Retinal Hemorrhages
Abusive Head Trauma or Not?

Jennifer B. Hansen, MD, Emily F. Killough, MD, Mary E. Moffatt, MD, and Jane F. Knapp, MD

Abstract: Abusive head trauma is an important cause of morbidity and mortality in infants and young children. Retinal hemorrhages (RHs) are frequently seen, particularly during dilated eye examination of these children. This review focuses on the evaluation of children with RH, with emphasis on the differential diagnosis, pathophysiology, and distinguishing features of RHs due to abusive head trauma. Many causes exist for RHs in infants and children. Most medical and accidental traumatic causes result in a pattern of RH that is nonspecific and not typical of the pattern and distribution of RHs seen in children with abusive head trauma. In children with intracranial hemorrhage and concerns for abuse, the finding of severe, multilayered RHs extending to the periphery of the retina is very specific for abuse as the cause of the findings, especially if retinoschisis is present. There are few other accidental traumatic mechanisms associated with retinoschisis, and the history of such a traumatic event is readily apparent. The indications for ophthalmologic consult, optimal timing of the eye examination, and significance of the findings are specifically discussed.

Key Words: retinal hemorrhage, abusive head trauma, retinoschisis, accidental head injury, ophthalmology consultation

(*Pediatr Emer Care* 2018;34: 665–672)

TARGET AUDIENCE

This CME activity is intended for all practitioners who may see children who have retinal hemorrhages that raise concern for abusive head trauma.

LEARNING OBJECTIVES

After completion of this article, the reader should be better able to:

1. Define the role of the primary, urgent, emergent, inpatient, or intensive care practitioner in the recognition and workup of retinal hemorrhages.
2. Describe the number, location, and pattern of retinal hemorrhages most characteristic of abusive head trauma.
3. Distinguish the differential diagnosis for retinal hemorrhages in infants and children.

The Centers for Disease Control defines pediatric abusive head trauma (AHT) as an injury to the skull or intracranial contents of an infant or young child (<5 years of age) due to inflicted blunt force impact and/or shaking.[1] The American Academy of Pediatrics Committee on Child Abuse and Neglect has recommended that the term AHT replace other terms, such as shaken baby syndrome, which imply a single injury mechanism. This standardization of terminology provides needed consistency in diagnosis, medical communication, and documentation.[2]

Infants younger than 1 year of age are the highest risk group for AHT at a rate of 30 per 100,000.[1] Retinal hemorrhages (RHs) are often found in infants with AHT but can be observed in other conditions. This review will focus on the finding of RH in infants with the goal of assisting the clinician in determining whether the presence of RH is due to AHT.

ANATOMY

Transverse section through the eyeball reveals that for the most part the eyeball is composed of 3 concentric layers, from outer to inner: sclera, choroid, and retina.[3] However, the anatomy of the anterior portion of the eyeball has a different structure and is not discussed here. The outermost component of the eyeball is the sclera, which is a firm layer of connective tissue providing attachment for the extraocular muscles. Moving inwards from the sclera, the next layer is the choroid, also called the uveal tract, a very highly vascularized organ, which supplies blood to the outer retinal layers and regulates the temperature of the eyeball. The innermost layer of the transected eyeball is the retina. The vitreous humor (vitreous body) occupies the interior of the eyeball, stabilizing it and preventing retinal detachment.[3]

Most of the retina is photosensitive and therefore involved in vision, but a smaller, nonphotosensitive, nonvisual portion extends from the ora serrata more anteriorly and lines the interior of the eyeball behind the posterior surface of the iris.[3] The ora serrata, involved in vision, is the peripheral retina and the point where the visual portion of the retina ends upon extension forward from the optic nerve and posterior pole of the eyeball.[3] The vitreous humor attaches most strongly to the retina at the ora serrata, macula, and along the retinal vessels.[4] At the posterior portion of the eyeball, a small area of the temporal retina called the macula lutea, contains a central depression where visual acuity is the highest, the fovea centralis. As well, at the posterior pole in an area called the optic disk, the optic nerve axons pierce through the sclera at the lamina cribrosa.[3] The retina consists of 10 layers: the outermost retinal pigmented epithelium and 9 other layers, which are called the neurosensory retina. The inner most layer is the inner limiting membrane (ILM).[5]

Hemorrhage can lie on the retina (preretinal), within the layers of the retina (intraretinal), or under the neurosensory retina (subretinal).[5] Preretinal hemorrhages, also called subhyaloid hemorrhages, form in front of the retina, between the ILM and the vitreous.[6] If the blood extends into the vitreous humor, the term vitreous hemorrhage is used. Intraretinal hemorrhages in the more superficial layers of the retina take on a linear streaking appearance, following the nerve fibers; these types of hemorrhages are often called flame or splinter hemorrhage due to their appearance. In contrast, intraretinal hemorrhages in the deeper layers of the retina are more round in shape, and are called dot or blot depending on their size.[5] Terms are also used to describe the distribution of the RHs regarding the area or region of the retina where they occur:

Assistant Professor (Hansen, Killough) and Professor (Moffatt), Divisions of Child Abuse and Neglect, Professor (Moffatt), Emergency Medicine, Assistant Professor (Hansen, Killough) and Professor (Moffatt, Knapp), Department of Pediatrics, Assistant Professor (Killough) and Professor (Knapp), Department of Graduate Medical Education, Children's Mercy Hospital, University of Missouri–Kansas City School of Medicine, Kansas City, MO.

