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Case Report

Spinal subdural tuberculous abscess

M Özateş*[,1], Ü Özkan[2], S Kemaloglu[2], S Hoşoglu[3] and I Sari[4]

[1]*Department of Radiology, Faculty of Medicine, Dicle University, Diyarbakir, Turkey;* [2]*Department of Neurosurgery, Faculty of Medicine, Dicle University, Diyarbakir, Turkey;* [3]*Department of Infectious Diseases, Faculty of Medicine, Dicle University, Diyarbakir, Turkey;* [4]*Department of Pathology, Faculty of Medicine, Dicle University, Diyarbakir, Turkey*

Objectives: Spinal subdural abscess is rare and only 48 cases have been described to date. In this report, we present an additional spinal subdural tuberculous abscess.
Method: Tuberculous meningitis was diagnosed with clinical and laboratory findings in a 45-year-old man. A spinal subdural abscess was demonstrated using MRI. Presence of the abscess was revealed by surgical intervention. The diagnosis was confirmed by pathological examination.
Results: The patient had been treated for tuberculous meningitis 2 years previously. The disease recurred when anti-tuberculous therapy was prematurely discontinued. During the second treatment, the patient also underwent a ventriculo-peritoneal shunt operation for hydrocephalus. Dizziness and weakness of both legs developed after the postoperative period. Spinal MRI showed a spinal subdural abscess as a iso-intense mass with spinal cord in the T1 and T2 weighted images, ring like enhancement and compression on the spinal cord at T3-T4 level. The patient underwent surgery and the abscess was drained.
Conclusion: Tuberculosis may cause a spinal subdural abscess and although it is a rare disorder, when encountered MRI is very useful in the diagnosis.
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Introduction

Spinal subdural abscesses are uncommon. The first spinal subdural empyema was reported by Sitting in 1927.[1] Up to now, only 48 cases have been published. The commonest agent is *Staphylococcus aureus*, our case was a tuberculous abscess. *Mycobacterium tuberculosis* as the causative organism in spinal subdural abscesses has been described but is very rare, but many clinical manifestations, including meningitis due to *Mycobacterium tuberculosis* are not rare in our region.[2]

We present this case clinically, radiographically and ultimately at operation. Patients presenting with neurological deterioration associated with subdural empyemas are rarely identified.

Case report

A 45-year-old man suffering from headache, nausea and vomiting was admitted to our hospital 2 years earlier and found to have tuberculous meningitis. The diagnosis was established by clinical and cerebrospinal fluid (CSF) examination. A miliary pattern was observed on chest radiography. There was a history of close contact with a patient with chest tuberculosis. The CSF and sputum cultures for tuberculosis were negative on admission. Cranial MRI showed basal cisternal enhancement, there was no hydrocephalus. The patient was treated with rifampicin (600 mg/day), isoniasid (500 mg/day), streptomycin (1 g/daily) and pyrazinamide (1500 mg/daily), and prednisolone (60 mg/day) at the hospital. Remission was established in 2 months and the patient was discharged.

One year later he was readmitted with the same complaints because he had discontinued anti-tuberculous therapy prematurely. The same treatment was repeated at the hospital. The CSF and sputum cultures were repeated and found to be negative. MRI also showed basal cisternal enhancement and hydrocephalus. He underwent ventriculo-peritoneal shunt operation for the hydrocephalus on the 40th day of this second treatment period.

One month later, urinary incontinence, lower extremity weakness especially on the right and loss

*Correspondence: M Özateş, Dicle Üniversitesi Tip Fakültesi, Radyodiagnostik Anabilim Dali, 21280, Diyarbakir, Turkey

of equilibrium when he closed his eyes (Romberg's sign) developed. He was fully conscious and cooperative. Neurological examination revealed spastic paraparesis, increased deep tendon reflexes in the lower limbs, hypoesthesia below the T4 level and a positive Romberg. MRI showed a spinal subdural abscess at T3-T4 level as an iso-intense mass on T1 and T2WI which showed compression of the spinal cord posteriorly and ring like enhancement after Gd-DTPA administration (Figure 1a,b).

The patient underwent surgery and the abscess was drained on the 78th day. The diagnosis was confirmed by surgery and pathologic examination (Figure 2). The culture of the surgical specimen was negative for tuberculosis. The patient's symptoms resolved progressively 1 month after surgery. He was discharged with significant clinical improvement on the 117th day after hospitalization. He has regained his ability to perform normal daily activities. He was followed-up at 1, 3, 6 and 12 months and was normal.

