Update on Non-accidental Trauma

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14.1 Introduction

Abusive head trauma (AHT), also known as non-accidental head trauma, is among the leading causes of infant death and long-term morbidity secondary to abuse [1, 2]. The previously used term "shaken baby syndrome" represents a syndromic subset of AHT in which there is evidence of central nervous system (CNS) injury, with or without obvious external signs of trauma. Head injury, in these cases, may be primary or secondary, exhibiting diffuse unilateral or bilateral subdural hemorrhages, diffuse multiplanar retinal hemorrhages, and brain injuries such as diffuse axonal injury or other parenchymal injuries. These injuries are likely secondary to either rotational or blunt force trauma, which have been confirmed by perpetrator accounts [3]. However, the initial history provided is commonly a minor fall or injury or even a complete lack of trauma. In many cases, infants can present without external manifestations of trauma, and injuries are usually revealed during the workup. Eye findings are noted in approximately 85% of AHT cases [4–6]. Ophthalmic manifestations can vary from retinal or vitreous hemorrhages to retinoschisis, retinal detachment, choroidal rupture, hyphema, orbital fractures, or

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periorbital ecchymoses. Victims of AHT can have lifelong physical, psychological, and academic consequences [7]. Every healthcare professional in pediatric and emergency departments has a responsibility to identify child abuse cases.

14.2 Epidemiology/Differential Diagnosis

As per the National Child Abuse and Neglect Data System (NCANDS), there were 656,000 national reports of child abuse and neglect in 2019, with an estimated 1840 fatalities stemming from abuse and/or neglect [8]. 70.3% of those fatalities occurred in victims under the age of 3 years, and 45.4% occurred in victims under the age of one [8]. Incidence of AHT in the United States has been estimated to occur in 29.7 per 100,000 person-years in infants, resulting in as many as 80 deaths per year, with evidence of head trauma evident through clinical history or objective exam findings [8]. Measuring incidence worldwide is fraught with limitations and inconsistencies. However, when assessing studies regarding the statistical frequency of AHT abroad, similar incidence rates are evident when compared to the US [9].

The majority of victims of AHT are under the age of 3 [8]. Often, misdiagnosis of nonspecific complaints, including irritability, fussiness, lethargy, or poor appetite, precede an AHT diagnosis. Risk factors identified by NCANDS include prior episodes of abuse, low family socioeconomic status, unstable home environment, including single-parent households, substance/alcohol abuse, and prevalence of domestic abuse among parental figures [8]. Interestingly, one study revealed a 1493% increase in AHT cases over a 30-day period of nationally mandated quarantining during the COVID19 pandemic in the United Kingdom when compared to the incidence rate in the same 30-day period over the previous 3 years [10]. This suggests that times of national strife in a pandemic could have exacerbated many of the known risk factors of AHT, including increased substance use among caregivers, worsened financial instability, and delayed presentation to the hospital to avoid increased exposure to contracting COVID19.

Retinal hemorrhages (RH) are not exclusive to head injury secondary to abuse. The most compelling differential diagnosis of RH in neonates and infants is hemorrhages related to normal vaginal delivery. In one prospective study, the incidence of RH in normal, nonassisted vaginal deliveries was 30.4%, up to 77.8% in vacuum-assisted deliveries [11]. However, all RH seen were intraretinal, and the majority of eyes were completely resolved in less than 30 days [11]. Beyond a neonate's first month of life, the most common cause of RH in infants is abuse. Less common diagnoses include accidental trauma (including falls, motor vehicle crashes, crush, and blunt force), congenital coagulopathic or bleeding disorders (including blood-borne neoplastic processes, clotting factor/inhibitor deficiencies, myelodysplastic syndromes, and cytopenias), elevated intracranial or intrathoracic pressure, or meningitis/encephalitis. See Table 14.1 for a complete list of common differential diagnoses of RH in infants. Table 14.1 Causes of Retinal Hemorrhages in infants and young children [12].

