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REVIEW

# Neuroimaging of abusive head trauma

**Gary L. Hedlund · Lori D. Frasier**

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**Abstract** The diagnostic process for evaluating suspected abusive head trauma in infants and children has evolved with technological advances in neuroimaging. Since Caffey first described a series of children with chronic subdural hematomas and multiple long bone fractures, radiologists have played an important role, along with pediatricians and pathologists, in evaluating abused children. Neuroimaging modalities include ultrasound, CT scans, and MRI technology. Each has distinct clinical applications, as well as practical uses in the clinical diagnostic process of AHT. Importantly, neuroimaging assists in the process of differential diagnosis of other conditions which may mimic AHT. Collaboration between neuroradiologists, clinicians, and pathologists remains critical to making the appropriate diagnosis. Careful history, physical examination, and investigation by legal authorities form the components that result in accurate assessment of any case. This paper reviews pertinent neuroimaging modalities currently utilized in the diagnosis of AHT, describing clinical indications and a collaborative approach to this process.

**Keywords** Child Abuse · Abusive head trauma · Neuroimaging · MRI · CT scan · Ultrasound · Subdural hematomas · Skull fracture · Metabolic disease

G. L. Hedlund
Pediatric Medical Imaging Department, Primary Children's Medical Center, 100 No. Mario Capecchi Drive, Salt Lake City, UT 84113, USA
e-mail: Gary.hedlund@imail.org

L. D. Frasier (✉)
Center for Safe and Healthy Families, Primary Children's Medical Center, University of Utah School of Medicine, 675 East 500 South, Salt Lake City, UT 84102, USA
e-mail: loridf@gmail.com

## Introduction

The diagnosis of abusive head trauma has evolved in recent years. Abusive head trauma results in the greatest risk of mortality and morbidity of infants due to physical child abuse [1]. This type of abuse is reflected in injury to the scalp, bony cranium, and intracranial contents, most specifically the brain and related structures. Much of what we currently know about clinical presentation and natural history of abusive head trauma depends upon neuroimaging techniques. In 1946, John Caffey, the undisputed 'father' of pediatric radiology, observed and published case reports of six infants whose principal disease was chronic subdural hematoma. Chronic subdural in 1946 was diagnosed by presentation of enlarged head, bulging fontanel, and neurologic symptomatology. These six patients also had 23 fractures and contusions of the long bones, that in some cases were much more acute than the chronic subdural hematoma [2]. It appeared to Caffey that these children had suffered some type of trauma. None of the bone lesions could be attributed to disease or conditions that caused the bones to be fragile. A history of trauma was not provided. It was rare in that era to consider child abuse as a cause of children's injuries. Caffey's observation was that "it is unlikely that trivial, unrecognizable trauma caused complete fractures of the femur." Caffey continued to observe and collect cases of children who were apparently seriously injured with fractures and head trauma. In 1974 he published his sentinel paper in which he linked violent manual shaking of an infant to brain damage, ocular hemorrhages, and residual mental retardation. In this study, he described cases, and reviewed the pediatric, neurosurgery, radiology and biomechanical literature available at the time [3]. Caffey emphasized that a prominent feature in many cases was the lack of external evidence of abuse. The lack of

external injuries, as well as misleading histories from the caregiver, often confounded the clinician as to the proper diagnosis of a child's symptoms. He also was one of the first physicians to point out that failure to recognize an abused child could result in repeated trauma, further disability, or death. This fact has been validated in more current studies of "missed" abusive head trauma, and clinician's recognition that many seriously injured and murdered children had earlier indicators that they were abused [4].

Prior to the development of radiologic modalities that could noninvasively image the brain, physicians were trained to recognize physical signs and symptoms of intracranial injury following trauma. The location of potential brain lesions was determined through careful examination of the patient. Treatment involved educated guesswork by the surgeon. The pathologist may have been the first to recognize unexpected signs of head trauma post mortem. Pneumoencephaography, angiography, and particularly in infants, ultrasound through open fontanel were earlier imaging techniques. Each of these modalities had their limitations. Computerized Axial Tomography (CT), introduced in the 1970s, opened a window into the brain and provided tremendous advantages in diagnosis of intracranial processes.

The subsequent introduction of magnetic resonance imaging (MRI), offers advantages over CT scanning, without the risk of radiation exposure. This article will discuss current neuroimaging techniques in the context of the evaluation of abusive head trauma. Advantages, disadvantages and clinical applications of different modalities will also be discussed.

CT brain imaging, being almost universally available and able to acquire images rapidly, remains the first-line imaging technique when intracranial pathology is suspected. CT also offers advantages for the unstable patient. In abusive head trauma, the brain CT may provide the first indication that a child is abused. As noted originally by Caffey, the lack of external physical findings of trauma without a history of a traumatic event may result in missing the diagnosis of abusive head trauma. Caffey, and subsequent others, have confirmed that missing a diagnosis of serious child abuse places a child at significant risk of further abuse resulting disability or death. The ubiquity of CT and its round the clock use, can quickly lead to recognition of intracranial pathology and correct diagnoses whether abusive or not.

CT has been suggested as a screening test for abusive head trauma in children who present with less specific symptoms. Apparent Life Threatening Events (ALTEs) are an example of a symptom that has many causes (infection, gastroesophageal reflux, seizure, endocrine etc.). Abusive head trauma is clearly an etiology of some ALTEs. Early detection of intracranial pathology utilizing CT scanning, as well as other adjunct assessments such as dilated fundoscopic examinations may be useful in the early detection of abuse [5–7]. The child abuse pediatrician or other clinicians with specialized expertise in evaluating child abuse collaborate extensively with many subspecialities. Neuroimaging of these children is standard medical practice. Therefore, consultation with radiologists and neuroradiolgists is critical in making an appropriate and accurate diagnosis.

## CT imaging in abusive head trauma

History: CT (computed tomography) invented in 1972 represents the collaboration between the British engineer, Godfrey Hounsfield—then working at EMI Laboratories in England, and Allen Cormach—a physicist working at Tufts University in Massachusetts.

CT imaging began to contribute to clinical care in 1974. By the early 1980s, CT units were widely available throughout North America. Original CT systems were dedicated to "head only" imaging. The time required to complete a head CT was on the order of 30 min.

Now almost 30 years after its invention, the original single slice "step and shoot" technology has been replaced by multi-slice CT systems capable of completing a pediatric head CT in less than 10 s.

