· Article ·

Heterogeneous etiologies and mechanisms of bilateral visual impairment in tuberculosis: a retrospective clini - cal analysis

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Abstract

Objective We aimed to systematically analyze the potential causes of bilateral visual impairment in patients with tuberculosis (TB), and to explore the underlying mechanisms involving infectious, inflammatory, immune-mediated, vascular, metabolic, and drug-related factors, thereby improving clinical awareness of this uncommon complication.

Methods We retrospectively reviewed seven patients with TB who developed bilateral visual decline at the Eighth Medical Center of the Chinese PLA General Hospital between October 2020 and July 2025. Clinical manifestations, neuroimaging features, immunological and nutritional profiles, medication history, therapeutic response, and prognosis were comprehensively analyzed.

Results Among the seven patients, three had tuberculous meningitis, three had pulmonary TB, and one had tuberculous peritonitis. The etiologies of visual impairment were diverse and included: (1) direct invasion of the optic pathway by TB infection; (2) bilateral occipital lobe infarction secondary to tuberculous vasculitis; (3) immune-mediated optic neuropathies triggered by infection—including myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), neuromyelitis optica spectrum disorder (NMOSD), and antibody-negative immune optic neuritis; (4) metabolic optic neuropathy caused by vitamin B1 deficiency; and (5) febuxostat-associated drug-induced neurotoxicity. Following mechanism-oriented individualized treatment, visual acuity was fully recovered in four patients, partially improved in two, and showed no improvement in the one patient with NMOSD.

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Conclusion TB-associated visual impairment has complex and heterogeneous etiologies beyond classic anti-TB drug toxicity, including direct infectious involvement, tuberculous vasculitis–related ischemia, infection-triggered immune-mediated demyelination, and nutritional/metabolic disturbances. A mechanism-oriented evaluation integrating clinical features, neuroimaging, antibody testing, nutritional assessment, and careful medication reconciliation may facilitate early targeted management. The potential association between febuxostat exposure and reversible optic neuropathy observed in this series is hypothesis-generating and requires further confirmation.

Keywords: tuberculosis, visual impairment, immune-mediated injury, vasculitis, metabolic abnormalities

Introduction

Tuberculosis (TB), caused by Mycobacterium tuberculosis, is a chronic infectious disease that can involve multiple organ systems, including the lungs, central nervous system, and peritoneum [1,2]. Visual impairment during TB treatment is often attributed to ethambutol toxicity [3–5]; however, TB can compromise the visual pathway through additional mechanisms such as optochiasmatic arachnoiditis, tuberculous vasculitis–related infarction, infection-triggered immune demyelination, nutritional/metabolic optic neuropathy, and toxicity from non–anti-TB medications [6–9].

Existing reports largely focus on single etiologies (most commonly ethambutol-associated optic neuropathy) or isolated case reports of TB-related optic neuritis [10,11], leaving clinicians without a practical, mechanism-oriented differential diagnosis when bilateral visual decline occurs in the context of TB. This gap is clinically important because timely mechanism-specific intervention (e. g., intensified anti-TB therapy for optochiasmatic involvement, immunotherapy for immune-mediated optic neuritis, or vitamin repletion for metabolic optic neuropathy) may improve visual outcomes.

Therefore, we retrospectively analyzed seven TB patients who developed bilateral visual impairment by integrating ophthalmologic findings, neuroimaging, immunological and nutritional profiles, medication exposure, treatment response, and prognosis. We aimed to (i) delineate a spectrum of etiological subtypes, (ii) summarize imaging and clinical “red flags” for rapid classification, and (iii) provide an evidence-informed diagnostic framework to support early targeted management in clinical practice.

Materials and Methods

Study design

This was a single-center retrospective clinical study. We consecutively collected information for patients diagnosed with TB at the Eighth Medical Center of the PLA General Hospital who developed bilateral visual impairment between October 2020 and July 2025. All enrolled patients met the following criteria: (1) confirmed diagnosis of TB infection; (2) onset of bilateral visual impairment during the disease course; (3) exclusion of primary ocular disorders and other systemic etiologies.

