# Tuberculoma With Visual and Vestibular Symptoms in A β-Thalassemia Patient: A Case Report

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**Abstract**

**Background:** Tuberculosis (TB) is an infectious disease caused by *Mycobacterium tuberculosis*, which can affect the lungs (pulmonary TB) as well as other organs (extrapulmonary TB), including the central nervous system (CNS). The number of TB cases in Indonesia remains high, with 969,000 cases reported in 2021. Tuberculoma is a manifestation of TB in the CNS, presenting as an intracranial mass due to the spread of TB from other organs. It is a rare and serious form of CNS TB infection. The presence of congenital β-thalassemia adds complexity to the pathogenesis, diagnosis, and treatment of tuberculoma.

**Case Description:** A 25-year-old female with a history of β-thalassemia HbE intermedia with a major phenotype presented with the main complaint of blurred vision in the lower right visual field. The patient also experienced vertigo, headache, and a history of seizures. Examination revealed inferior right quadrantanopia, anemia, leukopenia, and thrombocytopenia. Imaging and CSF analysis supported a diagnosis of intracranial tuberculoma. The patient was treated with anti-TB medication and intravenous dexamethasone.

**Discussion:** Patients with thalassemia may have an increased risk of TB infection, including CNS TB. Tuberculoma involves granuloma formation in the brain, often in the meninges and cerebrum. Its manifestations vary and can include headaches and seizures. Diagnosis is based on clinical history, radiological findings, and laboratory results. The patient's symptoms and imaging findings supported a diagnosis of tuberculoma, for which anti-TB and neurorestorative therapy was administered.

**Conclusions:** Tuberculoma, as a manifestation of CNS TB, presents with variable and non-specific symptoms. Visual field disturbances can be one of the presenting symptoms that aid in diagnosis. Clinical, radiological, and laboratory evaluations are necessary for prompt and accurate diagnosis. Treatment involves anti-TB therapy and dexamethasone. This case highlights the importance of early diagnosis and comprehensive management of complex extrapulmonary TB infections.

## Introduction

Tuberculosis (TB) is a contagious infectious disease caused by the bacterium Mycobacterium tuberculosis, which can affect the lungs (pulmonary TB) and other organs (extrapulmonary TB).[1] TB remains one of the major public health issues in Indonesian society due to its high morbidity and mortality rates. In 2021, there were

969,000 reported TB cases in Indonesia, according to the Global TB Report 2022.[2]

Tuberculoma is a manifestation of tuberculosis in the central nervous system, characterized by the presence of solid and multiple intracranial masses.[3] These intracranial masses form as a secondary process resulting from the spread of TB from organs other than the lungs.[4] Tuberculoma is a serious and rare form of central nervous system TB infection, alongside abscesses and meningitis. Dispositional factors such as age and immunosuppressive conditions play an important role in the development of extrapulmonary TB infections.[5] Thalassemia is one of the congenital diseases that can increase the risk of TB infection.[6]

## Case Description

A 25-year-old woman presented to the outpatient clinic at Dr. Cipto Mangunkusumo Hospital (RSCM) with a chief complaint of blurred vision in the lower right visual field that had been occurring intermittently for two weeks prior to hospital admission. The patient reported experiencing double vision and seeing rainbow-like shadows when exposed to sunlight. The symptoms were intermittent, lasting about five minutes each episode. Over the past week, the patient also complained of episodes of vertigo lasting 2–3 minutes, followed by pressing headaches on all sides of the head. These symptoms typically occurred while sleeping or lying down. The vertigo occurred once every two days and was not always accompanied by headaches. The patient denied any specific triggers.