The authors, faculty, and staff in a position to control the content of this CME activity and their spouses/life partners (if any) have disclosed that they have no financial relationships with, or financial interest in, any commercial organizations pertaining to this educational activity.

Reprints: Jennifer B. Hansen, MD, Division of Child Abuse and Neglect Office, Children's Mercy Hospital, 2401 Gillham Rd, Kansas City, MO, 64108 (e-mail: jbhansen@cmh.edu).

ISSN: 0749-5161

peripapillary meaning the area immediately around the optic nerve, posterior pole including the macula lutea and peripapillary areas, paravascular indicating along the retinal vessel arcades, midperipheral describing the area of the retina outside of the posterior pole to the peripheral retina, and the peripheral retina including the ora serrata.[4] Retinal hemorrhages in AHT can vary in severity, from none to mild (with few, intraretinal hemorrhages confined to posterior pole) to moderate or severe (with too numerous to count, multilayered hemorrhages that extend to the ora serrata).[4] (Figs. 1–4).

Retinoschisis is a term used to describe cleavage of the layers of the retina, with or without blood accumulating in the resulting cavity.[7] Blood may either partially or completely fill the cavity. In AHT, retinoschisis tends to be noted in the macula lutea and under the ILM. Retinoschisis has also been noted in other areas of the retina, including the peripheral retina, in cases of AHT.[7] If a major vessel supplying the retina has local bleeding, retinoschisis may form directly over the vessel.[5] In this instance, other disorders predisposing to vessel leak should be considered, in addition to trauma. A fold in the retina may develop as an arc at the edge of the schisis cavity and may be hemorrhagic or hypopigmented due to disruption of the retinal pigmented epithelium.[7] These folds are termed perimacular or paramacular folds. Pan retinal involvement, perimacular folds, and macular retinoschisis have been found to be nearly 100% specific for AHT and yield a very high positive predictive value (Fig. 5).[8] The only other published events associated with perimacular folds and macular retinoschisis in young children are fatal motor vehicle collisions,[9] fatal falls from a significant height,[10] and fatal crush head injuries.[11–13]

The number, types, and distribution of RHs visualized through a pharmacologically dilated pupil, by an ophthalmologist using the technique of indirect ophthalmoscopy, are essential information acquired through ophthalmological consultation. Although there is no standard for describing RHs, some protocols have been suggested, and experts agree that the documentation should include a description of the number, type, and extent of hemorrhage, and any diagnostic pattern should be reported.[5] Traumatic retinoschisis and retinal folds may also be visualized during the examination. All children with intracranial hemorrhage, for whom there is concern of AHT, should undergo ophthalmological consultation. A detailed descriptive note with schematic drawings, and accompanied by contact-based, wide field retinal photography if available, should result from the consultation. Dedicated eye examination should also be considered for children with external evidence of ocular and/or periocular injury, or in cases of unexplained loss of vision.[5]

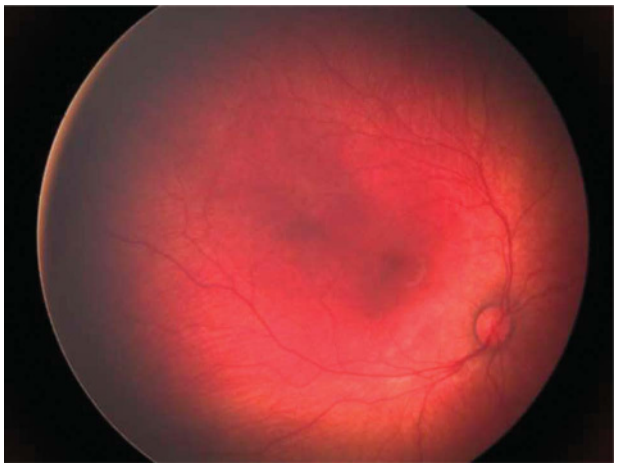

FIGURE 1. Normal fundus. Image credit, Laura S. Plummer, MD.

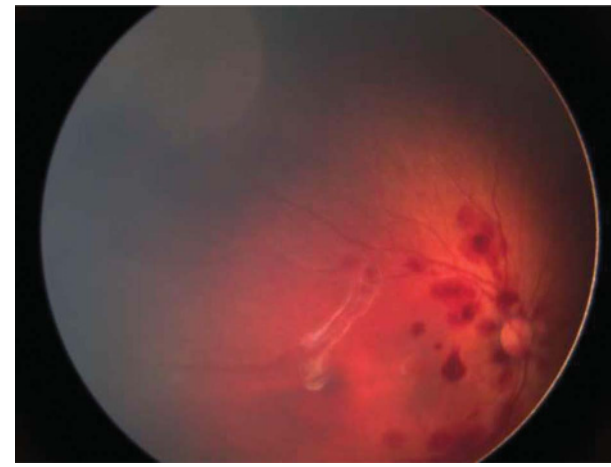

FIGURE 2. Nonspecific RH confined to the posterior pole. Image credit, Laura S. Plummer, MD.

