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Contrasting Neurological Presentations of Tuberculosis: Based on Two Clinical Cases (Inflammatory Myelopathy Secondary to Tuberculous Spondylitis and TB-Associated Cerebral Vasculitis)

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Abstract

Background: Tuberculosis (TB) is a multisystem infectious disease with protean manifestations. Although pulmonary TB is most common, involvement of the central nervous system (CNS) is rare and potentially devastating. Neurological complications may occur due to direct infection, immune-mediated inflammation, or vascular involvement.

Objective: To describe and analyze two uncommon neurological presentations of TB—non-compressive inflammatory myelopathy secondary to tuberculous spondylitis and TB-associated cerebral vasculitis—and to emphasize the importance of early recognition and treatment.

Methods: Two patients with active TB presenting with acute neurological deficits were evaluated clinically and radiologically using MRI of the spine and brain. Laboratory investigations and chest imaging supported the diagnosis of TB. Both patients received standard anti-tuberculosis therapy (ATT) with adjunctive corticosteroids.

Results:

- *Case 1: A 50-year-old man with pulmonary TB developed acute flaccid paraplegia with sensory and autonomic involvement. MRI spine revealed L1 vertebral compression fracture consistent with TB spondylitis, without direct spinal cord compression. Neurological deficits were attributed to inflammatory spinal cord involvement. Significant neurological recovery followed ATT and corticosteroid therapy.*

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- *Case 2: A 25-year-old man presented with seizures, altered sensorium, and right hemiparesis. MRI brain demonstrated multifocal ischemic infarcts. HRCT chest confirmed active pulmonary TB. TB endarteritis causing cerebral vasculitis was diagnosed. Combined ATT and corticosteroids led to marked clinical improvement.*

Conclusion: TB can cause rare but severe neurological complications through inflammatory and vascular mechanisms. Early diagnosis and combined anti-tubercular and anti-inflammatory therapy are crucial to prevent permanent neurological disability.

Keywords: *Tuberculosis, inflammatory myelopathy, cerebral vasculitis, TB endarteritis, spinal cord involvement*

Introduction

Tuberculosis remains a major global health problem, particularly in developing countries. While pulmonary TB accounts for the majority of cases, extrapulmonary TB contributes significantly to morbidity and mortality (Török ME, et al, 2024). Central nervous system involvement occurs in approximately 1–5% of TB cases and includes tuberculous meningitis, tuberculomas, spinal TB, and less commonly, immune-mediated myelopathy and cerebral vasculitis (Alberto Ortega-Rosales et al,2019).

Neurological manifestations of TB may result from direct infection, mechanical compression, immune-mediated inflammation, or vascular involvement (Huan Nie et al, 2025). Non-compressive inflammatory myelopathy and TB-associated cerebral vasculitis are particularly rare and often underrecognized, leading to delayed diagnosis and poor outcomes. We report two such cases with favorable recovery following timely intervention (Javaud N, et al ,2011).

Case Presentations

Case 1: Inflammatory Myelopathy Secondary to Tuberculous Spondylitis

A 50-year-old man with recently diagnosed sputum-positive pulmonary TB presented with acute onset weakness of both lower limbs for 5 days. He reported progressive difficulty in walking, numbness below the umbilicus, and urinary retention. There was no history of trauma.

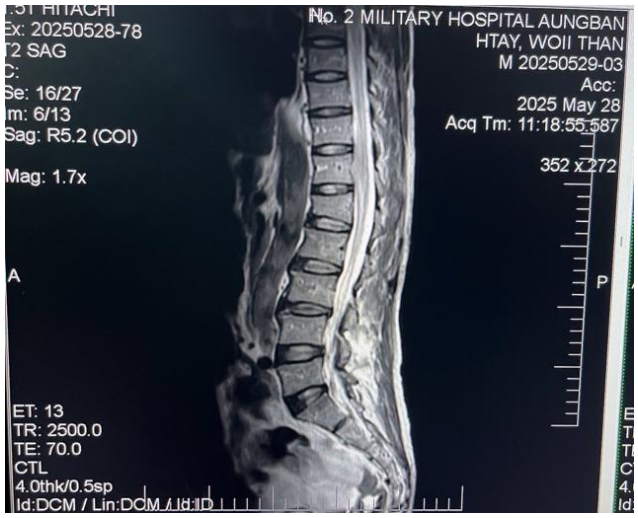
Examination

- Conscious and oriented
- Motor: Flaccid paraplegia (MRC grade 0/5)
- Sensory: Loss of pain and temperature below T10 level
- Reflexes: Absent deep tendon reflexes in lower limbs
- Autonomic: Bladder and bowel involvement

Investigations

MRI of the lumbar spine revealed:

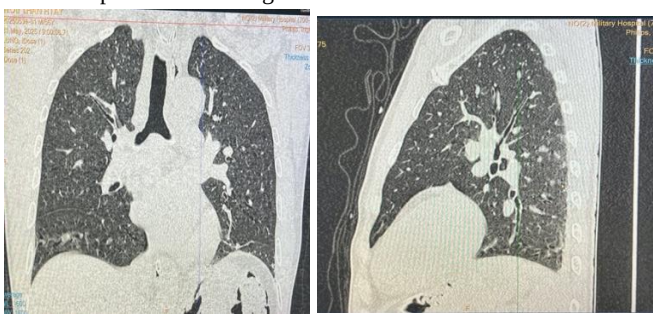
- Compression fracture of the L1 vertebral body
- Hypointense signals on T1 and hyperintense signals on T2/STIR sequences consistent with tuberculous spondylitis
- No evidence of direct spinal cord or nerve root compression
- spine-Severe wedge compression fracture of L1 vertebral body &Partial disc desiccation and minimal facet joint effusion



Laboratory findings showed elevated ESR and CRP. HIV testing was negative.

