Subdural Hemorrhage in Abusive Head Trauma: Imaging Challenges and Controversies

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Background of abusive head trauma

In the neonate, infant, or young child who has suffered from non-accidental injury, abusive head trauma (AHT) is acknowledged as the most common cause of fatality and long term morbidity with approximately 1,500 fatalities and 18,000 seriously disabled infants and children annually in the USA.[1-4] Ninety-five percent of serious CNS injuries among infants less than 1 year of age are attributed to AHT.[2] Up to 80% of fatal child abuse injuries are attributed to head injury.[2] Unfortunately, most authors agree that these statistics represent an underestimation of this national health problem. Beyond the tragedy of an injured or murdered child is the broader social and community impact of this national and international health blight. In addition to the emotional, family, and social costs caused by inflicted trauma, the societal financial burden is astounding. In 2008, in the United States, costs ascribed to child abuse were estimated at 103 billion dollars; $33 billion for immediate intervention services and $70 billion for long-term costs.[5]

Subdural hemorrhage: a marker of pediatric head trauma

Subdural hemorrhage (SDH) is the most common pathology associated with abusive head trauma.[6-8] The historical teaching describing the origin and location of subdural hemorrhage has been that the tearing of bridging veins leads to bleeding at the interface between the inner (meningeal layer) dural margin and the arachnoid membrane.[9] This explanation does not completely reflect the potential sites of subdural compartment hemorrhage. More recently, Julie Mack and colleagues have advanced our understanding of a more dynamic vascularized dura.[10] They describe the inner dural border zone region (inner meningeal dura) as a location where loose intercellular junctions exist, possesses a vascularized layer, and represents the location of subdural compartment (intradural) hemorrhage. Hemorrhage in this location conforms to the classic morphology of subdural bleeding (concavoconvex). The authors also point out that in the first two years of life, the inner dural border zone plays an important role in the resorption of CSF as the arachnoid granulations are maturing.[10] This expanded discussion of the inner dura, hemorrhage origin, and hemorrhage location gives guidance to the medical imaging physician to describe bleeding in this location as subdural compartment hemorrhage. Of course, from the brain CT or MRI examinations which depict intracranial hemorrhage the intent behind trauma cannot be inferred. It is only after a comprehensive child protection team evaluation that the determination of abusive versus accidental or non-traumatic causes of hemorrhage is determined.

Imaging goals in the evaluation of abusive head trauma

The goals for the medical imaging physician who is responsible for interpreting brain CT and MRI examinations for the pediatric patient with suspected abusive head trauma are clearly defined. These include: the determination of findings that require urgent and emergent treatment, fully assessing the extent of injury, estimating the timing of injury, detecting intracranial injuries in abused children who present with clinical manifestations of extracranial injury, and detecting mimics of SDH and underlying conditions which predispose to non-traumatic SDH.[1,2,4,7]

CT is the examination of choice in the initial evaluation of pediatric head trauma. Its availability, rapid examination times, and sensitivity for detecting intracranial hemorrhage, early herniation patterns, and fractures make it an indispensible tool.[1,2,7] Additionally, if vascular injury is suspected, intravenous contrast enhanced CT angiography and venography can be accomplished with ease. CT lacks sensitivity in the detection of cortical contusion, early edema, infarction, shear-strain

injury (diffuse axonal injury), and subtle petechial hemorrhage.[1,2]

Brain MRI yields full appraisal of intracranial hemorrhage, parenchymal injury, signs of early herniation, and vascular complications including stroke and vessel dissection. In addition to spin magnitude imaging (including gradient recall imaging [GRE] or susceptibility weighted imaging [SWI] and diffusion weighted imaging [DWI]) which represents the minimum standard examination for trauma, MR adjuncts such as magnetic resonance spectroscopy (MRS), perfusion MR imaging (pMRI), and vascular adjuncts including MRA and MRV may contribute useful diagnostic information.[1,2,8] At our pediatric medical center, brain MRI is performed for all pediatric patients suspected of having inflicted head trauma with abnormal CT examinations, the pediatric patient suspected of being abused with encephalopathy and focal neurological signs regardless of the CT findings, and for the infant with extracranial manifestations of abuse. From a timing standpoint, we strive to accomplish the MR examination 3 to 5 days following presentation. This allows for optimal patient stabilization and expression of intracranial injuries.[1,2,7,8]

