The Unique Features of Traumatic Brain Injury in Children. Review of the Characteristics of the Pediatric Skull and Brain, Mechanisms of Trauma, Patterns of Injury, Complications, and their Imaging Findings—Part 2

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ABSTRACT

Traumatic brain injury (TBI) is a major cause of morbidity and mortality in children. The unique biomechanical, hemodynamical, and functional characteristics of the developing brain and the age-dependent variance in trauma mechanisms result in a wide range of age specific traumas and patterns of brain injuries. Detailed knowledge of the main primary and secondary pediatric injuries, which enhance sensitivity and specificity of diagnosis, will guide therapy and may give important information about the prognosis. In recent years, anatomical but also functional imaging methods have revolutionized neuroimaging of pediatric TBI. The purpose of this article is (1) to comprehensively review frequent primary and secondary brain injuries and (2) to give a short overview of two special types of pediatric TBI: birth related and nonaccidental injuries.

Keywords: Pediatric traumatic brain injury, primary injury, secondary injury, skull fractures, facial trauma, epidural hematoma, subdural hematoma, intraventricular hemorrhage, subarachnoid hemorrhage, cortical contusion, diffuse axonal injury, computed tomography, magnetic resonance imaging, birth trauma, nonaccidental head injury.

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Introduction

Traumatic brain injury (TBI) is the leading cause of disability and mortality in childhood and adolescence. Children are not small adults. The anatomy, activity, and "life style" of children differ significantly from those of adults, resulting in very different types of injury. In addition, the progressing anatomical and functional development and maturation of the pediatric brain determine neurological deficits, recovery, and outcome. Birth trauma is responsible for almost all cases of neonatal trauma; nonaccidental injury is a frequent type of trauma in the first 2 years; whereas, falls and motor-vehicle accidents are responsible for the majority of TBI in toddlers and adolescents.

Pediatric TBI can be categorized into two types of injury: (1) primary injury, which is the consequence of the initial trauma or impact of force; (2) secondary injury, which occurs as a complication of the primary injury. Secondary injury is often underestimated but may be responsible for a significant secondary worsening of the prognosis and outcome.

Primary injuries can be extraaxial (eg, epidural hematoma [EDH], subdural hematoma [SDH], subarachnoid hemorrhage [SAH], and intraventricular hemorrhage [IVH]), intraaxial (eg, diffuse axonal injury [DAI], cortical contusion, and intracerebral hematoma), or vascular (eg, vascular dissection, carotid cavernous fistula, arteriovenous dural fistula, and pseudoaneurysm).[1]

Secondary injuries include both acute (eg, diffuse cerebral swelling, brain herniation, infarction, or infection), and chronic injuries (eg, hydrocephalus, encephalomalacia, cerebrospinal fluid [CSF] leak, and leptomeningeal cyst).[1]

In recent years, imaging has revolutionized the early and specific diagnosis of the full extent of TBI. Not only anatomical but also functional magnetic resonance imaging (MRI) sequences have proven to be highly sensitive in assessing quality and extent of brain injury. Various qualitative and quantitative imaging data have shown to correlate with the final mental, cognitive, and motor outcome. Allowing identification of possible chronic sequelae of brain tissue injury for children with TBI, these imaging techniques may not only facilitate efficacy and efficiency in delivering successful rehabilitation services, but also better clarify the pathogenetic link between trauma and long-term sequelae in terms of legal proceedings and indemnification of patients.

In this review, we summarize and present the principal imaging findings of various common and uncommon primary and secondary TBI's, with special emphasis on the characteristic differences between children and adults. In addition, two specific

pediatric types of TBI are discussed: (1) birth related TBI and (2) nonaccidental TBI.

Types of Injury

Concussion (Primary Injury)

Concussion is a trauma-induced alteration in mental status that may or may not involve the loss of consciousness.[2,3] The alteration of conscience is caused by either a diffuse cerebral dysfunction resulting from a rotational acceleration or deceleration or because of a more specific involvement of the brainstem. Concussion is a pathophysiologic process resulting in a self-limiting transient impairment of neurologic function that has an associated set of clinical symptoms including headache, dizziness, confusion, nausea/vomiting, and lethargy.[4] Theories regarding the biomechanics of concussion are still a matter of debate. In the laboratory setting, concussion can be more effectively achieved by nonimpact acute forceful rotation of the head than by a direct blow to the head.[5]

Twenty percent of TBI's with loss of consciousness occur during sports activities.[6] Brain concussion is considered a minor head injury, and the reported rates of surgical intervention approach zero in children with this type of injury.[7] However, proper assessment and management of sport-related concussions is crucial because repetitive concussions can result in a decreased neurocognitive functioning, progressive symptoms, and, at times, catastrophic outcomes.[8]

The diagnosis is typically established on the basis of clinical symptoms because patients generally do not have structural damage to the brain. Plain films, computed tomography (CT) and even conventional MRI are per definition unremarkable.[4,9] Newer functional MRI (fMRI) techniques (eg, diffusion tensor imaging [DTI] or fMRI) may prove relevant in the future. To date, the majority of DTI studies in acute TBI have been limited to adults and to patients with moderate or severe TBI. However, in a recent study of adolescents with mild TBI and negative CT, Wilde and colleagues found a strong relationship between increased fractional anisotropy (FA) values in the corpus callosum and more intense post concussion symptoms as well as emotional distress compared to a control group.[10] Therefore, DTI can be a promising imaging modality to objectively assess abnormal cerebral connectivity. Studies of fMRI performed in patients postconcussion are consistent with the concept that symptoms are related to neuronal dysfunction rather than purely anatomical lesions.[11] Chen and colleagues studied 16 concussed athletes and 8 controls subjects and demonstrated different activation patterns in 15 symptomatic athletes compared with the controls.[12] Data regarding the use of magnetic resonance spectroscopy (^{1}H-MRS) in patients with a concussion is very limited, and the clinical significance remains unclear.

Skull Fractures (Primary Injury)

Osseous lesions are significant not only as a direct sign of trauma but may also act as a pathway for the spread of infection. In addition, skull base fractures may compress or injure cranial nerves that leave the cranial vault.[13]

The reported incidence of skull fractures in pediatric head trauma series ranges from 2.1% to 26.6%.[5,6] Skull fractures occur in 75% of severe TBI, but in less than 10% of minor head traumas. Studies of pediatric head injury have shown that almost half of the intracranial injuries occur without a skull fracture identified on conventional plain films.[14]

The parietal bone is most commonly fractured (approximately 60–70%), followed by the occipital, frontal, and temporal bones.[15] The majority of the fractures are linear.

There are some aspects about skull fractures that have to be kept in mind. It may be difficult to differentiate linear fractures from normal sutures. However, the latter have typical anatomical locations, sclerotic borders, and a "zig-zag" configuration. In addition, the presence of an adjacent extracranial soft tissue swelling or an intracranial hematoma makes a fracture more likely. On CT, additional multiplanar reconstructions may help to identify linear fractures coursing in the plane of the primary CT acquisition (Fig 1A and B). The majority of frontal and occipital fractures are the result of direct impact. The calvarial bones have a membranous origin, and fractures typically heal without a periosteal reaction, making it nearly impossible to precisely date a calvarial fracture.

Skull fractures are typically described as linear, depressed, or basilar (skull base). Linear fractures are by far the most common and account for approximately 75% of all fractures. In 15–30% of the cases, they are associated with intracranial injury.[15] In depressed fractures, which occur in 7–10% of children with head injury, the bone fragment is depressed below the inner tabula. Depressed skull fractures are occasionally associated with injury to the underlying dural venous sinuses (Fig 1C and D), resulting in venous EDH or thrombosis. "Ping-pong" fractures (Fig 2) are a special variant of depressed skull fractures, in which the inner and outer skull table can be dented like a ping-pong ball. They are more commonly seen in the newborn when the cranium is less well mineralized and more prone to distortion. Basilar or skull base fractures are present in 6–14% of pediatric trauma patients and frequently extend from the temporal bone or from the paranasal sinus fractures. In up to 80% of the cases, secondary complications may occur including acute or delayed CSF-leakage with rhinorrhea, CSF otorrhea, ecchymosis over the mastoid bone (Battle's sign), periorbital ecchymosis (raccoon eyes), hematotympanum, or 7th cranial nerve palsy because of direct compression. Secondary meningitis may occur as complication of basilar fractures and occurs in .7–5% of the cases. Basilar skull fractures affecting the petrous bone (Fig 3) and middle ear may also dislocate the ossicular chain or disrupt the otic labyrinth leading to permanent hearing loss (Fig 4). Basilar fractures can also compress cranial nerves that pass through the skull base foramina. Ocular nerve and muscular entrapment should be ruled out in this particular clinical setting. Moreover, basilar fractures may cause lesions of the vessels, particularly the internal carotid artery, such as dissection or posttraumatic aneurysm. The great majority of basilar/skull base fractures resolve spontaneously without the need for surgical intervention.[16]

Leptomeningeal cysts (Fig 5) are unique pediatric lesions that occur as delayed complications in .05–1.6% of infants and children with skull fractures.[17] Leptomeningeal cysts are responsible for the so called "growing fractures" and typically occur in children younger than 3 years of age.[17,18] It results from

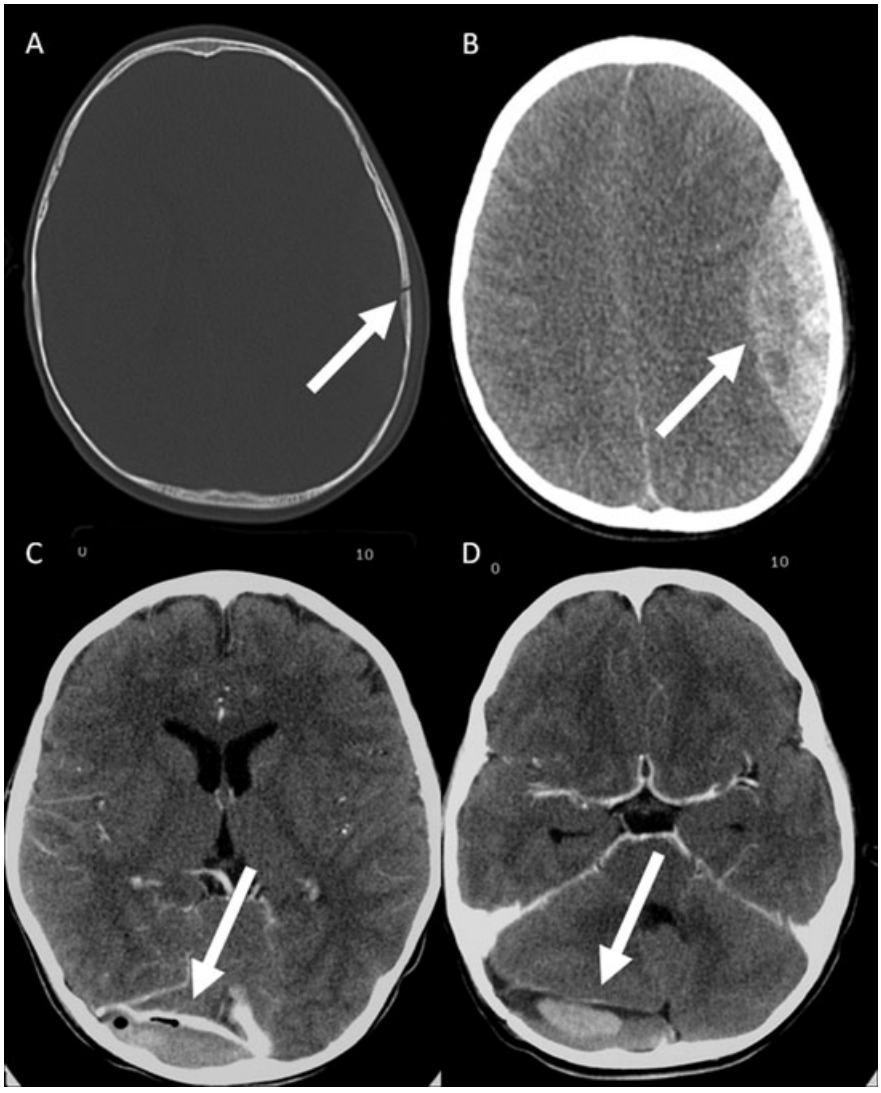

Fig 1. Axial CT images (A, B) of a 2-year-old boy who fell from 6 feet reveal a nondisplaced left temporal fracture with an underlying crescent shaped, hyperdense epidural hematoma (arrow). The extension of the EDH is limited by the coronal and lambdoid suture. Mild mass effect on the adjacent brain with mild midline shift is seen. Axial contrast enhanced CT images in a child with a supratentorial (C) and infratentorial (D) EDH because of a right sided skull base/temporal bone fracture. The EDH elevates and compresses the adjacent transverse sinus (arrow). Small air bubbles are seen within the hematoma which escaped from the fractured mastoid air cells.

a tear in the dura underlying the fracture. This interruption of the dura allows propagation of the systolic CSF pulsations across the fracture line with progressive meningeal herniation and widening of the skull defect.[18,19] In addition, it is believed that these cysts are especially frequent when the dura is entrapped within the fracture. There is often an accompanying area of encephalomalacia of the underlying brain.[20] Risk factors for the development of a leptomeningeal cyst include age less than 3 years and fracture diastasis of 4 mm or greater.[17] The diagnosis is usually suspected during follow-up when a palpable bulging mass is discovered. Diagnosis is usually made by the combination of CT to confirm skull fracture and brain MRI to depict parenchymal changes. An early diagnosis, however, is important because the brain damage and skull defect can be progressive over time.[19] MRI may make an early diagnosis of growing skull fracture showing indirectly the dural tear.[21] Ultrasonography can also diagnose the dural defect. Most leptomeningeal cysts require surgical correction of the dural defect.[15] Neurological/functional prognosis is determined by the degree of underlying brain injury.[16]

Plain films of the skull are unreliable in predicting the presence and degree of brain injury. Brain injury can be present in the absence of a skull fracture.[22] CT is consequently the primary, acute imaging modality of choice to study children who suffered TBI. Two-dimensional (2D) and three-dimensional (3D) reconstructions should be done not to overlook fracture lines parallel to the plane of scanning. MRI is more sensitive and specific when parenchymal brain lesions are suspected. To localize the exact site of a CSF leakage, high-resolution thin-sliced heavily T2-weighted MRI with fat suppression can be done.[23] Nuclear medicine studies may also be considered.

