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Original Research

Bilateral Lateral Rectus Palsy in Tubercular Meningitis: A Rare Neuro-Ophthalmic Manifestation

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ABSTRACT:

Background: Tubercular meningitis (TBM) is a life-threatening condition with diverse neurological manifestations. Cranial nerve palsies, particularly of the sixth nerve, are common; however, bilateral lateral rectus palsy is a rare and underreported presentation. **Objective:** To evaluate the clinical profile, imaging findings, and treatment outcomes in TBM patients presenting with bilateral sixth nerve palsy. **Methods:** Twelve patients with confirmed TBM and bilateral lateral rectus palsy were prospectively analyzed. Clinical features, CSF parameters, and MRI findings were documented, and treatment outcomes were followed over 6 months. **Results:** All patients presented with diplopia and headache; 83.3% had papilledema. MRI revealed basal exudates in 75% and hydrocephalus in 33.3%. Full recovery of nerve function was achieved in 83.3% of patients with anti-tubercular therapy and corticosteroids. **Conclusion:** Bilateral sixth nerve palsy in TBM is indicative of extensive basal meningeal inflammation. Early diagnosis and prompt therapy result in favorable outcomes.

Keywords: Tubercular meningitis, lateral rectus palsy, cranial nerve VI, neuro-ophthalmology, basal exudates, intracranial

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INTRODUCTION

Tubercular meningitis (TBM), a manifestation of Mycobacterium tuberculosis in the central nervous system (CNS), remains a significant cause of mortality and neurological sequelae in endemic countries like India [1]. Neurological complications include hydrocephalus, stroke, and cranial nerve palsies, which occur in up to 30–50% of TBM patients [2].

The sixth cranial nerve (abducens nerve), due to its long intracranial course, is particularly vulnerable to compressive and inflammatory insults in TBM. While unilateral abducens palsy is more frequently reported, **bilateral lateral rectus palsy is rare**, with very limited data available in medical literature [3-5]. The abducens nerve innervates the lateral rectus muscle, which abducts the eyeball; its impairment leads to diplopia and restricted outward gaze bilaterally.

This study presents a prospective series of TBM cases with bilateral sixth nerve involvement, exploring clinical presentation, imaging patterns, and treatment outcomes, with an emphasis on early diagnosis and neurological prognosis.

MATERIALS AND METHODS

Study Design and Setting

A prospective observational study was conducted at the Department of Neurology. Ethical clearance was obtained from the Institutional Review Board.

Inclusion Criteria

- Age >12 years
- Confirmed TBM based on consensus clinical, radiological, and laboratory criteria.
- Presence of bilateral lateral rectus palsy confirmed by neuro-ophthalmological assessment

Exclusion Criteria

- Diabetes or other systemic causes of cranial neuropathy
- HIV coinfection
- Pre-existing ophthalmological disease

Diagnostic Evaluation

Patients underwent:

- Neurological and ophthalmological examination
- Contrast-enhanced MRI brain with MR cisternography
- CSF analysis (protein, glucose, cell count, Ziehl-Neelsen stain, GeneXpert)
- Fundoscopy for papilledema

All patients were treated with a standard anti-tubercular regimen (HRZE for 2 months followed by HR for 10 months) and intravenous dexamethasone tapered over 6 weeks.

RESULTS

1. Demographic and Clinical Characteristics

A total of 12 patients were enrolled, with a mean age of 32.6 years (range 19–48 years). There were 7 males (58.3%) and 5 females (41.7%). The average duration of neurological symptoms before presentation was 9.3 ± 4.1 days. All patients (100%) presented with headache and horizontal diplopia, while fever was noted in 11 patients (91.6%), and neck stiffness in 9 patients (75%). Papilledema was observed on fundoscopy in 10 patients (83.3%). Fig 1

Table 1: Patient Demographics and Clinical Presentation (n = 12)

Parameter	Value
Mean Age (years)	32.6 ± 8.9
Sex (Male/Female)	7/5
Mean Symptom Duration (days)	9.3 ± 4.1
Headache	12 (100%)
Fever	11 (91.6%)
Diplopia	12 (100%)
Papilledema	10 (83.3%)
Neck Stiffness	9 (75%)
Altered Sensorium	4 (33.3%)

