CLINICAL PICTURE

26 years old male patient presented clinically with a grand male fit, confusion, fever, headache, and nausea. Examination showed bilateral papilledema and left sided extensor planter response. The patient gave a history of nasal sinusitis two weeks before clinical presentation.

RADIOLOGICAL FINDINGS

**Figure 1.** MRI T1 pre and post contrast scans showing two hypointense, oval, elongated, lentiform-like cystic lesions in the medial frontal (near the frontopolar region) and medial occipital regions. The lateral borders of the lesions are crescent shaped and the medial borders are flattened. The signal intensity of the intracystic fluid is higher than that of the CSF. The lesions are extraaxial in location, orientated along the falx cerebri and interhemispheric fissure and their capsules are densely enhanced after contrast injection. The pachymeninges are thickened and showed dense contrast enhancement. Notice poor or nonvisualization of the cortical sulci, and medial displacement of the white matter digitations ("white matter buckling") mostly due to diffuse brain edema. Some degree of venous congestion is probably present.
Figure 2. A case of subdural empyema (abscess). Postcontrast MRI T1 images showing bilateral oval and elongated extra-axial cystic lesions with densely enhanced capsules orientated along the falx cerebri, tentorial cerebelli and the interhemispheric fissure with medial fattened borders and lateral crescent-shaped borders. The pachymeninges are thickened and showed dense contrast enhancement. The nasal sinuses on the left side show thickened mucosa. Notice poor or nonvisualization of the cortical sulci, and medial displacement of the white matter digitations ("white matter buckling"), mostly due to diffuse brain edema. Some degree of venous congestion is probably present.

Figure 3. A case of subdural empyema (abscess). Postcontrast MRI T1 images showing bilateral oval and elongated extra-axial cystic lesions with densely enhanced capsules orientated along the falx cerebri, tentorial cerebelli, with medial fattened borders and lateral crescent-shaped borders. Notice the medial frontopolar abscess with enhanced capsule, another crescent-shaped abscess could be appreciated along the medial aspect of the tentorial ridge on the left side with a densely enhanced capsule. The pachymeninges are thickened with dense contrast enhancement. The nasal sinuses show thickened mucosa. Notice poor or nonvisualization of the cortical sulci, and medial displacement of the white matter digitations ("white matter buckling"), mostly due to diffuse brain edema. Some degree of venous congestion is probably present.
Figure 4. MRI T2, and FLAIR studies showing the frontopolar extra-axial abscess surround by edema. The abscess has a hypointense capsule. The mechanism of the capsule hypointensity on the T2 weighted scan is not certain. Proposals in this respect include: 1-Relative lack of water in the fibrous capsule. 2-The presence of blood products in the abscess capsule[deoxyhaemoglobin induces T2 hypointensity,while methemoglobin induces T1 hyperintensity]. 3- The presence of paramagnetic free radicals within the phagocytosing macrophages which are heterogeneously distributed in the periphery of the abscess, paramagnetic free radicals induce T2 hypointensity. The signal intensity of the intracystic fluid is different from that of the CSF on the FLAIR image (C). Notice the parenchyma edema that surrounds the frontal extra-axial abscess. The subdural abscess along the tentorial ridge could also be seen as a hyperintense crescent- shaped lesion.
Figure 5. The extraaxial subdural abscesses have a hypointense capsule on the T2 and FLAIR images. The capsule is densely enhanced on the postcontrast T1 scans. Notice the parenchyma edema that surrounds the frontal extra-axial abscess.

Box 1. Extra-axial subdural abscesses

1. Extraaxial empyemas usually develop 1-2 weeks following sinusitis or mastoiditis by retrograde thrombophlebitis of the transdiploic veins.

2. Subdural empyema (ie, abscess) is an intracranial focal collection of purulent material located between the dura mater and the arachnoid mater. About 95% of subdural empyemas are located within the cranium; most involve the frontal lobe, and 5% involve the spinal neuraxis.

3. The subdural extra-axial abscesses are multiple, scattered along the falx and tentorial cerebelli, the interhemispheric fissure and extra-axial in location.

