

Complete Recovery of Multiple Cranial Nerve Palsies in Tuberculous Meningitis Using Levofloxacin as a Second-Line Anti-Tuberculosis Drug: A Case Report

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ABSTRACT

Introduction: Tuberculous meningitis (TBM) is a severe form of extrapulmonary tuberculosis (TB) with high mortality and morbidity rates. Clinical manifestations of TBM vary, both typical and atypical. Second-line anti-TB drugs are generally used in cases of drug-resistant TB but may serve as an alternative if side effects occur from the standard regimen. Presentation: A 26-year-old woman presented with decreased consciousness, hallucinations, diplopia, and vertigo. Neurological examination revealed bilateral sixth nerve palsy and left eye ptosis. The diagnosis of TBM was confirmed based on clinical symptoms, brain magnetic resonance imaging (MRI) results showing basal leptomeningeal enhancement, and the detection of Mycobacterium tuberculosis (M. tuberculosis) in cerebrospinal fluid (CSF) and sputum. The patient initially received standard therapy with rifampicin, isoniazid, pyrazinamide, and streptomycin (RHZS), but developed side effects including drug-induced liver injury (DILI) and drug eruption. The rifampicin regimen was temporarily switched to levofloxacin as a second-line anti-TB drug for nearly two months, and streptomycin was discontinued. During this therapy, significant clinical improvement was observed, including the resolution of cranial nerve palsy. The rifampicin, isoniazid, pyrazinamide, and ethambutol (RHZE) regimen was then resumed and completed over 9 months without residual symptoms. Discussion: The use of second-line anti-TB drugs such as levofloxacin can be an effective temporary alternative in drug-sensitive TBM if there is intolerance to the first-line regimen. Fluoroquinolones are a compelling treatment option for TBM, primarily due to their favorable penetration into the central nervous system.

KEYWORDS: Tuberculous meningitis, Multiple cranial nerve palsies, Levofloxacin.

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INTRODUCTION

Tuberculosis (TB) cases in Indonesia rank second worldwide after India¹. Tuberculous meningitis (TBM) is one of the most common extrapulmonary TB cases, characterized by severe symptoms². It accounts for approximately 1% of all TB cases and has a global mortality rate of around 25%, reaching up to 70% in sub-Saharan Africa³. Clinical manifestations of TBM vary and may include headache, fever, seizures, signs of meningeal irritation, and cranial nerve involvement. The most commonly involved cranial nerves include cranial nerve II, III, VI, and VII⁴. This case report describes the clinical improvement of cranial nerve palsy in TBM with a second-line anti-TB drug regimen.

CASE REPORT

A 26-year-old woman initially presented with a progressive decline in consciousness, accompanied by behavioral changes and visual hallucinations. The patient had a history of intermittent fever, followed by a runny nose and headache. She denied having a prolonged cough or significant weight loss. After her consciousness improved, the patient complained of double vision and dizziness.

On neurological examination, nuchal rigidity was present, and cranial nerve abnormalities were observed, including bilateral sixth nerve palsy and left eyelid ptosis (Figure 1). Other neurological findings were within normal limits.

Brain magnetic resonance imaging (MRI) with contrast showed leptomeningeal enhancement in the basal cisterns (Figure 2). In addition, cerebrospinal fluid (CSF) analysis and GeneXpert of CSF also suggested TB infection. The results of chest X-ray and GeneXpert sputum analysis also confirmed *Mycobacterium tuberculosis* (*M. tuberculosis*). Based on medical history, radiological imaging, CSF analysis, GeneXpert of CSF and sputum specimens, she was diagnosed with TBM and new-onset pulmonary TB. The patient's clinical condition gradually improved, accompanied by improvement of multiple cranial nerve palsies with the standard TBM regimen and corticosteroids. However, several treatment challenges emerged during TBM therapy, including drug side effects. Initially, the intensive-phase regimen of rifampicin, isoniazid, pyrazinamide, and streptomycin (RHZS) was administered, but the patient developed drug-induced liver injury (DILI) and a drug eruption, necessitating discontinuation of

rifampicin and streptomycin. The patient was subsequently transitioned to the second-line agent, levofloxacin, for nearly two months. Upon evaluation, this alternative regimen yielded satisfactory outcomes, including complete resolution of the multiple cranial nerve palsies (Figure 1). Intensive-phase treatment was resumed with the RHZE regimen after resolution of medication side effects, followed by the maintenance phase until the 9th month. The patient completed treatment without residual symptoms.

