The articles in this summer supplement span the spectrum of medical conditions from psychosis to trauma, but all start from the same place: the emergency room.

In the emergency department (ED), teams of healthcare providers must be prepared for anything. While the cases in this issue were ultimately managed by many departments and specialists, the ED teams were on the front lines of care management and were responsible for appropriately coordinating care with other specialists. These articles remind us that each patient is unique, and that their medical management requires personalized plans, be they surgical, psychiatric, medical, or a combination of treatment modalities.

These case reports are not your typical day at the office. First, a patient repeatedly visits the ED with nonspecific abdominal symptoms to be eventually diagnosed with a rare case of mesenteric chylous lymphangiomatosis. In the next report, a unique case of psychosis is mitigated by treating suspected underlying attention deficit hyperactivity disorder. Finally, a 29-year-old man suffers severe hypothermic cardiac arrest after a car crash, but is successfully reanimated.

It is important to appreciate our fellow students for documenting these unique cases. They are disseminating the medical knowledge gained through clinical experiences and educating others about the quick decisions that ED teams and specialists made to change patient outcomes for the better. It is also important that we recognize the exceptional educational role undertaken by their supervising clinicians, who helped facilitate the first-hand clinical experiences.

Read on to discover the interesting cases with which students of the Schulich School of Medicine & Dentistry were involved over the summer of 2015.

— Ramona Neferu
Junior Associate Editor
Mesenteric chylous lymphangioma in the adult patient: a case report

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ABSTRACT
Mesenteric lymphangiomas are a rare cause of intra-abdominal mass in the adult population. The incidence is estimated to be 1 in 100,000 to 1 in 500,000 patients. For this reason, the diagnosis of mesenteric lymphangioma is often overlooked on the differential diagnosis of abdominal mass. We describe the case of a 29-year-old Caucasian male who presented with abdominal distention, nausea, vomiting, jaundice, and constipation, who was ultimately diagnosed with mesenteric lymphangioma. Furthermore, we discuss the etiology and epidemiology, presentation and diagnosis, and treatment of mesenteric lymphangioma.

INTRODUCTION
Mesenteric lymphangiomas are fluid-filled sacs which originate in the small bowel or large bowel mesentery and present as cystic intra-abdominal masses. These cysts may be pedunculated or sessile and hemorrhagic, serous, purulent, and/or chylous.1,2 Lymphangiomas are more prevalent in the pediatric population as they are often congenital malformations; presentation in the adult is exceedingly rare, with an estimated incidence of 1:100,000 to 1:500,000 patients.3 There are several theories pertaining to the etiology of mesenteric lymphangioma, of which one involves the aberrant proliferation of lymphatic tissue following abdominal trauma.1 Due to its relatively low incidence in the adult population, the diagnosis of mesenteric lymphangioma can easily be overlooked; however, it should be kept on the differential diagnosis for any patient presenting with an intra-abdominal mass. With this consideration, we present a case of mesenteric chylous lymphangioma in an adult patient and review the relevant background information and clinical management.

CASE REPORT
A 29-year-old Caucasian male presented initially to his local emergency department (ED) complaining of jaundice, nausea, vomiting, fever, and a distended abdomen. The patient stated that 4 days prior to his initial presentation he was shoveling snow from his driveway when he slipped on ice and fell onto his back. He reported no abdominal or gastrointestinal symptoms at the time of injury and proceeded to finish shoveling without complaint. Physical examination at that time revealed jaundice and a distended abdomen. He was subsequently admitted to his local hospital. The patient’s jaundice resolved after several days and he was subsequently discharged.

Two weeks later, he returned to his local ED with complaints of significant abdominal pain and further abdominal distention, nausea, and vomiting. Physical examination at that time revealed abdominal distention and a large intra-abdominal mass on palpation. He was readmitted to his local hospital for acute pain management; a computed tomography (CT) scan and magnetic resonance imaging (MRI) of the abdomen were arranged.

