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The ever-expanding breadth of medical expertise

Every once in a while it is worth taking a step back to acknowledge how vast the practice of medicine has become. I find myself reflecting on the career of Sir William Osler, sometimes referred to as the "Father of Modern Medicine", and the transformation that has taken place in the medical field since he earned that esteemed title. When Osler graduated medicine in 1872, post-graduate training was informal rather than a requirement to practice. The very notion of medical specialization was still developing in Osler's day. Indeed, in his farewell address to Canadian and American medical students, Osler bemoans the lost "polymathic student", the classic Renaissance man, whose area of study comprised not just the whole of medicine, but the whole of human knowledge.

Fast forward to today, where post-graduate training is not only required, but can be done in any of a dozen specialties and takes up to six years to complete. Subspecialisation through fellowships is now commonplace. As an old saying goes, physicians are now learning more and more about less and less until eventually, they will know everything about nothing.

Yet, this ever-increasing specialisation of knowledge demonstrates how far medical knowledge has advanced from Osler's time. Just as the prototypical Renaissance man became a thing of the past when the world grew too wide for one person to be an expert in all things, so too has medicine grown beyond the limit for any one physician to master all its elements. While a single physician may no longer be a comprehensive source of medical information, physicians and other clinicians working together now have the combined expertise to far surpass what was possible when Osler wrote of the demise of the "polymathic student".

This year's Summer Supplement showcases both the diversity and depth of knowledge which underlies the modern Canadian health care system. Drawing from multiple specialties in medicine, from otolaryngology to oncology, anesthesia to obstetrics, the cases in this issue highlight both how far medical knowledge has come – and how far it still has to go.

— Craig Olmstead
Junior Associate Editor

Oronasal Fistula with Palatal Involvement Secondary to Cocaine Use

Ayesha Raza (Meds 2015)
Faculty Reviewer: Dr Ali Hilal, MBChB, FRCSC, AFSA, FABMS, FRCSI (Department of Otolaryngology)

BACKGROUND

Cocaine abuse is associated with serious systemic complications, affecting the cardiovascular and nervous systems by activating the sympathetic pathway.[1] Vasoconstriction is a well-known local sympathetic effect of cocaine that may lead to ischemia, inflammation, ulceration and ultimately necrosis of the nasopharyngeal structures implicated by snorting cocaine.[2] Nasal septal perforation is present in 4.8% of abusers, making it the most common complication of cocaine abuse.[3] Chronic cocaine use may contribute to larger lesions and further destruction of the surrounding osteocartilaginous structures. Specifically, the destruction of nasal and midfacial bones and soft tissues can lead to a syndrome called cocaine-induced midline destructive lesion (CIMDL). The diagnosis of CIMDL requires at least 2 of the following clinical or radiologic findings: nasal septal perforation, lateral nasal wall destruction, and hard palate involvement.[4] We describe a patient with a history of cocaine abuse and signs of CIMDL, with midfacial destruction of the nasal septum and hard palate creating an oronasal fistula. To the best of our knowledge, we believe this is the first case of CIMDL with oronasal fistula reported in Canada.

CASE PRESENTATION

A 60-year-old male was referred to us with a 1-month history of a "hole" in his hard palate that caused fluids to leak out of his nose when drinking. Prior to presentation, he had been using denture adhesive gel as a temporizing measure for the palatal defect. He also disclosed that he had a nasal deformity, which previous physicians had attributed to his long history of cocaine snorting. The oral and nasal lesions were not associated with bleeding, pain, B-symptoms, nasal obstruction or recurrent sinusitis. His past medical history was notable for asthma and hypertension. His social history revealed a lifetime of tobacco use and many years of chronic cocaine use.

On physical examination, the patient looked well and was in no acute distress. The nasal vestibules were intact bilaterally with no erosion of the columellar skin. Anterior rhinoscopy revealed complete loss of the cartilaginous nasal septum, without saddle nose deformity. Flexible nasopharyngoscopy showed extensive mucosal crusting, severe atrophy of the turbinates bilaterally but no polyps or suspicious growths. Examination of the oral cavity showed normal mucosa and dentition overall, with a single 1 cm perforation at the midline of the hard palate, communicating with the floor of the nasal cavity. The hard palate was quite soft in the area surrounding the perforation, suggesting bone loss of 2–3 cm in circumference. The soft palate was intact and the posterior pharynx was normal, with no ulcerating or fungating growths. The remainder of the head and neck examination was unremarkable.

