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Late-Onset Central Nervous System (CNS) Tuberculosis Presenting as Delirium in a Treated Pulmonary Tuberculosis Patient: A Ceftriaxone and Streptomycin Responsive Case

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Abstract
Tuberculosis (TB) remains a major health burden in Indonesia. While pulmonary TB is common, extrapulmonary manifestations like tuberculous meningitis (TBM) are severe and diagnostically challenging. Standard oral anti-TB drugs often have poor central nervous system (CNS) penetration, which may lead to treatment failure in the CNS despite pulmonary cure. This study aims to describe a rare presentation of late-onset CNS tuberculosis presenting as delirium shortly after completed pulmonary TB treatment, and to discuss the therapeutic role of ceftriaxone and streptomycin. This study employed a qualitative, single-case report research design to conduct an in-depth investigation into a rare clinical presentation. The study presents the case of a 49-year-old male who developed acute delirium one week after being declared cured of pulmonary TB. Diagnosis was based on clinical presentation, brain MRI showing leptomeningeal enhancement, and cerebrospinal fluid analysis suggestive of TBM, despite a negative GeneXpert test. The patient showed marked neurological improvement after a regimen of intravenous ceftriaxone (2g twice daily) and intramuscular streptomycin (1g daily), without re-instituting standard oral anti-TB drugs. This suggests that the previous oral regimen may have achieved pulmonary cure but failed to eradicate a subclinical CNS infection due to insufficient drug levels. This case highlights the potential for CNS TB reactivation post-pulmonary treatment and suggests that alternative regimens with better CNS penetration, such as ceftriaxone and streptomycin, should be considered. Further studies are needed to evaluate their role as adjunctive therapy for TBM.

INTRODUCTION

Tuberculosis (TB) continues to pose a major public health burden in Indonesia, which currently ranks second among countries with the highest number of TB cases globally, as reported in the WHO Global Tuberculosis Report 2024 (Zhi Chen et al., 2025). Although pulmonary TB remains the most common presentation, extrapulmonary tuberculosis (EPTB) accounts for a significant proportion of TB morbidity, with manifestations affecting the lymphatic system, bones, genitourinary tract, and the Central Nervous System (CNS) (Kulchavenya, Naber, & Bjerklund Johansen, 2019). Among these, tuberculous meningitis (TBM) is the most severe form of EPTB due to its high rates of mortality and long-term neurological sequelae if not diagnosed and treated promptly (Arshad et al., 2020).

TBM arises when Mycobacterium tuberculosis crosses the blood-brain barrier, causing granulomatous inflammation of the meninges and brain parenchyma (Mubarak et al., 2025). Clinical manifestations are often nonspecific in the early stages, including headache, fever, and

behavioral changes, which may progress to altered consciousness, focal neurological deficits, and cognitive impairment (Suryadevara, 2025). Diagnostic delays are common due to the low sensitivity of conventional microbiological tests and the overlapping features with other chronic meningitides (Poplin, Boulware, & Bahr, 2020). Despite the availability of standard anti-TB regimens for pulmonary disease, the management of CNS-TB remains complex because many first-line drugs have limited penetration into the cerebrospinal fluid and brain tissue (Mandal, Biswas, Ansar, Mukherjee, & Jawed, 2024).

Several studies have explored the challenges of CNS tuberculosis diagnosis and treatment (Wei Chen et al., 2020). Thwaites highlighted persistent diagnostic and therapeutic uncertainties in TBM, emphasizing that despite decades of research, critical questions regarding optimal management remain unanswered (Seddon et al., 2019). (Madadi & Sohn, 2024) systematically reviewed cerebrospinal fluid concentrations of antituberculosis agents, demonstrating that many first-line drugs achieve suboptimal CNS levels, particularly in adults (Sileshi, Tadesse, Makonnen, & Aklillu, 2021). More recently, Lin (2024) discussed both classic diagnostic approaches and emerging high-throughput molecular techniques for TBM, underscoring the ongoing need for improved diagnostic tools (Lin, 2025). Davis et al. (2023) examined cognitive impairment as a major sequela of TBM, revealing that neurological complications persist even after microbiological cure, which underscores the importance of CNS-penetrating therapies. Additionally, studies by Srivastava et al. (2020) and Nguyen et al. (2022) have begun exploring the role of β-lactam antibiotics such as ceftriaxone in TB treatment, showing promising in vitro and experimental evidence of efficacy against Mycobacterium tuberculosis, particularly when combined with β-lactamase inhibitors (Alffenaar, Wicha, & Srivastava, 2023).