Dating intracranial hemorrhage using CT and MRI

Estimating the age of intracranial hemorrhage provides critical forensic information for the investigation of suspected abusive head trauma. I have found that CT and MRI findings are complementary when it comes to tackling the dating of an injury and characterization of intracranial hemorrhage. However, pinpointing the precise age of extraaxial hemorrhage is fraught with pitfalls and frankly, is unrealistic.[2,7,11,12] There are many factors that influence the CT and MRI appearance of subdural blood including the hemoglobin state, clot-serum separation, presence of an arachnoid tear with admixture of CSF and blood, RBC hydration, and MR technical considerations including magnetic field strength and the selection of scanning sequences.[13] The CT appearance of aging subdural hemorrhage is outlined in Table 1; this data represents a practical working tool for assessing the age of extraaxial hemorrhage. Here, a word of caution is in order. Note from Table 1, that the isodense appearance of hemorrhage could either represent hyperacute blood or early subacute hemorrhage.[14] Also, the patient with an acute SDH and a hemoglobin value of < 8 g/dl will exhibit an isodense hemorrhage.[13,14] Therefore, when the interpreting radiologist is assessing the initial CT examination the impression of the CT findings should be descriptive; emphasizing the appearance or density features of the hemorrhage rather than emphasizing the stage of hemorrhage (Table 1). Here is where an argument can be made for a short interval repeat CT examination (within 24 to 48 hours of the initial study) to clarify hypodense or isodense subdural components.

CT Evolution of Subdural Hemorrhage (SDH)	
≤ 3 Hours	Iso-to-Hypoattenuating
Few Hours → 7-10 Days	Hyperattenuating
2-3 Weeks	Isoattenuating
> 3 Weeks	Hypoattenuating

Table 1. (Modified from Reference 7)

Using MR as a means of dating subdural hemorrhage is even more complex than CT dating for reasons mentioned above. Although the work by Bradley has laid a foundation for our understanding of the MR evolution of intracranial hemorrhage, it must be kept in mind that the MRI evolutionary findings of intracranial hemorrhage are observations drawn from intraparenchymal hematoma aging (Table 2).[13] The relatively elevated parenchymal levels of tissue thromboplastin and higher tissue oxygen tension lead to more rapid degradation of blood than found within extraaxial hemorrhage.[13,15] Given this information, as medical imaging physicians, we must use the MRI guidelines for hemorrhage evolution as a dating estimate and always interpret MRI in conjunction with CT observations.[8]

Mixed density subdural hemorrhage

Interpretation of the mixed density subdural hemorrhage can be a source of confusion and inaccuracy when interpreting brain imaging.[2,7,8] Historically, dogma has stated that mixed density SDH represents a combination of new and old

Evolution of Intraparenchymal Hematoma – MRI				
Stage	Age	T1WI	T2WI	Hb State
Hyperacute	< 12-24 hours	Iso– to – Hypo	Hyper	Oxy-Hb
Acute	1-3 Days	Hypo	Very Hypo	Deoxy – Hb (Ic)
Early Subacute	2-3 Days → 1-2 wks	Very Hyper	Very Hypo	Met – Hb
Late Subacute	1-2 wks → 1-2 mo	Very hyper	Very Hyper	Met – Hb(Ec)
Chronic	Few wks → mos/yrs	Iso	Very Hypo	Hemosiderin (SD membrane)
Chronic	Few wks → mos/yrs	Hypo	Hyper	Nonparamagnetic hemochromes (SD content)