The advantages of CT in the evaluation of accidental or inflicted head trauma are many. Using current multi-slice scanners, characterization of brain anatomy, investigation of the brain coverings (dura and arachnoid), bony skull base and calvarium is very rapidly achieved even with an unstable patient, and usually without the need for sedation [8].

In the setting of suspected inflicted or accidental trauma, the availability of CT to rapidly answer clinical questions and aid in planning treatment including neurosurgical intervention remains unchallenged.

In addition to the standard assessment of axial CT data for intracranial hemorrhage, cerebral edema, early herniation and bony injury, rapid image reformatting (2D maximal intensity projections [MIP] or 3D surface rendered reconstructions) of the original axial data often gives insights into the precise location of hemorrhage, allows the distinction of normal venous sinus blood from adjacent subdural blood, and improves the detection and identification of the extent of fractures (Fig. 1). In our Department of Pediatric Medical Imaging, the 3D surface rendered reconstructions are performed on all patients with accidental or suspected abusive head trauma (Fig. 2a, b, c). In the setting of suspected AHT when CT of the brain has been accomplished, conventional skull

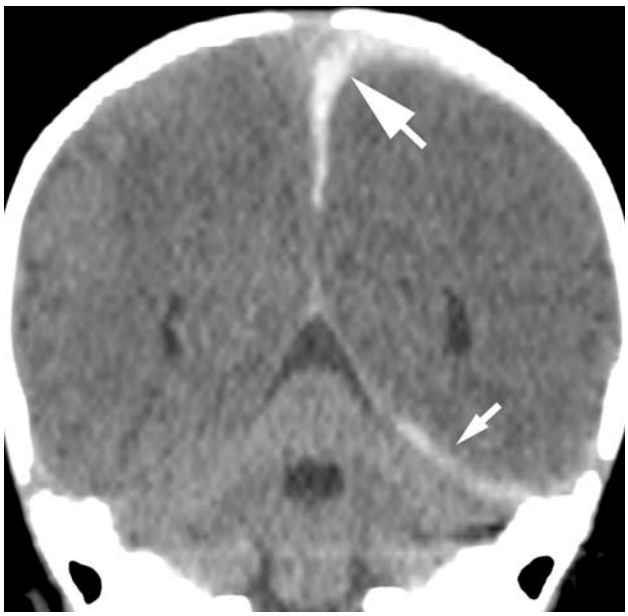

**Fig. 1** Acute parafalcine subdural hemorrhage. Coronal NCCT reconstruction shows a high attenuation left parafalcine SDH (*large arrow*), not to be mistaken for clot in the saggital sinus. Also note the smaller left paratentorial SDH (*small arrow*)

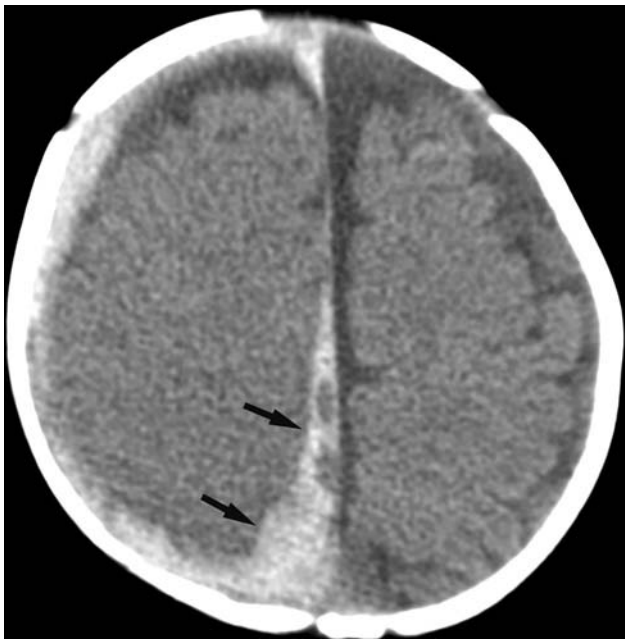

**Fig. 3** Mixed attenuation acute subdural hemorrhage (*arrows*). NCCT demonstrates heterogeneous falcine and right convexity SDH found to be acute at the time of surgical drainage

radiography is omitted in those patients who have a follow up osseous survey, thus sparing the child added radiation exposure. Additionally, in real time when the clinical and/or preliminary CT imaging is suspicious for neurovascular injury (carotid or vertebral arterial dissection), CT angiography (CTA), with multi-slice technique, can rapidly characterize craniocervical vasculature with high fidelity [9, 10].

## Typical features of SDH

Typical features of subdural hemorrhage (SDH) in abusive head trauma include mixed attenuation collections located over the cerebral convexities, within or adjacent to the falx cerebri, middle cranial fossa, subtemporal, and paratentorial locations (Fig. 3) [11]. Subdural hemorrhages may appear contracoup to the site of impact. Observing mixed attenuation acute subdural compartment collections carries a differential diagnosis. Considerations include mixed attenuation of acute subdural hemorrhage where heterogeneity reflects acute clot, active bleeding, serum—clot separation, mixture of blood and cerebral spinal fluid and the possible settling of more dense hemorrhage components (hematocrit effect) (Fig. 4). Alternatively, the mixed attenuation collection may represent the presence of acute hemorrhage in a pre-existing older subdural compartment collection. In my experience (GH), the pattern of acute

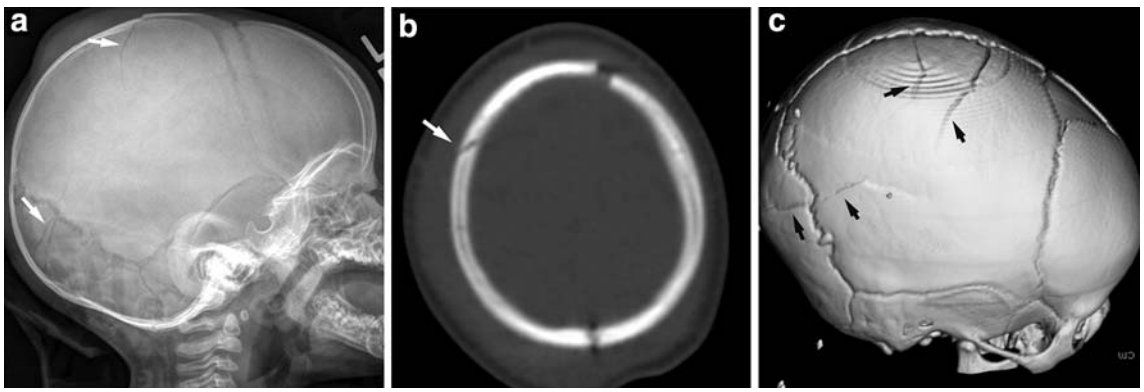


**Fig. 2** Skull fracture in the setting of abusive head trauma. **a** Lateral skull radiograph shows linear skull fractures involving the parietal, and occipital bones (*arrows*). **b** Axial CT underestimates the complexity of the skull fractures (*arrow*). **c** Surface rendered 3D CT clearly shows the complexity and extent of the fractures (*arrows*)