PATHOPHYSIOLOGY AND DIFFERENTIAL DIAGNOSIS

Abusive head trauma is the most common cause of subdural hemorrhage in infants, and RH may be seen in as many as 85% to 100% of fatal cases of AHT.[6,14–16] Consideration of AHT as a possible cause for intracranial hemorrhage in an infant or young child should prompt medical providers to evaluate for RH. Abusive head trauma should be considered in situations in which there are inconsistent histories to explain injuries, when the provided history does not adequately explain the severity or type of injury (such as a simple, short distance fall given as the explanation for a critically ill child), or when the reported mechanism of injury is inconsistent with the developmental abilities of the child.[17] When there is reasonable suspicion for abuse as a possible mechanism, medical providers are mandated by law to report the concern for abuse to Child Protective Services, law enforcement, or other designated governmental agency for investigation. Additional medical evaluation to assess for occult injuries is also needed, including detailed physical examination, skeletal survey, and laboratory evaluation for occult abdominal injury.[18–23] If there is no intracranial hemorrhage, dedicated eye examination

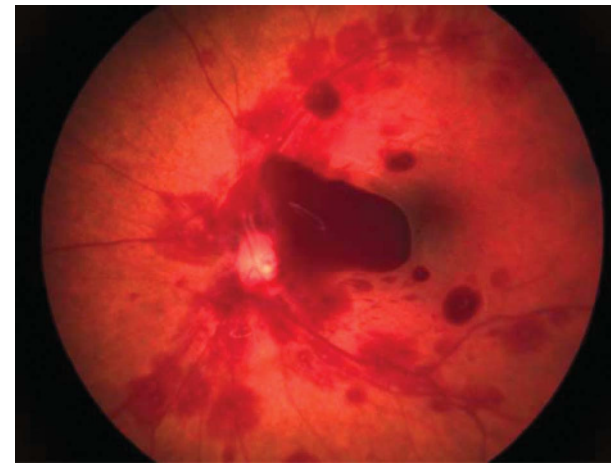

FIGURE 3. Multilayer RH, particularly distributed along the vascular arcades and with a large preretinal hemorrhage versus macular retinoschisis. Image credit, Laura S. Plummer, MD.

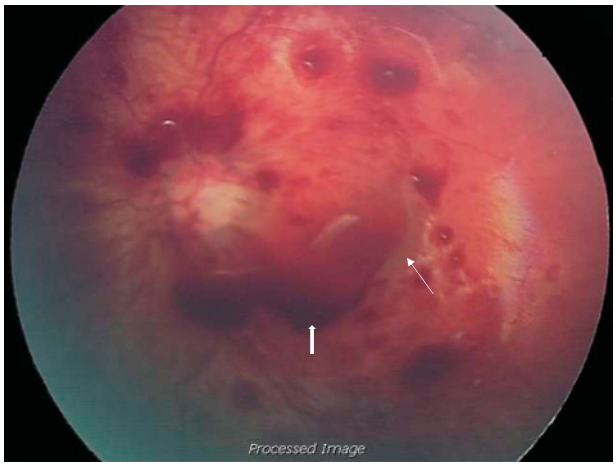

FIGURE 4. Too numerous to count, multilayer RHs with macular schisis (thick arrow) and a perimacular fold (thin arrow). Image credit, Laura S. Plummer, MD.

is generally not needed, even in the setting of other extracranial abusive injuries.[24]

In children with intracranial hemorrhage, consultation with an ophthalmologist for dilated fundoscopic examination with indirect ophthalmoscopy is indicated.[25] This consultation should preferably be performed within the first 24 hours of presentation, and certainly within 72 hours. Retinal hemorrhages cannot be specifically dated, but a recent study evaluated the natural history of RH due to accidental and abusive head injuries.[26] The authors found that intraretinal hemorrhages clear quickly, most within days, and nearly all by 1 to 2 weeks, but preretinal hemorrhages persisted much longer. Too numerous to count RHs were not seen beyond a few days, emphasizing the importance of prompt examination. Monitoring of a child's neurologic status should not preclude retinal evaluation. If needed, use of short-acting mydriatics, examination of 1 eye at a time, or examination through an undilated pupil allow for continued monitoring. For children who die from unexplained causes and children who die from abusive injury, postmortem examination is recommended.[24,25] A study of RH and related findings in AHT and accidental head injury (AHI) determined that when RHs are found in a child, there is a 91% (odds ratio, 14.7) probability that they are due to AHT.[27]

