Clinical Case Report

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Challenges in diagnosis of spinal epidural abscess

A case report

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Abstract

Rationale: Spinal epidural abscess (SEA) is a rare condition that shows a high prevalence in immunocompromised patients. The clinical presentation of SEA includes the "classic triad" of pain, fever, and neurological dysfunction. However, these nonspecific features can lead to a high rate of misdiagnosis. SEA may lead to paralysis or even death; thus, prognosis of these patients remains unfavorable.

Patient concerns: We report a case of a multilevel (T6–T12) SEA in a 22-year-old woman.

Diagnosis: The patient was initially diagnosed with spinal tuberculosis at a local hospital based on a history of tuberculosis exposure, as well as radiography and computed tomography. Histopathological examination of the tissue resected during laminectomy confirmed the diagnosis of SEA in this patient.

Interventions: The patient underwent multilevel laminectomy combined with long-term antibiotic therapy.

Outcomes: Physical examination performed 16 months postoperatively revealed that superficial and deep sensation was restored to normal levels in the lower extremities with improvement in the patient's motor function (muscle strength 2/5).

Lessons: This case report indicates that whole spine magnetic resonance imaging is warranted in patients with SEA and that prompt surgical intervention is important at symptom onset. Long-term antibiotic therapy is also essential postoperatively.

Abbreviations: CRP = c-reactive protein, CT = computed tomography; ED = emergency department; ESR = erythrocyte sedimentation rate; MRI = magnetic resonance imaging; SEA = Spinal epidural abscess; T1WI = T1-weighted imaging; T2WI = T2-weighted imaging.

Keywords: antibiotic therapy, laminectomy, magnetic resonance imaging, misdiagnosis, spinal epidural abscess, spinal tuberculosis

1. Introduction

A spinal epidural abscess (SEA) is a suppurative infection of the epidural space, which can cause injury to the spine by direct compression[1] or secondary to local ischemia.[2] Risk factors for SEA include intravenous drug abuse, diabetes mellitus, old age, and renal disease.[3] However, no definitive etiology can be identified in 20% of patients.[4] SEA is a rare condition. The incidence rate is approximately 0.51/1000 among admissions.[5] The clinical presentation of SEA includes the "classic triad" of pain, fever, and neurological dysfunction; however, these features are nonspecific and could lead to a high rate of misdiagnosis.[6] Moreover, 15% of patients with SEA show a poor prognosis (rate of development of paralysis = 8% and mortality rate = 7%).[5] Therefore accurate diagnosis and prompt treatment are important to treat patients with SEA.

Conventionally, SEA is diagnosed based on clinical symptoms, laboratory tests, imaging studies, and invasive diagnostic tests. Among these modalities, gadolinium-enhanced magnetic resonance imaging (MRI) shows 90% sensitivity and specificity and is the gold standard to diagnose SEA.[7] Computed tomography (CT) is rarely performed because of its lower sensitivity, particularly in patients with early-stage SEA. CT is performed only if MRI is unavailable.[8] Furthermore, other methods can be used to improve the diagnostic rate. Unfortunately, the misdiagnosis rate of SEA remains significantly high (≥50%).[9] Previous studies have reported several treatment options including antibiotic therapy and surgical intervention. However, the optimal treatment for SEA remains unclear.

In this case report, we describe a 22-year-old woman with SEA who was misdiagnosed at a local hospital as having spinal tuberculosis. She underwent emergency surgical intervention and a 4-week course of antibiotic therapy, which led to satisfactory results.

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XY, RG and XL contributed equally to this work and should be considered co-first authors.

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The study protocol was approved by the Ethical Institutional Review Board of the First Affiliated Hospital of Nanchang University, and written informed consent was obtained from all study participants.

There are no conflicts of interest to declare.

