NEUROSCIENCE RESEARCH NOTES

OPEN ACCESS | CASE NOTES ISSN: 2576-828X

Clinically confirmed tuberculous radiculomyelitis: a case report

Mohammad Kurniawan*, Tiara Kasih, David Pangeran and Kartika Maharani

Department of Neurology, Faculty of Medicine, Universitas Indonesia/Dr.Cipto Mangunkusumo Hospital, Jakarta, Indonesia
* Correspondence: m.kurniawan@ui.ac.id; Tel.: +62 813-3314-2408

Received: 26 Deecember 2024; **Accepted:** 7 September 2025; **Published:** 29 December 2025
Edited by: Battuvshin Lkhagvasuren (Mongolian National University of Medical Sciences, Mongolia)
Reviewed by: Hui Jan Tan (Universiti Kebangsaan Malaysia Medical Centre, Malaysia);
Nagarjuna Narayanasetti (Manav Rachna International Institute of Research and Studies, India);
Oyungerel Ravjir (Mongolian National University of Medical Sciences, Mongolia);
Siti Sarahdeaz Fazzaura Putri (Sriwijaya University, Indonesia).
https://doi.org/10.31117/neuroscirn.v8i4.421

Abstract: Tuberculous radiculomyelitis (TBRM) is a rare complication of tuberculosis which affects the nervous system. Common symptoms associated with TBRM include paraparesis or quadriparesis, urinary retention or constipation, and paraesthesia in the lower extremities. The ascending symptoms are often described as similar to Guillain Barre syndrome. A 21-year-old woman experienced progressive weakness from her lower to upper extremities, which eventually led to complete immobility within two days and was accompanied by autonomic dysfunction. Lumbar puncture demonstrated yellow and clear cerebrospinal fluid (CSF) with pleocytosis (214 cells/uL), neutrophil predominance (81%), increased protein (429mg/dL), and decreased CSF/serum glucose ratio (7 mg/dL vs. 159.3 mg/dL). Cervicothoracic MRI examination revealed multifocal hyperintense lesions with indistinct borders at the T5-T12 level and central predominance. These results indicate tuberculosis as the aetiology. This case illustrates the atypical manifestations of TB within the nervous system. In highly endemic countries like Indonesia, TB should be considered as one of the potential causes in the differential diagnosis of any progressive weakness involving the nervous system. The limited availability of high-sensitivity diagnostic tests for detecting *Mycobacterium tuberculosis* in the central nervous system remains a significant challenge.

Keywords: Spinal tuberculosis; CSF biomarkers in tuberculous radiculomyelitis; Guillain-Barré mimic; Radiculomyelitis.

1.0 INTRODUCTION

In the era of disease evolution towards the dominance of non-communicable disease, tuberculosis (TB) remains an infectious problem that cannot be completely eradicated. Tuberculosis is the second-highest cause of mortality in the world due to infectious diseases after COVID-19 (Mancuso et al., 2023). TB in Indonesia is the second highest after India, with 1,090,000 cases and 125,000 deaths per year (WHO, 2024). The composition of *Mycobacterium tuberculosis* (MTB) wall derived from lipoarabinomannan gives TB acid-resistant properties so that the bacteria can avoid

elimination both by enzymes in leukocytes and by antibiotics in general (Kalscheuer et al., 2019). In addition, tuberculosis can remain dormant in leukocytes and spread systemically through the bloodstream or lymphatics via leukocytes. Up to 15% of pulmonary tuberculosis cases result in extrapulmonary dissemination, with the highest incidence in the pleura, musculoskeletal system, gastrointestinal tract, peripheral lymphatic glands, genitourinary system, central nervous system, and skin (Gopalaswamy et al., 2021; Moule et al., 2020).

While not a primary extracranial dissemination, TB infection in the central nervous system (CNS) leads to higher mortality rates (22.8%) and increased morbidity in the form of residual neurological symptoms (28.7%) (Wen et al., 2019). These CNS manifestations include TB meningitis and TB myelitis. Since the blood-brain barrier protects the CNS, susceptibility to this infection is primarily observed in immunodeficiency cases and in children under 5 years old (Navorro-Flores et al., 2022).

