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Disseminated Tuberculosis with Tuberculous Meningitis And Multisegmental Tuberculous Sponsylitis: A Case Report

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ABSTRACT

Background: The combination of tuberculous meningitis and tuberculous spondylitis as a manifestation of disseminated tuberculosis (TB) is a rarely reported condition often associated with poor clinical outcomes due to delayed diagnosis. This case report aims to highlight the importance of maintaining clinical suspicion for disseminated TB in patients presenting with a combination of neurological symptoms and chronic back pain. Case Presentation: A 30 year old woman presented with a 15 month history of progressive chronic low back pain radiating to both legs, which subsequently progressed to headache and altered consciousness. Neurological examination revealed signs of meningeal irritation, paraparesis, and sensory impairment at the T4 level. Imaging studies demonstrated multiple foci of tuberculous spondylitis and meningeal involvement, while cerebrospinal fluid analysis and molecular testing confirmed tuberculous meningitis. Based on the combnation of clinical manifestations, imaging findings, and ancillary tests, the patient was diagnosed with disseminated TB. She received anti-tuberculosis therapy, corticosteroids, and underwent a laminectomy with posterior fusion. Discussion: This case demonstrates that the combination of chronic back pain and progressive neurological symptoms should raise suspicion of disseminated TB, even though these manifestations rarely occur concurrently. Early diagnosis achieved through the integration of clinical, radiological, and microbiological findings enables appropriate multidisciplinary therapy, thereby contributing to improved clinical outcomes. Conclusion: Disseminated tuberculosis should be considered in patients presenting with a combination of neurological symptoms and chronic back pain. Clinical vigilance, early diagnosis, and adequate multidisciplinary management play a crucial role in improving the patient's prognosis and clinical outcomes.

1. INTRODUCTION

Tuberculosis (TB) remains a major global health issue, particularly in developing countries like Indonesia. According to the World Health Organization (WHO) Global Tuberculosis Report 2024, an estimated 10.8 million new TB cases occurred worldwide in 2023. Although the majority of cases involve the lungs, the spread of *Mycobacterium tuberculosis* (MTb) to other organs remains a leading cause of morbidity and mortality, especially in patients with disseminated tuberculosis. Disseminated tuberculosis is a severe form of extrapulmonary TB resulting from the hematogenous spread of *Mycobacterium tuberculosis* from a primary focus to two or more anatomically non-contiguous organs. The dissemination of bacilli via the bloodstream allows for the involvement of various organs, including the central nervous system (CNS), spine, liver, spleen, and bone marrow, among others. While this condition is commonly found in immunocompromised patients, it can also occur in immunocompetent individuals (Wael et al., 2025). The diagnosis of disseminated TB is established based on a combination of clinical

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manifestations, radiological findings, microbiological or histopathological examinations, and evidence of multisystem involvement consistent with hematogenous spread.

One of the most severe manifestations of disseminated TB is tuberculous meningitis (TBM). Globally, an estimated 129,000–199,000 cases of tuberculous meningitis occur annually among adults. TBM is associated with a high mortality rate of approximately 23%, with mortality being substantially higher among patients with HIV infection than among those without HIV. Furthermore, nearly one-third of survivors experience permanent neurological sequelae, including cognitive impairment, motor deficits, epilepsy, and other neurological disabilities. Therefore, early diagnosis and prompt initiation of anti-tuberculosis therapy are crucial for reducing mortality and preventing long-term neurological complications.

In addition to central nervous system involvement, hematogenous dissemination of *Mycobacterium tuberculosis* may also result in tuberculous spondylitis (Pott's disease), the most common form of musculoskeletal TB. Tuberculous spondylitis accounts for approximately 2% of all TB cases and 15% of extrapulmonary TB cases. The infection typically begins in the vertebral body through Batson's venous plexus or the arterial circulation, subsequently leading to progressive bone destruction, vertebral collapse, kyphotic deformity, paravertebral abscess formation, and spinal cord compression. Multilevel vertebral involvement is relatively uncommon and is often associated with more extensive disease dissemination, a higher mycobacterial burden, and an increased risk of spinal instability and severe neurological deficits compared with single-level involvement.