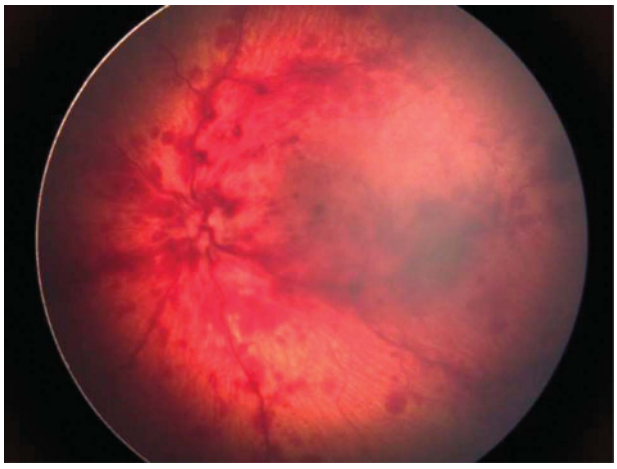

FIGURE 5. Too numerous to count, multilayer RHs extending beyond the posterior pole or to the peripheral limits of the retinal camera image. Image credit, Laura S. Plummer, MD.

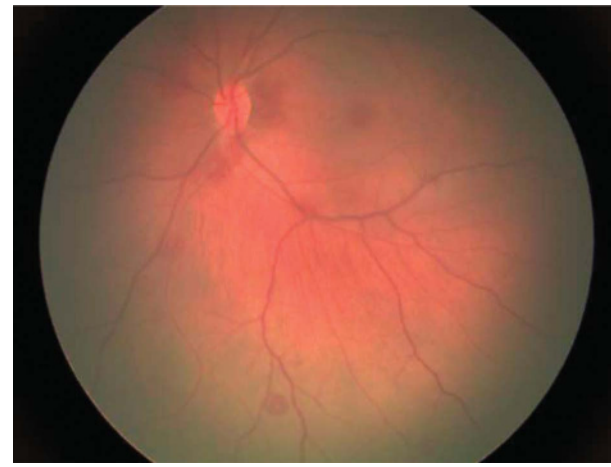

FIGURE 6. Retinal hemorrhages from birth. Image credit, Laura S. Plummer, MD.

Therefore, although the likelihood is high that RH in an infant are due to AHT, a differential diagnosis that considers other possible causes of RH is prudent. Although the presence of RH in a critically ill child is considered an important clinical indicator of abuse, RH in general is not specific to AHT, and other causes for RH do exist.[6] The number, pattern, and severity of RH are useful features that can help distinguish abusive injury from other possible causes.[15,16,28–35] The differential diagnosis for RH in young children is broad and could include birth trauma, coagulation disorders, AHI, meningitis, sepsis, leukemia, increased intracranial pressure, vasculitis, some retinal disorders, and metabolic disorders; other causes have also been theorized.[6,36] The majority of these conditions should be easily distinguished based on historical, laboratory, and clinical evaluation.

RETINAL HEMORRHAGE IN CHILD ABUSE

As many as 2/3 of infants diagnosed with AHT are found to have severe RH that are bilateral, involve multiple layers of the retina, are too numerous to count, and extend to the ora serrata.[6,30,35,37] Severe RH involving only one eye has also been reported.[38] Retinoschisis cavities are extremely sensitive for AHT as they are almost exclusively associated with AHT.[6,39] Evidence suggests that the major factor in the development of severe RH seen in AHT is vitreoretinal traction.[6,39] In infants and young children, the vitreous body is most securely attached to the retina along the vascular arcades, at the posterior pole, and at the ora serrata; these areas of attachment mirror areas commonly affected by RH and retinoschisis (posterior pole). When acceleration-deceleration forces are applied in a head injury event, traction is applied to the retina at these points of attachment, and the "pulling" forces then cause small points of tissue stress and hemorrhage, or physical separation of the layers of the retina leading to retinoschisis. Similar patterns of retinal injury are not observed in other types of traumatic injury or in medical conditions.

BIRTH

Birth is the most common cause of RH in infants with the majority resolving within the first few weeks of life. A systematic review found that RH in newborns correlated with the mode of delivery; occurring in 7% of cesarean deliveries, 25% of spontaneous vaginal deliveries, and 42% of vacuum extractions.[40] The mechanism for the development of RH during birth is not definitively known, but may be related to hemodynamic changes,

prostaglandin release, or compression and decompression forces on the head and chest, or to some combination of these.[36] Birth-related hemorrhages are characteristically bilateral, located primarily in the posterior pole, intraretinal, and range from mild to severe[40]; retinoschisis has not been reported.[6] In the systematic review by Watts et al, the majority of birth-related RH resolved within 2 weeks, with only severe RH persisting longer, and 97% of RH resolved completely within 42 days.[40] Based on the available evidence, it is generally concluded that RH observed after 6 weeks of age should not be attributed to birth.[6,26,40,41] (Fig. 6).