Data collection

Data were extracted from the electronic medical record system, including demographic information, clinical manifestations, laboratory results, imaging findings, medication exposure, and treatment regimens. Ophthalmologic assessment included ocular motility

(for diplopia/ophthalmoplegia), pupillary light reflex (including relative afferent pupillary defect when available), fundus examination, and visual field testing; ancillary tests such as visual evoked potentials and optical coherence tomography were reviewed when performed.

Statistical analysis

Purely descriptive methods were applied. The results are presented in narrative and tabular formats, and clinical features and outcomes were compared across different etiological categories.

Results

A total of seven patients were included (Table 1). The patients were 19–75 years old; five were men and two were women. The diagnoses included tuberculous meningitis (n=3), pulmonary TB (n=3), and tubercu-

Table 1. Clinical characteristics and outcomes of patients with tuberculosis-related visual impairment.

No.	Sex	Age (yrs)	TB type	Visual acuity (OD/OS)	CSF findings	EMB use (history)	Clinical manifestations	Etiology	Treatment	Outcome
1	M	75	TBM	OD: 0.3 OS: 0.2	WBC 18× 10^6/L; Protein 0.54 g/L	27 days	Fever Headache Diplopia	Direct tuberculous invasion of the optic nerve	Intensified anti-TB therapy	Complete improvement
2	M	59	PTB	OD: 0.5 OS:HM	WBC 9× 10^6/L; Protein 0.92 g/L	3 years	Blurred vision bilateral Ptosis Cognitive decline	Tuberculous vasculitis causing bilateral thalamic involvement	Anti-TB therapy; dexamethasone 10 mg; improved cerebral blood flow	Partial improvement
3	F	54	TBM RTB PTB	OD: 0.3 OS: 0.3	WBC 8× 10^6/L; Protein 3.39 g/L	None	Blurred vision Limb weakness Urinary retention	Immune-mediated (MOGAD)	Anti-TB therapy; methylprednisolone 500 mg	Complete improvement
4	M	19	PTB	OD: NLP OS: NLP	AQP4-Ab 1:100; WBC 5× 10^6/L; Protein 0.47 g/L	None	Bilateral blindness Limb weakness Respiratory disturbance	Immune-mediated (NMOSD)	Anti-TB therapy; methylprednisolone 500 mg; IVIG	No improvement
5	F	72	PTB	OD: 0.0 OS: LP	Normal	None	Bilateral visual decline progressing to blindness	Immune-mediated (suspected infection-triggered autoimmune optic neuropathy)	Anti-TB therapy; methylprednisolone 500 mg; IVIG	Partial improvement
6	M	19	TBP	OD: 0.3 OS: 0.2	Normal	1 month	Bilateral visual decline progressing to blindness; nausea and vomiting	Metabolic cause (vitamin B1 deficiency)	Anti-TB therapy; vitamin B1 supplementation	Complete improvement
7	M	21	TBM	OD: 0.4 OS: 0.3	None	8 months	Blurred vision; Diplopia (no structural ocular abnormalities)	Drug toxicity (febuxostat-associated optic neuropathy)	Discontinued febuxostat; neurotrophic therapy	Complete improvement

Abbreviations: EMB, ethambutol; IVIG, intravenous immunoglobulin; MOGAD, myelin oligodendrocyte glycoprotein antibody-associated disease; NMOSD, neuromyelitis optica spectrum disorder; TBM, tuberculous meningitis; PTB, pulmonary tuberculosis; RTB, renal tuberculosis; TBP, tuberculous peritonitis; CSF, cerebrospinal fluid; OD, right eye; OS, left eye; HM, hand motion; LP, light perception; NLP, no light perception; WBC, white blood cell; AQP4-Ab, Aquaporin-4 antibody.

lous peritonitis (n=1). Five patients developed bilateral visual impairment during anti-TB therapy, and two presented with visual symptoms at the time of TB diagnosis. Patients' visual disturbances manifested as blurred vision, diplopia, or vision loss. All patients received standard anti-TB treatment following confirmation of the diagnosis.

