Tuberculous spinal arachnoiditis involving cauda equina is rare. A patient with lumbar tuberculous arachnoiditis in the absence of both vertebral and meningeal tuberculosis, which was mimicking spinal intradural extramedullary tumor is described here. Diagnosis was made based on intraoperative findings and was confirmed by histopathology. Surgical decompression along with a combination of steroid and antitubercular therapy resulted in a good outcome. At 3 months follow-up, the patient regained bladder control and was able to walk with support. Clinical features, magnetic resonance imaging, and intraoperative findings are described. Pathology and the relevant literature are discussed. Based on the patient’s clinical and radiologic findings, it was believed that the patient had a conus cauda tumor and was operated on. Histologic examination of the mass revealed tuberculoma. Surgical decompression followed by antituberculosis medication resulted in good outcome. Hence tuberculous arachnoiditis should be considered in differential diagnosis of conus cauda tumors.

**Key words:** Conus cauda tumor, tuberculous spinal arachnoiditis, radiculomyelitis

### Introduction

Tubercular arachnoiditis and radiculomyelitis are the important causes of infectious spinal arachnoiditis.\(^1\) The thoracic spinal cord is the most frequently involved, followed by the lumbar and cervical spinal cord.\(^2\) Several atypical forms of tuberculous spinal arachnoiditis giving rise to confusing diagnosis are well known. We are reporting a case of conus cauda arachnoiditis mimicking as a tumor without any history of systemic tuberculosis or vertebral body lesions. We aim to illustrate the difficulties in the diagnosis and management of this potentially curable disease by reviewing the literature.

### Case Report

A 40-year-old man presented with dull aching, progressively increasing back pain, which was radiating to the left lower limb along the posterior–lateral aspect of the buttock up to the foot for 3 months. This was accompanied with tingling and paraesthesia of both the lower limbs. One month following illness, he developed asymmetric onset of weakness in both the lower limbs involving proximal and distal muscles. He became bedbound within 15 days. He noticed numbness in the lower half of the body, including the perianal areas. He also developed bladder disturbances with constipation and erectile dysfunction for 1 week before admission. On clinical examination, he had asymmetric lower motor neuron type paraparesis with bladder and bowel involvement. Sensory loss (30%–40%) was present below L1 dermatome, including perianal region. Spinal tenderness and deformity were absent. He had normal hemogram and ESR. X-rays of the lumbosacral spine and chest were normal. Contrast MRI (lumbosacral) revealed an ill defined lesion (7.5 cm long and 1.5 cm thick) in the intradural space at L1–L4, which was isointense in T1W and heterointense in T2W [Figure 1]. The lesion demonstrates near homogenous enhancement [Figures 2 and 3]. The conus was bulky with abnormal signals. He underwent L2–L3 laminectomy and subtotal decompression of mass. Peroperatively, there was yellowish white lesion extending from L2 to L4,
The patient was started on antitubercular medication and after 3 months he showed excellent improvement. He was able to walk without support and regained bladder control partially.

**Discussion**

TB infection of spine occurs in the form of tuberculous spondylitis, intradural tuberculosis, and tubercular myelitis in the decreasing frequency. Intradural tuberculosis has been variously termed as intradural extramedullary tuberculosis, spinal arachnoiditis, and chronic adhesive arachnoiditis. It has been suggested that all these atypical forms of tuberculosis should be designated as tuberculous radiculomyelopathy (TBRM).[3]
TBRM may develop from 3 different sources.
1. Primary TB lesion arising in the spinal meninges.
2. A downward extension from the intracranial TB meningitis.
3. A secondary spread from adjacent vertebrae disease.

Among these, downward spread from TBM is most common. The thoracic cord is more frequently involved, followed by lumbar and cervical cords.

TBRM passes through 3 stages.[4]
1. Radiculitis—inflammation of pia arachnoid with associated hyperemia and swelling of roots.
2. Arachnoiditis—progressive fibroblast proliferation and collagen deposition leading to nerve root adhesions to each other and pia arachnoid.
3. Adhesive arachnoiditis—dense collagen deposition with encapsulation of atrophied nerve roots.

The case described here belongs to the second stage. Our case is having several atypical features as there was extensive lumbar involvement from L1 to L4 and there was no primary focus either in the brain or in adjacent vertebral bodies.

The clinical signs and symptoms are mostly limited to monoradicular or polyradicular pain syndromes that may be accompanied by motor and sensory deficits. Symptoms usually evolve over several years, although rapidly evolving cases have been described.[5] This rapid onset of symptoms, as happened in our case (<3 months) led us to the misdiagnosis of intradural tumor rather than arachnoiditis.

Classically, 3 MR patterns of arachnoiditis have been described involving cauda equina.[6]

Type 1: Central type—roots are clumped to the centre of thecal sac.

