Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine

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Nonaccidental injury (NAI) is reportedly the most frequent cause of traumatic injury in infants (peak incidence age 6 months; 80% of traumatic brain injury deaths under the age of 2 years).1–4 NAI, non-accidental trauma (NAT), and nonaccidental head injury are more recently used terms instead of the traditional labels, child abuse, battered child syndrome, and shaken baby syndrome (SBS). The traditional definition of NAI/SBS is intentional or inflicted physical injury to infants characterized by the triad of (1) subdural hemorrhage (SDH), (2) retinal hemorrhage (RH), and (3) encephalopathy (ie, diffuse axonal injury [DAI]) occurring in the context of inappropriate or inconsistent history (particularly when unwitnessed) and commonly accompanied by other apparently inflicted injuries (eg, skeletal).1–4 This empirical formula is under challenge by evidence-based medical and legal principals.1–14

TRAUMATIC BRAIN INJURY

Traumatic brain injury has been categorized in several ways.1,4 Primary injury directly results from the initial traumatic force and is immediate and irreversible (eg, contusion or shear injury). Secondary injury arises from or is associated with the primary injury and is potentially reversible (eg, swelling, hypoxia-ischemia, seizures, or herniation). Traditional biomechanics describes impact loading as linear forces that produce localized cranial deformation and focal injury (eg, fracture, contusion, or epidural hematoma). Accidental injury (AI) is considered typically associated with impact and, with the exception of epidural hematoma, is usually not life threatening. Impulsive loading refers to angular acceleration/deceleration forces resulting from sudden nonimpact motion of the head on the neck (ie, whiplash) and produces diffuse injury with tissue disruption (eg, bridging vein rupture with SDH and white matter shear with DAI). Young infants are thought particularly vulnerable to the latter mechanism (ie, SBS) because of weak neck muscles, a relatively large head, and an immature brain. SBS is traditionally postulated to result in the triad of primary traumatic injury (ie, SDH, RH, and DAI), which has been reportedly associated with the most severe and fatal CNS injuries. Stated assault mechanisms

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in NAI include battering, shaking, impact, shaking-impact, strangulation, suffocation, and combined assaults (shake-bang-choke). Although the spectrum of injury in NAI overlaps that of AI, certain patterns have been previously reported as characteristic of or highly suspicious for NAI. These include multiple or complex cranial fractures (Fig. 1), acute interhemispheric SDH (Fig. 2), acute-hyperacute SDH (Fig. 3), DAI, chronic SDH, and the combination of chronic and acute SDH (Fig. 4). The latter combination is thought indicative of more than one abusive event. Imaging evidence of brain injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally higher-specificity imaging findings of abuse (eg, classic metaphyseal lesions or rib fractures) (Fig. 5). Therefore, clinical and imaging findings of injury out of proportion to the history of trauma and injuries of different ages have been the basis of making a medical diagnosis and offer expert testimony that such “forensic” findings are “proof” of NAI/SBS, particularly when encountered in premobile, young infants.

EVIDENCE-BASED MEDICINE

Evidence-based medicine (EBM) is now the guiding principle as medicine moves from an authoritarian to an authoritative era to overcome bias and ideology. EBM quality-of-evidence ratings of the literature (eg, classes I–IV) are based on levels of accepted scientific methodology and biostatistical significance (eg, P values) and apply to the formulation of standards and guidelines for every aspect of medicine, including diagnostics, therapeutics, and forensics. EBM analysis reveals that few published reports in the traditional NAI/SBS literature merit a quality-of-evidence rating above class IV (eg, expert opinion alone). Such low ratings do not meet EBM recommendations for standards (eg, level A) or for guidelines (eg, level B). Difficulties exist in the rational formulation of a medical diagnosis or forensic determination of NAI/SBS based on an alleged event (eg, shaking) that is inferred from clinical, imaging, or pathology findings in the subjective context of (1) an unwitnessed event, (2) a noncredible history, or (3) an admission or confession under dubious circumstances. This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS and because much of the traditional literature on child abuse consists of anecdotal case series, case reports, reviews, opinions, and position papers. Many reports include cases having impact injury, which

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**Fig. 1.** Nine-week-old infant with triad and alleged NAI; also, history of traumatic labor and delivery. Skull film (A), CT (B) plus FLAIR (C), T2 (D), and T1 (E) MR imaging shows bilateral skull fractures with left growing fracture (long white arrows), chronic bifrontal cerebral white matter clefts (short white arrows) (C) plus acute, subacute, and chronic SDHs/rehemorrhages (yellow arrows).
undermines the SBS hypothesis by imposing a shaking-impact syndrome. Also, the inclusion criteria provided in many reports are criticized as arbitrary. Examples include suspected abuse, presumed abuse, likely abuse, and indeterminate.\textsuperscript{21,22} Furthermore, the diagnostic criteria often seem to follow circular logic, such that the inclusion criteria (eg, the triad equals SBS/NAI) becomes the conclusion (ie, SBS/NAI equals the triad).

\textbf{Fig. 2.} Five-week-old infant with triad and alleged NAI; also, cold symptoms, vitamin D undersupplemented, acute choking episode during feeding, and status epilepticus. Chest film (A) shows bilateral lung opacities. CT (B, C) plus T2* MR imaging (D) shows bilateral cerebral edema with bilateral thin, acute-subacute hemorrhages (or thromboses) about the falx, tentorium, and convexities (arrows). Vertex CT (E) shows suture diastasis versus pseudodiastasis (arrows) (craniotabes?). DWI (F) shows global hypoxic-ischemic injury. Later CT (G) shows atrophy and chronic SDH.

