Clinical Challenge

Looking Beyond the Usual

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Abstract

Vision loss is a common association in tuberculosis. It may be due to direct damage to optic nerves and chaisma by the basal exudates and inflammation, tuberculomas affecting the visual pathway, optochiasmatic arachnoiditis, or papilloedema due to raised intracranial pressure. It may also be the result of toxic effects of anti-tubercular drugs like ethambutol or paradoxical response to ATT leading to expansion or development of new tuberculomas. The diagnosis is often challenging. In this case 13 year old child presented with vision loss in the left eye 2 months after starting the therapy. There was bilateral disc pallor that pointed towards ethambutol toxicity. But perimetry revealed bitemporal hemianopia and a subsequent neuroimaging showed optochiasmatic tuberculoma. Therefore, a proper neuro-ophthalmological work up supplemented with radiological examination can help arrive at a correct diagnosis and guide the appropriate management.

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Case Report

A 13-year-old female presented with complaint of subacute progressive diminution of vision in the left eye for 2 months. She was a diagnosed case of tubercular meningitis and was on anti-tubercular therapy/ Isoniazid (5mg/kg), Rifampicin (10mg/kg), Pyrazinamide (25mg/kg), Ethambutol (15mg/kg), (ATT /HRZE) for last 4 months. Neuroimaging films were not available with the patient. On examination, visual acuity in the right eye (OD) was 6/6 and left eye (OS) was 5/60. Relative afferent pupillary defect (RAPD) grade 4 was present in left eye and pupillary reaction was sluggish in right eye. Slit-lamp examination revealed normal anterior chamber. Fundus examination showed asymmetric temporal disc pallor OS>OD (Figure 1). We made a provisional diagnosis of toxic optic neuropathy. Ethambutol was stopped.

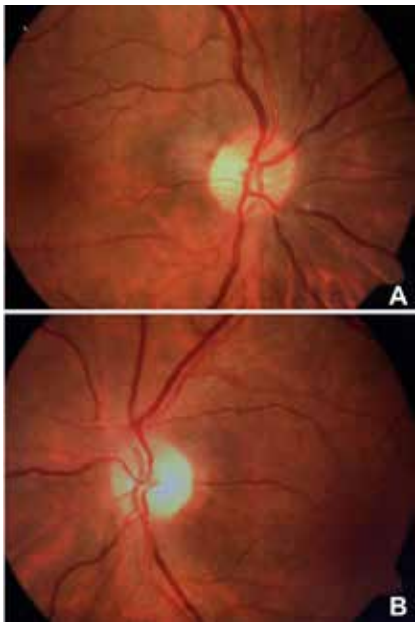

Figure 1: *Fundus picture of the patient showing (A) mild temporal pallor in the right eye (OD) and (B) significant temporal pallor in the left eye (OS).*

How should we proceed with further work up?

Dr Satya Karna: Asymmetric disc pallor with unilateral visual loss in a teenage girl raises the possibility of demyelinating, compressive, infective, ischemic, toxic, hereditary and nutritional optic neuropathy.

With a history of tubercular meningitis, assuming that the etiology of meningitis has been confirmed by laboratory tests, the possible causes of optic neuropathy are reduced to tubercular involvement of the visual pathway (optic chiasm and/or optic nerves), ischemia of the visual pathway by invasion of vasculature or toxic optic neuropathy (related to ethambutol or isoniazid).

Considering that the child had been suffering from tubercular meningitis and was also on antitubercular treatment (including ethambutol and isoniazid) for four months and the visual loss started 2 months on treatment, ischemic, infective and toxic, all three causes are possible. The asymmetry of visual loss suggests that toxic optic neuropathy is less likely as toxic levels in the body should affect both optic nerves equally or slightly asymmetrically.

A sudden loss of vision in the left eye would suggest ischemia as the cause and would give rise to a poorer prognosis. On the other hand a gradual progressive unilateral loss of vision would suggest compressive infective lesion which can theoretically have a better prognosis, if treated aggressively.

As is mandatory for all optic neuropathies, perimetry was done and revealed a bitemporal hemianopia. This is a very significant finding as it helps localise the lesion to the optic chiasm. Though it is rarely possible to have bitemporal defects in toxic optic chiasmal damage, hemianopia is not in favour of toxicity as it suggests complete involvement of crossing nasal fibres. Toxic medications affect the central fibres leading to loss of central vision in both eyes and the patient is unable to read or identify faces but is able to walk around and find his/her way.

Hence, the visual field leaves us with two possibilities, infective compressive and ischemic. Hence, neuroimaging of the brain is a must. The axial MRI brain shows multiple

ring-enhancing lesions, one of which is suprasellar and involving the optic chiasm. This tuberculoma is compressing the crossing nasal fibres leading to bitemporal hemianopia.

Case continued: Colour vision test (Ishihara) revealed colour perception defect bilaterally OS (3/14) >OD (5/14). Goldmann visual field (GVF) showed bitemporal hemianopia (Figure 2). In view of specific field changes, contrast enhanced magnetic resonance imaging (CEMRI) of brain and orbit was advised. Neuroimaging revealed a ring-enhancing lesion at the suprasellar cistern involving the optic chiasma and other multiple conglomerate ring-enhancing lesions in the left anterior temporal lobe (Figure 3). A diagnosis of Optochiasmatic tuberculoma was made and the patient was referred to the Neurology department for further management. At 3months follow-up, her vision improved to 6/36 in the left eye.