A 75-year-old man with tuberculous meningitis developed progressive blurred vision and diplopia during anti-TB therapy. Ophthalmologic examination revealed no fundus abnormalities, and both serum and cerebrospinal fluid (CSF) demyelinating antibodies were negative. Follow-up contrast-enhanced brain MRI (Figure 1B) showed obvious enlargement of the tuberculous lesions at the skull base and around the optic chiasm compared to the initial MRI (Figure 1A), suggesting that the visual impairment resulted from direct invasion of the visual pathway by TB infection rather than drug toxicity. After intensified anti-TB treatment, the diplopia returned to its pre-onset state, his blurred vision completely resolved, and brain MRI lesions showed marked resolution (Figure 1C).

A 59-year-old man with a 2-year history of pulmonary TB and irregular anti-TB treatment presented with a sudden onset of bilateral lower-limb weakness, blurred vision in both eyes, and cognitive decline. Neurological

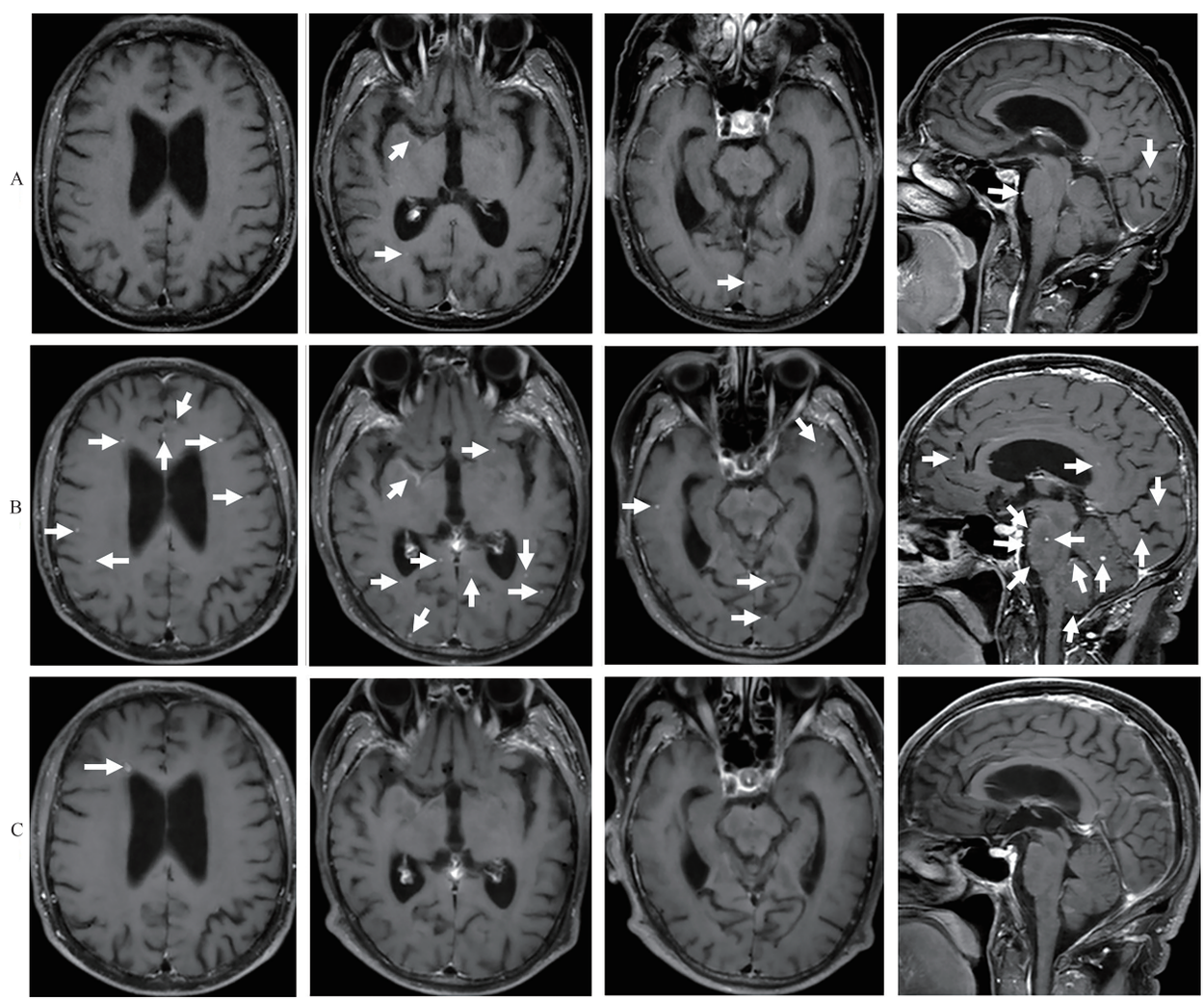


Figure 1. Evolution of contrast-enhanced brain magnetic resonance imaging (T1+C) in Case 1. (A) A few tuberculous-enhancing foci were visible in the basal cisterns at initial presentation. (B) One month later, the number of enhancing lesions were increased markedly (white arrows). (C) After intensified anti-tuberculosis treatment, the lesions were substantially reduced.

