

A rare case of tuberculous choroidal abscess

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Abstract

We report a case of ocular tuberculosis which presented as a subretinal abscess along with an active choroiditis patch and disc edema but with subtle ocular symptoms. Patient had a strongly positive Tuberculin Skin Testing but negative Interferon Gamma Release Assay. Based on the diagnosis of presumed ocular tuberculosis the patient was started on Category 1 Anti tuberculous therapy with a tapering dose of oral steroids, to which the patient responded in 4 weeks with complete resolution of lesions. This is a rare manifestation of ocular tuberculosis with panoramic unioocular manifestations in an immunocompetent male which healed rapidly without any residual damage. This case is reported to emphasize the importance of early detection and management of ocular tuberculosis as it aids in lesser ocular morbidity and visual impairment.

Keywords: Ocular tuberculosis, Choroidal abscess, Tuberculoma, Choroiditis

Introduction

Choroidal tuberculosis is the most common manifestation of ocular tuberculosis [1]. The lack of uniform diagnostic criteria and the difficulty in obtaining a tissue specimen makes the diagnosis challenging in many cases [2]. Choroidal tuberculomas may be associated with latent tuberculosis which has to be ruled out before treatment with ATT [3].

The demonstration of acid fast bacilli in ocular tissues is the gold standard for diagnosis. PCR also is a specific investigation for diagnosis of ocular tuberculosis but a negative PCR does not negate the diagnosis. This is a rare manifestation of Choroidal abscess with disc edema in an immunocompetent male.

Case Report

A 34 year-old male came with the complaints of painless diminution of vision in the left eye associated with metamorphopsia of 3 weeks duration. There was no history of trauma, fever or any systemic illness. Patient's father had pulmonary tuberculosis and was on irregular treatment. On examination, patient was normally built and nourished. There was no evidence of pallor or lymphadenopathy.

Best corrected visual acuity of the right eye was 6/6 and the left eye was 6/12. The examination of right eye was within normal limits. Anterior segment examination of the left eye was normal. Fundus examination revealed a mildly hazy media due to grade 1 vitritis. Blurring of disc margins was noted. A yellowish lesion with blurred margins of 2 DD suggestive of a choroiditis patch was seen superotemporal to the disc with a flame shaped hemorrhage. Another elevated subretinal lesion of 4DD in size with lifting of vessels was seen in the superotemporal quadrant just above the macula (Figure 1).

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Minimal exudation was seen temporal to the disc and temporal to the macula. ILM folds with serous detachment were seen in macular area. Complete blood count and ESR were within normal limits. Chest x-ray was normal but Mantoux test was strongly positive with an induration of 24 x 12 mm. ELISA for HIV1 and 2 were negative. Interferon gamma release assay was negative.

TORCH screening showed positive Ig-G for Rubella, Cytomegalovirus and Herpes Simplex Virus. Fundus fluorescein angiography of left eye in early phase showed leakage of dye around the disc, temporal to the macula and hypofluorescence corresponding to the choroiditis patch (figure 2).

Late phase images revealed blocked fluorescence with hyperfluorescence corresponding to active choroiditis patch with hemorrhage. Temporal lesion showed hyperfluorescence increasing in intensity due to leakage. Hyperfluorescence increasing in intensity temporal to disc and macula suggestive of exudation was also seen (figure 3). The crossover images of the right eye were normal.

B scan of the left eye revealed an elevated lesion with sub retinal fluid and minimal exudative retinal detachment with choroidal thickening. Optical Coherence Tomography also revealed subretinal fluid. Based on these features a diagnosis of tuberculous choroidal abscess was made.

