Seminars in
ULTRASOUND
CT *and* MRI

Pearls and Pitfalls in Imaging of Abusive Head Trauma

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Abusive head trauma (AHT) is the leading cause of fatal head injuries in infants. The mechanism of injury usually involves vigorous shaking of the infant, impact, or a combination of the 2. There are characteristic imaging findings of which the most common are subdural hemorrhages. Parenchymal injuries to the brain are common as well, including hypoxic ischemic injury, diffuse axonal injury, and cerebral contusions. Retinal hemorrhages are common with AHT and are best evaluated by fundoscopy, however, high grade retinal hemorrhages may be detected on cross-sectional imaging. Skull fractures are not specific to AHT but are present in third of the cases and tend to be complex in association with AHT. Injuries to the spine are more common than previously thought and typically involve soft tissues rather than bones, with the most common being ligamentous injuries in the craniocervical junction. In the setting of AHT, an affected infant would typically exhibit multiple findings. While CT of the head is the first modality of choice for assessment of an infant with AHT, magnetic resonance imaging examination of the brain and spine should follow due to its higher sensitivity for detection of these findings.
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Introduction

Abusive head trauma (AHT) is the leading cause of fatal head injuries in infants younger than 2 years.[1] It is the most serious form of physical child abuse with an associated mortality of 30% and morbidity in 50% of survivors.[2] As of 2000, child abuse was the most common cause of serious head injury among children younger than 1 year and it is estimated that 10% of cases of mental retardation and cerebral palsy result from child abuse.[3]

The mechanism of injury includes vigorous shaking of the infant, impact, or a combination of the 2.[1] While any type of injury may be caused by abuse, there are some characteristic imaging findings that are highly associated with AHT and should raise suspicion. Imaging findings are vital in the process of evaluation for AHT as the clinical presentation is nonspecific and the history may be misleading.[1,2,4] Nevertheless, it cannot be overemphasized that even in the presence of such findings the diagnosis of abuse cannot be made solely based on imaging. When AHT is suspected, a multidisciplinary approach should take place to evaluate the infant and caregivers in order to reach an accurate diagnosis. This includes but is not limited to interview of the caregivers, careful physical examination of the infant, ophthalmologic evaluation, additional imaging studies such as skeletal survey, neurosurgical and orthopedic consultations and evaluation of siblings.

In this review, imaging findings in the head and spine will be discussed, with emphasis on cross-sectional imaging modalities. Other skeletal injuries typically evaluated on skeletal surveys are beyond the scope of this review and will not be discussed.

Imaging Modalities

Computerized tomography (CT) of the head is the first modality of choice for assessment of traumatic brain injury. CT scan time is short, and it does not require sedation. CT provides valuable information with regard to intracranial hemorrhages, mass effect and skull fractures in the acutely injured patient and is essential for determining the need for neurosurgical intervention.[1,2,5]

All infants who present with signs of abuse and are acutely ill with neurological findings should undergo CT scans. 3D

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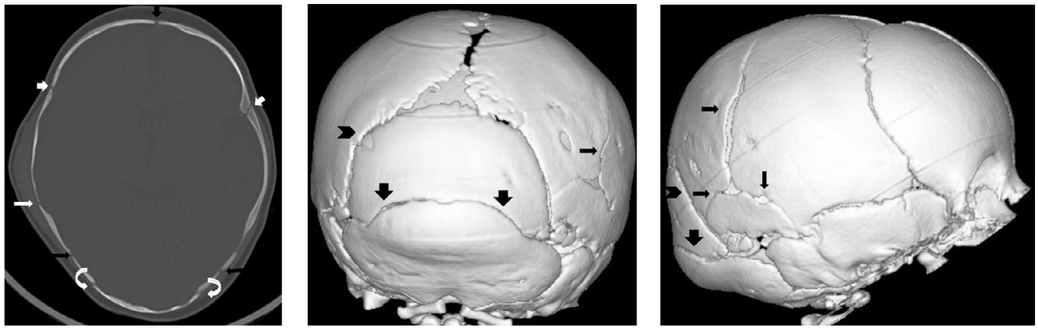


Figure 1 Skull fracture and sutures. (A) Axial cut from a brain CT of a 12-week-old female with history of fall and scalp swelling, demonstrates a right parietal cephalohematoma and an underlying skull fracture (long white arrow). The metopic suture (short black arrow), coronal sutures (short white arrows), and lambdoid sutures (long black arrows) can be identified. There are 2 additional lucencies (curved white arrows) in the occipital bone, which represent the bilateral mendosal sutures. (B and C) 3D reconstructed right lateral and posterior images of the skull demonstrate a right parietal fracture that branches into 2 fracture lines at its inferior aspect (thin arrows). The right lambdoid suture (arrowhead) and bilateral mendosal sutures (thick arrows) are also identified.

reconstructions of the skull increase the sensitivity and specificity for detection of skull fractures in infants and should be performed as part of the examination[1] (Fig. 1).

