

Spondilodiscitis and Lumbar Epidural Abscess Occurring after Orthopedic Epidural Anesthesia: A Case Report

ABSTRACT

Spinal epidural abscess is a rare complication of epidural catheterization with an estimated incidence of 0.2 to 2.8 cases per 10,000 per year, with the peak incidence occurring in patients in their 60's and 70's. Delay in diagnosis or treatment may have devastating consequences, resulting in severe and irreversible neurological impairment or even death. We present a patient with lumbar vertebral osteomyelitis, discitis, and epidural abscess who complained of persistent low back pain for 4 months after epidural anesthesia for an orthopedic procedure.

KEY WORDS: Epidural abscess, epidural anesthesia, osteomyelitis, discitis

INTRODUCTION

Spinal epidural abscess is a rare complication of epidural catheterization with an estimated incidence of 0.2 to 2.8 cases per 10,000 per year, with the peak incidence occurring in patients in their 60's and 70's. The most common causative agent is *Staphylococcus aureus*. 1,2 Associated predisposing conditions include a compromised immune system such as occurs in patients with diabetes mellitus (DM), AIDS (acquired immunodeficiency syndrome), chronic renal failure, alcoholism, or cancer, 3-5 or previous epidural anesthesia, spinal surgery, or trauma⁶ (Table I). Chronic diseases or conditions that compromise immunity are found in SEA patients. Associated conditions

Table I: Predisposing Factors for Epidural Abscess

Existence of an infectious source

- Local - Decubitus ulcer, Pott's disease (psoas abscess), pharyngitis, mediastinitis, pyelonephritis, and perinephric abscess
- Remote - Skin, respiratory (otitis, sinusitis, pneumonia), pharyngeal or dental infections, endocarditis, decubitus ulcer, UTI

Immunodeficiency

- AIDS
- Alcoholism
- Chronic Renal Failure
- Chronic Disease
- Diabetes Mellitus
- Intravenous drug abuse
- Malignancy

Spinal procedure or surgery

- Open procedures - All types of lumbar surgery
- Closed procedures - Catheter insertion, LP

Spinal Trauma

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are DM, iv drug abuse, chronic renal failure, alcoholism, cancer, Pott's disease, HIV positivity and recurrent urinary, respiratory, cardiac (endocarditis), skin (furuncle), pharyngeal and dental infections such as abscess. SEA may also occur following open (lumbar discectomy) or closed (lumbar puncture, epidural catheter insertion for epidural anesthesia) spinal procedures. No predisposing condition can be found in 20 percent of patients, and the condition has also been reported in patients with no predisposing risk factors. 7 Delay in diagnosis or treatment may have devastating consequences resulting in severe and irreversible neurological impairment or even death. 2 A small but steady number of case reports have been published over the years. 8 Infectious complications have been occasionally reported after epidural anesthesia and analgesia. These have included epidural abscess, meningitis, osteomyelitis, discitis and radiculoneuritis. 9, 10

We present a patient with lumbar vertebral osteomyelitis, discitis, and epidural abscess who complained of persistent low back pain for 4 months after epidural anesthesia for an orthopedic procedure.

CASE REPORT

A 66-year-old, 71 kg female had been operated on for bilateral total knee prosthesis with epidural anesthesia in a different institution about 4 months previously. One month after the surgery, she started having back pain and right leg paresthesia observed primarily paraspinally in the lumbar region. She was treated with medication for her moderately severe pain and paresthesia in a hospital and her complaints were reduced. However, she was hospitalized and immobilized in the neurosurgery ward of our hospital as her complaints continued and progressed. Systemic examination was unremarkable. Neurological examination revealed hypoesthesia in the right leg, local tenderness in the lumbar region, back pain and a temperature of 37.2°C.

Deminerlization, scalloping of endplates and spondylolysis in the L4 and L5 regions was revealed by lumbar X-Rays. Leukocytosis, elevated ESR, or other abnormal laboratory findings were not initially present. MRI of lumbar spine showed cystic necrotic changes with a hyper-intense and serpiginous contour, predominantly at the L4-5

intervertebral space that extended over the edge of the anterior epidural space (Figure 1). The inferior half of the L4 and superior half of the L5 vertebrae had spondylolytic changes. L4-L5 disc material was herniated and degenerated at the right side. A left-sided posterolateral and ventral epidural mass opacified peripherally after contrast administration indicating epidural abscess (Figure 2). Biochemical examination showed a progressively increasing sedimentation rate (110 and 119 mm/h) and CRP value (3.79, 6.14, and 6.00 ng/ml) and leukocytosis.