examination revealed finger-counting vision in the left eye, visual acuity of 0.5 in the right eye, and grade III muscle strength in both lower limbs. Brain MRI demonstrated multiple acute infarctions in the bilateral occipital lobes, hippocampi, and thalami, showing a symmetric distribution (Figure 2A-2C). Magnetic resonance angiography (MRA) revealed no stenosis or occlusion of major cerebral vessels (Figure 2D). The patient had no conventional risk factors for cerebral infarction, such as hypertension, diabetes, or atrial fibrillation. Given his long-standing inadequate anti-TB therapy and the characteristic infarct pattern (symmetric posterior circulation involvement), tuberculous vasculitis–related small arterial occlusion was considered the most likely etiology. After administration of low-dose dexamethasone (10 mg/day) and treatment to improve cerebral microcirculation, visual acuity improved to 0.8 in the right eye and to 0.4 in the left eye, and cognitive impairment partially resolved.

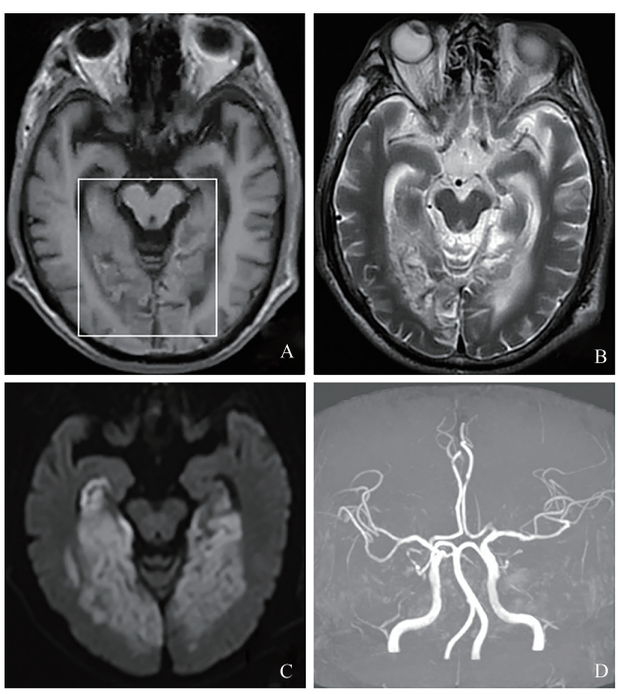


Figure 2. Brain magnetic resonance imaging findings in a patient with infarction related to tuberculous vasculitis. (A) T1WI showing slightly hypointense signals in the bilateral occipital lobes and hippocampi (within white rectangles). (B) T2WI demonstrating hyperintense signals in the same regions. (C) Diffusion-weighted imaging revealing corresponding hyperintensity. (D) Brain magnetic resonance angiography showing no significant large-vessel stenosis, suggesting a high likelihood of small-vessel vasculitic involvement.

