Imaging of traumatic intracranial hemorrhage

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Traumatic brain injury is a leading cause of morbidity and mortality in the United States, with an incidence of 95 per 100,000 population [1]. The most frequent causes are motor vehicle accidents, falls, assault, and firearm-related incidents [2]. The Centers for Disease Control and Prevention estimate that 22% of people suffering traumatic brain injury die from their injuries. Deaths attributable to intracerebral and intracranial hemorrhage range from 5.0 to 5.6 per 100,000 population [3].

Head trauma is particularly common in adolescents, young adults and the elderly. Increased age is an independent risk factor for acute traumatic intracranial hemorrhage, perhaps due to cerebral atrophy and an increased propensity for bridging veins to tear, or increased friability of vessels secondary to atherosclerotic disease or amyloid angiopathy [4]. The risk in males is twice the risk in females for all age groups [2]. Motor vehicle accidents are responsible for up to 35% of all cases of head trauma [2]. Polytrauma victims with signs of circulatory compromise have a relatively low incidence of traumatic intracranial hematomas, and should undergo prompt evaluation for serious injuries to the chest, abdomen and pelvis [5]. Falls cause up to 33% of all head trauma [6], and are the most common cause of hemorrhage in the elderly, particularly patients anticoagulated with sodium warfarin (Coumadin), who are at increased risk for hemorrhage [7]. Traumatic intracranial hematomas are common in head trauma patients with known or suspected seizure disorders, who appear postictal, or who are found convulsing after a fall [6].

Many patients may be managed medically [8]. Ten percent of medically managed patients may eventually require neurosurgical intervention. In a series reported by Patel et al [8], surgical evacuation was eventually required in 17% of epidural hematomas, 14% of subdural hematomas, and 10% of intraparenchymal hematomas, with frontal intraparenchymal hematomas particularly prone to early “medical failure.”

Imaging techniques

Computed tomography

Computed tomography (CT) is the imaging modality of choice in the evaluation of acute head trauma, due to its widespread availability, speed, and compatibility with life support and monitoring devices. Motion due to uncooperative patients is less important with the increasing availability of fast multidetector CT scanners [9]. If images are degraded by motion artifact, those particular slices can be selectively rescanned without repeating the entire scan. At our institution, we routinely view images in brain (window 80, level 40), subdural (window 200, level 70), and bone (window 3000/level 400) formats to evaluate for parenchymal, extra axial, and osseous injuries, respectively. Subdural windows are particularly useful in detecting superficial hemorrhage, shallow contusions, and small extra axial collections, where the high attenuation of blood may blend into the adjacent high attenuation bone (Fig. 1). Limitations of CT include beam hardening effects, which may partially obscure blood in the posterior fossa, subtemporal, and subfrontal regions; and volume averaging of small...
amounts of blood that occupy less than the acquired slice thickness with normal brain. In those instances where CT is equivocal, lesions such as small subdural hematoma will be more readily apparent on magnetic resonance (Fig. 2).

**Magnetic resonance**

Magnetic resonance (MR) is an alternative initial modality with greater sensitivity for detecting abnormalities and predicting prognosis [10], particularly in assessing injury to the brainstem. T2-weighted images are most useful for lesion detection, and T1-weighted images most useful for anatomic localization [11]. The appearance of blood on MR imaging depends on the predominant type of hemoglobin present within the hematoma.

In uncooperative, unstable, or claustrophobic patients, ultrafast sequences such as the gradient recalled echo single shot echo planar imaging sequence may be used, although characterization of hemorrhagic foci and sensitivity at the skull base are inferior to traditional gradient echo sequences [12]. Abbreviated MR studies using fast pulse sequences on ultra-low, low, or intermediate field strength systems, with less stringent ferromagnetic equipment restrictions, may offer answers to the crucial questions in the shortest time possible [13].

MR is inferior to CT in evaluation of injuries to the skull vault. Contraindications to MR imaging include pacemakers, noncompatible vascular clips, metallic implants, and ocular foreign bodies.