2. CSF Analysis and MRI Findings

Cerebrospinal fluid (CSF) analysis showed elevated protein and lymphocytic pleocytosis in most patients. The mean CSF protein was 123.5 mg/dL (range: 89–174 mg/dL), while the CSF glucose was reduced, with a mean value of 32.1 mg/dL (range: 24–43 mg/dL). The mean CSF cell count was 89 cells/mm³ (90% lymphocytes). Ziehl-Neelsen staining was positive in 3 cases (25%), and GeneXpert MTB/RIF was positive in 9 cases (75%).

MRI brain with contrast revealed basal meningeal exudates in 9 patients (75%), moderate communicating hydrocephalus in 4 cases (33.3%), and infarcts in the basal ganglia in 2 patients (16.6%).

Table 2: CSF Parameters and Radiological Findings

Parameter	Mean ± SD / Frequency (%)
CSF Protein (mg/dL)	123.5 ± 27.4
CSF Glucose (mg/dL)	32.1 ± 6.4
CSF Cell Count (cells/mm³)	89 ± 18.6 (90% lymphocytes)
ZN Stain Positive	3/12 (25%)
GeneXpert MTB Positive	9/12 (75%)
Basal Exudates (on MRI)	9/12 (75%)
Hydrocephalus	4/12 (33.3%)
Infarcts (Basal Ganglia or Thalami)	2/12 (16.6%)

3. Abducens Nerve Palsy Features

All 12 patients had bilateral lateral rectus weakness. Among them, 8 patients (66.6%) had complete bilateral palsy with severe restriction of lateral gaze in both eyes, while 4 patients (33.3%) had partial abduction restriction. No patients had pupil involvement or optic nerve palsy.

Table 3: Sixth Nerve Involvement and Initial Ophthalmic Findings

Clinical Feature	Number (%)
Bilateral Complete LR Palsy	8 (66.6%)
Bilateral Partial LR Palsy	4 (33.3%)
Diplopia on Lateral Gaze	12 (100%)
Nystagmus	0 (0%)
Optic Nerve Involvement	0 (0%)

4. Treatment Outcomes

All patients received WHO-recommended anti-tubercular therapy (ATT) and a 6-week tapering course of intravenous dexamethasone. Patients were monitored monthly for six months.

- **At 3 months**, **6 patients (50%)** had complete resolution of abducens palsy, **4 patients (33.3%)** had partial improvement, and **2 patients (16.6%)** had persistent complete palsy.
- **At 6 months**, full recovery was noted in **10 patients (83.3%)**, while **2 patients (16.6%)** continued to have persistent bilateral diplopia, attributed to fibrotic nerve entrapment or infarction.

Table 4: Recovery Status Post-Treatment (n = 12)

Outcome	3 Months	6 Months
Full Recovery	6 (50%)	10 (83.3%)
Partial Recovery	4 (33.3%)	0 (0%)
No Recovery	2 (16.6%)	2 (16.6%)
Visual Function Restored	9 (75%)	12 (100%)

