outlook
Trauma Corner

Snowboarder versus tree: Significant abdominal trauma

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A 20-year-old male, on a skiing holiday with family, lost control of his snowboard and hit a tree at high velocity. He was not wearing a helmet, and sustained a loss of consciousness. Initial transport was to a hospital close to the ski hill. On arrival, the patient was pale, cool, and diaphoretic with presenting vital signs: BP 94/60, HR = 124/minute, RR = 28/minute and shallow, and temperature of 35.8ºC. Treatment included 4 litres normal saline (NS) and two units packed red blood cells (PRBCs) for his hypotension and tachycardia. A Focused Assessment with Sonography for Trauma (FAST) revealed a rigid abdomen with fluid within the subhepatic space, Morrison's pouch. In the setting of acute trauma, this is one of the most dependent portions of the abdomen and should be examined closely for the presence of free intraperitoneal fluid (Net Medicine, 1996). He was transported by helicopter to an urban trauma centre.

Trauma centre initial care

On arrival to the urban emergency department (ED), the patient was combative with a Glasgow Coma Scale (GCS) of 14. Mottling of the abdomen was evidence of the degree of force from the impact with the tree. He continued to show signs of hypovolemic shock with a heart rate between 135-145/min and blood pressure dipping to 70 mm Hg systolic. The decision was made to intubate the patient using Rapid Sequence Induction (RSI); medications administered were Fentanyl, Etomidate and Succinylcholine. Treatment for the hypovolemic shock included another litre of NS, six units PRBCs and a litre of Pentospan. An orogastric tube was inserted; the Foley catheter inserted in the initial hospital showed gross hematuria. He received both calcium chloride and calcium gluconate for hypocalcemia. Zofran was given for nausea. Although the patient's temperature was 36.5ºC on arrival, the Bair Hugger™ warming blanket was applied to prevent hypothermia. After 50 minutes of emergency care, the patient was transferred to the operating room (OR).

Table One. Summary of traumatic injuries	
Head	Right temporal lobe contusion Small subarachnoid hemorrhage Small right temporal subdural hematoma
Abdomen	Avulsion of right side of liver (Grade 5 liver injury) Avulsion of right kidney Grade 2 spleen injury

Operative procedures

The patient's initial trip to the OR was to stabilize his sources of internal blood loss. He was found to have 2.5 litres of blood in the peritoneal cavity. He sustained an intraoperative Pulseless Electrical Activity (PEA) arrest, and required an emergency clamshell thoracotomy with CPR. The aorta was clamped for a total of 20 minutes. Hemorrhage was controlled and aggressive fluid resuscitation was required: 17 units PRBCs, 15 units Platelets, and 7 units of Fresh Frozen Plasma (FFP). A decision was made to do only 'damage control' surgery and transfer the patient to the Intensive Care Unit for further resuscitation and warming. Once further stabilized, he went to angiography where the hepatic artery was embolized. Table One summarizes all traumatic injuries while Table Two summarizes all operative procedures.

Table Two. Summary of operative procedures	
Timeframe	**Procedures**
Day of Injury	Laparotomy, right hepatectomy, right nephrectomy, emergency Clamshell thoracotomy, CPR, damage control closure
2 Days Post-Injury	Exploratory laparotomy and thoracotomy with closure of thoracotomy, cholecystectomy and damage control closure
3 Days Post-Injury	Laparotomy, washout and closure of abdomen
8 Days Post-Injury	Tracheostomy

Post-op complications

After his initial OR, the patient required norepinephrine in addition to large amounts of fluid to become hemodynamically stable. Respiratory complications included ventilator-associated pneumonia (VAP), and a paralyzed diaphragm, thought to be due to phrenic nerve contusion or diaphragm contusion related to the significance of the impact.

He developed jaundice, which was attributed to the major liver resection, the significant pigment load from the massive blood transfusion and the hypovolemic shock. All complications eventually resolved and the patient was transferred to the trauma ward 12 days post-injury.