ACCIDENTAL HEAD INJURY

Retinal hemorrhages are seen in 3% to 5% of children with AHI.[4,27,41] In distinguishing between AHI and AHT as the cause of RH, the clinician must consider the history and the mechanism of injury in conjunction with the appearance of the hemorrhages and the available research evidence. When RHs occur with AHI, the mechanism is usually a single blunt force impact, as in a simple short fall. The RHs of AHI are usually confined to the posterior pole, few in number and unilateral.[4,30,41,42] (Fig. 3) More severe RH, including retinoschisis, have been described only in severe, high-energy injury mechanisms, such as fatal high speed motor vehicle crashes, especially with roll-overs, fatal crushing head injury, or major falls (>10 ft); the history in these cases is clear and other injuries sustained are consistent with the severity of trauma.[9,11–13,35,43] The postulated mechanism for development of RH in AHI includes increased intracranial pressure as a contributor, which is described in more detail below. However, even in children with AHI who experienced increased intracranial pressure, RH is exceedingly rare, and, when present, is nonspecific with few hemorrhages confined to posterior pole.[39,42] (Fig. 2).

INTRACRANIAL PRESSURE

Courtroom arguments in AHT cases have raised the question of whether increased intracranial pressure (ICP) is a mechanism of RH in children. Terson syndrome is the association of intraocular blood with intracranial blood and increased ICP. It is seen in adults who experience rapid elevations of pressure, typically due to subarachnoid hemorrhage or ruptured cerebral aneurysm. The proposed mechanism by which elevated ICP can lead to RH is via increased resistance and/or obstruction of venous outflow from the eye due to elevated pressures in the head and optic nerve sheath, whereas arterial inflow continues unrestricted. Retinal hemorrhage patterns consistent with retinal vein occlusion are very specific and should be easily recognized by an ophthalmologist; this pattern of RH is not typical of AHT cases.[6,36,39] In addition, causes of increased ICP unrelated to trauma are not associated with significant RH in children. Binenbaum et al studied the patterns of RH in children 3 to 17 years (mean age, 12 years) with increased ICP from etiologies other than trauma.[44] They found that RH were rare and, when present, were associated with markedly elevated opening pressures, were intraretinal, and invariably located adjacent to a swollen optic disc. Shiau and Levin[45] explored the arguments for and against RH caused by increased ICP. They noted that extensive RH in the well-described pattern of AHT had not been observed in children with increased ICP from nonabusive causes. In addition, children with elevated ICP from hydrocephalus and/or shunt malfunction do not have RH.[45–47]

CARDIOPULMONARY RESUSCITATION WITH CHEST COMPRESSIONS

Chest compressions (CCs) have been proposed as a risk factor for RH, but multiple studies have shown that receiving cardiopulmonary resuscitation (CPR) does not increase the prevalence or cause RH.[35,48] Rather, current evidence consistently limits the finding of bilateral, multilayered RH post CPR CCs to children who have other known causes of RH, including major trauma, sepsis, or severe coagulopathy alone or in combination.[35,48–51] Two mechanisms have been proposed to explain the presence of RH after CPR. The first is reperfusion injury of the retina due to hypoxia-related tissue damage after prolonged CPR, but multiple clinical and animal model studies have not found this association.[36,39] The second proposed mechanism is that increased intrathoracic pressure caused by repeated CC can result in RH in young children. Purtscher syndrome is hemorrhagic retinopathy sometimes seen in adults who sustain severe crush injury to the chest; this results in a specific pattern of RH and hexagonal white retinal patches, which are only very rarely seen in children, and are characteristic and easily recognized by an ophthalmologist. Finally, multiple studies have evaluated the association between RH in infants and other medical conditions associated with increased intrathoracic pressure, including severe cough, respiratory distress, forceful vomiting, and seizures, and have found no association with significant RH.[6,36]

HEMATOLOGICAL DISORDERS AND ABNORMALITIES

Mild RHs occur as a nonspecific finding in 5% of children with leukemia, usually resulting from secondary complications such as thrombocytopenia or coagulopathy.[52] Severe RH have not been described in the setting of coagulopathy alone.[6,35,36] A prospective study of critically ill children without AHT found RH to be rare, occurring in 15% of children, with the majority of these having mild, nonspecific RH.[35] Severe, multilayer RH were only seen in critically ill children with severe coagulopathy and other significant conditions, including late-onset hemorrhagic disease of the newborn and trauma, leukemia and severe sepsis, and fatal accidental trauma.[35]

HYPOXIA

Hypoxia is a common reason for hospitalization in the pediatric population and has also been proposed as a possible cause of RH. It has been theorized that hypoxia leading to brain injury and swelling can lead to increased ICP, which was discussed previously, and that hypoxic damage to the endothelium of blood vessels can result in leaking of blood and subsequent hemorrhage formation.[6,36,53] There is no evidence to suggest any association between RH and other clinical presentations of hypoxia including acute life-threatening events, now referred to a brief resolved unexplained events, sudden unexplained infant death, congenital heart disease, or vascular fistulas. Hypoxia has not been credibly shown to be a cause of RH.[6,36,54]

The complete differential diagnosis for RH in infants is extensive and detailed reviews of the literature have been published.[41] The differential can be narrowed in the majority of cases, however, by a thorough history and physical examination: the extent, location, and appearance of the RH (Table 1) and on the results of accompanying laboratory testing and radiological imaging.