Key: Ic = Intracellular, Ec = extracellular, SD = subdural

Table 2. (Modified from reference 7)

blood. Four diagnostic considerations should come to mind for the radiologist in the setting of mixed density SDH. These include: hyperacute + acute blood, acute hemorrhage alone, hematohygroma (acute hemorrhage + CSF secondary to arachnoid tear), and the combination of new and old hemorrhage.[2,7,8] The first three examples of mixed density SDH can derive from a single traumatic event (Fig 1). In my experience, the mixed density SDH associated with ipsilateral cerebral edema is usually associated with one of the first three causes. Tung and colleagues reported that SDH in the context of abusive head trauma was more likely to be mixed density, bilateral in location, contrecoup, and affiliated with poor neurological outcome. SDH of accidental cause was more homogeneous, unilateral and coup to the site of impact (Fig 2).[16] Hymel and colleagues have also reported their CT observations in pediatric accidental and abusive head trauma.[17] A sediment or hematocrit layer may be seen shortly after trauma and may result from one traumatic event. For purposes of dating, the radiologist should focus upon the CT and MR features of the sediment for most accurately estimating hemorrhage age (Fig 3).[12]

The presence of membranes within the subdural hemorrhage is very helpful to strengthen the radiologist's diagnostic confidence of new and old subdural blood. Delicate incomplete membranes begin to form within the subdural hemorrhage within 2 to 3 weeks and mature by 4 to 5 weeks.[18,19] CT can suggest the presence of membranes but MR provides the most information regarding membrane structure and signal intensity (Fig 4). Membrane detection requires careful inspection of all pulse sequences. With older membranes, GRE and/or SWI will be helpful in detection. Membrane conspicuity may be heightened by the use of intravenous MR

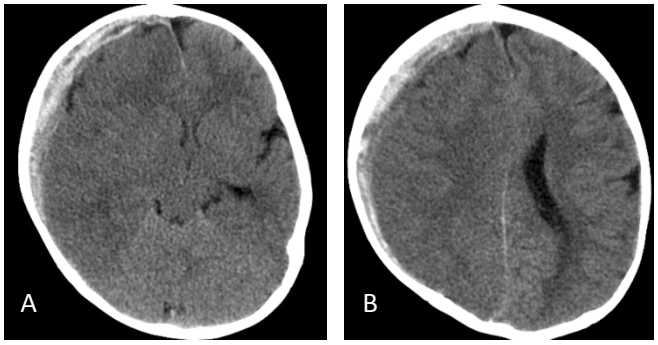

Figure 1: Mixed density subdural hemorrhage. (A). Non-contrast CT through the level of the frontal horns shows a heterogeneous right frontotemporal SDH. Note the associated right hemispheric cerebral edema and subfalcine herniation. (B). Non-contrast CT through the cerebral convexities demonstrates the cephalad extent of the subdural bleed and early obstruction of the left lateral ventricle due to compression at the left foramen of Monro as a result of the subfalcine herniation. At surgery the hemorrhage was all found to be acute. The perpetrator confessed to grasping the infant's neck and shaking.

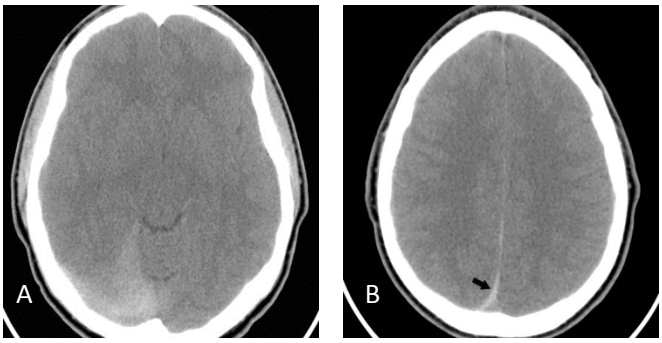

Figure 2: Accidental subdural hemorrhage. (A). Non-contrast CT shows a homogeneous increased attenuation SDH involving the right tentorium. (B). A small posterior parafalcine component of the SDH is also noted (arrow). This fifteen-year-old female had accidental closed head trauma ipsilateral to the SDH.