Twenty-five days after his injury, the CT and MRI scans of the abdomen were performed. They revealed a large cystic mass within the midabdomen, measuring 30 cm × 15 cm. The radiologist reported that the mass appeared to abut several regions of small bowel including the third part of the duodenum. There appeared to be some calcifications within the mass in addition to possible septations. The differential diagnosis of the mass favoured giant lymphangioma but also included duplication cyst, gastrointestinal tumour, lymphocele, and giant urachal cyst.

The patient was seen by a community general surgeon and a colonoscopy was performed, revealing no obvious pathology. The surgeon thus presented his case to the London Regional Cancer Program’s Gastrointestinal Multidisciplinary Tumour Board Case Conference. The patient was to be referred to our general surgery service for management and we saw the patient 1 month after his initial injury.

In our initial interview, the patient stated that he had a 20-pound weight loss and was experiencing constipation, nausea, fatigue, and exertional dyspnea. He reported being otherwise

Figure 1. CT scan of the abdomen at the L2 level showing a large cystic mass with several calcifications seen as white hyperdensities.
healthy with no significant past medical or surgical history. He was not taking any medications except Dilaudid 1 mg every 4 hours for pain. There were no known allergies. He denied any alcohol, smoking, or illicit drug use. There was no family history of abdominal mass or malignancy. Physical examination at this time revealed a large midline, nonfluctuant, periumbilical mass that was tender to palpation. Testicular examination was normal. Intervventional radiology was consulted, and together we determined that the patient would not benefit from percutaneous drainage of the mass. After a 3-week period of watchful waiting, we decided to perform an exploratory laparotomy for potential resection of the mass. The patient consented and was booked for surgery.

The surgery occurred 3 months after the initial injury. A midline laparotomy was performed. The tumour was visible immediately upon entering the abdominal cavity. It appeared grossly to be a lymphangioma. The dissection revealed the lymphangioma to originate from the small bowel mesentery at the level of the superior mesenteric artery. Additionally, there was extensive invasion of the colonic mesentery. During dissection, the tumour was inadvertently punctured; the contents were milky-white in colour and appeared chylous. We drained the cyst in vivo with simple suction. Further mobilization of the empty cyst sac revealed the stalk to be in close proximity to the superior mesenteric vein. The stalk was ligated, the cyst sac removed, and the specimen sent to the pathology department for investigation. Examination of the abdomen revealed a large defect in the colonic mesentery unsuitable for closure but with no compromise to the colonic blood supply. The abdomen was thoroughly washed and closed with no complications. Histopathology confirmed the diagnosis of lymphangioma.

The patient’s postoperative course was unremarkable. He experienced complete resolution of his symptoms, and aside from postoperative pain, reported no complaints. He was discharged from the hospital on postoperative day 3 with no concerns. He continues to be symptom-free at his 2-month postoperative visit.

**DISCUSSION**

**ETIOLOGY AND EPIDEMIOLOGY**

The first case report of a mesenteric lymphangioma was by Gardner in 1852. Since that time, the literature on these cysts has remained sparse. The etiology of mesenteric cysts in the adult is largely unknown. The most common hypothesized cause of mesenteric lymphangioma, due to the greater prevalence of mesenteric cysts in the pediatric age group; the incidence of pediatric mesenteric lymphangioma is estimated at 1 in 20,000 patients. Specific etiologies theorized in the pediatric population include failure of embryonic channels to join the venous system, failure of the leaves of the mesentery to fuse, and benign proliferation of ectopic lymphatics that lack communication with the normal lymphatic system. Presentation in the adult population is more exceptional, with estimates of the incidence of mesenteric lymphangiomas in adults between 1 in 100,000 to 1 in 500,000 patients. Theorized etiologies for development include neoplastic proliferation, lymph node degeneration, aberrant proliferation following local trauma, and benign proliferation of ectopic lymphatics.