Discussion

The exact incidence of spinal subdural abscess is unknown. Including our patient, only 48 cases have been reported. The most commonly affected region is the thoraco-lumbar spine. Fifty per cent of the patients were more than 50 years old. It occurred more frequently in men in the fifth and sixth decade of life.

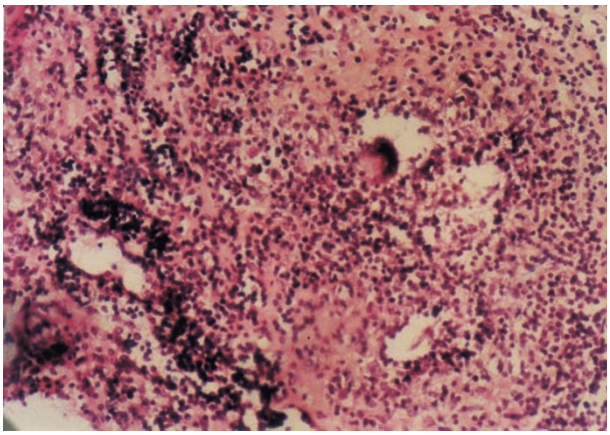

Figure 2 Langhans type giant cells, leucocytes, fibroblasts and epitheloid histiocytes with caseous micronecrosis in their centers

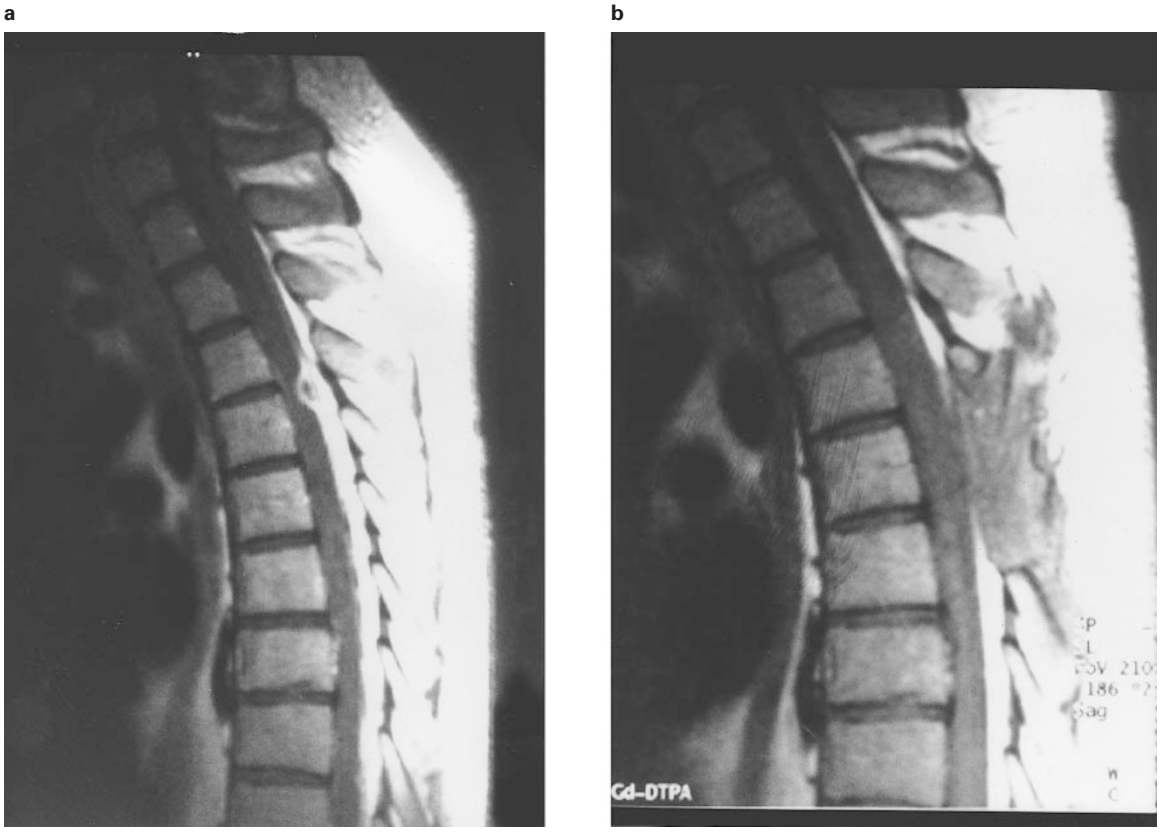

Figure 1 (a) Preoperative T1 weighted magnetic resonance image (contrast-enhanced), sagittal view, shows a subdural mass lesion 10×14 mm diameter posterior to the spinal cord at T4 level, and shows ring like enhancement after Gd-DTPA administration and compresses the spinal cord posteriorly. (b) When compared to former images, no abscess or arachnoid thickening is seen at the T4 level after operation

