Case study — Traumatic amputation

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Introduction

At approximately 2200h, on a summer evening, EMS received a call to respond to a motorcycle crash. Jim (pseudonym used), a 52-year-old motorcycle driver, was T-boned by a truck at highway speed. At the scene he was found supine on the grass, in no respiratory distress and GCS of 15. His left leg was mangled with a complete above-the-knee amputation and a laceration in the left femoral region. He was complaining of severe pain 10/10. The paramedics arrived on the scene and established their priorities: airway, breathing, circulation, and external hemorrhage control. Paramedics then applied large pressure dressings to the open wounds in an attempt to control bleeding from the amputated limb. Two large bore IVs were started and Jim was transported by land ambulance to the emergency department (ED) of a community hospital. The amputated limb was placed in a blanket at the scene and transported with the patient.

Case progression — Community emergency department

Fifty minutes post-injury, Jim arrived at the community hospital. His initial assessment revealed: A) airway patent, B) breathing spontaneously, but moderately distressed, C) pale, traumatic amputation left leg, vital signs: BP 84/60, HR 150, RR 23, and oxygen saturation 100% on supplemental oxygen, and D) GSC 15. Three litres of normal saline and eight units of uncrossed matched blood were rapidly administered as well as tetanus toxoid, Ancef and analgesia. Once stabilized, Jim was transferred to the regional trauma centre for definitive care.

Case progression — Arrival at the regional trauma centre

Two hours post-injury, Jim arrived in the ED of the regional trauma centre. Initial assessment revealed: A/B) RR 44, decreased air entry, C) systolic BP never greater than 80 mmHg, and D) increasing agitation. Therefore, Jim was intubated. In an effort to control bleeding from the left stump, orthopedics applied an auto tourniquet at 245 mmHg. Cuff pressure requires a physician order and needs to be high enough to completely suppress arterial circulation and control hemorrhage, yet low enough to minimize the risk of neuromuscular injury. Volume was administered in the ED using crystalloid, Pentaspan and blood products (see Table One). When transfusing units of PRBC it is critically important to also administer coagulation products to maintain the balance of plasma, proteins and coagulation factors. In addition, Recombinant Factor VII a (rFVIIa), Phenylephrine, and Calcium Gluconate were administered to control bleeding, increase BP and replace electrolytes depleted due to large amounts of blood transfusion. The left leg was stabilized with a Thomas Splint. Jim remained hemodynamically unstable throughout his ED stay despite indication of responsiveness to treatment (BP 110/60 and HR 110).

Hypothermia in trauma

Jim was admitted to ED with a temperature of 35.6°C, which remained low post-op (35.8°C), despite the Level 1 fluid warmer and blanket warmers. Hypothermia is defined as a core temperature from 35°C (mild) to below 32°C (severe). Jurkovich, Grerser, Luterman, and Curreri (1987) identified that up to 50% of trauma patients are hypothermic, which contributes significantly to morbidity and mortality, and that patients with temperatures less than 32°C are unlikely to survive. Hypothermia initially delays clotting factor activity, which contributes to prolonged clotting times and subsequent prolonged bleeding times (Watts, Trask, Soeken, Perdue, Dols, & Kaufmann, 1998; Cochrane, 2001). Persistent hypothermia can also contribute to increased platelet clumping potentiating the risk for consumptive coagulopathy and subsequent hemorrhage.

Both the ED resuscitation and the OR may increase the severity of the hypothermia. Trauma nurses must be vigilant in monitoring vital signs and ensuring that patients are kept warm. Inadequate tissue perfusion may prolong hypothermia, as an increase in metabolic heat production may be impossible during traumatic shock. Interventions such as administration of sedation and anesthesia will decrease the patient's metabolic rate, resulting in decreased heat

production and a lower core temperature. Anesthesia eliminates all shivering reactions, however, the body relies on shivering to increase heat production by up to five times normal. Anesthetic agents may also cause vasodilation, which will increase heat loss. Environmental interventions must be optimized to prevent hypothermia.

