Future Trauma Team Conference Dates:

Thursday May 22, 2014	18:00	Dolan CR
Tuesday June 24, 2014	12:30	First Colony CR 1
Tuesday August 26, 2014	12:30	First Colony CR 2
Thursday September 18, 2014	18:00	Dolan CR
Tuesday October 28, 2014	12:30	Bryant CR
Thursday November 20, 2014	18:00	Dolan CR

No December Conference Happy Holidays!

Case 1: Pediatric GSW

The first case we reviewed was a pediatric patient who presented to the ED as a level I alert after being shot in the abdomen. The full trauma team was in place on the patient's arrival. The patient arrived fully immobilized by EMS. Initial VS: HR 105, RR 20, B/P 112/80, 99% SpO2. The video showed great team work, leadership, and modeling of team roles. The patient had portable x-rays performed and then was promptly taken to CT.

The pelvis film showed a small metallic foreign body projecting over the right iliac bone.

CT scan revealed a bullet within the right lower anterior abdominal wall. There was a loop of colon within close proximity to the bullet and several small locules of extraperitoneal air; there was no definite colonic injury, intraperitoneal free air or free fluid seen on CT.

The patient was given IV Zosyn for antimicrobial coverage. Due to the need for continued observation and serial abdominal exams, it was decided to transfer this patient to a level I facility with PICU capabilities.

Learning Points:

Immobilization:

This patient arrived in full spinal immobilization. While immobilization is indicated for most of our trauma population, isolated penetrating injuries do not always require full spinal precautions. In penetrating injuries without a blunt mechanism, full exposure and examination of the patient is vital to determine additional wounds or injuries. C-collars in particular can easily hide penetrating injuries to the neck and upper chest – extra attention should be paid to examine these areas if a collar is in place. If there is concern for a spinal cord injury or bony fracture, please immobilize your patients. It is better to error on the side of caution for patient safety. However, in patients who are awake, alert, and neurologically intact with isolated penetrating abdominal injuries, full immobilization may not be necessary.

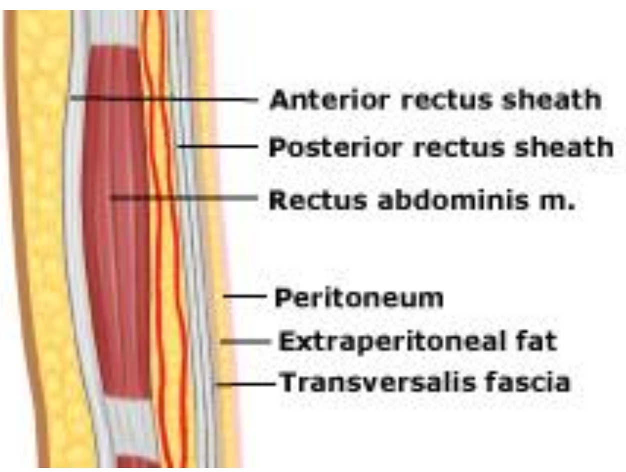
Managing Pediatric Abdominal Injuries

Penetrating trauma is less frequent in children compared to blunt trauma – but penetrating injuries account for more than 10% of admissions to most major pediatric trauma centers.

Initial assessment of these patients must include full exposure. It is not uncommon for pediatric patients with apparent isolated abdominal injuries to have other wounds found in the axilla, groin, perineum, scalp, or skin folds. It is important to roll these patients as well to evaluate for potential wounds on the back.

The key to the management of abdominal gunshot wounds is detection of peritoneal violation and injury.

The peritoneum is a single layer of serosa supported by a thin layer of connective tissue that lines the abdominal cavity.



Bibliography: *Uptodate.com. 2014. Anatomy of the abdominal wall. [online] Available at: http://www.uptodate.com/contents/anatomy-of-the-abdominal wall?source=search_result&search=abdominal+anatomy&selectedTitle=1%7E150#H15 [Accessed: 6 May 2014].*

If a patient is hemodynamically unstable or has signs of peritoneal injury, a laparotomy is required. If the patient does not have clear signs of peritoneal penetration, CT, plain film radiographs or diagnostic laparoscopy may be performed to further evaluate the wound. If no injury to the peritoneum is detected the patient can be observed for serial abdominal exams.