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2. Case report

A 22-year-old woman presented to our emergency department (ED) on March 8, 2015 with paraplegia. She reported that she had initially developed severe backache 8 days prior to presentation, for which she visited a local hospital. She had been the caregiver for her father who was diagnosed with tuberculosis. This history raised the suspicion of spinal tuberculosis, and chest radiography was performed at the previous hospital. Although the chest radiography was normal, she was diagnosed with spinal tuberculosis and was admitted to the hospital and received antituberculosis treatment. However, her symptoms showed aggravation, and she developed a mild fever and weakness of her bilateral lower extremities, which progressed overnight to paraplegia. She was transferred to the ED at our hospital for further treatment.

The patient's vital signs were within normal limits except a temperature of 37.4 °C. Physical examination in the ED revealed the absence of superficial abdominal reflexes and absence of superficial and deep sensation below the plane of the rib arch bilaterally. Gross examination showed that the muscle strength of the lower extremities was 0/5. Examination of both upper extremities revealed findings within normal limits. Additionally, emergency whole spine MRI revealed a fluid collection occupying the epidural space between the T6 and T12 vertebral levels (Fig. 1). Moreover, hyperintense spinal cord signals were observed between the T5 and T7 vertebral levels on T2-weighted imaging (T2WI), which were attributed to compression-induced edema and denaturation of the spinal cord.

Blood tests showed polymorphonuclear leukocytosis (13.45×10^9 cells/L, range 3.69–9.16 cells/L), an erythrocyte sedimentation rate (ESR) of 25.6 mm/h (range 0–20 mm/hour [h]), serum C-reactive protein (CRP) of 15.3 mg/L (range 0–8 mg/L), and mild hypocalcemia at 2.13 mmol/L (range 2.15–2.57 mmol/L). Her serum electrolyte levels were within normal limits. Electrocardiography and chest radiography were normal.

Based on the clinical features and imaging studies, the patient underwent an emergency T6–T12 laminectomy. After exposure of the lamina, gray-yellow tissue was visible on the left side of the spinal canal. This tissue was completely removed using micro-scissors, and histopathological examination of this specimen revealed an epidural abscess (Fig. 2). The operation time was approximately 3 hours, and the procedure was completed without any complications.

She received empirical antibiotic therapy with intravenous ceftriaxone (3rd generation cephalosporin, 2 g once a day) and vancomycin (500 mg 4 times a day) perioperatively. Culture of the purulent fluid grew *Staphylococcus aureus* sensitive to sulfamethoxazole, ceftriaxone, vancomycin, levofloxacin, and gentamicin, but resistant to clindamycin and erythromycin. The administration of ceftriaxone and vancomycin was continued at the same dosage for 4 weeks after confirmatory drug sensitivity testing.

Her back pain was relieved on the first postoperative day; however, her body temperature was raised to 39.5 °C, which gradually returned to normal 3 days later. Her serum CRP level was 5.92 mg/L, and the leukocyte count was 6.65×10^9 cells/L,

