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Spinal Tuberculosis as a Neurological Masquerader: A Clinicoradiological Case Series of Ten Patients

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Abstract: ***Background: Spinal tuberculosis is classically recognized as tuberculous spondylodiscitis, but neurological disease may arise from vertebral, epidural, meningeal, radicular, or intramedullary involvement and may imitate inflammatory, neoplastic, or peripheral nerve disorders. Objective: To describe the clinicoradiological spectrum of spinal tuberculosis encountered in a tertiary neurology service and emphasize patterns that created diagnostic mimicry. Methods: This descriptive case series included ten patients evaluated at a tertiary care centre between July 2025 and July 2026. Clinical localization and available spinal MRI were reviewed. Cases were classified on the basis of the documented clinical context, characteristic imaging pattern, evidence of tuberculosis at spinal or extra-spinal sites when available, treatment course, and follow-up information. Results: The series encompassed classical thoracic destructive spondylitis with compressive myelopathy, early vertebral disease, vertebral collapse with instability, acute thoracic compressive myelopathy, lumbar spondylodiscitis with paraspinal abscess presenting as cauda equina syndrome, intramedullary tuberculoma, longitudinally extensive myelopathic/meningeal disease, a lesion mimicking progression of multiple myeloma, disease mimicking inflammatory spondyloarthropathy, and a painful areflexic polyradiculoneuropathy mimicking chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). Conclusion: Spinal tuberculosis is anatomically and clinically heterogeneous. Recognition of the involved compartment and attention to discordant features - such as paravertebral collections in an apparent malignancy, meningeal abnormalities in longitudinal myelopathy, or severe radicular pain with active tuberculosis in a CIDP-like syndrome - can reduce diagnostic anchoring. Microbiological or histopathological confirmation should be pursued whenever feasible, particularly when the presentation is atypical.***

Keywords: spinal tuberculosis; tuberculous spondylitis; myelopathy; cauda equina; intramedullary tuberculoma; arachnoiditis; polyradiculoneuropathy; CIDP mimic

1. Introduction

Tuberculosis remains an important cause of spinal infection and neurological disability. The usual radiological paradigm is paradiscal vertebral disease with endplate destruction, disc involvement, paravertebral or epidural collection, vertebral collapse, and deformity. Magnetic resonance imaging is the principal modality for defining vertebral marrow disease, soft-tissue extension, epidural involvement, spinal cord compression, and intradural disease.

Neurological tuberculosis, however, is not confined to Pott disease. The spinal cord, meninges, nerve roots, and intramedullary compartment may be involved, and the resulting syndromes can resemble demyelinating myelitis, neoplasm, inflammatory spondyloarthropathy, or inflammatory polyradiculoneuropathy. In a tuberculosis-endemic setting, these overlaps create a recurrent diagnostic problem: excessive reliance on epidemiology risks empirical overdiagnosis, whereas excessive reliance on a competing radiological label can delay treatment of tuberculosis.

We present ten patients illustrating this spectrum. The purpose is not to propose a new disease entity, but to demonstrate how a familiar infection can produce different neurological localizations and clinicoradiological mimics.

2. Materials and Methods

This descriptive case series included ten patients evaluated in the Department of Neurology, Osmania Medical College and Osmania General Hospital, Hyderabad, between July 2025 and July 2026. Clinical records and available neuroimaging were reviewed with emphasis on symptom evolution, neurological localization, MRI pattern, relevant extra-spinal evidence of tuberculosis, treatment, and documented outcome.

Because microbiological or histopathological confirmation was not available uniformly across the series, diagnostic wording was deliberately graded. Cases with characteristic spinal imaging and a compatible clinical/tuberculosis context were described as clinicoradiologically consistent with or probable spinal tuberculosis rather than microbiologically proven disease. Atypical cases were retained because their principal value was the diagnostic masquerade. No missing clinical, pathological, or microbiological data were imputed.

Written informed consent for use of anonymized clinical information and images for academic publication was obtained. Institutional ethics committee approval was not obtained for this descriptive retrospective case series. All figures used in the manuscript were de-identified.

