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CASE REPORT

Spinal epidural abscess: report of five cases

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ABSTRACT

Background: Spinal epidural abscess (SEA) is a rare disease, associated with high morbidity and mortality. Recently, incidence of the disease is increasing because of increased predisposing factors.

Patients: Five cases with spinal epidural abscess admitted with neurologic deficits are reported here. Of these, one was an IV-drug abuser while in another case osteomyelitis resulted from tuberculosis. No source of infection was identified in other cases. All patients underwent urgent decompressive multilevel laminectomy followed by antibiotic therapy based on blood culture results. Finally, 3 patients could ambulate independently while the other two patients were left without improvement in neurologic status.

Conclusion: SEA is a devastating condition if not diagnosed and treated promptly and effectively. Preoperative MR imaging provides a noninvasive, accurate means of diagnosis in this condition. Recent advances in the surgical treatment of SEA have resulted in the increased use of less-invasive surgical techniques, thereby decreasing the morbidity associated with surgery in both the short and long term.

Keywords: *Spinal epidural abscess, Neurologic deficits, Laminectomy.*
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INTRODUCTION

Spinal epidural abscess (SEA) is a rare disease (1-13), but its high morbidity and mortality overcome its rarity (1,13,14). The classic manifestation of the disease is triad of fever, back pain, and progressive neurological deficit (1,7). SEA accounts for 0.18 to 2 cases per 10,000 hospital admissions (2-6,11,13,15). Though uncommon, it seems that the incidence of this problem has increased in the past years (3,4,6,10-15).

SEA represents the accumulation of purulent material in the space between the dura matter and the osseo-ligamentous confines of the vertebral canal (15). Despite advances in diagnostic imaging modalities, surgical techniques and antimicrobial therapy, SEA is still associated with a high morbidity and mortality varying from 4.6% to 31% (1,5,7,10-15) and remains a challenging problem that often eludes diagnosis and receives suboptimal treatment (3,7). Almost half of the survivors are left with neurologic deficiencies, including 15% with paresis or complete paralysis. Meanwhile, almost 90% of SEA patients are left with residual weakness, when motor deficiencies are present at the time of diagnosis (1).

Several risk factors have been reported to play a role for SEA, including endocarditis, use of epidural steroid before and during discectomy (9), use of immunosuppressive (2,9), aging population

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(11,15), a rise in the number of invasive spinal procedures (3,11,15), and growing numbers of intravenous drug abusers patients with human immunodeficiency virus (HIV) infection (11,15).

The most common way of infection is hematogenous spread with either direct seed to the epidural space or to the vertebral body with extension to the epidural space (3,6,7), however, primary site of infection remains undetected in up to 50% of the cases (6). Prompt diagnosis is the most important determinant of outcome (1,4,5,10, 11,15). If a delay in diagnosis and therapy occurs, prognosis is worsened (1,4,10). If prompt treatment is not instituted, irreversible neurologic deficit can and often does develop (11,15). Treatment usually involves surgical decompression with or without drainage of the abscess, followed by prolonged antimicrobial therapy. In this study, we present five cases with SEA.

PATIENTS

In a period of 5 years**,** five patients with acute SEA admitted to our hospital (table 1). There were 4 men and 1 woman, their age ranged from 14 to 50 years (28.4±13.3 years). IV-drug abuse and osteomyelitis were noted as major risk factors, each in one patient, while the other 3 patients presented without any identified risk factor.

SEAs were located in cervicothoracic (C2-T3) (figure 1), thoracic (T2-T6), thoracolumbar (T12-L3) and lumbar regions (L1-L5) (figure 2 and 3a,b).

Clinical presentation: All patients presented with pain around SEA area as the first complaint lasting several days to weeks. Meanwhile, all showed an acute or subacute deterioration in motor function occurred quickly within few hours, however, in 2 patients progressive neurological deficit resulted in quadriparesis (cases No: 1 & 2), in one resulted in paraparesis (case No: 3), in the other resulted in complete paraplegia (case No: 4), and, finally, in case 5 resulted in severe lower extremity weakness and loss of bladder control. Slight and progressive fever was noted in cases No 1 and 3.

