CASE REPORT

Disseminated Tuberculosis in An Immunocompetent Patient Presenting with Ptosis and Diplopia: A Case Report

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ABSTRACT

Introduction: Tuberculosis (TB) remains a global health burden to this day, with pulmonary TB (PTB) being one of its most common types. A lapse in the host immune response may lead to the dissemination of TB infection, causing extrapulmonary TB (EPTB) that is more difficult to manage, as it frequently shows atypical complaints. However, its manifestations among immunocompetent patients are rarely well-investigated.

Case: A 28-year-old human immunodeficiency virus (HIV)-negative woman presented with a history of chronic progressive bilateral headache, followed by diplopia and ptosis of her left eyelid. She also had classical TB symptoms 3 months prior. She had a history of household tuberculous contact for years, but she was never tested for TB nor given an anti-TB treatment (ATT). Chest X-ray revealed cavities and pleural thickening in the apex of the left lung. Contrast-enhanced magnetic resonance imaging (MRI) of the brain showed multiple intracranial tuberculomas and meningeal enhancement in most brain areas. GeneXpert tests from both sputum and cerebrospinal fluid (CSF) suggested TB infections. She was later diagnosed with simultaneous tuberculous meningoencephalitis (TBME) and PTB. After a 17-day course of oral ATT, streptomycin injection, and intravenous corticosteroid, she was discharged with improved clinical conditions and scheduled for outpatient follow-up until she completed 12 months of ATT.

Conclusion: In immunocompetent patients, disseminated TB is an uncommon occurrence and is frequently characterized by atypical symptoms. Findings from this case emphasize that comprehensive assessment and timely therapeutic intervention are critical for minimizing morbidity and mortality of TB infection.

INTRODUCTION

Tuberculosis (TB), an infectious disease caused by *Mycobacterium tuberculosis* (MTB), remains a significant public health issue in Indonesia. In 2019, 845,000 new cases and 96,700 TB-related deaths were reported in Indonesia, accounting for around 8% of the global TB burden.[1] While pulmonary TB (PTB) is the most common clinical presentation, extrapulmonary TB (EPTB) constitutes approximately 16% of all TB cases, with disseminated TB representing a rare and severe form of the disease.[2] Disseminated TB refers to the involvement of at least two non-contiguous organ systems or isolation of the bone marrow, liver, bloodstream, or cerebrospinal fluid by MTB.[2] Central nervous system (CNS) involvement represents one of the most severe manifestations of extrapulmonary tuberculosis (EPTB), occurring in approximately 10% of disseminated TB cases and associated with significant morbidity and mortality.[3]

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Disseminated TB is most frequently observed in immunocompromised individuals, particularly those with human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), malignancies, or undergoing immunosuppressive therapy.[2] It remains rare among immunocompetent individuals, comprising less than 2% of all reported TB cases.[4] Its occurrence in immunocompetent patients is uncommon and often leads to diagnostic challenges due to its non-specific and varied clinical manifestations. Neurological involvement, such as tuberculous meningoencephalitis (TBME) or cranial nerve palsies, may occur in disseminated TB but is rarely the initial presentation, particularly in patients without identifiable risk factors for immunosuppression.[3] Cranial nerve palsies, including oculomotor nerve involvement manifesting as ptosis and diplopia, are exceedingly rare as presenting features of disseminated TB.[5] A review of literature indicates only a limited number of such cases reported globally, highlighting the diagnostic complexity and clinical rarity of this presentation in immunocompetent individuals.[4,5]

This case report describes a rare presentation of disseminated TB in an immunocompetent adult who initially presented with isolated oculomotor nerve palsy manifesting as ptosis and diplopia, emphasizing the importance of maintaining a high index of suspicion for TB even in atypical clinical scenarios.

CASE

A 28-year-old woman was referred to Dr. Soetomo General Academic Hospital, Surabaya, following a 7-day admission at a peripheral hospital with a preliminary diagnosis of TBME and concurrent PTB. The patient reported a two-month history of progressively worsening bilateral headache, followed by the onset of binocular diplopia five days before admission at the referring hospital. She also noted the development of left-sided ptosis two weeks before referral, without associated visual loss or pain on ocular movement. Additional symptoms included frequent vomiting over the preceding week and a noticeable decrease in appetite. The patient denied neck stiffness, limb weakness, or other focal neurological deficits. Before admission, she had self-administered acetaminophen for headache relief, but with minimal symptomatic improvement. She also reported a three-month history of intermittent productive cough with yellowish, viscous, and foamy sputum, low-grade fever, and unintentional weight loss of approximately 4 kg. These symptoms had been previously self-limited and did not prompt medical attention. She denied hemoptysis, dyspnea, or night sweats. There was no history of trauma or pre-existing cardiac, respiratory, gastrointestinal, or neurological disorders.

