Atypical Presentation of Spinal Epidural Abscess—Prolonged and Intractable Abdominal Pain

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SUMMARY

Despite advances in medicine, early diagnosis of spinal epidural abscess remains a challenge to clinicians. The most common symptoms of spinal epidural abscess include back pain, fever, and neuralgic deficits. However, spinal epidural abscess can also present with vague and nonspecific symptoms. In this case, a 68-year-old male had abdominal pain in the right upper quadrant lasting 3 weeks and was diagnosed with a gastric ulcer. After treatment, his symptoms did not resolve. Fever and back pain became evident as his disease progressed, followed by right lower limb weakness and the inability to walk. He was taken to the emergency department of our hospital, and the weakness of his lower extremities worsened during hospitalization. His right leg became completely paralyzed despite the use of intravenous antibiotics. A spinal computed tomography scan was performed emergently (magnetic resonance imaging was unavailable) and revealed an epidural abscess involving T5–6 with adjacent osteomyelitis. The patient underwent posterior decompressive laminectomy with pus drainage in the T4–7 region. His neuralgic examinations improved soon after the operation, but ambulation remained limited. Early diagnosis is crucial to the prognosis of spinal epidural abscess, because delayed diagnosis usually results in complete paralysis even death. Thus, clinicians should be aware of atypical presentations of spinal epidural abscess. [International Journal of Gerontology 2009; 3(4): 244–247]

Key Words: abdominal pain, epidural abscess, osteomyelitis

Introduction

The early diagnosis of spinal epidural abscess (SEA), first described by Morgagni[1] in 1761, remains a challenge to clinicians despite advances in medical knowledge, imaging studies and surgical techniques[2,3]. The incidence of this disease was approximately 0.2–2 cases per 10,000 hospital admissions two decades ago[4]; however, the number has doubled in the last 20 years because of the increases in the aging population, increased use of spinal instrumentation, and the spread of injection drug use[2,5,6]. Most patients with SEA have predisposing conditions including diabetes, chronic liver disease, immunocompromised condition, intravenous drug use, spinal surgery or trauma, and a systemic source of infections[2,3,7–9]. With prompt diagnosis and therapy, SEA can be managed with little associated morbidity. However, the rate of irreversible paralysis or death remains high with a range of 18–31%[4,7,10].

The most common symptoms of SEA include back pain, fever, and neuralgic deficits[2]; however, the clinical triad is present only in a minority of patients[10–12]. This report describes a patient with intractable abdominal

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pain radiating to the back as the primary symptom of thoracic SEA.

Case Report

A 68-year-old man had history of hepatitis C infection and was otherwise very healthy. He had been admitted to a regional hospital because of right upper quadrant abdominal pain radiating to his back for approximately 20 days. An esophagogastroduodenoscopy examination showed a gastric ulcer. Despite treatment of the gastric ulcer, his back pain became severe. He then visited an orthopedic outpatient clinic for help before admission to our clinic. A thoracolumbar spine X-ray showed degenerative spondylosis (Figure 1). However, even with analgesics, the symptoms did not improve. Unfortunately, progressive weakness in the right leg became prominent. Soon, he was unable to walk. Two days prior to admission to our clinic, he developed fever with worsening pain in the right upper quadrant of his abdomen. There was no evidence of jaundice or abnormal changes in the appearance of his stool or urine.

As the abdominal pain continued to get worse, he was brought to our hospital for help. On admission, the patient's body temperature was 36.6°C. Examination of the abdomen revealed mild tenderness in the right upper quadrant and epigastric region. The patient's neck was slightly stiff and the lower spinal region showed no tenderness. The neurologic examination revealed muscle strength of 5/5 in the upper extremities, 4/5 in the left lower extremity, and 2–3/5 in the right lower extremity. The patient's peripheral white blood cell count was 16,200/mm^3 (90% neutrophils, 3% lymphocytes, 7% monocytes) and the alkaline phosphatase was 148 U/L (laboratory range, 38–126 U/L); albumin was 2.3 g/dL (range, 3.5–5.0 g/dL). The rest of the serum chemistries were normal.

The posteroanterior chest X-ray revealed increased bilateral lower lobe interstitial infiltration. An abdominal X-ray revealed lumbar spondylosis with marked spur formation and distended bowel loops at the upper abdomen. The cervical spine X-ray also revealed spondylosis. Blood and urine cultures were taken. Intravenous oxacillin and ciprofloxacin were given to treat the suspected spinal infection. Abdominal ultrasonography showed chronic liver disease and stones in the dependent portion of the gall bladder.

On day 4 after admission, a physical examination revealed progressive weakness of the left lower extremity (muscle strength, 1–2/5) and paralysis over the right lower extremity. We consulted a neurosurgeon. A spinal lesion at the T5 sensory level with multiple neurologic deficits in both lower extremities was identified. Because magnetic resonance imaging was not immediately available, emergent computed tomography scan of the thoracic spine was performed (Figure 2). Diskitis involving T5–6 with adjacent osteomyelitis was found. Abscess

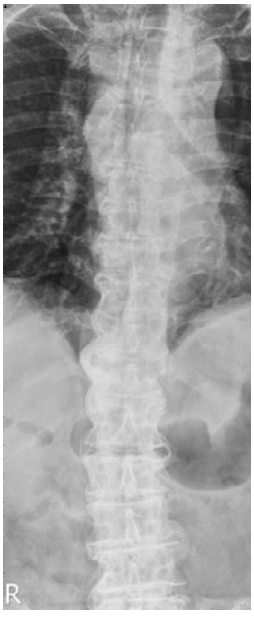

Figure 1. Spur formation of the thoracic and lumbar spine indicating degenerative spondylosis.