Table 14.1 Common differential diagnoses of retinal hemorrhages in infants and young children

Condition	Frequency of RH	Hemorrhages	Clinical features
Abusive head trauma	50–100% of cases	Numerous, extending to periphery, multiple layers	History inconsistent with injuries; commonly associated with SDH, fractures, bruising, intraabdominal injury
Perinatal RH	20–30% of newborns examined within first 24 h; 10–15% of newborns examined within first 72 h	Numerous, extending to periphery, usually intraretinal	More common in vacuum-assisted deliveries, resolves in 4–6 weeks
Unintentional head injury	0–10% patients	Typically few in number, confined to posterior pole	Associated with severe mechanism of injury (MVC, fall from height)
Hematologic conditions			
Bleeding disorder (hemophilia, von Willebrand disease, vitamin K deficiency)	Unknown	All layers of retina, vitreous	Characteristic history of bleeding disorder, abnormal coagulation studies
Leukemia	Unknown; most patients have funduscopic changes at some point in disease	Usually at posterior pole in deep retinal layers, but may occur in all layers and in vitreous	Characteristic laboratory features (lymphoblasts on peripheral blood smear)
Anemia (juvenile pernicious, iron deficiency, sickle cell)	Unknown, more common in adults than children	Intraretinal dot, blot, flame, or splinter	Anemia
Metabolic conditions			
Glutaric aciduria type 1	Rare	Few in number, confined to posterior pole	Macrocephaly, characteristic basal ganglia findings, may present with subdural hematomas; absence of bony abnormalities
Galactosemia	Rare	Vitreous	Failure to thrive, cataracts
Infection			
Cerebral malaria	20–60% of children with severe cerebral malaria	Multiple white lesions	Travel history; fever, anemia, characteristic blood smear findings
Meningitis	Rare	Various	Fever, characteristic cerebrospinal fluid findings

(continued)

Table 14.1 (continued)

Condition	Frequency of RH	Hemorrhages	Clinical features
Retinal infection (CMV, HSV, toxoplasmosis, rickettsiae)	Rare	Small, intraretinal areas of retinal necrosis; may span all layers of necrotic retina	Systemic signs of infection may be present; immunodeficiency
Endocarditis	Rare	White-centered oval hemorrhages	Other characteristic features (cardiac murmur, fever, splinter hemorrhages in nail beds, Janeway lesions)
Primary retinal disease			
Retinopathy of prematurity	Rare	Small, usually intraretinal on surface of neovascular ridge; may extend to vitreous	History of prematurity; retinal neovascularization
Coats disease	Rare	Usually intraretinal but may extend to vitreous	Usually unilateral; subretinal exudate; telangiectatic vessels
Persistent fetal vasculature	Rare	May present with vitreous hemorrhage	Usually unilateral; may be associated with mild microphthalmos; intralenticular hemorrhage; retrolental membrane
Other			
Intracranial hemorrhage	Rare in absence of abusive head injury	May be extensive	Characteristic neuroradiographic features (aneurysm, arteriovenous malformation)
Chest compression			
Cardiopulmonary resuscitation	Rare if ever	Few in number, small in size	History of cardiopulmonary resuscitation
Thoracic crush injury	Rare	Superficial; white retinal patches	History of thoracic injury

The presence of ocular findings varies widely in reports of AHT; however, RH play a vital part in contributing to a convincing clinical picture if AHT is suspected. In a retrospective study of perpetrator-confessed inflicted brain injury, RH were prevalent in 83% of 81 reports [13]. Other studies reveal a prevalence of RH in nonaccidental head trauma cases to be upwards of 94%, with one study reporting 100% of cases with evidence of RH [2, 14]. Early identification can be lifesaving, and an ophthalmological exam is imperative as intraocular pathology may be the only clinical sign of possible abuse.

Observational studies have been conducted in which AHT was diagnosed by clinical findings, along with inclusive criteria consisting of perpetrator confession,

legal conviction, autopsy data, or ancillary care team assessments. In a meta-analysis of these studies, intraocular (retinal or vitreous) hemorrhage had a sensitivity of 75%, and specificity of 94%, for AHT [15]. While intraretinal hemorrhages were found in both accidental and abusive groups, multilayered, numerous, and bilateral retinal hemorrhages were most specific for AHT [15]. Optic nerve sheath hemorrhages (ONSH) had a sensitivity of 72% and a specificity of 71% for AHT [15]. Lastly, retinoschisis and peri-macular folds were found only in cases associated with abuse, and not in the nonabuse head injury groups [15].

