An unusual presentation of neurotuberculosis: subdural empyema

Case report

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Tuberculosis continues to be a major public health concern, especially in developing countries. Many types of neurotuberculosis have been described, but there is only one previously reported case of subdural empyema caused by tuberculous bacilli. A 1-year-old boy who had been treated for pulmonary tuberculosis was referred to the authors’ institution with a diagnosis of right frontoparietal extraxial abscess formation. Computerized tomography and magnetic resonance imaging revealed an extraxial abscess with no evidence of calvarial infection. A craniotomy was performed to drain the pus, which was located subdurally. A polymerase chain reaction test yielded positive results, and histopathological examination revealed caseation. Antituberculous treatment was started after a diagnosis of subdural empyema with related neurotuberculosis had been made. At the end of a 12-month course of medical therapy, the patient was well with no evidence of tuberculosis.

KEY WORDS • empyema • neurotuberculosis • subdural space

Subdural empyema is a rare intracranial infection that usually arises as a complication of meningitis in infants and young children and as a complication of paranasal sinusitis, otitis media, or mastoiditis in older children and adults. This infection progresses to death if left untreated. The organisms responsible for subdural empyema depend on the origin of the infection. Aerobic and anaerobic streptococci, Haemophilus influenzae, Staphylococcus aureus, and S. epidermidis are the most commonly identified organisms.

Tuberculosis, which continues to be a major public health concern in many parts of the world, may involve the central nervous system either as meningitis or as parenchymal granulomas. Subdural empyema as a form of neurotuberculosis is an extremely rare condition. To our knowledge only one previous case has been documented. In that case, the tuberculous infection started as an osteitis of the cranium and spread to the subdural space in a retrograde fashion through diploic veins. The present paper is a unique report on a patient whose subdural empyema was demonstrated on MR imaging. In our case, there was no evidence of cranial involvement, and the infection resulted from direct hematogenous dissemination of tuberculous bacilli from the primary lesion in the thorax.

Case Report

History and Initial Treatment. This 1-year-old boy was admitted to another institution with primary symptoms of fever, difficulty breathing, and productive cough of 2 months’ duration. He was found to be infected with Mycobacterium tuberculosis, and antituberculous treatment was started after pulmonary tuberculosis was diagnosed. Approximately 2 months thereafter, the patient was admitted to the same center with complaints of nausea, vomiting, and lethargy. A CT scan revealed a hypodense extraxial fluid collection in the right frontoparietal region that mimicked an epidural abscess because a biconvex shape was seen. Empyema puncture was performed to reduce the volume of pus before the patient was referred to our hospital.

Examination. On admission, neurological examination revealed that the patient had a Glasgow Coma Scale score of 15, no focal deficits, and no clinical signs of meningism. Results of routine laboratory studies were normal, but the peripheral white blood cell count was 15,300/mm³ with 70% lymphocytes. The erythrocyte sedimentation...
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rate was 94 mm/hour. Results of testing for human immuno-deficiency virus antibody were negative. Gadolinium-enhanced MR images with T1-weighted sequences demonstrated a hyperintense extraaxial fluid collection with contrast enhancement of internal and external capsules in the coronal view (Fig. 1). Axial T1-weighted MR images additionally revealed a left frontal hypointense subdural fluid collection and nodular contrast enhancement adjacent to the subdural effusion in the subarachnoid space (Fig. 2).

Operation. A right frontoparietal craniotomy was performed, during which the cranium and dura overlying the lesion were found to be intact. When the dura was opened, a thick, rough, yellow capsule containing abscess fluid was exposed; the external capsule was totally excised. A yellowish purulent material with a total volume of 60 ml was drained and some necrotic material bearing a resemblance to cauliflower was seen in the cavity. We did not attempt to excise the inner capsule, to avoid the risk of cortical infarction.

Adjunctive Therapy and Histopathological Examination. Immediately after the operation, an empirical, high-dose antibiotic regimen consisting of penicillin (100,000 U/kg/6 hrs), chloramphenicol (15 mg/kg/6 hrs), and metronidazole (7.5 mg/kg/8 hrs) was initiated. Results of a polymerase chain reaction test applied to the material obtained from the empyema were positive, but no organisms could be cultured. Microscopic studies showed fibrotic tissue with abundant caseation on one surface. Next to this necrosis, groups of epithelioid macrophages with a granuloma-like organization and granulation tissue containing mixed inflammatory cell infiltrate were seen (Fig. 3). The positive polymerase chain reaction test, pathological findings, and history of pulmonary tuberculosis indicated that tuberculous bacilli were likely to be the causative organisms for the subdural empyema. The antibiotic regimen was stopped, and orally administered antituberculous therapy including streptomycin (200 mg/day), rifampicin (100 mg/day), and isoniazid (100 mg/day) was initiated.
Postoperative Course. The postoperative course was uneventful. The patient was discharged home 6 weeks after the operation and underwent follow-up review monthly at the hospital with CT scans and routine laboratory studies. At the end of the course of therapy, 12 months after the surgical intervention, no subdural collection or tuberculosis was seen on MR imaging (Fig. 4).

