Imaging of Head Trauma

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Traumatic brain injury (TBI) is a leading cause of mortality and morbidity in the world’s population, especially those under age 44.1 In the United States alone, the cost of head trauma has been estimated to be over 40 billion dollars annually.2 The National Center for Injury Prevention and Control has estimated that approximately 2% of the entire population of the USA currently lives with disabilities caused by TBI.3 TBI accounts for more than 500,000 emergency department visits annually.4 The etiologies of head trauma are usually associated with the patient age. In the elderly population, accidental falls are the most common causes. Motor vehicle crashes are common culprits in young patients. In children, abuse and neglect are common reasons.

TBI can be classified into primary and secondary injuries. Primary lesions are the direct result of trauma to the head, and secondary lesions arise as complications of primary lesions. Clinically, this classification is important because secondary injuries can be preventable, whereas primary injuries, by definition, have already occurred by the time the patient first presents for medical attention. TBI can be further divided according to location (intra- or extra-axial), mechanism (penetrating/open or blunt/closed), and clinical severity (minor, mild, moderate, or severe). The severity of head injury is usually based on the Glasgow Coma Scale (minor: GCS ≥ 15; mild: GCS ≥ 13; moderate: 9 ≤ GCS ≤ 12; severe: 3 ≤ GCS ≤ 8).5 Primary extra-axial lesions include epidural, subdural, subarachnoid, and intraventricular hemorrhage. Primary intra-axial lesions include cortical contusions, intracerebral hematomas, axonal shearing injuries, gray matter injury, and vascular injury. Acute and subacute secondary injuries include cerebral edema, ischemia, and brain herniation. Chronic secondary lesions include hydrocephalus, the cerebrospinal fluid (CSF) leak, leptomeningeal cyst, and encephalomalacia. These lesions will be discussed in further detail following a discussion about different methods for imaging TBI.

Imaging Techniques

Skull films are poor predictors of intracranial pathology and should not be performed to evaluate adult TBI.5-8 In the low-risk patient, skull films rarely demonstrate significant findings. In the high-risk patients, the lack of abnormality on skull films does not exclude major intracranial injury.9 Patients who are at high risk for acute intracranial injury must be imaged by CT. The “scout view” that is obtained with all CT exams can be used as a “pseudoskull film.” In cases of suspected child abuse, a skull series may occasionally identify a fracture that is not identified on the concomitant CT examination. While this finding may not have surgical importance, it, nevertheless, warrants removal of the child from the hostile environment.

Computed tomography (CT), in the setting of acute trauma, is indicated for severe TBI (GCS < 8), persistent neurologic deficit, antegrade amnesia, unexplained asymmetric pupillary response, loss of consciousness more than 5 minutes, depressed skull fracture, penetrating injury, or bleeding diathesis or anticoagulation therapy.10 The goal of imaging is to identify treatable injuries to prevent secondary damage. In the acute setting, CT is the modality of choice because it is fast, widely available, and highly accurate in the detection of skull fractures and intracranial hemorrhage. Life-support and monitoring equipment can easily be accommodated in the CT scanner suite. In addition, CT is usually superior to MR in revealing skull fractures and radio-opaque foreign bodies. With modern CT scanners, contiguous 3.75- or 5-mm sections from the skull base to the vertex can be obtained in less than 10 minutes. Thinner 1- or 2.5-mm sections are used to evaluate the orbits, maxillofacial structures, and skull base. With recent advances in multidetector CT (MDCT), thin cross-sectional slices can be performed which allowed for high-quality three-dimensional reconstruction. Thinner slices also improve the diagnostic accuracy of CT in the evaluation of maxillofacial, orbital, and temporal bone fractures. Intravenous contrast administration should not be performed because it can both mask and mimic underlying hemorrhage.

CT images must be reviewed using multiple windows and levels. With the widespread utilization of picture archiving and communication system (PACS), most imaging interpretation is now performed on computer workstations, allowing...
for rapid setting of windows and levels. A narrow window width (W: 80, L: 40) is used to evaluate the brain. A slightly wider window width (W: 150, L: 75) is used to exaggerate contrast between extra-axial blood and the adjacent skull. An even wider window (W: 2500, L: 500) is used to evaluate the osseous structures (Figs. 2 and 3).