\textbf{Fig. 3.} Eight-month-old infant with triad and alleged NAI; also, right occipital skull fracture (age indeterminate; not shown) and 4- to 6-week-old wrist fracture. Hyperacute right SDH versus chronic SDH with rehemorrhage? CT (A, B) shows mixed high- plus low-density right extracerebral collection (arrows) with right cerebral edema, mass effect, and left shift. Question of subdural membrane on autopsy.
RULES OF EVIDENCE AND EXPERT TESTIMONY

Regarding rules of evidence within the justice system, there are legal standards for the admissibility of expert testimony. The Frye standard requires only that the testimony be generally accepted in the relevant scientific community. The Daubert standard requires assessment of the scientific reliability of the testimony. A criticism of the justice system is that the application of these standards varies with the jurisdiction (e.g., according to state versus federal law). Additional legal standards regarding proof are also applied in order for the trier of fact (e.g., judge or jury) to make the determination of civil liability or criminal guilt. In a civil action (e.g., medical malpractice lawsuit), money is primarily at risk for the defendant health care provider, and proof of liability is based on a preponderance of the evidence (i.e., at least 51% scientific or medical probability or certainty). In a criminal action, life or liberty is at stake for the defendant, including the permanent loss of child custody. In such cases, the defendant has the constitutional protection of due process that requires a higher level of proof. This includes the principles of innocent until proved guilty beyond a reasonable doubt with the burden of proof on the prosecution and based on clear and convincing evidence. No percentage of level of certainty is provided, however, for these standards of proof in most jurisdictions. Furthermore, only a preponderance of the medical evidence (i.e., minimum of 51% certainty) is required to support proof of guilt whether or not the medical expert testimony...

Fig. 4. Six-month-old infant with macrocephaly, the triad, and alleged NAI: BECC versus chronic SDH with rehemorrhage versus acute SDHG plus SDH? CT (A) shows bilateral frontal isohypodense extracerebral collections (arrows) with minute high densities (not shown). T1 MR imaging (B) shows smaller extracerebral high intensities (arrows) superimposed on larger isohypointensities. T2 MR imaging (C) shows small extracerebral T2 hypointensities (arrows) superimposed on large isohyperintensities.

Fig. 5. Three-month-old infant with alleged NAI; also, history consistent with congenital rickets. Chest film (A) shows bilateral recent and old, healing rib fractures (pseudo fractures? rachitic rosary? [arrows]). Knee films before (B) and after (C) vitamin D supplementation show healing classic metaphyseal lesions (arrows).
complies with the Frye standard (ie, general acceptance requirement) or the Daubert standard (ie, scientific reliability requirement). Further criticism of the criminal justice process is that in NAI cases, medical experts have defined SBS/NAI as “the presence of injury (eg, the triad) without a sufficient historical explanation” and that this definition unduly shifts the burden to the defendant to establish innocence by proving the expert theory wrong.

THE MEDICAL PROSECUTION OF NAI AND ITS EBM CHALLENGES

Traditionally, the prosecution of NAI has been based on the presence of one or more aspects of the triad as supported by the premises that (1) shaking alone in an otherwise healthy child can cause SDH leading to death, (2) such injury can never occur on an accidental basis (eg, short-distance fall) because it requires a massive violent force equivalent to a motor vehicle accident or a fall from a multistory building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with prior head injury indicates newly inflicted injury and not a spontaneous re-bleed. Using this reasoning, the last caretaker is automatically guilty of inflicted injury, especially if not witnessed by an independent observer. Also, it has been asserted that RHs of a particular pattern are diagnostic of SBS/NAI.

Reports from clinical, biomechanical, pathology, forensic, and legal disciplines, within and outside of the child maltreatment literature, have challenged the evidence base for NAI/SBS as the only cause for the triad. Such reports indicate that the triad may also be seen with AI (including witnessed short-distance falls, lucid intervals, and rehemorrhage) as well as in medical conditions. These are the mimics of NAI and often present as acute life-threatening events (ALTEs). The medical mimics include hypoxia-ischemia (eg, apnea, choking, or respiratory or cardiac arrest) (see Figs. 2, 6, and 7), ischemic injury (eg, arterial versus venous occlusive disease) (Fig. 8), vascular anomalies (eg, arteriovenous malformation [AVM]) (Fig. 9), seizures (see Fig. 2), infectious or postinfectious conditions (Fig. 10), coagulopathies (Fig. 11), fluid-electrolyte derangement, and metabolic or connective tissue disorders, including vitamin deficiencies and depletions (eg, C, D, or K) (see Figs. 1 and 5; Fig. 12).

Many ALTEs seem multifactorial and involve a combination, sequence, or cascade of predisposing and complicating events or conditions. As an example, an infant may suffer a head impact, or choking spell, followed by seizures or apnea, and then undergo a series of interventions, including prolonged or difficult resuscitation and problematic airway management with subsequent hypoxia-ischemia and coagulopathy (see Figs. 2, 6, 7, and 11). Another example is a young infant with a predisposing condition, such as infectious illness, fluid-electrolyte imbalance, metabolic disorder, or a coagulopathy, who then suffers seizures, respiratory arrest, and resuscitation with hypoxia-ischemia (see Figs. 10–12; Fig. 13). In many cases of alleged SBS/NAI, it is often assumed that nonspecific premorbid symptoms (eg, irritability, lethargy, and poor feeding) in an otherwise healthy infant are indicators of ongoing abuse or that such symptoms become the inciting factor for the abuse. A thorough and complete medical investigation in such cases may reveal that the child is not otherwise healthy and is suffering from a medical condition that progresses to an ALTE.

BIOMECHANICAL CHALLENGES

The mechanical basis for SBS as hypothesized by Guthkelch, Caffey, and other investigators, was originally extrapolated from Ommaya, who used an animal whiplash model to determine the angular acceleration threshold (ie, 40 g) for head injury (ie, concussion, SDH, and shear injury). It was assumed that manual shaking of an infant could generate these same forces and produce the triad. Duhaime and colleagues measured the angular accelerations associated with adult manual shaking (ie, 11 g) and impact (ie, 52 g) in a 1-month-old infant anthropomorphic test device (ATD). Only accelerations associated with impact (4 to 5 times that associated with shakes) on an unpadded or padded surface exceeded the injury thresholds determined by Ommaya. In the same study, the Duhaime and colleagues reported a series of 13 fatal cases of NAI/SBS in which all had evidence of blunt head impact (more than half noted only at autopsy). The investigators concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. Their results contradicted many of the original reports that had relied on the whiplash mechanism as causative of the triad. They suggested the use of the new term, shaken-impact syndrome. More recently, Prange and colleagues, using a 1.5 month-old ATD, showed that inflicted impacts against hard surfaces were more likely associated with brain injury than falls from less than 1.5 m or from vigorous shaking. With further improvements in ATDs, more recent experiments indicate that maximum head injury (more than half noted only at autopsy)
accelerations may exceed injury reference values at lower fall heights than previously determined (Fig. 14). Critics of the Duhaime and Prange studies contend that there is no adequate human infant surrogate yet designed to properly test shaking versus impact. Other reports also show that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant injury to the neck, cervical spinal column, or cervical spinal cord, because these are the weak links between the head and body of the infant. Spinal cord injury without radiographic abnormality (SCIWORA), whether or not AI or NAI, is an important example of primary neck and spinal cord injury with secondary brain injury (see Fig. 7). For example, a falling infant experiences a head-first impact with subsequent neck hyperextension (or hyperflexion) from the force of the trailing body mass. There is resultant upper spinal cord injury without detectable spinal column injury on plain films or CT. Compromise of the respiratory center at the cervicomedullary junction results in hypoxic brain injury, including the thin SDH (see Fig. 7). CT often shows the brain injury, but only MR imaging may show the additional neck or spinal cord injury.
Fig. 7. Twenty-one-month-old with triad and alleged NAI; also, history of 4-ft fall. CT (A, B) with high-density SAH and thin SDH (arrows) plus cerebral edema. Sagittal plane photomicrograph (C) from autopsy shows upper cervical spinal cord disruption (arrows) resulting in global hypoxic-ischemic injury.

