Case Report

Isolated intramedullary tuberculoma as the initial presentation of disseminated tuberculosis: A case report

Rezwana Naher Begam[1], Ishan Sharma[2], Liyaqat Ali[1], Anjum Parvez[3], Syed Hasan Amir[4]

From [1]Doctor of Medicine, [2]Postgraduate Resident, [3]Professor, [4]Associate Professor, Department of Medicine, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, India

ABSTRACT

Spinal intramedullary tuberculoma is a rare manifestation of central nervous system tuberculosis (TB) and an uncommon cause of acute transverse myelitis. We report a 38-year-old man who presented with fever, low back pain, rapidly progressive paraplegia, sensory impairment, and urinary retention. Magnetic resonance imaging of the spine revealed intramedullary T2 hyperintensity extending from T11 to L1 with thick ring enhancement at T11, consistent with an intramedullary tuberculoma associated with tuberculous myelitis. HIV testing was negative. Additional imaging demonstrated pulmonary involvement with pleural effusion and multiple intracranial tuberculomas, suggestive of disseminated TB. The patient showed partial neurological improvement following initiation of corticosteroids and antitubercular therapy. This case highlights the importance of maintaining a high index of suspicion for TB in patients presenting with acute myelopathy in endemic regions, even in the absence of microbiological confirmation, as early diagnosis is crucial to prevent permanent neurological deficits.

Key words: Acute transverse myelitis, Disseminated tuberculosis, Magnetic resonance imaging spine, Spinal intramedullary tuberculoma

Extrapulmonary tuberculosis (TB) involving the central nervous system (CNS) most often presents as tuberculous meningitis or intracranial tuberculomas, with spinal manifestations occurring far less frequently. While most spinal TB presentations are related to osseous involvement (Pott's disease), spinal intramedullary tuberculoma (SIMT) represents one of the rarest presentations, with a reported rate of approximately 2/100,000 cases of TB [1]. Acute transverse myelitis (ATM) is an inflammatory condition of the spinal cord characterized by the rapid onset of motor, sensory, and autonomic dysfunction attributable to a lesion affecting the full cross-section of the cord at one or more vertebral levels. Although TB is an uncommon cause of ATM, it remains an important differential diagnosis in endemic regions, as delayed treatment may result in irreversible neurological deficits [2]. The intersection of disseminated TB, intramedullary tuberculoma formation, and ATM is an exceptional clinical phenomenon that poses significant diagnostic and therapeutic challenges. Diagnosis requires a high index of suspicion, especially in immunocompetent patients or in patients without systemic manifestations [3]. The present case illustrates an unusual but well-documented diagnostic challenge, where disseminated TB initially masqueraded as ATM.

Access this article online	
Received - 19 March 2026 Initial Review - 10 April 2026 Accepted - 27 April 2026	Quick Response code
DOI: 10.32677/ijcr.v12i5.8180	

CASE REPORT

A 38-year-old male, previously healthy, presented with a 10-day history of low-grade intermittent fever and dull aching non-radiating low back pain, followed by rapidly progressive weakness of both lower limbs and urinary incontinence over 7 days. The weakness was acute in onset, symmetric, and progressed to complete paraplegia with associated sensory loss below the groin level and early bladder involvement. There was no history of trauma, visual symptoms, cranial nerve involvement, or upper limb weakness. The patient denied weight loss, night sweats, cough, or prior contact with TB.

General and systemic examination was largely normal with a body mass index of 24.5 kg/m². Vitals on examination were stable with a blood pressure of 124/80, pulse rate of 110 bpm, temperature of 99°F, respiratory rate 17/min, and oxygen saturation of 97% in room air. Higher mental functions were intact, and no neck rigidity was observed. Neurological examination revealed flaccid paraplegia with reduced power in both lower limbs (1/5), accompanied by absent deep tendon and plantar reflexes.

Correspondence to: Dr. Rezwana Naher Begam, Department of Medicine, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, Uttar Pradesh, India. E-mail: rnb799001@gmail.com

Sensory examination showed a decrease in all modalities in both lower limbs. Based on the acute presentation, a provisional diagnosis of ATM was considered.