Upon further history taking, the patient disclosed a significant family history of TB. Her mother had died five years prior due to untreated PTB. Additionally, her sister had been diagnosed with PTB and had completed a full six-month course of anti-TB treatment (ATT). The patient had never undergone screening for TB nor received any form of ATT before her current illness.

During her 7-day admission at the referring hospital, the patient was diagnosed with PTB following a positive result on the sputum molecular rapid test (GeneXpert MTB/rifampicin [RIF]), which detected MTB and confirmed RIF sensitivity. A contrast-enhanced computed tomography (CT) scan of the brain was also performed, revealing leptomeningeal enhancement predominantly in the basal cisterns, suggestive of TBME (Figure 1). Due to the unavailability of cerebrospinal fluid (CSF) analysis, empirical ATT was initiated. The regimen included isoniazid 300 mg, RIF 450 mg, pyrazinamide 1,000 mg, ethambutol 500 mg, and daily intramuscular streptomycin 750 mg. By the time of her presentation to Dr. Soetomo General Academic Hospital, Surabaya, the patient had completed 8 days of oral ATT.

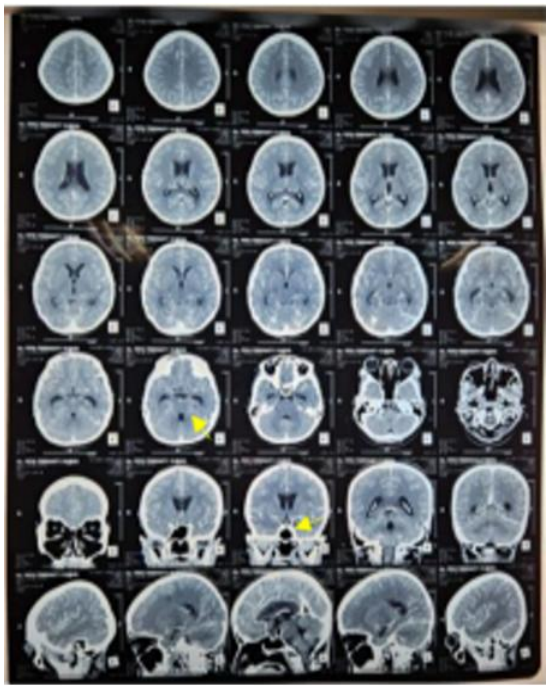

Figure 1. A contrast-enhanced computed tomography scan of the brain revealed leptomeningeal enhancement, particularly in the basal cistern (yellow arrows)

On admission, the patient was alert and fully conscious, with a Glasgow Coma Scale (GCS) score of 15 (E4, V5, M6). She reported a moderate bilateral headache, rated 6-7 on a 10-point numeric pain scale. Vital signs were within normal limits. Physical examination revealed conjunctival pallor bilaterally,

indicative of anemia. No lymphadenopathy was observed in the cervical, supraclavicular, or thoracic regions. Respiratory examination showed symmetrical chest wall movement. Tactile fremitus was increased over the upper third of the left hemithorax on both anterior and posterior assessment. Percussion of the same region revealed dullness, while the remaining lung fields were resonant. Auscultation demonstrated vesicular breath sounds throughout, with fine crackles and bronchophony localized to the upper third of the left hemithorax.

Neurological examination revealed no signs of meningeal irritation. Cranial nerve assessment demonstrated visual acuity of greater than 2/60 in both eyes, with normal visual fields. The pupils were equal, round, and reactive to light, measuring 3 mm bilaterally. Both direct and consensual light reflexes were intact. Left-sided ptosis was observed, with a markedly reduced marginal reflex distance (Figure 2A). Extraocular movements were preserved in all directions, with no evidence of ophthalmoplegia. Examination of the remaining cranial nerves yielded no abnormalities. Motor, sensory, and autonomic functions were intact. Muscle strength and tone were within normal limits across all extremities. Deep tendon reflexes were physiological, and no pathological reflexes (e.g., Babinski or Hoffmann signs) were elicited.

