J Fam Viol (2016) 31:797–804
DOI 10.1007/s10896-016-9840-0

ORIGINAL ARTICLE

Ophthalmologic Concerns in Abusive Head Trauma

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Published online: 8 September 2016

Abstract When Abusive Head Trauma (AHT) is suspected in a child, a dilated eye examination by an ophthalmologist is an essential part of the medical workup, as the presence and pattern of retinal hemorrhages can have a high positive predictive rate for abusive head injury. This article proposes to review the clinical presentation, pathophysiology, natural history, sequelae, and differential diagnosis of retinal hemorrhages and other ocular lesions associated with AHT.

Keywords Abusive head trauma · Ocular lesions · Retinal hemorrhages · Retinoschisis · Retinal folds · Dilated eye exam

Introduction

It has been estimated that roughly 4 % to 6 % of child abuse victims present first to an ophthalmologist, usually because of physical abuse. (Friendly 1971) In abusive head trauma (AHT) there may be no external evidence of direct ocular trauma such as periorbital ecchymosis, lid swelling, lacerations, or subconjunctival hemorrhages. The anterior segment examination of the eye is frequently unremarkable. (Vincent and Kelly 2010) Because of this, the ophthalmologist is usually not the first physician who examines the child victim of AHT. Instead, the ophthalmologist is more often consulted by other medical providers to assess for intraocular findings of abuse because of non-ocular physical findings suspicious for abuse (e.g. bruising, fractures), or the presence of unexplained neurologic symptoms in the child (e.g. apnea, seizures, or brief resolved unexplained events [formerly known as Apparent Life Threatening Event or ALTE]). (Levin 1990)

In a child with intracranial injury, retinal hemorrhages, along with rib fractures, are the physical findings most predictive of AHT (Maguire et al. 2009) Therefore, when AHT is suspected in a child less than 5 years old, a prompt complete ophthalmologic examination is an integral part of the medical evaluation. (Levin et al. 2010) This includes full indirect ophthalmoscopic examination through a dilated pupil, with detailed documentation of the description of any retinal hemorrhages found, preferably within 24 h of admission and no later than 72 h as hemorrhages may worsen or resolve in this time period. (Levin 2010; Levin et al. 2010) Photodocumentation of abusive ophthalmologic findings is often helpful, although not obligatory. Several hand-held digital cameras are available for this purpose. A detailed descriptive note with or without a hand-drawn picture is adequate in many cases. Additional testing with optical coherence tomography (Muni et al. 2010; Sturm et al. 2008) or intravenous fluorescein angiography (Bielory et al. 2012; Goldenberg et al. 2010) may also be useful.

Although non-ophthalmologists have a high rate of accuracy in determining whether retinal hemorrhage is present or absent, false positive and false-negative examinations have been reported. (Kivlin et al. 2000; Morad et al. 2003) Non-ophthalmologists

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also do not have the experience or tools to allow for complete descriptions of the pattern, number, and type of hemorrhages, all of which are important considering an appropriate differential diagnosis. (Levin 2010) In cases of child fatality when AHT is a possibility, removal of the eye and orbital tissue can reveal abnormalities not suspected before death such as optic nerve sheath hemorrhages or orbital fat hemorrhages. (Gilliland et al. 2007; Levin et al. 2010; Wygnanski-Jaffe et al. 2006)

Ocular Findings in Abusive Head Trauma

Retinal Hemorrhages In child physical abuse, the most common ophthalmologic finding is the presence of retinal hemorrhages. In AHT characterized by repetitive acceleration-deceleration with or without blunt impact trauma (shaken baby syndrome), the incidence of retinal hemorrhages is approximately 85 %. (Kivlin et al. 2000; Morad et al. 2002) Retinal hemorrhages tend to occur more frequently in children who have died versus unimpaired survivors. (Levin et al. 2010) Retinal hemorrhages are far less common in AHT characterized only by blunt impact, similar to the low incidence observed in accidental falls. (Bhardwaj et al. 2010; Levin 2000) In cases of child physical abuse — including patients without AHT — hemorrhages are much more frequent (19 % to 73 %) than in cases of severe accidental head trauma (0 to 20 %). (Bechtel et al. 2004; Pierre-Kahn et al. 2003) In the latter, the incidence is usually 0 % to 3 % and only rises higher with severe mechanisms such as motor vehicle accidents.

