Pediatric Abusive Head Trauma: A Review for Trauma Providers

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Abstract

Pediatric abusive head trauma (AHT), formally known as shaken baby syndrome, is the inflicted injury to a young child or infant through sudden forceful impact, violent shaking or a combination of these two, resulting in injury to the skull and intracranial contents. AHT involves a varying spectrum of cranial, cerebral and spinal damage. The majority of child abuse associated fatalities are consequent of AHT. Despite a high clinical suspicion, diagnosis can be challenging and often relies on a combination of both clinical and radiographic features specific to child abuse. Clinical findings of subdural and retinal hemorrhages are most common and hold a high positive predictive value for AHT. AHT is associated with high rate of morbidity and mortality when compared to other accidental pediatric traumas. Survivors of AHT are often left with long-term neurologic and developmental disabilities. This article aims to characterize the current prevalence of AHT, define associated risk factors, highlight mechanisms of injury and identify predictive clinical features that can aide in a timely accurate diagnosis. Furthermore, this review of AHT emphasizes the importance of early recognition and the initiation of evidenced based management of pediatric TBI in AHT in an effort to reduce poor long-term neurologic outcomes among these children.

ABBREVIATIONS

AHT: Abusive Head Trauma; TBI: Traumatic Brain Injury; ICH: Intracranial Hemorrhage; ICP: Intracranial Pressure; AAP: American Academy of Pediatrics; SBS: Shaken Baby Syndrome; CT: Computed Tomography; MRI: Magnetic Resonance Imaging; GCS: Glasgow Coma Scale; CPR: Cardiopulmonary Resuscitation; CPP: Cerebral Perfusion Pressure

INTRODUCTION

Pediatric AHT, formerly recognized as Shaken Baby Syndrome, is an inflicted injury from physical abuse to a child or infant that results in injury to the skull and intracranial contents. A spectrum of cerebral, spinal and cranial damage can occur [1]. AHT is the most common cause of traumatic death in children under the age of 1 year and accounts for most child abuse related fatalities [1,2]. Although the precise mechanisms of injury in pediatric AHT remains incompletely understood, it is believed to be caused by a combination of mechanisms including blunt impact, inertial (acceleration/deceleration) and hypoxic-ischemic [1,3].

The American Academy of Pediatrics (AAP) now endorses the diagnosis of AHT, as a less mechanistic terminology, to more clearly describe this specific type of pediatric TBI, which accounts for both the primary and secondary causes of injury not solely the mechanism of shaking [1]. The AAP does however continue to support the use of the term SBS for parental education and community preventative efforts to caution against the detrimental effects of infant shaking.

A diagnosis of AHT requires a high level of clinical suspicion accompanied by a meticulous clinical evaluation to avoid misdiagnosis and facilitate early intervention to improve outcomes. AHT compared to non-abusive head trauma is associated with worse outcomes, greater utilization of medical services and higher health care cost [2,4,5].

EPIDEMIOLOGY

The estimated incidence of pediatric AHT is 14-40 cases per 100,000 children under the age of 1 year although this may be an underestimation due to a high rate of misdiagnosis on initial presentation and does not account for those who are not brought to medical care [2,6,7]. A recent Centers for Disease Control and Prevention Report, noted a national AHT associated fatality rate of 0.68-0.88 per 100,000 children [8]. In the U.S between 2009-2014, 2,250 deaths in children less than 5 years were attributed to AHT however there was a reported 13% average annual decline in mortality secondary to AHT during that time [8]. This statistically significant decline in AHT associated mortality may be related to the dramatic increase in AHT incidence seen in the 3 years prior (2007-2009) during a time of a US economic recession. Despite this fluctuation in incidence, conflicting data exists in the literature in regards to the potential economic and financial impact on rate of AHT [8,9]. A retrospective study by Wood et al. (2016), showed no association between the rate of AHT and employment growth, mortgage delinquency or foreclosure rates [9]. Conversely, a study conducted by Nuno et al. (2015), evaluating socioeconomic factors associated with...
AHT, found a higher in come was associated with risk reduction in AHT related mortality [10]. Despite these findings, the literature supports that AHT occurs among all socioeconomic classes, cultures and educational backgrounds.