Concurrent involvement of the central nervous system and the spine, presenting as tuberculous meningitis or tuberculous meningoencephalitis accompanied by tuberculous spondylitis, is an uncommon manifestation that reflects a complex form of disseminated TB. The available literature is largely limited to individual case reports and small case series, resulting in limited evidence regarding the clinical characteristics, diagnostic strategies, and management of this condition. Establishing the diagnosis is often challenging because the clinical manifestations are insidious and nonspecific, while microbiological confirmation from cerebrospinal fluid or bone tissue demonstrates relatively low sensitivity. Moreover, imaging findings in the early stages of the disease are frequently nonspecific, which may contribute to delayed diagnosis. Delayed initiation of treatment has been associated with an increased risk of hydrocephalus, cerebral infarction, paraplegia, permanent spinal deformity, and death.

Reports describing the combination of tuberculous meningoencephalitis and multilevel tuberculous spondylitis remain exceedingly rare, particularly in immunocompetent adults. This case is of educational significance because it illustrates multisystem involvement resulting from hematogenous dissemination, highlights the diagnostic challenges posed by nonspecific clinical manifestations, and emphasizes the importance of integrating clinical, radiological, and laboratory findings to establish an accurate diagnosis and guide appropriate management.

In this report, we describe the case of a 30-year-old woman with disseminated tuberculosis presenting with tuberculous meningoencephalitis and multilevel tuberculous spondylitis. This case underscores that disseminated TB should be considered early in the differential diagnosis of patients presenting with neurological manifestations accompanied by back pain or spinal abnormalities, particularly in TB-endemic regions. Early recognition of multisystem involvement may facilitate timely diagnosis, enable prompt initiation of appropriate therapy, and reduce the risk of permanent neurological disability and mortality.

2. METHOD

Case Report

A 30-year-old woman was brought to the emergency department by her family with a chief complaint of decreased consciousness that had begun one day prior to admission. Four days before admission, she developed fever accompanied by a diffuse, throbbing headache. Three days before admission, she became increasingly somnolent. On the night before admission, she experienced high-grade fever with chills. The following morning, she was unable to communicate. Although she opened her eyes spontaneously, she was unable to follow commands and localized

only to painful stimuli. She was initially admitted to a local hospital, where she was diagnosed with a suspected central nervous system infection and subsequently referred to our tertiary referral hospital. Upon arrival two days later, her condition had improved, with a Glasgow Coma Scale (GCS) score of E4V5M6, and she was able to communicate appropriately.

Further history revealed that approximately 16 days before admission, the patient had developed a persistent cough that failed to improve. Nine days before admission, she also experienced urinary retention. Her illness had initially begun approximately 15 months before admission with lower back pain, initially localized to the mid-lumbar region. During the following year, the pain progressively radiated to both lower extremities and was described as a dull aching sensation accompanied by a burning feeling. Initially, the pain occurred 3–5 times per month, lasting approximately 30 minutes per episode, but gradually increased in frequency to 5–7 episodes per month, with each episode lasting 1–2 hours.

One year before admission, she began experiencing paresthesia extending from below the breasts to both lower extremities. Ten months before admission, progressive bilateral lower-limb weakness developed. Initially, she was still able to ambulate with assistance; however, her condition gradually deteriorated, and during the last 4–5 months before admission she became completely unable to move either lower extremity. During the preceding 1–2 months, she also developed lower urinary tract symptoms characterized by difficulty initiating micturition. The patient initially sought medical attention at a primary healthcare facility, where she was diagnosed with a pinched nerve. In December 2025, further evaluation at a hospital established the diagnoses of pulmonary tuberculosis and tuberculous spondylitis. Anti-tuberculosis therapy was initiated on December 22, 2025. She had no history of diabetes mellitus, hypertension, autoimmune disease, immunosuppressive therapy, or HIV infection. Anti-HIV testing was nonreactive. She denied any previous history of tuberculosis, known contact with patients with tuberculosis, or prior Bacillus Calmette–Guérin (BCG) vaccination.