4. The medial borders of the subdural abscesses are flattened and the lateral borders are crescent shaped. The medial growth of these abscesses are limited by the rigid dura resulting in flattening of the medial borders of the subdural abscesses along the the falx and tentorial cerebelli. SE is a primarily intracranial infection located between the dura mater and the arachnoid mater. It has a tendency to spread rapidly through the subdural space until limited by specific boundaries (eg, falx cerebri, tentorium cerebelli, base of the brain, foramen magnum). The subdural space has no septations except in areas where arachnoid granulations are attached to the dura mater. SE is usually unilateral.

5. Pachymeningitis is invariable present with thickening and enhancement of the pachymeninges.

6. The extraaxial subdural abscesses have a hypointense capsule on the T2 and flair images. The capsule is densely
**DISCUSSION**

- **Extra-axial Fluid and Pus Collections**
  - **Subdural Effusion and Hygroma**

Extra-axial collections associated with meningitis may be infected or sterile. The most frequent type of collection is a sterile subdural effusion or hygroma. Presumably, such subdural effusions are secondary to irritation of the dura mater by the infectious agent or by its products. Alternately, effusions can occur secondary to inflammation of the subdural veins with an attendant increase in protein and fluid within the subdural space. These collections are typically isodense to cerebrospinal fluid on CT scans and isointense to cerebrospinal fluid on all MR imaging sequences. Generally, subdural effusions do not require specific treatment and resolve spontaneously over weeks to months with effective specific therapy directed at the infectious agent responsible for the meningitis.

Subdural effusion appears as a low-density area over the surface of the cerebra hemispheres and seems to occur commonly in influenzal meningitis. The pathogenesis is not clear, but the presence of high albumin to gamma globulin ratio in the subdural effusion, compared with that in the serum, suggests that the effusion forms by passage of fluid through irritated or damaged blood vessels (increased capillary permeability). This concept seems to be further borne out by the passage into subdural effusion of the human serum albumin injected intravenously, indicating that subdural effusion is derived from plasma.

- **Subdural Abscess (Empyema)**

Purulent subdural infections (i.e., abscess, empyemas) account for about 13% to 20% of all cases of intracranial infection. Bacterial or fungal infection of the calvaria or paranasal sinuses can directly spread to the subdural space and produce an empyema. Frontal sinusitis is, in fact, statistically the most common cause of subdural empyema. The mechanism of subdural infection in these cases is usually secondary to retrograde thrombophlebitis via the calvarial emissary veins. Such extra-axial infections are less commonly due to direct spread of the bacterium after traumatic penetration of the skull.

Figure 6. Left sided isodense subdural effusion, notice the midline shift.

Figure 7. Five-month-old patient with subdural effusion and hygromas secondary to Haemophilus influenzae meningitis. Contrast-enhanced CT scan demonstrates bifrontal crescentic cerebrospinal fluid density of subdural effusion and hygromas.

The most common locations of subdural empyemas are the cerebral convexities and the interhemispheric fissure. Unenhanced CT scanning reveals a crescentic or lentiform extra-axial fluid collection that is slightly denser than
cerebrospinal fluid. On CT scan after intravenous contrast administration, an overlying peripheral rim of enhancement of varying thickness is identified. This rim represents an inflammatory membrane of granulation tissue on the leptomeningeal surface bordering the empyema. Hypodensity or contrast enhancement of the adjacent brain parenchyma may also be seen, secondary to thrombophlebitis of the bridging draining veins crossing the subdural space, resulting in venous occlusion and venous infarction of the involved brain. [7,8]

Subdural effusions are low-protein collections that are isointense relative to cerebrospinal fluid, whereas empyemas are more proteinaceous and have higher MR T1 imaging signal intensities than that of normal cerebrospinal fluid. MR imaging is now considered the modality of choice in the evaluation of subdural empyema. Although the findings on CT scanning may be equivocal, MR imaging helps differentiate between aseptic subdural effusions and infected subdural empyemas by virtue of the relatively shortened T1 relaxation times of purulent fluid, in comparison with otherwise normal cerebrospinal fluid signal within a sterile subdural effusion. Subdural effusions are low-protein collections that are isointense relative to cerebrospinal fluid, whereas empyemas are more proteinaceous and have higher MR imaging signal intensities than that of normal cerebrospinal fluid. After intravenous contrast administration, the internal and the external inflammatory membranes of subdural empyemas enhance on MR imaging. The external membrane often is not appreciated on CT scanning because the adjacent radiodense skull obscures the contrast enhancement. When present, concomitant parenchymal changes of edema and venous infarction are best visualized on T2-weighted or contrast-enhanced T1-weighted images. [1,2,3]

