Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics

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Abstract: Because of the widely acknowledged controversy in nonaccidental injury, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine. Children with suspected nonaccidental injury versus accidental injury must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate pattern of injury and timing issues and to consider the mimics of abuse. All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only central nervous system injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.

Key Words: child abuse, computed tomography, magnetic resonance imaging, nonaccidental injury, nonaccidental trauma

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Traumatic central nervous system (CNS) injury is reportedly the leading cause of childhood morbidity and mortality in the United States, resulting in about 100,000 emergencies annually and half the deaths from infancy through puberty.[1–5] The major causes are accidental injuries (AIs) and include falls, vehicular accidents, and recreational mishaps. However, nonaccidental, inflicted, or intentional trauma is said to be a frequent cause, with peak incidence at the age of about 6 months and accounting for about 80% of the deaths from traumatic brain injury in children younger than 2 years. Nonaccidental injury (NAI)—or nonaccidental trauma (NAT)—is the more recent terminology applied to the traditional labels *child abuse*, *battered child syndrome*, and *shaken baby syndrome* (SBS).[4,5] A modern restatement of the definition of SBS is that it represents a form of physical NAI 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 and commonly accompanied by other apparently inflicted injuries.[6] The short-term life-threatening presentations and long-term outcomes have become a major concern in health care, dating back to the original reports of Kempe,[7] Caffey,[8] and Silverman.[9] Later reports on the incidence rate of CNS trauma in alleged NAI estimate a range of 7% to 19%.[4,5]

However, a number of reports from multiple disciplines have challenged the evidence base (ie, quality of evidence [QOE] analysis) for NAI/SBS as the cause in all cases of the triad.[4,5,10] Such reports indicate that the triad may also be observed in AI (including those associated with short falls, lucid interval, and rehemorrhage) and in nontraumatic or medical conditions. These are the "mimics" of NAI that often present as acute life-threatening events (ALTE). This includes hypoxia-ischemia (eg, apnea, choking, respiratory or cardiac arrest), ischemic injury (arterial vs venous occlusive disease), seizures, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, and metabolic or connective tissue disorders. Many cases seem multifactorial and involve a combination or sequence of contributing events or conditions.[4,5,10] For example, an infant is dropped and experiences a head impact with delayed seizure, choking spell, or apnea, and then undergoes a series of prolonged or difficult resuscitations, including problematic airway intubation with subsequent hypoxic-ischemic brain injury. Another example is a young child with ongoing infectious illness, fluid-electrolyte imbalance, and coagulopathy, and then experiences seizures, respiratory arrest, and resuscitation with hypoxic-ischemic injury.

Often, the imaging findings are neither characteristic of nor specific for NAI. Because of the widely acknowledged controversy in NAI, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine (EBM).[4,5,10] Children with suspected NAI versus AI must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate the pattern of injury and timing issues and to consider the mimics of abuse.[4,5,10] All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only CNS injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.[4,5,10]

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MECHANISMS AND MANIFESTATIONS OF TRAUMATIC CNS INJURY

The spectrum of CNS injury associated with trauma (AI or NAI) has been classified into primary versus secondary, focal versus diffuse, and acute versus chronic categories.[4,5,10,11] The primary injury is immediate, irreversible, and is the direct result of the initial traumatic force (eg, contusion, shear injury). Secondary injury denotes the reactive phenomena that arise from or are associated with the primary injury (eg, swelling, hypoxia-ischemia, herniation). Direct contact or impact phenomena produce localized cranial distortion or deformation and thus produce *focal* injury (eg, fracture [Fx], contusion, epidural hematoma [EDH]). Accidental injury is said to be typically associated with this mechanism and result (Fig. 1). Although reported also in cases of NAI, it has been stated that impact injury, with the exception of EDH, is usually not life threatening.

It is *indirect* trauma (ie, independent of skull deformation) that has been considered responsible for the most severe CNS injury in SBS/NAI.[4,5,10–13] Inertial loading accompanying sudden angular acceleration/deceleration of the head on the neck (as with shaking) produces shear strain deformation and disruption at tissue interfaces, therefore *diffusing* the injury (Fig. 2). The young infant is said to be particularly vulnerable because of weak neck muscles, a relatively large head, and an immature brain. It is the shaking mechanism that is traditionally postulated to result in the triad, including primary traumatic injury (ie, SDH, RH, and DAI), with or without the secondary injury pattern (ie, edema, swelling, hypoxia-ischemia, herniation). Reportedly, such patterns are associated with the most severe and fatal CNS injuries and are readily demonstrated by means of neuroimaging, surgical neuropathology, and postmortem neuropathology.[4,5,10–13]

On a medical forensic basis, it is further stipulated that (1) retinal hemorrhages of a particular pattern are diagnostic of SBS/NAI, (2) such CNS injury on an accidental basis can only be associated with a massive force equivalent to a motor vehicle accident or a fall from a 2-story building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with previous head injury is caused by newly inflicted injury and not just a *rebleed*. Using this reasoning, the last caretaker is automatically guilty of abusive injury, especially if not witnessed by an independent observer.[4,5,10–13]

The range of acute primary and secondary CNS injury reported to occur with NAI significantly overlaps that of AI.[4,5,10,11] This includes multiple or complex cranial fractures, acute interhemispheric SDH (Fig. 2), acute-hyperacute convexity SDH, multiple contusions, shear injury (DAI, white matter tears), brain swelling, edema, and hypoxia-ischemia (Fig. 2). The range of chronic CNS injury includes chronic SDH, communicating hydrocephalus, atrophy, or encephalomalacia. The combination of acute and chronic findings suggests more than 1 traumatic event. Imaging evidence of CNS injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally *higher-specificity* imaging findings associated with violent shaking (eg, metaphyseal, rib, or other typical skeletal injuries).[4,5,10] Therefore, clinical and imaging findings of injury disproportional to the history, and injuries of differing age, have become 2 of the key diagnostic criteria indicating the *probability* of NAI/SBS, particularly when encountered in the premobile, young infant.[4,5,10] Such clinical and imaging findings have traditionally formed the basis from which health professionals, including radiologists, have provided a medical diagnosis and offered expert testimony that such *forensic* findings are *proof* of NAI/SBS.[10]

CONTROVERSY

Fundamental difficulties persist in formulating a *medical* diagnosis or *forensic* determination of NAI/SBS on the basis of a causative event (ie, shaking) that is inferred from clinical, radiological, and/or pathological findings in the often *subjective* context of (1) an unwitnessed event, (2) a *noncredible* history, or (3) an admission or confession.[4,5,10] This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS, and that the vast body of literature on child abuse is comprised of

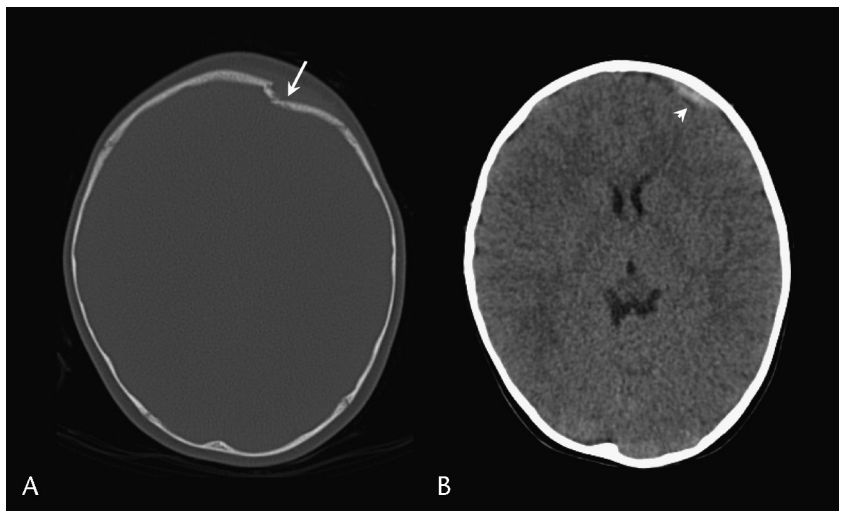

FIGURE 1. Images obtained from a 22-month-old female motor vehicle accident victim with depressed left-side frontal skull fracture (A, arrow), overlying scalp swelling, and a small, high-density epidural hematoma (B, arrowhead).

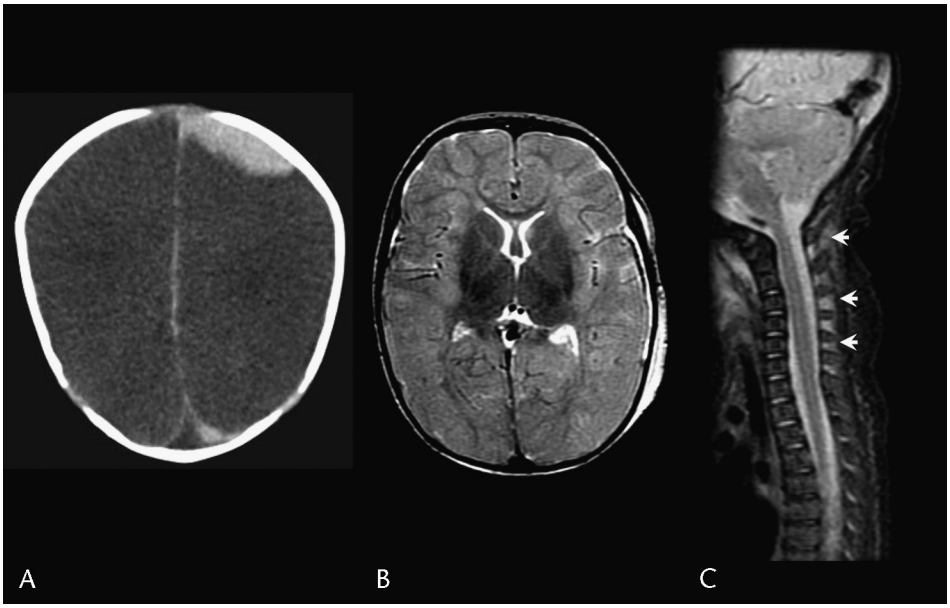

FIGURE 2. Images obtained from a 25-day-old female neonate with history of drop and RH (alleged NAI). A, Axial CT image shows high-density left-side frontal SDH (surgically drained before MRI), bilateral cerebral low densities with decreased gray-white matter differentiation (hypoxia-ischemia?), and interhemispheric high-density hemorrhage. B, Axial T2 MRI scan shows bilateral cerebral cortical and subcortical T2 high intensities plus interhemispheric T2 low intensities. C, Sagittal STIR cervical spine MRI scan shows posterior ligamentous high intensities (arrows) but no definite cord injury (NAI? SCIWORA?).

anecdotal case series, case reports, reviews, opinions, and position papers.[10,14] Furthermore, many reports include cases having impact injury that not only raises doubt regarding the *shaking-only* mechanism but also questions that this injury is always NAI based on a *shaken-impact* mechanism. From the perspective of EBM, QOE ratings for SBS/NAI diagnostic criteria reveal that few published reports merit a rating above class IV (ie, any design where test is not applied in blinded evaluation, or evidence provided by expert opinion alone or in a descriptive case series without controls).[10,14] The inclusion criteria provided in many reports often seem arbitrary, such as *suspected abuse*, *presumed abuse*, *likely abuse*, and *indeterminate*.[15,16] Furthermore, the diagnostic criteria often seem to follow *circular logic* (ie, SBS = SDH + RH [inclusion criteria], therefore SDH + RH = SBS [conclusion]). Such low QOE ratings hardly earn a EBM diagnostic recommendation level of *optional*, much less as a *guideline* or a *standard*.[10,14] This has traditionally been true of the neuroimaging literature, the clinical literature that uses neuroimaging, and the forensic pathology literature.[10,17–44]