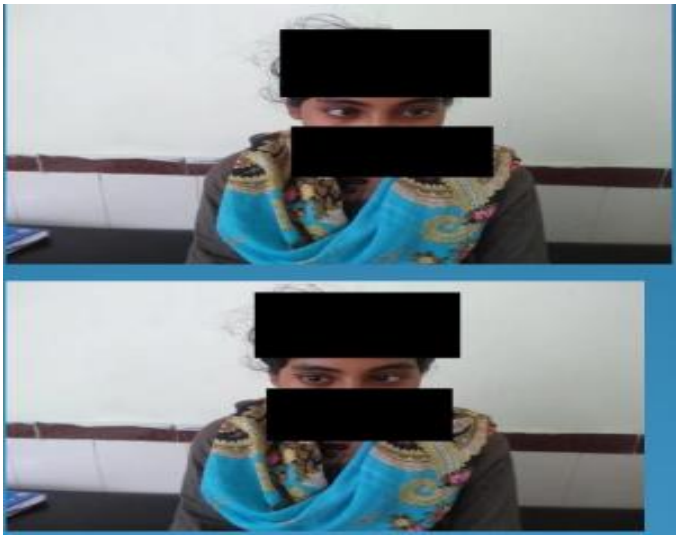

FIG 1: Clinical presentation

DISCUSSION

The occurrence of cranial nerve involvement in tuberculous meningitis (TBM) is well recognized, affecting nearly 30–50% of patients in reported series [6]. Among these, the sixth cranial nerve (abducens nerve) is particularly susceptible due to its long intracranial course and angulation at the petrous apex, making it vulnerable to compression by basal exudates and raised intracranial pressure (ICP) [7]. The bilateral presentation of sixth nerve palsy, however, is distinctly rare and typically indicative of extensive inflammation or hydrocephalus, both of which were evident in our cohort.

In our study, all patients presented with bilateral lateral rectus palsy, and 83.3% had MRI evidence of dense basal exudates. This aligns with the findings of Jinkins et al., who demonstrated that basal meningeal enhancement on MRI corresponds closely with cranial neuropathy in TBM [8]. The sixth nerve exits at the pontomedullary junction and travels upward along the clivus—an area often involved in basal meningitis—further explaining its vulnerability [9].

Dastur et al. reported that among 18 confirmed TBM cases, bilateral sixth nerve involvement was seen in only 11%, usually in association with raised CSF pressure and thick basal exudates [10]. Similarly, our study identified elevated CSF protein and reduced glucose in all patients, consistent with the inflammatory CSF profile described in classical TBM [6]. The correlation between bilateral sixth nerve palsy and high CSF protein (>100 mg/dL) further supports the hypothesis of pressure-related and exudative cranial nerve entrapment.

Heemskerk et al. emphasized that the addition of corticosteroids to ATT significantly reduces mortality and neurologic sequelae in TBM, including cranial neuropathies [11]. In our series, 10 out of 12 patients (83.3%) showed full or partial resolution of sixth nerve palsy with standard anti-tubercular therapy combined with corticosteroids. This is consistent with Misra et al.'s prospective study, where the resolution of cranial nerve deficits, particularly the sixth nerve, reached up to 85% after combination treatment [12].

Idiopathic intracranial hypertension (IIH) is a major differential diagnosis when bilateral sixth nerve palsy is encountered. However, IIH typically lacks the CSF pleocytosis and low glucose seen in TBM, and MRI often does not show basal exudates or infarcts [13]. In our cohort, the diagnostic distinction was clearly established by CSF analysis and MRI in all cases.

Visual outcomes are also of concern in TBM. Though optic neuropathy was not seen in our cohort,

papilledema was evident in 83.3% of patients. According to Donald [14], prolonged papilledema and raised ICP can lead to optic atrophy if not addressed promptly. The timely use of corticosteroids likely mitigated this complication in our series.

In cases refractory to systemic treatment, intrathecal therapy has been explored. Rajeswari et al. demonstrated the efficacy of intrathecal streptomycin in patients with persistent basal exudates and cranial nerve involvement [15]. Though we did not employ intrathecal regimens, the favorable outcome in the majority suggests early systemic intervention is often sufficient in reversing abducens palsy.

Our findings highlight that bilateral lateral rectus palsy, though uncommon, should prompt immediate neuroimaging and CSF evaluation in TB-endemic regions. It reflects a more aggressive basal meningeal pathology and may serve as an early clinical marker for potential complications such as hydrocephalus and infarction.

CONCLUSION

Bilateral lateral rectus palsy is a rare but significant neuro-ophthalmic manifestation of tubercular meningitis, indicating extensive basal meningeal inflammation and raised intracranial pressure. Early recognition through clinical examination and MRI findings, supported by CSF analysis, is crucial for prompt diagnosis. The combined use of anti-tubercular therapy and corticosteroids showed favorable neurological outcomes, with complete or partial recovery in most patients. Identifying such rare presentations can improve clinical vigilance and reduce long-term sequelae. Future studies should focus on neuroimaging correlations and long-term visual outcomes to optimize management protocols for cranial nerve involvement in TBM.

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