

Traumatic Subdural Empyema – A Rare Neurosurgical Emergency: Case Report and Review of Literature

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ABSTRACT

Subdural empyema is a neurosurgical emergency with potentially overwhelming complications. The prognosis is harmfully affected when early diagnosis is not made. Recent advancement in the imaging techniques, especially contrast enhanced CT Scans and MRI Scans, have improved the precision of radiological diagnosis of this condition, along with reduction in the mortality rate. Regardless of this, there may still be an impediment in diagnosis, partly due to the subtleness of early radiological signs, especially on non-contrast CT Scan. We present a case report which illustrates some of the radiological manifestations, complications, potential pitfalls in diagnosis and treatment of traumatic subdural empyema.

Conclusion: *Patient of severe head injury not improving with pharmacotherapy should get a repeat C.T Scan vault to look for delayed hematomas or empyema. Urgent diagnosis and prompt evacuation along with proper anti-biotic therapy can improve the survival of patient having traumatic subdural empyema.*

Keywords: *Subdural empyema, mortality rate, radiological sign, non-contrast CT scan.*

INTRODUCTION

Subdural empyema due to trauma is a relatively uncommon condition.¹ It is a neurosurgical emergency and the outcome may be prejudiced by delay in diagnosis.^{2,3} Pus may collect in the subdural space which may not be possible to localize this collection to either the subdural or the extra dural space.¹ Subdural empyema often occurs in young patients, most commonly secondary to paranasal sinusitis, middle ear sepsis and mastoiditis.¹ The frontal and ethmoidal sinuses are most commonly implicated.^{2,3} Other recognized causes include post-operative infection, as a complication of facial or scalp cellulitis, following bacterial meningitis or as a secondary infection of a sterile chronic subdural hematoma. Trauma, in particular if there are compound fractures of the paranasal sinuses, can also give rise to a subdural empyema. It may be due to complication of dental sepsis⁴ and may also be found in association with skull osteomyelitis. Secondary to sinus disease, pus collects over the convexity of the cerebrum, especially anteriorly, and also in a parafal-

cine area.³ It may collect inferiorly, particularly below the frontal lobes. Pus may track under the falx cerebri, giving rise to contra lateral or bilateral collections. The location of the causative pathology does not always consistently point to the site or even the side of the resultant intracranial collections.¹ Empyema arising as post-operative infections or following trauma occur more predictably at the site of surgery or injury. The underlying mechanism of brain injury in subdural empyema is related to thrombophlebitis and toxin-induced local effects, whereas mass effect may be of lesser importance.^{3,5} In contrast, if pus is restrained by the dura in the form of an extra dural empyema, mass effect is a more important cause.

The complications of subdural empyema are potentially devastating. Thrombosis of cortical veins or a major venous sinus (Figures 1, 2 and 5) may occur as a consequence of subdural empyema and cause cerebral edema and infarction.³ In subdural empyema occlusion of small cortical arteries and spasm of larger vessels at the base of the brain are known angiographic find-