3. Results

The ten patients demonstrated involvement across the vertebral, epidural, intramedullary, meningeal, and radicular compartments. Presentations ranged from classical compressive myelopathy to cauda equina syndrome and a CIDP-like polyradiculoneuropathy. The principal clinicoradiological features are summarized in Table 1.

Case	Age/Sex	Localization	Key clinical features	Key imaging/diagnostic clue	Principal mimic
1	47/F	Thoracic compressive myelopathy	Chronic mid-back pain, fever, paraplegia, T6 sensory level, bladder involvement	Destructive thoracic spondylitis with paravertebral/epidural disease and cord compression	Classical Pott disease
2	26/F	Vertebral disease without neurological deficit	Six months low-back pain and low-grade evening fever	Early vertebral/endplate marrow abnormality with relative disc preservation	Brucellosis / pyogenic infection
3	19/F	Lumbar compressive/instability syndrome	Back pain followed by progressive weakness	Advanced lumbar vertebral collapse and inflammatory change; L3 laminectomy with L2-L4 fixation documented	Mechanical collapse / other infection
4	40/M	Acute thoracic compressive myelopathy	Pulmonary TB; rapidly progressive spastic paraplegia over 10 days; T4 level	Thoracic epidural/vertebral disease with marked cord compression	Inflammatory acute myelopathy / pyogenic spondylitis
5	56/M	Cauda equina syndrome	Radicular low-back pain, bilateral leg weakness, hyporeflexia, dermatomal sensory loss, urinary frequency	L4-L5 spondylodiscitis with multiple paraspinal/psoas collections	Pyogenic, brucellar or fungal spondylodiscitis; metastasis
6	35/M	Cervical intramedullary syndrome	RVD positive on ART; previous pulmonary TB; focal seizures and progressive spastic quadriparesis	Focal ring-enhancing intramedullary cervical cord lesion with surrounding cord abnormality	Intramedullary neoplasm / demyelination / other infection
7	29/M	Longitudinal myelopathy with meningeal involvement	Progressive paraparesis, sensory symptoms and bladder dysfunction	Long-segment cord signal abnormality with associated meningeal/intradural abnormalities in source imaging	NMOSD / MOGAD / other infective myelitis
8	30/M	Thoracic vertebral lesion in known multiple myeloma	PET-active thoracic lesion raised concern for myeloma progression	Destructive discovertebral lesion with paravertebral/epidural soft-tissue component	Myeloma progression
9	61/M	Cervical myelopathy	Background psoriasis	C7-D1 endplate erosive disease with paraspinal soft-tissue extension and canal involvement in source imaging	Spondyloarthropathy / degenerative disease
10	20/M	Painful sensorimotor polyradiculoneuropathy	Severe bilateral radicular pain followed by progressive proximal and distal weakness, wasting and generalized hyporeflexia/areflexia	Lumbosacral root enhancement; CT chest with tree-in-bud change and bilateral upper-lobe cavitary consolidation; CSF protein 250 mg/dL, 1 cell	CIDP / nodopathy / vasculitic or infiltrative neuropathy

4. Case Vignettes

Case 1 - Classical thoracic Pott disease

A 47-year-old woman had six months of mid-back pain and intermittent fever followed by two months of progressive paraparesis, sensory loss to approximately T6, and bladder symptoms. Examination documented severe bilateral lower-limb weakness, absent knee and ankle reflexes, mute plantar responses, and thoracic vertebral tenderness. MRI demonstrated destructive thoracic spondylitis with a paravertebral/epidural soft-tissue component and marked spinal canal compromise, producing a classical compressive phenotype.

Case 2 - Early vertebral tuberculosis

A 26-year-old woman presented with six months of low-back pain, including pain at rest, and low-grade fever with evening rise. She had no focal neurological deficit or sphincter symptoms. MRI showed early vertebral/endplate inflammatory change with relative preservation of disc architecture. The differential included brucellosis and pyogenic spondylodiscitis; the pattern was considered compatible with early tuberculous vertebral disease in the documented clinical context.

Case 3 - Vertebral collapse with instability

A 19-year-old woman developed back pain followed by progressive weakness with mechanical instability. Imaging documented advanced lumbar vertebral collapse with inflammatory change and compressive features. She underwent L3 laminectomy with L2-L4 fixation. This case represented the destructive/instability end of the vertebral tuberculosis spectrum.

