

Spontaneous spinal epidural abscess: A case report

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Abstract

Introduction: Spinal epidural abscess (SEA) is an uncommon but serious infection involving the epidural space that may lead to irreversible neurological damage if not diagnosed and treated promptly. Early recognition is often difficult because clinical manifestations are variable and may occur without identifiable risk factors.

Case presentation: We report the case of a 58-year-old man with a history of chronic alcohol and tobacco use who presented with sudden bilateral lower limb paralysis associated with urinary dysfunction. In the months preceding admission, he described progressive fatigue, intermittent dorsal pain, and lower limb paresthesia without any history of trauma or invasive procedures. Neurological examination revealed complete motor deficit of both lower extremities with a sensory level below the umbilicus. Imaging studies, including computed tomography and magnetic resonance imaging, demonstrated a posterior epidural collection extending from the sixth to the twelfth thoracic vertebral levels, causing marked spinal cord compression with extension into adjacent paraspinal tissues. The patient underwent urgent decompressive laminectomy with evacuation of purulent material. Microbiological analysis identified *Staphylococcus aureus*, and appropriate intravenous antibiotic therapy was initiated. Partial neurological recovery was observed postoperatively.

Conclusion: This case highlights the importance of considering spinal epidural abscess in patients presenting with acute or progressive neurological deficits and emphasizes the role of early imaging and prompt combined surgical and antimicrobial management in improving outcomes.

Keywords: Spinal epidural abscess; Magnetic resonance imaging; Spinal cord compression; Neurological deficit; *Staphylococcus aureus*; Decompressive laminectomy

1. Introduction

Spinal epidural abscess is an uncommon infectious condition characterized by the accumulation of purulent material within the epidural space, resulting in compression of the spinal cord and nerve roots [1]. Its incidence ranges from 0.2 to 2 cases per 10,000 hospital admissions [1]. Although typically associated with predisposing factors such as diabetes mellitus, immunosuppression, intravenous drug use, or recent spinal interventions, spontaneous cases without an identifiable source remain rare [2].

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The clinical presentation is often nonspecific, contributing to frequent delays in diagnosis. Classical symptoms such as back pain, fever, and neurological deficit are not consistently present, and misdiagnosis at initial evaluation is common [3]. If untreated, spinal epidural abscess may lead to severe neurological impairment or death.

Magnetic resonance imaging plays a key role in diagnosis, allowing precise evaluation of the location, extent, and impact of the epidural collection. Management relies on urgent surgical decompression combined with appropriate antimicrobial therapy.

We report a case of spontaneous spinal epidural abscess presenting with acute paraplegia and sphincter dysfunction, highlighting the diagnostic challenges and the importance of early imaging.

2. Case presentation

A 58-year-old man presented to our emergency department with a sudden onset of bilateral lower limb weakness associated with sphincter disturbances. His medical history was notable for chronic tobacco use for approximately 50 years, ongoing at the time of presentation, and chronic alcohol consumption with abstinence for the past four years. No recent precipitating factors were identified, including trauma, infection, intravenous drug use, or invasive dental or spinal procedures. He also had no history of tuberculosis or known exposure.

The patient reported that his symptoms had begun approximately two months prior to admission, with progressive asthenia, anorexia, intermittent muscle cramps of both lower limbs, and paresthesia associated with exertional pain. These symptoms occurred in a context of intermittent episodes of fever and chills. One day before admission, his condition acutely worsened with the abrupt onset of heaviness and weakness of both lower limbs, associated with sphincter dysfunction, consisting of acute urinary retention and constipation, which prompted emergency hospital admission.

At presentation, the patient was conscious and clinically stable from a hemodynamic standpoint. Vital parameters showed a heart rate of 88 beats per minute, blood pressure of 125/70 mmHg, and a fever of 38.5°C.

