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COMMENTARY

Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI

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In a review by Fernando et al. [1] published in this journal on the pitfalls and controversies in the neuroimaging of nonaccidental trauma, the section addressing the age of subdural collections ("hyperacute versus acute on chronic subdural hematomas") did not fully explore the careful approach necessary for the proper assessment of the nature and age of subdural hematomas (SDH) evident in some infants suspected of having nonaccidental head injury (NAHI). The purpose of this commentary is to expand on this topic and bring further clarification.

Age determination with CT

The most common finding in abusive head injury in infants is subdural hemorrhage [2]. The age of a hematoma can be estimated with CT based on its density, and with greater accuracy with MRI. With CT, the density of hemorrhage is related to the degree of clot retraction, the hematocrit, the hemoglobin fraction and protein content (globin) [3]. Four stages can be identified (Table 1). Hyperacute hemorrhage implies that the blood products have not yet formed a clot —this can normally take up to 3 h. Unclotted blood is isodense on CT (assuming the individual is not severely anemic); clotted blood is hyperdense. Hyperacute hemorrhage is commonly encountered in epidural hematomas of arterial origin—the result of rapid bleeding from meningeal arteries. Hyperacute hemorrhage is less commonly encountered in SDH. The bleeding in SDH tends to be of venous origin and is therefore slower than arterial bleeding; by the time a patient is brought to the CT scanner following trauma, there is often a long enough lapse for the SDH to have clotted and present as acute, not hyperacute, blood (exceptions to this are patients with a coagulopathy or with very brisk hemorrhage).

Acute SDH remain hyperdense on CT images for about a week [3–5]. The density then diminishes progressively to isodensity ("subacute"), then hypodensity ("chronic" SDH); Table 1 gives approximate time intervals for this evolution. The age estimate and evolution of SDH, based on CT density, can be influenced by many factors, including:

1. The patient's hemoglobin level; acute hemorrhage in an anemic patient (Hb <8–10 g/dl) is isodense, not hyperdense [3].
2. Presence of a coagulopathy (e.g., DIC), which can delay blood clotting or development of a hyperdense clot.
3. Admixture of cerebrospinal fluid (CSF) with blood products can lead to early development of mixed isodense or hypodense hematomas (hematohygroma, see below).

Mixed density SDH: determination of etiology

In NAHI, acute SDH can be of uniform high density, though commonly show a mixture of high and low density. This acute presentation of mixed-density SDH is also seen

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Table 1 Density evolution of hemorrhage on CT images.

Stage	Appearance	Estimate of age
Hyperacute	Isodense	<3 hours
Acute	Hyperdense	Few hours → 7–10 days
Subacute	Isodense	2–3 weeks
Chronic	Hypodense	>3 weeks

Adapted from references [4] and [5].

in accidental injury, but is more common in NAHI [6]. Four scenarios commonly lead to the presence of mixed-density subdural hemorrhages (Table 2): co-existence of hyperacute (unclotted) and acute (clotted) blood; early clot retraction; acute admixture of hemorrhage and CSF into the subdural space ("hematohygromas"); and acute or chronic SDH/subdural hygroma. Difficulty and confusion arise most often in the radiographic differentiation of hematohygromas from the coexistence of acute and chronic SDH.

Hematohygromas result from an acute admixture of blood products (high density) and CSF (low density) within the subdural space [7]. Entry of CSF into the subdural space results from a traumatically induced tear in the arachnoid overlying the cerebrum, which allows effusion of CSF into the subdural space. CSF can accumulate in the subdural space acutely (within hours of trauma) or in the following days, as an acute subdural hemorrhage is resolving (Fig. 1) [2].

In normal children, most acute subdural hemorrhages resolve rapidly [2, 6]. Posttraumatic subdural hygromas usually resolve within a few weeks [2, 8]. Chronic, enlarging SDH are unusual in children; they are most often encountered in conditions where intracranial pressure is lowered (for example, in the presence of a ventriculoperitoneal shunt).