Several important factors in the evaluation of retinal hemorrhages include the depth, location, and extent of the hemorrhages. (Levin 2010) The retina is a multilayered structure extending from the internal limiting membrane (ILM) adjacent to the vitreous, to the retinal pigmented epithelium adjacent to the choroid. The appearance of retinal hemorrhages is somewhat different depending on their location within those layers. Superficial blood within the nerve fiber layer is flame or splinter shaped, whereas lesions in the deeper layers of the retina have a dot or blot configuration (Fig. 1). (Levin 2010) Blood in between the ILM and the vitreous (hyaloid) causes the ILM to become elevated, thus forming a cavity into which blood enters and may even layer with separation of the cells and serum (Fig. 2). This cavity is a form of retinoschisis. Blood which breaks through the ILM may enter the potential space between the vitreous and retina where it is called preretinal (subhyaloid). The term preretinal is often misused to include some smaller lesions where a small focus of blood remains trapped under the ILM without the characteristic appearance of traumatic macular retinoschisis (see Fig. 1). Blood may also be found under the retina (subretinal) or within the vitreous (Fig. 3).

The specificity of retinal hemorrhages for AHT also depends on extent and pattern of distribution. Hemorrhages that are too numerous to count, multilayered, and extending to the ora serrata, are highly specific for AHT. (Betz et al. 1996; Binenbaum et al. 2009; S. A. Maguire et al. 2013; Togioka et al. 2009) Unilateral and asymmetric retinal hemorrhages may occur in about 3 and 20 % of cases respectively. (Arlotti et al. 2007; Breazzano et al. 2014; Morad et al. 2002) The absence of retinal hemorrhage or a small number of hemorrhages confined to the region just around the optic

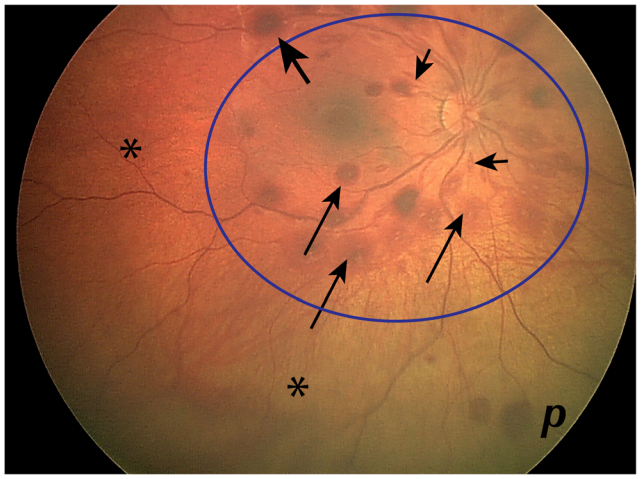

Fig. 1 Right eye of infant victim of abusive head trauma (AHT) with a moderate number of retinal hemorrhages. Blue circle denotes the posterior pole: that part of retina which encompasses the optic nerve and, temporal to the nerve, the macula. This region most commonly harbors retinal hemorrhages. Small numbers of hemorrhages in the posterior pole are less specific for AHT. Short arrows indicate flame (nerve fiber layer) retinal hemorrhages. Long thin arrows indicate blot intraretinal hemorrhages. Thick arrow indicates a hemorrhage over a blood vessel which is trapped beneath the internal limiting membrane. Asterisk (*) is the midperipheral region which lies between the posterior pole and the peripheral retina the posterior edge of which can just be visualized at p where a larger blot hemorrhage is seen

