

Imaging of Abusive Head Trauma: A Review and Update

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Abstract Abusive head trauma is the leading cause of severe traumatic brain injury in children <2 years of age. Because the clinical presentation of children with abusive head trauma can be nonspecific, CT and MR imaging play an important role in distinguishing these cases from episodes of accidental head trauma. In this article, we review the pathophysiology, imaging appearance, and specificity of patterns of traumatic brain injury associated with abusive head trauma, and highlight recent updates in the understanding of these injuries.

Keywords Abusive head trauma · Non-accidental trauma · Shaken baby syndrome · Inflicted brain injury · Subdural hematoma · Subdural hygroma

Abbreviations

AHT	Abusive head trauma
NAT	Non-accidental trauma
CCJ	Cranio-cervical junction
DWI	Diffusion-weighted imaging
SWI	Susceptibility-weighted imaging
SDH	Subdural hematoma

RH	Retinal hemorrhage
SIDS	Sudden infant death syndrome
ALTE	Acute life threatening event
DVST	Dural venous sinus thrombosis
DAI	Diffuse axonal injury
HII	Hypoxic-ischemic injury
nAHT	Accidental (non-abusive) head trauma
BESS	Benign enlargement of the subarachnoid space

Introduction

Abusive head trauma (AHT) is one of the commonest subtypes of non-accidental trauma (NAT). It accounts for a substantial proportion of NAT injuries in the first 2 years of life [1, 2] and is the leading cause of death among abused children [3]. AHT is responsible for the majority of severe traumatic brain injury in children <2 years of age [3–5], with case fatality rates above 20 % [6, 7]. Among children who survive, more than 75 % will have permanent neurologic impairment [8, 9].

AHT correlates with inconsolable crying behavior during infancy, which peaks in the second month of life [10]. Population-based studies in the UK and US suggest an annual incidence of 25–30 detected AHT cases per 100,000 children under the age of 1, with rapidly decreasing incidence over the first 5 years of life [6, 7, 11–13]. This is likely an underestimate due to the probability of missed and misdiagnosed cases [14].

Children with AHT can present with nonspecific signs and symptoms, making accurate recognition challenging. In a retrospective review of 173 children under the age of 3 diagnosed with AHT [14], 31 % of cases were initially missed by medical professionals. 28 % of these children sustained repeat injury prior to correct diagnosis. In 13 %

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of cases, missed AHT was due to misinterpretation of imaging studies. Improved awareness of the neuroimaging features of AHT is crucial for accurate diagnosis.

Imaging of AHT

Unenhanced CT of the head is the screening examination of choice for cases of suspected AHT [15•]. Multiplanar reformations and 3D volume rendering of the skull increase sensitivity for fracture and intracranial hemorrhage [16•]. Several studies have demonstrated an incidence as high as 27–39 % of unsuspected intracranial injury in neurologically asymptomatic patients with non-CNS injuries suspicious for NAT [3, 17, 18]. Many experts therefore recommend routine screening of the CNS in any child with sufficient clinical concern to warrant a skeletal trauma survey.

MRI of the brain is recommended for further evaluation of all abnormal screening examinations and in the cases of high clinical suspicion [19], generally between 3 and 5 after acute presentation. MRI in this time frame is sensitive for the detection of small volume extra-axial hemorrhage and for evolving parenchymal injury [20]. In addition to standard sequences, diffusion-weighted imaging (DWI) [21] and susceptibility-weighted imaging (SWI) [22] increase sensitivity for detection of parenchymal injury and microhemorrhage, and can provide prognostic information in AHT [23••]. Administration of gadolinium-based contrast material is not performed routinely but can be utilized in select cases to increase accuracy of dating extra-axial collections [24, 25].

MR of the cervical spine, including fat-suppressed fluid-sensitive sequences, should routinely be performed at the time of brain imaging, as unsuspected spinal injuries may be demonstrated in more than 75 % of cases [26, 27••, 28, 29•]. Whole spine imaging should be considered if there is suspicion for injury based on the trauma survey or an abnormal neurologic exam [30].

Terminology of AHT

Early publications [31–33] suggested the association of shaking-type abuse with a specific pattern of intracranial injuries, often in the absence of external signs of trauma. This association came to be known as “whiplash shaken infant syndrome” or simply “shaken baby syndrome,” terms that predominated in the literature for several decades and were considered synonymous with inflicted injury.

Over time, awareness of the diverse array of possible mechanisms of inflicted injury to the CNS has increased, particularly that of concurrent shaking and impact type injuries—the so-called “shaken impact syndrome” [5]. In addition, the contribution of less common mechanisms such as strangulation, suffocation, and penetrating trauma has been recognized.

In 2009, the term “abusive head trauma” was adopted by the American Academy of Pediatrics Committee on Child Abuse and Neglect, in an attempt to provide a more inclusive terminology for inflicted head injury that does not imply a specific mechanism of injury [34].

Mechanisms of Injury and Injury Patterns

AHT most commonly results from impulsive or impact loading [35]. Impulsive loading refers to nonimpact forces produced by rapid alternating angular acceleration and deceleration of the cranial contents, as with vigorous shaking. By contrast, impact loading refers to direct application of forces to the head. Both mechanisms can occur alone or in combination. Impulsive and impact loading are believed to result in distinct, but potentially overlapping, injury patterns.

Impulse loading produces differential angular acceleration of the intracranial contents, with risk for shearing injury to the brain and meninges [36]. The original reports of Guthkelch [31] and Caffey [32, 33] suggested the association of shaking-type injury with the triad of subdural hematoma (SDH), retinal hemorrhage (RH), and focal or diffuse parenchymal injury, often in the absence of external signs of injury. This association has been supported by numerous additional publications [37–40].

Impact loading typically results in soft tissue injury, skull fracture, subperiosteal hemorrhage, and parenchymal contusions [36]. In infants and young children, impact loading injury is less commonly observed in AHT than in accidental head trauma (nAHT) [41••, 42••, 43•]. When associated with AHT in young children, impact loading injuries are typically severe, and complex injury patterns can be present [44]. In older children, impact loading injuries becomes more common in AHT [44].

Goals of Imaging

The goals of imaging in suspected AHT are as follows: (1) detect pathology requiring emergent intervention; (2) assess the extent of the injury; (3) estimate timing of the injury; and (4) detect mimics or predisposing conditions.

Subdural Hematoma

SDH is the most commonly encountered imaging finding in AHT, present in 89 % of children on imaging in a recent prospective epidemiologic study [45]. This finding in isolation is only of moderate specificity for AHT, as it can be a feature of nAHT and non-traumatic conditions. The specificity of SDH for AHT is increased when associated with RH and underlying diffuse parenchymal injury [41••].

The typical SDH associated with AHT is thin and diffuse, without significant mass effect on underlying brain parenchyma [46]. The SDH is unlikely to be the cause of the clinical presentation but may serve as a marker for an impulsive mechanism of injury [47]. As discussed below, the prognosis is largely determined by the extent of associated parenchymal injury.

Pathophysiology

SDH has long been known to result from hemorrhage into the dural border cell layer—the innermost layer of the dura mater—and as such is actually an intradural hematoma [46, 48].

It has been considered axiomatic that traumatic SHD is the result of rupture of bridging veins [49]. Autopsy data on bridging vein rupture are sparse due to the technical difficulty of evaluating the bridging veins without introducing artifactual injury [50, 51]. Post-mortem imaging has occasionally been employed to demonstrate bridging vein rupture as the pathogenesis of SDH in fatal AHT cases [52–54], although it is not routinely used.

Recent imaging data suggests that this mechanism is actually more prevalent, with bridging vein thrombosis secondary to rupture demonstrated in between 40 and 45 % of AHT cases with SDH [55••, 56•]. When present, this confirms the traumatic (though not *per se* abusive) nature of the SDH [51, 57], and the vertex should be closely scrutinized in every case of unexplained SDH or suspected AHT.

