EJPMR, 2026, 13(7), 530-534 **Case Study** **Impact Factor: 8.158**

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

ISSN (O): 2394-3211
ISSN (P): 3051-2573
Coden USA: EJPMAG

CORTICAL BLINDNESS IN CNS TUBERCULOSIS: A DIAGNOSTIC IMPERATIVE — THE EYES SEE BUT THE BRAIN DOESN'T

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How to cite this Article: Dr. Harshavardhana U. H.[1]*, Dr. S. Rajkumar[2], Prof. Dr. Rathnakumar MD[3] (2026). Cortical Blindness In Cns Tuberculosis: A Diagnostic Imperative — The Eyes See But The Brain Doesn't. European Journal of Pharmaceutical and Medical Research, 13(7), 530-534.

Article Received on 15/06/2026 **Article Revised on 05/07/2026** **Article Published on 10/07/2026**

ABSTRACT

Cortical blindness — loss of visual perception arising from bilateral occipital cortical injury with intact peripheral visual apparatus and preserved pupillary light reflexes — is a rare but severe neurological complication of central nervous system (CNS) tuberculosis. Tuberculous meningitis (TBM) induces vasculitis of small and medium-sized cerebral arteries, producing ischaemic infarctions that preferentially involve the posterior circulation, including the basal ganglia, thalami, and occipital lobes. We present a middle-aged male weaver with a history of pulmonary tuberculosis at age 13 who presented with 20 days of low-grade fever with evening rise, unilateral headache, diminished vision (reported by attenders; denied by patient — consistent with Anton syndrome), and gait unsteadiness. Examination revealed Grade 2 clubbing, bilateral cerebellar signs, and a striking dissociation between subjective visual denial and objectively impaired visual function. All peripheral visual structures were intact. CSF analysis revealed the classical TBM profile: protein 126 mg/dL, glucose 73 mg/dL (CSF:blood glucose ratio reduced), lymphocyte-predominant pleocytosis, positive CSF globulin, and ADA 12 U/L. CBNAAT was negative. MRI brain demonstrated acute infarcts in bilateral occipital regions, right thalamus, and bilateral cerebellar hemispheres — the hallmark posterior circulation multi-territory pattern of TBM vasculitis. The patient was initiated on four-drug anti-tubercular therapy, adjunctive dexamethasone, and concurrent management of newly diagnosed diabetes mellitus. This case highlights that cortical blindness in TBM may be heralded by Anton syndrome, that CSF analysis demonstrating elevated protein, lymphocytic pleocytosis, and elevated ADA is essential to confirm TBM, and that early initiation of ATT with corticosteroids is critical to limit irreversible vasculitic injury.

KEYWORDS: CNS tuberculosis; Tuberculous meningitis; Cortical blindness; Anton syndrome; TBM vasculitis; Posterior circulation infarct; Occipital lobe infarction; Anti-tubercular therapy; CSF analysis; Cerebellar infarction.

INTRODUCTION

Cortical blindness is defined as loss of visual perception attributable to bilateral lesions of the primary visual cortex (V1) in the occipital lobes while the peripheral visual apparatus — retina, optic nerves, optic tracts, and lateral geniculate nuclei — remain structurally and functionally intact. It is characterised by the triad of visual loss, preserved pupillary light reflexes, and normal fundoscopy.[1] A clinically deceptive accompaniment is **Anton syndrome** — the paradoxical denial of blindness by the patient, arising from disconnection of cortical self-awareness mechanisms — in which the patient confabulates visual experiences and is unaware of the deficit.

Tuberculous meningitis (TBM) is the most severe form of extrapulmonary tuberculosis, with a case fatality rate of 20–30% in treated patients and up to 50% of

survivors sustaining permanent neurological deficits.[2] A major mechanism of injury in TBM is vasculitis of small and medium-sized cerebral arteries, in which inflammatory infiltration of the adventitia and media of basal perforating arteries produces endothelial injury, luminal narrowing, thrombosis, and posterior circulation ischaemic infarction. The posterior circulation — including posterior cerebral arteries, thalamoperforators, and cerebellar arteries — lies in close proximity to the basilar exudative process, making it particularly vulnerable to TBM vasculitis.[3]

Cortical blindness complicating TBM has been reported in fewer than 30 cases in the indexed English-language literature, making it a genuinely rare manifestation.[4] Its significance lies in the diagnostic trap it creates: patients with visual complaints may be investigated along an exclusively ophthalmological pathway, critically delaying the diagnosis of TBM and the institution of life-saving anti-tubercular therapy (ATT). We present this case to document cortical blindness with Anton syndrome as a manifestation of TBM posterior circulation vasculitis, to illustrate the pitfall of the falsely near-normal initial CSF in the context of concurrent hyperglycaemia, and to reinforce the importance of serial CSF analysis with MRI brain in suspected TBM.

