A case of subretinal tubercular abscess presenting as disc edema

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We report a case of ocular tuberculosis (TB) which initially presented with disc edema and was mistaken for optic neuritis. With no definite pathology being identified, the patient was treated on the lines of optic neuritis with intravenous (IV) steroid with beneficial effect. Ocular TB was suspected when he presented later with a subretinal abscess. Based on positive Mantoux, QuantiFERON TB gold results and radiographic findings, a diagnosis of subretinal abscess of presumed tubercular etiology was made. The patient was successfully treated with anti-tubercular therapy. To the best of our knowledge, this is the first case report of ocular TB presenting as disc edema followed by subretinal abscess.

Key words: Anti-tubercular therapy, disc edema, ocular tuberculosis, subretinal tubercular abscess

Ocular tuberculosis (TB) is an extrapulmonary form of TB. It can have a variable presentation depending on the site and severity of infection. The absence of pulmonary TB does not rule out the diagnosis of ocular TB.[1]

Case Report

A 33-year-old South Indian male presented with complaints of pain in the left eye (LE) with painful eye movements since 25 days and gradual progressive worsening of vision in his LE since 20 days. There was no history of fever, chronic cough, evening rise of temperature, lymphadenopathy, contact with TB, trauma, headache, vomiting, tinnitus, vitiligo or joint pains. He had no history of hypertension, diabetes, TB and no other significant medical history. On examination, his best corrected visual acuity (BCVA) of right eye (RE) was 20/15, N6 and LE was 20/30, N6. Intraocular pressure was normal in both eyes (BE). LE showed grade II relative afferent pupillary defect (RAPD) with disc edema [Figs. 1 and 2]. Both anterior and posterior segment were quiet with no anterior chamber cells or vitritis. His blood pressure was normal. Routine baseline investigations including erythrocyte sedimentation rate (ESR) and peripheral smear were normal. Special investigations like anti-nuclear cytoplasmic antibodies (P-ANCA, C-ANCA), homocysteine, angiotensin converting enzyme levels were all normal. Rheumatoid factor and venereal disease research laboratory was negative. B scan of LE showed disc elevation with normal choroidal thickness. No subtenon fluid or any mass lesion was noted. Automated perimetry with Humphrey field analyzer II 30–2 test showed enlarged blind spot in LE. Typical inflammatory optic neuritis of the LE was suspected. After physician's clearance, IV methyl prednisolone (IVMP) 1 g/day was given for 3 days and then substituted with oral prednisolone 1 mg/kg body weight with weekly 10 mg tapering dosage.

At 1-month follow-up, patient was symptomatically better with BCVA 20/16 BE. Anterior segment was normal in BE. Fundus showed resolved disc edema in LE. Patient was advised to continue oral prednisolone 10 mg for 4 weeks and then stop medication and review. However, the patient was lost to follow up.

Six months later, he presented again with sudden blurring of vision LE. There was no history of pain, fever, headache or any other complaints locally or systemically. BCVA RE was 20/16 and LE 20/400. LE showed RAPD with disc edema and a subretinal mass [Figs. 3 and 4]. Mantoux test was positive (13 mm × 14 mm). Laboratory investigations showed normal blood counts with normal ESR (13 mm/h). HIV was negative while QuantiFERON (Cellestis Limited, Carnegie, Victoria, Australia) TB gold test was positive. High resolution computed tomography (HRCT) chest showed multiple discrete sub-centimeter, noncalcified, pretracheal and circumaortic lymph nodes suggestive of old healed TB. B scan LE showed exudative retinal detachment with subretinal fluid in the peripapillary area and posterior pole. Peripapillary choroidal thickness was 2 mm and an elevated lesion was noted over the optic nerve head with moderate to high surface reflectivity and moderate internal reflectivity. Sputum evaluation was negative. Based on HRCT report suggestive of healed TB, a positive QuantiFERON TB and Mantoux test, patient was diagnosed to have subretinal abscess with exudative retinal detachment of presumed tubercular etiology. Patient was started on a 9 months course of anti-tubercular therapy (ATT) with tapering course of oral steroids over 6 weeks under the care of a physician. He was advised to review in 1-week but the patient reviewed after 1-month (his last follow-up). He was symptomatically better with his BCVA improving to 20/160 in LE with a healed tubercular granuloma and subretinal fibrosis [Figs. 5 and 6]. As the patient had shown good response to ATT, our diagnosis was confirmed and hence ocular fluid tap for polymerase chain reaction (PCR) test was avoided. The patient was advised to continue ATT course till 9 months and review monthly. However, the patient has been lost to follow-up.