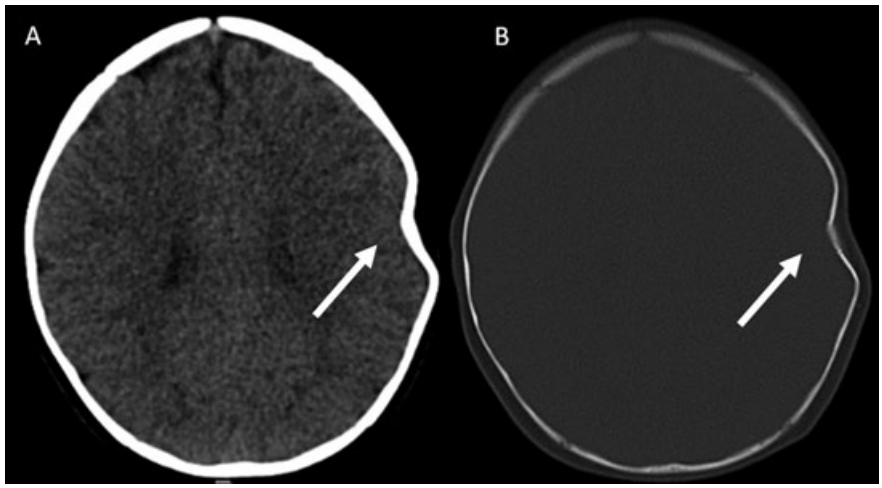

Fig 2. Axial CT images (A, B) of an 8-month-old female show a typical depressed "ping-pong" fracture (arrow). No underlying intraxial or extraxial brain lesion is noted.

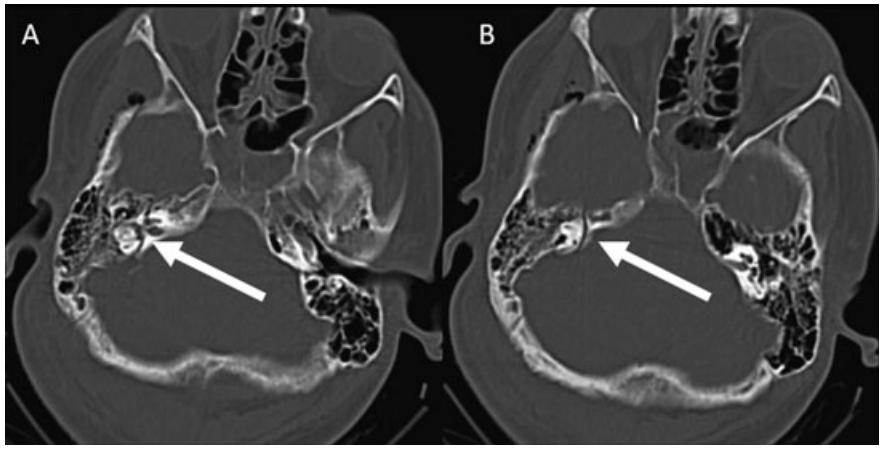

Fig 3. Axial CT images (A, B) show a transverse petrous bone fracture, crossing the cochlea and osseous canal of the facial nerve, extending into the region of the geniculate body. Small air bubbles are seen within the temporal and infratemporal fossa. The imaging findings match the clinical findings which include ipsilateral deafness and facial nerve palsy.

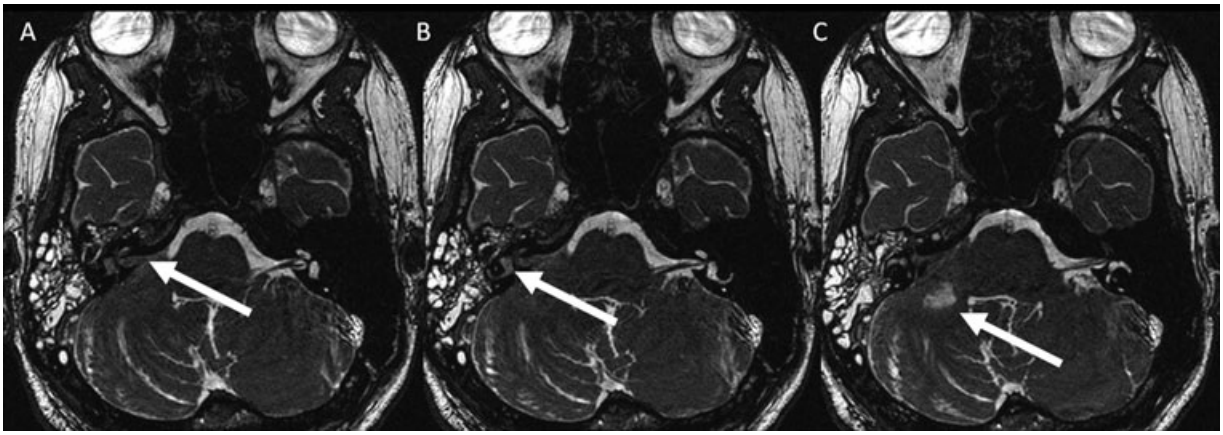

Fig 4. Axial thin sliced heavily T2-weighted MR images (A–C) of the same child as shown in Figure 3 confirm the right sided transverse petrous bone fracture. In addition, MRI shows T2-hypointense blood within the cochlea, vestibulum, semicircular canals, and internal auditory canal (arrows). Moreover, a nonhemorrhagic contusion is identified within the ipsilateral middle cerebellar peduncle (arrow). The right mastoid is completely filled with fluid.

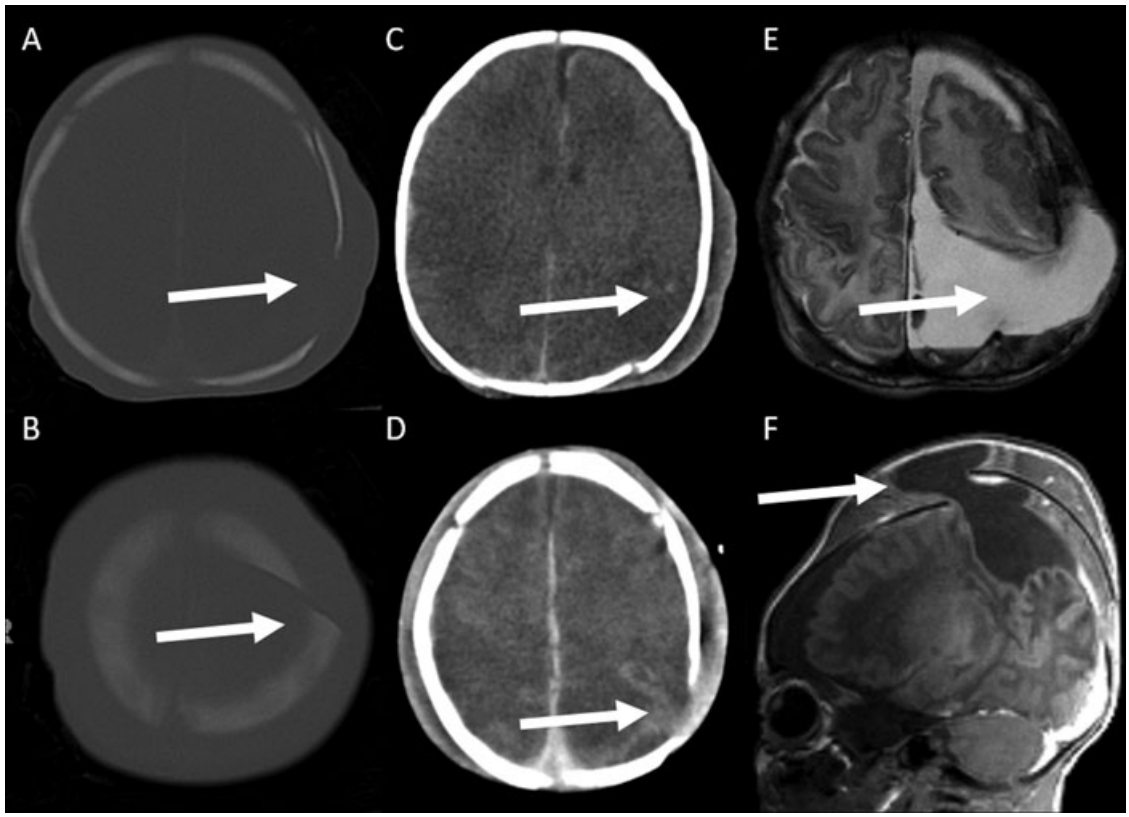

Fig 5. Axial CT images (A-D), axial T2-weighted (E) and sagittal T1-weighted (F) MR images of a 29-day-old boy with a growing fracture resulting from a nonaccidental injury. CT-images (A–D) show a diastatic left parietal fracture (arrow) with mildly hemorrhagic contusion of the adjacent parietal brain (arrow). Follow-up MRI shows interval appearance of a herniation of meninges and underlying brain (arrow) through the widened skull defect (E, F). In addition, a T1-hyperintense supratentorial and infratentorial SDH is noted.

Facial Trauma (Primary Injury)

Trauma-induced maxillofacial injuries are less common in children than in adults. The prevalence of pediatric facial fractures is the lowest in infants and increases progressively with age. Facial fractures rarely occur in children under 6 years of age. There is a male predominance ranging from 1.1:1 to 8.5:1 (male vs. female).[24]

Motor vehicle accident is the most common cause of facial trauma, followed by sports-related injuries and accidental causes.[24] Facial fractures are seen in 2.3% of abused children.[25]

Alcalá-Galiano and colleagues concluded based upon a case series of 262 patients (younger than 16 years of age) with 320 facial fractures that nasal fractures are the most common (58.6%), followed by mandibular (21.5%), orbital (9.5%), frontal skull (5.1%), and midfacial (3.8%) fractures.[24] Alveolar and dental fractures were seen in 19.4% of children in this age group. The true percentage may be underestimated in the literature. Recently, Gassner and colleagues found that 76.3% of children with facial trauma presented with dentoalveolar injuries.[26]

Knowledge of normal facial development and growth is important in understanding the patterns of pediatric facial trauma and the rationale behind its treatment. Facial growth, paranasal sinus development, dentition, and bone structure all affect the pattern of facial fractures in children. In children younger than 7 years, in whom the frontal sinus is not yet pneumatized, frontal fractures affect the orbital roof almost exclusively.[24] Midfacial or mandibular fractures entail a higher risk of associated intracranial injury, because these fractures patterns imply high-energy mechanisms. In these pediatric patients, the mandibular condyle is the most commonly injured region, which represents a therapeutic dilemma, because this area is considered to be a major growth center for the mandible. Temporomandibular joint dysfunction has been identified in a large proportion (72%) of patients after fracture healing.[27] Unlike the condylar region injuries, fractures in other areas of the mandible usually require surgical intervention. The midface is protected from fracture in children for several reasons: proportionately more prominent surrounding structures like the mandible and cranium, immature sinus development, increased bone malleability, and relative large amounts of facial subcutaneous fat.

Posttraumatic nasal edema often precludes adequate clinical evaluation of nasal fractures.[28] CT can give important information such as the recognition of septal fractures which may lead to saddle-nose deformities. Pediatric frontal sinus and naso–orbito–ethmoid fractures are uncommon, but carry a double risk for posterior table involvement and resultant CSF leakage compared to the adult patients.[29]

Finally, there is a wide variety of clinical presentations and fracture patterns after pediatric orbital trauma. The so called white-eyed orbital floor blowout fracture is associated with inferior rectus muscle entrapment in the presence of minimal direct

signs (ecchymosis, bony step-off) of the orbital floor fracture.[30] When muscular entrapment is present, early repair, generally within 2 days, is recommended for optimal outcome.[30]

Radiography provides limited diagnostic information about pediatric facial fractures, particularly for midfacial and condylar fractures, which may be easily overlooked on radiography.[24] CT with multiplanar reformatting and 3D reconstructions is mandatory for accurate diagnosis, especially in children with midfacial, orbital roof, mandibular, or temporomandibular joint fractures. High-frequency ultrasound (US) with either linear or curved array transducers may be performed as an alternative to radiography for the initial diagnostic work-up or can serve as a substitute for follow-up radiography. However, it is rarely used in clinical practice. MRI is also not included in the standard diagnostic work-up for facial trauma in children. MRI does provide reliable and adequate information about soft tissue injury but is limited for osseous lesions/fractures.[24] MRI should however be used when brain injury is suspected.

Retroclival EDHs and tectorial membrane injuries may be rare complications of pediatric head injuries, particularly motor-vehicle accidents. The clinical symptoms may be minimal or misleading and the radiologist should be aware of these injuries in children.

Extraaxial Hemorrhage

EDH (primary injury)

EDHs develop in the virtual space between the inner table of the skull and the dura mater. It occurs in about 3% of all head injuries, with the highest incidence in children of 10 years and older.[31]

EDHs (Fig 1A and B) are less common in infants and young children than in adults. The pediatric dura is more firmly adherent to the inner table of the skull.[32] In addition, the groove for the middle meningeal artery is shallow, allowing for more mobility of the artery.[19] EDHs in children are more frequently venous (from tears of a dural sinus or diploic veins) than arterial and, consequently have a better prognosis than EDHs in adults.[4,13,24] Venous EDH may slowly grow over 24 hours or more and tend to occur at three common locations: posterior fossa (from rupture of the torcula or transverse sinus), middle cranial fossa (from disruption of the sphenoparietal sinus), and along the vertex (from injury to the superior sagittal sinus; Fig 1C and D).[25] Arterial EDH typically rapidly grow in the first 6–8 hours after trauma. Skull fractures that cross the course of branches of the meningeal artery are most frequently the reason for arterial EDH. In neonates, arterial EDH are uncommon because the middle meningeal artery is not yet encased in bone and is not attached to the dura.[33]

The location of pediatric EDH is also different from adults. Pediatric EDH are more frequently seen in the posterior fossa or simultaneously above and below the tentorium.[13] Associated fractures are somewhat less frequent.[13,34] In the supratentorial cranial vault, EDH are more commonly located in the parietal and temporal regions.

