Case Report

Recurrent bilateral optic neuritis with multifocal choroiditis in a toxoplasmosis positive individual

D K Sindal[1], Smita Javadekar[2], Gaurav Paranzape[3], Palwinder Pal Singh[4*]

[1]Professor and Head, [2]Assistant Professor, [3]Associate Professor, [4]JR I, Department of Ophthalmology, Krishna Institute of Medical Sciences, Karad, Maharashtra, INDIA.
Email: pallwz007@gmail.com

Abstract

Toxoplasma is a leading cause of posterior uveitis in patients manifesting as a focal posterior retinochoroiditis. It also causes systemic disease with potentially lethal complications. This obligate intracellular parasite has worldwide distribution with an estimated 1 billion people affected.[1] The typical Toxoplasma lesion consists of fluffy gray or white-yellow retinal infiltrates adjacent to an old pigmented scar with overlying exudation of the vitreous.[2] Several unusual presentations in ocular toxoplasmosis have been reported, including: papillitis, neuroretinitis, retrobulbar neuritis, outer retinal toxoplasmosis, central serous retinopathy, retinal detachment, macular edema, scleritis, and multifocal diffuse necrotizing retinitis in the elderly. Complications of ocular toxoplasmosis include secondary glaucoma, central macular edema, vascular occlusion, retinal neovascularization, choroidal neovascularization, subretinal neovascularisation. We at KIMSDU karad (ophthalmology department) are reporting a case of ocular toxoplasmosis presenting initially as recurrent optic neuritis with minimal vitritis and severely diminished vision with multifocal chorioretinitis which is under treatment.

Keywords: Toxoplasmosis, Optic neuritis, Vitritis.

[*]**Address for Correspondence:**
Dr. Palwinder Pal Singh, JR I, Department of Ophthalmology, Krishna Institute of Medical Sciences, Karad, Maharashtra, INDIA.
Email: pallwz007@gmail.com
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INTRODUCTION

A female patient aged 42 years presented to the Opd with complaint of diminution of vision in both eyes since 8 to 10 days which was associated with pain in both eyes and frontal headache. She had two similar incident previously for which she was treated in the month of august2014 and was given inj. methy prednisolone in our hospital, and for the other incident she got admitted in some private hospital and was treated similarly. The patient had history of wearing spectacles since 5 years with no history of any ocular trauma. The patient also was a known case of hypertension since 6 months for which she had been advised by a physician to take Tab. CTD 12.5mg (Clorthiazide + Telmisartan) once daily and Tab Cinod 5mg (clinidipine) once daily at night. On general examination, her blood pressure was 150/90mmHg whereas other parameters including pulse rate ,respiratory rate and temperature were normal. Systemic examination was within normal limit. On ocular examination it was found that the patient had visual acuity of finger counting at 2 meters for both her eyes, and her colour vision on ishihara's type test was noted. There was also bilateral conjunctival congestion with circumcilliary congestion, keratitic precipitates and iris pigments on corneal endothelium. The anterior chamber of both eyes had grade 2 cells, there was bilateral cataract with iris pigments over the lens capsule. The I.O.P on Perkins's tonometer was 16mmHg in both eyes, and the regurgitation test was negative for either eye. On Dilatation of the pupils it was seen that the right eye was dilating up to 7mm and the left eye up to 8 mm. On fundus examination (initial as on admission as shown in fig1.1, 1.2)

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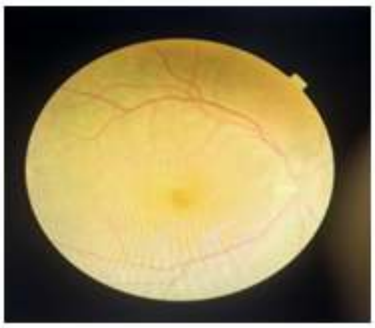
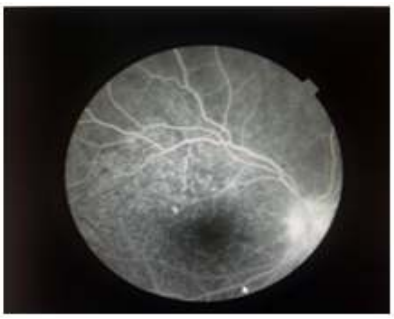
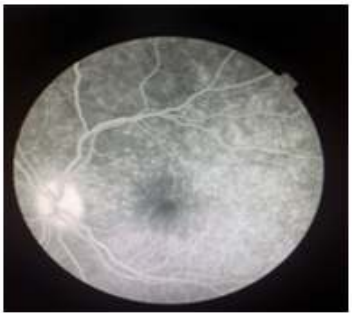



