Pearls and pitfalls of pediatric head trauma imaging

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Pediatric head trauma, whether it involves high-velocity impact, self-inflicted causes, or nonaccidental trauma, is a major cause of morbidity and mortality. It is estimated that the annual incidence of pediatric head trauma is on the order of 0.2% to 0.3% in the United States.[1,2] The radiologist's role on the trauma team is to promptly and accurately recognize and report the degree of injury. This not only allows clinicians to determine an appropriate strategy for treatment, but avoids delay in treatment that can result in significant morbidity and mortality.

Head trauma can be separated into two categories: primary brain injury, which occurs at the time of trauma, and secondary brain injury, occurring after the initial insult. Primary brain injury may be due to direct impact or indirect forces.[2,3] Examples of primary brain injury include skull fractures, subdural and epidural hematomas, hemorrhagic contusion, and diffuse axonal injury. Secondary brain injury is due to anoxia, as may be seen in the setting of birth trauma or drowning.[3] Nonaccidental trauma may take the form of primary and/or secondary brain injury. Obvious intracranial abnormalities are often picked up easily on imaging; however, it is important to be able to distinguish between true pediatric trauma and its mimics.

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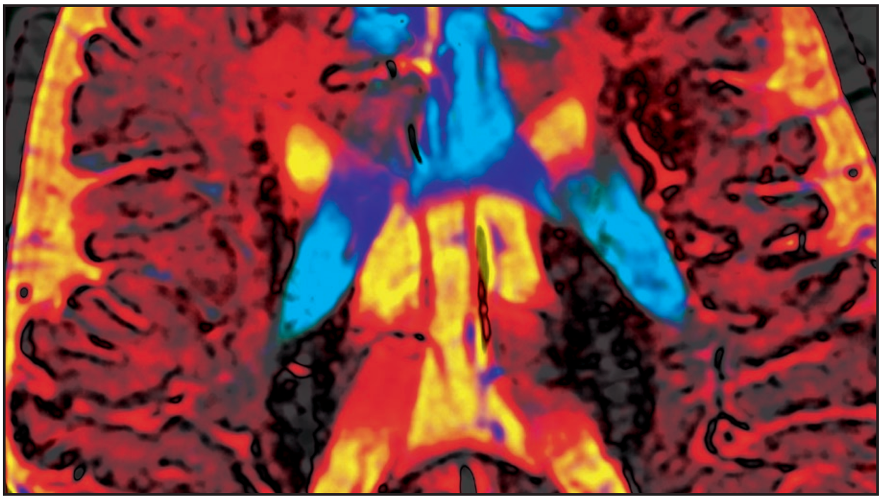

This article approaches pediatric head trauma in two parts; the first will compare and contrast the differences between true lesions and mimics, which may often represent normal variations in the pediatric age group and/or are considered "don't-touch" lesions. Finally, the authors will discuss lesions which should not be missed by any radiologist.

Calvarial fractures and associated mimics

Calvarial fractures are a common finding in both accidental and nonaccidental trauma and can have far-reaching effects in both clinical and social management.[4] However, the presence of sutures, accessory sutures, and wormian bones can confound their timely diagnosis. Fractures in the imaging plane can be extremely difficult to identify on conventional axial computed tomography (CT) slices, making a good case for always evaluating the CT scout images. Modern development of 3-dimensional reconstructions of CT data, which require minimal post-processing time and no additional radiation, have enhanced the ability of the radiologist to confidently diagnose and describe calvarial fractures, and accurately identify their mimics.[4]

Normal sutures are symmetric and found in expected locations. Thus, the radiologist reading pediatric trauma studies should be familiar with normal sutural anatomy, typical times of sutural closure, and common accessory sutures to avoid misdiagnosis. Normal sutures are usually identified on both axial CT images and 3-dimensional reconstructions by their typical zigzag course and sclerotic borders (Figure 1). One must also carefully evaluate normal-appear-

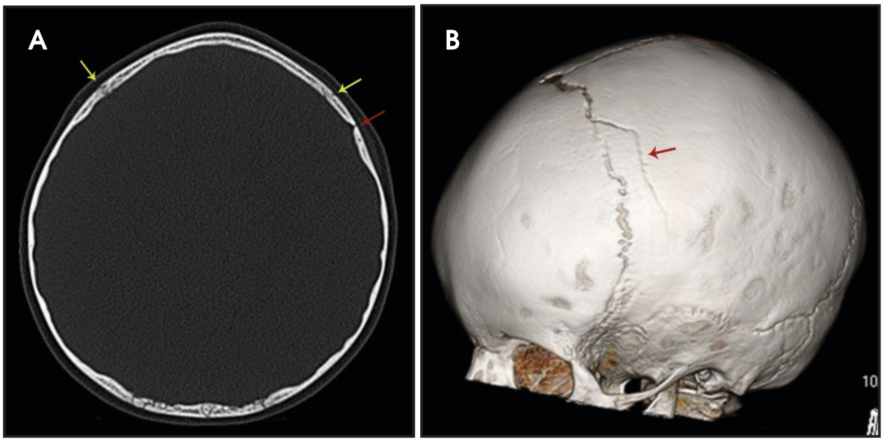

FIGURE 1. *(A) Axial CT scan in bone windows demonstrates the normal symmetric interdigitated pattern of the bilateral coronal sutures (yellow arrows), with an asymmetric linear defect paralleling the normal left coronal suture posteriorly (red arrow). This fracture was missed on initial review. Soft-tissue swelling was present inferiorly, not shown. (B) 3-dimensional reconstructions performed after high clinical suspicion definitively revealed a nondisplaced fracture of the left parietal bone paralleling the left coronal suture (red arrow).*