**PRESENTATION AND DIAGNOSIS**

Mesenteric cysts are often an incidental finding on imaging for other phenomena, and thus typically present asymptptomatically. When they do present with symptoms, these often include nausea, emesis, constipation, abdominal pain, and abdominal distension; larger cysts may result in obstruction and volvulus. In a review of 162 cases, 60% of mesenteric lymphangiomas were located in the small bowel mesentery, and 24% in the large bowel mesentery. Rupture of the cyst may lead to an acute surgical abdomen requiring laparotomy. Diagnosis of these lesions focuses on history, physical examination, imaging, and pathology. The physical examination reveals a distended abdomen with a palpable abdominal mass. Jaundice may be seen with obstruction of biliary outflow. Ultrasound, CT, and MRI can all be used to further define and localize the mass. On imaging, mesenteric lymphangioma are often seen as solitary,
loculated cystic lesions with sizes ranging from 4 cm to 30 cm. It is difficult to differentiate mesenteric lymphangiomas from duplication cysts, gastrointestinal tumours, lymphoceles, and giant urachal cysts on imaging alone. Intraoperative examination reveals a fluid-filled sac arising from either the small bowel or large bowel mesentery, possibly with adhesions to surrounding tissues and organs. Finally, pathological examination will confirm the diagnosis of lymphangioma.

TREATMENT

Complete surgical excision of the mesenteric lymphangioma remains the mainstay of treatment. Incomplete resection increases the likelihood of tumour recurrence. Depending on the size of the cyst, laparoscopy or laparotomy may be favored; both approaches have been used with success in the literature. In 20% to 60% of cases, bowel resection with anastomosis was required to achieve complete excision of the cyst.

REFERENCES

We report on a unique clinical case of psychosis precipitated by Attention Deficit Hyperactivity Disorder (ADHD) and explore its implications for clinical practice as well as our understanding of these conditions. We describe a clinical case of a 46-year-old male presenting with auditory, olfactory, tactile, and visual hallucinations. We reviewed the literature on reported cases in which psychotic symptoms were treated with stimulant medications for ADHD comorbidity. This case report reveals the potential for properly selected patients to benefit from a consideration of ADHD comorbidity and a trial of treatment with that focus. In addition, the literature reveals a pathophysiological association between psychosis and ADHD supported by neurobiological data. However, far more research is required to fully understand these conditions and their relationship. We conclude that ADHD and psychosis have some related pathophysiological mechanisms but their connection has not been adequately explored. This case adds support to literature suggesting that in refractory psychosis, clinicians should re-evaluate the diagnosis and one of the considerations should be ADHD. In certain cases, the presence of psychotic symptoms with ADHD should not exclude the use of stimulants.

BACKGROUND

Psychosis is a common presenting problem in emergency departments (ED). Such episodes may present with positive symptoms, which include delusions, thought disorder, disorganized thinking, and hallucinations. They may also present with negative symptoms, which include deficits in normal functioning such as flat affect, alogia, avolition, and anhedonia. Cognitive symptoms of psychosis are also well documented, including deficits in working memory, attention, and executive functioning.

The management of psychosis is dependent on the cause, and therefore establishing etiology may be the most significant step in caring for the psychotic patient. Several disorders associated with psychosis, including schizophrenia, are also associated with ADHD. However, there is a lack of available data examining the relationship between ADHD and psychosis. In addition, stimulants commonly prescribed for ADHD management are considered to be contraindicated during psychotic episodes as they have been shown to exacerbate or even trigger positive symptoms in some patients. These barriers have prevented a thorough examination of the connections between psychosis and ADHD, despite there being a theoretical relationship.

We describe a relatively unique case, in which the presentation of psychosis was managed successfully by treatment with stimulant medication even though criteria for adult ADHD was not met. The difficulty in meeting adult ADHD criteria in this case relates to the similarity of cognitive symptoms seen in psychosis with ADHD symptoms, which include poor attention and executive functioning, and to psychomotor effects of ongoing antipsychotic medication. Criteria for a history of childhood ADHD were likely met on retrospective review of the case.