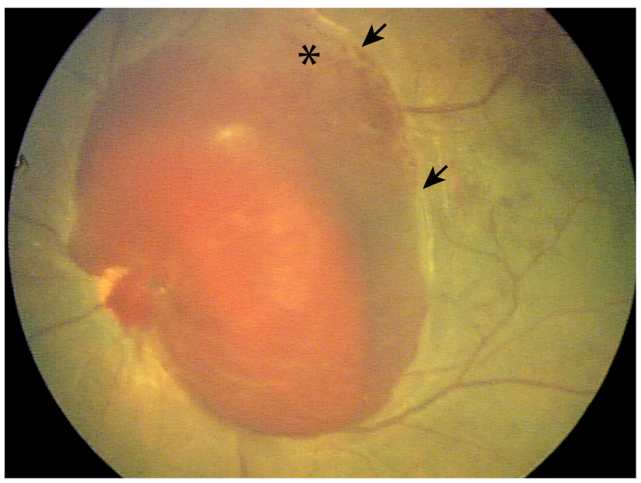

Fig. 2 Large area of macular retinoschisis in an infant victim of abusive head injury. Note that blood is filling a raised cavity, the surface of which is the internal limiting membrane. Asterisk (*) indicates area where blood is beginning to settle within the cavity leaving serum behind. Arrows indicate hypopigmented line at edge of the retinoschisis due to traumatic disruption of the retinal pigment from vitreo-retinal traction during the abusive act

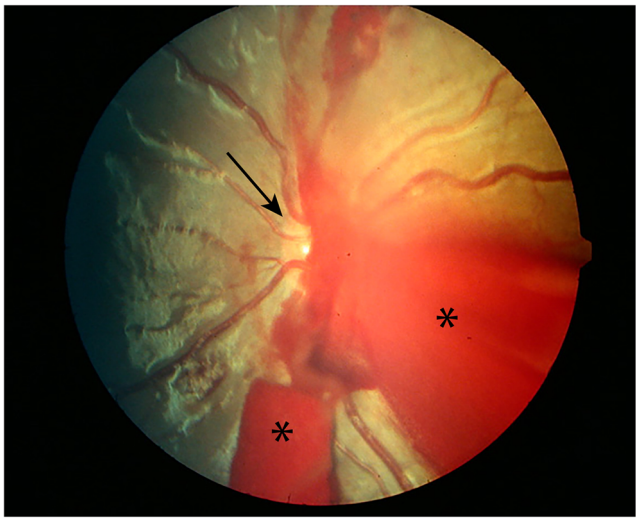

Fig. 3 Vitreous hemorrhage (*) in a victim of abusive head trauma. Note that blood is in front of the retina and obscures the view of the retina, retinal vessels and optic nerve (*arrow*)

nerve (peripapillary, Fig. 4) does not rule out AHT. Peripapillary hemorrhages may simply be a manifestation of papilledema.

Retinal hemorrhages cannot be accurately dated. Resolution rates depend on the type, location, and number of hemorrhages and may range from less than 24 h (some flame hemorrhages) to several months (vitreous hemorrhage). (McCabe and Donahue 2000) Multilayered hemorrhages rarely persist beyond a few days and a picture of predominantly preretinal hemorrhages suggests an event of several days to at least a week earlier. (Binenbaum et al. 2016) Although retinal hemorrhage severity is correlated with severity of brain injury, no single eye finding is correlated with poor long term vision. (Morad et al. 2002) The most common causes of vision loss in AHT are cortical visual impairment from the brain injury and optic nerve atrophy.