A review of the literature, showed risk factors for AHT included children who reside in households with unrelated adults, male gender caregiver (father, stepfather, mother’s boyfriend), children born to young mothers (<21 years), children who were product of multiples birth, age greater than 1 month but less than 2 years and male gender [5,9-12,13]. Infants have a higher incidence of AHT compared to children in the second year of life with a peak incidence at 2 months of age [5,10,12]. Onset of early crying also appears to be a trigger for AHT [14]. The highest reported mortality is among children aged 12-23 months; possibly related to smaller anterior fontanelle, closing cranial suture lines and thus more vulnerability to the effects of intracranial hypertension [10].

Survivors of AHT can have long-lasting and severe physical, neurologic and behavioral impairments leading to substantial excess health care cost and utilization of medical services for years after trauma [3,7]. Among children with AHT who present to medical care 75% present to an emergency department, 17% present to a primary care provider and half of those requiring admission necessitate intensive level care [3]. A recent study, demonstrated the estimated total medical cost for AHT in the 4 years after diagnosis was $47,952 per child [6].

PATHOPHYSIOLOGY

Primary injury in AHT, occurring at the time of abuse, results from direct or indirect mechanical forces to the brain parenchyma and surrounding vascular structures leading to structural failure and neurologic dysfunction [15]. Primary injury can be focal, such as with brain contusions, lacerations or intracranial hemorrhage, or diffuse in nature as seen with diffuse axonal injury or concussive syndromes [15,16]. A complex cascade of neural and vascular reactions ensues contributing to the clinical manifestations and long-term sequelae of AHT [16]. Following primary injury, there are increased cerebral metabolic demands, failure of cerebral auto-regulation, release of excitatory neurotransmitters, decreased clearance of metabolic by products and neural cellular energy failure [15-19]. During secondary injury, compromised perfusion to surviving brain tissue leads to ischemia and uncontrolled cerebral edema can progress to cerebral herniation syndrome with brain stem compression [19,20].

Infants are anatomically more susceptible to primary and secondary brain injury as a result of AHT. Developmentally, infants exhibit poor head control, weak neck musculature and relatively large head in proportion to body size, which makes them particularly vulnerable to acceleration/deceleration injuries of the brain and spinal cord [16,21,22]. Structurally, the immature infant skull sutures are poorly calcified allowing for more intense brain tissue displacement, increased shear stress and increased transfer of forces to brain parenchyma as a result of blunt impact [16].

Increased risk of secondary brain injury in infants occurs due to higher incidence of cerebral edema, impaired cerebral auto-regulation and altered neurotransmission. Age dependent differences in extracellular fluid space and aquaporin expression may contribute to disturbed brain water homeostasis and thus cerebral swelling [16,19,20]. Furthermore, following a primary brain injury the immature brain is more susceptible to oxidative stress secondary to compromised antioxidant defenses with immature scavenging enzymes leading to free radical mediated lipid peroxidation and supraphysiologic levels of excitatory amino acids contribute to excitotoxic brain injury [16,20].

CLINICAL & RADIOGRAPHIC MANIFESTATIONS

Infants and children with AHT can present with a wide range of clinical symptoms from subtle nonspecific symptoms to overt neurologic deficits. The severity of brain injury will largely dictate the clinical presentation and magnitude of symptoms. There is no single lesion diagnostic of physical child abuse thus a high clinical suspicion paired with a meticulous evaluation of history and physical examination can serve as clues to abusive trauma [3,5,22,23]. AHT should be considered in the differential diagnosis for all children under the age of 2 years who present with head trauma. Approximately one third of children with AHT have a missed diagnosis on initial presentation to medical care [3]. Misdiagnosis results in an increased risk of repeated abuse, medical complications from delayed diagnosis and fatal AHT [23,24]. A recognized challenge of AHT diagnosis is that the victim in often nonverbal and the history provided is often inaccurate or incomplete. Clinical predictive tools to identify children with AHT have been established and validated in the literature [25-26]. The Pediatric Brain Injury Research Network created a Clinical Prediction Rule (CPR) recommending abuse evaluation for high-risk children that meet 1 or more of the established predictors (Figure 1) [25]. Hymel et al. (2014), conducted a retrospective study to validate this 4 variable CPR which demonstrated theoretical use of the screening tool could increase detection of AHT from 87-96% and increase overall diagnostic yield of abusive evaluation in the PICU settings from 49-56% [25,27]. Another diagnostic tool is the Pittsburgh Infant

