BJMS
www.batnajms.net

Batna Journal of Medical Sciences 2026;13(8):25
https://doi.org/10.48087/BJMS.2026.130825

CASE REPORT

Tuberculous meningitis complicated by acute hydrocephalus and bilateral optic neuropathy: a case report

Nabila CHOUBANE

ABSTRACT

Tuberculous meningitis (TBM) is the most severe form of extrapulmonary tuberculosis and is associated with high mortality and long-term neurological sequelae. Early diagnosis remains challenging because of its nonspecific clinical presentation, particularly in endemic settings where delays in treatment are associated with poorer outcomes. We report the case of a 24-year-old female physician with no significant past medical history who presented with acute agitation and fever. Initial neurological examination was unremarkable, with no focal neurological deficits or meningeal signs. Cerebrospinal fluid analysis revealed lymphocytic pleocytosis (56 cells/mm³; 90% lymphocytes), hypoglycorrhachia, and elevated protein levels, while the initial brain computed tomography (CT) scan was normal. Empirical treatment with cefotaxime and acyclovir was initiated for presumed meningoencephalitis. On the third hospital day, the patient experienced rapid neurological deterioration, characterized by decreased consciousness and generalized tonic–clonic seizures, requiring endotracheal intubation and mechanical ventilation. A repeat brain CT scan demonstrated acute hydrocephalus, prompting emergency external ventricular drainage. Although TBM was strongly suspected, antituberculous therapy was initially deferred because of the absence of microbiological confirmation or evidence of tuberculosis at other sites. Following extubation, the patient developed severe visual impairment, and ophthalmological examination revealed bilateral optic neuropathy. Antituberculous therapy was subsequently initiated, and a ventriculoperitoneal shunt was placed. The patient's neurological status gradually improved, allowing discharge from the intensive care unit after a two-month hospitalization. At follow-up, visual acuity remained severely impaired and required corrective lenses. This case highlights the diagnostic challenges of TBM when the initial clinical and radiological findings are nonspecific and underscores the importance of early empirical antituberculous therapy in high-burden settings, even in the absence of microbiological confirmation, to reduce the risk of irreversible neurological and visual sequelae.

Keywords: Tuberculous meningitis, Hydrocephalus, Optic neuropathy, Neurocritical care, Central nervous system tuberculosis, Case report, Empirical therapy.

Received: 16 Jun 2026
Accepted: 06 Jul 2026

Correspondance to: Nabila CHOUBANE
E-mail: nabila.choubane@yahoo.com

1. INTRODUCTION

Tuberculosis remains a major global health burden, particularly in low- and middle-income countries. Although pulmonary disease accounts for the majority of cases, extrapulmonary tuberculosis contributes substantially to morbidity and mortality. Among these forms, tuberculous meningitis (TBM) represents the most severe manifestation of central nervous system tuberculosis, associated

with high case fatality rates and significant long-term neurological sequelae [1,2]. TBM results from hematogenous dissemination of Mycobacterium tuberculosis to the meninges and subpial spaces, triggering a subacute inflammatory process characterized by basal meningeal exudates, obliterative vasculitis, and impaired cerebrospinal fluid (CSF) circulation. These pathophysiological mechanisms explain the frequent occurrence of hydrocephalus, cerebral infarction, cranial neuropathies, and visual impairment [2,3].

Early diagnosis remains challenging due to the nonspecific and often misleading nature of initial clinical manifestations, which may mimic psychiatric, metabolic, or other infectious conditions. Furthermore, microbiological confirmation is frequently delayed or absent due to the paucibacillary nature of CSF infection and limited diagnostic performance of conventional tools [4,5].

Neuroimaging findings may be absent in early disease, further complicating diagnosis. When present, abnormalities such as basal meningeal enhancement, hydrocephalus, and ischemic infarctions are suggestive but not specific [6,7]. Consequently, TBM remains primarily a clinical diagnosis in many endemic settings, requiring a high index of suspicion and early empirical treatment to prevent irreversible neurological injury [8,9].

We report the case of a young female physician presenting with acute neuropsychiatric symptoms followed by rapid neurological deterioration, acute hydrocephalus, and severe visual impairment secondary to TBM. This case highlights the diagnostic complexity of TBM and underscores the critical importance of early empirical therapy in high-burden settings.