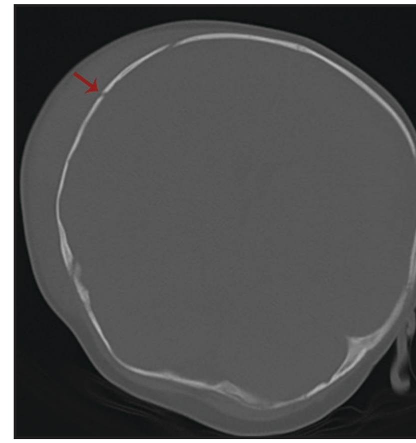

FIGURE 2. *A 6-month-old child who fell onto the floor from her grandfather's lap. Scalp hematoma along the right aspect of the calvarium overlies a subtle nondisplaced fracture of the right parietal bone (red arrow).*

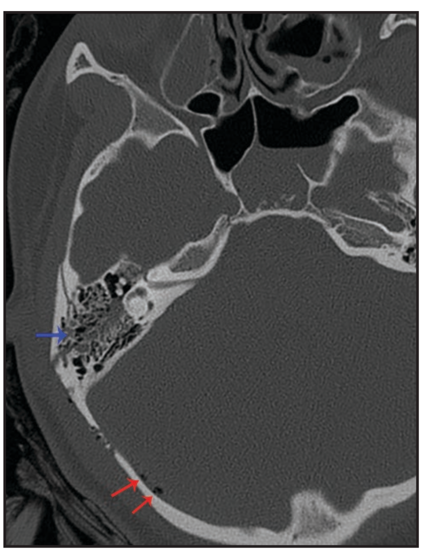

Figure 3. A 13-year-old female without a helmet fell off a horse. CT demonstrates fluid within the mastoid air cells (blue arrow) and foci of pneumocephalus (red arrows) secondary to a complex comminuted right temporal bone fracture.

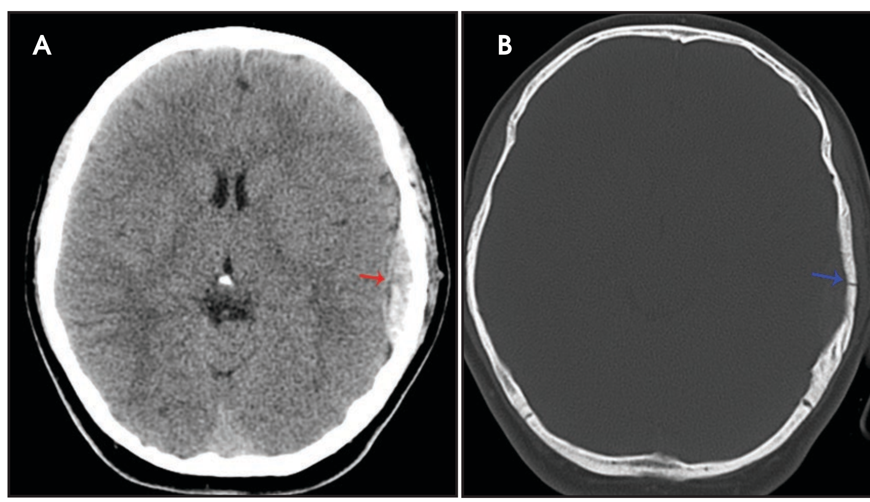

FIGURE 4. *(A and B) A 14-year-old boy who fell while skateboarding. CT of the head demonstrates a classic lentiform-shaped epidural hematoma (red arrow) with overlying calvarial fracture (blue arrow).*

ing sutures for the presence of diastasis, as these are the most common of pediatric skull fractures and also tend to be more subtle than their linear, comminuted, or depressed counterparts.[5] The use of symmetry is a helpful tool for identifying sutural diastasis.

Accessory sutures most commonly occur in the parietal and occipital bones, most likely as a consequence of their multiple ossification centers. Like normal sutures, they demonstrate a similar zigzag course without diastasis and merge with adjacent sutures. They are often bilateral, in which case they are relatively symmetric.[6]

True fractures generally show a linear course with lack of well-defined sclerotic borders. In contrast to normal or accessory sutures, they may cross, rather than merge with adjacent sutures. Fractures are more often unilateral and asymmetric. When bilateral, as sometimes seen in high-impact trauma, there is often associated displacement and/or comminution, with asymmetry as the rule.[6] The presence or lack of local soft-tissue swelling is an extremely valuable finding, as calvarial fractures rarely occur in the absence of an overlying soft-tissue abnormality (Figure 2).[7] However, one must be careful of dismissing a suspicious finding based on lack of soft tissue abnormality alone, especially in the setting of nonaccidental trauma, where late presentation is not uncommon.