Magnetic resonance imaging (MRI) is recommended for patients with acute TBI when the neurologic findings are unexplained by CT. MR is also the modality of choice for subacute or chronic injury. MR is usually comparable to CT in the detection of an acute epidural and subdural hematoma. However, MR is more sensitive to subtle extra-axial collections, nonhemorrhagic lesions, brainstem injuries, and subarachnoid hemorrhage (SAH) when using fluid-attenuated inversion recovery (FLAIR). Fluid-attenuated inversion recovery imaging improves the conspicuity of focal gray matter abnormalities (eg, contusions), white matter abnormalities, shear injuries, and SAH by eliminating (or “nulling”) the bright cerebrospinal fluid signal. Sagittal and coronal FLAIR images are particularly helpful in the detection of diffuse axonal injury (DAI) involving the corpus callosum and the fornix, two areas that are difficult to evaluate on routine T2-weighted images. On a more cautious note, abnormal high signal in the sulci and cisterns of ventilated patients receiving a high inspired oxygen fraction (>0.60) have been observed and should not be mistaken for hemorrhage.

Gradient-echo imaging is highly sensitive to local magnetic inhomogeneity caused by the presence of blood. Hemosiderin, a breakdown product of blood, is ferromagnetic. The presence of hemosiderin alters the local magnetic susceptibility of tissue, resulting in areas of signal loss on gradient-echo T2*-weighted images. Because hemosiderin can persist indefinitely, its detection on gradient-echo T2*-weighted images allows for improved evaluation of remote injury. However, due to the inherent inhomogeneity adjacent to the paranasal sinuses and mastoid air cells, gradient-echo images are limited in the evaluation of cortical contusions of the inferior frontotemporal lobes. This limitation is even more problematic at high magnetic field strength unless parallel imaging is used.

Diffusion-weighted imaging (DWI), which measures the random motion of water molecules in brain tissue, has improved the evaluation of TBI. Through its superior sensitivity to foci of acute shearing injury, DWI has especially improved detection of DAI. DWI reveals more DAI lesions than fast spinecho T2-weighted or gradient-echo T2*-weighted images in patients imaged within 48 hours of injury. The apparent diffusion coefficient (ADC), which measures the magnitude of water diffusion averaged over a three-dimensional space, is often reduced in chronic DAI. The fractional anisotropy (FA), which measures the preferential motion of water molecules along the white matter axons, is frequently reduced in chronic DAI. Tissue anisotropy is exploited in the new technique of diffusion tensor imaging (DTI).

Imaging Findings

Scalp Injury

When reviewing CT scans for head trauma, begin by examining the extracranial structures for evidence of soft-tissue injury and/or radio-opaque foreign bodies. Scalp injury is a reliable indication of the site of impact. Scalp injury includes soft-tissue lacerations, subgaleal hematoma, cephalohematoma, and residual foreign bodies. The subgaleal hematoma is far and away the most common manifestation of scalp injury. It can be recognized as focal soft-tissue swelling located beneath the subcutaneous fibrofatty tissue and above the temporalis muscle and calvarium (Fig. 1).

Skull Fractures

Nondisplaced linear fractures of the calvarium can be difficult to detect on CT when the fracture plane is parallel to the plane of section. Fortunately, isolated linear skull fractures usually do not require treatment unless they are associated with an epidural hematoma. Surgical treatment is usually only indicated for depressed and compound skull fractures, both of which are easily detectable on CT (Fig. 2). Depressed skull fractures can be associated with an underlying contusion; therefore, attention to the subadjacent parenchyma is essential (Fig. 14).

Thin-section (1-mm) CT using a bone algorithm is recommended for the evaluation of fractures in the skull base, orbit, or facial bones. Thin sections are helpful for evaluating the degree of comminution and depression of bone fragments.

Figure 1 Subgaleal, subdural, and epidural hematoma. Axial CT image demonstrates a left parietal subgaleal hematoma (arrow). Note that it is superficial to the temporalis muscle (*). Subjacent to the scalp injury is a biconvex, hyperdense, extra-axial collection consistent with an acute epidural hematoma (#). Anterior to the epidural hematoma is a crescent-shaped hyperdense collection consistent with an acute subdural hematoma (arrowhead).
MDCT with 3D reconstruction can elegantly display complex fractures and they can be generated expeditiously with sophisticated algorithms.19

Temporal Bone Fractures

Pneumocephalus, opacification of the mastoid air cells, and fluid in the middle ear cavity should always raise concern for a temporal bone fracture. Thin-section (1 to 1.5 mm) axial and direct coronal CT imaging with bone algorithm reconstruction is recommended for the evaluation of temporal bone fractures. With MDCT, thinner section axial imaging can be performed, and coronal reformats may be adequate for interpretation without the need for direct coronal imaging, which can be difficult in intubated or mentally altered patients, or in patients with suspected cervical spine injury.