Routine head CT in all children with signs of abuse but with no evidence of neurotrauma is controversial. According to The Royal College of Paediatrics and Child Health and the Royal College of Radiologists in the United Kingdom, CT is indicated in "any child under the age of 1 (year) where there is evidence of abuse."[5] The American College of Radiologists however, states that head CT in children without neurological symptoms should be strongly considered in patients that are at "high risk" for having suffered from AHT, including children with rib fractures, multiple fractures, facial fractures or those younger than 6 months.[6] The disadvantages of CT include exposure to ionizing radiation and low sensitivity for parenchymal lesions, particularly petechial hemorrhages seen with diffuse axonal injury (DAI), and acute ischemic changes.[2]

The second imaging modality for infants with AHT is Magnetic Resonance Imaging (MRI). Studies have shown MRI to be more sensitive than CT in the detection of intracranial hemorrhages and other parenchymal injuries in these patients.[2] According to a systematic review by Kemp et al[7] MRI examinations revealed additional findings including further subdural hemorrhage (SDH), subarachnoid hemorrhages, ischemic injuries, infarcts and shear injuries when compared to findings in initial CT, in at least 25% of all children with abnormalities on the initial CT scan. In a study by Foerster et al[4] on a cohort of 57 infants who suffered of AHT, in 12 of 40 patients with subdural hematomas undergoing MRI and CT scans, hemorrhage of different ages was detected on MRI, strengthening the case for non-accidental head trauma. Furthermore, MRI detected additional findings, not seen on CT, in 6 patients including 3 cases of DAI and 3 cases of ischemia. MRI should be performed whenever feasible in all infants with findings on CT and in those suspected of abuse with no CT findings but with encephalopathy or focal neurological signs.[1,2,5,7] MRI is usually delayed to the 3-5th day form presentation and is performed when the patient is stable.[2] A standard MRI protocol typically includes T1 and T2 weighted sequences as well as fluid attenuation inversion recovery (FLAIR) sequence, gradient echo (GRE) images and diffusion weighted images (DWI). FLAIR imaging is sensitive for detection of SDH, subarachnoid hemorrhages and cerebral edema.[2] GRE images include T2* and susceptibility weighted imaging (SWI). These MR sequences are sensitive for detection of the oxidation products of hemoglobin and are valuable in identifying older shear injuries and small petechial hemorrhages.[2,5,8] SWI has a higher sensitivity when compared to T2*. It depicts 3-6 times as many microhemorrhages associated with DAI, as well as retinal hemorrhages, when compared to standard T2 * GRE imaging.[2,8] DWI images are important for detection of ischemic changes associated with AHT. In DWI imaging the ability of water molecules to diffuse in the extracellular space is represented. When there is cytotoxic edema in the case of an infarct or hypoxic ischemic injury (HII), the ability of water molecules to diffuse is restricted. The signal intensity is increased in the affected area on DWI images and is decreased in the matching apparent diffusion coefficient images.[5]

MRI scanning using the standard protocol is lengthy and requires sedation. Limited MR protocols have been suggested to mitigate the need for sedation.[5,9] In a study by Flom et al[9] a limited protocol was developed and evaluated on a cohort of 78 infants of whom 25 were diagnosed with AHT. The protocol includes axial T2, axial GRE, and coronal T1- inversion recovery. This limited protocol demonstrated a sensitivity of 100% and a specificity of 83% for intracranial hemorrhage. The Royal College of Paediatrics and Child Health and Royal College of Radiologists from the UK have developed a limited protocol, which consists of standard T1- and T2-weighted imaging combined with SWI and DWI.[5]

Advanced imaging techniques may add information helpful in predicting the long-term outcome better than conventional imaging. One technique is diffusion tensor imaging (DTI), which is a form of DWI that allows better evaluation of white matter tracts by assessing the intrinsic directionality (anisotropy) of water diffusion in the brain. DTI has shown to be useful in identifying white tract abnormalities, associated with DAI when conventional imaging is normal.[2] In 1 study by Imagawa et al,[10] infants subjected to AHT and a group of age matched controls were imaged with DTI. In the AHT group it was shown that decreased values of mean diffusivity and axial diffusivity were associated with more severe outcome.

Subdural Hematomas and Other Extra-axial Hemorrhages

In young infants with AHT, SDH is the most commonly observed intracranial lesion, reported in up to 90% of cases.[1,3,11] Para-falcine, interhemispheric high convexity or posterior fossa SDH location is highly associated with AHT.[1,3,12] These hematomas presumably occur secondary to acceleration, deceleration and torque motion during vigorous shaking, which lead to tears of bridging veins draining into the superior sagittal sinus. There are generally about 15-20 bridging veins which are short and straight veins that cross the subdural and subarachnoid spaces to drain into the superior sagittal sinus. The segments of the veins within the subdural space are most fragile.[12] In addition, the arachnoid membrane may rupture leading to mixing of blood with Cerebrospinal fluid and consequently cause the subdural collections to exhibit heterogeneous densities on CT scans. Mixed attenuation SDH or hemato-hygromas were found to be more common with AHT when compared to accidental trauma.[1] Other factors that may lead to a heterogeneous appearance of SDH include hemoglobin degradation, red blood cell hydration, integrity of cell membranes, protein content of the blood clot, sedimentation rate and concentration of red blood cells[2] (Fig. 2).

Venous injuries involving the bridging veins are highly associated with AHT.[13] Thrombi within or around the bridging veins may develop within hours from the injury.[12,14] On imaging, tubular shaped blood clots can be identified in the subdural space. The thrombosed veins may exhibit the "lollipop sign,"[12,13] which consists of the tubular shaped thrombosed vein terminating with a focal area of a largest diameter representing the clot in the surrounding subdural or subarachnoid space. These findings are best depicted with MRI on SWI or GRE images, exhibiting hypointense signal[12] (Fig. 3).