Case progression — To the OR

Jim remained hemodynamically unstable and hypoperfused when transferred to the OR, despite aggressive management of his hypovolemic shock. Upon arrival at the OR, the vascular team noted a large expanding left groin hematoma and became suspicious that the patient's continuing hemodynamic instability was related to an arterial bleed in the left femoral artery. Surgery was postponed in favour of a CT scan. In CT scan, the team continued to aggressively fluid resuscitate Jim for ongoing bleeding and hypotension. Despite additional blood product and crystalloid infusion (see Table One), Jim remained tachycardic (HR 120) and hypotensive (BP 80/60).

Traditional assessment in hypovolemic shock

Valuable assessment parameters available to the RN caring for a patient who is in profound hypovolemic shock include HR, BP (including pulse pressure), urine output, the patient's general colour, capillary refill and level of consciousness (LOC).

Tachycardia is the first sign of hypotension in hemorrhagic shock. Sympathetic stimulation will increase the heart rate to compensate for a decrease in stroke volume (the volume of blood ejected with each beat of the heart). Keep in mind that cardiac output (CO) = HR (heart rate) × SV (stroke volume). In early hypovolemia, the increased heart rate will be sufficient to maintain the cardiac output within normal limits. In hypovolemic shock, up to 30% of total blood volume can be lost before hemorrhage becomes clinically evident (American College of Surgeons Committee on Trauma, 2004). It is critical that aggressive fluid resuscitation be initiated with tachycardia. Delaying fluid resuscitation until the patient becomes hypotensive may mean they have lost a significant volume of blood and that end organ perfusion may be severely compromised before resuscitation is begun.

Decreased blood volume in hypotensive shock causes decreased circulation to the organs, and will result in diminished organ function. A decrease in urine output is one of the early signs of hypovolemic shock, as perfusion to the kidney is quickly reduced in order to preserve blood flow to the brain and other vital organs. A decrease in LOC and agitation can also be ominous signs of hypoperfusion. This may indicate inadequate cerebral perfusion and subsequent hypoxia to brain cells, despite the body's attempt to shunt blood to the brain. Jim had been intubated on arrival to the ED due to increasing agitation, decreased air entry and tachypnea. It is recommended that all patients with severe traumatic injury be intubated prior to transfer from the community hospital, as their status can quickly deteriorate en route (American College of Surgeons Committee on Trauma, 2004).

Jim's narrowed pulse pressure of 20 mmHg is a cardinal sign of ongoing hypovolemic shock (McQuillan, Von Rueden, Hartsock, Flynn, & Whalen, 2002). In the hypovolemic patient, pulse pressure (systolic BP minus diastolic BP) will narrow to below the normal of 40 mmHg, as sympathetic compensatory mechanisms vasoconstrict blood vessels to maintain the diastolic pressure. This mechanism acts to increase diastolic pressure which, in turn, increases venous return, and produces an increased preload to the right side of the heart. The increased volume of blood returning to the heart will increase cardiac output, resulting in a clinically improved blood pressure (McQuillan et al.)

Perfusion deficit in hypovolemic shock

Porter and Ivatury (1998) emphasize that compensated shock can persist after the clinical signs of uncompensated shock have been corrected. In other words, a patient's vital signs and pulse pressure may return to a near normal state following resuscitation, but sympathetic compensation masks ongoing tissue hypoperfusion.

Additional assessment parameters that reflect tissue perfusion also include acid base status, including base deficit, lactate and $ScVO_2$ ((American College of Surgeons Committee on Trauma, 2004; Barquist, Pizzutiello, Burke, & Bessey, 2002; Shulman, 2002). The trauma RN should ensure all hemodynamically unstable trauma patients have an arterial line initiated in ED. This will allow for easy access to blood taking and will simultaneously allow for direct, continuous monitoring of the patient's systolic, diastolic and mean blood pressure values.