Computed tomography (CT) scan showed extensive bony erosion of the nasal septum and hard palate, with mucosal thickening of the maxillary and ethmoid sinuses. There was no evidence of orbital or intracranial extension of this disease. Nasopharyngeal swab was positive for *Staphylococcus aureus*. Biopsy of the nasal cavity mucosa showed non-specific granulation tissue with no evidence of malignancy or infection.

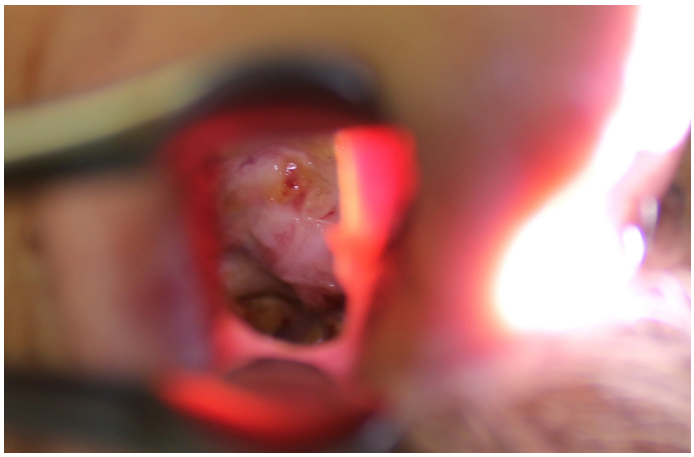

Figure 1. Anterior rhinoscopy showing a complete destruction of nasal septum and exposing the pale mucosa of the contralateral nasal cavity.

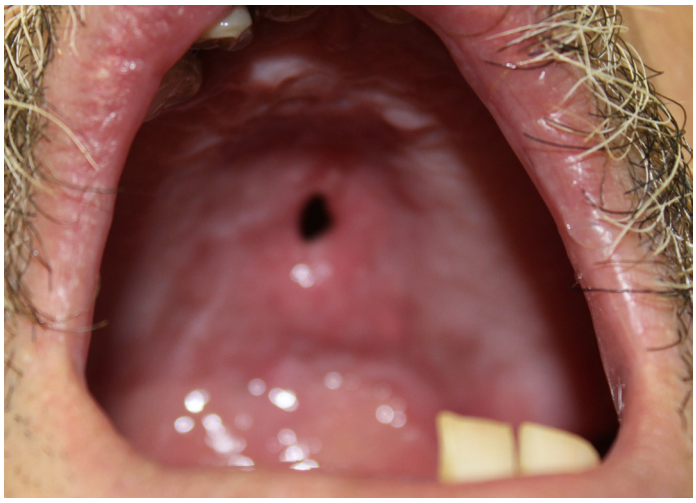

Figure 2. Photograph of hard palate with a midline oronasal fistula.

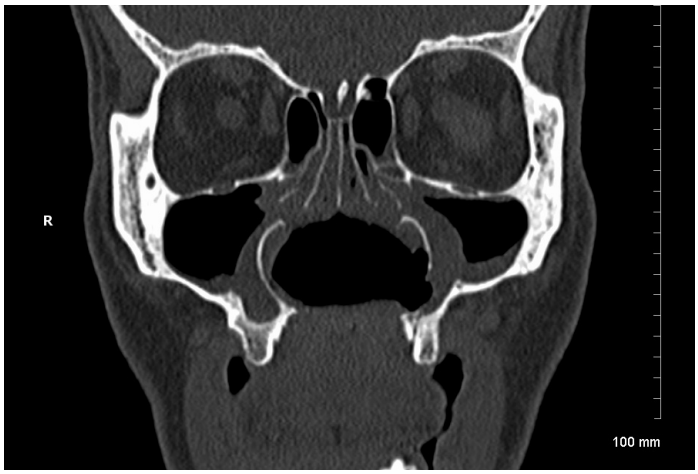

Figure 3. Coronal CT image of the nose and paranasal sinuses showing the complete loss of the nasal septum and turbinates.