Initial laboratory investigations revealed mild anemia and elevated inflammatory markers. Fever workup and viral markers were negative. Chest radiography revealed heterogeneous opacity in the left lung with bilateral pleural effusion (Fig. 1). Post-contrast T1-weighted magnetic resonance imaging (MRI) images, including those of the lower thorax, showed heterogeneous signal intensity in the left lung with associated pleural effusion, suggesting pulmonary parenchyma involvement. Pleural fluid analysis revealed elevated protein levels (6.4 g/dL; normal <3.0 g/dL), an increased total leukocyte count (1450 cells/mm^3; normal <1000 cells/mm^3) with lymphocytic predominance (95%), elevated lactate dehydrogenase levels (945.6 U/L; normal <200 U/L), and elevated adenosine deaminase level (104.5 U/L; normal range: 0–30 U/L) supporting tubercular etiology; however, the pleural fluid cartridge-based nucleic acid amplification test (CBNAAT) was negative (Table 1). Cerebrospinal fluid (CSF) analysis revealed a protein level of 40 mg/dL (normal range: 15–45 mg/dL), a total leukocyte count of 3 cells/mm^3 (normal range: 0–5 cells/mm^3), and an adenosine deaminase level of 5.1 IU/L (normal range: 0–10 IU/L). CSF Gram stain, culture, and CBNAAT for *Mycobacterium tuberculosis* were negative. MRI spine demonstrated intramedullary T2 hyperintensity extending from the thoracic segment T11 to the lumbar segment L1 with diffuse involvement of the lower thoracic spinal cord and thick ring enhancement at the T11 level on post-contrast images, suggestive of tuberculoma with tuberculous myelitis (Fig. 2a and b). Contrast-enhanced MRI of the brain showed multiple small ring-enhancing lesions involving the bilateral cerebral hemispheres and left cerebellar hemisphere, with surrounding perilesional edema and no evidence of meningeal enhancement (Fig. 3a-c). Funduscopic examination was normal. These findings established the diagnosis of disseminated TB involving the pulmonary, pleura, brain, and spinal cord.

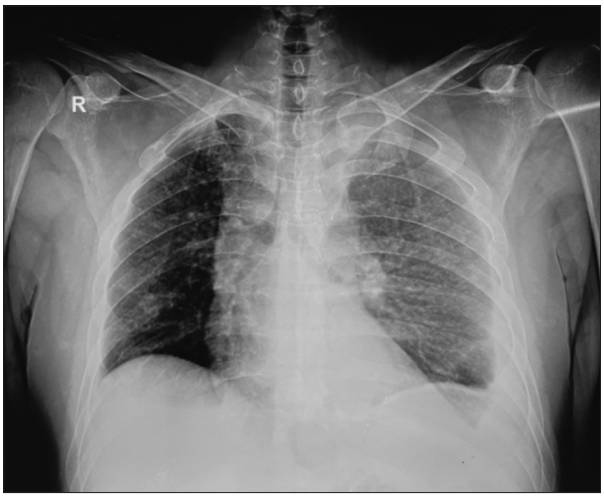

Figure 1: Chest radiograph (posteroanterior view) demonstrating heterogeneous opacity involving the left lung field with bilateral pleural effusion

The patient was initiated on standard antitubercular therapy under national TB guidelines [4], consisting of an intensive phase with isoniazid (5 mg/kg/day), rifampicin (10 mg/kg/day), ethambutol (15 mg/kg/day), and pyrazinamide (25 mg/kg/day), followed by a three-drug regimen (isoniazid, rifampicin, and ethambutol) in the continuation phase of treatment. High-dose intravenous corticosteroids, intravenous methylprednisolone (1 g/day) for 5 days, were administered initially to reduce spinal cord edema and inflammatory response, followed by gradual tapering to oral steroids for 8 weeks. Supportive management included bladder care with catheterization, thromboprophylaxis, and aggressive physiotherapy to prevent complications of immobility. Nutritional supplementation and monitoring for drug-related adverse effects were ensured. Following initiation of antitubercular therapy and corticosteroids, the patient showed gradual clinical improvement. Fever subsided within 2 weeks, and back pain significantly reduced. Over subsequent weeks, partial recovery of motor power in both lower limbs was noted, along with improvement in sensory symptoms. Bladder function showed gradual recovery, allowing intermittent catheter-free voiding. The patient continues antitubercular therapy with regular neurological follow-up and physiotherapy, showing steady functional improvement.