Fractures are classified as longitudinal or transverse, depending on their orientation relative to the long axis of the petrous bone. Longitudinal fractures parallel the long axis of the petrous pyramid, and transverse fractures are perpendicular to the long axis of the petrous bone. Longitudinal temporal bone fractures (Fig. 3) usually result from direct impacts to the side of the head. They represent more than 70% of temporal bone fractures.20 Complications include conductive hearing loss from dislocation or fracture of the ossicles.

Figure 2 Depressed skull fracture with an associated acute epidural hematoma (EDH). (A) Axial CT scan displayed in “bone window” shows a right frontal depressed skull fracture (arrowhead). Skull fractures can be associated with an underlying epidural hematoma (B, arrow) and/or contusion, especially depressed comminuted fractures.

Figure 3 Longitudinal temporal bone fracture. Axial CT scan shows a longitudinal left temporal bone fracture (arrowheads) with opacification of the mastoid air cells. Diastasis of the left lambdoid suture (open arrow) and fractures of the sphenoid sinus (curved arrow) and left lateral orbital wall (arrow) are also present. (Reprinted with permission from Gean AD: Imaging of head trauma. Philadelphia, PA, William & Wilkins-Lippincott, 1994, p 63.)
otorhinorrhea from CSF leak, and facial nerve palsy. Transverse temporal bone fractures typically derive from direct impacts to the occiput or frontal region. Complications include sensorineural hearing loss, vascular injury, and perilymphatic fistula. Vertigo, nystagmus, and facial palsy are also common complications. Mixed or complex temporal bone fractures involve a combination of fracture planes. They typically result from severe crushing blows to the skull. Patients with mixed temporal bone fractures have a high incidence of associated intracranial injury.

**Primary Head Injury**

**Extra-Axial Injury**

*The epidural hematoma (EDH)* occurs in the potential space located between the inner table of the skull and the dura (Fig. 2). The developing hematoma dissects the dura from the inner table of the skull, forming an ovoid mass that displaces the adjacent brain. They frequently result from a skull fracture, usually in the temporal squamosa, that disrupts the middle meningeal artery. In children, they may occur from stretching or tearing of meningeal arteries without an associated fracture. Venous EDHs are less common than arterial EDHs and tend to occur at three classic locations: the posterior fossa from rupture of the torcular or transverse sinus; the middle cranial fossa from disruption of the sphenoparietal sinus; and the vertex from injury to the superior sagittal sinus. Venous EDHs can be difficult to diagnose on axial imaging but can be readily confirmed on coronal reformatted images.

On CT, the acute EDH appears as a well-defined, hyperdense, biconvex, extra-axial collection (Fig. 2). It is usually associated with an overlying skull fracture. Mass effect with sulcal effacement and midline shift is frequently seen. Because the EDH is located in the potential space between the dura and inner table of the skull, it rarely crosses cranial sutures because the periosteal layer of the dura is firmly attached at sutural margins (Fig. 4). However, at the vertex, where the periosteum that forms the outer wall of the sagittal sinus is not tightly attached to the sagittal suture, the EDH can cross midline. An important imaging finding that predicts rapid expansion of an arterial EDH is the presence of low-attenuation areas within the hyperdense hematoma (the
so-called “swirl sign”), thought to represent active bleeding (Fig. 5). It is an ominous sign that needs to be followed closely.

Subdural hematomas (SDH) are generally venous in origin. They usually arise from laceration of bridging cortical veins during sudden head deceleration. Occasionally, they may also result from disruption of penetrating branches of superficial cerebral arteries. Because the inner dural layer and arachnoid are not firmly attached, SDHs are frequently seen layering along the entire hemispheric convexity from the anterior falx to the posterior falx (Figs. 6 to 10). The SDH is uncommonly seen in association with DAI. Because of the prominent extra-axial space in the elderly resulting from cerebral atrophy, increased motion between the brain parenchyma and the calvarium is permitted, and an increased incidence of SDH in these patients has been observed. Other causes of the SDH include rapid decompression of obstructive hydrocephalus, and injury to pial vessels and pachymeningeal granulations. In rapid decompression of the hydrocephalus, the brain surface recedes from the dura quicker than the brain parenchyma can re-expand after being compressed by the distended ventricles.