The most widely reported attempt of a scientific study to test NAI/SBS used a biomechanical approach, measured stresses from shaking versus impact in a doll model, and correlated those stresses with injury thresholds in subhuman primate experiments established in another study.[45–47] Only stresses associated with impact, whether using an unpadded or padded surface, exceeded the injury thresholds that correlated with the pathological spectrum of concussion, SDH, and DAI. The authors concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. These results obviously contradicted many of the original reports that had relied on the "whiplash" mechanism as causative of the triad.[47–49] These authors also concluded that fatal cases of SBS/NAI, unless occurring in children with predisposing factors (eg, subdural hygroma [SDHG], atrophy, etc), are not likely to result from shaking during play, feeding, and swinging, or from more vigorous shaking by a caretaker for discipline. A number of subsequent studies using various biomechanical, animal, and computer models have failed to convincingly invalidate this study, although many contend that there is no adequate model yet designed to properly test shaking versus impact.[50–61] Some of these reports also indicate that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant neck, cervical spinal column, or cervical spinal cord injury (Fig. 2).[53,54]

A number of past and more recent reports raise serious doubt that abuse is the cause in all cases of infant CNS injury using traditional SBS/NAI diagnostic criteria.[10,14,16,46,49,62–68] This includes reports of skull fracture or acute SDH from accidental simple falls in young infants, such as those associated with wide extracerebral spaces (eg, benign external hydrocephalus, benign extracerebral collections of infancy, SDHGs),[69–83] and fatal pediatric head injuries caused by witnessed, accidental short-distance falls, including those with a lucid interval and RH.[84–102] Recent neuropathologic studies in alleged SBS cases indicate that (1) the cerebral swelling in young infants is more often caused by diffuse axonal injury of hypoxic-ischemic origin rather than traumatic origin (traumatic origin is more appropriately termed *multifocal traumatic axonal or shear injury*); (2) although Fx, SDH (eg, interhemispheric), and RH are commonly present, the usual cause of death was increased intracranial pressure from brain swelling associated with hypoxia-ischemia; and (3) cervical EDH and focal axonal brain stem, cervical cord, and spinal nerve root injuries were characteristically observed in these infants (presumably caused by shaking, although most had impact findings).[103–109] Such upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the hypoxic-ischemic brain injury. Additional neuropathologic series have shown that dural hemorrhages are also observed in nontraumatic fetal, neonatal, and infant cases, and that the common denominator is likely a combination of cerebral

venous hypertension and congestion, arterial hypertension, brain swelling, and immaturity with vascular fragility further compromised by hypoxia-ischemia or infection.[107–109] Reports of neurosurgical, neuroradiological, and neuropathologic findings in head trauma, as correlated with biomechanical analyses, indicate that SDH and RH occur with rotational deceleration injuries, whether *accidental* (eg, axis or center of rotation internal to the skull, including those resulting from short-distance falls) or *nonaccidental* (ie, axis of rotation external to the skull [eg, at the craniocervical junction or cervical spinal level]).[50–53] There is no scientific basis to date to indicate how much or how little force is necessary to produce traumatic injury to the developing CNS.

Furthermore, the specificity of RH for child abuse and its dating has also been questioned.[4,5,10,16,49,67,68,73,84,110–113] Such hemorrhages have been reported with a variety of conditions, including AT, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage (SAH), sepsis, coagulopathy, certain metabolic disorders, systemic hypertension, and other conditions. Furthermore, many cases of RH (and SDH) are confounded by the existence of multiple factors or conditions that often have a synergistic influence on the type and the extent of RH. For example, consider the child who has trauma, hypoxia-ischemia, coagulopathy, and has undergone resuscitation.

IMAGING PROTOCOLS

Proper imaging evaluation includes not only computed tomography (CT) and a radiographic or radionuclide skeletal survey but also magnetic resonance imaging (MRI) and, in some cases, serial imaging.[4,10,114–118] Occasionally, ultrasonography (US) may be useful. The imaging protocols should be designed to evaluate not only NAI versus AI but also the nontraumatic mimics. Computed tomography is the primary modality in acute neurological presentations because of its access, speed (particularly using multidetector technology), and ability to demonstrate abnormalities requiring immediate neurosurgical or medical intervention (eg, an expanding hematoma, brain swelling, impending herniation) (Figs. 1, 2).[4,10,114] Nonenhanced head CT with soft tissue and bone algorithms is performed. Facial and spinal (eg, cervical) CT may also be needed, including reformatting. Three-dimensional computed tomographic reconstructions can be important to evaluate fractures versus developmental variants (eg, accessory sutures, fissures, synchondroses). Computed tomographic angiography (CTA) or computed tomographic venography (CTV) may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation, aneurysm) or infarction (eg, dissection, venous thrombosis). Intravenous contrast-enhanced CT or US with Doppler may be used to separate subarachnoid and subdural compartments by identifying bridging veins within the subarachnoid space; however, MRI is usually needed for more definite evaluation. In addition, in the unstable infant, initial and repeat cranial US (eg, transcranial Doppler) at the bedside may assist in evaluating structural abnormalities and monitoring alterations in cerebral blood flow and intracranial pressure.

Magnetic resonance imaging should be conducted as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.[4,10,114–118] Brain MRI should include 3 planes and at least T1, T2, fluid-attenuated inversion recovery (FLAIR), gradient-recalled echo (GRE) T2*, and diffusion imaging (diffusion-weighted imaging [DWI]/apparent diffusion coefficient [ADC]) (Fig. 3). Gadolinium-enhanced T1 images should probably be used along with MRA and magnetic resonance venography (MRV).

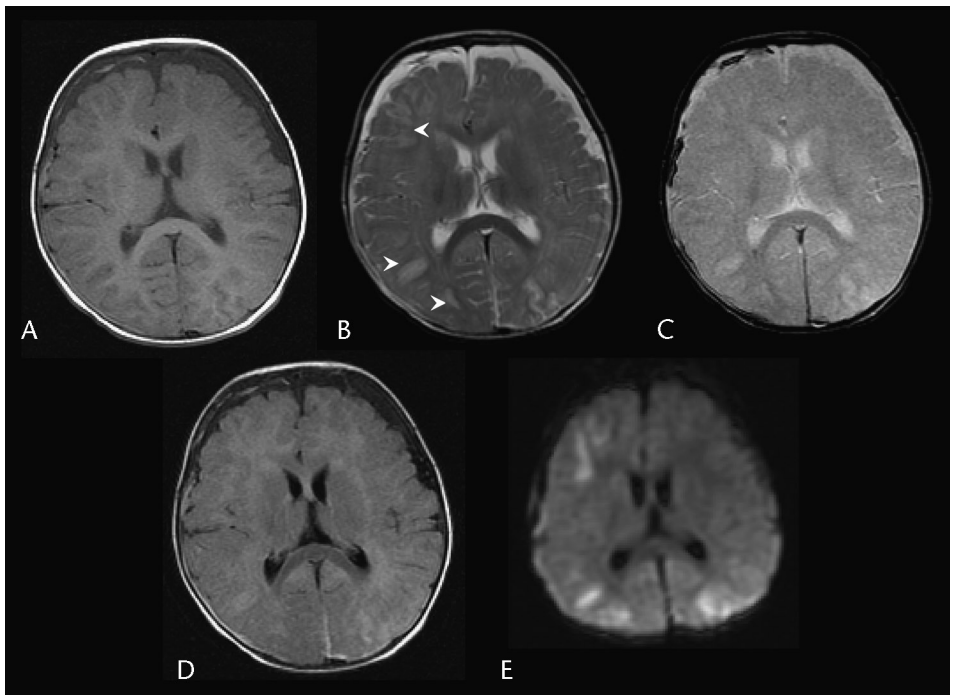

FIGURE 3. Images obtained from an 8-month-old male infant after viral illness, right-side humeral fracture, and RH (alleged NAI). Axial T1 (A), T2 (B), GRE (C), FLAIR (D), and DWI (E) images show bilateral frontal extracerebral CSF-intensity collections with right-side frontal extracerebral hemorrhage that is T1/FLAIR hyperintense and T2/GRE hypointense. Also seen are multifocal cerebral T2/FLAIR hyperintensities (arrowheads) that are DWI hyperintense (shear vs infarction?).

TABLE 1. Magnetic Resonance Imaging of Intracranial Hemorrhage and Thrombosis*

Stage	Biochemical Form	Site	T1 MRI	T2 MRI
Hyperacute (+ edema) (<24 hours)	Fe II oxyHb	Intact RBCs	Iso-low I	High I
Acute (+ edema) (1–3 days)	Fe II deoxyHb	Intact RBCs	Iso-low I	Low I
Early subacute (+ edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (− edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (− edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-low I	Low I

*Modified from Wolpert and Barnes,[119] Kleinman and Barnes,[4] Bradley,[120] and Zuerrer et al.[121]
RBCs indicates red blood cells; I, intensity; plus sign (+), present; minus sign (−), absent; Hb, hemoglobin; Fe II, ferrous; Fe III, Ferric; Iso, isointense.

The cervical spine should also be imaged, along with other levels when indicated, and especially by using short TI inversion recovery (STIR) (Fig. 2). T1- and T2-weighted imaging techniques are necessary for characterizing the nature and timing (whether hyperacute, acute, subacute, or chronic) of hemorrhages and other collections by using established criteria (Table 1). Gradient-recalled echo or other susceptibility-weighted (T2*) techniques is most sensitive for detecting hemorrhage or thromboses that are often not identified on other sequences. However, GRE cannot be used for timing alone because it shows most hemorrhages (new and old) as hypointense (eg, deoxyhemoglobin, intracellular methemoglobin, hemosiderin).[4,10,114] The FLAIR sequence suppresses cerebrospinal fluid (CSF) intensity and allows for a better assessment of brain abnormalities, especially when adjacent to a CSF space or collection. FLAIR is also sensitive (but nonspecific) for subarachnoid space abnormalities, which appear as high intensity (eg, hemorrhage, exudate, inflammatory or neoplastic leptomeningeal infiltration, occlusive vascular slow flow, and hyperoxygenation during sedation or anesthesia). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia. Magnetic resonance spectroscopy (MRS) may show a lactate peak. It must be remembered, however, that restricted or reduced diffusion may be observed in other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.[4,10,114] Gadolinium-enhanced sequences and MRS can be used to evaluate these other processes. In addition, MRA and MRV are important to evaluate arterial occlusive disease (eg, dissection) or venous thrombosis. The source images should be viewed along with the reprojected images. In some cases of partial occlusion/thrombosis, the abnormality may be more conspicuous on CTA/CTV, especially in infants. For evaluating arterial dissection by means of MRI, an axial fat-suppressed T1 sequence from the aortic arch to the circle of Willis may detect T1-hyperintense hemorrhage or thrombosis (ie, methemoglobin) within the false lumen, especially if the process is in the subacute phase.

INJURY EVALUATION

The range of CNS injury in childhood trauma, whether AI or NAI, often demonstrated by imaging may be categorized according to being primary or secondary (as previously described) and according to specific anatomical involvement, including scalp, cranial, intracranial, vascular, spinal, and head and neck.[2,4,5,10] A thorough analysis of the injury requires a systematic breakdown into injury components for both pattern of injury and timing parameters.

SCALP INJURY

Scalp injuries include hemorrhage, edema, or laceration and may be localized to any layer (SCALP [*s*kin, sub*c*utaneous,

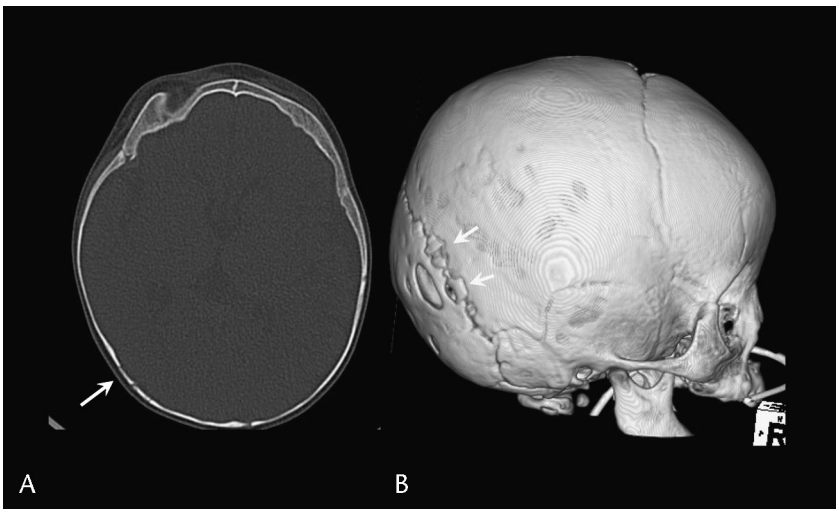

FIGURE 4. Images obtained from a 10-month-old male infant with intrasutural (wormian) bones versus fractures. A, CT image shows right-side parietal cranial defects (arrow). B, Three-dimensional computed tomographic surface reconstruction confirms intrasutural bones (arrows).