AREAS FOR RESEARCH

Retinal hemorrhages can be detected on magnetic resonance imaging (MRI), particularly well using gradient recalled echo imaging, which is an MRI sequence that uses the magnetic susceptibility of iron in red blood cells. An area of signal void larger on gradient recalled echo than the expected true signal abnormality in the orbit can help increase the detection of RH.[55] Correlation of RH described by ophthalmological examination or captured

TABLE 1. Differential Diagnosis for Retinal Hemorrhages and Typical Appearance[14]

Etiology of RH	Appearance of RH
Birth trauma	• Usually intraretinal, confined to posterior pole, but can be numerous and extend to the periphery resembling the pattern seen with AHT • Most resolve within 2 weeks, essentially all gone by 6 weeks of age • Retinoschisis is not reported
AHI	• Usually unilateral, confined to the posterior pole, few in number, with nonspecific pattern • Retinoschisis only reported in severe, fatal AHI
AHT	• Severe, multilayer, too numerous to count, extending to periphery • Usually bilateral, but may be unilateral • With or without retinoschisis
Terson syndrome	• Pattern of central retinal vein obstruction: RH (intraretinal and preretinal) radiating centripetally from the optic nerve • Dilated and tortuous retinal veins • Exceedingly rare in children • Retinoschisis is not reported
Purtscher retinopathy	• Few RH, with numerous polygonal white patches on the retina • Rarely seen in children • Retinoschisis is not reported
RH associated with coagulopathy and/or critically ill children	• Few, nonspecific pattern, typically limited to posterior pole • Retinoschisis is not reported
Increased ICP	• Few, on or adjacent to a swollen optic disc • Retinoschisis is not reported

by retinal camera imaging to visualization of RH on MRI in 77 children, aged 1 month to 2 years, demonstrated a specificity of 61% and sensitivity of 100%.[55] High-grade RH, per the classification scheme derived by Binenbaum et al,[56] were identified more often on MRI compared with low-grade RH in this retrospective study.[55] Magnetic resonance imaging may be advantageous over clinical examination in terms of examination of the peripheral retina as indirect ophthalmoscopy has limitations in visualization of the peripheral retina. Also, MRI demonstrative of RH may increase diagnosis of AHT in cases where young infants present with nonspecific symptoms.

Ophthalmic point-of-care ultrasonography (POCUS) has been used in children to detect increased ICP and in adults for several different retinal pathologies. Ophthalmic POCUS was used in the pediatric intensive care unit over a closed eyelid for a series of 11 critically ill infants, aged 6 weeks to 3 years and diagnosed with AHT, to identify traumatic retinoschisis when dilated eye examination was delayed.[57] In 7 of the 11 infants, both RH and retinoschisis was identified. Ophthalmic POCUS may be a promising tool to identify RH and retinoschisis, particularly when dilated eye examination must be delayed. Limitations may include the use of POCUS on awake, active young children, as well as cases of RH without accompanying retinoschisis.

OUTCOMES

Fortunately, visual outcome is good for nearly half of the surviving infants and young children who sustain ophthalmological injury as a result of AHT.[4] Severe RH and retinoschisis can resolve without long-term sequelae.[5] Visual potential can be limited by retinal scarring resulting from RH and retinoschisis, optic nerve atrophy, and most commonly by cortical visual impairment associated with brain injury involving the visual cortex.[4,5] Vitreous hemorrhage, if large, can cause amblyopia, and in some cases vitrectomy is required. Treatment of amblyopia and correction of refractive errors can improve visual function. Close follow-up by an ophthalmologist, after initial eye examination, is essential though to ensure best outcome.

SUMMARY

Many causes exist for RH in infants and children. However, most medical and accidental traumatic causes result in a pattern of RH that is nonspecific and not typical of that seen in AHT, and as such can be easily distinguished from the pattern and distribution of RH seen in AHT. In children with intracranial hemorrhage and concerns for abuse, the finding of severe, multilayered RHs extending to the periphery of the retina is very highly specific for abuse as the cause of the findings, especially if retinoschisis is present. Dilated eye examination by an ophthalmologist, preferably within 24 hours of presentation, is highly recommended for all children with intracranial hemorrhage and concern for AHT.

