Abusive head trauma (AHT), frequently termed shaken baby syndrome, refers to a constellation of clinical findings—classically the triad of subdural hemorrhage (Figure 1A), bilateral retinal hemorrhages, and anoxic encephalopathy—from which clinicians infer physical child abuse. Classic metaphyseal fractures of long bones (Figure 1B) and rib fractures (Figure 1C), especially when they are in different stages of healing, and skin ecchymoses are other clinical findings suggestive of inflicted trauma.

Several cases of AHT were reported in the early 1970s by Guthkelch and Caffey, and, ever since, multiple iterations in nomenclature have been applied to reflect the evolution in our understanding of AHT and its mechanisms of injury.\(^1,2\) The initial terminology, whiplash shaken infant syndrome, evolved to shaken impact syndrome, then infant whiplash-shake syndrome, battered-child syndrome, and, even later, nonaccidental head trauma. Recently, AHT was coined to describe infants with the aforementioned clinical findings and other evidence of physical abuse. Although shaken baby syndrome is the most widely known term, AHT is preferred by the American Academy of Pediatrics, as it is thought to encompass all mechanisms of injury.\(^3\)

A CHANCE TO MAKE A DIFFERENCE

The ophthalmologist’s role is pivotal in assisting in the diagnosis of AHT. It is estimated that approximately 5% of child abuse cases present first to an ophthalmologist.\(^4\) The anterior segment examination is typically unrevealing in AHT. Anisocoria, poor pupillary reaction, and mydriasis often indicate severe concurrent central nervous system (CNS) depression.\(^5\) Documentation of funduscopic findings by an ophthalmologist is critical in establishing a diagnosis in suspected cases of child abuse, given the medicolegal implications.

RETINAL FINDINGS IN AHT

The most common retinal manifestation of AHT is multiple retinal hemorrhages in multiple layers of the retina, seen in approximately 85% of cases. Sub–internal limiting membrane (sub-ILM), nerve fiber layer, intraretinal, and subretinal hemorrhages are seen frequently in different stages of healing, often with white centers (Figure 1D and E), which constitute fibrin aggregations.\(^6\) Retinal hemorrhages are frequently bilateral, although unilateral involvement does not rule out a diagnosis of AHT.\(^7\) Sub-ILM hemorrhages can break through into the posterior hyaloidal space or vitreous cavity, resulting in vitreous hemorrhage. Macular retinoschisis, often caused by intraretinal blood dissecting the outer nuclear layer from the inner retina, has been demonstrated with optical coherence tomography (OCT) and postmortem histopathologic analysis of victims of AHT.\(^8\) Retinal tears, retinal detachment, and (rarely) pigment epithelial detachments have been reported.\(^9,10\) Furthermore, the presence of circumferential retinal folds surrounding the macula has been regarded as a specific sign for AHT and has been associated with fatal outcomes.\(^11,12\) Optic disc hemorrhages and papilledema as a result of increased intracranial pressure may also be observed.

Recently, significant progress has been made in elucidating the underlying mechanisms leading to the retinal manifestations of AHT.
of AHT. Animal and computer simulation models support the most widely accepted theory, that repeated accelerations and decelerations of the head and globe result in shearing forces between the retinal blood vessels and the tightly adherent infant vitreous. This hypothesis has been further supported by histopathologic and OCT findings of victims of AHT showing vitreoretinal membrane formation, vitreoretinal traction, and persistent attachment of the vitreous at the apices of perimacular folds.

Retinal neovascularization in response to peripheral retinal ischemia and nonperfusion has been identified as an important late clinical manifestation in infants with AHT, and this underlines the importance of evaluating these patients using widefield fluorescein angiography (Figure 1F and G). The mechanism behind the peripheral nonperfusion is not well understood. It is believed that several factors are involved, including venous stasis, direct vitreous shearing of the capillary network, and autonomic dysregulation of the retinal vascular network secondary to brain hypoxia and damage.

**DIFFERENTIAL DIAGNOSIS**

The presence of multilayered retinal hemorrhages is not pathognomonic for AHT, as several other conditions can present similarly. Terson syndrome (intraocular hemorrhage associated with intracranial hemorrhage) is one of the most important masqueraders of AHT, given the overlapping ophthalmic and CNS findings. Retinal folds have been identified in patients with Terson syndrome, but they are certainly much less common than in AHT. Although the rare diagnosis of Terson syndrome has overlapping features with AHT, the literature and our experience both support the fact that AHT is more common and the likely diagnosis in almost all cases. Before a diagnosis of Terson syndrome can be made, a careful search (via clinical history, physical examination, skeletal survey, advanced CNS imaging, and consultations with a child abuse pediatrician and social services) for other findings of nonaccidental trauma must be made in order to avoid missing a potentially lethal diagnosis of AHT.

Multilayered retinal hemorrhages in infants can also be seen in the blood dyscrasias (eg, leukemia and chronic anemias), vaginal delivery, sepsis, and blunt trauma. Therefore, an appropriate workup, along with careful history, should be performed to rule out these entities. Note that the pattern and extent of retinal hemorrhages are usually highly suggestive of AHT, especially when other retinal findings such as perimacular folds and retinoschisis are present along with the classic systemic signs.

**MANAGEMENT STRATEGIES IN AHT**

When AHT is suspected, an ophthalmology consultation should be initiated early in the course of hospital admission in order to identify and document the retinal findings. Photodocumentation using a portable fundus camera is recommended. Depending on their extent, the majority of hemorrhages resolve spontaneously within a 1-to-3-week period; thus, observation only is usually recommended. Persistent hemorrhage involving the fovea or dense subretinal hemorrhage may result in deprivation amblyopia, especially if involvement is unilateral. In such cases, surgical intervention with vitrectomy may be necessary to clear the hemorrhage if it does not clear on its own within 2 months.

If available, widefield fluorescein angiography is recommended to assess for peripheral ischemia. If nonperfusion is identified, then laser photocoagulation may be considered, depending on its extent, to avoid neovascularization and late sequelae.

**CONCLUSION**

In nonfatal cases of AHT, long-term visual prognosis is further discussed. It is emphasized that the presence of multilayered retinal hemorrhages should not be considered diagnostic until other possible causes have been ruled out.
unfortunately often poor due to the high likelihood of concurrent traumatic brain injuries. Approximately 15% to 38% of victims succumb to their injuries, and, of the remaining patients, more than 80% will have permanent neurologic damage, possibly including blindness. 26, 27 Additionally, it has been shown that the severity and the extent of retinal hemorrhages correlates with the intracranial abnormalities of infants with AHT. 28

Despite recent advances in raising awareness and prevention, AHT continues to be a significant cause of preventable mortality and visual impairment in the pediatric population. AHT carries significant and often devastating implications that affect the patient’s immediate family, relatives, and caregivers, and it requires the involvement of a multidisciplinary team of physicians including pediatricians, radiologists, neurologists, neurosurgeons, orthopedic surgeons, child protective services, and law enforcement agencies. The ophthalmologist’s role is critical in confirming a diagnosis of AHT, as the dilated fundus examination findings (Figure 2) often provide compelling evidence. 29