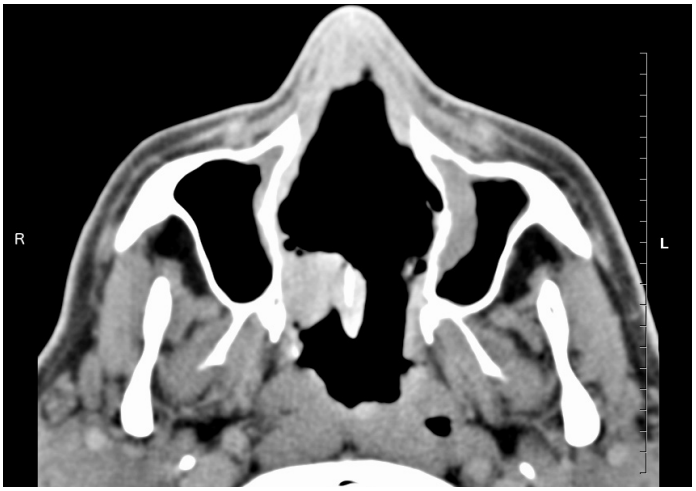

Figure 4. Axial CT image of the nasal cavity showing the soft tissue and bony loss of the nasal walls and septum.

We concluded that the patient's severe atrophic rhinitis and ischemic erosion of the nasal floor were due to years of intranasal cocaine use. Though the patient expressed interest in surgical closure of the perforation, the likelihood of a successful procedure is greatly reduced with a background of mucosal ischemia and continued local injury due to cocaine use. We discussed this with the patient and urged him to discontinue his drug use. He agreed to look into local addiction treatment programs with the help of his family physician. In the interim, we have advised frequent nasal saline rinses and referred him to an oral maxillofacial surgeon for fitting of a prosthetic obturator as a conservative measure for his oronasal communication. We felt that an obturator would stop his nasal regurgitation.

DISCUSSION

Though cocaine abuse has declined over the last decade, it remains among the top five most used substances among Canadian adults.[5] Well-known systemic effects of cocaine use include tachycardia, hyperthermia and mydriasis.[6] Snorting cocaine also causes local anesthesia and vasoconstriction, which predispose to trauma and mucoperichondrial ischemia of the nasopharyngeal system. Coupled with adulterant-induced mucosal irritation, these effects lead to erosion and destruction of the nasal septum.[7] Septal perforations are the best-known external manifestations of cocaine abuse and are generally well tolerated, thus delaying abusers' medical presentation. However, continued local ischemic necrosis causes progression of nasal manifestations to severe midline destruction involving the palate, causing an oronasal fistula in a condition called CIMDL. The most commonly reported symptoms in patients with CIMDL are rhinolalia and the passage of liquids and food through the nose when swallowing—both of which finally prompt patients to seek medical attention. Anosmia, pain, headache and halitosis with cacosmia are also frequent presenting complaints.[8]

The treatment of CIMDL is challenging due to patients' non-compliance and hesitancy to admit to cocaine use. The first line of management is to exclude other potential causes for midline nasal destruction, with CT, lesion biopsy, blood tests and toxicology screen. When considering restorative therapy, the importance of abstaining from drug use must be stressed to patients, as the midline destruction will progress until they have completely stopped using. Management during rehabilitation is therefore conservative, including analgesia, antibiotic therapy, surgical debridement of necrotic tissue and saline lavage to prevent further destruction.[9] The next treatment option is a prosthetic obturator, an oral device fitted to the patient's palate that artificially closes the oronasal fistula for temporary relief of symptoms. These can be adjusted to fit progressively widening fistulas, but their stability depends on the patient's dentition and palatal morphology. Nevertheless, some patients find adequate relief with an obturator and do not seek further therapy. Surgical reconstruction via local flap procedures is the ideal treatment and has been successful with non-cocaine related oronasal fistulas. However, the unpredictable vascularity of ischemic local tissues in CIMDL leads to a high risk of surgical failure and flaps may only be suited to small fistulas. To optimize outcomes, some authors have suggested that surgical reconstruction only be attempted after complete drug rehabilitation for at least one year—therefore, treatment must include psychiatric assistance.[10]

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Oral Squamous Cell Carcinoma in the Young Patient

Priya Sivarajah (Meds 2016)
Faculty Reviewer: Dr Kevin Fung, MD, FRCSC (Department of Otolaryngology)

INTRODUCTION

Oral cancer is primarily a disease seen in elderly men (50–80 years of age) with a decades-long history of smoking, and consumption of over 20 cigarettes and 100 g of alcohol a day.[1] Its presentation in younger patients (<35 years) is fortunately rare, with an incidence between 0.4% and 5.5%.[1] However, when cases do present, they are often misdiagnosed, leading to inappropriate delays in treatment, and worsening prognoses.[2] This bears significant implications for young patients, with the potential for long-term functionally debilitating sequelae. With this in mind, we report a case of oral tongue squamous cell cancer (OTSCC) in a young Caucasian female, with a focus on her diagnoses and subsequent post-surgical quality of life.