Subdural empyema probably represents infected subdural effusion. Compared with subdural effusion, in which development of a subdural membrane is possible but rare, the membrane is the hallmark of subdural empyema. Subdural empyema occasionally may be isodense with the brain. If it is not of a significant size or it is bilateral but small, then it might be "missed" on the plain CT scan. It is, however, possible to at least suspect significant isodense subdural empyema on the plain CT scan by compression and elongation of the lateral ventricles, poor or nonvisualization of the cortical sulci, and medial displacement of the white matter digitations ("white matter buckling"). It is, therefore, advisable to perform a high-dose contrast CT scan (60 to 80 gm of iodine) in all cases of suspected subdural empyema because its early recognition should initiate its prompt surgical evacuation. In exceptional cases in which diagnosis of subdural empyema cannot be made with confidence on the basis of CT findings alone, angiography may be performed. Hypertrophy of meningeal branches strongly favors the presence of subdural empyema under such circumstances. [4,5,6]

Subdural and epidural empyemas are collections of purulent material most commonly caused by anaerobic streptococci, staphylococci and Gram-negative enterics. These subdural and epidural infections are uncommon, accounting for only

**Aetiopathogenesis of subdural empyema**

Subdural and epidural empyemas are collections of purulent material most commonly caused by anaerobic streptococci, staphylococci and Gram-negative enterics. These subdural and epidural infections are uncommon, accounting for only
20-33% of all intracranial infections. Empyemas can result from the complications of meningitis or from haematogenous spread from a distant focus. Other causes include direct implantation through surgery or trauma. In adults, the most common cause is sinusitis or mastoiditis. In infants, a meningitis which induces an effusion is commonly the cause. [7,8]

Extraaxial empyemas usually develop 1-2 weeks following sinusitis or mastoiditis by retrograde thrombophlebitis of the transdiploic veins. The thrombophlebitis progresses to an irreversible thrombosis of the dural sinuses and venous structures leading to secondary parenchymal infection and infarction. The high morbidity and mortality rate (25-50%) along with the clinical and radiologic findings are related more to the response of the cerebral vasculature and brain to the inflammatory response and less to the mass effect of the extraaxial collection. Prompt surgical treatment is a requirement since systemically administered antibiotics do not penetrate the subdural space in therapeutic amounts. Aggressive surgical therapy is also important in limiting the amount of neurological deficits. [1,3,7,8]

Postoperative and posttraumatic empyemas, in contrast to otorhinologically induced empyemas, occur months to years after the initial incident with few or minimal signs and symptoms. The benign course is due to formation of a limiting membrane from the previous surgery or trauma which acts as a barrier protecting the underlying CNS structures. [2,3,7,8]

Because of the fulminant nature of extraaxial empyemas, prompt recognition is a necessity. MR imaging is superior to CT for demonstrating these lesions by enabling more sensitive detection, more accurate localization and more complete delineation of the disease. Superficial lesions are particularly easier to detect due to the absence of bony artifacts with MR imaging. Magnetic resonance imaging also allows the differentiation between benign and purulent effusions, because on both the T1- and the T2-weighted images, a higher signal intensity will be seen with empyemas. [7,8]

Figure 10. Subdural empyema, MRI T1

Posttraumatic empyemas are hypointense on both the T1- and the T2-weighted images when compared to most chronic subdural haematomas. Also, MR imaging is more specific in differentiating subdural from epidural empyemas. A hypointense medial rim is seen in epidural collections, but not in subdural effusions. Improvement in prognosis can be expected with the use of MR imaging because of early and accurate diagnosis as well as the ability to monitor therapy. [7,8]