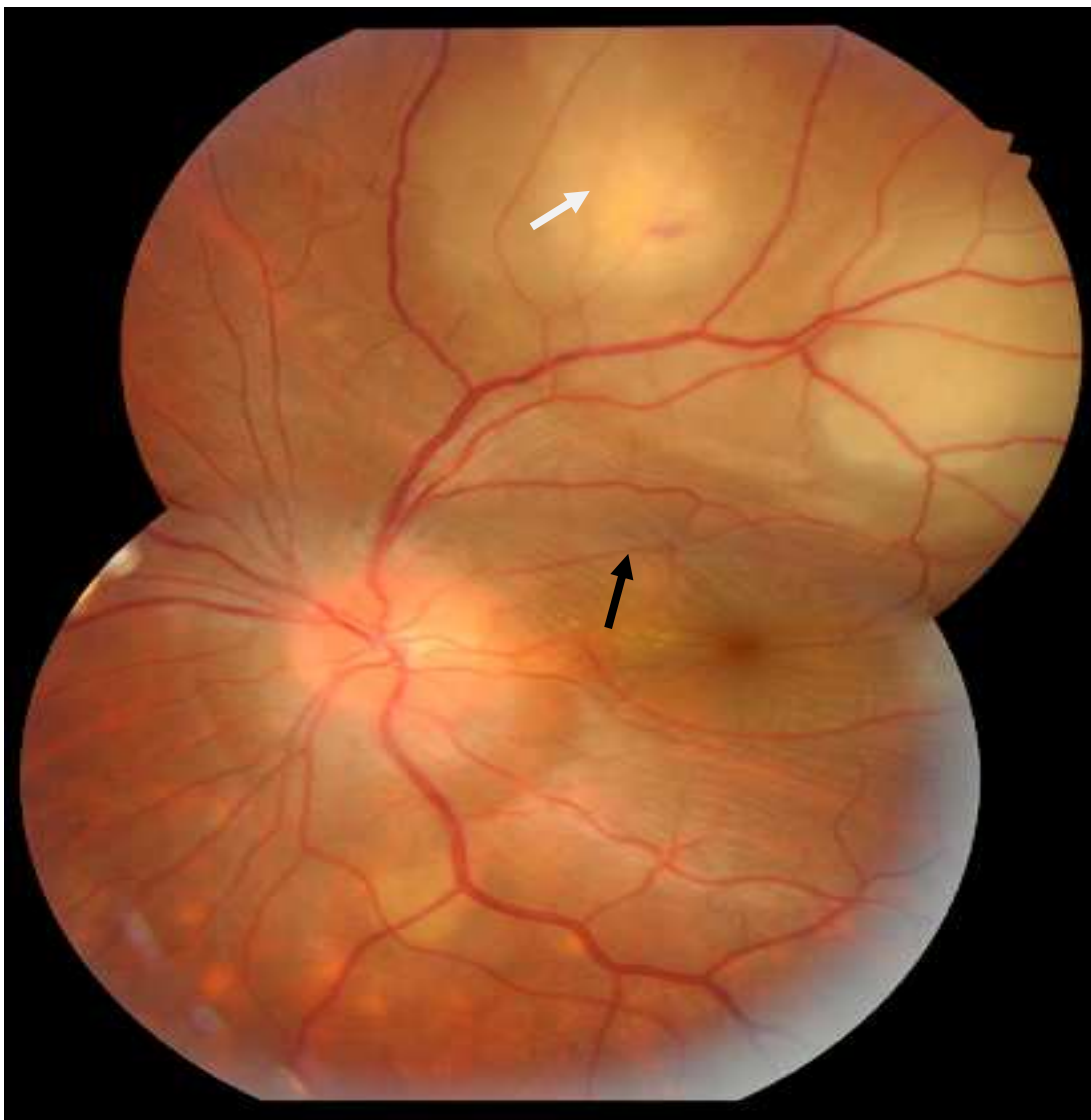


Figure-1: Showing Disc edema, an active choroiditis lesion (white arrow) superiorly with an elevated subretinal abscess (black arrow) and ILM folds at the macula.