CASE REPORT

A 25-year old Caucasian female presented to a dentistry clinic, complaining of a left-sided lesion on her tongue of 13 months duration, which had markedly increased in size within the past month. She reported an impact on her speech and eating patterns, and expressed worry about a malignant etiology. The patient had initially believed the lesion to be a chancre sore, but decided to seek help as the lesion had continued to grow.

The patient had an intermittent smoking history of an unspecified duration, during which she smoked up to 5–10 cigarettes daily. She also had a five-year history of drinking, a period through which she consumed 8 drinks per day, for four days a week. She denied experiencing night sweats or fevers, although she reported losing weight from a markedly reduced food intake. Her paternal grandfather had died from metastatic esophageal cancer, and her maternal grandfather was diagnosed with colon cancer.

The dental practitioner reassured her (allaying her fears of cancer), filed down her teeth, and scheduled a follow-up appointment in 2 weeks' time, during which her presentation changed significantly. She started experiencing constant headaches, and the lesion grew purulent, significantly affecting her ability to eat. Finding that conservative management with salt and water, as well as peroxide was futile, she visited her university walk-in clinic to receive antibiotics, noting once again that the physician failed to discuss the possibility of a malignant etiology. Re-visiting her dental practitioner at her follow-up appointment, the rapid change in presentation quickly prompted a referral to a dental specialist at an academic care centre. Although the appointment wasn't for another four months, the exponential growth of the tumor led the patient to successfully reschedule the appointment to a closer date, within two months' time. A physical examination prompted the specialist to quickly refer the patient to an otolaryngologist, who saw her within the week.

The surgeon prescribed her Tylenol-Codeine #3, which she took with Ibuprofen and Acetaminophen for pain management. Her diet was reduced to soft meals, consisting of pudding, apple sauce and yogurt. Three weeks later, after confirmation through biopsy of a T2N0 squamous cell carcinoma of the oral tongue, the patient underwent a selective neck dissection. A left-sided hemiglossectomy removed the carcinoma, and a fasciocutaneous radial forearm free flap from the right arm replaced the defect. The skin defect was replaced using a graft from the right hip. A tracheostomy was performed, and a feeding tube was inserted. The absence of metastasis precluded the need for chemotherapy or radiation treatments. The patient remained hospitalized for an additional two weeks.

Post-surgical QoL issues circled around dysfunction of the left shoulder muscles, sensitivity of the left sided face and right arm, and with problems inherent in having a tracheostomy and feeding tube. The patient's face was numb for an unspecified duration post-surgery, and her tongue was significantly swollen for a week. In addition to the cast on her right arm, and IV's in her left, communication (both verbal and written) during that week was significantly impaired.

The patient received integrated care from a team of inter-professional allied healthcare workers. A respiratory therapist aided her in coughing exercises to prevent aspiration pneumonia from her tracheostomy; a physiotherapist aided her transition off the tracheostomy, familiarizing her with breathing exercises, and encouraging the change from bed-rest to ambulation; a speech language pathologist performed pre- and post-op assessments to evaluate standard articulation and swallowing; a nutritionist made recommendations post-surgery to assist the patient's food intake; and a home care nurse assisted the patient in showering and re-dressing the cast on her right arm. The patient had pureed meals for 2 months, and continued her pain medications for one month post-surgery. With the assistance of her parents and her healthcare team, the patient was able to recover and return to school for the next academic year. Following surgery, the patient has had annual follow-up appointments with no evidence of recurrent disease to date. She subsequently quit smoking, and has since given birth to her first child.

The patient still suffers from ongoing QoL issues. The patient has a persistent dry mouth, and has reduced sensation of her left-sided tongue, occasionally not noticing when she bites on it. She is unable to differentiate between soft and hard bites, and has changed her eating patterns, shunting food from the right side of her mouth to make use of her remaining taste buds. This has spawned dentition problems, including right-sided gum loss and left-sided gum growth. She has recurrent neck and jaw pain, which she treats with Tylenol. She has also noticed changes in her facial features, with her left-sided face appearing 'droopier'.