Fig. 8. Fourteen-month-old infant with triad and alleged NAI; also, recent infectious illness: dural and cortical venous sinus thrombosis with dural hemorrhage: CT (A, B) shows high densities along the falx and dural venous sinuses (white arrows). (C) Gross specimen—reflected superior sagittal sinus and cortical venous thromboses with distended veins (yellow arrows); (D) photomicrograph of cortical venous thrombus with inflammatory reaction (black arrows) plus SDH with neomembrane (7–14 days old; not shown). (Pathology courtesy of J. Leestma, MD.)
The minimal force required to produce the triad has yet to be established. From the current biomechanical evidence base, however, it can be concluded that (1) shaking may not produce direct brain injury but may cause indirect brain injury if associated with neck and cervical spinal cord injury; (2) angular acceleration/deceleration injury forces clearly occur with impact trauma; (3) such injury on an accidental basis does not require a force that can only be associated with a motor vehicle accident or a multistory fall; (4) household (ie, short-distance) falls may produce direct or indirect brain injury; (5) in addition to fall height, impact surface and type of landing are important factors; and (6) head-first impacts in young infants not having developed a defensive reflex (eg, extension of a limb to break the fall) are the most dangerous and may result in direct or indirect brain injury (eg, SCIWORA).

**NEUROPATHOLOGY CHALLENGES**

In their landmark neuropathology study of 53 victims of alleged SBS/NAI,36,37 Geddes and colleagues showed in 37 infants (ages <9 months) that (1) 29 had evidence of impact with only one case of admitted shaking; (2) cerebral swelling was more often due to DAI of hypoxic-ischemic encephalopathy (HIE) rather than shear or traumatic axonal injury (TAI); (2) cerebral swelling was more often due to DAI of hypoxic-ischemic encephalopathy (HIE) rather than shear or traumatic axonal injury (TAI); (2) although fracture, thin SDH (eg, dural vascular plexus origin), and RH are commonly present, the usual cause of

**Fig. 9.** Twenty-month-old infant with triad and alleged NAI. Left SDH with cerebral cortical and pial AVM at autopsy. CT (A, B) shows left mixed-density SDH and SAH (long arrows) plus interhemispheric hemorrhage (short arrows) with marked left cerebral swelling and shift.

**Fig. 10.** Twenty-one-month-old infant with triad and alleged NAI. Pneumococcal meningitis, herniation, and hypoxic-ischemic injury confirmed at autopsy. CT (A–C) shows high-density thin SDH (arrows) plus cerebral edema.
death was increased intracranial pressure from brain swelling associated with HIE (see Fig. 2); and (4) cervical epidural hemorrhage and focal axonal brainstem, cervical cord, and spinal nerve root injuries were characteristically seen in these infants (most with impact). Upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the HIE. In the 16 older victims (ages 13 months to 8 years), the pathology findings were primarily those of the battered child or adult trauma syndrome, including extracranial injuries (eg, abdominal), large SDH (ie, bridging vein rupture), and TAI. Additional neuropathology series by Geddes and colleagues have shown that SDHs are also seen in nontraumatic fetal, neonatal, and infant brain injury cases and that such SDHs are actually of intradural vascular plexus origin rather than bridging cortical vein origin.

The common denominator in all these cases is likely a combination of vascular immaturity and fragility further compromised by HIE or infection, cerebral venous hypertension or congestion, arterial hypertension, and brain swelling (see Fig. 2). Although the unified hypothesis of Geddes and colleagues has received criticism, their findings and conclusions have been validated by the research of Cohen and Scheimberg, Croft and Reichard, and others. In their postmortem series, Cohen and colleagues described 25 fetuses (26–41 weeks) and 30 neonates (1 hour–19 days) with HIE who also had macroscopic intradural hemorrhage (IDH), including frank parietal SDH in two-thirds. The IDH was most prominent along the posterior falx and tentorial vascular plexuses (ie, interhemispheric fissure) (see Fig. 2). They concluded from their work, along with the findings of other cited studies.

Fig. 11. Nine-month-old girl with triad and alleged NAI; also, recent fall and coagulopathy (later confirmed platelet disorder). Initial CT (A) shows mixed-density right SDH (arrows) with right cerebral edema. Postoperative CT 5 days later (B) shows other cerebral and intraventricular hemorrhages (arrows). T1 MR imaging (C) 11 days postoperatively shows evolving right cerebral high-intensity cortical injury and hemorrhages.

Fig. 12. Twelve-month-old infant with triad and alleged NAI. Glutaric acidopathy type 1. CT (A) and T2 MR imaging (B) shows bilateral SDH of varying age (long arrows), wide sylvian fissures plus basal ganglia, and cerebral white matter abnormalities (short arrows).
researchers, that IDH and SDH are commonly associated with HIE, particularly when associated with increases in central venous pressure. This also explains the frequency of RH associated with perinatal events.42

From the current forensic pathology evidence base, it may be concluded that (1) shaking may not cause direct brain injury but may cause indirect brain injury (ie, HIE) if associated with cervical spinal cord injury; (2) impact may produce direct or indirect brain injury (eg, SCIWORA); (3) the pattern of brain edema with thin SDH (dural vascular plexus origin) may reflect HIE whether or not due to Al or NAI; and (4) the same pattern of injury may result from nontraumatic or medical causes (eg, HIE from any cause of ALTE). Furthermore, because the observed edema does not represent TAI (which results in immediate neurologic dysfunction), a lucid interval is possible, particularly in infants whose sutured skull and

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**Fig. 13.** Home-delivered newborn with seizures at 1 week of age; also, no vitamin K given at birth. T1 (A) and T2 (B) MR imaging shows acute-subacute left SDH (*long arrows*) plus right cerebral hemorrhage (*short arrows*); vitamin K deficiency confirmed and treated.