contrast and post-contrast T1 weighting and subtraction MR imaging techniques.[1,2,7]

Re-bleeding into subdural hemorrhage

Re-bleeding into a subdural hemorrhage remains a controversial topic and when observed brings to mind concern over whether the new blood represents: spontaneous hemorrhage, bleeding due to minimal trauma, or hemorrhage secondary to major trauma.[2,20] The corresponding clinical picture at the time of presentation is very important to consider as the encephalopathic child with new subdural hemorrhage is much more likely to have experienced significant trauma.[20] A careful child

protection team evaluation is warranted in this setting to determine if physical abuse is the likely cause of the new imaging findings. Additionally, the radiologist should always keep in the back of his or her mind the possibility of non-traumatic causes of SDH and re-bleeding (as one might see with a progressive neurodegenerative disorder) (Table 3).

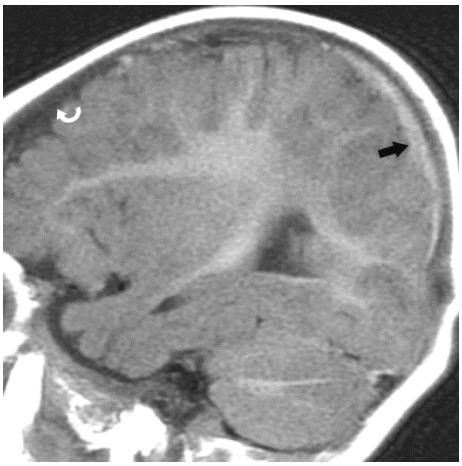

Figure 3: Early subacute subdural hemorrhage with sediment. Parasagittal T1 weighted MR image shows a thin parietooccipital hyperintense subdural hemorrhage (arrow). Note the thin hypointense frontal subdural fluid (curved arrow). The perpetrator confessed to three strong shaking episodes four days prior to the MRI. The redistribution of hemorrhage can occur within hours of the insult. For dating, the radiologist should focus attention upon the sediment.

Mimics and Non-Traumatic Causes of Subdural Hemorrhage

- Coagulopathies: vitamin K deficiency
- Hematologic disorders: leukemia, hemophilia
- Arachnoid cyst
- Vascular: aneurysm, arteriovenous malformation, dural AVF
- Meningitis
- Metabolic: glutaric aciduria type 1, galactosemia, pyruvate carboxolase deficiency
- Neoplasms: leukemia, leptomeningeal PNET, leptomeningeal melanoma
- Neoplastic like : hemophagocytic lymhohistiocytosis
- Other: Menkes disease

Table 3. (Modified from Reference 2)

Birth related subdural hemorrhage

Birth related SDH can lead to confusion and controversy particularly when SDH is detected in a young infant.[21] In a recent article by Rooks and colleagues, 101 asymptomatic newborns were studied with cranial sonography and MRI. The prevalence of SDH in their population was 46%. Take home points from their paper were that SDH was most common in the parietooccipital and tentorial locations, thin SDH (most < 3 mm in thickness), and nearly all SDHs had resolved by one month of life (Fig 5). Additionally, in the first three days of life, hemorrhage was most accurately detected with gradient recall imaging (GRE) at a time when acute hemorrhage was isointense on T1 weighted images.[22]

