iliac artery, vein, aorta, abdominal vena cava, and ureters were uninjured. The metal spear was removed slowly in a posterior direction by an OR nurse. The abdominal cavity was explored and a sigmoid colon resection with anastomosis and ileostomy were performed.

The case lasted 3 hours and had an estimated 300 mL blood loss. The patient was given a total of rocuronium 200 mg, sufentanil 25 mcg, midazolam 4 mg, normal saline 2000 mL, lactated ringers 2300 mL, and sodium bicarbonate 50 mEq. Vital signs remained stable throughout the case. The patient was taken to the intensive care unit (ICU) intubated and hypothermic at 35.8ºC despite warming measures, but in otherwise stable condition.

Discussion

This case describes a unique trauma case where the operating room team needed to be prepared for many potential problems. When removed, the metal rod was weighed at 40 lb. During the accident, it was attached to a hay fork and tractor loader. The attachment fell unexpectedly and went through the man in a posterior to anterior direction. The patient remained alert until he was intubated by emergency personnel. Co-workers used a heat torch to cut the fork off the loader and used special cooling gel to prevent thermal injury. Goals of managing this patient in the OR included avoiding the development of the triad of acidosis, hypothermia, and coagulopathy.[1]

Hypothermia is considered to be a core body temperature less than 35ºC.[1-4] Input from central and peripheral thermal receptors go the hypothalamus to stimulate heat production by shivering and increasing catecholamine, thyroid, and adrenal activity.[4] Sympathetic activity causes vasoconstriction which minimizes heat loss by reducing blood flow to peripheral tissues.[4] Other effects of hypothermia include reduced drug clearance, increased risk of infection, cardiac depression, and a blunted response to catecholamines.[3]

Heat loss for trauma patients can be from environmental exposure, cold intravenous fluids, hypovolemia, or from anesthetic drugs that interfere with thermoregulation.[1-4] Radiation contributes greatly to heat loss, therefore ambient room temperature was raised during surgery.[3]

The patient remained hypothermic with temperatures as low as 34.3ºC via urinary catheter temperature probe. An under, upper, and lower forced air warmer were set to the highest setting, 43ºC, and all fluids were given warm. While bladder temperatures are considered adequate in mild to moderate hypothermia (28 to 35ºC), they are slow to respond during rewarming.[4] The patient's core temperature may be increasing while the bladder temperature is still dropping.

Hypothermia can also cause abnormalities in coagulation by decreasing tissue factor activity, platelet aggregation, and platelet adhesion. Coagulation factor function decreases by about 10% for each 1ºC drop in temperature, ultimately slowing platelet plug and fibrin clot formation.[3] Platelet function and enzyme activity is further reduced with the severity of hypothermia.[3,5,6] Coagulation tests drawn in the OR showed a slightly elevated prothrombin time (PT) at 15 seconds and normal partial thromboplastin time (aPTT) at 29 seconds. It takes a 30-40% drop in factor activity for the aPTT/PT to be abnormal.[6] It is important to consider that blood samples drawn on hypothermic patients can be unreliable because they are tested pre-warmed at 37ºC in the laboratory, which may correct hypothermia-induced defects.[1,3] Thromboelastography was unavailable, but would be more reflective of platelet function and the patient's degree of coagulopathy if present.[3,5]

Acidosis contributes towards coagulopathy by interfering with the formation of thrombin. Thrombin is essential for activating platelets and for the conversion of fibrinogen to fibrin. A mixed respiratory and metabolic acidosis diagnosed by an arterial blood gas sample was corrected mid-case with intravenous fluids, increased ventilation, and 1 ampule of sodium bicarbonate.

It was important to achieve good intravenous access and monitoring before moving the patient to the OR table in case the impalement moved and caused injury to surrounding structures. The radial artery for intra-arterial pressure monitoring is a good choice in abdominal or chest trauma in case the aorta is cross-clamped, which would make a femoral arterial cannula nonfunctional.[1,2]

Intravenous fluids were given according to estimated blood loss, evaporative loss from a large open abdominal wound, and third-spacing from fluid redistribution. Hourly urine output, blood pressure, and heart rate were also used to guide intravenous fluids. Fluid requirements for a patient with moderate tissue trauma can be up to 6-8 mL/kg/hr on top of an hourly maintenance rate.[3] Some of the risks associated with infusing large volumes of fluid include dilution of coagulation factors, pulmonary/gastrointestinal edema, intra-abdominal hypertension, prolonged mechanical ventilation, and wound infections.[7] Restrictive fluid management has been associated with lower mortality rates when compared to patients where fluids were given more liberally.[7] For this reason, perhaps monitoring stroke volume variation with an arterial line would have been useful to help guide intravenous fluids. These parameters can predict fluid responsiveness with greater accuracy than central venous pressure or pulmonary artery occlusion pressure.[2,3]

The patient remained stable throughout the surgery and was transferred to the ICU for postoperative care. The patient was extubated 3 days later, neurologically intact. After multiple wound vacuum dressing changes, he was discharged home on post-operative day 15. Recognizing hypothermia, coagulopathy, and acidosis, and taking steps to control them is essential to decrease patient mortality.

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Mentor: James Stimpson, DNP, CRNA

Anesthesia Considerations for the Patient with Sturge-Weber Syndrome

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Keywords: Sturge-Weber syndrome, port-wine stain, staged laser treatment, vascular lesions, hemangioma, angiomatous leptomeninges

Sturge-Weber syndrome (SWS) is a nonhereditary congenital neurocutaneous disorder with an incidence of 1 in 50,000 births that roughly affects both sexes equally.[1-3] Typically, SWS involves the leptomeninges and the ophthalmic (V1) and maxillary (V2) distribution of the trigeminal nerve.[3] Classical presentation of SWS involves unilateral facial involvement of hemangiomas known as port-wine stains (PWS), progressive seizures, and ipsilateral glaucoma.[2] The anesthesia practitioner should maintain a high index of suspicion for oral hemangiomas that may only be discovered during laryngoscopy.[3] At the same time, it is important to maintain adequate seizure control and avoid increases in intraocular pressure (IOP) and intracranial pressure (ICP).[3]

Case Report

A 53-year-old, 173 cm, 72 kg male presented for staged laser treatment of vascular lesions of face, ears, and neck. The patient's clinical history was significant for SWS, seizures, cervicalgia, occipital neuralgia, hypothyroidism, glaucoma induced blindness, moderate cognitive impairment, obstructive sleep apnea, and gastroesophageal reflux. Facial presentation was significant for bilateral PWS from forehead to the neck region with marked left side involvement extending posteriorly to the ears. Airway exam revealed mild gingival purple-red hemangioma with poor dentition, Mallampati score of 3, adequate mouth opening, limited neck range of motion secondary to moderate cervicalgia, and a short, thick neck. However, previous anesthetic records documented successful direct laryngoscopy with a MAC 3 blade and Cormack-Lehane grade 2 view.

Past surgical history included multiple eye, dental, and gum surgeries as a result of soft tissue hyperplasia caused by hemangiomas. One year prior to the current surgery, he had the same procedure in an office-based setting where he developed seizures postoperatively. Due to concern for seizures, he was brought to the hospital for closer post-operative observation. The patient's current medications included lacosamide, levetiracetam, levothyroxine, pantoprazole, and pregabalin. A 12-lead electrocardiogram demonstrated sinus rhythm with heart rate (HR) of 63/min and right bundle branch block. All other laboratory results were within normal limits including therapeutic anticonvulsant serum levels.

Pre-induction vital signs included a blood pressure (BP) of 140/80 mm Hg, HR of 94/min, SpO_2 of 100% on O_2 2 L/min via face mask. Midazolam 2 mg intravenous (IV) was administered prior to anesthesia induction. After 4 minutes of preoxygenation, general anesthesia was induced using rapid sequence intubation technique with lidocaine 70 mg, propofol 120 mg, and succinylcholine 100 mg. Because of the proximity of the surgical field to the airway and the potential risk of airway fire, insertion of a 6.0 mm laser-resistant endotracheal tube (ETT) with traditional laryngoscopy was attempted, but unsuccessful due to engorged hypopharyngeal tissue. Video laryngoscopy was immediately performed; a Cormack-Lehane grade 2 view was noted and the trachea was intubated with a regular 7.0 ETT.

General anesthesia was maintained with sevoflurane 2.5% expired concentration in O_2 2 L/min. Cefazolin 2 g and acetaminophen 1 g was administered intravenously. Vital signs were stable throughout the procedure, ranging from 85/56 to 125/67 mm Hg, HR 57-79/min, and SpO_2 96-100%. At the end of surgery, the ETT was removed. The patient was transferred to the postanesthesia care unit; no perioperative complications occurred.

Discussion

The distinctive cosmetic feature of SWS is PWS caused by venous dilation of the ophthalmic (V_1) and maxillary (V_2) distributions of the trigeminal nerve. Facial hemangiomas are present at birth and tend to progress to larger ipsilateral facial areas as the patient ages.[4,5] The cutaneous hemangioma in this patient extended beyond the V1 and V2 distribution of the trigeminal nerve and involved the ipsilateral jaw and superior anterolateral neck.

In SWS, the embryonal vascular plexus around the cephalic plexus of the neural tube fails to regress by week nine of intrauterine life.[5] This commonly leads to formation of ipsilateral angiomas on the face, eye, and the space between the arachnoid and the pia mater (also called leptomeninges).[5] These vessels are characterized by abnormal blood flow patterns progressing to occlusions, thrombosis, tissue hypoxia, vascular steal phenomenon, and atrophy of surrounding tissues of the brain, eye, and facial skin.[5] Neurological manifestations may include seizures, blindness, contralateral symptoms of stroke, and progressive cognitive impairment.[4] The three principal manifestations of SWS are facial angiomas (PWS or facial nevus), followed by ocular degenerative changes manifested primarily as glaucoma, and lastly seizures which occur in up to 75% of cases within the first year of life.[4]

Anesthesia management begins with a thorough preoperative assessment of the airway, presence of oral hemangiomas, history of oral bleeding with or without surgery, history of seizures, use of anticonvulsants, identification of any neurological deficits, and degree of ocular involvement.[6] Extreme levels of anxiety or stress should be mitigated preoperatively by establishing good rapport with the patient, as well as incorporating sedatives when appropriate.[6] The anesthesia practitioner should make every effort to mitigate the emotional stress associated with the surgery because of the detrimental effects of elevated blood pressure. The effects of elevated blood pressure include increased IOP, increased ICP owing to enlargement of hemangiomas in the brain, and increased risk of airway bleeding during intubation.[6] Laboratory information should also include current therapeutic levels of anticonvulsant medication. When needed, anticonvulsant medication loading prior to surgery should be discussed with the surgeon and anesthesia team.[3] It is also important to assess for gingival hyperplasia which can be caused by chronic anticonvulsant medication use or the angiomatous proliferation of gingival tissue.[5]

In some patients, mask ventilation, and successful intubation may be challenging due to varying degrees of facial and oral deformities caused by chronic hypertrophy of hemangiomas.[6] When large facial hemangiomas with airway involvement are present, it is essential to avoid airway trauma. Increased airway edema and bleeding further complicate the process of securing the airway.[7] The above patient had only mild gingival hyperplasia, and a history of successful direct laryngoscopy. In spite of this, a video laryngoscope was made available and subsequently utilized. The management of the airway should include a high level of suspicion index for the possibility of a difficult airway.[6]

Tracheal intubation should follow adequate preoxygenation and adequate anesthetic level prior to airway manipulation to avoid increases in IOP and ICP.[6] Succinylcholine and ketamine are not recommended because of their effect on IOP and ICP.[6] However, the above patient already had blindness and no signs of denervation lesions; therefore, use of succinylcholine was considered appropriate. Propofol is a desirable drug for induction and emergence in these patients as it may reduce the incidence of bronchospasm and laryngospasm and lower the incidence of nausea and vomiting.[6] Neostigmine is not recommended due to increased salivation and postoperative nausea and vomiting . Additionally, anticholinergics are avoided in narrow angle glaucoma.[3,8]

The above patient was being treated for hypothyroidism with oral levothyroxine. It is important to evaluate for clinical signs of thyroid dysfunction. Hypothyroidism is known to be prevalent in SWS patients due to growth hormone deficiency; thereby, causing disruption of the

hypothalamic-pituitary axis.[1] The mechanism of this endocrine dysfunction in patients with SWS is not well understood, although it is hypothesized to be related to seizures and brain ischemia.[1]

Laser treatment of vascular lesions (PWS) is an important treatment modality in SWS. Early superficial laser treatments may help decrease the potential complications of reduced brain venous outflow through the PWS vessels.[2] However, due to the risk of airway fire the FiO_2 should be maintained as close as tolerable to room air. Utilization of laser-resistant tube is also recommended.

Patients with SWS may also exhibit stroke-like symptoms. Antiplatelet agents may be prescribed to SWS patients because of recurring thrombotic episodes. Therefore, regional anesthesia techniques should be avoided due to high risk of uncontrolled bleeding. Neuraxial block should also be avoided in SWS patients due to the possibility of enlarged venous plexuses in the epidural space. Neuraxial block could increase ICP and potentiate brain herniation.[3]

Important considerations when caring for patients with SWS include the risk of a challenging airway due to oral hemangioma, risk of airway fire during staged laser treatment, and perioperative seizures. Therefore, it is imperative that the anesthesia practitioner perform a thorough preoperative assessment and prepare a tailored anesthesia plan in order to optimize care of the patient with SWS.

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Opioid Sparing Technique for Colorectal Cancer Surgery

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Keywords: ketamine, transversus abdominis plane (TAP) block, non-steroidal anti-inflammatory drugs, multimodal analgesia, opioid sparing, colorectal cancer

Historically opioids have primarily been used to treat perioperative pain. Opioids can inhibit both cellular and humoral immune function in humans. Morphine is a proangiogenic and promotes breast tumor growth.[1] Angiogenesis is closely associated with the etiology and pathogenesis of several pathological conditions including tumor progression and metastasis.[2] Large opioid doses often correlate with prolonged hospitalization from untoward side effects such as: decreased gastric motility, respiratory depression, and postoperative nausea and vomiting (PONV). Multimodal pain management targeting different pain pathways is important in an opioid sparing technique. Adjuvants to opioids include, but are not limited to the following: ketamine, regional anesthesia, corticosteroids, and non-steroidal anti-inflammatory drugs.

Case Report

An 89-year-old, 50 kg, 160 cm female presented for an open partial colectomy. Past medical history included colon cancer, hypertension, and anemia. Current medications include ferrous sulfate 325 mg and amlodipine 5 mg. Her blood pressure was 180/90 mm Hg preoperatively. No medication was given preoperatively to manage her blood pressure.

The patient was transported to the operating room, standard noninvasive monitors were applied, and O_2 8 L/min was administered via face mask. Once end tidal oxygenation (EtO2) of 85% was achieved, intravenous (IV) induction with midazolam 1 mg, lidocaine 100 mg, propofol 75 mg, and rocuronium 50 mg was administered. The patient was ventilated with sevoflurane 2% inspired concentration for approximately 1.5 minutes. Esmolol 20 mg IV was administered and a drop in heart rate from 76 to 62/min was observed. Direct laryngoscopy was accomplished using a MAC 3 blade. The trachea was then atraumatically intubated with a 7.0 mm cuffed endotracheal tube (ETT) and taped at 21 cm at the lip. Respirations were controlled by a mechanical ventilator on a volume control setting. General anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of inspired air 1 L/min and O_2 1 L/min. Rocuronium was administered throughout the three hour case to maintain surgical neuromuscular blockade at 1 out of 4 on a train of four monitor.

A bilateral transversus abdominis plane (TAP) block with 20 mL of 0.9% normal saline and 10 mL of bupivacaine liposomal 133 mg was placed via ultrasound guidance for preemptive postoperative pain management. Under sterile conditions, an ultrasound machine with a 12-5 MHz linear transducer (Flex Focus 400 Anesthesia, BK Medical) was placed over the costal margin of the twelfth rib and superior iliac crest and moved slightly caudad. The three muscle layers: the external oblique, internal oblique and transverse abdominis were identified. A 100 mm 22-gauge needle was inserted lateral to the transducer in an in-plane direction. Once negative aspiration was confirmed, 1-2 mL of the diluted solution was administered to confirm

needle placement between the internal oblique and transverse abdominus muscle layers. Incremental doses of 5 mL were then administered for a total of 30 mL bilaterally. The dose administered was dependent on the patient's weight and body habitus.

Intraoperatively the patient remained hemodynamically stable. Intraoperative medication regimen included: ondansetron 4 mg IV, dexamethasone 10 mg IV, acetaminophen 750 mg IV, ketorolac 30 mg IV, and ketamine 50 mg IV.

Neuromuscular blockade was reversed with sugammadex 100 mg IV and flow of O_2 was increased to 10 L/min. The patient showed a 4/4 train of four count with sustained tetany and 3-5 mL/kg tidal volumes on spontaneous ventilation. The ETT was removed and the patient was transferred to the post anesthesia care unit (PACU) on O_2 8 L/min via face mask. Upon arrival to the PACU, the patient was following commands, pain free, and denied any nausea.

Discussion

Opioid sparing pain management of patients undergoing open abdominal colorectal surgery (CRS) can be challenging. The anesthesia professionals should be prepared for the challenges and complexity of the case which may exceed three hours. It is not uncommon to see excessive opioid use for pain management in patients undergoing CRS. Untoward side effects of opioids may be avoided by multimodal pain management. Although many institutions utilize patient controlled intrathecal catheters, it was not an option. Pain management for this patient was achieved by use of opioid sparing techniques which included but were not limited to a TAP block and ketamine IV.

A TAP block is an effective technique to reduce postoperative pain and opioid consumption during lower abdominal surgery. A reliable blockade of dermatomes T10-L1 can be achieved by guiding the needle with ultrasound and injecting a local anesthetic into the plane between the transversus abdominis and internal oblique muscles. In a systematic review comparing nine studies that included 413 patients, those who received a TAP block showed a reduced morphine requirement in a 24 hour period.[3] Incidents of PONV at 48 hours were also significantly reduced.[2] A meta-analysis compared seven randomized controlled trials that included 511 patients. The results indicated that patients who had CRS, and received TAP block, had significantly reduced postoperative pain and opioid consumption during post anesthetic recovery at rest and during movement.[4]

Ketamine was introduced into clinical practice in 1970 and has undergone dramatic fluctuations in popularity over the years. Today, ketamine-induced dissociative anesthesia has many unique advantages as an alternative anesthetic.[5,6] Recent research has found ketamine to have neuroprotective, anti-inflammatory and antitumor effects. The usefulness of low dose ketamine regimens have helped widen its clinical application.[6] Ketamine is a noncompetitive antagonist of N-methyl D-aspartic acid (NMDA) receptor preventing afferent pain transmission to the cortex. In addition, it also interacts with opioid receptors, monoamine, cholinergic, purinergic, and adrenoreceptor systems as well as having local anesthetic effects.[6] Intraoperative use of ketamine in multimodal opioid sparing techniques has been shown to reduce the need for opioids within

the first 24 to 48 hours postoperatively.[7] In a qualitative systematic review by Subramaniam, a single bolus dose of ketamine decreased opioid requirements in seven of 11 studies.[8]

Significant adverse events are a common problem when providing analgesia with opioids. Anesthesia professionals need better analgesic options for pain management such as a multimodal approach. Albeit the patient in this case report did very well, I would add a lidocaine infusion and intravenous magnesium to my future practice. A perioperative infusion of lidocaine reduces postoperative pain and speeds the return of bowel function. Magnesium is a well-known adjuvant to reducing pain by antagonizing N-methyl-D-aspartate (NMDA) receptors.

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Airway Management in Patients with Acromegaly

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Keywords: airway management, acromegaly, gigantism, awake fiberoptic intubation, difficult intubation

Patients diagnosed with acromegaly have a number of comorbidities with substantial anesthesia-related implications. The unregulated secretion of growth hormone seen in acromegaly results in hypertrophy of facial bones and supportive anatomical structures of the airway. This often results in prognathism, macroglossia, and reduction of the glottic inlet size.[1] These characteristics of the acromegalic airway make the alignment of laryngeal and pharyngeal axes difficult for purposes of laryngoscopic visualization. The incidence of difficult laryngoscopy is estimated to be anywhere from 20 to 34% in this patient population.[2] Therefore, judicious anesthetic management is imperative.

Case Report

A 63-year-old female presented for diagnostic dilation and curettage due to post-menopausal bleeding. The patient weighed 120 kg and was 163 cm tall with a body mass index (BMI) of 44.7 kg/m^2. Her preoperative vital signs were: blood pressure (BP) 107/68 mm Hg, heart rate (HR) 71/min, normal sinus rhythm (NSR), respiratory rate (RR) 14/min, and SpO_2 94% on room air. The patient's pertinent past medical history included diabetes mellitus type II, mixed connective tissue disease, hypertension, hyperlipidemia, fibromyalgia, hepatitis C, pituitary tumor, obstructive sleep apnea (OSA), and acromegaly. The patient also had a 20 pack year history of smoking, and successfully quit tobacco use in 2006. The patient's past surgical history included two caesarean sections, tonsillectomy, and pituitary tumor removal, without anesthesia-related complications. The patient's medications were: atorvastatin, budesonide/formoterol, bumetanide, insulin, losartan, metformin, and montelukast. The patient's allergies included penicillin and sulfa with no prior history of anaphylaxis.

The patient stated she was unaware of her documented difficult airway history on the electronic medical record (EMR). Given this information, the decision was made to have her airway assessed independently by three anesthesia practitioners in order to achieve consensus on her airway assessment. Each practitioner rated her airway as a Mallampati Class IV. Based on the airway assessment and history of OSA and acromegaly, the decision was made to proceed with an awake fiberoptic intubation. To facilitate airway topicalization, the patient was given gauze saturated with 4% lidocaine ointment affixed to a tongue depressor to self-administer orally over the course of 30 minutes. The patient was given midazolam 4 mg, glycopyrrolate 0.2 mg and a loading dose of dexmedetomidine 116 mcg IV infused over 10 minutes in preparation for the awake fiberoptic intubation.

Upon entrance to the operating room, the patient was transferred to the operating room table and standard monitoring devices were applied. Her head was elevated to a 30-degree angle to optimize the patient's functional residual capacity while O_2 12 L/min was administered by face

mask during pre-oxygenation. A dexmedetomidine infusion was initiated at 0.2 mcg/kg/hr. After 5 minutes of normal tidal volume breathing, a transtracheal block was administered using 4% lidocaine 4 mL. A pediatric fiberoptic bronchoscope was inserted orally by one anesthesia practitioner while a second anesthesia practitioner manually retracted the tongue. Once the full glottic aperture was in view, 2% lidocaine 2 mL was instilled through the working channel port on the bronchoscope. The endotracheal tube (ETT) was then advanced over the bronchoscope and through the glottis. Further confirmation of ETT placement was attained through auscultation of bilateral breath sounds and a positive end-tidal carbon dioxide tracing. The ETT was secured at 22 cm at the lip. A propofol infusion was started at 30 mcg/kg/min and the dexmedetomidine infusion was increased to 0.3 mcg/kg/hr. Once it was confirmed that the patient had lost her lid reflex, the surgical team proceeded with positioning and sterile preparation of the patient.

The patient did not receive any neuromuscular relaxants or opioids during the procedure. At the end of the procedure, all intravenous infusions were discontinued. The ETT was removed once consciousness was fully regained, and the patient consistently followed commands. Oxygen 15 L/min via non-rebreather mask was administered to the patient after extubation. Her postoperative vital signs were: BP 116/68 mm Hg, HR 68/min in NSR, RR 17/min, and SpO_2 98%. Her vital signs and condition remained stable throughout her post-anesthesia care unit stay. Once all supplemental O_2 was discontinued, the patient was discharged to home.

Discussion

Acromegaly is a disorder which results from hypersecretion of growth hormone (GH) due to, in 95% of cases, a benign pituitary adenoma.[3] The excess of circulating GH results in stimulation of the liver to produce insulin-like growth factor 1 (IGF-1), resulting in excessive bone growth, soft tissue hypertrophy, and water retention.[4] Of particular concern to the anesthesia practitioner is the problematic airway due to macroglossia, thickened soft palate and uvula, extended mandible, pharyngeal tissue hypertrophy, soft tissue edema around the glottis, cervical vertebra hyperostosis, and neck movement disorders.[3] Consequently, incidence of difficult laryngoscopy is a consideration in this population. [4]

The predictive value of difficult laryngoscopy from assessment tools such as the modified Mallampati (MMP) and extended Mallampati score (EMS) has been thoroughly examined in the literature. The EMS tool adds craniocervical extension to the MMP.[5] According to Bindra et al,[5] no additional predictive value of EMS was found when compared to using the MMP alone. However, the investigators suggested incorporating both tests into the preoperative evaluation.

While the MMP and EMS are valuable airway assessment tools, there is a possibility for interrater variability. Therefore, researchers have sought for more objective means to predict difficult laryngoscopy in acromegaly patients. Lee et al[2] hypothesized that difficult laryngoscopy could be predicted using radiographic indices indicating tongue size with skull x-ray and computed tomography (CT). Measurements such as tongue area on CT, linear distance from alveolar line of mandible to hyoid bone, and linear distance from interior border of mandible to hyoid bone were gathered retrospectively on patients with acromegaly.[2] The investigators noted a significant correlation ($P<.05$) with these characteristics and values and the incidence of

difficult laryngoscopy.[2] In another study, Zhang et al[3] obtained various hormone assays. While most hormone levels did not vary among the study population, fasting IGF-1 values were significantly (P<.001) higher in difficult laryngoscopy patients, leading to recommendations for their use in preoperative airway assessment of acromegaly patients.

