Case Report

Atypical Presentation of Tuberculous Meningitis with Early Quadriparesis Due to Tuberculous Myelo-arachnoiditis in a Young Female

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Introduction: Tuberculous meningitis (TBM) remains a major cause of neurological morbidity and mortality, particularly in tuberculosis-endemic regions like India. Tuberculous myelo-arachnoiditis is a rare but serious spinal complication of TBM, reported in approximately 2 to 5% of cases, often leading to progressive myelopathy and permanent neurological sequelae. Early neuroimaging may be normal, contributing to diagnostic delay.

Case Presentation: A 25-year-old immunocompetent female presented with an 8-day history of progressive headache, followed by fever and altered sensorium. Neurological examination revealed a Glasgow coma scale score of E3V3M5, generalized hypotonia, quadriparesis (Medical Research Council grade 2/5 in all limbs), diminished deep tendon reflexes, and bilateral extensor plantars. During hospitalization, she developed a right sixth cranial nerve palsy with progression to quadriparesis. Initial magnetic resonance imaging of the brain was normal. Initial cerebrospinal fluid (CSF) analysis showed mild abnormalities (glucose 52 mg/dL, protein 96 mg/dL, cells 45/mm^3 with mixed cellularity). With clinical deterioration, repeat CSF analysis revealed marked hypoglycorrhachia (glucose 23 mg/dL), elevated protein (667 mg/dL), and lymphocytic pleocytosis (110 cells/mm^3, 70% lymphocytes). A diagnosis of tuberculous meningitis with tuberculous myelo-arachnoiditis was established. The patient showed rapid clinical improvement following initiation of antitubercular therapy and adjunctive corticosteroids.

Discussion: This case highlights the diagnostic challenges of TBM, including the radiological–clinical dissociation with initially normal neuroimaging, the dynamic evolution of CSF findings, and the importance of recognizing spinal involvement in patients developing progressive motor deficits. The dramatic therapeutic response to antitubercular therapy further supported the diagnosis.

Conclusion: In tuberculosis-endemic regions, TBM should be suspected in patients presenting with subacute meningoencephalitis, even when initial imaging and CSF studies are inconclusive. Early recognition of spinal involvement, serial lumbar punctures, and prompt empirical antitubercular therapy are essential to prevent irreversible neurological disability.

Access this article online

Website: www.cijmr.com

DOI: 10.58999/cijmr.v5i02.331

Keywords: Tuberculous meningitis, Tuberculous myelo-arachnoiditis, Quadriparesis, Cerebrospinal fluid, Case report, CARE guidelines

Introduction

Tuberculous meningitis (TBM) is the most severe and debilitating form of extrapulmonary tuberculosis, accounting for approximately 1 to 5% of all tuberculosis cases and up to 10% of extrapulmonary tuberculosis worldwide. India bears a substantial burden of tuberculosis, and TBM remains a major cause of neurological morbidity and mortality, particularly among young adults. Despite appropriate therapy, mortality rates range from 20 to 30%, and nearly half of survivors are left with permanent neurological sequelae.

TBM results from hematogenous dissemination of *Mycobacterium tuberculosis* to the central nervous system, leading to chronic granulomatous inflammation predominantly involving the basal meninges. The resultant inflammatory exudates and vasculitic changes can cause raised intracranial pressure, hydrocephalus, cerebral infarction, cranial nerve palsies, and altered sensorium. While intracranial manifestations are well recognized, spinal involvement in TBM is relatively uncommon and frequently under diagnosed.

Tuberculous myelo-arachnoiditis is a rare but serious complication of TBM, reported in approximately 2 to 5% of cases. It is characterized by inflammatory involvement

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Submitted: 14/01/2026 Revision: 14/01/2026
Accepted: 02/03/2026 Published: 15/08/2026

How to cite this article: Agrawal U, Sharma A, Gupta N. Atypical Presentation of Tuberculous Meningitis with Early Quadriparesis Due to Tuberculous Myelo-arachnoiditis in a Young Female. Central India Journal of Medical Research. 2026;5(2):236-240.

of the spinal cord, nerve roots, and arachnoid mater, leading to adhesive arachnoiditis, vascular compromise, and progressive myelopathy. Clinically, patients may present with radiculopathy, sensory disturbances, sphincter dysfunction, and progressive motor weakness, which may evolve into paraparesis or quadriparesis. Early neuroimaging may be normal, contributing to diagnostic delay.

We report a case of a young female with confirmed tuberculous meningitis who developed progressive quadriparesis secondary to tuberculous myelo-arachnoiditis, despite initially normal neuroimaging, highlighting the importance of clinical vigilance and reliance on cerebrospinal fluid (CSF) biochemical analysis.