14.3 Pathophysiology

The pathophysiology of RH in AHT is not entirely understood. Vitreoretinal traction is widely accepted as the most likely mechanism for RH development. Other theories include repetitive acceleration-deceleration (with or without coup-contrecoup injury), increased intracranial pressure, intrathoracic pressure, hypoxia, coagulopathy, and intraorbital injury.

Force, and therefore pressure, at the vitreoretinal interface theoretically increases with repeated acceleration-deceleration. With vigorous shaking of an infant, vitreoretinal separation is most vulnerable at the posterior pole, along the retinal vasculature, and at the globe equator as tethering, areas which are subjected to greater shearing tangential forces [16]. In experimental studies, purely linear acceleration produced tenfold weaker tension stress than angular acceleration [16]. This differentiates the pathophysiological mechanism of a blunt force injury or purely linear acceleration that occurs along a single plane compared to tangential or angular forces seen during shaking.

Intracranial hemorrhage, and cerebral edema secondary to mechanical brain injury, can cause increased intracranial pressure (ICP). This pressure is purported to cause subsequent compression and downstream hemorrhages in the retinal vasculature due to increased venous congestion [17]. However, other entities that increase intracranial pressure do not present with the characteristic RH seen in AHT. In rare cases of increased ICP, RH are flame-shaped peripapillary intraretinal hemorrhages in conjunction with edematous optic nerves. Rarely do hemorrhages associated solely with increased ICP occur in the periphery, which are limited to the intraretinal pseudospace. In contrast, RH in AHT are usually described as multiplanar (i.e., evidence of subretinal, intraretinal, and preretinal) hemorrhages that can occur throughout the retina's panorama (Fig. 14.1) [18]. While the etiology of RH found throughout the retina layers is multifactorial. One study has shown that tangential shearing forces exert pressure on the retina transmitted throughout all layers with relatively congruent magnitude [16]. What is still not a consensus is if a rupture of the internal limiting membrane at the vitreoretinal interface is the instigator in permitting exaggerated pressure on the retina or if shaking causes enough localized pressure within all layers of the retina to cause internal limiting membrane rupture.

Increased intrathoracic pressure is also a proposed mechanism of ocular manifestations of AHT [17]. This theory assumes that AHT is accompanied by chest wall injury, including rib fractures, that would induce elevated intrathoracic pressure.

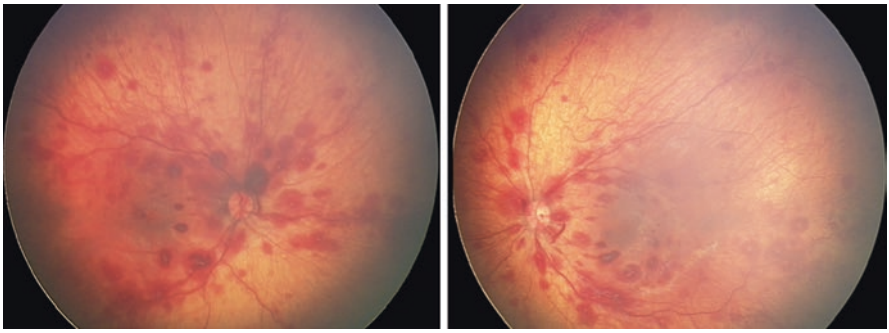

Fig. 14.1 Fundus photograph showing bilateral multiple Retinal Hemorrhages extending into the periphery

Acutely increased intrathoracic pressure is proposed to cause elevated retinal intravascular pressure, as seen in the Valsalva maneuver, or significant chest wall trauma, eponymously known as Purtscher retinopathy [19]. While Purtscher retinopathy was initially described by Otmar Purtscher in a man with head trauma secondary to falling from a tree, it is now more universally associated with pro-embolic systemic states. White retinal patches cotton-wool spots are seen in Purtscher retinopathy from vaso-occlusive processes, including fat emboli, circulating pancreatic enzymes during pancreatitis episodes, immune complex deposition, and also in disease states with decreased venous return, causing downstream retinal vein congestion. Purtscher retinopathy can be intrinsically associated with AHT, if superimposed by coagulopathy. However, it is more likely secondary to chest compressions performed during resuscitation events [20].