Accurate dating of SDHs based on imaging would provide valuable information and may help with identifying a certain perpetrator, however, based on the current scientific evidence this is not possible on CT or MRI. The current scientific evidence for dating intracranial hemorrhages on MRI is based on adult studies.[5] Studies on small cohorts of infants with AHT did not demonstrate any clear association between the timing of the injury and the CT density or MR intensity of hemorrhages.[5,15] SDHs in the setting of AHT are commonly of mixed attenuation. Furthermore, in some cases many episodes of shaking occur over a period of weeks or months and in others there might be a single episode with a certain caregiver. Evolution of the SDH over a serial short interval may be more valuable for dating than relying on the attenuation. Rapid change in the attenuation, redistribution of the hemorrhage and sedimentation of high attenuation product with formation of a fluid- fluid level suggest a more recent injury.[12] MRI examinations are more sensitive than CT in demonstrating membranes within the SDH which are seen in cases of subacute or chronic SDH.[2,16]

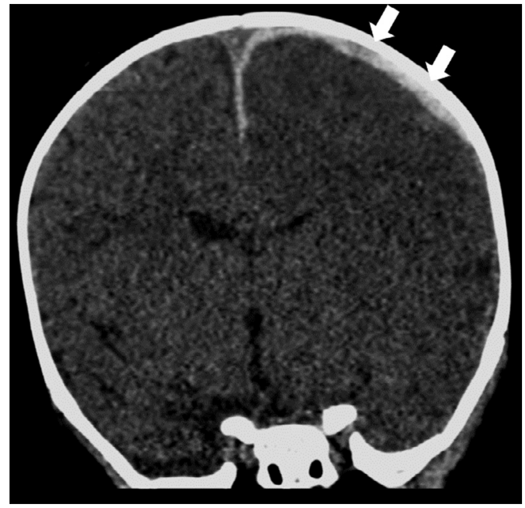


Figure 2 Mixed density SDH. Coronal reconstructed images from a brain CT scan of a 7 months old male subjected to AHT, with new onset of altered mental status and seizures, demonstrates a mixed density left subdural hematoma (arrows), which extends along the falx cerebri. In addition, there is edema in the left cerebral hemisphere with effacement of the left lateral ventricle.

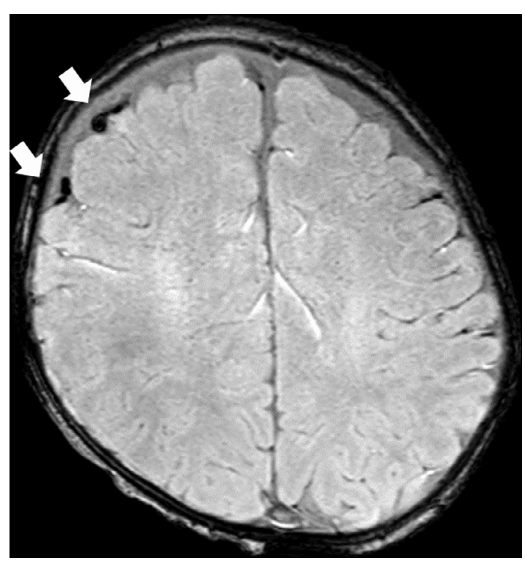


Figure 3 SDH with thrombosed subdural veins and "lollipop" sign. Axial SWI image from a brain MRI examination of a 9-week-old male subjected to AHT, who presented with an apneic episode, demonstrates a right SDH with thrombosed veins and a "lollipop" sign (arrows).

Parenchymal Injuries

Injury to the brain parenchyma may result from contact forces, inertial forces and from hypoxia and or ischemia. Diffuse parenchymal injury is rarely noted as an isolated finding in AHT except in cases of asphyxiation or strangulation. Although present in less than one-third of patients with AHT, parenchymal injury is the imaging finding most predictive of outcome.[17]

Contact forces may lead to contusions or lacerations. Inertial forces associated with shaking may lead to DAI. HII is caused by prolonged seizure activity or central apnea secondary to injury to the brainstem.

While contusions are not specific for AHT they are found in association with AHT and typically result from impact trauma. They tend to be located at the periphery of the cerebrum underlying scalp hematomas and/or skull fractures.[12]

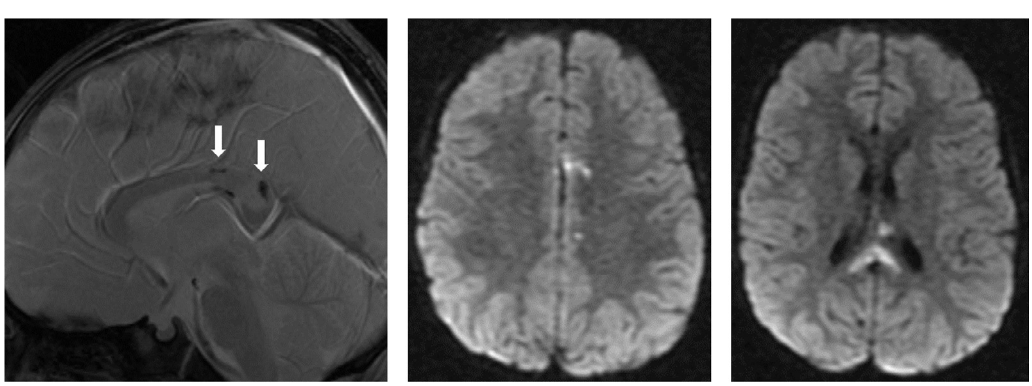
DAI is often a result of a rotational force leading to injury of neuronal axons. On MRI, numerous foci of abnormal signal are identified at characteristic locations, including the gray-white matter transition zones, the corpus callosum and the brainstem as well as the periventricular white matter.[2,12] The lesions typically exhibit high signal in T2 and FLAIR sequences. FLAIR is slightly more sensitive than T2 and SWI is substantially more sensitive than the former 2, as the foci may be hemorrhagic.[17,18] Microhemorrhages in SWI are associated with poor neurological outcome. Occasionally multiple punctate foci of restricted diffusion may be seen in DWI imaging[17,19] (Fig. 4).