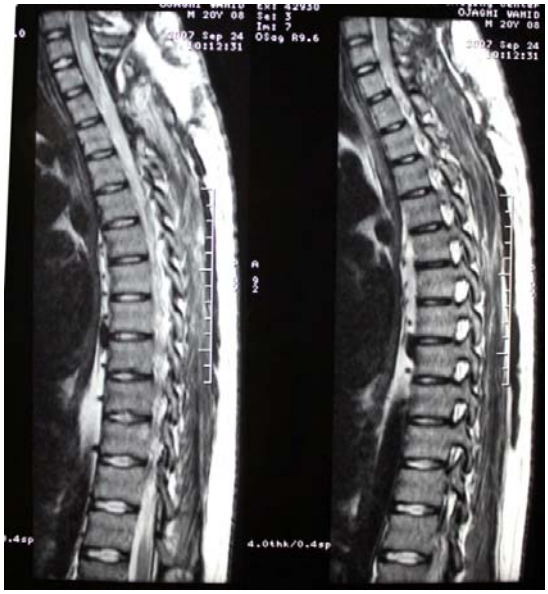

Figure 1. Cervical spinal epidural abscess. Sagital Gd –T1weighted MR images demonstrating an extensive heterogenous epidural collection

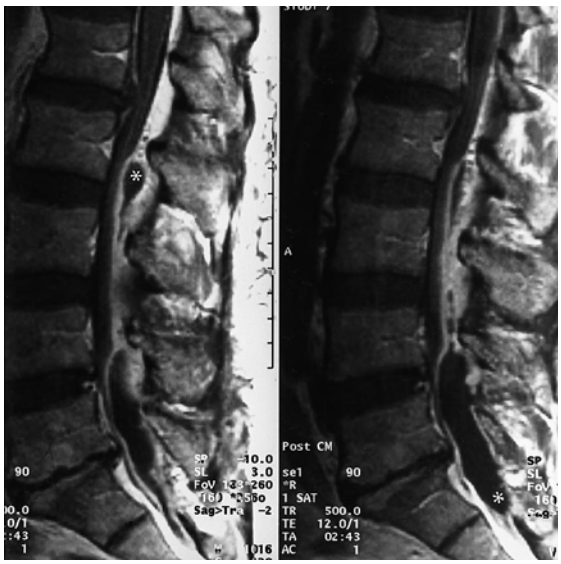

Figure 2. A spinal epidural abscess in thoracolumbar region is seen in sagittal T1-W MRI. Note epidural signal enhancement from T12 –L3. In this case the enhancement was in parts uniform and in other parts patchy. The implication was that the uniformly enhancing portions were liquid pus, with more solid phlegmon in the heterogeneous regions

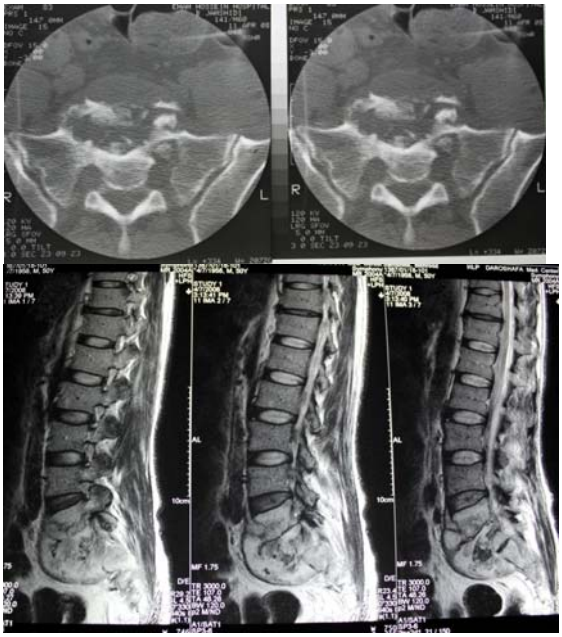

Figure 3. a, CT scan images of a patient with lumbar spinal epidural abscess which resulted from infection with TB that shows bone destruction; **b,** T1-W MRI of the same patient shows vertebral destruction and signal enhancement in lumbar region.