When shaking an infant, unique anatomical properties of the neonatal spinal cord and neck portend greater subjectivity to injury [21]. Spine ligaments have greater hydration, laxity, and elasticity, allowing greater displacement between vertebrae, and stretch significantly beyond subluxation limits of an adult without ligament rupture or spinal cord injury [22, 23]. Rigid limitations of mature bone provide a higher likelihood of spinal cord injury if repeatedly over-flexed and extended, and, thus, the infant's neck subtends a protective measure when shaking at low frequency, high displacement. Accordingly, spinal cord injuries may not be mutually inclusive with RH in AHT, and shaking does not requisitely lead to spinal cord injury in many cases.

Clotting disorders (Factor V Leiden, C/S deficiency, MTHFR mutation, homocysteinemia, etc., as well as conditions eliciting disseminated intravascular coagulation (DIC) response), could cause RH that may mimic those seen in AHT, given they may present with other systemic symptoms, including diffuse ecchymoses, erythema, swelling, and unlocalized pain [24]. Cytopenic or thrombophilic conditions superimposed on AHT may also complicate diagnosis.

The most common finding of abuse in postmortem autopsy examinations is subdural hemorrhages, and the presence of subdural hemorrhages has been reported in >70% of AHT victims [25–27]. RH are more commonly seen with subdural

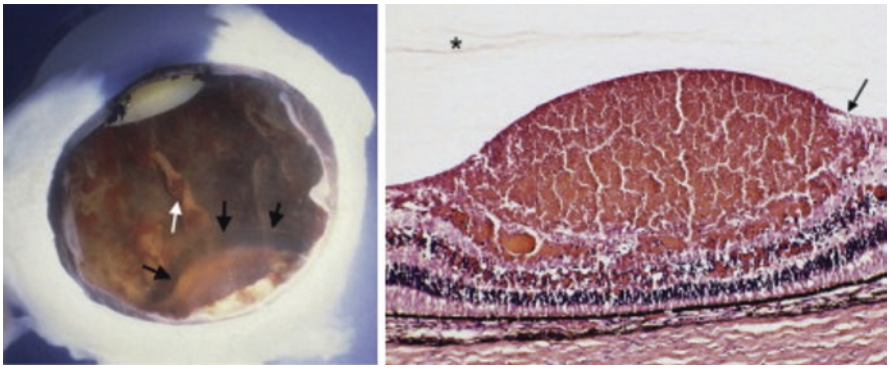

Fig. 14.2 Gross and histopathological section of eye post-shaking

hemorrhages (SDH), as both sequelae' mechanism depends on tangential and rotational inertia of the brain while shaking [28]. SDH develops as the bridging veins are sheared away from the arachnoid membrane with forceful rotational movement. Similarly, in the eye, histological evidence of internal limiting membrane (ILM) compromise suggests a shearing mechanism in which inertia of head contents produces a force that exceeds retinal adhesive composition. Erythrocyte extravasation through the ILM is the impetus for multilayered hemorrhages seen only with shearing forces, and, thus, a compelling diagnostic marker in this patient population. See Fig. 14.2 [29]

14.4 Clinical Presentation/Evaluation/Workup

Along with the physical signs and symptoms, presenting history is crucial for diagnosing AHT. Often, the account from caregivers is inconsistent or nonexistent, and a strong index of suspicion is required to make the diagnosis [30]. Initial presentation can vary widely, from minor nonspecific signs and symptoms to acute life-threatening complications. Often these signs can be mistaken for a virus or other minor illness. The presence of bruising in infants should alert a physician to the possibility of abuse. Examining physicians should pay particular attention to "TEN-4" bruising (bruising of the torso, ears, and neck in children younger than 4-years old or any bruising in an infant younger than 4 months) [31]. Findings such as apnea, retinal hemorrhages, and "TEN-4" bruising are much more common in abusive than accidental injury [31, 32]. A comprehensive medical evaluation includes a skin examination for bruising, a skeletal survey for fractures, head imaging for intracranial bleeds, and a prompt ophthalmology consultation scanning for retinal hemorrhages. This protocol remains the foundation of AHT assessments.

As mentioned above, the initial signs and symptoms of AHT can be nonspecific decreased interaction, lack of a social smile, including an acute decrease in the level of consciousness, poor feeding, vomiting, lethargy, hypothermia, increased sleeping, seizures, and failure to thrive, the latter of which is more chronic [33]. see Table 14.2.

