Multilevel Thoracic Spinal Epidural Abscess: Case Report
Çok Seviyeli Torakal Spinal Epidural Abse: Olgu Sunumu

ABSTRACT
We report a case of multilevel thoracic spinal epidural abscess without any evidence of predisposing factors. Methicillin-susceptible Staphylococcus aureus was identified as the causal pathogen. Infection was managed with surgical debridement and a prolonged course of antibiotic therapy.

KEY WORDS: Spinal epidural abscess

ÖZ
Infeksiyonu yatkınlığı olmayan bir hastada çok seviyeli torakal spinal epidural abse olgusu sunulmuştur. Metisiline duyarlı Staphylococcus aureus patojen olarak saptandı. Infeksiyon, cerrahi debridman ve uzun süreli antibiyotik ile tedavi edildi.

ANAHTAR SÖZCÜKLER: Spinal epidural abse
INTRODUCTION
Spinal epidural abscess is an uncommon, severe and generally pyogenic infection of the epidural space requiring urgent neurosurgical intervention. Spinal epidural abscess has been reported to have an incidence of 0.2-1.96 per 10,000 hospital admissions (1, 6). The abscess commonly occurs after lumbar puncture, epidural anesthesia or spinal surgery. The most common predisposing factors are greater numbers of spinal procedures, increased use of immunosuppressive agents and antibiotics, diabetes mellitus, trauma, intravenous drug abuse, alcoholism and septicemia. However no identifiable predisposing factor can be found in some patients (2, 3, 6, 7). The diagnosis of spinal epidural abscess is missed in about half of the cases. Delayed diagnosis and treatment result in an unexpectedly rapid neurological deterioration, even in patients without prior neurological deficit (4, 7).

We report a 17-year-old boy who presented with significant weakness of both lower limbs and incontinence after lifting heavy weights without evidence of the aforementioned predisposing factors.

CASE REPORT
A 17-year-old man presented with a twenty-five day history of back pain at rest and weakness of the lower limbs. The weakness slowly progressed and was combined with loss of sensation and bladder and bowel dysfunction. The patient only gave a history of lifting heavy weights 24 hours before the onset of the weakness. There was no history of trauma or predisposing factors associated with spinal epidural abscess. Seven days before his admission to our hospital, the patient was admitted to another hospital because of weakness of the lower limbs and fever.

On clinical examination the patient was afebrile and his vital signs were normal. The physical examination was unremarkable and there was no sign of meningeal irritation. On the neurological examination, the patient was alert and oriented. Superficial abdominal reflexes were absent, muscle strength in the lower limbs was diminished (1/5), and the patient had sensory loss below T6 with bladder and bowel involvement. Hematological and immunological parameters were within normal limits. The patient’s x-ray was normal. Magnetic resonance imaging (MRI) scan showed an abnormal mass formation located between the vertebrae and duramater extending from C7 to T9 (Figures 1, 2). An urgent two level (T6-T7) laminectomy was performed. The laminae had normal appearance and texture. Granulomatous and fibrinous material was present immediately under the ligamentum flavum and there was yellowish liquid pus in the extradural area. No additional pathological findings were observed in the extradural area. Culture of the pus revealed methicillin-susceptible Staphylococcus aureus. Vancomycin (2 gr/day) was administered for eight weeks. MRI scan made one month after surgery showed no pathological findings except the postlaminectomy scar (Figures 3, 4). After an 8-month rehabilitation period, the sensory loss resolved and muscle strength in the lower limbs improved (3/5).

Figure 1: T2-weighted sagittal MRI scan of the case showing posteriorly located epidural mass between C7-T9.

Figure 2: T1-weighted gadolinium-enhanced axial MRI scan of the case showing peripheral enhancement of posteriorly located epidural mass.
DISCUSSION

Spinal epidural abscess presents as a suppurative process localized between the duramater and the vertebral periosteum within the epidural space. Staphylococcus aureus is the most common causative agent.

The microorganism reaches the epidural area by direct inoculation through invasive procedures like a puncture, or a definitive connection between sources of infection near to and distant from the vertebral canal. Hematogenous spread into the epidural space is an important pathogenic factor. In published series, it was possible to identify a distant source in 35% of cases (6). The bacteremia is often unclear, and in some cases it is not possible to identify the source of infection. In this case, we found no source of infection, near or distant from the vertebral canal. Moreover, history or evidence of invasive procedures was not present in this case.

In different series, the predisposing factor could be identified in 6% to 100% of the cases (2, 5, 6, 8). There was no predisposing factor associated with spinal epidural abscess in this case. The patient was a young and healthy adult.

Trauma has been reported in 17%-34.7% of the patients with spinal epidural abscess in large series. Trauma can cause hematogenous spread of microorganisms by penetration of anatomic barriers. However, spinal trauma is especially important since it may create a site of entry for the microorganism into the epidural space. Spinal hematomas associated with severe blunt or penetrating spinal trauma represent an important pathogenetic factor that may cause development of spinal epidural abscess and could also explain the potential development of spinal epidural abscess following epidural hematoma (2, 6, 8). Spinal epidural abscess associated with lifting heavy weights without blunt or penetrating trauma could also be explained by the probable development of epidural hematoma following heavy weight lifting. However, predisposition to the development of epidural hematoma in our case might be suspected. The multiple level involvement of spinal epidural abscess in our case supports the idea of potential development of spinal epidural abscess following epidural hematoma. However, we did not find epidural hematoma in this case. This may be due to resolution of epidural hematoma in the twenty-five day period.

The pre-operative neurological condition, earlier surgical intervention, appropriate antibiotic, and intensive rehabilitation are the determinants for outcome in spinal epidural abscess. However, neurological recovery strongly correlates with the degree of initial neurological dysfunction. In this case, the patient recovered partially from his poor pre-operative neurological condition and was left with residual weakness.

This case presented here is exceptional because of following reasons: 1) there were no predisposing

Figure 3: T1-weighted gadolinium-enhanced sagittal MRI scan of the case one month after surgery showing no pathological findings except the postlaminectomy scar.

Figure 4: T1-weighted gadolinium-enhanced axial MRI scan of the case one month after surgery showing no pathological findings except the postlaminectomy scar.
factors and the patient was a young and healthy adult. 2) no source of infection near to or distant from the vertebral canal could be found. 3) the only tangible co-morbid condition was the patient’s history of lifting heavy weights which may have caused a spinal epidural hematoma

REFERENCES