**Other imaging modalities**

Ultrasound is portable, inexpensive and does not subject patients to ionizing radiation. Neonates are the most suitable patients, where the open anterior and posterior fontanelles are suitable imaging windows. Compared to CT and MR, however, ultrasound has relatively lower sensitivity and specificity for intracranial hemorrhage and ischemia [14].

The spatial resolution of positron emission tomography (PET) and 99m technetium hexamethylpropyleneamineoxime (HMPAO) brain perfusion with single photon emission computed tomography (SPECT) is inferior to that of CT and MR, but the former may be more sensitive in detecting brain abnormalities in chronic traumatic brain injury patients [15,16].

**Mass effect and herniation**

Regardless of the mechanism of injury, intra-axial and extra-axial hemorrhage may develop enough mass...
effect to compress adjacent vascular structures and cause ischemia or infarct. Alternatively, mass effect may herniate part of the brain from one compartment into another. In subfalcine herniation, increased pressure directed medially causes the cingulate gyrus to shift beneath and across the falx cerebri. This may cause compression of the anterior cerebral artery or internal cerebral veins. In descending transtentorial herniation, increased supratentorial pressure directed medially and inferiorly causes the temporal lobe to herniate over the tentorium, potentially compressing the oculomotor nerve, posterior cerebral, and anterior

Fig. 2. Acute subdural hematoma in a 37-year-old male. Contrast enhanced CT images viewed with (A) brain and (B) subdural windows reveal only mild left sulcal effacement. (C) Fluid attenuated inversion recovery MR image at similar level easily demonstrates the small left subdural hematoma.
choroidal arteries, or midbrain. In ascending transtentorial herniation, mass effect from the posterior fossa directed superiorly causes the cerebellum to herniate upward through the tentorial incisura. In uncal or medial transtentorial herniation, the uncus herniates through the tentorial incisura. Uncal herniation causes enlargement of ipsilateral basal cisterns and the contralateral temporal horn. In tonsillar herniation, increased pressure in the posterior fossa causes the cerebellar tonsils and cerebellum to herniate inferiorly through the foramen magnum, compressing the fourth ventricle, and thus producing acute obstructive hydrocephalus. The upper cervical cord may also be compressed.

**Traumatic intra-axial injury**

**Contusion**

Contusions are bruises of the brain. They occur in up to 43% of patients with blunt or nonpenetrating head injuries [11]. Neurosurgical intervention is more likely to improve outcome for hemorrhagic contusions in patients after a fall, patients who have a low Glasgow Coma score, patients with anisocoria, or patients older than 60 [4]. Contusions have traditionally been described as coup or contrecoup injuries due to deceleration/acceleration trauma. Coup contusions occur when the moving brain strikes the stationary calvarium, and the calvarium is deformed temporarily, physically distorting the underlying brain parenchyma [17]. There is mechanical damage to neurons, with or without petechial hemorrhage or torn capillaries. Contrecoup contusions occur distant or opposite from the site of initial impact, when the brain is set in motion relative to the stationary calvarium [17].

With frontal impact trauma, the brain moves over the roughened edges of the inner table of the skull, particularly the floor of the anterior cranial fossa, and slams into the sphenoid wings and petrous ridges, explaining why contusions occur most commonly in the inferior frontal, anterior temporal, and lateral temporal regions (Fig. 3). Paramedian bony irregularities may cause superior frontal and parasagittal contusions.

The acute contusion less than 12 hours old is composed mostly of intracellular oxyhemoglobin with the edematous brain undergoing necrosis. Noncontrast CT will show low attenuation if hemorrhage is absent and mixed, or high attenuation if hemorrhage is present. At this point, the high attenuation (50–70 Hounsfield units) is from high protein concentration within intact red blood cells and not iron content [18].

Fig. 3. Hemorrhagic contusions in a 38-year-old female. Noncontrast CT image shows bilateral inferior frontal and right anterior temporal hemorrhagic contusions.