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dural vascular plexus have the distensibility to tolerate early increases in intracranial pressure. Also, the lucid interval invalidates the premise that the last caretaker is always responsible in alleged NAI.

**CLINICAL CHALLENGES**

In the prosecution of NAI, it is often stipulated that short-distance falls cannot be associated with the triad, serious (eg, fatal) head injury, or a lucid interval. Traditionally, it has also been stipulated that nonintentional new bleeding in an existing SDH is always minor, that SDH does not occur in benign extracerebral collections (BECCs), and that symptomatic or fatal new bleeding in SDH requires newly inflicted trauma.1–4,7,8,11 Several past and current reports refute the significance of low level falls in children, including in-hospital and outpatient clinic series.43–51 There are other reports, however, including emergency medicine, trauma center, neurosurgical, and medical examiner series, that indicate a heightened need for concern regarding the potential for serious intracranial injury associated with minor or trivial trauma scenarios, particularly in infants.52–74 This includes reports of skull fracture or acute SDH from accidental simple falls in infants, SDH in infants with predisposing wide extracerebral spaces (eg, BECCs of infancy, chronic subdural hygromas, arachnoid cyst, and so forth) (see Fig. 4; Figs. 15 and 16), and fatal pediatric head injuries due to witnessed, accidental short-distance falls, including those with a lucid interval, SDH, RH, and malignant cerebral edema (see Fig. 6). Also included are infants with chronic SDH from prior trauma (eg, at birth) who then develop rehemorrhage (see Figs. 1, 4, and 15).

**Short-Distance Falls, Lucid Intervals, and Malignant Edema**

Hall and colleagues44 reported that 41% of childhood deaths (mean age 2.4 years) from head injuries associated with AI were from low level falls (3 feet or less) while running or down stairs. Chadwick and colleagues45 reported fatal falls of less than 4 feet in seven infants but considered the histories unreliable. Plunkett56 reported witnessed fatal falls of 2 to 10 feet in 18 infants and children, including those with SDH, RH, and lucid intervals. Greenes and Schultzman57 reported intracranial injuries, including SDH, in 18 asymptomatic infants with falls of 2 feet to 9 stairs. Christian and colleagues53 reported three infants with unilateral RH and SDH/SAH due to witnessed accidental household trauma. Denton and Mileusnic59 reported a witnessed, accidental 30-inch fall in a 9-month-old infant with a 3-day lucid interval before death. Murray and colleagues60 reported more intracranial injuries in young children (49% < age 4 y; 21% < age 1 y) with reported low level falls (<15 ft), both AI and NAI. Kim and colleagues61 reported a high incidence of intracranial injury in children (ages 3 mo to 15 y; 52% < age 2 y) accidentally falling from low heights (3 to 15 ft; 80% <6 ft; including 4 deaths). Because of the lucid intervals in some patients, including initially favorable Glasgow Coma Scale scores (GCS) with subsequent deterioration, Murray and colleagues60 and others expressed concern regarding caretaker delays and medical transfer delays contributing to the morbidity and mortality in these patients.53–56,58–61 Bruce and colleagues54,55 reported one of the largest pediatric series of head trauma (63 patients, ages 6 months to 18 years), both AI and NAI, associated with malignant brain edema and SAH/SDH (see Fig. 6). In the higher GCS (>8) subgroup,
there were 8 with a lucid interval and all 14 had complete recovery. In the lower GCS (≤ 8) subgroup, there were 34 with immediate and continuous coma, 15 with a lucid interval, 6 deaths, and 11 with moderate to severe disability. More recently, Steinbok and colleagues reported 5 children (4 < age 2 y; 3 falls) with witnessed AI, including SDH and cerebral edema detected by CT 1 to 5 hours post event. All experienced immediate coma with rapid progression to death (see Fig. 6).

**Benign Extracerebral Collections**

BECCs of infancy (also known as benign external hydrocephalus or benign extracerebral subarachnoid spaces) is a common and well-known condition characterized by diffuse enlargement of the subarachnoid spaces. A transient disorder of cerebrospinal fluid (CSF) circulation, probably due to delayed development of the arachnoid granulations, is widely accepted as the cause and develops from birth. BECC is typically associated with macrocephaly but may also occur in infants with normal or small head circumferences, including premature infants. As with any cause of craniocerebral disproportion (eg, BECC, hydrocephalus, chronic SDH or hygroma, arachnoid cyst, or underdevelopment or atrophy), there is a susceptibility to SDH that may be spontaneous or associated with trivial trauma (see Figs. 4 and 15). A recent large series report and review by Hellbusch emphasizes the importance of this predisposition and cites other confirmatory series and case reports (30 references). Papasian and Frim designed a theoretic model that predicts the predisposition of benign external hydrocephalus to SDH with minor head trauma. Platt’s case report of BECC with SDH (27 references), including RH, along with McNeely and colleagues’ case series are further warnings that this combination is far from specific for SBS/NAI.

**Birth Issues**

In addition to the examples discussed previously (eg, short-distance falls and BECCs), another important but often overlooked factor is birth-related trauma. This includes normal as well as complicated labor and delivery events (pitocin augmentation, prolonged labor, vaginal delivery, instrumented delivery, cesarean section, and so forth). It is well known that acute SDH often occurs even with the normal birth process and that this predisposes to chronic SDH, including in the presence of BECC (see Figs. 1, 4, and 15). Intracranial hemorrhages, including SDH and RH, have been reported in several CT and MR imaging series of normal neonates including a frequency of 50% by Holden and colleagues, 8% by Whitby and colleagues, 26% by Looney and colleagues, and 46% by Rooks and colleagues. Chamnanvanakij and colleagues reported 26 symptomatic term neonates with SDH over a 3-year period after uncomplicated deliveries. Long-term follow-up imaging has not been provided in many of these series, although Rooks and colleagues reported one child in their series who developed SDH with rehemorrhage superimposed on BECC (Fig. 17).
Chronic SDH and Rehemorrhage