CASE PRESENTATION

INITIAL VISIT

JP was a 46-year-old male seen for a consultation following an ED visit 6 months prior where he presented with depressive symptoms. At that time, he was started on psychotropic medication (including lithium and risperidone) for a possible mood disorder. At the time of consultation, his mood was stable. However, he also admitted to experiencing visual, auditory, olfactory, and tactile hallucinations, and paranoid delusions for several months.

JP’s visit to the ED was his first encounter with psychiatric care. He did have a history of alcohol abuse though he stated he had successfully stopped consuming alcohol 12 years ago. He also described a history of marijuana use which ended 2 years ago. His past medical history was otherwise unremarkable.

Following his initial consult, JP was tried on various medications in attempts to address his psychotic symptoms. These included Invega Sustenna (paliperidone palmitate), Loxitane (loxapine), Risperdal (risperidone), and Zyprexa (olanzapine). He was also tried on lithium, Epival (divalproex sodium), and Prozac (fluoxetine) to continue managing his mood. Unfortunately, over the next 12 months he continued to have paranoid and persecutory delusions, with the occasional return of his visual, auditory, olfactory, and tactile hallucinations.

NEW MANAGEMENT

At 18 months from his initial ED presentation, JP revealed that his brother was also seeing the same psychiatrist. It was discovered that he was being treated successfully for ADHD with Dexedrine (dextroamphetamine), which is an amphetamine-based psycho-stimulant.

JP did not have any overt symptoms of ADHD that would have led to a clinical diagnosis of ADHD. However, given evidence of the genetic heritability of ADHD and the successful treatment of JP’s brother, a trial of Dexedrine was decided upon.
CASE REPORT

JP’s dose of Dextedrine was gradually increased from 5 mg to 10 mg. His psychotic symptoms decreased, and he was eventually taken off all of his antipsychotic medications. Currently, JP’s medications include Dextedrine 10 mg twice daily, fluoxetine 40 mg once daily and temazepam 15 mg once daily. He experiences no more psychotic symptoms. His mood is stable and he is doing well. After being off work for at least 2 years, he has since returned and was promoted to a role as supervisor.

LITERATURE REVIEW

There are multiple studies suggesting a relationship between childhood ADHD and psychotic presentations later in life. However, there is limited evidence in the literature examining the use of stimulants in refractory psychosis in situations where a comorbid diagnosis of ADHD is suspected.

Huey and colleagues (1978) describe a patient with chronic paranoid schizophrenia with a childhood history of minimal brain dysfunction (minimal brain dysfunction was a past term used prior to ADHD becoming a diagnosis) who received methylphenidate and subsequently suffered from decreased anxiety and intensity of hallucinations. Bellak and colleagues (1987) describe a young man presenting with schizophrenic episodes with a poor response to phenothiazines subsequently having a good response to methylphenidate plus lithium. Pine and colleagues (1993) present 2 adults with ADHD and comorbid psychosis successfully treated with a combination of stimulants and antipsychotics. They were then tapered off of their antipsychotics and remained free of psychosis with methylphenidate treatment alone.

More recently, Tossell and colleagues (2004) describe 5 cases where the use of stimulants was beneficial in patients diagnosed with childhood onset schizophrenia and ADHD once their psychotic symptoms were well controlled. A 2009 literature review and case report by Sambhi and Lepping describes 2 cases of patients suffering from psychosis benefitting from stimulant use. Finally, Rittmannsberger and colleagues (2014) describe the case of a 21-year-old patient who presented with a substance-induced psychosis and a highly probable diagnosis of adult ADHD; there was no major improvement on antipsychotic medications, and symptoms only significantly improved with the addition of methylphenidate.

DISCUSSION

Although our literature search revealed several case studies showing the improvement of symptoms of psychosis with methylphenidate treatment, there is an important nuance to consider. Stimulant medications in general lead to an upregulation of neural catecholamines such as dopamine and norepinephrine. This mechanism of action is opposite of that typically used to treat psychotic presentations in other disorders which generally respond to blocking of catecholamines such as dopamine.