Table 14.2 Frequent symptoms and signs of abusive head trauma (AHT)

Apnea
Bulging fontanel
Bradycardia
Cardiovascular collapse
Chills
Decreased interaction
Decreased level of consciousness
Failure to thrive
Hypothermia
Irritability
Increased sleeping
Lack of a social smile
Lethargy
Microcephaly
Poor feeding
Vomiting
Respiratory difficulty and arrest
Seizures

Rib, long bone, and complex skull fractures can support a diagnosis of AHT but are not required. However, in all cases, retinal and subdural hemorrhages are the most specific findings in AHT [1]. A study by Binenbaum et al. reported an association between severe retinal hemorrhage and the presence of hypoxic-ischemic brain injury (HII) patterns by diffusion-weighted magnetic resonance imaging (DW-MRI) in infants with AHT [34]. In addition, orbital and ocular injuries can be present secondary to blunt trauma. Periorbital ecchymosis, subconjunctival hemorrhages, traumatic hyphema, and traumatic cataracts are possible due to blunt trauma sequelae. Direct impact to the eye can also lead to corneal abrasions or lacerations, globe rupture, or damage to the iris. RH may occur with accidental head trauma. However, they are uncommon, seen in less than 10% of cases, often unilateral, and generally limited to the posterior pole adjacent to the optic nerve and macula, to a single layer of the retina [1].

14.4.1 Workup

14.4.1.1 Ophthalmic Examination

Often, the ophthalmic examination is imperative for any child presenting with visible trauma, altered mental status or consciousness, or laboratory or clinical signs of coagulopathy and problems with bleeding. Given the transient nature of RH, consultations should ideally be completed within 24 h of patient presentation; however, no later than 72 h to enumerate the full extent of RH that may be present due to trauma. Exams should begin with standard eye vitals, including pupil measurements, extraocular motility, and intraocular pressure, especially taking note of pupils for incongruent reflexes suggesting an afferent defect. Next, anterior segment assessment should follow for possible external signs of trauma or bleeding,

followed by a dilated examination, ideally visualizing the fundus with indirect ophthalmoscopy. Ophthalmic consultation and fundus exam are helpful, even when dilated fundus exam is not feasible, especially in children who cannot undergo pupil dilation due to severe central nervous system injury.

When observing RH in the setting of suspected abuse, complete descriptions of the hemorrhages regarding location throughout the panorama and within which retinal layer(s), number, pattern, and bilaterality (if present in both eyes) is crucial to document. Fundus photographs may also be performed to aid in the characterization of RH. Intraretinal RH typically resolves more quickly than preretinal RH, the latter of which may last for a few days following the injury. Thus, it may be possible to roughly deduce the onset of injury—if there is a strong suspicion of abuse with the presence of preretinal RH but lack of intraretinal RH, it is reasonable to infer the incident occurred at least a few days to a week before the presentation [35].

14.4.2 Laboratory Tests

The laboratory evaluation aims to rule out medical conditions that may mimic AHT and evaluate for other abusive injuries. A list of workups can be seen in Table 14.3.

14.4.3 Imaging

A computerized head tomography (CT) scan is often the first study performed because it is easy and fast to complete; however, these scans are limited in evaluating cerebral injury. Therefore, a magnetic resonance imaging (MRI) examination of the brain and spine is recommended around 72 h post-injury [36, 37]. Cranial ultrasonography is an insufficient diagnostic neuroimaging modality in cases of suspected AHT [37].

A review by Kemp et al. looking at neuroimaging findings in children mostly over 3 years old has shown that multiple subdural hemorrhages (SDH) along the convexity, posterior fossa, interhemispheric location, hypoxic-ischemic injury (HII), and cerebral edema were significantly associated with AHT [38]. Chronic

Table 14.3 Blood workup for suspected abuse

CBC
CMP
Prothrombin time (PT)
Partial thromboplastin time (APTT)
Amylase
Lipase
Aspartate aminotransferase
Alanine aminotransferase
UA urinalysis