Chronic SDH is one of the most controversial topics in the NAI versus AI debate.\textsuperscript{1, 4, 12, 21, 22, 36–41} Unexplained SDH is often ascribed to NAI. By definition, a newly discovered chronic SDH started as an acute SDH that, for whatever reason, may have been subclinical. There is likely more than one mechanism for SDH that has prompted a revisiting of the concept of the subdural compartment.\textsuperscript{12, 40, 41, 90, 91} Mack and colleagues\textsuperscript{90} have provided an updated review on this important topic. In some cases of infant trauma, dissection at the relatively weak dura-arachnoid border zone (ie, dural border cell layer) may allow CSF to collect and enlarge over time as a dural interstitial (ie, intradural) hygroma. In other cases, there is bridging vein rupture within the dural interstitium that results in an acute subdural or intradural hematoma that extends along the dural border cell layer. Furthermore, traumatic disruption of the dural vascular plexus (ie, venous, capillary, or lymphatic), which is particularly prominent in young infants, may also produce an acute intradural hematoma. Some of these collections undergo resorption whereas others progress to become chronic SDH. Some progressive collections may represent mixed CSF-blood collections (see Figs 1, 4, and 15).

The pathology and pathophysiology of neomembrane formation in chronic SDH, including rebleeding, is well established in adults and seems similar, if not identical, to that in infants.\textsuperscript{83, 92–112} Although acute SDH is most often due to impact or deformational trauma, whether or not AI or NAI, it must be differentiated from chronic SDH with rehemorrhage. Progression of chronic SDH and rehemorrhage is likely related to capillary leakage and intrinsic thrombolysis.\textsuperscript{92, 93} Other factors include dural vascular plexus hemorrhage associated with increases in intracranial or central venous pressures (eg, birth trauma, congenital heart disease, venous thrombosis, or dysphagic choking) or with increased meningeal arterial pressure (eg, reperfusion after hypoxia-ischemia) with resultant acute hemorrhage (or rehemorrhage) in normal infants or superimposed on predisposing chronic BECC, hygromas, hematomas, or arachnoid cysts (see Figs. 1, 2, 4, and 15–17).\textsuperscript{12, 38, 40, 65–74, 90, 91} The phenomenon of acute infantile SDH, whether or not AI or NAI, evolving to chronic SDH and rehemorrhage, including RH, is well documented in several neurosurgical series reports, including those by Aoki and colleagues,\textsuperscript{97, 98} Ikeda and colleagues,\textsuperscript{99} Parent,\textsuperscript{94} Howard and colleagues,\textsuperscript{102} Hwang and Kim,\textsuperscript{95} Vinchon,\textsuperscript{103, 104} and others.

Conclusions

From the clinical evidence base, in addition to the biomechanical and neuropathology evidence bases, it may be concluded that (1) significant head injury, including SDH and RH, may result from low fall levels; (2) such injury may be associated with a lucid interval; (3) in some, the injury may result in immediate deterioration with progression to death; (4) BECC predisposes to SDH; (5) SDH may date back to birth; and (6) rehemorrhage into an existing SDH occurs in childhood and may be serious.
RH CHALLENGES

Many guidelines for diagnosing NAI depend on the presence of RH, including those of a particular pattern (e.g., retinal schisis, and perimacular folds) and based on the theory of vitreous traction due to inflicted acceleration/deceleration forces (e.g., SBS).1–4,113–132 The specificity of RH for NAI has been repeatedly challenged, however. Plunkett136 reported RH in two-thirds of eye examinations in children with fatal AI. Goldsmith and Plunkett132 reported a child with extensive bilateral RH in a videotaped fatal accidental short-distance fall. Lantz and colleagues122 reported RH with perimacular folds in an infant crush injury. Gilles and colleagues120 reported the appearance and progression of RH with increasing intracranial pressure after head injury in children. Obi and Watts125 reported RH with schisis and folds in two children, one with AI and the other with NAI. Forbes and colleagues126 reported RH with epidural hematoma in five infant AI cases. From a research perspective, Brown and colleagues128 found no eye pathology in their fatal shaken animal observations. Binenbaum and colleagues127 observed no eye abnormalities in piglets subjected to acceleration/deceleration levels greater than 20 times what Prange and colleagues30 predicted possible in inflicted injury. Emerson and colleagues129 found no support for the vitreous traction hypothesis as unique to NAI. The eye and optic nerve are an extension of, and therefore a window to, the CNS, including their shared vascularization, meningeal coverings, innervation, and CSF spaces. RH has been reported with a variety of conditions, including AI, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage, sepsis, coagulopathy, certain metabolic disorders, systemic hypertension, and other conditions.4,12,25,38,90 Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a rush to judgment regarding NAI.4–11 Such bias may have devastating effects on an injured child and family. It is important to be aware of these mimics, because a more extensive work-up may be needed beyond routine screening tests. Also, lack of confirmation of a specific condition does not automatically indicate the default diagnosis of NAI. In all cases, it is critical to review all past records dating back to the pregnancy and birth as well as the postnatal pediatric records, family history, more recent history preceding the acute presentation, details of the acute event itself, resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings. An incomplete medical evaluation may result in unnecessary cost shifting to

From the research and clinical evidence base, it may be concluded that (1) RH is not specific for NAI, (2) RH may occur in AI and medical conditions, and (3) predisposing factors and complicating cascade effects must be considered in the pathophysiology of RH.

MEDICAL CONDITIONS MIMICKING NAI

A significant part of the controversy is the medical conditions that may mimic the clinical presentations (i.e., the triad) and imaging findings of NAI.1,2,4,25,26,89,101 Furthermore, such conditions may predispose to or complicate AI or NAI, as part of a cascade that results in or exaggerates the triad. In some situations, it may be difficult or impossible to tell which of these elements are causative and which are the effects. These include HIE, seizures, dysphagic choking ALTE, cardiopulmonary resuscitation, infectious or postinfectious conditions (e.g., sepsis, meningoencephalitis, or postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation, and other conditions.1,25,89,101 Regarding pathogenesis of the triad (with or without other organ system involvement [e.g., skeletal]) and whether or not due to NAI, AI, or medical etiologies, the pathophysiology seems to be a combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or a collagenopathy imposed on the immature CNS, including the vulnerable dural vascular plexus as well as other organ systems.4,12,25,38,90 Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a rush to judgment regarding NAI.4–11 Such bias may have devastating effects on an injured child and family. It is important to be aware of these mimics, because a more extensive work-up may be needed beyond routine screening tests. Also, lack of confirmation of a specific condition does not automatically indicate the default diagnosis of NAI. In all cases, it is critical to review all past records dating back to the pregnancy and birth as well as the postnatal pediatric records, family history, more recent history preceding the acute presentation, details of the acute event itself, resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings. An incomplete medical evaluation may result in unnecessary cost shifting to
child protection and criminal justice systems and have further adverse effects regarding transplantation organ donation in brain death cases and custody/adoptive dispositions for the surviving child and siblings. Sirotnak’s recent review, along with others’, extensively catalogs the many conditions that may mimic NAIs.

