outlook
Trauma Corner

Case study: The link between severe traumatic brain injury and coagulopathy

By Margaret M. Dymond, RN, BSN, ENC(C)

Severe traumatic brain injury (STBI) and the subsequent development of coagulopathy increase mortality in trauma patients (Stein, Dutton, Kramer, & Scalea, 2009; Talving et al., 2009). The following case study will highlight the link between STBI and the development of coagulopathy as a risk factor for poor patient outcomes.

Pre-hospital care

Emergency Medical Services (EMS) transported a 50-year-old male to the emergency department (ED). The patient was struck by a car while crossing the street, and found to be combative on the scene. Cervical spinal immobilization was initiated and the primary and secondary survey performed. The primary survey indicated an intact airway, decreased breath sounds bilaterally, weak radial pulses, and a Glasgow Coma Score (GCS) of 7. Secondary survey findings included head/facial/limb abrasions, abdomen soft, and stable pelvis. Initial pre-hospital interventions included advanced airway management and initiation of intravenous (IV) therapy. The patient was difficult to intubate with an endotracheal tube (ETT), therefore a Combitube® was inserted. During transport, the patient deteriorated and arrived to the ED in a cardiac arrest. Total pre-hospital to ED time was 35 minutes.

Table One. Initial lab results

Lab Results	Time: 0821	Time: 0854
HgB	131g/L	95g/L
PTT	66 sec	
PT-INR	1.5 sec	
PLT	104 10**9/L	
PH	<6.8	7.01
PO2	110 mmHg	66 mmHg
PCO2	>104 mmHg	88 mmHg
BE	-18 mmol/L	-13 mmol/L
Lactate	15 mmol/L	13.6 mmol/L

**arterial samples

Emergency department care

The patient arrived in the trauma room in full spinal protection, bagged with a Combitube®, CPR in progress and one peripheral IV catheter in situ. A primary survey was conducted by the trauma team leader (TTL). Blood was apparent in the oral cavity requiring suctioning. Breath sounds were difficult to assess. The Combitube® was removed and bag valve mask (BVM) ventilation was initiated. The patient was intubated successfully with an ETT using a glidescope. Breath sounds were noted bilaterally, but decreased. The surgery team was requested to insert bilateral chest tubes. Initial chest tube drainage was minimal. The patient was placed on a cardiac monitor showing bradycardia. No central pulse was palpated. Slow Pulseless Electrical Activity (PEA) cardiac arrest was present. The TTL ensured high-quality CPR was continued with the addition of appropriate interventions and medications including IV fluid boluses, Epinephrine, and Atropine. Supportive care included insertion of a gastric tube and a urethral catheter draining yellow urine.

Complicating the resuscitation attempt was an interstitial IV. Attempts to gain peripheral IV access were unsuccessful. The TTL requested a central venous access. The femoral artery was cannulated on the first attempt. Standard trauma laboratory tests were obtained. An arterial line system was set up and indicated systolic B/P of 90 with chest compressions. Central venous access was quickly achieved permitting infusion of IV fluids and medications.

During the initial resuscitation, the patient's pulse became palpable at intervals following administration of Epinephrine IV. The patient arrested twice more in the first 30 minutes of

care in ED and required CPR for short intervals. Further interventions included x-rays of the chest, cervical spine and pelvis. The radiographic abnormalities included rib and c-spine fractures.

Significant initial lab results (Table One) indicated a developing coaguloapthy, acidosis, and hemorrhage. The patient's hemodynamic status was optimized by the administration of two units of unmatched packed red blood cells.

Further diagnostic imaging was obtained including computer tomography (CT) exams of the head and neck. Results included a subarachnoid hemorrhage, massive cerebral edema, marked effacement of sulci, and atlanto-cervical dislocation. It was determined that these injuries were incompatible with life. No further CT imaging was ordered.

On return to ED from CT, the patient's vital signs were: B/P 90/70, HR 104. Head-to-toe exam findings were continuous bleeding from head and facial abrasions, a large amount of bloody drainage on the chest tube dressings, 400 mLs bloody drainage from the left chest tube, 100 mLs from the right chest tube, and frank hematuria.

Pathophysiology

The brain is rich in tissue thromboplastin. Once brain tissue is injured, thromboplastin is released and activates the clotting cascade. Damage to the cerebral vessels can activate platelets producing intravascular coagulation leading to consumption of platelets and other clotting factors. Plasmin is also activated, which will break down clots forming around sites of vessel injury causing fibrinolysis. These factors can lead to further hemorrhage at local sites of injury and systemically (Carrick, Tyroch, Youmens, & Handley, 2005; Cohen et al., 2007; Kushimoto, Yamamoto, Shibata, Sato, & Koido, 2001).