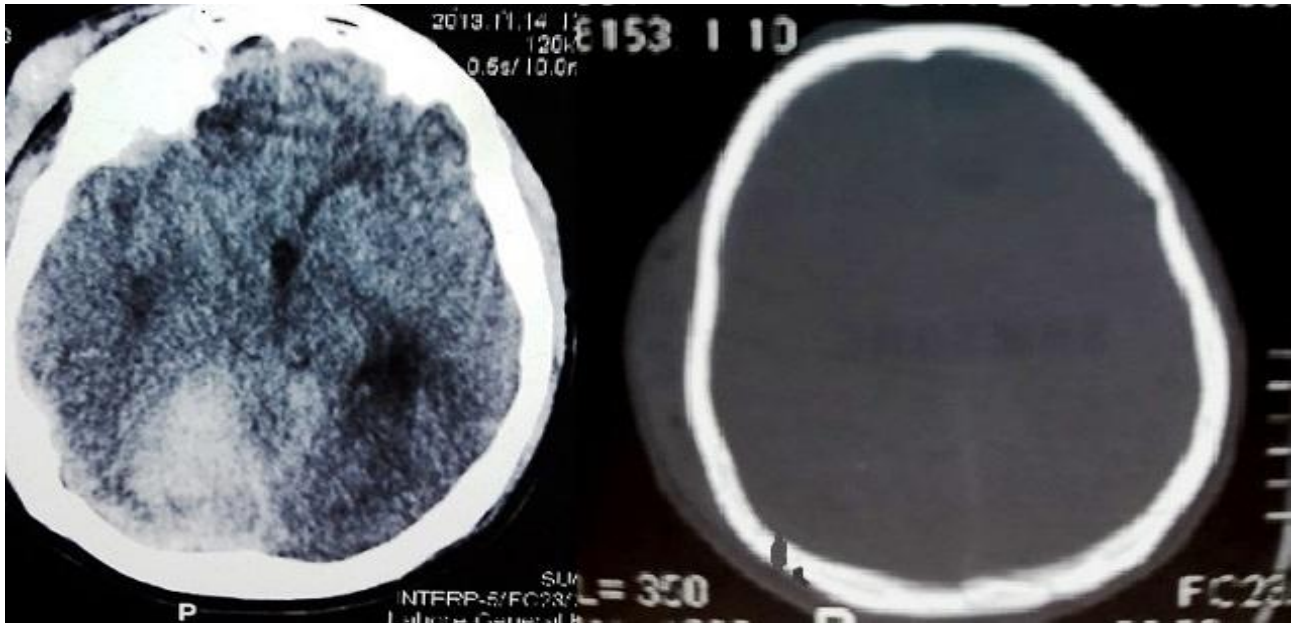


Figure 1: CT Scan Brain Plain showing right occipital fracture and contusion.

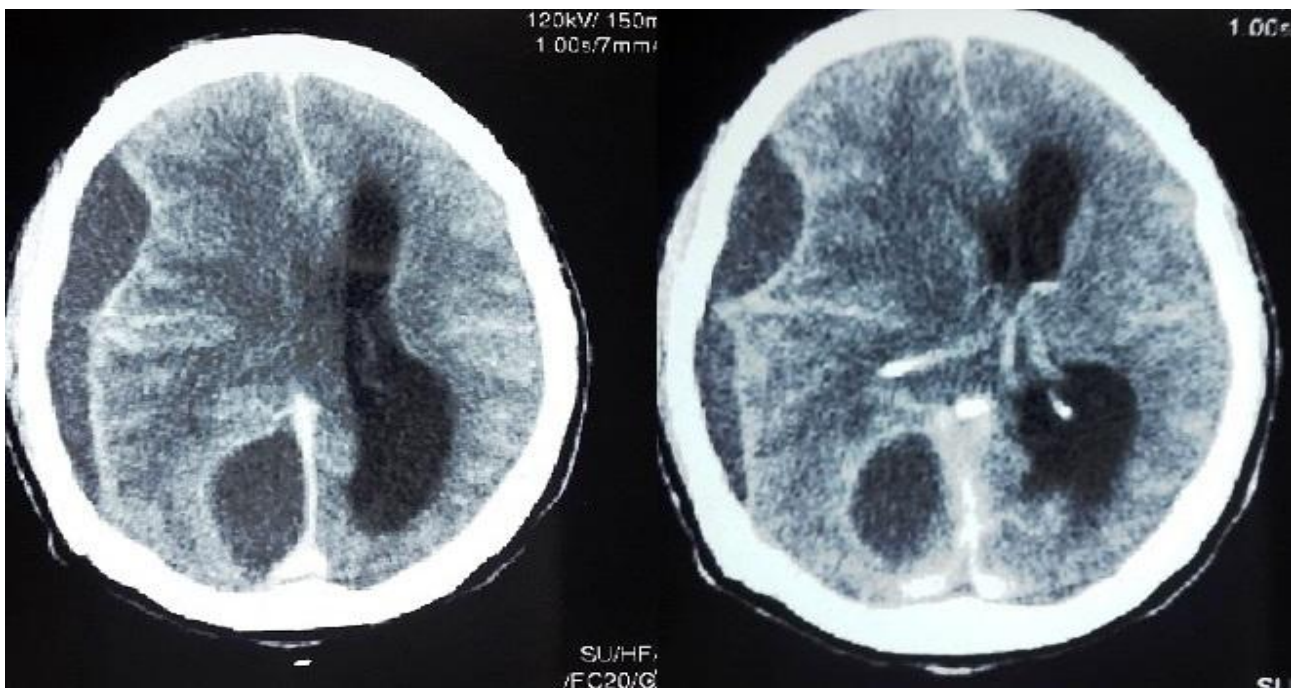


Figure 2: CT scan brain with I.V contrast showing right temporo parietal and parafalcine subdural empyema.