HII is significantly associated with AHT and is seen in about a third of AHT patients.[12] A study by Ichord et al[20] compared 30 infants with AHT to a control group of 22 infants with accidental trauma. They evaluated DWI MRI findings and demonstrated that HII was 4 times more common in the AHT group when compared to controls. In the early phase MRI is more sensitive than CT for ischemic injuries and the most sensitive MRI sequence is DWI. Infarcted areas will demonstrate restricted diffusion (ie, hyperintense signal on DWI and hypointense signal on apparent diffusion coefficient). The most common imaging finding is a diffuse ischemic supratentorial involvement and a watershed infarct pattern in the cerebrum.[12,19] The posterior circulation territories including the brainstem, cerebellum and the thalami may be spared secondary to autoregulation. On CT the term "reversal sign" was coined to describe the characteristic appearance of HII. This consists of diffuse hypodensity of the cerebrum with loss of gray white matter differentiation while the posterior fossa appears relatively hyperdense with preserved gray white matter differentiation.[12] If the injury is more severe, posterior circulation structures including the brainstem, cerebellum and thalami will be involved as well. Global HII is associated with poor neurological outcome and mortality[2,4,12] (Fig. 5).

Parenchymal lacerations, also known as contusional tears, contusional clefts, or gliding contusions, are a unique entity seen in young infants, most commonly younger than 5 months, secondary to the fact that their white matter and particularly the subcortical white matter is not myelinated, leading to a significant difference in the consistency between the gray and white matter.[12,17] They result from either shaking or blunt trauma which leads to shearing of the white matter while the gray matter is spared. Palifka et al[21] found that these lesions are highly associated with AHT. On MRI they appear as sharply marginated irregular clefts in the gray-white matter transition zone. The clefts may be surrounded by hemorrhage and edema and are usually filled with fluid but may contain blood. As with other parenchymal findings, clefts are better depicted and characterized on MRI when compared to CT[12] (Fig. 6).

Skull Fractures

Skull fractures are equally common following accidental trauma and AHT, however, complex skull fractures are more common with AHT.[1,17] The probability of abuse in children with skull fractures is approximately 30%.[2,12] In a study by Kelly et al[11] on a cohort of 255 children under the age of 2 years of which 180 were diagnosed with AHT, it was found that while fractures were more common in the accidental



(A) (B) (C)

Figure 4 Diffuse axonal injury. (A) Sagittal T2* sequence from a brain MRI examination of a 2 years old subjected to AHT, who presented with altered mental status, demonstrates edema and hemorrhagic foci in the posterior corpus callosum consistent with DAI. (B and C) on DWI there are multiple foci of restricted diffusion in the corpus callosum and in the left cerebral hemisphere.

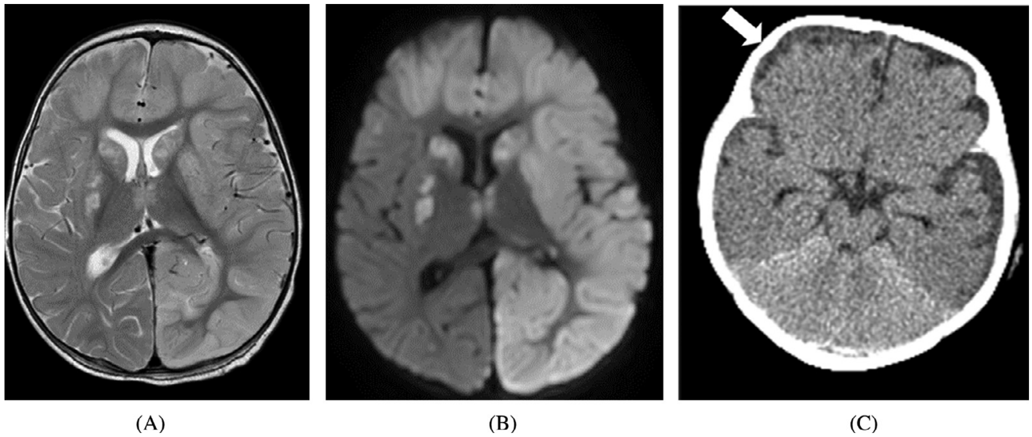


Figure 5 Hypoxic ischemic injury. (A) T2, and (B) DWI axial images from a brain MRI examination of 7 months old (same patient as in Fig. 2), demonstrating bilateral infracts involving the majority of the left cerebral hemisphere and the right frontal lobe, bilateral lentiform nuclei and medial thalami. (C) Axial cut from a brain CT scan of a 4 months old male, subjected to AHT, who presented with unresponsiveness, demonstrates diffuse hypodensity of the cerebrum with loss of gray white matter differentiation and a preserved cerebellum, consistent with the "reversal" sign. In addition there is a right frontal SDH (arrow).

injury group about a third of the AHT group patients had skull fractures. Fractures associated with AHT were more likely to be complex (ie, comminuted, depressed, compound, multiple, and skull base fractures) whereas fractures in the accidental trauma group were more likely simple (ie, linear, nondepressed) and most commonly located in the parietal bones.

