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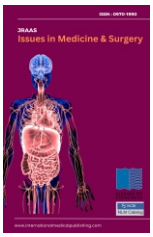

Case Report

Section: General Medicine

Tuberculous Meningitis with Longitudinally Extensive Transverse Myelitis: A Rare Case Report

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HIGHLIGHTS

- Rare TBM with LETM
- CSF confirmed TB infection
- MRI showed extensive myelitis
- Early treatment improved outcomes
- Neurological recovery occurred rapidly

Key Words:
Tuberculous meningitis
Central nervous system tuberculosis
Myelitis
Antitubercular therapy

ABSTRACT

Introduction: Tuberculous meningitis (TBM) is a severe form of central nervous system tuberculosis. Longitudinally extensive transverse myelitis (LETM), involving ≥3 vertebral segments, is a rare spinal complication. Their coexistence is exceptionally uncommon and can cause severe neurological deficits. **Aim & Objective:** To highlight the rare association of TBM with LETM and emphasize the importance of early diagnosis through clinical assessment, cerebrospinal fluid (CSF) analysis, microbiological confirmation, and magnetic resonance imaging (MRI). **Case Presentation:** A 20-year-old female presented with fever for 20 days, urinary retention for 14 days, headache and gait difficulty for 3 days, and altered sensorium for 1 day. Examination revealed decreased higher mental functions, positive Kernig's and Brudzinski's signs, upper-limb power of 4/5, lower-limb power of 0/5, and absent reflexes. CSF showed 30 cells/mm³ with 90% lymphocytes, protein 608 mg/dL, glucose 15.7 mg/dL, raised ADA, and CBNAAT positivity for *Mycobacterium tuberculosis*. Spinal MRI demonstrated heterogeneous signal alteration and enhancement from C7 to D5, consistent with LETM. Brain MRI showed enhancing lesions in the interpeduncular and perimesencephalic cisterns and bilateral parietal sulci. **Result:** Anti-tubercular therapy, physiotherapy, bladder management, intravenous methylprednisolone (30 mg/kg/day), and oral corticosteroids were administered. Fever and sensorium improved within 5 days, with marked neurological recovery within 2 weeks. **Conclusion:** TBM-associated LETM should be considered in patients presenting with meningitic features and acute myelopathy. Early combined anti-tubercular and corticosteroid therapy may promote favorable neurological recovery and prevent irreversible disability.

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INTRODUCTION

Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains a major global infectious disease despite the availability of effective antimicrobial therapy. The World Health Organization estimated that 10.7 million people developed TB in 2024, highlighting the continuing burden of disease, particularly in high-burden countries [1]. Central nervous system tuberculosis is one of the most severe forms of extrapulmonary TB, and tuberculous meningitis (TBM) represents its most important clinical manifestation because delayed recognition may lead to substantial neurological disability or death [2].

TBM develops after hematogenous dissemination of *M. tuberculosis* to the central nervous system, followed by formation and subsequent rupture of small subpial or subependymal tuberculous foci into the subarachnoid space. The resulting inflammatory response produces thick basal exudates, vasculitis, impaired cerebrospinal fluid circulation, cranial neuropathies, cerebral infarction, and hydrocephalus [3]. Clinically, TBM may initially present with nonspecific symptoms such as fever, headache, vomiting, altered sensorium, and meningism, while cerebrospinal fluid analysis typically demonstrates an inflammatory pattern compatible with chronic meningitis. Because microbiological confirmation may be difficult due to a low bacillary load in cerebrospinal fluid, diagnosis often relies on integration of clinical findings, neuroimaging, CSF characteristics, and targeted molecular or microbiological testing [2].

Spinal cord involvement in tuberculosis is uncommon and may occur through vertebral disease, arachnoiditis, tuberculoma, radiculomyelitis, vascular injury, or inflammatory myelitis [4]. Tuberculous transverse myelitis is particularly rare. When the inflammatory spinal cord lesion extends continuously across three or more vertebral segments on magnetic resonance imaging, it is classified as longitudinally extensive transverse myelitis (LETM) [5]. Patients may develop rapidly progressive paraparesis or quadriparesis, sensory deficits, a defined sensory level, and bladder or bowel dysfunction. The differential diagnosis of LETM is broad and includes neuromyelitis optica spectrum disorder, myelin oligodendrocyte glycoprotein antibody associated disease, autoimmune disorders, other infections, vascular disorders, and neoplastic conditions, making recognition of an infectious tuberculous etiology clinically challenging [6]. MRI is central to evaluating suspected LETM because it defines longitudinal extent, cord swelling, signal abnormalities, enhancement patterns, and meningeal or vertebral pathology. Recognizing this pattern in a patient with features of meningitis can potentially redirect investigation toward tuberculosis and prevent misclassification as a demyelinating disorder requiring a different therapeutic approach [4]. **Figure 1.** Pathogenesis of tuberculous meningitis (TBM) and longitudinally extensive transverse myelitis (LETM), illustrating hematogenous dissemination of *Mycobacterium tuberculosis*, basal meningeal inflammation, vascular injury, and associated spinal cord inflammation, swelling, and ischemia