In this particular case, there was no definite peritoneal injury seen on CT scan - but due to close proximity of the ballistic fragment to the adjacent colon, the patient needed to be observed. Typically any patient with a high-velocity abdominal GSW should be observed for a minimum of 12-24 hours before discharge and undergo serial abdominal examinations. Due to the patient's age, a pediatric center was the appropriate place to send the child.

Bibliography: *Uptodate.com. 2014. Initial evaluation and management of abdominal gunshot wounds in adults. [online] Available at: http://www.uptodate.com/contents/initial-evaluation-and-management-of-abdominal-gunshot-wounds-in-adults?source=see_link [Accessed: 6 May 2014].*

IV Access

There was some hesitation on whether or not to initiate a second IV site on this patient. For level 1 trauma patients or those with potentially unstable injuries, obtaining blood samples and/or starting a second IV site early is encouraged. We are great about utilizing methods to lessen the pain for IV access in stable pediatric patients (and props to our nurses for being such great advocates for our pediatric population), but this should not delay early resuscitative care.

Case 2: Bicycle Crash

The next case we reviewed was a middle aged male who crashed his road bike going approximately 30-40 mph. The ground crew initiated helicopter transport due to location of the accident and severity of the injury. The patient arrived as a level II trauma alert. VS on arrival: HR 70, B/P 130/88, RR 16, SpO2 100% on NRB. He had received 0.5mg of Ativan during his flight to LGH due to combativeness – his pre-hospital GCS was 10. On arrival to the trauma bay the patient was restless and combative and a decision was made to intubate him due to declining GCS. The trauma team upgraded the trauma to a level I alert.

The patient's CT scan showed several significant intracranial injuries (see the caption below).

Figure 1 & 2: Bifrontal intraparenchymal hemorrhages were seen on the top image. The bottom image shows a comminuted occipital bone fracture that extended down to both occipital condyles.

Neurosurgery was consulted and the patient was admitted to the intensive care unit. He had an ICP monitor and Licox placed for his traumatic brain injury. A follow up CT scan the following morning showed worsening progression of his hemorrhages.

A left subclavian cooling catheter and an arterial line were placed for further monitoring and thermoregulation. Over the next couple days his ICPs continued to worsen. Paralytics, hypertonic saline and pentobarbital infusion were initiated. Despite continued aggressive measures his status worsened and he developed ARDS with shock– and he ultimately expired from his injuries.

Learning Points

Management of TBI

(The following information is from Up to Date)

Pre-Hospital: The primary goal of pre-hospital management for severe head injuries is to prevent hypotension and hypoxia, two systemic insults known to be major causes of secondary injury after TBI. In a meta-analysis of clinical trials and population-based studies, hypoxia (PaO2 <60 mmHg) and hypotension (systolic BP <90 mmHg) were present in 50 and 30 percent of patients, respectively, and were each associated with a higher likelihood of a poor neurologic outcome.

Emergency Department: In the early hospital setting of patients with severe head injury, diagnostic assessment and treatment is done according to the ATLS (Advanced Trauma Life Support) protocol:

- Hemodynamics (HR, BP), respiratory status (including pulse oximetry and capnography) and temperature should all be closely monitored
- Adequate oxygenation (PaO2 >60 mmHg) and blood pressure support (systolic BP >90 mmHg) are paramount to prevent secondary injury.
- A neurologic examination should be completed as soon as possible to determine the clinical severity of the TBI. A GCS score of 8 or lower is considered a severe TBI. Deterioration is common in the initial hours after the injury.
- Coagulopathy is common in patients with severe TBI. If the INR is elevated, efforts to reverse the coagulopathy should begin immediately.

Efforts to evaluate and manage increased intracranial pressure (ICP) should begin in the emergency department. Patients with severe TBI (GCS ≤8) and clinical symptoms suggesting possible impending herniation from elevated ICP (unilaterally or bilaterally fixed and dilated pupil(s), decorticate or decerebrate posturing, bradycardia, hypertension, and/or respiratory depression) should be treated urgently, with head elevation and *possible* osmotic therapy (mannitol 1 g/kg iv) concurrently with neuroimaging and other assessments.

