

## Case Report

# Tubercular spinal epidural abscess involving the dorsal-lumbar-sacral region without osseous involvement

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### Abstract

Musculoskeletal tuberculosis is known for its ability to present in various forms and guises at different sites. Tubercular spinal epidural abscess (SEA) is an uncommon infectious entity. Its presence without associated osseous involvement may be considered an extremely rare scenario. We present a rare case of tubercular SEA in an immune-competent 35-year-old male patient. The patient presented with acute cauda equina syndrome and was shown to have multisegmental SEA extending from D5 to S2 vertebral level without any evidence of vertebral involvement on MRI. The patient made an uneventful recovery following surgical decompression and antitubercular chemotherapy. The diagnosis was confirmed by histopathological demonstration of *Mycobacterium tuberculosis* in drained pus. Such presentation of tubercular SEA has not been reported previously in the English language based medical literature to the best of our knowledge.

**Key words:** Tuberculosis; epidural abscess; cauda equina syndrome; laminectomy

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### Introduction

Spinal epidural abscess (SEA) represents a severe, generally pyogenic, infection of the epidural space which may compress neural elements and require urgent surgical intervention to avoid permanent neurological deficit [1]. Tubercular spondylitis and its resulting complications are common in developing nations [2]. Tubercular SEA usually is secondary to tubercular spondylitis, but may rarely develop by haematogenous spread from any primary foci [3,4]. Compression of neural elements may necessitate urgent surgical decompression. Early diagnosis and early decompression remain the two important predictors of successful neurological outcome in acute cauda equina syndrome [5,6]. The worldwide resurgence of tuberculosis and the process of “reverse migration” have made this a topic of interest for orthopedic and neurosurgeons of developing as well as developed nations. We present an extremely rare case of spinal epidural abscess of tubercular origin involving the thoracic-lumbar-sacral region of the spine without osseous lesions.

### Case report

A 35-year-old male patient presented to the orthopaedic emergency room with complaints of decreased power in both lower limbs and urinary retention for the past 24 hours. He had a history of prolonged cough with sputum, low-grade evening rise fever for the past six weeks, and low backache for the past three weeks. These complaints were associated with history of anorexia and weight loss. There was no history suggestive of radicular pain or band-like sensations over the trunk. There was also a history of close contact with a patient suffering from tuberculosis. There was no history of intravenous drug abuse, repeated infections, or receiving corticosteroids for prolonged duration in the recent past, or any other systemic illness.

The patient consulted his family physician and was diagnosed to have pulmonary tuberculosis. The patient was started on four-drug anti-tubercular therapy (ATT) 15 days prior to development of neurological symptoms. On general physical examination, vitals were stable. Higher mental functions, cranial nerve examination and upper limb neurology were unremarkable. There was no

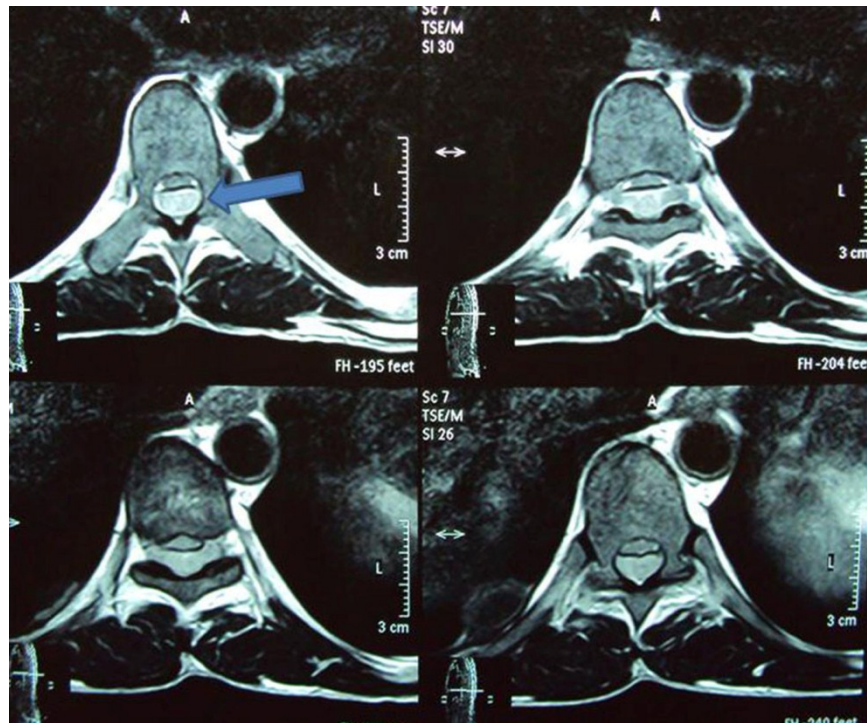
**Figure 1.** T1-weighted sagittal image of MR scan showing an elongated, diffuse, multisegmental collection in posterior epidural space involving dorsal, lumbar, and sacral region. All the visualized vertebrae are normal in morphology and signal intensity.