4-Variable AHT Clinical Predictive Rule

- Any clinically significant respiratory compromise at the scene of injury, during transport, in the emergency department, or before admission
- Any bruising involving the child’s ears, neck, or torso
- Any subdural hemorrhages or fluid collections that are bilateral or involve the interhemispheric space
- Any skull fractures other than an isolated, unilateral, nondiastatic, linear, parietal skull fracture

Figure 1 Pediatric Brain Injury Research Network Clinical Predictive Rule ‘Less than 3 years of age, not injured in a collision involving a motor vehicle. Acutely head-injured patient with definitive radiologic evidence of preexisting brain malformation, disease, infection, or hypoxia-ischemia were also excluded from analysis. Defined as infrequent or labored respirations, apnea, or any need for intubation or assisted ventilation. Including the patient’s chest, abdomen, genitourinary region, back, or buttocks – 1 or more of the 4 predictor variables warrants evaluation for abuse [20].
Brain Injury Score for AHT, which identifies at risk infants of abuse who would benefit from neuro-imaging to evaluate for inflicted brain injury (Figure 2) [26]. When evaluating a child with suspected abuse it is critical to consult a subspecialist in the field of child abuse to ensure that a complete medical evaluation and investigation is performed. In addition, a multidisciplinary approach to diagnosis is most effective, involving sub-specialties such as pediatric neurosurgery, neurology, ophthalmology and radiology whom are familiar with the characteristic signs of abuse [3]. Mandatory reporting to child protective services should be conducted simultaneously with medical evaluation once a reasonable suspicion of abuse has been established and should not be delayed for definitive testing.

It is highly recommended that all infants with suspected physical abuse undergo cranial imaging with CT, MRI or both to evaluate for intracranial pathology. Of note, as many as 25% of children with AHT are asymptomatic upon presentation and neuro-imaging reveals occult intracranial injury in 75% of asymptomatic children with AHT [3,28]. Subdural hemorrhage is the most frequent neuro-radiologic finding occurring in 75-81% of cases [2,5,12,29]. Heterogeneous or mixed density subdural hematomas are significantly more prevalent in non-accidental trauma compared to accidental trauma [12,29,30]. Head ultrasonography is another diagnostic tool that may be useful in infants with an open anterior fontanelle to screen for extra-axial fluid collections however, is insensitive for detecting small acute subdural hemorrhages and therefore should only be used in conjunction with CT or MRI [20]. MRI has the highest sensitivity for assessing intracranial hemorrhage and should be pursued in infants and children with ongoing clinical concerns despite normal CT findings [28]. Brain MRI with diffusion-weighted imaging can reveal extra-axial fluid collections, subtle brain contusions, shear injuries, intraparenchymal abnormalities, cerebral ischemia, diffuse axonal injury and cerebral edema [14,28,29]. Diffuse axonal injury affects the cerebral white matter and is caused by non-impact rotational forces on the brain. The immature infant brain is more vulnerable to repetitive mild non-impact load conditions and consecutive rotational injuries have been show to increase the distribution and density of cerebral injury in neonatal pig models [31].

Findings of diffuse multi-layered retinal hemorrhage occur in 85% of AHT cases [32]. The presence and pattern of retinal hemorrhage has been shown to be highly predictive of AHT. Presence of bilateral retinal involvement, lesions too numerous to count, lesions that are multi-layered and extend into the ora serrata, junction between the retina and ciliary body, are highly suggestive of AHT [5,32-34]. Conversely, birth related retinal hemorrhages are often intraretinal, located in the posterior pole and resolve within the first few weeks of life [32]. Birth-related retinal hemorrhages are more common in spontaneous vaginal delivery and instrumented assisted delivery [32]. Burkart et al. (2015), retrospectively evaluated risk factors for retinal hemorrhage in suspected AHT and found that retinal hemorrhages were almost never found in the absence of intracranial hemorrhage [35]. In addition, lethargy or altered mental status on presentation, subdural hemorrhage and radiologic findings of cerebral ischemia, diffuse axonal injury, hydrocephalus, or solid organ injury were strongly associated with findings of retinal hemorrhage [32]. A full indirect ophthalmoscopic examination, performed by an ophthalmologist with experience in pediatric abuse, is recommended preferably in the first 24 hours but no greater than 72 hours of presentation [9]. Characteristics of retinal hemorrhages may help establish a time frame in which abuse occurred. Presence of pre-retinal hemorrhage with few or no intra-retinal hemorrhage suggests days to weeks since trauma whereas too numerous to count intra-retinal hemorrhage suggests trauma occurred more recently [9,34]. One study among 52 children with TBI, showed that in all but one eye intra-retinal hemorrhages resolved to none or mild within 1-2 weeks while too numerous to count intra-retinal hemorrhages were resolved within a few days [34].