Indeed stimulant medications have been shown to induce psychosis in some patients, a phenomenon referred to as hallucinosis or stimulant toxicosis. For this reason it is important to better understand the conditions that dictate whether stimulant use will lead to improvement or worsening of symptoms of psychosis.

In order to better understand the relationships that underlie this decision, we must consider the pathogenesis of these 2 conditions. The prefrontal cortex acts as a gating mechanism that enhances goal-directed activities and inhibits irrelevant activations. In ADHD these prefrontal processes are impaired. This is thought to be related to underactivity in several areas. Brain imaging studies have shown patients with ADHD have reduced activity in the prefrontal cortex and superior prefrontal cortex, both areas that are important for executive function, attention, and working memory.

Deficits in these specific regions appear to be associated with hypometabolism and dysregulation of dopamine, particularly the prefrontal D1 receptors, and norepinephrine. The decreased dopamine activity in the prefrontal cortex in ADHD is also seen in schizophrenia. This is thought to be related to the cognitive impairment (negative symptoms) associated with the disease.

One manifestation of cognitive impairment common to both ADHD and schizophrenia involves salience identification. In schizophrenia-associated psychosis, patients lose the ability to perceive their environment accurately and to pick up key information. The inability to filter this information contributes to an altered sensory experience marked by an inability to attend to important stimuli in a normal fashion. Similarly, inattention is a significant symptom of ADHD due to above-described prefrontal cortex dysfunction. As a result, someone with ADHD may experience symptoms similar to those of psychosis via an inability to properly attend to salient information.

The link between ADHD and schizophrenia can be potentially explained by the idea that they share deficits in prefrontal functions such as salience identification, working memory, and response variability. In line with these common neurobiological dysfunctions, new research has revealed some genetic repertoires for ADHD that also overlap with those of schizophrenia.

In light of this information, it may be reasonable to consider an underlying diagnosis of ADHD in a patient such as JP with refractory psychosis and a strong family history of ADHD. As stimulant medications may be associated with psychotic symptoms themselves, the decision to use stimulant medication should be done only after multiple other avenues have been explored. As we continue to unravel the relationship between psychosis and ADHD, the use of stimulants for management in ADHD may become more clear.

CONCLUSION

ADHD and psychosis are both common psychiatric disorders and have related pathophysiologic mechanisms. However, there has been longstanding reluctance to consider stimulant treatment in psychotic patients, preventing its use from being adequately explored. This case adds support to previous case reports noted in the literature that suggest that in refractory psychosis, one should re-evaluate the diagnosis and consider ADHD. In certain cases, the presence of psychotic symptoms with ADHD should not exclude the use of stimulants.
REFERENCES


Back from the dead: reanimation following severe hypothermic cardiac arrest

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BACKGROUND

Hypothermia is clinically defined as a drop in core body temperature below 35°C in a previously healthy individual in the absence of pre-existing inherent underlying thermoregulatory pathology. Classification of hypothermia below 35°C is described as mild (32°C to 35°C) and moderate (28°C to 32°C). A temperature below 28°C is classified as severe. At 28°C cardiopulmonary function is likely to become compromised. The myocardium becomes vulnerable to arrhythmia, ventricular asystole and subsequently complete cardiovascular collapse. Concomitant cognitive impairment is most commonly associated with severe hypothermia and cold injury. These patients tend to be individuals with mental illness, homelessness, and alcohol and illicit drug use. Recreational accidents such as skiing comprise the second most common group for hypothermic injury.1

There has been much academic debate and speculation regarding the optimal approach to rewarming any patient suffering from hypothermia.2 Mild hypothermia responds well to external rewarming techniques like forced heated air blankets.3 Moderate hypothermia is more precarious from a physiological standpoint. The myocardium is exceptionally vulnerable at these temperatures. Consequently, rapid rewarming techniques are favoured. This includes warmed intravenous (IV) fluids and warm peritoneal lavage in those who have not demonstrated overt signs of cardiac instability.4 Less commonly, these modalities can be augmented with airway warming and pleural irrigation.4,5