Differentiation of an acute hematohygroma from coexistent acute and chronic SDH may not be possible on the very first imaging study, which is typically a CT scan obtained during a visit to the emergency room. Therefore, it is best to initially describe the subdural collections in terms of density (hyperdense, isodense, hypodense; uniform vs. mixed density) and avoid the use of "acute," "chronic," or "acute on chronic." Early, inaccurate labeling by these terms can further complicate the medical, social and legal aspects of an infant with suspected NAHI. If NAHI is suspected, MRI of the brain should be done as soon as feasible [5] because:

1. MRI is more sensitive than CT to the presence of SDH. While CT readily demonstrates even small amounts of subdural blood in the interhemispheric fissure, MRI is more sensitive to the presence of small SDH over the cerebral convexities, in the tentorium and in the posterior fossa.
2. MRI more accurately identifies the focal subarachnoid hemorrhages that commonly accompany SDH in NAHI [2]. These appear in patches over the cerebral convexities (and to some extent in the interhemispheric surfaces), and often extend into the subdural space, contiguous to or under areas of SDH.
3. The parenchymal lesions that often accompany NAHI are better characterized with MRI.
4. The craniocervical junction can be evaluated for signs of ligamentous injury with MRI; the brainstem and upper cervical cord can also be assessed for signs of traumatic injury. Injury of the cervicomedullary junction can cause central hypoventilation, which can lead to postanoxic brain injury [9].
5. MRI is more sensitive than CT in the identification of membranes within the subdural space. The presence of membranes is a very useful sign in determining the presence of an older subdural component. Contrast-enhanced MRI can demonstrate enhancing membranes surrounding SDH a week after hemorrhage, first along the dural border of the hematoma and later along the pial border [10].
6. The age of a hematoma can be determined more accurately with MRI than with CT. The age and

Table 2 Differential diagnosis and timing considerations of mixed hypo- and hyperdense subdural collections.

Differential diagnosis	Mechanism	Estimate of age	Number of hemorrhagic events
Coexistent acute + hyperacute hematoma	High-density acute hemorrhage and iso- to low-density unclotted blood or serum	Few hours	Single
Acute hematoma	High density acute hemorrhage or clot retraction, and low-density serum	Hours → 1 week	Single
Coexistent acute hematoma + subdural hygroma	Hematohygroma (acute admixture of blood and CSF)	Hours → 1 week	Single
Acute and chronic SDH	Acute hematoma and preexisting older hematoma/hygroma. Two separate hemorrhagic events	<1 week + >2–3 weeks	Two (or more)

Adapted from references [6] and [8].

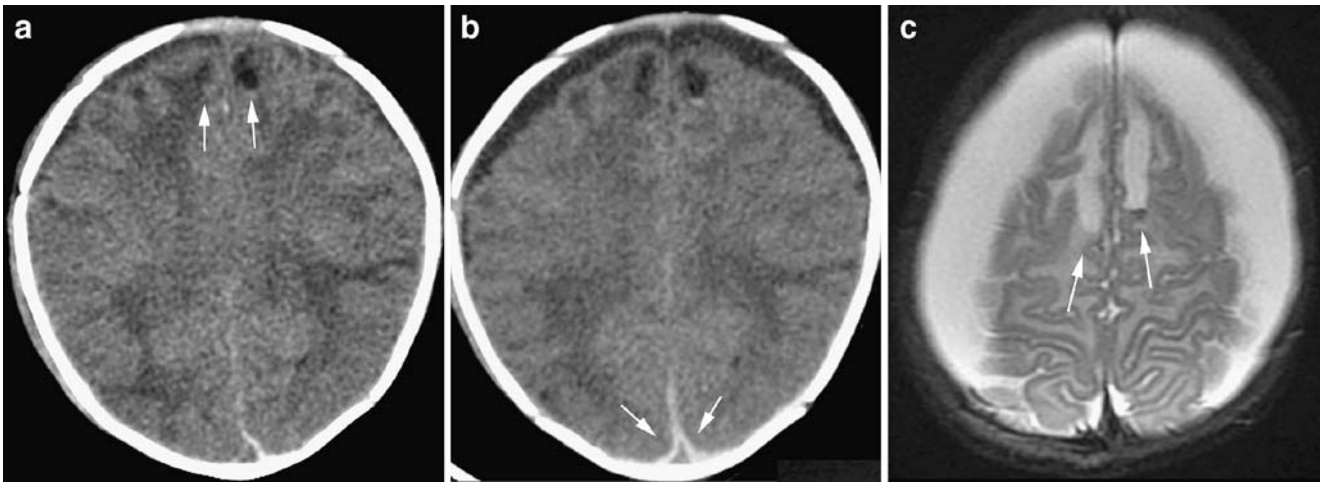

Fig. 1 Rapid accumulation of hematohygroma. CT images obtained on day 1 (**a**) and day 4 (**b**), and axial T2-W MR image obtained on day 12 (**c**) after NAHI. Accumulating, acute, low-attenuation subdural collections are evident around the frontal convexities. An increasing amount of SDH is also observed along the posterior sagittal falx between **a** and **b** (**b** *arrows*). Bifrontal cortical/subcortical shearing lacerations are evident (**a**, **c** *arrows*)