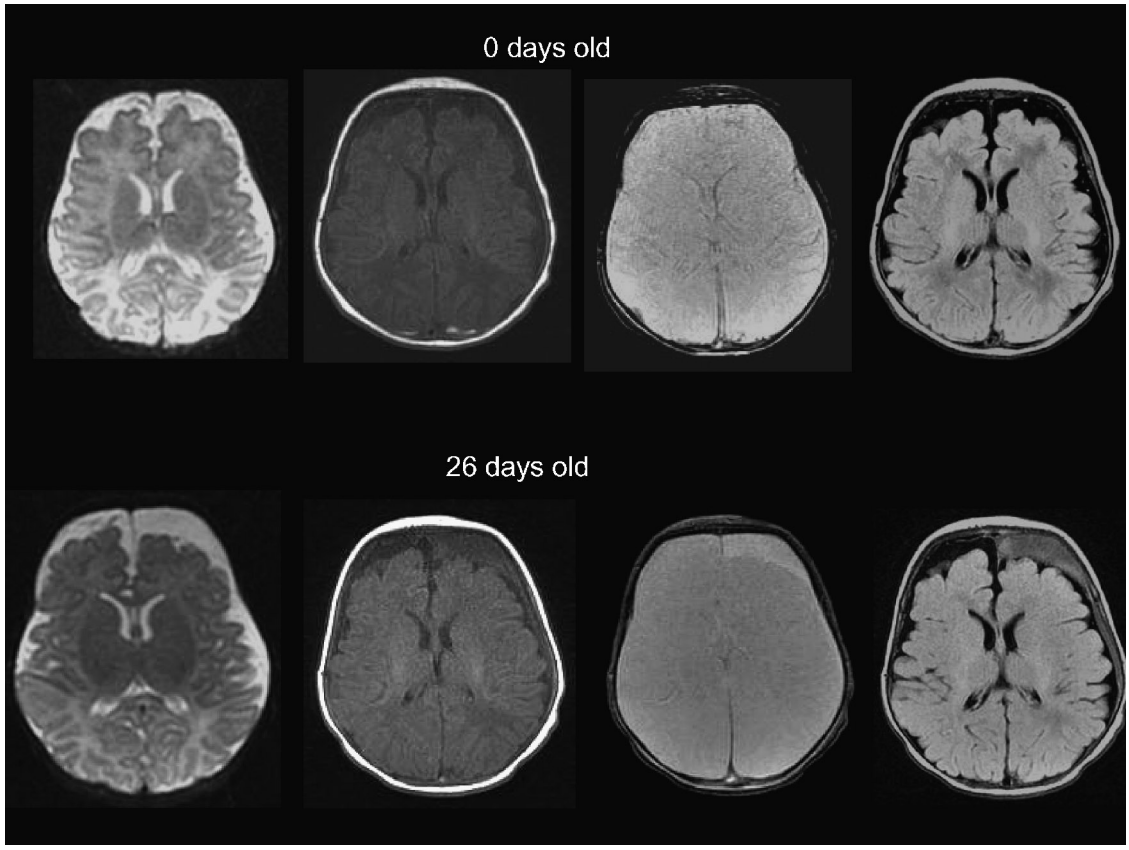

FIGURE 5. Images obtained from an infant with benign extracerebral collections of infancy and spontaneous subdural hemorrhage. Axial T2, T1, GRE, and FLAIR images (left to right) show CSF-intensity frontal subarachnoid collections at birth (top row). At 26 days postnatal age (bottom row), superimposed subdural collections that don't conform to CSF signal are present (courtesy of Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu HI).

galea *a*poneurotica, *l*oose or subga*l*eal space, *p*eriosteum]).[2,4,5,10] Although CT or MRI may not precisely resolve scalp layers, the site of a collection may be inferred by means of morphological findings (Fig. 1). Subperiosteal collections (eg, cephalohematoma) are usually confined by the sutures. Subcutaneous or subgaleal collections are not as contained, may be more extensive, and can contribute to circulatory compromise. Scalp injuries are difficult to precisely time on imaging studies, unless serial examinations are available; in addition, timing depends on the nature and the number of traumatic events or other factors (eg, circulatory compromise). Unless there is direct vascular injury that results in an acute hematoma, collections or edema may not be identified on early imaging. Scalp injuries may become evident several hours later or on the next day. Nonvisualization of scalp or skull abnormalities on imaging should not be interpreted as absence of impact injury.

SKULL INJURY

The spectrum of cranial injury includes Fx*s* and suture splitting.[2,4,5,10] Fractures may be simple (eg, single, linear, nondisplaced) or complex (eg, bilateral, multiple, diastatic, depressed, or growing [ie, leptomeningeal cyst]). Localized suture splitting may indicate traumatic diastasis where widening occurs as a part of Fx extension. Diffuse or multiple suture widening may indicate increased intracranial pressure from any cause to include edema, expanding collection, or hydrocephalus. Evaluating the skull in neonates, infants, and young children is challenging because Fx may not be distinguished from sutures, synchondroses, or their normal variations. This is particularly difficult in the parietooccipital region and skull base where accessory sutures, fissures, and synchondroses are common. The significance of this distinction is important because the reporting of a skull Fx is *evidence* of trauma (Fig. 1). In such cases, 3-dimensional computed tomography with surface reconstructions may provide clarification (Fig. 4). In general, the morphology of an Fx does not differentiate NAI from AI. Complex or bilateral skull Fx in this age group can arise from a single event under circumstances other than a 2-story fall or a motor vehicle accident. Such examples include a fall or a drop with impact to the skull vertex, impact against more than 1 surface (eg, table, wall, or floor), fall or drop downstairs, and an adult or older child falling with or onto a smaller child. Skull Fx*s* are also difficult to time by using plain films and CT because of the lack of periosteal reaction during healing. A simple skull Fx in an infant may require 6 months for complete healing. In an older child and adult, this may take up to a

year.[2,4,5,10] Intracranial air densities (ie, pneumocephalus) may be related to fracture involving the paranasal sinuses or otomastoid structures, caused by penetrating trauma (eg, open skull fracture), arise from CSF access (eg, lumbar puncture) or vascular access (eg, indwelling catheter), or may be associated with gas-forming infections.

EXTRACEREBRAL COLLECTIONS

The range of intracranial injury includes abnormal fluid collections and brain injury.[2,4,5,10] Abnormal collections may be subarachnoid, intraventricular, subdural, or epidural. These may contain hemorrhage of any age (eg, hyperacute, acute, subacute, chronic, combined), cerebrospinal fluid (CSF [eg, hygroma, hydrocephalus]), protein, exudate, or any combination of elements. On imaging, it may be impossible to specifically define the components or age of a collection (eg, SDHG vs chronic SDH). Subarachnoid and subdural collections may be localized or extensive and occur near the convexities, interhemispheric (along the falx), and along the tentorium. Epidural hemorrhage, whether arterial or venous in origin, tends to be more localized (limited by the periosteal layer of the dura mater along the inner calvarial table) and can cross midline (Fig. 1). Epidural (intradural) hemorrhage may split the leaves of dura and collect within the tentorium or falx. Epidural collections usually appear lentiform. Subdural collections tend to be crescentic and follow the contour of the adjacent cerebrum or cerebellum (Fig. 3). Subarachnoid collections may be less well defined (unless loculated) and extend into cisterns, fissures, or sulci. Occasionally, a collection cannot be determined to be specifically subarachnoid, subdural, or epidural because collections in multiple spaces may be present, owing to membrane layer disruption (Fig. 2). Intraventricular hemorrhage is a rare but reported finding in trauma. It may also be an indicator of associated hypoxia-ischemia, coagulopathy, or venous thrombosis.

Prominent subarachnoid CSF spaces may normally be present in infants (aka benign extracerebral collections [BECC], benign extracerebral subarachnoid spaces, benign external hydrocephalus).[10,79–83,114] These should be of the same density/intensity as CSF on CT and MRI (Fig. 5). This condition predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (Fig. 5). A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Cases of rapid resolution and redistribution of acute SDH for a few hours to 1 to 2 days have been reported.[117,122] A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space. An SDH may also redistribute within the subdural space as a gravity-dependent process (eg, a convexity SDH migrating to the peritentorial and posterior interhemispheric regions)[114,117] (Fig. 6). Subdural hemorrhage migration may lead to misinterpretation of a new hemorrhage. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may

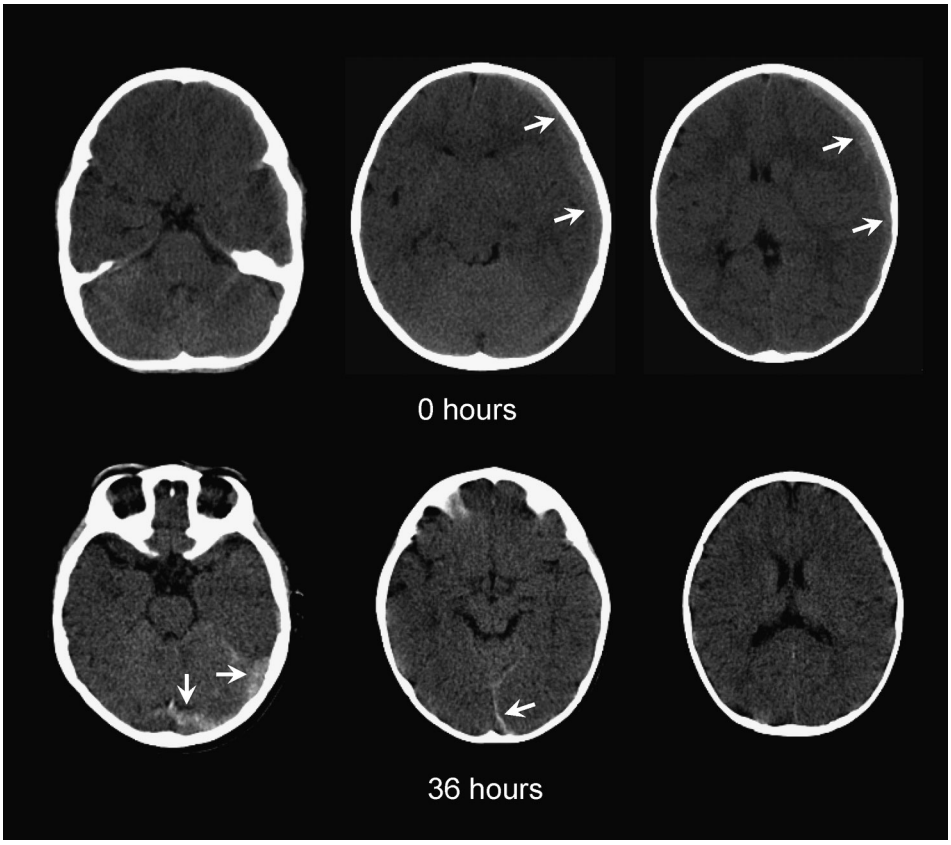

FIGURE 6. Images obtained from a 9-month-old female infant who had accidental trauma from left-side frontal impact. Computed tomographic images at presentation (top row) show left-side frontotemporal-convexity high-density subdural hemorrhage (arrows). Computed tomographic images obtained after 36 hours in the hospital (bottom row) show redistribution of the high-density hemorrhage to the peritentorial region and posterior interhemispheric fissure (arrows).