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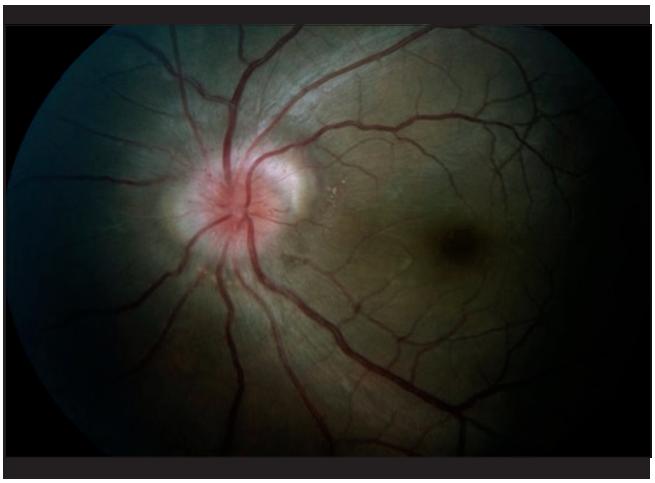

Figure 1: Colour photo of the patient at first presentation showing disc edema

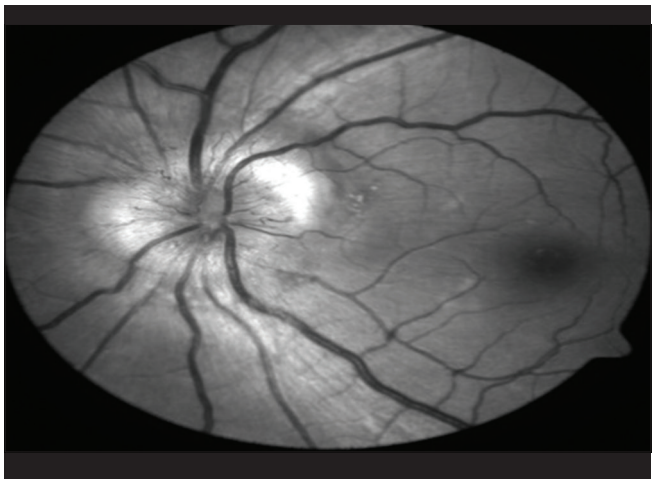

Figure 2: Autofluorescence picture of the patient at first presentation showing disc edema

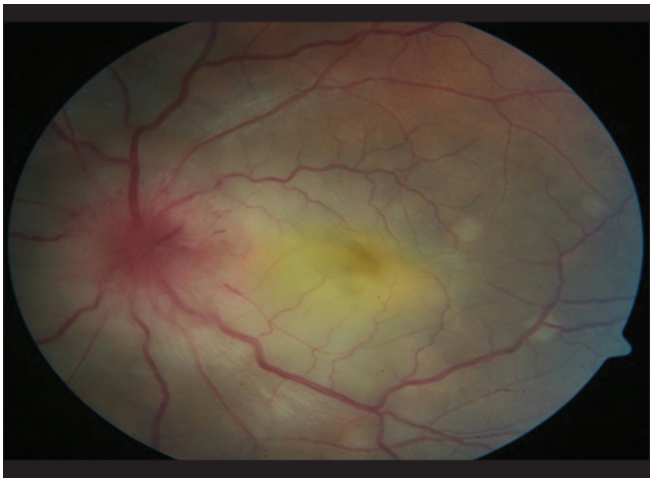

Figure 3: Colour photo of the patient at 7th month showing subretinal abscess and disc edema

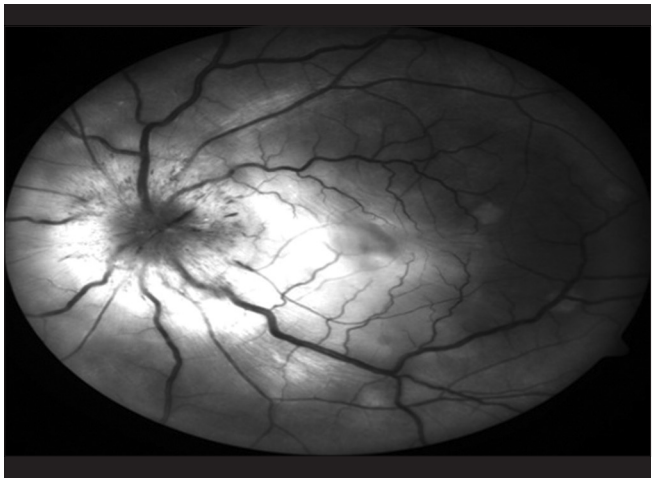

Figure 4: Autofluorescence picture of the patient at 7th month showing subretinal abscess and disc edema