Other helpful clues to subtle calvarial fractures include pneumocephalus, which sometimes may only be a few foci of gas, or fluid within the aerated spaces such as the mastoids (Figure 3). In rare difficult cases where a definitive diagnosis cannot be reached,

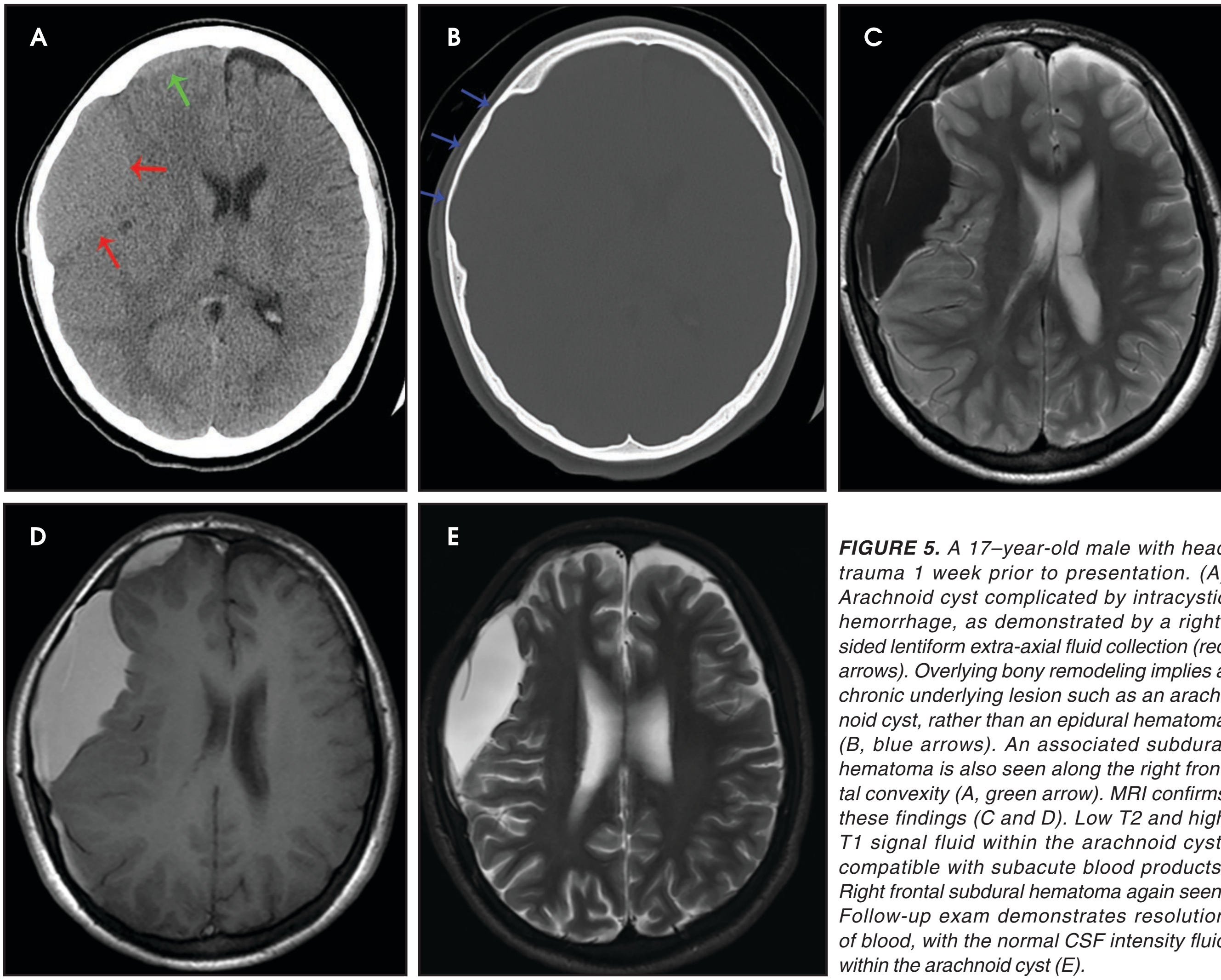

FIGURE 5. A 17–year-old male with head trauma 1 week prior to presentation. (A) Arachnoid cyst complicated by intracystic hemorrhage, as demonstrated by a right-sided lentiform extra-axial fluid collection (red arrows). Overlying bony remodeling implies a chronic underlying lesion such as an arachnoid cyst, rather than an epidural hematoma (B, blue arrows). An associated subdural hematoma is also seen along the right frontal convexity (A, green arrow). MRI confirms these findings (C and D). Low T2 and high T1 signal fluid within the arachnoid cyst, compatible with subacute blood products. Right frontal subdural hematoma again seen. Follow-up exam demonstrates resolution of blood, with the normal CSF intensity fluid within the arachnoid cyst (E).

close-interval follow up can be a helpful problem-solving tool, which can confirm presence of fracture healing at 2 to 3 months.[6]

Extra-axial hemorrhage

Extra-axial hemorrhage is a common primary manifestation of trauma in children. Although its appearance is classic and generally well known, several pitfalls can lead to misdiagnosis and subsequently to suboptimal clinical management. Extra-axial hemorrhage is classified by the space in which it occurs--epidural, subdural, and subarachnoid.

Epidural hemorrhage is characterized by its lentiform shape and respect of sutural boundaries; it may cross midline. Epidural hemorrhage is due to injury of either epidural arteries or veins and often associated with an overlying calvarial fracture and soft-tissue swelling (Figure 4).[8] Depending on its configuration, intracystic hemorrhage into an arachnoid cyst may mimic either epidural or subdural hemorrhage with associated mass effect. Although generally rare, a higher risk of this complication occurs in patients with larger cysts and those who have experienced trauma.[9] Treatment for this entity is unique to each patient, with some cases adequately managed conservatively, in which case a premature diagnosis of epidural hematoma could lead to unnecessary craniotomy.[10] This pitfall can be avoided by close attention to the overlying calvarium.