On T2-weighted MR images, acute hemorrhagic contusions will exhibit inhomogeneous signal due to hypointense deoxyhemoglobin and hyperintense, edematous cortical tissue. MR is less sensitive than CT in the hyperacute stage because diamagnetic intracellular oxyhemoglobin lacks unpaired electrons and thus clot signal is close to normal brain parenchyma—normal to slightly lower signal on T1-weighted images and slightly higher signal on T2-weighted images [19,20]. Repeat imaging is indicated to monitor the size of the hemorrhage and the development of delayed hemorrhage and vasogenic edema. Large hemorrhagic contusions commonly increase in size within the first 48 hours (Fig. 4).

Acute hemorrhagic contusions one to three days old are composed mostly of paramagnetic intracellular deoxyhemoglobin. The deoxyhemoglobin is formed by the dissociation of oxygen from hemoglobin, a process that begins within several hours. Because the deoxyhemoglobin within intact, clotted hypoxic red blood cells does not cause T1 shortening, the hemorrhagic contusion will have normal to slightly lower signal on T1-weighted images and slightly higher signal on T2-weighted images [19,20]. The concentration of red blood cells with clot and the concentration of fibrin cause T2 shortening, with areas of very low signal on T2-weighted spin echo and T2 *-weighted gradient echo images [20].
Within a few days, the subacute contusion will start to undergo liquefaction with development of vasogenic edema. As the edema increases over the first week, it may be great enough to cause herniation. The edema has fluid or water characteristics: iso- to hypointense on T1-weighted images, and hyperintense on T2-weighted images (Fig. 5). With oxidation of deoxyhemoglobin to strongly paramagnetic intracellular methemoglobin, proton-electron dipole-dipole interactions between hydrogen atoms and the paramagnetic centers of methemoglobin will cause marked T1 shortening and very high signal intensity on T1-weighted images [20] within the periphery of the hematoma (Fig. 6). The intracellular methemoglobin will cause T2 shortening and very low signal on T2-weighted images.

After erythrocyte membrane breakdown and extracellular migration of methemoglobin, there is neovascularization with removal of blood components and debris by macrophages. The new blood vessels at the periphery of the lesion lack the tight endothelial junctions of an intact blood brain barrier, and so there is intense enhancement of the margins on both contrast CT and MR [13]. The fragile granulation tissue vessels predispose the patient to additional episodes of acute hemorrhage. CT will show a decrease in the density of the contusion and decrease in the mass effect, the latter due to a decrease in

Fig. 4. Contusions in a 66-year-old male. (A) Initial noncontrast CT image demonstrates small hemorrhagic contusions in the left frontal lobe. (B) Noncontrast CT image 5 days later demonstrates a hemorrhagic contusion in the left temporal lobe and an increase in the size of the left frontal hemorrhagic contusions with surrounding edema.

Fig. 5. Hemorrhagic contusion in a 58-year-old female. Axial T2-weighted image shows a left temporal contusion with hypointense acute blood anteriorly (*) and hyperintense subacute blood posteriorly (x). Intermediate increased signal around the contusion represents edema (arrowheads).
edema. MR will exhibit the persistent high signal of extracellular methemoglobin on T1- and T2-weighted images [20] for up to a year. The peripheral rim of hemosiderin and ferritin has slightly low signal on T1- and marked low signal on T2-weighted images [20] from the susceptibility effect of hemosiderin within macrophage lysosomes.

Clot resorption begins from the periphery inward, and depending on the size of the hematoma, may vary from one to six weeks in duration. Necrotic tissue is sloughed and cystic cavities are formed over the next 6 to 12 months. Focal atrophy is characterized by a decrease in the size of cortical gyri, with compensatory enlargement of cerebrospinal fluid spaces and dilatation of the adjacent ventricle. Cystic cavities are surrounded by gliosis and hemosiderin scarring. Fibroglial scars may adhere dura to adjacent brain, and cause seizures in post-traumatic patients.