When diagnosing a skull fracture, knowledge of sutural anatomy is paramount, and it is important not to mistake variant accessory sutures for fractures. Sutures typically have zigzag interdigitating sclerotic margins, while fracture fragments are straight and not sclerotic. Fractures may cross the sutures and widen them while sutures do not. Accessory parietal sutures are the most common type of accessory sutures. Accessory occipital sutures, also known as mendosal sutures, are also commonly encountered. Accessory sutures tend to be bilateral and symmetric and are rarely unilateral. Fractures are best depicted and differentiated from anatomical variants on 3D-surface-shaded-volume-rendered reconstructions of the skull performed on thin sliced CT scans[12] (Fig. 1).

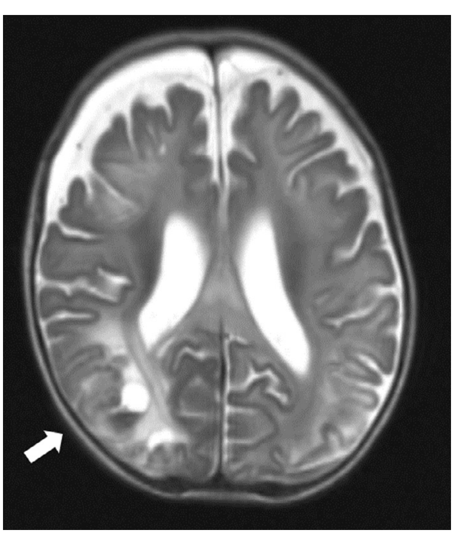


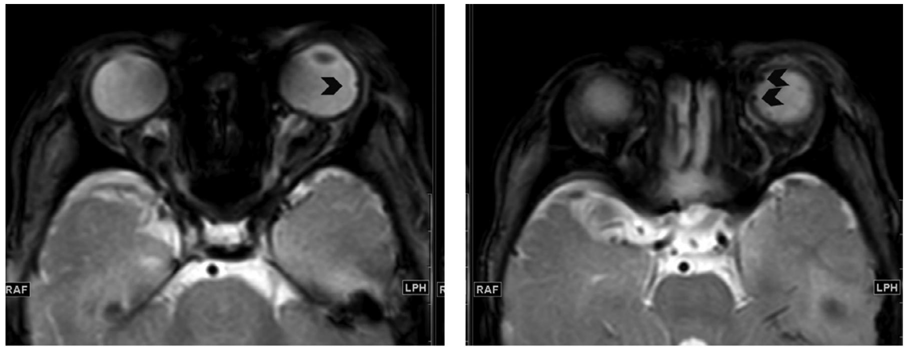
Figure 6 Parenchymal contusional cleft. Axial T2 sequence from a brain MRI examination of 4 months old (same patient as in Fig. 5C) demonstrates a right parieto-occipital cleft (arrow) in the gray white matter transition zone. The cleft contains hemorrhage forming a fluid/fluid level and is surrounded by edema.

Dating of skull fractures is inaccurate as imaging criteria used to date long bone fractures cannot be applied to those of the skull. In the skull there is no significant callus formation associated with healing of fractures.[12,17] Blurring of the fracture margins and new bone formation would suggest a subacute fracture, however these findings are not always present and therefore their absence does not exclude a subacute or chronic fracture.[17] While the presence of a scalp hematoma at the vicinity of a fracture is consistent with acuity, the absence of hematoma is nonspecific, as hematomas may not be present in the acute phase.[12,17]

Retinal Hemorrhages

Retinal and vitreous hemorrhages are presumably caused by rapid repetitive acceleration and deceleration forces from shaking or impact[12,17] and are common in association with AHT. They have been reported in 63%-92% of the cases in different series.[2,12,17,22] When seen in conjunction with AHT retinal hemorrhages tend to be numerous, bilateral, involve many layers of the retina and extend peripherally to the ora serrata. Vitreous hemorrhages and retinoschisis, a cavity secondary to separation of the retinal layers, may be also evident in fundoscopy in association with AHT.[12,23]

Retinal hemorrhages are associated with intracranial findings of AHT. A study by Binenbaum et al[24] reported an association between severe retinal hemorrhage and presence of HII in infants with AHT. Another study by Burkhart et al[25] evaluated a cohort of 168 children with AHT and reported



(A) (B)

Figure 7 Retinal hemorrhages. (A and B) axial T2* images through the orbits from a brain MRI examination of a 7 months old (same patient as in Fig. 2) demonstrate left retinal hemorrhages (arrowheads). On fundoscopy patient had sever left retinal hemorrhages and no right retinal hemorrhages.

an association of retinal hemorrhages with intracranial findings, including SDH, HII, and DAI and with intra-abdominal solid organ injuries in infants with AHT. They also reported that retinal hemorrhages were not found in the setting of skull or other fractures when intracranial hemorrhage was absent.

