Pediatric Trauma Update

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This article reviews the evaluation and management of the pediatric trauma patient while focusing on recent updates.

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Abstract

Trauma is the leading cause of mortality in children, accounting for over 11,000 deaths and more than 8 million nonfatal injuries in 2015 for ages 1-19 years.[1] Current issues garnering particular attention and research efforts include traumatic brain injury (TBI), blunt solid organ injuries, imaging guidelines and trauma-induced coagulopathy. This article reviews the evaluation and management of the pediatric trauma patient while focusing on recent updates.

Traumatic Brain Injury

Head injuries are the most common fatal injury in children and account for approximately 435,000 emergency department visits each year.[2] Compared with adults, children have a number of anatomical differences that may predispose them to injury including thinner cranial bones, a larger head-to-torso ratio, an immature immune system and differences in thermoregulation.[3] After evaluating the airway, breathing, circulation and disability of the injured child, evaluation for TBI should include a complete neurologic exam including a Glasgow Coma Score (GCS), pupil size, inspection of the head and spine, including the anterior fontanel, for gross deformity along with funduscopic and otoscopic evaluation. The pediatric GCS is modified for age-appropriate verbal development and should be used for children under two years of age. Children over two years should be evaluated with the standard GCS measurement.[4] (Table 1.)

Neuroimaging

Imaging of head injuries is an important focus of research as we are learning more about the effects of radiation exposure in children. A study by Rice et al. reported one lethal case of cancer for every 1,000 computed tomography (CT) scans of a child.[5] The CT scan is the preferred imaging modality for moderate to severe head injuries as it quickly provides detailed images that can guide management. However, for minor head trauma, fewer than 10% of children sustain a TBI. This has led to the creation of several clinical decision tools for neuroimaging, including CATCH, PECARN and CHALICE, to reduce unnecessary radiation exposure.[3] The CATCH study identified high-risk individuals who require CT scan as those who fail to reach GCS of 15 within two hours of injury, suspicion of open skull fracture, worsening headache and irritability, while moderate-risk individuals had a large scalp hematoma, signs of basilar skull fracture or a dangerous mechanism of injury. MRI and transcranial Doppler (TCD) are two other valuable modalities that are frequently used. TCD was shown to have high sensitivity for detecting intracranial hemorrhage (ICH) and abnormal cerebral perfusion pressure (CPP) after severe TBI in children.[6]

Table 1. Glasgow Coma Score

Pediatric GCS[4]	
Eye Opening	
Spontaneous	4
Speech	3
Pain	2
None	1
Verbal Response	
Coos, Babbles	5
Irritable cries	4
Cries to pain	3
Moans to pain	2
None	1
Motor Response	
Normal spontaneous movement	6
Withdraws to touch	5
Withdraws to pain	4
Abnormal flexion	3
Abnormal extension	2
None	1

Interventions

Walker and colleagues published an excellent update on the management of pediatric TBI in 2017 that highlighted a few modern interventions including controlled hypothermia and hypertonic saline (HTS) infusion. Controlled hypothermia has been shown to decrease mortality and improve neuronal function, presumably by decreasing neuronal metabolism. Current literature supports initiation within 24 hours of trauma and lasting up to 48 hours. HTS has been shown to decrease intracranial pressure (ICP) thereby increasing cerebral blood flow and providing neuroprotection. This mechanism is similar to that of mannitol; however, in a rat study, HTS was better at decreasing ICP and neurotoxicity than mannitol. A dosing range of 3 to 5 mL/kg is commonly used, but future randomized prospective trials with mannitol are needed before it is accepted as standard of care.[7]

Blunt Solid Organ Injury

Intraabdominal injury (IAI) is the third leading cause of pediatric trauma mortality with the majority of injuries sustained by a blunt mechanism (85%) versus penetrating (15%).[8] Motor vehicle crashes (MVC) are the major cause of blunt abdominal trauma, and a common physical exam finding that has been associated with IAI is the "seat belt sign" (SBS), which has been defined as an area of ecchymoses, erythema or abrasions sustained as a result of seat belt use. However, Chidester and colleagues did not find SBS to be significantly associated with IAI in a retrospective review of 331 children involved in an MVC. Fifty-four (16%) of the 331 children had SBS, but only 12 (22%) of those children had an IAI identified by CT scan or with an operation. Of the three children who required an operation, two had bowel injuries and one had a negative laparoscopy. They found the SBS to have a sensitivity of 25% and a specificity of 85%, concluding that SBS may not be as predictive of IAI as historically reported in adults.[9]

