Content available at: https://www.ipinnovative.com/open-access-journals

Indian Journal of Clinical and Experimental Ophthalmology

Journal homepage: www.ijceo.org

Case Report

A rare presentation of bilateral abducens nerve palsy with papilledema in tuberculous meningitis

Deekshanya J[1]*, Kale Satya Geethika[1], Hannah Ranjee Prasanth[1], Ramesh Kannan S[2]

[1]Dept. of Ophthalmology, Pondicherry Institute of Medical Sciences, Puducherry, India
[2]Dept. of Neurology, Pondicherry Institute of Medical Sciences, Puducherry, India

Abstract

We present a classical case of a 30-year-old female who presented with bilateral abducens nerve palsy and papilledema. Her initial presentation was a holocranial headache for the past 20 days and restricted eye movements in both eyes for the past 10 days. Her best-corrected visual acuity was 6/9 in both eyes. The Hirschberg test showed 15 degrees esotropia in the left eye. Dilated fundus examination revealed a hyperemic disc with blurred margins and dilated, tortuous vessels in both eyes. Visual evoked potentials suggest demyelinating axonal optic neuropathy involving both optic nerves. MRI was otherwise normal and showed no cerebral haemorrhage or cerebral venous thrombosis. A lumbar puncture was done. The cerebrospinal fluid opening pressure was less than 5cm of water. Cerebrospinal fluid analysis showed colourless, clear fluid with RBC-5/ul, WBC-247/ul, polymorph was 8%, lymphocytes were 92% with degenerated cells in the background. She was diagnosed with tuberculous meningitis.

Abducens nerve palsy is a common manifestation of intracranial conditions, especially in meningitis or idiopathic intracranial hypertension. Optic neuropathy in tuberculosis is mostly a result of chronic papilledema due to meningitis.

Tuberculous meningitis is known to cause papilledema and bilateral abducens nerve palsy, which can be worrying for the patient. Tuberculosis must be considered in such patients, as prevention is better than cure. Although there are many national programmes to identify and treat tuberculosis early, young people are afflicted by such debilitating forms of extrapulmonary tuberculosis. This public health challenge must be tackled as a national priority.

Keywords: Abducens nerve palsy, Lateral rectus palsy, Papilledema, Tuberculous meningitis, Optic neuropathy.

Received: 17-04-2024; **Accepted:** 24-10-2024; **Available Online**: 09-06-2025

1. Introduction

Abducens nerve palsy also known as sixth cranial nerve palsy is one of the most common cranial nerve palsies to occur in isolation. It innervates the ipsilateral lateral rectus (LR) which abducts the eye. Lateral rectus palsy can be due to lesions along the course of the nerve.

The sixth cranial nerve has the second longest intracranial course after the trochlear nerve. The abducens nerve originates from its nucleus in the ventral aspect of the pons near the seventh cranial nerve. It enters the subarachnoid space, and courses upwards between the pons and clivus to enter the Dorello's canal. It has a long journey from its origin before the nerve reaches the petrous apex of the temporal bone in the basal skull and enters the cavernous sinus.[1] Here it is susceptible to injury when intracranial tension is high. It enters the orbit via the superior orbital fissure and innervates the lateral rectus muscle.

The case report aims to acknowledge the varied clinical presentation of abducens nerve palsy which may have underlying intracranial disorders like raised intracranial pressure, meningitis, haemorrhage, giant cell arteritis, brain tumour or any granulomatous conditions.

2. Case Presentation

A 30-year-old female with subnormal intelligence was brought to the Ophthalmology OPD with complaints of holocranial headache for 20 days. She complained of restricted eye movements in the left eye greater than the right

*Corresponding author: Deekshanya J
Email: jdeekshanya14@gmail.com

https://doi.org/10.18231/j.ijceo.2025.064

eye for the past 10 days. No history of vomiting, trauma, fever, diplopia, limb weakness, slurring of speech, or bowel and bladder disturbances. There was no history of instability in walking, difficulty in breathing, swallowing, loss of consciousness, or seizures. She had been on treatment for hallucinatory behaviour for the past 2 years on T. Risperidone 3mg, T. Trihexiphenidyl 2mg, T.Clobetazole 1mg, T.Olanzepine 5 mg.

On general examination, the patient was conscious and oriented and vitals were stable. Central nervous system examination was normal and power in all limbs was 5/5. Deep tendon reflexes were normal. A provisional diagnosis was made as Idiopathic intracranial hypertension (IIH). The results of MRA/MRV brain were suggestive of papilledema. There was no cerebral oedema, haemorrhage, hydrocephalus or neuroparenchymal abnormality. To rule out idiopathic intracranial hypertension, a lumbar puncture was performed. The cerebrospinal fluid opening pressure was less than 5cm of water. Histopathological examination showed predominantly lymphocytes and empirical anti-tuberculosis therapy was started.

C-ANCA and other autoimmune tests were done and were reported negative which ruled out vasculitis in connective tissue disease patients and other conditions like pseudotumor cerebri.