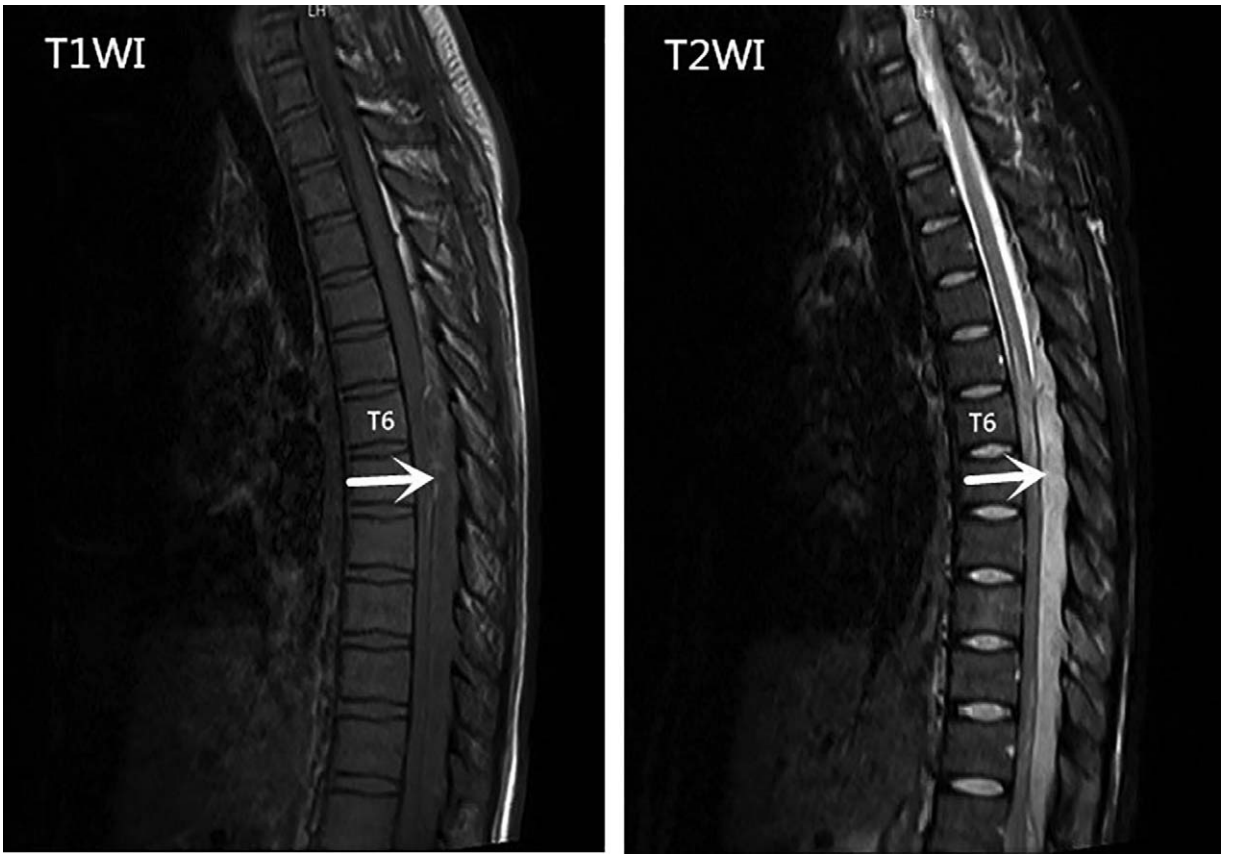

Figure 1. Preoperative magnetic resonance imaging (MRI) scan of the thoracic spine obtained in a 22-year-old woman shows a fluid collection between the 6th and 12th thoracic vertebral levels on sagittal T1-weighted and T2-weighted images.

