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Case Study

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AN UNUSUAL CASE OF TUBERCULAR NEURORETINITIS AND JUXTAPAPILLARY CHOROIDITIS

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ABSTRACT

OTB still represents a major diagnostic and therapeutic challenge, due to its heterogeneous clinical manifestations, mixed ocular tissue involvement, lack of diagnostic criteria and gold standard tests, and lack of international agreement on the therapeutic approach. We report a rare case of patient with tuberculous neuroretinitis and atypical juxtapapillary choroiditis who responded to antituberculosis treatment (ATT) and systemic corticosteroid with complete recovery of vision.

KEYWORDS: Choroiditis, neuroretinitis, juxtapapillary, corticosteroids.

INTRODUCTION

Tuberculosis is a global health challenge, affecting more than 2 billion people worldwide, with Asian countries like India and China being the hardest hit.[1-4] Ocular TB is a rare extra pulmonary form of the disease with a potential impact on vision in patients diagnosed with the disease. OTB still represents a major diagnostic and therapeutic challenge, due to its protean clinical manifestations, and mixed ocular tissue involvement.

The disease can present as granulomatous anterior uveitis, intermediate uveitis, choroidal tuberculomas, serpiginous-like choroiditis, multifocal choroiditis, retinal vasculitis, neuroretinitis, optic neuritis, panuveitis and scleritis. The choroid is the most commonly affected ocular structure in intraocular TB in the form of multifocal choroiditis.[6,9] It results from an immune-mediated hypersensitivity reaction against TB bacilli.[5] Patients with presumed tubercular serpiginous-like choroiditis, usually have unilateral multifocal irregular serpiginoid lesions involving the periphery, sparing the juxtapapillary area whereas individuals with classic SC are more likely to have bilateral, larger lesions in the posterior pole extending from the peripapillary area.

Serpiginous choroiditis is believed to be an autoimmune disease and responds well to systemic steroids; in contrast, treating serpiginous-like choroiditis with steroids can lead to serious systemic and local complications if not accompanied by antitubercular therapy (ATT).[10] The use of adjunctive systemic corticosteroid therapy may help reduce the inflammatory reaction, but its beneficial effect and safety remains controversial.[11,12,13]

Optic nerve involvement is a common complication of ocular TB. It may result from direct mycobacterial infection, by contiguous spread from the choroid or hematogenous dissemination, or from a hypersensitivity to the infectious agent.[11] The clinical spectrum of tuberculous optic neuropathy is wide, with papillitis being the most common followed by neuroretinitis and optic nerve tubercle.[12]

Here we report a rare case of patient with tuberculous neuroretinitis and atypical juxtapapillary choroiditis who responded to anti-tuberculosis treatment (ATT) and systemic corticosteroid with complete recovery of vision.

CASE REPORT

35 years female patient was referred from a local health centre with complain of sudden and progressive diminution of vision in right eye from last 1 week. Patient had no systemic complaints and no significant ocular or medical history.

On examination VA in right eye was HMCF and left eye was 6/6. Pupillary reactions elicited RAPD in right eye while left eye pupillary reactions were normal. On testing with Ischiara chart, patient was partially color blind with right eye. Anterior segment examination was normal. Fundus examination of right eye revealed optic disc edema, multiple peripapillary flame shaped haemorrhages, hard exudates around macula (macular

star), vascular sheathing with multiple active choroidal patches in periphery as well as juxtapapillary region (Fig. 1a). OCT macula showed lost foveal contour and multiple pockets of intraretinal fluid (Fig. 1b). All blood investigations were normal. Chest X-ray, USG abdomen, HRCT chest, MRI brain with orbits were normal. Mantoux test was positive with redness and induration of more than 15mm (Fig. 2).

After consultation with Pulmonologist, patient was started on Anti-Tubercular treatment under cover of tapering doses of oral steroids according to body weight. Patient was re-evaluated after two weeks of starting treatment, VA of patient had improved to 6/36, RAPD was still present and color vision was still abnormal. Anterior segment examination was again normal, and there was a marked reduction in optic disc edema with resolving haemorrhages. There was resolution in choroidal patches which now had well-defined margins but macular star persisted (Fig. 3). OCT showed juxtafoveal elevation, PVD and disrupted RPE-Bruch's membrane (Fig. 3).