A complete skeletal survey (Figure 3) is mandatory in all suspected cases of abuse in children less than 2 years of age to assess for skeletal trauma. Roughly half of all children with AHT will have associated skeletal fractures with metaphyseal (Corner or Bucket handle) fractures, long bone fractures and posterior rib fractures being the most common [20,28]. Radiographic dating of fractures based on stage of healing may also provide critical data for investigators by narrowing the time frame of abuse.

Blunt thoracoabdominal injuries may also be identified in children with AHT. Serum transaminases, amylase, lipase and urinalysis may be obtained to screen for intra-abdominal injuries. Abdominal CT imaging should be reserved for cases in which screening laboratory studies or physical exam findings suggest abdominal injury [19,24].

Cutaneous findings suggestive of physical abuse include bruising of the head, periorbit, face, buttocks, trunk, upper extremities, genitalia, hands, feet and ears. Patterned bruising caused by direct impact or friction with an objective and labial or frenulum laceration in an infant is also highly suspicious of physical abuse [3,21,36].

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<th>Pittsburg Infant Brain Injury Score for Abusive Head Trauma</th>
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<tr>
<td><strong>Predictor Variables</strong></td>
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<td>1.) Age ≥ 3 months (1 point)</td>
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<td>2.) Head Circumference &gt; 90th % (1 point)</td>
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<td>3.) Serum Hemoglobin &lt; 11.2 g/dL (1 point)</td>
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<td>4.) Physical Exam (2 point):</td>
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<td>Neurologic or Dermatologic abnormality</td>
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<td>5.) Previous ED visit for high risk symptom (1 point):</td>
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<tr>
<td>ALTE</td>
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<tr>
<td>Apnea</td>
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<tr>
<td>Vomiting without diarrhea</td>
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<td>Seizures</td>
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<td>Soft tissue swelling scalp</td>
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Figure 2 5-point Clinical Prediction Rule: Score of 2 associated with abnormal neuroimaging; *poor feeding, fussiness, lethargy [21].

Howes et al. (2017)  
Email: cclark@umm.edu
A history of non-specific complaints such as lethargy, in a child who is not yet walking requires further investigation that is contrary to a child's developmental stage such as bruising raise high suspicion for abuse [2,3,36]. Furthermore, history with the sustained injuries or inconsistencies in history should low fall trauma (less than 1.5 meters or 5 feet) that is incompatible red flags. Reported no history of trauma, history of low impact or activity may reduce morbidity and mortality associated with AHT children at risk for seizures and prompt treatment of seizure seizures among AHT victims [4,14,38]. Early identification of neuro-imaging is associated with an increased risk of clinical edema, midline shift or loss of grey-white differentiation on hemorrhage [16]. Moreover, presence of cerebral ischemia, TBI include age, abusive mechanism and presence of subdural hemorrhage [29]. Electrographic seizures and electrographic status epilepticus occurs in 55-67% of AHT cases [4,5]. Significant predictors of post-traumatic seizures following TBI include age, abusive mechanism and presence of subdural hemorrhage [16]. Moreover, presence of cerebral ischemia, edema, midline shift or loss of grey-white differentiation on neuro-imaging is associated with an increased risk of clinical seizures among AHT victims [4,14,38]. Early identification of children at risk for seizures and prompt treatment of seizure activity may reduce morbidity and mortality associated with AHT [38].