CASE REPORT

History

A middle-aged male weaver, a resident of Tirunelveli, Tamil Nadu, India, presented to Tirunelveli Medical College Hospital with a 20-day history of low-grade fever characterised by evening rise of temperature. He additionally reported a four-day history of unilateral headache (not associated with projectile vomiting), diminished vision (reported by attenders — the patient himself consistently denied any visual impairment, consistent with Anton syndrome), and unsteadiness while walking with a tendency to bump into.

Table 4: Serology, Microbiology, and Cardiac Investigations.

Investigation	Result
ANA	Negative — autoimmune excluded
VDRL	Negative — neurosyphilis excluded
HBsAg / HCV / HIV (HHH)	Negative — viral/HIV CNS excluded
ECG	Normal sinus rhythm
Echocardiography	Normal — cardioembolic source excluded
CV Doppler (carotid + vertebral)	Normal — large vessel disease excluded

Ophthalmological Examination

Pupils 3mm, round, reactive to light bilaterally; Lens clear; EOM full in all directions; No RAPD; Fundus: media clear, arteries and veins normal, foveal reflex present, no papilloedema. This intact peripheral visual apparatus — confirming the cortical origin of the visual loss — was the critical ophthalmological finding.

MRI Brain Findings

MRI brain with DWI/ADC demonstrated acute ischaemic infarcts involving

(1) bilateral occipital regions — bilateral posterior cerebral artery (PCA) territory, providing the anatomical substrate for cortical blindness; (2) **right thalamus** — thalamoperforating branch territory, a classical site of TBM vasculitis; (3) **bilateral cerebellar hemispheres** — PICA/SCA territory, explaining the bilateral cerebellar signs. This multi-territory posterior circulation infarction pattern is characteristic of TBM vasculitis rather than cardioembolic or atherosclerotic disease.[3,4]

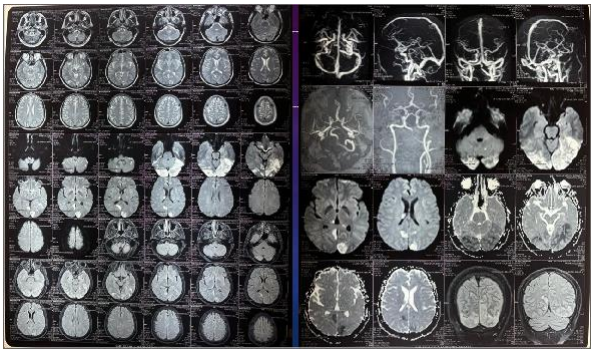

Figure 1: MRI Brain — T2/FLAIR and MRA sequences (axial). Left panel: multiple axial T2-weighted and FLAIR sequences demonstrating bilateral occipital, right thalamic, and bilateral cerebellar hyperintense signal corresponding to acute posterior circulation infarcts. Right panel: MR angiography (MRA) showing cerebrovascular objects.

There was no history of giddiness, speech disturbances, involuntary movements, seizures, loss of consciousness, behavioural disturbance, weakness of limbs, or bladder/bowel dysfunction. Detailed cranial nerve review was negative for diplopia, ptosis, dysphagia, hearing loss, or hoarseness. Past history was significant for pulmonary tuberculosis at age 13, fully treated and completed. No history of diabetes mellitus, hypertension, coronary artery disease, cerebrovascular disease, or chronic kidney disease was reported — however, newly

diagnosed diabetes mellitus was identified during the present admission. Personal history was unremarkable; no adverse social habits. Family history was non-contributory.

Examination Findings

On general examination the patient was conscious, oriented, comfortable at rest, well-built, and well-nourished.