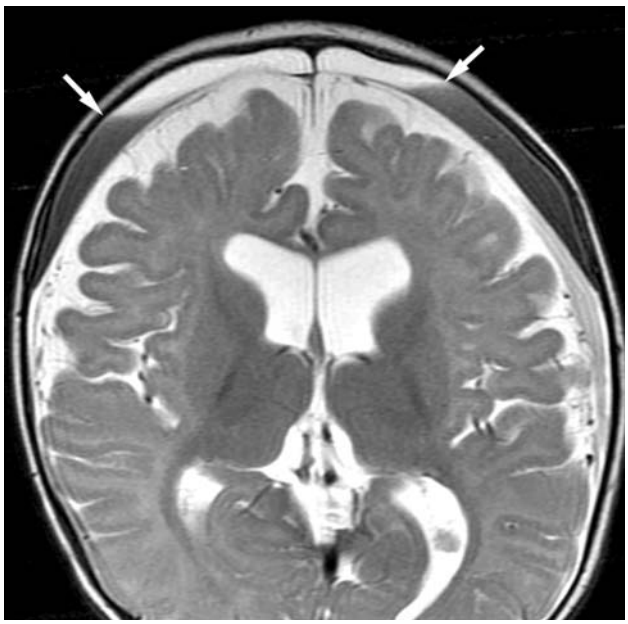

**Fig. 4** Hematocrit effect within acute subdural hemorrhage. Axial T2WI showing dependant hypointense hemorrhage (*arrows*)

mixed attenuation subdural hemorrhage is typically accompanied by underlying cerebral edema and/or ischemia. Homogeneous hyperattenuating subdural hematoma is more frequent in cases of accidental head trauma [9, 11, 12].

Although non-contrast brain CT is the preferred initial imaging exam in the investigation of abusive head trauma, there are occasions where supplemental contrast enhanced brain CT imaging will prove useful. At times, assigning intracranial extra-axial fluid to either the subarachnoid or subdural compartment on non-contrast brain CT may be confounding. Supplemental intravenous contrast enhanced brain CT with reformations in multiple planes can, in most cases, answer the question as to whether the fluid is subarachnoid or subdural in location (Fig. 5a, b). Additionally, contrast enhanced brain CT imaging can confidently differentiate between abnormalities of the intracranial venous sinus, such as sinus venous thrombosis from intradural or subdural compartment collections [9, 12].

The downside of early cranial CT in the setting of suspected abusive head trauma is its lack of sensitivity in detecting petechial hemorrhages, non-hemorrhagic strain and shear injury, ischemic edema, and ligamentous injuries of the craniocervical junction.

## Magnetic resonance imaging

Magnetic resonance imaging (MRI) is a modality particularly well suited for imaging the neuraxis (brain and spine). Although early understanding of the principals of nuclear magnetic resonance began in the 1930s, and culminated in the 1940s, it was not until 1977 that the first human patient was scanned.

In the setting of suspected abusive head trauma, CT remains the most practical imaging tool for rapid assessment and triage of the injured child [8]. MRI plays an important role in completely characterizing the extent of intracranial hemorrhage and CNS injury in symptomatic children as well as "asymptomatic" patients [9, 10].

Subdural hemorrhages are common in abusive head trauma. Most hemorrhages are parafalcine, situated over the cerebral convexities, inferior to the frontal and temporal lobes or found along the tentorium cerebelli. When they are small and older than 1 week in age, the SDHs may be difficult to detect with CT [9, 12].

Small accumulations of subdural, subarachnoid, intraventricular and intraparenchymal blood can be identified by using MRI sequences that are sensitive to detecting the oxidation products of hemoglobin. These sequences include gradient recall imaging (GRE) and susceptibility weighted imaging (SWI) techniques (Fig. 6) [13–15]. These blood sensitive scan sequences are currently offered by most MRI vendors.

In addition to compartmentalizing the intracranial hemorrhage, MRI is useful in depicting hemorrhagic characteristics such as the heterogeneity of signal intensity (acute or mixed age SDHs), membrane loculated subdural hemorrhage, and hematocrit effect within the subdural hemorrhage. Taken in the context of other intracranial injuries, these observations may give insight into the temporal continuum of abusive head trauma. Admittedly, MRI has some limitations in dating the age of intracranial hemorrhages. However, when clinical signs and symptoms are interpreted in conjunction with CT imaging and comprehensive findings of all MRI pulse sequences and MRI adjuncts, the neuroradiologist is often able to add useful chronological information beyond that of CT imaging [10, 16, 17].

The identification of intracranial hemorrhage in the setting of abusive head trauma is often the proxy for underlying brain injury. Standard MRI pulse sequences demonstrate the integrity of anatomic relationships, depict localized and generalized cerebral edema, and characterize the features of early cerebral herniation. Fluid attenuated inversion recovery imaging (FLAIR) is particularly helpful in detecting cerebral edema, contusions (coup and countercoup), shearing injuries (diffuse axonal injury [DAI]) and parenchymal lacerations. Another advantage of FLAIR imaging is its great sensitivity in detecting small SDHs adjacent to the subarachnoid space (Fig. 7) [9, 10].

Diffusion weighted imaging (DWI) and apparent diffusion coefficient (ADC) maps have added much to the neuroradiologists' understanding of traumatic brain injury.