In children, an impact to the head may cause a deformity of the skull without causing a fracture, but may be sufficient to separate the dura from the inner table with tearing of the small vessels.[32] Consequently, children younger than 15 years of age are less likely to have a fracture associated with an epidural hemorrhage.[32] As the child gets older, the etiology of the supratentorial EDH resembles that of adults.

EDH requires a direct impact to the head, mainly arising from falls in young children. Plain films are not reliable in evaluation of EDH as they can occur without skull fracture as mentioned previously. On CT, an acute EDH seems as a hyperdense, lentiform (biconvex) shaped fluid collection, and flattening/ compressing the adjacent gyri and sulci. It is widely accepted that EDH do not cross-cranial sutures because the periosteal layer of the dura is tightly adherent to the sutures. However, in a recent study by Huisman and colleagues studying 57 children with proven EDH, the authors showed that 11% of EDHs (7 patients) extended across cranial sutures.[35] Therefore, hematoma extension across a suture may not always allow differentiation between EDH and SDH.[35] Especially at the vertex, where the periosteum is not tightly attached to the sagittal suture, EDH can cross the midline.[1] CT low density areas within the hyperdense collection ("swirl sign") are thought to represent active bleeding within the hematoma and require a close follow-up monitoring.[36] MRI is helpful in depicting EDH because of the multiple image contrasts that can be generated which allow a better visualization of adjacent brain damage, and may offer a more reliable assessment of the time of injury.

SDH (Primary Injury)

SDHs are located in the space created by the traumatic separation of the arachnoidea from the dura mater. SDH usually cross sutures. The risk for SDH in the pediatric population ranges between 3.5% and 10.8%.[37,38]

Unlike EDH, SDH do not always require a direct impact of forces, but may also result from inertial shearing or rotational forces alone.[32] Therefore, they are more frequently associated with diffuse (axonal) brain injury.[18] However, in most circumstances, SDH occur when the head is abruptly decelerated by striking an object or surface. The most common cause of SDH in the young age group is abusive head injury.[32] The coexistence of SDH of different ages with the simultaneous occurrence of acute and chronic lesions may suggest child abuse, especially when a systemic hemorrhagic illness (coagulation disorder) has been excluded.

SDH (Fig 6) are more frequent in infants and younger children than in adolescents, being classically crescentic in shape. Most SDH are supratentorial and located along the convexity, the falx, or the tentorium. The bridging cortical veins are more easily torn, the plasticity/deformity of the pediatric skull is high, and the softness of the unmyelinated brain and the wider extraaxial spaces result in a higher degree of traction forces to the bridging veins facilitating development of SDH. SDH in children are more commonly associated with underlying parenchymal injury than EDH (Fig 7).[38]

Unlike in adults, SDH in children are often bilateral (80%),[13] are more common in the interhemispheric fissure and along the tentorium.[18] SHD can be extensive because of the lack of adhesions usually present in the adult.[19]

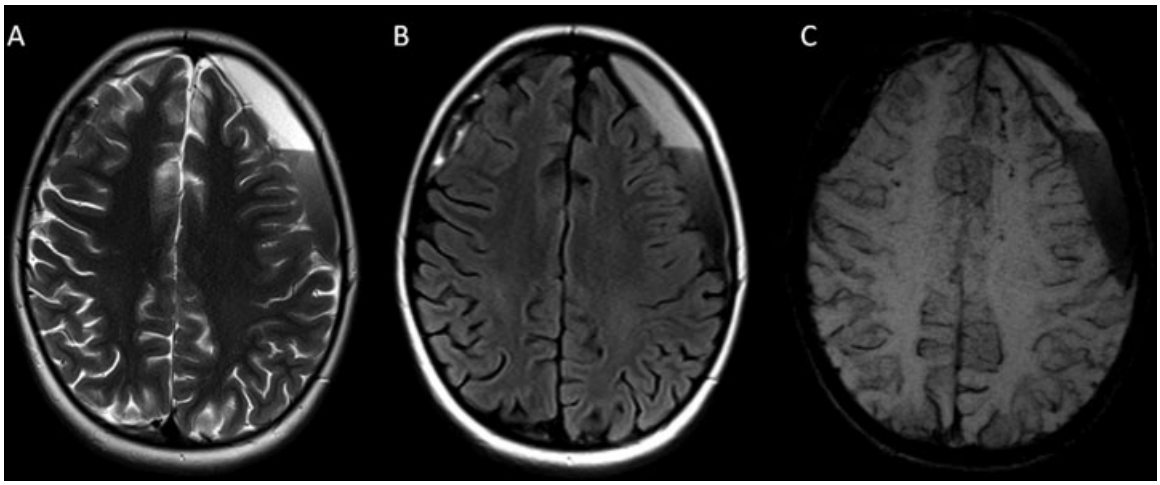

Fig 6. Axial T2-weighted (A), FLAIR (B), and SWI (C) MR images show a typical multilayered chronic, crescent shape subdural hematoma over the left frontal region. This SDH extends over the coronal suture. SWI images show prominent, engorged superficial veins along the left frontal lobe.

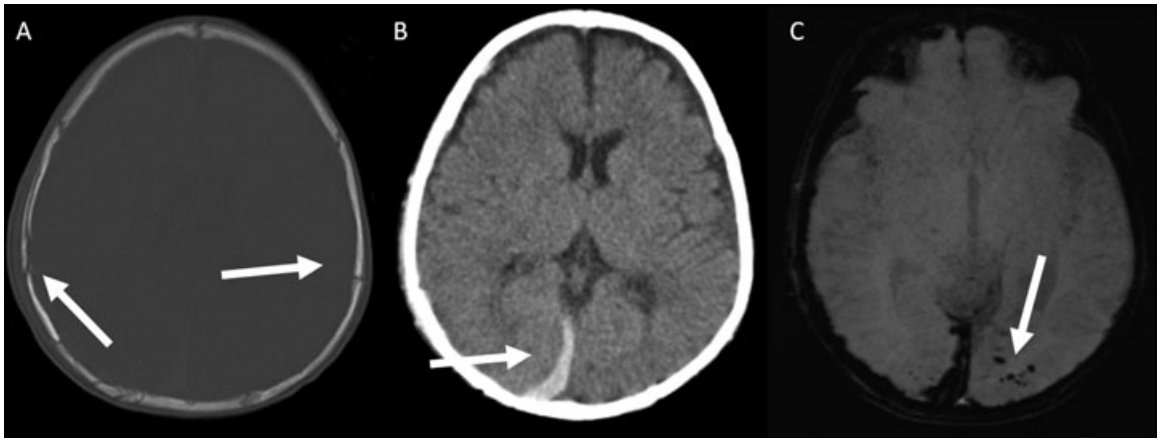

Fig 7. Axial CT images (A, B) and axial SWI MR image (C) of a child with confirmed nonaccidental TBI. Multiple bilateral linear fractures (arrows) (A) are seen as well as an acute, hyperdense subdural hematoma along the falx (arrow) (B). SWI image (C) reveals additional petechial intraparenchymal hemorrhagic shear lesions within the left occipital lobe (arrow).

It should be noted that in children and infants with brain atrophy, shunted hydrocephalus, benign external hydrocephalus, or previous subdural fluid collections in whom the bridging veins are already stretched, minimal forces may be sufficient to tear the veins.[39]

Occasionally, between 1 week and 3 weeks after trauma, depending on the patient's hematocrit level, clotting capability, and presence or absence of rebleeding, SDH may be isodense to gray matter on CT scan, limiting detection. If no MRI is available, the injection of contrast with opacification of the pial/cortical vessels, which are located between the isodense SDH and cortical gray matter may facilitate detection of isodense SDH. The MRI signal intensity of the SDH varies with the age of the blood products within the hematoma and the etiology of the SDH (venous vs. arterial). In addition, progressive dilution of the SDH by CSF leakage into the SDH also impacts the signal characteristics.[40] Because of the different image contrasts that can be generated and the lack of beam-hardening artifacts (which limits CT), MRI is useful in identifying small convexity and vertex SDH that might not be readily detected on CT.

Differential diagnosis between chronic SDH and prominent subarachnoid spaces in a child with macrocephaly, also known as benign enlargement of the subarachnoid spaces, is often difficult. In the latter condition, identification of vessels traversing the extraaxial space confirms the subarachnoid location of the fluid collection. Superficial US with linear transducer and color Doppler may be more reliable than CT to recognize the exact location of the fluid collection.[41]

SAH (Primary Injury)

Trauma related SAHs may result from the rupture of the subarachnoid, pial vessels, circulation/redistribution of IVH, or from an intraventricular or subarachnoid rupture of an intracerebral contusion or hemorrhage.

Imaging findings in SAH in children are identical to the adult counterparts (Fig 8A). On CT, acute, hyperdense blood extending into the brain sulci helps to confirm the diagnosis

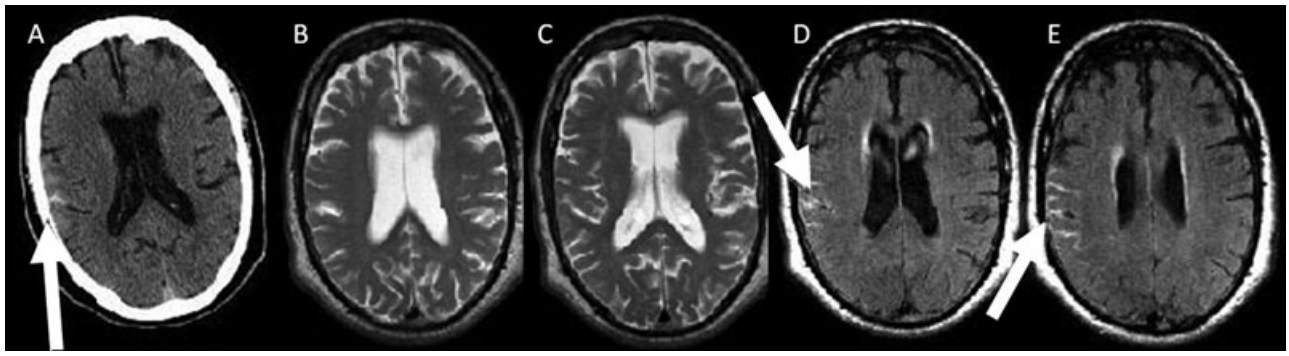

Fig 8. Axial CT (A), T2-weighted (B, C), and FLAIR (D, E) MR images of a child with posttraumatic subarachnoidal hemorrhage. The hyperintense blood within the brain sulci is best seen on the FLAIR images (D, E).

of SAH. Acute SAH can be difficult to detect on T1-weighted or T2-weighted images because SAH can be isointense with the brain parenchyma. FLAIR sequences facilitate detection of SAH. The blood products and proteins typically enhance the signal intensity of the subarachnoid CSF on FLAIR imaging (Fig 8).[19] Subacute and chronic SAH are better seen on MRI than on CT for different reasons: in the subacute phase, when the SAH is isodense to brain parenchyma on CT, it is usually hyperintense on T1-weighted and T2-weighted images; in the chronic phase, subarachnoid blood products (especially hemosiderin) result in a hypointense signal intensity on T2-weighted and T2*-weighted images as well as on the even more sensitive susceptibility-weighted imaging (SWI). A residual hypointense superficial hemosiderose typically outlines the cortical surface. SAH may coexist with SDH, EDH, and IVH in severe TBI.

IVH (Primary Injury)

IVH may result from the dissection of a large intracerebral hematoma into the adjacent ventricle, by the tearing of subependymal veins or periventricular structures such as the fornix, septum pellucidum, and corpus callosum or may result from the retrograde extension of a SAH into the ventricular system.

DAI is often associated with IVH from shear/rotational forces with tearing of the subependymal veins, typically along the ventral surface of the corpus callosum.[42]

IVH can be complicated by a secondary hydrocephalus because of resulting adhesions at the Sylvian aqueduct or because of obliteration of the arachnoid granulations (Fig 9). Patients with IVH are at risk for developing a "sterile" ependymitis from the chemical irritant effects of the blood products.

CT is very sensitive for diagnosing IVH in the acute setting because blood products typically result in a CSF-blood sedimentation level within the dependent parts of the ventricles (Fig 9). Special attention should be given to the existence of hemorrhage filling the aqueduct that can result in an obstructive hydrocephalus. CT is also useful in the assessment of the ventricular size. Similar to SDH, IVH can be difficult to be recognized in the acute phase because acute blood may

Fig 9. Axial CT images (A, B) show posttraumatic blood products within the ventricular system including Sylvian aqueduct after high-speed TBI. Moderate hydrocephalus is noted as complication of the IVH.

have similar signal intensity as CSF on MRI. Additional T2*-weighted or SWI sequences should be considered. During the subacute and chronic phase, MRI is highly sensitive.