The coexistence of TBM and LETM is exceptionally uncommon but clinically important because meningeal inflammation may extend to the spinal leptomeninges and cord, while immune-mediated inflammation, vasculitis, or direct mycobacterial involvement may further contribute to cord injury [7]. Published reports emphasize that tuberculosis should remain an important differential diagnosis of LETM in endemic regions, particularly when meningeal features and supportive CSF findings are present [8]. Early recognition is crucial because prompt initiation of antitubercular therapy with appropriate adjunctive corticosteroid treatment may limit irreversible neurological damage [2]. The present case is therefore reported to highlight the rare simultaneous occurrence of tuberculous meningitis with longitudinally

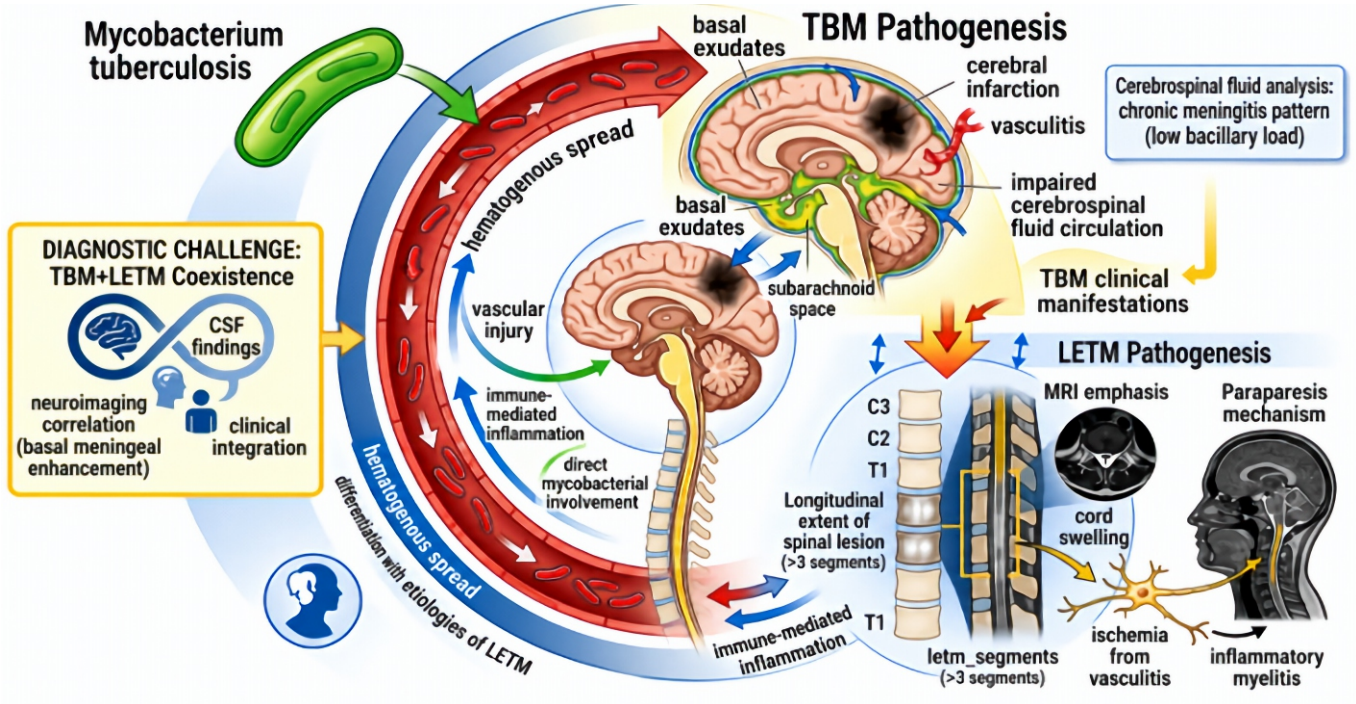

Figure 1: Pathogenesis of TBM with LETM, showing meningeal inflammation, vascular injury, and spinal cord involvement.

extensive transverse myelitis and to emphasize the importance of correlating meningeal signs, CSF findings, and spinal MRI abnormalities in establishing the diagnosis.