ings.⁶ Another serious lethal complication is cerebral abscess (Figure 3). The classical clinical presentation of subdural empyema is an acute febrile illness with neurological features such as decrease in conscious level, meningism, focal neurological deficits and epileptic fits. There may be a prodrome of paranasal sinusitis or mastoiditis. Subdural empyema related to

the other causes described above may give rise to a more indolent presentation.⁵

CASE REPORT

A 24 years male patient presented to the emergency department with a history of road traffic accident via a

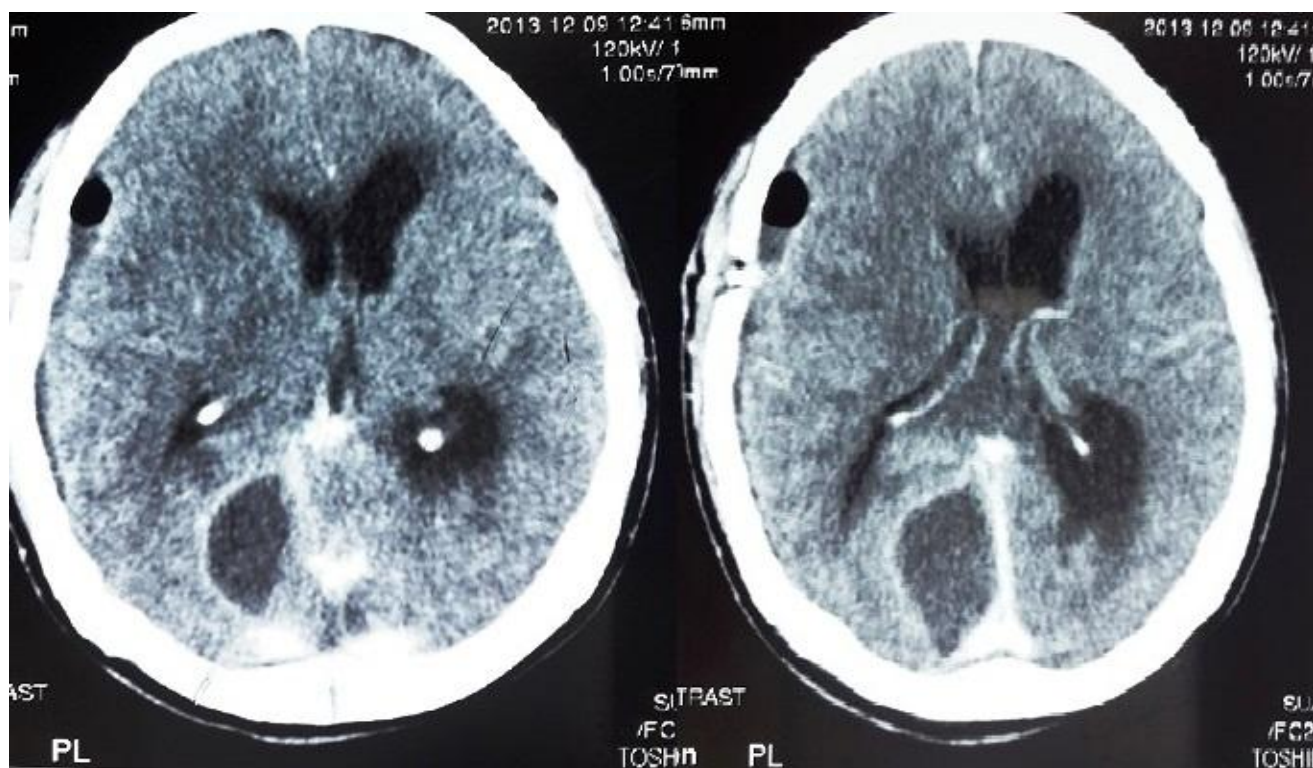


Figure 3: Post Operative initial CT scan showing the parafalcine occipital subdural empyema left.



Figure 4: Post operative CT scan brain and patient status.

motor bike. He had 3 episodes of vomiting, ear bleed and acute loss of consciousness on arrival. On examination he had anisocoria, right pupil mid dilated while

the left pupil was constricted. His Glasgow coma scale was 05/15, while there were no associated injuries. After resuscitation an urgent cranial CT scan was obtained.

ined that showed an occipital linear fracture and right sided occipital brain contusion.