DISCUSSION

Isolated SIMT is an exceptionally rare manifestation of CNS TB. SIMT typically develops following hematogenous dissemination from a primary focus, CSF seeding, or contiguous spread from pulmonary diseases [5]. In our patient, chest radiographic findings

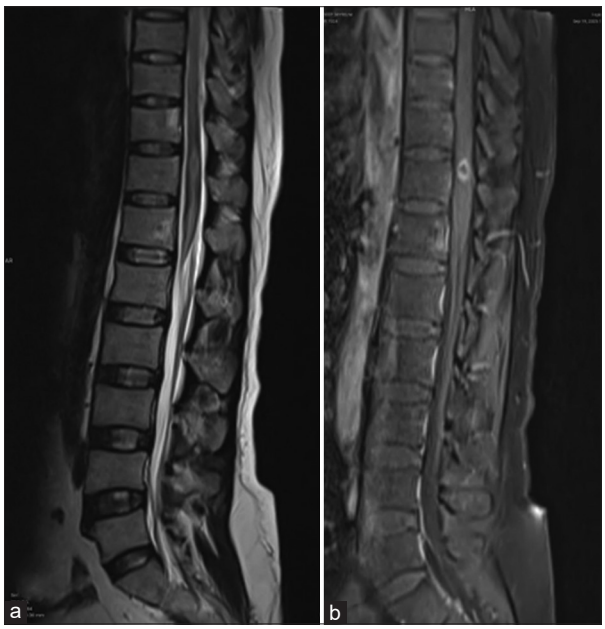

Figure 2: (a) Sagittal T2-weighted magnetic resonance imaging (MRI) of the spine demonstrating intramedullary hyperintensity at the T11–L1 level with diffuse involvement of the lower thoracic spinal cord; (b) post-contrast sagittal T1-weighted MRI of the spine demonstrating thick ring enhancement of the intramedullary lesion at T11 level (target lesion) suggestive of intramedullary tuberculoma

Table 1: Laboratory parameters of the patient

Parameters	Pleural fluid values	Normal range for pleural fluid parameters	CSF values	Normal range for CSF parameters
Protein	6.4 g/dL	<3.0 g/dL	40 mg/dL	15–45 mg/dL
Total leukocyte count	1450 cells/mm^3	<1000 cells/mm^3	3 cells/mm^3	0–5 cells/mm^3
Differential count	Lymphocytic predominance (95%)	-	-	-
Lactate dehydrogenase	945.6 U/L	<200 U/L	-	-
Adenosine deaminase	104.5 U/L	0–30 U/L	5.1 IU/L	0–10 IU/L
Gram stain	Negative	-	Negative	-
Culture	Negative	-	Negative	-
CBNAAT for *Mycobacterium tuberculosis*	Negative	-	Negative	-

CSF: Cerebrospinal fluid, CBNAAT: Cartridge-based nucleic acid amplification test

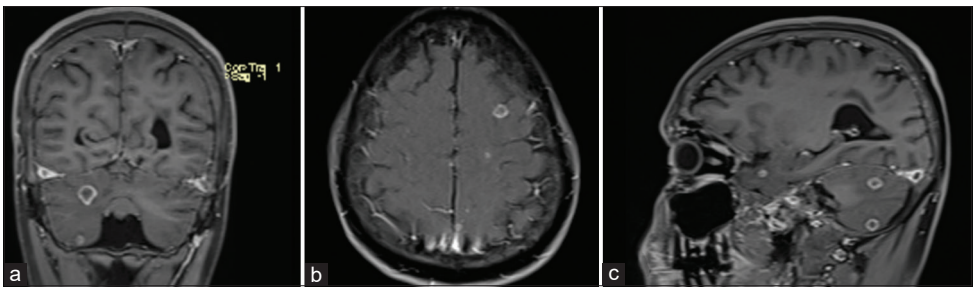

Figure 3: (a) Contrast-enhanced magnetic resonance imaging (MRI) brain (coronal section) showing multiple small ring-enhancing lesions in the left cerebellar hemisphere with surrounding perilesional edema; (b) Contrast-enhanced MRI brain (axial section) demonstrating multiple small ring-enhancing lesions involving bilateral cerebral hemispheres with surrounding perilesional edema and no evidence of meningeal enhancement; (c) Contrast-enhanced MRI brain (sagittal section) revealing multiple small ring-enhancing lesions in the left cerebellar hemisphere with surrounding perilesional edema