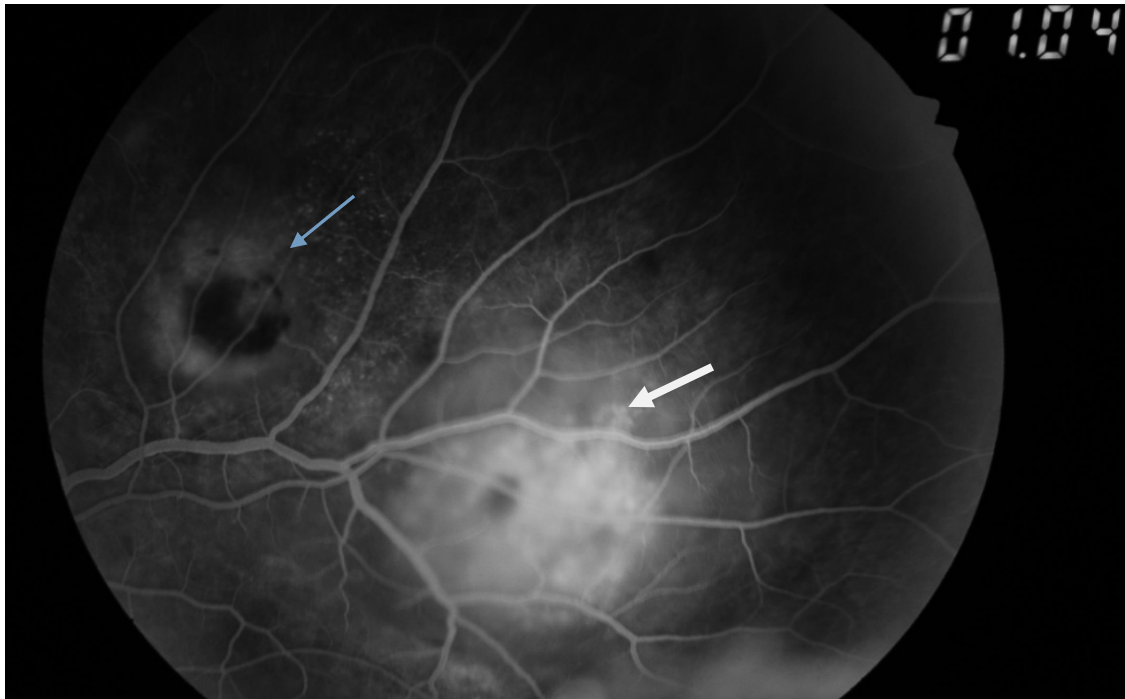


Figure-2: Early Phase showing hypofluorescence corresponding to choroiditis patch (blue arrow), with leakage corresponding to choroidal abscess (white arrow)

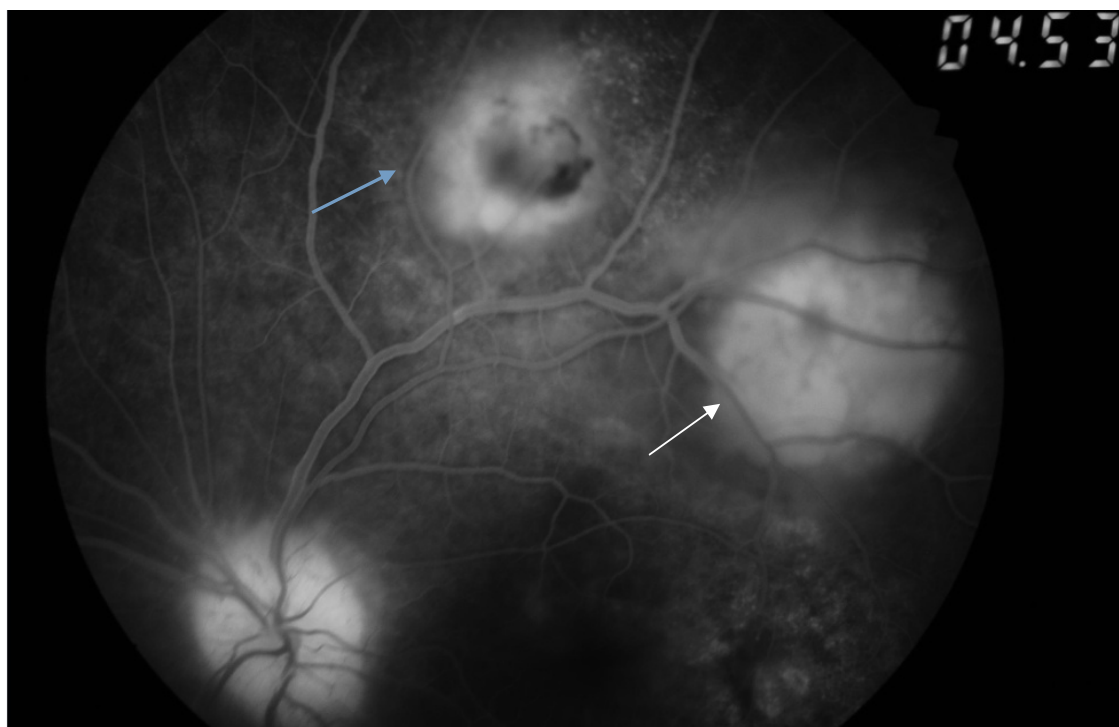


Figure-3: Late Phase FFA image showing leakage around disc, leakage in the abscess (white arrow), blocked fluorescence with leakage in choroiditis patch (Blue Arrow)