On admission, her blood pressure was 101/64 mmHg, heart rate was 76 beats/min, respiratory rate was 20 breaths/min, body temperature was 36.2°C, and oxygen saturation was 98% while receiving oxygen via a nasal cannula at 3 L/min. Neurological examination demonstrated that she was conscious (GCS E4V5M6) with nuchal rigidity. Motor strength of the upper extremities was normal (Medical Research Council grade 5/5 bilaterally), whereas complete paralysis of both lower extremities was noted (MRC grade 0/5 bilaterally). Deep tendon reflexes were brisk (+3) in all extremities, with bilateral positive Babinski signs. Sensory examination demonstrated hypoesthesia below the T4 dermatome. Urinary retention was present, requiring placement of an indwelling urinary catheter. Cranial nerve and cerebellar examinations were unremarkable.

3. RESULT AND DISCUSSION

Result

Cerebrospinal fluid (CSF) analysis revealed slightly turbid yellow fluid with a leukocyte count of 195 cells/μL, predominantly lymphocytes (67%), markedly elevated protein concentration (376 mg/dL), and severely decreased glucose concentration (6 mg/dL), while the simultaneous blood glucose level was 87 mg/dL. Both the Nonne and Pandy tests were positive. Cerebrospinal fluid culture showed no bacterial growth. Laboratory investigations demonstrated a hemoglobin level of 12.3 g/dL, leukocyte count of 13,040/mm^3, platelet count of 414,000/mm^3, serum sodium level of 126 mmol/L, potassium level of 4.0 mmol/L, aspartate aminotransferase (AST) of 40 U/L, alanine aminotransferase (ALT) of 26 U/L, and serum albumin level of 3.27 g/dL. Anti-HIV testing was nonreactive. Hyponatremia was corrected, resulting in an increase in serum sodium concentration to 132 mmol/L.

Chest radiography demonstrated extensive active pulmonary tuberculosis. Contrast-enhanced cranial computed tomography (CT) revealed bilateral frontoparietotemporal leptomeningeal enhancement, suggestive of meningoencephalitis, without evidence of increased intracranial pressure. Contrast-enhanced whole-spine magnetic resonance imaging (MRI) demonstrated destruction and abnormal signal intensity involving the T9, T10, L4, and L5

vertebral bodies, consistent with multilevel tuberculous spondylitis. Additional findings included grade II spinal canal stenosis at the T9–T10 and L4–L5 levels, narrowing of the T9–T10 and L4–L5 intervertebral disc spaces, and a left psoas abscess.