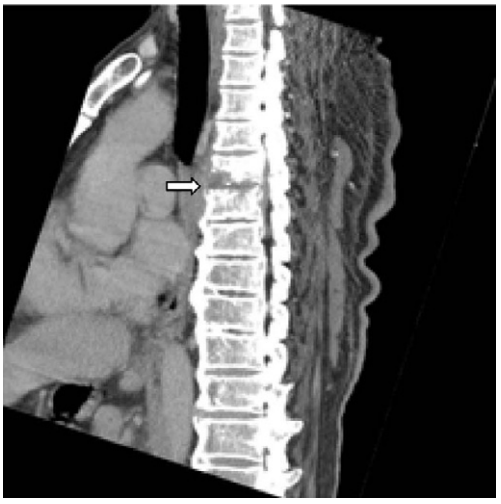

Figure 2. Diskitis involving T5–6 with adjacent osteomyelitis as well as paraspinal and epidural abscess formation (arrow) that resulted in spinal stenosis.

formation of the paraspinal and epidural areas resulted in spinal stenosis.

The patient was immediately scheduled for decompressive thoracic laminectomy for the epidural abscesses at T4–7. Purulent discharge was noted intraoperatively, and cultures were done. Cultures of blood yielded methicillin-sensitive *Staphylococcus aureus*, but the culture of drainage was negative. Antibiotics were changed to intravenous oxacillin and gentamicin. A transthoracic echocardiogram was performed to rule out endocarditis. Fortunately, no valvular vegetation was found. Subsequent blood cultures after the initiation of intravenous antibiotics and surgical decompressive laminectomies were normal.

The patient underwent physical therapy for muscle strength training after his condition stabilized. A follow-up magnetic resonance imaging (6 weeks after admission) revealed diskitis involving T5–6 with adjacent osteomyelitis and suspected diskitis involving T11–12. After a prolonged course of antibiotics (10 weeks), neurologic symptoms in his lower extremities improved. Two months after the operation, the patient had regained partial strength (2–3/5) in the left lower extremity. His right lower extremity, however, remained paralyzed.

Discussion

SEA is an uncommon disease with a low incidence of 0.2–2 cases per 10,000 admissions[4]. In an extensive review and meta-analysis, Reihsaus et al.[7] mentioned that roughly 70% of SEA occurred between the ages of 31 and 70 years, with no tendency toward any particular decade. However, some case series showed that SEA has a peak incidence in the sixth and seventh decades of life[13–15]. Reihsaus et al. also observed a preference for the male sex. Even though some authors stated that there was no association between age and outcomes[4,12,14], others believed that age was an important prognostic factor for outcomes[16].

The triad of fever, back pain and neurologic deficit has often been described in the literature. But, in fact, the clinical triad is present only in a minority of patients. There is a generally accepted staging system that outlines the progression of symptoms and signs in SEA: (1) stage 1, back pain at the level of the affected spine; (2) stage 2, nerve root pain radiating from the involved spinal area; (3) stage 3, motor weakness, sensory deficit, and bladder and bowel dysfunction; and (4) stage 4, paralysis[2,4]. The case discussed in this report was presented with prolonged and intractable abdominal pain. There were other cases of SEA presenting with intra-abdominal pathology that have been reported in the literature[17,18]. One of these cases was noted to have right upper quadrant and epigastric abdominal pain with referred pain in the T5–7 dermatomal distribution. SEAs are more commonly found in posterior areas and thoracolumbar areas[4], and are usually extended over three to four vertebrae[4,10,12,15,16].

Although the white blood cell count is not a reliable marker for SEA, leukocytosis is detected in about two-thirds of patients[4,14]. Generally, erythrocyte sedimentation rate and C-reactive protein are almost uniformly elevated[14,15]. *S. aureus* is the most common pathogen of this infection[3,7].

Magnetic resonance imaging is considered to be the gold standard imaging study for diagnosing SEA[4,10,15]. Even though computed tomography of the spine is highly sensitive for diagnosis, computed tomography was performed only because the magnetic resonance imaging was unavailable. Radionuclide scanning (gallium citrate Ga 67 inflammation scan) may show increased uptake and may help to identify the affected site; however, the findings are neither sensitive nor specific enough for SEA. After the diagnosis is established, the decompressive laminectomy and systemic antibiotics are the treatments of choice[2,7,10,13,14]. Typically, appropriate intravenous antibiotics are administered for 4–6 weeks, followed by 2–4 weeks of oral medications[8,13]. In patients associated with osteomyelitis, the duration of treatment should be extended to 8 weeks[8,10].

For patients with SEA, the prognosis is related to the degree of neurologic impairment and duration of infection[4,10,15]. With the presence of motor deficits, early decompressive surgery should be done promptly, because poor outcome is associated with the presence of motor deficits for more than 24–72 hours[4,16]. Despite the advances in the diagnosis and treatment of SEA, the mortality rates remain high, ranging between 5% and 32%. The morbidity rate can be high if an early diagnosis cannot be established.

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