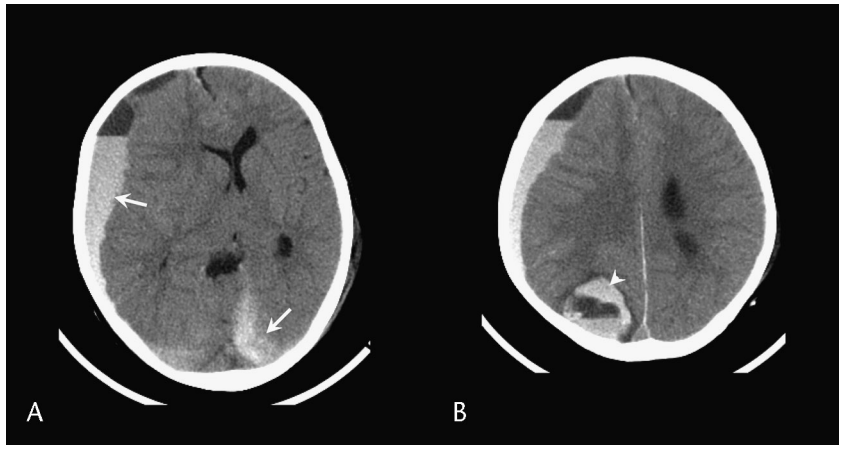

FIGURE 7. Images obtained from a 2-year-old boy with congenital heart disease and ECMO. Axial computed tomographic images show bilateral subdural hematomas (A, arrows) and right-side parietal intracerebral hematoma (B, arrowhead) with low-density over high-density fluid levels.

cause further confusion because the density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location (Figs. 7, 8).[117] In addition, more recent reports further substantiate that (1) the interhemispheric SDH may be observed in AI and, therefore, is not specific for NAI; (2) mixed-density SDH also occurs in AI; (3) SDH may occur in BECC either spontaneously or as a result of minor trauma (ie, AI); and (4) rehemorrhage within SDH may occur spontaneously or with minor AI.[10,82,114–118]

BRAIN INJURY

Traumatic brain injury includes contusion, shear injury, hemorrhage, and edema.[2,4,5,10] Contusions represent focal or multifocal impact injury, are usually hemorrhagic, and typically occur in cortical gray matter along brain surfaces that impact skull bone or dura mater (eg, falx, tentorium). The inner table of the immature, infant skull is not as *rough* as in older children and adults. Therefore, sliding contusions of the frontal or temporal lobes along the floor of the anterior or middle cranial fossa, respectively, occur less often. Infant contusions more commonly occur at the primary site of impact (ie, coup injury) or at a secondary, "recoil" site opposite the primary impact (ie, contracoup injury). Shear injury (ie, traumatic axonal injury, white matter tear) is also focal or multifocal and typically occurs at deep gray matter–white matter junctions, along the corpus callosum, and within the brain stem (Fig. 3). They are more often nonhemorrhagic but may become hemorrhagic. In severe cases, shear injuries may appear as gross tears. This type of injury has been previously referred to as *diffuse axonal injury* or DAI. It is more properly termed *multifocal or traumatic axonal injury* because diffuse axonal injury is more characteristic of hypoxic-ischemic injury (Fig. 2).[104–109]

Edema or swelling may be traumatic, hyperemic, hypoxic-ischemic, or related to other factors (eg, seizures, metabolic).[2,4,5,10] Traumatic edema is related to direct traumatic effects such as contusion, shear, or the result of a vascular injury (eg, dissection, herniation) (Figs. 2, 3). Malignant brain edema, a term used for severe cerebral swelling leading to rapid deterioration, may also occur in children with head trauma. The edema may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon. There may be rapid or delayed onset.[84–96] Predisposing factors are not well established but likely include a genetic basis. Global hypoxia (eg, apnea, respiratory failure) or ischemia (eg, cardiovascular failure or dissection) is likely a major cause of or contributor to brain edema in the child with head trauma (Fig. 2). Other contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus), fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus. The type (eg, cytotoxic, vasogenic, hydrostatic) and pattern of edema tend to conform to the nature and distribution of the causative insult. Traumatic edema is often focal or multifocal (eg, in areas of contusion, shear, or hemorrhage) (Fig. 3). Hyperemic edema is often diffuse and may appear early as accentuated gray-white matter differentiation on CT, then progressing to loss of differentiation (Fig. 2). Hypoxic-ischemic injury, depending on its severity and duration, may have a diffuse appearance acutely with decreased gray-white matter differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MRI (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (Fig. 2).[10,114,123–126] The subacute to chronic sequelae of traumatic brain injury include hydrocephalus, atrophy, encephalomalacia, gliosis, mineralization, and chronic extracerebral collections.

VASCULAR INJURY

Arterial trauma may result in dissection or pseudoaneurysm.[2,4,5,10,123, 127] The vascular injury may be the result of penetrating or nonpenetrating trauma, may be spontaneous, or caused by existing disease (eg, arteriopathy). Internal carotid artery dissection typically involves the cervical or supraclinoid segments. Vertebrobasilar dissection most commonly involves the distal cervical portion of the vertebral artery at the C1-C2 level. Intracranial or multiple dissections may rarely occur. Dissection may result in stenotic, thrombotic, or embolic infarction. Pseudoaneurysms may be associated with hemorrhage. The vascular injury may be initially detected by means of CT and CTA (Fig. 9) or of MRI (eg, DWI, axial fat-suppressed T1 sections of the neck and skull base) with MRA. Catheter

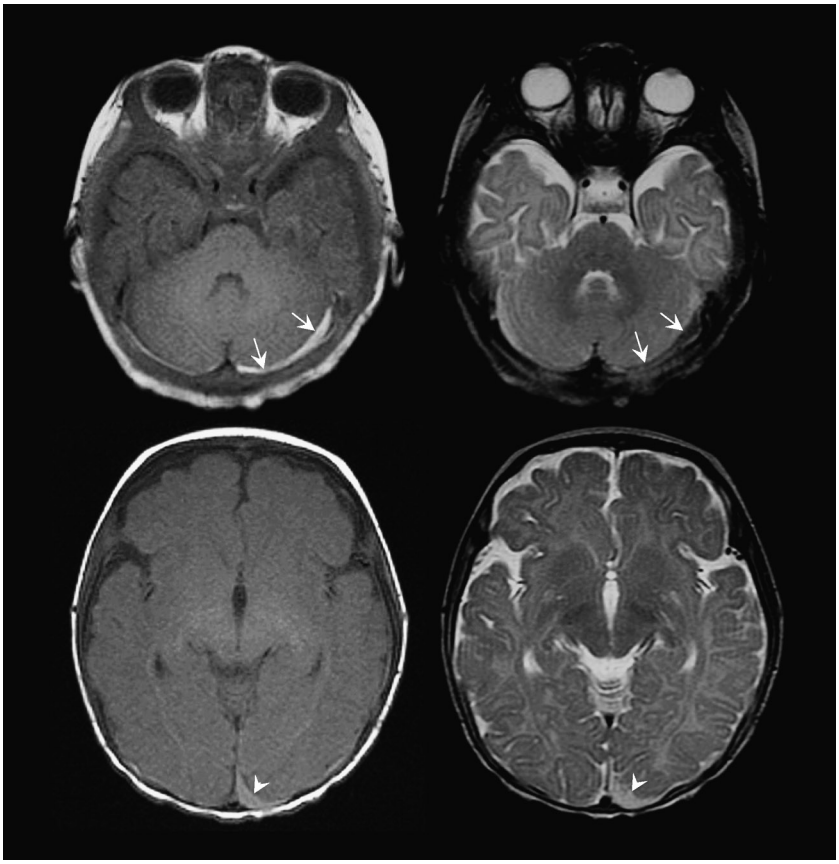

FIGURE 8. Images obtained from a 2-month-old female infant with left-side peritentorial and posterior interhemispheric subdural hemorrhage. Axial MRI images show T1-hyperintense and T2-hypointense sediment along the tentorium (top row, arrows) with T1- and T2-isohyperintense supernatant above (bottom row, arrowheads).

angiography may be necessary for definitive evaluation. Arterial occlusive infarction also occurs with the various types of herniation, in which relatively specific distributions are observed. Dural sinus and venous thrombosis may also occur with trauma (eg, adjacent to fracture, associated or predisposing coagulopathy) or as a mimic of NAI (eg, infection, coagulopathy).[128] Computed tomography may show hyperdensity within the venous system, a focal venous enlargement with associated subarachnoid or subdural hemorrhage, or infarction that is often hemorrhagic. A more definitive diagnosis may be made by means of CTV or of MRI and MRV.

SPINAL INJURY

The spectrum of spinal injury in NAI significantly overlaps that of AI.[2,4,5,10,123] This spectrum differs with age (degree of spinal development) and includes either single or

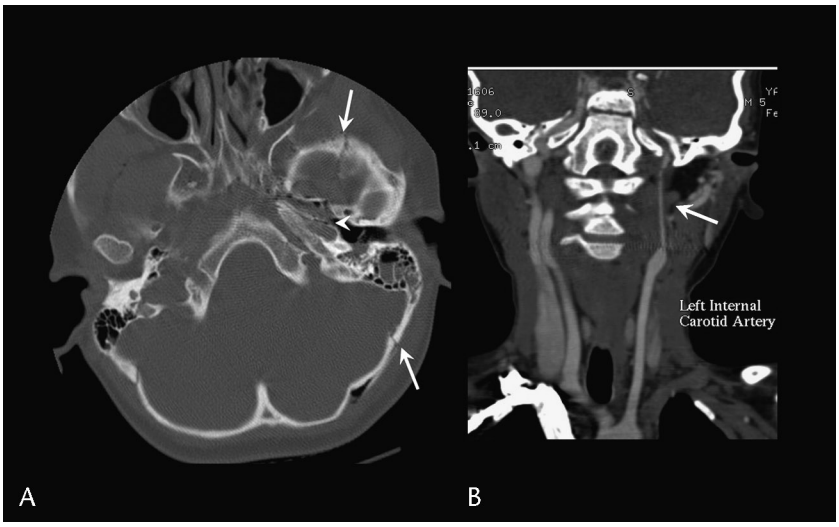

FIGURE 9. Images obtained from a 5-year-old boy. A, Computed tomographic image shows left-side skull base fractures involving left-side occiput, petrous bone, and sphenoid wing (arrows). Air densities are seen within the carotid canal (arrowhead). B, Computed tomography angiogram shows left-side cervical internal carotid arterial dissection with marked luminal narrowing (arrow).

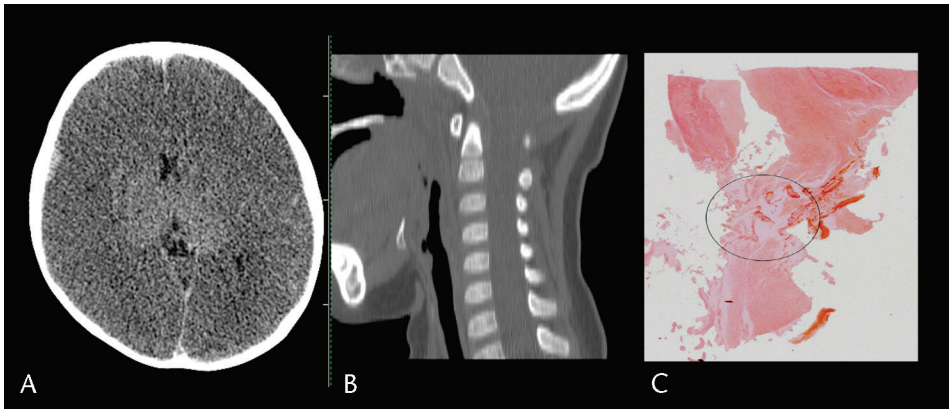

FIGURE 10. Images obtained from a 22-month-old boy with SCIWORA (caused by backward fall and parietal head impact) and hypoxic-ischemic injury and RHs. A, Axial brain CT image shows (1) bilateral cerebral low densities with decreased gray-white matter differentiation (edema) and (2) small high-density asymmetrical cerebral, extracerebral, and posterior interhemispheric hemorrhages. B, Sagittal reformatted cervical spinal computed tomographic image shows no spinal column abnormality (MRI not performed). C, Postmortem midsagittal section shows cervicomedullary disruption (circle). Diffuse hypoxic-ischemic axonal brain injury was also confirmed.

multiple lesions involving the cervical, thoracic, lumbar, or sacral level. The mechanisms of injury include hyperflexion, hyperextension, axial loading or rotation, and distraction. The range of spinal column and paraspinal injury includes vertebral or neural arch fractures, bony fragment or disk displacement, dislocations, instability, and paraspinal ligamentous, muscular, or vascular injury. Such injuries may not be apparent on plain films (eg, spinal cord injury without radiographic abnormality [SCIWORA]) and require additional CT plus MRI for complete evaluation.[129–131] Magnetic resonance imaging is particularly important for evaluating ligamentous injury and intraspinal injury. The range of intraspinal injury includes displaced bone or disk fragments and hematomas (eg, epidural) with spinal cord or nerve root compression. There may be edema, contusion, hemorrhage, transection of the spinal cord, or avulsion of 1 or more nerve roots. Computed tomographic angiography or MRA may be needed to evaluate vascular injury (eg, dissection). Cervical spinal cord injury may be associated with head injury or may be the unsuspected cause of respiratory failure and hypoxic-ischemic brain injury (eg, SCIWORA) (Fig. 10).[129–131] This should be evaluated by means of MRI in all such cases, whether AI or NAI. In addition, one must be aware of predisposing conditions that may result in major neurological deficits associated with *minor* head and neck trauma mechanisms (eg, craniocervical anomaly with instability Fig. 11; Chiari I malformation Fig. 12).