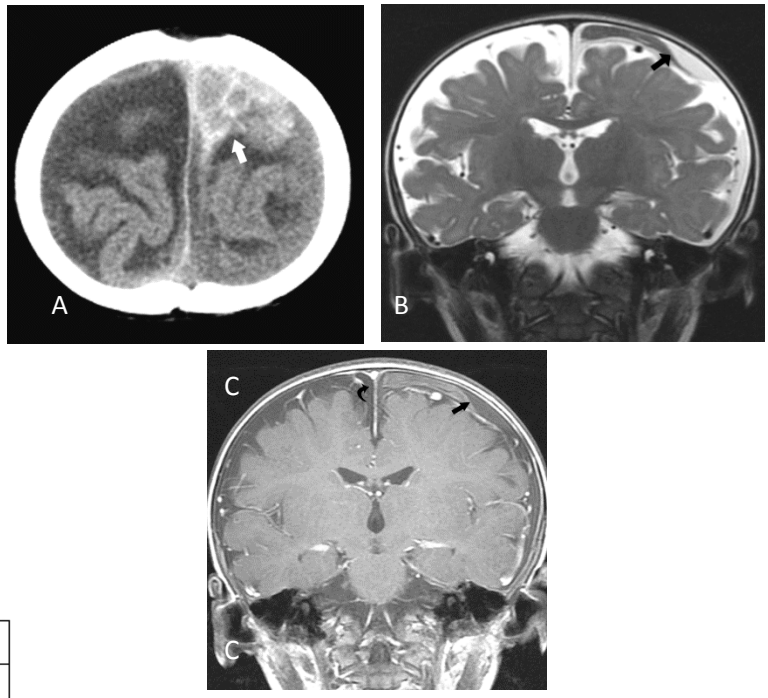

Figure 4: Membrane formation within subdural hemorrhage. (A). NCCT showing a heterogeneous left parafalcine subdural hemorrhage (arrow). This hemorrhage had been interpreted as an acute bleed at the referring hospital. (B). Coronal T2 weighted MR image shows a sharp transition (arrow) between the medial hypointense hemorrhage (early subacute) and lateral hyperintense to cortex (early chronic) subdural hemorrhage. (C). Coronal T1 weighted MR image with IV contrast shows T1 shortening (enhancement) within the subdural membrane (arrow). Also note the small T1 hypointense (chronic) right parafalcine subdural hemorrhage (curved arrow). Well defined membranes within subdural hemorrhages take 4 to 6 weeks to form.

Subdural hemorrhage with benign expanded subarachnoid spaces

Benign expanded subarachnoid spaces represent a common finding among infants with

macrocephaly who are otherwise normal. The etiology of these collections likely represents a transient mismatch between CSF production and resorption.[23,24] In the first two years of life, the arachnoid granulations are undergoing maturation. Additionally, during infancy, the inner dural border zone may play an important role in CSF resorption at a time of evolving arachnoid granulation maturation.[10]

When evaluating prominent extracerebral collections and considering the diagnosis of benign subarachnoid fluid, the radiologist should look for clues that allow assignment of the of the fluid to the subarachnoid space and thus exclude subdural compartment collections.[24] These findings include: visualization of corticodural veins traversing the fluid (positive cortical vein sign), interdigitation of the fluid into the cortical sulci, symmetry of the fluid interface with the dura, and iso-attenuation (CT) or isointensity (MRI) features of the fluid on imaging studies.[25]

Controversy arises when SDH is detected in association with these expanded subarachnoid spaces (Fig 6). There are authors who posit that in the context of benign expanded subarachnoid spaces that SDH can occur spontaneously or with minimal trauma.[26-30] My experience over twenty years of interpreting pediatric neuroimaging studies is that the occurrence of SDH with benign expansion of the subarachnoid spaces without a history of trauma is a rare event. Therefore, in my clinical practice, the detection of SDH in association with benign expanded subarachnoid CSF collections warrants a comprehensive child protection team evaluation.