Case 4 - Rapidly progressive thoracic compressive myelopathy

A 40-year-old man with pulmonary tuberculosis, started on antitubercular treatment 15 days earlier, developed rapidly progressive bilateral lower-limb weakness over ten days. Examination showed complete paraplegia, lower-limb spasticity, brisk knee reflexes, ankle clonus, extensor plantar responses, and a T4 sensory level. MRI demonstrated thoracic vertebral/epidural disease with severe cord compression. The rapid course emphasized that an apparently acute myelopathy may still be structural and infective.

Case 5 - Cauda equina syndrome with lumbar spondylodiscitis

A 56-year-old man presented with low-back pain radiating to both thighs, progressive bilateral leg weakness, buckling, and urinary frequency. Lower-limb hypotonia and hyporeflexia were accompanied by L4-L5-S1 sensory

abnormalities and a positive straight-leg-raising test, localizing clinically to the cauda equina. MRI showed L4-L5 spondylodiscitis with multiple collections involving the paraspinal musculature and psoas region. The principal competing diagnoses were pyogenic, brucellar, and fungal spondylodiscitis and neoplastic disease.

Case 6 - Intramedullary tuberculoma

A 35-year-old man living with HIV and receiving antiretroviral therapy, with a past history of pulmonary tuberculosis, developed right focal seizures followed by progressive weakness and stiffness involving all four limbs. Examination showed spastic quadriparesis, generalized hyperreflexia, ankle clonus, and Hoffmann signs. MRI demonstrated a focal ring-enhancing intramedullary cervical cord lesion with surrounding cord abnormality. In the documented tuberculosis context, an intramedullary tuberculoma was favored over intramedullary neoplasm, demyelination, or another opportunistic infection.

Case 7 - Longitudinal myelopathy with meningeal/ arachnoid involvement

A 29-year-old man developed progressive paraparesis with sensory and bladder involvement. The initial differential included NMOSD, MOGAD, and infective myelitis. Source imaging documented long-segment cord T2 signal abnormality with associated dural/meningeal and intradural abnormalities. The coexistence of longitudinal cord disease and meningeal pathology favored an infective meningomyelitic/arachnoid process over an isolated primary demyelinating myelitis.

Case 8 - Tuberculosis mimicking progression of multiple myeloma

In a 30-year-old patient with established multiple myeloma, a metabolically active thoracic spinal lesion raised concern for oncological progression. MRI, however, showed a destructive discovertebral process with a sizeable paravertebral/epidural soft-tissue component. The morphology prompted consideration of infection rather than automatic attribution to myeloma. This case illustrates the danger of diagnostic anchoring in a patient with a known malignancy; microbiological or histopathological confirmation is particularly important before escalation of oncological therapy.

Case 9- Tuberculous spondylitis mimicking inflammatory spondyloarthropathy

A 61-year-old man with psoriasis presented with cervical myelopathy. Source imaging documented C7-D1 endplate erosive disease with paraspinal soft-tissue extension and enhancement extending toward the spinal canal. Because the background psoriasis could bias interpretation toward inflammatory spondyloarthropathy, the associated soft-tissue component was an important clue to an infective process.

Case 10 - Tuberculosis-associated polyradiculoneuropathy mimicking CIDP

A 20-year-old man developed severe bilateral lumbosacral radicular pain followed by progressive lower-limb and subsequently upper-limb weakness, involving proximal and distal muscles, with truncal weakness and wasting. Examination showed generalized hypotonia, reduced upper-limb reflexes, absent knee and ankle reflexes, mild distal proprioceptive/vibration impairment, and a positive straight-leg-raising test. CSF showed albuminocytological dissociation (protein 250 mg/dL, 1 cell; ADA 2). MRI documented lumbosacral root enhancement. CT chest demonstrated a tree-in-bud pattern with cavitary consolidation in the posterior right upper lobe and apicoposterior left upper lobe, reported as likely tuberculosis; a brain tuberculoma was also documented in the source case record. CIDP was a compelling mimic because of the proximal-distal weakness, areflexia, root enhancement, and albuminocytological dissociation. Severe pain, asymmetry/wasting, constitutional features, and active tuberculosis argued for an infective/inflammatory association. He received a five-day course of IVIG together with antitubercular therapy, dexamethasone, physiotherapy, and nutritional support; he walked with support by two weeks and independently by one month. Because both IVIG and antitubercular therapy were administered, recovery cannot be attributed to either mechanism alone; the case is therefore described as probable tuberculosis-associated polyradiculoneuropathy rather than proven tuberculous radiculitis.