Diagnosis: Magnetic resonance imaging (MRI) with and without gadolinium was used to confirm the diagnosis. Imaging study showed mass in epidural area caused spinal cord compression.

ESR was elevated in all patients, ranged 60 to 82 mm/h. Culture obtained from the abscess showed *Staphylococcus aureus* and *Mycobacterium tuberculosis* as the causative agents in 4 and 1 cases, respectively. C-reactive protein (CRP) was positive in all while leukocytosis was found in tow cases (cases No. 3 and 5).

Management: All patients underwent surgery under general anesthesia, since they had progressive neurologic deficit. Multilevel decompressive laminectomies and abscess drainage via posterior approach were achieved. Postoperatively, patients (cases No. 1-4) received intravenous vancomycin for 6 weeks in addition to cefepim (case No. 1) and ceftazidime (cases No. 2-4), followed by oral ciprofloxacin for another 8 weeks.

Table 1. Clinical characteristics of patients with spinal epidural abscess

Case	Age/ Sex	Symptoms	Level of location	Microbiology	Management	Antibiotic therapy/duration	Follow up (month)	Outcome
1	28/F	Pain, fever, quadriparesis	C2-T3	*S. aureus*	Laminectomy C2-T3	IV vancomycin and cefepim/6W oral ciprofloxacin/8W	3	Resolution of epidural change, no improvement in neurologic status
2	14/M	Pain, slight fever ,quadriparesis	T2-T6	*S. aureus*	Laminectomy T1-T5	IV vancomycin and ceftazidime /6W oral ciprofloxacin/8W	6	Excellent neurologic recovery, independent walking, epidural collection
3	28/M	Pain, fever, paraparesis	T12-L3	*S. aureus*	Laminectomy T10-L4	IV vancomycin and ceftazidime /6W oral ciprofloxacin/8W	3	Paraplegia
4	22/M	Pain, complete paraplegia	L1-L5	*S. aureus*	Laminectomy L1-L5	IV vancomycin and ceftazidime /6W oral ciprofloxacin/8W	3	Complete cord decompression, recovery from paraparesis
5	50/M	Pain, lower extremity weakness, loss of bladder control	L1-L5 and T6-T10	*M. tuberculosis*	Laminectomy L1-L5 and T6-L1	Isoniazid, rifampin/12M	3	Slow neurologic recovery, independent walking

Case No. 5 underwent multilevel laminectomy from L1-L5, then observed postoperatively till symptoms exist. Further evaluation with MRI showed another abscess compressing the spinal cord in thoracic region (T6-T10) for which second multilevel laminectomy was scheduled. He received isoniazid and rifampin for 12 months, postoperatively.

Outcome: Postoperatively, patients were followed for 3 months, except case No. 2 who was visited for 6 months. We assessed the outcomes using previous laboratory tests and imaging studies. In all patients, inflammatory markers fell to normal except case No. 5. Two patients (cases No:1 and 3) left unable to walking following the surgery because of permanent spinal cord injury but the other 3 patients ambulated independently and neurologic deficits resolved postoperatively.

DISCUSSION

Although SEA is a rare disease, but its high mortality (4.6% to 31%) and morbidity necessitates finding appropriate diagnostic and management methods. This disease bears tendency toward men whose age above 30 years. Reihsaus et al. observed in their study that 70% of cases with SEA aged from 31 to 70 (14).

Pathogenesis: The etiology of SEA is not obvious but Phillips et al. demonstrated that there were several possible sources of infection (8). Most patients with SEA have one or more risk factors, either systemic such as diabetes mellitus, end-stage renal disease, alcoholism, infection with human immunodeficiency virus (HIV), septicemia, malignancy, morbid obesity, long-term corticosteroid use, intravenous drug abuse, a distant site of infection, and an indwelling catheter, or local such as a spinal degenerative joint disease, trauma, surgery, drug injection, or placement of stimulators or catheter (4,8,10,15,16). In about 20% of patients, no predisposing condition can be identified (15).