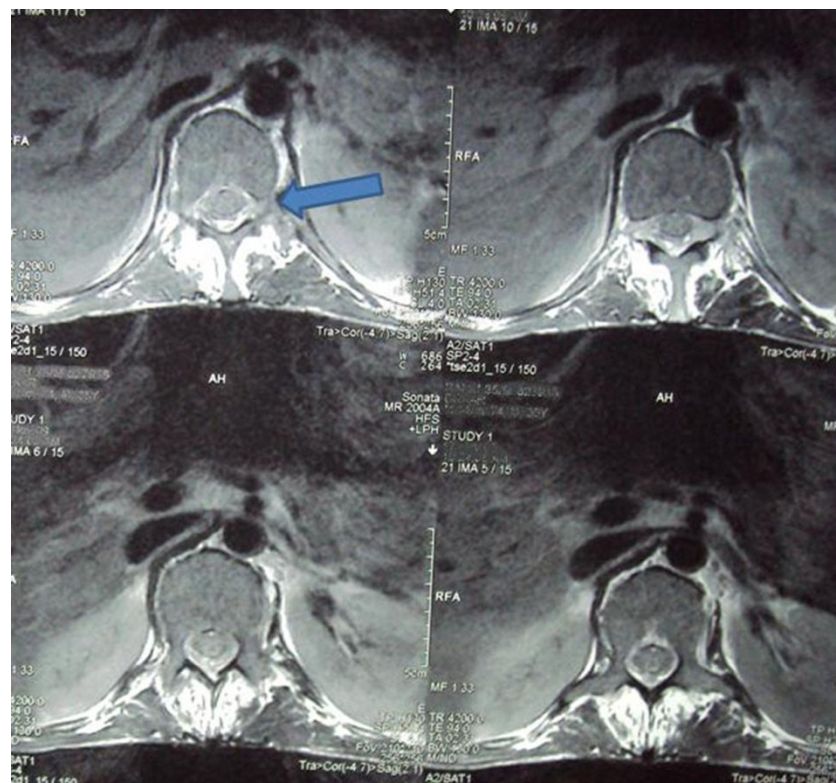
**Figure 2.** T2-weighted sagittal image of MR scan showing the proximal extent of the collection to D5 in posterior epidural space.



**Figure 3.** T2-weighted axial MR scans showing collection in posterior epidural space. The visualized vertebrae are normal in morphology and signal intensity.



**Figure 4.** Axial MR image showing the epidural abscess compressing the cauda equina





localized spinal tenderness, deformity, swelling, or discharging sinus in the back. Motor examination revealed loss of power, including bilateral tibialis anterior, extensor hallucis longus, flexor hallucis longus and tendoachilles, all of which had power Medical Research Council (MRC) grade 2/5 (*i.e.*, the patient was able to perform the action only when the effect of gravity has been excluded). Subjective sensory loss of 60-70% to touch, pinprick and temperature in L4 to S5 dermatome bilaterally was also observed. Ankle reflex was negative and plantars were down-going. There was loss of anal tone. A clinical diagnosis of cauda equina syndrome was made. The haematological investigations showed presence of leukocytosis (13,600/cumm) and raised sedimentation rate (80 mm after 1 hour by Wintrobe's method). The radiographs of the lumbo-sacral spine were unremarkable. Magnetic resonance imaging (MRI) of the lumbo-sacral spine with screening of the whole spine showed a multisegmental diffuse elongated collection in the posterior epidural region with peripheral rim enhancement extending from the D5 to S2 vertebra. However, it was not associated with changes in bone architecture (Figures 1-4). Chest radiograph showed right hilar lymphadenopathy with para-cardiac infiltrates suggestive of tuberculosis. ELISA test for HIV I and II antibodies was negative.