Grade 2 digital clubbing was present— relevant in the context of prior pulmonary TB and possible post-TB bronchiectasis. No pallor, icterus, cyanosis, pedal oedema, lymphadenopathy, neurocutaneous markers, or external markers of active TB/HIV were identified.

Vital signs: pulse 80/min (regular, normal volume, no delays); blood pressure 110/70 mmHg (supine, left arm); JVP not elevated; respiratory rate 16 cycles/min; SpO_2 99% on room air; temperature 37°C.

Cranial nerves: Optic nerve examination was critical — visual acuity formally recorded as normal (intact peripheral visual system), pupils 3 mm RTL bilaterally, EOM full, no RAPD, fundus: media clear, arteries and veins normal, foveal reflex present, no papilloedema. This intact peripheral visual examination in the context of reported visual impairment localised the dysfunction to the cortex. All other cranial nerves (I, III–XII) were intact bilaterally.

Motor, sensory, and reflexes: Muscle bulk symmetric, tone normal throughout, power 5/5 all groups, deep tendon reflexes 2+ bilaterally, plantar responses bilaterally flexor, all sensory modalities intact. **Cerebellar signs:** FNT and FNFT impaired bilaterally; gait broad-based and ataxic; tandem walking impaired; no nystagmus. **Meningeal signs:** Absent — neck stiffness, Kernig's and Brudzinski's signs all negative. No spinal tenderness.

INVESTIGATIONS

Table 1: Serial Haematological and Inflammatory Parameters.

Parameter	07/01/2025	13/01/2025	Ref. Range
Total Count (cells/mms)	14,900	10,600	4,000–11,000
Differential (N/L/E %)	66/25/9	68/25/7	40–70/20–40/1–6
Haemoglobin (g/dL)	16.0	16.1	13.5–17.5
Platelet Count (/mms)	4,80,000	4,20,000	1,50,000–4,00,000
ESR (mm/hr)	90	40	<20
CRP (mg/L)	4	——	<5

Table 2: Blood Glucose and Renal Function Tests.

Parameter	07/01/2025	13/01/2025	Ref. Range
Random Blood Sugar (mg/dL)	286	116	70–140
Fasting Blood Sugar (mg/dL)	146	89	70–100
Post-Prandial BS (mg/dL)	308	173	<140
Serum Urea (mg/dL)	21.0	20.3	15–40
Creatinine (mg/dL)	0.9	0.58	0.7–1.3
Sodium / Potassium (mEq/L)	139 / 4.2	139 / 4.6	135–145 / 3.5–5.0
TSH (µIU/mL)	1.34	——	0.4–4.0
Serum Calcium (mg/dL)	10.0	——	8.5–10.5

Newly diagnosed diabetes mellitus confirmed (RBS 286, FBS 146, PPBS 308 mg/dL). LFTs, coagulation, and thyroid function were within normal limits.

Table 3: Cerebrospinal Fluid Analysis.

Parameter	Result	Ref. Range	Interpretation
CSF Protein (mg/dL)	126	15–45	Markedly elevated — TBM inflammatory exudate
CSF Glucose (mg/dL)	73	>50% blood glucose	Reduced CSF:blood ratio — consistent with TBM
CSF Globulin	Positive	Negative	Positive — supports elevated protein content in TBM
CSF Cytology	Lymphocyte-predominant	0–5 cells	Hallmark of TBM —— T-lymphocyte inflammation
CSF ADA (U/L)	12	<10 U/L	Elevated — supports tuberculous aetiology
CSF CBNAAT	Negative	Negative	Does not exclude TBM; sensitivity 45–80%
CSF Culture and Sensitivity	No growth	Sterile	Negative — consistent with TBM

ESR 90 mm/hr. RBS 98 mg/dL. ECHO and CV Doppler: Normal. ANA, VDRL, HHH: Negative. architecture, with DWI/ADC sequences confirming restricted diffusion at infarct sites.

