Radiology Corner (Case #2)

Subdural Hematoma

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Note: This is the full text version of the radiology corner question published in the June 2006 issue, with the abbreviated answer in the July 2006 issue.

Subdural hematoma is a common injury in both military active duty and their dependents, including children subject to nonaccidental trauma. The subdural hematoma (SDH) is a potentially devastating, yet curable extraaxial fluid collection classically associated with head trauma including rapid acceleration or deceleration that produces tearing of the bridging veins as they cross from brain to dural sinus. Clinical manifestations may be nonfocal and nonspecific and a careful history taking and an appropriate degree of clinical suspicion are necessary for timely diagnosis. A noncontrast head CT is fast, highly accurate, and with few exceptions, should always be the first study obtained in the evaluation of acute head trauma. MR imaging – especially FLAIR and diffusion sequences - is useful for its ability to determine the age of the hematoma and is superior for the identification and characterization of more subtle associated CNS injuries. Treatment of nonacute subdural hematoma may involve craniotomy-guided hematoma evacuation, depending on the size and the amount of mass effect.

Introduction

Subdural hematomas are common injuries in both military active duty and dependent beneficiaries. It can be associated with high morbidity and fatality, depending on the size of the hematoma, the mass effect it causes, and the presence of and severity of other associated brain injuries. As many patients can experience rapid clinical improvement after successful evacuation of the extraaxial fluid collection, early and accurate diagnosis is paramount for timely treatment and recovery. The following case report reviews the typical clinical presentation, imaging findings, and treatment of subdural hematoma.

History

An 18-year-old Hispanic male was brought by ambulance to the Emergency Department status post high-speed motor vehicle collision in which the patient was ejected from the vehicle. He was lethargic on presentation, but without evidence of any focal neurological deficits. Physical examination in the trauma bay showed swelling over his left temporal region and scattered facial abrasions. A noncontrast head CT was immediately obtained (Fig. 1)

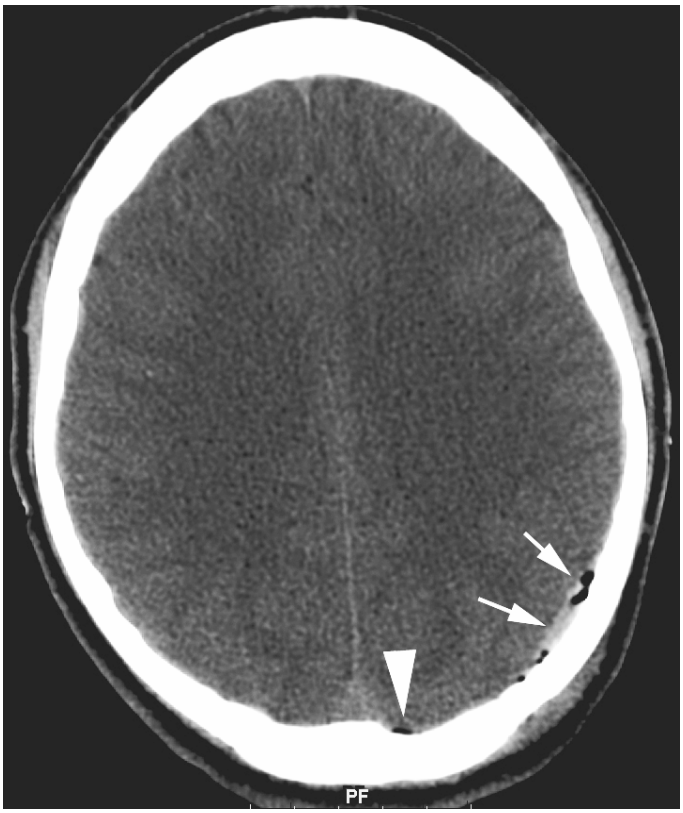

Figure 1a. Axial noncontrast head CT image shows a crescent-shaped hyperdense fluid collection in the left parieto-occipital subdural space, consistent with an acute subdural hematoma (arrows). The presence of several foci of air within the subdural collection as well as a single focus of air in the extraaxial space of the left occipital region (arrowhead) is indicative of the presence of a concomitant fracture communicating with a sinus or the skin surface.