Careful attention was given to preoperative airway evaluation of this patient. Owing to the patient's history of acromegaly and difficult larygoscopy, tracheal intubation via an awake fiberoptic was planned. If the patient did not have a history of difficult airway, video laryngoscopy might have been considered. Assessment of the patient's airway using the EMS was not employed in this case nor were radiographic indices of the patient's tongue size and serum IGF-1 values obtained preoperatively. No matter what assessment techniques are employed, a thorough preoperative airway assessment along with adequate preparation and anesthesia practitioner expertise are essential for successful acromegalic airway management.

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Vasoplegic Syndrome Treatment during Cystoscopy

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Keywords: Vasoplegic syndrome, vasopressin, hypotension, ACE Inhibitors, cystoscopy

Patients taking Angiotensin Converting Enzyme Inhibitors (ACE-Is) are at risk for intraoperative hypotension refractory to adrenergic vasopressors. Blood pressure is maintained by three separate systems: autonomic system, renin-angiotensin system (RAS), and the vasopressin system.[1] The induction of general anesthesia blunts the autonomic response.[1] Therefore, patients

who undergo general anesthesia and take ACE-Is must rely solely on the vasopressin system for blood pressure control. Vasoplegic Syndrome (VS) is associated with an increased morbidity and mortality.[2] This case demonstrates the successful identification of a patient with VS and prompt intervention with vasopressin to minimize the duration of intraoperative hypotension.

Case Report

A 61-year-old, 170 cm, 125 kg female with a body mass index of 43.3 kg/m^2 presented for a cystoscopy with insertion of a right ureteral stent to relive a ureteral stricture. Her past medical history was significant for morbid obesity, hypertension, hypothyroidism, depression, and endometrial cancer. Her surgical history included eye surgery and a hysterectomy. After her hysterectomy, she was informed of severe hypotension that transpired during the perioperative period and was subsequently diagnosed with VS. The patient's medication regimen included once daily doses of citalopram 20 mg, levothyroxine 75 mg, lisinopril-hydrochlorothiazide 10-12.5 mg, and a multivitamin. The last dose of these medications was taken the morning of surgery. Patient's medication allergies included penicillin and sulfadiazine. A 12-lead electrocardiogram (EKG) was completed and revealed sinus tachycardia with no ST segment abnormalities. All laboratory values were within normal limits. No further preoperative studies were performed.

The day of surgery, preoperative examination included stable vital signs of blood pressure (BP) 148/77 mm Hg, heart rate (HR) 106/min, respiratory rate (RR) 20/min, and SpO_2 99% on room air. Her airway assessment was unremarkable. A 20-gauge intravenous (IV) catheter was placed in her left hand. In the preoperative area, the patient expressed feeling anxious and was premedicated with midazolam 2 mg IV. Upon arrival to the operating room, standard noninvasive monitoring was instituted and preoxygenation was completed with O_2 10 L/min delivered via facemask for 5 minutes. General anesthesia was induced with lidocaine 60 mg and propofol 200 mg IV. During induction, the patient's BP ranged from 70/41 to 73/41 mm Hg, with a mean arterial pressure (MAP) of 52 mm Hg. Vasopressin 2 units was administered 3 minutes apart to maintain MAP greater than 65 mm Hg.

Secondary to the anticipated short duration of the procedure, the decision to place a laryngeal mask airway (LMA) was made. Successful mask ventilation was verified, a size #5 LMA was placed without incident, and positioning was confirmed with $ETCO_2$ capnography. Spontaneous ventilation returned within 2 minutes of induction. General anesthesia was maintained with sevoflurane 2% expired concentration in a mixture of O_2 1 L/min and air 1 L/min. The patient's legs were placed simultaneously in lithotomy position with arms supported on padded arm boards at 75 degrees. Prior to procedure start, fentanyl 50 mcg, and aztreonam 2 g were administered IV. Ten minutes into the case, the patient's blood pressure decreased to 66/53 (57) mm Hg after an administration of fentanyl 50 mcg. Vasopressin 2 units was administered and the patient's blood pressure returned to baseline. Additional doses of vasopressin 1 unit were administered at the 21 and 34 minute marks for MAP less than 60 mm Hg to which the patient responded accordingly. Additionally, sevoflurane was titrated down to an expired concentration of 1.5%. A total of lactated ringer's 600 mL was administered intraoperatively during the case.

Overall surgical time was 50 minutes. After completion of the procedure, oxygen flow was increased to 100% O_2 at 10 L/min. Ondansetron 4 mg was administered IV for postoperative nausea and vomiting prophylaxis. Once the patient maintained an average tidal volume of 500 mL, RR of at least 10/min and was responsive to commands, the LMA was discontinued. The patient was transferred to the post-anesthesia care unit (PACU) on O_2 4 L/min via nasal cannula for monitoring. Vital signs in PACU were BP 106/65 mm Hg, HR 88/min, RR 18/min, SpO_2 96% on 4L nasal cannula. The patient remained hemodynamically stable and no additional vasopressors were administered during her time in PACU.

Discussion

Vasoplegic syndrome is defined as refractory hypotension under general anesthesia with a MAP less than 50 mm Hg, a cardiac index greater than 2.5 L/min per m^2, and low systemic vascular resistance, despite adrenergic vasopressor administration.[1] It is most commonly associated with cardiac surgery with persistent hypotension following the cardiopulmonary bypass period. However, VS can occur during any surgery and following any anesthetic administration.[1] The development of VS includes 8-10% in patients undergoing cardiac surgery and upwards of 50% in patients that take ACE-Is and Angiotensin II Receptor Antagonists (ARAs).[1] Other potential risk factors that predispose patients to VS include blood transfusions, organ transplantation, trauma, sepsis, and the use medications such as heparin, amiodarone, aprotinin and protamine.[2]

The proposed mechanism for VS is attributed to the inhibition of the body's blood pressure regulatory mechanisms. Under normal physiologic conditions, the body is able to regulate its blood pressure via the autonomic, RAS and vasopressin systems. The induction of general anesthesia inhibits the autonomic nervous system (ANS) via the administration of volatile, hypnotic and amnestic agents.[3] Blunting the ANS renders commonly used vasoactive agents such as phenylephrine and ephedrine ineffective as these medications require adequate levels of circulating catecholamines to elicit a physiologic response. Additionally, patients who take ACE-Is or ARAs inhibit the RAS and must rely on vasopressin system alone for maintaining cardiovascular tone.

The current guidelines set forth by The American College of Cardiology and the American Heart Association in 2014 recommend continuing ACE-Is and ARAs up to and including the day of surgery.[4] However, this practice remains controversial. A retrospective cohort study that gathered data from six Veterans Affairs medical centers demonstrated that intraoperative hypotension was associated with a statistically significant increase in 30-day postoperative mortality.[5] A subsequent prospective international cohort study that included 16,079 patients from eight countries examined whether withholding ACE-Is and ARAs was associated with a 30-day decreased incidence of myocardial infarction (MI), stroke and death following non-cardiac surgery.[6] It was found that patients that withheld their ACE-Is and ARAs for 24 hours prior to surgery had a lower incidence of death and adverse vascular events.[6] The study concluded that if all patients who continue to take ACE-Is /ARAs on the day of surgery were to instead withhold them, over 500,000 patients per year would avoid death, MI, or stroke within 30 days of their operation.[6] This new literature could guide clinicians in their care of patients in the preoperative period.

In the case discussed above, the patient took her ACE-I/thiazide diuretic combination pill the morning of surgery. Her risk for hypotension was increased by the ACE-I as well as the potential volume depletion caused by the diuretic intake. Due to her existing history of VS, vasopressin was prepared preoperatively. A 0.5-1.0 unit bolus of vasopressin may be given to treat hypotension, followed by an infusion dose of vasopressin 0.03 units/min or terlipressin 1-2 mcg/kg/hr.[1] In this case, an infusion was not started due to the short duration of the procedure as well as the patient's responsiveness to vasopressin boluses. In retrospect, an infusion would have been favorable to promote hemodynamic stability. Decreasing the anesthetic agent, minimizing opioid administration and volume expansion may also aid in the treatment of VS.[1]

Lastly, methylene blue may be used as a rescue therapy given as a bolus dose of 1-2 mg/kg/hr over 10 to 20 minutes followed by an infusion of 0.25 mg/kg/hr for 48 to 72 hours, with a maximum dose of 7 mg/kg.[1] Methylene blue is thought to inhibit the nitric oxide-cGMP pathway, thus facilitating the constriction of vascular smooth muscle.[7] However, methylene blue is associated with worsening arterial oxygenation, pulmonary vasoconstriction, decreased renal and mesenteric blood flow and serotonin syndrome in patients concurrently taking serotonin reuptake inhibitors (SSRIs).[7] Despite this, in patients undergoing coronary artery bypass grafting, the use of methylene blue has been shown to decrease the incidence of VS.[8] The effect of this in patients undergoing noncardiac surgery has not yet been replicated. Due to the patient's SSRI intake the morning of surgery, methylene blue was not considered for this case.

This case report demonstrates the successful identification of a patient with VS and prompt intervention by the anesthetist. A thorough patient history, including medication regimen, will allow the anesthetist to recognize patients at significant risk for VS. Perioperative hypotension is multifactorial problem that is correlated with the use of ACE-Is and ARAs. Initiating volume expansion preoperatively as well as the preparation of necessary vasopressors can mitigate the duration of hypotension experienced by the patient. An infusion of vasopressors should be considered to maintain a steady-state concentration and adequate MAP. Conventional therapies, vasopressin and methylene blue should be available for rapid administration. More research is needed to develop a standardized, evidence-based approach for the treatment of VS.

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Management of Achondroplastic Patient Undergoing Robot-Assisted Hysterectomy

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Keywords: Achondroplasia, dwarfism, robot-assisted surgery, hysterectomy, difficult airway

Achondroplasia is the most common cause of disproportionate dwarfism.[1] It occurs predominantly in females, with an incidence of 1.5 per 10,000 births.[1] Anesthetic management of individuals with achondroplasia is complicated because of their physical characteristics.[1] Robot-assisted surgery for achondroplasia patients can be especially challenging due to the lack of clear evidence-based guidelines. Management of anesthesia in patients with achondroplasia is influenced by potential airway, ventilation, and surgical positioning complications. This case report describes the anesthetic management for a patient with achondroplasia undergoing an elective robot-assisted hysterectomy and bilateral salpingectomy.

Case Report

A 42-year-old, 127 cm, 53 kg, female presented for robot-assisted hysterectomy and bilateral salpingectomy related to uterine fibroid and abnormal uterine bleeding. The patient's past medical history included pulmonary embolism, spinal stenosis, neurogenic bladder, constipation, depression, and anxiety. Her past surgical history was significant for cervical and thoracolumbar decompression and fusion. A current list of medications included alprazolam, aspirin, clonazepam, escitalopram oxalate, and ibuprofen. The physical exam revealed a Mallampati grade 2 airway, macroglossia, prognathism, large mandible, short maxilla, and limited neck extension. Basic metabolic panel and complete blood count values were within normal limits. The anesthetic plan considered the potential for difficult ventilation and intubation as well as cervical spine precautions.

A 20-gauge intravenous (IV) catheter was placed into the patient's right hand by the preoperative nurse. The patient received midazolam 2 mg IV as sedative prior to entering the operating room (OR). When the patient entered the OR, standard noninvasive monitors were applied prior to positioning. The patient was assisted to the supine position. A shoulder roll was placed under the patient's shoulder blades in this case in order to achieve a neutral head position.

The patient was pre-oxygenated with O_2 10 L/min via a pediatric facemask. General anesthesia was induced with propofol 150 mg IV, fentanyl 100 mcg IV, and lidocaine 40 mg IV. Bag-mask ventilation was met with difficulty. After placement of an 80 mm oropharyngeal airway, successful ventilation was achieved with two-handed mask ventilation. Rocuronium 40 mg IV was administered and video laryngoscopy was performed with a GlideScope (Verathon Inc., Bothwell, WA). A 7.0-cuffed endotracheal tube (ETT) was placed and the position was confirmed by end-tidal capnography and bilateral lung auscultation. The patient was placed on volume-controlled mechanical ventilation with 6 ml/kg tidal volume and 5 mm Hg positive-end expiratory pressure (PEEP). Anesthesia was maintained with propofol infusion of 100 mcg/kg/min IV, lidocaine infusion of 1.5 mg/kg/hr IV, and hydromorphone boluses of 0.2-0.4 mg IV. Both bispectral index (BIS) and train-of-four monitors were utilized throughout the procedure.

The patient was repositioned in the lithotomy position with arms tucked and placed in steep Trendelenburg. Carbon dioxide was insufflated into the peritoneal cavity to a pressure of 15 mm Hg. The patient's vital signs were stable and peak inspiratory pressure was maintained less than 30 cm H2O. The surgery was prolonged by 30 minutes due to unfitting instruments and robotic positioning difficulty. The patient was maintained in steep Trendelenburg with insufflation while the surgeon was troubleshooting procedural instruments. Upon completion of the procedure, the propofol infusion was titrated off and lidocaine infusion was discontinued. After four out of four twitches without any fade were noted, neuromuscular blockade was antagonized with glycopyrrolate 0.6 mg IV and neostigmine 3 mg IV. Ondansetron 4 mg IV was given for antiemetic prophylaxis. Prior to extubation, the ETT cuff was deflated and cuff leak was confirmed. The ETT was removed after the patient appropriately followed commands and completed standard extubation criteria. The patient was transported to post-anesthesia care unit without supplemental O_2. The patient was alert with unlabored respiration and adequate pain control.

Discussion

Achondroplasia is a common cause of dwarfism, a condition whereby an individual's arms and legs are short in proportion to his or her body length. It is known that mutations in the fibroblast growth factor receptor 3 (FGFR 3) gene cause achondroplasia.[1] The defect in the FGFR3 gene decreases the rate of endochondral ossification that, when coupled with normal periosteal bone formation, produces short tubular bones.[1] Characteristic features of a patient with achondroplasia include small stature, short arms and legs with particularly short upper arms and thighs, limited range of motion at the elbows, and enlarged head with prominent forehead and occiput. In addition, the premature fusion of bones at the base of the skull can result in a shortened skull base and stenosis of the foramen magnum.[1]

Over the past decade, robotic surgery has gained in popularity.[2] The anesthesia-related management of achondroplasia patients undergoing robot-assisted surgery has not been described in the literature. Airway management is one of the greatest concerns for achondroplasia patients undergoing general anesthesia.[1,3,4,5] The presence of a large head, short neck, large tongue and mandible, and flat nose can pose difficulties with mask ventilation.[3] Due to the prominent occiput, neck hyperflexion occurs in achondroplasia patients when they are placed in the supine position, thus compressing the soft airway and resulting in upper airway obstruction.[4] A shoulder roll can be useful to help achieve optimal positioning for tracheal intubation.

Tracheal intubation can be difficult in patients with achondroplasia because of the inability to align the axes of the airway.[3-5] Hyperextension of the neck during direct laryngoscopy must be avoided because there is a high risk of foramen magnum stenosis.[1] For this reason, patients with achondroplasia may need to undergo a number of spinal surgeries.[1] In this case, the patient's past surgical history included cervical and thoracolumbar decompression and fusion. The literature suggests that awake fiberoptic intubation under airway block, in conjunction with topical anesthesia, is the safest option to secure the airway.[3-5] The video laryngoscopy, such as the Glidescope (Verathon Inc., Bothwell, WA), has been demonstrated to be useful in achondroplasia patients owing to its ability to facilitate superb laryngeal visualization without hyperextension of the neck.[5] The literature recommends the use of a smaller sized ETT than predicted by age for achondroplasia patients.[3] In this case, appropriate ETT size and length of ETT insertion were guided by laryngeal visualization using the GlideScope (Verathon Inc., Bothwell, WA). A fiberoptic bronchoscope was readily accessible as a backup technique after video laryngoscopy.

Robot-assisted surgery is associated with a high incidence of respiratory complications, including pulmonary atelectasis, pleural effusion, and V/Q mismatch.[2] Achondroplasia patients are especially at risk. Rib hypoplasia, a flattened rib cage, and pectus excavatum are common in achondroplasia patients, and can reduce their functional residual capacity, lung compliance, and ventilatory mechanics.[3] The use of steep Trendelenburg positioning and the creation of a pneumoperitoneum, required for robot-assisted surgery, may further decrease pulmonary compliance and functional residual capacity.[2] It is suggested that mechanical ventilation with a high respiratory rate, low tidal volume and high PEEP is an appropriate and safe strategy to use for achondroplasia patients. [3] The use of steep Trendelenburg positioning for a prolonged period can lead to upper airway edema. Furthermore, obstructive sleep apnea is prevalent in a large percentage of achondroplasia patients.[3] Awake extubation is preferred in these patients.

Patient positioning is a critical part of any robot-assisted surgery. The leg stirrups used to achieve the lithotomy position in adults were not suitable for use with a smaller physique. Pediatric leg stirrups were not readily available. Similarly, robotic instruments and equipment are generally designed for adults. The pediatric equivalent is not currently readily available. The robotic arms had limited space for movement owing to the small size of the patient's abdomen and pelvis. The robot had to be repositioned multiple times to determine the optimal operating space for the surgeon. The difficulties encountered with positioning significantly prolonged the surgery,

thereby exposing the patient to increased time not just with anesthesia, but also in steep Trendelenburg position with insufflation.

Robot-assisted surgery in achondroplasia patients should be approached with caution. The facility had very limited experience in this robot-assisted case because the amount of care administered to patients with achondroplasia at this institution is infrequent. However, this challenge can be mitigated by discussions among members of the multidisciplinary team prior to the patient's scheduled surgery.

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Myocardial Injury After Non-Cardiac Surgery

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Keywords: myocardial infarction, perioperative, myocardial injury after non-cardiac surgery, MINS

Myocardial injury remains one of the leading causes of post-operative mortality. After non-cardiac surgery, approximately 8% of patients develop signs of cardiac injury with a 10% mortality rate at 30 days. Perioperative analgesia can mask signs of myocardial damage, and 84.2% of affected patients will demonstrate no clinical indicators of ischemia.[1] Treatment strategies become challenging since clinicians must weigh competing goals of preventing thrombus formation while preventing surgical site bleeding.

Case Report

An 87-year-old, 83 kg, 175 cm female patient presented to the operating room on hospital day (HD)# 9 for a laparoscopic cholecystectomy due to persistent abdominal pain (5-8/10) despite antibiotic therapy, pain medications, and an endoscopic retrograde cholangio-pancreatography

under general anesthesia for gallstones on HD #4. The patient's medical history included diabetes mellitus type 2, hypertension, hyperlipidemia, and an unspecified hypercoagulation disorder leading to chronic deep vein thrombosis. Her surgical history included a lung resection from histoplasmosis and a carotid endarterectomy. Two years prior to admission, the patient was hospitalized with Guillian-Barre syndrome and continued to have generalized residual muscle weakness. Chronic medications included metformin, warfarin, and atorvastatin, all discontinued upon hospital admission.

The patient's hospital course was significant for chest pain on HD #1. Sublingual nitroglycerin 0.4mg was administered and the EKG showed no changes compared to baseline. Troponin T levels were negative for myocardial infarction (MI). On HD #3, the patient developed paroxysmal atrial flutter with a ventricular rate of 120/min. A transthoracic echo showed an ejection fraction > 70%, trace mitral and tricuspid regurgitation, and a mildly dilated left atrium. A venous duplex was negative for deep vein thrombosis. Troponin T levels were elevated to 20.7 ng/L, and a second troponin T was 18.5 ng/L. The patient was placed on an amiodarone infusion and developed one more episode of atrial flutter before returning to sinus rhythm.

During the patient's urgent laparoscopic cholecystectomy, standard noninvasive monitors were placed and induction of anesthesia was achieved via lidocaine 40 mg, propofol 30 mg, fentanyl 50 mcg, etomidate 6 mg, and rocuronium 45 mg. A 7.0 mm endotracheal tube was placed via video laryngoscope with a grade I airway and clear oropharynx. End-expiratory desflurane 5% was administered in a mixture of O_2 2 L/min and air 1 L/min. After intubation, a radial arterial line was placed along with 2 large bore IV catheters in the arms. An orogastric tube was placed and put on intermittent suction. No stomach contents were aspirated in the orogastric tube. A baseline arterial blood gas showed mild respiratory acidosis corrected by increasing the respiratory rate. Within 10 minutes of induction of anesthesia, the patient's systolic blood pressure dropped from 170 mm Hg to 110 mm Hg, which was then treated with calcium chloride 1g and incremental doses of ephedrine, totaling 45 mg. Throughout the surgery, the heart rate ranged between 50 and 70 beats per min. No episodes of cardiac arrhythmias occurred intraoperatively. After a 2 hour surgery, the patient was transferred to ICU with an F_{IO2} of 50%. Vital signs remained stable, and no further drugs were needed to support blood pressure. She was extubated the following day. Preoperative and post-operative laboratory values remained unremarkable.

On POD #2, the patient reported chest pain stating, "I feel like I am dying." Serial troponin T values were 63.9 ng/L, 75.5 ng/L, and 50.7 ng/L. A repeat EKG showed no changes compared to baseline. Chest x-ray showed pulmonary edema. Sputum cultures were sent for suspected aspiration pneumonia, and the patient was started on ceftriaxone antibiotics. Sputum cultures were positive for oxacillin-resistant staphylococcus aureus. The patient remained on telemetry monitoring for 4 more days before she was discharged home on antibiotics. Three weeks after discharge, an outpatient follow-up cardiology appointment documented no further episodes of chest pain or tachycardia, and the EKG remained unchanged. A subsequent x-ray showed continued pulmonary infiltrates, and the patient was managed with outpatient antibiotic therapy.

Discussion

Cardiovascular complications cause 1 in 3 deaths after surgery.[2] Under anesthesia, a heart is exposed to unusual stressors: drastic hemodynamic shifts, catecholamine release, sympathetic stimulation, and anesthetic medications. It is unclear whether anesthesia simply unmasks underlying cardiac disease, or whether it causes direct harm to the heart. Research has attempted to explore the complicated relationship between the heart and anesthesia. It is imperative for the anesthesia provider to understand the current research and adapt practice to minimize risk as best possible until clear guidelines are established.

Myocardial injury after non-cardiac surgery (MINS) is simply defined as cardiac troponin (cTn) levels exceeding the 99th percentile upper reference limit, with or without symptoms.[3] Lab values vary per manufacturer and test generation.[4] By contrast, the universal definition of myocardial infarction (MI) requires biomarkers, ischemic symptoms, and electrocardiogram changes in order to meet diagnostic criteria.[3]

Although causation of MINS is likely multifactorial and related to the patient's underlying conditions, one retrospective analysis of 57,345 patients found a strong link between episodes of intraoperative hypotension and subsequent MINS. While the common and accepted practice is to keep blood pressure within 20% of the patient's baseline values, Salmasi et al found that fluctuations relative to the patient's baseline did not correlate with MINS. Instead, maintaining mean arterial blood pressure above the absolute value of 65 mmHg corresponded to a reduced risk of MINS - even a minute of lower mean arterial blood pressures increased the risk of myocardial injury.[5] Further research could identify causation rather than simply correlation; however, it may be prudent for the anesthesia provider to maintain higher blood pressures to potentially protect against MINS.

Identification of MINS remains challenging since postoperative analgesia can mask classic signs of ischemia such as chest pain, or clinicians might attribute symptoms to surgical causes and miss the correct diagnosis.[1,2] Without pain symptoms to indicate periods of ischemia, EKG changes might have resolved and many cases of MINS might be unidentified MIs.[4] Several studies attempt to assess the actual frequency and severity of myocardial injury after non-cardiac surgery by monitoring serial troponin measurements on all post-operative patients in order to capture those without clinical symptoms.[1] One large international study of 15,065 patients found 1,194 experienced MINS (7.9%). Of the patients with MINS, 84.2% had no clinical indicators of cardiac ischemia. Patients with MINS suffered a 10% mortality rate.[1]

Should a patient develop an MI, clinicians must weigh competing clinical goals of preventing bleeding while also preventing/dissolving blood clots.[1,6] Several randomized controlled studies have examined possible treatment modalities for surgical patients at risk of cardiac complications, measuring rates of MINS after initiating post-operative treatment. With metoprolol therapy, a large study found an overall increased risk of death, with fewer MIs but greater numbers of strokes. The rate of sepsis increased in the metoprolol therapy group.[7] Aspirin therapy showed no mortality benefit while also increasing the risk of surgical site or gastrointestinal bleeding.[6] Dabigatran therapy showed the most promise, demonstrating no significant bleeding while reducing the risk of vascular complications by 25%.[8] At present there

appear to be no randomized controlled trials of revascularization procedures compared to medically managing MINS. Observational studies demonstrate mortality up to 25% with percutaneous intervention on patients with perioperative MIs.[4]

Currently, it is not standard clinical practice to monitor perioperative troponin levels and serial EKGs on all patients with high cardiac risk. However, a great many MINS cases could be detected with a baseline preoperative troponin and daily troponins for up to 72 hours after surgery. While further research is needed to determine the optimal treatment strategy for MINS, evidence suggests that careful clinical monitoring along with dabigatran therapy will improve outcomes for those at risk of cardiac events following surgery.[8] In addition, telemetry monitoring or continuous 12-lead EKG monitoring could allow identification of myocardial ischemia that might otherwise be missed in a post-operative setting.

In this case study, the troponins were monitored after the patient demonstrated unambiguous signs of myocardial ischemia. As a result, MI was diagnosed, and the patient was medically managed. Although only 15.8% of patients with MINS experience symptoms, the research does not investigate differences in outcome or severity of cardiac injury in patients who demonstrate signs of ischemia.[1] In theory, post-operative analgesia might prevent patients from feeling chest pain until the myocardial damage is quite severe, resulting in worse outcomes for that population. Alternately, it is possible that there is great variability in pain sensation. Patients more attuned to pain will seek help and receive the treatment that leads to better outcomes. Perhaps future research will address further prognostic indicators for MINS along with optimal prevention and treatment strategies.