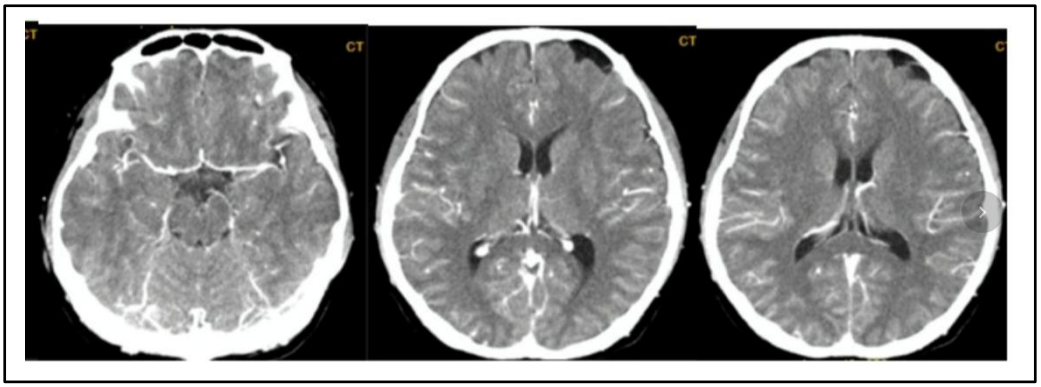

Figure 1. Contrast-enhanced head CT scan

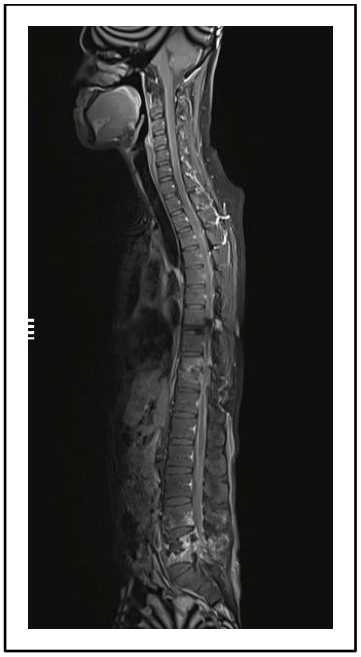

Figure 2. MRI Whole Spine T1+Contrast

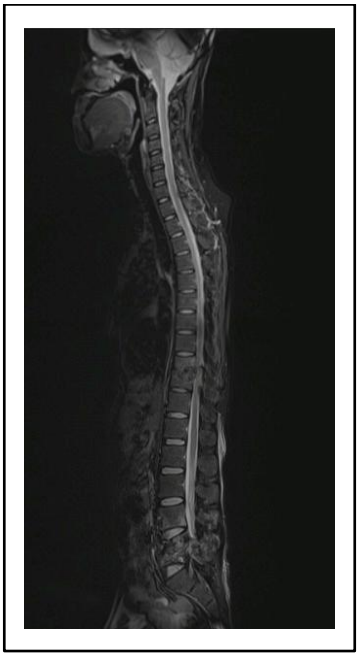

Figure 3 MRI Whole Spine T2 Non-Kontras

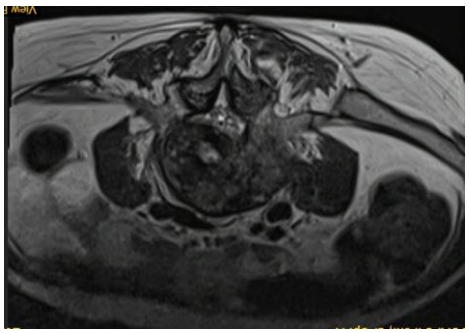

Figure 4 Transverse MRI

Based on the clinical presentation, cerebrospinal fluid findings characteristic of tuberculous meningitis, radiological evidence of leptomeningeal enhancement, active pulmonary tuberculosis, and multilevel tuberculous spondylitis, the patient was diagnosed with disseminated tuberculosis presenting as tuberculous meningoencephalitis and multilevel tuberculous spondylitis. Although cerebrospinal fluid culture showed no bacterial growth, the diagnosis was maintained because the microbiological diagnostic yield in tuberculous meningitis is known to be low, particularly in patients who have previously received anti-tuberculosis treatment.

The patient was treated with intravenous ceftriaxone (2 g every 12 hours), citicoline (500 mg every 12 hours), mecobalamin (500 µg every 12 hours), ranitidine (50 mg every 12 hours), paracetamol (1 g every 8 hours), and dexamethasone (5 mg every 8 hours). A pulmonology consultation was obtained for the management of pulmonary tuberculosis. Oral pyridoxine (once daily), curcuma extract (three times daily), acetylcysteine capsules (three times daily), and ongoing anti-tuberculosis therapy were continued. The patient's clinical condition gradually improved and remained stable throughout hospitalization. The patient was subsequently referred to the neurosurgery service for further management of tuberculous spondylitis. She underwent decompressive laminectomy, and her postoperative course was uneventful. On the third postoperative day, she was discharged from the hospital in stable condition.