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Imaging Findings

The noncontrast head CT revealed a small hyperdense subdural hematoma, consistent with the acute injury (Fig. 1a). There is pneumocephalus, seen as small bubbles of air within the left extraaxial parietal subdural collection as well as a single focus of air posteriorly at the left paramidline aspect of the extraaxial space shown (Fig. 1a).

These findings indicate that a fracture has occurred, allowing communication of outside air or a sinus cavity with the intracranial space. Indeed a complex adjacent temporal bone fracture was detected and is best appreciated using bone windows (Fig. 1b).

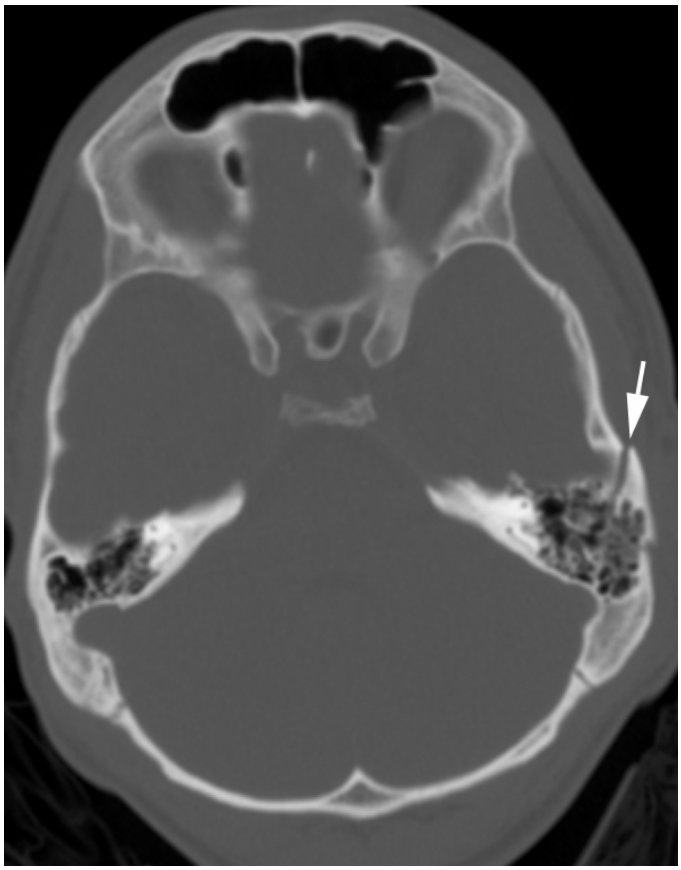

Figure 1b. Axial noncontrast head CT image with parameters optimized for evaluation of osseous structures (bone windows) shows a nondisplaced longitudinal left temporal bone fracture (arrow) with extension into the adjacent mastoid air cells. This fracture is complex as there is also a transverse component, which is not shown here.

Companion cases are provided for demonstration of the less common interhemispheric (Fig. 2) and subtentorial (Fig. 3a) locations for subdural hematomas; and, an acute frontal subdural hematoma in the setting of nonaccidental trauma (Fig. 3b), the difference in CT appearance between a subdural and an epidural hematoma (Fig. 4), and the typical appearance of a chronic subdural hematoma (Fig. 5).