Notricia and Linnaus published an excellent update on the nonoperative management of blunt solid organ injury (SOI) in pediatric surgery.[10] Solid organ injury includes the spleen, liver and kidneys; is more common than hollow organ injury and is most often treated nonoperatively. In the 1990's, the increasing utilization of the CT scan led to the development of organ injury scales (OIS) by the American Association for the Surgery of Trauma (AAST) as a means to guide SOI management. However, in recent years, studies have found hemodynamic status to be a better guide of management than OIS. For those with hemodynamic instability, attempts should first be made to restore adequate perfusion with isotonic fluid followed by blood products. If unsuccessful, a massive transfusion protocol should be considered along with operative exploration or interventional angiography. The current recommendation for maximum transfusion volume is 40 mL/kg based on a retrospective study of children injured in a combat zone. Children who respond to early resuscitation should be evaluated with CT scan and monitored for further hemodynamic instability. Patients admitted to the ICU may be transferred to a lower level of care after 24 hours with a stable hemoglobin and may be discharged the following day if the child is tolerating oral intake with minimal abdominal pain and stable vital signs. This recommendation replaces the initial guidelines of OIS grade plus one day for the length of hospitalization based on recent data demonstrating very low rebleeding rates in hemodynamically stable patients with a median time to rebleeding of 10 days.[11]

Blunt Liver and Splenic Injury

Nonoperative management (NOM) is the standard for blunt liver and splenic injury with a retrospective study by Holmes and colleagues showing a NOM failure rate of 3% for isolated liver injuries and 4% for isolated spleen injuries. Though rare, it is important to recognize failure of NOM quickly. Identified risk factors for NOM failure

include higher ISS, higher organ injury grade, multiple organ injuries and lower GCS on presentation. In two separate studies, the main cause of failure was hemorrhage. Holmes and colleagues found the median time of failure to be 2 hours with 76% of failures occurring within 12 hours of injury. Children with contrast extravasation from splenic injury are often treated successfully with nonoperative management but may benefit from angioembolization if conservative treatment fails. Angioembolization is rare for liver injury as very few of these injuries demonstrate contrast extravasation.[11]

Blunt Renal Injury

Most blunt renal injuries are successfully managed with conservative treatment, which may include percutaneous drainage, endourologic stenting and angioembolization. However, there is no consensus on the management of high-grade injuries. A systematic review by LeeVan and colleagues reported a success rate of 80% to 100% with nonoperative management for grades IV and V.[12] Therefore, it is recommended for all patients with renal trauma to initially be treated using a conservative protocol, while immediate surgical intervention is reserved for those with hemodynamic instability. LeeVan and colleagues also found minimal evidence to support ICU care for high-grade renal injury patients, urinary catheter placement, prophylactic antibiotics or mandatory bed rest. They recommend using ultrasound for assessment of injury progression as opposed to routine CT imaging. In cases of urinomas, two thirds of these will resolve spontaneously, and intervention should be reserved for large (greater than 4 cm), symptomatic or persisting urinomas.[11] The initial intervention should be ureteral stenting as isolated percutaneous drainage alone has repeatedly failed in several studies. For renal artery pseudoaneurysms and renal hemorrhage, angioembolization has been found to be a great tool in the conservative approach. Finally, discharge can be made on the basis of symptomatic control and stability as opposed to the commonly used criteria of resolution of gross hematuria.[12]

Pancreatic and Duodenal Injuries

Although solid organ injuries rarely necessitate an operation, pancreatic and duodenal injuries are more likely to require operative intervention. In a multicenter retrospective study of 1,823 patients with blunt abdominal trauma, Mattix and colleagues identified 173 (9.5%) patients who sustained pancreatic injury, of which 43 (26%) required operative intervention. The pancreas was cited as the cause of operation in 57.8% of all patients. This study found pancreatic ductal injury, defined as grades III to V, to be a predictor of nonoperative management failure with 23 of the 53 patients sustaining a ductal injury requiring an operation. Importantly, those with ductal injuries who were taken to the OR within 24 hours of injury experienced reduced rates of complications. Consequently, early identification of ductal injury by CT, MRI, or ERCP (endoscopic retrograde pancreatography) is critical. ERCP is an appropriate alternative diagnostic and potentially therapeutic study if CT imaging is equivocal.[13] Blunt duodenal injuries are rare and often occur with other abdominal injuries, most commonly the pancreas.[14] Their management is based on the hemodynamic stability of the patient with options of damage control and delayed repair, drainage or immediate definitive repair. The utility of duodenal diversion has been questioned in several studies and may be useful in a few specific situations. Antegrade and retrograde tube duodenostomies might be beneficial in cases requiring tenuous repair with mild contamination. There is no Level 1 evidence to support the routine use or nonuse of peri-duodenal drains. Moreover, Malholtra et al. do not recommend drains for Grade I or II injuries, rather they place them in the setting of a tenuous repair requiring a protective maneuver like a pyloric exclusion. Although uncommon, duodenal injuries can be a significant source of morbidity leading to fistula formation and obstruction and are often associated with multiple organ injuries which contribute to the overall outcome.[15]