Retinal hemorrhages may occur with accidental head trauma; however, they are uncommon, seen in less than 10% of the cases, are often unilateral and generally limited to the posterior pole adjacent to the optic nerve and macula, and to a single layer of the retina.[2,12]

Retinal hemorrhages are best detected with fundoscopy. In all cases suspected of AHT indirect fundoscopy should be performed by an ophthalmologist as it has a higher sensitivity for detection of retinal hemorrhages when compared to direct fundoscopy, even with pupillary dilatation. Indirect fundoscopy typically evaluates a larger field of view including the peripheral aspect of the retina, when compared to direct fundoscopy.[23]

Nevertheless, high grade retinal hemorrhages, typically associated with AHT, may be visualized on imaging. This may be useful in cases where fundoscopy is not feasible or difficult. On CT, retinal hemorrhages have been described as hyperattenuating foci in the posterior wall of the eye globe. The sensitivity of MRI for detection of retinal hemorrhages is significantly higher when compared to CT. On MRI retinal hemorrhages present as dark foci in GRE sequences.[26] Similar to other hemorrhagic foci, retinal hemorrhages are detected with higher sensitivity in SWI when compared to T2*[17] (Fig. 7). Zuccoli et al[26] conducted a study in which they evaluated a cohort of 28 infants suspected of AHT, and found that a standard SWI sequence had a sensitivity of 62% for detection of retinal hemorrhages when compared to the gold standard of fundoscopy. Moreover, a dedicated high resolution SWI of the orbits increased the sensitivity to 80%, and all the missed cases were graded as mild on fundoscopy.

Spinal Injuries

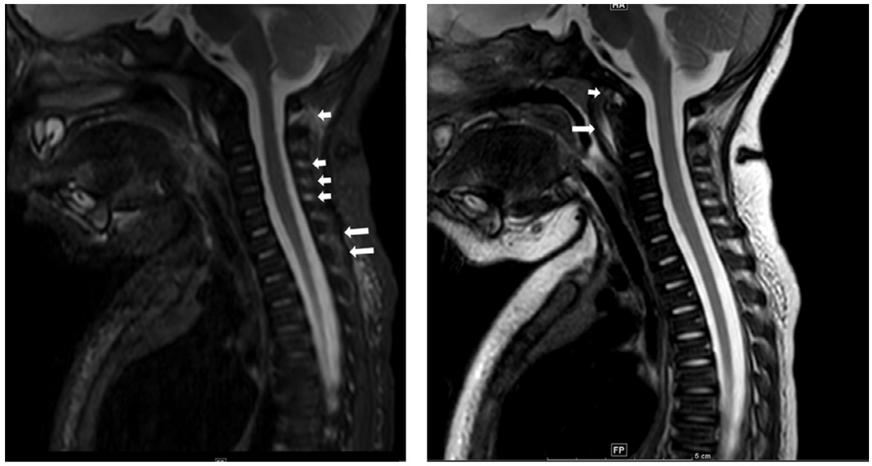
Spinal injuries were previously considered rare in association with nonaccidental trauma as they were not detected on skeletal surveys.[12,17] In recent years it has been recognized that spinal injuries frequently occur in infants subjected to AHT, however, they tend to involve soft tissues rather than bones hence they are detected only on MRI and in autopsies.[1,12]

In infants there are anatomical features in the craniocervical junction that lead to excessive motion with violent shaking. This includes a relatively large size of the head in proportion to the neck, low muscle tone, horizontally oriented facet joints, incomplete ossification of the vertebrae, and ligamentous laxity.[27] As a result, while fractures are uncommon, soft tissue injuries, including ligamentous injuries, SDH and injuries to the spinal cord are far more common.[1,27] Jacob et al[28] evaluated 89 young children subjected to AHT who had MRI examinations of the cervical spine and found injuries to the cervical spine in 69%, of which 98% were ligamentous injuries. Ligamentous injuries and spinal SDH were reported in other studies in 36%-78% and 44%-63% of AHT cases respectively.[1,17] Posterior ligamentous complex injuries to the craniocervical junction, involving the nuchal, atlanto-occipital as well as the atlanto-axial ligament, are more common with AHT than with accidental trauma. These injuries are typically seen in young infants and are consistent with hyperflexion mechanism.[12,27,29] Prevertebral edema may be seen as well (Fig. 8). Injuries to the anterior and posterior longitudinal ligaments are rare in association with AHT.[12]

Spinal SDH are more commonly identified in the thoracolumbar spine when compared to the cervical spine and are associated with posterior fossa SDH. They may be secondary to gravitation of intracranial SDH to dependent areas or may result from local venous injury to the thoracolumbar junction from hyperflexion/ hyperextension during shaking.[27]

Retroclival collections which extend to the craniocervical junction have been described in a high rate (32%) in association with AHT in a study by Silvera et al.[30]

Spinal fractures are relatively uncommon in association with AHT. They were reported in 0.8%-9.7% of positive skeletal surveys with the most common finding being compression fractures of vertebral bodies in the thoracic or lumbar spine,[27] which supports the theory of hyperflexion injury at this region during shaking.



(A) (B)

Figure 8 Ligamentous injury of the cervical spine. (A) STIR, and (B) T2, sagittal images from a cervical spine MRI examination of a 7 months old male (same patient as in Fig. 2) demonstrate ligamentous injury. There is high signal intensity at the interspinous ligaments (short arrows in figure A) and supraspinous ligaments (long arrows in figure A), as well as, in the anterior atlanto-occipital ligament and surrounding it (short arrow in figure B). There is also prevertebral edema at the cranio-cervical junction (long arrow on figure B). STIR = short tou inversion recovery.