Some of the challenges in the management of CNS TB infection include community and health worker recognition of symptoms and TB detection. Manifestations of TB can vary, and infection in the current episode may represent reactivation of TB, making it possible that pulmonary symptoms and evidence of TB are not always found. Symptoms may present as radicular pain, characterised by a sharp, shooting pain that follows the course of the nerve root. This pain is frequently accompanied by paresthesias, abnormal sensations such as tingling or numbness that indicate nerve dysfunction (Dydyk and Singh, 2022). In addition, there can also be areflexia, hypotonia, and muscle atrophy, which can lead to weakness and even paralysis due to denervation and lack of nerve input to the muscles. Autonomic involvement may cause bladder and bowel dysfunctions (Garg et al., 2023). This has led to TB being long recognised as the great imitator (Prapruttam et al., 2014).

Magnetic resonance imaging (MRI) and computed tomography (CT) are used for early detection of TBRM and are further confirmed through cerebrospinal fluid (CSF) analysis, acid-fast bacillus (AFB) culture, and polymerase chain reaction (PCR). The Xpert MTB/RIF assay on CSF samples demonstrates a sensitivity of 54% and a specificity of 93.8%. Recent studies on early TB detection highlight the use of the Xpert MTB/RIF Ultra assay and the TB lipoarabinomannan (LAM) antigen (Ag) test. These diagnostic tools can identify TB in both pulmonary and extrapulmonary specimens using different methodologies. In the brain and spinal cord, detection of TB is more difficult because the CSF is sterile. Severe symptoms can occur despite a low bacterial load of MTB, and standard diagnostic tests may result in negative findings (Alomatu et al., 2023; Chen et al., 2020; WHO, 2021). Delay in the diagnosis and treatment of CNS tuberculosis can lead to severe complications and permanent neurological damage (Gupta et al., 2025). However, immune reconstitution syndrome is often reported in CNS TB infection in the recovery process, so this case is not limited to the infectious aspect, but also the immunologic aspect.

2.0 CASE ILLUSTRATION

2.1 Clinical presentation and physical findings

A 21-year-old woman consulted a neurologist for weakness in both limbs that began in the preceding 2 days. The first symptoms started three weeks ago with intermittent low-grade fever, diarrhoea and weakness of the whole body. She was admitted to another hospital with a diagnosis of typhoid fever and treated for five days. After the treatment, she started to feel improvements in her fever and diarrhea but still felt weak throughout her body.

One week ago, she had recurrent fever accompanied by reddish spots on both palms, both arms, both sides of the face, and both feet and lower calves. The spots were itchy, but painless, non-indurated, non-festering, and non-bleeding. The patient was taken to the emergency department and was readmitted with typhoid fever. Three days ago, she felt burning pain and tingling in both limbs. The following day, the pain subsided, but she had numbness and weakness from toes to the thigh simultaneously. The weakness gradually worsened, culminating in complete immobility, accompanied by difficulty in breathing. She also began to have problems voiding and defecating. On the first day of monitoring, complaints began with weakness and numbness in the fingertips and toes. There were no cranial nerve disorder, imbalance, or seizures, along with a persistent cough, night sweats, or sudden weight loss in the past month.

She was physically active in her daily activities with no previous medical problems. Her mother had been diagnosed with systemic lupus erythematosus and tuberculous spondylitis postoperatively in 2016. She had no history of smoking, alcohol consumption, or hormonal contraceptive use, and her HIV status was negative.

Physical examination revealed stable vital signs and no wheezing, rales, or back tenderness. Dermatologic status showed multiple erythematous nodules on the palmar surfaces of both upper arms, neck, both sides of the face, and both plantar pedis, which were painless, without oedema, heat, or pus. She was fully alert, but with tetraparesis and sensory loss from L1 downwards, with urinary and bowel dysfunction. Physiological reflexes were also decreased in all extremities.

The prodromal features of diarrhea, followed by a predominance of ascending-type subacute weakness and decreased reflexes, raised suspicion for Guillain-Barré syndrome (GBS). However, the presence of autonomic involvement (urinary and bowel retention) raised suspicion for central aetiology, such as infectious or autoimmune myelitis.