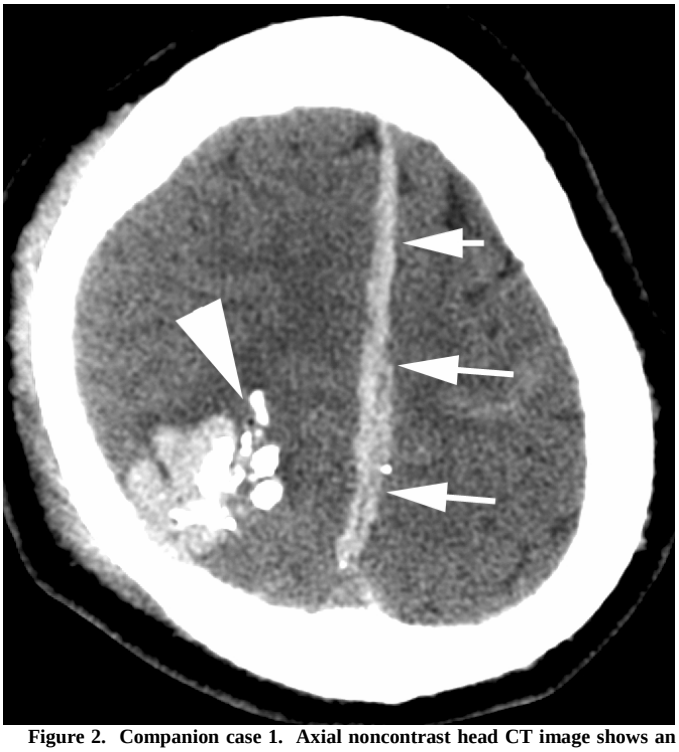

Figure 2. Companion case 1. Axial noncontrast head CT image shows an interhemispheric subdural dense collection (arrows) consistent with an acute hematoma. The irregular extremely dense material represents bone chips and bullet fragments (arrowhead), with some surrounding contusion and mild vasogenic edema of the right parietal parenchyma.

Discussion

Subdural hematomas are commonly occurring extraaxial fluid collections within the potential subdural space, between the arachnoid membrane and the dura mater. Aside from being caused by severe trauma with cortical bridging vein tearing, they can also occur with penetrating trauma, parenchymal contusions, and in patients who have a hemorrhagic diathesis. They represent approximately 5% of all head injuries and 65% of head injuries with prolonged loss of consciousness.

Subdural hematomas may be classified by age of the hematoma (which can be determined by clinical history, and estimated by CT density and MR signal intensity). Although clinical definitions vary, most acute subdural hematomas are equal to or less than three days of age (some report up to 7 days) and are often the result of high velocity acceleration or deceleration (usually motor vehicle-related) leading to tearing or shearing of cortical bridging veins or dural venous sinus. With an acute presentation, there may be associated parenchymal injuries that contribute to the poor prognosis associated with this injury. Subacute lesions are between 4 to 21 days old and in this setting a clear history of trauma may not be known. MR imaging is very sensitive for detecting blood products – like methemoglobin - that are commonly seen in subacute hemorrhage. Chronic subdural hematomas are greater than 21 days old. They have a tendency to present in older patients and after non-accidental trauma in children and may be associated with falls. They tend to have mild or

minimal symptoms and signs; and, the preceding trauma may not be remembered. Bilateral SDH collections are detected in 20 - 25% of cases. The chronic SDH is often organized within an encapsulating membrane – produced by fibroblasts from the dural border of the hematoma. Patients with chronic collections typically report trivial or no known history of head trauma. In children, chronic subdural hematoma may be caused by unrecognized or unreported accidental trauma, nonaccidental trauma or rarely, birth trauma. Rarely, subdural hematomas can be the result of metastasis or aneurysm rupture.