**Intraparenchymal hematoma**

Intraparenchymal hematomas unrelated to contusions are generally the result of penetrating trauma such as gunshot or stab wounds. Missile trauma with metallic objects may preclude MR imaging as a diagnostic modality. Noncontrast CT will demonstrate a homogenous high attenuation consolidation with well-defined margins (Fig. 7). Surrounding edema increases and peaks at one week.

Subacute hematomas three to seven days old may give the appearance on CT of layering fluid-blood levels within the hematoma or clot retraction. Late subacute hematomas 7 to 14 days old will decrease in attenuation from the periphery inward, of approximately one to two Hounsfield units each day. Chronic cerebral hematomas more than two weeks old are composed primarily of intracellular ferritin and lysosomal hemosiderin. On CT, the hematoma will continue to decrease in attenuation. Within 3 to 10 weeks, chronic hematomas will become isodense with normal brain parenchyma and very difficult to detect. Continued proteolysis, phagocytosis and adjacent atrophy will eventually replace the hematoma with an area of encephalomalacia.

**Diffuse axonal injury**

Diffuse axonal injuries occur in up to 48% of patients with closed head injuries [11] when the shearing forces of rapid rotational acceleration or deceleration cause axonal disruption. Axonal injury ranges from incomplete disruption detected at only the

Fig. 6. Early subacute hemorrhagic contusion in a 78-year-old male. Sagittal T1-weighted image demonstrates high signal intensity at the periphery of the contusion, consistent with extracellular methemoglobin.

Fig. 7. Intraparenchymal hemorrhage in a 54-year-old male. Axial CT image demonstrates acute hemorrhage in the left frontal lobe causing midline shift. There is a small left subdural hematoma. A subarachnoid hemorrhage outlines the basal cisterns.
microscopic level during postmortem analysis, to complete disruption associated with shearing hemorrhage from adjacent capillary lacerations. Severe diffuse axonal injury is responsible for coma and poor outcome in nearly half of patients with significant closed head trauma from motor vehicle accidents. Poor prognostic indicators include low Glasgow coma scale scores, concomitant shearing hemorrhages in the brainstem and corpus callosum, and hemorrhagic and nonhemorrhagic space-occupying lesions such as subdural hematomas, and hemorrhagic and nonhemorrhagic contusions [10,21].

Characteristically, diffuse axonal injuries are distributed at points of maximal shearing stress along white matter tracts. Nearly two-thirds of the lesions occur in lobar white matter at the corticomedullary junction, including frontal parasagittal region, temporal periventricular region, and less commonly the parietal and occipital lobes. The corpus callosum is commonly involved, especially the splenium and posterior body (Fig. 8). Intraventricular hemorrhage suggests injury to the corpus callosum with concomitant injury to the subependymal capillary and venous plexus along the ventricular surface of the corpus callosum, fornix, or septum pellucidum [22,23]. With more severe head trauma, there is involvement of the basal ganglia, including the internal and external capsules and corona radiata. Lesions may also involve the rostral dorsolateral brainstem adjacent to the superior cerebellar peduncles and medial lemnisci, within the midbrain and upper pons.

The initial CT is often normal, particularly when nonhemorrhagic shearing injury has occurred. Since more than 80% of shearing injuries are nonhemorrhagic [24], noncontrast CT underestimates diffuse axonal injury. Acute tissue tear hemorrhages, or shearing hemorrhages, occur in more severe instances or in up to 13% of diffuse axonal injuries and up to 8% of all head injuries [21]. With complete axonal disruptions and associated tissue tears, CT will demonstrate multiple 0.5 to 1.5 cm foci of high attenuation, representing hemorrhagic axonal injuries surrounded by rims of low attenuation edema (Fig. 9). Hemorrhagic foci are more easily detected on delayed than initial CT scans.