Table One – Crystalloid/plasma expander/colloid and blood product utilization

Fluids	Pre-hospital	ED	CT	OR	ICU	Total
Crystalloid	2L	4 L	2L	4L	8L	20L
Pentaspan		2 L				2L
PRBC	8U	7 U	6 U	14U	8U	43U
FFP		5U		5U	7U	12U
Platelets				5U	5U	10U
Cryoprecipitate					5U	5U

Prior to transfer to the OR, Jim's hemodynamic status had improved noticeably (BP 110/60 and HR 110). However, his acid base status demonstrated he remained seriously hypoperfused, and in a state of metabolic acidosis:

PH 7.08 CO_2 34 O_2 500 HCO_3 10 base deficit -19

Metabolic acidosis is common in hypovolemic shock as a result of inadequate tissue perfusion and anerobic metabolism. Severe refractory metabolic acidosis usually reflects inadequate resuscitation from long-standing shock or ongoing bleeding (American College of Surgeons Committee on Trauma, 2004). Base deficit may be useful in determining the severity of the acute perfusion deficit and can be used as an approximation of global tissue hypoperfusion. The severity of the base deficit has been correlated with the adequacy of fluid and blood resuscitation and mortality in trauma (Porter & Ivatury, 1998; Barquist et al., 2002). Rutherford et al. (1992) found that a base deficit of -15mmol/L or greater in a hemorrhagic trauma patient is associated with significantly increased mortality.

Lactate is a byproduct of anaerobic glycolysis and can also be useful in the assessment of global perfusion in hemorrhage (Abramson, Scalea, Hitchcock, Trooskin, Henry, & Greenspan, 1993). A normal lactate level is 2mmol/L or less. When a patient is hypoperfused, oxygenation to tissues is inadequate. This lack of oxygen requires energy (ATP) to be produced by anaerobic metabolism, which results in the production of lactic acid (lactate). Increased levels of lactate may indicate insufficient oxygenation due to hypoperfusion. Scalea et al. (1994) found that 80% of multisystem trauma patients in their study had elevated serum lactate levels and decreased mixed venous oxygen saturation despite normal HR, BP and urine output.

Case progression – In the OR

CT revealed left common femoral vein and left distal profound femoral artery injuries, which were ligated in the OR. A hematoma was evacuated and a hemovac drain was placed. The surgical team also revised the above-knee amputation.

In the OR, Jim's blood work revealed a prolonged clotting time: INR 3, PTT 39 and Hgb 82. Jim also had a critically elevated potassium level of 6.2 mmol/L, leaving him vulnerable to lethal cardiac arrhythmias. This elevation in serum potassium was likely a reflection of two physiologic responses: tissue damage and metabolic acidosis. With traumatic injury to skeletal muscle, the muscle breaks down, and potassium is released into the bloodstream, resulting in increased serum levels. In metabolic acidosis, an increase in hydrogen ions causes the ions to move into the cells. In a compensatory movement, potassium, the body's main intracellular cation, will move out of the cell to try to maintain the polarity of the intracellular fluid which, in turn, elevates serum potassium levels. Jim was given 10 units of humulin regular insulin as well as a bolus of 50% Dextrose and then started on an infusion of D5W. The insulin causes glucose to move into the cell and it will pull some of the serum K with it, dropping the critical serum K level and removing the risk of arrhythmia from hyperkalemia. Active resuscitation continued in the OR as Jim received further blood product, crystalloid and rFVIIa (see Table One).