DISCUSSION

EPIDEMIOLOGY

Oral tongue squamous cell carcinoma (OTSCC) is the most common tumor in the oral cavity.[2] Although the overall incidence of OTSCC has remained stable from 1975 to 2007, its incidence has been increasing in women, specifically in young, Caucasian females aged 18–44 years.[1] Unfortunately, as is evident in the case presentation, there is a stigmatized approach to younger female patients with suspicious oral lesions. Raising awareness on these rising incidence levels will alert healthcare practitioners to atypical presentations, and reduce any delays these patients may experience in receiving appropriate care.

RISK FACTORS FOR YOUNGER PATIENTS

Although the literature is uncertain and the topic still highly debated, it has been claimed that younger patients have more aggressive disease, with a higher incidence of local recurrence or regional lymph node involvement after treatment, and higher mortality rates.[1] Young patients often present with little-to-no risk factors (alcohol, tobacco and betel nut exposure), or with a history of tobacco and alcohol intake significantly shorter in comparison to their older counterparts. Given this disparity, researchers have proposed other risk factors to account for the etiology of these oral cancers, such as immune deficiency states, genetic factors, dietary factors, and viral infections (such as herpes simplex virus and human papilloma virus).[1] The association between oropharyngeal cancer and HPV infection is controversial at best, and significant research has been focused on screening for specific virally-associated mutations of known tumor suppressors, oncogenes, and replication pathways.[2] Unfortunately, no clear inciting pathway has been identified to this date.

QUALITY OF LIFE

Research into QoL issues for young oral cancer patients is a rather new and emerging concept. Thomas et al. performed a retrospective case series and questionnaire survey for patients treated for oral cancers during a 25-year period, when they were 40 years of age or less. They found that age at diagnosis and duration of follow-up did not correlate with overall QoL or health-related QoL. Approximately 77% of patients rated their overall QoL as outstanding, very good or good, with the key problematic domains lying within appearance, mood, salivation and shoulder function. Only radiotherapy adversely affected the overall QoL.[3] In addition, a systematic review by Carranza et al., found that younger oral cancer patients show more emotional and role dysfunctions than older patients, and are at higher risk for psychological stress and an increase in symptoms–such as dry mouth–in the first post-treatment year. Marital status seems to be a protective prognostic factor for survival and recurrence in comparison to those who live alone, possibly due to better hygienic habits, and fewer delays in diagnosis and treatment.[4]

CONCLUSIONS

Although there is adequate research into QoL for oral cancer patients, most of the current literature solely investigates elderly patients with significant histories for known risk factors. There is a noticeable lack of research into QoL specific to younger oropharyngeal cancer survivors, namely OTSCC survivors. The rising incidence of these cancers in the younger population raises the need for further studies to be done, in order to ensure that younger patients return to a fruitful life with limited functional disabilities.

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Anesthetic Management of Septic Shock

Konstantinos Alexopoulos (Meds 2016)
Faculty Reviewer: Dr Ramiro Arellano, MD, FRCPC (Department of Anesthesiology)

BACKGROUND

Severe sepsis, a systemic inflammatory response characterized by acute organ dysfunction in the face of an infectious process, is a major healthcare issue worldwide. Sepsis presents a unique challenge in the management of patients who require anesthesia and definitive surgical treatment. Anesthesiologists are involved in the multidisciplinary management of patients with severe sepsis throughout the patient's clinical course.[1] The mainstays of management include prompt administration of IV antibiotics in conjunction with preoperative fluid and pharmacological resuscitation. The goal is to optimize end organ perfusion through the judicious use of pressors, inotropes and IV fluids.[2] An example might include a patient with an acute small bowel obstruction complicated by sepsis. The patient would be appropriately resuscitated using a multi-modal approach as outlined above, while definitive management would include surgical decompression of the obstruction. Intra-operatively, these patients require conscientious induction of anesthesia, ample fluid resuscitation coupled with invasive hemodynamic/biochemical monitoring.[1] Efforts to promote a favourable surgical outcome of a septic patient begins before the surgeon picks up the scalpel.

CASE PRESENTATION

Patient SM is a 67-year-old retired female who presented to University Hospital with nausea, vomiting, constipation and acute kidney injury. SM's creatinine trended upwards for several days from 341 to 372 then 401. Kidney ultrasound was performed which revealed severe bilateral hydronephrosis. Subsequently, SM had bilateral ureteric stents to relieve the outlet obstruction. Despite the ureteric stents, SM's urine output remained poor and an IV fluid bolus was administered. Within 24 hours, SM developed increasing SOB and O_2 requirement, as well as a cough with fever. SM was started on IV Ceftriaxone and Piperacillin-Tazobactam to cover gram negative organisms that colonize the urogenital tract including *E. coli*, Proteus and Klebsiella.[3] Interventional radiology was consulted for the placement of bilateral nephrostomy tubes to relieve the bilateral ureteric outlet obstructions.