Intraaxial Hemorrhage

Cortical contusion (primary injury)

Cortical contusions usually involve the superficial cortical gray matter with relative sparing of the subcortical white matter, unless the contusion is large (Fig 10). Frontal/frontobasal and temporal/temporobasal regions are mainly affected because of their close proximity to the irregular inner contour of the skull base, ie, the cribriform plate and the petrous bone and the biomechanical distribution of the forces within the skull. Contusions can also occur along the margins of depressed skull fractures (Fig 10). Cortical contusions are less common in children than in adults because the inner table of the skull is usually smoother.[19]

Direct blows to a stationary skull can produce contusions (coup contusions). In contrast, when the head is in motion before coming to contact with a stationary object, contusions can also occur at a remote location relative to the point of contact (contrecoup contusions; Fig 11).[38] Coup and contrecoup contusions do not or rarely occur in children under 4 years of age. The very soft consistency of the young brain makes it less likely to contuse rather than to tear when significant force is applied to the head.[32]

In the subacute phase of the injury, approximately 30% of all contusions exhibit mass effect. Moreover, swelling of the adjacent brain also develops/increases when the brain reacts to the local release of toxic metabolites after the contusion. This is typically more severe 4–6 days after the injury.[38] Increased mass effect resulting from enlargement of the hemorrhage and brain swelling can lead to secondary brain injuries from cerebral herniation with focal ischemia resulting from compression of vascular structures. This is particularly important in lesions within the temporal lobe (uncal hernia-

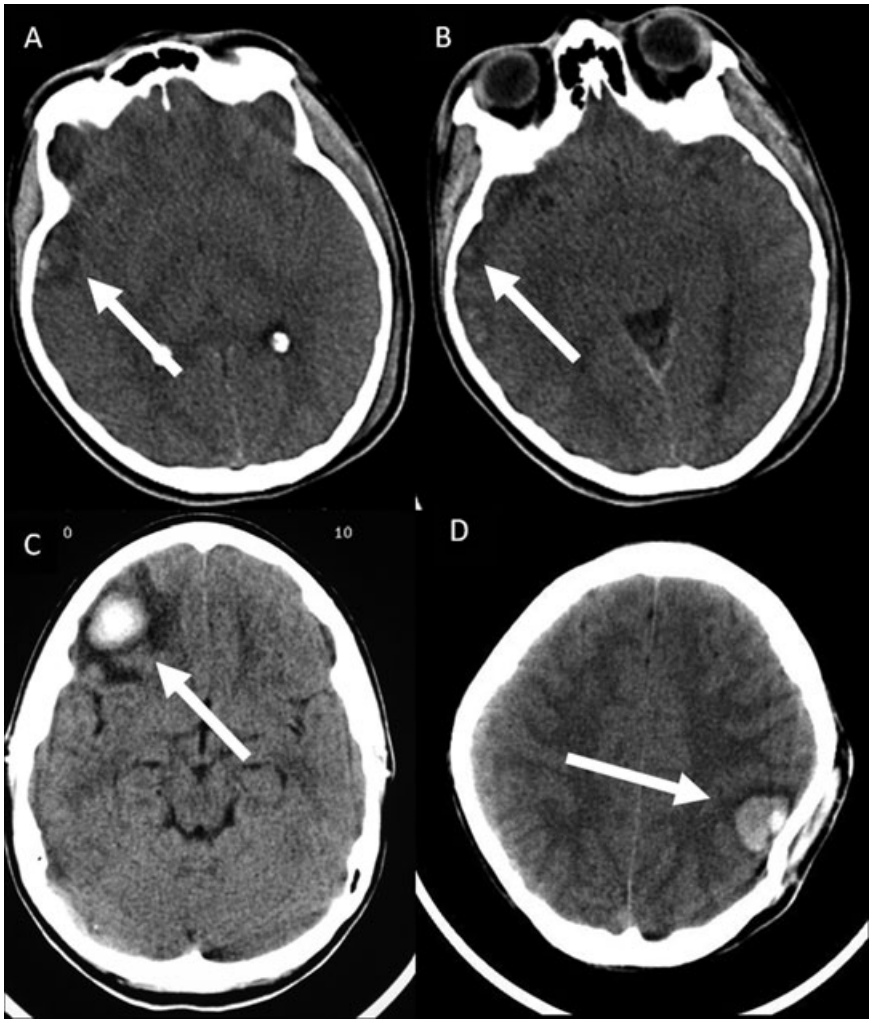

Fig 10. Axial CT images of two pediatric trauma patients with cortical contusions. In patient one (A, B) cortical contusions are seen in both temporal lobes, right > left with only mild subcortical edema. In patient two (C, D), a classical "coup and contrecoup" distribution of cortical lesions is seen. A cortical/subcortical coup is seen underlying a mildly depressed left parietal fracture in combination with a right frontobasal contrecoup lesion.

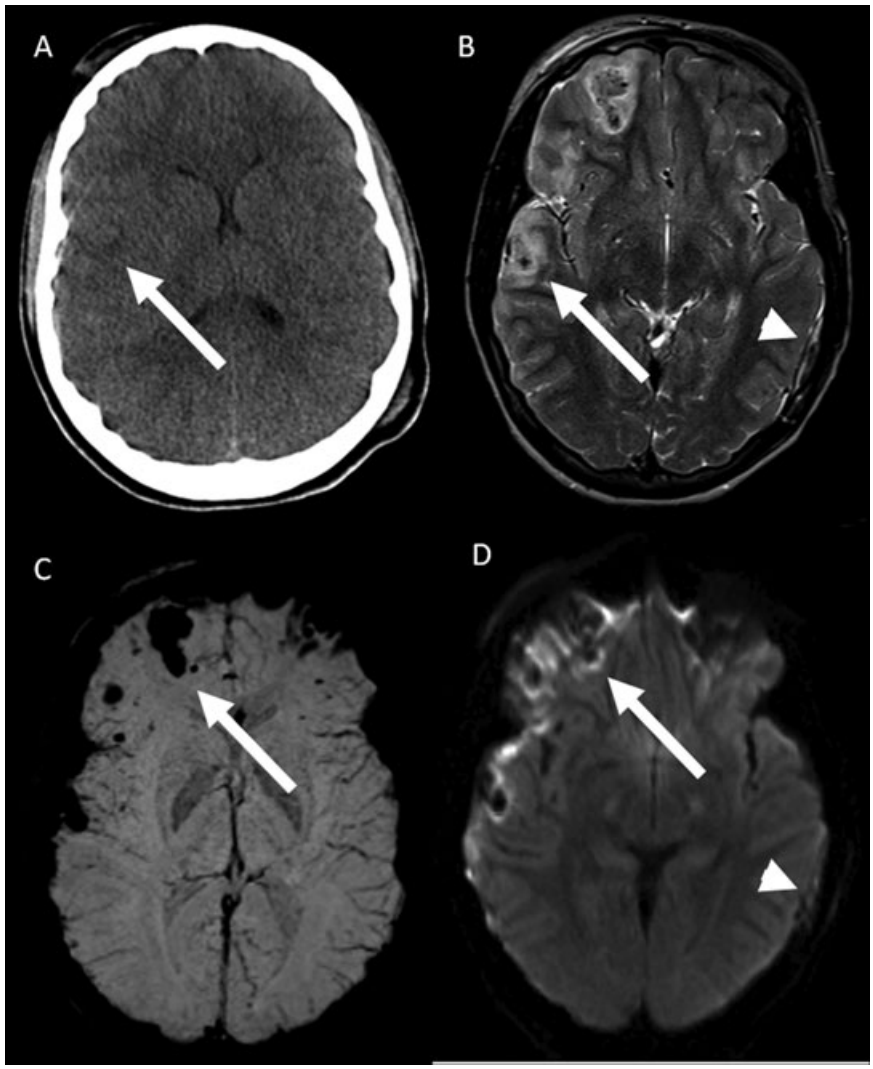

Fig 11. Axial CT (A) and matching T2-weighted (B), SWI (C), and DWI (D) MR images of a child with classical "coup and contrecoup" lesions. The primary impact of force resulted in minimal left temporal cortical (coup) and more extensive right fronto–temporal cortical contusions (contrecoup). The hemorrhagic lesions are best seen on the SWI images (C). The subcortical edema is best seen on the T2-weighted (B) and DWI MR (D) images. The blood products result in mild susceptibility artifacts on DWI. A small extraxial hematoma is seen overlying the left temporal region.

tion), the hypothalamus (downward transtentorial herniation), and the posterior fossa (tonsillar herniation with direct brainstem compression or occlusion of the fourth ventricle with hydrocephalus).

On CT, most acute/subacute hemorrhagic contusions are hyperdense. It can however be particularly difficult to diagnose nonhemorrhagic contusions, unless significant edema is present. Another limitation of CT is the posterior fossa and skull base beam-hardening artifacts, which may obscure traumatic lesions in the brainstem, cerebellum and frontobasal/temporobasal brain. Signal characteristics on MRI depend on the age of the lesion/hematoma. Multiple studies have described the signal evolution of hematomas on various MRI sequences, which allow to date the age of the hematoma. The hypointense signal from hemosiderin may persist indefinitely on T2-weighted or T2*-weighted sequences and on SWI images. MRI is significantly more sensitive than CT for the detection of nonhemorrhagic contusions.

DAI (primary injury)

Clinical hallmark of severe DAI is an immediate loss of consciousness that may be associated with decerebrate or decorticate posturing. DAI is of special interest because it is considered to be responsible for the majority of TBI related neurocognitive deficits.[34,35] DAI is histologically characterized by a widespread axonal damage in multiple brain regions including the dorsal brainstem, parasagittal white matter, corpus callosum, and at the gray–white matter junctions of the cerebral hemispheres.

DAI results from severe head injury in which sudden acceleration and deceleration forces, possibly combined with rotational forces are exerted to the brain. These forces result in

a differential motion of brain tissue of different densities or neuroarchitecture (gray vs. white matter, crossing of white matter tracts). These shear forces are believed to stretch and rupture vessels as well as fiber tracts.[18]

Hemorrhagic and nonhemorrhagic shearing lesions associated with DAI occur in up to 40% of children with TBI.[43] Compared to older children and adults, infants are more susceptible to this injury, because of their relatively large heads, weaker neck musculature, wider CSF spaces and thinner calvarium.[19] In children under 1 year of age, axonal damage can be confined to the cranio–cervical junction.[44]

DAI because of shear strains typically affects the axons of the subcortical white matter (more frontal and parietal), the corpus callosum (particularly the posterior body and splenium), the brainstem (mainly dorsolateral midbrain), the basal ganglia, and the internal capsules (Fig 12).[45] The overlying cortex is usually spared.

Accurate and complete detection of the extent of DAI is often challenging, especially because less than 40% of the lesions are hemorrhagic. In the acute phase, CT scan can be completely normal or reveal only small petechial hemorrhages at the interface of the subcortical white and gray matter, may show minimal amount of intraventricular blood (resulting from the shear of subependymal veins), or discrete SAH is seen in the perimesencephalic cisterns. CT often underestimates the severity of DAI. MRI should be considered when the CT imaging findings do not explain the neurological symptoms/deficits or when the trauma mechanism is suggestive of DAI. Conventional T1-weighted or T2-weighted and FLAIR images can detect nonhemorrhagic lesions with higher sensitivity than CT.[46,47] Diffusion-weighted imaging (DWI) is valuable because it identifies additional shearing injuries not visible on T2-weighted, FLAIR, or T2*-weighted sequences. However, DWI is less sensitive than T2*-weighted imaging for detecting hemorrhagic lesions.[48] SWI can detect 640% more lesions and 200% greater hemorrhagic volume than conventional 2D gradient-echo images (Fig 13),[49] making SWI an invaluable additional MRI sequence in the diagnostic work-up of DAI. In a recent volumetric MRI study, Wilde and colleagues observed decreased anterior commissure cross-sectional volume in pediatric patients with TBI.[50] They concluded that, similar to the corpus callosum, this can be a vulnerable region for shear injury. As stated before, DWI and DTI can have an important role in depicting pathological changes in the normal-appearing white matter on conventional MRI sequences.[48,51] Chronic DAI imaging findings include global atrophy, gliosis, and hemosiderin staining, which can persist on T2*-weighted or SWI images for many years.

Intracerebral hematoma (primary injury)

Trauma-related intracerebral hematoma is a shearing-straining injury because of rupture of small intraparenchymal blood vessels and may sometimes be difficult to distinguish from cerebral contusions.[13] It is commonly seen in the fronto–temporal white matter or in basal ganglia.[52] Pathologically, it is possible to distinguish intracerebral hematomas from contusions or DAI lesions if the hematoma is located within relatively normal appearing white matter.[13] Perilesional edema is usually less severe in intracerebral hematomas.

Intracerebral hematoma can have a delayed presentation in the setting of acute head trauma,[1] and it is the most common cause of clinical deterioration in patients who have experienced a lucid interval after the initial injury. Intracerebral hematomas often result in secondary injuries because of the mass effect with resulting cerebral herniation and from vascular lesions/complications because of compression.

On CT and in the acute setting, intracerebral hematoma seems as a hyperdense focal mass lesion. When fluid levels are present within the lesion it suggests hyperacute bleeding or a disturbance in hematocrit levels and coagulation status. The signal characteristics of an intracerebral hematoma depend on the percentage of oxyhemoglobin, deoxyhemoglobin, methemoglobin, and hemosiderin within the lesion. An exact description is beyond the scope of this article. In brief, acute hematomas seem T1-isointense and T2-hypointense; in the subacute phase, they are T1-hyperintense and T2-hyperintense; in the chronic phase, they are T1-hypointense and T2-hypointense.[53] These signal characteristics closely match the evolving signal characteristics of traumatic hemorrhagic contusions.

Vascular injury (primary injury)

Traumatic vascular lesions in children may result from skull base or vertebral body fractures, from a direct blow to the neck, or from sudden accelerations/decelerations and rotational forces exerting to the neck. They include arterial dissection/laceration/occlusion, carotid–cavernous (arteriovenous) fistulas (CCF), arterial pseudoaneurysm, carotid sheath hematoma, and venous occlusions or ruptures.