Arachnoid cysts are developmental lesions that are most often incidentally discovered. The chronic presence of an arachnoid cyst thins and remodels the inner table of the calvarium, which is suggestive of the diagnosis and is seen best on CT with bone windows (Figure 5). However, even when the diagnosis appears clear, care should be taken to exclude superimposed subdural hematomas that can be present in these patients.[11]

Subdural hematoma is characterized by its crescentic shape and ability to cross sutures.[8] Subdural hemorrhage is bounded by the midline falx and is due to tearing of bridging cortical veins (Figure 6). Benign enlarged subarachnoid spaces of infancy (BESSI) can look similar to chronic bifrontal subdural hematomas, but is a normal variant that should be considered in

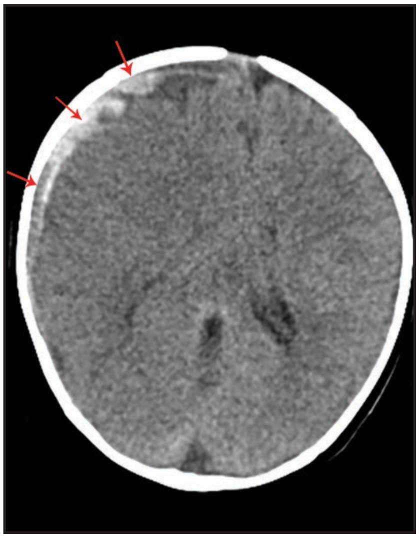

FIGURE 6. *Subdural hematoma along the right frontal convexity (arrows), crossing the coronal suture and bounded by the falx anteriorly. Heterogeneous nature of attenuation indicates evolution of blood products.*

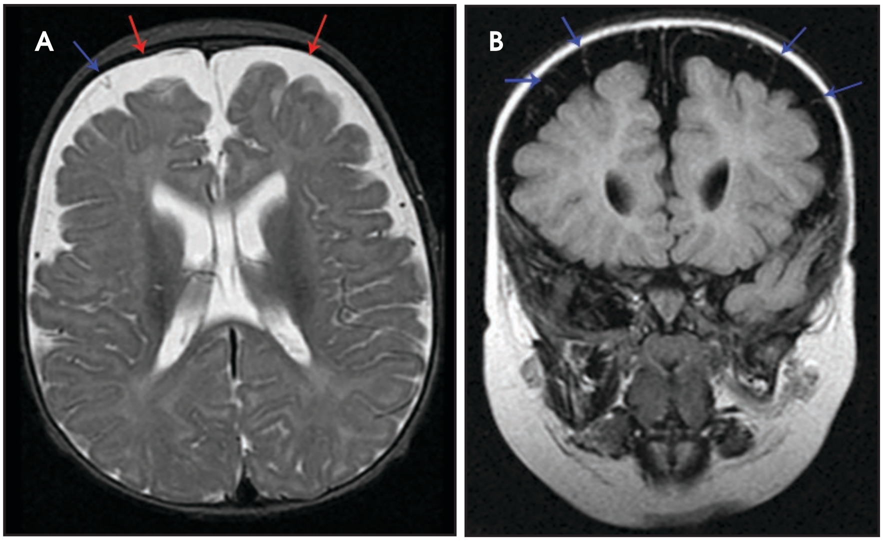

FIGURE 7. *(A and B) Axial T2 and coronal T1 images of an infant with minor trauma demonstrate benign enlargement of the subarachnoid space at the bilateral frontal convexities (red arrows), following CSF signal, which is symmetric in appearance. Traversing vessels confirm the diagnosis (blue arrows). Incidental cavum septum pellucidum et vergae.*

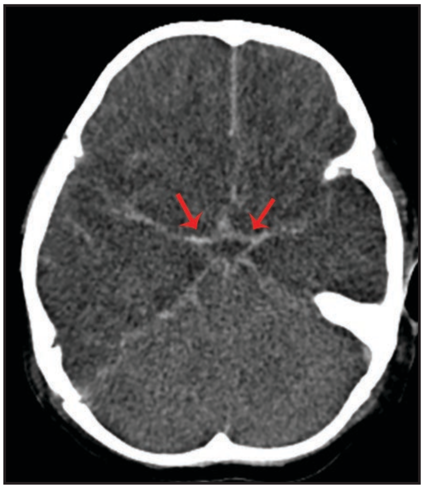

FIGURE 8. *Pseudosubarachnoid hemorrhage. There is relative hyperattenuation of the subarachnoid space due to hypoattenuation of the supratentorial brain from diffuse anoxic brain injury. White cerebellum sign also present.*

FIGURE 9. *Classic appearance of inferior frontal lobe hemorrhagic contusions with surrounding vasogenic edema following trauma (red arrow). Also seen is subarachnoid hemorrhage, most pronounced in the left Sylvian fissure.*

the appropriate clinical setting. Differentiating between these entities can be difficult and may require further evaluation with magnetic resonance imaging (MRI), where the unique features of each entity are better distinguished. BESSI is typically located along the frontal lobes and between the anterior interhemispheric fissure. Vessels are typically seen traversing the enlarged subarachnoid space, with lack of mass effect, normal to slightly enlarged ventricles, and no evidence of hemorrhagic products (Figure 7). In contrast, subdural hematomas may be asymmetric, demonstrate mass effect, have non-cerebrospinal fluid (CSF) intensity signal within or along the collections, and displace vessels along the inner table of the calvarium.[12]