Case Presentation

A 25-year-old female with no known comorbidities presented with an 8-day history of persistent, progressively worsening headache, followed by 6 days of intermittent fever and a 5-day history of progressive alteration in sensorium. The headache was diffuse, severe in intensity, and associated with nausea but no vomiting or photophobia. There was no history of seizures, loss of consciousness, head trauma, cough, weight loss, night sweats, or prior tuberculosis. There was no known contact with tuberculosis, and no history suggestive of immunosuppression, diabetes mellitus, or hypertension. Past medical, personal, and family histories were unremarkable.

On admission, the patient was febrile and disoriented to time, place, and person. Her vital parameters revealed blood pressure readings up to 150/100 mmHg, pulse rate of 104 beats per minute, temperature of 101.4°F, and oxygen saturation of 97% on room air.

Neurological Examination

Neurological examination revealed altered sensorium with a Glasgow coma scale score of E3V3M5. There were no signs of meningeal irritation at presentation. Cranial nerve examination was initially unremarkable. Motor examination demonstrated generalized hypotonia, reduced muscle strength (Medical Research Council grade 2/5 in all limbs), diminished deep tendon reflexes, and bilateral extensor plantar responses. Sensory examination was limited due to poor cooperation; however, pain sensation appeared reduced. These findings suggested involvement of the central motor pathways in the setting of a meningoencephalitic process.

During the course of hospitalization, the patient developed a right sixth cranial nerve palsy, manifested by impaired abduction of the right eye. Motor weakness progressed to involve all four limbs, evolving into quadriparesis, with further reduction in muscle tone and reflexes.

Systemic examination of the cardiovascular, respiratory, and abdominal systems was unremarkable.

Investigations

Neuroimaging

Magnetic resonance imaging (MRI) of the brain, including T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging (DWI), susceptibility-weighted angiography (SWAN), and contrast-enhanced sequences, demonstrated normal cerebral parenchyma, basal ganglia, and brainstem. There was no evidence of focal lesions, meningeal enhancement, infarcts, hemorrhage, or hydrocephalus. The ventricular system was normal in size and configuration (Figure 1).

Impression

No significant intracranial abnormality.

Other Imaging

Ultrasonography of the abdomen revealed mild right-sided hydronephrosis. The liver, spleen, pancreas, and urinary bladder were otherwise normal.

Cerebrospinal Fluid Analysis

Initial CSF examination

Lumbar puncture performed at admission revealed the following cerebrospinal fluid parameters:

- CSF glucose: 52 mg/dL (corresponding blood glucose: 94 mg/dL)
- CSF protein: 96 mg/dL (mildly elevated)
- CSF cell count: 45 cells/mm^3 with mixed cellularity (55% lymphocytes, 45% neutrophils)
- CSF adenosine deaminase (ADA): negative
- Gram stain, bacterial culture, fungal stain, and viral serology: negative

The initial CSF findings were non-conclusive, not fulfilling classical criteria for acute bacterial, viral, or tuberculous meningitis.

Initial management and clinical progression

In view of the clinical presentation of fever, altered sensorium, and equivocal CSF findings, the patient was empirically treated as a case of acute bacterial meningitis with broad-spectrum intravenous antibiotics along with

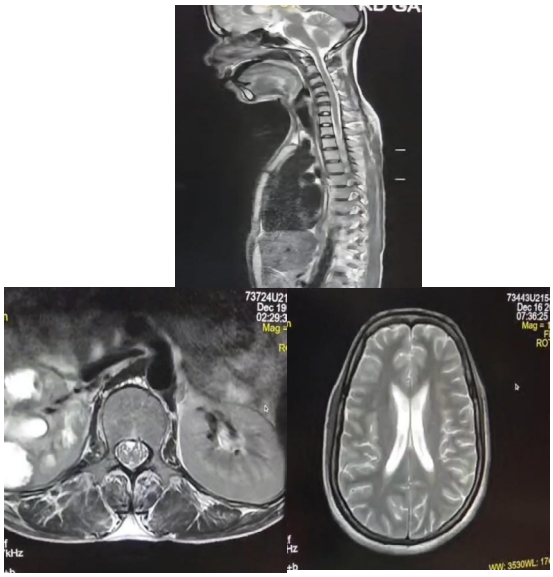

Figure 1: MRI brain and spine at the time of presentation showed no identifiable pathology

adjunctive corticosteroids. Supportive care, including antipyretics and close neurological monitoring, was provided.