HISTORY RED FLAGS

When eliciting a history of present illness for a child with suspected abuse it is important to pay close attention to historical red flags. Reported no history of trauma, history of low impact or low fall trauma (less than 1.5 meters or 5 feet) that is incompatible with the sustained injuries or inconsistencies in history should raise a high suspicion for abuse [2,3,36]. Furthermore, history that is contrary to a child's developmental stage such as bruising in a child who is not yet walking requires further investigation [36]. A history of non-specific complaints such as lethargy, poor feeding, vomiting, irritability seizures, apnea or difficulty breathing is common among AHT victims [33,39]. It is crucial to elicit the onset and progression of symptoms and to note inconsistent history among caregivers. The child’s past medical history should be reviewed noting high risk features including prior injuries or medical visits for bruising, intraoral trauma, fractures, failure to thrive or neglect [2,3,36]. Lastly, a discussion with the child’s primary care physician about social concerns and suspicion of abuse is vital.

DIFFERENTIAL DIAGNOSIS

Children presenting with vague symptoms associated with AHT can be confused for symptoms of other medical diagnoses. There are several misdiagnoses associated with the signs/symptoms of AHT the most common misdiagnoses include birth trauma, viral gastroenteritis, influenza, accidental head injury or trauma, sepsis, otitis media, increasing head circumference and seizure disorder [3,40,41]. If signs or symptoms are suspicious for potential abusive head trauma the AAP recommends consult with child abuse pediatricians, ophthalmologists, radiologists, neurologists, and neurosurgeons with training and expertise in pediatric non-accidental trauma [3]. The clinical findings and history should be carefully considered to determine if accidental injury is plausible. Presence of an isolated skull fracture, epidural hematoma and scalp swelling were found to be predictive of accidental head trauma among children with TBI [2,39,40]. Acker et al. (2014) conducted a retrospective review of pediatric trauma databases which showed after controlling for age, sex, illness severity score, GCS on presentation, need for CPR, and survival to hospital discharge, hematocrit of less than 30% and platelets of greater than 400,000 were predictive of AHT as the cause of TBI [40].

Many of the differential diagnoses associated with signs or symptoms of abusive head trauma can be investigated and potentially ruled out with a comprehensive medical history (i.e. medical interventions, genetic disorders) and thorough laboratory workup (i.e. coagulation disorders, infectious etiologies). Both acquired and congenital medical conditions should be considered. Coagulation disorders such as Hemophilia, thrombocytopenia, and vitamin K deficiency are associated with bleeding in infancy and can be ruled out with laboratory evaluation. Notably, most coagulation disorders with the exception of vitamin K deficiency are not commonly associated with intracranial hemorrhage [39]. It is also important to discern that children with severe TBI present in a transient coagulopathic state not necessarily attributable to an underlying congenital disorder [39,42]. Certain genetic and metabolic syndromes can also be associated with certain findings of AHT including osteogenesis imperfecta, sickle cell anemia, alagille syndrome, ehlers-danlos syndrome, glutaricaciduria type 1, and pyruvate carboxylase deficiency although these diagnoses have not been associated with subdural hemorrhage [3,44]. In addition, structural intracranial anomalies, infectious diseases, intoxications and malignancies should be considered and ruled out [3,32,42].

MANAGEMENT

Mortality associated with pediatric TBI is often the consequence of uncontrolled intracranial hypertension and its...
detrimental effects on cerebral perfusion [20]. A goal directed strategy for recognizing and treating intracranial hypertension is practiced in most tertiary pediatric centers however the optimal intracranial pressure (ICP) target for pediatric TBI has yet to be clearly defined in the literature. An ICP threshold of 20 mmHg for acute intervention has been widely accepted in both pediatric and adult neuro-critical care however it has been proposed that a lower ICP threshold may be advantageous in children secondary to immature mechanisms of cerebral auto-regulation [20,38]. A study among children who suffered TBI found there was no appreciable difference in ICP or CPP in abusive head trauma versus accidental head trauma [38]. The following recommendations are based on the Brain Trauma Foundation 2012 Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children and Adolescents.