On CT, the acute SDH appears as hyperdense, homogeneous, crescent-shaped, extra-axial collection (Fig. 1). Most are supratentorial and located along the convexity. They are also frequently identified along the falx and tentorium. Because the SDH is often associated with parenchymal injury, the degree of mass effect seen frequently appears more severe relative to the size of the collection.

The attenuation (density) of an acute SDH initially increases because of clot retraction. The acute SDH is hyperdense, measuring 50 to 60 Hounsfield Units (HU), relative to normal brain, which measures 20 to 30 HU. The density will progressively decrease as protein degradation occurs within the hematoma. Rebleeding during evolution of a SDH appears as a heterogeneous mixture of fresh blood and partially liquefied hematoma (Fig. 6). A sediment level or “hematocrit effect” may be seen either from rebleeding or in patients with clotting disorders (Fig. 7). The chronic SDH has a low-attenuation value similar to, but slightly higher than, CSF (Fig. 8). It can be difficult to distinguish from prominent subarachnoid space in patients with cerebral atrophy. In these patients, intravenous contrast administration can be helpful by demonstrating an enhancing capsule or displaced cortical veins.

A small thin convexity SDH can be difficult to appreciate adjacent to the hyperdense skull unless the images are viewed with a “wide window.” With most viewings and interpretations now performed on computer workstations, the radiologist can adjust the window setting readily to avoid this potential pitfall (Fig. 9). During the transition from the acute to the chronic SDH, an isodense phase occurs, usually between

![Figure 6](image-url) Rebleeding during evolution of a SDH (surgically confirmed). Axial CT image demonstrates a large, right, holohemispheric, extra-axial, fluid collection with mixed hyperdense (acute) and isodense (hyperacute) blood. An acute-on-chronic SDH would have a similar appearance.

![Figure 7](image-url) SDH with hematocrit level. Axial CT image shows a large, right, holohemispheric, extra-axial fluid collection. The collection has a hyperdense “sediment” level (*). The attenuation gradient is secondary to the presence of hemorrhage of different ages. There is effacement of the frontoparietal sulci and mild midline shift resulting from the SDH mass effect.
1 to 3 weeks after the acute event, depending on the patient’s hematocrit level, clotting capability, and presence or absence of rebleeding. Recognition of indirect imaging findings, such as effacement of sulci, displacement of gray matter with white matter “buckling,” and midline shift on a noncontrast CT scan can avoid this common pitfall.

The MR appearance of a SDH also evolves over time, depending on the biochemical state of hemoglobin. The acute SDH is isointense to brain on T1-weighted and hypointense on T2-weighted images. During the subacute phase, when the subdural hematoma may be isodense or hypodense on CT scans, T1-weighted images demonstrate high signal intensity due to the presence of methemoglobin in the subdural collection. The chronic SDH appears hypointense on T1-weighted and hyperintense on T2-weighted images relative to normal brain (Fig. 10). The signal intensity of chronic SDH is slightly higher than CSF signal intensity on T1-weighted, FLAIR, and proton-density T2-weighted imaging. Because of its multiplanar capability, MR is useful in identifying convexity and vertex hematomas that might not be detected on axial CT scans because of the similar attenuation of the adjacent bone.

Traumatic subarachnoid hemorrhage (SAH) can result from (a) the disruption of small pial vessels; (b) extension into the subarachnoid space by a contusion or hematoma; or (c) diffusion of intraventricular hemorrhage. SAH is very common with TBI, but it rarely causes mass effect. On CT, SAH appears as linear or serpentine areas of high attenuation that conform to the morphology of the cerebral sulci and cisterns.
Common sites for SAH include the sylvian and interpeduncular cisterns. The greatest accumulation of SAH tends to occur contralateral to the site of impact (ie, contrecoup). Subarachnoid hemorrhage along the convexity or tentorium can be difficult to differentiate from a SDH. A useful clue is the extension of the SAH into adjacent sulci. Occasionally, “effacement” of sulci due to the presence of intrasulcal SAH may be the only imaging clue of the presence of SAH. In patients who are found unconscious after an unwitnessed event, the detection of SAH in key cisterns (basilar, sylvian, and circle of Willis) may indicate a ruptured aneurysm, rather than trauma. In such cases, contrast-enhanced CT angiography (CTA) is particularly helpful in this situation. In this case, CTA shows a right middle cerebral artery bifurcation aneurysm (D). The aneurysm appears pointed, suggesting recent rupture. Intraventricular hemorrhage (IVH) is seen as a high attenuation fluid level within the occipital horn of the left lateral ventricle (B, white arrowhead).