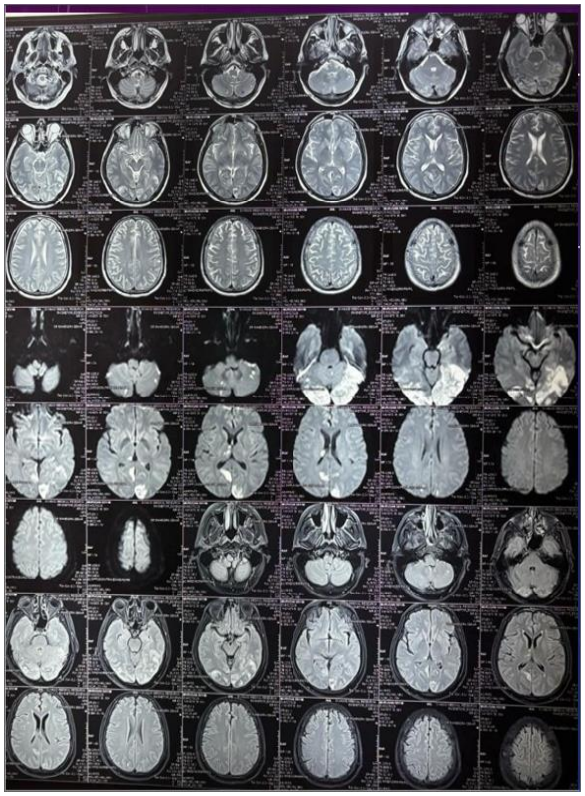

Figure 2: MRI Brain — Serial axial sequences (T2/FLAIR/DWI). Axial cuts from posterior fossa to vertex demonstrating the extent of signal abnormality in the bilateral occipital lobes, right thalamus, and bilateral cerebellar hemispheres, consistent with multi-territory posterior circulation ischaemic infarction from TBM vasculitis.

DIAGNOSIS

The combination of posterior circulation multi-territory infarcts on MRI, serially evolving CSF profile consistent with TBM (protein 126 mg/dL, lymphocytic pleocytosis, ADA 12 U/L), prior history of pulmonary TB, and exclusion of all alternative diagnoses established the diagnosis of Tuberculous Meningitis with posterior circulation vasculitis causing cortical blindness (bilateral occipital infarction) with right thalamic and bilateral cerebellar infarction.

MANAGEMENT AND OUTCOME

Anti-tubercular therapy (ATT) was initiated in accordance with standard recommendations for CNS tuberculosis, which requires a prolonged treatment course given the challenges of blood-brain barrier penetration and the severity of the disease. Pre-treatment liver function was documented as a normal baseline, with serial monitoring planned throughout the treatment course to detect drug-induced hepatotoxicity.

Adjunctive dexamethasone was administered concurrently with ATT and subsequently tapered, in accordance with current guidelines that recommend corticosteroids for all patients with TBM regardless of disease severity. A landmark randomised controlled trial demonstrated that adjunctive dexamethasone significantly reduces mortality in TBM.[6] Corticosteroids act by reducing the inflammatory exudate in the basal cisterns, decreasing vasogenic oedema, and suppressing the vasculitic process responsible for cerebrovascular complications. Antiplatelet therapy was initiated for the ischaemic infarcts following exclusion of haemorrhagic transformation. Newly diagnosed diabetes mellitus was managed with insulin during the acute admission; close glucose monitoring was maintained throughout the corticosteroid course, as steroid therapy adversely affects glycaemic control.

DISCUSSION

This case illustrates three important clinical principles. **First**, the classical triad of cortical blindness — visual loss, intact pupillary reflexes, and normal fundoscopy — must be recognised as a specific clinical syndrome indicating bilateral occipital cortical injury. When this triad is identified in a febrile patient with prior TB history and posterior circulation infarcts on MRI, TBM vasculitis must be considered the diagnosis until proven otherwise.[1,3]

Second, CSF analysis was the cornerstone of diagnosis in this case. The CSF demonstrated the classical TBM profile: markedly elevated protein (126 mg/dL), reduced glucose (73 mg/dL) with a correspondingly reduced CSF:blood glucose ratio, lymphocyte-predominant pleocytosis, positive CSF globulin, and ADA 12 U/L — crossing the commonly used diagnostic threshold. An important interpretive principle is that the absolute CSF glucose must always be evaluated using the CSF:blood glucose ratio rather than the absolute value in isolation, as the ratio reflects the actual metabolic perturbation regardless of systemic glycaemia.[2,5]