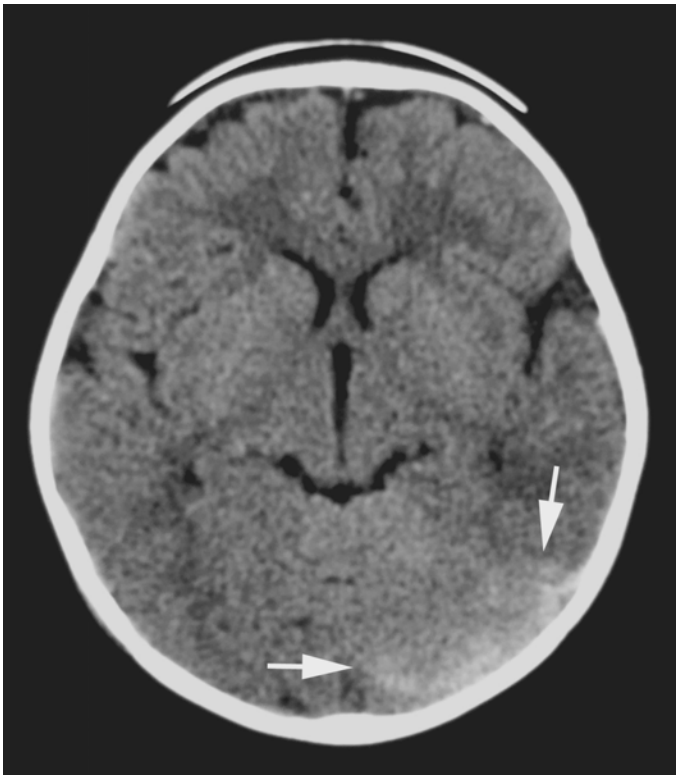

Figure 3a. Companion case 2. Noncontrast axial head CT in an infant, who was brought to the Emergency Center with decreased alertness and possible recent loss of consciousness of unknown cause reveals ill-defined increased density over the left cerebellar tentorium (arrows) consistent with an acute subdural hematoma in a location where subdural hematomas are more frequently missed.

Risk factors include coagulopathic state, alcohol abuse, seizure disorder, prior placement of a ventricular shunt, infancy, and anticoagulant use. The elderly are also predisposed to developing subdural hematomas due to the increased extraaxial CSF space caused by normal brain changes of aging. Falls are commonly the inciting factor, as noted in one study with showing 77% of elderly patients over 65 years of age who had chronic subdural hematomas reported a history of a preceding fall (1). The elderly are predisposed to developing subdural hematomas due to parenchyma atrophy, resulting in diffusely increased CSF space within the calvarium. The relative enlargement of the subarachnoid space allows greater movement of the brain relative to the dura – with resultant stretching and rupture of the bridging veins at their junction with the dural sinuses. Although associated with severe trauma in other groups, in the elderly population even minor trauma (such as vomiting and sneezing) can result in tearing or shearing of the cortical bridging veins, leading to slow accumulation of blood products within the subdural space – often bilateral.

In infants, the presence of subdural hematoma requires special attention and clinical suspicion for nonaccidental trauma, as it is the most common manifestation of head trauma associated with shaken baby syndrome (2). Infants, compared with adults, especially under the age of six months, have weaker neck muscles and large heads, and are unable to protect themselves from the damaging effects of a sagittal whiplash force caused by violent shaking. Subdural hematomas in infants are only rarely caused by accidental injury. A heterogeneous subdural hematoma, consisting of blood products of different ages, has been considered highly suggestive of repetitive nonaccidental head trauma. Recent literature has shown that heterogeneous signal intensity on MR can be a result of other factors, and not necessarily indicative of nonaccidental trauma. (3) In infants, subdural hematoma occurs most commonly in the interhemispheric fissure location.