Patient was started on category 1 Antituberculous therapy with tapering dose of oral steroids for a period of six weeks. When the patient reviewed with us after 3 weeks, BCVA had improved to 6/6. There was complete resolution of vitritis. There was complete resolution of the choroidal abscess and the choroiditis lesion (figure 4). Patient was recommended to complete the ATT regimen completely while reviewing with us every month.



Figure-4: Fundus picture after one month of ATT with complete resolution of disc edema, choroidal abscess and the choroiditis patch

Discussion

Ocular tuberculosis occurs due to the infection of the ocular tissues with *Mycobacterium Tuberculosis*. The association of tuberculosis with ocular disease was recognized in the 1700s, when iris lesions were noted in patients with pulmonary tuberculosis [4]. Between 1830 and 1844, tuberculosis was identified as the cause of choroidal tubercles in few patients. Usually patients with ocular TB have no evidence of Pulmonary TB. Ocular *M. tuberculosis* infection is most often a result of hematogenous spread during Pulmonary TB or Extrapulmonary TB. Primary ocular infection in which bacilli enter the body through the conjunctiva is rare and is most likely to occur in children [2]. Patients with ocular TB should be investigated for HIV and miliary tuberculosis. Ocular tuberculosis causes a wide variety of clinical manifestations from the eyelids to the posterior segment. Most common presentation of ocular tuberculosis is posterior uveitis. It can be seen both in immunocompetent and immunocompromised patients. Choroid is the most commonly involved site due to the extensive vascular networks which makes it susceptible during hematogenous spread of *M. tuberculosis* [1,3]. Choroidal involvements include

choroiditis, tubercles, tuberculomas and subretinal abscess with the most common manifestation being choroidal tuberculomas. Large tuberculomas may undergo liquefactive necrosis and form yellowish subretinal abscess with little vitreous inflammation. Rarely, subretinal abscess can rupture into vitreous cavity leading to endophthalmitis or panophthalmitis [4].

Microbiological (culture/acid-fast bacilli staining/PCR) evidence of mycobacterium TB from intraocular fluid or tissue constitutes the gold standard for diagnosing intraocular TB [4]. The difficulty in achieving ocular samples and the complications encountered while procuring a sample lessens the diagnostic utility of the test. 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 [4].

A scan usually reveals a low to medium reflectivity and B scan finds its purpose in differentiating a tuberculoma from malignancies. PCR is both a sensitive and specific method for early detection. But a negative PCR does not necessarily exclude the diagnosis of Ocular TB.

Subretinal tuberculomas and abscess, if diagnosed early, are amenable to medical treatment. Early treatment prevents complications like subretinal fibrosis and inflammatory choroidal neovascularisation membrane and also prevents recurrences. Active pulmonary tuberculosis should be ruled out in cases of ocular inflammations before starting steroids in order to avoid exacerbation of the lesions. Antituberculous therapy using four drugs for a period ranging from 6 to 18 months have been documented in literature but the results are variable and there is no standardized treatment protocol [5]. Four drugs namely, Isoniazid, Rifampicin, Pyrazinamide and Ethambutol are given during the initiation phase followed by Isoniazid and Ethambutol for a variable period of 4–7 months. In our case response to ATT was evident within four weeks of therapy. Steroids are given concomitantly with ATT for 4–6 weeks in tapering doses in order to reduce inflammation and tissue damage from delayed hypersensitivity.

Ocular tuberculosis poses a serious threat in our country due to the endemic nature of the disease. Early diagnosis and prompt treatment prevent sequelae and recurrences of inflammation. In our case the patient presented with very subtle clinical feature but with extensive lesions in the posterior segment. Very few

reports of choroidal tuberculosis presenting simultaneously with a choroidal abscess, choroiditis patch and disc edema are available. The diagnosis of ocular tuberculosis should be considered even in young immunocompetent patients in the appropriate clinical setup.

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