CASE PRESENTATION

A 20-year-old female presented with fever for 20 days, urinary retention for 14 days, headache and gait difficulty for 3 days, and altered sensorium for 1 day. Back pain, lower-limb weakness and bladder involvement with urinary retention/urgency were also documented. On clinical examination, higher mental functions were decreased, with positive Kernig's and Brudzinski's signs. Motor examination showed power of 4/5 in the upper limbs and 0/5 in the lower limbs, with absent reflexes. Fundus examination was normal **(Table 1)**.

CSF analysis showed TLC 30 cells/cu mm with 90% lymphocytes, markedly elevated protein (608 mg/dL), low glucose (15.7 mg/dL), and positive ADA. CBNAAT was positive for *Mycobacterium tuberculosis*. MRI spine revealed altered signal intensity with heterogeneous enhancement extending from C7 to D5 levels, consistent with longitudinally extensive myelitis. MRI brain demonstrated enhancing lesions in the interpeduncular and peri-mesencephalic cisterns and bilateral parietal sulci, supporting associated meningeal involvement **(Table 2)**. **Figure 2.** MRI brain showing enhancement in the interpeduncular cisterns and bilateral parietal sulci.

Figure 3: Sagittal MRI showing altered signal intensity and heterogeneous enhancement from C7 to D5. Anti-tubercular therapy (ATT) was started. Persistent paraparesis was followed by MRI confirmation of myelitis. Supportive care with physiotherapy and bladder management was initiated. Neurology consultation was obtained and intravenous steroids were advised. The patient received intravenous Solumedrol 30 mg/kg/day followed by oral steroids **(Table 3)**. Following initiation of ATT, fever and sensorium improved within 5 days. Paraparesis persisted initially, after which MRI confirmed myelitis and steroid therapy was administered. Following intravenous Solumedrol and subsequent oral steroids, marked neurological recovery was documented within 2 weeks **(Table 4)**.

RESULTS

A 20-year-old female presented with prolonged fever, headache, gait difficulty, urinary retention, altered sensorium, and progressive bilateral lower-limb weakness. On examination, positive Kernig's and Brudzinski's signs were noted, with profound lower-limb weakness and areflexia. CSF analysis revealed lymphocytic pleocytosis, markedly elevated protein (608 mg/dL), low glucose (15.7 mg/dL), elevated ADA, and positive CBNAAT for *Mycobacterium tuberculosis*, confirming tuberculous meningitis. MRI of the spine demonstrated a longitudinally extensive lesion from C7 to D5 with altered signal intensity and heterogeneous post-contrast enhancement, consistent with LETM. Brain MRI showed enhancement involving the interpeduncular and perimesencephalic cisterns and bilateral parietal sulci, indicating associated meningeal inflammation.

The patient was started on standard anti-tubercular therapy, following which fever and altered sensorium improved within 5 days. However, severe paraparesis and urinary dysfunction persisted, prompting further evaluation and corticosteroid therapy. Intravenous methylprednisolone followed by oral corticosteroids was administered along with physiotherapy and bladder management.

Marked neurological improvement was observed within 2 weeks, with improvement in lower-limb motor function and bladder symptoms. The favorable response suggests that, in addition to active tuberculous infection, a significant reversible inflammatory component contributed to the spinal cord dysfunction. Overall, early recognition and combined anti-tubercular and anti-inflammatory treatment resulted in substantial clinical recovery.

Table 1. Clinical Presentation and Examination Findings

Parameter	Finding
Patient	20-year-old female
Fever	20 days
Urinary retention	14 days
Headache and gait difficulty	3 days
Altered sensorium	1 day
Other symptoms	Back pain; lower -limb weakness; bladder involvement with urinary retention/urgency
Clinical examination	Decreased higher mental functions; positive Kernig's and Brudzinski's signs
Motor examination	Upper-limb power 4/5; lower -limb power 0/5; reflexes absent
Fundus	Normal

Table 2. Investigation Findings

Investigation	Finding
CSF cytology	TLC 30/cu mm; lymphocytes 90%
CSF biochemistry	Protein 608 mg/dL; sugar 15.7 mg/dL; ADA positive
CBNAAT	Positive for M. tuberculosis
MRI spine	C7-D5 altered signal and heterogeneous enhancement
MRI brain	Enhancing lesions in interpeduncular and peri -mesencephalic cisterns and parietal sulci