Almost one-third of patients with normal CT findings after head trauma will have evidence of diffuse axonal injury on MR [25]. T2-weighted images are especially sensitive for detecting axonal injuries, which are hyperintense on T2-weighted images and iso- to hypointense on T1-weighted images. They are round or ovoid, with the long axis parallel to fiber bundle direction. Because of their greater sensitivity

Fig. 8. Hemorrhagic shearing injury in a 77-year-old male. Sagittal T1-weighted image demonstrates high signal intensity representing subacute hemorrhage at the junction of the posterior body and splenium of the corpus callosum. In addition, a subacute subdural hematoma is seen posteriorly (arrows).

Fig. 9. Diffuse axonal injury in a 7-year-old. Noncontrast CT image demonstrates punctate hemorrhagic foci at the frontal gray-white junctions (arrows).

Fig. 11. Diffuse axonal injury in a 52-year-old male. (A) Isotropic diffusion weighted image demonstrates hyperintense signal within the splenium, consistent with cellular edema. (B) Diffusion trace image demonstrates corresponding decreased ADC values. (From Liu AY, Maaldjian JA, Bagley LJ, et al. Traumatic brain injury: diffusion-weighted MR imaging findings. Am J Neuroradiol 1999;20:1636–41; with permission. © by the American Society of Neuroradiology [www.ajnr.org].)
for susceptibility effects, gradient echo sequences such as 2D fast low angle shot (FLASH) are more sensitive for detecting hemorrhagic shear injuries [25,26] (Fig. 10). Acute to subacute nonhemorrhagic axonal injuries have increased signal on diffusion-weighted images and corresponding areas of decreased signal on trace apparent diffusion coefficient maps, consistent with cellular swelling or cytotoxic edema [27] (Fig. 11). Secondary Wallerian degeneration will occur in areas of axonal disruption and lead to diffuse degeneration and atrophy over months or years.

Deep cerebral gray matter and brainstem injury

Traumatic injury to the thalamus, basal ganglia, and brainstem is relatively uncommon. Severe shearing forces associated with diffuse axonal injury are usually responsible for the disruption of small perforating vessels. CT may be normal or may demonstrate multiple hemorrhagic foci near the lentiform nucleus and external capsule. The basal cisterns are often obliterated after brainstem injury, due to diffuse cerebral edema. MR provides better evaluation for brainstem contusion and hemorrhage, since it is more sensitive for nonhemorrhagic lesions and less susceptible to posterior fossa artifacts. Frequently coexisting lesions include subarachnoid hemorrhage, contusion, extra-axial hematoma, and intraventricular hemorrhage. Poor prognosis is reliably predicted by age greater than 60, low Glasgow coma scale score, abnormal pupil response, impaired oculocephalic response, and abnormal motor response to painful stimuli [13,28].

Traumatic extra-axial hemorrhage

Subdural hematoma

Subdural hematomas are seen in 10% to 20% of patients with head trauma. The mortality is between 50% and 85%. Acute subdural hematomas, with or without associated cerebral contusions, are the most commonly encountered operable intracranial hematoma [4]. Subdural collections, blood or cerebrospinal fluid, dissect into the potential space between the dura and arachnoid membranes. Subdural hematomas may cross suture lines, but not dural reflections such as the falk and tentorium. Subdural hematomas are usually caused by traumatic tears of cortical bridging veins between the temporal lobes and sphenoparietal or petrosal sinuses. Unlike epidural hematomas, subdural hematomas are not commonly associated with skull fractures in adults. Subdu-

![Fig. 12. Subdural hematoma in a 15-month-old male with multiple healing long bone metaphyseal fractures after child abuse. (A) Axial CT image shows faint high density subdural blood along the right tentorium. (B) Coronal T1-weighted MR image shows bright signal in the right suboccipital region (arrows) consistent with a subacute subdural hematoma. (Courtesy of C. Hilfer, MD, New York, NY.)](image)
Fig. 13. Acute subdural hematoma in an 81-year-old male. (A) Initial CT image demonstrates a left subdural hematoma causing midline shift. There is compression of the ipsilateral ventricle and dilatation of the contralateral ventricle and midline shift. (B) Follow-up CT image 15 days later reveals the left subdural collection to have significantly decreased in density, now similar to CSF attenuation.

