



Medical Treatment of a Spinal Epidural Abscess with Neurologic Deficits

Tzu-Tsao Chung¹, Cheng-Ta Hsieh², Ming-Ying Liu¹, and Cheng-Fu Chang^{1,3*}

¹Department of Neurological Surgery, Tri-Service General Hospital, National Defense Medical Center, Taipei;

²Division of Neurosurgery, Department of Surgery, Sijhih Cathay General Hospital, New Taipei;

³Division of Neurosurgery, Department of Surgery, Taipei Medical University Hospital, Taipei, Taiwan, Republic of China

In spite of advances in medical knowledge, imaging techniques, and surgical interventions, the diagnosis and treatment of spinal epidural abscess remains a challenge. The diagnosis is often elusive, but achieving diagnosis prior to the onset of neurological deficits is essential to preserving the vital neurological function. Traditionally, urgent surgical decompression and drainage remain the mainstay therapy for spinal epidural abscess. In recent years, with the improvement of antibiotics, conservative treatment with adequate antibiotics has been proposed as an effective method in selective patients. We report a case of spinal epidural abscess in a 50-year-old man, who complained of progressive weakness of lower extremities for 4 days before admission. After conservative treatment with antibiotics alone, the patient recovered uneventfully from previous neurological deficits. The relevant literature is also reviewed.

Key words: spinal epidural abscess, antibiotics, conservative treatment

INTRODUCTION

As a result of the work of Morgagni in the 17th century, clinicians have recognized spinal epidural abscess as a cause of devastating neurological dysfunction.^{1,2} The incidence of spinal epidural abscess has become higher, accounting for up to 2 per 10000 hospital admissions.³ Increasing numbers of drug addicts and new interventional procedures, such as regional spinal and epidural anesthesia, have been thought to be the cause of this disorder. The crucial issue for selecting appropriate treatment to reduce neurological sequelae and optimizing outcomes is prompt and accurate diagnosis. The treatment of urgent surgical decompression followed by 4 to 6 weeks of appropriate intravenous antibiotics has been advocated for patients with spinal epidural abscess.¹ However, antibiotic therapy alone and nonsurgical management in patients with limited neurological dysfunction have been reported.^{1,2,5,11} Here, we report a case of spinal epidural abscess which was successfully managed by non-surgical

approach. Nonsurgical treatment may be an alternative method in selective patients with spinal epidural abscess.

CASE REPORT

A 50-year-old male presented with progressive weakness of bilateral lower extremities and urinary incontinence for 4 days before admission. He had a history of heroin addiction for several months. At admission, his body temperature was 36.6 °C, blood pressure was 132/72 mmHg, pulse rate was 82/min, and respiratory rate was 16/min. Neurological examination demonstrated hypoesthesia below T10 level, decreased muscle power in lower extremities (MRC scale: 1/5), and loss of anal tone. Moreover, bilateral Babinski's signs were present while bulbocavernosus reflex was absent. His laboratory examinations revealed white blood cell count of $13.2 \times 10^3/\text{ul}$ (91.9% polymorphonuclear neutrophils, and 7.8% lymphocytes) and C-reactive protein (CRP) of 3.69 mg/dl. The radiograph of thoracic and lumbar spine revealed no remarkable finding. Magnetic resonance imaging (MRI) of the spine revealed a mass lesion located at the extradural space of T9-S1, compressing the spinal cord (Fig. 1A and 1B). The mass lesion showed iso-intensity on T1-weighted images and hyper-intensity on T2-weighted images. After injection of gadolinium, the mass had a peripheral enhancement. Another lesion with hyper-intensity on T2-weighted images in the pre-vertebral and intervertebral disc space of L5-S1 was also

Received: November 14, 2011; Revised: April 9, 2012;
Accepted: April 13, 2012

*Corresponding author: Cheng-Fu Chang, Department of Neurological Surgery, Tri-Service General Hospital, National Defense Medical Center, No. 325, Sec. 2, Cheng-Gong Road, Taipei 114, Taiwan, Republic of China. Tel: +886-2-8792-7177; Fax: +886-2-8792-7178; E-mail: kaloka@ndmctsgh.edu.tw