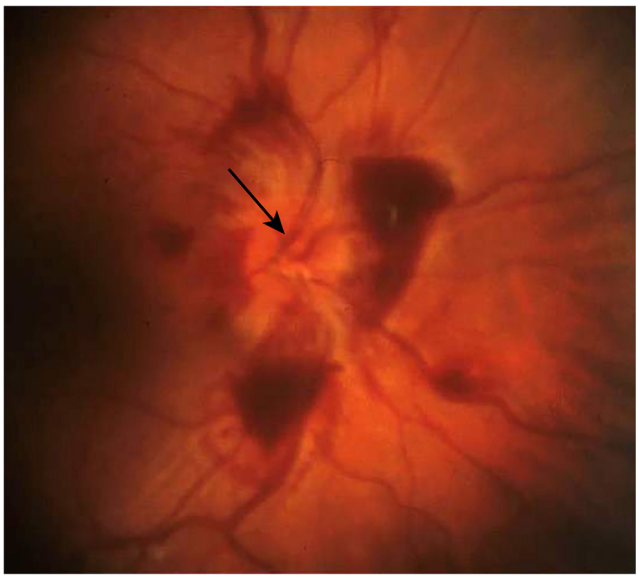

Fig. 4 Peripapillary hemorrhage. Arrow indicates the optic nerve

Risk Factors for Retinal Hemorrhages

There is an association between the severity of retinal hemorrhages (RHs) and the presence of a hypoxic-ischemic brain injury pattern as identified in Diffusion-Weighted-MRI (DW-MRI) in young children with both accidental and non-accidental head trauma. (Binenbaum et al. 2013) This is likely a proxy for the severity of brain injury rather than the true impact of hypoxia alone, as severe extensive RH do not occur in other disorders with cerebral hypoxia. Retinal hemorrhages also occur in AHT victims who do not exhibit such a pattern on DW-MRI; therefore, hypoxic-ischemic-injury is not a necessary factor for the development of severe retinal hemorrhages from inflicted head trauma. (Binenbaum et al. 2013)

A recent case control study found that in suspected victims of AHT, the presence of lethargy, altered mental status, subdural hematoma, cerebral ischemia, or diffuse axonal injury were strong risk factors for the presence of retinal hemorrhages. (Burkhart et al. 2015) Although it has been reported, RHs are rarely found in the absence of intracranial hemorrhage, even when a skull fracture was present. (Burkhart et al. 2015; Morad et al. 2004a, b; Morad et al. 2004a, b) The presence of epidural hemorrhage or a history of occipital impact may accentuate the chance of finding RH.

Retinal Folds and Traumatic Retinoschisis Retinal folds present as circular hemorrhagic or hypopigmented ridges or as lines usually around the macula (posterior retina temporal to the optic nerve) and less commonly in the more peripheral retina. They may be found at the edges of a traumatic retinoschisis but may also occur in the absence of a clinically evident schisis cavity (Fig. 5). Retinal folds occur in about 3 % of clinical cases of AHT and may be unilateral or bilateral. (Bhardwaj et al. 2010) The incidence of retinal folds is 23–42 % in severe or fatal AHT. (Bhardwaj et al. 2010; Breazzano et al. 2014) Although not required for diagnosis, in Breazzano et al.'s autopsy study of 30 infants with AHT, retinal folds were always associated with a tear of the ILM. (Breazzano et al. 2014) These authors and others have recognized the primary role of vitreoretinal traction in causing these pleats in the retina.

Traumatic retinoschisis is the splitting of the retinal layers, most often confined to the ILM, presenting as a blood filled cavity (see Fig. 2). The lesion is also thought to arise from vitreo-retinal traction as the child is submitted to repeated acceleration-deceleration forces. The vitreous may still be visualized postmortem attached to the apex of the folds or retinal cavity. Deeper splitting of the retina, and even focal retinal detachment may occur. Later, scarring can develop, leading to impaired vision if the macula is involved. Retinoschisis is not necessarily associated with a poor visual outcome.