After 1 month, optic disc edema and haemorrhages resolved with well defined and pigmented choroidal patches. After tapering steroids over two months, steroids were stopped. At four months follow up, there was no active choroiditis and VA of the patient was 6/12 with normal pupillary reactions and color vision. At six months follow up, ATT course was completed and fundus revealed multiple healed choroidal patches with central staining (Fig. 4). OCT was normal (Fig. 4), patient was kept on follow up for six months, when no recurrence was noted and after that patient was lost to follow up due to COVID-19.

DISCUSSION

Diagnosis and treatment of ocular TB is difficult due to its varied spectrum of presentation, coupled with the difficulty of making a definitive diagnosis, due to lack of a tissue sample and uncertain guidelines and protocols for anti-tuberculosis treatment. Many patients with ocular involvement have no systemic or pulmonary complaints and the diagnosis is usually presumptive, based on indirect evidence as was Mantoux test in our case.

The diagnosis of tuberculous chorioretinopathy remains a challenge and is usually presumptive. Patients presenting with multifocal choroiditis and optic neuropathy can be a diagnostic dilemma. Important diagnoses to be considered in the differentials when managing these patients include tuberculosis, sarcoidosis, histoplasmosis, syphilis, posterior scleritis, intraocular lymphoma, diffuse unilateral subacute neuroretinitis, and Lyme disease. An initial laboratory work-up should be performed in all patients. Thus, several lines of evidence should support the diagnosis of tuberculous chorioretinitis, such as history of systemic or previous TB, *Mycobacterium tuberculosis* detected in body fluids or tissues, the characteristic fundus lesions, PPD positive, good response to anti-tuberculosis therapy, and exclusion of other systemic diseases causing choroidal inflammation.[14,15,16]

Abrams and Schlaegel reported that out of 18 patients with presumed tubercular uveitis, chest X-ray showed no evidence of active or inactive tuberculosis in 17 cases, similar to the condition in our patient.[17] These authors followed Woods' guidelines in patients with chronic iridocyclitis, i.e. after searching for and finding no other cause for the inflammation, patients with positive TST were given a 6 to 12 month course of ATT.

In our case we made a clinical presumptive diagnosis of Intraocular TB based on positive Mantoux test, as our patient had no history of contact with a known pulmonary TB case, she had no significant systemic findings and her all investigations were essentially normal. Favorable clinical response to anti-tuberculosis therapy verified our presumptive diagnosis and patient's visual acuity gradually improved during the first 10 days of HRZE anti-tubercular regimen, confirming the presumed diagnosis.

Tubercular choroidal lesions may develop later as well in the course of optic neuropathy[18,24] as was seen in our case. Unilateral optic disc swelling may also be seen secondary to tubercular posterior scleritis. 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.[19]

Based on a recent meta-analysis by Kee et al.[20] on treatment of different types of ocular TB, there was minimal difference in the outcome between patients treated with anti-TB therapy alone (85% successful outcome) and those with concomitant systemic corticosteroid (82% successful outcome). One concern while initiating anti-TB without any anti-inflammatory drug is paradoxical worsening or progression of the lesions due to a Jarisch–Herxheimer-like reaction. It is generally due to immune response to the antigens released from dying bacilli.[23] It is important to be aware of the possibility of such reactions while using anti-TB, follow the patients in short intervals in first weeks of treatment and to start appropriate anti-inflammatory medication in this situation.[21,22]

Our patient was treated with systemic anti-tuberculosis therapy under cover of oral corticosteroids which were tapered over period of time and received a favorable clinical response. Combined applications of routine TB tests, fundus multimodal imaging and diagnostic therapy greatly contribute the clinician to establishing precise diagnosis and monitoring the therapeutic response.