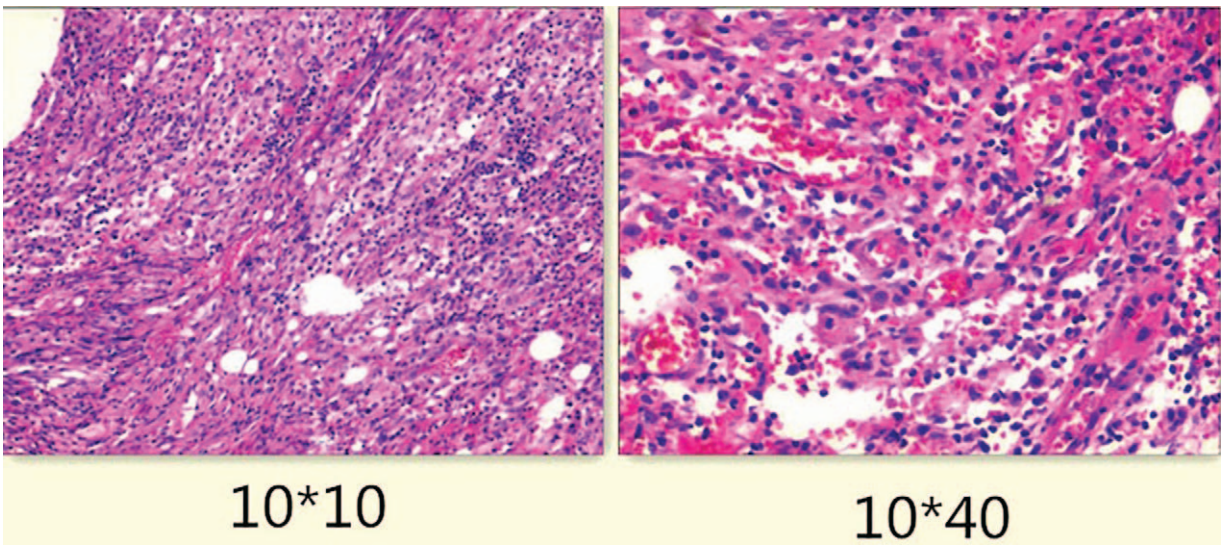

Figure 2. Image shows histopathological findings of an abscess with granulation tissue, inflammatory exudate, blood clot, and fibrous adipose tissue in addition to an infiltrate comprising abundant acute and chronic inflammatory cells.

and she showed an uneventful recovery. Spinal MRI (1 month postoperatively) showed disappearance of the purulent debris (Fig. 3). However, her sensory and motor function did not recover completely (muscle strength 1/5).

The patient returned for rehabilitation therapy 16 months after the neurosurgical operation (Fig. 4). Compared with the previous findings, her physical examination revealed restoration of sensory function (superficial and deep sensations), as well as improved motor function (muscle strength 2/5) in her lower extremities.

3. Discussion

SEA is a rare infectious condition that is diagnosed in 1/10,000 admissions to hospital.[10] However, its incidence rate has been increasing to 5.1 persons/1000 patients[5] secondary to the increasing number of risk factors associated with this condition including drug abuse, diabetes mellitus, renal disease, and an immunocompromised status. Typically, SEA presents clinically with back or neck pain, fever, and neurological dysfunction. Back pain is the most common feature observed in nearly all patients,