Subarachnoid hemorrhage appears as increased attenuation in the cerebral sulci and CSF cisterns on CT.[8] The presence of subarachnoid hemorrhage in the setting of trauma is not an unexpected finding. However, high attenuation in the basal cisterns often associated with subarachnoid hemorrhage is not pathognomonic, especially in the presence of other important imaging findings. Diffuse cerebral edema either primarily or secondarily related to trauma is one of several causes of pseudo-subarachnoid hemorrhage, which can confound the diagnosis of subarachnoid hemorrhage. This phenomenon is thought to be related to a combination of displaced CSF, distention of superficial vasculature, and low attenuation edema within the adjacent cortex. Although its appearance is that of subarachnoid hemorrhage, closer evaluation of density yields significantly lower Hounsfield units than true subarachnoid hemorrhage (Figure 8).[13]

Intraparenchymal contusions versus cavernoma or other underlying lesions

Cortical contusions account for approximately 50% of all intra-axial trauma in the pediatric population. Typical contusions tend to be multiple, bilateral, and occur along the inferior frontal lobes and anterior temporal lobes, where the brain has a greater chance of direct impact against

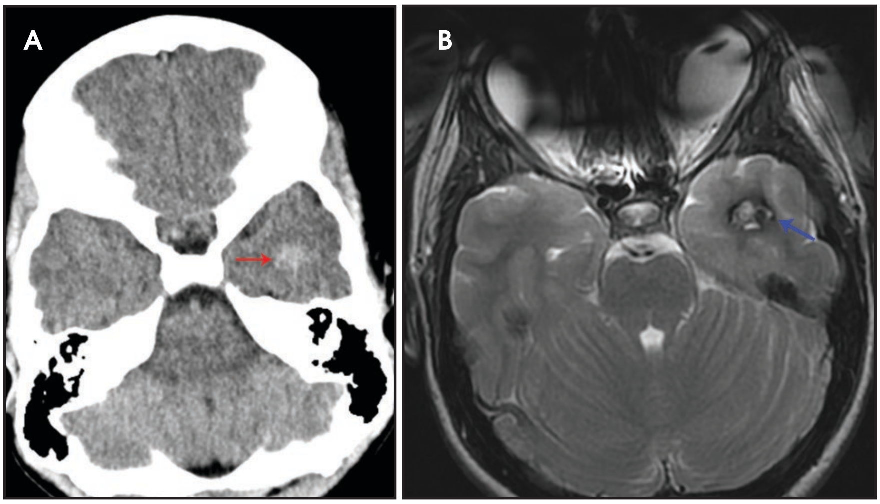

FIGURE 10. *A 13-year-old girl who struck her head during a fight at school. (A) CT demonstrates a focus of hyperattenuation in the left inferior temporal lobe (red arrow), a classic location for intraparenchymal hemorrhage, but with no significant surrounding low attenuation to suggest edema. (B) MRI demonstrates a "popcorn appearance" of the lesion, with a peripheral rim of hemosiderin (blue arrow), consistent with a cavernoma.*

rigid bone. Within the frontal lobe, injury is usually just above the cribriform plate and lesser sphenoid wing. In the temporal lobe, injury occurs adjacent to the petrous bone and greater sphenoid wing (Figure 9). In younger children, cortical contusions are less common because the inner table of the skull is smoother, and therefore, the above locations may not be typical.[1,2,14] It is important to look at the soft tissues of the scalp, as intraparenchymal contusions are classically coup (just below the point of direct impact) or contracoup (opposite to the site of direct impact) in location.

By definition, cortical contusions occur in the gray matter and tend to spare the adjacent subcortical white matter, unless the hemorrhage is large.[2] These may be difficult to identify on CT in the early stage, as one may only see faint areas of hypoattenuation reflecting early edema, with patchy punctate areas of hyperattenuation representing hemorrhage. Close follow-up imaging will unmask these subtle hemorrhages.[14] MRI of the brain is extremely useful, as even more subtle lesions can be identified; one must look for foci of high T2 signal edema and for punctate areas of low signal on gradient echo or susceptibility-weighted images reflecting hemorrhage.

Occasionally, a head CT may have a single focus of hyperattenuation, and the possibility of an incidental cavernous malformation must be considered. In patients with multiple hereditary cavernous malformations, multiple foci of hyperattenuation can be seen, although this is rare.

A cavernous malformation, also known as a cavernoma, cavernous angioma, or cavernous hemangioma, consists of a compact mass of sinusoidal-type vessels with no intervening brain parenchyma.[15,16] In those lesions that are not complicated by hemorrhage, areas of hyperattenuation on CT may represent foci of calcification. They tend to have no surrounding mass effect or vasogenic edema, unless complicated by superimposed hemorrhage. Cavernomas are typically centered in the subcortical regions and extend outwards toward the cortex, which is another key factor in distinguishing this incidental lesion from acute cortical contusion.[17]

MRI proves useful in questionable cases. Hemosiderin deposits within the lesion cause a rim of hypointense signal on all sequences. Postcontrast images will demonstrate variable enhancement.[17] T2-weighted images and susceptibility-weighted images demonstrate a central area of reticulation and a peripheral halo of hypointense signal, giving the characteristic "popcorn" appearance (Figure 10).[18,19] Cavernomas are often seen together with developmental venous anomalies, and identification of these enlarged medullary veins can help confirm the diagnosis.

Other mimics of intraparenchymal contusions include hemorrhage secondary to an underlying lesion, such as an arteriovenous malformation or tumor. In such cases, location, morphology, and degree of surrounding edema can be helpful clues to an accurate diagnosis.