In the absence of crush head injury, fatal motor vehicle accidents, or a fall of more than 10 m, retinal folds and traumatic retinoschisis remain specific for AHT. (Levin 2006) The

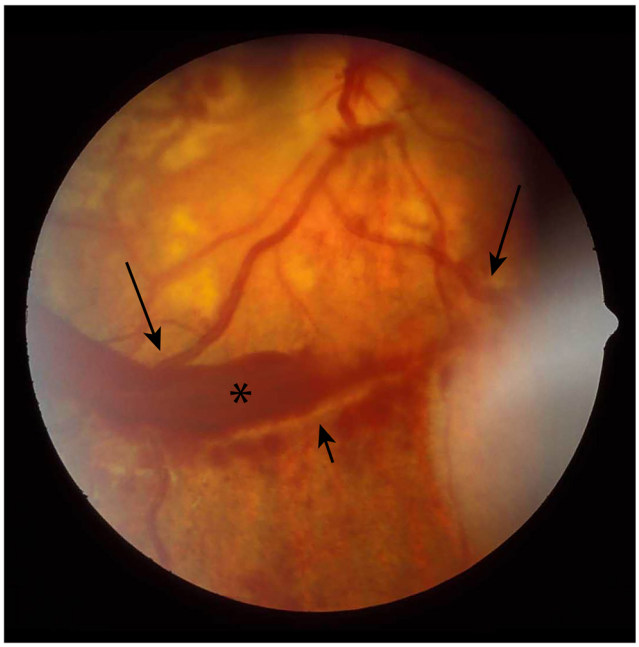

Fig. 5 Short arrow indicates a hypopigmented retinal fold in a victim of abusive head trauma. Note how blood vessels are rising (*long arrows*) to go over the fold. There is some subhyaloid hemorrhage (*) lying over the fold that has broken out of a retinoschisis cavity

same is not true for sub-ILM blood directly over retinal vessels, which may occur as a result of any cause of bleeding. Lesions similar to retinoschisis, with sub-ILM blood, have been reported after infantile ruptured aneurysm and leukemia. These differential diagnoses are readily apparent by history, examination and diagnostic testing.

Vitreous Hemorrhage A vitreous hemorrhage presents as curls, streaks, or patches of blood in the vitreous, obscuring the fundus vessels (see Fig. 3). It can occur as an extension of pre-retinal or sub-ILM hemorrhage, but may also reflect direct injury to retinal vessels and can be present at the first examination shortly after injury, or more commonly develops 1 to 3 days later from rupture of the ILM over a retinoschisis. A severe vitreous hemorrhage may require surgical intervention and is associated with a worse visual outcome.

Optic Nerve Sheath Hemorrhages Hemorrhages within the optic nerve sheath are frequently found at autopsy in cases of AHT (Fig. 6). The location can be subdural (78 %), intradural (61 %, i.e. within the dural tissue itself), subarachnoid (26 %), or a combination of these. (Wygnanski-Jaffe et al. 2006) Subdural and subarachnoid hemorrhage are non-specific and can be seen in fatal accidents and some medical causes of death. In AHT however, hemorrhage within the orbital tissues may be seen (see Fig. 6) including hemorrhage within the extraocular muscles and cranial nerve sheaths. Orbital hemorrhage is rarely seen in accidental injury, and then in association with direct orbit injury or mechanisms characterized by repeated acceleration-deceleration. (Wygnanski-Jaffe et al. 2006) The mechanism in AHT, and high incidence, seem to be related to translation of the globe in the orbit during the abusive event with direct orbital tissue injury.

Papilledema Papilledema is found in less than 10 % of abusive head trauma patients. (Morad et al. 2002).

Anterior Segment Injuries Anterior segment injuries are uncommon in AHT, but when present, they are rarely isolated and tend to be associated with severe trauma, intraocular disruption and a poor prognosis. (Levin 1990) Corneal or iris trauma, subconjunctival hemorrhages, (DeRidder et al.