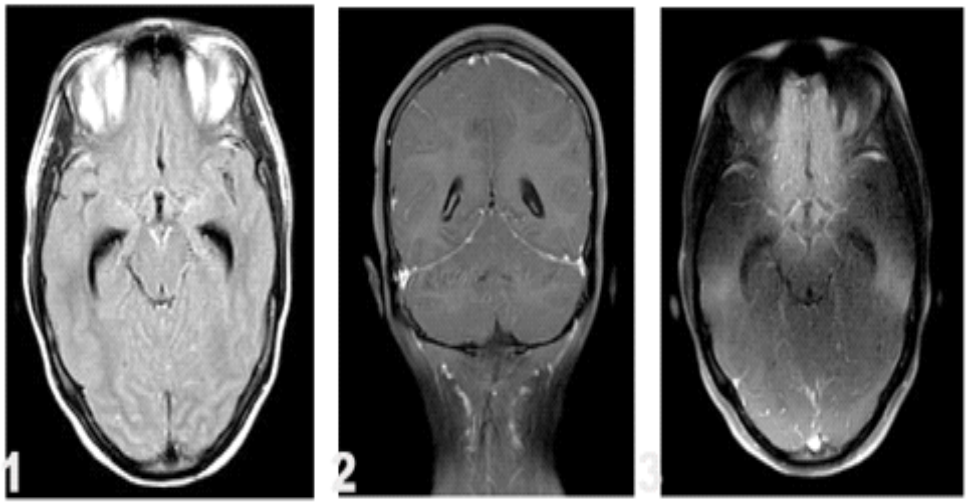

Figure 2: MRI brain showing mixed signal intensity and post-contrast enhancement in the interpeduncular cisterns and bilateral parietal sulci.

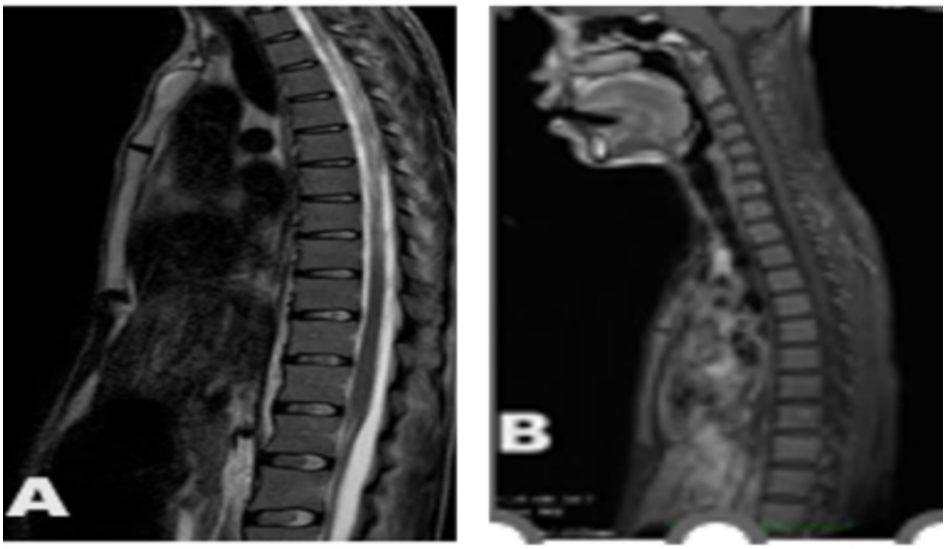

Figure 3: MRI spine sagittal views showing altered signal intensity and heterogeneous enhancement extending from C7 to D5 levels

Table 3. Treatment Methodology and Clinical Course

Management step	Documented detail
Anti-tubercular therapy	ATT started
Persistent neurological deficit	Paraparesis persisted; MRI confirmed myelitis
Supportive care	Physiotherapy and bladder management initiated
Neurology consultation	IV steroids advised
Corticosteroid therapy	IV Solumedrol 30 mg/kg/day followed by oral steroids

Table 4. Treatment Outcome

Time/Course	Documented Outcome
Within 5 days after ATT initiation	Fever and sensorium improved.
Initial neurological course	Paraparesis persisted, leading to MRI confirmation of myelitis.
Within 2 weeks after steroid therapy	Marked neurological recovery was documented.