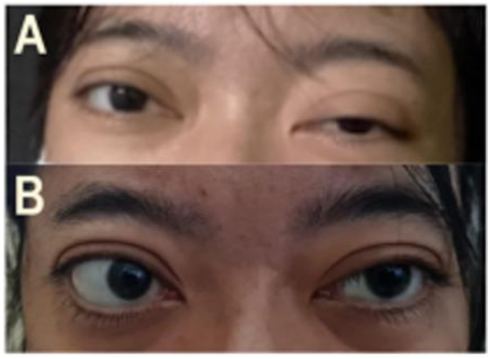

Figure 2. A) Left eye ptosis at the time of presentation (first day of care); B) Left eye ptosis improved on the fifteenth day of care.

Initial laboratory investigations revealed normochromic normocytic anemia, with a hemoglobin level of 8.6 g/dL, mean corpuscular volume (MCV) of 80.7 fL, mean corpuscular hemoglobin (MCH) of 24.4 pg, and mean corpuscular hemoglobin concentration (MCHC) of 30.3 g/dL. A mild leukocytosis was noted, with a total leukocyte count of 11,380 cells/µL. Serum albumin was decreased at 2.68 g/dL, and random blood glucose was within normal limits at 80 mg/dL. Serological testing for HIV and hepatitis B surface antigen (HBsAg) was both non-reactive.

Cerebrospinal fluid was macroscopically clear. Its analysis demonstrated pleocytosis with a total leukocyte count of 121 cells/µL, predominantly mononuclear cells (61.4%). The protein concentration was markedly elevated at 276 mg/dL, while the glucose level was reduced to 29 mg/dL, corresponding to a CSF-to-blood glucose ratio of 0.36. Cerebrospinal fluid adenosine deaminase (ADA) level was 8.6 IU/L (reference range: <10 IU/L). Both Nonne and Pandy tests were positive, indicating increased globulin content. No microorganisms were identified on Gram staining or aerobic culture. However, MTB was detected in the CSF using the GeneXpert MTB/RIF assay, confirming RIF-sensitive TB involvement of the CNS.

Chest radiography (CXR) revealed suprahilar fibroconsolidation in the left lung, along with multiple cavitary lesions and apical pleural thickening (Schwarte sign), consistent with chronic specific inflammation suggestive of PT (Figure 3).

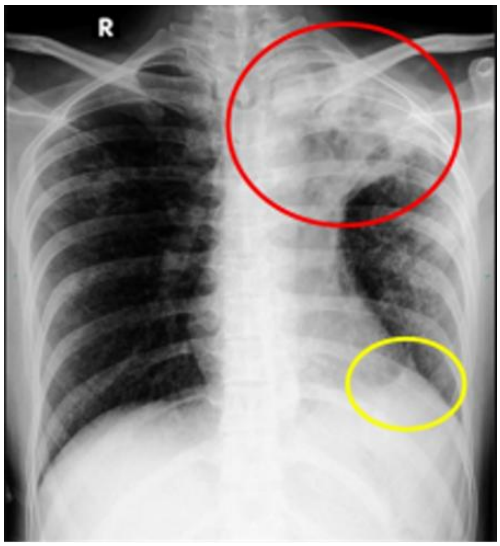

Figure 3. Initial chest X-ray result on the first day of care showed fibroconsolidation with multiple cavities, along with pleural thickening in the apex of the left lung (red circle). Tenting of the left hemidiaphragm can also be seen in this picture (yellow circle).

Contrast-enhanced magnetic resonance imaging (MRI) of the brain demonstrated multiple well-circumscribed lesions consistent with tuberculomas, distributed across the bilateral centrum semiovale, left corona radiata, bilateral frontal, parietal, and occipital lobes, as well as the left temporal lobe, cerebellum, medulla oblongata, and spinal cord. These lesions were accompanied by perifocal edema and diffuse abnormal meningeal enhancement. These findings are suggestive of disseminated CNS TB, including TBME and intracranial tuberculomatosis (Figure 4). The tuberculomas varied in size, with the largest measuring approximately 0.4×0.4×0.4 cm (anteroposterior, mediolateral, and craniocaudal dimensions,

respectively). Rim-enhancing lesions were observed on contrast-enhanced sequences.