Discussion

This case illustrates disseminated tuberculosis with simultaneous involvement of the central nervous system, presenting as tuberculous meningoencephalitis, and the spine, manifesting as multilevel tuberculous spondylitis, in a young adult. This presentation represents a rare form of extrapulmonary tuberculosis resulting from the hematogenous dissemination of *Mycobacterium tuberculosis* to multiple anatomically noncontiguous organs. Unlike most cases of tuberculous spondylitis, which are confined to a single spinal region, our patient exhibited concurrent thoracic and lumbar vertebral involvement accompanied by a psoas abscess and tuberculous meningoencephalitis, reflecting more extensive and complex disease dissemination (World Health Organization, 2024; Rajasekaran et al., 2022).

Several previous case reports have described the coexistence of tuberculous meningitis and tuberculous spondylitis; however, most occurred in patients with underlying immunodeficiency or involved only a single vertebral level. In contrast, our patient was a young immunocompetent adult with a nonreactive HIV test who presented with multilevel vertebral involvement accompanied by severe neurological deficits, including paraplegia and urinary retention. This case highlights that disseminated tuberculosis may also occur in immunocompetent individuals and may progress insidiously until advanced disease develops if the diagnosis is delayed (Goni et al., 2021; Gupta et al., 2025).

The clinical course demonstrated two distinct patterns of tuberculosis manifestation. The patient's chronic lower back pain, which had persisted for more than one year, was consistent with the slowly progressive nature of tuberculous spondylitis. In contrast, the acute onset of fever, headache, impaired consciousness, and nuchal rigidity several days before admission was characteristic of tuberculous meningoencephalitis. The coexistence of these manifestations

should raise clinical suspicion for disseminated tuberculosis, particularly in patients with established pulmonary tuberculosis (Wilkinson et al., 2023; Rajasekaran et al., 2022).

The diagnosis in this case required integration of the clinical presentation, laboratory findings, and imaging studies (Inggas et al., 2025). Although cerebrospinal fluid culture yielded no bacterial growth, the diagnosis of tuberculous meningoencephalitis was maintained. The diagnostic yield of cerebrospinal fluid culture in tuberculous meningitis is only approximately 40–60%, particularly among patients who have previously received anti-tuberculosis therapy. Cerebrospinal fluid analysis in our patient demonstrated lymphocytic pleocytosis, markedly elevated protein concentration, and severe hypoglycorrhachia, with a cerebrospinal fluid-to-blood glucose ratio of <0.4. These findings were further supported by contrast-enhanced cranial CT demonstrating leptomeningeal enhancement and the presence of active pulmonary tuberculosis. According to the Marais diagnostic criteria, the combination of clinical manifestations, cerebrospinal fluid characteristics, and neuroimaging findings fulfilled the criteria for probable tuberculous meningitis, thereby justifying the initiation and continuation of anti-tuberculosis therapy despite the absence of microbiological confirmation (Marais et al., 2010; Wilkinson et al., 2023; Lourens et al., 2025).

The diagnosis of tuberculous spondylitis was further supported by MRI findings demonstrating destruction of the T9, T10, L4, and L5 vertebral bodies, accompanied by narrowing of the intervertebral disc spaces, spinal canal stenosis, and a left psoas abscess. The combination of multilevel vertebral destruction and a psoas abscess is highly characteristic of tuberculous spondylitis and helps distinguish it from pyogenic spinal infection or degenerative spinal disease. Furthermore, the presence of active pulmonary tuberculosis strongly supported the conclusion that both the central nervous system and spinal involvement resulted from hematogenous dissemination of *Mycobacterium tuberculosis*, confirming the diagnosis of disseminated tuberculosis (Rajasekaran et al., 2022; Garg & Somvanshi, 2011).

