An unusual case of tuberculous optic neuropathy associated with choroiditis

Anindya K Majumder, Siddharth Sheth[1], Vidya Dharani[2], Parthopratim Dutta Majumder[3]

Tuberculous optic neuropathy that includes papillitis, neuroretinitis, and optic nerve tubercle is a rare presentation of ocular tuberculosis. Though contagious spread from choroid following the hematogenous dissemination of the bacilli has been implicated in the optic nerve involvement, unlike neuroretinitis, optic nerve and choroidal involvement are usually considered as two separate clinical entities. We report a case of optic nerve involvement in a 33-year-old male who had concurrent choroidal involvement at present and also in the past. A strong history of contact with patients of pulmonary tuberculosis, positive tuberculin skin test, interferon gamma release assay, and high-resolution computed tomography helped us to clinch the diagnosis. Patients responded to systemic corticosteroid therapy and anti-tuberculosis treatment. Choroidal involvement in a case of inflammatory optic neuropathy should arise suspicion of tuberculous etiology.

Key words: Choroiditis, neuroretinitis, ocular tuberculosis, tuberculous optic neuropathy

Ocular tuberculosis has a plethora of presentations. Posterior uveitis which comprises of retinitis, choroiditis, chorioretinitis, and neuroretinitis are the most common clinical manifestations of ocular tuberculosis.[1,2] Owing to the hematogenous spread of the bacilli, choroid remains the most commonly involved structure in ocular tuberculosis.[3] Optic nerve involvement in tuberculosis usually results from contagious spread from choroid or hematogenous spread from other remote sites.[3] Optic nerve involvement in tuberculosis is not very common, though reported in the literature.[4] Tuberculous optic neuropathy has a wide spectrum of presentations, and the majority of the cases are associated with posterior uveitis.[4] We report a patient with tuberculous optic neuropathy with multifocal choroiditis, who responded to anti-tuberculosis treatment (ATT) and systemic corticosteroid.

Access this article online

Quick Response Code:

Website: www.ijo.in

DOI: 10.4103/ijo.IJO_1707_18

Department of Uvea, [1]Paediatric Ophthalmology and Neurophtahlmology, Sankara Nethralaya, Kolkata, [2]Department of Neuophthalmology, [3]Department of Uvea Sankara Nethralaya, Chennai, Tamil Nadu, India

Correspondence to: Dr. Parthopratim Dutta Majumder, Department of Uvea, Sankara Nethralaya, 18, College Road, Nungambakkam, Chennai - 600 006, India. E-mail: drparthopratim@gmail.com

Manuscript received: 12.10.18; Revision accepted: 28.12.18

Case Report

A 33-year-old non-diabetic, normotensive male presented to us with a sudden onset, painless diminution of vision in his left eye. He denied any suggestive systemic history or recent onset of fever preceding his ocular symptoms. There was also no history of similar ocular complaints, such as redness, ocular pain or diminution of vision in the past. He had a history of contact with patients of pulmonary tuberculosis. His grandfather, grandmother, and uncle had pulmonary tuberculosis. On examination, best corrected visual acuity (BCVA) in the right eye was 6/6 and counting finger at a 1-metre distance in his left eye. Slit-lamp examination of both the eyes revealed quiet anterior chamber, clear lens, and cells in anterior vitreous of the left eye. Examination of pupil revealed a relative afferent defect in the left eye. The intraocular pressure and eye movements were normal. Colour vision with Ishihara chart was 21/21 in the right eye and 0/21 in the left eye. Fundus examination of the right eye showed multiple small areas of healed choroiditis sparing the macula [Fig. 1a]. Fundus examination of the left eye revealed an oedematous optic disc with large areas of peripapillary haemorrhages with surrounding subretinal fluid with hard exudates over macula [Fig. 2a]. Fundus fluorescein angiography (FFA) of the right eye delineated the healed areas of choroiditis with staining of the lesions [Fig. 1b]. FFA of the left eye showed hypofluorescence of choroidal involvement in early frame followed by the hyperfluorescence of the lesions and leakage of the dye from optic disc [Fig. 2b and c]. Magnetic resonance imaging (MRI) of the brain and orbit with contrast was essentially normal and revealed a small elevation at the left optic disc.

Extensive investigations were carried out to find out the underlying etiology and other causes of optic neuropathy. In these studies, neuroimaging and serologies for syphilis, toxoplasma, and human immunodeficiency viruses found negative. Serum

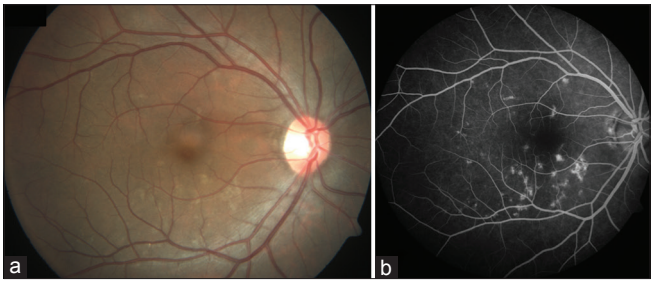

Figure 1: (a) Fundus photograph of the right eye showing multiple small areas of healed choroiditis sparing the macula. (b) Fundus fluorescein angiography of the right eye in late frame showing staining of the lesions which delineated the healed lesions more clearly than the clinical photograph

Cite this article as: Majumder AK, Sheth S, Dharani V, Dutta Majumder P. An unusual case of tuberculous optic neuropathy associated with choroiditis. Indian J Ophthalmol 2019;67:1210-2.