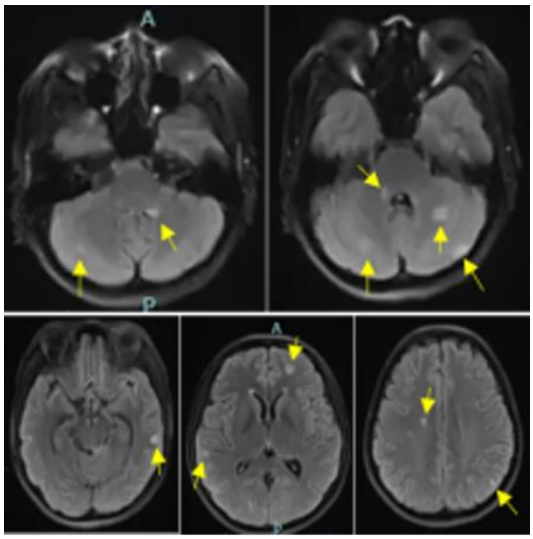

Figure 4. Brain magnetic resonance imaging revealed multiple lesions of various sizes, with well-defined borders, distributed over both cerebral hemispheres, cerebellum, medulla, and spinal cord, suggesting tuberculomatosis (yellow arrows).

Magnetic resonance spectroscopy (MRS) of the brain revealed no significant elevation in the choline-to-creatine (Ch/Cr) or choline-to-N-acetylaspartate (Ch/NAA) ratios. These findings may help differentiate tuberculomas from neoplastic processes. Additionally, magnetic resonance angiography (MRA) revealed bilateral vasculitis affecting the posterior cerebral arteries (PCA), while the circle of Willis remained patent.

The patient continued to receive ATT with a fixed-dose combination (FDC) regimen consisting of RIF 450 mg, isoniazid 300 mg, pyrazinamide 1,000 mg, and ethambutol 750 mg, administered as three tablets of the four-drug FDC (4FDC) daily for an intensive phase of two months. Pyridoxine 10 mg/day was co-administered to prevent isoniazid-induced peripheral neuropathy. Given the diagnosis of TBME, adjunctive therapy included intramuscular streptomycin and intravenous dexamethasone during the initial two months. Dexamethasone was initiated at a dose of 5 mg/day and gradually tapered according to clinical response.

Supportive management included blood transfusions and oral iron supplementation to correct normocytic normochromic anemia, with a therapeutic goal of maintaining hemoglobin above 10 g/dL. Nutritional support was provided in the form of a high-protein diet and oral albumin supplementation to address hypoalbuminemia.

The patient was hospitalized for a total of 17 days. Her management involved a multidisciplinary team comprising specialists from the Departments of Pulmonology, Neurology, Ophthalmology, and Internal Medicine. During the course of hospitalization, the patient showed significant clinical improvement. Left-sided ptosis demonstrated gradual resolution (Figure 2B), her headache intensity decreased substantially, and her diplopia resolved completely.

She was discharged in stable condition and scheduled for regular outpatient follow-up in both pulmonology and neurology clinics. The total planned duration of ATT was approximately 12 months, contingent upon clinical and radiological response. Follow-up evaluation of PTB included repeat sputum GeneXpert MTB/RIF testing at the second, fifth, and sixth months of therapy.

DISCUSSION

As MTB is transmitted via airborne droplets, prolonged or close contact with active TB patients significantly increases the risk of infection.[3] In individuals with weakened immune systems, a latent TB infection may progress to active disease, potentially allowing hematogenous dissemination of MTB to extrapulmonary sites, including the CNS.[4] This case illustrates concurrent PTB and TBME, a manifestation of disseminated TB, in an immunocompetent individual, which underscores the importance of early diagnosis and prompt initiation of ATT.

Cerebral TB is considered the most severe manifestation of EPTB, typically occurring in children and immunocompromised individuals. Tuberculous meningoencephalitis, one of its most life-threatening forms, results from the hematogenous dissemination of MTB following a failure of the host immune system to contain the primary infection.[3] Recent studies have shown that co-infection with HIV or hepatitis B virus (HBV) is associated with poor treatment outcomes in TB, likely due to impaired immune responses and delayed bacterial clearance.[2,6] In this case, the patient tested non-reactive for both HIV and HBsAg, and therefore was considered immunocompetent.