Case progression – To the ICU

Jim's hemodynamic status deteriorated immediately post-op on his arrival to the ICU. His admission vital signs demonstrated uncompensated hypovolvemic shock: HR 130, BP 75/50, pulse pressure of 25mmHg and a temp of 35.8. His ABGs (PH 7.33, CO_2 45, O_2 220, HCO_3 22, base deficit -2.3, and lactate 6 mmol/L) reflected an improved perfusion status in comparison to his pre-OR status. However, his sustained hypotension and Hgb of 39, INR 1.7, PTT 87, as well as overt signs of bleeding from his stump and operative site demanded immediate fluid and blood resuscitation by the ICU team. Orthopedics was called stat and the surgical team attempted exploration of the bleeding site in the ICU. The level 1 fluid warmer was deployed to transfuse additional crystalloid and colloid products while cryoprecipitate and platelets were also given through a separate line (these products should not be warmed when transfused [The Ottawa Hospital, 2002]). A third dose of rFVIIa, Factor X and transemic acid were added to the resuscitation. Phenylephrine was infused briefly when the team was unable to detect a blood pressure. Vasopressors are not routinely recommended for the hypovolemic patient as they may worsen perfusion (American College of Surgeons Committee on Trauma, 2004), however, when optimal fluid resuscitation is insufficient to maintain cardiac output, vasopressors may be used temporarily.

Forty-five minutes after his arrival in ICU, Jim was transferred back to the OR for exploration of the femoral artery and vein. The vessels were noted to be boggy, oozing, and were subsequently ligated. Before returning to the OR, ABG samples taken in the ICU provided evidence of worsening tissue perfusion: PH 7.31, CO_2 45, O_2 220, HCO_3 22, and base deficit -3.7. The worsening base deficit and the lower PH indicate tissue hypoperfusion despite administration of fluids, blood products, clotting factors and vasopressors. This change in base deficit, which occurred over the 45-minute resuscitation in ICU, demonstrates the usefulness of trending a base deficit in conjunction with vital signs and urine output in the hemorrhagic patient.

Coagulopathy in trauma

Exsanguination accounts for 38% of all hospitalized deaths of trauma patients (Sauaia, Moore, Moore, Moser, Brennan, Read, & Pons, 1995), and is the second leading cause of mortality in trauma (second only to head injury). Many trauma patients threatened by hemorrhagic shock and exsanguination develop some degree of coagulopathy due to

large amount of fluids, hypothermia, acidosis and multi-transfusion syndrome (Martinowitz, Holcomb, Pusatein, Stein, Onaca, Friedman, Macaitis, et al., 2001). Coagulopathy is defined as the inability to establish hemostasis with resultant excessive bleeding. Coagulopathy coupled with acidosis and hypothermia, often referred to as the lethal triad, significantly increases the risk of mortality in the hemorrhagic trauma patient (Mikhail, 1999).

Hemostasis in bleeding is achieved by the activation of a complex clotting cascade that comprises the intrinsic (plasma activated) and extrinsic (tissue activated) pathways. Chemical factors are released by damaged tissue and a platelet plug is formed to temporarily seal the hole in the blood vessel. Fibrin is eventually produced and crosslinks to anchor platelets in place to achieve a stable clot (Morton, Fontaine, Hudak, & Gallo, 2005). In massive hemorrhage and subsequent resuscitation, clotting factors may be diluted by massive fluid resuscitation or over-consumption. As well, both acidosis and hypothermia affect platelet function by inhibiting the enzymes that activate platelet aggregation, thus preventing stable clot formation (Mikhail, 1999; Klemcke, Delgado, Holcomb, Ryan, Burke, DeGuzman, et al., 2005; Reiss, 2000).

A promising therapy for hemorrhage in trauma patients has recently emerged — Recombinant Factor rVIIa, which complexes with Tissue Factor in the extrinsic pathway and leads to the activation of fibrin via the common pathway. Boffard et al. (2005) conducted a large multicentre international study of 301 trauma patients with both blunt and penetrating trauma. The amount of red blood cell transfusion was reduced with rFVIIa treatment. Many trauma centres are adding these pharmacological approaches to their management of hemorrhagic shock. Jim received Factor rVIIa in the ED, the OR and in the ICU for ongoing bleeding, as well as Factor X and tranexamic acid in the ICU.

Jim received significant fluid resuscitation in the first 24-hour period post-injury, as illustrated in Table One, as well as Factor X and tranexamic acid in the ICU. Thirty-six hours post-admission hemostasis was obtained. However, Jim continued to have ongoing instability due to complications related to his injuries: acute lung injury and sepsis.