The patient had a history of seizures, first occurring two years ago. The seizures were characterized by unknown preictal symptoms, ictal upward gaze to the right, head turning to the right, tonic-clonic movements in all four extremities, loss of consciousness for approximately five minutes, followed by postictal confusion for about 15 minutes before returning to baseline consciousness. The patient experienced several more seizures afterward but never took anti-epileptic medication. Additionally, the patient had a history of numbness throughout the body, including the face, and stiffness with pain in both hands. She had a known diagnosis of beta thalassemia HbE intermedia with a major phenotype and had been receiving regular transfusions every two weeks since the age of five. The patient had experienced gradual weight loss over the past three years, with a significant decrease of 10 kg in the past six months. She denied any history of fever, chronic cough, shortness of breath, asthma, or allergies.

On physical examination, the patient had a Glasgow Coma Scale (GCS) of E4M6V5, with stable vital signs (BP: 100/82 mmHg, HR: 108 bpm, RR: 20 bpm, Body temperature: 36.5ºC). Neuro-

ophthalmological examination revealed visual field disturbances with blurred vision in the lower right visual field.

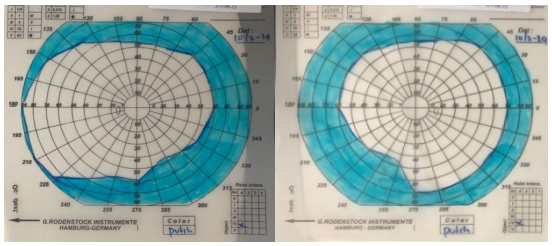

**Figure 1.** Campimetry Examination of Both Eyes (ODS)

Campimetry showed lower right quadrantanopsia in both eyes. Fundus examination (fundoscopy) revealed normofundus in both eyes. Neuro-otological examination did not yield significant findings to support the patient's complaint of vertigo.

**Figure 2.** Fundoscopy Examination of Both Eyes (ODS)

Additional investigations included blood laboratory tests. Results showed decreased hemoglobin (Hb) at 11.1 g/dL, hematocrit at 31.6%, leukocyte count of 3,200/µL, and platelet count of 43,000/µL. Cerebrospinal fluid (CSF) analysis showed cyto-protein dissociation and reduced CSF glucose compared to serum glucose. Gen Xpert CSF MTB was not detected, Acid-Fast Bacilli (AFB) smear was negative, and LAM TB was also negative.

Contrast-enhanced brain MRI revealed multiple intra-axial nodules with ring enhancement in the cortical regions of both cerebral and cerebellar hemispheres, as well as in the left mesencephalon, accompanied by perifocal edema and calcifications. A cystic lesion with CSF intensity was observed in the right middle cranial fossa, consistent with an arachnoid cyst. There were no signs of hydrocephalus, infarction, or intracranial hemorrhage. Compared to previous imaging, there was improvement in the left temporal rim-enhancing lesion, although perifocal edema remained. The patient was treated with first-line anti-tuberculosis therapy consisting of 2 months of RHZE followed by 4 months of RH, along with intravenous dexamethasone administered at a dosage of 5 mg three times daily.

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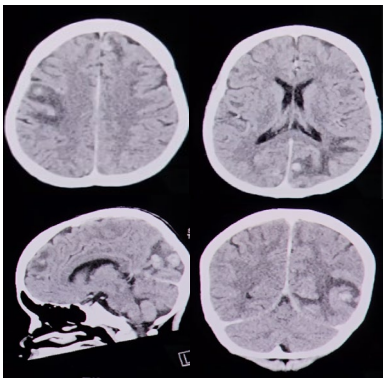