Neurosurgery was consulted regarding the patient's head injuries. It was decided surgical intervention was not required. No immediate neurological deficits were obvious, but due to the patient's tracheostomy, full cognitive assessment could not be performed.

Teaching points

Hypocalcemia during resuscitation

It is important to assess for and treat hypocalcemia in patients receiving massive blood transfusions. The citrate added to banked blood as an anticoagulant binds with calcium and renders it unavailable for use (Springhouse Corporation, 1997). Calcium can be measured in two ways—total serum calcium and an ionized calcium. In treating shock, the more important level is the ionized calcium, as it reflects the available calcium that can be used by the body for controlling cell membrane permeability, muscle contraction and in the clotting cascade (Calgary Laboratory Services, 2006). Since almost half the body's calcium is bound to albumin, serum protein levels will affect serum calcium levels. An ionized calcium is quickly available through a blood gas and the result is not influenced by albumin levels.

Table Three. Significant Lab Results

Timeframe	Lab Result		Normal Range[2]
Arrival to Trauma Centre	Hemoglobin	82 g/L	Males 137–180 g/L
	Hematocrit INR PTT Fibrinogen	0.24 L/L 1.8 50.7 0.9 g/L	Males 0.40–0.54 L/L 0.9–1.1 28.1–41.0 seconds 1.6–4.1 g/L
Initial Arterial Blood Gas	pH	7.11	7.36–7.44
Fi02 = 100	$pC0_2$ $p0_2$ Base Excess HC03 Lactate Ionized Calcium	41 274 -16 13 5.2 0.86	30–40 mm Hg 70–88 mm Hg -5 to -1 mmol/L 20–24 mmol/L < 2 mmol / L 1.15–1.35 mmol/L

Massive blood transfusions and coagulopathy in trauma

Patients involved in major trauma are at particular risk of developing blood coagulopathies. While attempting to compensate for hypovolemic shock due to potential blood loss, the patient is also at risk of developing disseminated intravascular coagulation (DIC) (Drews & Weinberger, 2000). Management becomes even more complicated if large amounts of blood products are required during the resuscitation process (Reed, Ciavarella, Heimbach, Baron, Pavlin, Counts, & Carrico, 1986).

DIC is the ongoing process of systemic and simultaneous coagulation and fibrinolysis. A noxious stimuli, such as trauma, causes the procoagulant tissue factor to initiate and then sustain the clotting cascade. Widespread intravascular coagulation occurs, which begins the natural counter process of fibrinolysis. As platelets, fibrinogen, prothrombin and clotting factors are consumed, the antithrombotic pathways are overwhelmed, exhausting the body's clotting mechanisms (Drews & Weinberger, 2000). This process results in widespread and uncontrolled bleeding in an already compromised trauma patient. While other therapies may be employed, definitive management of DIC requires identifying and treating the underlying cause (Drews & Weinberger).

Emergency resuscitation and stabilization of the trauma patient can include massive amounts of isotonic crystalloid and blood products. Studies have shown that after replacement of approximately 1.5 times the patient's blood volume in packed red blood cells (PRBCs), dilutional thrombocytopenia is increasingly more likely (Reed et al., 1986). Thrombocytopenia is defined as a platelet count >100 000/μL, while severe thrombocytopenia occurs when the count dips below 50,000/μL (Drews & Weinberger, 2000). Neglecting to administer platelets and/or plasma containing clotting factors during the resuscitation phase involving upwards of 15 to 20 units of PRBCs, can result in diffuse microvascular bleeding, characterized by the inability to clot and the loss of blood from trauma wounds, mucosa and puncture sites (Reed et al.). While evidence is equivocable, it is the practice of some trauma institutions to prophylactically administer a standard number of units of platelets and/or fresh frozen plasma (FFP) at a set ratio to PRBCs during massive transfusions in an attempt to avoid dilutional thrombocytopenia (Reed et al.).