IMAGING ANALYSIS—COMPUTED TOMOGRAPHY

Regarding the initial computed tomographic examination, the findings are often nonspecific with regard to pattern of injury and timing and require a differential diagnosis (DDX). To properly analyze such a case from an imaging perspective, each injury component must be addressed separately, and then collectively, and then correlated with clinical and other data.[4,10,114] The major findings are often (1) extracerebral and cerebral high densities, (2) extracerebral isohypodensities, (3) cerebral low densities, with or without (4) scalp or skull abnormalities. In general, the DDX may include trauma (AI vs NAI), hypoxia-ischemia, ischemic injury (arterial vs venous occlusive disease), seizure edema, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, metabolic or connective tissue disorder, and multifactorial.

Extracerebral high densities are often seen posteriorly along the tentorium, falx, interhemispheric fissure, and dural

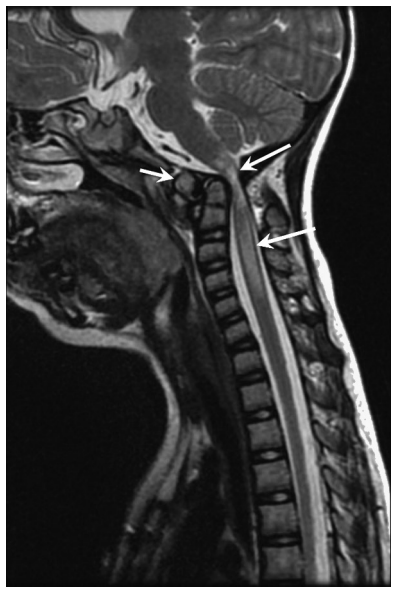

FIGURE 11. Image obtained from an 8-year-old girl with Down syndrome and minor trauma with quadriparesis. Sagittal T2 MRI scan shows hypoplastic dens, os odontoideum (anterior arrow), and anterior atlantoaxial instability (confirmed by means of CT) with cervicomedullary compression and high-intensity edema (posterior arrows).

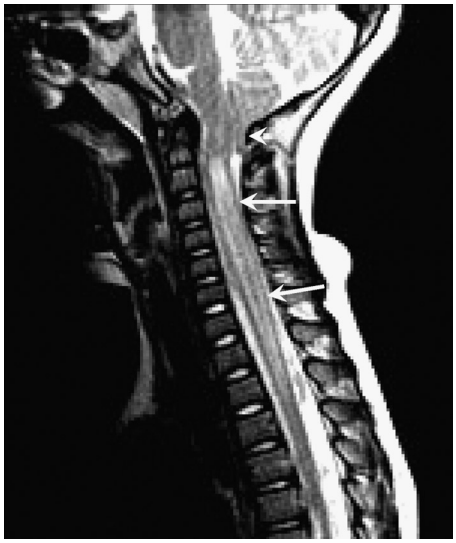

FIGURE 12. Image obtained from a 3-year-old boy with Chiari I malformation, minor trauma, and subsequent quadriparesis. Sagittal T2 MRI scan shows cerebellar tonsils extending into the upper cervical canal (upper arrowhead) and diffuse high-intensity edema of the cervical spinal cord (lower arrows). No abnormality was present on plain films or CT (SCIWORA).

venous sinuses that may vary in laterality and symmetry (Figs. 2, 6, 7, 10, 13–16). These and other extracerebral high densities may be laminar, linear, nodular, or punctate. Using published criteria and timing parameters (discussed in the succeeding sections), these represent either acute to subacute hemorrhages (subarachnoid, subdural) or thromboses (eg, venous).[4,10,114–118] For apparent intracerebral high densities, it may be difficult to differentiate cerebral from SAHs (including those within the perivascular spaces) from vascular thromboses (eg, cortical, subependymal, or medullary venous thromboses). Computed tomography may not be able to distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction (Figs. 13, 16, 18). Extracerebral isohypodensities may represent subarachnoid spaces (eg, BECC), SDHG, hyperacute SDH, or chronic SDH (Figs. 14, 17). According to the literature, the timing for any of the mentioned findings is as follows: (1) hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 3 hours to 7 to 10 days (Figs. 1, 2, 6, 7, 10, 13–18), (2) hemorrhage that is isohypodense on CT (ie, nonclotted) may be hyperacute (timing, <3 hours) or chronic (timing, >10 days) (Figs. 14, 17), (3) the low density may also represent preexisting wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF containing) that may be acute or chronic (Figs. 14, 17), (4) blood levels are unusual in the subacute unless there is coagulopathy (Fig. 7), (5) CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (Fig. 17), and (6) the interhemispheric SDH is no longer considered characteristic of NAI (Figs. 2, 6, 7, 13–16).[4,10,114–118]

Cerebral low densities may vary in bilaterality and symmetry and be associated with decreased gray-white matter differentiation or mass effect (Figs. 2, 10, 17). In general, this indicates edema/swelling, the timing of which depends on causation. If related to trauma, such edema/swelling may represent primary injury or secondary injury and be acute-hyperacute (eg, timing of few hours) or delayed (eg, timing of several hours to a few days), including association with lucid interval and short falls.[4,10,114,123–126] Bilateral diffuse edema is most commonly observed in hypoxia-ischemia but may also be observed in other diffuse processes (eg, fluid-electrolyte imbalance, status epilepticus, encephalitis, etc). Focal or multifocal edema may be observed in contusion (eg, gray matter), shear (eg, white matter), infarction (gray or white matter), encephalitis, or demyelination (eg, acute disseminated encephalomyelitis).

Cranial defects may represent Fx, and their timing range is very broad (eg, hours to months old) (Fig. 1).[4,10,114] Furthermore, Fx morphology (eg, multiple, growing) does not reliably distinguish accidental from nonaccidental causation. Scalp collections (hemorrhage, edema, blood level) are also nonspecific with regard to causation and timing (Fig. 1).[4,10,114] If caused by trauma, the timing range is also rather broad (eg, hours to days old). Sutural widening may indicate diastatic Fx

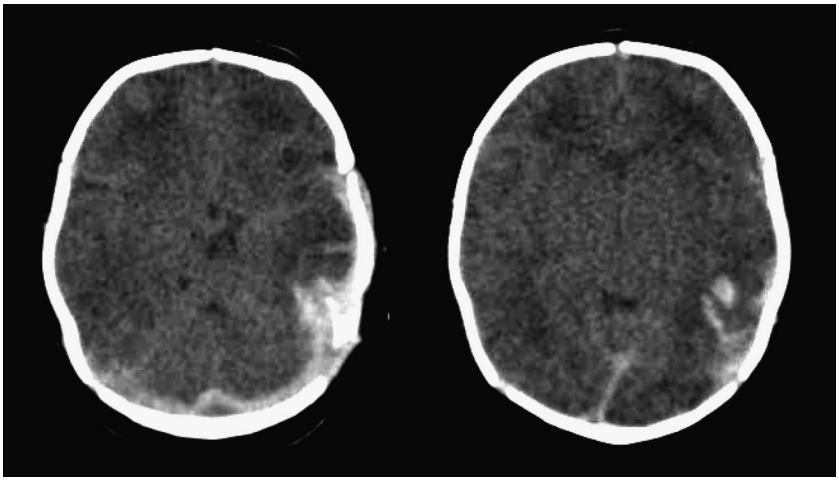

FIGURE 13. Images obtained from a 1-day-old female infant delivered by means of spontaneous vaginal delivery and with subsequent apneic episodes. Computed tomography demonstrates left-side temporal cerebral and extracerebral high-density hemorrhage (or thromboses); high-density hemorrhage is also demonstrated along the interhemispheric fissure, tentorium, and dural venous sinuses. The results of coagulopathy test and sepsis workup were negative (final diagnosis, birth trauma?).

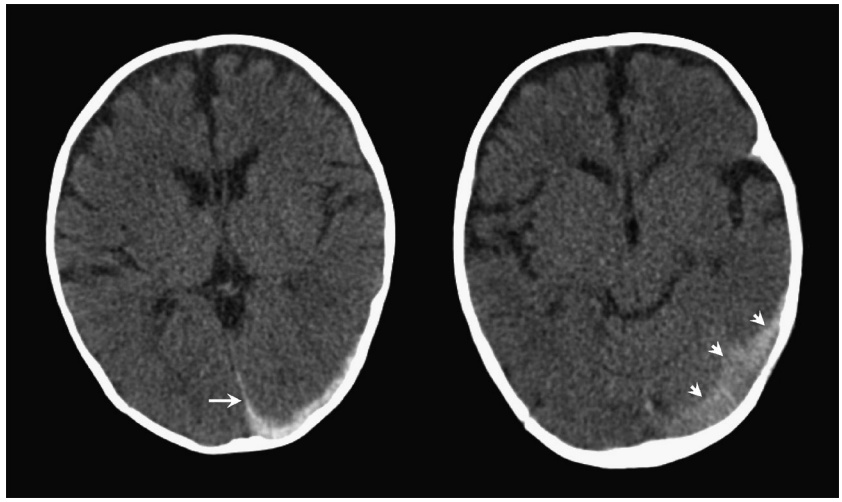

FIGURE 14. Images obtained from a 4-month-old male infant with 2-week viral illness who progressed to septic shock (*Staphylococcus aureus*), endocarditis, severe mitral regurgitation, and coagulopathy. Noncontrast axial CT images show high-density extracerebral hemorrhages (and/or thromboses) along the left-side tentorium, dural venous sinuses, falx, and interhemispheric fissure (arrows). In this case, the bifrontal low-density extracerebral spaces likely represent slightly prominent infantile subarachnoid spaces (BECC?) or underdevelopment, rather than chronic SDH or subdural hygroma.

or increased intracranial pressure. Accessory sutures or synchondroses and developmental fissures may mimic Fx. Intrasutural bones (eg, wormian) may be associated with a skeletal dysplasia or metabolic disorder (Fig. 4).

Subsequent or follow-up computed tomographic examinations may show surgical changes (eg, postevacuation, ventricular catheter, pressure-monitoring device), evolving, redistributing, or recurrent/new hemorrhages, and evolving cerebral densities (edema/swelling). Subsequent CT examinations during the weeks or months may show evolution to permanent cerebral tissue loss (ie, atrophy, encephalomalacia).