Third, CBNAAT negativity (on both taps) must not delay ATT in a patient with high clinical and radiological probability of TBM. The sensitivity of Xpert Ultra on CSF is 45–80%; a negative result excludes neither the diagnosis nor the need for immediate treatment.[8]

Anton syndrome in this patient — the consistent denial of visual loss — reflects cortical anosognosia from bilateral occipital injury. This feature may delay medical consultation (as occurred here, with the visual complaint coming from attenders rather than the patient) and may also create medico-legal complexity. Clinicians should be aware that patient self-report is unreliable in cortical blindness; attender history and objective neuro-ophthalmological testing are essential.[1,4]

Concurrent newly diagnosed diabetes is a clinically important finding. Diabetes increases TB risk approximately 3-fold through impaired T-lymphocyte function and macrophage dysfunction.[9] It may have contributed to the failure to adequately control residual

mycobacterial burden after the index TB episode at age 13. Additionally, corticosteroid therapy will worsen glycaemic control throughout the 6–8-week taper, requiring vigilant glucose monitoring. The interaction between TBM, diabetes, and steroid therapy represents a management challenge that should be explicitly addressed in the treatment plan.

CONCLUSION

Cortical blindness with Anton syndrome is a rare but diagnostically important manifestation of CNS tuberculosis, arising from TBM posterior circulation vasculitis causing bilateral occipital infarction. This case underscores three essential clinical lessons: (1) visual loss with intact pupillary reflexes and normal fundoscopy in a febrile patient with prior TB history and posterior circulation MRI infarcts should be treated as TBM vasculitis until proven otherwise; (2) CSF analysis demonstrating elevated protein, lymphocytic pleocytosis, and elevated ADA constitutes a classical TBM profile that should prompt immediate ATT; and (3) CBNAAT negativity in CSF does not exclude TBM and must not delay ATT. Early four-drug ATT with adjunctive corticosteroids is critical to limit irreversible vasculitic injury and improve neurological outcome.

DECLARATIONS

Conflict of Interest: The authors declare no conflict of interest.

Patient Consent: Written informed consent obtained from the patient and family for publication of clinical data and neuroimaging.

Funding: None declared.

Author Contributions: Dr. Harshavardhana UH (primary clinical management, data collection, manuscript drafting); Dr. S. Rajkumar (clinical supervision, diagnostic reasoning, manuscript revision); Prof. Dr. Rathnakumar MD (overall clinical oversight, senior management decisions, final approval). All authors meet ICMJE criteria for authorship.

REFERENCES

1. Aldrich MS, Alessi AG, Beck RW, Gilman S. Cortical blindness: aetiology, prognosis, and clinical course. *Ann Neurol*, 1987; 21(2): 149–158.
2. Thwaites GE, van Toorn R, Schoeman J. Tuberculous meningitis: more questions, still too few answers. *Lancet Neurol*, 2013; 12(10): 999–1010.
3. Bhatt M, Silber E. Stroke in patients with HIV and CNS tuberculosis. *J Neurol*, 2002; 249: 1355–1358.
4. Shukla R, Abbas A, Kumar P, et al. Evaluation of cerebral infarction in TBM by DWI. *J Infect*, 2008; 57(4): 298–306.
5. Thwaites GE, Nguyen DB, Nguyen HD, et al. Dexamethasone for tuberculous meningitis in adolescents and adults. *N Engl J Med*, 2004; 351(17): 1741–1751.
6. World Health Organization. *WHO Treatment Guidelines for Drug-Susceptible TB, 2017 Update*. Geneva: WHO, 2017.
7. Kashyap RS, Kainthla RP, Mudaliar AV, et al. CSF adenosine deaminase in early diagnosis of TBM. *Cerebrospinal Fluid Res*, 2006; 3: 5.
8. Bahr NC, Nuwagira E, Evans EE, et al. Diagnostic accuracy of Xpert MTB/RIF Ultra for TBM in HIV-infected adults. *Lancet Infect Dis*, 2018; 18(1): 68–75.
9. Jeon CY, Murray MB. Diabetes mellitus increases risk of active tuberculosis: systematic review. *PLOS Med*, 2008; 5(7): e152.
10. Arboix A, Masson I, Codina A, et al. Cortical blindness in ischaemic stroke. *Eur J Neurol*, 2004; 11(3): 163–168.