In instances when bridging vein rupture is not demonstrable by imaging, bridging vein injury without thrombosis remains possible. Several authors have described a well-developed intradural vascular plexus, particularly prominent in young children, which may provide an alternative source for SDH [46, 48].

Imaging

SDH is well demonstrated on screening CT, but detection and characterization both benefit from the improved spatial and contrast resolution of MR [19, 20, 58]. Associated bridging vein thrombosis may be evident as hyperdensity on CT or hypointensity on susceptibility-sensitive MR sequences (T2* or SWI), with linear intravascular thrombus extending into more globular extravascular clot at the site of vessel rupture near the vertex. This appearance has been termed the “tadpole” [55••] or “lollipop” [56•] sign (Fig. 1).

The timing of SDH based on imaging should be approached with caution. A recent systematic review of studies evaluating the imaging appearance of SDH relative to time of trauma demonstrated considerable overlap—particularly in children, in AHT, and on MR [59••].

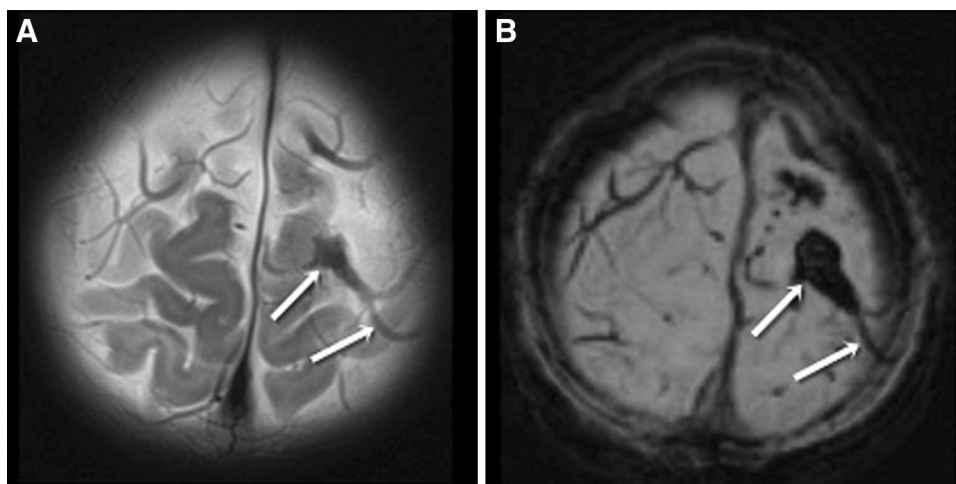


Fig. 1 3-month-old male presented with seizures, bruising, subconjunctival hemorrhages, and multilayered RH. **a** Axial T2-weighted image shows focal enlargement of a parasagittal bridging vein near the vertex (*white arrows*), suggesting vein rupture. **b** Axial SWI image demonstrates marked hypointensity and blooming within the

distal vein and at site of venous rupture (*white arrows*), confirming thrombosis. This appearance has been termed the “tadpole” or “lollipop” sign. Thin left SDH is evident as crescentic blooming artifact on the SWI image

Although classically the density of SDH on CT has been correlated with age, it is clear that acute SDH can present with a variety of imaging appearances. Unclotted blood can result in a SDH isodense to cortex, reflecting either hyperacute bleeding (typically in the first 3 h) or delayed clotting in coagulopathy [24, 25]. (Hemato)hygroma related to meningeal tear or arachnoid cyst rupture can present as an acute iso- or hypodense subdural collection [60, 61, 62•]. Anemia (hemoglobin <8–10 g/dL) can result in hypoattenuation of acute hemorrhage [24]. SDH hyperdensity and acute bridging vein thrombosis are reliable indicators of acute hemorrhage (up to approximately 10 days), but their absence cannot be used to infer the opposite.

The specific case of mixed-density collections warrants discussion. Although mixed-density SDHs may reflect acute-on-chronic bleeding, they should not be taken as *a priori* evidence of injury of multiple ages. Hyperacute bleeding, acute hemorrhage with sedimentation levels, and inhomogeneous hematomas can all present as mixed-density collections [24, 25] (Fig. 2). A descriptive approach to the density or attenuation of SDH on CT is recommended, rather than a dogmatic assertion of timing.

More specific estimates of the timing of SDH may be possible with MR, but certain caveats apply. The timing of evolution of blood products on MR suggested by Bradley et al. were derived from longitudinal observation of intraparenchymal hematomas [63]. SDHs evolve over a



Fig. 2 5-week-old female presented with an acute onset of obtundation, fixed and dilated right pupil, and coagulation studies consistent with hemorrhagic disease of the newborn. Axial CT image demonstrates a right mixed-density SDH (white arrows), found to be admixed clotted and unclotted blood at subsequent surgical decompression. Note that unlike the typical SDH of AHT, there is significant associated mass effect with midline shift, right lateral ventricular effacement, and left lateral ventricular entrapment

generally longer time course with slightly different imaging characteristics [24, 64].

As recently emphasized by Wittscheiber et al. and others, the fate of acute SDH is to either quickly resolve or to rapidly evolve into subdural hygroma through interstitial fluid or CSF transudation, which in most cases will eventually undergo resorption [62•, 65]. As discussed above, subdural hygromas may present acutely as well, secondary to an arachnoid tear, and should not be considered markers of chronicity. Subdural hygromas are characterized by CSF-like signal intensity and absence of mass effect, enhancing neomembranes, and loculations.

During their evolution, SDHs often demonstrate a layering sedimentation level, and the imaging characteristics of the serous fraction will typically approximate that of simple fluid, which can be erroneously interpreted as reflecting chronicity. Dating should be estimated from the dependent sediment layer [66] (Fig. 3).

In the presence of negative intradural pressure related to parenchymal volume loss or intracranial shunting, a small subset of hygromas may persist and form enhancing vascular neomembranes along the outer then inner margins [24] (Fig. 4). Repeated spontaneous hemorrhage from these membranes transforms the collections into chronic SDHs [46, 62•]. They may be relatively simple in appearance initially, but can assume an increasingly loculated configuration with increasing mass effect over time. Enhancing neomembranes are estimated to become apparent between 10 days and 3 weeks after injury [46, 65], and are one of the few reliable indicators of non-acuity in subdural collections.

The possibility that acute SDH may represent rebleeding into a chronic subdural collection, spontaneously or with trivial trauma, is a common dilemma in imaging of AHT. Based on the pathophysiology proposed by Whittscheiber and others, spontaneous hemorrhage into pre-existing subdural hygromas can be inferred to occur [62•, 64]. Identification of enhancing subdural membranes and associated parenchymal volume loss or shunting are supportive imaging features (Fig. 4). Acute neurological deterioration, new parenchymal injury, new subarachnoid hemorrhage, and new discontinuous SDH should not be present, however, and are suspicious for repeat AHT [65, 67].