Subdural hemorrhage and intracranial venous thrombosis

In the differential diagnostic consideration of non-traumatic causes of SDH, some authors opine and testify to the fact that intracranial venous thrombosis (ICVT) may lead to the development of SDH that mimics the SDH of abusive head trauma.[4,31,32] At the 2011 American Society of Neuroradiology (ASNR) meeting in Seattle Washington, Dr Logan McClain and colleagues reported their observational retrospective CT and MRI study of 36 pediatric patients with non-traumatically acquired intracranial venous thrombosis, looking for the presence of SDH. None of the 36 were found to have SDH [AJNR In Press]. Of course, trauma can be a cause for ICVT and subdural hemorrhage alike.

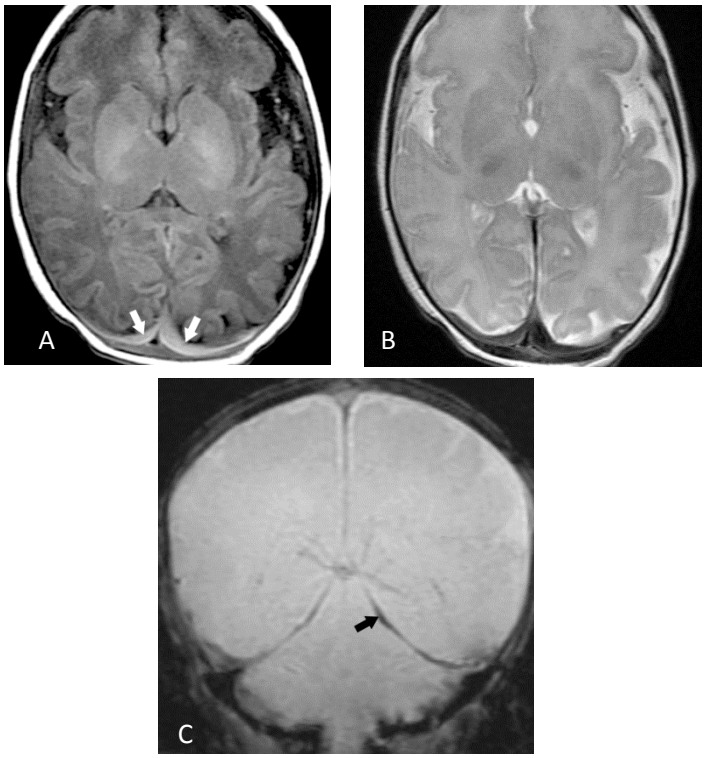

Figure 5: Birth related subdural hemorrhage in a four-day-old newborn. (A). Axial T1 weighted image shows bilateral thin occipital hyperintense subdural hemorrhages (arrows); typical for size and location of birth related subdural hemorrhages. (B). Axial T2 weighted MR image shows these subdural bleeds to be hypointense (early subacute). (C). Coronal gradient recall (GRE) (T2*) MR image shows the presence of a thin left tentorial subdural hemorrhage (arrow). This is also a common location for birth related SDH. GRE images are particularly helpful in the first few days following birth when the T1 signal intensity of birth related hemorrhage will be isointense.

Hypoxic ischemic encephalopathy and subdural hemorrhage

Finally, there has been recent controversy raised over whether hypoxic ischemic encephalopathy (HIE) is a potent cause of SDH which may mimic the features of abusive head trauma.[33,34] In my experience and in that of other authors, HIE may certainly accompany other findings consistent with abusive head trauma. Of course, child birth related subdural hemorrhage may occur in conjunction with HIE without a causal relationship. Several large