temporal evolution of cerebral parenchymal hematomas using MRI is well documented [11]. In general, parenchymal hematomas resolve faster than epidural hematomas as brain tissue contains high concentrations of tissue thromboplastin, which accelerates hematoma resolution; fibrinolytic enzymes are also present in CSF after trauma [4]. The temporal signal modification of SDH is slowed (compared to parenchymal hematomas) due to the oxygen tension within the subdural space (the oxygen originates from the dura, which is well vascularized, and also from admixture of oxygen within the CSF, if present). In addition, dilution of hemorrhagic products with CSF further modifies the MRI (and CT) appearance of SDH. The T1 and T2 features of evolving SDH are summarized in Table 3; the suggested time periods of the different phases are modified (lengthened, with overlap) from published evolution of intraparenchymal hematomas to account for the effects of oxygen within the subdural space [11].

Evolution of subdural collections

Insight on the age and nature of the subdural collections is also augmented by their dynamic evolution, assessed on follow-up CT or MR scans obtained in the days after admission. Repeat CT scans are easily performed as sedation is rarely needed; however, CT exposes the child to ionizing radiation. Repeat MRI scans avoid exposure to radiation, but sedation is usually needed in infants and young children. Many useful evolving features of a subdural collection can be observed:

1. Further accumulation of high-density hemorrhagic components within hours to 1 day of admission suggests interval bleeding and/or clotting of blood products; such rapid change points to a very recent origin to the hematoma seen on the admission CT.
2. Redistribution of blood products within the subdural space as a gravity-dependent process to the dependent

Table 3 Signal evolution of SDH on MRI.

Stage	Hemoglobin breakdown product/distribution	Time period	T1	T2
Hyperacute	Oxyhemoglobin/intracellular	<12–24 h	↓ or ↔	↑
Acute	Deoxyhemoglobin/intracellular	1–3 days	↓ or ↔	↓↓
Early subacute	Methemoglobin/intracellular	2–3 days → 1–2 weeks	↑	↓↓
Late subacute	Methemoglobin/extracellular	1–2 weeks → 1–2 months	↑↑	↑
Chronic	Hemosiderin (subdural membrane)	Few weeks → months/years	↔	↓↓
Chronic	Nonparamagnetic hemichromes (subdural content)	Few weeks → months/years	↓ (>CSF)	↑

↔ Intermediate (iso) signal, ↓ Dark (low) signal, ↑ Bright (high) signal.

Note: In the presence of sedimentation of blood products within a subdural collection, the most precise dating with MR comes from signal analysis of the blood sediment [12].

Adapted from reference [11].

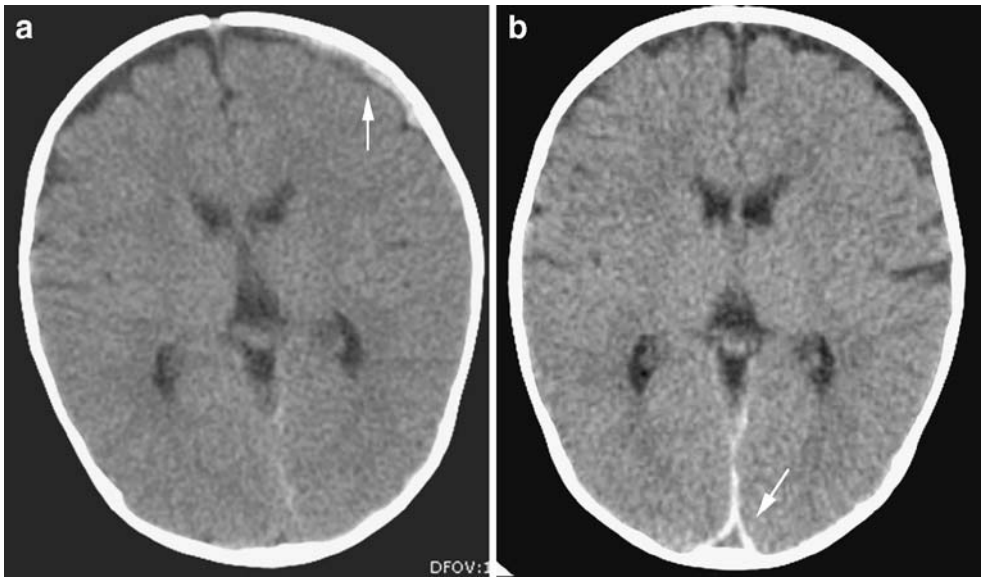

Fig. 2 Redistribution of SDH. Axial CT scan of a 4-month-old infant on admission to the emergency room (**a**) and 16 h later (**b**). The SDH initially evident over the left frontal convexity (**a** *arrow*) has shifted posteriorly (**b** *arrow*)