Arterial dissections are more commonly affecting the internal carotid artery (ICA) and the vertebral artery (VA; Fig 14). ICA dissections most commonly involve the extracranial segment distal to the bifurcation of the common carotid artery. The dissection may extend to the skull base. In nonpenetrating trauma, VA dissection usually occurs at the C1–C2 level or at the site of cervical spine fractures.[54] In addition, in rare cases of head trauma arterial dissection may affect the middle cerebral artery.[55]

MRI findings of traumatic dissection include: (1) presence of an intramural hematoma, which is best seen on T1-weighted images with fat suppression; (2) intimal flap; and (3) absence of a normal vascular flow void secondary to slow flow or thrombosis. An associated parenchymal infarction in the territory supplied by the injured vessel should raise the possibility of a traumatic vascular dissection (Fig 15).[1] Conventional angiography has been historically considered the gold standard for the diagnosis of cervical artery dissection, but MRI/MR angiography (MRA) and CT/CT angiography (CTA) are currently used as valuable, highly sensitive noninvasive alternatives. Vertinsky and colleagues recently concluded that multidetector CT/CTA visualized more features of cervical artery dissection than MRI/MRA, whereas in many cases, MRI/MRA provided complementary or confirmatory information,

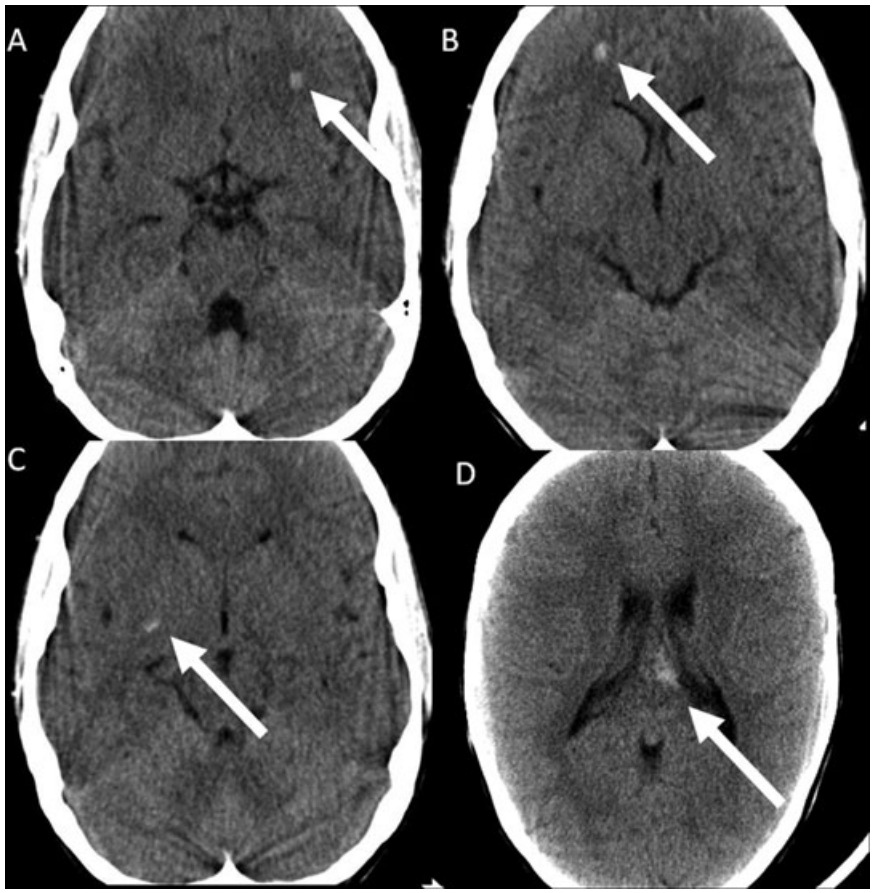

Fig 12. Axial CT images (A–D) of a 9-year-old girl who suffered extensive shear injuries because of a high speed MVA. Hyperdense shear injuries (arrows) are noted at the cortico–medullary junction within both hemispheres as well as along the right cortico–spinal tract within the internal capsule. In addition, significant shear injury is seen within the trunk and splenium of the corpus callosum.

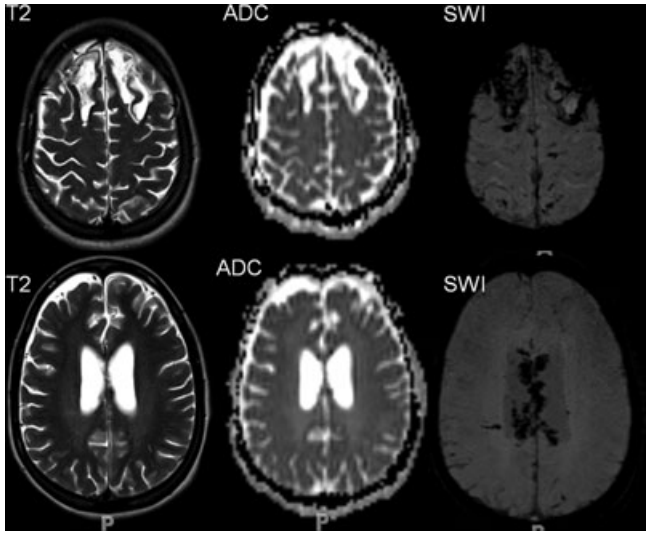

Fig 13. Axial T2, ADC, and SWI MR images of a 16-year-old female with diffuse axonal injury after high-speed motor-vehicle accident. Images are acquired several months after trauma. CSF filled T2-hyperintense (high ADC values) brain defects are noted within both frontal lobes. SWI is especially helpful to identify the extensive hypointense shear injuries within the corpus callosum as well as the blood products along the periphery of the brain defects. SWI shows, in addition, multiple small cortical/subcortical petechial hemorrhagic shear injuries.

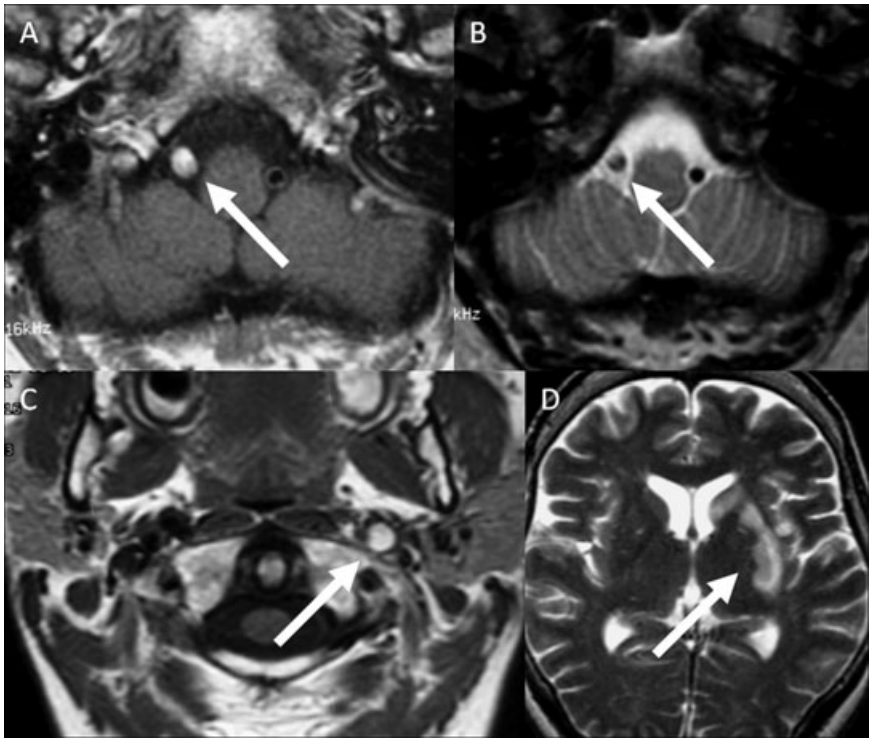

Fig 14. Axial T1-weighted (A, C) and T2-weighted (B, D) MR images of two children with posttraumatic vascular dissection. The first patient (A, B) suffered from a right vertebral artery dissection (arrows). The second patient (C, D) suffered from a thromboembolic left middle cerebral artery infarction because of a posttraumatic left sided internal carotid artery dissection (arrows). In both patients, the normal hypointense flow related signal void is replaced by a hyperintense thrombus within the false lumen.

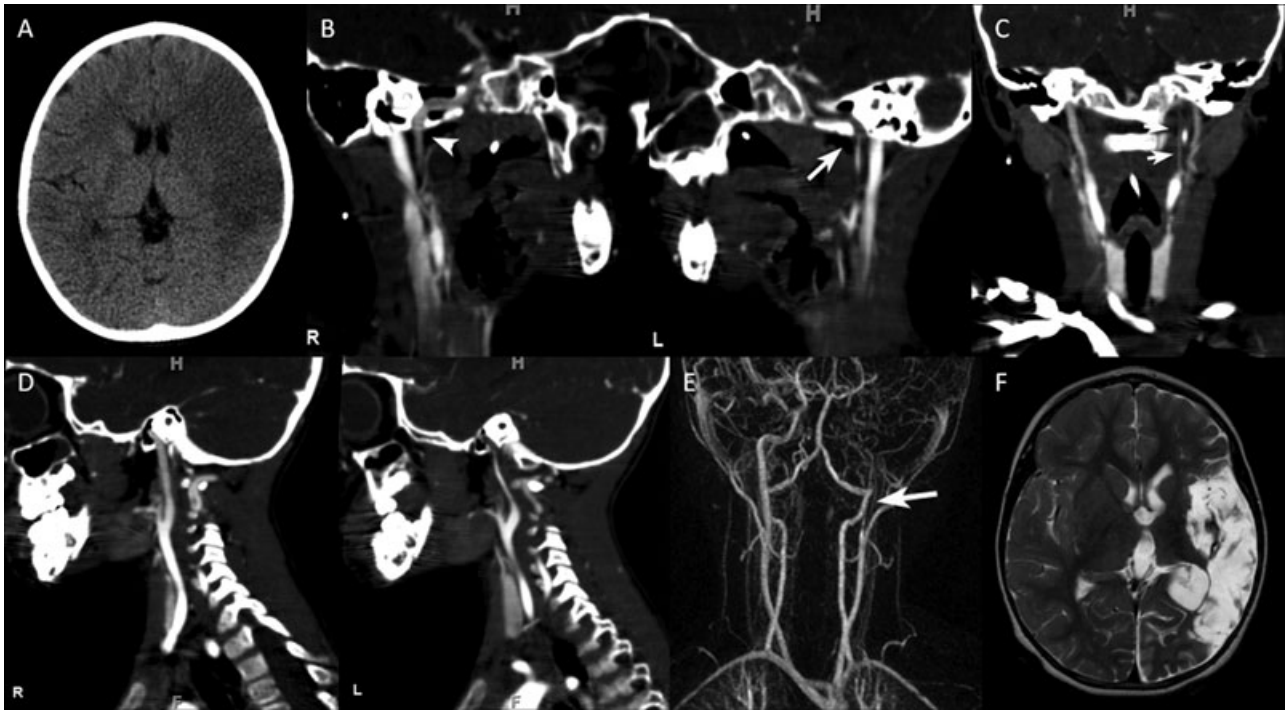

Fig 15. Axial CT (A), multiplanar CT-angiography (B–D), follow-up coronal MR-angiography (E) and axial T2-weighted (F) MR images of a male infant with posttraumatic left internal carotid artery dissection. The child had presented with an acute right focal neurological deficit. CT shows a large subacute, hypodense left middle cerebral artery infarction. CTA show an acute dissection with tapering of the left ICA with complete occlusion of the left ICA at the level of the skull base. Follow-up MRA and MRI show the occluded left intracranial ICA and the chronic left MCA ischemia.

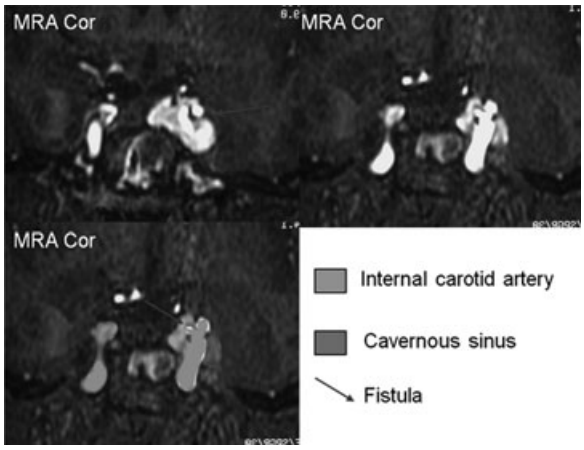

Fig 16. Coronal 3D-TOF MRA images (raw data) show a left sided internal carotid artery–cavernous sinus fistula after TBI. Arterial flow signal (red) is noted within the left venous cavernous sinus. The wall of the intracavernous segment of the internal carotid artery is interrupted (arrow). The right cavernous sinus is unremarkable.

particularly given its better and early depiction of ischemic complications.[56]

CCF (Fig 16) represent an abnormal communication (fistulous connection) between the cavernous segment of the ICA and the venous cavernous sinus. These CCF mostly result from skull base fractures, especially those that are extending into the sphenoid bones. The arteriovenous fistula leads to an arterial recruitment and dilatation of the affected cavernous sinus. The ipsilateral superior ophthalmic vein and inferior petrosal sinus typically enlarge because of the arterialization of the cavernous sinus. The intercavernous sinus may also distend the contralateral cavernous sinus. Clinically, CCF may present with a pulsatile exophthalmos or cranial nerve palsies.[19] CCF occasionally become apparent weeks or months after the acute trauma and can be bilateral because the cavernous sinuses are connected by a network of venous channels/shunts.

Pericallosal aneurysm (Fig 17) is a typical vascular traumatic injury, resulting from shearing forces between the falx, pericallosal artery, and the brain at the moment of trauma.[13] In addition, intracranial vascular injuries with formation of pseudoaneurysms may also result from penetrating injuries that affect/reach intracranial vessels.