Fig. 14. Bilateral subacute subdural hematomas in a 52-year-old male. (A) Noncontrast CT image demonstrates bilateral isodense subdural hematomas displacing the gray-white junction medially (arrows). There is sulcal effacement, but the two subdural hematomas balance each other and result in no midline shift. (B) Contrast enhanced CT image demonstrates enhancing cortical veins along the surface of the brain (arrows), medial to the bilateral subdural hematomas.
rual hematomas are common in cases of child abuse (Fig. 12).

The typical appearance of an acute subdural hematoma on CT is a crescentic hyperdense collections between the cerebral hemisphere and inner table of the skull, extending from front to back around one hemisphere. Mass effect from the subdural hematoma will displace the gray-white junction away from the inner table and may cause midline shift, with compression of the ipsilateral lateral ventricle and dilatation of the contralateral ventricle (Fig. 13A). Bilateral subdural hematomas may have a balanced effect without midline shift despite significant mass effect (Fig. 14A).

Small subdural hematomas may be missed because of high convexity locations, beam hardening artifacts, or narrow window settings. As noted above, wider “subdural” windows help to differentiate pixels of similar brightness, thus differentiating acute blood from bone (see Fig. 1). Acute subdural hematomas may uncommonly present as isodense or hypodense collections because of marked anemia and paucity of hemoglobin within the hematoma, clotting abnormalities, or cerebrospinal fluid dilution from associated arachnoid tears, similar in appearance to subacute hematomas.

The right and left interhemispheric subdural spaces are separated by the attachment of the falx to the inferior sagittal sinus. Disruption of bridging veins feeding into the superior sagittal sinus causes blood to collect in the subdural space along one side of the falx cerebri. The medial border will be straight as it is bounded by the rigid falx, and the lateral border will be convex as it displaces brain away from the midline. The interhemispheric subdural hematoma is particularly common in abused children.

Hematomas may collect in the potential subdural space along the tentorium. Supratentorial extra-axial collections are more commonly subdural hematomas than epidural hematomas, and infratentorial extra-axial collections are more commonly epidural hematomas than subdural hematomas. The subdural hematoma will appear as hazy high attenuation along the tentorium. Volume averaging and beam hardening artifacts may partially obscure the subtemporal hematoma.

The predictable evolution of blood within unevacuated subdural hematomas produces typical changes on noncontrast CT scan. The subdural collection will gradually decrease in size and attenuation as the hemoglobin, platelet, or fibrin clot, and cellular components are resorbed. The attenuation of subacute subdural hematomas 4 to 20 days old will approach the attenuation of normal brain parenchyma. Isodense subdural hematomas are difficult to detect with non-contrast CT, but should be suspected when the gray-white matter interface is displaced away from the ipsilateral inner table of skull and the sulci are effaced or difficult to trace to the brain surface (Fig. 15). Contrast CT will demonstrate enhancement of the inner membrane or cortical veins defining the surface of the brain (see Fig. 14B). Subacute subdural hematomas are more easily detected with MR imaging because of their high signal intensity on T1-weighted images [29] (see Fig. 12B).

Chronic subdural hematomas more than two to three weeks old are typically lower in attenuation than brain on CT (see Fig. 13B). Acute on chronic subdural hematomas develop from rehemorrhage into previous subdural collections. Fluid-blood levels are seen when sedimented fresh blood gravitates dependently and the proteinaceous fluid layers on top.

Subdural hygromas are cerebrospinal fluid collections that develop 6 to 30 days after traumatic arachnoid tears. On CT, subdural hygromas will have the same density as cerebrospinal fluid. Blood vessels crossing through the extra-axial collection will help differentiate subdural hygromas from chronic subdural hematomas. On T1-weighted MR images, subdural hygromas will have the same signal as cerebrospinal fluid, although slightly higher signal from increased contrast CT.
protein content may be observed on occasion. Since subdural hygromas lack the methemoglobin present in chronic subdural hematomas, they are hypointense, similar to cerebrospinal fluid (CSF), on T1-weighted images [30].