Acute subarachnoid hemorrhage may be more difficult to detect on conventional MR than on CT because it can be isointense to brain parenchyma on T1- and T2-weighted images. However, FLAIR has been shown to be more sensitive than CT in detecting acute subarachnoid hemorrhage in an animal model, especially when a high volume (1 to 2 mL) is present. Subacute subarachnoid hemorrhage may be better appreciated on MR because of its high signal intensity when the blood is isointense to CSF on CT. Chronic hemorrhage on MR scans may show hemosiderin staining in the subarachnoid space, which appears as areas of decreased signal intensity on T1- and T2-weighted sequences (“superficial hemosiderosis”). Hemosiderins are best detected on gradient-echo T2*-weighted images. SAH may lead to communicating hydrocephalus by impeding CSF resorption at the level of arachnoid villi.

Traumatic intraventricular hemorrhage (IVH) can also occur by one of three mechanisms. First, it can result from rotationally induced tearing of subependymal veins along the surface of the ventricles. Another mechanism is by direct extension of a parenchymal hematoma into the ventricular system. Third, IVH can result from retrograde flow of SAH into the ventricular system via the fourth ventricular outflow foramina (the reverse mechanism in which IVH can extend into the subarachnoid space). Patients with IVH are at risk for developing both communicating and noncommunicating hydrocephalus secondary to obstruction at the level of the arachnoid villi or aqueduct, respectively. They are also at risk of ependymitis from the irritant effects of the blood.

On CT, IVH usually appears as CSF-hyperdense fluid level, layering within the ventricular system (Fig. 11B). Indeed, tiny collections of increased density layering in the occipital horns may be the only clue to IVH. Occasionally, the IVH may appear “tumefactive” as a cast within the ventricle.

Figure 11 SAH and IVH secondary to aneurysm rupture. (A) On CT, acute SAH appears as linear or serpentine areas of high attenuation that conform to the morphology of the sulci and cisterns. Although SAH is common with TBI, if the SAH is identified in certain locations (Sylvian, interhemispheric, or basilar cisterns), an underlying ruptured aneurysm must also be considered (B, C, D). Contrast-enhanced CT angiography (CTA) is particularly helpful in this situation. In this case, CTA shows a right middle cerebral artery bifurcation aneurysm (D). The aneurysm appears pointed, suggesting recent rupture. Intraventricular hemorrhage (IVH) is seen as a high attenuation fluid level within the occipital horn of the left lateral ventricle (B, white arrowhead).

Figure 12 DAI on CT and MR. In Grade II DAI, the corpus callosum is injured. Note the low attenuation within the splenium of the corpus callosum on CT (A). The FLAIR image demonstrates abnormal hyperintensity in the same region (B). The diffusion-weighted image (DWI) shows a focus of bright signal intensity (C); the apparent diffusion coefficient (ADC) was correspondingly reduced (not shown). The T2*-weighted image gradient-echo image shows scattered foci of susceptibility staining (signal loss) (D).
Intra-Axial Injury

Diffuse Axonal Injury (DAI) results from rotational acceleration and deceleration forces that produce shearing deformations of brain tissue. Clinically, DAI is characterized by loss or severe impairment of consciousness beginning at the moment of direct impact. DAI in the chronic stage can result in overwhelming cognitive and psychiatric problems. The affected areas of the brain are often distant from the site of direct impact. DAI, which occurs in about half of all severe head trauma cases, is of special interest because it tends to be underdiagnosed by current imaging techniques.