Extrapulmonary TB often presents with non-specific symptoms depending on the affected site. In TBME, common features include persistent headache, vomiting, low-grade fever, and, in advanced cases, altered mental status.[7] Neurological signs such as cranial nerve palsies, motor or sensory deficits, and visual disturbances may also occur, alongside systemic TB symptoms including chronic cough, fever, fatigue, and weight loss.[2,8] The patient had classic PTB symptoms three months before admission. The symptoms included a productive cough, low-grade fever, fatigue, anorexia, and weight loss. However, these symptoms resolved on their own and were initially

ignored. Her immune system might have initially contained the infection in granulomas, resulting in latent TB. A later decline in immune control was likely to lead to hematogenous spread and subsequent CNS involvement, manifesting as TBME.

The patient presented with a two-month history of progressive bilateral headache, followed by diplopia and left-sided ptosis, neurological features suggestive of TBME. A retrospective cohort study in Indonesia reported headache in 84.0% and cranial nerve palsy in 37.1% of TB meningitis cases.[9] The headache was likely due to increased intracranial pressure from multiple tuberculomas, while cranial nerve III involvement explained the ptosis and diplopia. Based on the British Medical Research Council (BMRC) classification, she was categorized as grade II TBME (GCS 15 with focal neurological deficits), indicating moderate disease severity.[10]

Chest examination revealed signs of consolidation in the upper third of the left hemithorax. Although physical findings in TB are generally non-specific, a 2020 review noted vesicular breath sounds and crackles in 80% and 52.5% of TB cases, respectively.[11] The observed signs in the patient suggested chronic pulmonary inflammation, likely from prolonged disease.[8]

Neurological examination revealed left-sided ptosis and diplopia, indicative of involvement of cranial nerve III (oculomotor nerve). The preservation of other extraocular movements suggested partial rather than complete nerve involvement. Cranial nerve palsy occurs in approximately 30% of TBME cases.[5] However, isolated third nerve palsy remains a rare occurrence.[5] In this case, the symptoms were likely due to inflammatory processes related to TB infection, causing compression or irritation of the oculomotor nerve. Additionally, intracranial tuberculomas may have exerted a mass effect, disrupting the oculomotor pathway and contributing to the ocular manifestations.[5]

Interestingly, the patient did not exhibit meningeal signs, which are commonly associated with TBME. Neck stiffness, the most typical sign of meningeal irritation, is reported in approximately 40-80% of TBME cases.[12] However, its absence does not rule out the diagnosis. In early TBME, inflammation may not yet involve the cervical musculature, leading to a lack of clinical signs.[13] Additionally, the sensitivity of physical examination for meningeal signs is estimated to be below 40%, further limiting their diagnostic utility.[14] Thus, the absence of meningeal signs in this case does not contradict the diagnosis of TBME.

Further laboratory and radiological findings supported the diagnosis of both PTB and TBME. Chest radiography revealed fibroconsolidation with multiple cavities and apical pleural thickening in the left lung. Although radiographic features of TB can mimic other pulmonary conditions, cavitation and fibrosis are characteristic of chronic TB infection.[2] Cavitary lesions are commonly seen in post-primary TB, including cases of reactivation or reinfection.[2] In this patient, chronic undiagnosed and untreated PTB was likely to contribute to the hematogenous spread of MTB, resulting in TBME.

Neuroimaging and CSF analysis further supported the diagnosis of TBME. Contrast-enhanced brain CT revealed leptomeningeal enhancement, raising suspicion for TBME. The brain MRI showed features consistent with both TBME and tuberculomatous disease. Magnetic resonance spectroscopy revealed no increase in the Ch/Cr and Ch/NAA ratios, suggesting that the lesions were non-neoplastic and likely of infectious origin.[15] Tuberculous CNS involvement typically presents with meningitis, hydrocephalus, or tuberculomas.[16] Parenchymal and leptomeningeal enhancement are the most frequent MRI findings (38-80%) in TBME, while tuberculomas are seen in 46-70% of cases.[17]

These radiologic findings aligned with the patient's clinical presentation and CSF analysis, which confirmed RIF-sensitive MTB. Cerebrospinal fluid results showed pleocytosis with mononuclear dominance, elevated protein, and low glucose levels, which are typical findings in TBME. Mononuclear predominance reflects a T-cell-mediated inflammatory response, while increased protein levels result from blood-brain barrier disruption. Glucose depletion in CSF is attributed to impaired glucose transport and bacterial metabolism.[18] These CSF abnormalities are frequently observed in TBME patients.