Urgent surgical decompression was performed. Considering the patient's presentation as cauda equina syndrome, it was planned to primarily relieve the clinically evident compression. Hence, laminectomy was done at L3-L5 levels and pus collection was drained, decompressing the cauda equina and dura. The spinal canal was irrigated with normal saline using an infant feeding tube. The pus was sent for culture and histopathological examination. The patient received standard post-operative care including adequate pain management, antibiotic coverage, and standard nursing care. Nutritional supplementation and antitubercular treatment was continued postoperatively (Isoniazide 5 mg/kg; Rifampicin 10 mg/kg; Pyrazinamide 25 mg/kg; Ethambutol 15 mg/kg; along with pyridoxine 10 mg). Acid fast bacilli were found from the pus evacuated during surgery and histopathological examination revealed necrotic tissue with granulomatous reaction and Langerhans giant cells. Polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* was positive [7]. Complete motor and sensory recovery was seen approximately 15 days after surgical decompression. The patient regained

his bladder sensations and voluntary control after three weeks of surgical decompression; however, a detailed urodynamic study was not performed. ATT was continued for a period of twelve months. At 30 months' follow up, the patient was asymptomatic.

The patient was informed that data concerning the case would be submitted for publication, and written, informed consent authorizing radiologic examination and photographic documentation was taken.

## Discussion

Infections of the spine can pose a wide range of problems for both the patient and the surgeon. There is no more devastating complication than the neurological deficit in the form of cauda equina syndrome due to spinal infection [6,8]. Most spinal infections in developed regions are the result of pyogenic organisms, whereas non-pyogenic organisms are responsible for most spinal infections in areas of the developing world, and in the immunocompromised population of developed nations [1,8]. *Mycobacterium tuberculosis* is the most common organism among these areas [2]. Tuberculosis demonstrates a variety of clinical and radiological findings and has a known propensity for dissemination from its primary site; therefore, it can mimic a number of disorders.

Spinal tuberculosis most commonly involves anterior elements, which causes pus to accumulate in pre- and para-vertebral space. The pus can further travel through intervertebral foramen and can cause pressure over the cauda equina. Tubercular SEA may develop secondary to involvement of the vertebral body or its appendages, and rarely by hematogenous dissemination from a primary focus in the body [3,4,9]. Hematogenous spread from pulmonary tuberculosis appears to be the cause in our case since no osseous lesion was apparent on radiographs or MRI. A long epidural abscess involving the thoracic-lumbar-sacral spine without osseous lesion may be considered an extremely rare manifestation of tuberculosis. Atypical presentation in this form may pose a diagnostic and therapeutic dilemma for treating clinicians [10-12]. Acute cauda equina syndrome in tuberculous spondylodiscitis and fungal spine infections is a rare clinical entity [6,11,13].

MRI displays the greatest diagnostic accuracy and is the method of choice for early diagnosis of these cases. Characteristic findings of tubercular spondylitis on MRI include the presence of multilocular, calcified abscess in the paraspinal

region and having subligamentous spread and contiguous multilevel involvement with a thick, enhancing, irregular rim in the presence of vertebral body fragmentation [14]. Tubercular spondylitis must be differentiated from pyogenic infections and malignant metastatic lesions. Tubercular affliction of the spine tends to have contiguous multilevel osseous involvement with paravertebral and epidural abscesses having subligamentous spread in contrast to pyogenic lesions. Metastases characteristically spare the disc space and may involve multiple noncontiguous vertebrae [15-17]; however, certain conditions, especially rheumatoid arthritis, brucellosis, sarcoidosis, fungal infection, and lymphoma, may produce a similar radiological appearance [14]. The clinical presentation in our patient suggested a cauda equina syndrome with the highest level of involvement at L4, but MRI showed that the highest level of involvement was D5, which further demonstrates that a high index of suspicion for multiple level involvements is needed in spinal tuberculosis and a screening of the whole spine may be required when spinal tuberculosis is suspected.

