Spinal Epidural Abscess: Common Symptoms of an Emergency Condition

A Case Report

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SUMMARY – *Spinal epidural abscess (SEA) is a severe pyogenic infection of the epidural space that leads to devastating neurological deficits and may be fatal. SEA is usually located in the thoracic and lumbar parts of the vertebral column and injures the spine by direct compression or local ischemia. Spinal injury may be prevented if surgical and medical interventions are implemented early. The diagnosis is difficult, because clinical symptoms are not specific and can mimic many benign conditions. The classical triad of symptoms includes back pain, fever and neurological deterioration. The gold standard in the diagnostic evaluation is magnetic resonance imaging with gadolinium enhancement, which determines the location and extent of the abscess. Increased awareness of the disease is essential for rapid recognition and immediate implementation of treatment. Here we describe the case of a 26-year-old woman with SEA with fever, back pain in the thoracic region and delayed symptoms of a transverse spinal cord injury.*

Introduction

Spinal epidural abscess (SEA) is a severe pyogenic infection of the epidural space. The rapid accumulation of purulent material in the space between the dura matter and the osseoligamentous confines of the vertebral canal may injure the spine by direct compression¹ or local ischemia^{2,3}. Early medical and surgical intervention is necessary to prevent devastating neurologic deficits, however, the rate of progression of neurologic impairment is difficult to predict^{4,7}. Despite the advances of modern diagnostic and management methods the associated morbidity and mortality remain high⁷.

The diagnosis of SEA is a challenge, largely because of its rarity: it is found in 0.2-2.8 cases per 10000 hospital admissions^{8,9}. Initial clinical symptoms may be misinterpreted, since they are not specific and can mimic many benign conditions. Prompt diagnosis based on neuroimaging procedures is an important determinant of outcome because if proper treatment is not timely implemented irreversible neurologic deficits can develop. Additionally, if the diagno-

sis is delayed, the patient may receive suboptimal treatment⁵. Here we describe the case of a 26-year-old woman with SEA who presented at our hospital with symptoms of fever and back pain in the thoracic region, and further developed lower paraplegia within a few days.

Case Report

A 26-year-old woman with a history of a bronchial asthma and bacterial angina three weeks before admission presented at our Emergency Department (ED) with the complaints of headache and severe back pain in the thoracic region. The pain lasted for three days, intensified on deep breathing, radiated to the left upper extremity and pericardial area and was accompanied by high fever (40°C). Prior to admission she was diagnosed as having intercostal neuralgia and was treated with non-steroid analgesics but without improvement. On clinical examination, the patient was anxious and tearful, but without any deficits in consciousness or mental function. She showed pathological meningeal

signs: stiffness of the neck and Kernig's sign bilaterally. There were no other neurological deficits within cranial nerves, sensory, motor or coordination systems, or any other symptoms of spinal cord injury. Except for an elevated C-reactive protein (CRP) serum level (128 mg/L), laboratory parameters were normal. Contrast-enhanced CT of the head revealed the left maxillary sinus inflammatory exudate, however, the brain tissue was normal. Magnetic resonance imaging (MRI) of the head was not performed in the ED due to the presence of metallic orthodontic braces. Lumbar puncture was performed for suspected meningitis. Cerebrospinal fluid (CSF) pressure and Queckenstedt test were normal. Laboratory CSF examination revealed elevated pleocytosis (36/mm³) and protein level (444 mg/dl), which was suggestive of parameningeal inflammation and/or viral meningitis. Intravenous anti-herpetics and broad-spectrum antibiotics were initiated (acyclovir, third generation cephalosporin and vancomycin). Initially the patient's condition slightly improved and was stable during the first days of hospitalization. After three days' incubation CSF cultures were negative. Up to the fifth day of hospital stay meningeal signs were reduced and there were no symptoms of spinal cord injury. On the sixth day a flaccid lower paraparesis, superficial and deep hypoaesthesia beneath the Th6 segment and sphincter dysfunction appeared. After removing the metallic orthodontic braces spinal cord MRI was performed and revealed the SEA located in the epidural space posteriorly to the spine, extending from the level of vertebrae Th2 to Th5 and compressing the spinal cord (Figure 1). Within the next few hours the patient underwent decompressive laminectomy (level Th4-

Th5) and surgical evacuation of the abscess. The post-operative cultures from the abscess were positive for *Staphylococcus aureus*. Intravenous antibiotics were continued, antiviral medication was stopped and active neurorehabilitation was initiated. Immediate improvement of the paresis and sphincter function was observed as a result of the surgical decompression. Post-operative MRI performed on the eighth day after surgery showed decompression of the spine and local ischemic changes of the spine at the Th2-3 and Th4-5 levels (Figure 2). The follow-up MRI on the 65th day after surgery revealed localized swelling of the spinal cord with a hyperintense signal suggestive of past focal ischemia and/or mechanical injury. Nine weeks after surgery the patient could walk with minor assistance and was discharged home with minor lower paraparesis and with no sphincter dysfunction or sensory loss.