REFERENCES

1. Parks SE, Annest JL, Hill HA, et al. Pediatric abusive head trauma: recommended definitions for public health surveillance and research. Atlanta (GA): Centers for Disease Control and Prevention; 2012. Available at: https://www.cdc.gov/violenceprevention/pdf/pedheadtrauma-a.pdf. Accessed September 19, 2016.
2. Christian CW, Block R Committee on child abuse and neglect; American Academy of Pediatrics. Abusive head trauma in infants and children. *Pediatrics*. 2009;123:1409–1411.
3. Schuenke M, Schulte E, Schumacher U. Eye and orbit. In: Ross LM, Lamperti ED, Taub E, eds. *Atlas of Anatomy Head and Neuroanatomy*. New York, NY: Thieme; 2010:120–139.
4. Binenbaum G, Forbes BJ. The eye in child abuse: key points on retinal hemorrhages and abusive head trauma. *Pediatr Radiol*. 2014;44(suppl 4): S571–S577.
5. Levin AV, Morad Y. Ocular manifestations of child abuse. In: Reece RM, Christian CW, eds. *Child Abuse Medical Diagnosis & Management*. 3rd ed. Oak Grove, IL: American Academy of Pediatrics; 2009:211–225.
6. Levin AV. Retinal hemorrhage in abusive head trauma. *Pediatrics*. 2010; 126:961–970.
7. Levin AV. Eye injuries in child abuse. In: Jenny C, ed. *Child Abuse and Neglect Diagnosis, Treatment, and Evidence*. 1st ed. St Louis, MO: Elsevier Saunders; 2011:402–412.
8. Bhardwaj G, Jacobs MB, Martin FJ, et al. Photographic assessment of retinal hemorrhages in infant head injury: the Childhood Hemorrhagic Retinopathy Study. *J AAPOS*. 2017;21:28–33.e2.
9. Kivlin JD, Currie ML, Greenbaum VJ, et al. Retinal hemorrhages in children following fatal motor vehicle crashes: a case series. *Arch Ophthalmol*. 2008;126:800–804.
10. Reddie IC, Bhardwaj G, Dauber SL, et al. Bilateral retinoschisis in a 2-year-old following a three-storey fall. *Eye (Lond)*. 2010;24:1426–1427.