5. Discussion

This series demonstrates that spinal tuberculosis is best approached anatomically before it is approached etiologically. Vertebral and epidural disease produces the familiar compressive myelopathy, whereas intramedullary disease produces a focal cord syndrome, meningeal disease produces longitudinal myelopathy and arachnoid/radicular abnormalities, and root involvement may produce a lower-motor-neuron polyradiculoneuropathy. The same infection can therefore occupy different neurological compartments and generate very different examination findings.

Classical tuberculous spondylitis usually involves adjacent vertebral bodies and may progress to disc destruction, collapse, kyphosis, paravertebral cold abscess, and epidural extension. MRI is particularly valuable for defining marrow involvement, soft-tissue spread, epidural disease, cord compression, and noncontiguous lesions. Compared with pyogenic infection, a relatively indolent course, thoracic predilection, smooth thin-walled paravertebral collections, multilevel involvement, and delayed disc destruction may support tuberculosis, but none of these features is individually pathognomonic.

The intramedullary case extends the spectrum beyond vertebral disease. Intramedullary tuberculoma is uncommon and can resemble neoplasm or demyelination. A T2 iso-/hypointense or target-like center in a caseating lesion with ring enhancement can be suggestive, but interpretation must remain integrated with immune status, extra-neural tuberculosis, and competing infections.

The longitudinal myelopathy case highlights another common diagnostic trap. Longitudinally extensive cord signal abnormality often triggers an NMOSD/MOGAD framework. Associated meningeal enhancement, intradural abnormalities, CSF loculation, or root involvement should broaden the differential toward infection, granulomatous

inflammation, and neoplastic meningitis. In tuberculosis-endemic regions, tuberculous meningomyelitis and arachnoiditis remain important considerations.

The malignancy mimic is clinically important because fluorodeoxyglucose avidity and vertebral destruction are not specific for cancer. In a patient with known multiple myeloma, a new spinal lesion can be prematurely labelled relapse. Disc-endplate involvement and a prominent paravertebral collection should prompt an infective differential. Tissue diagnosis is especially valuable when treatment pathways diverge substantially.

Case 10 represents a different type of masquerade. Albuminocytological dissociation and cauda equina root enhancement are compatible with CIDP but are not specific to it. Severe radicular pain, wasting, asymmetry, systemic clues, and active tuberculosis should trigger reconsideration of an infectious or infiltrative polyradiculoneuropathy. Importantly, the patient received both IVIG and antitubercular therapy with corticosteroids; therefore the case supports an association and a diagnostic warning rather than proof that tuberculosis directly caused the demyelinating phenotype.

The practical lesson is to avoid two opposite errors. First, not every destructive spinal lesion in a tuberculosis-endemic setting is tuberculosis; pyogenic infection, brucellosis, fungal infection, metastasis, lymphoma, and inflammatory disease remain important alternatives. Second, tuberculosis should not be excluded merely because the syndrome resembles a well-recognized noninfectious neurological disorder. A compartment-based localization followed by targeted microbiology/histopathology, where feasible, is the safer strategy.

6. Limitations

This is a small, single-centre descriptive case series selected to illustrate clinicoradiological diversity and is not suitable for estimating prevalence or diagnostic accuracy. Microbiological or histopathological confirmation was not uniformly available. Imaging was obtained during routine clinical care and the available publication images were derived from stored clinical images/films rather than a standardized research imaging protocol. Follow-up duration was not uniform. In Case 10, simultaneous IVIG, corticosteroid, and antitubercular therapy prevents attribution of clinical improvement to a single treatment. These limitations require cautious use of causal language.

