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RESEARCH ARTICLE

RECURRENT OCULAR TOXOPLASMOSIS AND NORMALISATION OF SERUM IgG VALUE

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ABSTRACT

Toxoplasma retinochoroiditis has been reported as a leading cause of posterior uveitis. *Toxoplasma gondii* is ubiquitous obligate intracellular parasite with two clinical types, namely, a congenital and a postnatally acquired disease[1] Ocular toxoplasmosis is a progressive and recurring necrotizing retinitis, with vision-threatening complications such as retinal detachment, choroidal neovascularization, and glaucoma, which may occur at any time during the clinical course. In the case of acquired toxoplasmosis, which rarely causes ocular disease, the antibody titres are usually very high, and therefore serology for this diagnosis is indispensable. There lies a matter of controversy about diagnosis and treatment for ocular toxoplasmosis, and to date, many treatment options are applied clinically.[2]

INTRODUCTION

Case

A 29 years old male presented with history of redness, blurring of vision and floaters in left eye for 1 week. The diminution of vision was sudden, painless and progressive.

Patient had similar episodes of sudden painless diminution of vision in left eye in past. His past records revealed that he had been diagnosed as a case of toxoplasma retinitis with positive serological tests 9 years back. He had visual acuity of 1/60 in the left eye and 6/6 in the right eye with 4+ cells and flare in anterior chamber and vitreous. Fundus examination showed grade 2 media haze with macular edema and localised macular detachment. On investigations, the serum titres of IgG against toxoplasma were raised to 26.00 (normal value <6.0) and IgM 1.18 (normal <0.8) while patient had normal haemogram with negative rheumatoid factor, and ANA. X-ray chest and sacroiliac joint showed no abnormality. Patient was admitted for 10 days and put on clindamycin 300 qid for 10 days, Septran DS for 6 weeks, oral prednisolone (60mg) in tapering dose, topical prednisolone acetate 1% 1hourly then tapered slowly over 6 weeks and atropine tds for 2weeks. On discharge his visual acuity was 6/12 and improved to 6/6 after 3 months in left eye. The patient remained asymptomatic for 9 years after treatment.

His present general physical and systemic examinations were within normal limits. On examination his visual acuity was 6/9 in left and 6/6in right eye. Slit lamp examination showed circumcilliary injection, old pigmented keratic precipitates, normal anterior chamber and 2+ cells in anterior vitreous. Fundus examination showed grade 2 media haze with old healed pigmented patch of size 3DD inferotemporal to macula along with a yellowish satellite lesion with indistinct margins of size ¼ DD inferotemporal to the old healed lesion. (Figure 1,2)

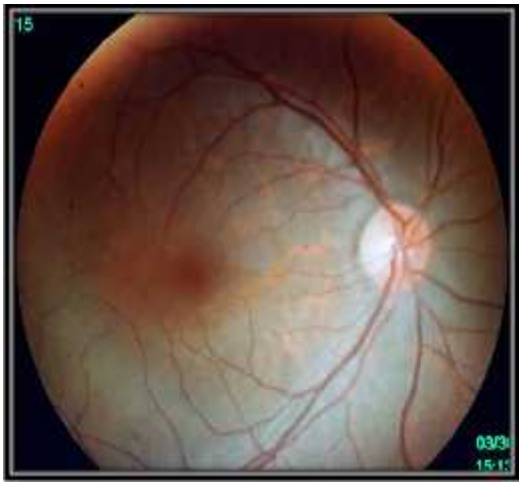

Figure 1 Normal fundus of right eye

Complete haemogram was normal, rheumatoid factor, ANA was negative. IgG titre against toxoplasma were raised to 29.44 (normal value <6.0) and IgM 1.20 (normal <0.8). X-ray chest and sacroiliac joint showed no abnormality. Montoux test was also negative.

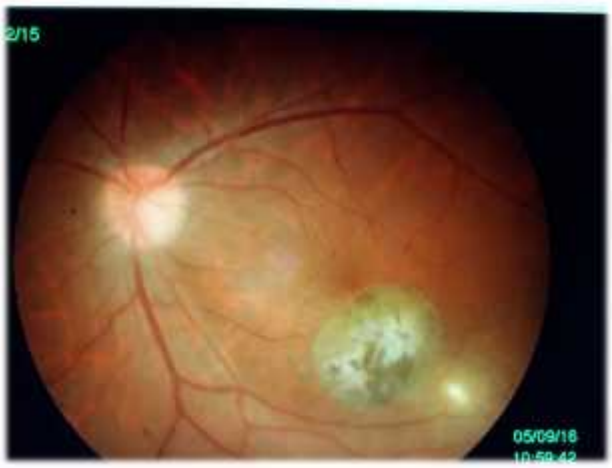

Figure 2 Fundus photograph of left eye showing old healed pigmented patch along with yellowish white active satellite lesion inferotemporally, choroidal folds radiating from the old scar.