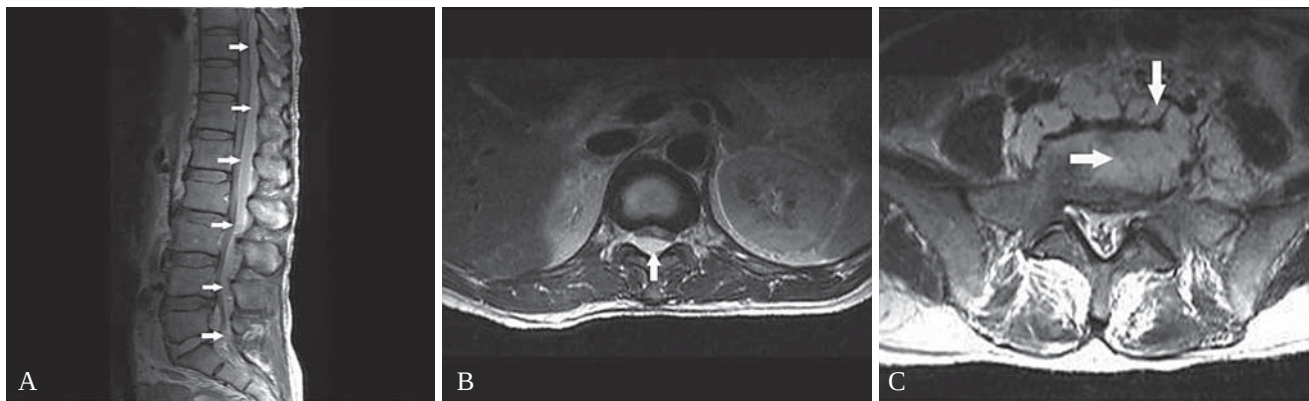


Fig. 1 (A) Sagittal view of T1-weighted MRI of thoracic-lumbar spine revealed a high-intensity lesion (arrows) extending from T9 to S1 dorsal epidural space. (B) Axial view of T1-weighted MRI of thoracic-lumbar spine revealed the enhanced mass (arrow) located in the right posterior aspect of spinal canal compressing the spinal cord. (C) Axial view of T1-weighted MRI of L5-S1 disc space revealed the hyper-intense lesion (arrows) located in disc space and prevertebral space.

found (Fig. 1C). Methicillin-sensitive *Staphylococcus aureus* was cultured from the blood sample. According to the clinical presentations, laboratory examinations and radiological findings, spinal epidural abscess was highly suspected. Because of progressive neurological deficits, surgical intervention was recommended. However, the patient refused it. Conservative treatment with antibiotics (oxacillin, 6 g/day and gentamicin, 160 mg/day) was prescribed. After a six-week course of intravenous oxacillin and gentamicin treatment, the patient recovered uneventfully from previous neurological deficits, was discharged and returned to his normal activities.

DISCUSSION

The overall incidence of spinal epidural abscess accounts for 0.2-1.2 cases per 10000 hospital admissions.^{1,2} It primarily occurs in individuals over 30 years of age and more likely in males.^{1,4} The predisposing factors of spinal epidural abscess were intravenous drugs injection, diabetes mellitus, end-stage renal disease, malignancy, usage of steroids, infection or blunt trauma.^{4,5} Spinal epidural abscess is mainly the result of either contiguous spread or hematogenous dissemination. The source of infection can be identified in 71% to 78% of cases.^{4,6} Most posterior spinal epidural abscesses seem to originate from a distant focus such as a skin infection, pharyngitis, or dental abscess.⁴ Anterior spinal epidural abscesses are commonly associated with discitis or vertebral osteomyelitis caused by direct extension from retropharyngeal or retroperitoneal abscess through communication with

intervertebral foramina.⁷ The location of spinal involvement is 14%-26% in the cervical spine, 31%-63% in the thoracic spine and 21%-44% in the lumbar spine.^{4-6,8} Spinal epidural abscess located in the thoracic spine has been shown to be a poor prognostic indicator in previous studies.^{3,9} This has been attributed to the tenuous blood supply and the small subarachnoid space in the thoracic region that may facilitate rapid neurological compromise. The most frequent causative pathogen for spinal epidural abscess is *Staphylococcus aureus*, implicating in 45%-62% of cases.^{3,5,6} The methicillin-resistant *Staphylococcus aureus* (MRSA) is found in 15% of staphylococcal spinal epidural infections.³ *Streptococci*, *Staphylococcus epidermidis*, *Escherichia coli* and *Pseudomonas aeruginosa* are less common causative pathogens.^{1,4,8}

The classic triad of clinical manifestations of spinal epidural abscess is often stereotyped as localized back pain (presented in about three quarters of patients), progressive neurological deficit (detected in about one third of patients), and fever (documented in half of patients).^{1,3} However, this classic triad is present only in a minority of patients.¹⁰ The coexistence of axial back pain and progressive neurological symptoms remained the most reliable clinical indicators for the presence of spinal epidural abscess.³ The laboratory findings may include leukocytosis, accelerated erythrocyte sedimentation rate, and elevated CRP.^{6,9} MRI with intravenous gadolinium is the best radiological method for the diagnosis of spinal epidural abscess.⁶ It is superior in delineating both the longitudinal and paraspinal extension of the abscess, and may help differentiate infection from other disease enti-

ties in the spinal cord, such as a herniated disc. If MRI is not available, computed tomography (CT scan) with myelogram is advised.