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Advanced Maternal Age and HELLP Syndrome

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Keywords: elderly primipara, advanced maternal age, HELLP syndrome, hemolysis, elevated liver enzymes, low platelets

Hypertensive disorders complicate up to 8% of pregnancies and contribute to 19% of pregnancy-related mortality.[1] The condition of hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome) arises from gestational hypertension and preeclampsia. HELLP syndrome can be attributed to 70% of preterm births, disseminated intravascular coagulation (DIC), placental abruption, renal failure, and both cerebral and liver hemorrhage.[1] This report discusses a case of HELLP syndrome in which delivery of the fetus effectively reversed complications in the mother.

Case Report

A 49-year-old 76 kg, 168 cm primipara (G5T0P0A4L0) presented to the labor and delivery unit for a scheduled cesarean section. She was 38 weeks and 4 days pregnant with a single fetus following donor sperm and donor egg in-vitro fertilization. She had no known allergies. Past medical history included asthma and anxiety. Past surgical history included hysteroscopy, dilation and curettage and in-vitro fertilization. The patient's current medication regimen consisted of prenatal vitamins, vaginal progesterone suppositories, and albuterol as needed. There were no reported health issues or concerns to the parturient during this pregnancy.

Upon assessment of the parturient in the labor suite, blood pressure (BP) was found to be 168/88 mm Hg, heart rate (HR) 92/min, respiratory rate (RR) of 16/min, temperature of 98.7°F, with an SPO2 of 98% on room air. An 18 gauge intravenous (IV) catheter was inserted into the left metacarpal vein and 1,000 mL of Lactated Ringers (LR) was administered as a bolus. Laboratory results were as follows: Hemoglobin 8.2 g/dL, Hematocrit 29%, platelet count 65,000 per microliter, AST 224 units/L and ALT 182 units/L. The previous week during routine examination, the patient's BP was 132/70 mm Hg, platelets were 160,000/μL, hemoglobin was 10.5 g/dL, and both AST and ALT were within normal range. HELLP syndrome was then diagnosed and the patient was transferred to the operating room (OR) for emergency cesarean delivery.

The patient was placed in the supine position with left uterine displacement. Noninvasive standard monitors were applied; 2 grams of cefazolin was initiated as well as free flowing LR

IV. Oxygen 12 L/min was applied via simple face mask. Vital signs were recorded as BP 157/95 mm Hg, $Sp0_2$ 99% and HR 105/min. A rapid sequence intubation was performed with cricoid pressure; propofol 200 mg of and succinylcholine 100 mg were administered intravenously. The trachea was intubated with a 6.5 mm cuffed endotracheal tube and placement was confirmed via auscultation. A nasogastric tube was inserted into the esophagus and placed on low intermittent suction to decompress the abdomen. Sevoflurane 2% was administered in O_2 2 L/min. In approximately 60 seconds, the fetus was delivered. Midazolam 2 mg and fentanyl 100 mcg were administered IV as well as oxytocin 20 units in 1 liter of normal saline. Sevoflurane was decreased to 1% with N_2O 1 L/min and O_2 1 L/min.

The neonate was given Apgar scores of 7 and 8 at 1 and 5 minutes after delivery. Mild uterine atony and bleeding was treated with another 20 units of Pitocin in 1L of Normal Saline IV. Once suturing was complete, all anesthesia gases were discontinued and patient was extubated to 3 liters of oxygen via nasal cannula. Vital signs at the end of the procedure were BP 138/77 mm Hg, HR 82/min, RR 13/min and SpO_2 98%. The patient was transferred to the recovery room where contact with the neonate was initiated. The mother remained in the hospital with the neonate until all laboratory values and vital signs returned to baseline and no lasting complications of HELLP syndrome were diagnosed.

Discussion

Advanced maternal age has been associated with hypertension, whether chronic or pregnancy-induced. Studies suggest the instance of preeclampsia increases by 4% each year for every year over the age of 32.[2] The aging process has been correlated with oxidative stress, which adversely impacts relaxation of the endothelium, increasing the risk of pregnancy-induced hypertension.[2] Maternal hypertension is defined as a BP of greater than or equal to 140/90 mm Hg at or after 20 weeks gestation, new onset of proteinuria (>300 mg/dl), organ dysfunction, and/or restriction in fetal growth.[3]

Clinical presentation can differ on a case by case basis, however prompt recognition and treatment ensures the health of the parturient and fetus. Hypertensive disorders are one of the leading causes of maternal and perinatal mortality and morbidity.[3] In this case report, upon the discovery of the patient's hypertension, stat labs were drawn to confirm the diagnosis. Immediately following the resultant labs, anesthesia was notified and the OR staff prepared to deliver the fetus immediately.

Current literature shows timely delivery to ensure stability of the mother as the only concrete evidence pertaining to treatment.[4] Diagnosing and distinguishing HELLP Syndrome from other pregnancy-related disorders poses a challenge and can compromise treatment. Differential diagnoses include hemolytic uremic syndrome, acute fatty liver, antiphospholipid syndrome, and thrombotic thrombocytopenic purpura.[4] The clinical presentation is variable and may consist of nausea and vomiting, malaise, epigastric pain, disturbances in vision, headache and weight gain.[5] None of these symptoms were reported in this patient. The only evidence of HELLP syndrome were her blood pressure and laboratory values.

A recent study demonstrated that in order to best prevent adverse outcomes from pregnancy induced hypertension, it is crucial to evaluate patient factors that put them at increased risk. Early onset preeclampsia is associated with a higher perinatal death rate compared to those diagnosed with preeclampsia later in pregnancy.[3] Other factors associated with adverse outcomes include: maternal age over 35 years, the use of assisted reproductive technology, living in a rural area, history of pregnancy induced hypertension, multiple births, multigravida, having polycystic ovary syndrome, intrahepatic cholestasis of pregnancy, cardiovascular disease, gestational diabetes mellitus, systemic lupus erythematosus, thyroid disease, and liver disease.[3] The prompt recognition of these associative factors leading to poorer outcomes for mothers and neonates demonstrates the importance of prenatal care and advanced assessment by healthcare professionals. The patient in this case study possessed multiple risk factors as stated above. The prompt recognition of this patient as being high risk for developing HELLP Syndrome facilitated the appropriate actions to diagnose and deliver the fetus, preventing adverse outcomes.

The coagulopathies resulting from HELLP syndrome contraindicate lumbar neuraxial analgesia, therefore general anesthesia is the preferred anesthesia method used for cesarean delivery.[5] In this case report, the patient was intubated using a rapid sequence induction with cricoid pressure due to the full stomach from delayed gastric emptying in pregnancy.[6] Best practice protocols were followed, including left uterine displacement to prevent compression of the vena cava and hypotension, opioid administration after delivery of fetus, and Pitocin administration to contract the uterus to prevent further bleeding.[1,7,8] The patient in this case report was given a cesarean section promptly after the diagnosis was confirmed.

The anesthetic objectives should facilitate the birth of a thriving neonate and complete restoration of hemodynamic stability to the mother.[5] Anesthetic problems in HELLP Syndrome are contributed to effects on the cardiovascular, respiratory, neurologic, renal, hematologic, hepatic and utero-placental systems.[5] Aggressive management requires delivery as soon as possible as delivery is the only curative treatment.[4] Retrospectively, this case report demonstrates the prompt diagnosis and appropriate anesthetic interventions based on current evidence for best practice in treating HELLP Syndrome.

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Airway Securement after Laryngeal Fracture

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Keywords: laryngeal trauma, laryngeal fracture, epiglottitis, airway obstruction, cricoid cartilage fracture, tracheostomy

Laryngeal fractures are a rare and potentially life-threatening occurrence, with a calculated incidence of approximately 1 per 30,000-125,000 emergency room visits.[1] Common mechanisms of injury are blunt trauma, motor vehicle trauma, and falls.[1] Presenting signs and symptoms can vary widely: dyspnea, dysphonia, stridor, neck pain, hemoptysis, neck crepitus, and diminished lung sounds; some patients may exhibit no signs or symptoms at all.[1] Patients with laryngeal fracture frequently have concomitant injuries requiring treatment. The overall mortality rate associated with laryngeal fractures is 3.8%.[1] Anesthesia professionals must be aware of the implications a laryngeal fracture has for securing a safe airway.

Case Report

A 36-year-old alcohol-intoxicated male without significant past medical history presented to the emergency room after being struck by a car at approximately 20 mph while riding an electric scooter. The patient was placed into a cervical collar, and computed tomography (CT) scans of his head and neck performed. The head CT showed the patient had a small subarachnoid hemorrhage and small subdural hematomas. The patient was transferred to the neurological intensive care unit for monitoring. Overnight, the patient was reported by nursing staff as being calm, mostly non-verbal, with a hoarse voice when the patient attempted to speak. Laboratory work and vital signs were within normal limits. The practitioners reviewed the neck CT approximately 7 hours after admission and found the patient had a non-displaced, posterior cricoid cartilage fracture, epiglottitis, with significant airway swelling and obstruction. The airway management team were called by the patient's primary team to secure the patient's airway before patency was lost.

Assessment of the patient demonstrated a young, healthy male without obvious external injuries, resting peacefully in bed with a cervical collar in place. Vital signs indicated the patient was normotensive with a respiratory rate of 16-20/min and SpO_2 of 99% on room air. The patient was slow to respond to questions, rarely attempted to speak, and his voice was low and hoarse. The cervical spine was stabilized while assessing the neck, but overt edema was not appreciated. Initially, the anesthesia professionals planned to intubate the trachea via awake fiberoptic

intubation, but after reviewing the neck CT, they requested a consultation with the on-call otolaryngologist to determine the extent of airway swelling and the possibility for orotracheal intubation.

Upon arriving to the patient's bedside, the otolaryngologist performed a flexible endoscopic fiberoptic exam via the nare and determined that attempting orotracheal intubation would be very dangerous due to extreme narrowing of the airway beneath the epiglottis. It was estimated that the largest endotracheal tube the airway could accommodate would be 4.0 mm. The otolaryngologist recommended an emergent awake tracheostomy for both airway securement and potential future treatment of the cricoid fracture. The patient's brother provided consent for emergent awake tracheostomy and the patient was taken to the operating room within 30 minutes.

The surgeon performed the tracheostomy utilizing local anesthesia without complication. The patient remained calm during the majority of the procedure without premedication or sedation. Once the tracheostomy tube was placed into the trachea and there was confirmation of $EtCO_2$, the patient was anesthetized with propofol 200 mg and general anesthesia was maintained with sevoflurane 2% inspired concentration in a mixture of O_2 1L/min and air 1 L/min. The anesthesia professionals performed an asleep oral fiberoptic examination of the patient's airway, which revealed a swollen uvula, epiglottis, and a large hematoma. The hematoma protruded into the airway under the epiglottis which prevented the advancement of the fiberoptic endoscope. The vocal cords could not be visualized. The patient was subsequently emerged from anesthesia and transported back to the neurological intensive care unit in stable condition.

Discussion

Owing to the scarcity of laryngeal trauma, the frequency with which it accompanies other injuries, and the lack of correlation between presenting symptoms and severity of the injury, laryngeal fractures often go unrecognized and undertreated.[2] The anesthesia professional must appreciate the airway implications of laryngeal trauma. In addition to the threat of airway edema, laryngeal fractures can result in vocal cord immobility leading to problems with airway protection, phonation, swallowing, airway obstruction, and distress.[1] Treatment decisions vary as they are based on the severity of the injury, which may range from mild edema or hematoma without detectable fracture to complete laryngotracheal separation.[2] Non-displaced fractures with moderate to severe edema or hematoma do not often require a tracheostomy and can be treated with steroids, humidified oxygen, head of bed elevation, voice rest, and anti-reflux medications.[2,3]

In the case presented, the diagnosis of the laryngeal fracture, and subsequent treatment was delayed. The decision to further prolong airway securement, until after otolaryngology evaluation, was made due to concerns for a difficult airway and fear of inadvertent tissue damage leading to bleeding, increased edema, or complete loss of airway patency in the context of a patient without respiratory distress.

Patients with significant laryngeal trauma and fracture(s) commonly have orotracheal intubation prophylactically in the emergency room or with general anesthesia for surgical repair, which is

converted to tracheostomy for repair and healing.[3] In the case presented, the decision to forgo attempting orotracheal intubation was based off of two factors: severe airway narrowing and the possibility that a tracheostomy would be required in the future for surgical repair of the cricoid fracture. Surgical placement of a tracheostomy is considered to be the safest choice for acute airway obstruction. However, for those with laryngeal trauma, a fiberoptic or video laryngoscope assisted orotracheal intubation has demonstrated to be a safe choice.[4] Anesthesia considerations for attempting orotracheal intubation include a skilled practitioner,[3] and avoiding cricoid pressure as it is contraindicated due to the risk of causing further airway damage and compromise. If orotracheal intubation attempts result in failure, surgical or needle cricothyroidotomy is not recommended for the same rationale as avoiding cricoid pressure.[4] While non-displaced laryngeal fractures may not require a tracheostomy for healing,[2] in this case, the consulting otolaryngologist felt confident a tracheostomy would be required for several months.

Awake tracheostomy with local anesthesia can be performed with sedatives to help the patient tolerate the procedure. Possible drug choices for an awake tracheostomy include midazolam, fentanyl, and ketamine.[5] It is recommended that caution is taken for those with respiratory compromise or at risk for developing respiratory compromise as general anesthesia decreases both pharyngeal and laryngeal muscle tone, potentially leading to complete airway obstruction.[5] In the case presented, waiting to sedate the patient until after the tracheostomy tube was placed within the trachea and determined to be patent was deemed to be the safest choice of anesthetic while minimizing patient discomfort as much as possible which is consistent with current standards.

Laryngeal fractures are rare, possibly life-threatening injuries, and a high suspicion of injury should be present as to not have a delay in diagnosis and treatment. In the presented case, the patient's cricoid fracture and airway edema went untreated for several hours, potentially allowing for the condition to worsen. An important lesson taken from this case is the extreme disconnect between the patient's symptoms and the severity of his airway edema. The patient had no overt outward appearance of having respiratory compromise but had such severe edema and hematoma as to prevent orotracheal intubation with an endotracheal tube appropriate for his size. The importance of anesthesia professionals taking the extra time to review the CT neck for themselves is also striking, as this prompted emergent consultation with otolaryngology that was likely critical to patient surviving his injuries. Signs of laryngeal trauma should be investigated thoroughly before attempting orotracheal intubation.

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Pediatric MRI Sedation and Food-Protein Induced Enterocolitis Syndrome

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Keywords: food-protein induced enterocolitis syndrome (FPIES), pediatric anesthesia, propofol allergy, remote location anesthesia

Performance of "off-site" anesthesia, such as procedures in the magnetic resonance imaging (MRI) suite, can be harrowing experiences for anesthesia professionals due to their remote locations. The ensuing case study examines an off-site MRI procedure for a child diagnosed with food-protein induced enterocolitis syndrome (FPIES). FPIES encompasses a series of non-IgE-mediated food allergies which leads to profuse, intractable vomiting and diarrhea several hours after food ingestion.[1] Although rare, the incidence of FPIES is sharply rising with recent evidence of approximately 90 cases per year and an overall incidence of about 1 in 10,000 infants less than 2 years of age.[2]

Case Report

A 13-month-old female (7.7 kg and BMI 17.7) presented for an MRI of the brain for seizure-like activity and developmental delays. Past medical history was significant for FPIES and her extensive allergy list included both IgE-mediated and FPIES triggers. IgE-mediated allergies included cefdinir (hives), eggs (anaphylaxis), and green beans (hives and urticaria). FPIES allergies included milk products, soy, oats, corn, watermelon, and wheat. There was no prior anesthetic history and no home medications were taken on the day of the procedure. The patient's most recent FPIES incident was less than one month prior when she presented to the emergency room after ingesting soy and developed fever, nausea, vomiting, and bloody diarrhea. The patient presented to the MRI suite with her parents. She was mildly lethargic and avoided eye contact with the anesthesia practitioners. A thorough airway exam was deferred as the patient was uncooperative. Pre-procedural vital signs were blood pressure of 90/69 mmHg, pulse of 118/min, respirations 20/min, 97% SpO_2, and temperature of 36.9 °C. She showed no signs of separation anxiety upon being separated from her parents.

On arrival to the MRI suite and while sitting on an anesthesia practitioner's lap, an inhalation induction was initiated with sevoflurane 8% inspired concentration. The induction took longer than usual as her breathing was slow and shallow. Once the patient's eyes drifted shut, her extremities started to relax, and she was positioned supine on the MRI table. Standard

noninvasive monitors were applied, and sevoflurane continued to be administered while an intravenous (IV) catheter was placed in her left hand. Per institutional protocol guidelines for all pediatric MRI procedures, a facemask with 6 L/minute of oxygen was applied and an intravenous infusion of propofol was initiated at 175 mcg/kg/min. Approximately 25 minutes into the case, the propofol drip was reduced to 50 mcg/kg/min. The propofol was incrementally decreased and then completely shut-off at the end of the procedure. The patient received a total of 20.44 mg of propofol. She woke up slowly and calmly, opening her eyes to her name but returning to a resting state when not stimulated.

After the 45-minute procedure was complete, the patient was taken to the recovery area of the MRI suite on room air in a crib-stretcher. Post-procedure vital signs were blood pressure of 91/59 mmHg, pulse of 144/min, respirations 20/min, 97% SpO_2, and a temperature of 36.7 °C. After approximately 90 minutes of standard monitoring, the patient was discharged at her baseline level of consciousness. Her parents had no concerns at the time of discharge. The MRI results included no signs of intracranial abnormalities.

Discussion

Because of the low incidence of FPIES, many anesthesia practitioners are possibly unaware of its pathophysiology and implications on the anesthetic care plan. The disorder is typically discovered in infancy with exposure to formula and solid foods.[1] The delayed onset of the reaction and lack of cutaneous and respiratory effects differentiate FPIES from true food-allergy driven anaphylaxis.[1] According to the international consensus guidelines for FPIES diagnosis, the 2 hallmarks of this chronic disease include asymptomatic patients exhibiting normal growth when trigger foods are eliminated from the diet and acute exacerbations when trigger foods are reintroduced.[3] Affected infants often present to emergency rooms with pallor, lethargy, and severe dehydration (possibly to the point of hypovolemic shock) on multiple occasions before a diagnosis is made by connecting the incidents through relation of common FPIES trigger foods. Such common trigger foods include cow's milk, dairy products, soy, grains, eggs, fish, rice, oats, and barley products.[1]

The exact immunological and pathological mechanisms of FPIES remain largely uncertain at this time. Patients typically test negative for the allergen immunoglobulin IgE biomarker and, therefore, the traditional epinephrine autoinjectors prescribed for patients with food allergies are not routinely recommended for patients with FPIES.[3] Based on endoscopic studies, it is believed that antigen-specific T cells, antibodies, tumor necrosis factor, and cytokines cause colon and ileum inflammation.[3] These inflammatory markers are indicative of increased intestinal permeability which results in fluid shifts into the gastrointestinal tract, providing the genesis for the subsequent hallmark symptoms of vomiting and diarrhea. Because episodes of FPIES are self-limiting, treatment is typically supportive and focuses on maintaining hydration status and avoiding known trigger foods in the future.[4] Per guidelines, methylprednisolone at 1 mg/kg dosing can be used for severe cases to mitigate cell-mediated intestinal inflammation.[3] Ondansetron, a 5-HT_3 receptor antagonist, at doses of 0.2 mg/kg has been shown to be effective for the treatment of nausea.[4]

When caring for a patient with FPIES undergoing anesthesia, the primary implication is related to propofol administration. The original preparation of propofol included a polyethoxylated castor oil, but this was abandoned due to high allergic potential.[5] Currently, most formulas of propofol involve a mixture of soybean oil, glycerol, and egg phospholipid which is considered safe to administer in IgE-mediated egg and soybean allergic individuals.[5] However, as noted above, FPIES involves a non-IgE mediated reaction. Unfortunately, due to the rare nature of the disorder, adverse reactions to anesthetic drugs, including propofol, are currently unknown in the FPIES population.[4] At present, there are no case reports in the existing scientific literature that include evidence of an adverse reaction to propofol in FPIES patients. However, FPIES reactions are often delayed for 4 hours or more. This timeframe exceeds the postoperative stay for most outpatient pediatric procedures; therefore, potential reactions may go unrecognized by healthcare practitioners.

In the case of this particular atopic and hypersensitive patient who presented with both an anaphylactic reaction to eggs and an FPIES intolerance to soy, a different pharmacologic plan may have been optimal to avoid the use of propofol. Because of the potential complications of propofol administration in this patient, the remote location of the procedure was not ideal and could have contributed to an adverse outcome. Perhaps a safer option for this patient might have been a general anesthetic with a laryngeal mask airway (LMA) and sevoflurane administration. Although the patient was discharged symptom-free, it is possible that she may have developed vomiting, diarrhea, or discomfort once she was home. Additionally, no anti-emetic medication was administered before discharge. A prophylactic dose of ondansetron could have optimized her condition for discharge to home.

Preoperatively, the patient was briefly identified as allergy-prone, but her hypersensitivities were largely dismissed from the planning process. This case study presents a possible a societal bias that could have formed over recent years as children are presenting with higher incidences of intolerances and allergies when compared to prior generations. Because the phenomenon is becoming more common, anesthesia practitioners may not consider it a noteworthy finding with potential anesthetic implications. Despite the concerns in her past medical history, this patient received a standardized anesthetic that followed the institutional protocols. Implications for future research include a more thorough exploration of the disease process and the need for pharmacologic management studies. Only then can best anesthesia practices for patients diagnosed with FPIES be identified and optimal anesthesia care be provided in this growing segment of the pediatric patient population.

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Anesthetic Implications for Patients with Friedreich's Ataxia

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Keywords: Friedreich's ataxia, frataxin, neurodegenerative, autosomal recessive

Friedreich's ataxia (FRDA) is an autosomal recessive inherited neurodegenerative ataxic condition that is often accompanied with cardiac myopathies, scoliosis, diabetes mellitus, atypical reflexes, and dysarthria.[1] The majority of FRDA cases are caused by a defect in the GAA triplet repeat expansion in the frataxin (FXN) gene found on chromosome 9.[1,2] The multisystem effects of FRDA can make the delivery of anesthesia a challenging task to the provider in regard to the pharmacologic, physiologic, and pathologic effects that occur with this disorder. Specific concerns to anesthesia include cardiac abnormalities, pulmonary function, neurodegenerative effects, and endocrine dysfunction.

Case Report

A 30-year-old, 71 cm, 70 kg Caucasian male patient presented for a posterior spinal fusion and instrumentation of thoracic 2 through lumbar 4 with thoracic osteotomies. His past medical history included FRDA, scoliosis, wheelchair, chronic pain, methicillin-resistant staphylococcus aureus, and daily tetrahydrocannabinol use. Medication allergies included codeine. His home medications included ferrous sulfate 325 mg, acetaminophen 500 mg, and sildenafil 20 mg. Surgical history included cardiac defibrillator placement and teeth extraction.

The patient's preoperative vital signs included: blood pressure 95/59 mm Hg, room air SpO_2 96%, and all other vital signs within normal limits. A preoperative 12-lead electrocardiogram was performed and interpreted by a cardiologist as sinus rhythm with ST and T wave abnormalities. The most recent echocardiogram demonstrated preserved left ventricular function with an ejection fraction of 59% in addition to concentric hypertrophy and mild tricuspid regurgitation. Thoracic x-ray exhibited a 27-degree convex left curve of T6 through T11 and chronic wedging of right T8 and T9 with osteophytes.

Preoperatively, a 20-gauage intravenous (IV) catheter was placed in the dorsum of the patient's left hand, a Lactated Ringer's infusion was initiated, and midazolam 2 mg IV were given prior to transport to the operating room (OR). Upon entering the OR, standard noninvasive monitors were attached to the patient while still on transfer cart and preoxygenation via a face mask was initiated at 100%. A 20-gauge right radial arterial line was placed, and an additional 18-gauge IV was placed and was connected to blood tubing primed with 0.9% normal saline. An IV induction was performed with fentanyl 100 mcg, lidocaine 100 mg, propofol 200 mg, rocuronium 30 mg, and phenylephrine 100 mcg. An elective C-MAC video laryngoscope (KARL STORZ, El Segundo, CA) was utilized with a D-blade and airway was secured with 8.0 mm endotracheal tube. Endotracheal tube placement was confirmed via video visualization, presence of $EtCO_2$, and positive bilateral breath sounds. The patient was then placed in the prone position, bilateral lung sounds were reconfirmed posteriorly, and then placed on volume control ventilation. Neuromonitoring leads were placed by the technician for the purpose of somatosensory (SEPs) and motor evoked potentials (MEPs).

Anesthesia maintenance included 0.5 minimum alveolar concentration (MAC) of sevoflurane, propofol infusion of 75 mcg/kg/min, and remifentanil infusion of 0.2-0.3 mcg/kg/min. Prior to surgical incision, cefazolin 2 g and vancomycin 1 g were administered, as well as a 1 g tranexamic acid bolus, followed by 2 mg/kg/hr infusion of the tranexamic acid. A low dose phenylephrine infusion was used to maintain a mean arterial pressure greater than 65 mmHg. Antiemetics included IV dexamethasone 5 mg and IV ondansetron 4 mg. The total amount of fluids given included 1,500 mL of Lactated Ringer's, 200 mL of 0.9% normal saline, 750 mL of 5% albumin, and 500 mL of cell saver blood products. The estimated blood loss totaled 700 mL.