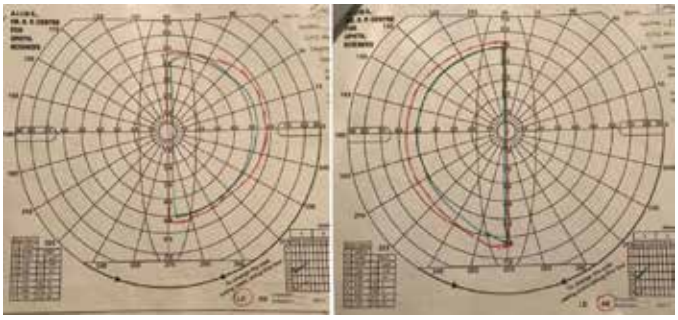

Figure 2: *Goldmann visual field of the patient with target III4e and V4e, showing bi-temporal hemianopia.*

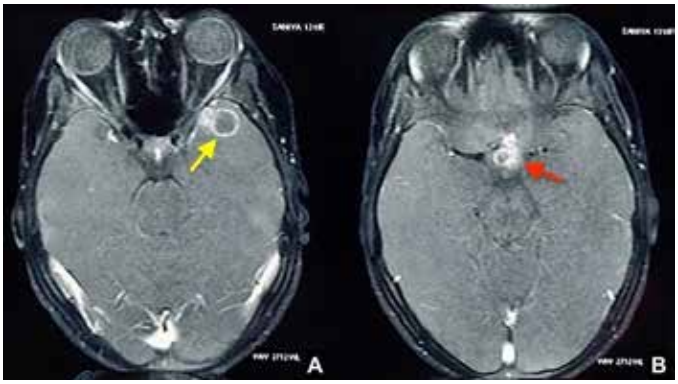

Figure 3: *Post-Gadolinium T1 weighted axial MRI images show (A) conglomerate ring-enhancing lesions in the left anterior temporal lobe (yellow arrow). The intraorbital and intracanalicular segments of optic nerve appear normal. (B) Similar ring-enhancing lesions noted in the suprasellar cistern involving the optic chiasma (red arrow).*

What should be the further course of management?

Dr Satya Karna: It is prudent to stop Ethambutol as it has been given for 4 months already and compromised optic nerves will not be able to withstand the toxic effects of the drug, which might lead to further vision loss. The antitubercular drugs need to be modified by the neurologist to control the intracranial lesions better. Also, a course of intravenous methylprednisolone followed by oral steroids to prevent further damage to the chiasmal fibres would help. The presence of temporal disc pallor in both eyes implies that the lesion has been compressing the optic chiasm for a period more than 4 weeks. This implies that the visual acuity and visual field recovery with appropriate treatment may not be excellent. As noted, after appropriate treatment by the neurologist, the vision in the left eye had marginally improved to 6/36. Perimetry test would show the improvement in visual fields. Also, the patient needs repeat neuroimaging to confirm resolution of the intracranial tuberculomas. Also, the patient has to complete the entire antitubercular regimen.

This case illustrated the extremely important role of color vision, visual acuity and visual field testing in a patient with tubercular meningitis to detect and treat any early optic neuropathy.

Section editor's comment (Rebika Dhiman): Ethambutol toxicity is the most common cause of vision loss after ATT. It mainly depends upon the dose and duration of the treatment. It usually occurs between 4 to 12 months of starting treatment1. Bilateral involvement is commonly seen; however, onset may be unilateral. Dyschromatopsia can be the first symptom in few patients2. Therefore, colour vision testing should be done for all patients. In our case, in view of bilateral disc pallor and colour vision defect we made a provisional diagnosis of ethambutol toxic optic neuropathy. Static or kinetic visual field evaluation is important to unravel underlying visual field defect. Bitemporal hemianopia in the current case, led to the suspicion of chiasmal involvement and a subsequent CEMRI revealed an optochiasmatic tuberculoma. The incidence of CNS tuberculoma has been reported as high as 20% in our country3. Although rare, tuberculomas can involve visual pathways directly- either optic nerve or optic chiasma4. Development of optochiasmatic tuberculoma or the expansion of pre-existing ones are known to occur as a paradoxical response in patients undergoing ATT therapy for central nervous system or pulmonary tuberculosis. This is a rare delayed type of hypersensitivity response seen against the mycobacterial proteins released by the dying bacteria.5 Due to unavailability of previous neuroimaging film in this case, it was difficult to comment whether the optochiasmatic tuberculoma was pre-existing or developed following ATT therapy. But as vision loss started after the initiation of anti-tubercular drugs, this goes more in favor of development of a paradoxical response.

Therefore, we must keep in mind an alternate diagnosis other than toxic optic neuropathy in TBM especially in cases presenting with asymmetrical eye involvement and evaluate the patient accordingly. A simple test like perimetry can suggest alternate pathology and neuroimaging helps in arriving at the diagnosis.

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