It was seen that that the media was hazy because of vitritis (grade II cells in vitreous), disc was hyperaemic with optic cup margins being elevated with optic disc margins blurred, venous pulsation were negetive in both eyes, there was macular odema with absent foveal reflex in both the eyes. The blood vessels appeared normal with general visual fundus showing retinal folds radiating from disk towards macula with subretinal fluid with no elevation. On Fluorescein fundus angiography as shown in fig 1.2

Revealed hyperfluroscence secondary to leakage. Biochemical (TORCH) examination revealed
Rubella IgG (+), IgM(+)
CMV IgG (+) IgM(-)
HSV IgG(+) IgM(+)
Toxoplasma IgG(+) IgM(+)
After 1 week of treatment with inj. Methyprednisolone 1g given in 100 cc of normal saline over 3-4 hrs, with tab. Septran (trimethoprine + sulphamethoxazol) once daily, the vision was 6/24 in right eye and 6/18 in the left eye. Fundal examination showed decreased grade cells in vitreous and decreased vitritis.

DISCUSSION

Diagnosis of ocular toxoplasmosis is mainly based on clinical grounds and biochemical investigations. Differential diagnosis include entities resulting in retinitis and secondary vitritis including ARN(Acute Retinal Necrosis) from herpes simplex and zoster, cytomegalovirus, candidiasis, syphilis, TB, Lyme disease and sarcoidosis. Retinal detachment and hemorrhages are rare in toxoplasma but common in Acute Retinal Necrosis. Toxoplasma may be differentiated from CMV retinitis by the presence of more prominent anterior and vitreous reactions, lack of retinal hemorrhage and fluffy thick borders. CMV has relative quiet chambers, prominent hemorrhages and dry, granular borders. Attempts at parasite identification in tissue and body fluid specimens are often unproductive. Parasites may be cultured by inoculation of patient tissues into mice peritoneum or tissue culture cells. Alternatively, polymerase chain reaction analysis (PCR) can be used to detect the toxoplasma DNA in tissues. Fluorescein angiography is a useful adjunct in the diagnosis and management of ocular Toxoplasmosis. Active lesions show hyperfluorescence; early leakage and late staining is observed in areas of vasculitis. Inactive lesions manifest as dye blockage and late staining. Other FA findings include window defects, later scleral staining and presence of choroidal neovascularization.[3] Unfortunately the treatment of ocular manifestation in man is not as easy as that in experimental infections in animals; the cysts are not destroyed by drugs and recurrence are still possible after the course of treatment, moreover the strain of the organism may have considerable influence on the result of therapy thus kaufman and his colleagues in 1958-59 have shown that pyrimethamine is less effective against slow growing strains and that large doses are necessary to obtain concentrations sufficiently high to have a therapeutic effect on them. To avoid the development of thrombocytopenia and leucopenia a blood

count should be repeated at frequent intervals during the course of treatment[4]. There are some grounds for giving folic acid 10-15 mg daily or brewer's yeast as prophylactic measure against side effects (Frenkel and Hitchings 1957;Giles, 1964. It should be noted that corticosteroids are given to counter the inflammatory response to toxoplasma. They should never be given without concomitant antibiotics and should not be given by depot steroid injections which may lead to uncontrolled infection. Sometimes steroids treatment may be delayed to allow systemic antibiotic levels to rise.[5] Clindamycin (Cleocin) is a semisynthetic antibiotic with protozoacidal activity. It acts by a different mechanism from the folic acid antagonists and may be synergistic with them. Clindamycin has high ocular tissue concentration, has been shown to have activity against the cyst form. The usual dosage is 150-300 mg four times a day. Clindamycin does not carry the same risk of toxicity as the folic acid antagonists however, its use is associated with pseudomembranous colitis in a small number of patients. This is readily reversible with vancomycin. One way of avoiding systemic administration is by giving clindamycin subconjunctival injections of 50 mg on alternate days for a month.[6] Spiramycin is a macrolide antibiotic with proven antiprotozoal activity. It has minimal side effects is used in pregnancy because it has been shown to reduce the incidence of congenital infection. However, it has been reported to have a relatively higher rate of reinfection.[7] Though no particular combination has been definitely shown to be superior to others, the use of pyrimethamine may result in smaller scars and may be of benefit to those with macular, juxtamacular, papillary, and peripapillary lesions[8]. New drugs are being tested for use against toxoplasmosis. One promising agent is atovaquone (Mepron) which is a hydroxynaphthoquinone used in the treatment of malaria. It acts by interfering with the parasite mitochondrial electron transport system. The recommended dosage is 150 mg qid for 4 weeks. Trials are currently being conducted with this agent It is hoped that the new agents currently under study such as new antibiotics and cytokine treatment will prove to have fewer adverse reactions and to be more effective against the cyst form thus decreasing the risk of relapse.

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