A 54-year-old woman was admitted with blurred vision, bilateral lower-limb weakness, and urinary retention. Chest CT revealed multiple patchy opacities with cord-like calcifications in both lungs. Contrast-enhanced brain MRI (T1+C) demonstrated heterogeneous enhancement of the bilateral optic nerves (Figure 3A, white arrows). Spinal MRI revealed linear and patchy T2 hyperintense lesions at T3 – T4 (Figure 3B and 3C, white arrows). Serum myelin oligodendrocyte glycoprotein (MOG) antibody was positive (1:10), and *M. tuberculosis* was detected in the CSF, suggesting MOG antibody-associated disease (MOGAD) triggered in the setting of tuberculous infection. High-dose methylprednisolone (500 mg/day for 5 days) was initiated alongside anti-TB therapy, resulting in marked clinical

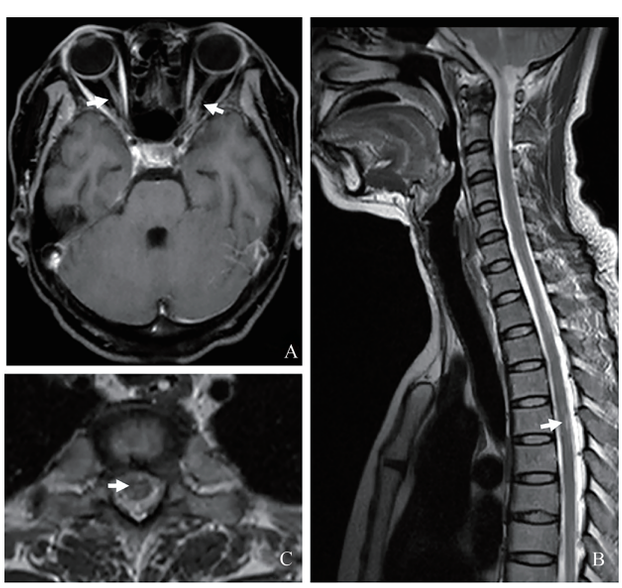


Figure 3. Imaging findings in a patient with tuberculosis-associated MOG antibody-associated disease. (A) Contrast-enhanced brain magnetic resonance imaging (T1+C) showing heterogeneous enhancement of both optic nerves (white arrows). (B, C) Spinal T2WI revealing linear and patchy hyperintensity at the T3 – T4 levels (white arrows), consistent with demyelinating changes.

improvement and complete visual recovery.

A 19-year-old man developed intractable hiccups, vomiting, progressive vision loss, and limb weakness 1 month after being diagnosed with pulmonary TB. Both serum and CSF were positive for Aquaporin-4 (AQP4) antibodies (1∶100). Brain MRI showed bilateral enlargement and tortuosity of the optic nerves (Figure 4A), with patchy enhancement (Figure 4B). Cervical MRI revealed intramedullary lesions at C3 and C5–C7 with heterogeneous enhancement (Figure 4C), consistent with neuromyelitis optica spectrum disorder (NMOSD). Owing to the delayed diagnosis (1 month after symptom onset) and sustained optic nerve injury, the patient's vision did not improve despite treatment with methylprednisolone and intravenous immunoglobulin.

A 72-year-old woman presented with progressive bilateral visual decline. Chest CT from another hospital revealed a right hilar mass with multiple enlarged mediastinal and hilar lymph nodes. Patchy high-density opacities with calcification were observed in the right upper lobe, with mild enhancement and bronchial wall thickening in both lungs. *M. tuberculosis* was detected in bronchoalveolar lavage fluid. Orbital contrast-enhanced MRI showed abnormal enhancement of the intracranial segments of both optic nerves and the optic chiasm (Figure 5). On admission to our department, the patient was nearly completely blind, with only faint light perception in the left eye. Serum and CSF demyelinating antibody panels were negative, and no spinal lesions were found. The binocular visual evoked potential examination showed no abnormalities. Given the prominent optic neuritis features, infectious background, and significant response to corticosteroids plus intravenous immunoglobulin (IVIG), infection-induced autoimmune optic neuritis was considered. Visual acuity gradually improved to 0.6 after treatment.