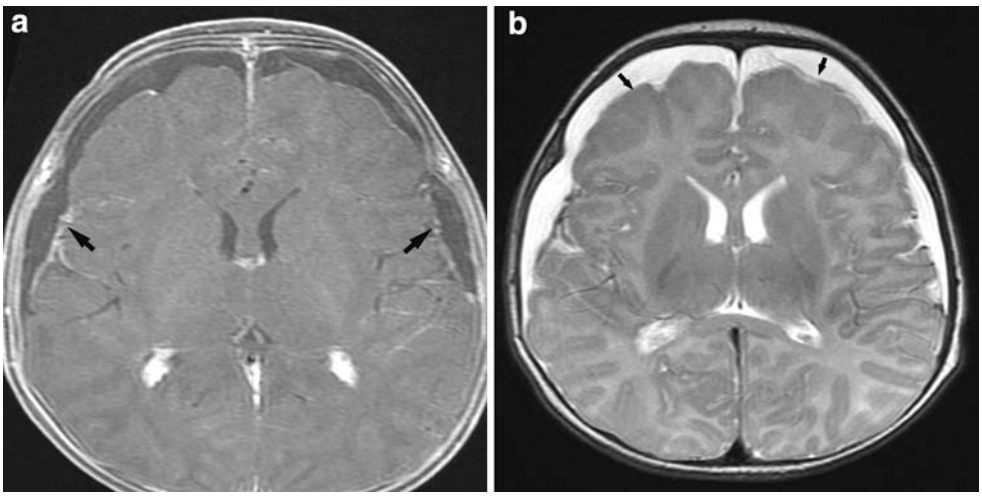


**Fig. 5** Chronic SDH mimics benign enlarged extraaxial subarachnoid fluid spaces. **a** CECT shows symmetric fronto-temporal subdural fluid collections. Note the enhancing cortical veins hugging the surface of the temporal lobes (*arrow*). **b** Axial T2WI shows minimal gyral compression by the hyperintense chronic subdural collections (*arrows*)

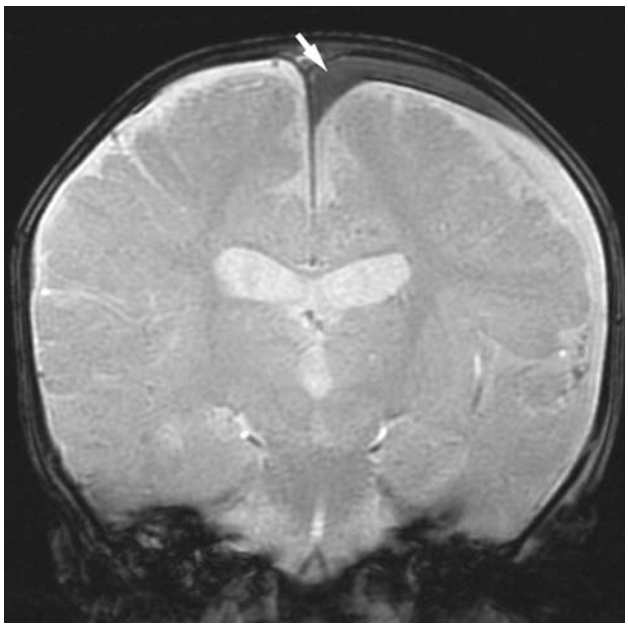

**Fig. 6** Coronal GRE (blood sensitive) MR sequence demonstrates a hypointense left frontal subdural hemorrhage (*arrow*)

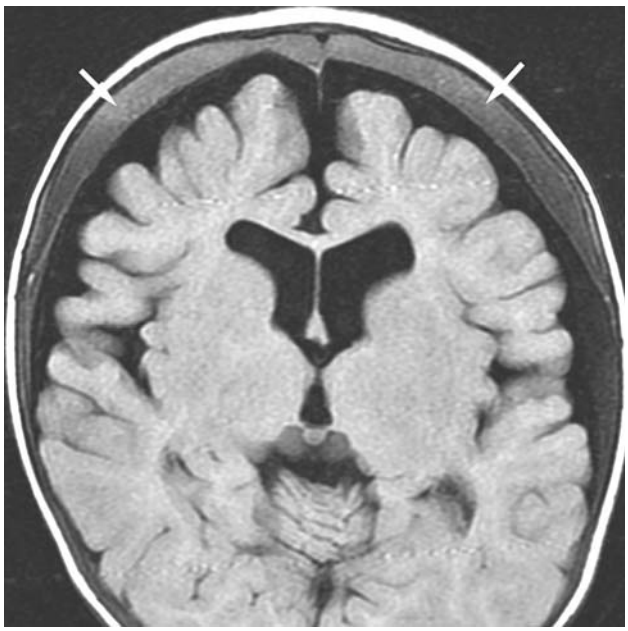

**Fig. 7** Axial FLAIR MR image shows bilateral frontal subdural collections of increased signal (*arrows*) adjacent to expanded hypointense subarachnoid spaces

In our medical imaging practice, we have found diffusion imaging particularly helpful in assessing for hypoxic-ischemic injury that may follow apnea, respiratory arrest, and transient brainstem or upper cervical cord stretching injury in the setting of AHT (Fig. 8). DWI provides insights into CNS tissue strain, shear, axonal disruption and parenchymal laceration. Diffuse axonal injury as a manifestation of tissue shear strain is associated with alterations of molecular water movement and resultant signal alterations on diffusion weighted imaging (cytotoxic edema) [15, 18]. Observing diffusion restriction at the margins of a cerebral parenchymal laceration gives insight into the age of the disruptive injury (Fig. 9a, b). This diffusion information routinely helps in dating the injury and in prognosticating patient outcome [10]. Diffusion tensor imaging (DTI) and tractography represent advances in display of diffusion data.

