Imaging in Head Trauma: Current Concepts

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1. Introduction

Intracranial injury is frequently a factor in trauma-related fatalities. The radiologic findings of intracranial trauma are important to recognize for a number of reasons: a) so life-threatening injuries can be treated immediately, b) to adequately explain the findings on neurologic examination and c) to help establish prognosis. Because CT is readily available, fast, and sensitively detects injuries which require urgent surgical treatment, it is still the preferred means of initial diagnosis of intracranial injuries. Important types of intracranial injuries, e.g. subdural or epidural hematoma, subarachnoid hemorrhage and hemorrhagic contusion, can all be seen on CT. MRI has also proven to be helpful in evaluation of intracranial injuries, often by helping to solve problems which cannot be answered by CT examination (rather than as a first-study screening technique).

A number of intracranial injuries which are difficult to detect on CT, including nonhemorrhagic diffuse axonal injury, brainstem injury, and deep gray matter injury, can be demonstrated easily using MRI. Detection of these injuries can be helpful in determining long-term prognosis. Traumatic intracranial injuries can be classified as penetrating (i.e. penetration of the intracranial compartment by foreign bodies or bone) and non-penetrating (or "closed-head ") forms. The latter category consists of those in which there is forceful contact of the skull with another object as well as non-impact trauma, e.g., injury due to acceleration/deceleration or rotational motions. The following review summarizes the main types of intracranial injuries.

2. Fractures

The three main types of fractures include linear (the most common), depressed and basal fractures. The most important linear fractures are those extending across the temporal bone (which, in a small percentage of cases lacerate the middle meningeal artery and cause an epidural hematoma) and occipital fractures (which can result in laceration of dural venous sinuses and venous epidural hematoma). In general, however, linear fractures have a low incidence of associated intracranial injury in the absence of cognitive changes, altered level of consciousness, or focal neurologic signs and are associated with a low incidence of long-term neurologic sequelae.

Depressed fractures are more commonly associated with parenchymal injury than linear fractures. These wounds are typically surgically explored because of the risk of infection, in order to exclude a dural tear and, in some cases, to insure a satisfactory cosmetic result.

The most important complication of basal skull fractures is injury to the cranial nerves and vessels at the base of skull. Temporal bone fractures can result in injury to the seventh or eighth cranial nerves, ossicular disruption or a persistent cerebrospinal fluid (CSF) leak, sometimes causing recurrent meningitis.

Unlike linear and depressed skull fractures, basal skull fractures can be difficult to detect even on CT, unless thin section images are performed.

3. Extra-axial Hematomas

Subdural and epidural hematomas are commonly seen following severe head injury. Subdural hematomas (SDHs) are seen on CT or MRI in approximately 15% of patients with acute closed head injury. Epidural hematomas are less common, but more frequently require emergency surgical evacuation. Either type of hematoma can be associated with fracture of the adjacent skull or parenchymal injury.

3A. Subdural Hematoma

SDHs most commonly occur at one of three locations: over the convexities, along the falx cerebri and along the tentorium cerebelli. They undergo a characteristic temporal evolution, and are characterized according to their age, i.e. acute, subacute or chronic. Each of these categories has a typical CT appearance, with some exceptions.

Acute SDHs are often seen following trauma (of either the penetrating or nonpenetrating type) in which there is forceful contact of the skull with a foreign object, and are frequently associated with a skull fracture. Elderly patients, those on anticoagulant therapy or undergoing long-term hemodialysis, and thrombocytopenic patients are at increased risk for development of SDH following relatively minor head trauma. Acute SDHs can rapidly enlarge and cause rapid neurologic deterioration and death. These SDHs require emergency surgical evacuation, whereas small SDHs are usually clinically stable, and do not require surgical evacuation. Small acute SDHs are frequently relatively findings which may be accompanied by significant mass effect (out of proportion to the volume of the SDH) due to associated parenchymal injury and edema.

Subacute and chronic SDHs typically occur in the elderly population, and can have various clinical presentations, e.g. slowly progressive intellectual decline (mimicking dementia), transient or permanent focal neurologic deficits (simulating a transient ischemic attack or cerebrovascular accident), or chronic headache. Age-related brain atrophy is thought to play an important role in development of these SDHs, because as the distance between brain cortex and the dura increases (due to atrophy) small cortical ("bridging") veins become stretched and under tension. Minor impact trauma or even nonimpact motions of the head (e.g. violent coughing or sneezing) can cause tears in these veins and produce a SDH. The SDH can slowly increase over a few weeks or months, due to repeated small hemorrhages and result in a subdural collection composed of blood products of different ages, often having a thick proteinaceous (“motor oil”) consistency.