During his stay in the ICU, Jim showed evidence of sepsis and septic shock, which were treated aggressively with fluid, vasopressors, antibiotics and surgical revisions. Jim returned to the OR for incision and drainage of his left thigh stump four times before his discharge from ICU.

Progress towards rehabilitation

Seventeen days post-admission to the ICU, Jim was transferred to the trauma unit. He progressed very well and did not experience any complications. He was subsequently discharged to his community hospital to await healing of his stump prior to admission to the rehabilitation centre. Unfortunately, because of Jim's initial hemodynamic instability, ongoing bleeding and sepsis, the revisions of his stump resulted in decreased preservation of limb length. The goal in amputation is to optimize stump length for a more ideal prosthesis and improved walking capacity, while maximizing tissue integrity and healing. Jim's prosthesis was optimized and his physical and psychological needs were addressed during rehabilitation. Several months later, Jim returned to visit the staff of the trauma unit, walking independently and ready to face new challenges!

Motorcycle injuries — An ounce of prevention...

Motorcycle sales in Canada have more than tripled in the past few years (Canada Safety Council, 2005). The typical rider in Canada has increased in age (46 years), and they are buying more powerful and expensive machines. With this increase in popularity comes the potential for increased injury. Motorcycles typically offer very little protection for the rider. The fatality rate for motorcycle riders involved in a collision is 34 times greater than for those riding in a car, and the risk of serious injury is about eight times greater (National Highway and Traffic Association's National Centre for Statistics and Analysis, 2005). Other factors that contribute to the risk of injury while riding motorcycles include: speeding, impairment by drugs or alcohol, invalid licensing, and neglecting to wear a helmet. Typical injuries seen are to the head, arms and legs. With this increased risk of injury, there is a clear role for injury prevention.

So, what can you do? The ED may provide a unique opportunity to implement injury prevention information to motorcyclists (Blanchard & Tabloski, 2006), but what can you teach to patients in order to prevent future injury? Here are some important points to cover with your patients:

Get trained: Take a course on how to operate this powerful machine safely. Courses such as The Canadian Safety Council's *Gearing Up* teaches riders how to pick the right bike for their needs, and to learn about speed control, balance and breaking, basic traffic behaviour, urban riding tactics, and emergency driving techniques. Driver education has been shown to reduce injury and collision rates (Savage, 2001).

Drive sober: Alcohol is a factor in nearly one-third of all collisions involving motorcycles (Canada Safety Council, 2005). Motorcycles require more skill and coordination to operate than a car, so even small amounts of drugs or alcohol affect your ability to operate the bike safely.

Wear the gear: Protective clothing and equipment is key. Long-sleeved jacket and pants should be worn and should be made of a strong, durable fabric such as leather. This will offer more comfort when riding and serves to protect against abrasion. Bright colours allow other drivers to see you more readily. Pants should not be flared to avoid getting caught in the gears, foot pegs or starter. Gloves are recommended for all seasons. Proper footwear, such as leather boots, protects the

ankle, foot and lower leg, which are commonly injured in collisions involving motorcycles. Perhaps the most important piece of gear is the helmet. In 1996, 41% of motorcyclists killed in Canada were not wearing a helmet (Canada Safety Council).

Conclusion

Jim's case exemplifies the importance of the trauma care continuum, from pre-injury to rehabilitation, with all health care providers working together to return the patient to optimal family and work life. Trauma nurses are reminded that key assessment parameters beyond the traditional vital signs and pulse pressure measurements, such as acid base status (including base deficit, lactate levels and uses of $ScVO_2$ monitoring) can lead to the early identification of the under-resuscitated trauma patient. This case review also highlights complications in the trauma population: hypothermia, hypovolemic shock and coagulapathies. Emergency nurses have a key role in the identification and management of these potential complications as early aggressive identification and treatment plays a critical role in the patient's morbidity and significantly impacts their eventual health status.

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