Symmetric-appearing brain versus anoxic brain injury

One of the unique aspects of reading a CT or MRI exam of the head is that there is an internal standard of symmetric brain structures. At first glance, it is easy to pick up small findings just by comparing the left and right hemispheres; however, this technique can also be a major pitfall in pediatric imaging.

A normal pediatric head CT should have normal gray-white matter differentiation, with a distinct appearance of the basal ganglia. Any loss of blood flow or profound hypoxia can cause diffuse cerebral swelling, which will appear symmetric and can potentially be called normal by the untrained eye. CT will demonstrate loss of the gray-white matter differentiation, sulcal and ventricular effacement, as well as effacement of the cisterns.[2] One should look for low attenuation of the deep gray nuclei because these structures are extremely vulnerable to hypoxic ischemic insults, even if the event is short in duration, due to their inherent high metabolic activity and oxygen requirement (Figure 11). It is also important to remember that the midbrain and cerebellum are the least vulnerable to hypoxic injury, and therefore, diffuse cerebral injury may result in the appearance of a relatively lower attenuation cerebrum, when compared to the cerebellum, causing the "white cerebellum sign" (Figure 11).[20] In cases of trauma with anoxia, CT is usually the first imaging modality employed. Therefore, correctly identifying the di-

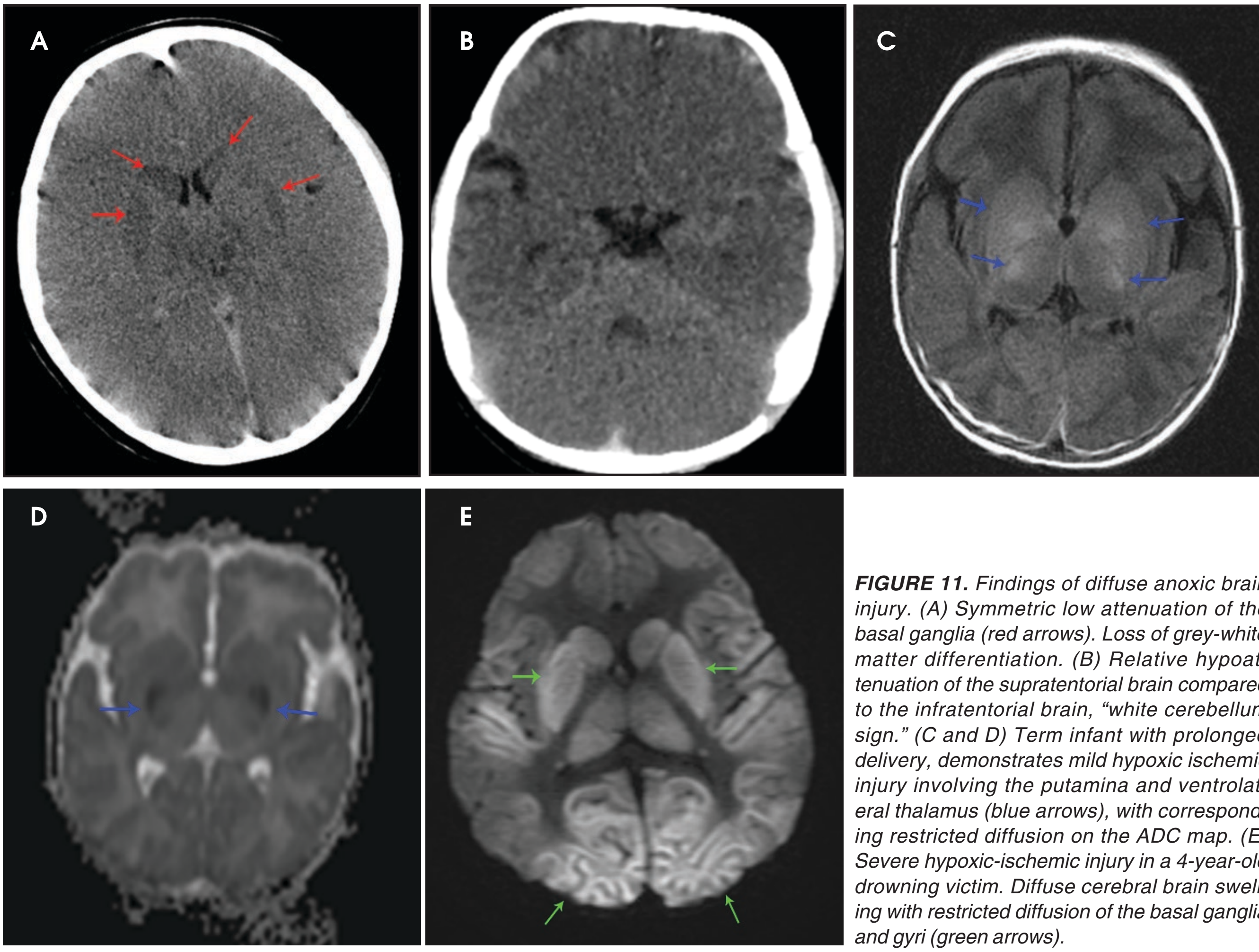

FIGURE 11. Findings of diffuse anoxic brain injury. (A) Symmetric low attenuation of the basal ganglia (red arrows). Loss of grey-white matter differentiation. (B) Relative hypoattenuation of the supratentorial brain compared to the infratentorial brain, "white cerebellum sign." (C and D) Term infant with prolonged delivery, demonstrates mild hypoxic ischemic injury involving the putamina and ventrolateral thalamus (blue arrows), with corresponding restricted diffusion on the ADC map. (E) Severe hypoxic-ischemic injury in a 4-year-old drowning victim. Diffuse cerebral brain swelling with restricted diffusion of the basal ganglia and gyri (green arrows).

agnosis on CT should appropriately prompt further evaluation with MRI to characterize the extent of injury.