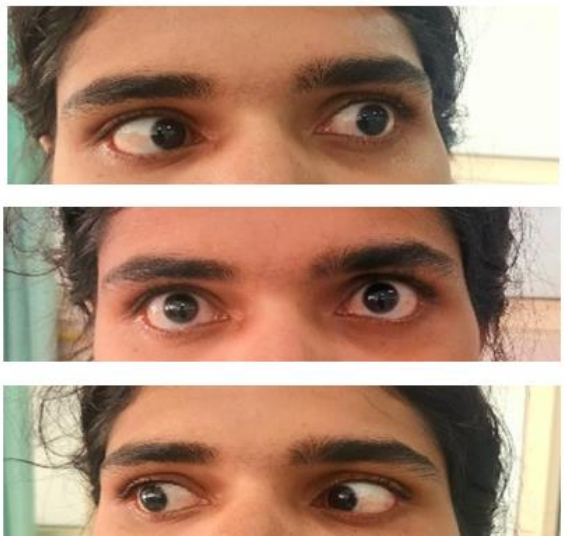

Figure 1: Resolving lateral rectus palsy

Ocular examination revealed best corrected visual acuity of both eyes 6/9, direct and consensual reflexes were normal. Intraocular pressure and colour vision were normal. On the Hirschberg test, 15 degrees of esotropia was noted in the left eye. Extraocular movements were tested, and abduction was restricted in the left eye (**Figure 1**). The anterior segment was normal. Dilated fundus in both eyes showed tortuous vessels and established papilledema. There were no choroidal tubercles (**Figure 2**). OCT showed a normal macula (**Figure 3**).

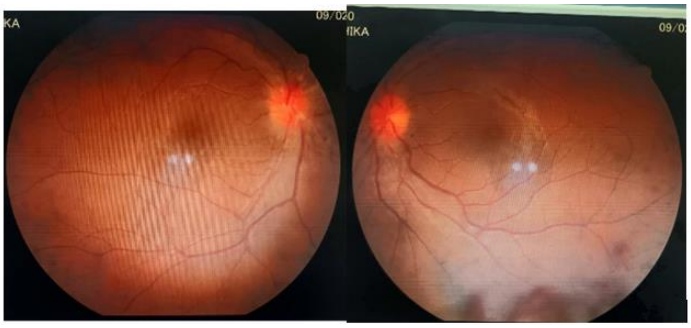

Figure 2: Fundus photography of both eyes

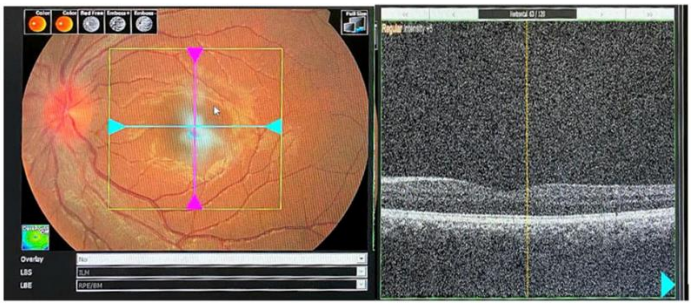

Figure 3: Optical coherence tomography was normal

Visual evoked potentials (**Figure 4**) suggest bilateral demyelinating axonal optic neuropathy involving both optic nerves. On admission the patient had bilateral papilledema and abducens nerve palsy, after empirical treatment with anti-tuberculous drugs, the bilateral lateral rectus palsy and papilledema showed resolution, and the patient improved symptomatically.

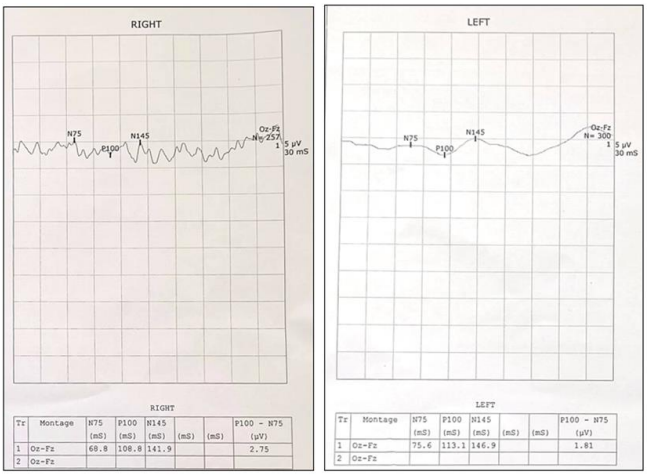

Figure 4: Visually evoked potentials show bilateral demyelinating axonal neuropathy

3. Discussion

Clinical presentation of the patient with abducens palsy usually raises suspicion of an underlying neurological disorder like raised intracranial pressure, meningitis, brain tumour or granulomatous conditions. Tuberculosis (TB) is one of the major global causes of morbidity and mortality and the majority of these deaths are in developing countries.