Laboratory findings further reflected the chronic nature of the patient's TB infection. She exhibited hypochromic microcytic anemia and hypoalbuminemia. Chronic TB infection promotes increased iron sequestration by macrophages, impairing erythropoiesis and contributing to anemia.[19] Additionally, decreased nutritional intake was likely to exacerbate this effect. Hypoalbuminemia is attributed to both infection-driven increased protein catabolism and poor nutritional status, as protein is essential for tissue repair and regeneration in chronic disease states, including TB.[20]

According to the Indonesian guidelines of TB diagnosis and management, disseminated TB is treated similarly to PTB, as first-line anti-TB drugs (ATD) exhibit excellent CSF penetration.[10] The patient received three tablets of 4FDC for two months, followed by three tablets of two-drug FDC (2FDC) for 7-10 months during the continuation phase, adjusted based on clinical response, disease severity, and immune status.[21] Ethambutol was administered under supervision due to potential ocular side effects that

could exacerbate her symptoms. Streptomycin, given parenterally for meningoencephalitis, has demonstrated synergistic bactericidal activity with RIF and superior CSF penetration compared to ethambutol.[22]

The use of corticosteroids remains controversial due to their immunosuppressive effects. However, in this case, the benefits outweighed the risks. Corticosteroids reduce meningeal inflammation and vasogenic edema, thereby lowering intracranial pressure and potentially preventing pleural complications, such as thickening or adhesions.[23,24] Management of disseminated TB poses diagnostic and therapeutic challenges due to nonspecific constitutional symptoms, underscoring the importance of bacteriological confirmation.[25] In endemic regions like Indonesia, prompt initiation of ATT is critical when TB is suspected.

This case represents bacteriologically confirmed TB with concurrent TBME and PTB. Tuberculous meningoencephalitis is a rare form of EPTB, particularly uncommon in immunocompetent individuals like the patient, who lacked known immunosuppressive risk factors. Histopathologically, intracranial tuberculomas, as observed in this patient, are challenging to diagnose due to the invasiveness of brain biopsy. Therefore, after excluding other intracranial pathologies such as tumors, parasitic infections, and aneurysms, and considering the patient's significant history of TB exposure, prompt initiation of ATT was warranted. A previous study indicated that household contacts of infectious TB patients have a substantially increased risk of developing active TB compared to non-household contacts.[26] Another report documented a 76.6% rate of EPTB within five years following close contact with active TB cases.[27] Given the diagnostic and therapeutic challenges of EPTB, emphasis on rigorous contact tracing and screening is critical to prevent ongoing transmission.[28]

The cause of this patient's ptosis and diplopia was initially unclear, as gaze palsy and anisocoria were absent. Emergency etiologies, including posterior communicating artery aneurysm and vertebrobasilar occlusion, were ruled out due to normal pupillary function and unremarkable MRA findings.[29] A Pancoast tumor causing Horner syndrome was also considered due to left apical opacity, but was excluded given the absence of miosis, anhidrosis, and enophthalmos.[30] The most likely cause was identified on brain imaging, which showed leptomeningeal enhancement near the oculomotor nerve pathway, suggesting TB-related exudative inflammation and subsequent nerve compression.[31] Although multiple tuberculomas were also present, their contribution to raised intracranial pressure remains uncertain. Notably, the patient showed marked improvement in ocular symptoms after 25 days of ATT, supporting a tuberculous origin. Early diagnosis and treatment are essential to reduce TB-related complications.

CONCLUSION

Currently, TB remains a significant health concern in Indonesia. This case demonstrates that TB dissemination can occur in the absence of immunosuppression, indicating that additional risk factors may contribute to its pathogenesis. High-intensity exposure to MTB resulting from years of household contact may facilitate disease progression, with patients often exhibiting atypical manifestations that pose diagnostic challenges. As portrayed in this case, the presence of ptosis and diplopia may mimic other neurological or ophthalmologic conditions, potentially leading to delayed diagnosis of the underlying disease. Early diagnosis of TB following active contact tracing and timely initiation of ATT could have prevented the progression to TBME in this patient. Nevertheless, favorable outcomes are still achievable with the administration of first-line ATT, even after the onset of TBME. This underscores the critical importance of prompt recognition and early treatment in improving prognosis among patients with TB.

Consent

Written informed consent was obtained from the patient.

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Conflict of Interest

The authors declared there is no conflict of interest.

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Authors' Contributions

Methodology: NAZ, AASPP. Investigation: NAZ, AASPP. Data gathering: NAZ. Data analysis: NAZ, AASPP, TK. Drafting: NAZ, AAS. Approval: TK.

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