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**SUMMARY**

Subdural empyema (ie, abscess) is an intracranial focal collection of purulent material located between the dura mater and the arachnoid mater. About 95% of subdural empyemas are located within the cranium; most involve the frontal lobe, and 5% involve the spinal neuraxis. This case record focuses on the intracranial type, which causes clinical problems through extrinsic compression of the brain by an inflammatory mass and inflammation of the brain and meninges. Subdural empyema (SE) is a life-threatening infection that was first reported in the literature about 100 years ago. It accounts for about 15-22% of focal intracranial infections. The mortality rate approached 100% before the introduction of penicillin in 1944 and has declined since that time. Because the symptoms might be very mild initially, rapid recognition and treatment are important; the early institution of appropriate treatment gives the patient a good chance of recovery with little or no neurological deficit.

SE is a primarily intracranial infection located between the dura mater and the arachnoid mater. It has a tendency to spread rapidly through the subdural space until limited by specific boundaries (eg, falx cerebri, tentorium cerebelli, base of the brain, foramen magnum). The subdural space has no septations except in areas where arachnoid granulations are
attached to the dura mater. SE is usually unilateral.

With progression, SE has a tendency to behave like an expanding mass lesion with associated increased intracranial pressure (ICP) and cerebral intraparenchymal penetration. Cerebral edema and hydrocephalus also may be present secondary to disruption of blood flow or cerebrospinal fluid (CSF) flow caused by the increased ICP. Cerebral infarction may be present from thrombosis of the cortical veins or cavernous sinuses or from septic venous thrombosis of contiguous veins in the area of the SE. In infants and young children, SE most often occurs as a complication of meningitis. In such cases, SE should be differentiated from reactive subdural effusion (ie, sterile collection of fluid due to increased efflux of intravascular fluids from increased capillary wall fenestrations into the subdural space). In older children and adults, it occurs as a complication of parasanal sinusitis, otitis media, or mastoiditis.

Infection usually enters through the frontal or ethmoid sinuses; less frequently, it enters through the middle ear, mastoid cells, or sphenoid sinus. This often occurs within 2 weeks of a sinusitis episode, with the infection spreading intracranially through thrombophlebitis in the venous sinuses. Infection also may extend directly through the cranium and dura from an erosion of the posterior wall of the mastoid bone or frontal sinus. Direct extension also could be from an intracerebral abscess. Rarely, infection spreads hematogenously from distant foci, most commonly from a pulmonary source or as a complication of trauma, surgery, or septicemia. The sphenoid sinus also could be a source of infection.

- Cranial MRI is now the imaging study of choice, being superior to cranial CT scan in outlining the extent of SE and demonstrating the convexity and interhemispheric collections.
  - MRI also shows greater morphological detail than CT scan.
  - The sensitivity of MRI is improved by using gadolinium contrast medium.
- Cranial CT scan was the standard technique for quick diagnosis before the advent of MRI. The use of high-resolution, contrast-enhanced CT scan increases diagnostic yield, although it sometimes gives equivocal or normal results.
  - On CT scan, SE shows as a hypodense area over the hemisphere or along the falx; the margins are better delineated with the infusion of contrast material. Cerebral involvement also is visible.
  - Cranial osteomyelitis may be seen.
  - CT scan is the modality of choice if the patient is comatose or critically ill and MRI is not possible or is contraindicated.
- Cranial ultrasound has been helpful in differentiating SE from anechoic reactive subdural effusion in infants with meningitis accompanied by complex features (eg, increased echogenicity in the convexity collections, presence of hyperechoic fibrinous strands or thick hyperechoic inner membrane, and increases in echogenicity of the pia-arachnoid).

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**Addendum**

- A new version of this PDF file (with a new case) is uploaded in my website every week (every Saturday and remains available till Friday.)
  - To download the current version follow the link "http://pdf.yassermetwally.com/case.pdf".
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  - At the end of each year, all the publications are compiled on a single CD-ROM, please contact the author to know more details.
  - Screen resolution is better set at 1024*768 pixel screen area for optimum display
References