In general, with neonates, mild cases of hypoxic-ischemic injury tend to involve only parts of the deep gray nuclei, with progressively severe forms involving a majority of the deep gray nuclei with peripheral extension to involve the cortex. With mild injury, T1-weighted images will demonstrate high signal within the ventrolateral thalami and posterolateral putamina. The T1 hyperintensity of the posterior limb of the internal capsule seen in full-term infants may or may not be preserved (Figure 11). Moderate injury tends to cause T1 hyperintensity of the anterior putamen, and the posteromedial thalamus, with usual loss of the normal T1 hyperintense signal in the posterior limb of the internal capsule. Severe injury causes T1 hyperintensity in the perirolandic cortex, insula, or possibly even involving all the cortices (Figure 11).[21.22] In neonates, there may be no appreciable T2 signal change except for subtle indistinctness of gray-white matter junctions; later in life, one will see areas of T2 hyperintensity. Diffusion-weighted images will demonstrate corresponding areas of restricted diffusion and may be the only images that reveal subtle injury.[1]

"Don't-miss" lesions

Diffuse axonal injury

When assessing trauma, it is important to look at the history and mechanism of injury. Depending on the location of edema and/or hemorrhage, one should be able to make the diagnosis of diffuse axonal injury — a diagnosis which has critical prognostic implications due to its poor clinical outcome and nonsurgical management. These patients tend to present with loss of consciousness directly after head injury.[14,23] The mechanism of injury is a shear-strain deformation of the brain due to rotational acceleration and deceleration, and therefore, this entity is also known as "shearing injury".[23,24]

As the name implies, these lesions occur within the axonal white matter. The most common location is the lobar white matter, followed by the corpus callosum (a majority of which are in the posterior body and splenium), and finally the brainstem (Figure 12). Less common locations include the cerebellar peduncles and basal ganglia.[14,23,24] CT may not have any appreciable findings, or only possible faint foci of hemorrhage or edema. For this reason, or in the setting of high clinical suspicion, it

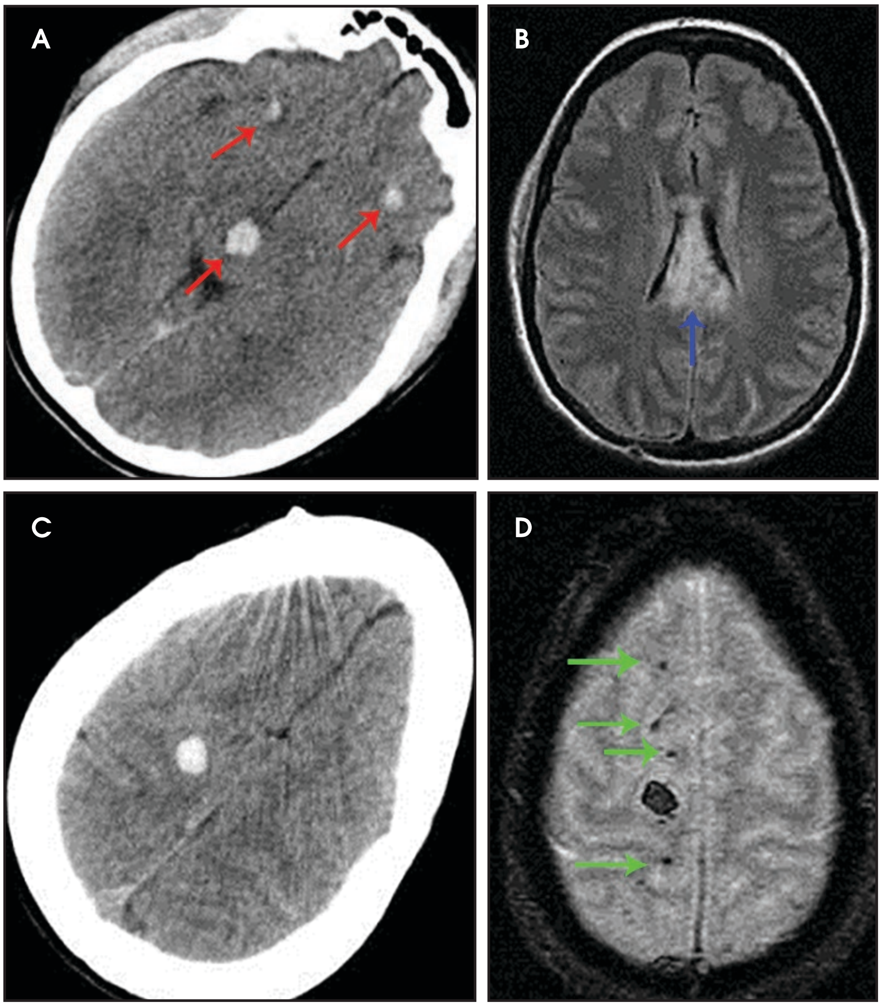

FIGURE 12. *Motor vehicle accident victim. (A) Lobar white matter hemorrhages near the gray-white matter junction and within the brainstem, the largest of which are labeled (red arrows). (B) Axial fluid attenuated inversion recovery (FLAIR) image demonstrates shearing injury of the corpus callosum (blue arrow). (C and D) CT and gradient recalled echo (GRE) images are shown at the same level. Note that the GRE image demonstrates blooming of additional microhemorrhages not readily apparent on CT scan (green arrows).*