There is an association between spinal injuries and HII in infants with AHT and it has been theorized that this is secondary to involvement of regulatory centers in the medulla and superior cervical cord, which lead to prolonged apnea.[3,27]

In conclusion, soft tissue injuries to the spine are common in the setting of AHT and therefore MRI examinations of the spine should be considered in all infants undergoing MRI of the brain for the assessment of AHT. MRI examinations should include T1 and T2 weighted sequences as well as fat saturated fluid sensitive sequences (ie, T2 with fat suppression or short tau inversion recovery). While MRI of the cervical spine may show ligamentous injury to the craniocervical junction and injury to the spinal cord, MRI of the thoracic and lumbar spine may present an added value. This is secondary to the fact that SDHs have been detected with higher rates in the thoracic and lumbar spine than in the cervical spine in infants with AHT, and since vertebral body fractures which are also more common in the thoracic and lumbar spine in association with AHT, are detected with higher sensitivity in MRI with fat saturated fluid sensitive sequences when compared to skeletal surveys.[17,27,29]

Alternative Theories and Mimickers

Entities exhibiting imaging findings which mimic findings of AHT may present a diagnostic or legal dilemma. In many of the cases the differentiation between AHT and another entity is clear since infants who were subjected to AHT exhibit many of the findings described in the prior sections including SDH, parenchymal intracranial injuries, retinal hemorrhages, spinal, and skeletal injuries. In contrast, other entities typically present with a single finding, most commonly SDH. In addition, the clinical presentations are commonly different. This strengthens the notion that the diagnosis of AHT should be made with a multidisciplinary approach. Several entities will be described in this section.

SDH are common in neonates secondary to birth trauma. In most cases they are present at the posterior fossa or in the parieto-occipital regions and are asymptomatic. They usually resolve by the age of 4-6 weeks and do not tend to rebleed. Their density is homogenous in contrast to the mixed density of SDH typically observed with AHT. Acute decompensation cannot be attributed to birth related SDH after the first days of life[1,31] (Fig. 9).

Benign enlargement of the subarachnoid spaces (BESS) is a common condition in infants who present with macrocephaly. On imaging these infants have prominent subarachnoid spaces along the convexities, and mild enlargement of the lateral and third ventricles. It is theorized that disequilibrium between production and resorption of cerebrospinal fluid leads to this finding and it usually resolves by the age of 2 years and requires no intervention.[5,31] Reports of spontaneous SDH in infants with BESS exist in the literature[1,12,31,32] and it is thought to be secondary to excessive motion of the brain within the skull and stretching of bridging veins due to the enlarged extra axial space. Latest reviews reveal that less than 6% of infants with BESS will develop SDH.[1] In these cases, SDH is typically an isolated finding, with no associated parenchymal brain injuries or retinal hemorrhages and with no associated neurological collapse as typically observed in infants subjected to AHT[31] (Fig. 10).

Glutaric aciduria type 1 (GA1) is a rare metabolic disorder of glutaryl-CoA-dehydrogenase enzyme deficiency. Children with GA1 are reported to be predisposed to SDH development due to stretching of bridging veins secondary to cerebral atrophy and expansion of the extra-axial spaces.[33] Clinical presentation of GA1 during infancy includes developmental delay, dystonia and occasionally movement

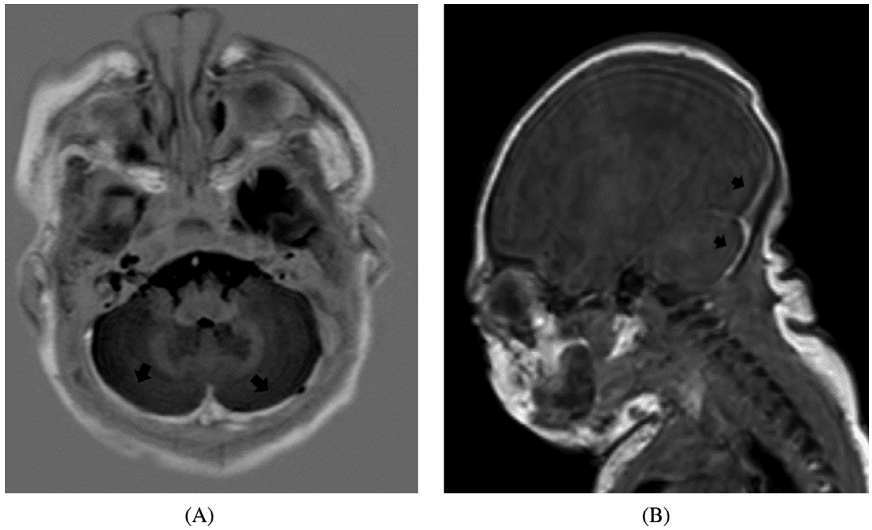


Figure 9 SDH in a newborn. (A) axial T1 IR and (B) sagittal T1 images from a brain MRI examination performed on a 4-day old newborn female who presented with a seizure, demonstrate an incidental finding of an SDH in the occipital region and in the posterior fossa (arrows). No other abnormalities were identified on this examination.

disorders. MRI findings in the majority of patients include atrophy of the frontal and parietal lobes with prominence of the subarachnoid spaces and open opercula. Other findings include white matter and basal ganglia signal abnormalities, arachnoid cysts and ventriculomegaly.[32,33] The differentiation between an infant with GA1 presenting with an SDH and an infant subjected to AHT should be clear based on clinical presentation and MRI findings of GA1 that would typically be present in an association with SDH in the former[34] (Fig. 11).