Controversies

SDH has traditionally been considered a marker of possible traumatic injury. Several recent publications have postulated that a variety of relatively common non-traumatic pathologies may result in SDH and RH, thereby mimicking AHT. Proposed theories include hypoxic-ischemic injury

Fig. 3 4-month-old male presented with status epilepticus, facial bruising, and diffuse multilayered RH after fall from bed. **a, b** Axial T2-weighted and T1-weighted images obtained 1 day after presentation demonstrate bilateral holo-hemispheric subdural collections which approximate CSF signal intensity (*white arrows*), likely subdural hygromas. There is a dependent T2 hypointense, T1 isointense sediment layer on the left (*black arrows*), suggesting recent hemorrhage. Watershed HII was also present, not shown

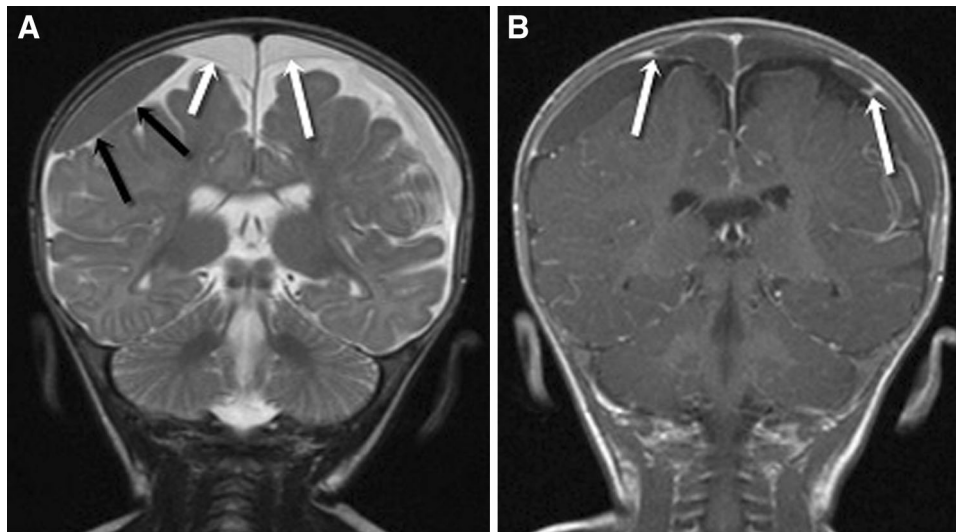
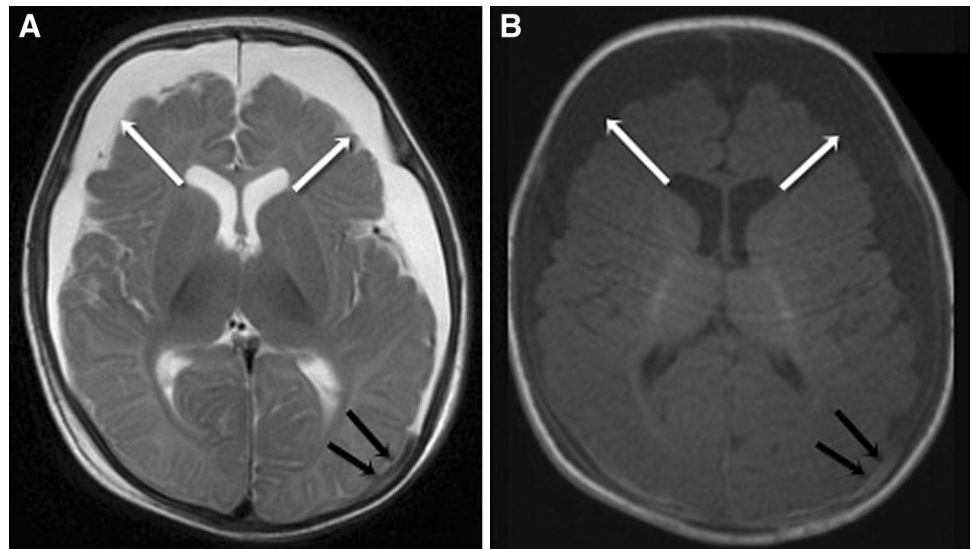


Fig. 4 4-month-old male imaged 1 month following acute bilateral SDH related to AHT. **a** Coronal T2-weighted image shows bilateral subdural collections. The dura can be identified as a thin hypointense line (*white arrows*) between the collections and the subarachnoid space. A loculation is present on the right (*black arrows*) containing blood products with different signal intensity. Note the mass effect on

the subjacent brain surface. **b** Coronal contrast-enhanced T1-weighted image shows enhancing neomembranes (*white arrows*) in continuity with the loculation, confirming chronic SDH with superimposed rebleeding. Such rebleeding can occur with minimal or no trauma and does not in itself suggest repeated abuse

(HII), elevated central venous pressures, and primary dural venous sinus thrombosis (DVST).

Geddes et al. have suggested that SDH and RH seen in the setting of AHT might be the result of the HII rather than a direct consequence of trauma [68]. This was based on the observation that microscopic intradural bleeding was noted in 36/50 children in their series who died of non-traumatic causes, including HII and intracranial infection. This line of reasoning has been coopted to support the

hypothesis that HII from any cause—including choking events, viral lung infections, and sudden infant death syndrome (SIDS) [69, 70]—can result in the classic imaging features of AHT.

It is important to note that the intradural hemorrhage that was described by Geddes et al. in their article was in all cases microscopic [68]. The extrapolation to cases of macroscopic SDH such as those seen on imaging studies in AHT is not supported by current evidence. In fact, large

case series examining imaging studies of children with severe HII related to drowning [71] and cardiac arrest [72] did not report macroscopic SDH in any case.

Dysphagic choking acute life threatening events (ALTEs) resulting in paroxysmal cough and elevated central venous pressures have also been postulated to result in SDH and RH in two reports [70, 73]. Scant clinical data exist to evaluate this contention. Supporting evidence is based on computer modeling and two reported cases of pertussis-related SDH that predate neuroimaging [74–76]. No recent well-documented clinical cases exist to support this association, however [77].

DVST has been suggested to result in non-traumatic SDH. Case reports [78] and a small case series [79] in adults support this possibility. However, a cases series of 36 consecutive infants and young children with DVST found no association with SDH [80•]. A second case series of 9 consecutive infants with DVST demonstrated only 2 SDH, one 8 days after forceps delivery and another in a child after MVA [81]. The evidence base does not support DVST as a cause of spontaneous SDH in young children at this time. Nonetheless, some centers routinely include a 2-D time of flight MRV through the head in cases of suspected abuse to excluded DVST for medicolegal purposes.

Diffuse Parenchymal Injury

In isolation, diffuse parenchymal injury is not specific for trauma of any etiology. In the presence of SDH, RH, and/or cervicomedullary injury, diffuse parenchymal injury is highly associated with AHT [27•, 29•, 41•, 42•, 43•, 44]. Thomas et al. recently demonstrated in a large series of nAHT victims that parenchymal injury was not present in the absence of a high-force mechanism, essentially excluding a history of trivial trauma as an explanation for diffuse parenchymal injury [82].

Excepting with pure asphyxiation, diffuse parenchymal injury is rarely present in the absence of SDH and RH in AHT [83]. Diffuse parenchymal injury is noted in less than a third of patients with suspected AHT, but it is the most important imaging feature predictive of outcome [23•, 43•, 83].

Pathophysiology

The etiology of parenchymal injury associated with impulsive-type AHT is incompletely understood. It was initially presumed to reflect traumatic diffuse axonal injury (DAI) [84, 85], based to some extent on extrapolation from the neuropathology of adult traumatic parenchymal injury [36, 86, 87]. Subsequent studies have suggested based on patterns of immunoreactivity to beta-amyloid precursor

protein (BAPP) that the predominant parenchymal injury in children with AHT is more likely HII rather than traumatic DAI [86–89].

These studies further described focal cervicomedullary traumatic axonal injury, which the authors suggested may have resulted in apnea and consequent HII. This association is supported by both animal models [90] and clinical series of suspected AHT [91–93]. Traumatic brain injury may also directly result in ischemic injury secondary to altered excitotoxicity and oxidative stress [94, 95], possibly mediated by clinical or subclinical seizure activity [96].

Several authors have reported unilateral HII [83, 97] as a characteristic injury pattern in AHT. The etiology of unilateral HII is difficult to understand from the mechanistic standpoint of cervicomedullary injury but has been explained by transient unilateral vascular occlusion [93]. Another possibility is “second impact syndrome”—an injury pattern described primarily in adolescents subject to repeated head trauma—characterized by catastrophic brain edema with a unilateral HII pattern [98]. The etiology of this syndrome is unknown but is hypothesized to reflect disordered cerebral autoregulation.