**Birth Trauma and Neonatal Conditions**

Manifestations of birth trauma, including fracture, SDH, and RH, may persist beyond the neonatal period. Other examples are the sequelae of extracorporeal membrane oxygenation therapy, at-risk prematurity, and congenital heart disease. When evaluating a young infant with apparent NAIs, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues. These include hemorrhage or rehemorrhage into extracerebral collections existing from birth (see Figs. 1, 4, 13, and 15). There may be associated skeletal findings of birth trauma (eg, new or healing clavicle, rib, or long bone fractures), particularly in the presence of a bone fragility disorder (see Figs. 1, 2 and 5).

**Developmental Anomalies and Congenital Conditions**

Vascular malformations are rarely reported causes for the triad but may be underdiagnosed (see Fig. 9). BECCs and arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (see Figs. 4, 15–17).

**Genetic and Metabolic Disorders**

Several conditions in the genetic and metabolic disorders category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I (see Fig. 12), Menkes’ kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others.

**Hematologic Disease and Coagulopathy**

Conditions in the hematologic disease and coagulopathy category predispose to intracranial hemorrhage and RH (see Figs. 11 and 13). The bleeding or clotting disorder may be primary or secondary. A more extensive work-up beyond the usual screening tests is needed, including a hematology consultation. Conditions in the category include the anemias, hemorrhagic disease of the newborn (vitamin K deficiency), the hemophilias, thrombophilias, disseminated intravascular coagulation and consumption coagulopathy, liver or kidney disease, hemophagocytic lymphohistiocytosis, and anticoagulant therapy. Venous thrombosis includes dural venous sinus thrombosis (DVST) and cerebral venous thrombosis (CVT). DVST or CVT may be associated with primary or secondary hematologic or coagulopathic states. Risk factors include acute systemic illness, dehydration, fluid-electrolyte imbalance, sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncologic disease and therapy, head and neck infection, hypercoagulable, and trauma states. Infarction, SAH, SDH, or RH may be seen, especially in infants. High densities on CT may be present along the dural venous sinuses, tentorium, falx, or the cortical, subependymal, or medullary veins and be associated with SAH, SDH, or intracerebral hemorrhage (see Fig. 8). There may be focal infarctions, hemorrhagic or nonhemorrhagic, intraventricular hemorrhage, and massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be present. The thromboses and associated hemorraghes have variable MR imaging appearances depending on their age. CT venography (CTV) or magnetic resonance venography (MRV) may readily detect DVST but not CVT. The latter may be better detected as abnormal hypointensities on susceptibility-weighted T2* sequences but difficult to distinguish from hemorrhage (SDH or SAH), hemorrhagic infarction, contusion, or hemorrhagic shear injury.

**Infectious and Postinfectious Conditions**

Meningitis, encephalitis, or sepsis may involve the vasculature resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage. SDH and RH may also be seen (see Fig. 10). Postinfectious illnesses may also be associated with these findings. Included in this category are the encephalopathies of infancy and childhood, hemorrhagic shock and encephalopathic syndrome, and postvaccinial encephalopathy.

**Toxins, Poisons, and Nutritional Deficiencies**

The category of toxins, poisons, and nutritional deficiencies includes lead poisoning, cocaine, anticoagulants, over-the-counter cold medications, prescription drugs, and vitamin deficiencies or depletions (eg, K, C, or D). Preterm neonates, and other chronically ill infants, are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily effect bone development. Furthermore, the national and...
international epidemic of vitamin D deficiency and insufficiency in pregnant mothers, their term fetuses, and their undersupplemented breastfed term neonates predisposes them to rickets (ie, congenital). Such infants, who have also been subjected to the trauma of birth, may have skeletal imaging findings (eg, multiple healing fractures or pseudofractures) that are misinterpreted as NAI, especially in the presence of the triad (see Figs. 2 and 5).\(^{136,137}\)

**Dysphagic Choking ALTE as a Mimic of NAI**

Apnea is an important and common form of ALTE in infancy whose origin may be central, obstructive, or combined.\(^{25}\) The obstructive and mixed forms may present with choking, gasping, coughing, or gagging due to mechanical obstruction. When paroxysmal or sustained, the result may be severe brain injury or death due to a combination of central venous hypertension and hypoxia-ischemia. It is this synergism that produces cerebral edema and dural vascular plexus hemorrhage with SDH, SAH, and RH (see Fig. 2; Fig. 18). Examples include dysphagic choking (eg, aspiration of a feed or gastroesophageal reflux), viral airway infection (eg, RSV), and pertussis, particularly when occurring in a predisposed child (eg, prematurity, Pierre Robin syndrome, or sudden infant death syndrome).\(^{25,160–167}\)

**IMAGING CHALLENGES AND THE IMPORTANCE OF A DIFFERENTIAL DIAGNOSIS CT**

Because of the evidence-based challenges to NAI, imaging protocols should be designed to evaluate not only NAI versus AI but also the medical mimics. Noncontrast CT has been the primary modality for brain imaging because of its access, speed, and ability to show lesions (eg, hemorrhage and edema) requiring immediate neurosurgical or medical intervention.\(^{4,77,83–99,102–112,168–181}\) Cervical spinal CT may also be needed. CT angiography (CTA) or CTV may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation or aneurysm) or infarction (eg, dissection or venous thrombosis). A radiographic or scintigraphic skeletal survey should also be obtained according to established guidelines.\(^{179,180}\)