IMAGE ANALYSIS—MAGNETIC RESONANCE IMAGING

On an imaging basis, only MRI may provide more precise information regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses, and (2) brain injury. The MRI should be performed as soon as feasible, and the findings be compared with the findings from the earlier CT. As a result, MRI has become the standard for such evaluation in these matters.[4,10,114–117,121,123–126]

Hemorrhages and Thromboses

Using published MRI guidelines (Table 1), in general, the evolutionary timing for hemorrhages or thromboses (eg, venous) are as follows: (1) hyperacute phase (timing, <12 hours): T1 isohypointense, T2 hyperintense; (2) acute phase (timing, 1–3 days): T1 isohypointense, T2 hypointense; (3) early subacute phase (timing, 3–7 days): T1 hyperintense, T2 hypointense; (4) late subacute phase (timing, 7–14 days): T1 hyperintense, T2 hyperintense; (5) early chronic phase (timing, >14 days): T1 hyperintense, T2 hyperintense; (6) late chronic phase (timing, >1 to 3 months): T1 isohypointense, T2 hypointense.[4,10,114–117,121,123–124] Mixed intensity collections are problematic regarding timing. Matching the MRI findings with the computed tomographic findings may help, along with follow-up MRI. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. In addition, a single MRI may not reliably differentiate T1-hypointense/T2-hyperintense collections as representing CSF collections (eg, BECC, acute SDHG) versus hyperacute SDH versus chronic collections (SDH, SDHG). Gradient-recalled echo hypointensities are iron sensitive but do not assist with timing unless matched with

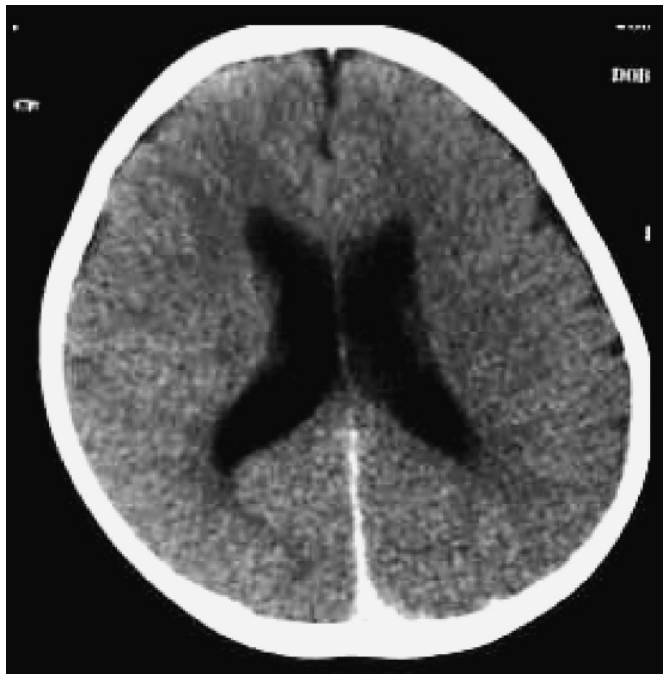

FIGURE 15. Image obtained from a 23-month-old girl who had recent viral gastrointestinal illness, ALTE, RHs, then brain death. Computed tomographic image shows posterior interhemispheric high densities at the level of portions of the inferior sagittal, straight, and superior sagittal sinuses, plus poor cerebral gray-white matter differentiation and moderate ventriculomegaly. Autopsy showed extensive dural and cerebral venous sinus thrombosis with extensive hypoxic-ischemic diffuse axonal brain injury.

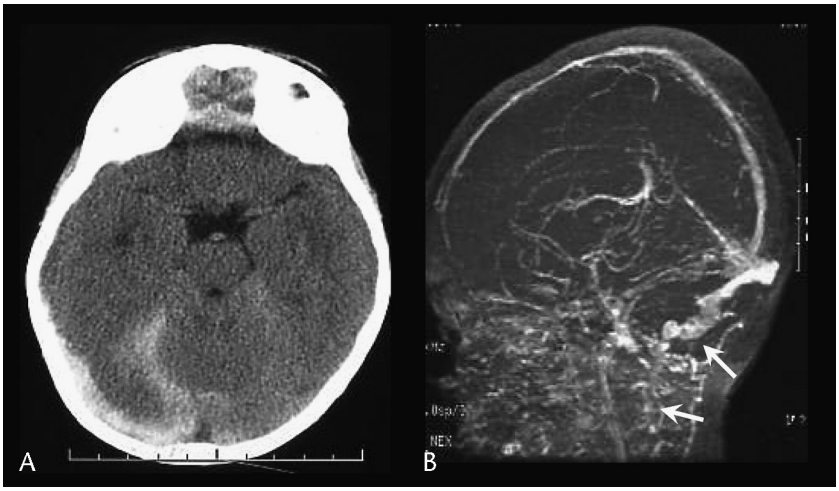

FIGURE 16. Images obtained from a 19-month-old boy who had 1 week of febrile illness (treated with antibiotics), followed by ALTE with RHs. A, Computed tomographic image shows high-density hemorrhages (or thromboses) along the right tentorium and dural venous sinuses. B, Magnetic resonance imaging with MRV shows irregular flow gaps with incomplete opacification of the right-side internal jugular vein and sigmoid sinus. Other flow gaps were demonstrated within the superior sagittal and straight sinuses, along with multiple venous collaterals (diagnosis, DVST).

T1, T2, and computed tomographic densities. Gradient-recalled echo and other magnetic susceptibility sequences are also sensitive to venous thromboses (eg, cortical, medullary, subependymal) that are not detected by means of MRV.

Brain Injury

With regard to brain injury, MRI may distinguish hypoxic-ischemic injury (diffuse relatively symmetrical DWI/ADC restricted diffusion with or without matching T1/T2 abnormalities) from shear and contusional injury (focal/multifocal restricted diffusion, GRE hypointensities, with T2/FLAIR edema). Shear and contusional injury, however, may not be reliably differentiated from focal/multifocal ischemic or hemorrhagic infarction (eg, dissection, vasculitis, venous, embolic) without supportive MRA, CTA, MRV, or angiography.[4,10,114,123–125] In addition, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed in encephalitis, seizures, and metabolic disorders. Using published MRI criteria and parameters,[114,123–126] in general, the evolutionary timing for ischemic injury is as follows: (1) hyperacute phase (timing, <1 day): DWI hyperintense, ADC hypointense; MRS result, lactate peak; (2) early acute phase (timing, 1–2 days): additional T2 hyperintensity; (3) late acute phase (timing, 2–4 days): additional T1 hyperintensity; (4) early subacute phase (timing, 6–7 days): additional T2 hypointensity; (5) late subacute phase (timing, 7–14 days): additional DWI isohypointense, ADC isohyperintense; (6) chronic phase (timing, >14 to 21 days): additional atrophy. If related to trauma, focal/multifocal ischemic findings may be caused by arterial injury (eg, dissection), venous injury (eg, tear, thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus). Hypoxia-ischemic brain injury caused by apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA), whether AI or NAI.[114,123,129–131] It may also occur with any nontraumatic cause (eg, choking, paroxysmal coughing, aspiration).[132] In addition to the diffuse brain injury, there may be associated subarachnoid and subdural hemorrhage without mass effect.[104–109]

CONDITIONS MIMICKING NONACCIDENTAL INJURY

Traumatic and nontraumatic conditions may mimic the clinical presentations (ie, the triad) and imaging findings of NAI. These include accidental trauma (as previously discussed), birth trauma, hypoxia-ischemia, cardiopulmonary

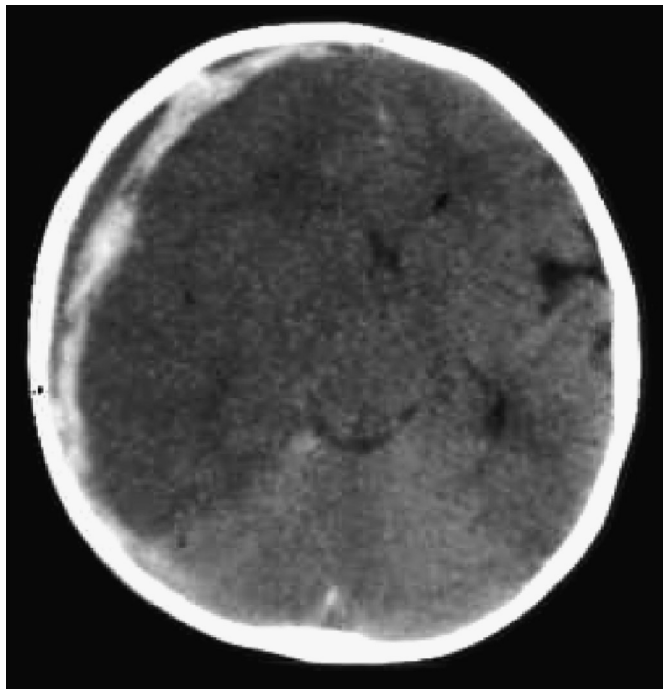

FIGURE 17. Image obtained from an 8-month-old male infant who had ALTE, right-side occipital skull fracture (not shown), a healing right-side distal radial fracture, and then had brain death. Computed tomographic image shows a right-side, mixed density extracerebral collection with right-side cerebral low density, mass effect, and leftward shift. High-density hemorrhages (or thromboses) are also present along the tentorium. There was disagreement among the forensic experts regarding hyperacute-acute SDH versus chronic SDH with rehemorrhage.

resuscitation, infectious or postinfectious conditions (eg, sepsis, meningoencephalitis, postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation (ECMO), and other conditions.[4,5,10,114,115,133] Regarding the pathogenesis of the triad (with and without other organ system involvement [eg, skeletal]), and whether caused by NAI, AI, or nontraumatic etiologies, the pathophysiology seems to be some combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or collagenopathy superimposed on the immature CNS and other systems.[107,115,123,132–146]

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. It is important to be aware of these mimics because a more extensive workup may be needed beyond the routine *screening* tests. In addition, the 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 records dating back to the pregnancy and birth, the postnatal pediatric records, the family history, the more recent history preceding the short-term presentation, the details of the short-term event itself, the resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings.[4,5,10,115,133]

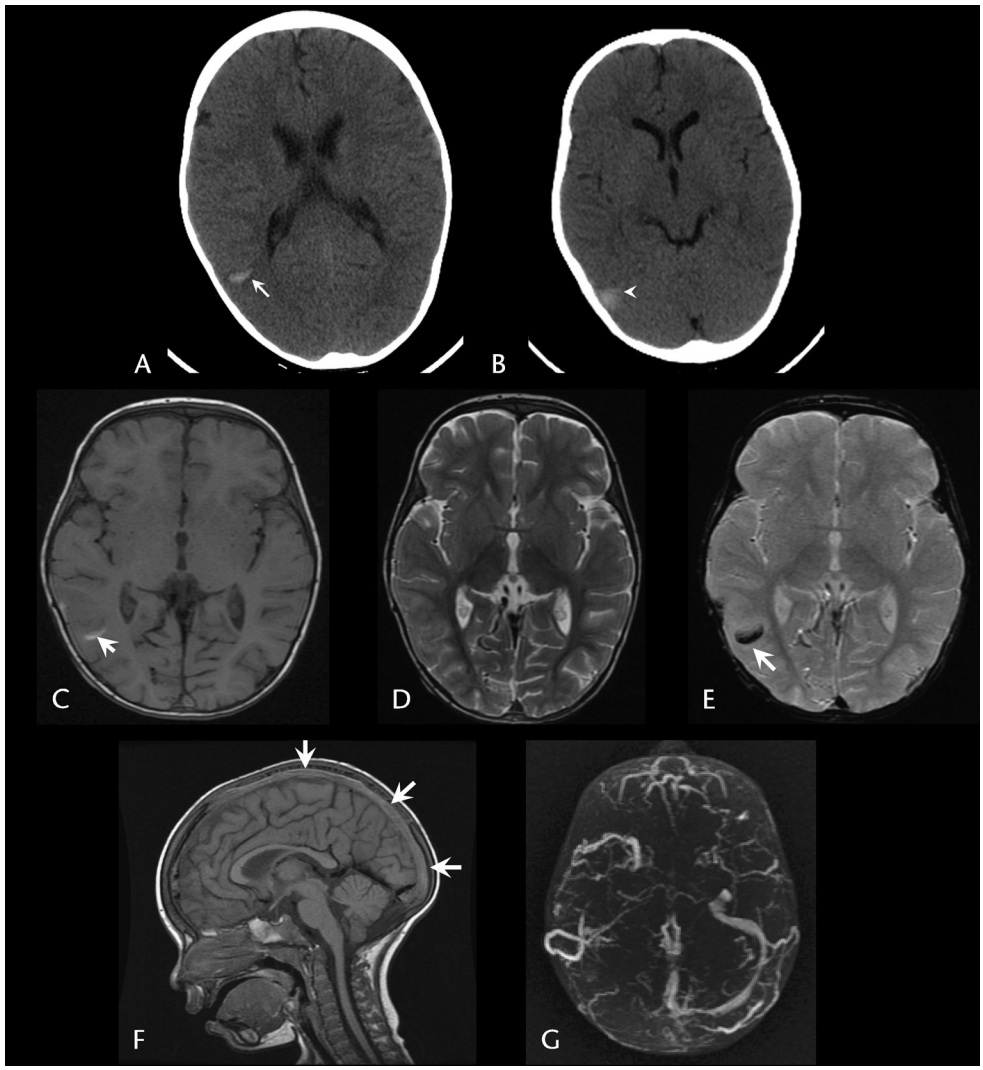

FIGURE 18. Images obtained from a 22-month-old boy who experienced lethargy, vomiting, and seizures after a viral illness, plus thrombocytopenia and iron deficiency anemia. A–B, Computed tomographic images show right-side posterior temporal and peritentorial high-density foci of hemorrhage or thrombosis (arrows). Axial T1 (C), T2 (D), and GRE (E) images show corresponding T1-hyperintense and GRE-hypointense foci with associated T2 hyperintensity (arrows). F, Sagittal T1 MRI scan shows hyperintensity along the superior sagittal sinus (arrows [thrombosis vs slow flow]). G, Axial MRV projection image shows nonvisualization of the superior sagittal, right-side transverse, and right-side sigmoid sinuses (diagnosis, postviral dural and cerebral venous thrombosis [extensive coagulopathy workup continues]).