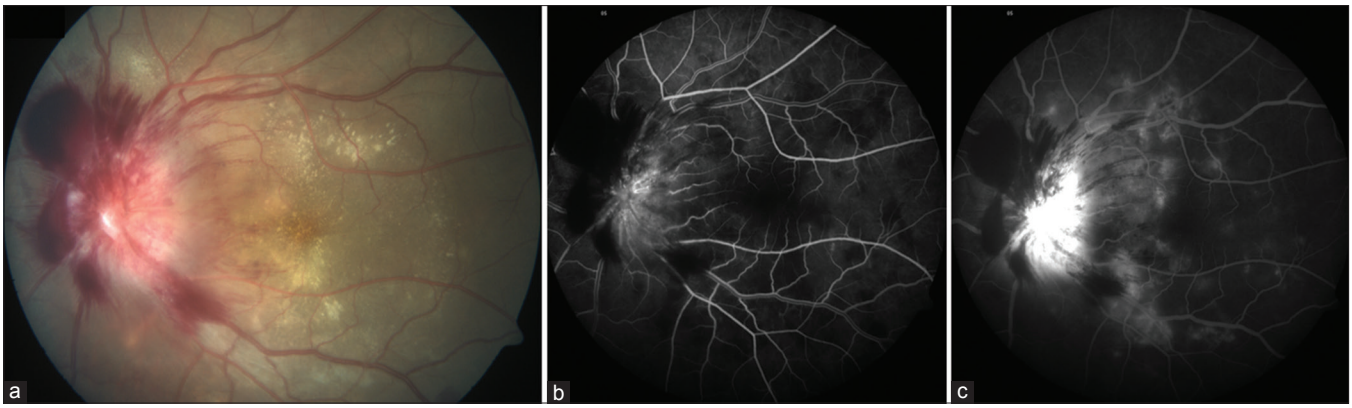

Figure 2: (a) Fundus photograph of the left eye showing an oedematous optic disc with large areas of peripapillary haemorrhages with surrounding subretinal fluid with hard exudates over macula. (b and c) Fundus fluorescein angiography of the left eye showing hypofluorescence of choroidal involvement in early frame followed by hyperfluorescence of the lesions and leakage of the dye from the optic disc

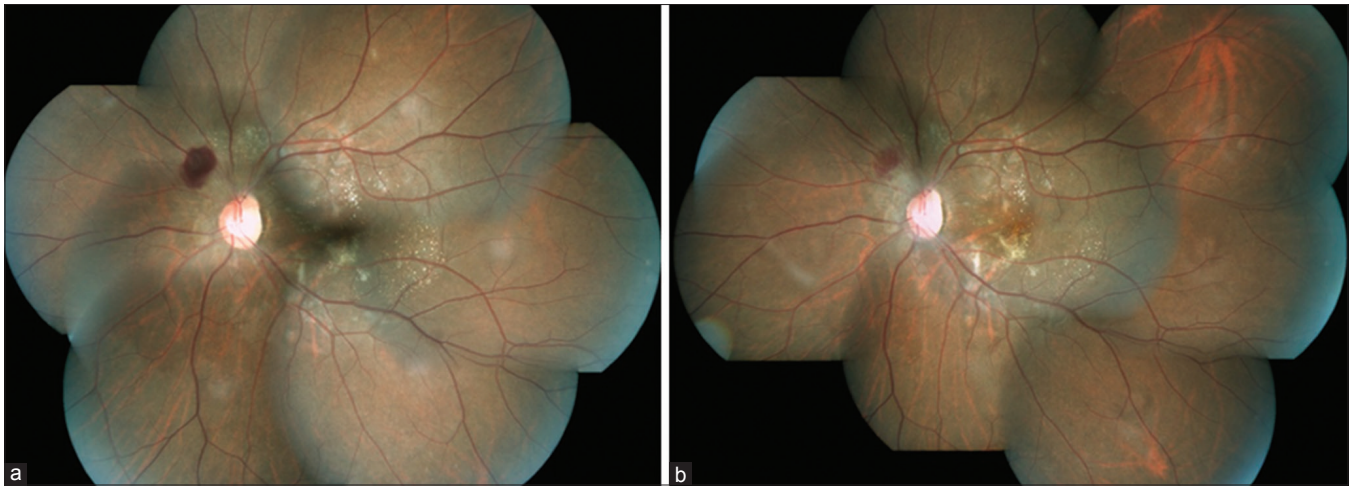

Figure 3: (a and b) Fundus photograph of the left eye at two-week and two-month follow-up