Several differential diagnoses were initially considered, including bacterial meningitis, leptomeningeal metastasis, spinal epidural abscess, and pyogenic spondylodiscitis. Bacterial meningitis was considered less likely because of the subacute clinical course, lymphocytic predominance in the cerebrospinal fluid, profoundly decreased cerebrospinal fluid glucose concentration, and negative cerebrospinal fluid culture. Leptomeningeal metastasis was unlikely because there was no history of malignancy or imaging findings suggestive of neoplastic disease. Likewise, pyogenic spondylodiscitis typically presents with a more acute course, more pronounced intervertebral disc destruction, and is rarely associated with concomitant active pulmonary tuberculosis and meningitis. Collectively, the overall clinical, laboratory, and radiological findings were more consistent with disseminated tuberculosis (Bernaerts et al., 2003; Thwaites et al., 2020).

Management was undertaken using a multidisciplinary approach. The patient continued standard anti-tuberculosis therapy in accordance with national treatment guidelines and received adjunctive corticosteroid therapy for tuberculous meningitis. Adjunctive corticosteroids have been shown to reduce mortality and neurological complications by attenuating the inflammatory response within the subarachnoid space. In addition, decompressive laminectomy was performed because the patient had progressive paraplegia with MRI evidence of spinal canal stenosis. Previous studies have demonstrated that spinal cord decompression in patients with severe neurological deficits improves the likelihood of neurological recovery when combined with adequate anti-tuberculosis therapy (WHO, 2022; Thwaites et al., 2020; Zhang et al., 2023).

Empirical antibiotic therapy was also initiated during the early stage of treatment to cover the possibility of bacterial meningitis while awaiting diagnostic confirmation. In this patient, ceftriaxone was administered in accordance with standard clinical practice for suspected bacterial meningitis. Following treatment with anti-tuberculosis therapy, adjunctive corticosteroids, and empirical intravenous antibiotics, the patient's level of consciousness improved substantially. This favorable clinical response is consistent with previous studies demonstrating that the prognosis of tuberculous meningitis depends largely on early diagnosis and prompt initiation of treatment, whereas neurological outcomes in tuberculous spondylitis are strongly influenced by

the duration of spinal cord compression before surgical decompression (Nuswantoro et al., 2025; Rajasekaran et al., 2022; Wilkinson et al., 2023).

This case has several limitations. First, microbiological confirmation was not obtained from all sites of infection; therefore, the diagnosis relied primarily on the integration of clinical manifestations, laboratory findings, imaging studies, and therapeutic response. Second, the negative cerebrospinal fluid culture may have reflected the inherently low sensitivity of microbiological testing as well as prior exposure to anti-tuberculosis therapy before referral. Third, long-term follow-up data after hospital discharge were unavailable, precluding comprehensive assessment of long-term neurological outcomes (Wilkinson et al., 2023).

This case highlights the importance of considering disseminated tuberculosis in patients from TB-endemic regions who present with acute neurological manifestations accompanied by chronic progressive back pain and neurological deficits, even when initial microbiological investigations are inconclusive. A multidisciplinary approach integrating clinical assessment, cerebrospinal fluid analysis, neuroimaging, and timely medical and surgical management is essential for achieving an early diagnosis, preventing permanent neurological disability, and improving patient outcomes (World Health Organization, 2024; Thwaites et al., 2020; Wilkinson et al., 2023).

4. CONCLUSION

This case report demonstrates that disseminated tuberculosis may present as the uncommon combination of tuberculous meningoencephalitis and multilevel tuberculous spondylitis in an adult patient. Acute neurological manifestations accompanied by a history of chronic progressive back pain and evidence of tuberculosis in other organs should raise a high index of suspicion for disseminated tuberculosis, particularly in TB-endemic regions. This case underscores the importance of a multidisciplinary approach that integrates clinical findings, cerebrospinal fluid analysis, and imaging studies to establish an early diagnosis, even in the absence of microbiological confirmation. Prompt diagnosis and appropriate management are essential to avoid delays in treatment, reduce the risk of permanent neurological disability, and improve patient outcomes.

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