Imaging of Traumatic Intracranial Hemorrhage

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Intracranial hemorrhage (ICH) is a common entity encountered in clinical emergency medicine. Imaging is the cornerstone in the diagnosis of traumatic ICH. In a large study of patients with a head injury and a decreased Glasgow Coma Scale (GCS), 46% of patients demonstrated intracranial hemorrhage. Of these, 30% were subdural hematomas (SDH), 22% were epidural hematomas (EDH), 22% were intraparenchymal hemorrhages (IPH), and 14% were subarachnoid hematomas (SAH). Current literature reports up to 72% incidence of diffuse axonal injury (DAI) in moderate to severe head injury. Timely and accurate diagnoses of ICH is key to successful patient management given the emergent nature of ICH.

CT is the initial modality of choice due to its accuracy, short study time, low cost, and robustness against artifacts. MR is also used given its increased sensitivity in detecting DAI, as well as subacute and chronic hemorrhage. Although lumbar punctures were previously more common in the diagnostic workup of SAH, there has been a paradigm shift away from performing this procedure, as nonenhanced CT (NECT) has been shown to be highly accurate in detecting SAH when performed within 6 hours of symptom onset.

This article will review the key vascular anatomy associated with ICH followed by a strategy for imaging evaluation and reporting. This is followed by a detailed review of the different types of traumatic ICH with an emphasis on imaging findings.

Meningeal Vascular Anatomy

The three meningeal layers (dura, arachnoid and pia mater) are perfused by the anterior, middle and posterior meningeal arteries. The anterior and posterior meningeal arteries originate from the anterior ethmoidal and ascending pharyngeal arteries, respectively, and perfuse dura of the anterior and posterior cranial fossa. The middle meningeal artery, a maxillary artery branch, enters the skull at the foramen spinosum. Trauma to the parietotemporal skull can lacerate these vessels resulting in hemorrhage.

Imaging Evaluation and Reporting

Given the critical nature of ICH, the interpreting physician must take necessary steps to prevent overlooking ICH on imaging. Although axial imaging is the standard for evaluating head CT, using additional reconstructions should be considered in routine clinical practice. ICH detection rate increases when axial and coronal images are viewed. Among missed acute intracranial hemorrhages, SDH accounts for 39%, followed by SAH with 33%. A customized checklist approach (Table 1) may be beneficial to the radiologist, particularly to ensure classic areas for subtle findings are evaluated (Figure 1). CT windows should also be considered when interpreting studies as subtle findings may be obscured due to windowing. In addition to standard CT head windows, the authors’ institution has found a width

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Table 1: Example Checklist for a Systematic Search Pattern for Subtle Intracranial Hemorrhage

<table>
<thead>
<tr>
<th>Axial images</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Check the posterior tip of occipital horns and interpeduncular cistern</td>
</tr>
<tr>
<td>• Check for asymmetric increased gyrus to inner table distance</td>
</tr>
<tr>
<td>• Check for asymmetrically effaced Sylvian fissure</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Coronal images</th>
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</thead>
<tbody>
<tr>
<td>• Evaluate for falcine/tentorial hemorrhage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Axial and coronal images</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Check for extra-axial fluid collection of the anterior and middle cranial fossa floor</td>
</tr>
<tr>
<td>• Check for vertex bleed</td>
</tr>
<tr>
<td>• Check for skull fracture and, if present, evaluate for contralateral injury</td>
</tr>
</tbody>
</table>

Imaging Features of Intracranial Hemorrhage

Diffuse Axonal Injury

Unequal rotational and acceleration-deceleration forces cause the brain to pivot around the brainstem, causing stretching and shearing of axons and resulting in the clinical diagnosis of DAI. Shear forces peak at the grey-white matter interface, due to its peripheral location and differences in tissue density; hence, 67% of DAI lesions are in this region. In more severe cases, additional sites of injury occur in the brainstem. Within hours, axonal deformation damages its cytoskeleton, resulting in arrest of axoplasmic flow on a microscopic level, followed by axonal swelling and subsequently axon rupture. Mechanisms of injury vary, but include high-impact falls and motor vehicle accidents.

Foci of axonal edema with or without hemorrhage are imaging findings of DAI and these foci may vary in size as the injury progresses.
Despite DAI being present in up to 72% of patients with moderate to severe head injuries, 50% to 80% of initial CT and MRI studies are negative. MRI is more sensitive than CT in detecting lesions (Figure 4). Gradient echo (GRE) and susceptibility-weighted imaging (SWI) are the most sensitive sequence, revealing the highest number of lesions at the highest number of locations compared to other MR sequences. The classic triad seen in DAI is diffuse damage to axons, a focal lesion in the corpus callosum, and a focal lesion in the dorsolateral quadrant of the rostral brainstem adjacent to the superior cerebellar peduncles. DAI lesions detected within 4 weeks after injury improve functional status and long-term outcome due to changes in patient management. Therefore, MRI is recommended within 4 weeks of traumatic brain injury (TBI).