7. Conclusion

Spinal tuberculosis can present far beyond classical Pott disease. In this series it involved vertebral, epidural, intramedullary, meningeal, and radicular compartments and mimicked demyelinating myelitis, malignancy, inflammatory spondyloarthropathy, and CIDP. The most reliable approach is neurological localization first, pattern recognition second, and etiological confirmation whenever feasible. Recognizing discordant clinical and imaging clues may prevent both delayed tuberculosis treatment and inappropriate treatment of a mimic.

8. Learning Points

1) Localize the neurological syndrome before assigning the etiology: spinal tuberculosis can involve bone, epidural space, cord, meninges, and nerve roots.
2) Paravertebral or epidural soft-tissue disease accompanying a destructive vertebral lesion should keep infection in the differential even in a patient with known malignancy.
3) Longitudinally extensive myelopathy with meningeal, intradural, or root abnormalities is not a typical isolated demyelinating pattern and should prompt evaluation for infection and granulomatous disease.
4) Root enhancement and albuminocytological dissociation are not specific for CIDP; marked radicular pain, wasting, asymmetry, and systemic clues should trigger a search for infectious or infiltrative causes.
5) Microbiological or histopathological confirmation should be pursued whenever feasible, particularly in atypical presentations or when treatment pathways differ substantially.

Figures

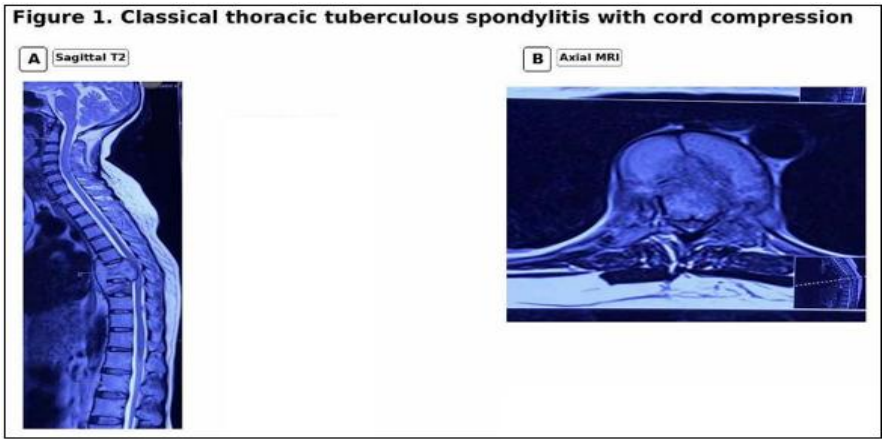

Figure 1: Classical thoracic tuberculous spondylitis with compressive myelopathy. Composite MRI demonstrates destructive mid-thoracic spondylitis with vertebral collapse, angular deformity, pre/paravertebral soft-tissue disease and severe spinal canal compromise with cord compression; the axial panel demonstrates associated canal compromise/epidural extension

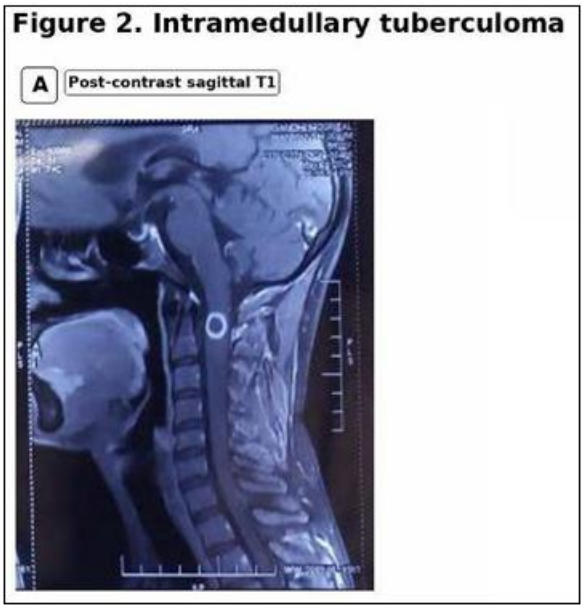

Figure 2: Intramedullary tuberculoma. Post-contrast sagittal T1-weighted MRI demonstrates a focal ring-enhancing intramedullary cervical cord lesion. In the documented clinical and tuberculosis context, the morphology was considered suggestive of intramedullary tuberculoma.