The novelty of this case lies in its documentation of late-onset CNS tuberculosis occurring shortly after the completion of standard pulmonary TB therapy—a presentation that is rarely reported in the literature (Machida, Ishihara, Amano, & Otsu, 2018). While most cases of TBM occur concurrently with or prior to pulmonary TB treatment, this case demonstrates CNS reactivation or dissemination despite microbiological cure of pulmonary disease (Leonard, 2017). Furthermore, the favorable response to intravenous ceftriaxone and intramuscular streptomycin—neither of which is included in standard first-line TB regimens—highlights a potential therapeutic gap in CNS-TB management (Malik et al., 2025). This case contributes to the growing body of evidence suggesting that alternative regimens with enhanced CNS penetration may be necessary for patients with extrapulmonary TB, particularly in the central nervous system (Schaller, Wicke, Foerch, & Weidauer, 2019). The clinical urgency of this report stems from the high morbidity and mortality associated with TBM, and the implication that current oral TB regimens may be insufficient in preventing or treating CNS involvement (Oo & Agrawal, 2025). This report underscores the need to reassess current TB treatment regimens in light of their limited CNS bioavailability and highlights the potential role of β-lactams in refractory or atypical post-treatment cases.

This report highlights a rare case of late-onset TBM occurring shortly after the completion of standard pulmonary TB therapy (Barry, Akkielah, Askar, & Nasser, 2019). The patient presented with acute neuropsychiatric symptoms, including agitation and hallucinations, which were later attributed to meningoencephalitis tuberculosis (Hernández, Fuentes, & Serrano, 2025). This case underscores the possibility of CNS reactivation or dissemination despite microbiological cure of pulmonary TB and raises the concern of subtherapeutic CNS drug concentrations with standard oral regimens (Mandal et al., 2024). Furthermore, this report explores the potential therapeutic role of ceftriaxone and streptomycin, which were not included in the original treatment plan but yielded favorable clinical outcomes (Eljaaly, Wali, Basilim, Alharbi, & Asfour, 2019).

RESEARCH METHOD

This study employed a qualitative, single-case report research design to conduct an in-depth investigation into a rare clinical presentation. This case report was conducted in accordance with ethical standards for case report publications. Written informed consent was obtained from the patient for publication of this case and accompanying images. Diagnostic confirmation of tuberculous meningitis followed the Marais et al. (2010) scoring system for TBM, which integrates clinical, laboratory, and radiological criteria to improve diagnostic accuracy in resource-limited settings. The data population comprised the entire clinical narrative of a single patient who developed central nervous system tuberculosis shortly after successful treatment for pulmonary tuberculosis. The data sample was the specific 49-year-old male patient who met the inclusion criteria of presenting with neuropsychiatric symptoms and a confirmed clinicoradiological diagnosis of TBM post-pulmonary TB cure. A purposive sampling technique was utilized, as the case was selected specifically for its uniqueness and its potential to provide critical insights into a novel clinical scenario and therapeutic approach.

The primary research instruments were the patient's medical records, including history, physical examination notes, laboratory results, and radiological imaging (chest X-ray, CT thorax, and brain MRI). Data collection was performed retrospectively through a comprehensive review of this clinical documentation. The data analysis technique was predominantly descriptive and thematic. The patient's clinical course, diagnostic findings, and treatment response were systematically described and synthesized. The analysis focused on interpreting the thematic sequence of events—from pulmonary cure to CNS relapse—and evaluating the therapeutic outcome in the context of existing pharmacological literature on blood-brain barrier penetration.