MRI is clearly superior to CT for detecting DAI, and therefore, provides increased success at explaining neurologic deficits after trauma and predicting long-term outcome. Even with MR, the incidence of DAI is thought to be underestimated. Newer imaging methods, such as DWI and DTI with 3D tractography, have shown potential in improving the detection of white matter damage.\(^{17,24,25}\)

On MR, nonhemorrhagic DAI lesions appear as multiple small foci of increased signal on T2-weighted images (Fig. 12) and as decreased signal on T1-weighted images within the white matter. Petechial hemorrhage causes a central hypointensity on T2-weighted images and hyperintensity on T1-weighted images as a result of intracellular methemoglobin in the subacute stage. The conspicuity of DAI on MR eventually diminishes as the damaged axons degenerate and the edema resolves. Residual findings may include nonspecific atrophy, gliosis, or hemosiderin staining, which can persist indefinitely on gradient-echo T2*-weighted images (Fig. 12D).

The location of the DAI tends to correlate with the severity of the trauma. Grade I DAI involves only the periventricular gray–white junctions of the lobar white matter. The parasagittal regions of the frontal lobes and periventricular regions of the temporal lobes are commonly affected. Patients with more severe trauma have DAI involving the lobar white matter as well as the corpus callosum, particularly the posterior body and splenium (Fig. 12). The corpus callosum is susceptible to DAI because the falx prevents displacement of the cerebral hemispheres. In severe DAI, the dorsolateral midbrain, in addition to the lobar white matter and corpus callosum, is affected.

The cortical contusions is a focal brain lesion primarily involving superficial gray matter, with relative sparing of the underlying white matter. Regions of the brain that are in close contact with the rough surface on the inner skull table are commonly affected. These areas include the orbitofrontal and temporal lobes, and less so, the parasagittal convexity. The temporal lobes above the petrous bone or posterior to the greater sphenoid...
wing, and the frontal lobes above the cribriform plate, planum sphenoidale, and lesser sphenoid wing are commonly affected (Fig. 13). Contusions are associated with a better prognosis than DAI, unless they are accompanied by brainstem injury or significant mass effect. Contusions can also occur at the margins of depressed skull fractures (Fig. 14). The cerebellum is involved less than 10% of the time.30

On CT, nonhemorrhagic contusions are difficult to detect initially until the development of associated edema (Fig. 15). Hemorrhagic contusions are more easily identified and appear as foci of high attenuation within superficial gray matter (Fig. 14). They may be surrounded by larger areas of low attenuation from the associated vasogenic edema. As the contusion evolves, the characteristic “salt and pepper” pattern of mixed areas of hypodensity and hyperdensity becomes more apparent. Contusions with severe mass effect may require surgical decompression to prevent secondary injury.

On MRI, contusions appear as ill-defined areas of variable signal intensity on both T1- and T2-weighted images, depending on the age of the lesions (Fig. 13). They are limited to the surface and often have a “gyral” morphology. Hemosiderin from an old contusion leads to decreased signal intensity on T2- and especially T2*-weighted images, especially at higher field strengths. The signal loss can persist indefinitely and serves as an important marker of prior hemorrhage.

The intracerebral hematoma is the most common cause of clinical deterioration in patients who have experienced a lucid interval after the initial injury. In contrast to the common contusion described above, the intracerebral hematoma is due to shear-induced hemorrhage from the rupture of small intraparenchymal blood vessels. Because the bleeding occurs into areas of relatively normal brain, intracerebral hematomas tend to have less surrounding edema than cortical contusions. Most traumatic intracerebral hematomas are located in the frontotemporal white matter. Involvement of the basal ganglia has been described; however, hemorrhage in basal ganglia should alert the radiologist that an underlying hypertensive bleed may be the culprit (Fig. 16). Intracerebral hematomas are often associated with skull fractures and other primary neuronal lesions, including contusions and DAI, especially in patients who are unconscious at the time of injury. Symptoms typically result from the mass effect associated with an expanding hematoma. Indeed, delayed hemorrhage is a common cause of clinical deterioration during the first several days after head trauma.

Vascular Injury

Vascular injuries are mentioned here because they are causes of both intra- and extra-axial injuries, including the cause of hematomas and subarachnoid hemorrhages. Additional traumatic vascular injuries include the arterial dissection, pseudoaneurysm, and arteriovenous fistula. Arterial injuries are usually related to skull base fractures. The most often injured artery is the internal carotid artery, especially at sites of fixation, where it enters the carotid canal at the base of the petrous bone, and at its

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Figure 15 Nonhemorrhagic contusion on CT. Axial CT image demonstrates an ill-defined area of low attenuation within the right temporal lobe (arrow). The low attenuation is due to edema from the nonhemorrhagic contusion. The inferior temporal lobe is commonly injured because of its proximity to the petrous ridge.