**MR Imaging**

Brain and cervical spinal MR imaging should be done as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.\(^{4,104,181–190}\) Brain MR imaging should include T1, T2, T2*, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted imaging/apparent diffusion coefficient (DWI/ADC). Gadolinium-enhanced T1 images should probably be used along with MRA and MRV. T1 and T2 are necessary for estimating the timing of hemorrhage, thrombosis, and other collections using published criteria.\(^{4,104,181}\) T2* techniques are most sensitive for detecting hemorrhage or thromboses but may not distinguish new (eg, deoxyhemoglobin) from old (eg, hemosiderin). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia.\(^{4,154,189,190}\) Restricted or reduced diffusion, however, may be seen with other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.\(^{4,154,189,190}\) Gadolinium-enhanced sequences and MRS can be used to evaluate for these other processes. Additionally, MRA and

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**Fig. 18.** Six-month-old infant with triad and alleged NAI; acute choking event while feeding. CT (A–D) shows bilateral cerebral edema with acute SAH and SDH (arrows), including along the falx, and tentorium. Autopsy confirmed the hemorrhages, a subdural membrane, and hypoxic-ischemic brain injury. (Courtesy of The Wisconsin Innocence Project.)
MRV are important to evaluate for arterial occlusive disease (eg, dissection) or venous thrombosis, although they cannot rule out small vessel disease. The STIR technique is particularly important for cervical spine imaging.

**Scalp and Skull Abnormalities**

Scalp injuries (eg, edema, hemorrhage, and laceration) are difficult to precisely time on imaging studies and depend on the nature and number of traumatic events or other factors (circulatory compromise, coagulopathy, medical interventions, and so forth).\(^1,4\) Skull abnormalities may include fracture and suture splitting. Fracture may not be readily distinguished from sutures, synchondroses, their normal variants, or from wormian bones (eg, osteogenesis imperfecta) on CT or skull films. 3-D–CT surface reconstructions may be needed. In general, the morphology of a fracture cannot differentiate NAI from AI and must be correlated with the trauma scenario (eg, biomechanically) (see Fig. 1). Skull fractures are also difficult to time because of the lack of periosteal reaction.\(^1,4\) Suture diastasis may be traumatic or a reflection of increased intracranial pressure but must be distinguished from pseudodiastasis due to a metabolic or dysplastic bone disorder (eg, congenital rickets) (see Fig. 2).\(^1,4,136,137\) The growing fracture (eg, leptomeningeal cyst) is not specific for NAI and may follow any diastatic fracture in a young infant, including birth related (see Fig. 1).\(^1,2,4\) Nondetection of scalp or skull abnormalities on imaging should not be interpreted as the absence of impact injury.

**Intracranial Collections**

It should not be assumed that such collections are always traumatic in origin. A differential diagnosis is always necessary and includes NAI, AI, coagulopathy (hemophilic and thrombophilic conditions), infectious and postinfectious conditions, metabolic disorders, and so forth.\(^2,4,22,89,90,101,106–110\) It may not be possible to specify with any precision the components or age of an extracerebral collection because of meningeal disruptions (eg, acute or subacute subdural hygroma [SDHG] versus chronic SDH, or subarachnoid versus thin SDH).\(^1,4,103,104,173–176,181\) Vezina\(^181\) has recently summarized the literature regarding the complexity of timing of intracranial collections. Subarachnoid and subdural collections, hemorrhagic or nonhemorrhagic, may be localized or extensive and may occur about the convexities, interhemispheric (along the falx), and along the tentorium. With time and gravity, these collections may redistribute to other areas, including into or out of the spinal canal, and cause confusion.\(^4,177,181,191\) For example, a convexity SDH may migrate to the peritentorial and posterior interhemispheric regions or into the intraspinal spaces. SDH migration may lead to a misinterpretation that there are hemorrhages of different timing. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may cause further confusion because density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location.\(^4,104,178,181\) Prominent subarachnoid CSF spaces are commonly present in infants (ie, BECCs). This entity predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (eg, dysphagic choking ALTE) (see Figs. 4, 5, and 17).\(^4,65–73\) A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Rapid spontaneous resolution and redistribution of acute SDH over a few hours to 1 to 2 days has been reported.\(^4,177,191\) A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space.

For apparent CT high densities, it may be difficult to differentiate cerebral hemorrhage from subarachnoid hemorrhage or from venous thrombosis (see Figs. 2, 3, 6–11, 15, 16, and 18).\(^4\) According to the literature, hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 0 to 3 hours up to 7 to 10 days.\(^4,104,178,181\) Hemorrhage that is isohypo-dense on CT (ie, nonclotted) may be hyperacute (<3 h) or chronic (>10 d) (see Figs. 3 and 11). The low density may also represent pre-existing, wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF-containing) that may be acute or chronic (see Figs. 3, 12 and 15).\(^4,103,104,175,181\) Blood levels are unusual in the acute stage unless there is coagulopathy.\(^4,104,181,188\) CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (see Figs. 3 and 15).\(^4,66,72,92–104,173,178,181\) Traditionally, the interhemispheric SDH as well as mixed-density SDH were considered characteristic, if not pathognomonic, of SBS/NAI.\(^1,2,4,168,171–173\) This has been proved unreliable. Interhemispheric SDH may be seen with AI or with nontraumatic conditions (eg, HIE, venous thrombosis, venous hypertension, or dysphagic choking ALTE) (see Figs. 2, 6–10).\(^178\) Mixed-density SDH also occurs in AI as well as in other conditions (see Figs. 3, 9, and 11).\(^178\) Furthermore, SDH may occur in BECC
spontaneously or result from minor trauma (ie, AI), and rehemorrhage within SDH may occur spontaneously or with minor AI (see Figs. 1, 4, 15, and 17).1,2,12,38,40,72,90,104,178,181

Only MR imaging may provide more precise information than CT regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses (Table 1) and (2) brain injury.104,181 As a result, MR imaging has become the standard and should be done as soon as possible. Mixed-intensity collections, however, are problematic regarding timing.181 Matching the MR imaging findings with the CT findings may help along with follow-up MR imaging. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. With mixed-intensity collections, MR imaging cannot reliably differentiate BECC with acute SDH from acute SDHG/SDH, from hyperacute SDH, or from chronic SDH or chronic SDHG with rehemorrhage (see Figs. 1, 4, and 13–17).4,104,181 T2* hypointensities are iron sensitive but may not differentiate hemor rhages from venous thromboses that are not detected by MRV (eg, cortical, medullary, or subependymal).