DISCUSSION

This case illustrates a rare but clinically important association between tuberculous meningitis (TBM) and longitudinally extensive transverse myelitis (LETM). The patient was a 20-year-old female presenting with prolonged fever, headache, gait difficulty, urinary retention, altered sensorium, positive Kernig's and Brudzinski's signs, profound lower-limb weakness, and areflexia. CSF showed lymphocytic pleocytosis, markedly elevated protein (608 mg/dL), low glucose (15.7 mg/dL), positive ADA, and CBNAAT positivity for *Mycobacterium tuberculosis*. MRI spine demonstrated a long-segment lesion extending from C7 to D5 with heterogeneous enhancement, while brain MRI showed enhancement involving the interpeduncular and peri-mesencephalic cisterns and bilateral parietal sulci, supporting simultaneous meningeal and spinal cord involvement. Sahu et al. (2014) similarly described four patients with tuberculosis-associated LETM & emphasized tuberculosis as an important consideration when extensive spinal cord involvement is encountered [9]. Jain et al. (2015) reported tuberculous LETM associated with brain tuberculoma, demonstrating that intracranial and extensive spinal manifestations may coexist in CNS tuberculosis [10]. Kasundra et al. (2016) described distal cord-predominant LETM with diffuse spinal meningitis and dural abscesses secondary to occult tuberculosis, further supporting the association between meningeal inflammation and extensive spinal pathology [11].

Liu et al. (2019) highlighted MRI as a key diagnostic modality in tuberculous meningomyelitis because abnormalities of the spinal cord and meninges may facilitate early diagnosis when the clinical presentation is nonspecific [12]. Md Noh et al. (2020), in their clinicoradiological review, concluded that tuberculous myelopathy associated with longitudinally extensive lesions is very rare but potentially treatable when identified promptly [13]. Zafar et al. (2021) reported a 24-year-old man with TBM and LETM extending from T2 to T9 who had fever, headache, urinary difficulty, and 0/5 lower-limb power, with brain imaging demonstrating basal meningeal enhancement. Their presentation closely parallels the severe motor and bladder involvement observed in the present case, although microbiological testing was negative in their patient, whereas our patient had positive CSF CBNAAT [14].

Khan et al. (2022) prospectively evaluated tuberculous myelitis and found headache, markedly elevated CSF protein, and spinal meningeal enhancement to be distinguishing features; LETM was common, and tuberculous myelitis showed a favorable response to corticosteroid-containing treatment [15]. Jiang et al. (2022) identified myelitis in 19 of 114 patients with TBM, with urinary retention or constipation occurring in 77.8%; all affected patients had spinal lesions extending over more than three segments. High CSF protein was independently associated with myelitis, making the markedly elevated protein in the present case particularly relevant [16]. More recently, Garg et al. (2025) systematically reviewed 39 reported patients with tuberculous myelitis and found longitudinally extensive cervical and thoracic hyperintensities to be common, while 87.2% demonstrated clinical improvement [17].

Therapeutically, ATT produced improvement in fever and sensorium within five days in our patient, while persistent paraparesis prompted MRI confirmation of myelitis. Intravenous methylprednisolone followed by oral corticosteroids, together with physiotherapy and bladder management, resulted in marked neurological recovery within two weeks. The rapid neurological response after steroids also suggests a substantial reversible inflammatory component alongside active tuberculous infection.

CONCLUSIONS

Tuberculous meningitis with longitudinally extensive transverse myelitis is a rare but important manifestation of central nervous system tuberculosis that may cause profound neurological disability if recognition is delayed. In this case, meningeal signs, characteristic CSF abnormalities, CBNAAT positivity, and MRI evidence of C7–D5 myelitis established the diagnosis. Early anti-tubercular therapy improved fever and sensorium, while persistent paraparesis responded markedly to intravenous methylprednisolone followed by oral steroids, physiotherapy, and bladder care. This case highlights the importance of integrating clinical, microbiological, and neuroimaging findings and initiating timely combined antimicrobial and anti-inflammatory treatment to achieve favorable neurological recovery and effectively prevent irreversible sequelae.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical health-.care, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patientworld relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

TBM: Tuberculous Meningitis
LETM: Longitudinally Extensive Transverse Myelitis
CNS: Central Nervous System
CSF: Cerebrospinal Fluid
MRI: Magnetic Resonance Imaging
ADA: Adenosine Deaminase
CBNAAT: Cartridge-Based Nucleic Acid Amplification Test
ATT: Anti-Tubercular Therapy
IV: Intravenous
IM: Intramuscular

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research diretions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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