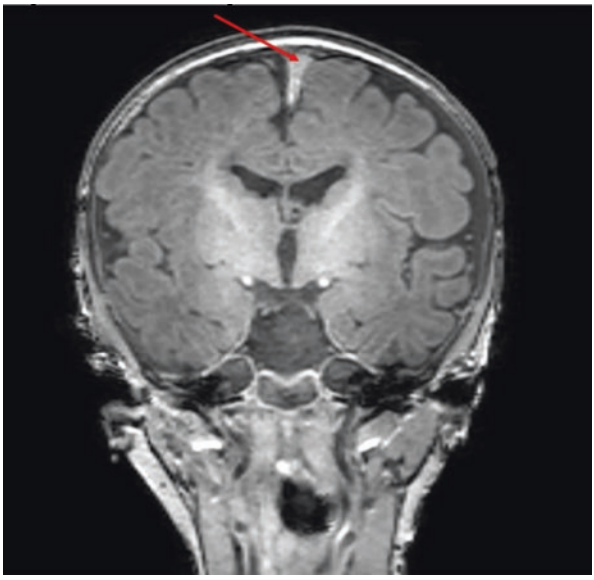

Fig. 14.3 MRI image showing Subdural Hematoma (SDH)

SDH appears specific for AHT, if not particularly sensitive, with less than half of identified AHT cases presenting with chronic SDH [38, 39] (Fig. 14.3).

A complete skeletal survey consisting of plain radiographs of the skull, spine, ribs, and long bones is performed in children younger than 2 years with concerns of AHT to rule out occult fractures. A limited skeletal survey (excluding the spine, pelvis, and skull) should be considered 2–3 weeks after initial presentation to assess for healing fractures that may not be apparent in the acute stage [40, 41].

Severe head injuries can rarely occur from a fall. Short falls (less than 1.5 m or 5 ft) generally do not cause a severe head injury. When the history of a short fall is provided for a child with a severe head injury, AHT should be considered in the differential diagnosis. Falling from a bed height is usually considered minor, although some children may suffer from a fractured arm, leg, or skull [42]. Vertical falls of 1–4 feet rarely cause severe head trauma or multiple injuries. One study evaluating children who died following a fall from that height found other concurrent evidence of abuse [43]. Even falls from 10 feet rarely result in death [44]. Generally however the greater the height, the greater likelihood of fractures and other injuries.

14.5 Management/Expected Sequelae or Complications/ Ancillary Consults

Management of AHT may involve the expertise of a number of medical specialists, including neurosurgery, trauma surgery, neurology, child abuse pediatrics, critical care physicians, and ophthalmology. The initial treatment of a patient with AHT should involve stabilizing the airway to support oxygenation, ventilation,

hemodynamics, and mitigation of intracranial pathology. A more detailed evaluation for other abusive injuries of pathology can wait until the patient is stable.

Pediatricians' role in diagnosing and reporting abuse is crucial since often they are the first point of contact. Pediatricians must report suspected abuse or neglect to state child protective services. The diagnosis of abuse is sometimes straightforward but at times may not be informed or may often go unnoticed, especially when there are subtle signs and symptoms [33]. Sometimes, primary care physicians or pediatricians are not forthcoming in reporting all of the highly suspicious abuse cases. The decision to report is greatly influenced by the mechanism of injury, familiarity with family, and previous encounters with child protective services [45]. Pediatric practitioners should be aware of and must remain observant of the possibility of AHT in infants to protect and prevent any future cases. They must promptly inform relevant authorities of suspected abuse for further investigation.

Before making a diagnosis or reporting to the child protective agency, practitioners should consider various conditions that can present with findings indicative of AHT. Physicians should objectively communicate the findings and not overemphasize the impact of specific medical findings. Nevertheless, they should effectively convey child safety concerns. Early consultation with a child abuse physician may be sensible since verbal and written dialog with collaborative investigative agencies can be challenging.

In children with abusive head trauma, mortality rates ranges up to 35.7%. Forty-two to ninety-six percent of survivors suffer long-term neurologic morbidity [46]. High rates (72%) of any form of disability are commonly reported among the survivors, such as delayed psychomotor development; motor deficits (spastic hemiplegia or quadriplegia in 15–64%); epilepsy, often intractable (11–36%); microcephaly with corticosubcortical atrophy (61–100%); visual impairment (18–48%); language disorders (37–64%), and cognitive, behavioral, and sleep disorders, including intellectual deficits, agitation, aggression, tantrums, attention deficits, memory, inhibition or initiation deficits (23–59%) [7]. Due to significant short- and long-term complications, survivors, need to have frequent monitoring and prompt referral to a subspecialist to achieve the best possible outcomes. These sequelae may not manifest for years, as cognitive and behavioral consequences may only become apparent at school entry or later [47].