2. CASE PRESENTATION

A 24-year-old female physician with no significant medical history presented to the emergency department with acute agitation and fever. On admission, vital signs were stable, with a blood pressure of 129/80 mmHg, heart rate of 99 beats/min, respiratory rate of 19 breaths/min, oxygen saturation of 97% on room air, and temperature of 38°C. Initial neurological examination revealed psychomotor agitation without focal neurological deficits or meningeal signs.

Laboratory investigations showed leukocytosis (14,000/mm³) and an elevated C-reactive protein level of 10 mg/L. Chest radiography showed no abnormalities (Figur1). Cerebrospinal fluid (CSF) analysis revealed a lymphocytic pleocytosis (56 cells/mm³, 90% lymphocytes), elevated protein concentration, and hypoglycorrhachia (0.21 g/L), with a corresponding blood glucose level of 1.20 g/L. Initial brain computed tomography (CT) findings were normal. Empirical intravenous therapy with cefotaxime and acyclovir was initiated for presumed meningoencephalitis. CSF Gram stain and mycobacterial culture were requested. Interferon-gamma release assay as well as GeneXpert MTB/RIF testing on cerebrospinal fluid were not available at the time of patient management. On day 3, the patient experienced neurological deterioration with seizures and coma, requiring endotracheal intubation and admission to the intensive care unit. Repeat brain CT revealed acute obstructive hydrocephalus (Figure 2). Emergency external ventricular drainage (EVD) was performed.

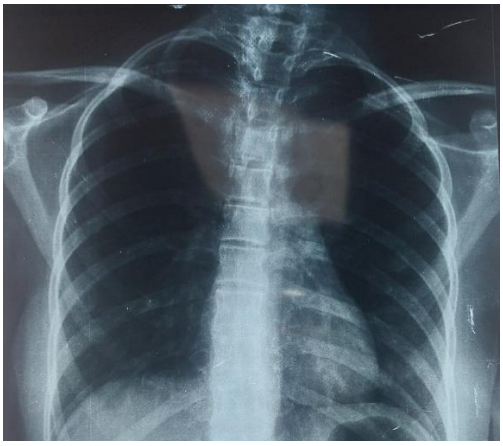

Figure 1. Normal chest radiograph.

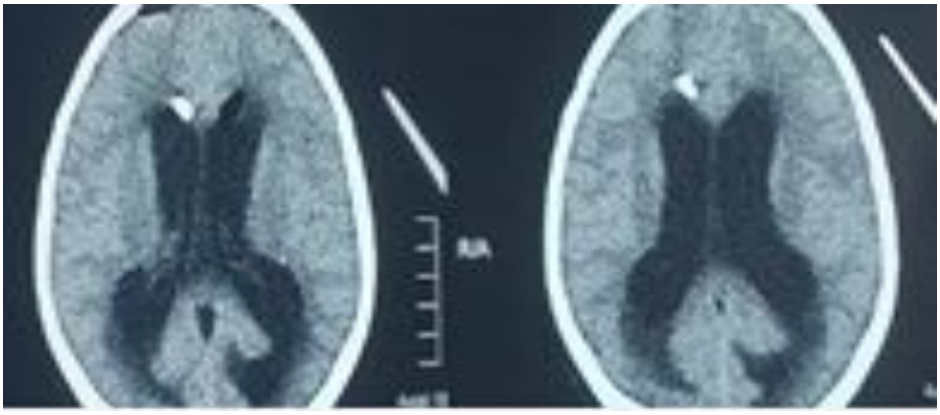

Figure 2. Brain CT scan showing acute tetraventricular hydrocephalus.

Despite a strong clinical suspicion of TBM, antituberculous therapy was not initiated immediately due to the initial atypical presentation, absence of microbiological confirmation, and the need for multidisciplinary validation in accordance with local

therapeutic protocols. Following extubation, the patient complained of severe bilateral visual impairment. An ophthalmological consultation was therefore requested and revealed bilateral optic neuropathy.This finding further strengthened the clinical suspicion of tuberculous meningitis. Consequently, standard antituberculous therapy consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol was initiated, together with adjunctive dexamethasone.Due to persistent hydrocephalus, a ventriculoperitoneal shunt was subsequently placed.