During closing of the surgical site, the remifentanil, propofol, and subsequently the phenylephrine infusions were discontinued. Total procedure duration was 9 hours and 25 minutes. The patient did not require neuromuscular blockade antagonism. Train of four monitoring produced 4/4 twitches, sustained tetany, and adequate tidal volumes were observed prior to extubation. The patient was extubated awake and transferred to the post-anesthesia care unit (PACU) on 10 L/minute of oxygen via a simple mask.

Discussion

Nikolaus Friedreich, a German pathologist, was the first person to detail the characteristics of FRDA in 1863.[2] The diagnosis of FRDA at this time is uncurable and is associated with a decreased life expectancy of 35 to 40 years.[1] The main causes of mortality in FRDA are related to cardiac dysfunction; congestive heart failure and arrhythmias being the leading causes, followed by stroke, ischemic heart disease, and pneumonia.[3] Prevalence of FDRA in the United States of America is unknown but, it is predicted to be 1 in 80,667.[4] Countries with the highest prevalence of FRDA include: northern Spain with 1 in 20,000, Ireland 1 in 23,000, France 1 in 43,000, and Germany 1 in 47,000.[4] FRDA is very uncommon in individuals of Sub-Saharan and Far East descent, occurring primarily in those of Caucasian descent.[4]

Two types of FRDA exist, with the most predominant form (96 - 98%) being a homozygous GAA trinucleotide repeat expansion in the first intron of the FXN gene on the long arm of chromosome 9q21.11 and the least predominant form (2 - 4%) being heterozygous, characterized

by a point mutation or exonic deletion.[2,5] Normal chromosomes in patients unaffected by FRDA have repeat GAA trinucleotide expansions containing typically less than 12 repeats, with 60 being the upper limit of normal. [3,5] Those affected with FRDA have repeat trinucleotide expansions on average between 600 to 900, however, they can range from 6 to 1,500 repeats.[3,5] Thus, the greater proportion of GAA repeats is linked to decreased measures of the frataxin protein, which accelerates the disease process at an earlier age and determines the severity of the disease.[1]

Hypertrophic cardiomyopathies are prevalent among FRDA patients and left ventricular outflow obstructions should be avoided.[5] During induction, sympathetic stimulation should ideally be avoided with direct laryngoscopy, premedication with a beta blocker or volatile anesthetic can be considered to blunt this response. Alpha-adrenergic agonists should be used to treat decreases in preload or afterload caused by hypotension, whereas beta-adrenergic agonists should be avoided related to potential increases in inotrophy and chronotrophy. Normal sinus rhythm should be maintained and therefore, beta blockers such as esmolol and metoprolol can be considered to reduce tachycardia. Additionally, a cardioverter-defibrillator should be present within the OR, in the event the patient develops supraventricular tachydysrhythmia.[6]

Scoliosis is a common feature in patients presenting with FRDA and can cause varying degrees of pulmonary dysfunction.[7,8] The curvature of the spine can result in the lungs becoming constricted in the chest cavity, which decreases the individual's vital capacity and increases work of breathing, airway resistance, and dyspnea.[7] Obstructive sleep apnea has also been associated with FRDA, which increases with severity of the disease.[3] No contraindications were found for neuraxial anesthesia in FRDA patients, however, it may present a challenge related to the presence of scoliotic changes. Respiratory status should be closely monitored in neuraxial blocks higher than the level of T10, as respiratory muscle involvement may become compromised.[7]

Neuromuscular blocking agents for induction and maintenance should take into account the FRDA patient's progressive neurodegenerative disease.[1,7] The use of succinylcholine should be avoided, as FRDA is a neurodegenerative disease and anesthetic management should be treated in a similar fashion to amyotrophic lateral sclerosis patients.[7] The patient may be at risk for a hyperkalemic episode after administration of succinylcholine, thus a nondepolarizing neuromuscular blocker should be considered. A reduced dose of nondepolarizing neuromuscular agent should be used, as patient response to muscle relaxation may be variable.[7]

Patients with FRDA may be more sensitive to sevoflurane than other agents, however, if muscle relaxation is needed, sevoflurane or desflurane would be the ideal choice.[9] Isoflurane or desflurane may be advantageous if cardiac output needs to be preserved, though caution should be exercised with use of desflurane, related to the risk of tachycardia.[7] The use and consideration of electroencephalogram monitoring should be considered in order to administer the minimum amount of anesthetic needed for the proposed procedure. Lastly, inhalation agents may be safer than intravenous sedation, as volatile anesthetics are able to be exhaled and do not require extensive metabolism.[9] In the case presented, a combination of low inhalation MAC and IV sedation was utilized for the purpose of neuromonitoring.

Motor evoked potentials and SEPs information is limited in patients with FRDA. Research has found that MEPs and SEPs are usually significantly reduced or absent.[8] If SEPs/MEPs monitoring is planned, low dose inhalation agents combined with intravenous agents should be utilized in addition to the avoidance of long acting neuromuscular blockers. If a wake-up test is planned, neuromuscular blockade should be avoided or antagonised.[7]

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Management of General Anesthesia in the Kratom-Using Patient

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Keywords: kratom, mitragyna speciose, opioids, substance abuse

An increasing number of Americans are using mitragyna speciose, kratom, for both recreational and medicinal purposes. Kratom is an indigenous plant originating from Southeast Asia whose leaves can be consumed.[1,2] The primary active ingredients, mitragynine and 7-hydroxymitragine, exhibit opioid-like effects by stimulating mu and delta opioid receptors. Mitragynine also stimulates alpha-2 adrenergic receptors and produces sedative and analgesic effects. Kratom is used to relieve pain, increase energy, and alleviate depression.[3] There are no published studies

describing drug interactions or anesthetic implications of kratom use beyond its sedative and analgesic effects.

Case Report

A 31-year-old, 162.5 cm, 59 kg female presented for a laparoscopic myomectomy. Her past medical history included uterine fibroids, depression, anxiety, anemia, and current daily cigarette smoking. She denied taking prescription medications, but she reported using kratom as a supplement. Her past surgical history included a hysteroscopy and hysteroscopic resection of a submucosal fibroid. She reported social alcohol use and no illicit drug use. Significant preoperative laboratory values included hemoglobin 9.3 g/dL, hematocrit 26.4%, platelets 309 k/microliter, sodium 136 mEq/L, potassium 4.2 mEq/L, and creatinine 0.71 mg/dL. Preoperative vital signs were as follows: blood pressure 114/71 mmHg, heart rate 74 beats per minute, respiratory rate 15 breaths per minute, oxygen saturation 99% on room air.

General anesthesia with an endotracheal tube was discussed and agreed upon by the patient, family, and surgeon. The surgeon anticipated the potential for significant blood loss and that the procedure would take five to six hours to complete. It was therefore decided that an arterial line and a second IV would be placed after induction. The patient received midazolam 2 mg intravenously in the preoperative area. After transfer to the operating room, standard monitors were applied and the patient was preoxygenated. General anesthesia was induced with propofol 150 mg, fentanyl 100 mcg, and lidocaine 60 mg. Following the administration of rocuronium 40 mg and two minutes of mask ventilation, the trachea was successfully intubated. The patient was maintained under general anesthesia with sevoflurane 2% inspired concentration and a fresh gas mixture of oxygen 1 L/min and air 1 L/min. Approximately thirty minutes after induction, the patient became increasingly hypertensive and tachycardic with a systolic blood pressure of 150 mmHg and a heart rate reaching 113 beats/minute. Propofol 50 mg, fentanyl 100 mcg, and hydromorphone 1 mg were administered intravenously. Thirty minutes later the patient remained hypertensive. Additional doses of fentanyl 100 mcg and hydromorphone 1 mg were administered. The patient's preoperative evaluation confirmed that she did not take any opioids at home but was a current every day smoker, reported social alcohol use, and reported kratom as a home medication. The anesthesia team was unsure if the patient underreported her alcohol and tobacco use, leading to enzyme induction, or if kratom was contributing to her opioid requirement. Two hours into the procedure the decision was made to initiate the hospitals' multimodal pain protocol for opioid dependent patients. An intravenous ketamine infusion was initiated at 5 mcg/kg/min and an intravenous lidocaine infusion was initiated at 1.5 mg/kg/hr.

At the conclusion of surgery, the neuromuscular blockade was antagonized with sugammadex 200 mg, the ketamine infusion was stopped, and the patient was extubated without complication to O_2 8 L/min via facemask. The patient was transferred to the post anesthesia care unit with the lidocaine infusion continuing. The patient appeared comfortable in the immediate postoperative period and rated her pain a 4 out of 10. Throughout the 5-hour case, the total analgesic given included hydromorphone 6 mg, fentanyl 600 mcg, ketamine 140 mg, and lidocaine 350 mg.

Discussion

Anesthesia professionals can tailor their anesthetic plan for opioid dependent patients based on clinical judgement and hospital protocols for multimodal pain management. However, there is a lack of knowledge regarding how some substances, such as kratom, interact with anesthetics and how to best care for patients taking these non-prescription supplements. Because opioid agonists and kratom work on similar receptors, it is possible that a kratom user may behave similarly to an opioid user under general anesthesia.

From the beginning of surgery, the patient exhibited a higher than anticipated opioid requirement based on her history and physical. Her preoperative evaluation, including the pharmacology of kratom, was reviewed again in the operating room approximately two hours into surgery. Prior to this, the anesthesia team was unaware that kratom agonizes opioid receptors and produces opioid-like effects. The team was also unaware of the kratom dosage or the amount of time that the patient had been taking the supplement. Based on these findings, the team decided to initiate the hospital's multimodal pain protocol that is typically used for opioid-dependent patients with chronic pain.

Although the patient was not taking prescription opioids prior to surgery, the opioid-like effects of kratom could have rendered this patient opioid dependent and increased her opioid tolerance. Case reports of kratom use suggest that it can result in abuse, addiction, and "fatal interactions with other psychoactive drugs."[1] A cross sectional study conducted in Malaysia explored the prevalence of addiction and withdrawal symptoms in kratom users. Approximately 79% of the study participants stated they needed to use kratom daily. In addition, kratom users reported withdrawal symptoms that included sleeping difficulty, nausea, vomiting, sweating, fever, and shakiness.[1] These withdrawal symptoms are similar to opioid withdrawal symptoms, further suggesting its opioid agonist properties.

In addition to understanding the opioid-like effects of kratom, it is important to gain knowledge about its side effect profile and adverse effects. There was an assessment of calls to U.S. poison control centers from 2010 through 2015. Six hundred and sixty kratom exposure cases were reported and 65% of the cases were solely related to kratom. The most common adverse effects included "tachycardia (25%), agitation or irritability (24%), drowsiness (19%), nausea (15%), and hypertension (12%)".[2] A recent report from the Centers for Disease Control (CDC) stated there were 152 overdose deaths related to kratom between July 2016 and December 2017 in the United States. Furthermore, approximately 80% of the decedents who passed from "kratom-positive" or "kratom-involved deaths" during this time period had a history of substance abuse. Fentanyl and heroin were the two most common co-substances found on toxicology testing following these overdoses.[5] Although kratom was not the sole cause of death, these statistics suggest the potential for dangerous drug interactions between opioids and kratom. Patients who present for surgery and test positive for illegal substances such as heroin may also be consuming kratom, although it is not included in routine toxicology screening.

Kratom gained public attention in 2016 when the US Drug Enforcement Agency (DEA) attempted to classify the substance as a schedule I drug.[4] Retaliation from groups such as the American Kratom Association persuaded the DEA to withdraw the movement to reclassify the

substance. However, the DEA still classifies kratom as a "drug of concern," which means it is "not regulated but poses a risk to persons abusing it."[4] Currently, kratom is illegal in six states and it can be purchased on the internet or at smoke shops. As of April 2019, kratom is not classified as a scheduled or controlled drug.[6]

More research regarding the pharmacological effects and drug interactions of kratom in humans is needed, the implication of which is that available knowledge is limited to case reports and qualitative studies. Anesthesia professionals are well versed in the anesthetic implications of common prescription drugs such as opioids and antihypertensive agents. However, caring for patients taking less studied substances such as kratom poses a significant challenge. The emerging evidence that kratom has opioid-like effects could result in more anesthesia professionals applying multimodal analgesia protocols for patients actively taking kratom. In addition, anesthesia professionals must be aware of the rise of new legal and illegal substances consumed by their patient population. It is imperative that the anesthesia professionals consider how supplements, non-prescription substances, and illicit drugs can interact with anesthetic agents and affect patient care.

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Management of the Pediatric Patient with Autistic Spectrum Disorder

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Keywords: Autism spectrum disorder, multidisciplinary preoperative preparation, intramuscular midazolam, child life specialist, general anesthesia

A paucity of literature exists on the perioperative anesthetic management of children with autism spectrum disorder (ASD), leading to a knowledge deficit for anesthesia professionals and

potentially negative patient outcomes. This case study reports the perioperative management of an autistic girl requiring general anesthesia for dental extractions. The management of pediatric patients with ASD relies on an understanding of the disorder, multidisciplinary preoperative preparation, therapeutic communication, and strategic anesthetic management. Parental involvement, development of a multidisciplinary plan, and the administration of intramuscular midazolam led to a successful outcome for the patient.

Case Report

A 6-year-old female presented to the ambulatory surgery department for dental extractions under general anesthesia. The patient had a history of autism spectrum disorder (ASD), was non-verbal, and understood sign language. Her surgical history included bilateral myringotomy and tympanostomy tube insertion at the age of 2. Her weight was 22.4 kg and her height was 119.38 cm, yielding a BMI of 15.8 kg/m^2. On first encounter with the patient, she was severely anxious and would not cooperate with physical examination or premedication. It was made known by the patient's mother that the patient had a severe phobia to hospitals and hospital personnel. The child life specialist (CLS) was immediately contacted to assist with the management of the patient. A conversation was held between the patient's parents, CLS, preoperative nurse, and the anesthesia team on how to best manage the patient so that she would be more cooperative and less anxious. A plan was made to give the patient midazolam via the intramuscular (IM) route because she was not going to tolerate oral administration. The parents explained that the injection could be successful if they communicated the steps of the procedure to her in sign language, while music was playing.

Prior to administering IM midazolam, a physical examination was performed with careful attention to maintaining a quiet, non-threatening, and therapeutic environment. The mother was asked to hold the patient during the examination and the CLS stood next to the patient, holding her favorite toy from home. During the physical examination, she remained quiet and appeared inattentive. She did not respond to verbal communication, had unclear speech, and displayed repetitive motions with her hands. Her head-to-toe assessment was negative.

Immediately after the physical examination, the CLS began to sing and play a soothing melody on the guitar to distract the patient from the administration of IM midazolam 3.5 mg (0.15 mg/kg) which was administered in the right deltoid. She did not react to her injection and continued to be engaged in the music making the intervention successful. Approximately 20 minutes after the injection, the patient appeared increasingly sleepy while maintaining a patent airway. After review of the anesthetic plan and answering the parent's questions, she was transferred to the operating room (OR) with the anesthesia staff, and CLS in attendance.

In the OR, after placement of the pulse oximeter, the patient was induced with sevoflurane 8% inspired concentration in a mixture of O_2 3 L/min and N_2O 7 L/min. Following a successful inhalational induction, standard noninvasive monitors were applied and intravenous access was established. Propofol 100 mg IV and fentanyl 25 mcg IV was administered and then the trachea was intubated with a cuffed 5.0 mm nasal Ring-Adair-Elwyn (RAE) endotracheal tube and mechanical ventilation was initiated. General anesthesia was maintained with sevoflurane 2.5% inspired concentration in a mixture of O_2 2 L/min. The patient breathed spontaneously

throughout the procedure. The patient was hemodynamically stable during the anesthetic course and was successfully extubated and transferred to the pediatric room in the post-anesthesia care unit (PACU). In the PACU, the patient recovered smoothly without any signs of postoperative emergence delirium. As the patient started to wake up, the parents were called to the bedside and she continued to recover in a calm and uneventful state.

Discussion

Pediatric patients with ASD have difficulties coping with surgical environments because of impaired social skills, communication barriers, sensory issues, and poor problem-solving skills.[1] Anesthesia professionals may view the perioperative management of these patients to be challenging because of the limited understanding of how to properly manage these patients. The combination of the diagnostic characteristics displayed by autistic children and anesthesia professionals' inexperience with the management of these patients can create a stressful situation, especially during the preoperative stage. Literature continuously states the use of oral midazolam or ketamine for preoperative sedation in uncooperative children; however, little is known about preoperative sedation specific to children with ASD.[2] Traditional administration of oral anxiolytics may not be feasible in autistic children, requiring the use of alternative plans.

Autism spectrum disorder is a biologically based neurodevelopmental disorder ranging in severity, characterized by persistent deficits in social interaction and social communication and restricted, repetitive patterns of behavior, interests, and activities.[1,3] These children may respond to sensory stimuli in the hospital setting with verbal or physical aggression, disruptive behavior, panic-attacks, and self-injurious behavior.[3] To prevent adverse events and provide successful anesthetic management of these children, the anesthesia professional must understand what ASD is, implement proper multidisciplinary preoperative preparation, engage in therapeutic communication, and appropriately tailor the anesthetic management.

To properly care for children with ASD, it is not only important to know the pathophysiology of the disorder but to truly understand the theory of mind deficit. The mind deficit theory states that children with ASD have difficulty seeing another's perspective, difficulty in determining the intentions of others, and lack the understanding of how their behavior affects those around them.[4] Understanding this theory and the social aspects of ASD will allow anesthesia professionals to enhance their interactions with ASD children.

Surveys of family views on the care of their autistic children have shown perceived inadequacies in care delivery, specifically regarding social aspects much more than medical treatment.[4] Strategies to improve interactions with autistic children include: minimizing waiting times, warning before making physical contact, eliciting information from parents, speaking quietly and gently, recognizing a patient may not wish to communicate, giving clear explanations, and providing a clear plan for the day.[4] In addition to these strategies, it is important to be mindful of both environmental and behavioral interventions, such as dimming the lights, maintaining a quiet room, and implementing appropriate distractions.[5]

To provide a safe and stress-free perioperative environment, a careful multidisciplinary plan is required.[3] The social aspect of this multidisciplinary plan is made possible with the help of a

CLS, whose role is to help children and families cope with the challenges of being a patient. The CLS assesses the child's routines, special interests, sensory sensitivities, level of understanding, and social needs through direct observation and in collaboration with parents, to develop an individualized social plan for the child. This social plan is communicated to other health care professionals to improve interactions with the child and prevent potential adverse outcomes.[4] A social plan helps everyone who is part of the child's health care team to understand the child's deficits in social communication, response to sensory hypersensitivity, possible associated mental health problems such as anxiety and anger, and potential intellectual impairment. Anesthesia professionals can use the CLS's assessment on the child's social needs and limitations to better tailor the anesthetic and perioperative interaction. The benefit of working with a CLS may not be available to all anesthesia departments and the CLS's may need to be taken up by a collaborative effort between nursing and anesthesia, throughout the perioperative continuum.

Successful anesthetic management of this population not only requires in-depth knowledge on the social aspects of ASD, it also requires knowledge of the appropriate pharmacological interventions. A thorough assessment of the patient's home medications is crucial because some of these patients are on antipsychotics, such as risperidone and clozapine, which can interact with general anesthesia. Risperidone may cause hypotension and have pro-arrhythmic properties with general anesthesia.[6] Clozapine may cause agranulocytosis, hyperthermia, cardiac conduction problems, and hypotension with general anesthesia.[6]

Premedication for children with ASD is strongly recommended; however, there is limited literature to provide evidenced-based recommendations. There are case studies that outline how oral ketamine, dose ranging from 7 mg/kg to 10 mg/kg, was successfully used to premedicate these children; however, with prolonged recovery times of up to 4-6 hours.[6] Ketamine is also associated with emergence phenomenon, disorientation, sensory and perceptual illusions, vivid dreams, nausea and vomiting in the pediatric population, nystagmus, hypersalivation and laryngospasm.[6] Oral midazolam, dose ranging from 0.25 mg/kg to 0.5 mg/kg, has also been successfully used for premedication of these children, without the potential adverse effects of ketamine.[6] It must be noted that children with ASD range in intellectual ability and respond differently to certain tastes associated with oral medications, which may make the oral administration of premedication impossible. In situations where children cannot tolerate oral medications, such as this case study, the use of intramuscular medications is warranted.

There are two studies that conclude midazolam 0.15-0.3 mg/kg IM is an effective dose in sedating children with ASD, without adverse effects to hemodynamics or prolonged recovery time.[2,7] Midazolam 0.15 mg/kg IM was successfully used for the child in this case study. The decision to use midazolam 0.15 mg/kg IM was based on the anesthesia professional's prior success using it in children with ASD and because of the potential adverse effects of ketamine that were previously mentioned. It is important to note children in general are at increased risk of developing emergence delirium after the administration of sevoflurane, and preoperative administration of midazolam may mitigate this.[8] Sevoflurane, regardless of the risk of causing emergence delirium, should continue to be used in pediatric anesthesia for induction and maintenance because it offers several advantages. Advantages include a relative lack of airway

irritation, a more rapid onset and recovery, and greater hemodynamic stability than other potent inhaled agents.[6]

Understanding ASD in children and the anesthetic management of this population will allow for successful outcomes in this unique subclass of patients. The successful anesthetic management of this patient was due to an understanding of ASD, development of a multidisciplinary plan, therapeutic communication, and a properly tailored premedication and anesthetic regimen. To increase the success of anesthetic management of patients with ASD, updated protocols need to be developed and further research needs to be done on the impact of anesthetic agents in this population.

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Anesthetic Management of Patient with Idiopathic Hereditary Angioedema

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Keywords: hereditary angioedema, C-1 esterase, bradykinin, FFP, HAE C1-INH

Hereditary angioedema (HAE) due to C-1 esterase deficiency can present with a number of potential complications that require precautionary measures to be considered for anesthetic and airway management. HAE with C-1 esterase deficiency has a recommended treatment plan. However, there is a subset of patients with normal C-1esterase levels that have similar episodes of angioedema in response to stress or trauma that are bradykinin-mediated, which do not have clear recommendations for management. This case report details the anesthetic management of a patient with hereditary angioedema with normal C-1 esterase levels, who presented for elective surgery.

Case Report

A 41-year-old, 180 cm, 93 kg male with a complex medical history presented for a laparoscopic ostomy closure under general anesthesia. Past medical history was significant for hereditary angioedema with normal C-1 esterase, Ramsay Hunt syndrome, complicated by esophageal dysmotility and chronic failure of the small intestine, requiring total parenteral nutrition; non-insulin dependent diabetes mellitus, chronic pain syndrome, and mild asthma. Past surgical history was significant for elective tracheostomy for airway protection, roux-en-Y gastrojejunostomy, J-tube placement, and biopsy of laryngeal lesion. Allergies included penicillin (rash), nifedipine (rash and lower extremity swelling), prochlorperazine (rash), and lidocaine (reaction uncertain). The patient was hospitalized due to recurrent episodes of angioedema, with C-1 esterase levels testing normal on multiple occasions. Factor XII analysis was also normal. The patient reported a family history of angioedema. Current medication regimen included lanadelumab 300 mg subcutaneously every 2 weeks, and ecallantide 10 mg subcutaneous as needed for acute angioedema episodes. Past angioedema episodes had responded to fresh frozen plasma (FFP) infusion.

The patient was evaluated the day prior to the scheduled surgery. An uncuffed tracheal tube was in place. The patient was able to participate in the interview via a Passy-Muir (Passy-Muir, Inc. Irvine, CA) valve while breathing room air. General anesthesia and the need to replace the tracheal tube with a cuffed tube in the operating room (OR) was discussed and agreed upon.

On the day of surgery, the patient was premedicated in the preoperative area with intravenous (IV) diphenhydramine 50 mg, methylprednisone 40 mg, and ranitidine 50 mg after being placed on standard noninvasive monitors. Two units of type and crossmatched FFP were present in a cooler in the OR. Pre-oxygenation was administered through the anesthesia circuit and tracheal tube. Midazolam 2 mg and fentanyl 50 mcg were administered IV. The surgeon replaced the tracheal tube with a cuffed tracheal tube with the assistance of an otolaryngologist. Placement was verified with $ETCO_2$ and flexible fiberoptic bronchoscope. The tracheal cuff was inflated, and induction of anesthesia was initiated with sevoflurane, and then augmented with propofol

100 mg, fentanyl 100 mcg, rocuronium 30 mg. Positive pressure ventilation was achieved with volume control auto flow on the anesthesia machine. An end tidal sevoflurane concentration of 2.3% was maintained with a mixture of O_2 1 l/min and air 1l/min. An arterial line was placed in the right radial artery and a second 18 gauge IV was placed in the left forearm after induction.

The patient was maintained in a deep plane of anesthesia with an expired sevoflurane concentration from 2.2% to 2.5% throughout the case, and a total of 250 mcg of fentanyl and 1 mg of hydromorphone were administered. An additional 40 mg of rocuronium was given to maintain a train of four count of 0/4 twitches throughout the procedure. The patient was monitored for any sign of airway edema throughout the case. Ondansetron 4 mg IV was administered, and the neuromuscular blockade was antagonized with neostigmine 3 mg and glycopyrrolate 0.6 mg IV. Sevoflurane was discontinued at the end of surgery and the patient was weaned to pressure support ventilation with progression to spontaneous ventilation via the tracheal tube.

Discussion

Hereditary angioedema is a rare genetic disorder that is characterized by hyperactivity of the complement, clotting and kinin systems.[1] This produces edema of cutaneous and mucosal tissues in response to mental or physical stress, minor trauma, illness, or infection.[2-4] Edema can be pronounced in the upper airway, putting a patient at risk for airway obstruction.[4] Surgical stimulation, including laryngoscopy and intubation, can be triggering factors that may initiate an acute episode of airway edema. Treatment of life-threatening angioedema reactions requires understanding the specific type and cause of HAE.