Discussion

Neurotuberculosis remains a major cause of neurological morbidity and mortality and is the most dangerous type of systemic tuberculosis. Many types of neurotuberculosis have been described, of which tuberculous meningitis and parenchymal granulomas are the most commonly seen. Hematogenous spread of M. tuberculosis from primary lesions is the main cause of neurotuberculosis. Rupture of a subpial or subependymal granuloma into the subarachnoid space or hematogenous spread of the bacilli directly to the leptomeninges from any source may be the cause of tuberculous meningitis. Bacilli may enter the subarachnoid space in regions with no blood–brain barrier, such as the choroid plexus. Granulomatous inflammatory reaction involves the meninges. Besides diffuse meningitis, in most patients the brain is also directly involved, with tuberculous infection seen in the form of granulomas or, less frequently, tuberculous abscesses or focal tuberculous meningitis.

Other forms of tuberculosis, such as formation of epidural abscess or subdural empyema, are extremely rare; there are only two reported cases of extradural abscesses caused by M. tuberculosis. One of them occurred as a result of an intrasellar tuberculous abscess, presumably from hematogenous spread. The other arose from an untreated pulmonary infection caused by M. tuberculosis. Hematogenous dissemination from the primary lesion caused cranial osteomyelitis and epidural abscess in both cases.

There is only one previously reported case of subdural empyema caused by tuberculosis. This patient had a 5-year history of treatment for pulmonary tuberculosis. Rim calcification, cranial osteitis, and scalp swelling overlying the affected zone were the CT findings in this case. It was thought that subdural empyema started as an osteitis of the cranium and spread to the subdural space in a retrograde fashion through diploic veins. Hematogenous dissemination from a primary lesion, usually a pulmonary lesion, is responsible for this condition.

In our case there was no clinical or radiological evidence of cranial involvement. Surgical exposure revealed that the cranium and underlying dura mater were intact. It is known that tuberculous bacilli can involve the cerebral parenchyma with tuberculous granuloma formation and that granulomas are most commonly located at the corticomedullary junctions and in periventricular regions. From this point of view, the possible mechanism in our case might be a hematogenous dissemination of tuberculous bacilli from the lung to the subdural space by an indirect route. Perhaps a small subpial tuberculous granuloma occurred by hematogenous spread of pulmonary tuberculosis, and rupture of this granuloma to the subarachnoid space formed a small tuberculous granuloma. Arachnoid membrane adjacent to the granuloma was affected by the microorganism, as shown in Fig. 2, which reveals contrast enhancement. Rupture of the affected arachnoid, which would allow the microorganism to pass to the subdural space, is a possible mechanism of formation of the subdural empyema. This mechanism is implicated by the contralateral subdural effusion, granuloma, and arachnoid enhancement.

A thick, gelatinous exudate-forming pus rapidly accumulates and may remain localized or spread throughout the subdural space. Although the arachnoid acts as a barrier to the deeper spread of infection, unrestricted access in the supratentorial subdural space allows a thin layer of purulent material to be deposited diffusely over the cerebral convexity. Whatever the mechanism, a granulomatous inflammatory reaction occurred only between the dura mater and arachnoid. The dura mater and arachnoid restricted the dissemination of the bacilli through the subarachnoid space, which could have caused meningitis, epidural abscess, or cranial osteitis.

Neuroimaging has dramatically contributed to early diagnosis and management of subdural empyema. Magnetic resonance imaging has been shown to be superior to CT scanning in the detection of subdural empyemas, and it can also be used to differentiate sterile, bloody, and infected subdural collections more clearly. Parenchymal edema, ischemia, and parenchymal spread of infection are also more clearly demonstrated on MR imaging, because this modality is more sensitive than CT scanning in the detection of edematous tissue. Our case is the first reported one of a tuberculous subdural empyema identified on MR imaging. The sensitivity of this modality in this case also helped to reveal the possible mechanism of formation of subdural empyema in which there was no evidence of meningitis or osteitis of the overlying cranium.

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