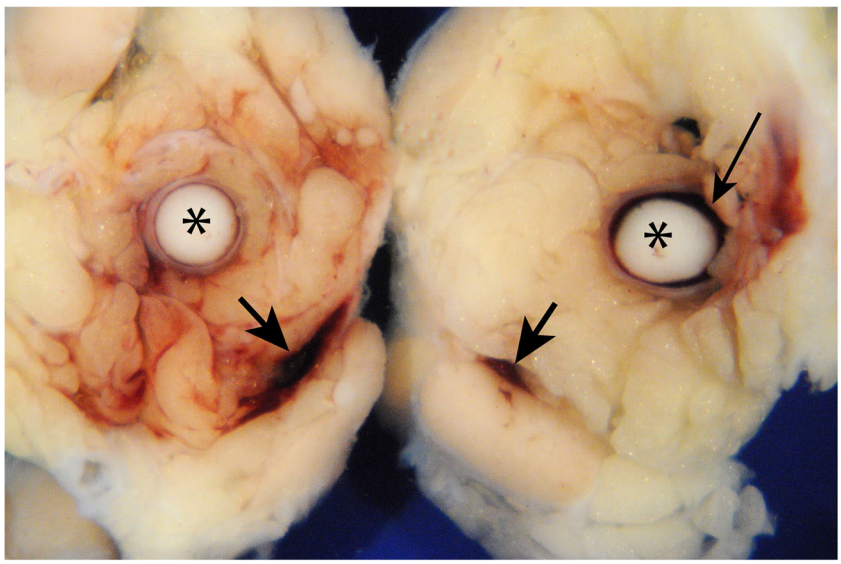

Fig. 6 Postmortem specimen from both eyes of an infant victim of abusive head trauma. Asterisks (*) indicate optic nerves. Thin arrow indicates optic nerve sheath hemorrhage of the left eye (right specimen). There is much less sheath hemorrhage of the other eye. Thick arrows indicate foci of orbital fat hemorrhage. (Specimen prepared and photographed by Dr. Ralph Eagle, Wills Eye Hospital)

2013) hyphema, glaucoma, lens subluxation, cataract, and anisocoria, have all been reported as a result of child physical abuse. (Levin 1990, 2000).

Pathophysiology of Ocular Injury in Abusive Head Trauma

Pathophysiology of Retinal Hemorrhages The pathophysiology of retinal hemorrhages has been the subject of lengthy debates in the courtroom where mechanisms including isolated blunt trauma, short falls, increased intracranial pressure, increased intrathoracic pressure related to cardiopulmonary resuscitation, and hypoxia have all been implicated. The present scientific consensus based on multiple lines of research clearly implicates repetitive acceleration-deceleration forces leading to vitreo-retinal traction as the primary etiologic factor. (Levin 2010) The robust medical literature does not support those courtroom theories. The severe rotational trauma associated with shaking apparently leads to asynchronous movements at the vitreo-retinal interface, inducing shearing stress. The pediatric vitreous is tightly adherent to the retina, in particular at the macula, blood vessels, and retinal periphery. It is interesting to note that in woodpeckers, the vitreous lacks attachments to the retina. This is one of several anatomic adaptations presumably protecting them from retinal injury during their rapid, forceful, and repetitive pecking behavior. This finding is only one of many lines of research which support the understanding that in AHT, retinal hemorrhages are caused by vitreous traction. (Wygnanski-Jaffe et al. 2007).

Using a dummy doll model, Yamazaki and colleagues studied the stress exerted on the retina and posterior pole of the retina during shaking as compared to a fall. During a single cycle of shaking, the integral of the stress was 103 Pa•s (Pascal second) as compared to 60–73 Pa•s during a single fall. This might explain why retinal hemorrhages are much more frequent during repetitive shaking than after an accidental fall. (Yamazaki et al. 2014) Finite element analysis has also pointed to the importance of vitreoretinal traction. (Rangarajan et al. 2009)

Another important factor may be disruption of retinal vessel autoregulation either at a local level due to vitreo-retina traction or due to intraorbital damage to the cranial nerves carrying autonomic supply to the globe. (Levin 2010) This may in part explain the reported potential for worsening of retinal hemorrhages after admission, aggravated perhaps by other systemic factors such as coagulopathy from brain injury. (Gilles et al. 2003) This underscores the need for prompt ophthalmic examination.