Hereditary angioedema has been classified into a number of categories depending on its cause. Type I and Type II have either insufficient or abnormal C-1 esterase, an inhibitor of C-1 complement protein that initiates the complement cascade. Angioedema with normal C-1esterase inhibitor (HAE C1-INH, formerly known as Type III HAE), is less understood, but is known to be a bradykinin-mediated response.[2-4] HAE may be acquired, as in use of angiotensin-converting enzymes, or hereditary. Hereditary causes include either a defect of coagulation factor XII, an activator of the kinin system, or an unknown genetic cause.[4]

The primary mediator in all types of HAE is bradykinin. Overactivation of the β2 bradykinin receptors result in increased vascular permeability, vasodilation and tissue edema.[2,3] HAE C1-INH shows a higher percentage of edema and swelling in the upper airway than HAE I/II, which may cause edema in extremities, bowel, genitourinary tract, as well as face and upper airway.[3] Histamine is not a key factor in any type of HAE, precluding the routine use of antihistamines or glucocorticoids as effective treatment.[2-4] Edema may be severe enough to cause airway obstruction.[4]

Treatment pathways for HAE Type I/II have been well-documented. A three-tiered system is the mainstay of treatment, including long term prophylaxis, treatment of an acute exacerbation, and treatment in the setting of increased triggers, such as in the perioperative setting.[5] Medication that disrupts the kinin and complement cascade to inhibit the overproduction of bradykinin are paramount. Plasma derived C1-INH concentrate is used in C1-INH deficiency. Plasma kallikrein inhibitors (ecallantide) and bradykinin β2 receptor antagonists (icatibant) are other modalities

used for both short- and long-term prophylaxis. Antifibrinolytics and 17α-alkylated androgens are also used as therapeutic adjuncts but are considered less effective with more potential for side effects.[3] FFP has historically been an accessible option to treat angioedema when other first-line agents are unavailable. The presence of angiotensin II in FFP acts to degrade bradykinin. However, the use of FFP must be weighed against the risk of viral transmission and the presence of a substrate, which may exacerbate edema.[2]

The patient described in this report falls into the category of idiopathic hereditary angioedema with normal C-1 inhibitor (HAE C1-INH). He had a family history of angioedema, normal C-1 inhibitor levels and function, normal Factor XII analysis, and has a negative history of taking ACE inhibitors.

There are currently no approved treatments for HAE C-1 INH. However, an Angioedema Expert Consensus Meeting held in 2013 in Budapest provided some recomendations.[2] First and foremost, the cause of HAE must be determined if possible, to best guide treatment. IgE mediated reactions, which typically present with urticaria, must be ruled out, as these reactions follow a treatment pathway consisting of antihistamines and glucocorticoids. Despite normal levels and function of C1-INH, it has been found that C1-INH concentrate, ecallantide, and icatibant have all had some success in resolving bradykinin-mediated angioedema. Success has also been found with FFP, although it is recommended that this is used with caution due to the presence of substrate, which may exacerbate swelling.[2]

In the case of the patient presented in this report, we researched the patient's medical records and collaborated with the allergist/immunologist following his case. The patient had been maintained on lanadelumab, a plasma kallikrein inhibitor, for long-term prophylaxis, and was not due for another injection until four days following surgery. Ecallantide was being used for acute exacerbations, however it was noted to show decreasing efficacy. FFP had been used with success, despite a minor allergic reaction to a recent FFP infusion.

Although this patient already had an uncuffed tracheal tube in place for airway protection, replacement with a cuffed tracheal tube was required to facilitate positive pressure ventilation. We considered the necessary tracheal tube change to be a potential trigger for exacerbation. Our plan to prevent an acute exacerbation during the procedure was to medicate the patient for anxiety and pain, without completely suppressing his respiratory drive. Reassurance, fentanyl, and midazolam proved to be effective, and the tracheal tube was replaced successfully. Type and crossmatched FFP was present in the room to be given in the event of acute exacerbation. The patient was premedicated with an antihistamine and glucocorticoid in the preoperative setting based on his prior allergic reaction to FFP and on recommendation of his allergist. This was done with the understanding that it would not help any potential HAE exacerbations, but rather allow for the administration of FFP in the event of an HAE attack. Once anesthesia was induced, the patient was purposefully maintained in a deep plane of anesthesia with an inhalation MAC >1, in addition to opioid supplements. Positioning for abdominal surgery allowed for close visual monitoring of the oropharynx throughout the procedure.

The primary treatment in HAE Type I/II for an exacerbation is recombinant C1-INH. It has been noted that C1-INH, ecallantide, icatibant, and FFP have all successfully resolved attacks of

angioedema in individuals with HAE C1-INH.[2] Without guidelines, the decision was made to utilize that which had worked for this particular patient, historically.

Recommendations from the currently available literature integrate the specifics of a patient's diagnosis and history with known therapeutic regimens. There are physiologic nuances within the umbrella diagnosis 'HAE' that influence treatment. Further research is necessary to develop a recommended pathway to treat patients with HAE C-1 INH, and each subset of the condition, such as HAE with normal C-1 INH but abnormal Factor XII, or HAE in the presence of increased estrogen. An anesthesia professional caring for an HAE patient should avoid potential triggers, such as mental stress, anxiety, trauma, infection and surgical stimulation while preparing a plan for use of available pharmacological agents to be used in the case of an attack.

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Pierre Robin Airway Management

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Keywords: Pierre Robin sequence, anesthesia, airway management, natural airway.

Pierre Robin sequence is a rare disease presenting with the clinical orofacial abnormality triad of micrognathia, glossoptosis, and airway obstruction.[1-4] Airway distress and obstruction ranges from mild to severe and is most prominent at birth and in the first few months of life.[2,3] An essential skill for all anesthesia professionals is to be able to successfully manage the airways of neonates and children that present with disease processes that predispose them to possessing a difficult airway. With the use of clinical judgment and skill the airway of a Pierre Robin sequence patient can safely be managed.

Case Report

A 10-year-old, 122 cm, 20.4 kg, male with a calculated body mass index of 13.7 kg/m^2 presented to the pediatric clinic to undergo anesthesia for the removal of a lower extremity cast and pins following a left cavovarus foot reconstruction that took place one month prior to this visit. The current procedure and anesthetic included recasting and x-raying the left foot as well as a magnetic resonance imaging (MRI) study of his brain due to recent headaches accompanied with nausea and an underlying diagnosis of craniosynostosis with concern for increased intracranial pressures. Past medical history included; non-syndromic Pierre Robin sequence (PRS), cleft palate, obstructive sleep apnea (OSA), craniosynostosis, complex de novo unbalanced chromosomal translocation with multiple duplications and deletions, acquired cavovarus deformities of both feet, attention deficit hyperactivity disorder, seizures, asthma, feeding difficulty, hearing loss, esophageal reflux, and delayed developmental milestones. He had an extensive surgical history including mandibular reconstruction, cranioplasty, cleft palate repair, and multiple ENT procedures. Past successful airway management included tracheal intubation using both video and direct laryngoscopy techniques with miller blades; achieving a grade 1 Cormack-Lehane view with all attempts and without complication. Daily medications consisted of methylphenidate 10 mg once a day and melatonin 5 mg once a day as needed for sleep. Preoperative evaluation revealed a cooperative, small for his age, 10-year-old male, with left lower leg cast. Airway assessment showed a Mallampati IV class airway, short thyromental distance consistent with micrognathia associated with PRS, and surgical scaring of the hard palate related to previous cleft palate repair.

Induction of anesthesia was achieved by mask induction using N_20 6 L/min, O_2 3 L/min; Sevoflurane 8% inspired concentration was added to the mixture when patient began to show signs of sedation. Induction took place on the parent's lap; the patient was then lifted to the stretcher and the airway was maintained with jaw thrust and minimal positive pressure from the reservoir bag. Standard noninvasive monitors were applied to the patient, eyes were taped closed, and peripheral intravenous (IV) access was placed in his left hand. General anesthesia was maintained with a propofol infusion at 300 mcg/kg/min. A 60 mm oropharyngeal airway was placed, patient was spontaneously breathing, and anesthesia circuit mask replaced with a simple facemask on O_2 8L/min. After release of jaw thrust, the airway obstructed slightly and was resolved with the placement of a shoulder roll.

The patient's anesthetic depth was maintained with the propofol infusion and with a single bolus of propofol 30mg at the time of the pin removal. After the left foot was recast, the patient was transported to the MRI holding area on facemask O_2 8 L/min, propofol 300 mcg/kg/min, with SpO_2 and $EtCO_2$ monitors. The MRI was completed without issues and the propofol infusion was stopped prior to transportation back to the pediatric recovery area. Patient remained on facemask O2 8L/min upon initial transfer to recovery, vital signs were stable throughout anesthetic time, heart rate 100-120BPM, and SpO_2 99-100%. He was discharged later that morning to home.

Discussion

Pierre Robin sequence occurs in 1 in 5,000 to 85,000 births.[2-4] In addition to the triad of clinical orofacial abnormalities associated with PRS other anatomical features such as retrognathia and cleft palate may be present in some cases. PRS can be syndromic, most commonly associated with Stickler's syndrome, or non-syndromic. Airway obstruction and distress from orofacial abnormalities are hallmark symptoms in all cases of PRS. Most severe obstruction occurs in the first few days to months of life and will require varying degrees of intervention for correction. Management is initially aimed at restoring a patent airway and then correcting the underlying cause of obstruction. Ideally non-surgical intervention measures can be employed until the patient's airway has developed enough to not need intervention. The orofacial abnormalities that cause airway obstruction and distress may also lead to feeding difficulties resulting in failure to thrive, delayed growth, and delayed development. [2,3]

Properly managing the airway of the neonate with PRS is crucial to their care. Depending on the severity of their abnormalities, non-syndromic patients are often managed less aggressively than those with other comorbidities. Patients with non-syndromic isolated PRS are more likely to grow out of airway obstruction as their mandibles grow and develop later into life.[4] The severity of the syndrome will dictate the treatment course for PRS patients. Less severe obstructions can often be managed with positioning the patient prone or lateral, which displaces the tongue forward and clears the airway obstruction. Feeding difficulties may still be present and necessitate the placement of invasive means of feeding or non-invasive feeding tools. More severe cases of PRS, especially those associated with other syndromes, will often require the need for more invasive means of airway management. These interventions range from the placement of naso- or oropharyngeal airways all the way to tracheostomy.[2-4] Most patients with PRS will need some form of surgical correction in the first few months of life, to alleviate airway obstruction and facilitate feeding.

Prior to any intervention or anesthetic, it is essential to complete a comprehensive airway exam. This may consist of placing the patient in different positions to evaluate obstruction, along with a full airway, head, and neck exam. All airway scans and studies should be reviewed to locate areas of obstruction. Inclusion of a multidisciplinary team, including ENT, in planning can be another helpful tool in formulating the safest anesthetic plan. Yin et al.[2] introduced a comprehensive scoring system for predicting the difficulty of ETT placement in the PRS patient. It described the relationship between four assessment criteria which included severity of clinical manifestation (orofacial abnormalities), weight loss since birth (poor feeding), severity of dyspnea at rest (severity of obstruction), and Cormack-Lehane score. Those scoring high in all four categories were at high risk of a failed intubation attempt and should be progressed to the most reliable from of placing an ETT, which is under direct visualization using a fiberoptic scope.

Airway management can be accomplished in many different ways. In less severe cases, placing the patient lateral or prone may be enough to alleviate airway obstruction. Progressing to more invasive forms of airway management, a nasal or oropharyngeal airway may be placed. Supraglottic airways have been shown to be successfully and reliably placed in patients whose airways are unable to be secured with a traditional ETT.[2-5] In the aforementioned patient, an ETT

had been successfully placed with both direct (Miller blade) and indirect video (fiberoptic scope) placement. In most cases of PRS, induction of anesthesia is achieved via mask induction, while keeping the patient spontaneously breathing until the appropriate airway device is secured. With a thorough understanding of the syndrome, a complete airway assessment and exam, the availability of the proper tools and equipment, and a well devised anesthetic plan, patients with Pierre Robin sequence can be more safely managed.

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Case Study of a Patient with Mitochondrial Cytopathy

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Keywords: Mitochondrial disease, mitochondrial cytopathy, anesthesia, neurologic disease

Mitochondrial cytopathy (MC) is a form of multi-systemic mitochondrial disease with complications that often require surgery.[1,2] This disorder affects the DNA of mitochondria, resulting in a decreased production of energy for cellular metabolism. Body systems that have higher energy requirements are most affected, including the central nervous system (CNS), muscular system, and respiratory system.[1,3] This patient population has unique risks involved with undergoing general anesthesia. All volatile anesthetics depress the activity of mitochondria and intravenous anesthetics carry their own risks for patients with mitochondrial disease.[1-3] Anesthetic planning for this patient population requires careful choices to avoid perioperative complications when caring for patients with MC.

Case Report

The patient was a 17-year-old female with a clinical history of mitochondrial cytopathy, epilepsy, developmental delay, hypotonia, and expressive delay. She weighed 33.9 kg with a body mass index of 13.24 kg/m^2. She had no known drug allergies. The patient arrived for removal of hardware from her left hip and adductor muscle chemical denervation of the left thigh for relief of spasms. All laboratory values, including an anion gap, were within normal limits. The patient was wheelchair-bound with muscular contractions and was non-verbal.

The presumptive diagnosis of mitochondrial cytopathy in the patient's medical record had not been confirmed by either muscle biopsy or genetic testing. The anesthetic plan was devised on the acceptance that the patient had the disorder, taking the necessary precautions for a patient with this disease. She had a smooth intravenous (IV) induction with lidocaine 50 mg, propofol 100 mg and fentanyl 50 mcg, and was easily mask ventilated and intubated. No muscle relaxants were used. Anesthesia was maintained with a propofol infusion at 50 mcg/kg/min and sevoflurane 1.2% expired concentration. She had received 300 mL of lactated ringers, but the infusion was changed to 0.9% saline. Dexmedetomidine 12 mcg was titrated for a smooth emergence. Antiemetics including ondansetron and dexamethasone were given. Acetaminophen 340 mg IV was administered for analgesia. The procedure was completed, and the patient recovered without incident in the postoperative area.

Discussion

Mitochondria are the organelles that conduct cellular metabolism and supply the energy for the body via the Krebs cycle. This organelle is responsible for the metabolism of fatty acids, fatty acid oxidation, and oxidative phosphorylation. Electrons enter the transport chain through complex I or complex II and are then transferred to other subsets of the Krebs cycle to eventually form water. This allows phosphorylation of ADP to ATP through complex V. Mitochondria are the only organelles to have their own DNA. It is possible that both defective and normal DNA are present in body tissues, allowing the defects to be unevenly distributed throughout the body. Various body systems have different thresholds before physiologic defects from mitochondrial cytopathy are evident.[1-3] This may cause a wide range of physiologic effects, making the diagnosis of mitochondrial disease difficult.

Mitochondrial cytopathy is a diagnosis that causes a range of physiological issues, many of which affect the care given during the perioperative period. The organ systems most affected by a mitochondrial defect are those with the highest metabolic demand, including the respiratory system, the heart, the GI system, the muscular system, and the central nervous system.[1,3] These are also the body systems most affected by anesthesia.[2] Due to the fact that anesthesia depresses mitochondrial function, patients with this diagnosis are more prone to complications including organ system damage and death.[3]

This population is at risk for respiratory depression, both intraoperatively and in the post-operative period. With a mitochondrial defect, patients have chronic muscular weakness or hypotonia. This combined with anesthetics that can exacerbate muscle weakness and decrease

respiratory drive places MC patients at higher risk for respiratory depression in the postoperative period.[1-3]

Mitochondrial cytopathy can cause chronic electrolyte imbalances. Due to the decreased energy production by the mitochondria, it is important to minimize any increase in the metabolic demand of these patients. This includes keeping blood glucose levels within normal limits, maintaining normothermia intraoperatively, and avoiding prolonged fasting before surgery.[1-5] It is therefore recommended that patients with MC be scheduled as the first case of the day.[2] This population does not metabolize lactate well and often have a persistently high lactate level, leading to chronic lactic acidosis. Because of this deficiency in lactate metabolism, fluids containing lactate such as lactated Ringers should be avoided so as not to increase acidosis.[1-5]

Patients with MC have distinct anesthetic requirements. This population may be very sensitive to volatile anesthetics[1-3] requiring a lower concentration of inhaled anesthetic. They also have an underlying metabolic acidosis, which will decrease the minimum alveolar concentration of inhaled anesthetics.[1] Although this patient had a diagnosis of mitochondrial cytopathy in her medical record, she had not had a muscle biopsy or genetic testing to confirm this diagnosis. The diagnosis was therefore assumed, and appropriate precautions were observed.[1-3]

An advantage to the use of volatile anesthetics is that, although they depress mitochondrial activity, they are rapidly excreted when the anesthetic is discontinued. This allows for the rapid return of mitochondrial function after the end of anesthesia.[3] Parental anesthetics such as propofol, benzodiazepines, ketamine, and etomidate all affect mitochondrial function by either direct or indirect inhibition of oxidative phosphorylation.[1,3] Etomidate, midazolam, and barbiturates affect mitochondria primarily by inhibiting complex I.[3] Research suggests that ketamine also inhibits complex I, but this finding remains inconclusive at this time.[3] Propofol affects mitochondrial function through multiple mechanisms. The greatest effect is through inhibition of acylcarnitine esters by inhibiting acylcarnitine transferase. The inhibition of these esters is implicated as the cause of propofol infusion syndrome.[1,3,5] Because of the multiple inhibitory effects of propofol, high dose or long-term infusions of propofol should be avoided in the patient with MC. Prior case reports regarding the anesthetic care of patients with MC do suggest that boluses of propofol are well tolerated by this patient population.[1,3] Parental anesthetics which require metabolism should be used with caution, as these medications may produce mitochondrial depression that is sustained.[3]

Patients who have pre-existing hypotonia are at risk for airway obstruction, hypoventilation, hypercarbia, and respiratory acidosis.[1-3] These patients have a high sensitivity to non-depolarizing muscle relaxants and will require lower doses of these medications.[3] Because of the hypotonia and muscle-wasting caused by this disease, depolarizing agents such as succinylcholine should be avoided due to the upregulation of nicotinic receptors in the skeletal muscles. This upregulation can lead to an increase in potassium levels when depolarizing agents are used, leading to hyperkalemia and myolysis.[2-3]

There is a hypothetical delayed post-operative risk of metabolic decompensation from the increase in body stress caused by perioperative care. Common contributors include fasting, anesthetic medications, catabolic metabolism, and exposure to pain.[1,3] It is important in patients

diagnosed with MC to minimize all of these stressors. It is also suggested that patients with this diagnosis have a prolonged post-operative observation period as this metabolic decompensation may occur after the standard post-operative care unit stay.

Patients with mitochondrial disease require that certain precautions be taken when undergoing anesthesia. They should be the first case of the day to avoid prolonged fasting. Succinylcholine should be avoided due to muscle wasting and upregulation of nicotinic receptors. This patient population has an increased sensitivity to both volatile anesthetics and muscle relaxants, necessitating increased vigilance in monitoring intra- and post-operatively. Due to a decreased ability to metabolize lactate and an underlying acidosis, lactated Ringers should be avoided. The use of long-term, high dose proprofol infusions must be avoided as these patients are prone to propofol infusion syndrome. With proper precautions, patients with MC can safely undergo surgery and general anesthesia without complication.

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Arachnoiditis: A Rare but Serious Complication of Epidural Blood Patch

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Keywords: post-dural puncture headache, sphenopalatine ganglion block, epidural blood patch, arachnoiditis, chronic adhesive arachnoiditis

Post-dural puncture headache (PDPH) complicates postpartum recovery with disabling pain. The unintended breach of the dural membrane during epidural needle placement results in a cerebrospinal fluid (CSF) leak, CSF hypotension, and "sagging" of the intracranial structures.[1,2] An epidural blood patch (EBP) is the definitive treatment for PDPH.[2] Though largely safe and effective, EBP is not a benign treatment and carries with it risks of acute and chronic injury.

Arachnoiditis, also referred to as aseptic meningitis, is one such risk associated with EBP. Arachnoiditis can result in severe, chronic neurologic deficits.[3-5]

Case Report

A 40-year-old, 90.7 kg, gravida 3, para 2 female presented with request for continuous labor epidural (CLE) analgesia. Pertinent medical history included migraines, tension headaches, obsessive-compulsive disorder (OCD), generalized anxiety disorder, major depressive disorder, and two previous uncomplicated CLEs. For the CLE procedure, the patient was positioned sitting. Standard monitors were applied. Sterile technique was used. A 2% chlorhexidine gluconate and 70% isopropyl alcohol solution was used to prepare the skin. A 17-gauge Tuohy epidural needle was inserted at the L4-L5 interspace with some difficulty. The epidural space was located at 8 cm via midline approach using normal saline and the loss of resistance technique. An epidural catheter was inserted and secured at 15 cm at the skin. There was no evidence of dural puncture. A test dose of 1.5% lidocaine 45 mg with 1:200,000 epinephrine 15 mcg was administered and produced no signs of intrathecal or intravascular injection. A continuous infusion of 0.1% ropivacaine with fentanyl 2 mcg/ml was initiated and discontinued 85 minutes later following spontaneous delivery. The patient reported effective pain relief.

Throughout the following night, the patient complained of a severe headache that pulsated and was greater on the right side of her face. She had a feeling of fullness in her ears and neck and upper back pain that worsened with movement. The patient was encouraged to lie flat and she reported some improvement of symptoms in the recumbent position. Recumbent relief was limited to positions that did not place pressure on the occiput such as with her head turned laterally. Oxycodone/acetaminophen 5/325mg, 2 tablets by mouth, given every 4 hours as needed and alternating hot and cold packs provided no relief. Symptoms continued to be managed by nursing until the following morning when anesthesia was notified.

In the morning on postpartum day (PPD) 1, anesthesia met with the patient to assess and discuss treatment options. EBP was described and transnasal sphenopalatine ganglion block (SPGB) was offered as a less invasive measure. The patient elected to proceed with the SPGB. The patient was positioned in low Fowlers for the procedure. Due to the small volumes of anesthetic being used, monitors were not applied. A topical SPGB was performed using hollow cotton-tip applicators saturated with a 1:1 mixture of 2% lidocaine and 0.5% bupivacaine prior to placement. An applicator was advanced slowly through each nare until resistance was met. They were left in place for 5 minutes, after which, the mixture was reapplied via the hollow applicators and left in place for an additional 5 minutes. The patient reported immediate headache relief, but the pain in her neck and upper back persisted.

Upon assessment approximately six hours later, the patient reported that her headache had returned. An EBP was performed with 20 mL autologous blood injected into the epidural space using sterile technique. The patient was instructed to remain recumbent for 1 hour following the procedure. The EBP did not provide any relief of symptoms. Ketorolac 30 mg IV was administered and was also ineffective. Meperidine 25 mg IM and promethazine 25mg IM were subsequently administered and resulted in improvement.

On PPD 2, symptoms were improved. The patient remained hospitalized for 24 hours of monitoring. On PPD 3, her severe headache returned. IM meperidine 25 mg and promethazine 25 mg was administered and she reported some relief. Several hours later she reported the pain was "moderate" and centralized in the base of her neck. She was unable to move her head without moving her upper body. A hospitalist was consulted, and an MRI was ordered.

The results from the MRI findings included "some clumping and enlargement of the nerve roots of the cauda equina. This raises possibility of arachnoiditis." The anesthesia practitioner explained to the patient that performing a subsequent blood patch in the presence of possible arachnoiditis carries the risk of worsening arachnoiditis and chronic neurologic problems. The patient opted to continue pharmacologic treatments. Following consultation with anesthesia, she was discharged home.

Discussion

Post-dural puncture headache occurs as a result of puncturing the dural membrane separating the epidural space from the intrathecal space. The perforation allows cerebrospinal fluid (CSF) to leak from the intrathecal space.[2,6] This loss of CSF results in intracranial hypotension and sagging of intracranial structures. Traction applied to the meninges accounts for much of the pain that occurs and explains the postural nature of the condition.[2,6] It is believed that the intracranial hypotension leads to cerebral vasodilation which contributes to the pain.[2] The pain can be significantly debilitating and interfere with the ability of a new mother to care for her newborn child. During placement of the CLE, there were no signs of dural puncture in the patient though it could not be ruled out due to subsequent symptoms.

Several modalities exist for treating PDPH. These include conservative options like bed rest, pharmacologic management (simple oral analgesics, opioid analgesics, caffeine, theophyllines, ACTH and analogues, steroids, triptans, gabapentinoids, and others), as well as more invasive procedures like nerve blocks (greater occipital nerve block and SPGB) and epidural administration of crystalloids and "glue." Fibrin glue 3-5 mL via epidural needle has been used to treat PDPH though there are no case reports in obstetric patients and further investigation is required regarding its efficacy and safety in this population.[2] Initial attempts to manage the patient's symptoms with bed rest, cold and hot compresses, analgesics, and a sphenopalatine ganglion block (SPGB) proved inadequate. Because of the severity of the patient's persistent, refractory postural headache, it was decided to proceed to EBP. EBP is considered the definitive treatment for PDPH, though rates of > 90% success reported 40-50 years ago have not been reproduced in more recent studies.[5] Regardless, it remains the most effective treatment option.