Figure 16 Intracerebral hematoma. (A) Axial CT shows a hyperdense lesion within the right putamen. The surrounding low attenuation is due to vasogenic edema. (B) On the T1-weighted image, the periphery of the hematoma is hyperintense due to the presence of methemoglobin. (C) The hematoma is also hyperintense on the T2-weighted image due the presence of extracellular methemoglobin. (D) Gradient-echo T2*-weighted image shows a surrounding hemosiderin rim, which is dark due to local magnetic susceptibility inhomogeneity.
exit from the cavernous sinus beneath the anterior clinoid process.

MR findings of vascular injury include (a) the presence of an intramural hematoma, which is best seen on T1-weighted with fat suppression (Fig. 17); (b) intimal flap with dissection; and (c) absence of a normal vascular flow void secondary to slow flow or occlusion. An associated parenchymal infarction supplied by the injured vessel may also be seen. Conventional angiograms remain the gold standard for confirmation and delineation of the vascular dissection and may also show spasm or pseudoaneurysm formation. However, magnetic resonance angiography and MDCT angiography serve as important screening tools in the evaluation of patients with suspected vascular injury.

The acquired carotid cavernous fistula (CCF) typically results from full-thickness arterial injury. The injury leads to communication between the cavernous portion of the internal carotid artery and the surrounding venous plexus, resulting in venous engorgement of the cavernous sinus (Fig. 18) and its draining branches: the ipsilateral superior ophthalmic vein and inferior petrosal sinus. Skull base fractures, especially those involving the sphenoid bone, should alert the radiologist to search for associated cavernous carotid injury. Another cause of CCF is the rupture of a cavernous carotid aneurysm. On imaging, CCF can present as an enlarged superior ophthalmic vein, cavernous sinus, and/or petrosal sinus. Other findings include proptosis, preseptal soft-tissue swelling, and extraocular muscle enlargement. The findings may be bilateral because venous channels connect the cavernous sinuses. Again, definitive diagnosis often requires selective carotid angiography with rapid filming to demonstrate the site of communication. Patients can present with findings weeks or even months after the initial trauma. Therefore, a CCF can be overlooked if a detailed clinical history and ophthalmic examination is not performed.

Another traumatic vascular injury is the dural fistula, usually caused by laceration of the middle meningeal artery with resultant meningeal artery to meningeal vein fistula formation. The dural fistula generally drains via the meningeal veins; therefore, they rarely lead to the formation of an EDH. Patients are often asymptomatic or present with nonspecific complaints such as tinnitus.

### Secondary Head Injury

#### Acute

Diffuse cerebral swelling arises from an increase in cerebral blood volume (hyperemia), vasogenic edema, or an increase in tissue fluid (cerebral or cytotoxic edema). Imaging demonstrates effacement of the cerebral sulci and cisterns and
compression of the ventricles. Hyperemia is thought to be the result of cerebral dys-autoregulation, and cerebral edema usually occurs secondary to tissue hypoxia. In cerebral edema, the gray–white differentiation is lost, which is in contrast to cerebral hyperemia where the gray–white differentiation is preserved. The cerebellum and brainstem are usually spared in cerebral edema and may appear hyperintense relative to the affected cerebral hemispheres (Fig. 19).

Brain herniation occurs secondary to mass effect produced by other causes. In subfalcine herniation, the most common form of herniation, the cingulate gyrus is displaced across the midline under the falx cerebri. Compression of the ipsilateral ventricle due to mass effect and enlargement of the contralateral ventricle due to obstruction of the foramen of Monro can be seen on imaging (Fig. 8). The anterior cerebral arteries (ACA) are displaced to the contralateral side, trapping the callosomarginal branches of the ACA, and may lead to ACA infarction. In uncal herniation, the medial temporal lobe is displaced over the free margin of the tentorium. Effacement of the ambient and lateral suprasellar cisterns is an important clue of the presence of uncal herniation. In severe cases, displacement of the brainstem can cause compression of the contralateral cerebral peduncle against the tentorium (“Kernohan’s notch”), leading to peduncular infarction or hemorrhage. Occasionally, the third cranial nerve is compressed; these patients present with a “blown pupil” and ipsilateral hemiparesis. In transentorial herniation, the brain herniates either upward or downward. Upward herniation typically occurs with large posterior fossa hematomas that displace portions of the cerebellum and vermis through the tentorial incisura. The mass effect of the hematoma can also cause downward herniation of the cerebellar tonsils through the foramen magnum. Downward herniation of the cerebrum manifests as effacement of the suprasellar and perimesencephalic cisterns. Inferior displacement of the pineal calcification is an additional imaging clue for the presence of downward herniation.