Diffuse Cerebral Edema (Acute Secondary Injury)

Diffuse cerebral edema can be a catastrophic secondary complication after TBI and is often present in severe TBI cases. The age-related mechanisms underlying diffuse swelling in pediatric TBI are only partially understood. It is believed that hyperemic cerebral swelling occurs more frequently in children than in adults because of the immature or impaired autoregulation of the pediatric brain perfusion.[57] Animal models also showed that there is an increased release of excitotoxic neurotransmitters in the developing/immature pediatric brain after injury,[58] as well as an enhanced inflammatory response of the developing brain.[59] Furthermore, an increased blood–brain barrier permeability has been discussed in the immature brain.[59] In addition, children generally have a lower mean arterial blood pressure which increases the likability for critically decreased cerebral blood flow and consequent hypoperfusion of the brain facilitating development of brain swelling.[19] Finally, the more compliant skull compared to the adults favors a more diffuse pattern of brain distortion in children after TBI.[60]

Diffuse cerebral edema usually manifests within 24–48 hours after trauma. Both CT and MRI are sensitive in diagnosing cerebral edema. Effacement of the cerebral sulci and cisterns and compression of the ventricles are typical imaging findings. Cytotoxic edema is believed to result from a simultaneous occurrence of tissue hypoxia and hypoperfusion which can lead to a loss of the normal gray–white matter differentiation. When the intracranial pressure (ICP) increases, the brain may suffer from additional secondary injury because of progressive brain herniation possibly leading to brain death.[61] Even when cerebral cytotoxic edema is diffuse, the cerebellum and brain stem are usually spared, which can lead to the so called "white cerebellar sign" on CT (Fig 18). In addition, diffuse hypoxic injury may result from traumatic lesions affecting the chest with possible hypoventilation or cardiac arrest. DWI has proven to be a very sensitive MRI technique to differentiate vasogenic edema from cytotoxic edema. Finally, transcranial Doppler US may be useful to monitor brain perfusion in diffuse cerebral edema.

Herniation (Acute Secondary Injury)

Subfalcine, uncal, or ascending/descending transtentorial herniation can occur if a significant mass effect develops either from the primary injury or secondary from global cerebral edema (Fig 19).[18]

Subfalcine herniation (Fig 19B) is the most common form of brain herniation and results in the displacement of the cingulate gyrus across the midline underneath the falx cerebri. Enlargement of the contralateral ventricle and hydrocephalus because of obstruction of the contralateral foramen of Monro and compression of the anterior cerebral arteries with consequent ischemia are the most important consequences of subfalcine herniation.

Uncal herniation (Fig 19B) consists of a displacement of the medial temporal lobe and hippocampus over the free margin of the tentorium. Effacement of the ambient cistern and compression of the mesencephalon are typical imaging characteristics.

In transtentorial herniation (Fig 19A), brain can herniate either upward or downward according to the primary location of the lesion (posterior fossa or supratentorial cranial vault). Upward herniation results in the displacement of the cerebellum and vermis through the tentorial incisura. Downward herniation can lead to displacement of the supratentorial brain through the tentorial incisura or downward displacement of the cerebellar tonsils through the foramen magnum. It is often a life-threatening condition because of compression and subsequent dysfunction of respiratory and circulatory nuclei in the medulla oblongata. Descending transtentorial herniation can also result in Duret hemorrhages. They are typically located in the ventral and paramedian aspects of the upper brainstem (mesencephalon and pons). Their pathophysiology remains unclear and may be of arterial (from laceration and stretching of pontine branches of the basilar artery) or venous origin (thrombosis and venous infarction).

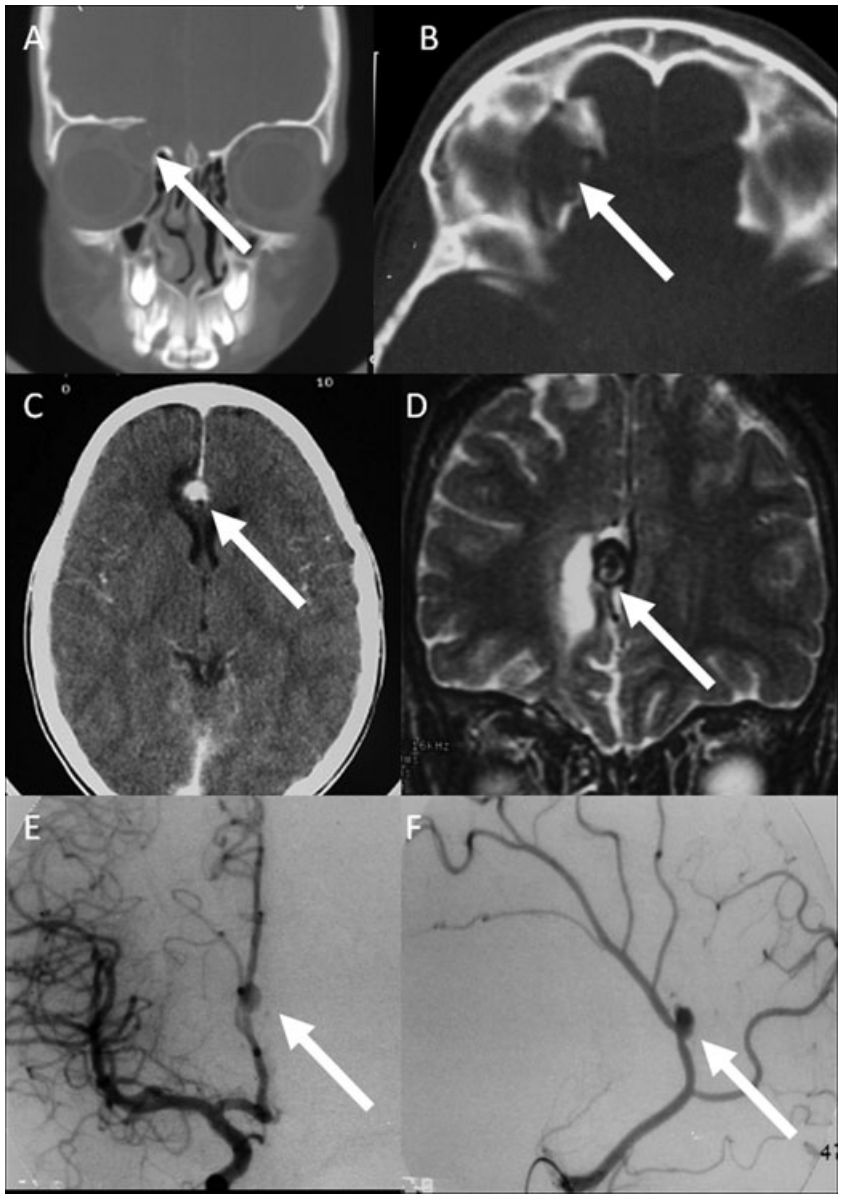

Fig 17. Coronal (A), axial (B), and contrast enhanced axial (D) CT images, coronal T2-weighted MRI (D) and AP and lateral digital subtraction angiography (DSA) (E, F) images of a 6-year-old boy who fell down while running with a pencil in his hand. The pencil had penetrated the right orbital roof (arrow) resulting in a bony defect (A, B) as well as a small brain defect within the right paramedian frontal lobe (C). Contrast enhanced CT showed a posttraumatic contrast enhancing right pericallosal artery aneurysm (arrows). The aneurysm was confirmed by the coronal T2-weighted MRI (hypointense flow void) and DSA (E, F).

Cerebral Ischemia (Acute Secondary Injury)

Cerebral ischemia can result from global cerebral edema/hypoxia, from focal compression of vascular structures because of herniation or mass effect of large hematomas, or from primary vascular injury. As described previously, subfalcine herniation can lead to compression of branches of the anterior cerebral artery, mainly the callosomarginal and anterior perforating arteries with subsequent hypothalamic and cingulate infarction. Uncal herniation can compress the cerebral peduncle against the tentorium (Kernohan's notch) and compression of the posterior cerebral artery may result in subsequent focal ischemia. Tonsillar herniation rarely causes ischemia in the territory of the posterior inferior cerebellar artery.

Secondary global cerebral ischemia can involve one or both hemispheres and may be a significant cause of chronic brain damage in child abuse and usually happens in the context of global anoxia due to chest compression or strangulation.

Hydrocephalus (Acute or Chronic Secondary Injury)

Trauma related hydrocephalus occurs secondary to impaired CSF absorption at the level of the arachnoid granulations (communicating hydrocephalus) or secondary to obstruction of the cerebral aqueduct and fourth ventricular outflow tracts

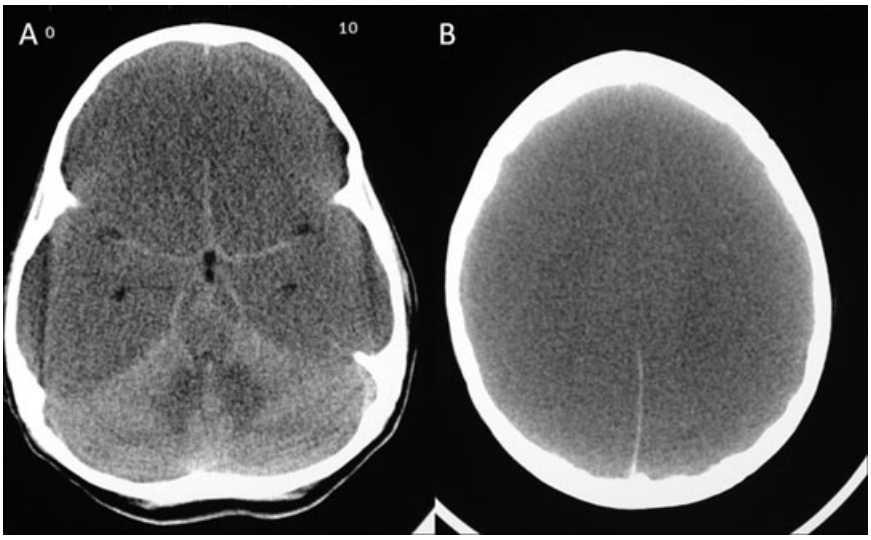

Fig 18. Axial CT images show the classical "white cerebellar sign" (A) in a patient with diffuse cerebral edema (B). The intact, non-ischemic/edematous cerebellum appears hyperdense relative to the edematous, hypoattenuated supratentorial brain. In addition, the cortico–medullary differentiation is obscured and the brain sulci are effaced.

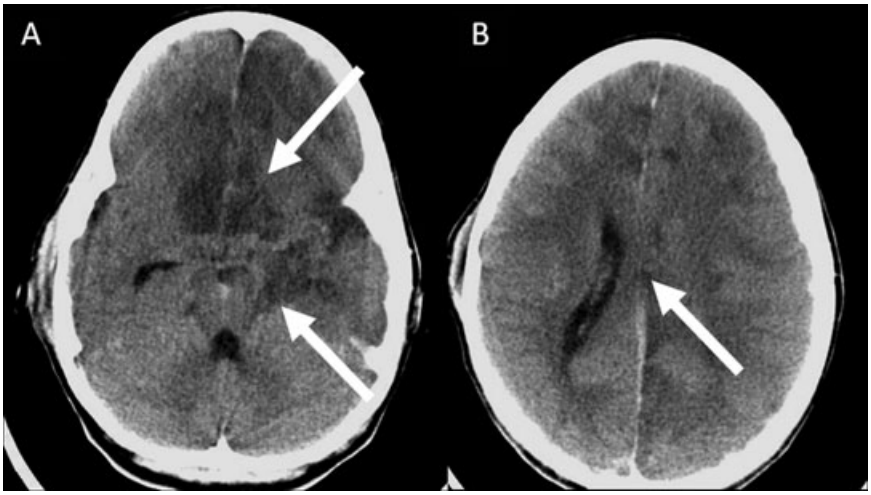

Fig 19. Axial CT images (A, B) show the complications of subfalcine and descending transtentorial herniation because of a left sided convexity SDH. The midline is pushed to the right, the left ventricle is compressed, the right ventricle obstructed. Downward herniation with compression of branches of the anterior circulation and anterior choroid artery have resulted in focal ischemic infarction of the hypothalamus bilaterally, both frontal lobes (left > right) and of the left cingulate gyrus and hippocampus. A focal hypodense cortical contusion is seen in the left temporal lobe.

(noncommunicating hydrocephalus).[1] Blood products are believed to obstruct/occlude the arachnoid granulations as well as the Sylvian aqueduct. Typically, adhesions are seen in the lower third of the Sylvian aqueduct or in the posterior fossa along the outlet channels of the fourth ventricle (foramen of *Magendie* or *Luschka*).

In the acute setting, hydrocephalus can also result from compression of the foramen of Monro, Luschka, or Magendie or because of aqueductal obstruction/compression related to brain herniation. Chronic hydrocephalus is a common complication seen in patients with prior SAH or IVH. T2*-weighted, and SWI images are especially helpful for identifying the hemosiderin depositions along the ventricles and subarachnoid spaces and thin T2-weighted images of phase-contrast imaging may help to find the site of obstruction along the CSF pathways.

CSF Leak (Chronic Secondary Injury)

CSF leaks are well known, rare complications of skull fractures, especially when the frontobasis or skull base is involved. CSF otorrhea and rhinorrhea result from trauma related direct communications between the subarachnoid space and the middle ear or paranasal sinuses. Pneumococcus is the pathogen agent in the majority of the cases.

A CSF leak should be suspected in patients that present with recurrent meningitis and a prior history of moderate/severe head trauma (Fig 20). In addition, high index of suspicion is needed in patients with previous Pneumococcus meningitis.