14.6 Primary/Secondary Prevention and Public/Parent Education

The diagnosis of abuse is intertwined with significant ramifications. Diagnosing AHT has severe legal and social implications, including for children who can be removed from their homes and adults imprisoned for their actions. However, missing a case of abuse can result in a child being returned to a potentially unsafe environment. Various strategies are implemented to decrease the incidence of AHT. Two primary areas include parental education about the crying infant and the risks of shaking a baby. Parents with newborn infants are provided with mandatory "shaken

baby syndrome" education in some states. Dias et al. demonstrated success in reducing the number of AHT in a region of New York; however, similar results could not be replicated with statewide implementation of the education in Pennsylvania [48–50].

Another program is the Period of PURPLE Crying, where the acronym "PURPLE" stands for the characteristics of infant crying: P: Peak of crying, U: Unexpected, R: Resists soothing, P: Pain-like face, L: Long-lasting, E: Evening and late afternoon. This program provides parents and caregivers information about normal infant crying patterns and expected normal behavior in these infants based on scientific evidence. A three-step approach involves an in-hospital immediate postpartum education in the maternity ward to both parents, which provides written and video education, followed by public health nurses and annual community education in the second phase, public awareness and mass media promotion, as the third step. This program was successfully implemented in British Columbia. Still, performance in the state of North Carolina did not match up. It was not able to demonstrate a reduction in the incidence of AHT [51, 52]. Although it has been challenging to show a decrease in AHT rates with educational interventions consistently, some prevention programs have found self-reported, improved understanding of infant crying, improved emotional self-regulation, and increased parental knowledge of AHT [53]. Even though there has been no significant reduction in AHT rates with the Nurse-Family Partnership (an in-home visitation program), there has been a long-term decrease in child maltreatment reported, and this may be a helpful methodology in tackling AHT [54].

14.7 Ongoing New Research

Current research is ongoing to determine the force, frequency, pressure, and stress required to cause RH when shaking an infant. As noted in previous sections, research is underway to determine inciting factors that cause multilayered and distinctive RH patterns seen uniquely with AHT [16]. Several studies have been performed on dummy dolls or computer simulation models. However, the translation to animal and, eventually, human studies is complex and limited due to ethical concerns precluding the experimentation on in vivo subjects [55–58]. Autopsy data is limited, given an incomplete clinical picture and natural tissue decomposition postmortem [59, 60]. Early Ex vivo animal models have been developed in conjunction with more refined finite element (FE) eye simulated models since the initial description of FE eyes in 2014 [16, 61–63]. One preliminary study isolates the vitreoretinal interface in an intact globe and indirectly measures the force required to separate vitreous from the retina by pulling anchor points from opposite axes away from each other [16].

In addition to mechanistic studies, further research is currently being conducted to study long-term follow-up in infants who undergo ophthalmic intervention at the time of presentation, including corticosteroid therapy, hyperbaric oxygen, and surgery, such as pars plana vitrectomy [16]. Public health/epidemiologic studies are

being undertaken to determine if a significant link exists between incidence and prevalence of brief unexplained events (BRUE), sudden infant death syndrome (SIDS), and shaken baby syndrome (SBS) [64]. While some have argued determining the exact mechanisms of ocular consequences post shaking may provide a theoretical manual for future perpetrators, the risk may outweigh the benefit of public education in showing how little force is required to overcome vitreoretinal adhesion.

The unifying goal of ongoing research about AHT is public education and determining primary and secondary prevention strategies to reduce rates and cases per year.

Key Points

1. Abusive head trauma in infants and children can be fatal. Even if they survive the trauma, children can have a substantial permanent disability.
2. Abusive head trauma can be missed initially or frequently misdiagnosed. Clinicians should remain mindful of infants with bruising or nonspecific symptoms, such as vomiting.
3. Children suspected to have abusive head trauma should undergo a thorough workup, including laboratory and imaging studies.
4. Child abuse patients will benefit from a multidisciplinary approach.

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