Fatal Subdural Empyema Following Pyogenic Meningitis

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Subdural empyema is a rare form of intracranial sepsis associated with high morbidity and mortality. The most frequent cause is extension of paranasal sinuses through emissary veins or of mastoiditis through the mucosa, bone, and dura mater. Development of subdural empyema after pyogenic meningitis is known to be very unusual in adults. We report a rare case of fatal subdural empyema, an unusual complication of pyogenic meningitis. Our bitter experience suggests that subdural empyema should be borne in mind in patient with pyogenic meningitis who exhibit neurological deterioration.

Key Words: Subdural empyema • Pyogenic meningitis.

INTRODUCTION

Subdural empyema is an intracranial infection that can rapidly lead to death. Although acute subdural empyema is imperative surgical emergency to neurosurgeons, the symptoms usually may be very mild initially. The most frequent cause is extension of paranasal sinuses through emissary veins or of mastoiditis through the mucosa, bone, and dura mater. Subdural empyema complicating pyogenic meningitis is quite rare in adults. Although accurate diagnosis and adequate treatment can lead to better results, its diagnosis may be still challenging. These difficulties are mainly due to nonspecific initial symptoms such as headache or fever, and are also due to subtle changes that are often ignored on computed tomography (CT) scans. Here, we report a rare case of fatal subdural empyema, an unusual complication of pyogenic meningitis.

CASE REPORT

A previously healthy 53-year-old male patient was admitted to the neurology ward with a history of febrile sense, headache, nausea, and vomiting. At that time, he had mild fever of 37.2°C. He was alert and his vital signs were stable. There was no history of trauma and any focus of infection including paranasal sinuses. However, he had focal neurological signs of stiff neck. Hematological investigation revealed mild leukocytosis with a total white blood cell (WBC) count of 9,760/μL and mild elevation of erythrocyte sedimentation rate (ESR) to 32 mm/hr. Computed tomography (CT) scan of his brain revealed no evidence of abnormalities (Fig. 1) and the lumbar cerebrospinal fluid (CSF) analysis was performed. Lumbar CSF analysis revealed 2916 WBC cells/mm³ (poly : mono = 75% : 25%) with a glucose level of 151.5 mg/dL. With the diagnosis of pyogenic meningitis, the patient was placed on broad-spectrum antibiotics. CSF cultures showed growth of gram positive Streptococcus pneumoniae. First generation cephalosporin antibiotic, cefazolin, and vancomycin were given according to antibiotics susceptibility test. Despite the aggressive fluid and antibiotics therapy,
the patient did not respond to any treatment and, in fact, his symptoms worsened. Fourteen days after admission, he was still febrile and had developed recurrent seizures. Magnetic resonance imaging (MRI) of the brain showed the collection of left fronto-parietal extracerebral fluid with meningeal enhancement (Fig. 2). He was transferred to neurosurgical department with a diagnosis of subdural empyema. His body temperature was 39.1°C, WBC count steadily rose to 23.700/µL, and ESR had climbed to 102 mm/hr by the time of referral to the neurosurgical service. A fronto-temporal craniotomy was performed promptly for evacuation of the subdural empyema. A yellowish purulent material was identified and large amount of pus was drained. Otherwise, the operation was performed as planned without any problem (Fig. 3, 4). Streptococcus pneumoniae was also isolated from subdural pus. Three days after the operation, the patient's level of consciousness deteriorated gradually to a semicoma state. A brain CT scan revealed intracerebral hemorrhage and severe brain swelling with midline shift (Fig. 5). Emergent decompressive craniectomy was performed, the result of which was further aggravation of swelling. His condition deteriorated rapidly, culminating in cardiorespiratory arrest with dilated non-reactive pupils, and died shortly after surgery (Fig. 6).

**DISCUSSION**

Subdural empyema is a diagnosis that constitutes a neurosurgical emergency. It is one of the most imperative in neurosurgical
field due to its high mortality and this fatal result can be preventable if diagnosis is made at an early phase of the disease and appropriate management is implemented\(^9\). Unless diagnosed early, the condition can propagate rapidly, leading to neurologic deterioration. It evolves from the spread from any adjacent focus. Among them, parasanal sinusitis accounts for the majority of cases, and the spread of infection is centripetal via mucosal, emissary, and bridging cortical veins\(^{2,3,10}\). The pathogenesis of subdural empyema in adults differs from that observed in younger children and infants. In the infants, subdural empyema occurs as a concomitant or complicating feature of meningitis, whereas in adults it is mostly due to spread from a contiguous focus such as parasanal sinusitis or mastoiditis and about 15% is cryptogenic\(^{12}\). The subdural space lacks septations, except where the arachnoid membranes embed into the dura mater. This anatomical characteristics allow for the possible spread of inflammatory processes within the subdural space\(^9\). The adhesions from this inflammatory process may impede the spread of infection and loculate pus, which can result in subdural abscess formation. With the onset of neurological symptoms and signs, however, the picture should be clearer in diagnosing intracranial sepsis, although it would not be possible on clinical grounds alone to differentiate subdural empyema from other types of intracranial infections such as brain abscess, meningitis, cerebritis, or viral encephalitis\(^{13,14}\). Subdural empyema has a tendency to spread rapidly through the subdural space until blocked by specific boundaries such as falx cerebri or tentorium cerebelli. With progression, it has a inclination to behave like an expanding mass lesion with associated increased intracranial pressure (ICP). Severe brain swelling may be present as a result of disruption of blood flow or cerebrospinal fluid flow by increased ICP. Moreover, cerebral infarction or hemorrhagic infarction may be present from thrombosis of the cortical veins or cavernous sinuses or from septic venous thrombosis of contiguous veins near subdural empyema. Under these conditions, the possibility of subdural empyema should be kept in mind, prompting immediate neuroimaging including CT or MRI. CT scans show a classic (extra-axial) hypodensity area compared to brain parenchyma, almost isodense compared to CSF. Contrast enhanced CT increases conspicuity of the collections and leads to finer delineation\(^{15,16}\). Subtle initial CT scan changes and false-negative CT scans are well documented in cases of subdural empyema and are potential pitfalls for the treating neurosurgeon. Reasons for missing the lesion based on CT scan results also include lack of experience and unfamiliarity with this condition, leading to misinterpretation of the findings. Interval follow-up imaging is necessary in those cases where subdural might not appear in the initial studies seen in our case. MRI provides much better sensitivity and specificity as compared to CT scans. In addition, MRI also provides better visualization of, the precise localization, and the differentiation of subdural empyema from other noninfected subdural effusions or hygromas\(^9\).

**CONCLUSION**

Our bitter experience suggests that subdural empyema should be borne in mind in patient with pyogenic meningitis who exhibit focal neurologic deficits or deteriorate neurologically.

**References**