In adolescent cases of second impact syndrome, thin ipsilateral SDH is a universal associated feature, not unlike AHT cases. This suggests that SDH itself may have a direct or compounding effect on the subsequent development of HII. Animal studies have shown altered metabolism underlying SDH [99]. These findings suggest that the HII seen in AHT is likely multifactorial.

Imaging

The parenchymal injury associated with AHT may be occult on early screening CT, or may be seen as diffuse or hemispheric hypodensity with evidence of mass effect. Thin SDH associated with disproportionate ipsilateral hemispheric mass effect on screening CT should raise the possibility of early unilateral HII and prompt consideration of AHT and further evaluation with MRI (Fig. 5).

Early injury is better demonstrated on MRI. T2 hyperintensity reflecting tissue edema may be subtle in the unmyelinated brain, and DWI will dramatically increase conspicuity of cytotoxic injury [100]. Studies examining the patterns of diffusion restriction in AHT have suggested a watershed pattern of HII in a majority of cases [93, 100, 101]. However, multiple patterns of parenchymal injury are potentially consistent with AHT (Fig. 6).

A recent study that examined white matter microstructural changes in AHT using diffusion tensor imaging demonstrated reductions in mean diffusivity largely as a result of decreased axial diffusivity, with preserved radial diffusivity and fractional anisotropy [102•]. These findings are presumed to reflect hypoxic-ischemic axonopathy and

Fig. 5 Two and half-year-old male presented with seizure and decreased level of consciousness following unwitnessed fall down 2 stairs. Initially misdiagnosed as stroke at OSH and transferred to tertiary care hospital for rehabilitation, patient was noted to have multiple healing fractures and diffuse multilayered RH on further evaluation. **a** Initial CT from OSH demonstrated thin right SDH with disproportionate mass effect resulting in right to left midline shift. **b** Axial FLAIR image from subsequent MR demonstrates diffuse unilateral cortical hyperintensity on the right. **c** Axial ADC map demonstrates a predominately subcortical pattern of diffusion restriction

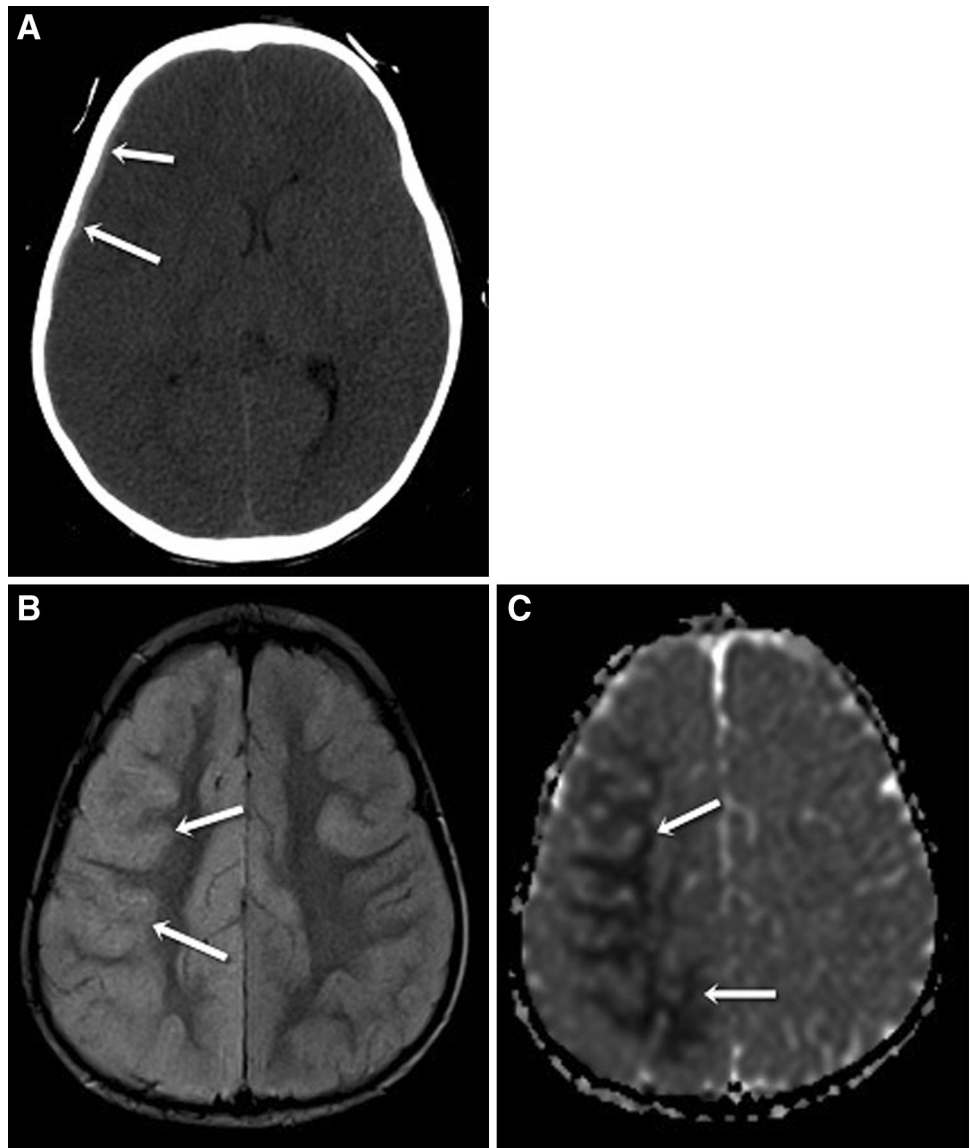
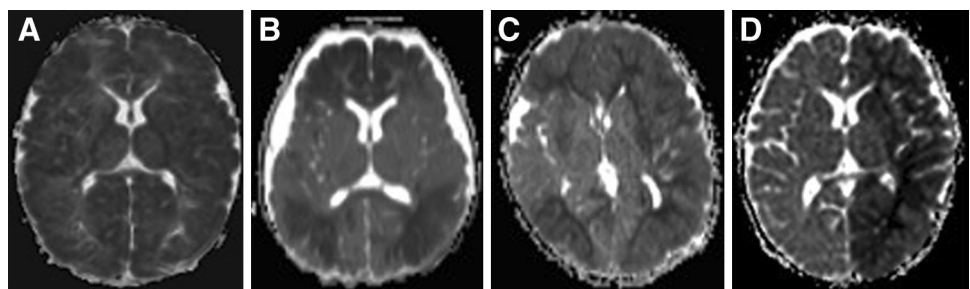


Fig. 6 Patterns of diffusion restriction in AHT on axial ADC map images. **a** Whole brain, **b** watershed, **c** subcortical, **d** unilateral



were found to be of prognostic value in stratifying functional outcome.

The time course of the evolution of HII on DWI is variable, but may be delayed relative to that seen in closed-vessel ischemic injury typical in adults. This reflects variability in

the underlying pathologic substrate of HII in the immature brain, ranging from acute tissue necrosis to necrosis-like cell death to delayed apoptosis [103], depending on multiple factors including the severity of the initial insult, contribution of inflammation and excitotoxicity, and superimposed

seizures. This variability must be considered when attempts are made at dating parenchymal injury related to AHT.

Controversies

There is continued medicolegal debate regarding the feasibility of isolated shaking as a viable etiology for parenchymal injury in pediatric patients. A study by Duhaime et al. using a 1-month-old baby model found that shaking injury without impact was unable to generate sufficient angular acceleration to meet expected thresholds for parenchymal injury including concussion and DAI [104].

Other authors [105] have called into questions this conclusion, pointing out problems with the biofidelity of the model used. They further noted that injury thresholds were scaled from single high-frequency impulsive loading studies in adult primates, and their comparability to repeated low-frequency shaking injury in infant children is unknown.

In light of the evolving understanding of the nature of parenchymal injury in abused children and the diversity of probable underlying mechanisms, the continued relevance of early estimates of the biomechanical feasibility of shaking alone as a cause for traumatic DAI and concussion is of questionable importance.