suggested pulmonary and pleural TB as the likely primary focus. ATM, as the initial manifestation of TB, is rare and poses significant diagnostic challenges, particularly when systemic features are minimal and microbiological tests are negative. The pathogenesis of tuberculous ATM likely involves a combination of direct mycobacterial spread and immune-mediated inflammatory injury, leading to spinal cord edema and neuronal damage [6]. In the current case, the presence of fever, elevated inflammatory markers, and pleural effusion provided early clues to an underlying infective etiology, despite the absence of classic constitutional symptoms such as weight loss or night sweats, a pattern also highlighted by the previous literature [5].

CSF examination was non-contributory, and CBNAAT was negative, reflecting the paucibacillary nature of extrapulmonary TB. Neuroimaging, therefore, played a decisive role in diagnosis. MRI revealed intramedullary lesions with characteristic features suggestive of tuberculous pathology, along with multiple intracranial tuberculomas. Importantly, our patient was immunocompetent, emphasizing that severe neurological manifestations of TB are not limited to immunosuppressed individuals in endemic regions. While histopathological confirmation remains the diagnostic gold standard, it is often not feasible in intramedullary lesions. In endemic regions, a diagnosis can be reasonably established based on characteristic MRI findings, evidence of systemic TB, and clinicoradiological correlation, even in the absence of microbiological confirmation. The thoracic predilection, particularly around the T10–T12 level, as seen in this case, has also been frequently reported by previous studies [1].

Management of intramedullary tuberculoma is primarily medical. Early initiation of antitubercular therapy with adjunctive corticosteroids is recommended to reduce cord edema and inflammatory damage. Surgical intervention is generally reserved for cases with diagnostic uncertainty or progressive neurological worsening despite adequate medical therapy, although it has shown variable benefit [7]. The neurological improvement observed following antitubercular therapy with corticosteroids highlights the importance of early diagnosis and prompt treatment in achieving functional recovery [1].

CONCLUSION

This case reinforces the need for a high index of suspicion for spinal TB in patients presenting with ATM–like syndromes, particularly in TB-endemic regions, even in immunocompetent patients. This highlights the importance of timely systemic evaluation, targeted imaging, and early medical management to prevent permanent neurological disability.

REFERENCES

1. Jaiswal M, Gandhi A, Sharma A, Mittal RS. Experiences and conceptualisation of spinal intramedullary tuberculoma management. Korean J Spine 2015;12:5-11.
2. Garg D, Radhakrishnan DM, Agrawal U, Vanjare HA, Gandham EJ, Manesh A. Tuberculosis of the spinal cord. Ann

Indian Acad Neurol 2023;26:112-26.
3. Muthukumar N, Venkatesh G, Senthilbabu S, Rajbaskar R. Surgery for intramedullary tuberculoma of the spinal cord: Report of 2 cases. Surg Neurol 2006;66:69-74; discussion 74.
4. Central TB Division. India TB Report 2023. New Delhi: Ministry of Health and Family Welfare, Government of India; 2023.
5. Iftikhar S, Ijaz N, Iftikhar S, Khan S. Intramedullary spinal tuberculoma: An uncommon presentation of a common disorder. Cureus 2022;14:36-9.
6. Turgut M. Spinal tuberculosis (Pott's disease): Its clinical presentation, surgical management, and outcome. A survey study on 694 patients. Neurosurg Rev 2001;24:8-13.
7. Arseni C, Samitca DC. Intraspinal tuberculous granuloma. Brain 1960;83:285-92.

Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Begam RN, Sharma I, Ali L, Parvez A, Amir SH. Isolated intramedullary tuberculoma as the initial presentation of disseminated tuberculosis: A case report. Indian J Case Reports. 2026;12(5):326-329.