Case report

Spontaneous spinal epidural abscess in a normoglycemic diabetic patient – Keep it as a differential

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ABSTRACT

Spinal epidural abscess (SEA) is a rare and serious condition which can lead to permanent neurological deficit. Spontaneous SEA is even rarer condition with an incidence of less than 1 per 10,000 person-year. Being spontaneous, it has high chances of being misdiagnosed, more so when the risk factors are not clearly explainable for the condition. This is a case report of such a case in a middle aged normoglycemic recently diagnosed diabetic man with atypical presentation. The diagnosis was made after magnetic resonance imaging and confirmed after surgical intervention in form of spinal decompression and patient recovered after appropriate antibiotics. This article also sums up the risk factors of spontaneous SEA.

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1. Introduction

Spinal epidural abscess (SEA) is a rare but potentially life threatening condition which can cause permanent neurological disability if it is not diagnosed and treated in time. Approximately half of the cases of SEAs are either misdiagnosed or a delayed diagnosis is made.¹ The rare nature of spontaneous spinal epidural abscess (SSEA) combined with the fact that only half of the patients present with the classical clinical triad of back pain, fever and neurological deficit, leads to diagnostic difficulty.¹ But these clinical features are neither specific nor sensitive.² We are reporting a case of SSEA with atypical presentation.

2. Case report

A 57 year old male patient was admitted in the emergency department of our hospital with history of 15 days long low back ache. He was recently diagnosed with diabetes mellitus and was on oral hypoglycemic drugs since three months. His symptoms began 15 days back, acutely whilst picking up a heavy object. He experienced sudden catching pain in the lower back radiating to bilateral lower limbs. At that time he did not report of any neurological deficit. He went to a local general practitioner who

diagnosed him clinically of having acute prolapsed intervertebral disc and prescribed oral analgesics which gave him no relief. After 8 days of the acute episode, he experienced sudden loss of motor power in bilateral lower limbs without any bowel and bladder disturbance. He was reviewed by the local general practitioner, who noted weakness in bilateral lower limbs and ordered for an MRI of LS spine and the patient was then referred to our emergency department. During the entire course of 15 days the patient was afebrile. No antibiotics were given to the patient at any point of the treatment. On admission, he was afebrile, blood pressure was 140/90 mm Hg, random blood sugar was 128 mg/dl and heart rate was 77/min. Examination revealed diffuse tenderness over the lumbo-sacral region. Straight leg raise test was positive at 70° bilaterally. Power in both lower limbs was 3/5. No sensory deficit was there. Bilateral plantar reflex was flexor. He had normal anal tone and intact perianal sensations. Bulbocavernosus reflex was present. Total counts were in the normal range. Erythrocyte sedimentation rate and C-reactive protein were raised. Other blood investigations like hemoglobin, differential leukocyte counts were normal. Chest X-ray was within normal limits. But MRI showed epidural space-occupying lesion, which was hyperintense in both T1- and T2-weighted sequence, at the level of L2 to L4 vertebral body (Figs. 1 and 2).

The patient was immediately taken up for surgery. Posterior midline incision was given. Skin subcutaneous tissues were cut and the paraspinal muscles were elevated to expose the spinous process over L2–L4 centered over L3. Spinous process of L3

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Fig. 1. Magnetic resonance T2 image of sagittal section of spine showing heterogeneous signal changes in the epidural space from L2 to L4.

Fig. 2. Magnetic resonance T2 image of axial section of spine showing increased signal intensity around the cord, spinous process and the right facet, suggestive of an abscess.

vertebra was removed to expose the dura. Most of the lamina and whole of the facet joints were intact and not resected. Intraoperatively frank pus was found in the epidural space (Fig. 3) which was drained and sent for the culture and sensitivity test both against bacteria and mycobacteria. Proper saline wash using infant feeding tube 6 Fr, cephalad and caudad to the exposed cord after pus drainage, was given and no cord compression was evident. Paraspinous bone grafting using the resected spinous process was done and the wound was closed in layers.

Post-operatively patient was started on intravenous amoxicillin-clavulanic acid. Post operatively the patient had relief in pain on post operative day 1. In next 2 days the motor power of the lower limbs also became 5/5. The culture report came out to be *Staphylococcus aureus* sensitive to amoxicillin and the antistaphylococcal antibiotics was given for a week intravenously and for another week orally. The patient was advised bed rest for first five days and then encouraged for weight bearing. The inflammatory marker C-reactive protein was within normal limits by sixth post operative day. In the last follow up at fifteen months the patient came walking to us with no low back pain, no neurological deficit and no history of fever.

3. Discussion

SEA is a rare entity, incidence being 2.5–3 per 10,000 hospital admissions.^{3,4} SSEA is one which is not associated with any

Fig. 3. Intraoperative picture showing the epidural abscess after opening up the spinal canal after removing the spinous process of L3.

neurosurgical procedures or instrumentations of the spine⁵ and its incidence is further less estimated to be 0.88 cases per 100,000 person-year.⁶ SSEA is different from non spontaneous SEA, where later is most often than not iatrogenic in nature.⁶

This might suggest that Clinical progression of SEA is divided into four stages: (1) back pain, (2) radiation along nerve root from involved spinal region, (3) incomplete neurological deficit, (4) complete paralysis.⁴ Neuro-imaging is essential for determining the extent of the lesion. MRI is the gold standard for diagnosing spinal epidural abscess.⁷

In SSEAs, there can be normal blood counts and elevated inflammatory markers, in as much as 69% of the cases according to one case series.^{8,9} Similar pattern was seen in our case. Absence of fever with no leucocytosis may indicate that the pathogens in SSEA are of low virulence strains and this may be also the reason why they respond so quickly and dramatically with the antibiotics.