Initial Treatment and ICP Monitoring

Several approaches are used in the intensive care setting to prevent and treat elevated ICP. Simple techniques should be started as soon as possible:

- Head of bed elevation to 30 degrees
- Optimization of venous drainage: keeping the neck in neutral position, loosening neck braces if too tight
- Monitoring central venous pressure and avoiding excessive hypervolemia

Indications for ICP monitoring in TBI are a GCS score ≤8 and an abnormal CT scan showing evidence of mass effect from lesions such as hematomas, contusions, or swelling

Most guidelines and clinical protocols recommend that treatment for elevated ICP should be initiated when the ICP rises above 20 mmHg. Ventricular drainage, osmotic therapy, additional sedation and/or ventilatory changes are all possible therapeutic options. In refractory cases, barbiturate coma, induced hypothermia, and decompressive craniectomy may be considered.

Osmotic Therapy

The intravascular injection of hyperosmolar agents (such as mannitol or hypertonic saline) creates an osmolar gradient, drawing water across the blood-brain barrier. This leads to a decrease in interstitial volume and a decrease in ICP.

- Mannitol is administered in boluses of 0.25 to 1 g/kg every four to six hours as needed. Monitoring of serum osmolality (maintained <320 mMol/L), fluid balance, renal function, and electrolytes is required
- Hypertonic saline is being used increasingly in this setting, but with varying volumes and tonicity (3 to 23.4 percent)

Hyperventilation

Most patients with severe TBI are sedated and mechanically ventilated during the first several days. Regarding ICP management, control of ventilation helps prevent increases in intrathoracic pressure that may elevate central venous pressures and impair cerebral venous drainage.

Hyperventilation can be used to reduce ICP. With hyperventilation, $PaCO_2$ decreases thereby leading to cerebral vasoconstriction, which then results in decreased cerebral blood volume and ICP. However, hyperventilation-induced vasoconstriction may also cause secondary ischemia and thereby worsen outcomes. Hyperventilation can also increase extracellular lactate and glutamate levels that may contribute to secondary brain injury

Based upon these competing concerns, guidelines recommend avoiding hyperventilation, especially in the acute phase (the first 24 to 48 hours) following TBI. Mild to moderate hyperventilation can be considered at later stages, but a $PaCO_2$ of less than 30 mmHg should be avoided.

Sedation

Sedative medications and pharmacological paralysis are often used in patients with severe head injury and elevated ICP. The rationale is that appropriate sedation may lower ICP by reducing metabolic demand. These possible beneficial effects are counterbalanced by the potential for these drugs to cause hypotension and cerebral vasodilation that in turn may aggravate cerebral hypoperfusion and elevate ICP.

While there are a number of agents that can be used, propofol is frequently used because of its short duration of action, which allows intermittent clinical neurologic assessment. Anecdotally, propofol sedation can produce ICP reductions.

Barbiturate coma has been used traditionally in this setting. However, there is little clinical data to support its use. Pentobarbital remains a treatment option for elevated ICP refractory to other therapies. A loading dose of 5 to 20 mg/kg is given as a bolus, followed by 1 to 4 mg/kg per hour. Continuous EEG monitoring is used, with the pentobarbital infusion titrated to produce a burst-suppression pattern.

Temperature Management

Fever worsens outcome after stroke and probably severe head injury, presumably by aggravating secondary brain injury. Current approaches emphasize maintaining normothermia through the use of antipyretic medications, surface cooling devices, or even endovascular temperature management catheters. However, this approach has not been systematically tested with regard to clinical outcome. Similarly noninduced hypothermia has been associated with an increase in mortality after TBI but the efficacy of efforts to avoid this complication have not been evaluated.

Induced Hypothermia

Induced hypothermia has been a proposed treatment for TBI based upon its potential to reduce ICP as well as to provide neuroprotection and prevent secondary brain injury. Induced hypothermia has been shown to be effective in improving neurologic outcome after ventricular fibrillation cardiac arrest.

Two trials of hypothermia therapy in children with TBI have shown no improvement in neurologic or other outcomes; one showed a nonsignificant increase in mortality.

Given the uncertainties surrounding its appropriate use, therapeutic hypothermia treatment should be limited to clinical trials, or to patients with elevated ICP refractory to other therapies.

Bibliography: *Uptodate.com. 2014. Management of acute severe traumatic brain injury. [online] Available at: http://www.uptodate.com/contents/management-of-acute-severe-traumatic-brain-injury?source=search_result&search=ICP&selectedTitle=2%7E150 [Accessed: 7 May 2014].*