Discussion

Spinal epidural abscess (SEA) is still considered a rare condition, but the incidence of SEA has doubled in the past two decades⁵. The mortality in SEA dropped from 34% in the late 1950s to 15% in the 1990s⁷, although it is still estimated to range from 2% to 20%⁷. Regardless of the availability of advanced diagnostic and neurosurgical procedures, diagnosis of SEA remains challenging. To quote Grobovskhek: "the problem with spinal epidural abscess is not treatment, but early diagnosis – before massive neurological symptoms occur"¹⁰.

Identification of the risk factors may help in the early establishment of the diagnosis. The

Table 1 Types of risk factors and examples of sources of infection in patients with spinal epidural abscess (modified after Reih-saus et al. ⁹).

Risk factors and sources of infection	Prevalence (%)
<i>Localized or systemic infections:</i> Skin and soft tissue abscesses; Vertebral osteomyelitis/discitis; Paraspinal infection; Pulmonary/mediastinal/urinary infections; Sepsis; Endocarditis; Pharyngitis; Others	44
<i>Immunodeficiency in the course of:</i> Diabetes mellitus; Intravenous drug abuse; Alcohol abuse	29
<i>Invasive procedures:</i> Epidural anaesthesia; Extrapinal/spinal surgery; Vascular access; Corticosteroid injections; Paravertebral injections; Others	22
<i>Trauma:</i> Extrapinal/spinal trauma	10
<i>Disorders of different organs, systems or body regions:</i> Chronic spondylosis; Chronic renal insufficiency; Colitis ulcerosa/Crohn's disease; SLE; Pregnancy/Delivery; Malignancy; Others	10

Figure 1 T1-weighted, fat-suppressed, gadolinium enhanced magnetic resonance images of the thoracic spine in a 26-year-old woman. Axial (A) and sagittal (B) images show posterior spinal epidural abscess at the Th3 to Th6 segments (arrows). Maximum measured thickness of the abscess was 8.0 mm at the level of Th3/Th4 vertebral bodies, whereas the spine in the narrowest region was 2.0 mm thick.

Figure 2 Post-operative magnetic resonance images of the thoracic spine in a 26-year-old woman. Sagittal T1-weighted, fat-suppressed, gadolinium enhanced (A) and sagittal T2-weighted (B) images show sufficient decompression of the spine after abscess evacuation and a local pathological signal intensity within the spine at the Th2-3 and Th4-5 levels (arrows) suggestive of a past local ischaemic process.

most common risk factors of SEA are listed in Table 1, though it is estimated that almost 20% of cases are idiopathic⁵. The leading comorbidities are localized or systemic infections, often with coexisting immunodeficiency e.g. in the course of diabetes mellitus¹¹. In half of SEA cases bacteria reach the epidural space by haematogenous dissemination, where the primary sources are skin, soft tissue, urinary or respiratory tract infections. In 10-30% of cases SEA develops by direct expansion from adjacent tissues, e.g. in the case of a vertebral osteomyelitis or psoas muscle abscess. Neurosurgical interventions, lumbar puncture, epidural an-

algnesia and other invasive procedures are estimated to be responsible for about 15% of SEA¹¹. Bacterial angina was probably the source of infection in our patient, but we also identified a predisposing factor such as compromised immune system caused by chronic glucocorticoid intake for bronchial asthma treatment.

As SEA is well separated from the surrounding tissues, identification of the exact pathogen may not always be possible from the blood or CSF cultures. *Staphylococcus aureus* is responsible for about 70% of SEA cases^{5,7,11-13}. Infection by methicillin-resistant *S. aureus* (MRSA) is particularly high in patients with implant-

able spinal or vascular devices. Other less common agents are: *Staphylococcus epidermidis*, *Escherichia coli* (in particular in patients with urinary tract infection), *Pseudomonas aeruginosa* (in injection-drug users), anaerobes, mycobacteria, fungi and parasites⁵. *S. aureus* was responsible for the development of SEA in our patient, but it could be confirmed only after surgery, not by CSF examination.

In managing the SEA patient, prompt diagnosis is crucial to achieve a favourable outcome. A non-specific clinical picture may result in diagnostic difficulties. Four stages may be identified in SEA development: 1) back pain at the level of the affected spine, fever, spine tenderness; 2) radicular pain radiating from the affected part of spinal cord, nuchal rigidity, hyper-reflexia; 3) neurological deficits such as hypoaesthesia, motor weakness, bowel or bladder dysfunction; 4) paralysis^{2,5,11}. The rate of progression from one stage to another and the duration of symptoms vary from a few hours to several days. The sudden development of the neurological deficit in our patient was delayed about a week after the occurrence of thoracic back pain.