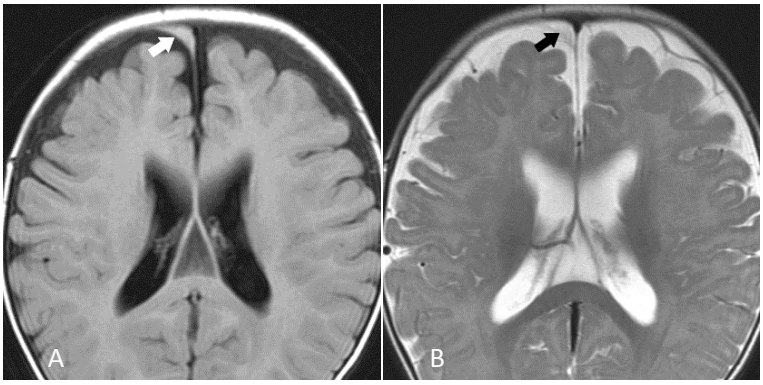

Figure 6: Subdural hemorrhage in the setting of macrocrania and benign expansion of the subarachnoid spaces. (A). Axial T2 fluid attenuated inversion recovery image (FLAIR) shows a small slightly hyperintense right parafalcine frontal subdural hemorrhage (arrow). (B). Axial T2 MR image shows heterogeneity of the SDH (arrow) and hypointense cortical veins coursing through the expanded subarachnoid spaces (positive cortical vein sign).

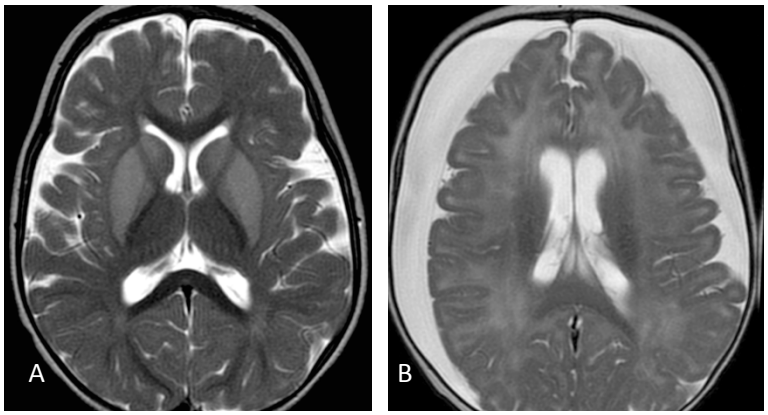

Figure 7: Glutaric aciduria type I. A non-traumatic cause of SDH (A). Axial T2 weighted MR image in a child with the metabolic disorder of glutaric aciduria type I shows bilateral basal ganglia swelling and hyperintensity. These regions also showed evidence for cytotoxic edema on diffusion weighted MR imaging. (B). Six months later, new onset seizures prompted a repeat MRI. The T2 MR image shows the interval development of large chronic subdural hemorrhages. Brain parenchymal volume loss (due to nueodegeneration) was confirmed on other sequences. When subdural collections are detected, the radiologist must closely inspect the brain parenchyma for signs of atrophy.

non-traumatic observational patient cohort studies have failed to substantiate HIE as a cause of SDH.[35]

Non-traumatic causes of subdural hemorrhage

Finally In addition to the key observations that the radiologist must make in the setting of suspected abusive head trauma, there must be an awareness that some disorders may either as a result of mechanical distortion or neurodegeneration predispose to the development of non-traumatic SDH (Table 3).[36-42] To avoid this pitfall, the radiologist must be alert to key clinical features, laboratory abnormalities, and imaging clues that suggest an underlying cerebral parenchymal disorder (Fig 7).[36-42] Of course, a comprehensive clinical, and laboratory evaluation of the patient with a chronic neurologic disorder and SDH is mandatory. It is worth remembering that physical abuse is more common among children with chronic illness.[43]

Reporting responsibilities for the radiologist when AHT is suspected

The radiologist shoulders an important responsibility when it comes to reporting imaging findings suggesting abusive head trauma. The law is clear in this regard. For the radiologist, there is a legal responsibility to report findings suspicious for AHT. These guidelines are outlined by the American College of Radiology, and can be reviewed at (www.acr.org/guidelines). Documentation of the individual contacted, the method of communication, the date and time are minimal requirements. As a mandatory reporter, the radiologist is protected from civil and criminal prosecution by Shield Laws that exist within the United States. The radiologist should inquire with their local child protection team and/or county medical association to review specific state statutes.

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