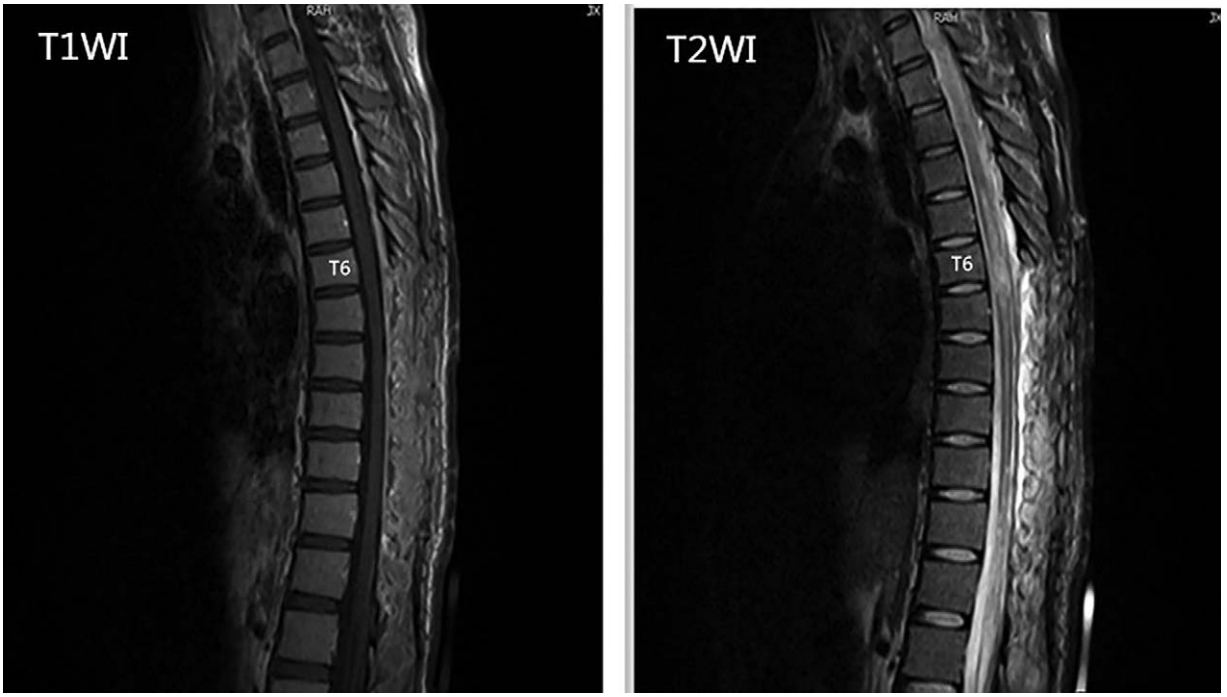

Figure 3. A repeat magnetic resonance imaging (MRI) scan obtained a month postoperatively shows that compared with the previous scan, the abscess in the spinal canal has disappeared but denaturation is observed at the T5–T7 levels of the spinal cord.

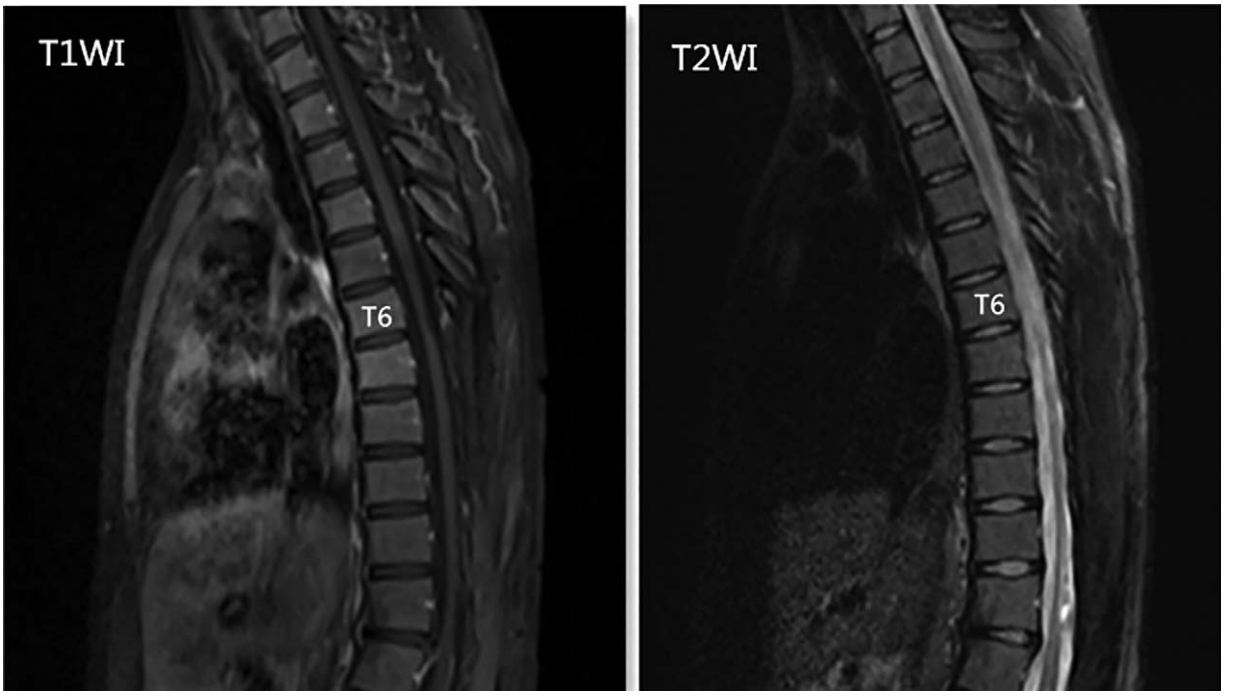

Figure 4. Image obtained 16 months postoperatively shows that the abnormal signal of the thoracic marrow has disappeared.

whereas neurological dysfunction occurs in >50% of patients, and few patients present with fever.[1,11] Neurological dysfunction can be severe and may even include paralysis.