Abscess may form in the epidural space through two important mechanisms: 1) hematogenous spread, 2) direct extension from an infected contiguous structure (4,10,15,16). Although sometimes formation is iatrogenic (15) or a complication caused by a spinal procedure results in abscess formation (10).

Hematogenous spread: Infections which are in another portion of body may contaminate the epidural space via the blood circulation. The skin and soft tissue are the most common source of infection, accounting for 15–25% of reported cases. Abscesses in posterior epidural space are formed by this mechanism generally (15).

Direct extension: Infection near the spinal canal may spread to the epidural space. This mechanism usually results in producing abscesses near the site of osteomyelitis in anterior portion of canal (15). However, Lu et al found that the mechanism which produces the abscess has no effect on prognosis (10).

Most common organism causes this problem is *Staphylococcus aureus* (3-5,8,10,15- 17) followed by *Streptococcus species* (3,15,16). This could be in part explained by the fact that most risk factors allow for invasion through risk flora. Masanovu et al. found that hyperhydrosis is a potential risk for epidural abscess, since sweat and sweat glands provide appropriate environment for organisms to reside (8). However, the source of infection remains undiagnosed in 50% of SEA patients (12).

Pathophysiology of neurologic compromise: The pathophysiologic basis of the neurologic deficits in patients with SEA is obscure (15). One explanation is that epidural abscess injures the spinal cord by mechanical compression or by vascular occlusion resulted from septic thrombophlebitis (9,15,16). Indeed, in many patients decompression of spinal cord elicited neurologic deficits. Nevertheless, the quick progression of symptoms and irreversibility of them may be due to vascular occlusion (15).

Clinical manifestations: SEA has a variable clinical presentation (15). Early common signs and symptoms of SEA are nonspecific and are like many other conditions (11). The main triad of SEA are back pain, fever and progressive sensory and motor deterioration (3,4,8-10,14, 15). Occasionally the backache is misdiagnosed as a bad posture pain (8). The pain may develop within days or weeks but neurologic deficits progression is rapid. In many of these patients, especially those with cervical involvement, diagnosis is very difficult since fever or neurologic deficits are absent (3-6). This leads to initially misdiagnosis in about half of the SEA patients (3). It is necessary to state that the classic triad is seen in only 8-37% of these patients (15). SEAs are seen more commonly in lumbar region followed by thoracic and cervical regions (4,5,10). Some authors found that the outcomes of cervical abscesses are worse than others but Lu et al. found no difference in outcomes of abscesses in different regions (10).

Heusner provided a staging classification for SEA which is generally accepted. In stage 1, patient has severe back pain, fever and local tenderness; in stage 2 radicular pain, fever and neck rigidity manifests, while in stage 3 progressive sensory and motor deficits initiate and patients in stage 4 will experience paralysis (1,10-12,14,17).

Diagnosis: In cases suspected of suffering from SEA, diagnosis is made by physical examination, laboratory tests and imaging studies. The options that should be considered in physical examination, were presented above briefly, hence, laboratory tests and radiographic assessments will be discussed in summation.

Laboratory tests: For diagnosing SEA in suspected patients, infection related indices should be assessed. These indices are white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), and the C-reaction protein (CRP) level (15).

Prior investigators found that WBC count is within normal limit in many of patients with SEA, thus, this index is not a reliable, strong marker of presence of SEA (5,14). However, Lasker et al. found that 15% of patients with SEA had a WBC count less than 10,000/mm^3 (4). Also, Rigamonti et al. reported that approximately 40% of patients with SEA had normal WBC count (normal range: 4500-11000/mm^3) (14).