The patient was taken to surgery due to congeniality of neurological and radiological proofs. She was intubated after induction with 2 mg/kg propofol, 1 mug/kg fentanil and 0.6 mg/kg

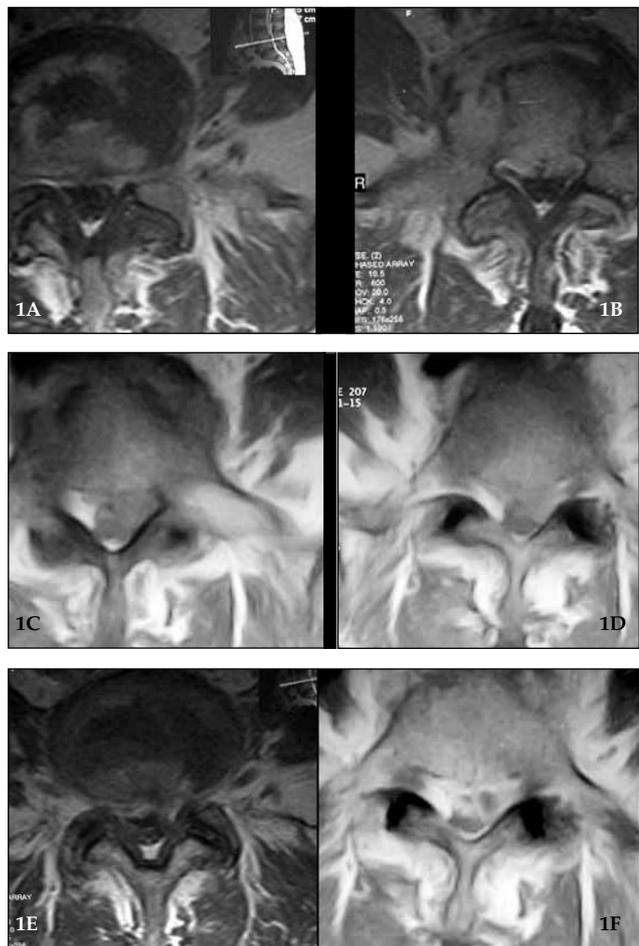


Figure 1: T1-weighted axial images (A,B) show heterogeneous appearance of end-plate fragmentations and T2-weighted axial images (C,D) reveal obstruction of the left posterolateral epidural space by a slightly hyperintense lesion at the L4-L5 intervertebral disc space. Coexistence of right-sided disc herniation (E) and peripheral contrast fixation of infective lesion on the left (F)

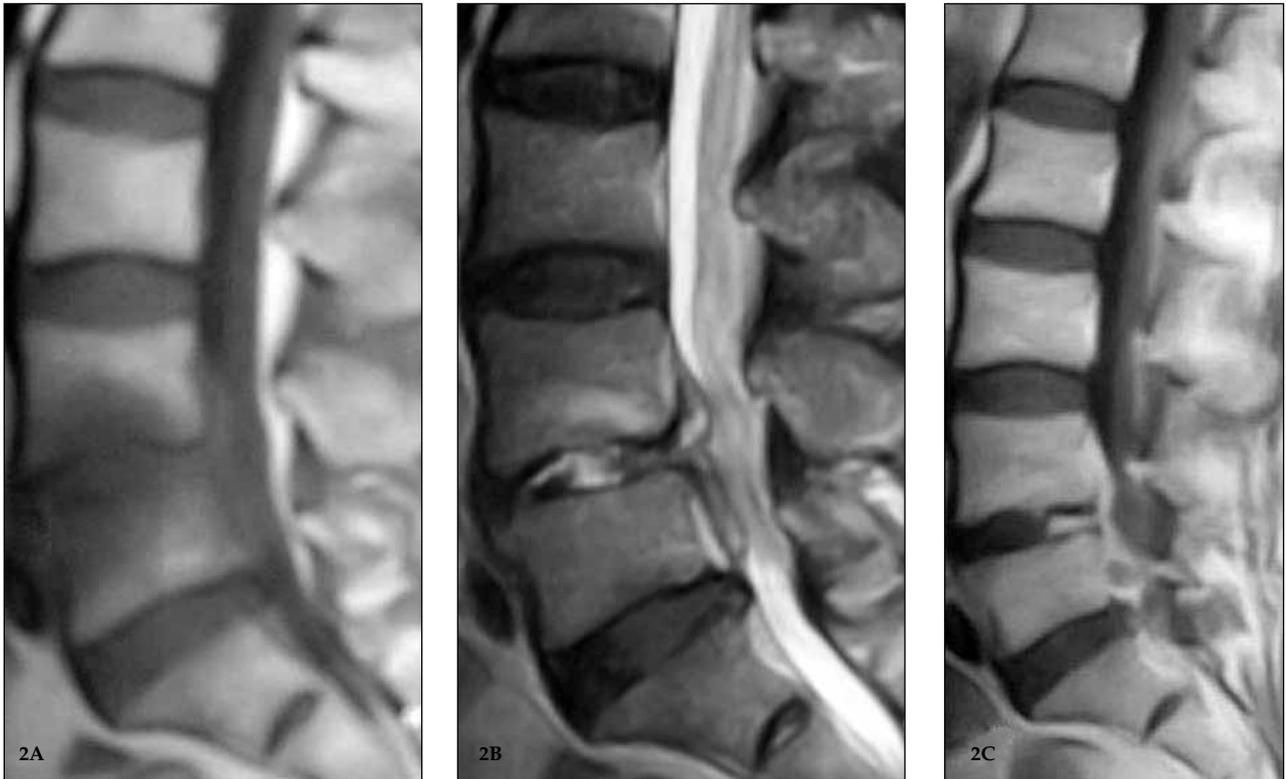


Figure 2: T1- (A) and T2-weighted (B) sagittal images show spondylolytic changes of the inferior part of L4 and the superior part of L5. Please note the coexistent disc herniation and obstruction of the anterior epidural space. T1 contrast sagittal cut (C) shows peripheral contrast enhancement of the same level.