A recent review presented by Sirotnak[133] extensively catalogues the many conditions that may mimic abusive head trauma. These include perinatal conditions (birth trauma and congenital malformations), accidental trauma, genetic and metabolic disorders, hematologic diseases and coagulopathies, infectious diseases, autoimmune and vasculitic conditions, oncological disease, toxins, poisons, nutritional deficiencies, and medical and surgical complications. The reader is encouraged to read this review.[133] An abbreviated discussion is presented in this article along with some examples.

Birth Trauma and Neonatal Conditions

Manifestations of birth trauma, including Fx, SDH, and RH, may persist beyond the neonatal period and mimic CNS findings of abuse.[145–151] Other examples are the cases of infants following ECMO therapy, at-risk preterm neonates, and infants with congenital heart disease.[4,5,10,123,124,152] When evaluating the condition of a young infant with apparent NAI, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues. This includes hemorrhage or rehemorrhage into collections existing at birth (Figs. 5, 8, 13).

Developmental Anomalies

Vascular malformations of the CNS in neonates and infants are relatively rare.[115,133,153,154] The most common are the vein of Galen malformations. Aneurysms are also rare in childhood but may arise within the circle of Willis. Aneurysms outside the circle are usually mycotic or traumatic in origin. Increased risk of aneurysm is associated with certain conditions, such as coarctation of the aorta, polycystic kidney disease, neurofibromatosis, and a family history positive for aneurysm. A number of syndromes in childhood are associated with vascular anomalies and may present with intracranial hemorrhage. These syndromes include, as examples, PHACE (*p*osterior fossa brain malformations, *h*emangiomas, *a*rterial anomalies, *c*oarctation of the aorta, *c*ardiac defects, and *e*ye abnormalities), Sturge-Weber, Beckwith-Wiedemann, Klippel-Trenaunay-Weber, Maffucci, and Olser-Weber-Rendu. Arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (Fig. 19).[133,155]

Genetic and Metabolic Disorders

A number of conditions in this category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I, Menkes kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others (Fig. 19).[115,133,135,136,156]

Hematologic Disease and Coagulopathy

Many conditions in this category predispose to intracranial hemorrhage and RH.[4,5,10,114,115,133,140–143,157] The bleeding or clotting disorder may be primary or

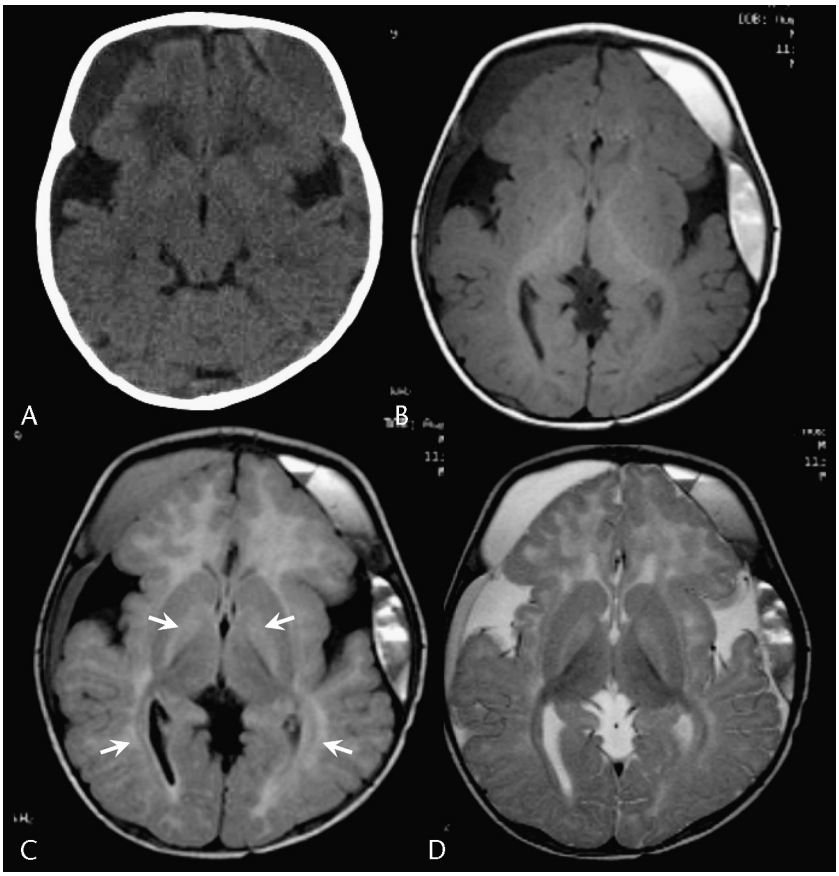

FIGURE 19. Images obtained from a 9-month-old male infant with glutaric aciduria type 1, SDHs, and RHs. CT (A), T1 (B), FLAIR (C), and T2 (D) MRI images show bilateral mixed-density and mixed-intensity extracerebral collections with fluid levels and septations, especially on the left side. Other characteristic findings for glutaric aciduria type 1 include bilaterally wide sylvian fissures (arachnoid cysts) plus abnormal basal ganglia (globus pallidus) and cerebral white matter intensities (arrows).

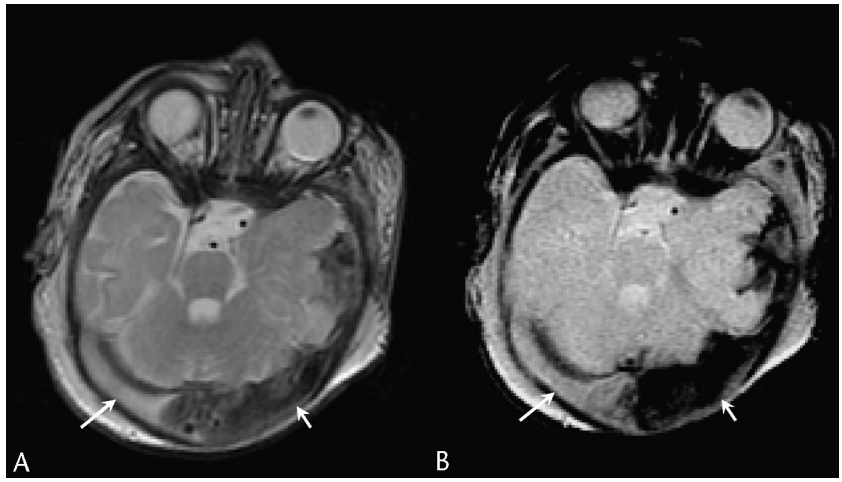

FIGURE 20. Images obtained from a 1-week-old male neonate with seizures, thrombocytopenia, antithrombin III deficiency, and ECMO for pulmonary hypertension. Axial T2 FSE (A) and GRE (B) MRI images show bilateral, mixed-intensity SDHs (arrows).

secondary (Figs. 7, 14–16, 18, 20, 21). In some cases, a more extensive workup beyond the usual *screening* tests will be needed, including a hematology consultation. Included in this category are the anemias, hemoglobinopathies (eg, sickle cell disease), hemorrhagic disease of the newborn (vitamin K deficiency Fig. 21), hemophilia A and B, factor V and XII deficiencies, von Willebrand disease, idiopathic thrombocytopenic purpura, disseminated intravascular coagulation and consumption coagulopathy associated with other conditions (eg, trauma, infection), liver disease, nephrotic syndrome, hemophagocytic lymphohistiocytosis, anticoagulant therapy, and others. Venous thrombosis may involve the dural venous sinuses (ie, dural venous sinus thrombosis [DVST]) and/or the cerebral veins (ie, cerebral vein thrombosis [CVT]) and be associated with primary or secondary hematologic or coagulopathic state.[10,123,124,133,158–161] Risk factors include acute systemic illness, dehydration (fluid-electrolyte imbalance), sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncological disease and therapy, head and neck infection, and hypercoagulable states. Seizure and/or neurological deficit are common, and hemorrhagic infarction is characteristic. Subarachnoid hemorrhage, SDH, or RH may also be observed, especially in infants (Figs. 15, 16, 18, 22). Relative high densities anywhere along the dural venous sinuses, tentorium, and falx (interhemispheric fissure and inferior sagittal sinus) may be seen on initial CT. Linear high densities may also be present along the distribution of the cortical ("cord sign"), subependymal, or medullary veins and give the impression of SAH, SDH, or intracerebral

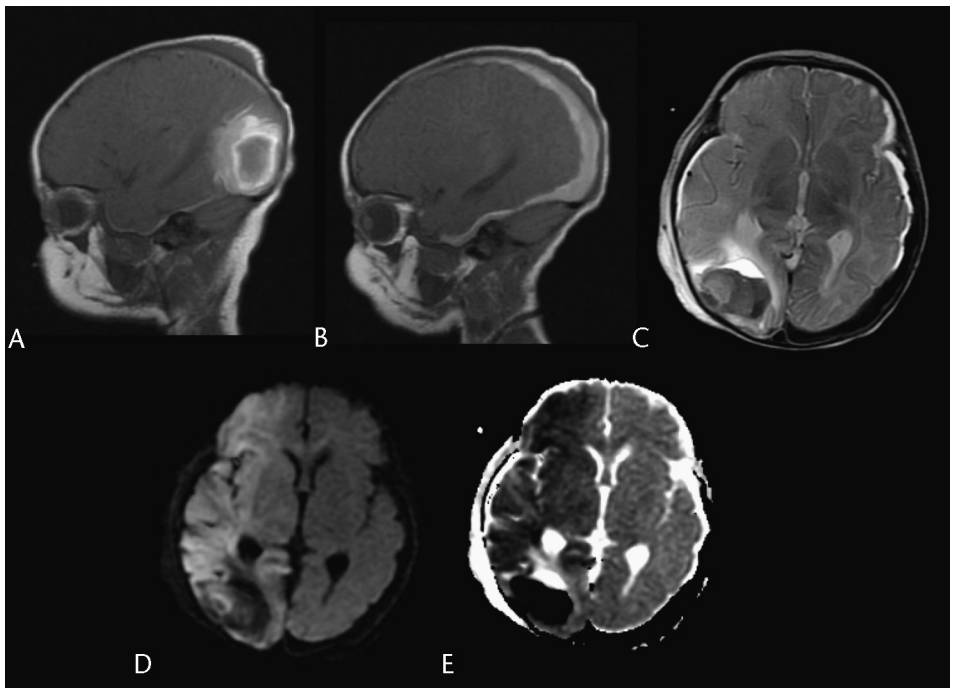

FIGURE 21. Images obtained from a 1-week-old male neonate who had seizures after delivery at home (no vitamin K administered). After surgical evacuation of large, right-side SDH, sagittal T1 (A, B), axial T2 (C), ADC (D), and DWI (E) images show bilateral mixed-intensity extracerebral and intracerebral hemorrhages and right-side cerebral hemispheric restricted diffusion (likely infarction) (diagnosis, hemorrhagic disease of the newborn [vitamin K deficiency]).