Contrast enhanced cerebral MRI may on occasion contribute useful information in the setting of suspected abusive head trauma. As was mentioned in the section on CT imaging, assigning the precise location of extra-axial fluid collections can be enhanced with intravenous contrast

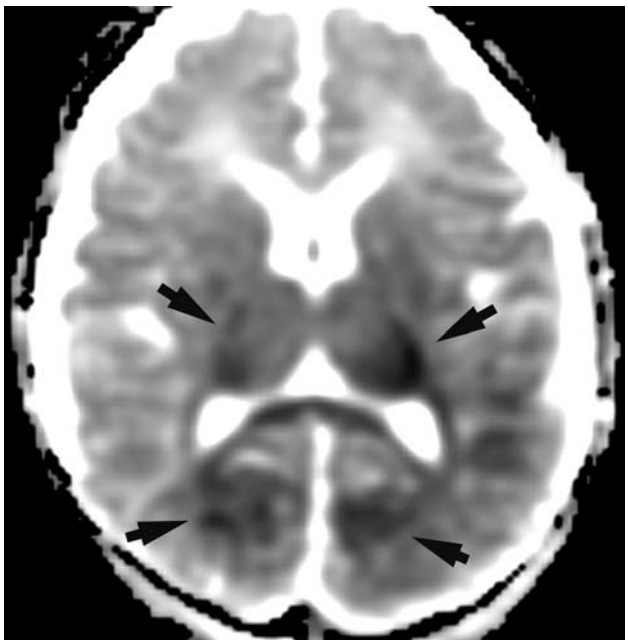

**Fig. 8** Axial ADC map from diffusion MRI sequence showing thalamic and temporal-occipital regions of hypointensity (*arrow*) reflecting cytotoxic edema secondary to a hypoxic insult that followed a shaking injury

imaging. In confounding cases where leptomeningeal infectious debris or cellular material (leukemic cells) mimic hemorrhage, the use of intravenous contrast can prove invaluable. In the context of atypical intracranial hemorrhage, the detection of intracranial venous thrombosis or the presence of an intracerebral or dural arteriovenous malformation may be illuminated by performing contrast enhanced MRI sequences.

Vascular imaging MRI adjuncts include magnetic resonance venography (MRV) and magnetic resonance angiography (MRA). These sequences add only minutes to the routine MRI examination. MRV is typically performed following intravenous contrast and is capable of detecting thrombi in superficial and central venous structures. MRA techniques exploit the movement of protons in flowing blood. Current MRA techniques are particularly helpful in characterizing cervical vasculature, skull base arterial structures, and the circle of Willis arterial anatomy. Beyond the sylvian cisterns, the arterial branches are seen with variable resolution.

Magnetic resonance spectroscopy (MRS) yields useful biochemical information acutely in characterizing ischemic of infarcted tissue (Fig. 10). Additionally, subacute or chronic injury shows decline of *N*-acetylaspartic acid (NAA); a marker of neuronal integrity and density [16, 17].

Another important contribution of MRI in the setting of suspected AHT is evaluation of the craniovertebral junction with particular attention to strain or disruption of the stabilizing ligaments, detection of atlanto-axial separation, injury of the interspinous ligaments, vertebral body compression, and epidural and intradural hemorrhages [19]. The use of heavily T2-weighted fat suppressed sequences such as a short tau inversion recovery (STIR) enhances detection of bone, ligament, and adjacent soft tissue edema. It has become our practice to include supplemental craniocervical junction, cervical spine and upper to mid thoracic spine magnetic resonance imaging at the time of cerebral MRI in cases of suspected AHT (Fig. 11).

Magnetic resonance imaging has been reported to identify subdural hematoma in infants where CT was negative. Sato demonstrated that MR was superior to CT in detecting SDH due to child abuse [20]. Levin and colleagues reported on a child with seizures and retinal hemorrhages in whom the CT was normal and the MR

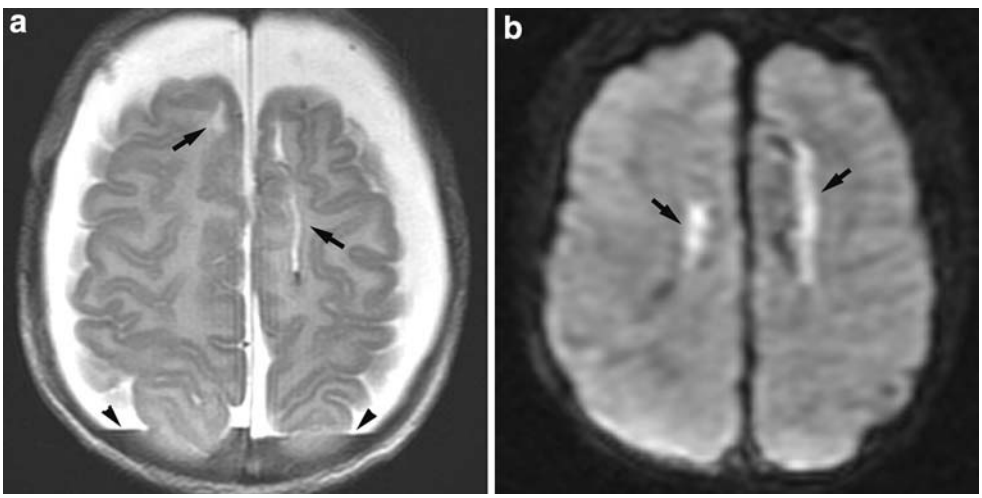


**Fig. 9** Parenchymal laceration. **a** Axial T2WI shows bilateral frontal sub-cortical linear parenchymal lacerations (*arrows*). Also note the dependant subdural hemorrhage hematocrit effect (*arrowheads*). **b** Axial DWI demonstrates corresponding linear diffusion hyperintensity (*arrows*). This reflects cytotoxic edema and axonal disruption. These findings are likely to be less than 7–10 days of age

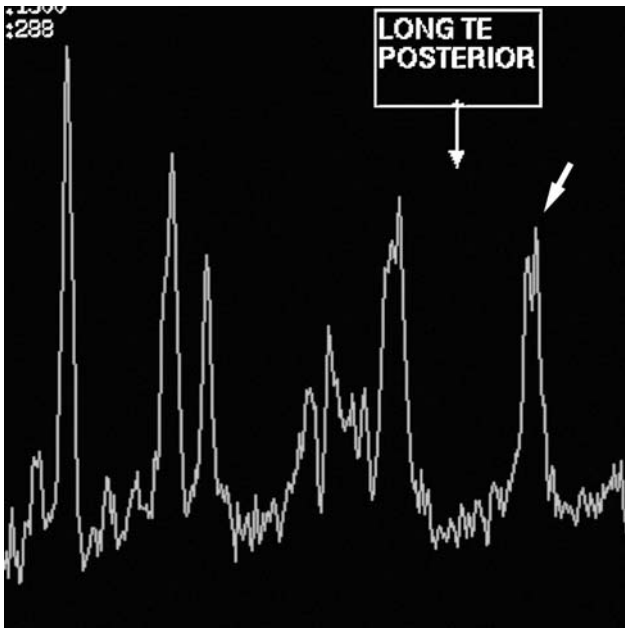


**Fig. 10** Magnetic resonance spectroscopy (MRS) of the left occipital lobe from Fig. 8, shows a large lactate peak reflecting disordered oxidative metabolism (*arrow*)