If the nerve is damaged bilaterally, this usually would present with persistent diplopia, large-angle esotropia, and severe restrictions of abduction. However, our patient did not have diplopia because it was more prominent only in distance and lateral gaze.

In a case reported by Zuhaimy H,[2] a patient presented in the early stage of TB meningitis with visual impairment, papilledema and abducens nerve palsy without classical signs of meningitis such as headache, fever, vomiting and neck rigidity.

Rival G et al.[3] presented a case of a 29-year-old male who came to the emergency with diplopia, headache, strabismus, and recent onset seizure, after an eye examination he was found to have bilateral abducens nerve palsy. A lumbar puncture revealed cryptococcal meningitis.

In a study by Crum OM et al.[4] idiopathic intracranial hypertension was found in patients who presented with papilledema with an unknown cause.

In a study by Verma R et al.[5] in patients with tuberculous meningitis a wide spectrum of ophthalmological manifestations was present. The visual impairment was more evident in complicated tuberculous meningitis. Ocular findings like optic atrophy, papilledema and RNFL thinning were associated with poor visual outcomes on univariate but not multivariate analysis.

In a study by Caire Estévez JP et al.[6] a 29-year-old woman with headaches for several days, loss of visual acuity and pain in her left eye. She had a 3-year history of type 1 diabetes mellitus. Fundus examination revealed papilledema. She showed a marked improvement after treatment with anti-TB drugs.

Secondary optic atrophy results from untreated optic neuropathy in TB, which is mostly caused by prolonged papilledema from meningitis. One of the most prevalent infectious diseases in developing countries is tuberculosis. Even though it's primarily a lung infection, extrapulmonary tuberculosis has been on the rise in recent years. Around 1% of cases involve the central nervous system which often results in significant complications. TB foci trigger an inflammatory reaction in a susceptible host. TB bacilli cause neuro-tuberculosis by three mechanisms, vasculitis, arachnoiditis, and obstructive hydrocephalus.[7] Sporadic tuberculous foci are established in the brain as a result of the bacilli's hematogenous spread that occurs after the primary infection or late reactivation.[8]

4. Conclusion

Abducens nerve palsy is a common manifestation of intracranial conditions, especially in meningitis or idiopathic intracranial hypertension. Optic neuropathy in tuberculosis is mostly a result of chronic papilledema due to meningitis.

Tuberculous meningitis is known to cause papilledema and bilateral abducens nerve palsy, which can be worrying for the patient. Tuberculosis must be considered in such patients, as prevention is better than cure. Although there are many national programmes to identify and treat tuberculosis early, young people are afflicted by such debilitating forms of extrapulmonary tuberculosis. This public health challenge must be tackled as a national priority.

5. Source of Funding

None.

6. Conflict of Interest

None.

7. Acknowledgement

I thank my guides and the Department of Ophthalmology and Department of Neurology, Pondicherry Institute of Medical Sciences, for their constant support, and encouragement.

References

1. Graham C, Gurnani B, Mohseni M. Abducens nerve palsy. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [updated 2023 Aug 24]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482177.
2. Zuhaimy H, Leow SN, Vasudevan SK. Optic disc swelling in a patient with tuberculous meningitis: a diagnostic challenge. *BMJ Case Rep*. 20179;2017:bcr2017221170.
3. Rival G, Okorji O, Kern R, Patel P, Fradeneck K, Cullen D. Bilateral Cranial Nerve VI Palsies in Cryptococcal Meningitis, HIV, and Syphilis: A Case Report. *Clin Pract Cases Emerg Med*. 2021;5(4):515–8.
4. Crum OM, Kilgore KP, Sharma R, Lee MS, Spiegel MR, McClelland CM, et al. Etiology of Papilledema in Patients in the Eye Clinic Setting. *JAMA Netw Open.* 2020;3(6):e206625.
5. Verma R, Sarkar S, Garg RK, Malhotra HS, Sharma PK, Saxena S. Ophthalmological manifestation in patients of tuberculous meningitis. *QJM*. 2019;112(6):409–19.
6. Caire Estévez JP, González-Ocampo Dorta S, Sanz Solana P. [Papilledema secondary to tuberculous meningitis in a patient with type 1 diabetes mellitus]. *Arch Soc Esp Oftalmol.* 2013;88(10):403–6.
7. Narayanan S, Prakash D, Subramaniam G. Bilateral primary optic neuropathy as the presenting manifestation of tuberculosis in an immunocompetent patient. *IDCases.* 2019;18:e00579.
8. Gupta M, Tobin EH, Munakomi S. CNS tuberculosis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [updated 2024 May 6]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK585138/

Cite this article: Deekshanya J, Kale Satya Geethika, Hannah Ranjee Prasanth, Ramesh Kannan S. A rare presentation of bilateral abducens nerve palsy with papilledema in tuberculous meningitis. *Indian J Clin Exp Ophthalmol*. 2025;11(2):362–364.