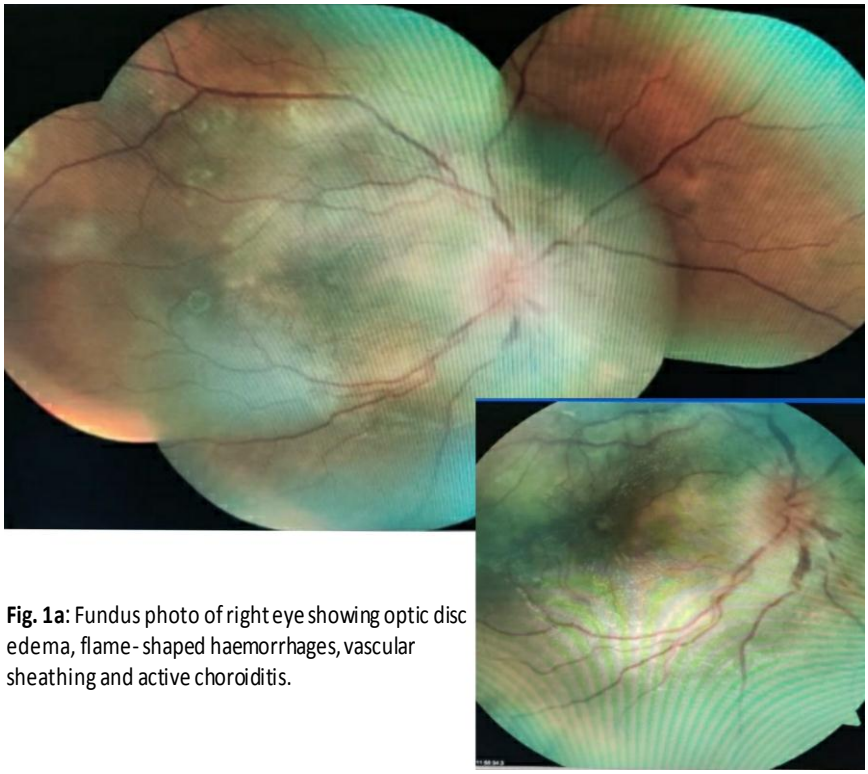


Fig. 1a: Fundus photo of right eye showing optic disc edema, flame-shaped haemorrhages, vascular sheathing and active choroiditis.

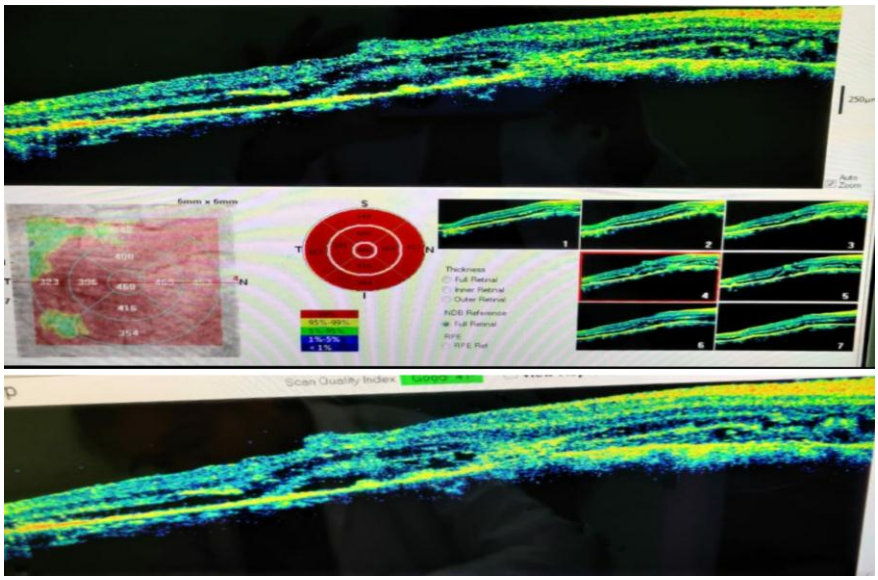
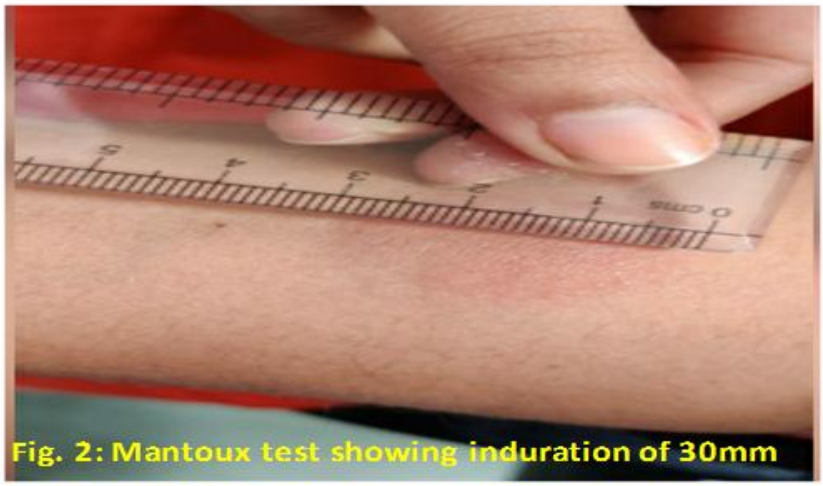