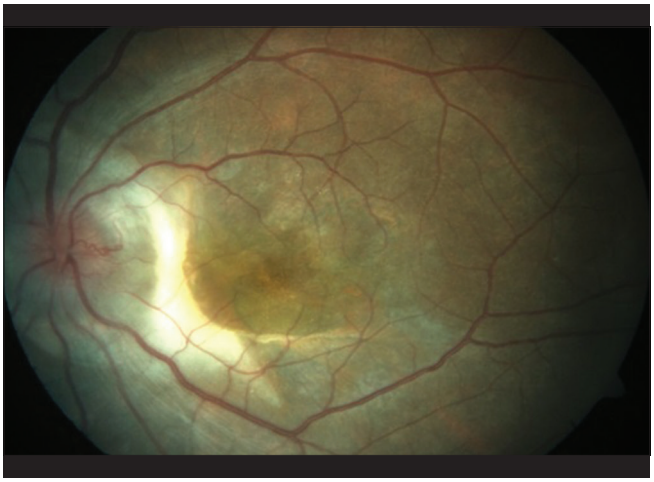

Figure 5: Colour photo at 8th month showing resolution of inflammation with subretinal fibrosis, one month after starting anti-tuberculosis therapy

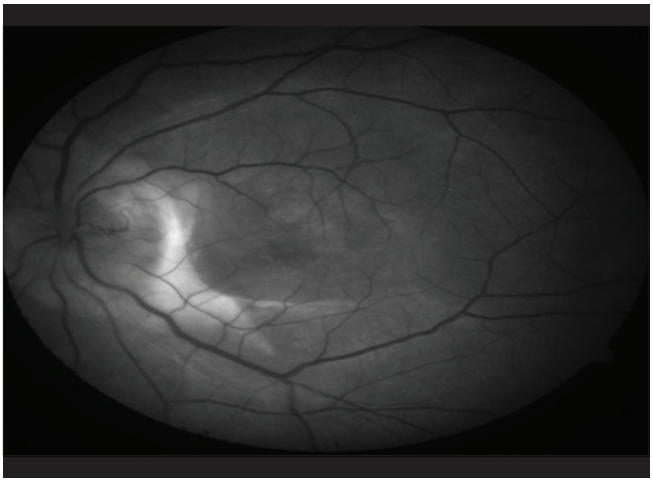

Figure 6: Autofluorescence picture at 8th month showing resolution of inflammation with subretinal fibrosis, 1-month after starting anti-tuberculosis therapy

Discussion

Ocular TB can result from hematogenous spread, direct local extension from respiratory tract, or it can be delayed hypersensitivity reaction. Choroid is the most commonly involved site with posterior uveitis being the most common presentation.[2,3] The main types of choroidal involvement include choroiditis, tubercles, tuberculomas and subretinal abscess.[3] Choroidal tubercle is the most recognized finding of ocular TB. Large tuberculomas may undergo liquefactive necrosis and form yellowish subretinal abscess with little vitreous inflammation.[4] It can be seen both in immunocompetent and immunocompromised patients. Such patients need to be investigated for miliary TB and HIV. Rarely, subretinal abscess can rupture into vitreous cavity leading to endophthalmitis or panophthalmitis. With timely ATT, they can be curtailed to form an atrophic scar. Subretinal neovascularization may develop within the scar leading to choroidal neovascularisation and choroidal hemorrhages.[2]

Microbiological (culture/acid-fast bacilli staining/PCR) evidence of mycobacterium TB from intraocular fluid or tissue constitutes the gold standard for diagnosing intraocular TB. Usually the diagnosis is presumptive as ocular specimens can rarely be obtained easily. The current criteria of making a presumptive diagnosis of intraocular TB is based on a combination of clinical features suggestive of ocular TB with corroborative evidences such as a positive Mantoux test, positive interferon-gamma release assay, radiographic findings, exclusion of known nontubercular uveitic entities, and a positive response to ATT.[2] PCR is both a sensitive and specific method for early detection.[2] B scan is useful in differentiating tuberculomas from tumors. It usually has a low to medium internal reflectivity on A scan.[3,5]

Subretinal tuberculomas and abscesses have been successfully managed surgically.[6] If diagnosed early, they are amenable to medical treatment.[2] The regimen consists of isoniazid, rifampicin, ethambutol, and pyrazinamide for 2 months and then isoniazid and rifampicin for 9–12 months. Early treatment with ATT not only takes care of the active infection but also decreases the risk of developing recurrences of uveitis.[1] A favourable response to ATT was evident within 4 weeks in our case. Low dose steroids are given concomitantly with ATT for 4–6 weeks since they have a protective effect against Jarisch–Herxheimer reaction and tissue damage from delayed hypersensitivity.[2,4]

Ocular TB, presenting as disc edema and followed by subretinal abscess has not been reported in literature to the best of our knowledge. It is possible that IV steroids could have caused a flare up of latent TB which manifested later. It is also possible that that initial disc edema and the later subretinal tubercular abscess were two separate disease processes. We can never reach a conclusion on this as Mantoux and QuantiFERON TB gold tests were not done initially. This case report shows that before starting IVMP, TB has to be ruled out at least by a Mantoux and/or QuantiFERON TB gold test. In cases where IVMP has to be administered and TB is strongly suspected, IVMP may be given under the cover of ATT. The clinician should always have a high index of suspicion for ocular TB. Since the disease is treatable, a timely diagnosis and treatment is of paramount importance to salvage the eye for a good outcome.

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