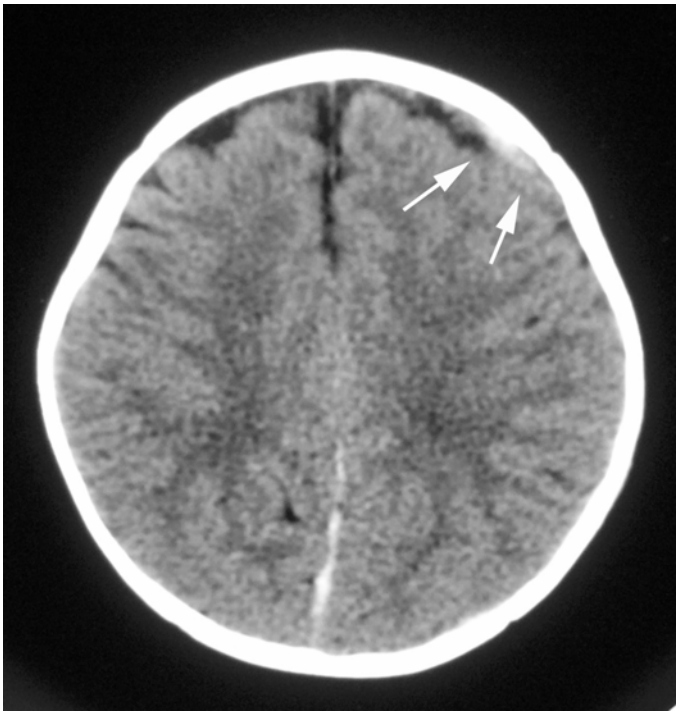

Figure 3b. Companion case 2. Noncontrast axial head CT of the brain in the same patient shows a small amount of asymmetric high density material with a crescentic shape, located at the left frontal subdural space (arrows), consistent with an acute subdural hematoma. The presence of the acute subdural hematomas and suspicious clinical history led to a skeletal survey (not shown), which revealed multiple skeletal fractures of different ages, consistent with the diagnosis of nonaccidental trauma.

Other CNS findings supportive of nonaccidental head trauma include cerebral contusion, subarachnoid hemorrhage, diffuse axonal injury, and intraoccular retinal hemorrhage; the

latter finding is significantly more common in nonaccidental head trauma than in infants who have suffered head impaction (77.5% versus 20%) (4). According to some authors, the combination of subdural hematoma and retinal hemorrhage is believed to be very suggestive of non-accidental trauma (5). Additional imaging evaluation in suspected nonaccidental trauma involves a skeletal survey, with the intent of identifying other possible fractures, which in the setting of nonaccidental trauma, are classically of different ages and therefore varying stages of healing.

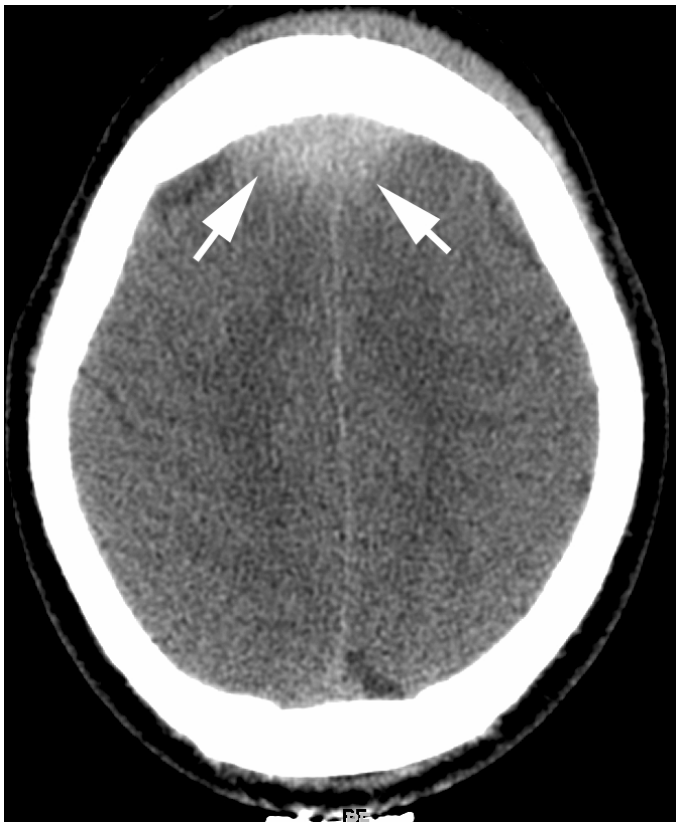

Figure 4. Companion case 3. Noncontrast axial head CT of the brain shows a classic acute epidural hematoma, which is lens-shaped or biconvex with extension across the midline (arrows), distinguishing it from a subdural hematoma, which is limited by the interhemispheric falx.