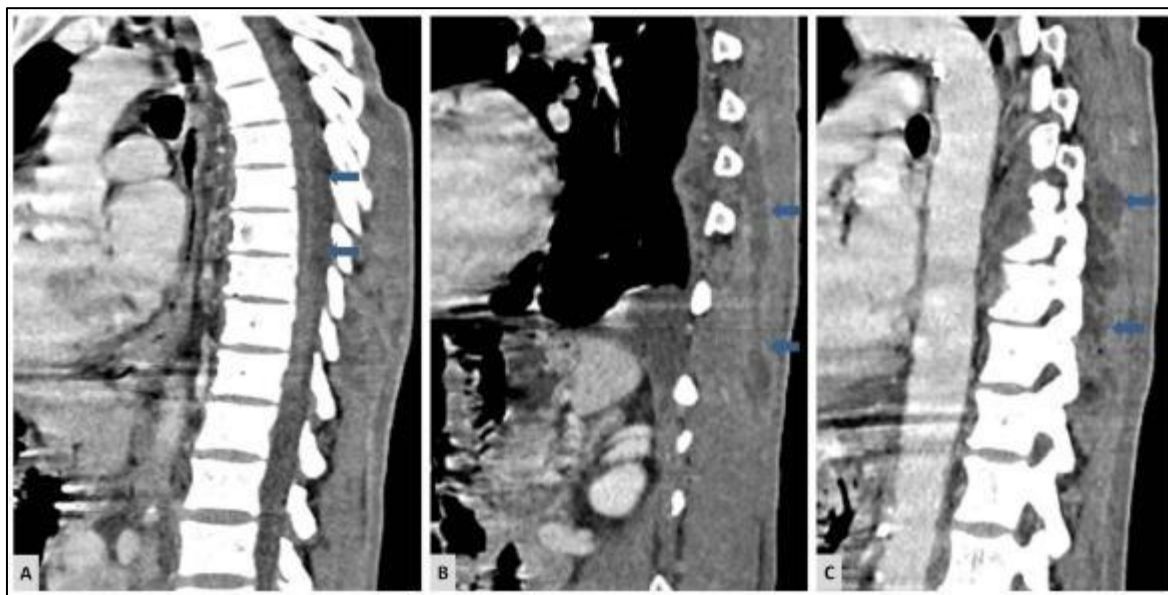


Figure 1 Post-contrast sagittal computed tomography images of the thoracic spine demonstrating a posterior epidural collection extending from T6 to T12, with peripheral enhancement and significant mass effect causing anterior compression of the spinal cord (A). Extension through the intervertebral foramina into the bilateral paraspinous muscles is noted (B, C)

Neurological examination revealed an inability to stand or walk. Motor strength in the upper limbs was normal (5/5), whereas a complete motor deficit was noted in both lower limbs, with muscle strength graded 0/5 proximally and distally. Deep tendon reflexes were absent in the lower limbs, with an indifferent plantar response. Sensory examination demonstrated a clearly defined sensory level below the umbilicus, with bilateral impairment of proprioception. Muscle tone was decreased in both lower limbs. The Lasègue test was negative, and examination of the spine reveals no spinal

tenderness. A distended bladder was also noted. Initial laboratory evaluation revealed significant leukocytosis ($17.620/\text{mm}^3$) associated with a markedly increased C-reactive protein concentration of 319 mg/L.

An initial computed tomography (CT) scan of the spine was performed in the emergency setting. CT demonstrated a posterior epidural lesion extending from the Th6 to the Th12 vertebral levels. The lesion showed hypodense fluid attenuation with peripheral wall enhancement after contrast administration, consistent with an abscess. The collection measured up to 9 mm in maximal anteroposterior diameter within the spinal canal and exerted a significant mass effect on the spinal cord, resulting in anterior compression. Extension of the collection was noted through the intervertebral foramina into the paraspinal erector spinae muscles bilaterally, more pronounced on the left side. No vertebral body fracture or obvious destructive bone lesion was identified (Figures 1, 2 and 3).



Figure 2 Axial post-contrast computed tomography image showing extension of the epidural collection through the intervertebral foramina into the paraspinal muscles and the vertebromediastinal recess



Figure 3 Sagittal (A) and coronal (B) computed tomography images in bone window showing no evidence of vertebral fracture or osseous destructive lesions

Subsequent magnetic resonance imaging (MRI) of the spine was performed and confirmed the presence of a posterior epidural collection extending from Th6 to Th12. On T1-weighted images, the lesion appeared isointense relative to the spinal cord. On T2-weighted and STIR sequences, it demonstrated heterogeneous hyperintensity with associated compression and anterior displacement of the spinal cord. Contrast-enhanced images revealed peripheral rim enhancement of the epidural collection with a non-enhancing central component. No evidence of vertebral body destruction, disc involvement, or associated spondylodiscitis was observed (Figures 4 and 5).