**Figure 3.** Contrast-Enhanced CT Scan of the Brain (Axial, Sagittal, and Coronal Views).

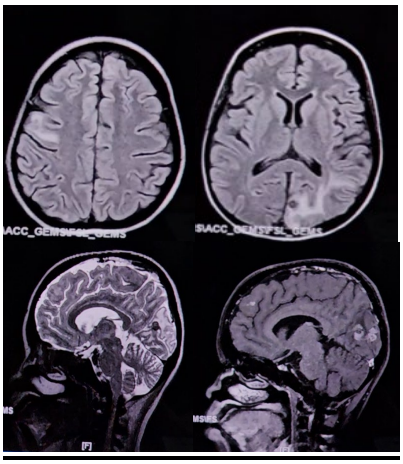


**Figure 4.** Contrast-Enhanced Brain MRI (Axial and Sagittal Views)

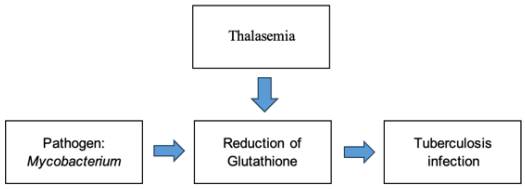
## Discussion

Tuberculoma is a mass lesion in brain tissue formed from granulomas consisting of tuberculous cells and other inflammatory tissues. Tuberculomas frequently occur in the meninges and cerebrum, and more rarely in the cerebellum. They can present as either solitary or multiple lesions in the brain.[7] Extrapulmonary TB infections that can affect the central nervous system (CNS) include tuberculoma, TB meningitis, TB encephalopathy, TB vasculopathy, TB brain abscess, Pott's disease, and spinal meningitis. The incidence of extrapulmonary TB infection in the CNS is approximately 6.3%.[8]

Tuberculoma does not present with specific symptoms. Its manifestations result from mass effect and elevated intracranial pressure, and they depend on the size, number, and location of the lesions. In adults, lesions are commonly found in supratentorial regions, while in children, infratentorial locations are more typical. Clinical symptoms can vary widely, including headache, seizures, cranial nerve paresis, and other neurological signs. Diagnosis of tuberculoma requires a comprehensive approach that includes patient history, radiological imaging, and laboratory investigations.[9]

This patient has a congenital condition—β thalassemia HbE intermedia with a major phenotype—requiring regular blood transfusions, which may be a risk

factor for TB infection. The link between thalassemia and TB infection lies in the antioxidant system, a critical biological process for protecting against various health issues. The antioxidant system contributes to TB prevention through antimicrobial and immune-enhancing effects mediated by the glutathione pathway. In thalassemia, this pathway is clinically significant as glutathione levels are reduced, increasing the patient's susceptibility to medical conditions, including infection.[6]




**Algorithm 1.** Antioxidant Pathways Involved in Thalassemia and TB Infection.

In this patient, multiple clinical diagnoses were established: contralateral inferior right quadrantanopia with macular sparing due to an upper occipital brain lesion, spontaneous episodic vestibular syndrome (EVS), and a history of seizures. The patient's subacute and progressive focal neurological symptoms suggest an infectious etiology. The lesion in the upper occipital brain due to tuberculoma manifested as right inferior contralateral quadrantanopia with macular sparing, as shown in the visual field map in Figure 5. The spontaneous EVS can also be explained by a tuberculoma lesion in the left mesencephalon. Radiological findings, together with clinical history and laboratory results, supported the diagnosis of tuberculoma.

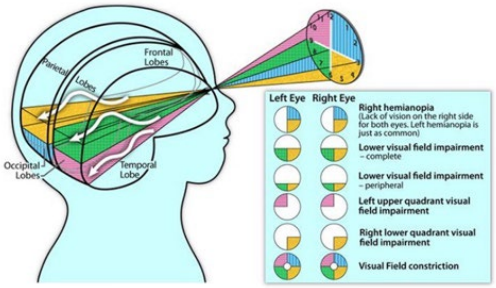



**Figure 5.** Occipital Visual Field Map

This case report emphasizes the patient's visual field disturbance. A lesion in the left occipital lobe caused right inferior quadrantanopia with macular sparing. Macular sparing indicates that the damage in the occipital region is incomplete and that the primary visual cortex is still functioning. This differs from visual field disturbances caused by temporal lobe lesions (involved in the "what" pathway of visual information processing), which typically present with superior quadrantanopia ("pie in the sky") with macular involvement and may include visual agnosia. In contrast, parietal lobe damage (involved in the "where" pathway) can cause inferior quadrantanopia ("pie on the floor") with macular involvement, optokinetic nystagmus impairment, and optic ataxia.