Radiation Exposure and Imaging Guidelines

Imaging studies have an increasingly important role in the evaluation and management of trauma patients. CT scan utilization has increased from 3 million scans per year in the early 1980s to over 60 million in 2006. However, the radiation exposure produced by CT scans is not trivial, especially in the pediatric population. A retrospective review by Brunetti and colleagues calculated the total effective dose of radiation received by 719 injured children who underwent 4,603 radiographic studies (67.3% x-rays, 31.7% CT scans). The mean dose for all children was 12.8 mSv $\pm$ 12.0 mSv. For comparison, the average ionizing radiation exposure a person receives each year from the environment is 3 mSv.[16] While CT scans only accounted for 31.7% of the total radiographic studies, they produced 91% of the total radiation dose. One study found that a total radiation exposure dose of 50 to 60 mGy to the head and bone marrow resulted in a threefold-increased risk for brain tumors and leukemia, respectively.[17] For perspective, two to three head CT scans from a current scanner on

typical settings would provide a dose of 50 to 60 mGy to the brain.[18] Unsurprisingly, guidelines are being developed to limit the unnecessary radiation exposure. The Pediatric Trauma Society supports the Level 2 recommendations by Rozzelle et al. for clearing children of the need for cervical spine imaging. They report cervical spine imaging is not recommended for injured children greater than 3 years of age who are alert, have no neurological deficit, have no midline cervical tenderness, have no painful distracting injury, do not have unexplained hypotension and are not intoxicated. Children under 3 years of age must meet the above criteria as well as a GCS greater than 13 and were not involved in an MVC, fall greater than 10 feet or non-accidental trauma.[19] Children who have experienced trauma and do not meet either set of criteria should have cervical spine radiographs or high resolution CT scan.

Trauma-Induced Coagulopathy

Trauma-induced coagulopathy (TIC) is a complex process resulting from tissue injury and ischemia that is further exacerbated by dilution, acidosis and hypothermia. It is well known that children have different hemostatic systems that mature with age. The majority of trauma patients become hypercoagulable and can benefit from coagulation monitoring devices. In addition to conventional coagulation tests (CCT), coagulation screens including thromboelastography (TEG®) rotational thromboelastometry (RoTEM®) and impedance aggregometry are being implemented in the acute evaluation of the injured child and are necessary to better inform clinicians on TIC. These devices monitor the viscoelastic properties of whole blood and the endothelium along with platelet function testing. TEG® and RoTEM® are able to evaluate all of the stages of clotting.

An important mechanism associated with TIC in children is TBI, which may have a different pathophysiology than TIC of hemorrhage. A study by Vavilala and colleagues reported fibrin degradation product levels over 1000 μg/mL on admission predicted a poor outcome in children less than 16 years with an isolated brain injury and GCS between 7-12.[20] A retrospective 10-year review of 803 children after severe trauma with an INR of 1.2 or greater at presentation was an independent predictor of mortality, especially in those with TBI.

Treatment strategies for TIC

Early identification of coagulopathy with the screens mentioned above facilitate a targeted approach to blood product resuscitation. Christiaans and colleagues published an excellent review on coagulopathy after severe pediatric trauma focusing on novel hemostatic adjuncts.[21] Massive blood transfusion has evolved in recent years to include the early and liberal use of blood products, often with a goal of a 1:1:1 ratio of PRBC to FFP to platelets for severely injured patients. However, massive transfusion occurs less commonly in children than adults in large part due to their greater physiological reserve and tolerance to blood loss. Although a definition for massive transfusion in children is difficult due to varying body volumes by age, gender and weight, a commonly cited definition is 40ml/kg. Interestingly, in a single center study of 55 children by Chidester and colleagues, mortality was not significantly different between children who received massive transfusion protocol and children who were given blood at the physician's discretion.[22] Randomized prospective trials are needed to better evaluate the role of massive transfusion, optimal blood product ratios and blood component therapies in pediatric trauma patients.[21]

