Supratentorial subdural empyema

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The supratentorial subdural empyema is a subclassification of a intracranial subdural empyema.

Etiology

see Subdural empyema etiology.

Pathophysiology

The underlying arachnoid and subarachnoid spaces are usually unaffected, but a large subdural empyema may produce a mass effect. Further, a thrombophlebitis may develop in the bridging veins that cross the subdural space, resulting in venous occlusion and infarction of the brain.

Symptoms

Include those referable to the source of the infection. In addition, most patients are febrile, with headache and neck stiffness, and, if untreated, may develop focal neurologic signs, lethargy, and coma. The CSF profile is similar to that seen in brain abscesses, because both are parameningeal infectious processes.

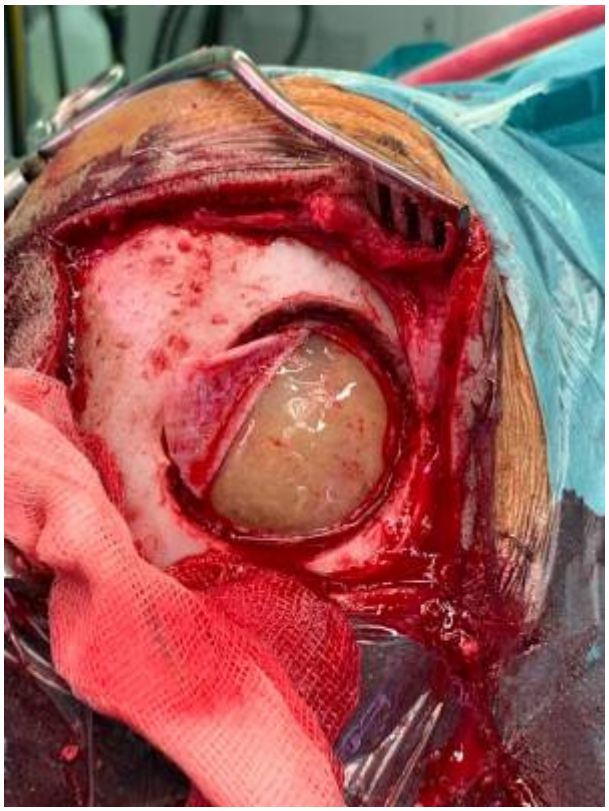
Diagnosis

Diagnosis of SDE is based on a strong clinical suspicion and the clinical features include fever, altered mental state, focal neurological deficits, and seizures with a fulminant and rapid downhill course.

CT typically demonstrates a crescent-shaped extra-axial fluid collection that can be either iso- or hyper-attenuating compared with the cerebrospinal fluid. Classically, there is also enhancement of the inner membrane with contrast administration [1].

MRI has become the imaging modality of choice. Increased signal intensity is usually seen on T1-weighted and fluid-attenuated inversion recovery (FLAIR) MRI sequences because of the increased protein concentration of an empyema relative to cerebrospinal fluid. A fluid collection surrounded by a contrast-enhancing rim is often the feature [2].

Differential Diagnosis



The main difficulties, in term of diagnosis, result from differential diagnosis between hematoma, hygroma, and empyema [3].

Treatment

Subdural empyema represents a neurosurgical emergency and if left untreated is invariably fatal. Rapid diagnosis, surgical intervention and intensive antibiotic therapy improve both morbidity and mortality [4].

Treatment consists of surgery to establish bacteriologic identification and subsequently guide antibiotic therapy.

With treatment, including surgical drainage, resolution of the empyema occurs from the dural side, and, if it is complete, a thickened dura may be the only residual finding.

Drainage by craniotomy is associated with better outcome and lower mortality as it ensures maximal drainage of the loculated pus and also allows inspection of adjacent structures, and removal of the bone flap if necessary. However, some reports advocate drainage via burr-holes [5) 6)].

Outcome

They result in significant morbidity and mortality despite improvements in neuroimaging, surgical techniques and antibiotic therapy.

Case series

see Supratentorial subdural empyema case series.

Case reports

see Supratentorial subdural empyema case reports.

Videos

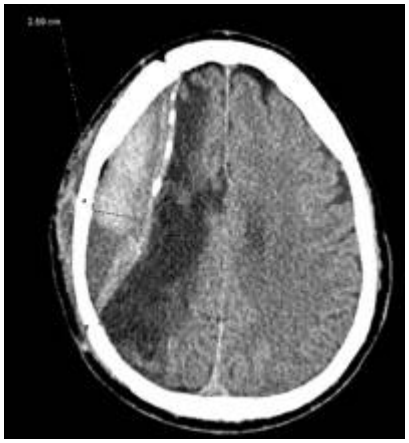
<html><iframe width="560" height="315" src="https://www.youtube.com/embed/-DziqU6wnz8" frameborder="0" allow="accelerometer; autoplay; encrypted-media; gyroscope; picture-in-picture" allowfullscreen></iframe></html>

Case report from the HGUA

A male, 47 years old, with a history of a malignant middle cerebral artery infarction, underwent a decompressive craniectomy exactly one year ago. He subsequently had a cranioplasty

Reports experiencing an unusual headache, swelling with increased temperature in the cranioplasty area, and the discharge of serosanguinous fluid that was not present before.

An urgent non-contrast and contrast-enhanced cranial CT scan is performed.



Compared to the last study there is a presence of an extra-axial collection on the right convexity underlying the cranioplasty. It has a subdural morphology, approximately 2.7 cm in the coronal plane, with a heterogeneous content predominantly hyperdense, likely related to hematic residues, as well as some minimal air bubbles. There is a striking enhancement of the dura mater, and radiological signs suggestive of subdural empyema. This collection causes a mass effect on the underlying cerebral sulci but does not cause midline deviation or clear signs of herniation.

A discrete increase in extracranial soft tissues of about 2 cm thickness in the coronal plane adjacent to the cranioplasty is also identified. It shows heterogeneous contrast enhancement and ill-defined hypodense foci inside, suggesting soft tissue infection with associated myositis and subgaleal collections.

Extensive corticosubcortical hypodensity in the territory of the right middle cerebral artery is identified, indicating an old ischemic infarction. This results in slight retraction of the right occipital and temporal horns, as well as slight hypodensity of the right mesencephalic peduncle related to Wallerian degeneration.

References

1)

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4)

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