Acute SDHs located over a cerebral convexity appear as a hyperintense crescentic collection with a sharp margin between the collection and adjacent brain. The shape is not a reliable distinguishing feature because SDHs can have a biconvex (lentiform shape) like that of a typical epidural hematoma. In unusual circumstances, e.g. severe anemia, acute SDHs can be isointense (rather than

hyperintense) with gray matter. SDHs along the falx cerebri are seen as hyperintense linear blood collections between the hemispheres. Thin SDHs layering along the tentorium cerebelli can be difficult to detect on CT, because of volume averaging with brain, and may be seen on only a single axial CT slice. Their presence can be definitively determined by thinner axial slices, or coronal slices (obtained by imaging directly in the coronal plane or on coronal images reconstructed from axial images).

The CT appearance of SDHs undergoes a typical progression, becoming isointense to brain after a few (ca. 2 weeks), making identification difficult. Clues to the presence of an isodense SDH include mass effect and the absence of sulci over the affected hemisphere. However, the mass effect can be balanced when bilateral isodense SDHs are present, making subtle signs of mass effect, e.g. displacement of the gray/white interface away from the inner table of the skull, and bilateral absence of sulci, even more important. As with any radiologic examination, comparison to previous studies can be important in establishing the diagnosis.

Chronic SDHs (approximately 3-4 weeks old) typically appear hypointense relative to gray matter, but can have a two layer appearance with a hypointense (chronic blood component) layer on top of a hyperintense (acute blood component) layer. When membranes form within the SDH (a relatively frequent occurrence in chronic SDHs) multiple compartments, each having different attenuation characteristics due to containment of blood components of differing ages, can be seen.

MRI is more sensitive than CT in detection of small SDHs and tentorial and interhemispheric SDHs. Detection of these SDHs can be important in certain circumstances, e.g. when the presence of even a small SDH is considered significant (e.g. suspected child abuse). The MRI signal intensity of SDH also has a characteristic progression, being hypointense relative to gray and white matter on T2-weighted images during the first few days (due to blood in the stage of intracellular deoxyhemoglobin). SDHs become hyperintense on T1-weighted images after a few days, due to blood in the stage of intracellular methemoglobin. After the first few weeks, SDHs are hyperintense on both T1- and T2-weighted images, a finding which can persist for many months.

3B. Epidural Hematoma

Epidural hematomas (EDHs) are hemorrhagic collections which occur between the dura (which serves as the periosteum to the inner table of the skull) and the inner table of the skull. Because the dura is tightly adherent to the inner table at cranial suture sites, EDHs characteristically do not cross cranial suture lines (unlike SDHs, which can freely cross these sites). Whereas the extension of SDHs is restricted by the falx cerebri and tentorium cerebelli, this is not true of EDHs. These features, as well as the fact that EDHs usually clearly have a biconvex shape (and SDHs usually do not), help to distinguish EDHs from SDHs on CT and MRI.

EDHs are often neurosurgical emergencies which require immediate drainage in order to avert permanent neurologic deficit or death. They usually

occur after low-velocity, blunt trauma to the head, often to the temporal region. The onset of loss of consciousness is frequently immediate, but may be delayed following a so-called "lucent" interval while the EDH expands. The source of hemorrhage is arterial in 85%. Venous EDHs are seen more frequently in an occipital location, at the point of laceration of venous sinuses, and often have a delayed onset of many hours.

The typical CT appearance is a biconvex extra-axial fluid collection which has high attenuation due to the fresh clot. In rare cases, EDHs are isointense with gray matter if imaged before clot formation. Prominent mass effect, manifested by shift of midline structures, compression of the ipsilateral lateral ventricle, subfalcine herniation, and uncal herniation, is often present. CT is the best means of establishing the diagnosis because it is quick, sensitive and readily available and less expensive than MRI. MRI is rarely obtained to establish the diagnosis but, on occasion, small EDHs can be demonstrated when MRI is performed for non-emergency evaluation of brain injury. The signal intensity of EDH on MRI follows the same progression as that of SDH.

4. Subarachnoid and Intraventricular Hemorrhage

A small amount of subarachnoid hemorrhage (SAH) or intraventricular hemorrhage (IVH) is often seen following head trauma. The CT and MRI findings of SAH and IVH can be very subtle; CT is more sensitive than MRI in the detection of either form of hemorrhage. A high level of suspicion with special attention to particularly common sites of SAH (e.g. basilar cisterns such as the prepontine, ambient, interpeduncular and perimesencephalic cisterns) and IVH (e.g. in the dependent portions of the atria of the lateral ventricles) can help in detection. SAH is usually (but not invariably) accompanied by other intracranial injuries. In some circumstances, e.g. when a large amount of hemorrhage is present following only a small degree of trauma, cerebral angiography is often performed to exclude rupture of an aneurysm or vascular malformation.

5. Intraparenchymal Injuries

5A. Cortical Contusion

Cortical contusions are the one of the most common intraparenchymal injuries, accounting for slightly more than 40% of traumatic brain lesions. They occur on gyral surfaces, although they may sometimes extend into the adjacent white matter. The terms “coup” and “contre-coup” are often used to refer to cortical contusions. When the skull strikes an external object, the brain may itself strike the inner table of the skull at the site of impact (causing a "coup" injury) or slide forward over the rough contours of the floor of the anterior and middle cranial fossae. At the latter sites, the undersurface of the frontal lobes may be contused by the cribiform plate and lesser sphenoid wing and the undersurface of the temporal lobe by the petrous bone and greater sphenoid wing. The brain may also strike the inner table of the skull at a point 180 degrees from the site of impact, causing a so-called "contre-coup" injury. Contusions often have small amounts of petechial hemorrhage and surrounding edema.

