Spinal epidural abscess

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ABSTRACT

Spinal epidural abscess is an uncommon condition. Predisposing factors include spinal surgery, recent trauma, immunosuppression, a distal site of infection and intravenous drug use; however, these are not always present, as illustrated by this case report describing a patient who had repeated emergency department visits and delayed diagnosis that was ultimately confirmed via magnetic resonance imaging.

Key words: spinal epidural abscess; urinary retention; back pain

RÉSUMÉ

L'abcès rachidien épidural est une atteinte peu courante. Les facteurs prédisposants sont la chirurgie rachidienne, un traumatisme récent, l'immunosuppression, un site distal d'infection et l'usage de drogues intraveineuses; cependant, ces facteurs ne sont pas toujours présents, comme le démontre la présente observation d'un patient ayant visité le département d'urgence à plusieurs reprises et chez qui le diagnostic fut finalement confirmé au moyen de l'imagerie par résonance magnétique.

Introduction

Spinal epidural abscess (SEA) occurs at an incidence rate of 0.22.8 cases per 10 000 hospital admissions.[1–4] Predisposing factors include spinal surgery, recent trauma, immunosuppression, a distal site of infection and intravenous drug use;[1–3,5–7] however, 20% of patients will have no clear predisposing factor.[2] The typical symptoms of back pain and fever are common daily presenting complaints in the emergency department (ED); therefore the condition is easily missed if physicians fail to consider it in the differential diagnosis. Multiple ED visits and diagnostic delays occur in 68% of patients, contributing to a 45% morbidity rate and 15% mortality.[1,7] We describe a patient with no significant predisposing factors who presented with the unusual chief complaint of urinary retention.

Case report

A 63-year-old woman presented to the ED with a 3-week history of urinary retention, abdominal pain, back pain, bilateral leg weakness, sweats and facial flushing. She had been seen in the ED twice during the preceding 3 weeks with similar symptoms. On these occasions she was discharged after urinary catheterization with an unexplained diagnosis of urinary retention. Her lower-back pain was worse on the right side and radiated as a burning sensation to her lateral thighs. Her medical history included hyper-

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tension, osteoarthritis, hiatus hernia repair, left knee arthroscopy, appendectomy, cholecystectomy and hysterectomy. She denied smoking, alcohol consumption, drug abuse, trauma, immunodeficiency, spinal surgery or dysuria. Medications included ramipril, valdecoxib and cyclobenzaprine.

On examination, she was conscious, alert and oriented, with a blood pressure of 179/103 mm Hg, pulse of 88 beats/min, respiratory rate of 16 breaths/min and temperature of 36.2°C. Physical examination revealed moderate obesity as well as tenderness over the lower spine and left para-lumbar region. Neurological examination revealed brisk deep tendon reflexes, proximal left leg weakness, decreased pinprick sensation in the left leg, and a left-sided sensory level at T7–T8. Rectal tone and sensation were normal, as was the remainder of her examination. The initial clinical impression was neurogenic bladder secondary to a cauda equina syndrome.

Her white cell count was 28.6×10^9/L. Serum chemistry and coagulation parameters were normal. Urinalysis was positive for leukocytes, blood and nitrates, and the urine culture later grew *Staphylococcus aureus*. An urgent magnetic resonance imaging (MRI) study of the thoracic and lumbar spine was performed, showing an extensive multiloculated epidural abscess encircling the thecal sac from T9 to L3, causing mild to moderate cord compression. Cord changes were also apparent in the lower thoracic area, and there was a paraspinal collection at L2–4 on the right side (Fig. 1).

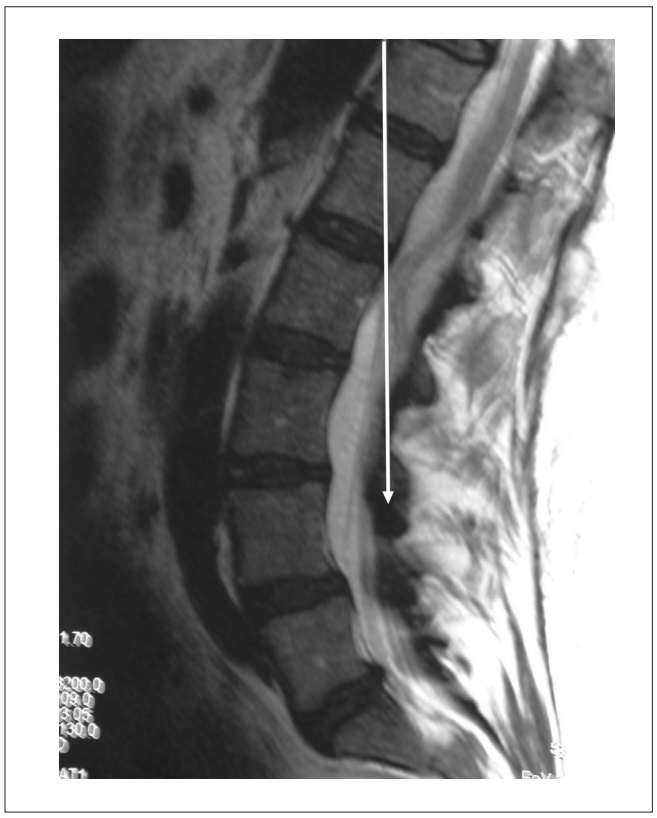

Fig. 1. MRI study of the thoracic and lumbar spine showed, among other things, a paraspinal collection at L2–4 on the right side.