SM was evaluated by the consultant anesthesiologist, resident physician and author KA. SM's past medical history included GERD, chronic fatty liver, hypothyroidism, irritable bowel syndrome, dyslipidemia, psoriasis, migraines and anxiety. SM appeared much older than her stated age with central morbid obesity and a BMI of 48 kg/m^2. With this information SM was assigned the American Society of Anesthesiologists number 5 physical status classification. Class 5 patients are not expected to survive 24 hours without surgical intervention.4 SM's vitals at this time were: NIBP 95/42 mmHg, MAP calculated at 60 mmHg, HR of 130 bpm, SpO_2 of 88% on 10 L nasal cannula, febrile with a temperature of 39.2 °C. Neurologically the patient was intact, alert and oriented to person, place and time. However, as the exam continued SM became increasingly obtunded, showing signs of laboured breathing. Her airway exam revealed a Mallampati Class IV with decreased thyromental distance. Intubation was predicted to be extremely difficult. SM's most recent blood work and chemistries showed a leukocytosis with a developing coagulopathy due to urosepsis. As the patient was a Jehovah's Witness, all blood products were refused. The patient had a 20 Ga IV in situ. Plan for induction of anesthesia included an awake arterial line for accurate hemodynamic monitoring, and an additional large bore IV (> 18 Ga), modified rapid sequence induction and oral endotracheal intubation using the Glidescope and transfer to the Medical Surgical Intensive Care Unit (MSICU) for postoperative care.

An arterial line was established in the right radial artery using sterile technique. A good waveform was observed. The dicrotic notch indicates closure of the aortic valve and the wash back of blood against the aortic valve. The waveform showed a lower than normal dicrotic notch indicating that the patient was severely hypovolemic.[5] SM was having difficulty cooperating with our instructions as she was extremely somnolent. Tracheal topicalization was achieved with 2% lidocaine spray. BP had dropped to 85/40 mmHg. A 1 litre fluid bolus of 0.9% NaCl was administered prior to intubation. Hypnotic agents like etomidate were discussed for the maintenance of what little hemodynamic stability SM had, but ultimately was not chosen. Evidence in the literature indicated that etomidate in sepsis can precipitate marked adrenal insufficiency and increase rates of mortality.[6] The patient's potassium was 3.8 mmol/L which made succinylcholine our choice for neuromuscular blockade. Remifentanil, a potent ultra-short-acting opioid was chosen to assist visualization of the glottis during intubation. Remifentanil in rat models has demonstrated a protective effect against sepsis. The proposed mechanism is a decreased release of inflammatory mediators IL-1, IL-6, TNF-alpha, while also decreased the inducible nitric oxide synthase which is a potent endogenous vasodilator.[7]

A defasciculating dose of Rocuronium 3 mg IV was administered to prevent fasciculations and myalgias from succinylcholine. Midazolam 1 mg was administered for anxiolysis. Cricoid pressure was applied to the larynx. IV Lidocaine was not administered since it was deemed that the pharyngeal response of increased BP to laryngoscopy would be favourable in this patient. Remifentanil 30 mcg was administered, followed by Propofol 30 mg and Succinylcholine 100 mg. At this point, SM's SpO_2 was 85% despite administration of 100% O_2. The Glidescope was used and a McCormack-Lehane Grade II view was obtained. Intubation was successful and confirmed with end tidal CO_2 ($ETCO_2$) return and equal bilateral

air entry. Patients with sepsis have a decreased anesthetic requirement, thus, maintenance of anesthesia was achieved using Sevoflurane at a concentration of 1% end-tidal Sevoflurane, which is one half its minimal alveolar concentration.[2] SM's $ETCO_2$ was 21 mmHg, indicating poor perfusion likely due to decreased cardiac output and developing pulmonary edema secondary to acute respiratory distress syndrome (ARDS). Phenylephrine 200 mcg, a pure alpha-1 agonist was administered, with little to no effect. Another 300 mcg was given with no observable clinical effect.