Fig. 1b: OCT of right eye showing intraretinal fluid



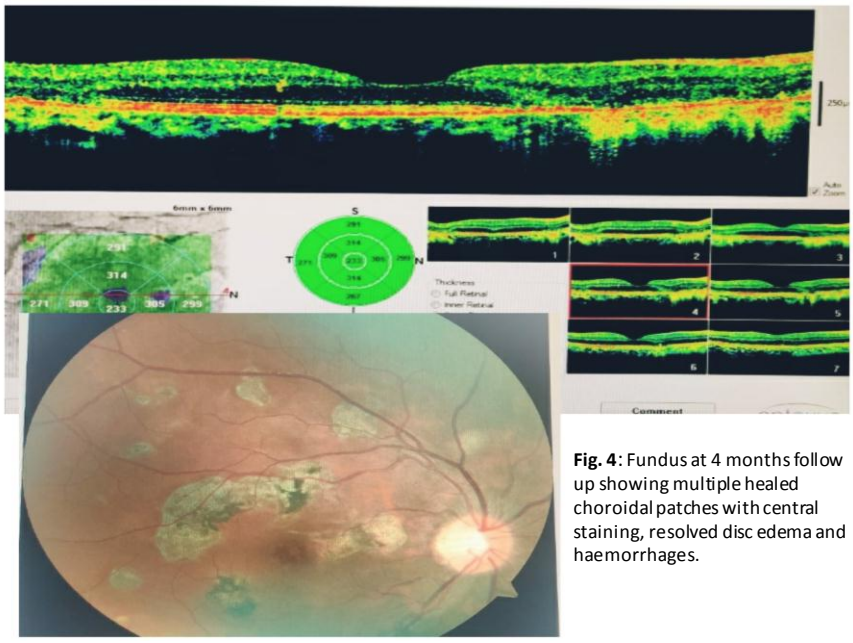


Fig. 4: Fundus at 4 months follow up showing multiple healed choroidal patches with central staining, resolved disc edema and haemorrhages.

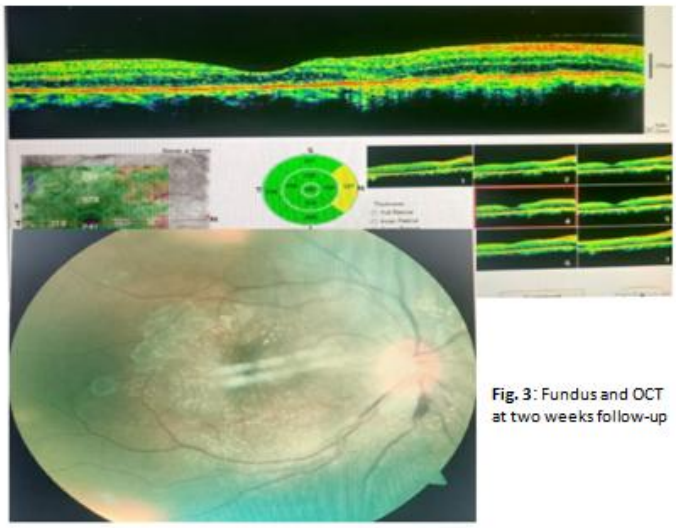


Fig. 3: Fundus and OCT at two weeks follow-up

CONCLUSION

Our case highlights the contagious spread of the infection from choroid to optic nerve head and its successful management with systemic steroid and ATT based on presumptive diagnosis. In summary, in patients with tuberculous choroiditis, early diagnosis is crucial for prompt and complete visual recovery and to decrease the likelihood of relapse. Therefore, in such patients, a relatively short course of ATT (6 months) may be adequate and cover of steroids may prevent paradoxical worsening.

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