Invasive ICP monitoring is recommended in severe pediatric TBI and may prove helpful in children at risk for neurologic deterioration secondary to traumatic mass lesions or in circumstance in which a neurologic exam maybe unreliable due to the effects of sedation, neuromuscular blockade or anesthetics [20]. A prospective observational study by Dixon et al. (2016) found that ICP monitoring and ICP directed therapies were more common among children with a GCS of less than 8 and showed 50% of children who received one or more therapies for intracranial hypertension (barbiturate coma, active cooling, hyperventilation, or hypomolar therapy) did not have an ICP monitor in place [41]. This suggests that clinicians are using clinical markers other than invasive monitoring to recognize and treat intracranial hypertension. The current pediatric TBI literature does not clearly show the relative value of ICP monitoring in preventing cerebral herniation and no surrogate markers studied have consistently been shown to detect intracranial hypertension in the absence of ICP monitoring [20,38,41].

Hyperosmolar therapy with hypertonic saline is recommended in the treatment of intracranial hypertension in the setting of severe pediatric TBI creating an osmolar gradient promoting movement of water across the blood brain barrier decreasing intravascular volume thus decreasing ICP [20]. There currently is insufficient evidence to support the routine use of mannitol in this patient population. Effective bolus doses of 3% hypertonic saline range from 6.5-10 ml/kg with continuous infusion dose of 0.1-1.0 ml/kg/hr if needed. The minimum dose needed to maintain ICP less than 20 mmHg should be utilized with a target serum osmolarity of less than 360 mOsm/L and age-appropriate CPP [20,38]. Intracranial pressures of greater than 20 mmHg sustained for more than five minutes necessitate aggressive treatment. Prolonged periods of intracranial hypertension with ICP greater than 20 mmHg and CPP of less than 45 mmHg have been shown to be strong predictors of poor patient outcomes [20,38,41]. CPP, calculated as mean arterial minus ICP, is a surrogate indicator for global cerebral perfusion. Systemic hypotension should be corrected rapidly to prevent secondary injury from cerebral hypoperfusion and ischemia. Lower limits of acceptable systolic blood pressure for age can be estimated using the formula 70 mmHg + (2 x age in years) [20]. An age specific CPP threshold for pediatric between 40-50 mmHg should be targeted with infants at the lower end and adolescents at the upper end of this recommended range [20]. A recent study evaluating age specific CPP thresholds on short term survival in pediatric TBI supported CPP goals above 50-60 mmHg in adults, above 50 mmHg in 6 to 17 year olds, and above 40 mmHg in 0 to 5 year olds [45]. Furthermore, among all age groups an elevation in ICP was associated with lower CPP whereas the effects of systemic hypertension on CPP was inconsistent emphasizing the significance of controlling intracranial pressure [45].

Adequate oxygenation and ventilation should be ensured in pediatric TBI patients to decrease the risk of secondary brain injury resulting from hypercapnia and hypoxemia. Early tracheal intubation is often warranted to establish airway control and is recommended in children with a GCS of less than 8 with increased risk of aspiration [20,26,42]. Adequacy of oxygenation and ventilation should be monitored with, serial blood gases, continuous pulse oximetry and end tidal carbon dioxide. Of note, prophylactic hyperventilation is not recommended and should only be considered in patients with acute signs of impending cerebral herniation [20].

Response to pain, noxious stimuli and stress can cause significant increases in cerebral metabolic demands and cerebral blood flow consequently raising ICP. Sedation and analgesia should be optimized ideally with an agent that has minimal or no cardiovascular effects to avoid systemic hypotension and with rapid half-life to allow dose monitoring of neurologic exam. Neuromuscular blockade can be used in refractory cases of intracranial hypertension despite adequate sedation to decrease metabolic demands however this will significantly hinder neurologic exam and thus should be used in conjunction with invasive ICP monitoring [20].