Focal Parenchymal Injury

Focal parenchymal injuries in AHT may result from impulsive or impact loading injury and are less common and less well described than diffuse injury. The pattern of focal parenchymal injury secondary to impulsive loading evolves with increasing age, likely due to progressive myelination and changes in brain parenchymal viscosity.

In children under 5–6 months of age, shear injury can result in very distinctive parenchymal lacerations or contusion clefts [36]. First described by Lindenberg and Freytag in 1969 [106], these lacerations have received very little attention in the imaging literature, largely limited to an older cases series using cranial ultrasound [107]. A recent report by Palifka et al. reaffirmed that these lesions are highly associated with AHT and reported them on MR in children up to 11 months of age [108•].

In older children, multifocal parenchymal lesions may uncommonly occur with severe trauma, likely representing classic DAI [101]. At any age, focal hemorrhagic contusions may be present reflecting an impact loading mechanism, typically with overlying skull fracture and occasionally with a coup-contrecoup pattern.

Imaging

Parenchymal lacerations or contusional clefts appear as linear tears in the brain parenchyma, predominately within the cortex or at the gray-white junction of the gyral crests. They predominate in the frontal and temporal lobes. They frequently demonstrate layering sedimentation levels on both CT and MR (Fig. 7). When superficial, they can be associated with overlying subpial hemorrhage.

Focal parenchymal lesions in older children may occur and are presumed to be related to DAI. They are characterized by multifocal T2 prolongation, most common in the white matter of the frontal lobes and splenium. FLAIR is incrementally more sensitive for demonstration of lesions than T2, while SWI is substantially more sensitive [22, 109]. Presence of parenchymal microhemorrhage on SWI in AHT has been shown to correlate with poor clinical outcome, particularly when associated with underlying diffuse parenchymal abnormality [23••]. Occasionally, a pattern of multifocal punctate diffusion restriction is evident [101].

Retinal Hemorrhage

RH is estimated to occur in up to 85 % of AHT, but similar to SDH is of only moderate specificity [110]. Specificity is increased when RH is bilateral, multilayered (involving the pre-, intra-, and subretinal layers), and when it extends peripherally to the ora serrata on fundoscopic examination [111]. A recent report by Binenbaum et al. found that RH severity was correlated with severity of HII in children with traumatic brain injury [112].

Pathophysiology

RH in AHT is believed to result from traumatic retinoschisis or splitting of the retinal layers by shearing forces [113]. This mechanism can sometimes be suggested on fundoscopic examination by identification of retinal folds at the periphery of the hemorrhage, though this is inconstant [111].

Imaging

RH has not traditionally been considered an imaging diagnosis, although it has occasionally been reported [114]. It presents as high-attenuation foci at the posterior globes on CT, and low-signal foci on MR sequences (Fig. 8).

Data recently published by Beavers et al. demonstrated a 61 % sensitivity for confirmed RH [115••] using concurrent analysis of T2*, T2, FLAIR, T1, and T1 post-contrast sequences. They demonstrated decreasing sensitivity of

Fig. 7 6-week-old female presented with vomiting, seizures, abnormal breathing, abdominal bruising, and facial abrasions. **a** Axial-unenanced head CT image shows hyperdense (acute) right frontal subdural hemorrhage (*black arrows*) and a hypodense left frontal cleft containing dependent hyperdense blood (*white arrows*). **b, c** Axial T2-weighted and SWI images show the acute blood products as T2 hypointense material in the subdural space (*black arrows*) and layering within the contusional tear (*white arrows*)

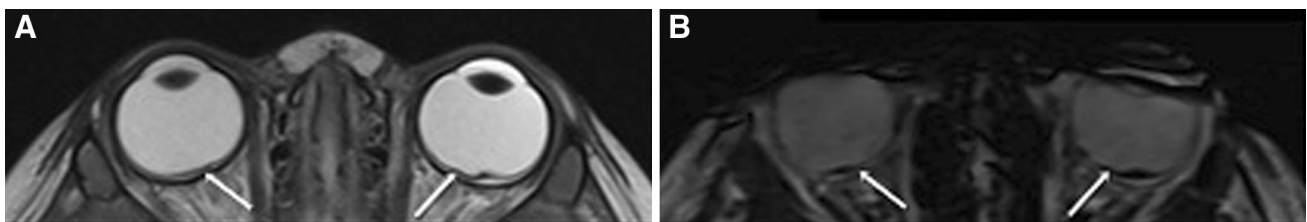
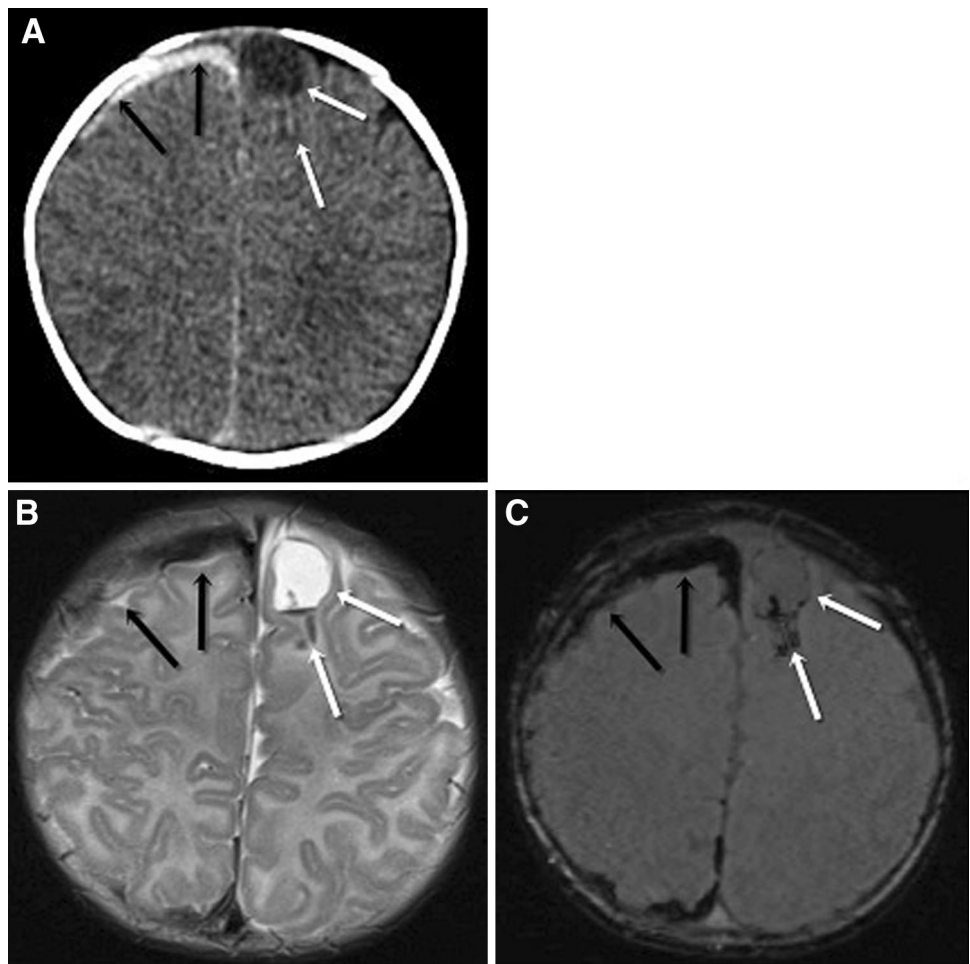


Fig. 8 4-month-old male presented with seizures and decreased level of consciousness. Dilated fundoscopic examination demonstrated multilayered retinal hemorrhage with a macular schisis cavities in the bilateral eyes. **a** Axial T2-weighted image demonstrates retinoschisis

of the posterior globes bilaterally (*white arrows*). **b** Axial SWI images demonstrate increased conspicuity due to blooming artifact (*white arrows*), confirming RH

sequences in the order listed. They went on to correlate MR detection rates with fundoscopic severity, concluding that high-grade hemorrhage was significantly more likely to be detected by MR than low-grade hemorrhage, at 76 versus 14 %.