Pathophysiology of Optic Nerve Injuries The optic nerve is longer than the distance between its entry point at the apex of the bony orbit and its insertion into the eyeball. This slack allows the eyeball and orbital contents to move rhythmically when the child is submitted to repeated acceleration-deceleration forces. As the optic nerve is firmly attached both to the eyeball and the apex of the orbit, injury may occur at these tethering locations as a result of shearing forces at the points of fixation. In woodpeckers, the optic nerve is extremely short and straight with no redundancy, and the orbital bones encase the globe so that no translation of the globe is possible. (Wygnanski-Jaffe et al. 2007). This finding, along with studies of the postmortem orbital contents, support the understanding that in AHT, optic nerve injuries are caused by the translation of the globe within the orbit. (Wygnanski-Jaffe et al. 2006; Wygnanski-Jaffe et al. 2007).

Visual Outcome in Cases of Abusive Head Trauma

Retinal hemorrhages do not seem to be a major cause of visual loss unless the fovea is directly involved. (McCabe and Donahue 2000) If the fovea is obscured for a prolonged period of time, especially when the contralateral fovea is not involved, amblyopia may result.

Visual impairment and blindness are significant in long-term survivors of AHT. (Levin 2000) In addition to visual loss, patients may experience visual field loss, color vision impairment, decreased contrast sensitivity, decreased binocularity, and secondary amblyopia. Retinal detachment, cataract, and neovascular glaucoma have been reported as sequelae of AHT. (Levin 1990) Bilateral retinal non-perfusion may also occur in AHT survivors and may lead to optic nerve or retinal neovascularization. (Bielory et al. 2012) Sixth nerve palsy, nystagmus and ophthalmoplegia have been reported. Strabismus develops in up to 30 % of AHT victims. (Levin 1990) However, the main causes of serious visual impairment are severe vitreous hemorrhage, retinal scarring or fibrosis secondary to retinoschisis, optic nerve atrophy, and cortical blindness. Collectively, they occur in 25 % to 30 % of AHT survivors. (McCabe and Donahue 2000)

Differential Diagnosis of Retinal Hemorrhages

The most common cause of retinal hemorrhages in infancy is the normal birth process. A review of 13 clinical studies revealed that 25.6 % of newborns born via spontaneous vaginal deliveries had retinal hemorrhages. The rate is higher in instrument assisted delivery: 42.6 % with vacuum extraction, and 52 % in double-instrument deliveries (forceps and vacuum). (Watts et al. 2013) In Cesarean deliveries, the rate is approximately 6 %. (Watts et al. 2013) This study found that birth hemorrhages are commonly bilateral (59 %), predominantly intraretinal, and confined to the posterior pole. (Watts et al. 2013) The severity varies from "mild" (22 % to 56 %) to "severe" (18 % to 37 %). (Watts et al. 2013) The majority of RH (83 %) resolved within 10 days although isolated intraretinal hemorrhages can persist up to 58 days. (Watts et al. 2013) Those that persist are usually deeper intraretinal,

in particular foveal. Although less common, subretinal, preretinal, vitreous, and even sub-ILM bleeding can occur, but neither the retinal folds nor typical macular retinoschisis seen in AHT have ever been described. It is important to note that babies with flame hemorrhages within the first month, or deeper intraretinal hemorrhages within the first 4 to 6 weeks after birth may otherwise be indistinguishable from AHT. Hemorrhages of these types outside these time windows should not be ascribed to birth. Likewise, these time windows do not apply to calculate the timing of AHT injuries. This underscores the need for a multidisciplinary multisystem evaluation to rule out abuse.