The location of SEA defines the clinical picture. It predominantly locates in the thoracic and lumbosacral regions^{7,8,11,12}, may extend throughout three to four vertebrae, but it may also involve the entire vertebral column. Abscesses located anteriorly to the spine usually coexist with osteomyelitis^{11,13}. The classical triad of symptoms includes: back pain, fever and neurological deterioration, but is present only in 10-15% of patients at first contact¹¹. As severe back pain is the most common symptom¹², every patient with back pain, fever and predisposing risk factors should be evaluated as suspected of SEA¹². Common conditions which should be differentiated at first clinical evaluation are: disc prolapse, degenerative joint disease, demyelination, spinal haematoma or tumour, osteomyelitis, discitis, meningitis, urinary tract infection or endocarditis⁵. In our patient SEA occurred in the thoracic region, but was limited only to two to three vertebrae.

Different mechanisms are suggested to be responsible for the spinal cord lesion such as: direct compression by an extending epidural abscess, an ischaemic process associated with the compression of spinal arteries and/or veins, or indirectly, by septic thrombophlebitis^{3,14}. These factors may occur at different times of the disease and usually cause additive adverse effects. Detailed pathogenesis of spinal cord injury remains uncertain. Although the surgical

decompression immediately improved the clinical condition of the reported patient, follow-up MR imaging confirmed the local ischaemic changes to the spinal cord probably caused by compression (Figure 2). The Th4 cord level is known to have the poorest blood supply called a watershed region, which could cause some residual postoperative neurological deficits. As the compression of the spinal cord in our patient was mainly from the dorsal side, isolated paraparesis as a sequela without dorsal column symptoms implies an additional mechanism of injury. The post-operative MRI demonstrated cord swelling with T2 signal elevation at the Th2-3 and 4-5 levels, which may match with that of venous infarction of the spinal cord. We may suspect, that not solely due to compression or ischaemia, but also phlebitis in the spinal cord could participate in the injury.

The diagnosis of SEA should be based on neuroimaging studies. The gold standard of imaging diagnostics in SEA is MRI with gadolinium, having a sensitivity and specificity higher than 90%¹⁵⁻¹⁷. MRI determines the extent of the abscess in both longitudinal and axial planes, which is essential for planning surgery (see Figure 1). It may also help to differentiate infection from neoplasms. The delay with spinal cord MRI is unacceptable if the procedure is available. We could not perform MRI on admission because of the presence of metallic orthodontic braces. However, on admission there were no symptoms of spinal cord injury and also no pathological results in lumbar puncture suggesting spinal canal stenosis. Consequently, within first days we did not urge the removal of orthodontic braces for MRI examination. This delay could have contributed to worsening the patient's condition.

Imaging findings may be supported by clinical and laboratory data. Markers of inflammation like: leukocytosis, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR) may be elevated, but none of these is specific for SEA. Elevation of protein level and/or pleocytosis in the CSF examination may be suggestive of parameningeal infection, but CSF cultures are positive in less than 25% of patients⁵. Nevertheless, lumbar puncture is not required to confirm SEA, and it increases the risk of meningitis or subdural infection if the needle traverses the abscess. Blood cultures may remain negative in about 40% of cases⁸. Poor prognosis is recognized to be associated with elevated levels of ESR, CRP, leukocytosis or thrombocytopenia on admission^{13,17}.

The only effective therapy for SEA is evacuation of the abscess and microorganism eradication. Decompressive laminectomy and debridement of infected tissues is the method of choice. Surgery should be performed as soon as possible^{4,7,11,13} and followed by specific culture-guided intravenous antibiotics for four to six weeks. The final neurological outcome corresponds strongly with the duration and the degree of neurological deficit prior to surgery. Patients in stage 1 or 2 may have a full recovery, patients in stage 3 may have no weakness or a lesser degree of weakness, whereas patients in stage 4 may benefit from surgery only if they undergo decompression in 24-36 hours after the onset of neurological symptoms. Nevertheless, even timely surgical intervention does not guarantee full recovery.

Surgery may be withheld in specific conditions such as patient's refusal, high operative risk, paralysis for more than 24-36 hours and panspinal infection. In these cases antibiotic

therapy must be guided by the results of blood cultures or CT-guided needle aspiration. In neurologically intact patients with identified pathogen conservative treatment with intravenous antibiotic administration is allowed, although they need careful monitoring. Empirical therapy should provide coverage against staphylococci (including MRSA), streptococci and Gram-negative bacilli (e.g. intravenous vancomycin and third or fourth generation cephalosporin)⁵.

The treatment is successful if follow-up MRI in four to six weeks after therapy reveals changes only in soft tissue.

In conclusion, early intervention improves the prognosis in patients with SEA. Despite the advances of modern diagnostic and management methods, about 30% of patients with SEA still do not have a good outcome⁷. Increased awareness of the disease is essential for rapid recognition and immediate introduction of treatment.

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