After two months of hospitalization, the patient achieved substantial neurological recovery and was discharged from the intensive care unit. She was subsequently oriented to the infectious diseases department for continuation of antituberculous therapy. Although neurological status markedly improved, severe persistent visual impairment persisted, requiring ophthalmological follow-up and prescription of corrective lenses.

3. DISCUSSION

Tuberculous meningitis (TBM) remains the most severe form of central nervous system tuberculosis, with persistently high mortality and substantial long-term neurological disability despite advances in diagnostics and critical care management [1,8]. Prognosis is primarily determined by the delay between symptom onset and initiation of appropriate therapy.

Early recognition of TBM is particularly challenging due to its highly variable and often nonspecific presentation. Initial symptoms may include fever, behavioral changes, or altered consciousness, frequently in the absence of meningeal signs or focal neurological deficits. In this case, the initial presentation was dominated by acute neuropsychiatric manifestations, a pattern that can easily mimic psychiatric, metabolic, or other infectious encephalopathies, leading to diagnostic delay.

Cerebrospinal fluid analysis typically reveals lymphocytic pleocytosis, elevated protein, and hypoglycorrhachia; however, these findings are nonspecific and overlap with other chronic meningitides [1,4]. In the present case, CSF abnormalities were highly suggestive but not diagnostic, reflecting the inherent limitations of early TBM diagnosis. Microbiological confirmation remains a major challenge in routine clinical practice. In many endemic and resource-limited settings, access to rapid diagnostic tools such as interferon-gamma release assays and advanced molecular platforms is limited. Smear microscopy has poor sensitivity, while culture remains the reference standard but is slow and often unavailable or incompatible with urgent clinical decision-making.

Although molecular assays such as GeneXpert MTB/RIF have improved diagnostic yield, their sensitivity remains suboptimal, and their negative predictive value is insufficient to exclude TBM in clinically compatible cases [5]. Consequently, diagnostic confirmation is frequently delayed, and clinical decision-making must rely on an integrated assessment combining CSF findings, neuroimaging, and validated scoring systems such as the Marais criteria [4].

Neuroimaging findings in TBM are variable and may be normal in early stages, as observed in this patient. When present, typical abnormalities include basal meningeal enhancement, infarctions secondary to vasculitis, and hydrocephalus due to impaired CSF circulation [6,7]. Hydrocephalus represents one of the most severe complications of TBM. It results from obstruction of CSF flow by inflammatory exudates within the basal cisterns and is associated with increased intracranial pressure, neurological deterioration, and poor prognosis. Management often requires urgent neurosurgical intervention, including external ventricular drainage or ventriculoperitoneal shunting [10–12].

Visual loss in tuberculous meningitis (TBM) is multifactorial and may result from prolonged intracranial hypertension, ischemic optic neuropathy, or inflammatory involvement of the optic pathways. The severe bilateral visual impairment observed in this case was first noted following the episode of acute hydrocephalus and intracranial hypertension and persisted during follow-up, consistent with secondary optic neuropathy. Detailed ophthalmological assessments, including visual acuity quantification, fundoscopic examination, or visual evoked potentials, were not available in the medical record. Timely initiation of antituberculous therapy is the most important determinant of outcome in TBM. Current WHO guidelines strongly recommend early empirical treatment in all patients with a high clinical probability of TBM, even in the absence of microbiological confirmation [9].

In this case, therapy was initially deferred due to diagnostic uncertainty and the absence of definitive microbiological or radiological evidence at presentation. In addition, in high-burden endemic settings, therapeutic hesitation may also reflect concerns regarding overtreatment, drug toxicity, antimicrobial stewardship, and emerging resistance patterns. However, retrospective analysis suggests that delayed therapy likely contributed to the severity of neurological deterioration and permanent visual sequelae.

Optimal management of TBM requires a multidisciplinary approach involving intensive care, infectious diseases, neurology, neurosurgery, and ophthalmology. Early cerebrospinal fluid diversion procedures, combined with appropriate antituberculous therapy and adjunctive corticosteroids, are essential to improve survival and reduce neurological sequelae [11,14].