BRAIN INJURY

Edema or swelling in pediatric head trauma may represent primary injury or secondary injury and be acute-hyperacute (eg, minutes to a few hours) or delayed (eg, several hours to a few days), including association with short-distance falls and lucid intervals.4,53–62 The edema or swelling may be further subtyped as traumatic, malignant, hypoxic-ischemic, or related to (or combined with) other factors. Traumatic edema is related to areas of primary brain trauma (ie, contusion or shear) or to traumatic vascular injury with infarction (eg, dissection, herniation, or spasm) (see Figs. 3, 6, 9, and 11). Traumatic edema is usually focal or multifocal, whether or not hemorrhagic. CT, however, may not distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction.4 Focal or multifocal low density edema may also be seen with infarction (eg, arterial or venous occlusive), encephalitis, demyelination (eg, ADEM), or seizure edema.4,89,146–154 Also, MR imaging often shows sheet and contusional injury as focal/multifocal restricted diffusion, GRE hypointensities, and/or T2/FLAIR high intensities.4 Focal/multifocal ischemic findings may also be due to traumatic arterial injury (eg, dissection) or venous injury (eg, tear or thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus) (see Figs. 2, 6, and 11).4,64,154,189,192 These may not be reliably differentiated, however, from focal/multifocal ischemic or hemorrhagic infarction from nontraumatic causation (eg, dissection, vasculitis, venous, or embolic) even without supportive MRA, CTA, MRV, or angiography. Also, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed with

Table 1
MR imaging of intracranial hemorrhage and thrombosis

<table>
<thead>
<tr>
<th>Stage</th>
<th>Biochemical Form</th>
<th>Site</th>
<th>T1−MR Imaging</th>
<th>T2−MR Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute (+ edema)</td>
<td>Fe II oxyHb</td>
<td>Intact RBCs</td>
<td>Iso-low I</td>
<td>High I</td>
</tr>
<tr>
<td>(&lt;12 hours)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute (+ edema)</td>
<td>Fe II deoxy Hb</td>
<td>Intact RBCs</td>
<td>Iso-low I</td>
<td>Low I</td>
</tr>
<tr>
<td>(1–3 days)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early subacute (+ edema)</td>
<td>Fe III metHb</td>
<td>Intact RBCs</td>
<td>High I</td>
<td>Low I</td>
</tr>
<tr>
<td>(3–7 days)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Late subacute (− edema)</td>
<td>Fe III metHb</td>
<td>Lysed RBCs (extracellular)</td>
<td>High I</td>
<td>High I</td>
</tr>
<tr>
<td>(1–2 weeks)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early chronic (− edema)</td>
<td>Fe III transferrin</td>
<td>Extracellular</td>
<td>High I</td>
<td>High I</td>
</tr>
<tr>
<td>(&gt;2 weeks)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic (cavity)</td>
<td>Fe III ferritin and</td>
<td>Phagocytosis</td>
<td>Iso-low I</td>
<td>Low I</td>
</tr>
<tr>
<td></td>
<td>hemosiderin</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Fe II, ferrous; Fe III, ferric; Hb, hemoglobin; I, signal intensity; Iso, isointense; RBCs, red blood cells; +, present; —, absent.

Data from Refs. 4,188,189
encephalitis, seizures, and metabolic disorders. Therefore, a differential diagnosis is always required.\textsuperscript{4,154,189,192}

Malignant brain edema, a term used for severe cerebral swelling after head trauma, may lead to rapid deterioration.\textsuperscript{1,4,5,4,5,5,5,5,5,6,6,6} The edema is usually bilateral and may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon and associated with global ischemia. A unilateral form may also occur in association with an ipsilateral SDH that progresses to bilateral edema (see Figs. 3 and 6).\textsuperscript{64} There may be rapid or delayed onset (ie, lucid interval). Predisposing factors are not well established but likely include a genetic basis. Hyperemic edema may appear early as accentuated gray-white matter differentiation on CT, then progresses to loss of differentiation.

Global hypoxia (eg, apnea or respiratory failure) or ischemia (eg, cardiovascular failure or hypoperfusion) is likely a major cause of or contributor to brain edema in a child with head trauma (eg, malignant edema).\textsuperscript{4,38,40,5,5,6,6,6} HIE, depending on its severity and duration, may have a diffuse appearance acutely (ie, diffuse or vascular axonal injury) with decreased gray-white differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MR imaging (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (see Figs. 2, 6, 7, 10, and 18).\textsuperscript{4,189} It is typically bilateral but may not be symmetric. This more diffuse pattern may distinguish HIE from the multifocal pattern of primary traumatic injury, although they may coexist. Hypoxia-ischemic brain injury due to apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA) whether or not AI or NAI (see Fig. 7).\textsuperscript{4,38,40,5,5,6,6,6} It may also occur with any nontraumatic cause (choking, paroxysmal coughing, aspiration, and so forth) (see Figs. 2 and 18).\textsuperscript{4,25,160–166} In addition to the diffuse brain injury, there may be associated subarachnoid and SDH without mass effect (see Figs. 2, 7, 10, and 18).\textsuperscript{4,38,40,5,5,6,6,6} MR imaging shows hypoxic-ischemic injury, depending on timing, as diffuse-restricted diffusion on DWI/ADC plus matching T1/T2 abnormalities as the injury evolves (see Figs. 2, 6 and 11).\textsuperscript{4,189} Other important contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus) [see Fig. 2], fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus.\textsuperscript{4} It is well known that many of these may also be associated with restricted diffusion along with other nontraumatic processes (encephalitis, seizures, and metabolic disorders).\textsuperscript{4,154,186,187,189} Again, a differential diagnosis is required.

**SUMMARY**

An extensive review of the literature to date fails to establish an evidence base for reliably distinguishing NAI from AI or from the medical mimics. The medical and imaging findings alone cannot diagnose intentional injury. Only a child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathologic data. The duty of a radiologist is to give a detailed description of the imaging findings, provide a differential diagnosis, and communicate the concern for NAI, directly to the primary care team in a timely manner. Radiologists should be prepared to consult with child protection services; other medical and surgical consultants, including a pathologist or biomechanical specialist; law enforcement investigators; and attorneys for all parties as appropriate. Radiologists must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse. These should be properly evaluated, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered. Furthermore, a negative medical evaluation does not make NAI the default diagnosis. A timely and thorough multidisciplinary evaluation may be the difference between appropriate child protection versus an improper breakup of a family or a wrongful indictment and conviction.

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Barnes