The patient was started on intravenous vancomycin and metronidazole, and admitted to the neurosurgery service. Blood cultures and aspirates from the paraspinal abscess both demonstrated *S. aureus*. Initially a non-operative approach was taken, but by day 6 there was minimal improvement and surgical decompression was performed. The patient did well postoperatively and was discharged on oral cloxacillin for 1 month. She remained stable, with no residual deficits at follow-up.

Discussion

Spinal epidural abscess is uncommon, but its incidence has been rising since the 1980s, reflecting increases in spinal surgery, a rising injection drug abuse problem and a growing number of immunocompromised patients.[2] Previous literature suggested that SEA was most prevalent during the 6th and 7th decades;[2,7] however, a recent review demonstrated that it is most common between the ages of 30 and 60, with a preference for males at a ratio of almost 2:1.[1] *Staphylococcus aureus* is the primary causative organism, although gram-negative bacteria and other opportunistic organisms occasionally cause these infections (Table 1).[1,2,3,5,8] Table 2 summarizes the SEA risk factors described above.[1]

Pathophysiology

The epidural space is a continuous vertical casing located outside the dura mater and within the wall of the bony vertebral canal. This space is normally occupied by fat, areo-

Table 1. Causative organisms in cases of spinal epidural abscess[5,12]

Organism	Frequency, %
Staphylococcus aureus	63
Methicillin-resistant *S. Aureus*	15
Gram-negative bacilli	16
Streptococci	9
Coagulase-negative staphylococci	3
Anaerobes	2
Others	1
Unknown	6

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lar tissue and a network of veins (Fig. 2).[6,9] Anterior to the spinal cord, the dura adheres to the vertebral bodies, leaving only a potential space. As a result, 80% of spinal epidural abscesses lie posteriorly. Within the epidural space, longitudinal extension is common; abscesses often involve 3 to 5 spinal cord segments and can affect the whole length of the spinal cord. Spinal epidural abscess most often starts in the thoracic (50%), followed by the lumbar (34%) and cervical regions (15%).[2,5,8]

The effects of SEA progress locally, from reversible neurological deficit and destruction within the spinal cord, to permanent central nervous system dysfunction and death. The underlying pathophysiologic mechanisms are poorly understood, because neurologic dysfunction is often disproportionate to the degree of spinal cord compression.[1,2,6] Previous research has led to the hypothesis that edema and inflammation involving the cord and epidural space eventually involve the epidural venous plexus, compromising the circulation and causing cord ischemia.[1,2,6] Most likely, the synergistic effects of compression and ischemia are responsible for the neurological deterioration seen with SEA.

Clinical diagnosis

Spinal epidural abscess is characterized by the classic triad of localized back pain, neurological deficits and abnormal inflammatory parameters (fever, leukocytosis and elevated erythrocyte sedimentation rate);[1–7] however, a recent study demonstrated that the "classic triad" was present in only 8 (13%) of 63 SEA patients. Most of these patients suffered diagnostic delays as a result[10] and two-thirds required multiple ED visits (from 1 to 8) before a diagnosis was made. During these delays, 57% experienced further neurological deterioration,[10] and residual motor weakness was 3 times more likely (45% v. 13%) in patients with diagnostic delays.[10]

Clearly, diagnostic delay can have a tragic affect on outcome. One-third of SEA patients suffer permanent muscle weakness, incontinence and sensory deficits. Paralysis and death occur in 16% and 15% of patients, respectively. Danner and Hartman reported that only 39% of 188 SEA patients made a full recovery if treatment was delayed more than 36 hours after the onset of motor weakness.[11]

Because the prognosis for complete recovery is poor once neurological deficits are present,[10] the diagnostic value of the "classic triad" has been challenged. Dependence on it may lead to diagnostic delays, multiple ED visits and adverse neurological outcomes like those described above. Instead, Davis and colleagues recommend using risk factors to screen ED patients for SEA, since they are more sensitive than the classic diagnostic triad (98.4% v. 7.9%).[10] These authors also note that the erythrocyte sedimentation rate is more sensitive and specific than the total white blood cell count.[10] It is important to point out that the most important step in diagnosing SEA is consideration of the entity.[1]