With the clinical implementation of SWI, MR has become more sensitive for the detection of RH. Zuccoli et al. demonstrated a sensitivity of 62 % for the detection

of RH with this single sequence using a gold standard of dilated funduscopy [116]. Sensitivity increased to 80 % when a dedicated high-resolution orbital SWI sequence was employed, and all missed cases were of mild severity on fundoscopic evaluation.

Given that increasing severity of RH has been shown to correlate with increasing risk for AHT, MR-visible RH must be viewed as suspicious. Detection of signal

abnormality in the posterior globes in children absent adequate clinical history of severe trauma should prompt close scrutiny for additional intracranial abnormalities and consideration of dilated fundoscopic examination.

Controversies

Many alternate theories of causation for RH and SDH have been proposed, many of which have already been discussed in the section on SDH.

Skull Fractures

Skull fractures are not specific for AHT. Non-displaced linear skull fractures and associated small volume subperiosteal hemorrhage are common findings in low-impact nAHT, such as with short-distance falls [42••]. Contradictory data exist regarding which features, if any, may increase the specificity of skull fractures for AHT [117–119]. Fractures which are multiple, complex, diastatic, or growing suggest a high-energy mechanism and are concerning in the absence of appropriate history (Fig. 9).

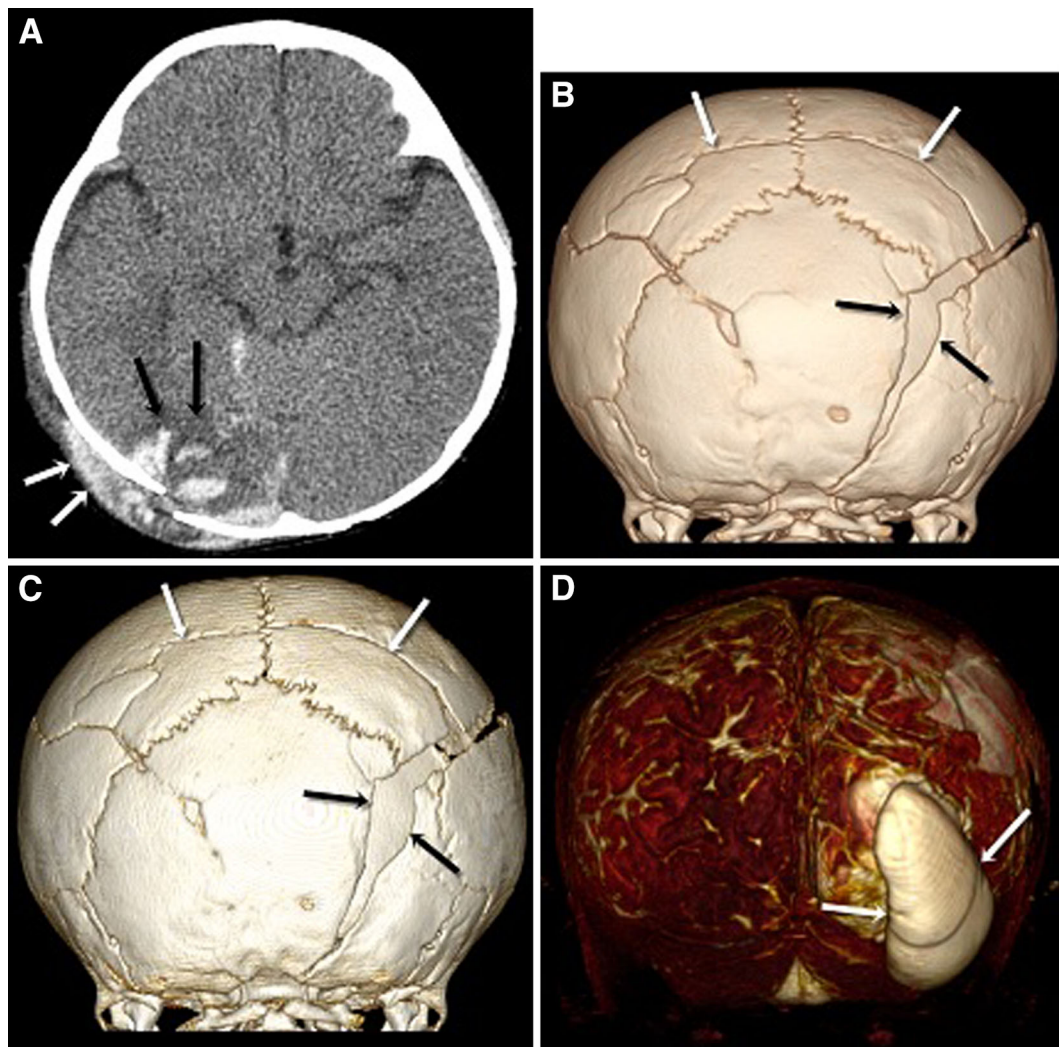


Fig. 9 1-year-old male presented with head injury after mother’s boyfriend “fell on him” while removing him from his car seat. **a** Axial-unchanged CT image shows impact injury pattern with scalp swelling and cephalohematoma (*white arrows*), diastatic right occipital fracture, and hemorrhagic contusion (*black arrows*). **b** Volume-rendered CT image demonstrates complex occipitoparietal fractures crossing sutures (*white arrows*), with fracture diastasis (*black*

arrows). **c** Volume-rendered CT image at 2-month follow-up redemonstrates complex fractures (*black arrows*), with increasing diastasis of the displaced fracture suggesting a “growing fracture.” **d** Volume-rendered T2-weighted MR image after skull stripping demonstrates a large posterior pseudomeningocele or “leptomeningeal cyst” as the cause of the growing fracture

Pathophysiology

Skull fractures occur when impact forces result in deformation of the calvarium in excess of the failure strength of the bone. The calvarium of young children is considerably more pliable than that of older children and adults, and significant deformation can occur without consequent fracture. In addition, a plastic “ping-pong” type fracture can occur.

Imaging

Skull fractures in children are best evaluated on CT reconstructed at submillimeter slice thickness. Multiplanar reconstructions and volume-rendered images of bone should be routinely performed and are helpful for identifying “in plane” fractures which are easily missed on axial images only.

Dating of skull fractures on CT can be difficult, as expected imaging findings associated with healing long bone fractures are not typical. In our experience, blurring of the fracture margins and new bone formation by the intact periosteal layer of dura are features of subacute healing fractures and are inconsistent with recent trauma (Fig. 10). Absence of these features cannot reliably be used to conclude acuity, however. Soft tissue swelling and subgaleal hemorrhage are suggestive of more recent injury but are not universally present in acute injury.

Spine Injuries

Until recently, spine injury had been considered rare in NAT, with a reported incidence of <2 % [120, 121]. Incidence of spine fracture is increased to almost 10 % in the setting of a positive skeletal survey and is significantly associated with intracranial injury [122]. Occult spine injuries are often not routinely sought in evaluation of AHT, however, and their prevalence is likely significantly underestimated.

Indeed, recent evidence supports that spine injury in children with AHT is not uncommon. Ligamentous injury at the craniocervical junction (CCJ) and spinal SDH are reported in 36–78 % [27•, 29•] and 44–63 % [24, 26] of AHT cases, respectively, in several recent retrospective studies. Fracture [122], bone contusion [122], epidural hematoma [123], and cord injury have also been reported [29•].

CCJ injury in particular is highly correlated with abusive HII and can be helpful in establishing the traumatic etiology of SDH [27•, 29•].