The pathophysiology of retinal hemorrhages in the newborn is not completely understood. The higher frequency in instrument assisted deliveries suggests that head compression may play a role, as it does in the retinal hemorrhages of infant head crush injury. The surge in prostaglandins associated with the normal labor process is also implicated, as evidenced by a higher rate of retinal hemorrhages when labor is induced with prostaglandin versus oxytocin. (Schoenfeld et al. 1985)

There are multiple other causes of retinal hemorrhages in infants (See Table 1). Note that almost all of these circumstances can be diagnosed by a thorough history and attention to the presence or absence of other clinical, laboratory, and radiological findings. Extensive retinal hemorrhages have been reported infrequently in the literature in the absence of AHT. In a review of 159 non-abused, critically ill children, retinal hemorrhages were identified in 24 children (15 %), but higher numbers and extent of retinal hemorrhages were only observed after severe high-impact head trauma — such as from motor vehicle accidents or crush head injuries — or in the presence of severe coagulopathy, leukemia, or sepsis, all circumstances that would be readily distinguished by history and laboratory testing from AHT. (Adams et al. 2013) Several excellent studies have independently shown the high correlation between retinal hemorrhage and AHT as opposed to accidental injury. (Bhardwaj et al. 2010; Binenbaum et al. 2009; S. A. Maguire et al. 2013; Togioka et al. 2009)

Conclusion

When a child is evaluated for head trauma of unknown cause, ophthalmological consultation should be sought as soon as

Table 1 Differential Diagnosis for Retinal Hemorrhages*

Injury or Condition	Discussion
Accidental trauma	— Few in number except in very severe trauma (see below) — Usually limited to posterior pole — Predominantly intraretinal and pre-retinal — Extremely rare (most studies <3 % incidence) after short falls except if there has been an epidural hemorrhage [16,50] or occipital impact
Motor vehicle crash or severe crush injury	— Easily determined by history[47]
Intraocular surgery	— Easily determined by history
Cardiopulmonary Resuscitation (CPR)	— Extremely rare,[48] few in number, posterior pole
Extracorporeal Membrane Oxygenation (ECMO)	— 5 out of 37 (13 %) of ECMO patients had retinal hemorrhage.[49]
Birth	— Between 19.2 % and 37.3 % incidence in vaginal birth — 6 % incidence after C-section — see text regarding timing
Prematurity	— Retinal hemorrhage occur at the peripheral circumferential demarcation between the vascularized and avascular retina
Intracranial hypertension or Papilledema	— Small number of retinal hemorrhage on or around the optic disc[51]
Cancer	— Leukemia -usually with white retinal infiltrates
Coagulopathy/anemia	— Uncommon, few in number, posterior pole severe anemia and usually thrombocytopenia required, often with cotton wool spots
Meningitis	— More often if coagulopathy or sepsis is present — Only severe retinal hemorrhage if purulent meningitis[52] otherwise ew in number, posterior pole
Cytomegalovirus	— Necrotic retinitis
Ruptures aneurysm/arteriovenous malfomation	— May have severe extensive RH easily recognized on neuroimaging
Cyanotic congenital heart disease	Few in number, posterior pole with tortuous retinal veins
Endocarditis	Few in number, usually with white centers (Roth spots)
Hypoxia	— Few in posterior pole
Menkes disease	Causes blue sclera
Galactosemia	Vitreous hemorrhages reported
Glutaric aciduria	Rarely occurs and is confined to posterior pole
Carbon monoxide poisoning	Few in posterior pole
Other retinal disorders (e.g. juvenile X-linked retinoschisis, subretinal neovascular membrane retinal hemangioma)	Recognized by other characteristic features

*This list is incomplete

possible because the presence of retinal hemorrhages and other retinal findings beyond the neonatal period may be highly suggestive of AHT, and some of these lesions may resolve quickly. The pattern of retinal hemorrhages carries important diagnostic information, and the specificity of their association with AHT increases with the severity of the ophthalmologic findings. Ocular findings should always be interpreted in the context of the clinical presentation as assessed by a multidisciplinary multisystem evaluation.

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