Epidural hematoma

Only 1% to 4% of patients with head trauma have epidural hematomas. The overall mortality in these patients is 5%. More than 90% of epidural hematomas in adults are associated with fractures [31]. Fractures of the inner table may cause lacerations of the middle meningeal artery because the dural blood supply lies within the epidural space. Less commonly, venous bleeding can occur from disruption of a meningeal vein or a dural venous sinus—the transverse or sigmoid sinus in the posterior fossa and superior sagittal sinus in the parasagittal region. Blood dissects into the potential space between the inner table of the skull and periosteal dura. The periosteal dura has its strongest attachment at the sutures. Therefore, in contrast to subdural hematomas, epidural hematomas do not cross sutures and have a characteristic convex shape. They do, however, cross dural reflections such as the falx. Epidural hematomas classically occur unilaterally in the temporoparietal region. Ninety-five percent are supratentorial. Epidural hematomas in the posterior fossa are rare, and have a higher morbidity and mortality.

Acute epidural hematomas are typically seen on noncontrast CT as hyperdense lenticular (Fig. 16A) or biconvex (Fig. 17) extra-axial collections adjacent to a fracture. (see Fig. 16B). Areas of low attenuation within the hematoma represent mixing of unclotted blood and serum separating from clotted blood [31,32], and are suggestive of active arterial bleeding. (see Fig. 17). Hyperacute and chronic epidural hematomas demonstrate exhibit signal intensity similar to CSF on T1- and T2-weighted images. The acute epidural hematoma is isointense on T1-weighted images and iso- to hypointense on T2-weighted images. Subacute and early chronic epidural hematomas are hyperintense on T1- and T2-weighted images.

Large acute epidural hematomas with significant mass effect may displace and compress adjacent brain, and lead to brainstem herniation and death. These cases are neurosurgical emergencies that require prompt evacuation. Small epidural hematomas that are asymptomatic, located along the convexities, are less than 1.5 cm in maximum width, and have minimal or no midline shift require no intervention, provided they do not increase in size [33,34]. Slow bleeds may increase pressure within the hematoma before blood has time to dissect further into the epidural space, leading to tamponade of the bleeding vessel. Depend-
ing upon the series, enlargement of epidural hematomas has been reported in 10% to 64.9% [35–37] of patients, usually within 48 hours after injury [35,37]. Therefore, serial neurological exams with low thresholds for repeat scanning are indicated to detect enlarging epidural hematomas [38].

Subarachnoid hemorrhage

Subarachnoid hemorrhage occurs in up to 11% of traumatic brain injuries [39]. It is the result of injury to small bridging cortical vessels on the pia or arachnoidal leptomeninges crossing the subarachnoid space. The very young and very old are especially vulnerable to subarachnoid hemorrhage because of their relatively larger subarachnoid spaces. Alternatively, blood from an intracerebral hematoma may decompress directly into the subarachnoid space or dissect into the ventricular system. When occurring in conjunction with other forms of traumatic brain injury, subarachnoid hemorrhage is often focal, next to a contusion.

CT will demonstrate high attenuation blood within the basal cisterns and subarachnoid spaces such as the sylvian fissures, superior cerebellar cisterns, and sulci over the cerebral convexities (Fig. 18). Nonvisualization of the interpeduncular cistern may be a clue that a small amount of isodense subarachnoid blood is present. Small amounts of subarachnoid hemorrhage may not be visualized, since blood that occupies less than the full thickness of a single CT slice will be volume averaged with brain.

The low hematocrit and low deoxyhemoglobin of acute subarachnoid blood gives signal similar to brain parenchyma on T1- and T2-weighted spin echo images [30]. Therefore, MR may underestimate or fail to detect acute subarachnoid hemorrhage. Fluid attenuated inversion recovery imaging (FLAIR) may detect small areas of acute or subacute subarachnoid hemorrhage, however, that are not detected by conventional MR images or CT scans, as hyperintense signal within the sylvian fissures or cerebral sulci [40] (Fig. 19).