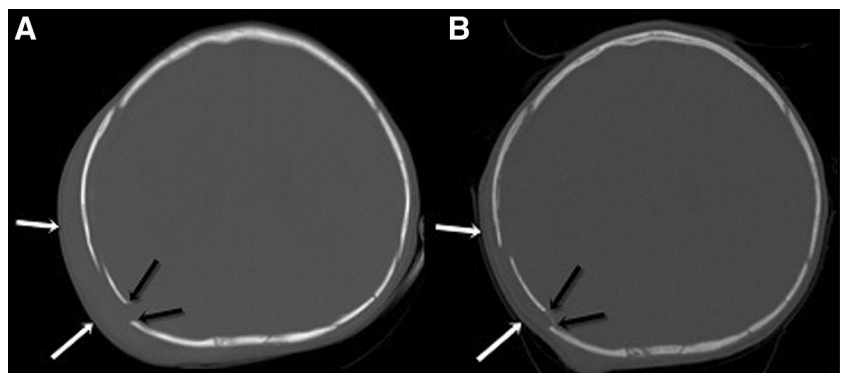
Pathophysiology

The mechanisms of spine injury in AHT can be diverse. Of particular importance in children under 12 months of age is ligamento-osseous injury at the CCJ, as it may be expected to result from the whiplash-like motion of the cervical spine associated with impulsive loading of the cranium [124]. Pathologic studies have demonstrated CCJ injury in >75 % of fatal AHT cases [86, 87, 91, 125–127]. Specific injuries included subdural and epidural hemorrhage; cervical cord contusion, laceration, and hemorrhage; cervical nerve root avulsions; and ligamentous or soft tissue injuries.

Imaging

MR imaging of the cervical spine is recommended routinely along with MR brain imaging in all cases of suspected AHT. Although an early report demonstrated low sensitivity for detection of CCJ injury with MR in cases of severe AHT [126], this study predated the widespread clinical availability of STIR and other fat saturation techniques that increase the conspicuity of soft tissue and subtle bony injury. Recent studies have demonstrated improved rates of detection of abnormalities at the CCJ with MR [27•, 29•, 120], with ligamentous injury the most commonly reported finding (Fig. 11). Routine utilization of fat-saturated fluid-sensitive sequences is therefore recommended.

Fig. 10 6-month-old male presented with head injury after being stuck with a hammer. **a** Axial-unenhanced CT image shows an acute diastatic right parietal fracture (*black arrows*) with associated scalp swelling (*white arrows*). **b** Axial-unenhanced CT image performed after 4 weeks shows linear periosteal reaction consistent with healing (*black arrows*), as well as decreased scalp swelling (*white arrows*)



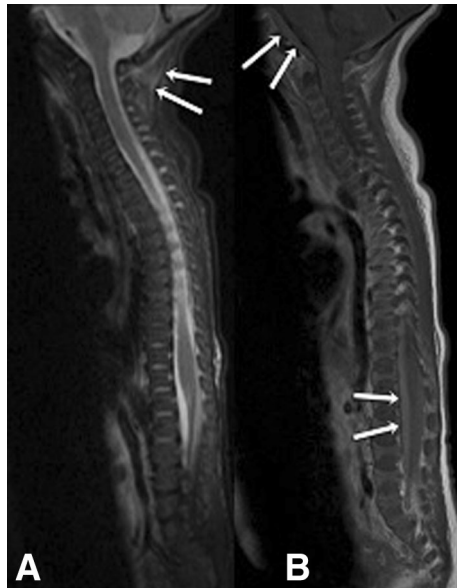


Fig. 11 6-week-old male found down at home. MR of the brain revealed diffuse HII, bilateral SDH, and bridging vein thrombosis. Left intraretinal RH on dilated fundoscopy. **a** Sagittal STIR image shows hyperintense signal in the nuchal ligament and underlying posterior cervical fat (*white arrows*), consistent with ligamentous injury. **b** Sagittal T1-weighted image shows mildly hyperintense retroclival and thoracolumbar extra-axial collections (*white arrows*), consistent with subdural hemorrhage

A recent report by Koshy et al. highlighted the association of head trauma and retroclival hematoma, both epidural and subdural [128]. Retroclival epidural hematoma occurs anterior to the tectorial membrane and is associated with ligamentous injury at the CCJ [128, 129]. It should be differentiated if possible from SDH at the CCJ which occurs posterior to the tectorial membrane, as this latter finding likely represents redistribution of posterior fossa SDH. Although classically associated with severe nAHT, Silvera et al. demonstrated that retroclival hematomas were present in a third of cases with AHT in their series [123] (Fig. 11).

The value of routine screening of the whole spine in suspected AHT is being increasingly recognized. Studies of MR limited to the cervical spine have demonstrated a relatively low incidence of spinal SDH [29, 126]; detection is significantly increased when imaging is extended through the thoracolumbar spine [26, 28] (Fig. 11). Although detection of spinal SDH is rarely associated with a change in management, Choudhary et al. have reported that the presence of spinal SDH is highly associated with an inflicted rather than accidental mechanism (present in 46 % AHT vs. 1 % nAHT in their cohort), and therefore, may be of diagnostic value.

Routine MR screening may also help to identify occult bony injury. Barber et al. found that spine fractures were

present in up to 10 % of children with positive skeletal surveys [122]; spine injuries were reported at all levels in a recent review of spine injuries in AHT by Kemp et al. [30]. As it is known that MR increases the yield for detection of spine fractures in AHT [130], whole spine MR may have added value for identifying subtle bony injury in this population. For these reasons, some authorities now suggest that routine screening of the whole spine be performed in any child receiving an MRI of the brain for suspicion of AHT [28, 30, 120, 121].

Differential Considerations of AHT

Accidental HT

The primary differential consideration for AHT is nAHT. A number of studies have analyzed the imaging injury patterns present in children with AHT and nAHT in an attempt to establish the association of particular injuries or patterns of injury with AHT.

Two recent comprehensive systematic reviews [42, 43] demonstrated that SDH, HII, RH, and skull fracture associated with intracranial injury were associated with AHT; epidural hematoma, isolated skull fracture, and scalp swelling were associated with nAHT; subarachnoid hemorrhage, focal contusion, and DAI were not consistently associated with either etiology of trauma.

These findings were corroborated by Roach et al. who recently published the largest single cases series of AHT to date [131]. Among 2015 children with traumatic head injury under age 5, they found that patients with AHT were significantly less likely to have skull fracture and epidural hematoma, and significantly more likely to have SDH and parenchymal injury.

Another recent case series of 345 children with traumatic head injury published by Kelly et al. [44] found that patients with AHT were significantly more likely to have absence of impact injuries, SDH, and HII. They found that this association held only for children under 2 years of age, suggesting that older abused children suffer more impact-related AHT. When AHT was associated with impact injuries in younger children, however, they were more likely to be severe with associated intracranial injury (Fig. 9).

It is important to address the potential effect of ‘circularity of reasoning’ as it impacts the data on patterns of injury with AHT and nAHT. To the extent that presence of certain injuries (e.g., SDH) or particular patterns of injury (e.g., SDH, RH, and HII) themselves led to the initial categorization of head trauma as abusive or accidental, an analysis of the association of these same features with abuse will be spuriously strengthened.

In an attempt to control for this, Vinchon et al. published an analysis of prospectively identified, corroborated cases of head trauma in children under age 2 (45 confessed AHT and 39 publicly witnessed nAHT) [132••]. Identical to the aforementioned studies, they reported that SDH, HII, RH, and absence of scalp injury were strongly associated with AHT, while extracranial hemorrhage, fracture, and contusion were highly associated with nAHT. These findings suggest that the relationship between impulsive injury patterns and AHT in children under age 2 is robust.

Birth-Related Trauma

A subset of nAHT is related to birth trauma. In a series of 41 consecutive patients with symptomatic birth injury, Pollina et al. reported 73 % SDH, 25 % extracalvarial hemorrhage, 20 % subarachnoid hemorrhage, 20 % intraparenchymal hemorrhage, and 5 % fracture [133]. There was an increased risk of birth trauma with vacuum- and forceps-assisted vaginal delivery.