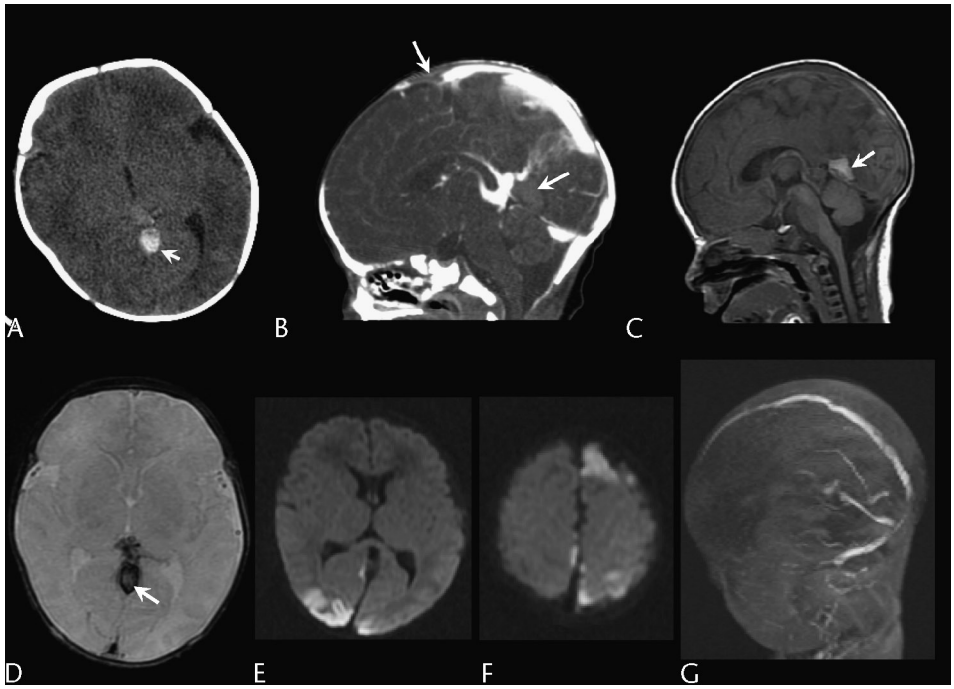

FIGURE 22. Images obtained from a 2-week-old male neonate with lethargy in emergency room (ER). Computed tomographic image (A) shows a focal midline hyperdensity at the level of the straight sinus (arrowhead). Sagittal CTV image (B) shows luminal masses along the straight and superior sagittal sinuses (arrows). Sagittal T1 (C) and axial GRE (D) images show the thrombus within the straight sinus (arrows). Axial DWI images (E–F) show restricted diffusion in multiple cortical areas (likely infarction vs suppuration). Magnetic resonance venography (G) is of poor diagnostic quality as compared with CTV (diagnosis, group B streptococcal meningitis with DVST).

hemorrhage. The "empty delta" sign may be seen within the superior sagittal sinus on contrast-enhanced CT. There may be multifocal infarctions (hemorrhagic or nonhemorrhagic) or intraventricular hemorrhage. With extensive dural venous sinus or cerebral venous thrombosis, there may be massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be associated with basal venous sinus thrombosis (eg, cavernous, petrosal, sphenoparietal). The thromboses and associated hemorrhages have variable MRI appearance depending on their age (see Image Analysis–Magnetic Resonance Imaging section and Table 1). Computed tomographic venography or MRV may readily detect DVST but not cerebral vein thrombosis, which may be suspected because of the characteristic distribution of hemorrhage or thromboses along venous structures, as demonstrated on susceptibility-weighted sequences (eg, GRE hypointensity). Depending on the clinical context, treatment may be directed only to the specific cause (eg, infection) or may also include anticoagulation or thrombolysis.

Infectious and Postinfectious Conditions

Meningitis, encephalitis, or sepsis (eg, bacterial, viral, granulomatous, parasitic) may involve vascular structures resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage (Figs. 3, 14–17, 22, 23). Subdural hemorrhage and RH may also be observed.

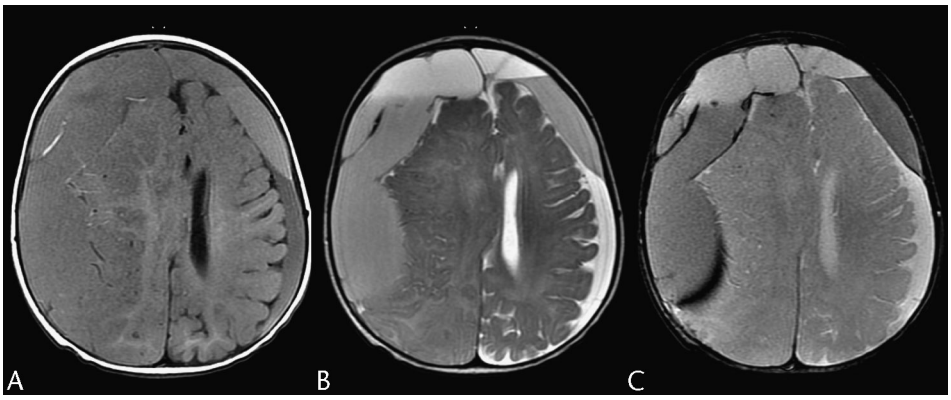

FIGURE 23. Images obtained from a 5-month-old male infant who had macrocephaly and seizures after having group D streptococcal (nonenterococcal) meningitis at the age of 3 days. Axial T1 (A), T2 (B), and GRE (C) images show bilaterally large and mixed-intensity extracerebral collections with septations and asymmetrical mass effect (likely chronic subdural effusions or hygromas with rehemorrhage).

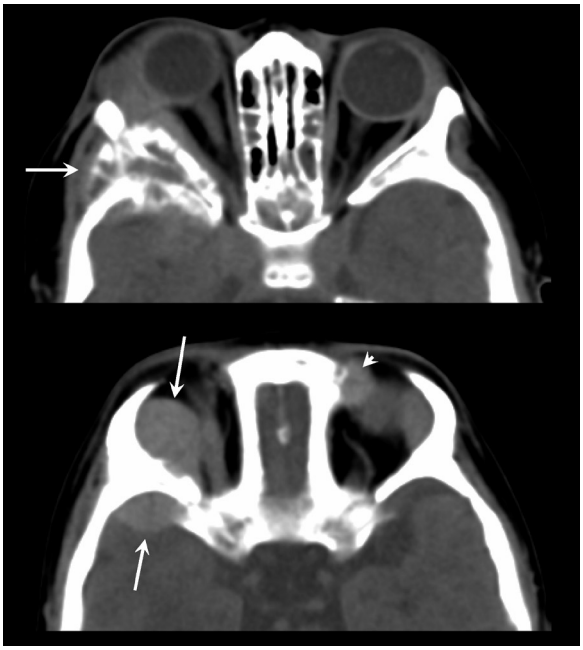

FIGURE 24. Images obtained from an 18-month-old girl with periorbital and facial ecchymoses in ER, evaluated for NAI. Computed tomographic image shows bilateral iso-high-density orbital soft tissue masses with bone destruction (arrows) and extension into the right-side middle cranial fossa (diagnosis, neuroblastoma).

Postinfectious illnesses (eg, postvaccinial) may also be associated with these findings.[139] Included in this category are the *encephalopathies of infancy and childhood* and *hemorrhagic shock and encephalopathy syndrome*.[115,133]

Autoimmune and Vasculitic Conditions

These include Kawasaki disease, systemic lupus erythematosis, moyamoya disease, Wegener granulomatosis, and Behçet syndrome.[115,133]

Oncological Disease

Hematologic malignancies, solid tumors of childhood, and their attendant therapies (including transplantation) are commonly associated with a variety of sequelae or complications that predispose to hemorrhage (eg, SDH and RH).[115,133] This includes vascular invasion by tumor, immunocompromise, infection, and coagulopathy. The clinical presentation and image findings may be mistaken for NAI (eg, leukemia, neuroblastoma) (Fig. 24).

Toxins, Poisons, and Nutritional Deficiencies

This category includes lead poisoning, cocaine, anticoagulants, and vitamin deficiencies (eg, vitamins K, C, D) (Figs. 21, 25). Preterm neonates and other chronically ill infants are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily affect bone development. Such infants may have skeletal imaging findings (eg, multiple healing fractures) that are misinterpreted as NAI, particularly if they present with AI that is complicated by SDH and RH (Fig. 25).[162–174]

Medical and Surgical Complications

This category includes (1) anticoagulant therapy or treatment-induced coagulopathy and (2) morbidity from medical or surgical interventions.[115,133]

CONCLUSIONS

In view of the currently available data, it is clear that we do not have an established EBM platform from which to

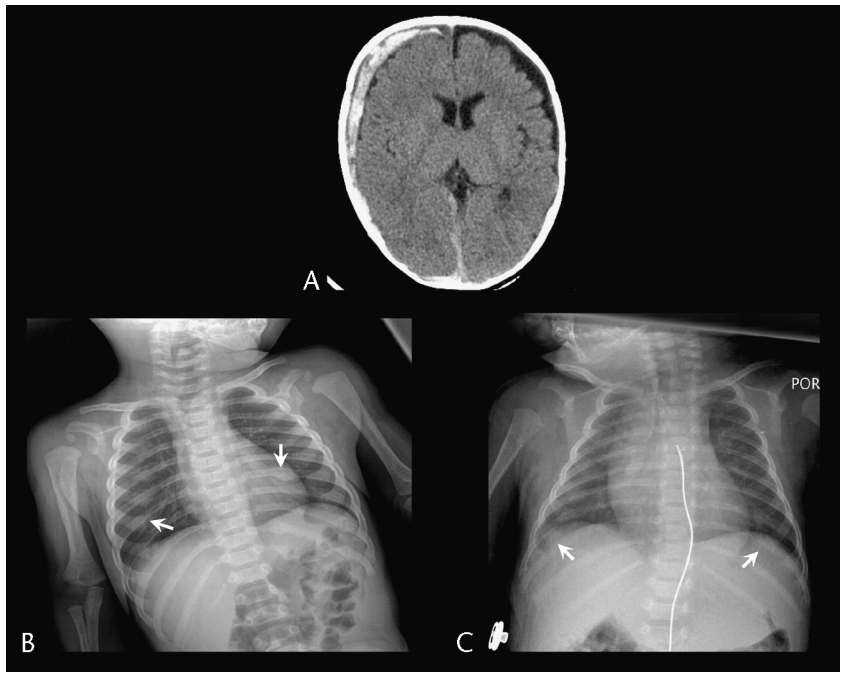

FIGURE 25. Images obtained from a 7-month-old male infant (25-week preterm birth) dropped with head impact to floor, RHs, evaluated in ER. Computed tomographic image (A) shows right-side mixed high-density extracerebral collection, left-side low-density extracerebral collection, posterior interhemispheric high-density hemorrhage, and right-side cerebral low-density edema. Chest radiograph in ER (B) shows bilateral anterior and posterior old, healing rib fractures. Comparison with earlier chest radiograph (C) at discharge from neonatal intensive care unit shows diffuse osteopenia and anterior rib flaring (arrows). Diagnosis: rickets of prematurity vs NAI?; AI with acute SDH superimposed on BECC vs NAI?

distinguish NAI from AI and, in some cases, traumatic from nontraumatic CNS injury. More reliable research is needed to establish a sound scientific foundation for CNS injury in NAI. The young infant is assumed more vulnerable to traumatic CNS injury, whether accidental or not, as compared with the older child or adult, and relies on the attention of caretakers for safety. However, as the infant becomes more mobile (rolling, crawling, walking, etc), the risk of AI (eg, from falls) increases. Furthermore, the interaction with older siblings or other children becomes a factor. The medical and imaging findings cannot diagnose intentional injury. Only the child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathological findings. Furthermore, biomechanical factors must be taken into consideration regarding the mechanism of trauma.

The radiologist should describe the imaging findings in detail, including the pattern, distribution, and severity of injury. A DDX is given, and timing ranges are provided if possible. If NAI is at issue, then the radiologist must directly communicate the imaging findings to the primary care team and be available to consult with child protection services and other medical or surgical consultants, including the pathologist or biomechanical specialist, law enforcement investigators, and attorneys for all parties, as appropriate.[1–5] The pattern of injury and the timing parameters, as may be provided by MRI, are particularly important with regard to correlation of events as reported by witnesses and potential suspects. The radiologist must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.[1–5] These should be properly ruled out, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered (eg, predisposing condition plus trauma). A timely and thorough multidisciplinary evaluation may be the difference between an appropriate child protection and an improper breakup of the family or a wrongful indictment and conviction.

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