2.2 Diagnostic workup

Laboratory examination showed leukopenia with neutrophil predominance, with a white blood cell (WBC) count of 1,780 cells/µL and neutrophilia of 81%. The chest radiograph was normal. **Figure 1** shows a normal chest X-ray (CXR), while sagittal T2-weighted MRI of the cervicothoracic region reveals multifocal hyperintense lesions with indistinct borders extending from T5 to T12, predominantly involving the central spinal cord. CSF analysis showed a yellow, clear colour, with pleocytosis (214 cells/µL), defined as >5 leukocytes/µL, neutrophil predominance (81%), elevated protein (429mg/dL), and a decreased CSF to serum glucose ratio (7 mg/dL vs 159.3 mg/dL). The patient was diagnosed with tuberculous radiculomyelitis based on clinical symptoms, imaging, and CSF analysis.

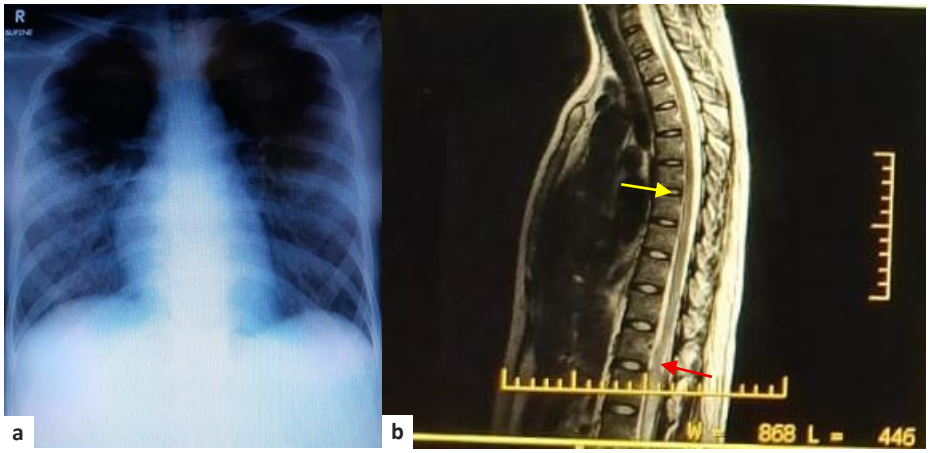

Figure 1: Diagnostic examination. **(a)** Chest X-Ray shows no abnormalities. **(b)** Cervicothoracic MRI image (sagittal view) showing an intramedullary T2-hyperintense lesion with indistinct borders (yellow arrow), suggestive of inflammatory spinal cord involvement; posterior leptomeningeal thickening and T2 hyperintensity along the spinal canal (red arrow), suggestive of arachnoiditis with inflammatory exudates involving the spinal cord surface.

2.3 Treatments and outcomes

The patient received rifampicin 600mg/day, isoniazid 225mg/day, pyrazinamide 1,200mg/day, ethambutol 825mg/day, and methylprednisolone 1 g intravenously for 5 to 14 days, followed by a tapering regimen. During the first week of monitoring, the skin lesions had become hyperpigmented macules, and the weakness in both hands had improved, but both limbs could contract only minimally. After one month, the skin lesions partially diminished. Power in the upper extremities returned to normal but persisted in the lower limbs.

3.0 DISCUSSION

TB radiculomyelitis (TBRM) is a rare disease involving the spinal cord in extrapulmonary TB (WHO, 2021). Typical symptoms of TBRM include radicular pain, paraparesis, quadriparesis, urinary retention, constipation, and paraesthesias in the lower extremities or paraplegia, which can vary depending on the extent of spinal and radicular involvement (Tai et al., 2017). These symptoms are frequently likened to those of GBS (Dalai et al., 2019). In this case, the main complain is weakness in both limbs (paraplegia) accompanied by urinary disorders and constipation. A visible characteristic of TBRM is the presence of extensive exudate in the cerebrospinal fluid (Katrak, 2021). The primary causes of late-onset paraparesis and paraplegia in spinal TB include rigid pressure exerted by kyphotic bone ridges, granulomatous scars, and contractures (Li et al., 2021).

The diagnosis of CNS TB is usually based on clinical, CSF, and CT/MRI findings. In this case, CSF analysis showed a typical tuberculous aetiology, with xanthochromia, pleocytosis, increased protein, and a low glucose ratio. CSF examination showed a yellow, clear colour with pleocytosis (214 cells/uL), neutrophil dominance (81%), increased protein (429mg/dL), and decreased CSF glucose ratio compared to serum (7 vs 159.3mg/dL). These results suggest that the cause of the myelitis was tuberculosis. CSF may also be tested for the presence of acid-fast bacilli by the Ziehl-Neelsen method. *Mycobacterium tuberculosis* can be specifically identified by PCR (Leonard, 2017).