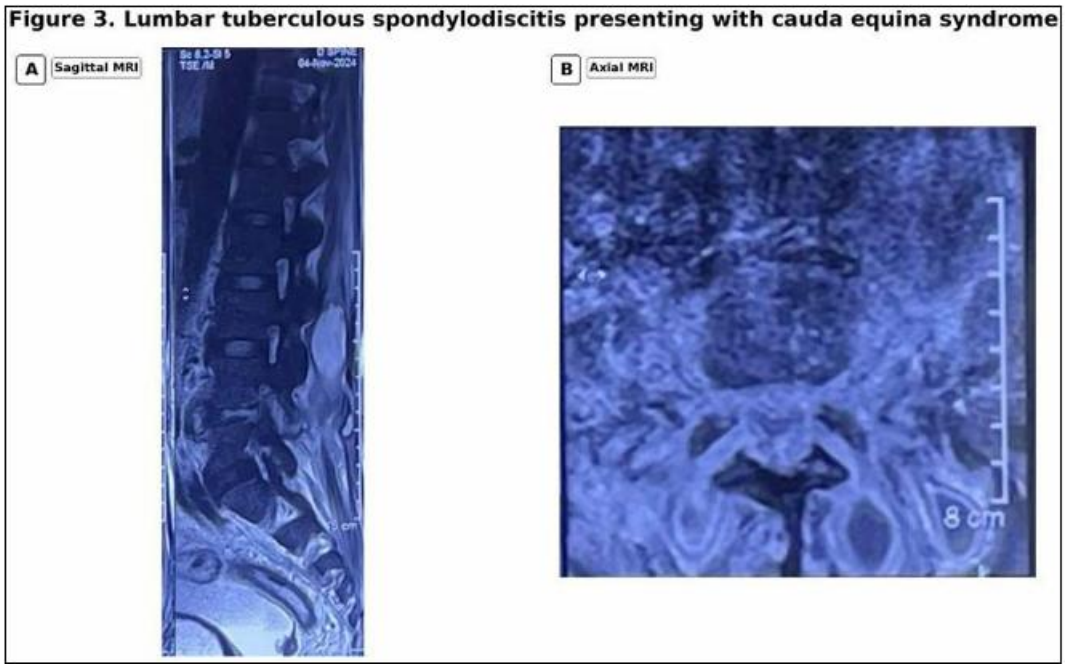

Figure 3: Lumbar tuberculous spondylodiscitis presenting with cauda equina syndrome. Sagittal and axial MRI demonstrate lower lumbar spondylodiscitis with extensive paravertebral soft-tissue involvement and a large paraspinal/psoas-region collection. The patient clinically localized to the cauda equina.