Treatment options for SSEA include intravenous antibiotics and surgical decompression by laminectomy, especially for patients with neurological deficit.¹⁰ This procedure not only decompresses the neural structures but also provides samples for the cultures and helps in starting accurate antibiotics according to the sensitivity. Considering high rate of delayed and misdiagnosis of SSEA, which can lead to deteriorating neurological deficit, surgery should be performed as soon as the diagnosis has been made. Though surgical decompression with antibiotic therapy is the standard treatment but there is a series which suggest only antibiotic therapy or in combination with percutaneous drainage have equivalent results.¹¹ Most common organism associated with SEA is *S. aureus*.⁴ Others include *E. coli*, *Pseudomonas*, *Brucella*, *Mycobacterium tuberculosis*.¹² No specific guidelines exist for antibiotic treatment in spinal epidural abscess. The standard principle is to treat with antibiotic having bactericidal action against the most common organism *S. aureus* empirically till the culture sensitivity report is available.⁹ To start with it is generally penicillin group drugs or second generation cephalosporins. Using third generation cephalosporin and a penicillin with or without metronidazole is another alternative till the sensitivity report.¹³ The recommended duration of antibiotic in SEAs extends up to 6–8

Table 1

Table enumerating the risk factors associated with spontaneous spinal epidural abscess with their incidence in two different studies (Study 1 by Gonzalez-Lopez et al.⁵ and Study 2 by Adogwa et al.¹¹).

Risk factors associated with spontaneous spinal epidural abscess	Study 1	Study 2
Acute infection	66.7%	9.3%
Osteomyelitis		
Urosepsis		
Retropharyngeal abscess		
Previous dental trauma		
Soft tissue infection		
Pneumonia		
Catheter related infections		
Hypertension	33.3%	
Liver disease/cirrhosis	26.7%	5.3% ^a
Smoking	20%	
Malignant neoplasm	20%	
Recent non neurological surgery	20%	
Alcoholism	13.3%	5.3% ^a
Illicit intravenous drug abuse	13.3%	1.3%
Diabetes mellitus	13.3%	41.3%
HIV infection	6.7%	1.3%
End stage renal disease		26.7%
Endocarditis		9.3%
Rheumatoid arthritis		5.3%

^a Study 2 mentioned combined percentage of liver cirrhosis and alcoholism as the risk factor of SSEA; their individual association was not clearly mentioned in the article.

weeks depending upon the virulence and presence of vertebral osteomyelitis.^{9,13} Since in our case, by 1st week C-reactive protein was normalized, parental administration was stopped and oral antibiotic was started for further one week.

The clinical features and subtle symptoms of sometimes only back pain initially may hinder a clinician to make a diagnosis of SEA. As reported the correct diagnosis of SEA is suspected clinically in only 40% of the patients at initial presentation.⁹ The other mimicking diagnoses like acute prolapsed intervertebral disc, degenerative disc disease, tuberculosis, primary or secondary spinal tumors, and even transverse myelitis in an immunocompromised patient⁹ create a diagnostic challenge and that's why the suspicion for SEA should be high.

It is prudent to know the risk factors of SSEA considering its sudden nature of onset and serious complications. Gonzalez-Lopez et al.,⁵ observed sixteen risk factors many of which were overlapping among the patients, Adogwa et al.,¹⁴ observed twelve risk factors in 80 patients but non spinal risk factors were ten in 75 patients (Table 1). Diabetes mellitus is one of the main predisposing factors for spinal epidural abscess. The patient in our study was recently diagnosed diabetes mellitus with normal blood sugar level at the time of presentation while he was on hypoglycemic medications. Still the patient developing spinal epidural abscess emphasizes that hyperglycemic period for few days can also lead to a sudden spontaneous epidural abscess.

Hence suspicion of SSEA should be high even in a diabetic patient with a well-controlled blood sugar.

4. Conclusion

Spinal epidural abscess can have an insidious onset with clinical presentation mimicking that of an acute prolapsed intervertebral disc. But it is a diagnosis which calls for urgent intervention and may present as diagnostic dilemma. Hence in patients with risk factors, as was diabetes in our case, presenting with acute back pain with or without radiculopathy, spontaneous spinal epidural abscess should always be kept in the differential diagnosis and suspicion should still be there despite normal blood sugar level.

Authors' contributions

Avinash Kumar involved in writing the manuscript and editing the manuscript, Vishal Kumar involved in data collection and supervision of manuscript, Sarvdeep S. Dhath involved in concept, supervision of manuscript and performed the surgery, Hitesh Lal involved in revision and editing of manuscript, Raj Bahadur involved in concept and performed the surgery.

Conflicts of interest

The authors have none to declare.

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