Optic perineuritis secondary to tuberculosis: A rare case presentation

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1. Introduction

Tuberculosis (TB) is a disease, most often involving the lungs and caused by infection with Mycobacterium tuberculosis (M. tuberculosis). Extrapulmonary involvement, including lesions of the gastrointestinal tract, genitourinary tract, cardiovascular system, skin, central nervous system, and eyes, may occur in association with clinically apparent pulmonary TB or in isolation, with no clinical or laboratory evidence of pulmonary infection. Ocular involvement is an uncommon extrapulmonary manifestation of TB (1%-2%)[1].

Optic perineuritis (OPN) also termed periopitic neuritis, is an uncommon inflammatory disorder involving the optic nerve sheath[2] that occurs occasionally as a manifestation of TB[3]. We described a patient with optic perineuritis as a rare initial presentation of TB and discussed the diagnosis and management.

2. Case report

A 58-year-old Malay lady, presented with gradual loss of vision in the right eye for 1 month duration. It was associated with right sided headache, nausea and vomiting. Patient had a history of contact with tuberculosis patient. On examination, visual acuity in right eye was no perception of light with presence of relative afferent pupillary defect. The left eye visual acuity was 6.0/7.5. There was a total restriction of extraocular movement in all direction (frozen eye) in the right eye. Both anterior segments were unremarkable. Fundoscopy in both eyes showed normal appearance of optic disc and retinal vessels. Other cranial nerves were normal. Lungs were clear and had no lymphadenopathy. Chest radiograph was normal. Erythrocyte sedimentation rate was elevated by 35 mm/h and Mantoux test was positive (20 mm). MRI of brain and orbit demonstrated thickening and irregularity of the right optic nerve on axial view with doughnut sign on coronal view. Patient was diagnosed to have optic perineuritis secondary to tuberculosis. She was treated with anti-TB drugs. Systemic corticosteroid was commenced after 2 weeks. The visual acuity was improved to hand motion after 1 month with almost complete resolution of extraocular movement.
and heterogenously enhanced. There was thickening of the right optic nerve on axial view, with presence of "doughnut sign" on coronal view suggestive of OPN (Figure 3A and 3B). Erythrocyte sedimentation rate was elevated by 35 mm/h and Mantoux test was positive (20 mm). Electrocardiography (ECG) and chest radiograph was normal.

Patient was diagnosed to have OPN secondary to TB. She was treated with anti-TB drugs, T. isoniazid 300 mg OD, T. ethambutol 1 200 mg OD, T. rifampicin 600 mg OD, T. pyrazinamide 1 500 mg OD and T. pyridoxine 10 mg OD. Systemic corticosteroid (T. prednisolon 50 mg OD) was commenced after 2 weeks after anti-TB treatment. The right extraocular movements were markedly improved after 10 days. The steroid was tapered down after 2 weeks. The visual acuity was improved to HM (hand motion) after 1 month with almost complete resolution of extraocular movement (EOM). Fundoscopy showed pale disc in right eye (Figure 4). During follow up after 6 months, the visual acuity was improved to 6/60. There was complete resolution of EOM. Patient was on
anti-TB treatment for one year. There was no history of recurrent episode even after 1 year follow up.

Figure 4 A: Fundoscopy showed pale disc (after 6 weeks) in right eye. B: The left disc is normal.

3. Discussion

TB is an infectious disease responsible for significant morbidity and mortality worldwide[4]. Although ocular involvement is an uncommon extrapulmonary manifestation of infection, it is important to recognise it because a 12% incidence has been reported. *M. tuberculosis* (66.6%) is in cases of granulomatous panuveitis, followed by multifocal choroiditis, granulomatous iridocyclitis, and lastly vasculitis (33.3%). Optic neuritis caused by TB does not occur very frequently and OPN occurs occasionally as a manifestation of TB[3].

Definitive diagnosis of ocular TB is with finding the *M. tuberculosis* in ocular fluid or tissue, but this is difficult to confirm because it is usually achieved by enucleation of the eye. Diagnosis of ocular TB is difficult also due to its widely variable clinical presentation and the lack of uniformity in diagnostic criteria. In most cases, diagnosis requires both corroborative evidence, such as a positive PPD (purified protein derivative) and chest X-ray, and the exclusion of other causes[1]. In OPN, inflammation involving the optic nerve sheath is evidenced by the characteristic pattern of enhancement around the optic nerve (“tramtrack” on axial views and “doughnut” on coronal views) in MRI scan[2,6]. Furthermore, the absence of clinically evident pulmonary TB does not rule out the possibility of ocular TB, as approximately most patients with extrapulmonary TB have no evidence of pulmonary TB.

In this case, the diagnosis of ocular TB was presumptive based on ocular findings, positive history of contact with TB patient, and a positive Mantoux skin test. Researchers showed the combination of high-dose corticosteroids and anti-TB medication was rapidly effective and obtained a good outcome in cases with optic neuritis secondary to TB[5]. On the other hand, differentiation between OPN and retrobulbar optic neuritis is needed before starting any treatment. This is because of the different treatment and prognosis between these two[6], and the steroid treatment for OPN is necessary and longer than optic neuritis[1,6].

This case is unique as OPN is a rare association of TB and is also an interesting case due to association of orbital apex syndrome and OPN due to TB without any systemic evidence of TB that is also a rare presentation. Therefore, OPN should be considered in cases of atypical presentation of optic neuritis. TB is important to rule out the possible cause of OPN.

Conflict of interest statement

We declare that we have no conflict of interest.

References