Symptoms of subdural hematomas are relatively nonspecific, often presenting as insidious global decline in mental status and without focal neurological deficits. Headache is present in up to 80% of cases. Other nonspecific symptoms include memory impairment, confusion, lethargy, weakness, nausea, vomiting, seizure activity, and visual changes. Larger hematomas may be associated with paralysis and coma. Conditions that may clinically mimic subdural hematoma include dementia, cerebrovascular accident, transient ischemic attack, encephalitis, brain tumor, and abscess.

Imaging findings

Head CT, due its high sensitivity and specificity for subdural hematoma, should be the first imaging study obtained in the evaluation of head trauma. Subdural hematoma has characteristic imaging findings on CT. Classically crescentic in morphology, they can extend across suture lines and into the paramidline interhemispheric space. They may dive into the interhemispheric fissure to cross the midline, by wrapping around the interhemispheric falx and also the tentorium cerebelli. The density of the subdural collection can vary based on the age of the traumatic injury. Acute hematomas usually have high attenuation, unless the patient is anemic or has a clotting disorder, in which case the density of the fluid may be low. Acute SDH can spontaneously disappear or progress to subacute and chronic types. Subacute hematomas show intermediate attenuation or are isodense to brain tissue, and chronic subdural hematomas may have a density identical to that of cerebrospinal fluid – unless they have been complicated by re-bleeding.

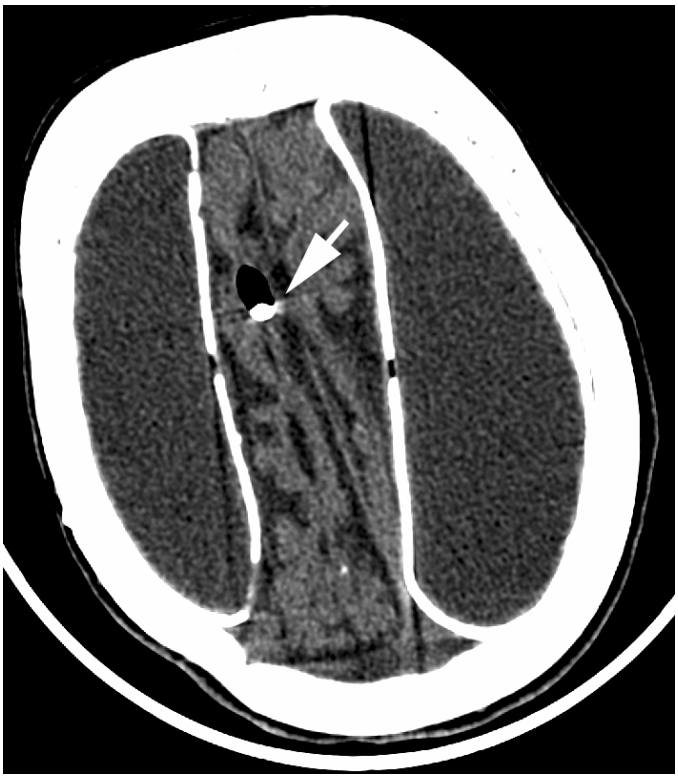

Figure 5. Companion case 4. Noncontrast axial head CT of the brain shows bilateral large, homogeneously low attenuating, extraaxial fluid collections that extend across suture lines but not into the interhemispheric fissure. These findings are consistent with chronic bilateral subdural hematomas, showing marked compression of both lateral ventricles. These subdural collections demonstrate a thin, interrupted rim of dystrophic calcification, which also supports the chronic nature of this process. Note the ventricular shunt (arrow) in place for drainage of hydrocephalus, caused by significant mass effect on the ventricular system.