Based on the clinical condition and radiological findings we subject the patient to conservative management. The patient was shifted to the neuro ICU where by the pharmacotherapy along with hyperventilation was initiated. He was ventilated for a period of three days whereby he was weaned off from the ventilatory support and his Glasgow coma scale improved to 08/15 with bilaterally equal pupils and fairly stable vitals. He was shifted to the general ward for proper nursing care and physiotherapy. On the seventh day of admission the patient started to develop high grade fever along with bilateral crepitation on chest auscultation. Consultation with the pulmonologist was done and tracheostomy was offered to the patient under local anesthesia. Based on the culture and sensitivity of the sputum proper antibiotics were administered. With this maneuver the patient remained well for 2 days but then suddenly deteriorated with a high grade fever and at the same time Glasgow coma scale declining to 06/15. His routine hematological investigation revealed an elevated leucocyte count. His check CT Scan brain was ordered and obtained.

After the subdural empyema diagnosis was established, the patient was operated. In the first setting the parietal empyema was evacuated by placing two burr holes, while an attempt was made to evacuate the parafalcine occipital empyema but due to profused bleeding the procedure was abandoned.

On the next operative list the parafalcine occipital subdural empyema was evacuated by a small craniectomy. On gross appearance there was a frank pus. The culture and sensitivity of which turned out to be negative, there was no organisms of growth.

Post operatively there was a remarkable recovery in the patient neurological status, his Glasgow coma scale improved to 11/15 though there was left sided weakness. The patient remained in the hospital after the last surgery for a period of 5 days. He was discharged and physiotherapy was advised.

DISCUSSION

Subdural empyema is a potentially fatal condition if prompt diagnosis is not made and immediate treatment is not offered to the patients. Infection of the paranasal or mastoid sinuses is the commonest associated cause of subdural empyema. In most studies, 60% of patients suffered from underlying sinus disease. The highest mortality (43%) occurred in conjunction with mastoid disease. A more benign form of empyema rarely occurs

following surgery for subdural hematoma.⁶ Spontaneous empyema is very rare, but in some cases it occurs with minor head trauma some days to week after the trauma. It presents with severe headache, rapidly advancing neurological deficit, and epilepsy, combined with evidence of underlying infection. The majority of patients in different studies showed that the patients were drowsy at the time of referral, but physical signs could not be correlated with mortality because of the small number of cases. With increased experience and the introduction of later generation scanners, computed tomography provided a specific diagnosis in all cases. Subdural empyema was almost always fatal before penicillin was available for topical and systemic use. Schiller vividly showed its beneficial effects on survival in 1948.⁷ Chloramphenicol and penicillin remain the primary treatment until specific antibiotic sensitivities are known and treatment is continued for several weeks. Streptococcal infection is still found in a high proportion of fatal cases. Metronidazole readily crosses into intracranial pus and is effective against the anaerobic organisms commonly found in association with mastoid air cell disease.⁸⁻¹⁰ The exhibition of antibiotics prior to culture of the infected material leads to a poor yield of organisms. There is a recent trend towards wide decompressive craniotomy, as an initial maneuver in all cases of subdural empyema. This has been advocated by several authors emphasizing surgical technique^{11,12} rather than aspects which some believed to be of more crucial importance, namely early diagnosis and speedy surgical intervention. Deteriorating patients are likely to receive the lesser procedure of burr hole aspiration.

In Bannister's series those who survived craniotomy had a higher incidence of neurological deficit (29%) at late follow-up than those managed with burr holes. This did not reach statistical significance. There is also a possibility that pre-existing deficits are exacerbated by major neurosurgical procedures which have not yet been shown to improve survival. Epilepsy remains a significant cause of late morbidity. In this series 31% of patients discharged from hospital continued to suffer fits. This compares with 26% reported by Hitchcock and Andreadis,¹³ 37% by Borzone et al¹⁴ and 33% by Cowie and Williams¹⁵ despite the prescription of anticonvulsant therapy.

CONCLUSION

Patient of severe head injury not improving with pharmacotherapy should get a repeat C.T Scan vault to

look for delayed hematomas or empyema. Urgent diagnosis and prompt evacuation along with proper antibiotic therapy can improve the survival of patient having traumatic subdural empyema.

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