If patients with severe hypothermia have a sustainable cardiac rhythm, they are at increased risk for cardiovascular collapse.6 The optimal modality for rewarming a patient with severe hypothermic cardiac arrest remains a highly controversial topic.7 The most recent retrospective comparative study in 68 patients demonstrated cardiopulmonary bypass (CPB) rewarming to be far superior to conventional methods of rewarming, with mortality rates of 15.8% and 53.3%, respectively. Reanimation on CPB allows for several advantages, including rapid rewarming, but most importantly oxygenation and perfusion are preserved. A retrospective study analyzed the long-term neurological outcomes of survivors who reported normal neuropsychological findings in 93.3% of cases and normal brain magnetic resonance imaging in 86.7% of cases.8

CASE PRESENTATION

Patient MG is a 29-year-old gentleman with no previous medical history who presented to the emergency department in a peripheral hospital. MG was found near his vehicle which had crashed into a nearby tree. His core temperature was 27°C. There was minimal damage to the vehicle. He reportedly left his partner’s home at midnight and had been drinking alcohol and consuming illicit drugs.

The patient had been intubated on scene and peripheral vascular access obtained. Cardiopulmonary resuscitation (CPR) had also been initiated before he was transferred to hospital. CPR was continued in conjunction with external rewarming techniques which proved futile. The physician in the periphery contacted CritiCall Ontario and was put in contact with the nearest facility with CPB capabilities and a cardiac surgery team. The decision was made to transfer MG to University Hospital (UH) for reanimation on bypass. Throughout the resuscitative efforts, MG remained asystolic, with a Glasgow Coma Scale (GCS) score of 3. His neurological exam at that time showed dilated pupils with a normal Babinski sign. No other obvious injuries were appreciated on secondary survey.

Upon arrival to UH, MG was taken directly to the operating room (OR). Core temperature was repeated and confirmed at 21°C. The patient was switched from manual respiration to mechanical ventilation. He had elevated airway pressures and was difficult to ventilate. IV access was in situ and a radial arterial line and right internal jugular vein triple lumen central line were placed under ultrasound guidance. Rocuronium 50 mg IV was administered for paralysis along with 40 000 units of heparin. Initial arterial blood gas (ABG) showed pH 6.15, pCO2 67 mmHg, pO2 431 mmHg, bicarbonate 12 mEq/L, sodium 145 mmol/L, potassium of 6.4 mmol/L.

Concurrently, the surgery team attempted a cutdown of the right groin, but the patient’s right femoral artery could not be cannulated using a Seldinger technique. This was likely due to truncation and collapse proximally. Therefore, the left femoral artery was cut down and cannulated. 10 mg of midazolam was administered and CPB was initiated. MG was successfully rewarmed to 35°C and his cardiac rhythm showed ventricular fibrillation. MG was cardioverted once externally and converted to a normal sinus rhythm at a rate of 78 beats per minute with a mean arterial pressure (MAP) of 65 mmHg. ABG at that time showed pH 7.15, pCO2 39 mmHg, pO2 472 mmHg, bicarbonate 12 mEq/L, sodium 145 mmol/L, potassium of 6.4 mmol/L, lactate >15 mmol/L. He initially required no vasopressor support.

Following cardioversion and weaning from CPB, MG’s airway pressures remained high on the anesthesia machine. Pink froth began to emanate from the endotracheal tube and around the patient’s mouth. Videolaryngoscopy (Glidescope) showed the endotracheal tube between the vocal cords, and it was determined that the endotracheal tube cuff had ruptured. The patient was suctioned and reintubated using the Glidescope with an 8.0 mm internal diameter.
endotracheal tube. A ventilator from the medical-surgical intensive care unit (MSICU) was employed with little improvement in ventilation or airway pressures. Solumedrol 125 mg IV along with furosemide 40 mg IV was administered. There was mild improvement in ventilation. At this point in time, norepinephrine was started at 4 µg/min for hemodynamic support.