Despite appropriate therapy, the patient continued to have persistent fever and progressive neurological deterioration. Her sensorium worsened, and she developed new focal neurological deficits in the form of right sixth cranial nerve palsy. Motor weakness progressed to quadriparesis over the subsequent days, raising concern for an evolving chronic inflammatory meningoencephalitic process.

Repeat cerebrospinal fluid analysis

In view of the lack of response to antibacterial therapy and progressive neurological involvement, a repeat lumbar puncture was performed, which revealed:

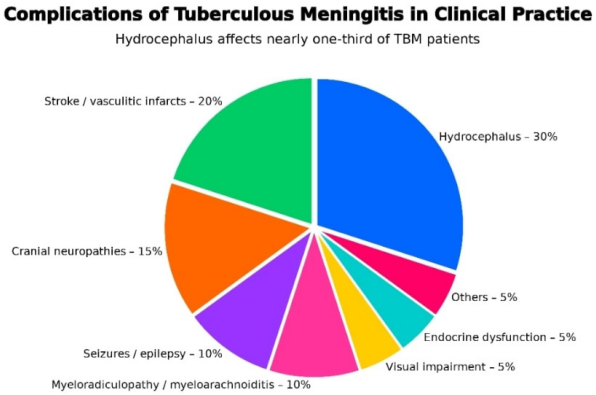

Figure 2: Complications of tubercular meningitis in clinical practice

- CSF glucose: 23 mg/dL
- CSF protein: 667 mg/dL
- CSF cell count: 110 cells/mm^3 with predominant lymphocytosis (70%)
- CSF ADA: negative
- CSF cultures and microbiological studies: sterile

The repeat CSF demonstrated marked hypoglycorrhachia, significantly elevated protein levels, and lymphocytic pleocytosis, a pattern strongly suggestive of tuberculous meningitis, despite the absence of microbiological confirmation.

Final diagnosis and outcome

Given the subacute progression, development of cranial nerve palsy and myelopathic features, evolution of CSF findings, and failure to respond to antibacterial therapy, a diagnosis of tuberculous meningitis with associated tuberculous myelo-arachnoiditis was established following multidisciplinary discussion.

The patient was initiated on antitubercular therapy (ATT) along with adjunctive corticosteroids. Following initiation of ATT, the patient demonstrated rapid clinical improvement, including defervescence, gradual improvement in sensorium, and stabilization of motor deficits. The marked therapeutic response further supported the diagnosis of tuberculous meningitis (Figure 2).

Discussion

Tuberculous meningitis (TBM) remains one of the most severe manifestations of extrapulmonary tuberculosis and continues to pose significant diagnostic and therapeutic challenges, particularly in the early stages of disease. The heterogeneity of clinical presentation, frequent absence of characteristic neuroimaging findings at presentation, and low microbiological yield from cerebrospinal fluid (CSF) samples often result in delayed diagnosis and treatment, contributing to high morbidity and mortality. This case illustrates an atypical yet clinically important presentation of TBM in a young immunocompetent female, complicated by early-onset quadriparesis and sixth cranial nerve palsy, despite initially non-conclusive investigations.

The neurological manifestations of TBM are primarily mediated by intense basal meningeal inflammation, formation of thick exudates, obliterative vasculitis, and immune-mediated neuronal injury. Cranial nerve involvement is a well-recognized feature, with the abducens nerve being most frequently affected due to its long intracranial course and vulnerability to compression

by inflammatory exudates in the basal cisterns. The development of sixth cranial nerve palsy in this patient is consistent with these mechanisms and often serves as a clinical marker of basal meningeal involvement.

The early development of quadriparesis in the present case is noteworthy and suggests extensive spinal axis involvement in the form of tuberculous myelo-arachnoiditis. Spinal involvement in TBM is relatively uncommon and frequently underdiagnosed, particularly in the absence of overt spinal symptoms at presentation. The pathophysiological mechanisms underlying tuberculous myelo-arachnoiditis include granulomatous inflammation of the arachnoid mater, spinal cord edema, obliterative endarteritis leading to ischemia, venous congestion, and formation of adhesions within the subarachnoid space. These processes culminate in progressive myelopathy, manifesting clinically as motor weakness, sensory deficits, and sphincter dysfunction. Early recognition is critical, as delayed treatment can result in irreversible neurological damage.

Neuroimaging plays a pivotal role in the evaluation of TBM; however, early imaging studies may be deceptively normal. Contrast-enhanced MRI of the brain and spine is considered the imaging modality of choice, yet radiological abnormalities such as basal meningeal enhancement, infarcts, hydrocephalus, or spinal cord involvement may be absent in the early phase of the disease. This radiological–clinical dissociation, as seen in the present case, can further complicate early diagnosis and emphasizes the need for a high index of clinical suspicion.