rocuronium. Anesthesia was continued with %50 O₂ %50 N₂O and 1-2% sevoflurane. The dorsolumbar fascia was opened and the paravertebral muscles were dissected off from the right side due to the herniated disk and to opinion of easy leakage of the purulent abscess in spite of its opposite (left side) location. A fenestration was performed at the level of L4-L5, and the dissection was continued until the dura. The dura looked healthy, intact and pulsatile. Standard discectomy was performed at the L4-L5 level. Leakage of the abscess was very mild due to the granulomatous form of the infection, and it was therefore partially skimmed off. A suction drain was inserted, and the wound was closed. Operative cultures of the purulent material revealed the presence of *Staphylococcus aureus* organisms, sensitive to sulbactam-ampicillin. The patient was administered sulbactam-ampicillin (Ampicid 1g injectable vial, Mustafa Nevzat, Türkiye) intravenously, 3g per day in three divided doses for 3 weeks after the surgery. The back pain and leg paresthesia of the patient improved progressively after the exploration but the paresis unfortunately

persisted. She was mobilized with a TLSO (thoracolumbo-sacral orthosis) girdle and discharged to a nursing home after 3 weeks of IV antibiotic treatment. IV therapy was converted to an oral form of sulbactam-ampicillin (Ampicid 1g tablet, Mustafa Nevzat, Turkey), 3g per day in three divided doses for 3 more weeks. A repeat MRI of the spine showed resolution of the infection.

DISCUSSION

The hematoma developing following a vertebral fracture may become infected in a trauma patient. The mechanisms leading to back pain and right leg paresthesia may be compression and destruction of the spinal cord or cauda equina roots within the spinal canal due to the spinal epidural abscess. Cord and nerve root compression from the extradural mass within the rigid spinal canal might appear to be the obvious explanation of clinical findings but neurological dysfunction is often disproportionate to the observed degree of compression.⁶ Many authors have postulated that edema and inflammation in the epidural space may involve the valveless epidural venous plexus (Batson's plexus), which may

compromise circulation and result in cord ischemia.^{5,11} A combination of compressive and ischemic effects may result in the disastrous sequelae of epidural abscess.

The diagnosis of epidural abscess must be made immediately because of a delay in treatment can lead to irreversible neurological damage or death.² Cranial or spinal epidural abscesses are emergency situations. Development of arterial and/or venous ischemia and infarction secondary to infective thrombophlebitis are the main causes of the neurological findings rather than compressive effects. The development of neurological dysfunction may take a few hours to several months. The presentation of a spinal epidural abscess can be nonspecific. Fever, malaise, and back pain are the earliest symptoms. ¹² Local tenderness, with or without a neurological deficit, is the usual physical finding, and leukocytosis may be the only abnormal laboratory finding. Epidural abscess should be suspected in patients presenting with fever and back pain, especially if the risk factors listed in Table I are present. Age and epidural anesthesia were the predisposing factors in the present case. Once the diagnosis is suspected, emergency neurosurgical consultation is recommended. The average time from back pain to root symptoms, and from root symptoms to weakness can be short (3-4 days for each). Only 24 hours is sufficient for total paralysis to develop from weakness. Emergency decompression should therefore be considered if the patient does not have major paresis and presents with minor finding such as back pain and fever . A blood culture should be obtained (blood cultures are positive in more than 60 percent of the patients) to confirm the diagnosis.⁵

Surgical decompression is the mainstay of treatment for spinal epidural abscess. Endoscopy-assisted surgery¹³ and percutaneous drainage ¹⁴ are other reported successful treatments. A recent study ¹⁵ suggests that surgery provides a good outcome when patients' cord symptoms (bowel and bladder dysfunction, paresis, or "plegia") are present for less than 72 hours, when the extent of the abscess (the degree of thecal sac compression) is less than 50 percent, and when patients are younger than 60 years of age. In our case, duration of cord symptoms (leg paresthesia) for three months indicated a poor outcome but our patient healed completely. This

situation can be explained by the patient's symptoms. Our patient only had pain and paresthesia. These finding can appear due to a compressive effect of the disease and irreversible ischemic effects secondary to thrombophlebitis might not be present.

In conclusion, it is very important to provide aseptic conditions during insertion of epidural catheters because of the severity of this emergency infective complication. General anesthesia may be a better choice, especially for patients that have predisposing factors.

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