Intraparenchymal Hemorrhage

Traumatic acute IPH, commonly referred to as a cerebral contusion, usually occurs after a significant direct head injury. It commonly involves brain parenchyma adjacent to bony protuberance/dural fold. CT displays a patchy, irregular, hyperdense area of acute hemorrhage on an edematous background (Figure 5). Hemorrhages may be multiple and bilateral. Cerebral edema may be relatively mild acutely; however, progression of cerebral edema is common and can result in midline shift and herniation syndromes. IPH can be complicated by continued enlargement and/or re-hemorrhage. Likewise, new lesions can be detected on follow-up imaging. MRI is useful to identify traumatic intraparenchymal damage without blood products, termed nonhemorrhagic cerebral contusion (Figure 6). Sequelae after resolution are common and include gliosis, encephalomalacia and associated volume loss.

Subdural Hematoma

Acute SDH is characterized by an extra-axial crescent-shaped hemorrhage in a potential space between the dura and arachnoid. A direct blow to the head is not necessary to incur an SDH. Instead, an SDH may be related to acceleration-deceleration forces secondary to the impact from a fall or shaking of an infant. Regardless of the mechanism, shear forces on the bridging veins or cortical arteries can lead to injury and rupture. Elderly are predisposed to develop SDH due to cortical atrophy. Additionally, anticoagulants and antiplatelet agents (Figure 7) raise concern for increased risk of hemorrhage. Contrary to epidural hematomas, classic SDH are limited by dural duplications, but can cross suture lines and, therefore, can extend over an entire hemisphere. The vast majority of small, atypically shaped, extra-axial

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**FIGURE 3.** Typical CT appearance of diffuse axonal injury. (A-D). Axial unenhanced CT images in a young male patient in a motor vehicle accident demonstrates numerous punctate hyperdensities indicating hemorrhage (yellow arrows) at junctions of the grey and white matter. Additional microhemorrhage in corpus callosum (blue arrow) and intraventricular hemorrhage (red arrow) are noted.

**FIGURE 4.** Improved sensitivity of MR over CT to detect DAI in an adolescent in a motorcycle accident. (A) Axial unenhanced CT demonstrates a subtle solitary punctate hyperdensity (arrow) suggestive of DAI. (B) Susceptibility-weighted MR image of the same patient later the same day at a similar level demonstrates considerably more extensive DAI involving bifrontal lobes (yellow arrows) and basal ganglia (red arrows) as demonstrated by small areas of susceptibility artifact.
hemorrhages are due to a low-pressure venous injury causing an SDH with continued CT follow-up suggested if clinical doubt remains as to whether an extra-axial blood collection reflects an EDH or SDH.

It is valuable for the radiologist to be generally aware of the appearance of blood products on CT based on the acuity. Although imaging may not occur this early, in the first few hours (hyperacute phase), SDH might be isodense to the underlying cortex, as it is still liquid (40-50 HU), and may not be detectable. Hemorrhage becomes increasingly dense as it coagulates and condenses during the acute phase (0-3 days) due to clot retraction by a decreased fluid component and relative increase in iron content in the red blood cells. Those clots measure between 80-100 HU and show the typical hyperattenuation. The attenuation decreases in the subacute phase as the blood products degrade and eventually becomes similar to CSF density in the chronic phase (Figure 8).21

SDH requires heightened vigilance and follow-up imaging to ensure early detection of complications, such as continued bleeding, rebleeding (8% of cases), midline shift, or herniation.22 Mixed attenuation blood in which isodense areas are intermixed by hyperdense areas suggest active (re)bleeding, which is termed the “swirl sign” on NECT. Cerebral spinal fluid
in the subdural space (subdural hygromas) is thought to be related to subdural hematomas. An arachnoid membrane tear with a “flap-valve” mechanism results in CSF leakage into the subdural space.\textsuperscript{23,24}

**Epidural Hematoma**

Hemorrhage into a potential space between the inner table of the skull and the dura mater occurs in 1% of minor head injury and 10% following head injury in patients who present in a comatose state.\textsuperscript{25,26} A provided history of head injury days-to-months prior to imaging should not eradicate the possibility of an EDH, as 20% to 50% of patients experience a symptom-free lucid interval.\textsuperscript{27} Of patients with an EDH, 91% suffer from an associated skull fracture.\textsuperscript{28} Typically supratentorially located, EDH frequently involves injury to the middle meningeal artery or anterior ethmoidal artery. Rapid hematoma expansion is common due to the relatively high pressure of arterial blood.\textsuperscript{29} Classic CT findings are a high-density convex or biconvex shaped extra-axial fluid collection that does not cross the sutures (Figure 9).