S aureus is the most common causative organism isolated from cultures in two-thirds of all cases of SEA,[12] although other organisms including the *Brucella* and *Streptococcus* species have also been implicated as causative agents.[13–15] Pathogens can invade the epidural space through hematogenous seeding, direct spread from contiguous structures, or via direct inoculation from trauma or invasive procedures.[16] Levofloxacin-sensitive and clindamycin-negative *Staphylococcus* was cultured in the specimen obtained from our patient.

The rate of misdiagnosis of SEA is ≥50% despite advances in medical imaging technology.[9] Spontaneous SEA is rare with nonspecific clinical symptoms leading to delayed diagnosis and treatment. A combination of various modalities including clinical symptoms (thorough history taking), laboratory tests, and imaging studies are important to improve the diagnostic rate of SEA. Among these modalities, gadolinium-enhanced MRI shows 90% sensitivity and specificity and is the gold standard to diagnose SEA[7] Hypointense spinal cord signals on T1-weighted imaging and hyperintense signals on T2WI characterize SEA.[17] However, diagnosis of multifocal SEA can be challenging and may easily be missed. Therefore, whole spine imaging is important.[18] Radiography and CT can accurately diagnose bone lesions, which can serve as indicators of other conditions, such as spinal fractures and spinal tuberculosis. Sendi et al reported leukocytosis in 60% to 80% and ESR >20 mm/h in >95% of cases of SEA,[7] which may be useful diagnostic indicators of SEA. Our patient presented with fever, severe back pain, and paraplegia, which led to a high index of clinical suspicion for spinal cord injury. Emergency whole spine MRI showed a fluid collection occupying the epidural space between the T6 and T12 vertebral levels and hyperintense signals between the T5 and T7 vertebral levels on T2WI (Fig. 1), which confirmed the diagnosis. Emergency surgical intervention was performed to prevent further progression of the spinal cord injury, and we identified an SEA.

Optimal treatment for SEA remains controversial, and there is lack of consensus regarding the efficacy of conservative (systemic antibiotic therapy) vs surgical management (laminectomy). Conflicting reports are available in the literature in this context. Pathak et al[19] reported a case of a 13-year-old boy who presented with paraparesis, as well as bladder and bowel involvement and showed an excellent response to 6-week antibiotic therapy with only minimal residual gait disturbance. However, Arko et al[20] reported that exclusive antibiotic use fails in patients with risk factors, including white blood cell counts $>12 \times 10^9$ cells/L and severe neurological involvement. Epstein NE et al[21] reported failure rates of 41% to 42.5% associated with nonoperative treatment (including a 22% risk of permanent paralysis and 3–25% risk of mortality). These authors proposed surgical intervention to avoid these risks. Furthermore, a 5-year prospective study performed by Khursheed et al[22] showed that delayed surgical management (>36 hours after symptom onset) was associated with a poor prognosis. Therefore, they recommend prompt surgical intervention in such cases. Other researchers have reported similar findings.[23,24]

In this case, based on the serious neurological symptoms and imaging studies, we combined conservative and surgical treatment. Considering that *S aureus* is the most common pathogen causing SEA, we initiated treatment with antibiotics sensitive to this organism using the following regimen: intravenous ceftriaxone (3rd generation cephalosporin, 2 g once a day) and vancomycin (500 mg 4 times a day). She underwent an uncomplicated emergency laminectomy at T6–T12 levels with an operation time of approximately 3 hours. Culture of the purulent fluid obtained intraoperatively grew *S aureus* sensitive to sulfamethoxazole, ceftriaxone, vancomycin, levofloxacin, and gentamicin, but resistant to clindamycin and erythromycin. She continued the

ceftriaxone and vancomycin regimen for 4 weeks based on drug sensitivity testing, to ensure complete clearance of the bacteria and prevent recurrence. Her motor and sensory functions were restored to satisfactory levels 16 months postoperatively.

4. Conclusion

In summary, we describe a rare case of a spontaneous SEA, which was initially misdiagnosed as spinal tuberculosis. Multilevel laminectomy combined with long-term antibiotic therapy led to satisfactory prognosis of neurological symptoms and ensured survival of this patient. This case highlights our experience regarding the diagnosis and treatment of SEA and would benefit physicians in clinical practice.

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