portion of the cranium (sedimentation) can be observed over a span of a few hours to 1–2 days after a bleed—a good indication of the recent nature of the bleed (Fig. 2). Such migration of SDH may lead to misinterpretation of a new hemorrhage [5]. Care must be taken not to mistake signal intensity or density differences of supernatant and sediment as SDH of different ages [12].

3. A decrease in the amount of hyperdense products within a day or so of admission usually results from redistribution of blood products, or from hemodilution by low attenuation fluid (CSF) within a hematohygroma, again indicating a recent bleed.
4. An increase in the amount of hypodense products within the subdural space over the first few days or week following trauma can be observed in acute hematohygromas. This is presumably caused by ongoing acute leakage of CSF into the subdural space [7]; at times CSF can accumulate quite rapidly (Fig. 1). With coexistent acute and chronic SDH, the low attenuation chronic subdural compartment does not undergo a similar increase in size.
5. Dynamic changes in the density and signal intensity of a SDH occur in the first 2 weeks after a bleed (see Tables 1 and 3).

Subdural vs subarachnoid collections

As a final point, one needs to stress the importance of differentiating low-attenuation subdural fluid collections from the subarachnoid fluid collections that are commonly observed in infants (e.g., "benign hygromas of infancy"). On CT images, and more accurately on MR images, one can localize extra-axial fluid by the examination of the pial

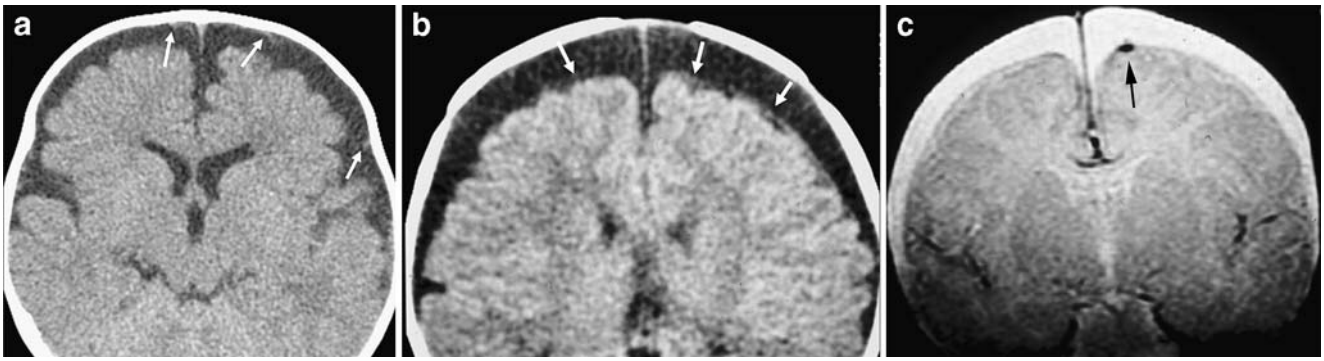

Fig. 3 Differentiation of subdural fluid collection from benign hygroma of infancy. **a**, **b** Axial CT images of benign hygroma of infancy (**a**) and a chronic SDH in an abused infant (**b**). In **a**, note the position of the pial vessels (*arrows*) away from the surface of the cerebrum (subarachnoid fluid). In **b**, note the preferential position of the vessels (*arrows*) against the surface of the cerebrum (subdural fluid). **c** Coronal gradient echo T2-W image of the abused infant (same patient as **b**) confirms subdural location of the fluid and reveals a focal subarachnoid clot (*arrow*) at the vertex

Table 4 SDH – differential diagnosis.