ESR increase is a more reliable and sensitive indication of abscess formation in epidural space than WBC count (1,5,15). However, ESR increases in many of diseases and conditions such as infectious problems, malignancies, rheumatological and autoimmune conditions, though is not a specific test. Only two studies documented use of ESR as a specific test for SEA (1). In patients with SEA, CRP level returns to normal level earlier than ESR.

To determine the causative pathogen, blood culture and sometimes urine analysis are required. However, approximately in 40% of patients with SEA, the blood culture remains negative (15).

Plain radiography is not efficient and appropriate tool for detection of SEA, but it may be useful to evaluate the presence of osteomyelitis or fracture in vertebrae (1,12). Both gadolinium-enhanced MRI and myelography followed by computed tomography (CT) of the spine are highly sensitive (more than 90%) in diagnosing spinal epidural abscess, but currently computed tomography after myelography is used only when MRI has contraindication (12,14,15). Nevertheless, MRI is the imaging method of choice because it is less invasive, delineates both the longitudinal and paraspinal extension of the abscess (which is essential for planning surgery), and may help differentiate infection from cancer on the basis of the appearance and the signal intensity of the image (5,8,10,12,14,15,17).

Radionuclide scanning using technetium, gallium, or indium may show increased uptake, helping to identify the affected site, however, has little role in diagnosis of SEA (15).

Treatment: Treatment of spinal epidural abscess has three major goals including return to normal neurologic status, prevention of progression of disease, and enabling the patient to obtain independence in ADL (Activity of Daily Living) (14). Due to the rare occurrence and serious outcome of spinal epidural infection, it is both impractical and ethically difficult to conduct prospective, randomized clinical trials to determine the optimal treatment. Treatment of SEA is controversial, however, the majority of retrospective studies provide support for the overwhelming consensus that surgical decompressive and drainage associated with systemic antibiotics is the choice of treatment (2-7, 10-16). Indications of surgery in patients with SEA are spinal cord decompression, histologic evaluation and microbiologic culture, and vertebral stabilization (12).

Patients who are neurologically intact may also qualify for nonsurgical therapy if the microbial cause is identified and the patient's clinical condition is closely monitored. Although controversial, this approach may be reasonable especially when the radiologic epidural abnormality and the symptoms can be explained by findings that the inflammation is not caused by a true abscess (8,10,12,16). Literature includes a few good results from only antibiotic therapy in patients for whom surgery was contraindicated (5). Curry et al. demonstrated that although in some patients antibiotic therapy alone may be efficient, urgent surgical treatment is the best (14).

Prompt diagnosis and treatment are important factors to gain best results, however, preoperative neurologic status is the most important predictor of the final outcome, and progression of the neurologic deficit is easily predictable, with some patients become paralyzed within hours after the onset of neurologic deficit, decompressive laminectomy and debridement of infected tissues should be done as soon as possible (2,3,5,6,10,11, 14-16). Khanna et al. demonstrated that only 33% of patients with plegia and 45% of patients who had paresis got better after surgery (5). Gonzales et al. found that patients with paralysis have worse outcome than others (3).

Figure 4 shows an algorithm for treating patients diagnosed as SEA. In clinical scenarios in which decompressive laminectomy is declined by the patient, contraindication because of high operative risk, or is unlikely to recover from paralysis that is presented for more than 48-72 h which is irreversible, or considered impractical because of paraspinal infection, patients may be treated medically, however, surgery may still be required to treat the epidural infection and control sepsis (10,12,14,15).

Before exact diagnosis, patients suspected to suffer from SEA must receive empirical antibiotic therapy which should provide coverage against *staphylococci* (usually with vancomycin to cover MRSA) and, because of the potentially serious consequences, gram-negative bacilli (potentially with a third- or a fourth-generation cephalosporin, such as ceftazidime or cefepim), particularly in the presence of documented or suspected gram-negative bacterial infection of other sites, such as the urinary tract (15), but when the diagnosis is established, antibiotic therapy must be guided by the results of blood cultures or CT-guided needle aspiration of the abscess. Because vancomycin is less active than β-lactam agents against methicillin-susceptible *S. aureus* (MSSA), nafcillin or cefazoline is preferred for treatment of documented MSSA infection. The antibiotics should be injected at least for 4-6 weeks followed by 2-4 weeks oral antibiotic therapy (5,12,14). In cases associated with osteomyelitis this treatment should continue for 8 weeks (5). Neurologic function and imaging findings should be closely monitored while the therapy is commenced, particularly in patients who are treated medically. Subsequent development of immunocompromising condition or intake of immunosuppressive agents may result in recurrence of SEA long after the antibiotic therapy.