Patient was started on clindamycin 300 mg qid for 10 days along with cotrimoxazole double strength BD for 3 weeks and a course of oral steroids was given after 24 hours of initiating above treatment in tapering dose. He was put on topical prednisolone acetate 1% 4 hourly and then tapered slowly over 6 weeks along with atropine. The patient responded well, having visual acuity of 6/6 in left eye (Figure 3) at the time of discharge. At 6 months follow up visual acuity was 6/6 in both eyes and no evidence of active inflammation. His IgG titre were 4.94 and IgM titre were 0.67(below the normal range).

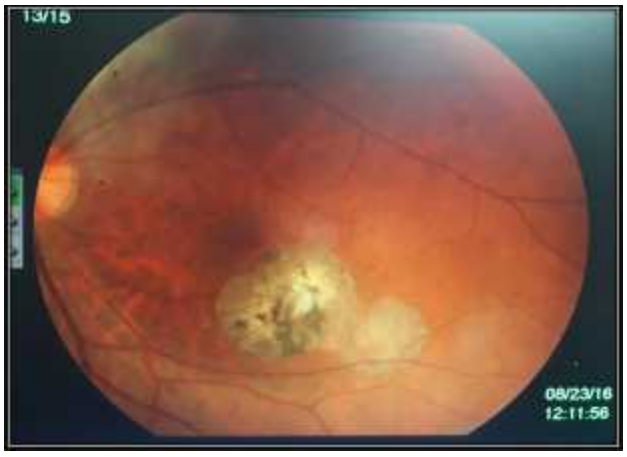

Figure 3 Fundus photograph of left eye showing well defined healed patch inferotemporal to old healed pigmented chorioretinal scar.

DISCUSSION

T. gondii primarily exists in three forms: oocysts, tachyzoites, and bradyzoites. The definitive hosts are members of the family Felidae, including domestic cats. Various warm-blooded animals serve as intermediate hosts. *Toxoplasma gondii* is transmitted by three known modes: congenitally, through the consumption of uncooked infected meat, and via fecal matter.Oocysts are only produced in the definitive host, members of the family Felidae, when passed in feces and then ingested, the oocysts can infect humans and other intermediate hosts. They divide rapidly in cells, causing tissue destruction and spreading the infection. Eventually tachyzoites localize to muscle tissues and the CNS where they convert to tissue cysts, or bradyzoites. This is thought to be a response to the host immune reaction.[3]

In our patient intially there was non granulomatous anterior uveitis with associated vitritis and localised macular detachment with macular odema. With treatment the active lesion subsided only to recur after 9 years as whitish foci of retinochoroiditis adjacent to old healed pigmented atrophic scar with associated vitritis. These features corborate with the classical clinical presentation of retinochoroiditis in ocular toxoplasmosis in previous studies.[2]

The studies have postulated rising titres of antibodies in parasitemia in acute and chronically infected patients and in reactivation of disease. However there is no immune response once the patient has inactive bradyzoite cysts in ocular tissue, nervous system or any other tissue.[4] Smith *et al*. have found that retinal vascular endothelial cells are more readily infected with *T. gondii* compared with endothelial cells from the other sites of the body, which suggests a preferential infection of the retina by the parasite.[5,6]

The use of serologic tests for demonstration of specific antibody to *T. gondii* is the initial and primary method of diagnosis. Different serologic tests often measure different antibodies that possess unique patterns of rise and fall with time after infection A panel of tests (the *Toxoplasma* Serological Profile [TSP]) is available, the most commonly used tests for the measurement of IgG antibody are the Sabin-Feldman dye test DT, the ELISA, the IFA, and the modified direct agglutination test. In these tests, IgG antibodies usually appear within 1–2 weeks of acquisition of the infection, peak within 1–2 months, decline at various rates, and usually persist for life. IgM antibodies may appear earlier and decline more rapidly than IgG antibodies.

Rising titre of serial IgG and IgM antibodies obtained at interval 3–4 weeks apart provides the best diagnostic clue, while a single high titer of any immunoglobulin is insufficient. Negative results in both tests virtually rule out the diagnosis of toxoplasmosis. IgG antibodies may persist at high titres for many years and IgM antibodies may be detectable for >12 months.[7,8] In this case there was normalisation of serum titre in 6 months.

There are multiple treatment options for ocular toxoplasmosis that could vary from no treatment in extra macular lesion to multi drug therapy in visual threatening lesion and in immunocomprosed host for 4-6 weeks[9]. This treatment has shown similar efficacy to classical therapy and was effective in achieving normal serum level of IgG even after recurrence of disease.

CONCLUSION

This case report suggests that the combination of cotrimoxazole and clindamycin is an effective alternative to the commonly used regimen consisting of pyrimethamine, sulfonamides. It may be stated that the combination of cotrimoxazole and clindamycin is effective in achieving normal serum level of IgG even after recurrence of disease.

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