A 19-year-old man undergoing treatment for tuberculous peritonitis for 4 months developed recurrent vomiting and reduced oral intake due to intestinal obstruction, followed by blurred vision and diplopia that rapidly progressed to bilateral blindness. Ophthalmic examination revealed no specific fundus abnormalities; immunological testing was negative; and MRI showed no demyelinating lesions. Serum vitamin B1 level was markedly reduced (9.8 mmol/L). Considering his poor nutritional status and symptom pattern, metabolic optic neuropathy was diagnosed. Visual improvement occurred rapidly within 3–5 days of vitamin B1 supple-

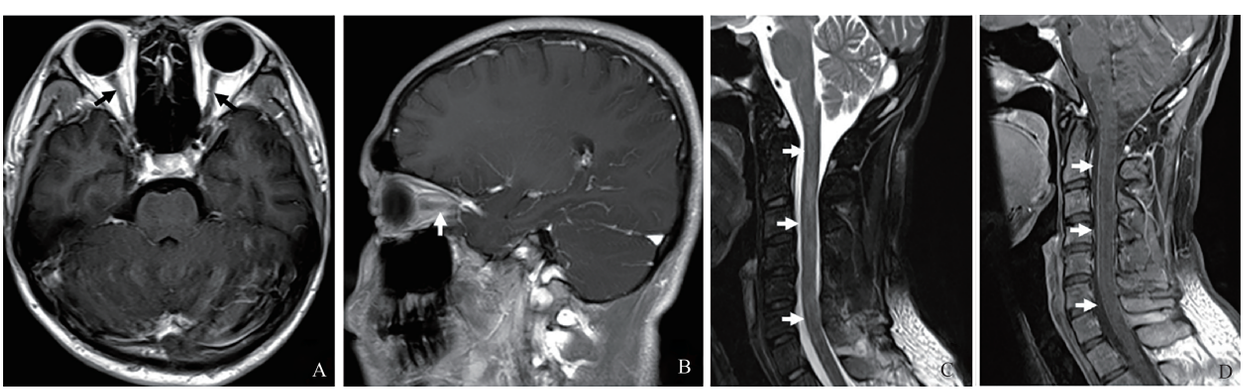


Figure 4. Imaging findings in a patient with neuromyelitis optica spectrum disorder associated with tuberculosis. (A) Brain magnetic resonance imaging demonstrating bilateral optic nerve thickening and tortuosity (black arrows). (B) T1+C images showing patchy enhancement of the optic nerves (white arrows). (C) Cervical spine T2WI showing multisegmental hyperintensity spanning C3 – C7. (D) T1+C images revealing heterogeneous enhancement of the cervical spinal cord accompanied by linear meningeal enhancement.

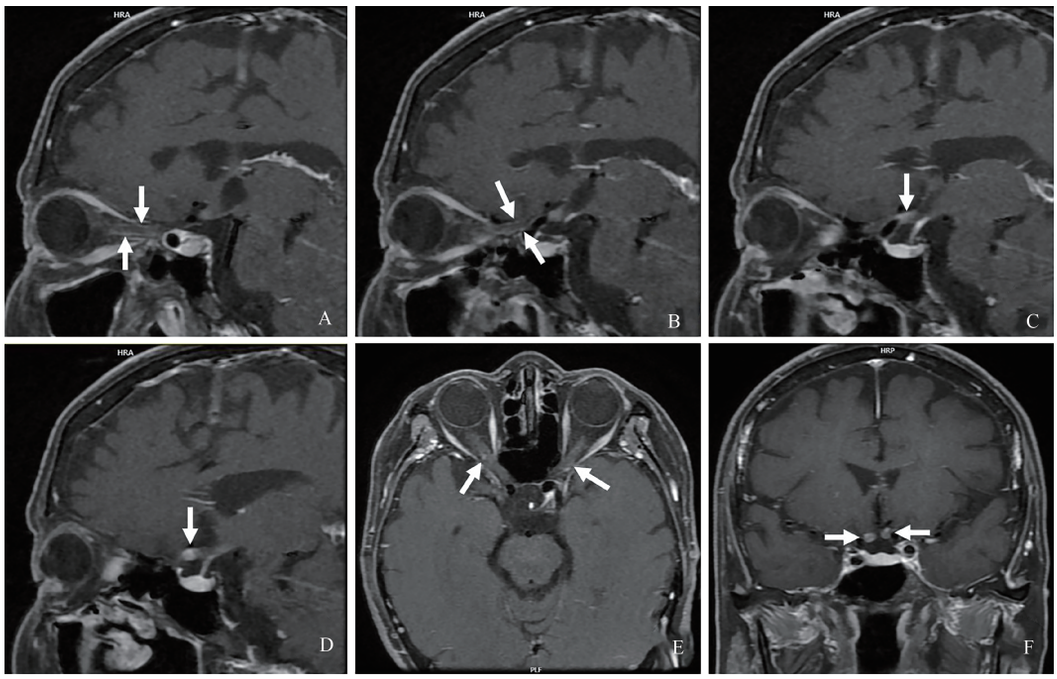


Figure 5. Orbital magnetic resonance imaging (T1+C) in a patient with infection-triggered immune-mediated optic neuritis showing marked enhancement of the bilateral optic nerves and optic chiasm (white arrows), consistent with active optic neuritis.

mentation, and the patient's vision returned to normal within 2 weeks.