Isodense subdural hematomas are commonly missed; factors contributing to this failure to diagnose include: small size, orientation in the plane of injury, narrow CT window settings, anemia, coagulopathic state, subacute or chronic age of the fluid showing a density similar to adjacent cortex or CSF respectively, or dilution with CSF due to presence of an arachnoid tear (true hygroma). Note that following administration of intravenous contrast, a thin enhancing margin may be appreciated in organized collections with membranes. In addition, there is normal brain enhancement that may highlight the fluid collection –which should not enhance. The SDH collection is more commonly homogeneous. Heterogeneity or "swirling," when present, reflects the mixing of blood products of different ages and/or acute unclotted blood intruding into clot. A fluid-fluid level within the subdural collection can result from layering of the higher density material in the dependent location and the hypodense serum superiorly.

Unlike epidural hematomas, subdural hematomas are not usually associated with skull fracture. Associated parenchymal changes are seen with larger subdural collections and include mass effect with sulcal effacement, medial buckling of the gray-white interface away from collection, ipsilateral ventricular compression, and midline shift/subfalcine herniation due to ipsilateral parenchyma contusion and edema.

MR imaging is useful for confirming smaller suspected chronic subdural hematomas that may have a CT density that is similar to the cerebrospinal fluid. MR imaging also allows assessment of the size and extent of the lesion in multiple planes (the coronal plane is very useful) and is superior for the evaluation of associated parenchymal brain injuries – such as deep white matter injury and contusion. Moreover, MR imaging signal characteristics of the extraaxial collection can also suggest information on the age of the collection. Acute blood appears T1 isointense and T2 hypointense. Subacute blood can be T1 hyperintense and T2 hypo- or hyperintense. Chronic blood is hypointense (or isointense to CSF) on both T1- and T2-weighted MR imaging sequences. An additional GRE sequence is useful for identifying suspected parenchymal hemorrhage on any MR imaging study. Yet another advantage of MR imaging is the detection of very small subdural hematomas that are easily missed on CT, particularly when the hematoma is oriented in the plane of the CT scan (e.g. tentorial subdural hematomas).

In the young infant with open fontanelles, head sonography may show a curvilinear fluid space between the calvarium and adjacent brain, flattening of the gyri, ventricular distortion, and extension into the interhemispheric fissure space.

Differential considerations for subdural hematoma include epidural hematoma, subdural hygroma, and subdural empyema. Epidural hematomas are classically lens-shaped ("lenticular") but, unlike subdurals, are usually confined or limited by the cranial sutures. Subdural hygromas are frequently a delayed complication of head trauma and can mimic the appearance of a subdural hematoma (6). They result from a tear in the arachnoid membrane, with CSF dissecting into the subdural space. Subdural empyema, like chronic subdural hematoma, can show rim enhancement with intravenous contrast. Diffusion-weighted imaging may be used to differentiate between the two entities, as empyemas will show restricted diffusion.

Treatment

The goal of treatment of subdural hematoma is the removal of the fluid collection. Acute collections may require craniotomy. Because SDH may be under high intracranial pressure resultant from associated injuries, patients with the acute form of hematoma do not tend to do as well, particularly if the collection is bilateral or if surgery occurs greater than four hours following the initial insult. Nonacute collections can be drained via a catheter placed into the subdural space, across burr hole or twist drill craniostomy, because clot lysis tends to increase with increasing age of the hematoma. Drainage via craniostomy has become the treatment of choice because of its low morbidity and mortality (7). The mortality associated with acute subdural hematomas is generally higher than that seen with epidural hematomas due to the association with other injuries and ranges from 50 to 85%. However, dramatic clinical improvement can be seen in many cases with early detection and successful drainage of the subdural hematoma.

Note: Follow this link for a case in the MedPix™ *digital teaching file with more information and Category 1 CME and CNE*:

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