4. CONCLUSION

This case underscores the protean and misleading presentation of tuberculous meningitis, particularly when manifesting with neuropsychiatric symptoms and minimal early radiological abnormalities. It reinforces that in endemic settings, where diagnostic delays are frequent and microbiological confirmation is often unavailable, clinical suspicion must remain paramount. Early empirical antituberculous therapy remains the cornerstone of management to prevent irreversible neurological and visual sequelae.

Competing interests: The authors declare that they have no competing interest.

Funding: This research received no external funding.

Informed consent: Informed verbal consent was obtained from the patient for publication of this case report. Patient confidentiality was strictly maintained.

REFERENCES

1. Thwaites GE, van Toorn R, Schoeman J. Tuberculous meningitis: more questions, still too few answers. Lancet Neurol. 2013 Oct;12(10):999-1010. doi:10.1016/S1474-4422(13)70168-6. PMID:23972913.
2. Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. Clin Microbiol Rev. 2008 Apr;21(2):243-61. doi:10.1128/CMR.00042-07.
3. Brancusi F, Farrar J, Heemskerk D. Tuberculous meningitis in adults: a review of a decade of developments focusing on prognostic factors for outcome. Future Microbiol. 2012 Sep;7(9):1101-16. doi:10.2217/fmb.12.86.
4. Marais S, Thwaites G, Schoeman JF, Török ME, Misra UK, Prasad K, Donald PR, Wilkinson RJ, Marais BJ. Tuberculous meningitis: a uniform case definition for use in clinical research. Lancet Infect Dis. 2010 Nov;10(11):803-12. doi:10.1016/S1473-3099(10)70138-9.
5. Bahr NC, Marais S, Caws M, van Crevel R, Wilkinson RJ, Tyagi JS, Thwaites GE, Boulware DR; Tuberculous Meningitis International Research Consortium. GeneXpert MTB/Rif to diagnose tuberculous meningitis: perhaps the first test but not the last. Clin Infect Dis. 2016 May 1;62(9):1133-5. doi:10.1093/cid/ciw083.
6. Bernaerts A, Vanhoenacker FM, Parizel PM, Van Goethem JW, Van Altena R, Laridon A, De Roeck J, Coeman V, De Schepper AM. Tuberculosis of the central nervous system: overview of neuroradiological findings. Eur Radiol. 2003 Aug;13(8):1876-90. doi:10.1007/s00330-002-1608-7.
7. Maria M, Kankara SR, Reddy KS, Kancharla M, Jayanna D. CNS tuberculosis: insights into its multifaceted imaging manifestations. Educational exhibit presented at: ECR 2025; doi:10.26044/ecr2025/C-17284. Available from: https://dx.doi.org/10.26044/ecr2025/C-17284
8. Török ME. Tuberculous meningitis: advances in diagnosis and treatment. Br Med Bull. 2015 Mar;113(1):117-31. doi:10.1093/bmb/ldv003.
9. World Health Organization. Global tuberculosis report 2022. Geneva: WHO; 2022.
10. Caliman-Sturdza OA, Cucu A. Hydrocephalus in tuberculous meningitis. IntechOpen. 2023. doi:10.5772/intechopen.110251.
11. Figaji AA, Fieggen AG. The neurosurgical and acute care management of tuberculous meningitis: evidence and current practice. Tuberculosis (Edinb). 2010 Nov;90(6):393-400. doi:10.1016/j.tube.2010.09.005.
12. Rajshekhar V. Management of hydrocephalus in patients with tuberculous meningitis. Neurol India. 2009 Jul-Aug;57(4):368-74. doi:10.4103/0028-3886.55572.
13. Thwaites GE, Chau TT, Farrar JJ. Improving the bacteriological diagnosis of tuberculous meningitis. J Clin Microbiol. 2004 Jan;42(1):378-9. doi:10.1128/JCM.42.1.378-379.2004.
14. Heemskerk AD, Bang ND, Mai NT, et al. Intensified antituberculosis therapy in adults with tuberculous meningitis. N Engl J Med. 2016 Jan 14;374(2):124-34. doi:10.1056/NEJMoa1507062.