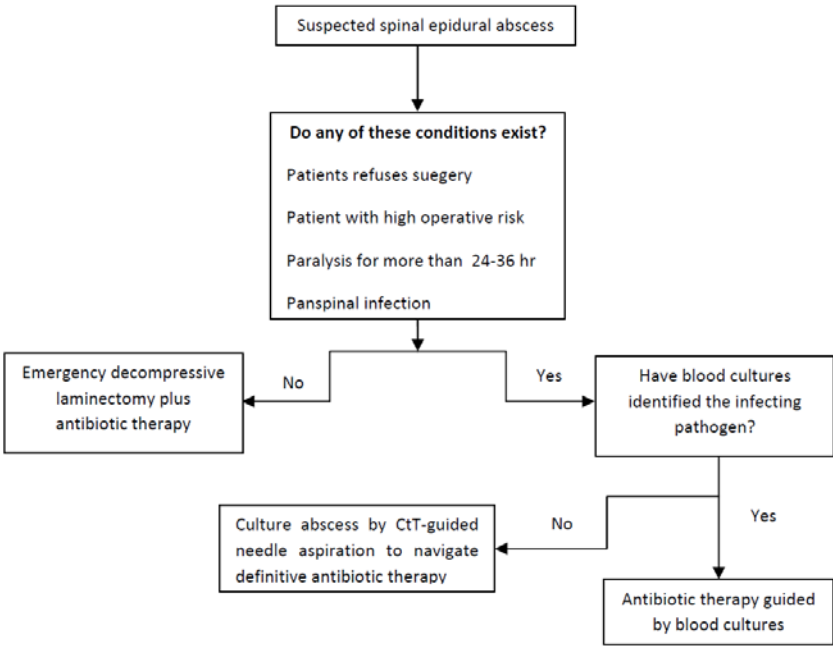

Figure 4. Management of spinal epidural abscess

In patients with SEA associated with an infected spinal cord stimulator, it is crucial to remove the whole stimulator system (including the subcutaneously placed generator and epidural electrodes) to reduce the likelihood of recurring implant related epidural infection. Patients with unexplained persistent or recurrent epidural infection may be assessed for rare sources of infection, such as esophageal tear (in the case of cervical epidural abscess) or intestinal–spinal fistula (in the case of thoracolumbar abscess).

Although there have been sporadic reports in which glucocorticoid therapy has been associated with an adverse outcome in patients who already had a severe case of SEA, it may help to reduce swelling in patients with progressive neurologic compromise who are awaiting surgical decompression.

In conclusion, SEA is a devastating condition if not diagnosed and treated promptly and effectively. Preoperative MR imaging provides a noninvasive, accurate means of diagnosis in this condition. Recent advances in the surgical treatment of SEA have resulted in the increased use of less-invasive surgical techniques, thereby decreasing the morbidity associated with surgery in both the short and long term. The single most important predictor of the final neurologic outcome is the patient's neurologic status immediately before surgery. Unless perioperative complications occur, the final neurologic condition in patients in whom SEA is adequately and promptly decompressed, is as good as or better than the preoperative condition. Patients who undergo surgery during stage 1 or stage 2 are expected to remain neurologically intact and possibly have a decreased risk of back and radicular pain, and those in stage 3 may have no weakness or a lesser degree of weakness versus before surgery. Patients in stage 4 who have been paralyzed for up to 24 to 36 hours are likely to regain some neurologic function postoperatively.

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