SM was now markedly hypotensive at 70/35 mmHg as per the arterial line. Treatment for pulmonary edema caused by ARDS is positive end expiratory pressure (PEEP), but PEEP decreases venous return and results in hypotension. At this point, the need for adequate ventilation did not outweigh the need for hemodynamic stability. Vasopressin was considered but ultimately was not administered in favour of more powerful direct acting inotropes. 10 mcg of IV epinephrine was administered in conjunction with a fluid bolus. The radiologist began to scrub for the procedure. SM developed tachycardia at 145 bpm, with a BP 55/30 mmHg. SpO_2 was 78%. Sevoflurane was switched off; oxygen at 100%, flows at 15 L/min. IV epinephrine was increasingly administered in 10 mcg boluses escalating to 30 mcg boluses. BP improved slightly to 70/40 mmHg. An infusion of epinephrine/norepinephrine was established.

The anesthesiologist advised the radiologist that the patient was too unstable for the procedure. SM's coagulopathy and sepsis were far too severe, and in the event of blood loss the risk for mortality was greatly increased.[2] SM was transferred to the MSICU on the infusion of epinephrine/norepinephrine and cardiac monitors. SM was started on continuous renal replacement therapy and maintained on an epinephrine/norepinephrine infusion. 24 hours later, the ureteric stents were changed at the bedside in the MSICU. Prior to the procedure, SM received DDAVP (Vasopressin) 20 mcg IV and Vitamin K 10 mg IV for her coagulopathy. The procedure was a success and the patient's coagulopathy started to resolve. SM remained in the MSICU for 4 days requiring decreasing doses of pressors. After a 24 day stay, she improved markedly and was discharged home in stable condition.

DISCUSSION

This case presented some difficult decisions from a management perspective. As always it is important to remember that physicians inadvertently can cause harm to their patient(s). From iatrogenic infections, to ill-informed decision making, in some cases, the gold standard treatment for the patient may result in deleterious consequences. In SM's case, the decision to forego the procedure in the face of marked hypoxemia and hypotension was the most appropriate management. In the event that plan A begins to fail, plan B must already be formulated and ready to implement, while remembering: First, do no harm.

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Who is to Blame?

A Case of Medical Negligence in Obstetrical Care

Erica Hoe (Meds 2015)
Faculty Reviewer: Dr Ellen Tsai, MD, MHSc (Bioethics), FRCPC (Canadian Medical Protective Association)

CASE SUMMARY

A 30-year-old nulliparous pregnant female presents to a large urban teaching hospital at 35 weeks gestation in active labour following spontaneous membrane rupture. She is initially assessed by her obstetrician, who after assessing the fetal heart rate to be normal and reactive, prescribes oxytocin by infusion to augment her contractions. The patient is then followed by both the obstetrician and a junior obstetrics resident.

The obstetrician reassesses the patient several hours later and orders the oxytocin infusion to be increased because the contractions are only moderately strong. Epidural anesthesia is initiated by an anesthesiology resident prior to increasing the oxytocin. The nurse then calls the obstetrics resident to reassess the patient, following an episode of fetal bradycardia lasting several minutes—a possible indicator of impaired fetal oxygenation—subsequent to the epidural insertion. The resident places a fetal scalp electrode to monitor the heart rate more accurately, which demonstrates decreased heart rate variability and variable decelerations. The nurse expresses concern to the resident when the variable decelerations persist. The resident then calls the obstetrician to recommend monitoring the patient a little longer because the cervix is already 8-9 cm dilated, rather than proceeding directly to a Cesarean section. The obstetrician accepts the resident's plan without reviewing the fetal heart rate tracing.

Within the hour, the patient's cervix is fully dilated and the resident instructs the patient to begin pushing. The obstetrician arrives shortly thereafter. Upon delivery of the infant's head, the umbilical cord is found to be wrapped tightly around the neck and thus is cross-clamped and cut. The baby boy is then delivered easily and spontaneously, but is pale and flaccid with gasping respirations immediately after birth.

The obstetrics resident and nurses begin resuscitation of the infant. The infant's colour becomes pink within one or two minutes, but his tone remains flaccid. The anesthesiology resident is paged, intubates the infant at 10 minutes of life, and manages the resuscitation until the arrival of the on-call pediatrician at 45 minutes of life. The infant is subsequently transferred to a tertiary care neonatal unit, where he is diagnosed with hypoxic brain injury, eventually resulting in severe quadriplegic cerebral palsy, cortical blindness and recurrent seizures.

A legal action follows some years later.