Microbiological confirmation of TBM remains challenging due to the paucibacillary nature of the infection. Conventional CSF smear microscopy and cultures have limited sensitivity, and nucleic acid amplification tests may also yield false-negative results. Although CSF adenosine deaminase (ADA) is frequently used as a supportive diagnostic marker, it lacks adequate sensitivity and specificity, particularly in early disease or immunocompetent individuals. Consequently, CSF biochemical parameters—namely markedly elevated protein levels, profound hypoglycorrhachia, and lymphocytic pleocytosis—continue to be of paramount diagnostic importance, especially in tuberculosis-endemic regions.

This case further underscores the dynamic nature of CSF findings in TBM. Initial CSF analysis may be non-conclusive, reflecting early or evolving disease, whereas repeat examination often reveals the characteristic biochemical profile of TBM. Therefore, repeat lumbar puncture should be strongly considered in patients with progressive neurological deterioration and inconclusive initial investigations.

Therapeutically, prompt initiation of antitubercular therapy, along with adjunctive corticosteroids, remains the cornerstone of management. Corticosteroids play a crucial role in reducing inflammatory exudates, cerebral edema, vasculitis, and spinal cord inflammation, thereby improving neurological outcomes and survival. The dramatic clinical improvement observed following initiation of antitubercular therapy in this patient further supports the diagnosis and highlights the importance of early empirical treatment when TBM is strongly suspected.

In conclusion, this case highlights the need for heightened clinical vigilance for TBM in patients presenting with subacute meningoencephalitis, particularly in endemic regions, even when initial CSF and imaging findings are inconclusive. Early recognition of spinal involvement and timely initiation of appropriate therapy are essential to prevent irreversible neurological sequelae and improve patient outcomes.

Epidemiology and burden of TBM have been widely reported.[1] WHO estimates continue to highlight the global burden of tuberculosis.[2] CNS tuberculosis pathogenesis has been extensively described.[3] Neuroendoscopy may be useful in TBM-associated hydrocephalus.[4] Uniform diagnostic criteria for TBM have been proposed.[5] WHO guidelines provide updated recommendations for drug-resistant tuberculosis treatment.[6]

Conclusion

Tuberculous meningitis remains a formidable diagnostic and therapeutic challenge owing to its heterogeneous clinical presentation, insidious onset, and frequent absence of definitive early radiological or microbiological evidence. This case highlights that TBM may present with atypical and rapidly progressive neurological manifestations, including early quadriparesis and cranial nerve palsies, even in young, immunocompetent individuals and in the absence of demonstrable neuroimaging abnormalities. Such presentations may lead to diagnostic uncertainty and delay in initiating appropriate therapy.

Spinal involvement in TBM, particularly tuberculous myelo-arachnoiditis, though relatively rare, should be actively considered in patients who develop evolving or unexplained motor deficits during the course of illness. Normal early neuroimaging does not exclude spinal

axis pathology, and clinical deterioration should prompt reassessment, repeat investigations, and reconsideration of differential diagnoses.

Cerebrospinal fluid biochemical analysis continues to serve as a cornerstone of diagnosis when imaging and microbiological studies are inconclusive. The dynamic evolution of CSF parameters underscores the importance of serial lumbar punctures in patients with worsening neurological status and initially non-conclusive findings. In tuberculosis-endemic regions, characteristic CSF biochemical patterns should carry significant diagnostic weight and may warrant early empirical initiation of antitubercular therapy.

Early recognition, meticulous serial neurological examination, and prompt initiation of antitubercular therapy with adjunctive corticosteroids are essential to mitigate inflammatory injury, prevent irreversible neurological sequelae, and improve functional outcomes.

Future Recommendations

Future clinical practice should emphasize the development of standardized diagnostic algorithms for suspected tuberculous meningitis, particularly in patients with atypical presentations or non-diagnostic initial investigations. Greater emphasis should be placed on early recognition of spinal axis involvement, with a low threshold for spinal imaging and repeat CSF evaluation in patients exhibiting progressive motor deficits.

There is a pressing need for more sensitive and rapid diagnostic biomarkers, including advanced molecular techniques and immunological assays, to facilitate earlier and more reliable diagnosis of TBM. Prospective studies evaluating the role of serial CSF analysis, early spinal MRI, and optimal duration and dosing of adjunctive corticosteroids in TBM-associated myelo-arachnoiditis are warranted.

Finally, increased clinician awareness and multidisciplinary collaboration are essential to ensure timely diagnosis and treatment, particularly in tuberculosis-endemic settings, where early empirical therapy based on clinical and biochemical evidence may significantly reduce morbidity and long-term neurological disability.

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