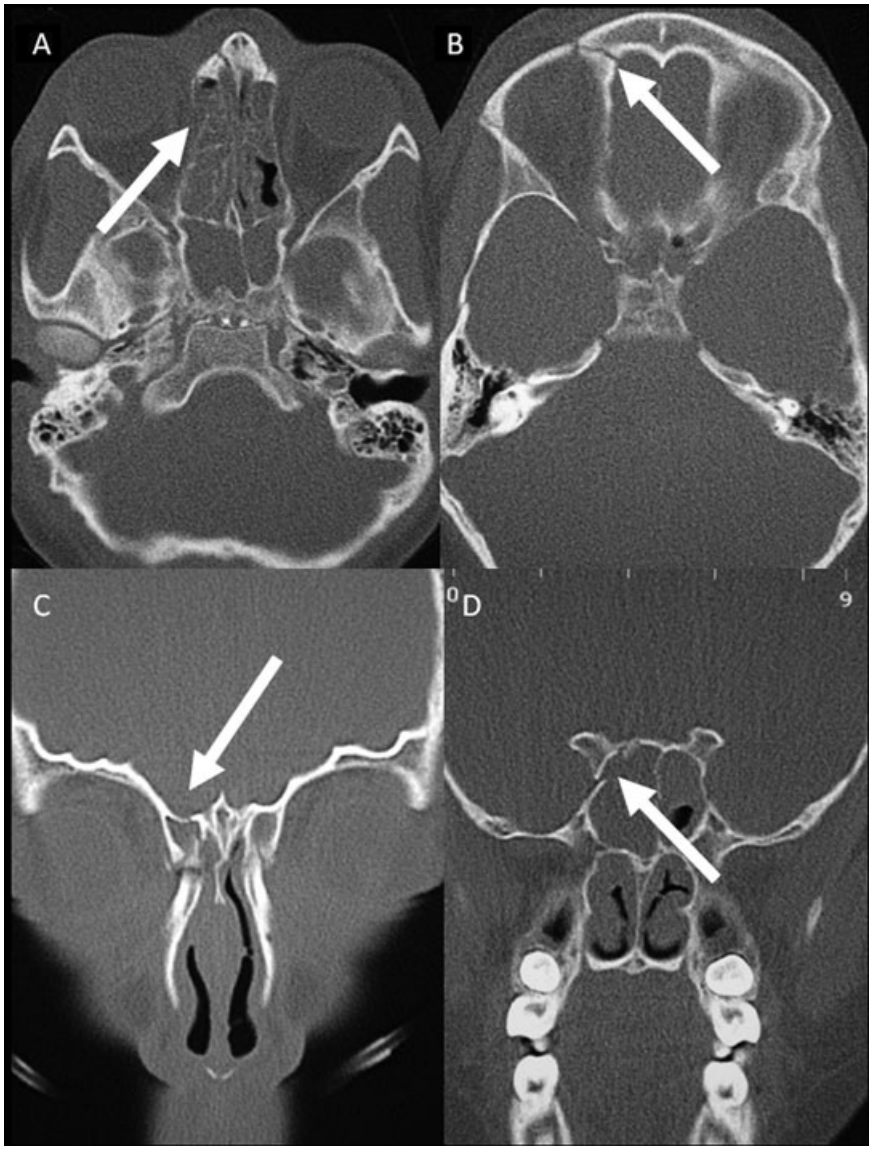

Fig 20. Axial (A, B) and coronal (C, D) CT images show multiple fracture lines (arrows) through the anterior and middle skull base which result in a direct communication between the paranasal sinuses and cranial vault. The left medial orbital wall is depressed, the roof of the left frontal sinus is fractured and the left lateral wall of the sphenoid sinus is also fractured. These open connections may act as port of entry for intracranially extending paranasal sinus infections.

Thin sliced, high resolution CT taking advantage of bone algorithm reconstructions, and multiplanar images is the method of choice to identify osseous defects responsible for a CSF leakage. When present, complementary MRI studies help to exclude brain complications like meningitis, or secondary meningo–encephaloceles through the osseous defect. Stone and colleagues concluded that noncontrast, high-resolution CT allowed to detect an osseous defect in 70% of the patients with CSF leak.[62] CT cisternography and radionuclide cisternography should be reserved for patients in whom high-resolution CT did not identify a bone defect despite a history of recurrent meningitis or for patients with multiple complex skull base fractures or postoperative defects. Defects are occasionally diagnosed with a significant delay, even many years later, when the patient presents with so-called "unexplained" meningitis. Patient records should be carefully studied for prior minor or major TBI. A secondary meningo–encephalitis may be devastating (Fig 21).

Encephalomalacia (Chronic Secondary Injury)

Encephalomalacia can be a late complication from a trauma related tissue injury and is usually characterized by a focal brain defect filled with CSF, frequently accompanied by an area of focal gliosis and volume loss. Symptoms depend on the affected area (eloquent or not). Seizures are especially frequent when the cortex is involved. Knowledge of the trauma mechanism is essential to identify subtle cortical lesions that may go undetected if imaging is not adapted to, or optimized for the expected distribution and location of lesions. Typically, fronto–basal and temporo–basal cortical injuries may be overlooked on CT

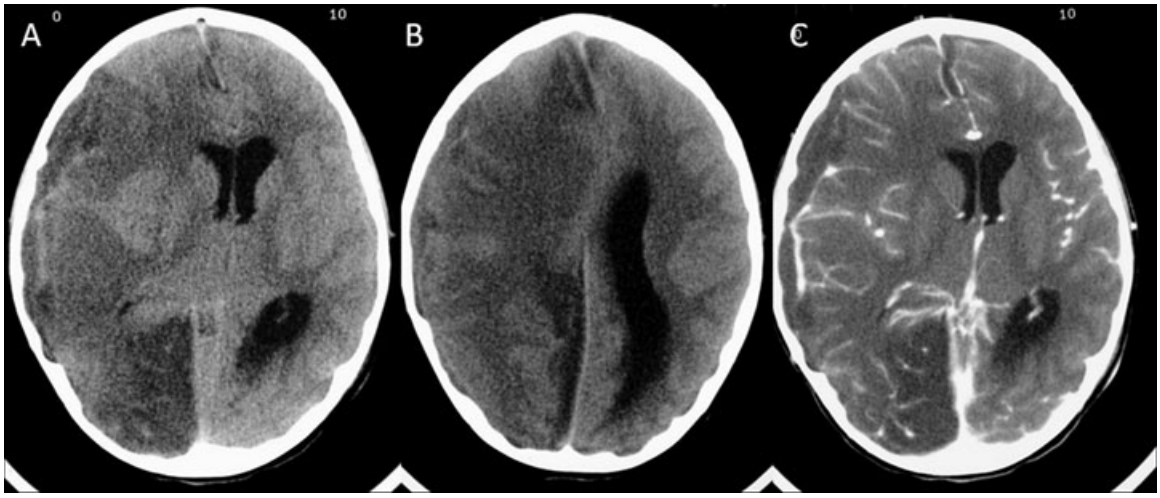

Fig 21. Precontrast (A, B) and postcontrast (C) enhanced axial CT images in a child with severe meningoencephalitis and right sided subdural empyema after previous skull base fracture. In addition, a significant cortical and subcortical edema is seen because of the meningoencephalitis. A significant midline shift is noted with subfalcine herniation of the right lateral ventricle and obstruction of the left lateral ventricle. Focal ischemia in the right occipital lobe resulted from compression of the right posterior cerebral artery.

because of the beam hardening artifacts from the adjacent, irregular skull base and partial volume effects. Additional imaging planes both in CT and MRI, may show subtle lesions that better explain neurological symptoms.

Age Related Trauma

Birth Trauma

Birth trauma represents a unique type of neonatal TBI. It usually results from instrumental delivery, which may be performed for fetal or maternal indications. The incidence of instrumental vaginal delivery varies between countries, and the use of forceps has significantly decreased in the last years in favor of vacuum extraction or cesarean section.[63] Scalp, skull, and intracranial injuries may arise in this setting.

Scalp Injury

The majority of injuries because of vacuum extraction are minor and of no clinical significance. There are three principal patterns of extracranial hemorrhage associated with birth trauma: caput succedaneum, subgaleal hemorrhage, and cephalohematoma.

Caput succedaneum is the most common complication, which typically occurs during vaginal delivery, and consists of hemorrhage and edema within the fibrofatty layer just beneath the neonatal skin. It is a soft palpable mass along the vertex, extending across the midline and over the sutures. It is usually an incidental-imaging finding. These lesions typically resolve within the initial days of life without complications.

Subgaleal hemorrhage (Fig 22) is an accumulation of blood in the subaponeurotic space between the periosteum of the skull and the galea aponeurotica. This space extends to the orbital margins anteriorly, the nuchal ridge posteriorly, and the temporal fascia laterally.[64] Subgaleal hematomas are superficial to the temporal muscle, consisting of a firm palpable mass, and are asymptomatic in the majority of cases. Rarely, they can be large, leading to significant blood loss and hypovolemia.[65] Surgery may be required to cauterize the bleeding vessels. Unlike cephalohematomas, subgaleal hematomas can cross-suture lines.[19] They usually resolve within a couple of weeks.

Cephalohematomas are subperiosteal in location beneath the temporal muscle. They are seen in about 1–2% of spontaneous vaginal deliveries, in 6–10% of vacuum extractions, and in 4% of forceps deliveries.[65] They are usually associated with forceps or vacuum extractions and are secondary to the rupture of bridging blood vessels. They present as a firm mass, do not cross sutures and are usually smaller than subgaleal hemorrhages. They can calcify resulting in a hard disfiguring mass on follow-up, but usually resolves within a few weeks and rarely require treatment. Infection of a cephalohematoma is a rare complication.

Skull Injury

Skull fractures (Fig 22) rarely result from birth trauma. The incidence of skull fractures after vacuum extractions is higher in newborns from nulliparous mothers compared to multiparous mothers. Skull fractures can be linear, affecting the parietal bones, or depressed, resembling the so-called "ping-pong ball-type" fracture.[66] This name is because of an inward buckling of the bone without loss of the bony cortical limits. The most common location is the parietal bone. A skull fracture must be suspected if a cephalohematoma or SAH is present.[66] The overlapping of calvarial bones at the sutures is a unique skull injury in newborns. It usually resolves without complication with progressive growth.

Intracranial Injury

Symptomatic intracranial hemorrhages (Fig 23) are rare and occur in approximately 5–6 neonates per 10,000 live births.[63] Asymptomatic intracranial hemorrhages are, however, more common and recently a prevalence of 26% for asymptomatic intracranial hemorrhages in vaginal births has been reported.[67]

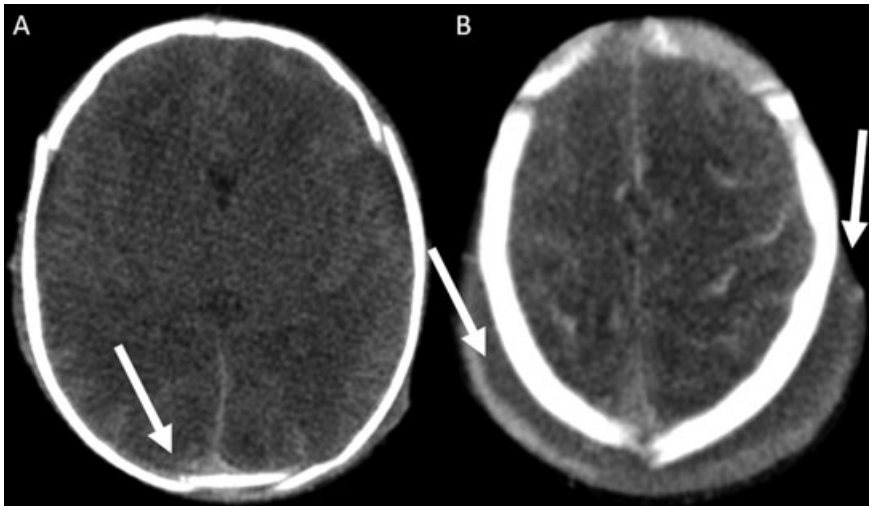

Fig 22. Axial noncontrast enhanced axial CT images (A, B) of a newborn baby (35 week gestation). CT shows parietal bone fractures, subgaleal hematoma crossing the suture lines, subarachnoid blood within the sulci near the vertex and mild global edema as complication of a failed vacuum extraction.

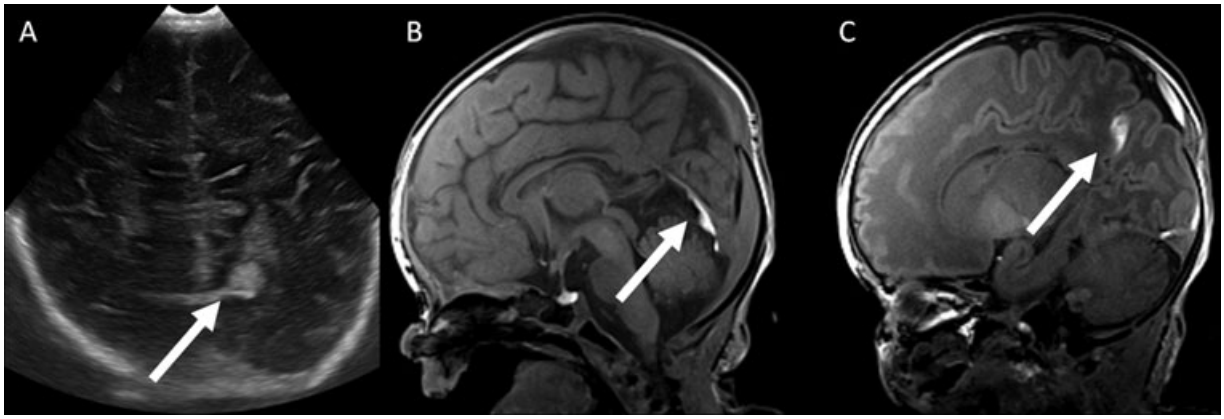

Fig 23. Coronal head ultrasound (A) shows a birth related focal, hyperechogenic left occipito–parietal subarachnoid hemorrhage (arrow) and an adjacent cortical contusion in a several days old neonate. Sagittal T1-weighted MRI shows a small T1-hyperintensee infratentorial subdural hematoma (B, arrow) that follows the contour of the tentorium as well as the focal subarachnoid hemorrhage along the parieto–occipital sulcus (C, arrow) next to the cortical contusion. In addition, a small T1-hyperintense epidural hematoma is seen along the supratentorial parieto–occipital bone.

Intracranial hemorrhage is mostly related to abnormal labor and not to the mode of delivery.[68]

Epidural hemorrhage is rare in neonates, accounting for 2% of all intracranial hemorrhages, and can result from injury of the middle meningeal artery, being frequently associated with cephalohematomas or skull fractures.[63]

Subdural hemorrhage (Fig 23) is the most common intracranial hemorrhage after instrumental birth,[64] accounting for 73% of all cranial brain injuries in term neonates according to a study by Pollina and colleagues.[69] There are four common intracranial lesions associated with SDHs in this age group: tentorial laceration, occipital osteodiastasis, falcine laceration, and rupture of bridging cortical and meningeal veins.[70,71] They usually subside within 4 weeks.