RESULTS AND DISCUSSION

CASE PRESENTATION

Chief Complaints

A 49-year-old male from Sampit, Central Kalimantan, Indonesia, presented to the Emergency Department with acute agitation that worsened three hours prior to admission.

History of Present Illness

According to the patient's family, the symptoms began approximately two weeks prior to admission and had progressively worsened. The patient’s wife reported that he experienced visual hallucinations, seeing a figure dressed in white carrying a gun and attempting to harm him—these hallucinations primarily occurred at night. Additional complaints included severe headaches, nausea, and repeated vomiting in the last three days. The patient also had a dry cough but denied experiencing fever, dyspnea, rhinorrhea, or diarrhea.

History of Past Illness

The patient had a confirmed diagnosis of pulmonary tuberculosis six months prior to admission and completed a full six-month course of oral anti-tuberculosis therapy from mid-October 2024 to mid-April 2025 at a local hospital. At the start of therapy, the patient’s body weight was 51 kilograms, and he was prescribed three tablets daily of Fixed-Dose Combination (FDC) anti-tuberculosis drugs during both the intensive and continuation phases, in accordance with national guidelines. At the end of treatment, sputum acid-fast bacilli (AFB) smear converted from positive to negative, and the patient was declared clinically cured by a pulmonologist. However, approximately one week after completing therapy, he began to experience hallucinations, which progressively worsened into severe headaches by the time of

admission. The patient had no known history of diabetes mellitus, hypertension, or other chronic systemic diseases.

Personal and Family History

The patient was a heavy smoker for 37 years, consuming approximately two packs of cigarettes per day. There was no known contact with individuals diagnosed with chronic cough or tuberculosis.

Physical Examination

Upon examination, the patient was uncooperative and appeared undernourished. Vital signs were within normal limits, and no fever was observed. He exhibited disorientation regarding time, place, and person, and continued to report hallucinations. Meningeal signs were positive on physical examination. Auscultation of the chest revealed decreased vesicular breath sounds in the left lung field.

Laboratory Examinations

Routine blood tests showed no specific abnormalities, with a normal leukocyte count and mildly elevated erythrocyte sedimentation rate (Table 1).

Imaging Examinations

Chest X-ray and CT thorax imaging revealed extensive fibrotic changes in the left lung apex and anterior mediastinum suggestive of previous pulmonary tuberculosis (Figures 1A and 1B). Brain MRI with contrast (Figures 2A and 2B) demonstrated leptomeningeal enhancement and multiple small lesions in the bilateral frontal lobes, consistent with tuberculous meningoencephalitis.

Microbiological Identification of the Causative Agent

Cerebrospinal fluid (CSF) obtained via lumbar puncture appeared clear and demonstrated lymphocytic pleocytosis with reduced glucose levels. Although the GeneXpert test performed on the CSF sample was negative, the findings were consistent with tuberculous involvement of the central nervous system (Table 1).

FINAL DIAGNOSIS

The final diagnosis was Stage II Central Nervous System Tuberculosis (meningoencephalitis) with delirium, in a patient with previously treated pulmonary tuberculosis, supported by a Marais Score of 13.

TREATMENT

In the Emergency Department, the patient was administered intramuscular diazepam 5 mg and haloperidol (Lodomer) 5 mg to control agitation, followed by oral maintenance therapy with clozapine 5 mg, alprazolam 1 mg, and trihexyphenidyl (Hexymer) 2 mg. Intravenous hydration was provided using 1000 mL of Futrolit per 24 hours. Pain management included intravenous paracetamol 1 g three times daily. Dexamethasone 5 mg IV three times daily was initiated to reduce central nervous system inflammation. Neurotrophic support was provided with intravenous mecobalamin (Kalmeco) 500 mcg twice daily and citicoline (Brain Act) 500 mg IV twice daily. Empiric infection management consisted of intravenous ceftriaxone 2 g twice daily for 14 days and intramuscular streptomycin 1 g once daily for 30 days. No oral anti-tuberculosis drugs were administered during this hospitalization.