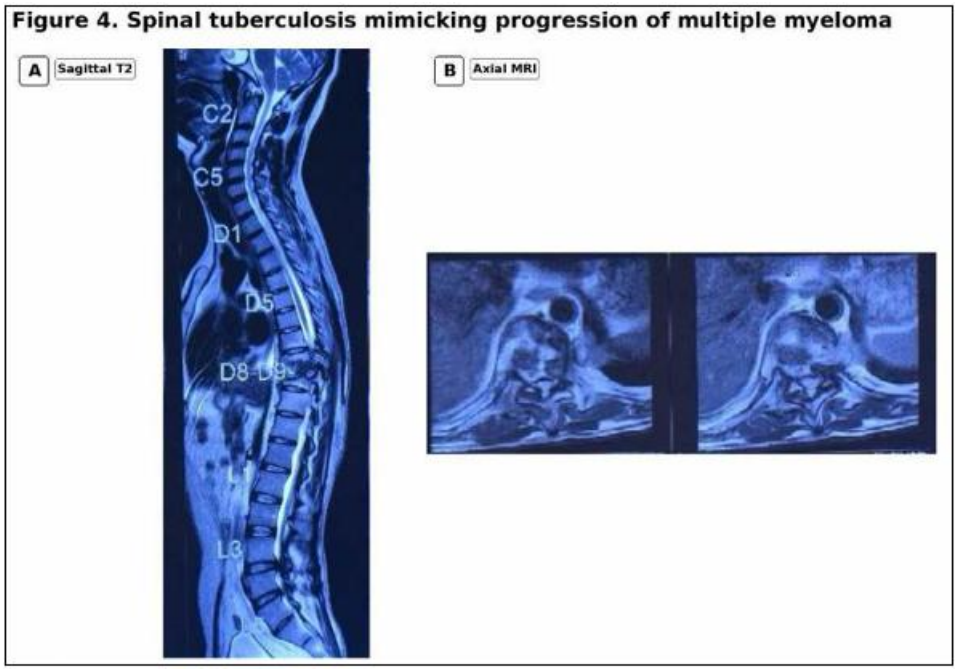

Figure 4: Spinal tuberculosis mimicking progression of multiple myeloma. Sagittal and axial MRI demonstrate a destructive thoracic discovertebral lesion with an associated paravertebral/epidural soft-tissue component. In a patient with established multiple myeloma, the lesion initially raised concern for malignant progression; the morphology prompted consideration of infection

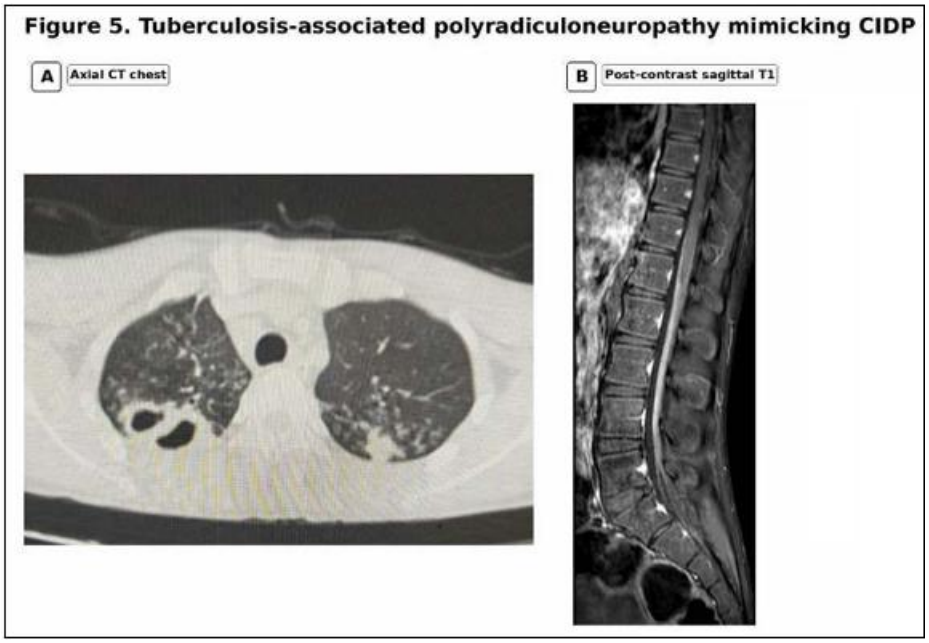

Figure 5: Tuberculosis-associated polyradiculoneuropathy mimicking CIDP. Axial CT chest demonstrates upper-lobe predominant cavitary pulmonary disease with surrounding centrilobular/tree-in-bud-type nodularity. Post-contrast sagittal lumbosacral MRI demonstrates enhancement along the cauda equina/root region. The clinical phenotype was a painful progressive areflexic sensorimotor polyradiculoneuropathy with albuminocytological dissociation.

Declarations

Ethics approval: Institutional ethics committee approval was not obtained for this descriptive retrospective case series.

Consent for publication: Written informed consent for publication of anonymized clinical information and images was obtained.

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Conflict of interest: The authors declare no conflict of interest.

Data availability: The clinical data underlying this case series are contained within the article; additional patient-level data are not publicly shared to protect confidentiality.

Author contributions: Dr. Rutuja Gajbhar contributed to case identification, clinical data compilation, literature review, manuscript drafting, and figure preparation. Dr. N.

Veena supervised the clinical work, critically revised the manuscript, and approved the final version.

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