HRCT chest showed:

- consistent with active pulmonary TB with bilateral basal pleural thickening



Diagnosis

Inflammatory myelopathy secondary to tuberculous spondylitis (non-compressive).

Treatment and Outcome

The patient was treated with:

- Standard four-drug ATT (isoniazid, rifampicin, pyrazinamide, ethambutol)
- High-dose intravenous corticosteroids followed by oral taper

Over 6 weeks, he showed marked improvement in motor strength (MRC 4/5), sensory function, and bladder control.

Case 2: TB-Associated Cerebral Vasculitis

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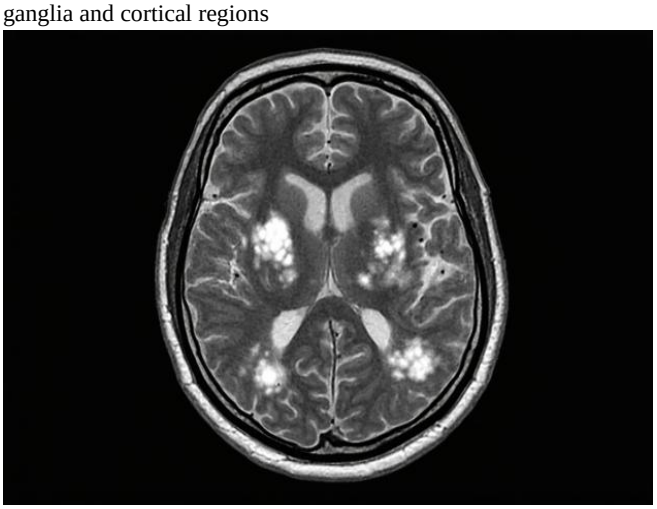
A 25-year-old man with no known comorbidities presented with recurrent generalized tonic–clonic seizures, confusion, and sudden onset weakness of the right side of the body for 3 days. There was a history of low-grade fever, weight loss, and chronic cough.

Examination

- Altered sensorium
- Right-sided hemiparesis (MRC grade 3/5)
- Upper motor neuron signs on the right
- No meningeal signs

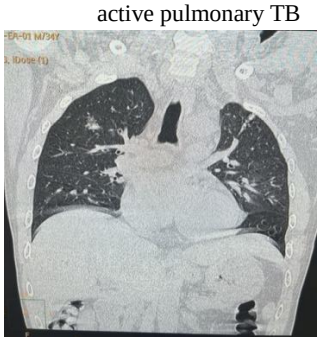
Investigations

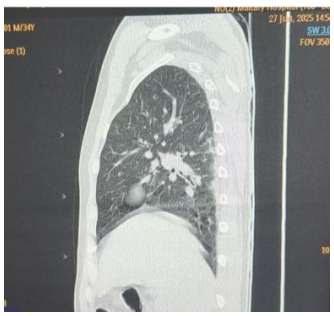
MRI brain revealed:
Multiple acute and subacute infarcts involving bilateral basal ganglia and cortical regions



HRCT chest showed:

- Tree-in-bud nodules and cavitary lesions consistent with active pulmonary TB





CSF analysis was unremarkable, excluding meningitis.

Diagnosis

TB endarteritis causing cerebral vasculitis with ischemic strokes.

Treatment and Outcome

The patient received:

- Standard ATT
- Adjunctive corticosteroids
- Antiepileptic drugs

Over 4 weeks, there was significant improvement in sensorium, seizure control, and motor power (MRC 4+/5).

Discussion

Neurological complications of TB can occur through diverse mechanisms. In spinal TB, neurological deficits are usually attributed to mechanical compression by abscesses or vertebral collapse. However, inflammatory myelopathy without direct compression, as seen in Case 1, is rare and likely immune-mediated (Khatiwada A et al,2024).

TB-associated cerebral vasculitis results from granulomatous inflammation of cerebral vessels, leading to luminal narrowing, thrombosis, and infarction. Infarcts commonly involve the basal ganglia and internal capsule, as observed in Case 2. Early imaging is crucial, as CSF findings may be normal (*Ann Indian et al, 2023).*

Corticosteroids play a key role in reducing inflammation and preventing irreversible neuronal damage when used alongside ATT (Schaller MA et al,2019).

Conclusion

Tuberculosis should be considered in the differential diagnosis of unexplained myelopathy or ischemic stroke in young patients, especially in TB-endemic regions. Recognition of inflammatory and vascular mechanisms is essential. Prompt initiation of ATT combined with corticosteroids can result in favorable neurological outcomes.

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