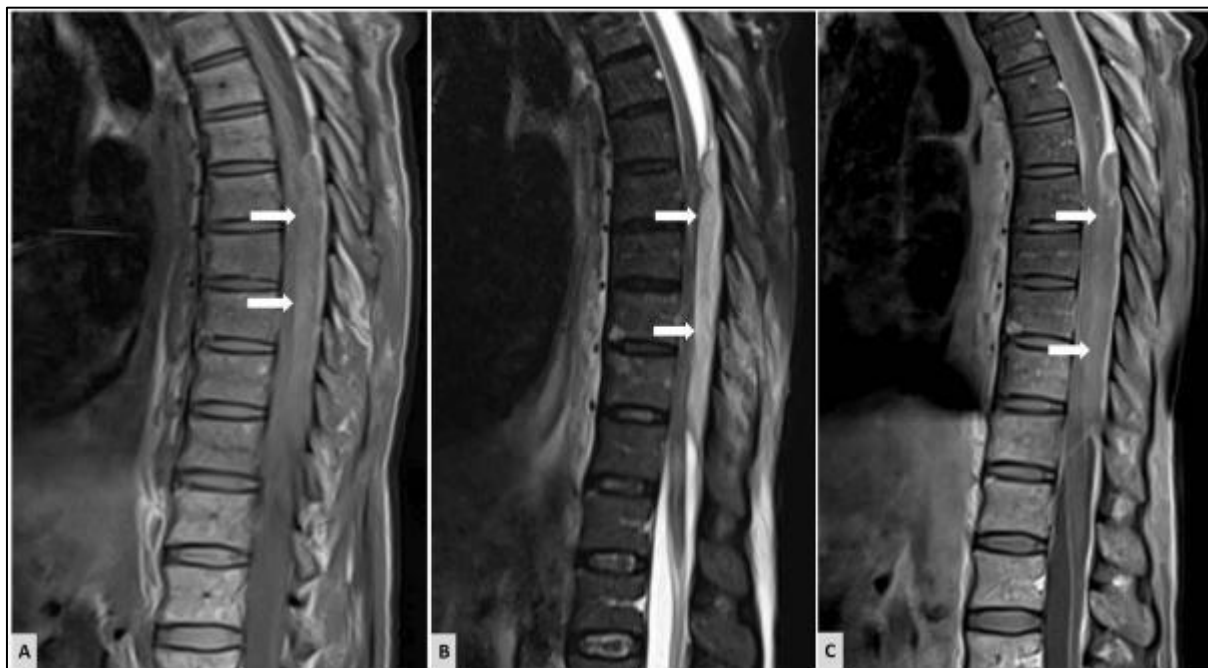


Figure 4 Sagittal magnetic resonance images: T1-weighted (A), T2-weighted (B), and post-contrast T1-weighted (C).

The posterior epidural collection extends from T6 to T12, appearing isointense on T1-weighted images and heterogeneously hyperintense on T2-weighted images, with marked anterior displacement and compression of the spinal cord. Post-contrast images demonstrate peripheral rim enhancement with a non-enhancing central component. No associated vertebral body destruction or disc involvement is observed

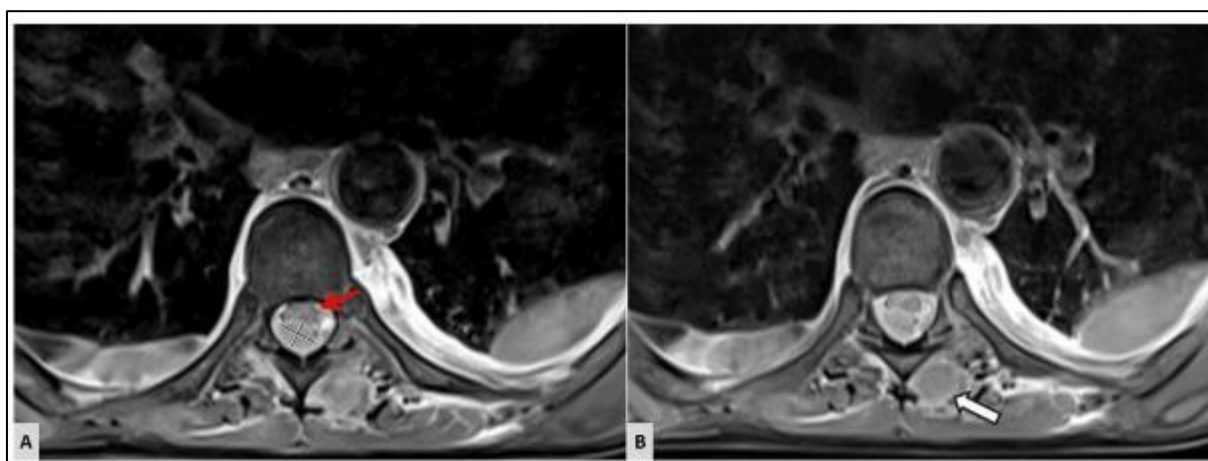


Figure 5 Axial post-contrast fat-suppressed T1-weighted magnetic resonance image demonstrating a posterior epidural collection causing anterior displacement of the spinal cord (red arrow), with extension into the paravertebral soft tissues (white arrow)

Given the severity of the neurological deficits and the imaging evidence of spinal cord compression, the patient underwent urgent surgical management. A decompressive laminectomy was performed with evacuation of the posterior epidural abscess. Intraoperative findings revealed purulent material, and a sample of the pus was sent for microbiological analysis.