A 21-year-old man with tuberculous meningitis developed hyperuricemia (569 μmol/L) secondary to pyrazinamide during anti-TB treatment. After insufficient response to benzbromarone, febuxostat was initiated, and on day 3 he developed bilateral blurred vision. Ophthalmologic and neurologic examinations showed no structural abnormalities, and MRI revealed no specific findings. His symptoms completely resolved within 1 week after discontinuing febuxostat. Based on temporal association, reversibility, and exclusion of other causes, febuxostat-associated toxic neuropathy was considered. This case highlights the need to recognize potential optic nerve toxicity from urate-lowering agents in patients with TB.

Discussion

This study provides a systematic analysis of the etiologies underlying bilateral visual impairment in seven patients with TB. Our findings demonstrate distinct multi-mechanistic patterns of visual pathway injury, including direct infectious involvement, ischemia secondary to tuberculous vasculitis, infection-induced immune-mediated demyelination, metabolic optic neuropathy due to vitamin B1 deficiency, and potential drug-induced neurotoxicity. These mechanisms may occur simultaneously or sequentially, resulting in complex clinical presentations and substantial diagnostic challenges. Integrating clinical features, imaging findings, immunological data, and treatment responses allowed for in-depth mechanistic characterization.

Direct infectious involvement of the visual pathway

Tuberculous meningitis can involve the basal cisterns and optic chiasm, representing a major mechanism of TB-related visual impairment. The underlying pathology includes inflammatory exudate leading to compression, granulomatous infiltration, and perivascular

inflammation causing focal ischemia [6]. In the patient with direct infectious optic pathway involvement, the clinical and radiological evolution closely paralleled an anti-TB treatment response, supporting direct infectious injury rather than drug toxicity. Typical imaging features include optic nerve or chiasmal enhancement, prominent adjacent meningeal inflammation, and gradual resolution following anti-TB therapy. Recent reports further emphasize optochiasmatic involvement in TBM and highlight that early recognition of optic nerve/chiasmal enhancement can be vision-saving [12,13].

Tuberculous vasculitis–related ischemia of the visual centers

Tuberculous vasculitis primarily affects small vessels, particularly perforators supplying the posterior circulation [7,8]. The patient with vasculitic ischemic injury exhibited symmetric acute infarctions in the occipital lobes, thalami, and hippocampi without large-vessel abnormalities on MRA and lacked conventional vascular risk factors. Combined with his history of irregular anti-TB therapy, the findings strongly suggested small-vessel occlusion secondary to tuberculous vasculitis. Symmetrical involvement of posterior circulation structures is a key radiologic clue. Low-dose corticosteroids may alleviate vascular inflammation. The predilection for posterior circulation structures and small-vessel involvement in TBM-related vasculitis has been repeatedly documented and supports our imaging-based inference in the vasculitic ischemia subgroup [7,8].

Immune-mediated demyelinating mechanisms

Three patients in this cohort indicate that TB infection may trigger autoimmune responses through molecular mimicry, bystander activation, or disruptions in T helper 17/regulatory T cell balance [14–17], leading to MOGAD, NMOSD, or antibody-negative immune-mediated optic neuritis. The patients in these cases shared several features: optic nerve enlargement or chiasmal enhancement on imaging, long-segment spinal cord lesions in some cases, positivity for MOG or AQP4 antibodies in two cases, and significant responses to steroids or IVIG. In the antibody-negative patient, the imaging findings and treatment response were strongly suggestive of immune-mediated optic neuritis. Emerging evidence suggests an association between TB and NMOSD, although the certainty of evidence remains low; clinicians should maintain diagnostic vigilance when optic neuritis or longitudinal spinal lesions coexist with TB [18].