Tubercular SEA is amenable to both non-surgical and surgical management depending upon the individual case. Antitubercular chemotherapy remains the mainstay of treatment for tubercular infection or abscess [2]. Surgical drainage is indicated if it is not regressing on antitubercular therapy or causing pressure symptoms. In general, the goals of treatment in such cases include disease eradication, pain relief, preservation of neurological function, and spinal stability. Indications for surgical intervention include the presence of pressure symptoms, failure of medical management to control the disease process, cases with spinal instability, and the need to obtain diagnostic tissue in doubtful cases [2,6,8,10,13]. Since cauda equina syndrome is an acute surgical emergency, early decompression (within 48 hours) is vital for neurological recovery of such patients [3-6,8,11,13]. Kennedy *et al.* (1999) evaluated several predictors responsible for successful outcome in cauda equina syndrome and concluded that early diagnosis and early decompression are the two most important predictors of a successful outcome [5]. We decompressed the cauda equina by performing laminectomy L3-L5 to relieve the clinically evident compression and the higher abscess was flushed using a catheter.

This case report highlights the necessity of early diagnosis and early treatment of tubercular SEA for successful outcome. A high index of suspicion is

essential for making the correct diagnosis in such cases and one must bear in mind that the atypical presentations of common diseases are more common than the typical presentations of uncommon diseases.

## References

1. Reihnsaus E, Waldbaur H, Seeling W (2002) Spinal epidural abscess: a meta-analysis of 915 patients. *Neurosurg Rev* 232: 175-204.
2. Tuli SM (2004) Tuberculosis of the spine. In: Tuli SM, editor. *Tuberculosis of the Skeletal System (Bones, Joints, Spine and Bursal Sheaths)*. 3<sup>rd</sup> edition. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd. 191-343.
3. Prakash A, Kubba S, Singh NP, Garg D, Pathania A, Makhija A, Prakash N, Agarwal SK (2004) Tuberculous epidural abscess- an unusual presentation. *Ind J Tuberc* 51: 157-158.
4. Kumar A, Singh AK, Badole CM, Patond KR (2009) Tubercular epidural abscess in children: report of two cases. *Ind J Tuberc* 56: 217-219.
5. Kennedy JG, Soffe KE, McGrath A, Stephens MM, Walsh MG, McManus F (1999) Predictors of outcome in cauda equina syndrome. *Eur Spine J* 8: 317-322.
6. Arora S, Kumar R, Batra S, Nath R (2011) Transpedicular drainage of presacral abscess and posterior decompression of acute cauda equina syndrome in caries spine: a case series of 3 patients. *J Spinal Disord Tech* 24: E26-E30.
7. Tevere VJ, Hewitt PL, Dare A, Hocknell P, Keen A, Spadaro JP, Young KK (1996) Detection of Mycobacterium tuberculosis by PCR amplification with pan-Mycobacterium primers and hybridization to an M. tuberculosis-specific probe. *J Clin Microbiol* 34: 918-923.
8. Cohen DB (2004) Infectious origins of cauda equina syndrome. *Neurosurg Focus* 16: e2.
9. Pareyson D, Savoiaro M, D'Incerti L, Sghirlanzoni A (1995) Spinal epidural abscess complicating tuberculous spondylitis. *Ital J Neurol Sci* 16: 321-325.
10. Pande KC and Babulkar SS (2002) Atypical spinal tuberculosis. *Clin Orthop Relat Res* 398: 67-73.
11. Kapoor SK, Garg V, Dhaon BK, Jindal M (2005) Tuberculosis of the posterior vertebral elements: a rare cause of compression of the cauda equina. A case report. *J Bone Joint Surg Am* 87: 391-394.
12. Mathew J, Tripathy P, Grewal S (2009) Epidural tuberculosis involving the entire spine. *Neurol Neurochir Pol.*:470-474. {article in Polish}
13. Batra S, Arora S, Meshram H, Khanna G, Grover SB, Sharma VK (2011) Cauda equina syndrome: a rare etiology. *J Infect Dev Ctries* 5: 79-82.
14. Sharif HS, Clark DC, Aabed MY, Haddad MC, al Deeb SM, Yaqub B, al Moutaery KR (1990) Granulomatous spinal infections: MR imaging. *Radiology* 177: 101-107.
15. Moorthy S and Prabhu NK (2002) Spectrum of MR imaging findings in spinal tuberculosis. *Am J Radiol* 179: 979-983.
16. Smith AS, Weinstein MA, Mizushima A, Coughlin B, Hayden SP, Lakin MM, Lanzieri CF (1989) MR imaging characteristics of tuberculous spondylitis vs vertebral osteomyelitis. *Am J Radiol* 153: 399-405.
17. Stabler A and Reiser MF (2001) Imaging of spinal infection. *Radiol Clin North Am* 39: 115-135.

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