Type 2: Peripheral type—roots are adherent to the margins of the dural sac.

Type 3: Adherent roots to one side of thecal sac resembling a soft tissue mass.

MRI of our case is of type 3 showing adherent roots filling entire subarachnoid space resembling the conus cauda tumor. Differential diagnoses of contrast enhancing tumors in the conus cauda lesions in this age group include myxopapillary ependymoma, schwannoma, and paraganglioma.

Preoperatively, dura was thick, roots of cauda equina were adherent to each other and to the overlying dura. The roots of cauda equina were entangled in the granulation tissue. Similar intraoperative findings have been reported by Tanriverdi et al and Sreeharsha et al,[7] for spinal arachnoiditis.

Treatment of TBRM is mainly medical (9–12 months). Surgery should be considered only when HPE confirmation is required or there is evidence of spinal cord compression with neurologic deficit or spinal instability. Treatment of spinal tuberculous arachnoiditis may be medical or surgical. Medical treatment remains the mainstay of the treatment. Antituberculosis therapy with a combination of drugs should be started once the diagnosis is established.[8] Previous studies have shown a promising outcome with intrathecal hyalurondase—an enzyme that hydrolyses the glucosaminic bonds of hyaluronic acid and other mucopolysaccharides of the ground substance.[9] High-dose corticosteroid is another efficient adjuvant medical treatment, either given orally or, rarely, via the intrathecal route.[10,11] The entire course of therapy should continue for at least 9–12 months.[12]

The outcome of treatment has been unpredictable, with some reports observing good recovery[13] and some reporting unfavorable outcomes after surgical decompression.[17,14] However, a few case studies showed good recovery after surgical decompression. So surgery—decompressive laminectomy—should be considered if histologic diagnosis is necessary or there is evidence of spinal cord compression with neurologic deficit or spinal instability.[15]

Conclusions

A possibility of tuberculous arachnoiditis should be suspected despite the absence of primary focus anywhere in the body, particularly in TB endemic regions.

We emphasize the need for careful interpretation of MRI images and that spinal arachnoiditis may closely resemble a spinal cord tumor.

References

1. Medonca RA. Spinal infection and inflammatory disorders. In: Atlas SW, editor. MRI of the Brain and Spine. 3rd ed. Philadelphia: Lippincott Williams and Wilkins Publishers; 2002. p. 1855-969.

2. Poon TL, WS Ho, Pang KY, Wong CK. Tuberculous meningitis with spinal tuberculous arachnoiditis. Hong Kong Med J 2003;9:59-61.

3. Wadia NH, Dastur DK. Spinal meningitides with radiculo-myelopathy: Clinical and radiological features. J Neurol Sci 1969;8:239-60.

4. Burton CV. Lumbosacral arachnoiditis. Spine 1978;3:24-30.

5. Hoffman GS. Spinal arachnoiditis: What is the clinical spectrum? Spine 1983;8:538-40.
6. Ross J, Masaryk T, Modic M, Delamater R, Bohlman H, Wilbur G, et al. MR imaging of lumbar arachnoiditis. AJR Am J Roentgenol 1987;149:1025-32.
7. Sree Harsha CK, Shetty AP, Rajasekaran S. Intradural spinal tuberculosis in the absence of vertebral or meningeal tuberculosis: A case report. J Orthop Surg 2006;14:71-5.
8. Vidyasagar C, Murthy HK, Spinal tuberculosis with neurological deficits. Natl Med J India 1996;9:25-7.
9. Gourie-Devi M, Satishchandra P. Hyaluronidase as an adjuvant in the management of tuberculous spinal arachnoiditis. J Neurol Sci 1991;102:105-11.
10. Freilich D, Swash M. Diagnosis and management of tuberculous paraplegia with special reference to tuberculous radiculomyelitis. J Neurol Neurosurg Psychiatry 1979;42:12-8.
11. Hernandez-Albajar S, Arribas JR, Royo A, Gonzalez-Garcia JJ, Pena JM, Vazquez JJ. Tuberculous radiculomyelitis complicating tuberculomeningitis. Clin Infect Dis 2000;30:915-21.
12. Zuger A, Lowy FD. Tuberculosis. In: Scheld MW, Whitley RJ, Durack DT, editors. Infections of the central nervous system. 2nd Ed. Philadelphia: Lippincott-Raven Publishers; 1997. p. 428-36.
13. Kim MS, Kim KJ, Chung CK, Kim HJ. Intradural extramedullary tuberculoma of the spinal cord. J Korean Med Sci 2000;15:368-70.
14. Gimenez-Roldan S, Esteban A, Benito C. Communicating syringomyelia following cured tuberculous meningitis. J Neurol Sci 1974;23:185-97.
15. Dastur HM. Diagnosis and neurosurgical treatment of tuberculous disease of the CNS. Neurosurg Rev 1983;6:111-7.