FFP (Fresh Frozen Plasma) is the most common blood component used to treat coagulopathy, but it is associated with several risks including transfusion-related acute lung injury (TRALI), transfusion associated circulatory overload (TACO) and adverse immunological reactions. It is recommended that FFP be used only in cases of active bleeding and not for abnormal coagulation screens alone. A retrospective study of patients receiving FFP alone and patients receiving coagulation factor concentrates without FFP showed the patients receiving FFP alone had an increased frequency of multi-organ failure.[21]

Recombinant Factor VIIa (rFVIIa) has gained support for off-label use in pediatric trauma patients in addition to its use in patients with hemophilia and acquired inhibitors. In one case series of 135 pediatric patients who received rFVIIa for off-label use, there was a decrease in the 24-hour median transfusion volume after receiving rFVIIa, and the mortality rate was significantly lower in surgical/trauma patients (16%) versus medical patients (58%). Dosing for rFVIIa has been extrapolated from adult literature and the pediatric hemophiliac population with bolus doses ranging from 40-100 μg/kg in the non-hemophiliac population and repeat dosing intervals reported from two to six hours. An analysis of the Western Trauma Association web-based registry suggests that rFVIIa may be most beneficial when given early in the setting of hemorrhage as acidosis (pH $<$ 7.2), thrombocytopenia (platelets $<$100,000) and hypotension (systolic $\leq$ 90) were indicators of poor response to rFVIIa.[21]

Prothrombin complex concentrate (PCC) is another blood product garnering attention for treatment of TIC, although very few studies exist for the pediatric trauma population as it was developed for treatment of hemophilia B and consequently also known as factor IX complex. PCC contains 25-30 times the concentration of clotting factors as FFP and can be a three or four factor concentrate. Three factor PCC contains factors II, IX and X while four factor PCC also includes factor VII. PCC has Level 2C evidence for its use in patients with massive bleeding who also receive FFP. Several guidelines and recommendations provide a recommended dosing range of 20-50 units/kg with a risk of thromboembolism at doses greater than 50 units/kg.

Tranexamic acid (TXA) acts to prevent the breakdown of a fibrin clot by competitively inhibiting tissue plasminogen activator (tPA), which converts plasminogen to plasmin. Plasmin then degrades fibrin, which is responsible for the clot stability. TXA has been well studied in the adult population and even the non-traumatic pediatric population.[23] The Clinical Randomization of an Antifibrinolytic in Significant Hemorrhage (CRASH-2) study demonstrated a significant decrease in mortality from bleeding when TXA was given early after trauma. Beno and colleagues feel that TXA should be considered for adolescent trauma patients with the same indications and dosing regimen as adults.[23] They also suggest younger children with hemodynamic instability and ongoing risk of hemorrhage may also benefit from TXA with recognition that evidence in this population is lacking. Dosing recommendations for children over 12 years of age is the same as the adult protocol which is a 1-gram loading dose over 10 minutes within 3 hours of injury followed by a 1-gram infusion over 8 hours. For children 12 years and younger, the dosing recommendation is a loading dose of 15 mg/kg (max 1g) over 10 minutes followed by 2 mg/kg/h for at least 8 hours or until bleeding stops.[23]

Conclusion

While many lessons learned from the management of adult trauma patients can be applied in the pediatric population, due diligence must be practiced in the areas of TBI, blunt solid organ injury, radiographic imaging and traumatic coagulopathy in the context of age-specific research that is briefly summarized here. Randomized prospective trials are needed to better evaluate current methods of treatment and produce pediatric-specific guidelines.