STBI results from the initial impact and injury, plus secondary factors post-insult (Carrick, Tyroch, Youmens, & Handley, 2005). Tissue hypoxia is a secondary factor that activates the inflammatory mediators and tissue thromboplastin in the brain. These factors cause further damage to cerebral tissues after initial impact. Inflammatory mediators further compromise brain tissue resulting in severe secondary injury to the brain (cerebral edema, ischemia, and infarction) (Miner, Kaufman, Graham, Haar, & Gildenberg, 1982). Hypoxia, hypotension, hypercarbia, and hyperglycemia were all present in this patient, which led to progressive damage to the cerebral structures. Patients who are acidotic are less able to clot due to inhibition of thrombin and platelet malfunction (Cohen et al., 2007; Martini, 2009). This can contribute to ongoing coagulopathy.

Discussion

The development of coagulopathy in STBI contributes to higher rates of mortality (Talving et al., 2009). The literature suggests that patients who have an STBI and coagulopathy have an increased risk of an adverse outcome and death. Talving et al. reported that patients with STBI who developed coagulopathy had a 50% mortality compared to 7% who had incurred an STBI with no coagulopathy. Miner et al. have reported the incidence of developing coagulopathy increases with the severity of the STBI in the range of 33% to 93%. Similar groups of studies have reported the incidence of coagulopathy after STBI between 15% and 100% (Cohen et al., 2007).

Some investigators have used GCS as a prognostic indicator: the lower the GCS, the greater the risk of coagulopathy with limited success. The assumptions were the lower the GCS, the greater the brain insult. Cohen et al. (2007) investigated the link between STBI with hypoperfusion and the development of early coagulopathy. They concluded that not all patients with STBI developed coagulopathy and that the onset of coagulopathy is closely associated with hypoperfusion in patients with base deficit >6. Patients with a low GCS and no hypoperfusion did not develop coagulopathy.

Several investigators have studied markers of coagulation and inflammation in patients with STBI and coagulopathy. Some research studies have concluded that markers of coagulation can be a prognostic indicator. Olson et al. (1989) reported that elevated PTT, and/or INR, and/or low platelet counts in patients with STBI was an indicator of increased mortality. Kushimoto et al. (2001) evaluated the components of the fibrinolytic system in patients with STBI and found that patients with coagulopathy had poorer outcomes.

A study by Talving et al. (2009) found independent risk factors in assessing outcomes in patients with STBI and coagulopathy. They include GCS<8, Injury Severity Score (ISS) of >16, hypotension upon admission, cerebral edema, subarachnoid hemorrhage, and midline shift. All of these factors were present in this case study.

Patients can become coagulopathic from multiple factors during resuscitation. Large volumes of crystalloid administration can lead to dilutional effects of clotting factors. Bleeding from other major injuries can lead to consumption of clotting factors (Stein, Dutton, Kramer, & Scalea, 2009). Hemorrhage from internal injuries could not be ruled out in this case, as a complete trauma diagnostic exam was not performed due to the severity of the head and neck injuries.

Treatment

Patients with STBI and coagulopathy are treated with replacing clotting factors and platelets to attempt to reverse bleeding. This is important to prevent ongoing hemorrhage in the brain and is required if surgical intervention is planned. Administration of fresh frozen plasma, cryoprecipitate, packed red blood cells, and vitamin K may be required (Carrick, Tyroch, Youmens, & Handley, 2005). A novel intervention currently being debated and studied is administration of recombinant Factor V11a as an option in some patients. Stein et al. (2009) have reported that administration of Factor V11a in patients with STBI and coagulopathy has not been proven to show a mortality benefit, but the investigators report a decrease in length of stay in ICU and decreased use of blood products in this population of patients.

Outcomes of case study patient

Due to the severity of the brain injury, the trauma team discussed the prognosis with the family. Care was withdrawn. The patient died several minutes later.

Summary

Patients with STBI who develop coagulopathy have an increased risk of morbidity and mortality. Not all patients with STBI develop coagulopathy, but patients with hypotension and hypoperfusion on admission to ED are at increased risk (Cohen et al., 2007). Trauma nurses should be monitoring standard coagulation profiles on their patients and must be prepared to intervene early to minimize complications and optimize outcomes (Carrick, Tyroch, Youmens, & Handley, 2009).

About the author

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