Rooks et al. reported SDH in 46 % of asymptomatic newborns MR within 3 days of birth, with lowest rates reported with caesarian section and the highest rates reported in assisted vaginal deliveries [134]. Lower rates of detection have been reported in other studies with decreased field strength [135] and longer time to initial imaging [136]. Birth-related SDH in these studies were generally dependent and thin (< 3 mm) (Fig. 12). All resolved within the first postnatal month [134, 136].



Fig. 12 4-day-old term female delivered by Cesarean section, imaged for hypotonia. Sagittal T1-weighted image shows thin subdural blood products along the occipital convexity and in the posterior fossa (arrows), without mass effect. This finding is common in the first postnatal month

Evolution to chronic SDH was not demonstrated, as expected given lack of predisposing factors [62•]. One patient in the study by Rooks et al. had a new separate SDH incidentally noted on a 2-week follow-up examination in the context of benign enlargement of the subarachnoid space, which subsequently resolved by 3 months.

Laghmari et al. reviewed over 2000 children born by spontaneous vaginal delivery and demonstrated that nearly one-third of children had RH [137]. These tended to be few in number and localized to the optic disks and posterior pole of the retina. There is currently no imaging data to suggest that such RH is routinely visible by MR. Similar to SDH, all cases of birth-related RH resolved within 1 month.

Non-Traumatic SDH

SDH is reported to occur in the context of numerous underlying medical conditions, though these are rare. Potential etiologies include vascular malformations, hematologic malignancies, coagulopathy (Fig. 2), and metabolic disorders such as Menkes kinky hair syndrome and glutaric aciduria type I (Fig. 13). Awareness of underlying medical conditions that might present with spontaneous hemorrhage that can mimic AHT is crucial for accurate diagnosis, and reinforces the notion that the evaluation of AHT must be accomplished in a multi-disciplinary fashion on a case-by-case basis.

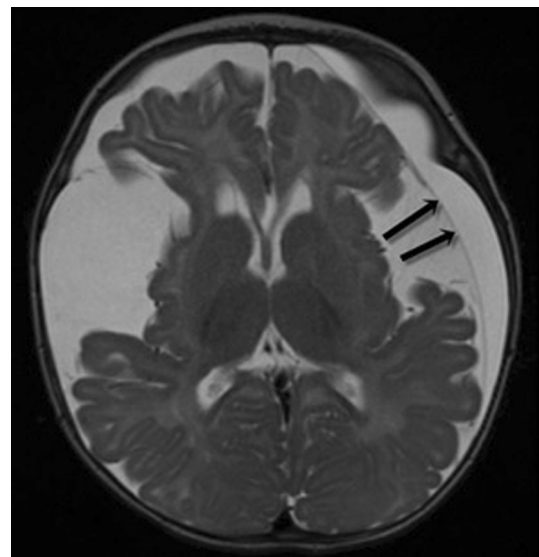


Fig. 13 6-month-old male with developmental delay and macrocephaly, subsequently confirmed to have glutaric aciduria type I. Axial T2-weighted image shows bilaterally enlarged perisylvian subarachnoid spaces secondary to frontal and temporal opercular hypoplasia and a left subdural collection (black arrows)

Mimics

Normal Neonatal Imaging Features

Care must be taken not to interpret normal imaging features of neonates and young infants as evidence of AHT. Normal sutural prominence in the neonatal period, hypodense or T2 hyperintense unmyelinated white matter, and dense dural venous sinus blood are potential pitfalls that can mimic sutural diastasis, parenchymal edema, and sinovenous thrombosis or layering SDH, respectively. Accessory sutures can occasionally mimic fractures in children but can usually be distinguished based on their frequent bilaterality, sclerotic borders, and serpiginous course [138].

Benign Enlargement of the Subarachnoid Space (BESS)

From 0.5 to 0.8 in 1000, children will demonstrate mild to moderate enlargement of the subarachnoid spaces in the first 18 months of life, resulting in macrocephaly [139]. This appearance can easily be confused with hypodense bifrontal SDHs, particularly on CT. A key discriminating feature is the presence of normal subarachnoid vessels traversing the enlarged fluid space in BESS; these vessels are notably absent in SDH, in which case the subarachnoid space is compressed over the cerebral convexities (Fig. 14). Slight differences in signal intensity between the different fluid spaces is often accentuated on the FLAIR sequence, and the displaced dural-arachnoid membrane can be well visualized on high-resolution steady-state free

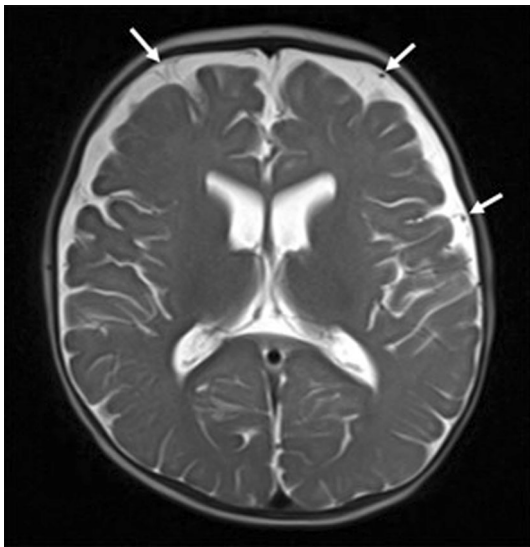


Fig. 14 7-month-old male imaged for macrocephaly. Axial T2-weighted image demonstrates prominent bifrontal extra-axial spaces with traversing vessels (*white arrows*), consistent with enlarged subarachnoid spaces

precession sequences (CISS, FIESTA, and b-FFE) in unclear cases.

It is worth noting that some publications suggest that BESS can itself predispose to SDH in the setting of minimal or no trauma, and that the two can occasionally co-exist. In two retrospective reviews of children diagnosed with BESS on imaging, asymptomatic SDH was identified in between 3 and 4 % [140••, 141].

Several retrospective case reviews have reported the existence of acute symptomatic SDH in the setting of trivial trauma in young children, with a majority occurring in the setting of BESS [142–144]. In a prospective evaluation of 164 children under age 2 presenting with symptomatic SDH, 16 (10 %) were categorized as spontaneous, among which 75 % were associated with BESS and macrocephaly [41••]. Additional risk factors included severe dehydration and lumbar puncture with intracranial hypotension.

These data should be interpreted with caution. 25 % of patients with SDH in the setting of BESS in the series by McKeag et al. manifested additional injuries suggestive of physical abuse [140••]. In addition, many cases of SDH deemed secondary to trivial trauma in retrospective case reviews were associated with RH or went on to develop diffuse encephalomalacia [142, 143], suggesting a likelihood of missed AHT in some cases. Given this, thorough clinical, ophthalmologic, laboratory, and radiologic evaluation of all children with unexplained SDH is warranted.

Conclusion

AHT is a common cause of morbidity and mortality in the pediatric population, and imaging evaluation is crucial for accurate diagnosis and prognostication. A comprehensive review of the imaging of AHT has been provided. Recent advances in the field of imaging of AHT have been emphasized, including limitations of imaging for dating AHT, emerging concepts in the evolution of SDH over time, the importance of parenchymal lacerations as high-specificity injuries in AHT, and the increasing utility of MR for the detection of RH and spine injuries in AHT. Understanding of the common patterns of abusive and accidental injury can help increase accuracy of diagnosis, both by increasing recognition of high-specificity injuries and by preventing unwarranted concern in cases of clearly concordant history and injuries.

Compliance with Ethics Guidelines

Conflict of Interest Jason N. Nixon declares no potential conflicts of interest. Bruno P. Soares is a section editor for *Current Radiology*

Reports.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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