Challenges in diagnosis of spinal epidural abscess
A case report

Xiaowei Yang, MD, Runsheng Guo, MD, Xin Lv, MD, Qi Lai, MD, Banglin Xie, MD, Xiaozhen Jiang, MD, Min Dai, MD, Bin Zhang, PhD

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.
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 × 10⁹ 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 2.15–2.37 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 microscissors, 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, 2g 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 × 10⁹ cells/L.
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,
whereas neurological dysfunction occurs in >50% of patients, and few patients present with fever.\textsuperscript{1,11} Neurological dysfunction can be severe and may even include paralysis. \textit{S. aureus} is the most common causative organism isolated from cultures in two-thirds of all cases of SEA,\textsuperscript{12} although other organisms including the \textit{Brucella} and \textit{Streptococcus} species have also been implicated as causative agents.\textsuperscript{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.\textsuperscript{16} Levoﬂoxacin-sensitive and clindamycin-negative \textit{Staphylococcus} was cultured in the specimen obtained from our patient.

The rate of misdiagnosis of SEA is ≥50% despite advances in medical imaging technology.\textsuperscript{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.\textsuperscript{7} Hypointense spinal cord signals on T1-weighted imaging and hyperintense signals on T2WI characterize SEA.\textsuperscript{17} However, diagnosis of multifocal SEA can be challenging and may easily be missed. Therefore, whole spine imaging is important.\textsuperscript{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,\textsuperscript{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\textsuperscript{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\textsuperscript{20} reported that exclusive antibiotic use fails in patients with risk factors, including white blood cell counts >12 × 10\textsuperscript{9} cells/L and severe neurological involvement. Epstein NE et al\textsuperscript{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\textsuperscript{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.\textsuperscript{23,24}

In this case, based on the serious neurological symptoms and imaging studies, we combined conservative and surgical treatment. Considering that \textit{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 \textit{S. aureus} sensitive to sulfamethoxazole, ceftriaxone, vancomycin, levofloxacin, and gentamicin, but resistant to clindamycin and erythromycin. She continued the

![Figure 4](image-url). Image obtained 16 months postoperatively shows that the abnormal signal of the thoracic marrow has disappeared.
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.

Acknowledgments
We greatly appreciate the assistance of the staff from the Department of Imaging, The First Affiliated Hospital of Nanchang University, and thank them for their efforts. We greatly appreciate the assistance of Editag which is Language Service Company.

Author contributions
Investigation: Qi Lai, Banglin Xie, Xiaozhen Jiang.
Writing – original draft: Xiaowei Yang, Xin Lv.
Writing – review & editing: Xiaowei Yang, Runsheng Guo, Min Dai, Bin Zhang.
Bin Zhang orcid: 0000-0002-5876-1238.

References
[1] Khanna RK, Malik GM, Rock JP, et al. Spinal epidural abscess: evaluation of factors influencing outcome. Neurosurgery 1996;39:958–64.
[2] Heusner AP. Nontuberculous spinal epidural infections. N Engl J Med 1948;239:845–54.
[3] Shweikeh F, Saeed K, Bukavina L, et al. An institutional series and contemporary review of bacterial spinal epidural abscess: current status and future directions. Neurosurg Focus 2014;37:E9.
[4] Louis A, Fernandes CM. Spinal epidural abscess. Can J Emerg Med 2005;7:351–4.
[5] Vakili M, Crum-Gianfalone NF. Spinal epidural abscess: a series of 101 cases. Am J Med 2017;130:1458–63.
[6] Alerhand S, Wood S, Long B, et al. The time-sensitive challenge of diagnosing spinal epidural abscess in the emergency department. Intern Emerg Med 2017;12:1179–83.
[7] Sendi P, Bregenzer T, Zimmerli W. Spinal epidural abscess in clinical practice. Q J Med 2008;101:1–2.
[8] Lury K, Smith JK, Castillo M. Imaging of spinal infections. Semin Roentgenol 2006;41:363–79.
[9] Hsieh PC, Liu JC, Wang MY. Introduction: vertebral osteomyelitis and spinal epidural abscess. Neurosurg Focus 2014;37:E1 preceding E1.
[10] Darouiche RO, Hamill RJ, Greenberg SB, et al. Bacterial spinal epidural abscess: review of 43 cases and literature survey. Medicine (Baltimore) 1992;71:369–85.
[11] Curry WT, Hoh BL, Hanjani SA, et al. Spinal epidural abscess: clinical presentation, management and outcome. Surg Neurol 2005;64:279.
[12] Darouiche RO. Spinal epidural abscess. N Engl J Med 2006;355:2012–20.
[13] Lampen R, Bearman G. Epidural abscess caused by Streptococcus milleri in a pregnant woman. BMC Infect Dis 2005;5:100.
[14] Zhang Q, Yang X, Yu H, et al. Spinal epidural abscess caused by Brucella species: a review of 17 cases. J Neurosurg Sci 2017;61:271–6.
[15] Ramhdirani S, Bydon A, Streptococcus intermedius: an unusual cause of spinal epidural abscess. J Spine Surg 2017;3:243–9.
[16] Hwang DM, Lee CW, Nam HT, et al. Long level (T4-L1) spinal epidural abscess in a diabetic patient - a case report. Asian Spine J 2008;2:55–8.
[17] Akalan N, Ozgen T. Infection as a cause of spinal cord compression: a review of 36 spinal epidural abscess cases. Acta Neurochir (Wien) 2000;142:17–23.
[18] Ju KL, Kim SD, Melikian R, et al. Predicting patients with concurrent non-contiguous spinal epidural abscess lesions. Spine J 2015;15:95–101.
[19] Pathak A, Singh P, Gehlot P, et al. Spinal epidural abscess treated with antibiotics alone. BMJ Case Rep 2013: 2013.
[20] Arko L, Quach E, Nguyen V, et al. Medical and surgical management of spinal epidural abscess: a systematic review. Neurosurg Focus 2014;37:E4.
[21] Epstein NE. Timing and prognosis of surgery for spinal epidural abscess: a review. Surg Neurol Int 2015;6(Suppl 19):S475–86.
[22] Khursheed N, Dar S, Ramzan A, et al. Spinal epidural abscess: report on 27 cases. Surg Neurol Int 2017;8:240.
[23] Ma H, Kim I. Clinical outcomes of spinal epidural abscess. Korean J Spine 2012;9:6–11.
[24] Patel AR, Alton TB, Bransford RJ, et al. Spinal epidural abscesses: risk factors, medical versus, surgical management, a retrospective review of 128 cases. Spine J 2014;14:326–30.