References

1. Fatal Injury Data | WISQARS | Injury Center | CDC. https://www.cdc.gov/injury/wisqars/fatal.html. Accessed January 30, 2018.
2. Walker PA, Harting MT, Baumgartner JE, Fletcher S, Strobel N, Cox CS. Modern Approaches to Pediatric Brain Injury Therapy. doi:10.1097/TA.0b013e3181ad323a
3. Alexiou G a, Sfakianos G, Prodromou N. Pediatric head trauma. J emergencies, trauma Shock. 2011;4(3):403-408. doi:10.4103/0974-2700.83872
4. Badjatia N, Carney N, Crocco TJ, et al. Guidelines for prehospital management of traumatic brain injury 2nd edition. Prehosp Emerg Care. 2008;12 Suppl 1:S1-S52. doi:10.1080/10903120701732052
5. Rice HE, Frush DP, Farmer D, Waldhausen JH. Review of radiation risks from computed tomography: essentials for the pediatric surgeon. J Pediatr Surg. 2007;42(4):603-607. doi:10.1016/j.jpedsurg.2006.12.009
6. Amyot F, Arciniegas DB, Brazaitis MP, et al. A Review of the Effectiveness of Neuroimaging Modalities for the Detection of Traumatic Brain Injury. J Neurotrauma. 2015. doi:10.1089/neu.2013.3306
7. Brenkert T, Estrada C, McMorrow S, Abramo T. Intravenous Hypertonic Saline Use in the Pediatric Emergency Department. Pediatr Emerg Care. 2013;29(1):71-73. doi:10.1097/PEC.0b013e31827b54c3
8. Drexel S, Azarow K, Jafri MA. Abdominal Trauma Evaluation for the Pediatric Surgeon. Surg Clin North Am. 2017;97(1):59-74. doi:10.1016/j.suc.2016.08.004
9. Chidester S, Rana A, Lowell W, Hayes J, Groner J. Is the "seat belt sign" associated with serious abdominal injuries in pediatric trauma? J Trauma. 2009;67(1 Suppl):S34-6. doi:10.1097/TA.0b013e3181a93630
10. Notrica DM, Linnaus ME. Nonoperative Management of Blunt Solid Organ Injury in Pediatric Surgery. Surg Clin North Am. 2017. doi:10.1016/j.suc.2016.08.001
11. Notrica DM, Linnaus ME. Nonoperative Management of Blunt Solid Organ Injury in Pediatric Surgery. Surg Clin North Am. 2017;97(1):1-20. doi:10.1016/j.suc.2016.08.001
12. LeeVan E, Zmora O, Cazzulino F, Burke R V., Zagory J, Upperman JS. Management of pediatric blunt renal trauma: A systematic review. J Trauma Acute Care Surg. 2016;80(3):519-528. doi:10.1097/TA.0000000000000950
13. Mattix KD, Tataria M, Holmes J, et al. Pediatric pancreatic trauma: predictors of nonoperative management failure and associated outcomes. J Pediatr Surg. 2007;42(2):340-344. doi:10.1016/j.jpedsurg.2006.10.006
14. Siboni S, Benjamin E, Haltmeier T, Inaba K, Demetriades D. Isolated blunt duodenal trauma: Simple repair, low mortality. Am Surg. 2015;81(10):961-964.
15. Malhotra A, Biffl WL, Moore EE, et al. Western Trauma Association Critical Decisions in Trauma: Diagnosis and management of duodenal injuries. J Trauma Acute Care Surg. 2015;79(6):1096-1101. doi:10.1097/TA.0000000000000870
16. Brunetti M a, Mahesh M, Nabaweesi R, Locke P, Ziegfeld S, Brown R. Diagnostic radiation exposure in pediatric trauma patients. J Trauma. 2011;70(2):E24-E28. doi:10.1097/TA.0b013e3181e80d8d
17. Pearce MS, Salotti JA, Little MP, et al. Radiation exposure from CT scans in childhood and subsequent risk of leukaemia and brain tumours: a retrospective cohort study. Lancet (London, England). 2012;380(9840):499-505. doi:10.1016/S0140-6736(12)60815-0
18. Radiation Risks and Pediatric Computed Tomography - National Cancer Institute. https://www.cancer.gov/about-cancer/causes-prevention/risk/radiation/pediatric-ct-scans. Accessed March 10, 2018.
19. Rozzelle CJ, Aarabi B, Dhall SS, et al. Management of pediatric cervical spine and spinal cord injuries. Neurosurgery. 2013;72(SUPPL.2):205-226. doi:10.1227/NEU.0b013e318277096c
20. Vavilala MS, Dunbar PJ, Rivara FP, Lam AM. Coagulopathy predicts poor outcome following head injury in children less than 16 years of age. J Neurosurg Anesth. 2001;13(1):13-18. doi:10.1097/00008506-200101000-00003
21. Christiaans SC, Duhachek-Stapelman AL, Russell RT, Lisco SJ, Kerby JD, Pittet J-F. Coagulopathy after severe pediatric trauma. Shock. 2014;41(6):476-490. doi:10.1097/SHK.0000000000000151
22. Chidester SJ, Williams N, Wang W, Groner JI. A pediatric massive transfusion protocol. doi:10.1097/TA.0b013e318265d267
23. S. B, A.D. A, J. C, S. R. Tranexamic acid in pediatric trauma: Why not? Crit Care. 2014;18(4):1-5. doi:10.1186/cc13965

Disclosure

None reported.

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