angiotensin-converting enzyme and serum lysozyme were within normal limit. Laboratory investigations revealed a positive tuberculin skin test (TST), a positive interferon gamma release assay (IGRA), and high-resolution computed tomography (HRCT) of the chest revealed multiple lymphadenopathy involving mediastinal, axillary lymph nodes with fibrotic strands in the lung parenchyma. Consultation from a pulmonologist was sought who started the patient on anti-tuberculosis treatment (ATT). The patient subsequently underwent pulse corticosteroid therapy with intravenous methylprednisolone (IVMP) for three days followed by tapering doses of oral prednisolone starting at 1.5 mg/Kg body weight/day. He was examined again after two weeks. He felt minimal visual improvement, and BCVA in his left eye was recorded as 3/60. Examination revealed quiet anterior chamber, and anterior vitreous but left relative afferent pupillary defect persisted. Fundus examination revealed marked resolution of optic nerve edema with resolving haemorrhage and subretinal fluid. He was advised to continue tapering oral steroid under ATT cover [Fig. 3a].

Two months after the initial presentation, the patient was seen again and BCVA had improved to 6/24. Examination of the anterior segment of the left eye was unremarkable and no relative afferent pupillary defect noted. Fundus examined revealed resolved optic disc edema and resorption of subretinal fluid with hard exudates and few retinal pigment epithelium alterations in macula [Fig. 3b]. Visual field of the left eye by 24–2 Humphrey automated perimetry showed nasal peripheral and superotemporal defects. Colour vision with Ishihara chart was 21/21 in the right eye and in the left eye it improved to 20/21. He was advised to continue ATT and oral steroid in low maintenance dose. The patient is under follow-up with us for the last eight months and till now he did not develop any recurrence.

Discussion

Inflammatory optic neuropathy is characterized by optic disc edema, surrounding edema of the retinal layers and subsequent formation of hard exudates in the outer plexiform layer, arrangement around the macula giving a star-like appearance.[5,6] Our patient had a strong history of contact with patients of pulmonary tuberculosis among family members, had evidence of healed multiple choroidal tubercle in another eye, subsequent investigations revealed positive Mantoux test, and IGRA and HRCT Chest suggesting possible pulmonary involvement. The patient subsequently responded to ATT and systemic steroid. Thus, even in the absence of definitive tissue diagnosis, the association of tubercular etiology was

straightforward in our case. In an analysis of clinical data of 63 eyes of 49 patients with tuberculous optic neuropathy, papillitis was the most common involvement followed by neuroretinitis and optic nerve tubercle.[4] Uveitis was a frequent association in these patients – posterior uveitis (62%) being the most common subtype followed by panuveitis (31%).[4] In our case, it was predominantly choroidal and optic nerve involvement and does not fit into the optic involvement described earlier.[4]

However, the exact mode of optic nerve involvement in ocular tuberculosis remains unclear. Ocular involvement in *Mycobacterium tuberculosis* infection can result from direct invasion of the bacilli through hematogenous route or believed to incite intraocular inflammation by a cross-reactive immune response to the bacilli. In the absence of tissue fluid analysis by polymerase chain reaction, we cannot speculate the possible mechanism of optic nerve involvement in our case. However, predominant choroidal involvement in the affected eye and another eye may be correlated as a possible contagious spread of bacilli from the choroid.

Conclusion

In summary, we report a case of presumed tuberculous optic neuropathy with concurrent choroiditis which presented following choroidal involvement in another eye. Our case highlights once again the possible contagious spread of the infection from choroid to optic nerve head and its successful management with systemic steroid and ATT.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Singh R, Gupta V, Gupta A. Pattern of uveitis in a referral eye clinic in north India. Indian J Ophthalmol 2004;52:121-5.
2. Jabs DA, Nussenblatt RB, Rosenbaum JT. Standardization of Uveitis Nomenclature (SUN) Working Group. Standardization of uveitis nomenclature for reporting clinical data. Results of the First International Workshop. Am J Ophthalmol 2005;140:509-16.
3. Gupta V, Gupta A, Rao NA. Intraocular tuberculosis--an update. Surv Ophthalmol 2007;52:561-87.
4. Davis EJ, Rathinam SR, Okada AA, Tow SL, Petrushkin H, Graham EM, *et al*. Clinical spectrum of tuberculous optic neuropathy. J Ophthalmic Inflamm Infect 2012;2:183-9.
5. Stewart MW, Brazis PW, Barrett KM, Eidelman BH, Mendez JC. Optical coherence tomography in a case of bilateral neuroretinitis. J Neuro-Ophthalmol Off J North Am Neuro-Ophthalmol Soc 2005;25:131-3.
6. Purvin V, Sundaram S, Kawasaki A. Neuroretinitis: Review of the literature and new observations. J Neuro-Ophthalmol Off J North Am Neuro-Ophthalmol Soc 2011;31:58-68.