Damage control surgery and the 'Trauma Triad of Death'

One of the major advances in surgical practice in the last 20 years is damage control surgery following trauma (Ghosh, Banerjee, Banerjee, & Chakrabarti, 2004). The overall concept is to strategically stage resuscitation and surgical efforts to reduce early mortality from the extreme physiological changes following severe hemorrhagic polytrauma that have become

known as the 'Trauma Triad of Death"—hypothermia, coagulopathy and acidosis (Ghosh et al.). There is a complex relationship between these factors and cascades of events that cause one factor to accentuate the other—the vicious cycle leading to death. To break the cycle, a three-stage approach was designed (Ghosh et al.):

Stage 1: Prehospital to OR

- Resuscitation starting in the field and continuing until the patient reaches the OR
- Abbreviated laparotomy or 'bail-out' surgery
- Control of hemorrhage is priority
- Temporary closure of abdomen with packing and compression

Stage 2: Intensive Care Unit
(24-48 hours post injury)

- Focus is on stabilization of patient and correction of hypothermia, coagulopathy and acidosis
- Focused assessment to identify any occult or missed injuries

Stage 3: Planned Return to OR
(36-48 hours post injury)

- Timing is important to decrease incidence of onset of multiple organ dysfunction syndrome (MODS)
- Removal of packing and control of small bleeding vessels
- Thorough exploration for hidden injuries
- Vascular repair
- Restoration of GI continuity; provision for enteral feeding
- Washout of abdominal cavity.

The patient in this case study was appropriate for damage control surgery. Table Three outlines initial significant laboratory results that showed acidosis and coagulopathy.

Abdominal Compartment Syndrome (ACS)

Signs and symptoms of ACS include elevated intra-abdominal pressure (IAP), a tense, distended abdomen, an increase in airway pressures with inadequate ventilation and impaired renal function (Ivatury, 2004). Certain medical and traumatic conditions increase a patient's risk for ACS: pre-operative hypovolemic shock and massive fluid resuscitation, increased intra-abdominal fluid accumulation, e.g., ascites, and any mechanical increase in pressure such as with damage control surgery. The patient in this case study was certainly at risk for ACS. Through judicious monitoring of fluids, this complication was fortunately avoided. One challenge is early detection; treatment of this potentially lethal complication is prompt decompression of the abdomen.

Outcome

Continuing post-operative care of pain management, nutrition and mobilization resulted in steady improvement. His tracheostomy was downsized, de-cannulated and four chest tubes were removed. As this patient had no long bone fractures, he was able to mobilize without too many barriers. Although the focus of care was on the abdominal injuries, it was not forgotten that this patient did suffer a head injury (Table One). Occupational therapy did conduct a Montreal Cognitive Assessment (MoCA©) prior to discharge. The MoCA© is a cognitive screening test designed to assist health professionals in the detection of mild cognitive impairment (Montreal Cognitive Assessment, 2003). This patient scored 21/30 whereas normal cognitive functioning is a score of above 26/30. Further cognitive assessments were recommended.

As the patient's home was in the United Kingdom, efforts were focused to stabilize the patient enough to be transferred back home to the care of another surgeon and a rehabilitation team. He was discharged to a local hotel three weeks post-injury, with arrangements for air medical transfer home for follow-up care. The main issue was eliminating the gas in the patient's chest so he could fly. His two liver drains were still draining bile on discharge. This patient's age and pre-injury health and fitness status certainly played a role in his overall survival and healing.

And, we're hoping that along the way, someone had a discussion with the patient and his family about helmets and future head injury prevention…

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Abdominal trauma is not the only risk from engaging in snowboarding. WipeOut is a one-hour documentary produced through a collaboration of the Brain Injury Association of Canada and the Knowledge Network (which has aired it several times on its channel). It is produced by award-winning filmmaker Lionel Goddard and premiered at the Doxa Film Festival in Vancouver this past June. WipeOut features Olympic medallist Ross Rebagliati and Dr. Brian Hunt, and follows three young men after their brain injuries sustained while pursuing extreme sports—including snowboarding. It is a film well worth watching and can be viewed online through a link at ***www.biac-aclc.ca****. For further information on injury prevention, or to order a copy of the film please email* ***info@biac-aclc.ca****.*