Establishing the diagnosis is essential because a favorable prognosis was found to be directly proportional to prompt accurate diagnosis and initiation of appropriate therapy. Once the diagnosis is confirmed, there are three goals for therapy of the spinal epidural abscess: (1) preservation of normal neurological function; (2) prevention of worsening of existing neurologic deficits; and (3) optimization of opportunities for improvement and return of function.^{1,3} Therefore, urgent surgical decompression, drainage of spinal epidural abscess, and long-term antibiotic therapy remain the mainstay of treatment for spinal epidural abscess.⁴⁻⁶ Meanwhile, surgical intervention with culture can also provide more information of the causative pathogen of spinal epidural abscess. Some authors observed improved outcome if the decompressive surgery was performed within first 24-36 hours after onset of neurological symptom.^{4,10} However, permanent neurological sequelae remain common.¹¹

In recent years, with the advance of antibiotics, some patients with spinal epidural abscess were successfully treated with antibiotics alone.² In a retrospective review of 52 patients with spinal epidural abscess, Savage *et al.* demonstrated good or excellent neurological outcome in 24 of 29 medically treated patients (83%) harboring normal or stable neurological functions and no systemic sepsis.¹¹ This result showed that conservative treatment with antibiotics alone seems to be an alternative treatment of spinal epidural abscess in selective conditions. The decision for medical treatment can be made when the patient presented with back pain alone, with stable neurological functions for more than 72 hours, or higher surgical risks such as co-morbidity condition.^{11,12} If the patient is treated medically, repeated MRI and close neurological examination should be performed to ensure the diminishing and healing of the abscess.¹⁰ If the neurological deficit progresses or the MRI shows an increase in the size of the abscess, prompt surgical intervention is indicated.

In general, empirical antibiotics therapy should provide the coverage against staphylococci (usually vancomycin to treat methicillin-resistant *Staphylococcus aureus*; nafcillin or cefazolin is the preferred management of methicillin-sensitive *Staphylococcus aureus*).⁴ Sometimes, a third- or fourth-generation cephalosporin such as ceftazidime or cefepime can be administered in the presence of documented or suspected gram-negative bacilli infection. Typically, appropriate intravenous antibiotics are administered for 4 to 6 weeks depending on the

culture results.² If vertebral osteomyelitis is suspected or exists, the duration of antibiotics therapy should be at least 6 to 8 weeks.

In this patient, although surgical intervention was indicated for a decrease in muscle power of bilateral lower extremities and urinary incontinence, conservative treatment with adequate antibiotics was administered according to the request of the patient, and good recovery of neurological functions was observed after a six-week course of antibiotics therapy. This successful outcome was compatible with previous reports.¹¹ It seems that non-surgical intervention could be an alternative approach to treating patients with spinal epidural abscess if the neurological deficits have remained stable for more than 72 hours and there are no signs of systemic sepsis.

In conclusion, in patients presenting with back pain and abnormal inflammation parameters (fever, leukocytosis, high ESR, high CRP), we should always include spinal epidural abscess in the differential diagnosis. A patient who presents with stable neurological symptoms for more than 72 hours and no signs of systemic sepsis may be treated with intravenous antibiotics alone as an alternative strategy of management.

DISCLOSURE

All authors declare no competing financial interests.

REFERENCES

1. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. *Neurosurg Rev* 2000;23:175-204.
2. Curry WT Jr, Hoh BL, Amin-Hanjani S, Eskandar EN. Spinal epidural abscess: clinical presentation, management, and outcome. *Surg Neurol* 2005;63:364-371
3. Rigamonti D, Liem L, Sampath P, Knoller N, Namaguchi Y, Schreiber DL, Sloan MA, Wolf A, Zeidman S. Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. *Surg Neurol* 1999;52:189-196.
4. Darouiche RO. Spinal epidural abscess. *N Engl J Med*. 2006;355:2012-2020.
5. Danner RL, Hartman BJ. Update on spinal epidural abscess: 35 cases and review of the literature. *Rev Infect Dis* 1987;9:265-274.
6. Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: a ten-year perspective. *Neurosurgery* 1990;27:177-184.

7. Chao D, Nanda A. Spinal epidural abscess: a diagnostic challenge. *Am Fam Physician* 2002;65:1341-1346.
8. Kaufman DM, Kaplan JG, Litman N. Infectious agents in spinal epidural abscesses. *Neurology* 1980;30:844-850.
9. Tang HJ, Lin HJ, Liu YC, Li CM. Spinal epidural abscess--experience with 46 patients and evaluation of prognostic factors. *J Infect* 2002;45:76-81.
10. Davis DP, Wold RM, Patel RJ, Tran AJ, Tokhi RN, Chan TC, Vilke GM. The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. *J Emerg Med* 2004;26:285-291.
11. Savage K, Holtom PD, Zalavras CG. Spinal epidural abscess: early clinical outcome in patients treated medically. *Clin Orthop Relat Res* 2005;439:56-60.
12. Leys D, Lesoin F, Viaud C, Pasquier F, Rousseaux M, Jomin M, Petit H. Decreased morbidity from acute bacterial spinal epidural abscesses using computed tomography and nonsurgical treatment in selected patients. *Ann Neurol* 1985;17:350-355.