The EBP is generally considered safe, though it does carry risks that vary in incidence and severity. Two of the more common risks are repeat dural puncture and back pain.[5] Several neurologic complications have been reported including: spinal hematoma, seizures, cerebral venous sinus thrombosis, facial nerve palsy, infective meningitis, and others.[5] An extremely rare, but serious neurologic complication that has been associated with EBP is arachnoiditis or chronic adhesive arachnoiditis (CAA), depending on etiology.[3-5]

Very few cases of arachnoiditis attributed to EBP have been documented.[3,4] Arachnoiditis following EBP has been described to occur via two different mechanisms. The first involves local inflammation that occurs as a result of the presence of blood degradation products in the intrathecal space.[3] The second, implicated as the cause of CAA, typically involves repeated EBPs using chlorhexidine and isopropyl alcohol solutions for skin preparation.[4] Both mechanisms involve chemical irritation that results in aseptic meningitis. Administration of multiple large-volume EBPs appears to be a significant risk factor.[5] It is possible that either or both mechanisms contributed to the development of the arachnoiditis that was suspect in the MRI findings for the patient. The patient did not receive multiple EBPs, however, she was exposed to chlorhexidine/isopropyl solution during both CLE and EBP. The literature does not specify a minimum number of incidental chemical introductions into the intrathecal space so this cannot be ruled out as a contributing factor.

Arachnoiditis is not well-defined but the generally accepted symptoms include chronic and persistent pain in the lower back and legs, and neurological abnormalities such as hyporeflexia.[4] Symptoms have persisted for longer than two years in patients who have been diagnosed with the complication.[5] Despite the MRI findings, our patient did not exhibit symptoms of arachnoiditis.

Until recently, arachnoiditis was considered untreatable.[7] Greater understanding has led to the development of a treatment regimen that involves the use of medications and physical therapies. These measures aim to restore CSF flow and to prevent tissue remodeling that can result in neurologic impairment and pain.[7] Medications are aimed at suppressing inflammation, inducing neuroregeneration, and pain relief, and include steroids, non-steroidal anti-inflammatory drugs (NSAID), and analgesics.[7] Physical measures include various types of exercise.[7] These treatments are considered “first-generation” and agents used are expected to change as our understanding of the condition improves.[7]

The patient’s postnatal symptoms may not have been a result of PDPH. Her history was significant for tension headaches and migraines. She obtained relief when lying recumbent but only when not putting pressure on her occiput. Each time the anesthesia service encountered the patient, she was sitting upright, which led us to believe that the postural relief was not significant. Additionally, by the time MRI results were available her symptoms had improved, and pharmacologic management was providing tolerable relief. For these reasons and due to the increased risk of CAA with repeated EBPs in combination with the MRI findings, it was determined with patient agreement that a subsequent EBP could possibly do more harm than good. EBP continues to be the best treatment option for relieving symptoms associated with PDPH. But as with most treatments, it carries the risk of iatrogenic harm. Recognizing these risks is important if we are to minimize that harm.

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Complex Obese Pediatric Patient Undergoing Tonsillectomy

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Keywords: pediatric obesity, pediatric tonsillectomy, pediatric obstructive sleep apnea, dexmedetomidine

The increasing prevalence of pediatric obesity necessitates an understanding by the anesthesia professional of commonly associated comorbidities, anesthesia related risks, and evidence-based anesthesia management recommendations.[1] For the obese pediatric patient undergoing tonsillectomy, a thorough preoperative assessment including polysomnography when obstructive sleep apnea (OSA) is suspected but undiagnosed, and evaluation of additional comorbidities such as asthma is necessary. The anesthesia plan should include adequate pre-oxygenation immediately prior to induction, rapid detection and intervention for obstruction and/or hypoxia, and use of an opioid-sparing anesthetic technique that includes intravenous acetaminophen and dexmedetomidine[2,3] Postoperative observation and/or admission should also be considered.

Case Report

A 6-year old obese female (41 kg, 121 cm, >99th percentile) presented to the emergency department for intractable somnolence. The patient's legal guardian reported a history of asthma, snoring, and nasal congestion. Home medications included fluticasone 0.05% nasal

spray, one spray daily, and fluticasone HFA 44 mcg inhaler 10.6 grams, 2 puffs per day. Physical exam revealed 4+ bilateral tonsillar hypertrophy. Laboratory findings were unremarkable. Chest radiography revealed prominent bronchovascular markings compatible with bronchiolitis or active airway disease. Polysomnography revealed severe OSA with a recorded apnea hypopnea index (AHI) of 128, an oxygenation nadir of 60%, and a maximum $EtCO_2$ of 60 mmHg. Echocardiography revealed normal cardiac size and function. Physical assessment findings included a short, thick neck, a large head, and a normal thyromental distance. The patient was uncooperative for assessment of Mallampati classification, however a large tongue and the potential for a difficult airway were noted.

A plan for surgical excision of bilateral tonsils and adenoids was initiated; however, surgery was delayed for three days due to the unavailability of a pediatric intensive care unit (PICU) bed for post-operative monitoring. The patient remained hospitalized in the telemetry unit with intravenous (IV) access until surgery could be scheduled. Bi-level positive airway pressure (BiPAP) therapy was attempted as a bridge to tonsillectomy, but the patient was uncooperative due to the inability to tolerate wearing the mask. While awaiting surgical intervention, hypoxic drive was maintained by avoiding supplemental oxygen and stimulating the patient with every desaturation, which occurred two to three times per hour per nursing staff report.

The anesthetic plan of care included preoperative administration of albuterol inhaler and pre-oxygenation in an upright position facilitated by IV sedation as needed. A rapid sequence induction was planned to secure the airway. Due to the potential for difficult mask ventilation and direct laryngoscopy, a variety of oral airways and blades were made available as well as the C-MAC video laryngoscope. Sevoflurane was planned for anesthesia maintenance to provide bronchodilation as well as dexamethasone IV to reduce airway inflammation. An opioid-sparing technique utilizing acetaminophen and dexmedetomidine for pain management, followed by an awake extubation were planned.

On the day of surgery, the patient was brought to the operating room. Standard non-invasive monitors were applied. Dexmedetomidine 12 mcg was given intravenously for preoperative anxiolysis. Albuterol 2 puffs were administered to induce bronchodilation. The patient was placed in a semi-sitting, sniffing position and pre-oxygenation was initiated using a handheld mask and 10 L/min of oxygen for 5 minutes. Intravenous induction of general anesthesia was performed using lidocaine 30 mg, propofol 100 mg, fentanyl 20 mcg, and succinylcholine 60 mg. Under direct laryngoscopy a grade 1 view was visualized, and the trachea was easily intubated with a 5.0 mm cuffed oral RAE tube. Following induction, dexamethasone 8 mg, ondansetron 3 mg, and acetaminophen 615 mg were administered intravenously. Hemodynamics were maintained within 20% of baseline throughout the procedure. Anesthesia was maintained with 3% sevoflurane, O_2 0.5 L/min, and air 0.5 L/min. The patient was ventilated using the synchronized intermittent mandatory ventilation mode until return of spontaneous respirations, at which point the patient required minimal additional manual ventilatory support for prevention of atelectasis.

Prior to emergence an additional dose of dexmedetomidine 10 mcg IV was administered. Upon completion of the procedure thorough suctioning of the oropharynx was performed by the surgical team. Once protective airway reflexes returned and purposeful movement was

observed, the trachea was extubated. Adequate airway control and respirations were observed, and no desaturations or distress were noted. The patient was transferred directly to the PICU on O_2 6 L/min via simple mask.

In the PICU under close supervision and non-invasive monitoring, the patient remained stable on room air overnight without requiring BiPAP assistance. The patient was subsequently transferred to the medical ward the following day, and discharged home on post-operative day two.

Discussion

Anesthetic considerations for tonsillectomy and adenoidectomy typically include the potential for difficult intubation, need for adequate pain control, prevention of post-operative nausea and vomiting (PONV), and the need for an emergence with minimal coughing and bucking.[2,4] This surgery is common in the pediatric population, and as in this case, comorbidities frequently include a history of OSA and obesity.[2,4] The combination of these factors warrants careful assessment and planning by the perioperative team.

In 2019 the American Academy of Otolaryngology–Head and Neck Surgery Foundation revised their 2011 guidelines for the perioperative management of pediatric patients undergoing tonsillectomy in an effort to educate clinicians and optimize perioperative care.[2] Recommendations pertinent to this case study include early assessment and identification of specific comorbidities including asthma, the conduction of polysomnography preoperatively for patients with suspected OSA, and the recommendation of tonsillectomy for children with overnight polysomnography confirming OSA. Parents and caregivers should be informed that tonsillectomy may not completely resolve OSA and that further treatment may be necessary. Additional recommendations include intraoperative administration of intravenous dexamethasone for PONV and pain reduction, and the overnight observation of oxygen saturation and respiratory status for patients with severe OSA. The authors define severe OSA as an AHI of $\geq$10 obstructive episodes per hour and/or an oximetry nadir of <80%. The patient's AHI of 128, oximetry nadir of 60%, and peak $ETCO_2$ of 60, combined with her preoperative physical status and somnolence heavily influenced the implementation of the aforementioned guidelines.

Descriptive classification of obese pediatric patients deviates from the standardized adult reporting metric of body mass index (BMI).[1] Because children's body composition changes rapidly and at a variable rate influenced by gender and age, average BMI fluctuates significantly. To compensate for these variations, pediatric BMI is measured and reported in percentiles relative to other children of the same gender and age. Obesity is defined as greater than 95th percentile, and severe obesity, greater than 99th percentile.

To optimize the perioperative care of obese children, preoperative oxygenation with 100% oxygen with the head of the bed elevated 25 degrees increases functional residual capacity and reduces incidence of postoperative atelectasis.[1] Additionally, the use of multimodal analgesia including intravenous acetaminophen, dexmedetomidine or clonidine, magnesium, remifentanil, and an opioid-sparing technique can provide optimal pain relief and decrease the incidence of post-operative respiratory depression. Close monitoring and observation are essential

postoperatively, preferably while the patient is asleep, in order to assess for respiratory compromise and facilitate prompt intervention. In this case, due to the severity of the OSA combined with obesity and asthma, the surgical team recommended overnight observation in the PICU.

Repeated hypoxia alters mu opioid receptors and intrinsic pain pathways resulting in hypersensitivity to opioids.[5] For children with OSA, preoperative SpO_2 nadir is reported to correlate with the degree of opioid sensitivity.[5] Respiratory depression and overdose may be avoided by reducing opioid dosing to one-third the normal weight-based dosing, combined with careful titration including maintenance of spontaneous respirations prior to extubation.[6] In this case, the patient received the lower dose of the standard range of dosing for fentanyl (0.5 mcg/kg) based on their actual body weight, with the intent of administering only a one time dose. Acetaminophen and dexmedetomidine were used to decrease opioid requirements.

Dexmedetomidine is an alpha-2 agonist that is becoming increasingly popular in the perioperative setting because of its ability to provide sedation, anxiolysis, and analgesia with minimal respiratory depression.[3] These characteristics make dexmedetomidine an excellent adjuvant to anesthesia for the obese pediatric population despite the fact that it is not currently approved by the Food and Drug Administration for pediatric use. For tonsillectomies specifically, dexmedetomidine 1 mcg/kg IV has demonstrated a similar analgesic effect as morphine 100 mcg/kg with a comparable time to discharge and overall reduction in opioid administration.[7] Additional benefits of dexmedetomidine include prevention of emergence delirium, reduction in the incidence of PONV, and blunting of the sympathetic response to direct laryngoscopy, intubation, and surgical stimuli.[8]

With a reported incidence of approximately 289,000 pediatric outpatient tonsillectomies in the United States in 2018, tonsillectomies are one of the most common pediatric surgical procedures performed.[2] Therefore it is essential for anesthesia professionals to be informed of the practice recommendations and evidence-based interventions that have been shown to provide optimal outcomes, especially in complex populations.[2,4] When caring for pediatric patients with obesity, asthma, and severe OSA undergoing tonsillectomy, a careful preoperative assessment and perioperative plan are necessary. This includes polysomnography, adequate pre-oxygenation, multimodal and opioid-sparing analgesia, combined with careful postoperative observation with overnight admission to optimize outcomes.[1,2]

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C2-Pelvic Fusion in Patient with Hypertrophic Obstructive Cardiomyopathy

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Keywords: Hypertrophic obstructive cardiomyopathy, cardiomyopathy, hemodynamic management

With an incidence of nearly 1 in 500, hypertrophic obstructive cardiomyopathy (HCM) is a pathology that should be familiar to every anesthesia professional.[1] HCM is unique among cardiovascular disorders, both for its potential clinical presentation during any stage of life and the potentially adverse clinical consequences it portends.[1] Proper anesthetic management requires knowledge of the physiologic derangements conferred by the disease. Pharmacologic and hemodynamic considerations play integral roles in maintaining sufficient myocardial function. Anesthetists play a critical role in ushering these patients through their operative course. The following is a case study of a patient with diagnosed HCM undergoing a C2-pelvis fusion.

Case Report

A 52-year-old, 75 kg, 165 cm female presented to the operating room for C2-pelvis fusion for chronic scoliosis and a sub-acute C-4 fracture. Her past medical history included hyperlipidemia, hypertension and hypertrophic obstructive cardiomyopathy with systolic anterior motion (SAM). Her surgical history included a prior C2-pelvic fusion/decompression. Current medications included hydrocodone-acetaminophen, diltiazem, gabapentin, metoprolol and trazadone. A preoperative electrocardiography from one-month prior revealed normal sinus rhythm and laboratory testing was insignificant but for a BNP of 222 mg/dL. Recent echocardiography confirmed a diagnosis of HCM with SAM, a left ventricular ejection fraction of 75% and a left ventricular outflow gradient of 6 mm Hg. Confirmed in the report was evidence of diastolic

dysfunction (prolonged early mitral inflow suggesting impaired relaxation), and severe left ventricular (LV) and septal hypertrophy.

The patient's preoperative hemodynamics were as follows: non-invasive blood pressure 133/81 mm Hg, heart rate (HR) 75/min, respiratory rate 14/min, and SpO_2 99% on room air. The patient was typed and crossmatched for 4 units of packed red blood cells (PRBC), two units of fresh frozen plasma (FFP), and one pack of platelets (PLT).

An 18-gauge intravenous catheter (IV) was secured and a fluid bolus of Plasma-Lyte 600 mL was completed prior to entering the operating room. Standard noninvasive monitors including electrocardiogram, pulse oximetry, and non-invasive blood pressure cuff were applied. Oxygen 10 L/min was administered via simple facemask. Midazolam 2 mg IV was administered and lidocaine 1% was injected intradermally to anesthetize the left radial site and facilitate the arterial line placement. After securing the arterial line, intravenous induction of anesthesia was initiated with lidocaine 80 mg, fentanyl 50 mcg, methadone 10 mg, and ketamine 25 mg. After establishing effective bag-mask ventilation, rocuronium 50 mg IV was administered. The patient's trachea was successfully intubated with a 7.0 mm endotracheal tube (ETT), after which the patient received additional methadone 10 mg and ketamine 25 mg. Throughout induction the patient's arterial blood pressures were maintained between 110/80 and 140/80 mm Hg, HR between 75 and 98/min and SpO_2 95-100%. A propofol infusion was initiated at 50 mcg/kg/min and desflurane was set at 3% in O_2 and air, both set at 1 L/min.

Invasive central venous access lines were placed following intubation. This included a right internal jugular multi-lumen access catheter and a left internal jugular quad-lumen central venous catheter. The patient was turned to the prone position without event. A cell-saver blood salvage transfusion system was established at the patient's bedside.

Throughout the maintenance phase of anesthesia, desflurane 2-3% and propofol 50-100 mcg/kg/min were administered to maintain bispectral index numbers between 30-60. At the start of the procedure, magnesium 2 g IV was administered over 30 minutes as a non-opioid augmentation for analgesia as well as a tranexamic acid (TXA) bolus of 600 mg IV to mitigate blood loss. Medications administered during the maintenance phase included ketamine 0.5 mg/kg/hr IV bolus, TXA 50 mg IV bolus every hour, and infusions of vasopressin 0.01-0.02 mcg/kg/min and propofol 50-100 mcg/kg/min. Approximately 45 minutes into the procedure estimated blood loss (EBL) reached 150 mL, and it was decided to begin colloid replacement, with one unit of PRBCs administered. Arterial blood pressures were maintained between 95/60 and 150/90 mm Hg, HR between 68 and 110/min, BIS values between 15 and 65, and SpO_2 between 96 and 100%. Normal sinus rhythm was maintained. The vasopressin infusion, along with colloid and crystalloid fluids were titrated to keep hemodynamic values within approximately 20% of baseline values. Attention was given to the avoidance of precipitous or large changes in values. Colloids were administered throughout the procedure, with totals as follows: PRBC 3 units, PLT 2 units, FFP 3 units, 4 cell saver units (225 mL each), 750 mL of albumin 5% and 3 L of Plasma-Lyte solution. Final EBL was 2.5 L.

Discussion

This case describes the successful anesthetic course and physiologic management of hypertrophic obstructive cardiomyopathy with systolic anterior motion in a non-cardiac surgery of highly invasive nature. After a surgical duration of 8 hours it was decided the patient would benefit from a more gradual extubation course than could be provided in the surgical suite. The patient was transferred to the intensive care unit with the ETT in situ, on mechanical ventilation. This decision accounted for the large fluid shifts, long surgical time and large surgical insult bestowed upon the patient. The patient was extubated on post-op day 1 without notable sequalae, including absence of myocardial or pulmonological compromise.

The genetic cardiac disorder of hypertrophic obstructive cardiomyopathy is characterized by myocardial anatomic distortion. Although all variations of LV thickening are possible, anterior ventricular septal wall thickening is the predominant feature.[2] This asymmetric hypertrophy is due to muscle cell disarray that creates mismatching between myocytes and arterioles with subsequent small vessel disease and scarring.[2] Consequential mass proliferation results in mitral valve abnormalities, with anterior mitral valve leaflet drag being a characteristic feature. The systolic anterior motion of the leaflet may obstruct the left ventricular outflow tract (LVOT) obtunding cardiac output and precipitating mitral regurgitation.[2] Left ventricular outflow tract obstruction can be easily instigated by decreases in preload or systemic vascular resistance, as well as increases in myocardial contractility or HR.[2] Surgery and anesthesia may precipitate LVOT obstruction, provoking sudden intraoperative death.[3]

New clinical insights afford anesthesia professionals strategies for optimizing perioperative care to help avoid the life-threatening nature of this disease.[2] Management begins in the preoperative setting. As with this case, the cardioprotective effects of anti-arrhythmic and beta-blocking drugs are desired and should be continued, whereas ACE/angiotensin-inhibitors and diuretics should be stopped for their contributions to intraoperative hemodynamic lability.[4] Although benzodiazepines are implicated in a measure of cardiovascular depression, adequate premedication with midazolam alleviates anxiety and the harmful effects of the resulting sympathetic stimulation.[2] Volume expansion in the preoperative setting was implemented to reduce the incidence of hypotension on induction, as well as mitigate the effects of positive pressure ventilation. Although exact volume prescriptions are not provided, addressing a majority of the patient's nil-per-os deficit is recommended to restore preload.[2] When compared to propofol, narcotics such as fentanyl and methadone provide hemodynamic stability for induction.[2] The addition of ketamine provides minimal hemodynamic effect, with added analgesia. A 'slow' induction, with maintenance of a sinus rhythm, reduction in sympathetic stimulation and maintenance of preload and afterload was employed. This avoided increases in contractility and HR or contraction of an underfilled LV, all of which can precipitate SAM and LVOT obstruction. [5] The awake arterial line allowed direct arterial pressure monitoring for early identification of hemodynamic changes during the induction period.

Recommended goals during the maintenance phase of anesthesia with HCM include minimizing the negative inotropic effects of anesthetic drugs.[2] Desflurane was limited to 2-3% and the propofol infusion to 50-100 mcg/kg/min during this case. Desflurane was selected for its rapid and predictable recovery. Maintaining preload, preventing increases in afterload, maintaining

perfusion, controlling arrhythmias and avoiding hypotension and tachycardia must be continuously employed.[2] Variability in blood pressure and HR were limited, and precipitous or dramatic changes were avoided. Hypotension was treated with volume expansion and titration of the vasopressin infusion, as opposed to inotropic agents. Vasopressor selection should avoid drugs that increase HR or contractility.[2] Vasopressin was selected for its absence of effect on both contractility and HR. Smaller tidal volumes (6-8 mL/kg, [350mL]) with increased respiratory rates were elected to prevent reductions in venous return.

Hemodynamic instability may be precipitated by large fluid shifts or blood loss.[2] The near continual administration of colloids and crystalloids in this case prevented LV under-filling and deficits of perfusion. Resuscitation was guided by EBL, hemodynamic values and periodic laboratory sample findings. Hematocrit (HCT) was trended throughout the case at intervals of approximately 90 minutes, with a resuscitation goal of maintaining the HCT > 26%. This course of action was based on a maximum HCT reduction of 25% from baseline. This approach aims to maintain an approximation of adequate oxygen carrying capacity while preferentially preserving volume status over hemoconcentration. Recommendations for exact values of hemoglobin/hematocrit goals are unavailable, and as such resuscitation efforts should be guided by clinical findings. In this case, HCT was maintained between 28-36%, with a final HCT of 30%.

In addition to ASA standard monitors, central venous pressure (CVP) monitoring via invasive central access provides assessment of preload as well as access for vasopressor infusions and prompt fluid administration.[2] Other useful measures of volume status include stroke volume variation and pulse pressure variation, as measured via pulse index continuous cardiac output monitoring.[2] Transesophageal echocardiography (TEE) has value in providing real-time assessment of preload, fluid responsiveness, CO, and wall and valve function.[2] In this case TEE was reserved for hemodynamic deterioration which did not arise. The decision to implement two central access catheters provided the ability to concurrently administer blood, crystalloids, vasopressin and propofol infusions, as well as monitor the CVP.

In summary, the anesthesia for this case was conducted in accordance with recommended strategies for success. The patient's myocardial pathophysiology was considered at each stage of the procedure, and actions taken prevented disastrous decompensation. This case presented the particular challenges of lengthy and profound fluid shifts. Vigilant fluid resuscitation, adequate invasive monitoring and access, along with careful pharmaceutical selection guided the successful management of the case.

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Anesthetic Management for a Parturient with Brugada Syndrome

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Keywords: Brugada syndrome, anesthetic management, parturient, pregnancy

Brugada syndrome is an arrhythmogenic autosomal dominant genetic disease affecting cardiac sodium channels. Sudden cardiac death results from polymorphic ventricular tachycardia or ventricular fibrillation, usually originating from the right ventricle. Diagnosis is made via electrocardiogram and presence of symptomatology like syncope or nocturnal agonal respirations. Increased susceptibility to cardiac dysrhythmias occurs during certain conditions such as fever or situations causing increased vagal tone. Medications that trigger tachycardia or bradycardia should be administered cautiously as stimulation of cardiac electrolyte channels can exacerbate patient condition.[1] Palliative treatment for Brugada syndrome is isoproterenol; definitive treatment requires automatic implantable cardioverter defibrillator (AICD) placement.

Case Report

A 30-year-old female presented for a repeat cesarean section. The patient's current pregnancy was uncomplicated. The patient's history was remarkable for Brugada syndrome elucidated via genetic testing; however, 12- lead electrocardiogram showed no signs of cardiac arrhythmias. The patient denied any abnormal breathing patterns or occurrence of seizures or syncope and exhibited no signs of cardiac or respiratory distress. During the interview, the anesthetic plan was discussed, and the patient agreed to placement of external defibrillation pads prior to delivery.

Upon entering the operating room, the patient was placed in the sitting position. Defibrillator pads were placed in upper right and lower left chest and contact was verified by intact monitoring through the defibrillator. Ondansetron 4 mg IV was administered followed by a 500 mL bolus of lactated Ringers. After the patient's lower back was prepped and draped, a 25-gauge pencil-point needle was inserted into the subarachnoid space. Spinal anesthesia was achieved with the intrathecal administration of bupivacaine 0.75% 1.4 mL, fentanyl 15 mcg and preservative-free morphine 150 mcg. The patient was moved to the supine position with left uterine displacement and immediately received prophylactic ephedrine 10 mg IV.

The patient remained conscious and conversational with the anesthesia provider throughout the procedure. The anesthetic plan included avoidance of the use of phenylephrine due to the patient's condition; therefore, ephedrine was used sporadically to maintain the blood pressure within 20% of baseline. Isoproterenol was available in case of signs of cardiovascular distress or

arrhythmias. ECG leads II and V5 were monitored from patient entry into the operating room until transfer back to the patient's room.

The patient received a total of 120 mg IV ephedrine throughout the case, administered approximately every 5 minutes to maintain blood pressure. After the fetus was delivered, oxytocin 30 units in normal saline 500 mL was initiated. The patient denied nausea and the blood pressure was maintained within the lower 20% of baseline vitals. The heart remained in normal sinus rhythm throughout the procedure, confirmed via 2-lead electrocardiogram. The neonate was examined and was given back to mother for skin-to-skin contact and return transport to the patient's room.

Discussion

Brugada syndrome occurs as the result of defective electrolyte channels in a structurally normal heart leading to the development of fatal arrhythmias and sudden death.[2] Anticipation of potential intraoperative problems that may arise from Brugada is crucial to the prompt identification and treatment of syndromic exacerbations. Additionally, a knowledge of conditions that may exacerbate this condition is important. Patients who are positive for Brugada exhibit a Type 1 electrocardiogram (ECG) rhythm, expressed as a "coved" ST segment with an inverted T wave larger than 1mm. This pattern may be elicited either spontaneously or through medication trigger.[3] This patient's familial history of Brugada strongly suggested that she may have the syndrome; however, her ECG was normal. Signs and symptoms of syncope, seizures, nocturnal agonal respirations, or a history of polymorphic ventricular tachycardia or ventricular fibrillation were also absent in the patient's history.

Patients with Brugada syndrome are most susceptible to life threatening dysrhythmias when the heart rate is either elevated or depressed. Therefore, careful management of cardiac chronotropy is of utmost importance.[1] Moreover, exacerbations of Brugada syndrome typically occur at night, likely due to the predominance in vagal tone during sleep.[3] Consequently, it may be prudent to avoid spinal anesthesia due to the subsequent sympathectomy that it may cause. However, when considering the risks and benefits of spinal anesthesia as compared to general anesthesia for the parturient with Brugada syndrome, spinal anesthesia was still determined to be the best anesthetic choice for this patient.[4] General anesthesia poses a danger for the fetus and the mother. Stimulating the patient during instrumentation of the airway during a necessary rapid-sequence intubation poses too many unnecessary risks to the patient that can be otherwise avoided in this overall healthy mother.