DISCUSSION

TB cases in Indonesia rank second globally, after India¹. TBM is among the most common forms of extrapulmonary TB and is associated with severe clinical manifestations². Geographical differences affect treatment outcomes and mortality rates in TBM, which are linked to healthcare accessibility, diagnostic and treatment capacity, nutritional status, and socioeconomic conditions³. The pathogenesis of TBM begins with the spread of *M. tuberculosis* from a primary pulmonary infection through the bloodstream to the central nervous system, penetrating the blood-brain barrier and forming granulomas in the leptomeningeal and cortical regions, known as Rich foci. The ruptured granuloma enters the subarachnoid space, spreading infection and triggering neuroinflammatory responses that cause nerve tissue damage, leading to neurological deficits³.

Clinical symptoms of TBM vary and may include headache, fever, seizures, signs of meningeal irritation, and cranial nerve involvement. The most commonly involved cranial nerves are cranial nerve II, III, VI, and VII⁴. Clinical manifestations that appear in TBM can be typical or atypical. Typical presentations display classic meningeal signs accompanied by neurological deficits, while atypical presentations resemble psychiatric disorders³.

The diagnosis of TBM can be established through supporting tests such as CSF analysis, molecular testing, and neuroimaging. CSF analysis may reveal nonspecific findings such as elevated protein levels, reduced glucose levels, and pleocytosis. Molecular testing using GeneXpert MTB has nearly 100% specificity and moderate sensitivity (46.5%–71.1%) for detecting *M. tuberculosis*. TBM is also supported by MRI as the primary diagnostic modality, showing basal meningeal enhancement, hydrocephalus, infarcts in the thalamus, basal ganglia, and tuberculoma³.

Extrapulmonary TB treatment in adults generally uses the same standard regimen (2RHZE/4RH for six months), with important exceptions for certain severe and difficult-to-treat forms of extrapulmonary TB. For TBM, a longer treatment duration (9 to 12 months) is recommended. It is associated with high mortality and neurological morbidity. Therefore, extended therapy is considered safer and offers a better chance of cure⁵. The fluoroquinolones, including moxifloxacin, gatifloxacin, and levofloxacin, are second-line drugs used in multidrug-resistant TB (MDR-TB) and TBM cases. Fluoroquinolones can be used in TBM cases due to their good penetration ability through the blood-brain barrier⁶. In the case described, the standard TB regimen was temporarily switched to levofloxacin in response to drug-related adverse effects. Clinical improvement was obtained during the initial TB therapy, temporary fluoroquinolone substitution, and repeated TB treatment until the 9th month, at which point treatment was declared complete. However, routine use in drug-sensitive TB is currently not recommended as a replacement for the first-line regimen, RHZE, except in cases where contraindications are present⁶.

CONCLUSION

This case report shows that the use of levofloxacin as a temporary second-line anti-TB therapy can provide clinical improvement in TBM cases. Management of TBM requires special consideration due to treatment-related challenges, including suspected adverse drug reactions. Effective alternative treatment is essential to mitigate the risk of mortality and neurological morbidity associated with TBM.

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Data availability statement

The data used to support the findings of this study are included within the article.

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Consent

Written informed consent was obtained from the patient for submission and publication of this case report and accompanying images.

Conflict of interest

none declared.

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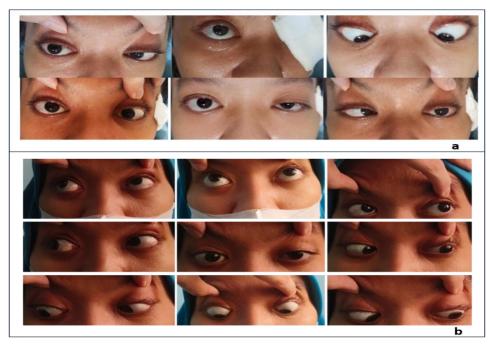
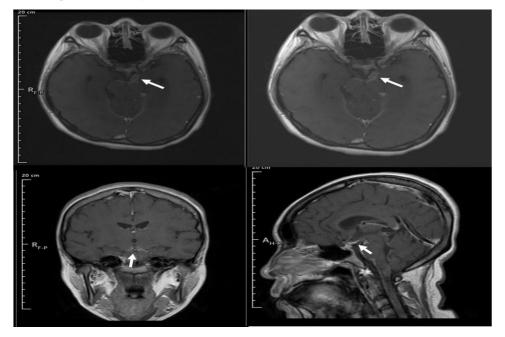


Figure 1. (a) Examination of cranial nerve III, IV, VI showed ptosis oculi sinistra and bilateral sixth nerve palsy before treatment. (b) Clinical improvement of eyeball movement in all directions after treatment.



Figure~2.~Basal~leptomeningeal~enhancement~(white~arrows).~(a~and~b)~Axial~view,~(c)~coronal~view,~and~(d)~sagittal~view.