Women were more affected most often in seventh decade.[1,3]

A spinal subdural empyema may develop primarily by hematogenous spread secondarily by iatrogenic contamination and locally by spread from an adjacent abscess. The cause of neurodeficits with spinal empyema is thromboembolic and/or due to compressive ischemia. It has been shown that spinal cord inflammation can also uncommonly be a reason.[4] *Staphylococcus aureus* is the most common causative organism, but other organisms may be involved. Lesions secondary to tuberculosis are uncommon.[1,3]

Spinal subdural empyema usually presents with fever, spinal or root pain, and a neurological deficit, depending on the spinal cord compression. Analogous to the stages of progression of symptoms proposed by Heusner[5] for spinal epidural abscess, symptoms of spinal subdural abscess appear to occur in a certain sequence; Stage 1: fever, either in combination with or without spinal and/or root pain; Stage 2: motor deficit, sensory loss, and/or sphincter disturbances; and Stage 3: paralysis and complete sensory loss below the level of the lesion. In our case, symptoms developed in the order outlined above. The rate of progression from one stage to another is not predictable.[1]

Differential diagnosis of spinal empyema, without specific laboratory findings includes acute transverse myelitis, vertebral osteomyelitis, epidural hematoma, epidural abscess, subdural abscess and intraspinal tumor.[1,3]

The CT finding of extraaxial empyemas have been well described in previous reports.[6] Early in the course of the disease, CT findings can be subtle and may easily be overlooked. Many studies report that MRI is more definitive in the early demonstration of the abscess in spinal cord.[6–8] Frequently, loculations of pus cannot be appreciated on CT scanning. MRI is superior to CT in evaluating granulomatos lesions and other soft tissues lesions in spinal cord. It is more sensitive in detection, more accurate in localization, and more complete in delineation of the process.

It is reported that, on the basis of signal intensity differences, the MRI can differentiate extraaxial empyemas from most sterile effusions and most chronic hematomas.[6–8] Increased signal intensity on the intervertebral disk can be helpful in diagnosing disk-space infection on MR scans. MRI was more sensitive in detecting disk-space infection than CT. It was sensitive as radionuclide studies and more specific.[9]

In subdural abscess, contrast-enhanced images clearly depicted thickening of the neighboring dura mater and a co-existent abscess. In epidural empyemas, part of the displaced dura mater did not enhance, which facilitated differentiation from subdural empyemas. Contrast-enhanced MRI was thus of value in diagnosis.[10] There is greater specificity of MR as compared with CT in differentiating a subdural from an epidural empyema and from a parenchymal abscess. A hypointense medial rim, representing inflamed dura, is seen in an epidural but not a subdural empyema, as in our case.[6] Spinal tuberculosis may also be seen as an arachnoiditis.[11]

A specific MRI signal characteristic allows the detection of the lesion level as well as its extent, thus replacing the need for both myelography and CT myelography.[3] Magnetic resonance imaging is a noninvasive superior imaging modality for demonstrating spinal infections, and has a high sensitivity for detecting lesions in the bone, disk space, subdural space, epidural space, or adjacent soft tissue. The degree of cord compression is better seen on MR imaging than myelography. Furthermore, the entire extent of the extraaxial lesions, as well as the presence of paraspinal lesions, can be demonstrated. The presence or absence of discitis is readily demonstrated on MR imaging and, with gadolinium enhancement and a fat suppression technique, it has been proven to be a superior technique for evaluation of spinal infection.[3]

After MRI examination, the diagnosis of the subdural abscess and exudative collection located in the upper thoracal level was made. A definitive diagnosis must be made at the time of surgery or autopsy.

Conclusion

Although rare, tuberculosis may cause a spinal subdural abscess. MRI scanning is useful in establishing a diagnosis.

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