Imaging

Radiological modalities are the basis for SEA diagnosis. Magnetic resonance imaging, with 91% sensitivity, is now considered the diagnostic gold standard, although this is based largely on case reviews and retrospective studies.[1,3] Magnetic resonance imaging, especially with gadolinium

Table 2. Predisposing factors for spinal epidural abscess[1]

Predisposing factors	Frequency, %
Infections	44
Skin abscess	
Vertebral osteomyelitis/discitis	
Pulmonary/mediastinal infections	
Sepsis	
Immunodeficiency	34
Diabetes mellitus	
Intravenous drug use	
Alcoholism	
Chronic renal failure	
AIDS	
Spinal procedure or surgery	22
Trauma	10
No predisposing factors	20

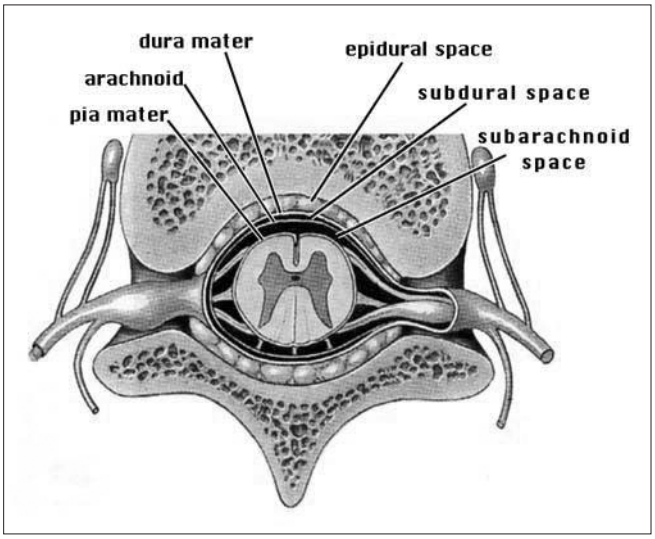

Fig. 2. The epidural space is a continuous vertical casing located outside the dura mater and within the wall of the bony vertebral canal. This space is normally occupied by fat, areolar tissue and a network of veins.

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enhancement, is superior to computed tomography (CT) because it better delineates SEA from contiguous structures and differentiates SEA from spinal tumours, hematomas, transverse myelitis, spinal cord infarction and intravertebral disk prolapse.[1] Lumbar puncture is now regarded as an unnecessary procedure likely to increase the risk of spreading the infection to the subarachnoid space.[1]

The case we have discussed here illustrates several of the problems described above. Spinal epidural abscess was not considered in the differential diagnosis until the patient's 3rd ED visit and, therefore, diagnostic imaging was delayed. Her chief complaint of urinary retention did not fit the classic triad; nor did she have relevant risk factors. In fact, her symptoms were more suggestive of a cauda equina syndrome. Urinary retention, with or without overflow incontinence, is the most common finding in cauda equina syndrome, with a sensitivity of 90% and a specificity of 95%.[12] Cauda equina syndrome is similarly associated with bilateral weakness of the lower extremities, sensory deficits and gait disturbance.[10] Despite the potential for diagnostic confusion, a MEDLINE search revealed no literature describing the overlap and differentiation of cauda equina syndrome and SEA.

Therapy

Rapid treatment improves outcomes. When SEA is clinically suspected, even prior to diagnostic imaging, immediate ED administration of dexamethasone is recommended to minimize the progression of compressive edema, cord ischemia and neurological damage.[13] Parenteral antibiotics effective against staphylococci, streptococci and Gram-negative bacilli should be initiated as early as possible. Appropriate initial empiric regimens include cloxacillin with metronidazole and either cefotaxime or ceftazidime.[1,5] Vancomycin can be substituted for cloxacillin in cases of penicillin allergy or if methicillin-resistant *S. aureus* is considered likely.[1,5] The duration of antibiotic administration is usually 4–6 weeks but can be up to 12 weeks.[1,8] Traditional surgical treatment consists of decompressive laminectomy and surgical debridement.[1,2,5,7,8]

Pediatric perspectives

Spinal epidural abscess is rare in children. Unlike adults, only 35%–38% of children have a predisposing medical condition, most often sickle cell anemia or immunosuppression from chemotherapy or malignancy.[14] Clinical presentation, bacteriology, diagnostic approach and treatment strategies are otherwise similar to adults. Fortunately, 75%–85% of children with SEA have a favourable outcome.[14]

Conclusion

Spinal epidural abscess is a rare disease, usually associated with specific predisposing factors; however, patients may present atypically. Diagnostic delays lead to adverse neurological outcomes. Magnetic resonance imaging is now the diagnostic procedure of choice, if available. Emergency department management should include early administration of dexamethasone and appropriate parenteral antibiotics.

Competing interests: None declared.

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