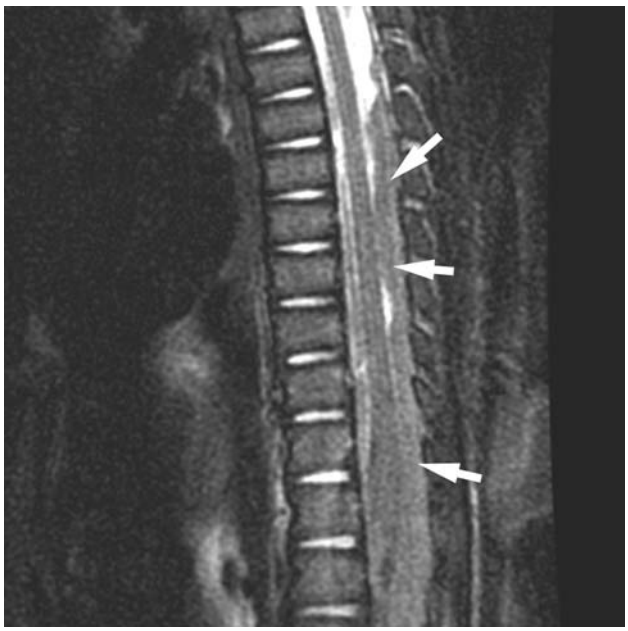

**Fig. 11** Intraspinal hemorrhage. Saggital STIR thoracic MR image demonstrates hypointense epidural and intradural hemorrhage in a shaken infant (*arrows*)

demonstrated subdural hemorrhages, and hemorrhagic contusions [21]. MRI has become standard in the imaging of AHT. In general, MRI is reserved for the stable patient, and delayed 3–5 days following the acute injury. Table 1 summarizes CT and MRI nomenclature, outlining specific features of the various modalities.

## Cranial ultrasound

Cranial sonography has utility in evaluation of the newborn and infant where it has been particularly useful in the evaluation of intracranial hemorrhage in the preterm newborn. The anterior fontanelle is a useful acoustic window for cranial ultrasonography to 6 months of age. Some clinicians will refer the infant with macrocrania for sonography evaluation as an initial imaging test. The presence of asymmetric convexity extra-axial fluid collections or extra-axial collections with complex fluid characteristics should be further investigated with MRI [22]. The use of color power Doppler may aid in the assigning of intracranial extra-axial fluid collections [23].

## Intracranial hemorrhage in asymptomatic newborns

The prevalence of intracranial hemorrhage among asymptomatic newborns ranges between 26 and 43% [24, 25]. Newborns delivered after vaginal birth or assisted vaginal birth (forceps, vacuum extraction) constitute the largest group with asymptomatic intracranial hemorrhage [26]. Subdural hemorrhage has been documented in babies delivered by cesarean section; typical after a trial of labor involving oxytocin. Risk factors for birth related intracranial hemorrhage include assisted vaginal delivery (forceps or vacuum), maternal parity, fetal weight, and prolonged duration of labor [24].

Subdural hemorrhage is common, particularly posteriorly involving the parietal and occipital regions and the tentorium cerebelli [27]. The mechanical deformity of the skull, resultant shear strain on the dura, particularly at sites of suture overlap, may lead to disruption of the dural venous plexus located within the inner portion of the dura with resultant subdural hemorrhage. These hemorrhagic collections tend to be small and rapidly resolving. In the series reported by Rook et al. [27] most birth related SDHs were $\leq$ 3 mm in thickness and resolved by 4 weeks of age. Recognition of these characteristic features of birth-associated hemorrhage helps in distinguishing them from subdural hemorrhages seen in the setting of abusive head trauma.

## Benign enlargement of the intracranial subarachnoid spaces

Enlargement of the ventricular and extraventricular cerebrospinal fluid (CSF) spaces is fairly common in infants. A variety of terms are in use to describe this finding. Benign extracerebral fluid collections in infancy, benign extra-axial collections of infancy, benign subdural effusions of infants, subdural hygromas, benign communicating

**Table 1** Imaging nomenclature

| | | |
|---|---|---|
| *CT imaging* | | |
| NCCT | (Non-contrast CT) | Detects blood, calcium, identifies fractures |
| CECT | (Contrast enhanced CT) | Tissue enhancement, venous sinuses |
| CTV | (CT venography) | Detects venous sinus clot or injury |
| CTA | (CT angiography) | Identifies arterial injury, dissection, aneurysm, AVM |
| 3D CT | (Surface rendered algorithm) | Helpful in detection and characterization of fractures |
| *MR imaging* | | |
| T1WI | (T1 weighted imaging) | Characterizes anatomy, injuries, and hemorrhage |
| T2WI | (T2 weighted imaging) | Identifies edema, characterizes hemorrhage |
| GRE | (Gradient recall imaging) | Detects hemorrhage, oxidation products |
| SWI | (Susceptibility weighted imaging) | Identifies slow venous flow, blood, shear injury |
| FLAIR | (Fluid attenuated inversion recovery) | Characterizes contusion, shear injury, small SDH |
| DWI | (Diffusion weighted imaging) | Characterizes edema (cytotoxic vs. vasogenic), identifies shear injuries, diffuse axonal injury (DAI) |
| ADC | (Apparent diffusion coefficient) | Reflects true diffusion restriction or decreased anisotropy |
| STIR | (Short tau inversion recovery) | Detects injury of ligaments and edema in tissue (bone, soft tissue) |
| MRV | (Magnetic resonance venography) | Identifies venous sinus clot and sinus injury |
| MRA | (Magnetic resonance angiography) | Detects dissection, occlusion, stenoses, aneurysms |

hydrocephalus, extraventricular obstructive hydrocephalus, and ventricular obstructive hydrocephalus are some of the more commonly used terms [28]. A preferable term is benign enlargement of the subarachnoid spaces. This condition typically comes to clinical awareness due to the association with macrocephaly; increase in occipitofrontal circumference (OFC).

The precise cause of benign enlargement of the subarachnoid spaces of infancy is yet to be completely determined. Some authors propose an anatomical or functional obstruction at the level of the arachnoid villi. Fortunately, this benign process is transient, typically normalizing by the age of 2 years with minimal developmental issues [29].