In this case, the inability to confirm tuberculosis through PCR or culture is due to the difficulty in isolating MTB from CSF samples. CSF analysis in GBS revealed albuminocytologic dissociation, characterised by elevated protein levels in the absence of pleocytosis (Rath et al., 2021). MRI examination is important for the early diagnosis of TBRM because CSF analysis and culture require an extended period. The most common MRI findings are basilar meningitis, parenchymal lesions, tuberculomas (usually T2 hypointense, hyperintense), ventriculitis, and spinal involvement (Saxena et al., 2021). In this case, cervicothoracic MRI examination revealed multifocal hyperintense lesions with indistinct borders at the T5-T12 level and central predominance. MRI findings in GBS showed thickening of the nerve roots involving the conus medullaris and cauda equina (Alkan et al., 2009).

CNS tuberculosis is a medical emergency. The British Infectious Diseases Society recommends starting treatment with a combination of isoniazid, rifampicin, pyrazinamide, and either ethambutol, streptomycin, or a fluoroquinolone for an initial period of 2-3 months, followed by isoniazid and rifampicin for a consolidation phase lasting up to 12 months. In some cases, therapy may be prolonged to 18 months (Dian et al., 2021). There was a clinically significant improvement in motor and sensory function after administration of antituberculosis for 1 year. **Table 1** outlines the recommended treatment regimen for central nervous system tuberculosis.

Corticosteroids are used as adjuvant therapy to Anti-Tubercular Therapy (ATT), and function as anti-inflammatory agents by suppressing pro-inflammatory factors including cytokines, chemokines, and related mediators (Prasad et al., 2016; Schutz et al., 2018). Previous studies have shown its significant clinical and radiological improvement, and its ability to reduce the mortality rate of CNS TB (Gundamraj and Hasbun, 2020). Steroids are often used as pulse therapy with a duration of 8 weeks to 6 months (Prasad et al., 2016; Schutz et al., 2018). Clinical improvement was observed by the fifth day of steroid administration, particularly when administered alongside anti-tuberculosis therapy. The patient's clinical outcome showed significant improvement, with disappearance of upper-limb weakness, pain relief, and enhanced motor skills within the first month of therapy.

Table 1: Treatment protocol for CNS tuberculosis caused by drug-susceptible *M. tuberculosis* (WHO, 2014)

Drug	Daily Dose	30-35 kg	36-45 kg	46-55 kg	56-70 kg	>70 kg	Duration
Isoniazid	4-6 mg/kg once daily	150 mg	200 mg	300 mg	300 mg	300 mg	10-12 months
Rifampicin	8-12 mg/kg once daily	300 mg	450 mg	450 mg	600 mg	600 mg	10-12 months
Pyrazinamide	20-30 mg/kg once daily	800 mg	1000 mg	1200 mg	1600 mg	2000 mg	2 months
Ethambutol	15-25 mg/kg once daily	600 mg	800 mg	1000 mg	1200 mg	1200 mg	2 months

4.0 CONCLUSIONS

Tuberculous radiculomyelitis presents a diagnostic challenge due to its rarity and clinical similarity to other spinal disorders, including GBS. Due to the limited sensitivity of the rapid diagnostic test for the detection of MTB in the CNS, it cannot be used as a single diagnostic tool. Clinical features, CSF analysis with or without culture for MTB, and imaging—especially MRI or CT scans—are necessary to establish the diagnosis. Delay in the diagnosis and treatment of CNS TB can lead to severe complications and permanent neurological damage. In this case, the patient showed significant neurological improvement following antituberculosis and corticosteroid therapy. This case may serve as a valuable reference to support diagnostic strategies in TB-endemic areas with limited facilities.

Author Contributions: Concept and design: M.K. and T.K.; data collection: M.K. and D.P.; manuscript drafting: M.K., T.K., D.P. and K.M.; critical revision of the manuscript: M.K., D.P. and K.M.; final approval of the article: M.K., D.P. and K.M.; overall responsibility: M.K..

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

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