After identifying possible triggers for dysrhythmias in the parturient with Brugada syndrome, proper monitoring and preparation should be employed in case of emergency. Brugada syndrome primarily involves the right ventricle; electrocardiogram monitoring should focus on anterior precordial leads V1-V3.[3] For this case, the patient did not have any history of dysrhythmias, nor did she have any issues with her first cesarean section. Likewise, she denied any incidence of or changes related to dyspnea or palpitations. In retrospect, the patient may have been better optimized in monitoring for exacerbations of Brugada if leads V1, V2, or V3 were followed as opposed to the conventional II and V5 leads observed during the case.

Finally, interventions should be planned in order to optimize a patient with Brugada syndrome.[2] For this case, these included speaking with the patient regarding the anesthetic plan and the placement of defibrillator pads on the patient before the institution of the spinal anesthetic. A discussion was held regarding the possible need for rapid change to a general anesthetic if the patient was to convert into a life-threatening arrhythmia. The optimal treatment for Brugada syndrome is an AICD to convert from lethal rhythms.[2] This patient did not have a defibrillator or cardioverter implanted. Transcutaneous cardioversion/defibrillation was the chosen method to treat any potential arrhythmias. Moreover, since both bradycardia and tachycardia can trigger an exacerbation, phenylephrine was avoided throughout the case due to potential production of bradycardia. Instead, ephedrine was the vasopressor utilized, due to its dual effect in stimulating beta receptors and inducing catecholamine storage release. These effects would blunt the sympathectomy that the spinal anesthetic would create. It should be noted that small titrations of phenylephrine do not guarantee the triggering of a dysrhythmia, while large doses of ephedrine can produce tachyphylaxis and acidemia in the baby during delivery.[4,5] Despite that, the anesthetic plan was determined to be the same as was administered for the patient's previous delivery: spinal anesthesia and use of only ephedrine as the pressor of choice. Ondansetron was used as a vagolytic prior to spinal anesthesia placement, potentially blunting the profound sympathectomy from the spinal while also reducing the possibility of nausea and vomiting.[5]

The medication of choice for patients with an exacerbation of Brugada syndrome is isoproterenol or phosphodiesterase III inhibitors. These medications do not affect the sodium, potassium or calcium channels which, depending on the type of Brugada syndrome present, could make the condition worse.[6] Avoidance of Class I and II antiarrhythmic medications, such as procainamide and flecainide, is another imperative to minimize chances of triggering an arrhythmia in this patient population. Lastly, maintenance of normothermia is important for Brugada syndrome patients as fever can trigger ventricular arrhythmias. Aggressively treating large fluctuations in temperature is vital to optimizing these patients intraoperatively.[2]

Patients with various rare conditions will present to anesthesia providers. The goal is to have a comprehensive knowledge of the comorbidity, obtain a thorough patient history and create a plan to optimize the patient before and during the procedure. Taking time to build a rapport with the patient is a significant factor in properly preparing and implementing a tailored anesthetic plan. As with anything done in anesthesia, identifying risks, monitoring for untoward events, and intervening both before and during changes in patient status will help to ensure that patients receive the best care possible.

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The Role of Telehealth in Pre-anesthesia Practice

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Keywords: Anesthesia, peri anesthesia, pre-anesthesia interview, telehealth, telemedicine

Introduction

According to the Institute of Medicine (IOM), telehealth is defined as "the use of electronic information and communications technologies to provide and support health care when distance separates participants."[1] The advent of telehealth can be traced back to the 1960s by the National Aeronautics and Space Administration (NASA) scientists.[2] To monitor the physiological conditions of the astronauts, NASA scientists created technologies that allowed long-distance transmissions of medical data through space.[2] Many healthcare facilities are now incorporating telehealth technology into how anesthesia providers collect and formulate pre-anesthetic evaluations (PAE). The PAE is performed before elective surgical procedures and includes an airway evaluation, review of the patient's medical records, laboratory data, and diagnostic test results. The objective of the PAE is to assess co-morbidities, reveal disease processes, and synthesize a patient-specific anesthetic plan designed to guide perioperative care.[3] The goal of this evidence-based practice review is to examine the role telehealth can provide in pre-anesthetic interviews and evaluations. Researchers are finding the use of telehealth for PAE can provide benefits to both patients and clinicians.

Methodology

Evidence-based Practice Model
As the framework for this evidenced-based review, the population, intervention, comparison, outcome, and time (PICOT) were identified to formulate a research question. For surgical

patients and their providers (P), can telehealth be used to effectively perform or supplement (I) pre-anesthetic evaluations (T), compared to traditional face-to-face interviews (C), to increase efficiency of anesthetic management and decrease overall cost (O)?

Search Models

An electronic literature search was performed using the following online databases and search engines: PubMed, The Cochrane Library, Cumulative Index to Nursing and Allied Health Literature (CINAHL), MEDLINE, and Google Scholar. Boolean operators were utilized in the database inquiries. PubMed's "Related Articles" was utilized to examine further evidence. Keywords included telehealth, telemedicine, mHealth, peri anesthesia, preanesthetic evaluation, anesthesia, surgery, presurgical, difficult airway. Systematic reviews, literature reviews, retrospective analyses, and randomized controlled clinical trials (RCT) were included. The literature search yielded current, high-level evidence by limiting the sources to English text, scholarly peer-reviewed journals, academic journals, and studies published between 2011 to 2019.

Levels of Evidence

The sources utilized for this review were critically appraised and assessed. The method presented by Melnyk and Fineout-Overholt[4] was used to determine the levels of evidence of the obtained sources. A summary of the literature analyzed for this review is presented below (see Table).

Literature Review

Access to Care

As technology has evolved and become increasingly inter-connected, the demand for remote, electronically integrated healthcare has increased by both the patients and the providers. Physicians and other healthcare providers routinely made house calls in the past; however, this practice is no longer feasible. Rather than creating a new health service, telehealth provides a more efficient method of delivering existing services. In geographical locations where specialists and access to healthcare are limited, telehealth can provide a tangible solution. The implementation of telehealth services has been shown to increase access to care and convenience to patients in both rural and urban settings.[2-6]Additionally, telehealth can bridge the gap and provide healthcare access to those with impaired mobility and transportation challenges.

Powell et al. retrospectively analyzed the implementation of a scheduled telehealth video visit program at a large urban multihospital health system in Philadelphia, PA. This mixed-method study provided surveys to eligible participants following their scheduled telehealth visit. A total of 3,018 scheduled video visits took place, and 764 participants responded to the survey. The researchers concluded that 86.0% (652/758) responded that the scheduled video visit facilitated access to care.[7]

A prospective randomized controlled trial (RCT), conducted by Applegate et al., examined the effectiveness of pre-anesthetic evaluations, via telehealth, in a group of 155 adults scheduled for head and neck surgery at Loma Linda University Medical Center (LLUH) in California. The LLUH is an academic medical center that draws patients from across several hundred miles.[6]

The study found that participants randomized to the telehealth PAE benefitted from fewer appointments, less travel time, and less time off work; these findings imply the improved access to care, particularly to those living in rural areas who travel several hours for each appointment. [6]

Financial Impact

In more traditional settings, patients scheduled for elective surgery are required to make an additional trip to the surgeon's office or hospital, prior to surgery, to provide information regarding their PAE. Additionally, for some individuals in remote areas, the nearest hospital may be hundreds of miles away making an additional trip an extreme burden to the patient. The costs, accrued by the patient, for transportation and wages lost from work absence, are not reimbursed by health insurance companies. In the RCT previously mentioned by Applegate et al., researchers concluded that the elimination of an in-person PAE and associated time off from work, accompanied by potential costs related to travel and childcare, could provide significant cost benefits to patients. [6]

The recent shift to payment for performance, as outlined by the Centers for Medicare & Medicaid Services (CMS) reimbursement, has forced organizations to re-structure their clinical and billing processes to provide more efficient, effective, patient-focused care. A 2019 proposal, by the CMS, was designed to expand Medicare reimbursement for healthcare services, via telehealth. The new reimbursement regulations will allow practitioners to provide remote visits to patients in both rural and urban settings.[8] According to the systematic review on the use of telemedicine in surgical care, conducted by Asiri et al., researchers concluded the development of this technology [telehealth] is not yet accessible to all hospitals. Asiri et al. supports the previous claim by citing a 2017 clinical trial by Mandzuka et al. in which researchers concluded that the direct cost savings of telehealth are still not significant for the healthcare system.[1]

Experience

Numerous studies have examined provider and patient satisfaction with telehealth services. The previously mentioned RCT, Applegate et al. found PAE staff reported high satisfaction with the ability to obtain history, discuss anticipated problems, and provide instructions for all patients during the telehealth enabled PAE. Additionally, the PAE clinicians reported that the telemedicine cameras provided excellent views to allow completion of the airway examination.[6]

Schoen and Prater systematically reviewed the role of telehealth in pre-anesthetic evaluations. Researchers utilized PubMed, Cochrane Library, online medical data, ancestry approach, and Google Scholar to identify a total of 115 potential evidence sources. Of the seven sources meeting criteria for the inclusion of their review, five sources concluded that telehealth consultations for PAE were not only as reliable as those conducted by traditional in-person methods but also provided the pertinent information needed to develop a safe anesthesia plan. Interestingly, the same five sources previously mentioned indicated that patients have a positive perception of the virtual PAE, are more accepting of this technology, and prefer it to a face-to-face evaluation.[3]

Asiri et al. systematically reviewed the use of telemedicine in surgical care. MEDLINE, EMBASE, CINAHL, and Science Direct were utilized to yield 24 studies included in the systematic review out of the 6678 potential sources identified. Three studies found preoperative

diagnosis via telemedicine was as accurate as interventions carried out in usual conventional clinics. Additionally, researchers concluded that nine studies reported high patient satisfaction with the use of telemedicine.[1]

Anesthesia Implications

Telehealth technology is becoming an essential tool in healthcare delivery.[1] Using telehealth, healthcare providers can remotely assess and address their patient's health-related issues with the use of devices such as computer webcams, handheld devices, and smartphones. Video conferencing technology allows for peripheral devices, such as incentive spirometry, ECG monitoring, high-definition airway photography, and electronic stethoscopes to be attached to a computer in which an interactive examination can be performed.[3]

Applegate et al. conducted an RCT investigating the impact of telemedicine pre-anesthesia evaluation on perioperative processes. Findings from this RCT provide the highest-level of evidence source for this review. Two hundred participants consented to participate in the study, but 40 subjects were eliminated before randomization. The one hundred and sixty subjects were randomly assigned an in-person PAE or telemedicine PAE. The primary outcome measure was inadequate PAE as a result of missing documentation, testing, consultation, or physical examination findings should have been identified during the PAE that resulted in a preventable day of surgery delay. The secondary measures included prediction of a difficult airway, concordance of PAE with day of surgery physical exam findings, and satisfaction scores. The preanesthesia clinic had telemedicine equipment installed for this trial. A wireless, mobile cart was suited with a high-definition pan-tilt-zoom camera capable of real-time videoconferencing with two-way video and audio communications. An electronic stethoscope and specialized headphones, capable of detecting the low frequencies of heart and lung sounds, were also mounted to the mobile cart. The electronic stethoscope was used for the cardiopulmonary exam, whereas the general examination camera was used to complete the adapted American Society of Anesthesiologists (ASA) 11-point airway examination.[6] Staff anesthesiologists were blinded to group assignment and evaluated all patients on the day of surgery. The anesthesiologist found the documented findings of the PAE staff to be consistent with their heart and lung examination results. Incomplete PAE, due to the unavailability of echocardiogram results the morning of surgery, resulted in a day of surgery delay in 1 telemedicine patient. The in-person airway examinations identified more instances of airway management difficulty (4 of 10) compared to telemedicine (3 of 15); however, no significant intergroup difference was found ($P = .54$).[6]

Table. Recent Literature Related to Telehealth Use in Pre-anesthesia Evaluations

Articles	**Description**	**Level of Evidence**	**Outcome Measures**	**Results**	**Strengths and Weaknesses**
Asiri et al., 2018	Investigation into the broad range of telemedicine technologies used in surgical care	Systematic Review, Level I evidence	Examine telemedicine use and surgery for pre-, peri-, or post-surgery periods.	Telemedicine in surgical care can provide benefits to both patients and clinicians.	Strengths: High-level evidence Weaknesses: List of excluded studies was not provided

Clancy SP., 2015	Law review in support of the use of telehealth in the state of Oklahoma	Systematic Review of descriptive & qualitative studies, Level III evidence	Telehealth ability to increase access to care and improve health outcomes	Telehealth increases access to health care, improves health outcomes, lowers health care costs, but could breakdown patient-provider relationships	Strengths: Robust methods Objective was clearly stated Inclusion criteria were stated Weaknesses: Lower level evidence Narrow audience Overt bias
Schoen DC, Prater K., 2019	Examined the evidence evaluating the effectiveness of using telehealth when performing the PAE	Systematic Review, Level I evidence	Evaluate the efficiency and reliability of telehealth for PAE consultations	PAE can be carried out successfully using telehealth and that both subjects and investigators reported satisfaction with the use of telehealth when performing the PAE	Strengths: Robust methods Current evidence cited Weaknesses: Conflicting data List of excluded studies was not provided
Mullen-Fortino et al., 2018	Retrospective analysis of 7,803 evaluated; 361 with telemedicine and 7,442 without telemedicine	Retrospective Analysis, Level III evidence	Examine the impact of telemedicine on the presurgical assessment	Evidence supports the use of telemedicine for PAT in terms of access (efficiency, patient experience, and effectiveness	Strengths: Robust methods Current evidence cited Weaknesses: List of excluded studies was not provided Measurement of time saved was biased toward the null
Applegate et al., 2013	Pre-surgical screening performed with the use of	RCT, Level I evidence	Examining the adequacy of telehealth PAE, including	The information gathered via telehealth	Strengths: Robust methods Controlled variables

	general examination camera and electronic stethoscope. Exam conducted by a nurse practitioner or first- or second-year anesthesiology resident		difficult airway predictions, in order to prevent day-of-surgery (DOS) delays	was deemed adequate, and no DOS delays or cancellations were observed	High-level evidence Weaknesses: Study participants were from a single specialty clinic
Powell et al., 2018	Retrospective analysis of 3018 scheduled video visits at one extensive urban academic-affiliated health system in Philadelphia, PA	Retrospective Analysis, Level III evidence	Investigate and report the health system and patient experiences with the implementation of a telehealth scheduled video visit program across a health system	Patients found the use of scheduled video visits made it easier to get care and the majority perceived time saved, suggesting that use of telehealth for scheduled visits can improve potential access to care	Strengths: Objective was clearly stated Inclusion criteria were stated Robust methods Current evidence cited Weaknesses: List of excluded studies was not provided

Conclusion

This literature review was conducted to provide evidence related to the benefits and barriers of implementing PAE telehealth services into anesthetic management. The potential time and cost savings from alternative method of healthcare delivery have been proven to provide similar or supplemental services to in-person evaluations. As internet connections, telecommunications, smart phones, wearables, tablets, and other electronic devices become more affordable and faster, anesthesia providers and their patients will benefit from the added ability to collect and analyze the data needed to produce a safe, individualized, and more efficient anesthetic plan. In addition to the high patient satisfaction with telehealth PAE, evidence supports the distinct advantages telehealth can provide to individuals in remote and rural areas where healthcare access can be difficult.[1-3, 5-8]

Despite the evidence supporting the safe and effective use of telehealth in the PAE, distinct implementation challenges still exist. For example, it would be discriminatory to presume all patients have access to this technology. Other possible barriers to the integration of telehealth might include: the associated upfront cost required to install the necessary equipment and train the staff who likely have varying degrees of digital literacy; patient apprehension stemming from concerns of inferior care and/or the deterioration of the patient-doctor relationship; lack of reimbursement to the providers; anesthesia provider data overload; data quality, security, and liability concerns; as well as the need for heightened regulatory and accreditation agency oversight.[9] In an effort to provide national direction to advance health information technology to make healthcare more effective and safer for all Americans, the National Quality Forum (NQF), a project funded by the U.S. Department of Health and Human Services (HHS), released a detailed report, in August of 2017, offering methods to assess and improve telehealth quality and interoperability.[10]

This literature review has found sufficient evidence to support the claim that telehealth can be used to effectively perform or supplement pre-anesthetic evaluations, compared to traditional face-to-face interviews, to increase efficiency of anesthetic management and decrease overall cost to providers and their patients. Due to its documented success, telehealth will inevitably play a vital role in the future delivery of many anesthesia services; therefore, further research, pertaining to the barriers of implementation, should be explored.

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Mentor: Maria Ledbetter, DNAP, CRNA, COI

The Clinical Utility of Preoperative Pharmacogenomic Testing

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Keywords: pharmacogenomics. clinical utility. preoperative testing

Introduction

Pharmacogenomic testing has become a rapidly growing topic in the world of healthcare and advances in science and technology have opened doors to personalizing medicine.[1] Certified Registered Nurse Anesthetists (CRNAs) are involved in this process of applying pharmacogenomics to patient care.[2] It is important for the anesthetist to understand the difference between pharmacogenomics and pharmacogenetics; two topics that often unintentionally become intertwined. Pharmacogenomics is the technology and foundation that studies how an individual's entire genetic makeup affects drug response.[3] Pharmacogenetics is defined as the study of a single gene and how it affects drug response.[3] Pharmacogenomics can be used as a clinical tool in order to gauge how an individual's genetic makeup will guide metabolism by evaluating the cytochrome P450 (CYP450) system for drug metabolism.[4] The results gained from the pharmacogenomic testing of the CYP450 system can then be integrated with the anesthesia provider's pharmacologic plan of care.[4] More specifically, the results can give the provider insight regarding what drug and what dose might be best for this specific patient.[5] Pharmacogenomic testing allows for personalized medicine by allowing providers to tailor pharmacotherapy to a specific patient based on the predicted response to therapy.[5] Blood tests are used in order to predict drug therapy responses based on how the individual metabolizes certain various medications. This is a novel application to the perioperative setting but may prove to be beneficial in the perioperative setting by better understanding an individual's genetic makeup and response to therapy.[1]

Although the field of pharmacogenomics has been expanding, the integration in preoperative testing has been delayed.[4] In the psychiatric population, patient outcomes have been shown to be improved when pharmacologic decisions are made based on genetic results.[5] One of the major contributing factors associated with this delay deals with anesthesia providers lack of knowledge. Riddle et al.[7] found that CRNA's do not have enough knowledge about pharmacogenomic testing and the interpretation of results deeming this tool clinically irrelevant. A current review of pharmacogenomic testing and its clinical utility in anesthesia practice was conducted and summarized.

Methodology

Purpose and Evidence-Based Practice Model

The purpose of this literature review was to determine the clinical utility of pharmacogenomic testing among Certified Registered Nurse Anesthetists. In this review, clinical utility refers to anesthesia practices and how pharmacogenomic testing can benefit perioperative outcomes. The PICOT format was used in order to formulate the following question for this literature review: For Certified Registered Nurse Anesthetists (P), is pharmacogenomic testing (I) versus non-pharmacogenomic testing (C) useful at guiding anesthetic plans (O) throughout the perioperative period (T)?

Search Terms and Databases

Search terms for this literature review included anesthesia, pharmacogenomic testing, clinical utility, and preoperative testing. Databases used in this search include CINAHL, PubMed, and Allied Health Source. The literature reviewed ranged from the years of 2011 - 2019 and involved preoperative pharmacogenomic testing in perioperative subjects.

Levels of Evidence and Inclusion Criteria

The level of evidence included in this literature review is a combination of mixed-method analysis, randomized control trials, systematic reviews, and meta-analysis that met level I - V criteria. Studies with low level evidence were excluded from this literature review.

Literature Analysis

Greden et al. [5] conducted a double-blinded randomized control trial to assess the impact of pharmacogenomics on clinical outcomes in psychiatric patients diagnosed with major depressive disorder. One thousand one hundred and sixty-seven outpatient subjects were enrolled in the trial and each patient was randomly assigned drug therapy based on pharmacogenomic testing versus drug therapy with non-pharmacogenomic testing.[5] The primary outcome assessed in this trial was symptom improvement based on the Hamilton Depression Rating Scale. Secondary outcomes assessed were response and remission. Greden et al.[5] found no significant difference between the two groups regarding symptom improvement (27.2% vs 24.4% $p = 0.107$). Response (26.0% vs 19.9% $p = 0.013$) and remission (15.3% vs 10.1% $p = 0.007$) outcomes measured were significantly different after eight weeks. Although pharmacogenomic testing did not significantly improve mean outcomes, it did improve response and remission rates for difficult-to-treat depression patients over non-pharmacogenomic treatments.[5]

Huang et al.[6] used genetic testing in metastatic colorectal cancer patients receiving chemotherapy. This study explored three specific single-nucleotide polymorphisms (SNPs) in order to evaluate the predictive role of these nucleotides throughout the chemotherapy course. Three SNPs were used and genotyped in 137 patients with metastatic colorectal cancer. Chi-squared tests, logistic regression models, and operating characteristic analysis were used to evaluate correlations. Genotype GA/AA of SNP rs2306283 of the SLCO1B1 gene and genotype GG of SNP rs1051266 of gene SLC19A1 were linked to higher response rates (odds ratio = 3.583 and 3.521, 95%CI = 1.301-9.871 and 1.27-9.804, $p = 0.011$ and $= 0.013$).[6] The response rate in patients containing both genotypes was 70% compared to 19.7% in remaining patients

(odds ratio = 9.489, 95%CI = 2.191-41.093, p = 0.002).[6] The authors found that these polymorphisms of solute carriers may be beneficial to predict a response to this specific chemotherapy regimen in those with metastatic colorectal cancer.

Riddle et al.[7] conducted a mixed-method study searching for impressions of pharmacogenomic testing within Certified Registered Nurse Anesthetists. The authors used a qualitative-quantitative study with interviews to create a survey geared toward thoughts anesthesia providers have about pharmacogenomics and preoperative testing. In order to formulate the survey, 10 anesthesia providers were interviewed, and emerging themes were used at the end to compose the survey. The survey was then sent to a randomly selected 6000 AANA members in a blinded fashion. Individuals were able to fill out this survey with any electronic device. Of the 6000 eligible CRNAs, 325 responded (about 5%). Incomplete surveys were discarded and a total of 262 surveys were used to conduct data analysis. The survey contained questions based on the following three factors: benefits, knowledge, and concerns of pharmacogenomic testing. Fourteen questions were composed, and each question consisted of a unidirectional, Likert-style type question that aimed to measure a single concept. Surveyors were prompted to answer questions based on a 0-10 scale where 0 represented “completely disagree” and 10 represented “completely agree”. After factor analysis, the authors found that barriers to pharmacogenomic testing include the anesthesia providers lack of knowledge on the topic, training on specific testing, and how to order and use tests (mean = 1.23 on 10-point scale). Therefore, its clinical utility is low and in order to increase the utility there must be an increase in the anesthesia providers knowledge of this topic. Strengths of this study involve the random selection of interviewees and how easy the survey was to complete. Weaknesses include the small sample size and the initial 10 providers that helped formulate the survey.

Sengore et al.[8] aimed to provide the first assessment of the impact pharmacogenomics guided pain management has following major abdominal surgery with an enhanced recovery protocol. The authors looked at data following open and laparoscopic ventral hernia repairs after patients were given a guided analgesic regimen based on assessment of individualized CYP metabolism. A series of 50 open and laparoscopic colon resections or major ventral hernia repairs had a pharmacogenomic guided medication therapy based on assessment of various genes obtained from buccal cells. Patients in the study were compared to 47 patients who had previously undergone the same surgeries but were managed with an enhanced recovery protocol. Pain scores and the Overall Benefit of Analgesia Scores (OBAS) were compared between groups. The study found that cases with patients who underwent CYP450 assessment often required frequent analgesia modifications which would not have normally been done with the normal enhanced recovery protocol. The data obtained showed the study populations were similar in age, gender, and case type pharmacogenomic group (mean age 64.5 years; female/male ratio 64%/35%; colon/hernia: 44/5) and non-pharmacogenomic group (mean age 60.6 years; female/male: 47%/53%; colon/hernia: 42/5). The mean overall OBAS pain scores were compiled from post-operative day one through postoperative day five. The data showed a significantly (p = 0.01) lower OBAS rating from postoperative day one through day five (day one, 3.8 vs 5.4 day five, 3.0 vs 4.5).