Culture of the abscess identified *Staphylococcus aureus*, and the patient was subsequently treated with targeted intravenous antibiotic therapy, adjusted according to antimicrobial sensitivity results.

Following combined surgical and medical management, the patient showed partial neurological recovery.

3. Discussion

Spinal epidural abscess (SEA) is an uncommon yet serious infectious entity, with a reported incidence of approximately 0.2 to 2 cases per 10,000 hospital admissions [1]. Despite advances in diagnostic imaging and antimicrobial therapy, SEA remains associated with significant morbidity, particularly when diagnosis and treatment are delayed. The thoracic spine is less commonly affected than the lumbar region, and spontaneous epidural abscesses without an identifiable predisposing factor are uncommon [4].

Spontaneous spinal epidural abscess is most commonly associated with underlying risk factors that predispose patients to hematogenous spread of infection. The most frequently reported risk factors include diabetes mellitus, chronic renal failure, immunosuppression, intravenous drug use, alcoholism, and advanced age [5]. Chronic infections, skin and soft tissue infections, and bacteremia-particularly due to *Staphylococcus aureus*-are also well-recognized predisposing conditions [4]. However, a significant proportion of cases occur in patients without identifiable risk factors or obvious sources of infection, which may contribute to diagnostic delays [5].

The variable and non-specific presentation of spontaneous SEA often leads to delays in diagnosis and treatment. The clinical presentation of SEA is frequently vague, and the full triad of back pain, fever, and neurological impairment is observed in only a small proportion of patients. Misdiagnosis is frequent, notably in patients with an initially preserved neurological status, especially when classical signs of infection such as fever, elevated inflammatory markers, or leukocytosis are lacking [3]. In the present case, the patient initially developed insidious systemic and neurological symptoms over several months, followed by abrupt neurological deterioration with acute paraplegia and sphincter dysfunction, reflecting sudden spinal cord compression. This biphasic evolution has been described in the literature and underscores the diagnostic challenge of SEA, particularly in the absence of spinal pain or clear infectious signs [6].

Imaging plays a central role in the diagnosis and management of SEA. Magnetic resonance imaging (MRI) is considered the gold standard, with reported sensitivity and specificity exceeding 90% [7]. MRI allows precise assessment of the epidural collection, including its location, size, and characteristics, while also assessing spinal cord compression and any related complications. Typical MRI features include a T1 hypointense and T2 hyperintense epidural collection with peripheral rim enhancement after gadolinium administration, as observed in our patient. Diffusion-weighted imaging, when available, further improves diagnostic confidence by demonstrating restricted diffusion within the abscess cavity [8]. Computed tomography (CT), although less sensitive than MRI, remains useful in emergency settings and can demonstrate an epidural collection, associated mass effect, and paraspinal extension, particularly when MRI is not immediately available.

The differential diagnosis of epidural space-occupying lesions includes epidural hematoma, metastatic disease, lymphoma, and inflammatory or granulomatous conditions. In this context, the presence of rim enhancement, fluid characteristics, foraminal extension, and an infectious clinical background strongly favor the diagnosis of an epidural abscess [9].

Management of SEA requires a multidisciplinary approach combining urgent surgical decompression and targeted antibiotic therapy, especially in patients presenting with neurological deficits. *Staphylococcus aureus* is the most frequently isolated pathogen, accounting for approximately 60-70% of cases, consistent with the microbiological findings in this patient [2]. Early surgical intervention aims to relieve spinal cord compression, obtain microbiological samples, and prevent irreversible neurological damage. Despite prompt treatment, neurological recovery may be incomplete, particularly in patients with severe preoperative deficits or delayed diagnosis, which explains the partial recovery observed in our case [9].

4. Conclusion

Spontaneous spinal epidural abscess is an uncommon but potentially severe condition that demands a high degree of clinical suspicion, especially in patients presenting with acute or subacute neurological deficits, even when traditional risk factors are absent. Early identification, rapid MRI evaluation, and prompt surgical decompression along with targeted antibiotic therapy are essential to prevent permanent neurological injury.

This case emphasizes the need to include SEA in the differential diagnosis of acute paraplegia and highlights the importance of a multidisciplinary approach to optimize patient outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflicts of interest.

Statement of informed consent

Written informed consent was obtained from the patient for publication.

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