Cross-sectional brain imaging (CT and/or MRI) will demonstrate fluid prominence of the anterior interhemispheric fissure region, chiasmatic cistern, frontal subarachnoid spaces, lateral and third ventricles. The CT attenuation and/or MRI signal features of the fluid should appear simple and uncomplicated. In addition, the CT attenuation and MR signal intensity of the underlying cortex and deep brain parenchyma should appear normal [29]. MRI, in particular, is helpful in distinguishing subarachnoid space enlargement from subdural space fluid collection within the regions of subarachnoid space enlargement; veins are identified as linear or curvilinear structures traversing the fluid spaces running between the surface of the brain and the inner table of the calvarium (Fig. 12). With subdural space fluid collections, the veins are displaced inwardly against the brain surface [30].

Patients with benign enlargement of the subarachnoid spaces occasionally are found to have associated subdural hemorrhage (Fig. 13). This situation presents a clinical

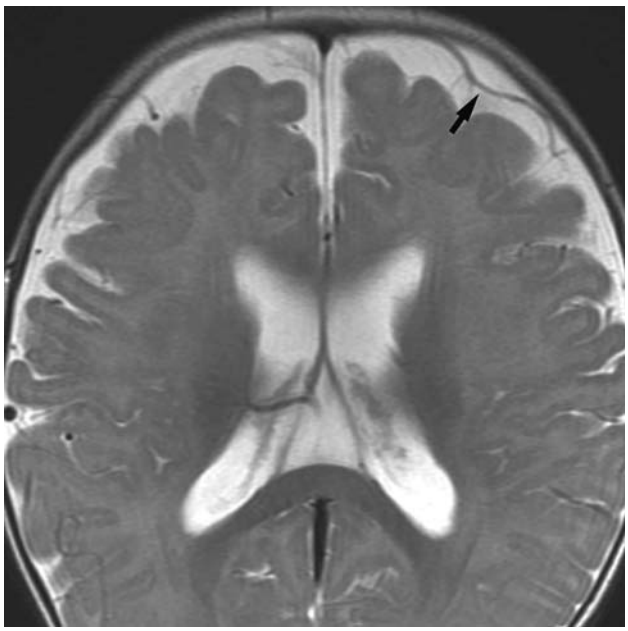

**Fig. 12** Benign enlargement of the Subarachnoid spaces. Axial T2WI shows hyperintense frontal subarachnoid spaces. Note the tubular hypointense veins traversing the subarachnoid fluid (*arrow*)

conundrum as such hemorrhages are proposed to occur in the setting of slight trauma or no trauma at all. Some clinicians have postulated that benign enlargement of the subarachnoid spaces may predispose to spontaneous subdural hemorrhage. Stretching of bridging veins or shear strain upon the dural membrane are possible explanations although this remains controversial [31]. Often extensive multidisciplinary investigation reveals no evidence of trauma [32]. Detection of subdural hemorrhage in the

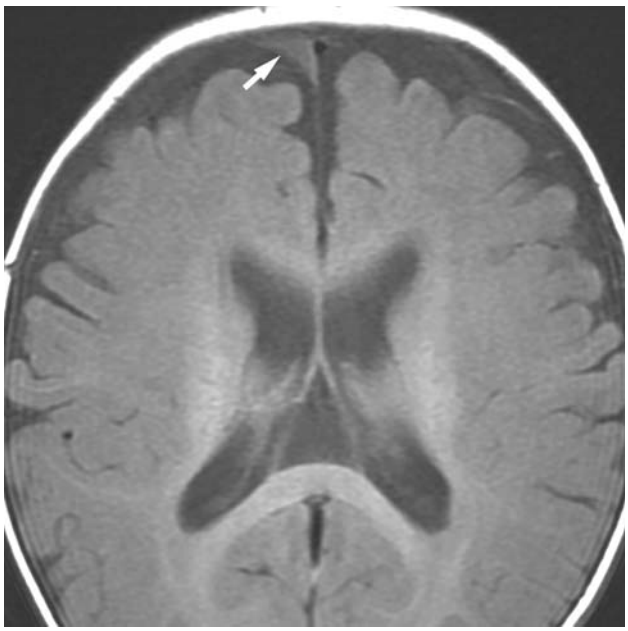

**Fig. 13** Benign extraaxial subarachnoid fluid and small SDH. Axial T1WI shows a small right frontal medium signal intensity SDH adjacent to the anterior inferior falx cerebri (*arrow*). Also note the expanded subarachnoid spaces

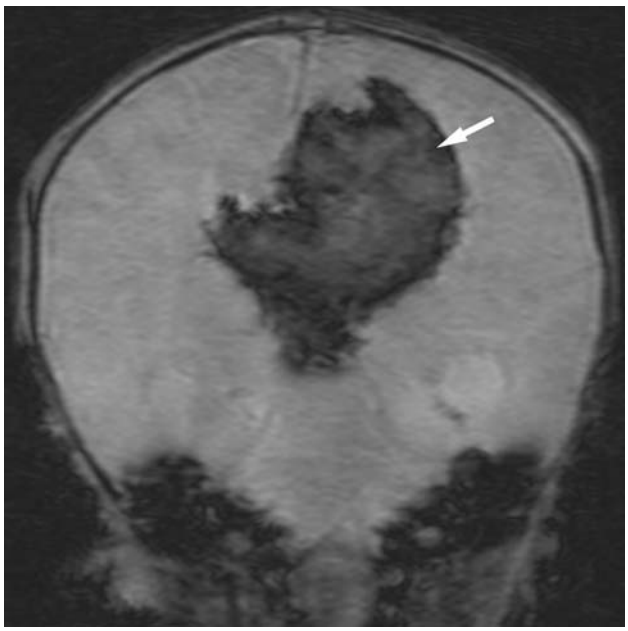

**Fig. 14** Hemorrhagic disease of the newborn. Coronal GRE MR sequence shows a large medial left hemispheric hypointese hematoma (*arrow*). Moderate intraventricular hemorrhage is present

context of benign enlargement of the subarachnoid spaces in the infant warrants a prudent evaluation of the child for any other signs of physical abuse or neglect. This should include a skeletal survey, dilated fundoscopic examination, and careful history, and plotting of growth parameters, including occipital frontal circumference. A sudden post natal acceleration of OFC may provide a rough window of possible traumatic injury.