Articles	Description	Results	Conclusion
Greden JF, Parikh SV, Rothschild AJ, et al.	1167 outpatient subjects underwent a double-blind randomized control trial to determine the impact of pharmacogenomics on clinical outcomes in patients with major depressive disorder.	The authors found no significant difference in symptom improvement in patients when treated with pharmacogenomic guided practice vs non-pharmacogenomic guidance. Significant differences were observed in response and remission rates in subjects.	Pharmacogenomic guided practice did not improve mean outcomes but it did improve responses and remission rates in difficult-to-treat depression patients versus non-pharmacogenomic practice. Pharmacogenomic based practice may be beneficial in difficult-to-treat populations.
Huang L, Zhang T, Xie C, et al.	Three single nucleotide polymorphisms (SNPs) were used and studied in 137 subjects with metastatic colon cancer. The SNPs were used to evaluate the predictive role of nucleotides throughout chemotherapy regimens.	The researchers observed a significant difference in the SLCO1B1 and SLC19A1 genes which were associated with higher rapid response rates. The response rate was 70% in subjects with both genes compared to 19.7% in subjects with the genes.	The polymorphisms studied showed relevance in predicting responses to chemotherapy courses in those with metastatic colon cancer.
Riddle D, Gregoski M, Baker K, et al.	This mixed-method study searched for impressions of pharmacogenomic testing among Certified Registered Nurse Anesthetists. A survey was generated and completed by 262 AANA members and factor analysis was used to determine the anesthetists' knowledge of pharmacogenomic testing.	The authors found that pharmacogenomic testing is rarely utilized due to the anesthesia provider's lack of knowledge on the topic. The survey showed five common themes present referring to this lack of knowledge. These themes included a lack of understanding on the topic, lack of facilities ability to interpret findings, lack of access to technology, economic concerns, and legal and ethical implications regarding genetic testing.	Providers showed they lack the knowledge required to use pharmacogenomic testing in practice. Barriers to this practice include cost and legal/ethical implications that the testing might bring. Providers expressed the usefulness of pharmacogenomic testing and stated it could result in better patient outcomes. Providers understand the concept and usefulness behind pharmacogenomic testing but stated they lack the knowledge for interpreting results for testing to be useful at this time

Sengore AJ, Champagne BJ, Dosokey E, et al.	This study observed the impact of pharmacogenomics-guided analgesia following major abdominal surgery. 63 patients undergoing colorectal or major ventral hernia repairs were treated with pharmacogenomic guidance and compared to 47 patients with previous operations without pharmacogenomic guidance.	The study observed significant differences in pain scores between the two groups and lower pain scores were recorded in the pharmacogenomic guided group. Postoperative opioid use was evaluated, and researchers found a significantly lower need for narcotics in the pharmacogenomic guided group.	Patients who underwent pharmacogenomic testing required frequent modifications intraoperatively which were not done in the non-pharmacogenomic group. This lead to lower pain scores and narcotic requirements postoperatively.

Conclusions

Pharmacogenomic testing has been shown to be useful in determining what medications are best for each individual patient. The studies above demonstrate a variety of reasons why providers may benefit from preoperative pharmacogenomic studies but the barriers that exist at the current time are devaluing the clinical utility of this tool. A common theme throughout this review is that anesthesia providers simply do not have the knowledge base concerning this technology for it to be a routinely used tool. The studies above show that providers are aware of the benefit this tool can provide but do not have the means to interpret the results to provide better patient outcomes.

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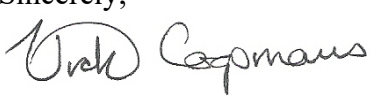
Mentor: David Fort, DNP, CRNA

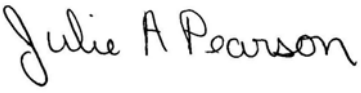
Editorial

We are living in times unlike anything we have experienced before. The degree of disruption is unprecedented, and we have all been affected in a myriad of ways involving health, finances, and education to name a few. The volume of information on COVID-19 is overwhelming, and challenging to absorb as it seems to change on a daily basis. On the front lines, many CRNAs continue to provide care in the stressful healthcare environment, or they are not working due to reduced surgical volume. Most nurse anesthesia students have had their clinical experience suspended. I want to express my solidarity with all practicing and student nurse anesthetists. My heart goes out to everyone facing stress, anxiety, or frustration for any reason due to this pandemic, and I pray for timely resolution.

Sincerely,



Vicki C. Coopmans, PhD, CRNA, CHSE
Editor



Julie A. Pearson, PhD, CRNA
Associate Editor

"***The International Student Journal of Nurse Anesthesia*** **is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.**"

INTERNATIONAL STUDENT JOURNAL OF NURSE ANESTHESIA
GUIDE FOR AUTHORS

MISSION STATEMENT

The International Student Journal of Nurse Anesthesia (ISJNA) is produced exclusively for publishing the work of nurse anesthesia students. It is intended to be basic and introductory in its content. Its goal is to introduce the student to the world of writing for publication; to improve the practice of nurse anesthesia and the safety of the patients entrusted to our care.

ITEM PREPARATION & SUBMISSION

Case reports, research abstracts, evidence-based practice (EBP) analysis reports, evidence-based practice project abstracts, and letters to the editor may be submitted. These items must be authored by a student under the guidance of an anesthesia practitioner mentor (CRNA or physician). Case and EBP analysis reports must be single-authored, while abstracts may have multiple authors. Submissions may list only one mentor. **Mentors should take an active role** in reviewing the item to ensure appropriate content, writing style, and format prior to submission. The mentor must submit the item for the student and serve as the contact person during the review process. Items submitted to this journal should not be under consideration with another journal. Authors and mentors should critically evaluate the topic and quality of the writing – multiple reviews of the item by the mentor, faculty, and peers (fellow graduate students) prior to submission is recommended. If the topic and written presentation are beyond the introductory publication level we strongly suggest that the article be submitted to a more prestigious publication such as the *AANA Journal*.

The journal is committed to publishing the work of nurse anesthesia students. The review process is always initiated with the following rare exceptions. We are conservative in accepting reports where the patient has expired, realizing that you can do everything right and still have a negative outcome. Submissions that report a case demonstrating failure to meet the standard of care (by any practitioner involved in the case) will not be accepted. Unfortunately, while the experiences in these cases can offer valuable insight, these submissions will not be accepted for review due to potential legal risks to the author, journal, and anyone else involved in evaluating the report.

It is the intent of this journal to publish items while the author is still a student. In order to consistently meet this goal, all submissions must be received by the editor at least **3 months prior** (4-6 months recommended) to the author's date of graduation. Manuscripts must be submitted by the mentor of the student author via e-mail to **INTSJNA@aol.com** as an attachment. The subject line of the e-mail should use the following format: ISJNA Submission_submission type_author last name_mentor last name. The item should be saved in the following format – two-three word descriptor of the article_author's last name_school abbreviation_mentor's last name_date (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)

REVIEW PROCESS

Items submitted for publication are initially reviewed by the chief editor. If the chief editor does not acknowledge receipt of the item within two weeks, please inquire to ensure receipt. Upon receipt, the chief editor will review the submission for compliance with the Guide to Authors. If proper format has not been followed, the item will be returned to the mentor for correction. This is very important as all reviewers serve on a volunteer basis. Their time should be spent ensuring appropriate content, not making format corrections. It is the mentor's responsibility to ensure formatting guidelines have been followed prior to submission.

All accepted submissions undergo a formal process of blind review by at least two reviewers. After review, items may be accepted without revision, accepted with revision, or rejected with comments. Once the item has been accepted for review the chief editor will send a blinded copy to an editor, who will then coordinate a blinded review by two reviewers who are not affiliated with the originating program. The editor will return the item to the chief editor, who will return it to the mentor for appropriate action. Every effort is made to complete the process in an efficient, timely matter. Again, the goal is for all articles submitted by students to be published while the author is still a student. If an item is not ready for publication within 6 months after the student author has graduated it will no longer be eligible for publication. Mentors will be listed as contributing editors for the issue in which the item is published.

PHOTOS

Photos of students for the front cover of the Journal are welcome. Please contact the chief editor at intsjna@aol.com to submit photos for consideration. Only digital photos of high quality will be accepted. If the photo is accepted, consent forms must be completed and returned by all identifiable individuals in the photo, and the individual who took the photo.

ACADEMIC INTEGRITY

Issues of academic integrity are the responsibility of the author and mentor. Accurate and appropriate acknowledgement of sources is expected. The two most common breaches of academic integrity that have been identified in submissions to this journal are (AMA 10th ed., p. 158):

1. Direct plagiarism: verbatim lifting of passages without enclosing the borrowed material in quotation marks and crediting the original author.
2. Paraphrase: restating a phrase or passage, providing the same meaning but in a different form without attribution to the original author.

Please note that changing one or two words in a reference source passage (e.g. 'of' for 'in', or 'classified' for 'categorized') and then citing it as a paraphrase or summary is also not appropriate, and still falls within the definition of direct plagiarism. If plagiarism in any form is identified, review of the item will be suspended and it will be returned to the mentor. Repeated instances of plagiarism will result in rejection of the item.

Plagiarism detection software (TurnItIn, PlagScan, SafeAssign, etc . . .) can be used to analyze the document prior to submission to ensure proper citation and referencing, but is not required.

"Plagiarism is the presentation of someone else's ideas, writings, or statements as one's own. Plagiarism is a serious breach of academic integrity, and anyone who is found to have committed plagiarism will be subject to disciplinary action.
Paraphrase is the act of putting someone else's ideas into one's own words. The use of paraphrase can be an acceptable practice under some circumstances if it is used sparingly and if the original text is properly acknowledged. Unacknowledged paraphrase, like plagiarism, is a serious breach of academic integrity. Any improper use of sources may constitute plagiarism. Every quotation from another source, whether written, spoken, or electronic, must be bound by quotation marks and be properly cited. Mere citation alone is not sufficient when a scholar has used another person's words. Similarly, every paraphrase or summary (a more concise restatement of another's ideas) must be properly cited."
https://sites.google.com/a/georgetown.edu/gsas-graduate-bulletin/vi-academic-integrity-policies-procedures

GENERAL GUIDELINES

Items for publication **must adhere to the *American Medical Association Manual of Style*** (AMA 10th ed., the same guide utilized by the *AANA Journal* and such prominent textbooks as *Nurse Anesthesia* by Nagelhout and Plaus). Page numbers are provided for easy reference in the AMA Manual of Style throughout this document. The review process will not be initiated on items submitted with incorrect formatting and will be returned to the mentor for revision. Please note the following:

1. Use complete sentences.
2. Acronyms/Initialisms (p. 379) - spell out with first use, do not capitalize the words from which the acronym/initialism is derived unless it is a proper noun or official name. If you are using the phrase only once, do not list the acronym/initialism at all. Avoid beginning sentences with acronym/initialisms.
3. Abbreviations (p. 441)
4. Use *Index Medicus* journal title abbreviations (p. 472, http://www.ncbi.nlm.nih.gov/nlmcatalog/journals)
5. Always provide units of measure (p. 521 & 795). In most cases The International System of Units (SI) is used. Abbreviations for units of measure do not need to be spelled out with first use. Report height in cm, weight in kg, temperature in ^{o}C, pressure in mm Hg or cm H_2O. Report heart and respiratory rate as X/min (e.g. the patient's heart rate increased to 145/min).
6. In general, first use of pulmonary/respiratory abbreviations should be expanded, with the following exceptions: O_2, CO_2, PCO_2, $PaCO_2$, PO_2, PaO_2, $EtCO_2$, N_2O. Please use SpO_2 for oxygen saturation as measured by pulse oximetry.

7. Use the nonproprietary (generic) name of drugs (p. 568) - avoid proprietary (brand) names. Type generic names in lowercase. When discussing dosages state the name of the drug, *then* the dosage (midazolam 2 mg).
8. Use of descriptive terms for equipment and devices is preferred. If the use of a proprietary name is necessary (for clarity, or if more than one type is being discussed), give the name followed by the manufacturer and location in parenthesis (p. 583, e.g. a GlideScope (Verathon Inc., Bothell, WA) was used) Please note, TM and ® symbols are not used per the AMA manual.
9. Infusion rates and gas flow rates:
    a. Use mcg/kg/min or mg/kg/min for infusion rates. In some cases it may be appropriate to report dose or quantity/hr (i.e. insulin, hyperalimentation). If a mixture of drugs is being infused give the concentration of each drug and report the infusion rate in ml/min.
    b. Report gas flow of O_2, N_2O and Air in L/min (not %) and volatile agents in % as inspired or expired concentration (e.g. General anesthesia was maintained with sevoflurane 3% inspired concentration in a mixture of O_2 1 L/min and air 1 L/min.)
10. Only Microsoft Word file formats will be accepted with the following criteria:
    a. Font - 12 point, Times New Roman
    b. Single-spacing (except where indicated), paragraphs separated with a double space (do not indent)
    c. One-inch margins
    d. End the sentence with the period before placing the superscript number for the reference.
    e. Do not use columns, bolds (except where indicated), or unconventional lettering styles or fonts.
    f. Do not use endnote/footnote formats.
11. Do not use Endnotes or similar referencing software – any embedded formatting must be removed prior to submission.
12. Remove all hyperlinks within the text.
13. Avoid jargon and slang terms. Use professional, scholarly, scientific language.
    a. *'The patient was reversed'* - Did you physically turn the patient around and point him in the opposite direction? "Neuromuscular blockade was antagonized."
    b. *The patient was put on oxygen.* "Oxygen 2 L/min was administered via face mask."
    c. *The patient was intubated and put on a ventilator*. "The trachea was intubated and mechanical ventilation was initiated.
    d. *An IV drip was started.* "An intravenous infusion was initiated."
    e. Avoid the term "MAC" when referring to a sedation technique - the term sedation (light, moderate, heavy, unconscious) may be used. Since all anesthesia administration is monitored, pharmacologic, rather than reimbursement, terminology should be used.
14. Direct quotes are discouraged for reports of this length – please express in your own words.
15. Use the words "anesthesia professionals" or "anesthesia practitioners" when discussing all persons who administer anesthesia (avoid the reimbursement term "anesthesia providers").
16. Do not include ASA Physical Status unless it is germane to the report.
17. Do not use the phrase "ASA standard monitors were applied". Instead, "standard noninvasive monitors" is acceptable – additional monitoring can be detailed as needed.
18. References
    a. The **AMA Manual of Style must be adhered to** for reference formatting.
    b. All sources should be published within the past 8 years. Seminal works essential to the topic being presented will be considered.
    c. Primary sources are preferred.
    d. **A maximum of one textbook (must be most recent edition available) may be used as reference for case report submissions only**.
    e. All items cited must be from peer-reviewed sources – use of sources found on the internet must be carefully considered in this regard. URLs must be current and take the reader directly to the referenced source.

Heading – for all submission types (Case Report, Abstract, EBPA Report) use the following format.

1. **Title** is bolded, centered, 70 characters (including spaces) or less
2. Author name (academic credentials only) and NAP are centered, normal font,.
3. *Graduation date and email address* are centered, italicized, and will be removed prior to publication)
4. **Keywords** is left-justified, bolded – list keywords that can be used to identify the report in an internet search

Title

Author Name
Name of Nurse Anesthesia Program
Anticipated date of graduation
E-mail address

Keywords: keyword one, keyword two, etc . . .

Case Reports - The student author must have had a significant role in the conduct of the case. The total word count should be between 1200 – 1400 words (references not counted). Case reports with greater than 1400 words will be returned to the mentor for revision prior to initiation of the review process. The following template demonstrates the required format for case report submission.

Heading (see above)

A brief introductory paragraph of less than 100 words to focus the reader's attention and interest them to continue reading. This may include historical background, demographics or epidemiology (with appropriate references) of the problem about to be discussed. It is written in the *present tense*. Although it is introductory, the heading word '***Introduction'*** is not used. Be certain to cite references in this section, especially statistics and demographics.
[space]
Case Report (bold, 400-600 words)
[space]
This portion discusses the case performed and is written in the *past tense*. Do not justify actions or behaviors in this section; simply report the events as they unfolded. Present the case in an orderly sequence. Some aspects need considerable elaboration and others only a cursory mention. Under most circumstances if findings/actions are normal or not contributory to the case then they should not be described. Events significant to the focus of the report should be discussed in greater detail. The purpose of the case report is to set the stage (and 'hook' the reader) for the heart of your paper which is the discussion and teaching/learning derived from the case.

- Give dosage and schedule only if that information is pertinent to the consequences of the case.
- **Significant** laboratory values, x-rays or other diagnostic testing pertinent to the case. Give the units of measure after the values (eg. Mmol/L or mg/dL).
- Physical examination/pre-anesthesia evaluation - **significant** findings only.
- Anesthetic management (patient preparation, induction, maintenance, emergence, post-operative recovery).

[space]
Discussion (bold, 600-800 words)
[space]
Describe the ***anesthesia*** implications of the focus of the case report citing current literature. Describe the rationale for your actions and risk/benefits of any options you may have had. This section is not merely a pathophysiology review that can be found in textbooks. *Relate the anesthesia literature with the conduct of your case noting how and why your case was the same or different from what is known in the literature.* Photographs are discouraged unless they are essential to the article. Photos with identifiable persons must have a signed consent by the person photographed forwarded to the editor via first class mail. Diagrams must have permission from original author. This is the most important part of the article. In terms of space and word count this should be longer than the case presentation. End the discussion with a summary lesson you learned from the case, perhaps what you would do differently if you had it to do over again.
[space]
References (bold)
[space]
A minimum of 5 references is recommended, with a maximum of 8 allowed. One textbook may be used as a reference – it must be the most recent edition. All references should be no older than 8 years, except for seminal works essential to the topic. This is also an exercise in searching for and evaluating current literature.
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

EBP Analysis Reports - Evidence-based practice analysis reports are limited to 3000 words. Please do not include an abstract. The report should provide a critical evaluation of a practice pattern in the form of a clinical question about a specific intervention, population, and outcome. The manuscript should:

1. Articulate the practice issue and generate a concise question for evidence-based analysis. A focused foreground question following either the PICO or SPICE format should be used.
2. Describe the methods of inquiry used in compiling the data.
3. Critically analyze the quality of research reviewed and applicability to different practice settings.
4. Draw logical conclusions regarding appropriate translation of research into practice.

The same general format guidelines apply with the exception of the section headings as below. Textbooks and non-peer reviewed internet sources may not be used, and sources of reference should be less than 8 years old unless they are seminal works specifically related to your topic of inquiry. A maximum of 16 references is allowed.

Heading

Introduction (bold)
[space]
Briefly introduce the reader to the practice issue or controversy, describe the scope or significance or problem, and identify the purpose of your analysis. Describe the theoretical, conceptual, or scientific framework that supports your inquiry.
[space]
Methods (bold)
[space]
Include the format used for formulating the specific question you seek to answer, search terms and methods used, and levels of evidence.
[space]
Literature Analysis (bold)
[space]
Analyze and critique the literature relevant to your question, determining scientific credibility and limitations of studies reviewed. Your synthesis table is included in this section. Your review and discussion of the literature should logically lead to support a practice recommendation. Subheadings may be used if desired.
[space]
Conclusions (bold)
[space]
Summarize the salient points that support the practice recommendation and make research-supported recommendations that should improve the practice issue, while also acknowledging any limitations or weaknesses
[space]
References (bold, 16 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Evidence Based Practice Project Abstracts - Evidence-based practice project abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a project proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose (what change is intended) and rationale (why change is needed/evidence to support the change) here.
[space]

Design and Methods (bold)
[space]
Include population, intervention, and measures
[space]
Outcome (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Conclusion (bold)
[space]
Discuss results (implications). Optionally include limitations, suggestions for future projects/research.
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Research Abstracts - Research abstracts are limited to 600 words. References do not impact the word count - a maximum of 5 are allowed. Note that the abstract is different from a research proposal. The following format should be used:

Heading

Introduction (bold)
[space]
A brief introductory paragraph including purpose and hypotheses.
[space]
Methods (bold)
[space]
Include sample and research design
[space]
Results (bold)
[space]
Present results from statistical analysis – do not justify or discuss here.
[space]
Discussion (bold)
[space]
Discuss results (implications, limitations, suggestions for future research)
[space]
References (bold, 5 maximum)
[space]
Mentor: (bold, followed by mentor name and credentials in normal text)
E-mail address: (normal text, will be removed prior to publication)

Letters to the Editor - Students may write letters to the editor topics of interest to other students. Topics may include comments on previously published articles in this journal. Personally offensive, degrading or insulting letters will not be accepted. Suggested alternative approaches to anesthesia management and constructive criticisms are welcome.
The length of the letters should not exceed 100 words and must identify the student author and anesthesia program.

AMA MANUAL OF STYLE

The following is brief introduction to the *AMA Manual of Style* reference format along with some links to basic, helpful guides on the internet. The website for the text is http://www.amamanualofstyle.com/oso/public/index.html. It is likely your institution's library has a copy on reserve. Some helpful websites are listed below:
https://guides.nyu.edu/amastyle
https://owl.english.purdue.edu/owl/resource/1017/01/

Journal names should be in *italics* and abbreviated according to the listing in the PubMed Journals Database. The first URL below provides a tutorial on looking up correct abbreviations for journal titles; the second is a link to the PubMed where you can perform a search.
http://www.nlm.nih.gov/bsd/viewlet/search/journal/journal.html
http://www.ncbi.nlm.nih.gov/pubmed

The International Student Journal of Nurse Anesthesia (ISJNA) is not listed in the PubMed Database. For the purpose of citing the ISJNA *in this Journal* use "**Int Student J Nurse Anesth**" as the abbreviation.

Journals - A comma is placed after the first initials until the last author, which has a period. If there are six or less authors **cite all six**. If there are more than six authors **cite only the first three** followed by "et al." Only the first word of the title of the article is capitalized. The first letters of the major words of the journal title are capitalized. There is no space between the year, volume number, issue number, and page numbers. If there is no volume or issue number, use the month. If there is an issue number but no volume number use only the issue number (in parentheses). Page numbers are inclusive - **do not omit digits** (note - some online journals do not use page numbers). Some journals may be available both as hard copies and online. When referencing a journal that has been accessed online, the DOI (digital object identifier) or PMID (PubMed identification number) should be included (see example below).

Journal, 6 or fewer authors:
Han B, Liu Y, Zhang X, Wang J. Three-dimensional printing as an aid to airway evaluation after tracheotomy in a patient with laryngeal carcinoma. BMC Anesthesiol. 2016;16(6). doi:10.1186/s12871-015-0170-1.

Journal, more than 6 authors:
Chen C, Nguyen MD, Bar-Meir E, et al. Effects of vasopressor administration on the outcomes of microsurgical breast reconstruction. *Ann Plast Surg*. 2010;65(1):28-31. PMID: 20548236.

Elayi CS, Biasse L, Bai R, et al. Administration of isoproterenol and adenosine to guide supplemental ablation after pulmonary vein antrum isolation. *J Cardiovasc Electrophysiol*. 2013;24(11):1199-1206. doi: 10.1111/jce.12252.

Electronic references - Only established, peer-reviewed sources may be referenced. Please do not reference brochures, fact sheets, or informational websites where a peer-review process cannot be confirmed. The URL must be functional and take the reader directly to the source of the information cited. The accessed date may be the only date available.

Author (or if no author, the name of the organization responsible for the site). Title. *Name of Website*. Year;vol(issue no.):inclusive pages. URL. Published [date]. Updated [date]. Accessed [date].

Examples:
Kamangar N, McDonnell MS. Pulmonary embolism. *eMedicine*. http://www.emedicine.com/med/topic1958.htm. Updated August 25, 2009. Accessed September 9, 2009

Howlader N, Noone AM, Krapcho M, Garshell J, Miller D, et al. SEER Cancer statistics review, 1975-2012. National Cancer Institute. http://seer.cancer.gov/csr/1975_2012/. Published April 2015. Updated November 18, 2015. Accessed February 29, 2016.

Textbooks - There are two types of books – 1) those that are fully authored by one or more individuals, and 2) those that are edited by one or more individuals, with chapters authored by different individuals. Edited textbooks give

primary credit to the chapter authors, who are listed first, and the inclusive page numbers of the entire chapter are provided at the end. Textbooks that are authored do not have different chapter authors and the chapter titles are not listed, but the inclusive page numbers where the information was found are provided, unless the entire book is cited.

Authored text:
Shubert D, Leyba J, Niemann S. *Chemistry and Physics for Nurse Anesthesia*. 3rd ed. New York, NY: Springer; 2017:405-430.

Chapter from an edited text:
Pellegrini JE. Regional anesthesia. In Nagelhout JJ, Elisha S, eds. *Nurse Anesthesia*. 6th ed. St. Louis:Elsevier; 2017:1015-1041.

SUBMISSION CHECK LIST

___ **Adheres to AMA Manual of Style and all other format instructions**
___ Total word count not exceeded (1400 for case report, 600 for abstracts, 3000 for EBPA report)
___ The item is one continuous Word document without artificially created page breaks
___ All matters that are not common knowledge to the author are referenced appropriately
___ Generic names for drugs and products are used throughout and spelled correctly in lower-case
___ Units are designated for all dosages, physical findings, and laboratory results
___ Endnotes, footnotes not used
___ Jargon/slang is absent

Heading
___ Concise title less than 70 characters long
___ Author name, credentials, nurse anesthesia program, graduation date and email are included
___ Three to five **Keywords** are provided

Case Report
___ Introduction is less than 100 words.
___ Case Report section states only those facts vital to the account (no opinions or rationale)
___ Case report section is 400-600 words and not longer than the discussion
___ Discussion section is 600-800 words
___ Discussion of the case management is based on a review of current literature
___ Discussion concludes with lessons learned and how the case might be better managed in the future

Abstracts
___ The 600 word count maximum is not exceeded
___ Appropriate format used depending on type of abstract (research vs. EBP project)

EBPA Report
___ The 3000 word count maximum is not exceeded
___ A critical evaluation of a practice pattern in the form of a precise clinical question about a specific intervention, population, and outcome is presented
___ A focused foreground question following either the PICO or SPICE format is used
___ Includes Introduction, Methodology, Literature Analysis (with synthesis table), and Conclusion sections

References
___ Adheres to AMA Style format
___ Reference numbers are sequenced beginning with 1 and superscripted
___ References are from anesthesia and other current (within past 8 years) primary source literature
___ Journal titles are abbreviated as they appear in the PubMed Journals Database
___ Number of references adheres to specific item guidelines (1 textbook allowed for case reports only)
___ Internet sources are currently accessible, reputable, and peer reviewed

Transmission
___ The article is sent as a attachment to **INTSJNA@AOL.COM**
___ The file name is correctly formatted (e.g. PedsPain_Smyth_GU_Pearson_5.19.09)
___ Item is submitted by the mentor
___ Subject heading format - ISJNA Submission_submission type_author last name_mentor last name