NEGLIGENCE IN MEDICAL PRACTICE

This real-life case illustrates the complexity of medico-legal actions, where there are often several physicians—and other health professionals—involved in the patient's care. The court found the care of both the attending obstetrician and the obstetrics resident in this case to be negligent. Negligence is determined when the patient-plaintiff can prove on the balance of probabilities that the physician had a duty of care, and that the harm experienced was as a result of a breach of the standard of care.[1] The standard of care is based on what a normal, prudent physician of similar training and experience would do in similar circumstances.[2] Courts will judge whether the standard of care was breached after considering the testimony of physician peers, otherwise referred to as medical experts.[3] This case illustrates how the standard of care is determined differently for each physician, based on their level of training and the particular clinical circumstances.

The attending obstetrician breached the standard of care on several counts. The primary criticism was that he did not call the pediatrician earlier. As such, the obstetrician failed to display situational awareness. Although physicians are not expected to accurately predict outcomes, they have the responsibility to anticipate critical situations that may arise and act according to what a reasonably prudent physician would be able to foresee. There were several risk factors present—including prematurity and abnormal fetal heart rate tracings—that should have mandated a request for pediatric attendance at delivery based on the national practice standards in place at that time.[4]

The obstetrics resident's actions also fell below the standard of care in this case. The court determined that a reasonably prudent resident at the same level of training should have been able to recognize the warning signs of fetal distress and alert the attending obstetrician sooner. Medical trainees, including medical students, are responsible for knowing the limits of their own knowledge and experience. Similar to the staff obstetrician, the resident failed to recognize the significance of the abnormal fetal heart rate tracing and subsequently the gravity of the infant's condition after birth.

Another criticism of the obstetrician was in regard to supervision of the resident. Supervising physicians can reasonably rely upon residents to carry out the duties properly delegated to them; however, they must be aware of circumstances when more active supervision is required. This will depend on such factors as the patient's condition, the complexity of the procedure, and the level of experience and skill of the resident. Failure to adequately supervise a trainee can result in liability for the supervising physician, even if the harm was caused by the trainee's actions.

The resident did not alert the attending obstetrician to persistent fetal heart rate abnormalities in a timely manner. Nevertheless the obstetrician was still responsible for overseeing the overall

care of the patient, including the resident's work. In this case, the obstetrician overly relied on the skills and knowledge of this junior resident, and did not personally review the abnormal fetal heart rate tracing. The principle of shared responsibility is reflected in the court's apportionment of liability, which in the end was 75% for the attending obstetrician and 25% for the obstetrics resident.

Interestingly, the anesthesiology resident was not found to be negligent and was dismissed from the legal action. As a first-year resident who was in the third month of a pediatric anesthesia block, this resident would have had limited experience in the care of premature neonates at the time of the events. Any contribution to the infant's outcome was considered to be negligible; in fact, it was the anesthesiology resident who called for the pediatrician's help and managed the resuscitation to the best of his abilities until the pediatrician's arrival.

DISCUSSION

Even when physicians have strived to provide the best possible care, harm to the patient can ensue. On review of this case, it is easy to identify actions that could have led to a better outcome. However, these actions may not have been as obvious at the time. This is called hindsight bias, and it is comforting to know that the Courts do consider it when adjudicating cases of alleged medical negligence.[5]

The inability to predict the occurrence of a legal action may lead some physicians to practice "defensive medicine". Defensive medicine refers to making clinical decisions based on fear of litigation rather than on the clinical needs and the best interests of the patient. Thus defensive medicine is not good medicine. Instead, physicians should practice medicine according to evidence-based or peer-reviewed standards and implement risk management techniques. Consulting colleagues in uncertain situations, appropriately applying current clinical practice guidelines and documenting the rationale for one's decisions can all improve a physician's defensibility.

Finally, as medical trainees, we should know the limits of our knowledge and experience. It is expected that we will be given greater responsibility and autonomy with increasing training and experience. The Courts have previously commented that it makes intuitive sense to have residents gradually left more on their own over the course of training. But it also means that we should not be afraid to ask for help. In failing to contact our supervisor, or to consult colleagues and other trusted resources, not only may the patient be harmed, but we may also be held legally liable for our actions.

During my summer internship at the Canadian Medical Protective Association (CMPA), these were just a few of the key lessons that I learned. If you want to learn more about ways you can promote safe medical care, visit the CMPA Good Practices Guide at www.cmpa-acpm.ca/gpg or just ask me for more information!

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