Current patient management includes pharmacological treatment with dexamethasone and anti-tuberculosis therapy, and non-pharmacological

intervention through neurorestorative transcranial magnetic stimulation (TMS) once no contraindications remain.

## Conclusion

Tuberculoma, as a manifestation of TB in the central nervous system, can cause diverse and non-specific symptoms, as seen in this 25-year-old patient. She presented with visual disturbances, seizures, and other neurological symptoms, leading to a tuberculoma diagnosis based on clinical, radiological, and laboratory findings. Her β thalassemia may have contributed as a risk factor for TB infection through compromised antioxidant mechanisms. Treatment involved anti-TB drugs and dexamethasone, with referral for neurorestorative therapy. This case highlights the importance of recognizing symptom s and risk factors for early diagnosis and comprehensive management of complex and rare extrapulmonary TB infections such as tuberculoma.

## References


1. Zahrou F, Elallouchi Y, Ghannane H, Ait Benali S, Aniba K. Diagnosis and management of intracranial tuberculomas: about 2 cases and a review of the literature. *Pan Afr Med J*. 2019 Sep 11;34:1–6. https://doi.org/10.11604/pamj.2019.34.23.17587
2. Collins D, Hafidz F, Mustikawati D. The economic burden of tuberculosis in Indonesia. *Int J Tuberc Lung Dis*. 2017;21(9):1041–8. https://doi.org/10.5588/ijtld.16.0898
3. Perez-Malagon CD, Barrera-Rodriguez R, Lopez-Gonzalez MA, Alva-Lopez LF. Diagnostic and neurological overview of brain tuberculomas: a review of literature. *Cureus*. 2021;13(12):e20579. https://doi.org/10.7759/cureus.20133
4. Mukherjee S, Das R, Begum S. Tuberculoma of the brain—a diagnostic dilemma: magnetic resonance spectroscopy a new ray of hope. *J Assoc Chest Physicians*. 2015;3(1):3–8. https://journals.lww.com/ascp/fulltext/2015/03010/tuberculoma_of_the_brain___a_diagnostic_dilemma_.2.aspx
5. Yogi P, Andrika IP, Sajinadiyasa IGK, Bagiada IM. Diagnosis dan penatalaksanaan tuberkulosis sistem saraf pusat. *Intis Sains Medis*. 2021;12(3):912–6. https://doi.org/10.15562/ism.v12i3.1173
6. Sriwijitalai W, Wiwanitkit V. Tuberculosis in patients with underlying thalassemia: a consideration of common antioxidative pathway − an expressional analysis. *Egypt J Chest Dis Tuberc*. 2021;70(1):38–9. https://doi.org/10.4103/ejcdt.ejcdt_159_19

7. Gregol BM, Berres TO, Barreto T, Giacomelli R, Schwingel D, Oleksinski CG, et al. Brain tuberculoma as a differential diagnosis of single intracranial lesion: case report. *Arq Bras Neurocir*. 2020;39(2):142–5. http://dx.doi.org/10.1055/s-0040-1708895
8. Moussa AA, Mahmoud ME, Yousef HA. Intracranial tuberculoma and recent advances in magnetic resonance imaging. Egypt J Neurosurg. 2018;33(13):1–5. https://doi.org/10.1186/s41984-018-0013-8
9. Kumar EA, Bai PJ. A clinical study of CNS tuberculomas. *Int Arch Integr Med*. 2016;3(6):101–6. http://iaimjournal.com/
10. Blumenfeld H. Neuroanatomy through clinical cases. 2nd ed. Sunderland (MA): Sinauer Associates; 2010.

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