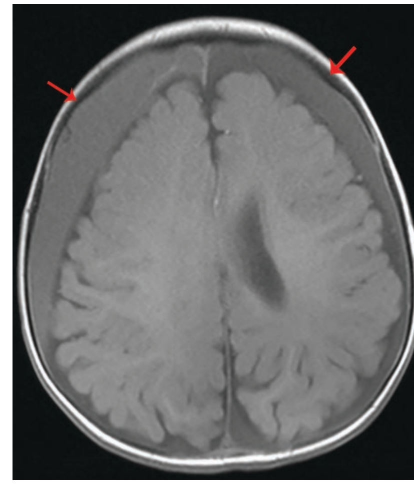

FIGURE 13. *T1-weighted image demonstrates bilateral subdural hematomas with differing signal intensity, signifying hemorrhages of different ages in a child abuse victim.*

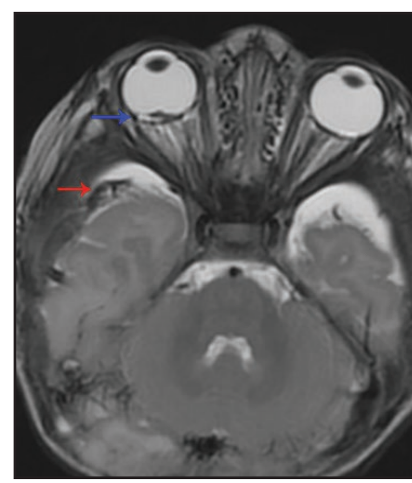

FIGURE 14. *Child abuse victim. Right subdural hemorrhage (red arrow) and right retinal hemorrhage (blue arrow).*

is important to recommend MRI for further evaluation. On MRI, it is critical to look for foci of low signal on susceptibility-weighted images representing hemorrhage (Figure 12).[25] Diffusion-weighted images may also show areas of restricted diffusion.[26]

Nonaccidental trauma

Due to the medical, legal, and social implications, another "don't-miss" lesion is nonaccidental head injury. When looking at a pediatric head scan, it is important to look at both the pattern and various stages (acute versus chronic) of injury. Homogeneously hyperattenuating subdural hematoma on CT is more commonly seen in children with accidental head trauma than those with nonaccidental head trauma. Mixed attenuation within a subdural hematoma or two (or more) subdural hematomas of varying ages should raise the suspicion for nonaccidental head injury (Figure 13).[27]

One term frequently used in literature is "shaken-baby syndrome;" it implies a rotational acceleration type of injury. The bridging veins that connect the dural sinuses to the brain are stretched, which causes hemorrhage into the subdural and subarachnoid space. The same stretching mechanism occurs along the axons in the white matter and in the retina. Therefore, the constellation of subdural hematoma, diffuse axonal injury, and retinal hemorrhage should alert the radiologist to suspect child abuse (Figure 14).[28,29] This suspicion must be promptly relayed to the referring physician.

It is also critical to look at both the history of the trauma, as well as other organ systems to get a global sense of injury. Look for fractures in various stages of healing, posterior rib fractures, vertebral body-compression fractures, duodenal hematomas, and pancreatic injury.[29] These patients may present with apnea, irritability, and decreased consciousness.[1,2]

Atlanto-occipital dissociation/ dislocation

Radiologists in training are often reminded to look at the edges of all imaging studies, a pearl that proves crucial in pediatric head trauma imaging. Due to concern about radiation exposure in the pediatric population, some clinicians may only perform a CT scan of the head without the cervical spine. It is important in these situations that the radiologist takes time to evaluate the craniocervical junction.

Osteoligamentous injuries at the craniocervical junction are of critical importance and can have devastating clinical consequences that are avoidable in some cases with early diagnosis and treatment. Ligamentous laxity, large size of the head relative to the body, relative weakness of the cervical musculature, and hypoplastic occipital condyles predispose children less than 10 years of age to atlanto-occipital injury.[30,31] Reportedly, 20% of patients with atlanto-occipital injuries initially present neurologically intact.[32] In some cases, dissociation of the atlanto-occipital joint is clear. In more subtle cases, perhaps in the setting of secondary signs of injury such as prevertebral soft-tissue swelling, measurements and ratios are often used to help make the diagnosis. Recent studies have demonstrated the usefulness of the atlanto-occipital joint interval (condyle-C1 interval, or CCI) above other commonly used ratios/measurements in identifying subtle craniocervical dissociation, with measurements above 2.5 mm in the coronal or sagittal planes considered abnormal.[33-35]

In cases where cervical spine injury is suspected in the absence of CT findings, MRI can be an excellent problem-solving tool that may help to identify ligamentous injury that is occult on other imaging modalities.[36]

Conclusion

The radiologist clearly plays an integral role in the setting of pediatric trauma. Prompt identification of the site and severity of injury can prove to be lifesaving for the pediatric patient. It is important to be aware of not only the patient's mechanism of injury, but also to the location and pattern of injury, paying special attention to the peripheral aspects of the imaging study. In instances of a mimic, proper recognition can prevent unnecessary treatment or surgery.

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