Venous epidural hemorrhage accounts for 5% to 10% of all EDH and occurs at specific locations including the vertex (superior sagittal sinus injury), anterior middle cranial fossa (sphenoparietal sinus injury), and
posterior occipital region (transverse/sigmoid sinus injury). The latter can rapidly cause tonsillar herniation. However, the majority of venous EDH are unlikely to expand because venous pressure is insufficient to further strip off the dura from the skull. Similar to arterial EDH, an associated fracture/diastasis is common. Venous EDH typically displaces the sinus away from the fracture and, in contrast to arterial EDH, can occasionally cross suture lines (Figure 10). One should suspect a venous EDH if a skull fracture runs through the expected location of a dural sinus.

Traumatic Subarachnoid Hemorrhage

Traumatic SAH (tSAH) results from disruption of pial vessels with bleeding into the subarachnoid space. It is often coexistent with other pathologies in the setting of trauma. Vessel injury more commonly arises in the cerebral convexities/sulci following a high-impact mechanical force to the head. Of patients with severe TBI, 40% develop tSAH. Be cautious with SAH in the setting of acute trauma as differential diagnosis includes tSAH as well as nontraumatic SAH (eg, ruptured aneurysm) that may have preceded (and perhaps led to) the reported trauma (Figure 11). Differentiating traumatic from nontraumatic SAH by imaging is not always possible. However, SAH identified around the cerebral convexities with relative sparing of the basilar cisterns around the circle of Willis would favor trauma. Similarly, SAH associated with other signs of trauma on CT such as a countercoup pattern associated with a parenchymal contusion may also favor a traumatic etiology. When doubt exists, the CT or traditional angiography may be performed for further evaluation to exclude an aneurysm.

Imaging characteristics that portend a worse prognosis include other evidence of brain trauma including brain contusions, especially if enlarging. Complications of tSAH include hydrocephalus and cerebral vasospasm. Table 2 summarizes differentiating key features of EDH, SDH and SAH.

Conclusion

Traumatic intracranial hemorrhage is an urgent finding requiring prompt and accurate evaluation by the interpreting radiologist with excellent communication and documentation of key findings that may affect patient management. Given the trauma and further complicating patient management, additional critical coexistent injuries may also be present and require urgent treatment. Thus, at many medical centers a multidisciplinary neurotrauma team

![Figure 11. Subarachnoid hemorrhage in three trauma patients with different etiologies. (A) Axial unenhanced CT demonstrates isolated, focal tSAH in asymptomatic elderly with supratherapeutic INR (10.4) following minor ground level fall without external head injury with high-density blood in the subarachnoid space on the right (arrow). (B) Axial unenhanced CT demonstrates a left tSAH (arrow) in a young patient after a blow to the right temple demonstrating a coup-contrecoup pattern. (C, D) Axial unenhanced CT (C) and right carotid approach cerebral angiogram (D) in a young patient who fell off a chair and minor facial trauma to the nose following a seizure. CT shows symmetric, midline SAH (arrow in C) is typical for aneurysmal SAH. Subsequent angiography confirmed nontraumatic etiology, a ruptured anterior communicating artery aneurysm (arrow in D). This case is a reminder to always consider aneurysmal etiology in SAH patients regardless of the history of trauma.](image-url)
FIGURE 12. Epidural hematoma with evacuation. (A) Axial unenhanced CT in an elderly patient with right parietal depressed skull fracture (arrow) following a ground-level fall. (B) On the same CT, an acute, right epidural hematoma is present as a largely high-attenuating biconvex extra-axial fluid collection with a central hypoattenuating component representing active bleeding (arrow). Note that the blood extends but does not cross the coronal suture anteriorly and lambdoid suture posteriorly. The patient was taken to surgery for evacuation. (C) Follow-up CT 6 weeks post-trauma demonstrates significant improvement with minimal residual blood products/dural thickening (arrow).

guides management and determines the need for surgical decompression/evacuation (Figure 12).

Furthermore, the radiologist must be vigilant about new and/or worsening edema, midline shift, signs of hydrocephalus, worsening hemorrhage (rebleeding), or other complicating factors on follow-up studies that may alter patient management. Medical and surgical management in these patients is complex and multifactorial requiring timely and accurate imaging information, thus solidifying the radiologists’ key role in the management of traumatic ICH.

**REFERENCES**


| Table 2. Classic Mechanisms and Imaging Feature of Intracranial Hemorrhage |
|---------------------------------|-----------------|-----------------|
| **Classic Trauma Mechanism**    | **Imaging Features**                       |
| Epidural hematoma               | High-impact blow to head | Biconvex fluid collection |
|                                 |                               | Crosses dural duplication |
|                                 |                               | Confined by sutures       |
| Subdural hematoma               | Fall, low-impact trauma       | Crescent-shaped           |
|                                 |                               | Crosses sutures but not dural duplications |
| Subarachnoid hemorrhage         | High-impact blow to head      | Blood products in subarachnoid spaces including sulci and basal cisterns |