11. Lantz PE, Sinal SH, Stanton CA, et al. Perimacular retinal folds from childhood head trauma. *BMJ*. 2004;328:754–756.
12. Lueder GT, Turner JW, Paschall R. Perimacular retinal folds simulating nonaccidental injury in an infant. *Arch Ophthalmol*. 2006;124:1782–1783.
13. Watts P, Obi E. Retinal folds and retinoschisis in accidental and non-accidental head injury. *Eye (Lond)*. 2008;22:1514–1516.
14. Case ME. Inflicted traumatic brain injury in infants and young children. *Brain Pathol*. 2008;18:571–582.
15. Bhardwaj G, Chowdhury V, Jacobs MB, et al. A systematic review of the diagnostic accuracy of ocular signs in pediatric abusive head trauma. *Ophthalmology*. 2010;117:983–992.e17.
16. Morad Y, Kim YM, Armstrong DC, et al. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. *Am J Ophthalmol*. 2002;134:354–359.
17. Chadwick DL, Bertocci G, Castillo E, et al. Annual risk of death resulting from short falls among young children: less than 1 in 1 million. *Pediatrics*. 2008;121:1213–1224.
18. Rubin DM, Christian CW, Bilaniuk LT, et al. Occult head injury in high-risk abused children. *Pediatrics*. 2003;111(6 pt 1):1382–1386.
19. Laskey AL, Holsti M, Runyan DK, et al. Occult head trauma in young suspected victims of physical abuse. *J Pediatr*. 2004;144:719–722.
20. Lane WG, Dubowitz H, Langenberg P. Screening for occult abdominal trauma in children with suspected physical abuse. *Pediatrics*. 2009;124: 1595–1602.
21. American Academy of Pediatrics Section on Radiology. Diagnostic imaging of child abuse. *Pediatrics*. 2009;123:1430–1435.
22. Expert Panel on Pediatric ImagingWootton-Gorges SL, Soares BP, et al. ACR Appropriateness Criteria® suspected physical abuse-child. *J Am Coll Radiol*. 2017;14(5S):S338–S349.
23. Lindberg DM, Shapiro RA, Blood EA, et al. Utility of hepatic transaminases in children with concern for abuse. *Pediatrics*. 2013;131:268–275.
24. Greiner MV, Berger RP, Thackeray JD, et al. Dedicated retinal examination in children evaluated for physical abuse without radiographically identified traumatic brain injury. *J Pediatr*. 2013;163:527–531.
25. Levin AV, Christian CW American Academy of Pediatrics Committee on Child Abuse and Neglect and Section on Ophthalmology. Clinical report—the eye examination in the evaluation of child abuse. *Pediatrics*. 2010;126:376–380.
26. Binenbaum G, Chen W, Huang J, et al. The natural history of retinal hemorrhage in pediatric head trauma. *J AAPOS*. 2016;20:131–135.
27. Maguire SA, Watts PO, Shaw AD, et al. Retinal haemorrhages and related findings in abusive and non-abusive head trauma: a systematic review. *Eye (Lond)*. 2013;27:28–36.
28. Feldman KW, Bethel R, Shugerman RP, et al. The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics*. 2001;108:636–646.
29. Kemp AM. Investigating subdural haemorrhage in infants. *Arch Dis Child*. 2002;86:98–102.
30. Bechtel K, Stoessel K, Leventhal JM, et al. Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. *Pediatrics*. 2004;114:165–168.
31. Hobbs C, Childs AM, Wynne J, et al. Subdural haematoma and effusion in infancy: an epidemiological study. *Arch Dis Child*. 2005;90:952–955.
32. Vinchon M, deFoort-Dhellemmes S, Desurmont M, et al. Accidental and nonaccidental head injuries in infants: a prospective study. *J Neurosurg*. 2005;102(suppl 4):380–384.
33. Maguire S, Pickerd N, Farewell D, et al. Which clinical features distinguish inflicted from non-inflicted brain injury? A systematic review. *Arch Dis Child*. 2009;94:860–867.
34. Maguire SA, Kemp AM, Lumb RC, et al. Estimating the probability of abusive head trauma: a pooled analysis. *Pediatrics*. 2011;128:e550–e564.
35. Agrawal S, Peters MJ, Adams GG, et al. Prevalence of retinal hemorrhages in critically ill children. *Pediatrics*. 2012;129:e1388–e1396.
36. Forbes BJ, Rubin SE, Margolin E, et al. Evaluation and management of retinal hemorrhages in infants with and without abusive head trauma. *J AAPOS*. 2010;14:267–273.
37. Vinchon M, de Foort-Dhellemmes S, Desurmont M, et al. Confessed abuse versus witnessed accidents in infants: comparison of clinical, radiological, and ophthalmological data in corroborated cases. *Childs Nerv Syst*. 2010; 26:637–645.
38. Gilles EE, McGregor ML, Levy-Clarke G. Retinal hemorrhage asymmetry in inflicted head injury: a clue to pathogenesis? *J Pediatr*. 2003;143:494–499.
39. Levin AV. Retinal hemorrhages: advances in understanding. *Pediatr Clin North Am*. 2009;56:333–344.
40. Watts P, Maguire S, Kwok T, et al. Newborn retinal hemorrhages: a systematic review. *J AAPOS*. 2013;17:70–78.
41. Levin AV. Retinal hemorrhages and child abuse. In: David T, ed, Recent Advances in Paediatrics, *Volume 18.* London, England: Churchill Livingstone; 2000:151–219.
42. Forbes BJ, Cox M, Christian CW. Retinal hemorrhages in patients with epidural hematomas. *J AAPOS*. 2008;12:177–180.
43. Moran K, Reddie I, Jacobs M. Severe hemorrhagic retinopathy and traumatic retinoschisis in a 2 year old infant, after an 11 metre fall onto concrete. *Acta Paediatr*. 2008;97(suppl 459):149.
44. Binenbaum G, Rogers DL, Forbes BJ, et al. Patterns of retinal hemorrhage associated with increased intracranial pressure in children. *Pediatrics*. 2013;132:e430–e434.
45. Shiau T, Levin AV. Retinal hemorrhages in children: the role of intracranial pressure. *Arch Pediatr Adolesc Med*. 2012;166:623–628.
46. Tzekov C, Cherninkova S, Gudeva T. Neuroophthalmological symptoms in children treated for internal hydrocephalus. *Pediatr Neurosurg*. 1991–1992; 17:317–320.
47. Nazir S, O'Brien M, Qureshi NH, et al. Sensitivity of papilledema as a sign of shunt failure in children. *J AAPOS*. 2009;13:63–66.
48. Gilliland MG, Luckenbach MW. Are retinal hemorrhages found after resuscitation attempts? A study of the eyes of 169 children. *Am J Forensic Med Pathol*. 1993;14:187–192.
49. Odom A, Christ E, Kerr N, et al. Prevalence of retinal hemorrhages in pediatric patients after in-hospital cardiopulmonary resuscitation: a prospective study. *Pediatrics*. 1997;99:E3.
50. Kanter RK. Retinal hemorrhage after cardiopulmonary resuscitation or child abuse. *J Pediatr*. 1986;108:430–432.
51. Pham H, Enzenauer RW, Elder JE, et al. Retinal hemorrhage after cardiopulmonary resuscitation with chest compressions. *Am J Forensic Med Pathol*. 2013;34:122–124.
52. Russo V, Scott IU, Querques G, et al. Orbital and ocular manifestations of acute childhood leukemia: clinical and statistical analysis of 180 patients. *Eur J Ophthalmol*. 2008;18:619–623.
53. Hansen JB, Frazier T, Moffatt M, et al. Evaluation of the hypothesis that choking/ALTE may mimic abusive head trauma. *Acad Pediatr*. 2017;17: 362–367.
54. Richards PG, Bertocci GE, Bonshek RE, et al. Shaken baby syndrome. *Arch Dis Child*. 2006;91:205–206.
55. Beavers AJ, Stagner AM, Allbery SM, et al. MR detection of retinal hemorrhages: correlation with graded ophthalmologic exam. *Pediatr Radiol*. 2015;45:1363–1371.
56. Binenbaum G, Mirza-George N, Christian CW, et al. Odds of abuse associated with retinal hemorrhages in children suspected of child abuse. *J AAPOS*. 2009;13:268–272.
57. Riggs BJ, Trimboli-Heidler C, Spaeder MC, et al. The use of ophthalmic ultrasonography to identify retinal injuries associated with abusive head trauma. *Ann Emerg Med*. 2016;67:620–624.