Metabolic optic neuropathy

Anti-TB medications, reduced appetite, and chronic infection can result in nutritional deficiencies, particularly of B vitamins [19]. Vitamin B1 plays an essential role in neuronal development and metabolic function, and its deficiency may lead to optic nerve degeneration [20]. The patient with vitamin B1 deficiency highlights the often overlooked risk of vitamin deficiency in patients with TB. The rapid visual recovery following vitamin B1 supplementation supports the diagnosis. Metabolic optic neuropathy is reversible and should be considered in patients with malnutrition, chronic inflammation, or prolonged treatment.

Drug-induced neurotoxicity

In the patient exposed to febuxostat, bilateral blurred vision developed 3 days after initiating febuxostat and resolved promptly after drug withdrawal. Although febuxostat-associated optic neuropathy has not been previously reported, the temporal association and reversibility suggest potential neurotoxicity, possibly via

oxidative stress, mitochondrial dysfunction, or immune susceptibility. This observation underscores the importance of including urate-lowering therapies in the differential diagnosis of visual impairment in patients with TB. Given the absence of established evidence for febuxostat-associated optic neuropathy, our case should be considered a signal requiring further pharmacovigilance rather than definitive proof of causality.

Integrative differential diagnosis in multi-mechanistic etiologies

Based on our findings, the differential diagnosis of TB-associated visual impairment can be summarized as follows. Direct infection: optic nerve/chiasmal enhancement; improvement with anti-TB therapy. Vasculitic ischemia: symmetric posterior circulation infarcts; normal MRA. Immune-mediated demyelination: optic nerve swelling, demyelinating antibody positivity, or steroid/IVIG responsiveness. Metabolic: malnutrition; rapid recovery with vitamin B1. Drug toxicity: onset shortly after medication; rapid improvement after discontinuation.

This framework can facilitate mechanism-based classification and guide targeted therapeutic strategies in complex clinical scenarios.

Clinical relevance and novelty

This work contributes to the understanding of visual impairment in TB by categorizing five major mechanisms and detailing the clinical features of immune-mediated optic neuropathies. Importantly, it also presents preliminary clinical evidence suggesting that febuxostat may be associated with reversible optic neurotoxicity, a finding that warrants further investigation.

Limitations

This study has several limitations. First, the sample size was small and derived from a single center, limiting generalizability and precluding robust statistical inference; selection bias is possible. Second, as a retrospective analysis, the ophthalmologic workup was not fully standardized across cases: some patients lacked complete color vision testing, OCT, visual evoked potentials, or detailed visual field documentation, which may have led to under-characterization of certain phenotypes. Third, follow-up duration and timing of imaging and laboratory testing varied, limiting longitudinal comparisons. Finally, the potential association between febuxostat and optic neuropathy is based on temporal correlation and exclusion of alternatives; causality cannot be established. Prospective multicenter studies with standardized neuro-ophthalmic protocols are needed to validate the proposed mechanism-based classification and to refine management strategies.

Conclusion

Bilateral visual impairment in patients with TB may arise from multiple etiologies, including direct optochiasmatic infection, vasculitic ischemia, infection-triggered immune-mediated demyelination, nutritional/metabolic disturbance, and medication-related toxicity. We propose a mechanism-oriented diagnostic approach that integrates symptom pattern, neuroimaging, antibody testing, nutritional assessment, and careful medication reconciliation to enable early targeted intervention. Our observation of reversible visual decline temporally associated with febuxostat exposure should be interpreted as hypothesis-generating and warrants further pharmacovigilance and confirmatory studies.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Eighth Medical Center of the PLA General Hospital. Written informed consent for publication was obtained from the patients' families.

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Conflict of interest

All authors declare no conflicts of interest.

Author contributions

Jinli Feng and Ming Fang: study design, manuscript drafting. Zhuangzhuang Ren: manuscript drafting. Lihong Wu: literature review. Shuang Wang and Jun Zhao: figure and table preparation. Hongfen Wang: clinical data collection and organization. Jinming Han and Feng Qiu: manuscript revision and supervision.

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