Trauma (inflicted, accidental)[a]
Genetic disorders
Menkes kinky hair disease
Glutaric acidemia type I
Osteogenesis imperfecta, Ehlers-Danlos syndrome
Hematologic disorders/coagulopathy
Hemophilia, hemophagocytic lymphohistiocytosis
Dural arteriovenous malformation
Sinovenous thrombosis
Rupture of arachnoid cyst
Meningitis/encephalitis/vasculitis
Oncological disease
Leukemia, neuroblastoma
Hypoxic-ischemic injury

[a] Birth trauma usually not relevant after 3–4 weeks [15].

Adapted from reference [5].

vessels that overlay the cerebrum. Displacement of these vessels away from the brain, against the inner surface of the skull by a fluid collection, indicates a subarachnoid location. Conversely, positioning of the pial vessels principally along the surface of the brain, with the fluid collection interposed between the vessels and the inner layer of the skull, is diagnostic of a subdural fluid collection (Fig. 3).

Such differentiation should be made on every CT and MR study in the infant patient population: bilateral, symmetric subarachnoid fluid collections that overlay the anterior cerebral hemispheres in an otherwise normally developing infant with an enlarged head is typical of a benign subarachnoid fluid collection of infancy ("benign hygroma of infancy"), usually a self-limited condition. Whereas the presence of subdural fluid in an infant raises the possibility of a hematohygroma or a chronic SDH; discovery of an unexplained subdural collection should trigger a search for an explanation and concern that the collection is the result of NAHI. Interestingly, enlarged subarachnoid fluid spaces appear to predispose infants to development of subdural hemorrhage [13], although this is still debated [14]. Incidental subdural bleeding next to a subarachnoid hygroma is usually minor in extent and does not necessarily indicate NAHI [13].

Conclusion

Evaluation of head injury in suspected abused children is radiographically and clinically challenging. In the case of SDH, a differential diagnosis should be explored when appropriate (Table 4); most nontraumatic causes of SDH can be diagnostically excluded or confirmed by history, physical examination, and radiological or laboratory studies [8]. The intent behind a posttraumatic SDH cannot be inferred from CT or MRI alone; a competent child protection investigation is the only basis to determine if an injury is inflicted or accidental, supported by the medical, imaging, biochemical and/or pathological findings [5].

References

1. Fernando S, Obaldo RE, Walsh IR et al (2008) Neuroimaging of nonaccidental head trauma: pitfalls and controversies. Pediatr Radiol 38:827–838
2. Case ME (2008) Inflicted traumatic brain injury in infants and young children. Brain Pathol 18:571–582
3. Huisman TA (2005) Intracranial hemorrhage: ultrasound, CT and MRI findings. Eur Radiol 15:434–440
4. Lee KS, Bae WK, Bae HG et al (1997) The computed tomographic attenuation and the age of subdural hematomas. J Korean Med Sci 12:353–359
5. Barnes PD, Krasnokutsky M (2007) Imaging of the central nervous system in suspected or alleged nonaccidental injury, including the mimics. Top Magn Reson Imaging 18:53–74
6. Tung GA, Kumar M, Richardson RC et al (2006) Comparison of accidental and nonaccidental traumatic head injury in children on noncontrast computed tomography. Pediatrics 118:626–633
7. Zouros A, Bhargava R, Hoskinson M et al (2004) Further characterization of traumatic subdural collections of infancy. Report of five cases. J Neurosurg 100(5 Suppl Pediatrics):512–518
8. Hymel KP, Jenny C, Block RW (2002) Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. Child Maltreat 7:329–348
9. Geddes JF, Hackshaw AK, Vowles GH et al (2001) Neuropathology of inflicted head injury in children. I. Patterns of brain damage. Brain 124:1290–1298
10. Williams VL, Hogg JP (2000) Magnetic resonance imaging of chronic subdural hematoma. Neurosurg Clin N Am 11:491–498
11. Bradley WG Jr (1993) MR appearance of hemorrhage in the brain. Radiology 189:15–26
12. Vinchon M, Noulé N, Tchofo PJ et al (2004) Imaging of head injuries in infants: temporal correlates and forensic implications for the diagnosis of child abuse. J Neurosurg 101(1 Suppl):44–52
13. Ravid S, Maytal J (2003) External hydrocephalus: a probable cause for subdural hematoma in infancy. Pediatr Neurol 28:139–141
14. Raul JS, Roth S, Ludes B et al (2008) Influence of the benign enlargement of the subarachnoid space on the bridging veins strain during a shaking event: a finite element study. Int J Legal Med 122:337–340
15. Rooks VJ, Eaton JP, Ruess L et al (2008) Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. AJNR 29:1082–1089