In other cases, there are unsubstantiated alternative theories, not backed by scientific literature, that typically arise in courts. Examples include cerebral sinus venous thrombosis or coughing and choking as a cause for retinal hemorrhages and SDH, and intracranial hypotension from lumbar puncture as a cause of SDH. All of these theories are not supported scientifically by large series of cases and rely on anecdotal case reports.[1] In addition, in all these entities, parenchymal injuries to the brain including DAI and HII, spinal injuries, and other skeletal injuries are not present.

Summary

There are characteristic imaging findings of AHT in the head and spine and usually many of these findings are present in an infant subjected to AHT. Most common findings are SDH and parenchymal injuries to the brain. Spinal injuries occur commonly at the craniocervical junction and involve soft tissues rather than bones.

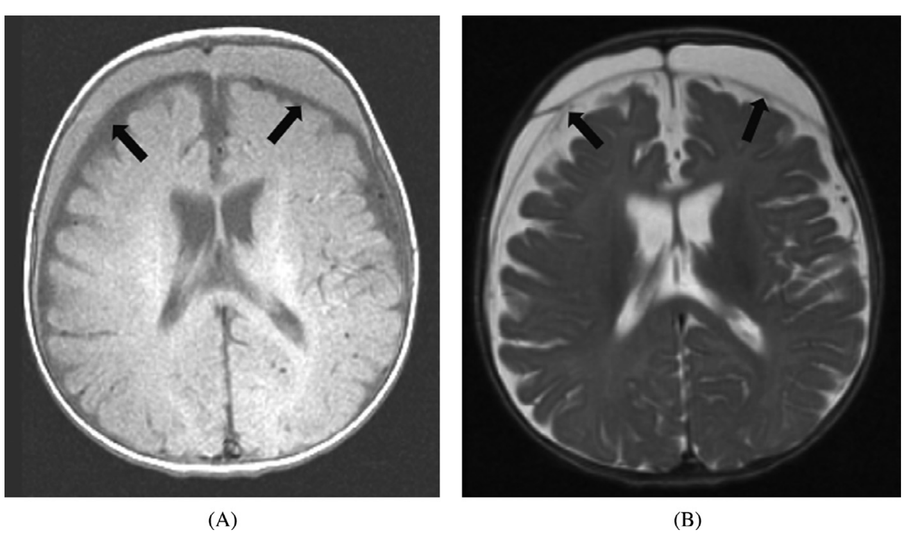


Figure 10 Benign enlargement of the subarachnoid spaces (BESS) with chronic SDH. (A) axial T1, and (B) axial T2 images from a brain MRI examination of a 4 months old male with macrocephaly and otherwise asymptomatic, demonstrate bilateral subdural collections (arrows) and enlarged subarachnoid spaces. The subdural collections were drained and were found to be chronic SDH.

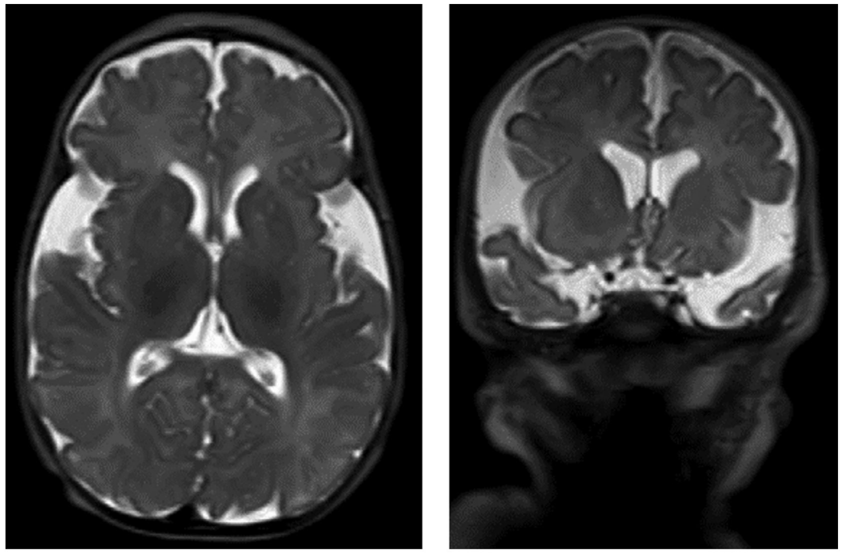


Figure 11 Glutaric aciduria type 1. (A) axial, and (B) coronal T2 images from a brain MRI examination of a 3 months old female infant who presented with macrocephaly and was later diagnosed with Glutaric aciduria type 1, demonstrate prominent subarachnoid spaces in the frontal and temporal regions with enlarged sylvian fissures.

CT of the head is the initial modality of choice in the acute phase, as it is fast, readily available and does not require sedation. CT provides valuable information with regard to the need for an emergent intervention. Skull fractures are best evaluated with 3D-surface-shaded-volume-rendered reconstructions of the skull performed on thin sliced CT scans.

MRI is the most sensitive modality for detection of intracranial and spinal injuries and is indicated in infants subjected to AHT. MRI scans of the brain should include spin echo as well as DWI and SWI sequences to maximize the detection of SDHs and parenchymal injuries typically observed in infants subjected to AHT. MRI of the Spine should be considered as well in all infants who undergo MRI examinations of the brain for evaluation of AHT.

It is important to acknowledge that the diagnosis of AHT should be based on multidisciplinary evaluation and that accurate dating of injuries is usually not possible based on imaging.

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