## Cerebral hemorrhage due to hemorrhagic disease of the newborn

The deficiency of vitamin K predisposes to hemorrhagic disease of the newborn (HDN). This occurs particularly in exclusively breastfed infants, but may also occur as a result of mothers who have been taking anticonvulsants or antituberculous therapy. The classic or early form of vitamin K deficiency leading to bleeding in the newborn typically occurs between the second day of life and the end of the first week of life. A late onset form of hemorrhagic disease of the newborn typically occurs between the second and 12th week of life; or at times may be delayed as long as 6 months [33]. It is this late or delayed form of HDN that may be associated with serious and life threatening intracranial hemorrhage. These hemorrhages may be intraparenchymal or extra-axial (subdural, subarachnoid, epidural) (Fig. 14) [34]. Differentiation of hemorrhage due to HDN from that due to AHT requires an analysis of birth history and maternal factors, as well as laboratory investigation.

## Metabolic diseases

Other conditions that present with intracranial findings that lead to a consideration of abusive head trauma can be explained by alternative diagnoses. Although trauma, specifically abusive head trauma, is the leading cause of subdural hemorrhage in infants, it is important to consider other potential, although rare diagnoses [35, 36]. Cross sectional cranial imaging can provide initial clues to the appropriate diagnosis of metabolic disease. As in any case, the history, physical examination and specialized testing for specific inborn errors of metabolism will confirm the diagnosis suspected on image. Glutaric aciduria type 1, a defect in the metabolism of lysine and tryptophan caused by deficiency of glutaryl-CoA dehydrogenase has specific imaging features that will assist the clinician in making an appropropriate diagnosis. Children often present with developmental delays and macrocephaly. CT may demonstrate wide sylvian fissues and mesencephalic cisterns, fronto-temporal atrophy, and edema within the deep nuclei (caudate, globus palladus, and putamina). Atrophy can predispose the patient to developing subdural hemorrhages. The MRI in GA1 may demonstrate increased T2 signal and acutely, reduced diffusion within the caudate, putamina and to a lesser degree the globus pallidus (Fig. 15). Fronto-temporal atrophy is common, often mimicking sylvian fissure arachnoid loculations or cysts. Myelin maturation delay is also common [37].

Other conditions that have been reported to present with neuroimaging findings that may initially be diagnosed with

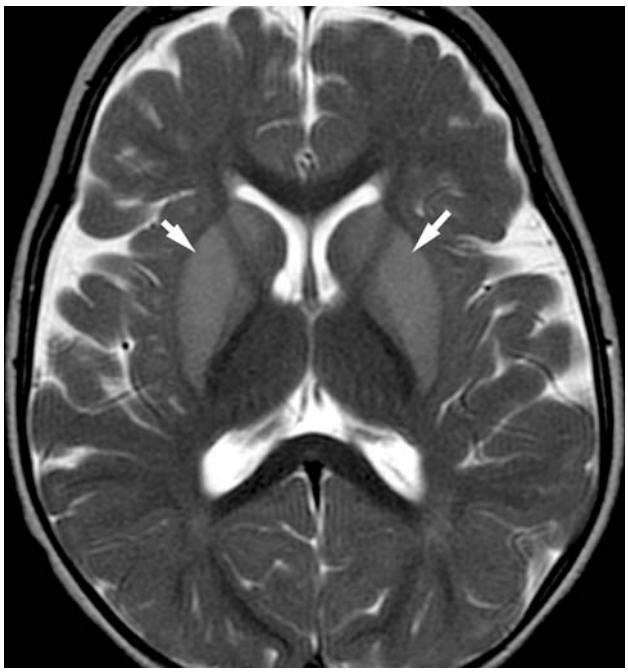

**Fig. 15** Glutaric aciduria type 1. Axial T2WI shows prominent sylvian fissures reflecting temporal lobe atrophy. Also note the hyperintense and swollen caudate nuclei and putamina reflecting early striatal necrosis (*arrows*)

AHT include galactosemia, homocystinuria, osteogenesis imperfect, Menkes kinky hair syndrome, and Alagille syndrome [38]. Typically, neuroimaging does not assist in differentiating many of these conditions from AHT. However, excellent history and physical examination, as well as family history may lead the clinician to the appropriate laboratory and genetic testing.

Collaborations

The assessment of a child who may be abused requires the skills of many medical specialists. The pediatrician can assist the neuroradiologist in providing clinical context in the interpretation of neuroimaging studies. The neuroradiologist can make recommendations to the child abuse team regarding additional studies that may clarify the case. Correlations of neuroradiologic findings with those seen at post mortem and then on histopathology, may be helpful, in the unfortunate event that the child dies from the injuries. In the medical/legal arena, the expertise of the neuroradiologist in court may be invaluable. In some jurisdictions where non radiologists are allowed to testify as to radiologic findings, assisting pediatricians and other clinicians in preparing their testimony is extremely important.

Neuroimaging in suspected child abuse has become essential in making an appropriate diagnosis. This has become standard practice as imaging modalities and techniques have advanced and become more available. Presence of blood products, location, aging of blood, parenchymal injuries and alternative diagnoses to abuse are only part of what neuroimaging offers to clinicians. Radiologic studies often do not stand alone in the evaluation of child abuse. Clinical correlation that includes detailed medical, family, and social history, onset of symptom development and progression is critical in determining an accurate diagnosis. Collaboration and collegial communication between all members of a clinical team from pediatrician, to neuroradiogologist, pathologist and other specialists that may be involved in the care of children is necessary in both the clinical and legal settings.

## Key points

1. Pediatricians, neuroradiologists, pathologists, and other pediatric specialists must collaborate in cases of child maltreatment to ensure accurate diagnoses.
2. Cranial CT is the first choice for imaging the child suspected to have abusive head trauma.
3. 3D CT fully characterizes complexity and extent of skull fractures.
4. Acute heterogeneous SDH is often accompanied by underlying cerebral edema.
5. By day 7 following cerebral insult, diffusion weighted MR imaging abnormalities may normalize.
6. Intravenous contrast enhancement (CT or MRI) may clarify the location of extraaxial fluid.
7. In the setting of suspected AHT, MRI of the craniocervical junction, cervical and thoracic spine may reveal additional unsuspected findings of injury.
8. Birth related SDH typically resolves by four weeks of age.
9. SDH found in the setting of benign enlargement of the subarachnoid spaces warrants a thoughtful clinical evaluation.

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