Occipital osteodiastasis occurs when there is a traumatic tear and separation along the synchondrosis between the squamosal and the lateral portions of the occipital bone.[19,70,72] This may lead to the rupture of the occipital sinus or direct cerebellar trauma and brain stem compression.[63] Feto–maternal/pelvic disproportion can be an important factor contributing to this injury. Laceration of the falx occurs at the junction of the falx and the tentorium, with tearing of the inferior sagittal sinus resulting in SDH along the inferior aspect of interhemispheric fissure.[19]

SAH (Fig 23) is the second most common intracranial hemorrhage after instrumental deliveries and, unlike SAH in adults, usually results from rupture of small bridging veins of the leptomeninges.

Nonaccidental Head Injury

It is estimated that 6–10% of young children brought to the emergency department (ED) with traumatic injury are victims of child abuse. The population-based estimate of inflicted TBI

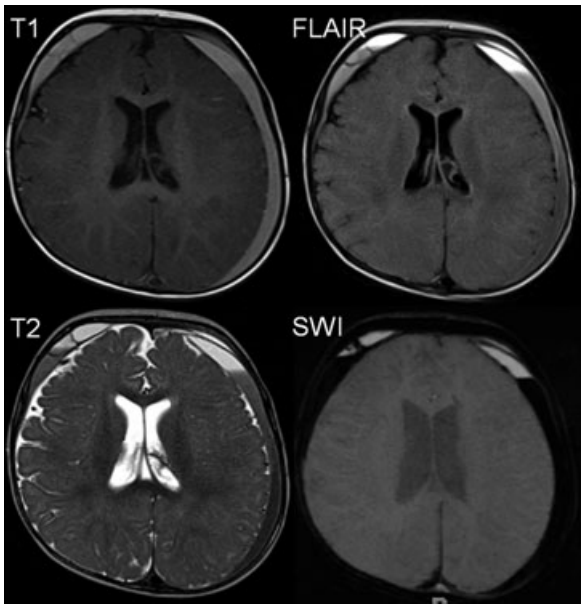

Fig 24. Different MRI sequences (T1, FLAIR, T2, SWI) can help to better characterize, grade, and possibly date subdural effusions/hematomas. This valuable information may assist diagnosis of nonaccidental TBI.

Fig 25. High resolution T2 (A) and T1-weighted (B) orbital MRI may directly show posttraumatic retinal hemorrhages related to vigorous shaking of a newborn. These findings may render valuable, additional evidence suggesting nonaccidental injury.

in children less than 1 year of age leading to hospitalization in an intensive care unit is 30 per 100,000 children yearly.[73] The median age for inflicted TBI is approximately 2–4 months of age.[73] Apart from being the most common cause of traumatic death in the first 2 years of life, nonaccidental head injury is responsible for significant neurological morbidity.

Nonaccidental head trauma is a specific issue in children and a major concern in this age group. It has been extensively studied in the literature in the past years. Because this paper is focused on the imaging aspects of traumatic injuries in children and differences of those from adults, a detailed discussion about this unique type of injury is beyond the scope of this review.

Nonaccidental injury should however be suspected when there is a discrepancy between the trauma history and encountered imaging findings; if there are hemorrhages of different ages (Fig 24); if there are unusual findings; when retinal hemorrhages (Fig 25) are present (they should be confirmed by an ophthalmological examination, which is mandatory in the suspicion of nonaccidental head trauma); or if the child has evidence of overall poor care. Clinical presentation may be subtle and include various degrees of decreased levels of consciousness, irritability, respiratory distress, poor feeding, and seizures. The radiologist should alert the clinician if there is the slightest suspicion of nonaccidental injury to prevent additional injury on follow-up.

Mechanisms of Injury

Either abuse or an accident may cause head injury, and from the biomechanical point of view, it is not possible to differentiate a deliberate impact from an accidental fall under the same circumstances because the mechanism can be identical.[74]

The mechanisms of injury in abusive head trauma typically involve blunt impact (such as striking the child with an object, dropping, or throwing of the child) and/or acceleration/deceleration injuries (shaking of a neonate).[75] Another mechanism, frequently underestimated, is neck strangulation that can lead to asphyxia and compression of major cervical arterial vessels with consequent brain ischemia (Fig 26). Moreover, chest compression may occur during the forceful shaking of a baby resulting in additional hypoxic brain injury next to the classical shear injuries.

The most common mechanism of injury leading to the extraaxial hemorrhages in shaking-impact syndrome is thought to be the rupture of parasagittal draining veins resulting from angular accelerations/decelerations. Under these circumstances, the brain rotates relative to the dura and skull, placing the parasagittal draining veins on stretch until they avulse from the cortical surface. This results in a typical hemorrhage pattern into both subdural and subarachnoid spaces.[38]

Melvin and colleagues summarized in 1993 the various mechanisms of brain injury as follows: (1) direct contusion from skull deformation, (2) brain contusion from motion of the brain relative to the internal fractured skull, (3) reduced blood flow caused by neck or chest compression with infarction, (4) indirect contusion of the brain opposite to the side of impact (contrecoup), (5) tissue strain produced by the relative motion of the brain with respect to the skull or hemisphere, and (6) rupture or tearing of the blood vessels coursing between the brain and the dura mater.

Types of Injury

Nonaccidental head trauma is responsible for several types of injury, most of them discussed earlier. Some are typical or

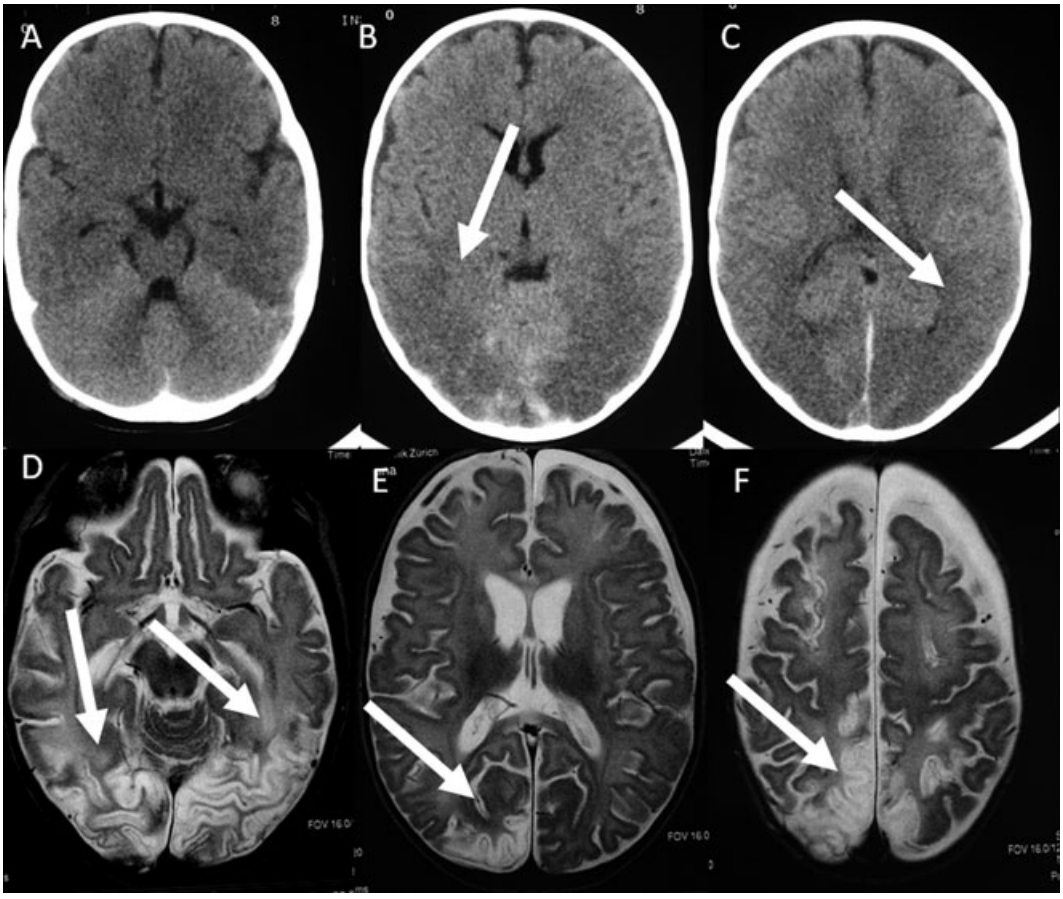

Fig 26. Acute axial CT (A–C) and follow-up T2-weighted (D–F) MR images of a child who suffered from nonaccidental injury with simultaneous strangulation. The supratentorial brain is edematous (hypodense, arrows) relative to the preserved cerebellum (white cerebellar sign) suggesting global brain edema (A–C). Hyperdense subdural blood is seen along the falx and tentorium. Follow-up MRI (D–F) shows extensive global and partially focal tissue loss. The occipital lobes and hippocampi are predominantly injured with laminar necrosis. The CSF spaces are diffusely widened because of global tissue loss.

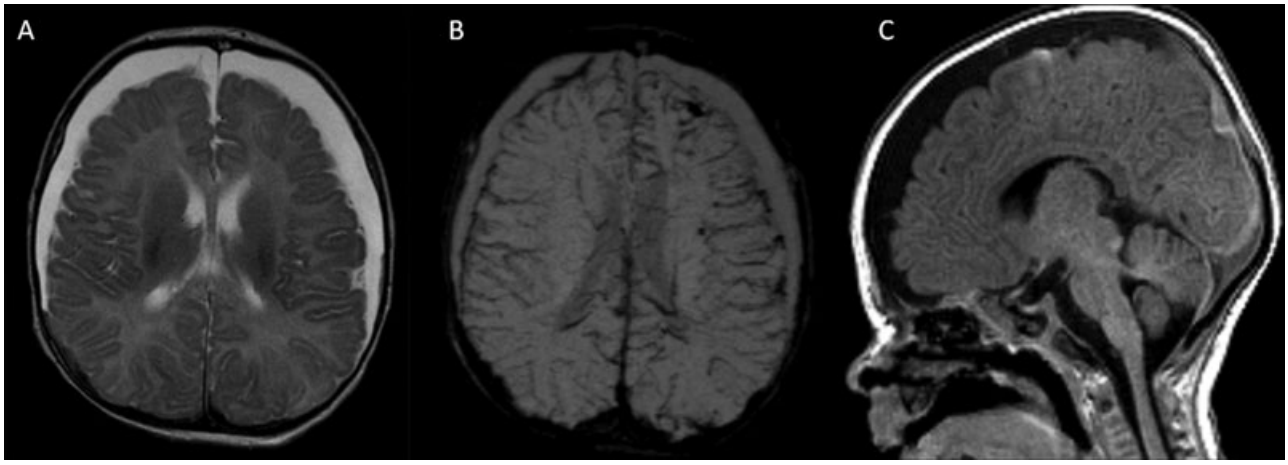

Fig 27. Axial T2-weighted (A), SWI (B), and sagittal T1-weighted (C) MR images of a child with chronic T2-hyperintense subdural hygromas as complication of child abuse. SWI shows small hypointense hemosiderin depositions (B) along the compressed brain surface. The hemorrhagic components are mildly T1-hyperintense.

specific for this kind of trauma and deserve further characterization.

Scalp abrasions, swelling, and bruising result from direct impact. A soft surface may dissipate the force of impact to the head, resulting in brain injury without visible signs. In a study conducted by Duhaime and colleagues, all children who died from abusive head trauma had evidence of blunt head trauma, but in several of the cases, evidence of impact was found only at autopsy.[76]

Skull fractures result from direct impact. They are usually linear or depressed. Basilar fractures result from propagated stress waves. Correct estimation of the age of skull fractures is difficult, but the presence of an overlying soft tissue swelling indicates that the injury is acute. Multiple fractures, bilateral fractures, and fractures crossing sutures are more commonly associated with abuse cases.[77] Parietal and occipital bones are most often involved in nonaccidental trauma, and depressed occipital fractures have been found to be highly suspicious for abuse.[78]

SDH (Fig 24) and SAHs are the most common findings in nonaccidental head injury. SDH are commonly seen in the occipital region and posterior interhemispheric fissure.[79] SDH can result from impact injuries or acceleration/deceleration forces. MRI is particularly helpful for detecting hemorrhages of different ages. Dating of hemorrhages, however, remains a difficult task and radiologists should be aware of this point particularly in the context of legal proceedings.[80]

SDH are generally associated with bad outcome in child abuse because they may result from or indicate acceleration/deceleration forces and are often associated with DAI. MRI and superficial US may have a crucial role in helping to distinguish SDH from dilated subarachnoid spaces that may be seen as a normal variant in children (Fig 27).

EDHs are less common than SDH and SAH in child abuse. They result from an impact injury and are often associated with a skull fracture.

Intracerebral injuries include contusions, hematomas, ischemic lesions, and DAI. CT is the diagnostic study of choice for evaluating acute traumatic head injury; however, it has low sensitivity for the detection of acute nonhemorrhagic lesions and white matter shearing injuries. MRI is especially helpful in depicting these lesions when there is a disproportion between the severity of the neurological symptoms and encountered CT imaging findings. Moreover, DWI can be especially useful in detecting acute ischemic changes as well in differentiating vasogenic edema from cytotoxic edema.

Cortical contusions are the result of direct impact and are mostly located in the frontal and temporal lobes, close to the skull base. There are no differences in the neuroimaging findings between contusions resulting from accidental or abusive injuries.

DAI is the hallmark injury of acceleration/deceleration forces, and are typically seen in shaken baby injuries. There are no recognizable differences between DAI injuries in accidental and abuse injuries.

Further diagnostic work-up should include a skeletal survey and possible additional cross-sectional studies to exclude other solid organ injury.

Conclusion

A detailed knowledge and familiarity with the various, child-specific TBI mechanisms and patterns of injury is mandatory as well as a proper understanding of the unique facial, skull, and brain properties of children. Primary and secondary injuries are responsible for a wide variety of acute and chronic lesions as well as delayed complications. Pediatric TBI differs significantly from adult TBI. In addition, the radiologist should be familiar with several unique trauma patterns like nonaccidental injuries and birth related injuries. High-end CT and/or MRI should be performed to guide and monitor treatment. In addition, anatomical and functional imaging data may better predict functional outcome.

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