Post-traumatic seizures have been shown to occur in approximately one-fourth of children with severe TBI and up to 57% of children who suffered abusive head injury [4,17,46]. Children under the age of 2 years have the highest incidence of seizures following TBI [40]. Hashani et al (2013) found evidence of ischemia on neuro-imaging to be associated with presence of electrographic seizures whereas parenchymal and extra-axial abnormalities were not [4]. Another retrospective study conducted by Bennett et al. (2017), showed the triad of young age, injury by abuse, and subdural hemorrhage conferred the greatest estimated probability for posttraumatic seizures among children with TBI [17] Chung et al. (2016), found similar results, noting early posttraumatic seizures were more common in younger infants (median age 4 mo) and those who suffered from AHT compared to accidental trauma [46]. Clinical observation alone in patients with severe non-accidental trauma or TBI is not sensitive enough to appreciate seizure activity as subclinical seizures can occur. Therefore, continuous in addition to seizure prophylaxis should be considered in patients following TBI [46-49]. Radiologic findings on initial CT such as midline shift, cerebral edema and loss of gray white differentiation can help to identify patients at increased risk for both clinical and non-clinical seizure activity [40]. Non-convulsive seizures occur frequently in association with imaging findings of subarachnoid hemorrhage or abnormal T2 imaging on MRI [47]. Seizures are most likely to occur forty-eight hours after occurrence of intracranial hemorrhage coinciding with time frame for maximal swelling [31]. Seizure prophylaxis can include either the use of
Levetiracetam or Fosphenytoin. There is no clear advantage of using Levetiracetam or Fosphenytoin; although in some patient’s difficulty in maintaining therapeutic levels with Fosphenytoin may make the use of Levetiracetam advantageous [49].

OUTCOMES

Historically, pediatric studies have demonstrated that AHT is associated with worse outcomes and a higher mortality compared to other forms of non-inflicted head trauma [12]. Associated spinal cord injury and secondary hypoxic ischemic insults further contribute to these poor outcomes [1]. A recent large multi-center study demonstrated conflicting findings, reporting similar mortality rates and deaths from refractory intracranial hypertension among children with abusive and non-abusive mechanisms of head injury [50]. They did however report a higher prevalence of pre-hospital intubation, apnea and seizures during resuscitation in those with AHT suggesting presence of increased ICP and need for early ICP directed therapies [50].

A review of the literature has revealed a number of factors associated with increased mortality among infants and children with AHT. A low initial GCS (3-5), retinal hemorrhage, intraparenchymal hemorrhage and cerebral edema are independently associated with mortality in AHT [51]. Non-survivors of AHT are more likely to have received CPR and undergone pre-hospital intubation suggesting a higher severity of illness on presentation [52]. An elevated base deficit on admission was also associated with increased mortality noting a two fold increase in mortality for every 5 meq/L increase in base deficit [55]. A blood glucose elevation (>200 mg/dL) in the first 12 hours and prolonged INR (>1.3) on admission were found to be independent predictors of associated poor outcomes and increased mortality [53,55]. Identifying these risk factors associated with increased morbidity in children with AHT may be useful in providing early prognostic information.

The neurologic consequences of AHT can be severe and long lasting. Studies have shown, among AHT survivors, 45% suffer permanent neurologic impairment and two-thirds are at least moderately disabled following traumatic brain injury [1,2,6,34,56]. Lind et al., (2016) evaluated long term outcomes of children following AHT noting reports of sustained epilepsy (38%), motor deficits (45%), visual deficits (45%), sleep disorders (17%), language abnormalities (49%), attention deficits (79%) and behavioral disorders (53%) [5,7]. Visual field loss, color vision impairment, secondary amblyopia, strabismus, retinal detachment and neovascular glaucoma are among the visual impairments encountered [32]. Among those studied, 83% required on-going rehabilitation with 30% requiring special education services at an 8-year follow [5,7].

Although the associated morbidity and mortality in pediatric AHT, compared to non-inflicted head trauma, is not entirely understood it is presumed to be multifactorial including characteristics of injury, delays in seeking medical attention by caregivers, delays in identification of AHT by providers, effects of prior maltreatment particularly prior AHT with associated developmental disabilities.

CONCLUSIONS

Pediatric AHT, a severe and often devastating form of child maltreatment, remains a challenging diagnosis with both profound social and legal implications [57-61]. Clinicians should understand the mechanism of injury associated with this type of non-accidental trauma as well as be familiar with the clinical spectrum of injury and associated clinical manifestations to avoid misdiagnosis. A thorough review of systems and medical history is necessary to investigate the possibility of abusive head trauma in pediatric patients. Management of patients with AHT should include treatment recognition and treatment of increased intracranial hypertension, optimization of cerebral perfusion pressure, support of oxygenation and ventilation, and recognition and treatment of seizures. A multidisciplinary approach with development of child abuse protective teams is highly recommended. Future pediatric research efforts should focus on AHT specific treatment strategies aimed to improve clinical outcomes and decrease the incidence of long-term neurologic and developmental sequelae.

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