A transesophageal echocardiography (TEE) probe was inserted to assess heart function. The examination overall revealed no gross wall motion abnormalities, and the left ventricular function was mildly globally depressed. Given the ABG results and frothing from the tube, which constituted acute noncardiogenic pulmonary edema, the decision was made to reinitiate CPB for exploratory laparotomy. Intraoperative physical exam had revealed that MG’s abdomen was becoming quite distended and tense. General surgery was consulted and performed an exploratory laparotomy with no acute findings. The abdomen was left open with an ABThera vacuum dressing in place.

Following the laparotomy, the decision was made to convert the CPB circuit to an extracorporeal membrane oxygenation (ECMO) circuit and transfer the patient to the cardiac surgery recovery unit (CSRU). A venovenous ECMO circuit was considered. However, given MG’s extended period of cardiac arrest, an arteriovenous (AV) circuit was placed. He remained on the norepinephrine infusion at 8 µg/min and after conversion to ECMO his ABG showed pH 7.31, pCO₂ 41 mmHg, pO₂ 263 mmHg. Throughout the evening his vasopressor requirements increased and other injuries began to present themselves.

In the coming weeks, MG developed compartment syndrome in all limbs. Despite fasciotomy and surgical management, he required amputation of his right leg above the knee and his left arm above the elbow. Despite all of the sequelae from the initial hypothermic insult, MG regained neurological function with no focal neurological deficits. MG survived his extended stay in the CSRU and MSICU and was discharged to a rehabilitation facility.

DISCUSSION

This case presented some very unique challenges from a surgical and anesthetic management perspective. The case was treated as a trauma and the principles of resuscitation were employed at all levels of care. However, it is apparent that access to tertiary and quaternary hospitals is paramount to successful resuscitation. Had this gentleman remained in the periphery, he would likely not have survived. Despite the obvious morbidity MG faces with his quality of life being impacted by his amputations, he remained neurologically intact.

His airway and ventilation were of major concern throughout the reanimation. Despite correct placement of the endotracheal tube, it is likely that the aggressive resuscitative efforts increased pressure around the cuff of the endotracheal tube. More importantly the development of noncardiogenic pulmonary edema/acute respiratory distress syndrome (ARDS) upon rewarming posed significant management issues. This has been described in the literature upon rewarming of hypothermic patients. At this point in time there has been no trial which advocates for a specific treatment modality. Positive pressure ventilation in severe ARDS with IV steroids and diuretics are the mainstay of treatment. AV ECMO was preferred as this would permit bypass of the lungs entirely so that tissue integrity might be preserved during convalescence.

Furthermore, the patient presented with a profound acidosis with an associated hyperkalemia of 7.5 mmol/L. The potassium level prior to initiating CPB was associated with greater mortality for the patient. This is likely due to the fact that in mild hypothermia there is an initial inward shift of potassium, which is likely contributory to the arrhythmias that develop in those with moderate hypothermia. As the tissue becomes hypoperfused during cardiac arrest, cell necrosis will elevate the serum potassium. Profound hyperkalemia is a grave prognostic indicator as mentioned earlier on. Experts in reanimation have considered a potassium level greater than 10 mmol/L as the point of no return and subsequently a decision is made to withhold reanimation on CPB in that population.

Neurologically the patient did remarkably well considering his downtime was anywhere from 6 to 12 hours. Following deep hypothermic cardiac arrest, the emergent use of CPB has become advocated for as the preferred method of rewarming. It has shown superiority to conventional rewarming methods, particularly in adult patients. The survival benefits and reduced morbidity with the use of CPB in hypothermic arrest is significant. However, there has been no trial which compares outcomes in a randomized manner. Therefore, management is left up to the discretion of the trauma team in consultation with cardiac surgery and anesthesia. One thing remains certain: it has been said that those who work and live in extreme northern climates say, “You’re not dead until you’re warm and dead.”

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