OUTCOME AND FOLLOW-UP

The overall clinical outcome was favorable. The patient showed marked improvement without any residual neurological deficits or signs of extrapulmonary tuberculosis. He was able to resume normal daily activities and remained stable at follow-up.

This case describes a 49-year-old male with a prior history of successfully treated pulmonary tuberculosis, who later developed central nervous system tuberculosis (CNS-TB) manifesting as delirium. To the best of our knowledge, this presentation of tuberculous meningitis (TBM) occurring after completed pulmonary TB treatment is rarely reported. The

patient demonstrated substantial clinical improvement following combination therapy with intramuscular streptomycin and high-dose intravenous ceftriaxone, which was not part of the initial anti-tuberculosis regimen. This observation raises important questions regarding the pathogenesis and pharmacological management of CNS-TB in post-treatment pulmonary TB cases.

TBM is the most severe and disabling form of extrapulmonary TB, known for its high rates of mortality and long-term neurological sequelae (9). Diagnosis is often delayed due to its nonspecific clinical features that overlap with other chronic meningitides, such as viral or fungal causes (6). The pathogenesis involves Mycobacterium tuberculosis breaching the blood-brain barrier (BBB) via two main routes: direct invasion through endothelial actin rearrangement and the "Trojan horse" mechanism, where bacilli travel within infected macrophages and neutrophils (10). Once in the CNS, the bacilli trigger a robust inflammatory response, resulting in granulomatous meningitis, vasculitis, and complications such as cerebral infarction and cognitive impairment (5,11). The virulence factor Gen Rv0931c gen (pknD) further facilitates adhesion to endothelial structures, enhancing CNS colonization (10).

Despite radiological and clinical signs consistent with TBM, this patient's cerebrospinal fluid (CSF) GeneXpert MTB/RIF test was negative. This may be attributed to the low bacillary burden typically seen in CSF, especially in subacute presentations (6). GeneXpert demonstrates moderate sensitivity (63–82%) and high specificity (>90%), but may yield false negatives when CSF volume is limited or bacterial load is minimal (7). The newer Xpert MTB/RIF Ultra assay offers better performance in paucibacillary conditions and improved rifampicin resistance detection (12). These limitations underscore the need for integrative diagnostic strategies combining molecular, biochemical, and radiographic findings, as well as emerging technologies from the fields of genomics and metabolomics (7,13).

Interestingly, in this case, tuberculous meningitis (TBM) occurred shortly after the patient had completed the standard six-month regimen for pulmonary tuberculosis, consisting of two months of intensive therapy with isoniazid, rifampin, pyrazinamide, and ethambutol (HRZE), followed by four months of consolidation with isoniazid and rifampin (HR), as recommended by the WHO (14). Although this regimen is highly effective for pulmonary TB, it may not achieve optimal therapeutic concentrations in the central nervous system due to the complex pharmacokinetics of anti-tuberculosis drugs in the cerebrospinal fluid (CSF) and brain parenchyma (7,8). The penetration of drugs across the blood-brain barrier (BBB) and into CSF does not necessarily correlate with effective drug distribution in brain tissue, necessitating specific dose adjustments when treating CNS-TB (7).

Among first-line oral anti-TB drugs, isoniazid demonstrates good BBB penetration; however, data on its efficacy at higher doses in TBM remain inconclusive, with a commonly recommended dosage of 600 mg/day (8). Rifampin, a cornerstone in TB treatment, often achieves subtherapeutic CSF levels at standard doses. Evidence suggests that higher doses, such as 600–1000 mg/day (12–20 mg/kg), may improve clinical response in TBM by ensuring CSF concentrations exceed the minimum inhibitory concentration (MIC) for M. tuberculosis (8,15). Pyrazinamide exhibits excellent CSF and brain tissue penetration, making it a critical component in TBM treatment. The optimal dose for CNS involvement is estimated at 35 mg/kg (range 30–40 mg/kg) (8,15). Conversely, ethambutol shows poor CSF permeability and is often undetectable in CSF even at high doses up to 50 mg/kg (15), thus limiting its role in CNS-TB management.