About 50% of contusions are hemorrhagic on macroscopic examination of the brain or on CT. The CT appearance of hemorrhagic contusions is round or oval high attenuation foci at gyral surfaces, with surrounding \low attenuation edema and, sometimes, mild mass effect. Non-hemorrhagic contusions are often difficult to initially detect on CT, but become more conspicuous after a few days, when they have a hypointense appearance due to edema within the contused tissue. Delayed hemorrhage can be seen within initially non-hemorrhagic lesions a few days following trauma. After a few weeks, contusions become less conspicuous on CT examination, as hemorrhage and edema are resorbed, making contusions more difficult to detect. A residual focal region of tissue loss (encephalomalacia) is often seen, due to clearance of contused tissue by macrophages.

Contusions (especially subacute and chronic contusions) are more conspicuous on MRI for a number of reasons. First, the multiplanar capability of MRI allows imaging of lesions on the undersurface of the temporal or frontal lobes in the coronal and sagittal planes. These lesions are often difficult to detect on CT examination because of volume averaging with the floor of the anterior or middle cranial fossa. Second, the intrinsic contrast between contusions and normal brain tissue is greater on MRI examination, in large part because of increased water content within these lesions. Third, MRI is generally more sensitive to the presence of intraparenchymal hemorrhage than CT. Contusions which appear nonhemorrhagic on CT are often demonstrated to have hemorrhagic components on MRI. More importantly, the products of hemorrhage can be detected by MRI for years (rather than a few weeks, as on CT). Thus, the contusion (rather than merely the resultant areas of encephalomalacia) can be demonstrated by MRI over long periods of time. During the first few days following trauma, hemorrhagic contusions are hypointense on T2-weighted images and become hyperintense on T1-weighted, and subsequently T2-weighted images. Nonhemorrhagic contusions are hypointense on T1-weighted images and hyperintense of T2-weighted images. Chronic hemorrhagic contusions are hypointense on T2-weighted images due to the magnetic susceptibility effects of the blood products, a feature which can be accentuated by gradient echo pulse sequences.

5B. Diffuse Axonal Injury

Diffuse axonal injury (DAI) is relatively common in patients who suffer closed head injury. Patients with this type of injury often have immediate and sustained loss of consciousness. This injury results from shear/strain forces exerted primarily on brain white matter. These forces are produced by acceleration/deceleration and rotational motions of the head, even without direct impact of the skull with another object. The gray/white matter interfaces, e.g. the cortico-medullary junction, and white matter tracts (especially the corpus callosum and the corona radiata) are the preferential sites of injury. Most DAI lesions are nonhemorrhagic, and can be very difficult to detect on CT. Specific attention to preferred sites of involvement can optimize detection of these lesions. DAI lesions are usually more conspicuous on MRI than CT. Nonhemorrhagic DAI

lesions are hypointense on T1-weighted images and hyperintense on T2-weighted images, while hemorrhagic DAI lesions have various signal intensities corresponding to the age of their blood products.

5C. Subcortical Gray Matter Injury

This category refers to injury in the thalamus and basal ganglia, and accounts for only about 5% of intraparenchymal traumatic lesions. These lesions, however, are associated with severe initial neurologic impairment and poor neurologic outcome. The lesions often contain small regions of hemorrhage, possibly due to disruption of small perforating blood vessels. Nonhemorrhagic lesions are demonstrated on CT as hypointense regions of variable size within the lentiform or caudate nuclei or the thalamus. When hemorrhagic, these lesions are seen on CT as hyperintense foci, often with surrounding edema and mass effect. Like many other traumatic lesions, they are more conspicuous on MRI.

5D. Brainstem Injury

Brainstem injuries may be categorized according to whether they occur as the immediate result of trauma (so-called "primary" injury) or delayed in onset and due to mechanisms other than the force of impact (so-called 'secondary" injury). Primary brainstem injury can the result of forces directed on the brainstem from other structures (e.g. contusion or laceration by the incisura) or shear/strain (i.e. indirect forces) which can result in DAI. Brainstem DAI typically occurs in the dorsolateral portion of the pons or midbrain (sparing the medulla), and is usually associated with DAI lesions in the supratentorial compartment. Brainstem injuries are difficult to detect on CT because of beam-hardening artifact from the base of the skull, and are more easily seen on MRI because of the absence of this artifact and the better tissue contrast on MR imaging. Examples of secondary brainstem injury include hypoxic/ischemic injury or infarction (e.g. due to respiratory dysfunction, systemic hypotension or vascular thrombosis) and compression of the brainstem by uncal herniation.

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