Hydrocephalus is the most common complication after subarachnoid hemorrhage. Acute obstructive hydrocephalus may develop within the first week from ependymitis or intraventricular blood obstructing the aqueduct of Sylvius or outlet of the fourth ventricle. Communicating hydrocephalus may develop within hours or after the first week, if the arachnoidal villi are blocked by the corpuscular elements of the blood, leading to fibroblastic proliferation in the subarachnoid space and blockage of the

Fig. 17. Acute epidural hematoma in a 41-year-old male. Noncontrast CT image demonstrates a biconvex left fronto-temporal epidural hematoma. Epidural blood does not cross the coronal suture. Small areas of lower attenuation represent mixing of clotted and unclotted blood. No fracture was present, but the location is typical for an injury to the main branch of the middle meningeal artery.

Fig. 18. Acute subarachnoid hemorrhage in a 31-year-old female. Noncontrast CT image demonstrates high density blood filling the interpeduncular cistern (arrows).
pacchionian granulations. Blood is a direct irritant to vessels. Subarachnoid hemorrhage may induce cerebral vasospasm. The risk of severe ischemia and subsequent infarction peaks at 5 to 15 days. Vasospasm is more likely when subarachnoid hemorrhage is accompanied by subdural hematoma, intraventricular hemorrhage, cerebral contusion, or intracerebral hemorrhage [41].

Intraventricular hemorrhage

Intraventricular hemorrhage occurs in 2.8% of all patients with blunt head trauma [42]. The incidence is higher, ranging from 9.5–22% [22,43], in those patients with Glasgow coma scores less than or equal to eight. Isolated intraventricular hemorrhage is relatively uncommon. Superficial contusions and subarachnoid hemorrhage are common associated injuries. Intraparenchymal hematomas may dissect along white matter tracts into the ventricular system. Hemorrhagic injuries to the corpus callosum and brain stem, often due to diffuse axonal injury, are associated with intraventricular hemorrhage and poor outcome [42]. LeRoux et al [42] report a series where intracranial pressure monitors were placed in 39 patients with intraventricular hemorrhage. Intracranial pressure rose in 46%, acute hydrocephalus developed in 7%, and ventricular drainage was required in 10% of the patients [42]. Mortality in patients with intraventricular hemorrhage has been reported to range from 21% to 77% [43,44], although outcome is likely related more to severity of the primary brain injury than directly to intraventricular hemorrhage [43].

The intrinsic antithrombotic properties of fibrinolytic activators within CSF often cause intraventricular hemorrhage to layer within the ventricular system rather than clot [45]. Noncontrast CT will demonstrate a fluid-fluid layer with the high attenuation blood layering dependently within the ventricle and the lower attenuation, lower density supernatant, or CSF on top.

On T2-weighted MR imaging, the layering of acute blood will appear isointense to hypointense inferiorly and CSF intensity superiorly. Acute blood will appear hyperintense on FLAIR sequences sensitive to T2 prolongation, while nulling the normal cerebrospinal fluid background. There is evidence that FLAIR and fast spin echo FLAIR (fast FLAIR) sequences more conspicuously show acute intraventricular hemorrhage during the first 48 hours than noncontrast CT [46]. T1- and T2-weighted images will show acute intraventricular hemorrhage as isointense or hypointense. Cerebrospinal fluid pulsation artifacts within the ventricles, particularly within the posterior fossa, may be confused for intraventricular hemorrhage [46].
Summary

Traumatic intracranial hemorrhage is a leading cause of morbidity and mortality in the United States. CT remains the primary imaging modality for initial evaluation of patients who have sustained head trauma. MR imaging, which has always been important for the evaluation of subacute and chronic head trauma, has been gaining popularity and recognition as an alternative primary imaging modality.

References