Infarction or ischemia can complicate TBI as a result of increased intracranial pressure or mass effect on cerebral vasculature by herniation or hematoma. In addition to ACA infarction secondary to the subfalcine herniation described above, uncal herniation and tonsillar herniation can cause ischemia in the territory of the posterior cerebral artery and the posterior inferior cerebellar artery distributions, respectively.

Chronic
As mentioned above, traumatic hydrocephalus occurs secondary to impaired CSF reabsorption at the level of the arachnoid villi or secondary to obstruction of the cerebral aqueduct and 4th ventricular outflow by SAH. Mass effect from cerebral herniation or a hematoma can also cause noncommunicating hydrocephalus via compression of the aqueduct and ventricular outflow foramina (Fig. 8).

Encephalomalacia is a common, but nonspecific, sequela of prior parenchyma injury. It may be clinically asymptomatic, but it can also be a potential seizure focus. On CT, the imaging appearance consists of an area of low attenuation

![Figure 19](image19.png)

**Figure 19** Diffuse cerebral edema. Noncontrast CT scan in an infant with diffuse cerebral edema following strangulation. There is a diffuse decrease in attenuation of the cerebral hemispheres with loss of gray–white differentiation. Sparing of the brainstem and cerebellum causes these structures to appear dense relative to the rest of the brain. An acute SDH overlies the tentorium (arrows).

![Figure 20](image20.png)

**Figure 20** CSF leak. Coronal CT cisternography performed before (A) and after (B) intrathecal contrast demonstrates a meningocele and suspected bony defect involving the left cribiform plate with contrast leakage into the upper left nasal vault (arrowhead).
with volume loss. It typically follows CSF signal intensity on both CT and MR, except for the areas of gliosis, which appears as low intensity on T1- and high intensity on T2-weighted images. Encephalomalacia within the orbitofrontal (especially the gyrus rectus) and anteroinferior temporal lobes is characteristic of remote traumatic injury.

A CSF leak usually results from a dural tear secondary to a skull base fracture. When communication between the subarachnoid space and middle ear occurs in association with a ruptured tympanic membrane, CSF otorrhea may be noted. CSF rhinorrhea occurs when there is communication between the subarachnoid space and the paranasal sinuses. CSF leaks are often difficult to localize and can lead to recurrent meningeal infection. Radionuclide cisternography is highly sensitive for the presence of CSF extravasation. However, CT scanning with intrathecal contrast is required for detailed anatomic localization of the defect (Fig. 20).

The leptomeningeal cyst or “growing fracture” is a pediatric lesion that is also caused by a tear in the dura in association with a calvarial defect. The dural defect allows expansion of the arachnoid at the site of the bony defect, presumably as a result of CSF pulsations. Such expansion leads to progressive, slow widening of the skull defect or suture. The leptomeningeal cyst appears as a lytic skull defect on CT or plain skull films, which can enlarge over time (Fig. 21).

**Summary**

The goal of imaging in the management of head trauma is to identify treatable injuries to prevent secondary damage. CT continues to be the modality of choice in the evaluation of acute head injury. CT is preferred in the acute setting because it is fast, is widely available, and can easily accommodate life-support and monitoring equipment. CT can accurately identify space-occupying lesions, acute hemorrhages, mass effect, midline shift, hydrocephalus, ischemia, and herniation. MRI is indicated for patients with acute TBI when the neurologic findings are unexplained by CT. MRI is also the modality of choice for subacute or chronic injury. Advances in MR methods, such as diffusion-weighted imaging, further improve the neuroradiological evaluation of traumatic brain injury and enhance our understanding of the pathophysiological manifestations of head trauma.

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