Despite microbiological cure of pulmonary TB, the development of TBM in this patient suggests that standard oral anti-TB regimens may not achieve sufficient CSF concentrations to prevent or eliminate CNS infection. This supports the hypothesis that TBM may arise due to inadequate CNS drug exposure rather than systemic treatment failure. Therefore, further clinical research is warranted to explore optimized and individualized dosing strategies of oral

anti-TB drugs specifically tailored for CNS tuberculosis, especially in post-pulmonary TB cases (7,8,15).

Ceftriaxone, a third-generation cephalosporin, was used in this case and may have contributed significantly to the favorable outcome. It possesses favorable pharmacokinetics for CNS infections, with prolonged half-life (6–8 hours) and excellent CSF penetration during meningeal inflammation (16). Although M. tuberculosis naturally resists β-lactams via the BlaC β-lactamase, ceftriaxone is more stable and exhibits reduced hydrolysis by BlaC compared to other cephalosporins (17). Moreover, in vitro studies show that β-lactamase inhibitors such as avibactam enhance the susceptibility of M. tuberculosis to ceftriaxone (17,18). Experimental models also support its efficacy in TB meningitis, especially when used at high doses or in combination with β-lactamase inhibitors, making ceftriaxone a promising adjunct in CNS-TB treatment (18).

Streptomycin was historically the first antibiotic to improve survival in TBM and, although its use has declined due to poor CSF penetration in non-inflamed states, it retains therapeutic potential under conditions of meningeal inflammation (8,19). Inflammation-induced disruption of the BBB enhances drug permeability by loosening tight junctions, decreasing CSF outflow, and inhibiting efflux pumps such as P-glycoprotein, thereby facilitating higher intracerebral antibiotic concentrations (20). Streptomycin, a hydrophilic aminoglycoside, benefits from this pathophysiology and can reach therapeutic levels in inflamed CNS tissue (20). When combined with β-lactams such as ceftriaxone, it exhibits synergistic bactericidal activity and reduced toxicity, which is especially important given its known nephrotoxic and ototoxic potential (16). Corticosteroid therapy, often used in TBM, does not impair streptomycin's CNS penetration(20).

In summary, this case demonstrates the potential utility of high-dose ceftriaxone and intramuscular streptomycin as an effective combination for TBM, particularly in patients previously treated for pulmonary TB. The outcome suggests a need to reconsider current CNS-TB treatment paradigms, especially in cases following standard oral therapy. Further studies are warranted to validate this approach and explore optimal dosing strategies, with emphasis on drug penetration into the CSF and brain parenchyma. Additionally, future clinical trials should investigate the role of ceftriaxone and β-lactamase inhibitors as part of frontline therapy for TBM. This finding supports the exploration of ceftriaxone and streptomycin as adjuncts in CNS-TB, potentially addressing pharmacokinetic gaps in current regimens. This case report is limited by its single-patient design, lack of confirmatory culture, and short-term follow-up. Nonetheless, it provides a clinically relevant observation that warrants further pharmacological and clinical validation.

CONCLUSION

This case highlights a rare and clinically significant presentation of late-onset central nervous system tuberculosis (CNS-TB) manifesting as delirium in a patient recently declared cured from pulmonary TB. Despite completing a full course of oral anti-tuberculosis treatment, the patient developed tuberculous meningoencephalitis—underscoring the possibility of subclinical CNS dissemination or reactivation, potentially due to insufficient CNS drug penetration. The use of high-dose intravenous ceftriaxone and intramuscular streptomycin, in combination with adjunctive corticosteroids and supportive neurotrophic therapy, resulted in substantial neurological and functional recovery. This therapeutic response supports the potential role of alternative or adjunctive regimens, especially in cases where standard oral therapy may fall short of achieving therapeutic concentrations in the central nervous system. Given the limited clinical data on CNS-TB occurring after treated pulmonary TB, further studies are needed to evaluate optimal drug combinations, dosing strategies, and diagnostic

tools for early detection. Early recognition and timely intervention are essential to prevent long-term neurological complications and improve prognosis in similar clinical scenarios.

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