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Case Report Of Reactivation Of Unilateral Ocular Toxoplasmosis In An Immunocompetent Patient

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ABSTRACT
Ocular toxoplasmosis can cause potentially blinding necrotizing retinitis with a progressive and relapsing course. It is the most common cause of posterior uveitis worldwide, and usually presents as a localized retinochoroidal lesion. Despite its high incidence, ocular infection with toxoplasmosis tends to be subclinical and goes unnoticed until it is too late. To highlight this blinding disease and to emphasize the importance of preventive measures to avoid grave visual disability, we report this case of a middle aged gentleman with a history of toxoplasmal retinitis 6 years ago with acute onset blurring of vision and floaters. The best-corrected vision for the affected eye was 20/30. Fundus examination showed a well demarcated, atrophic, hyperpigmented healed toxoplasmosis scar with an adjacent new retinochoroidal lesion and mild focal vasculitis. Serology demonstrated acquired toxoplasma gondii infection with IgM and IgG antibodies. He responded well to oral sulfamethoxazole and trimethoprim combination, clindamycin, oral methylprednisolone and topical steroids. Since it is a potentially blinding disease with recurrences, preventive measures are important. Proper washing of hands and strict food hygiene is crucial. Patients with retinochoroidal scar harbouring cysts should be monitored periodically for recurrence.

Keywords: *Ocular toxoplasmosis, retinochoroiditis, vasculitis*

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I. INTRODUCTION

Toxoplasma gondii, a protozoan parasite is the most common cause of posterior uveitis worldwide that can infect upto one third global population.[1]. Even though posterior uveitis being a self-limited disease, it may affect the vision secondary to the optic nerve or macular involvement and/or severe vitreous inflammation.[2] Multifocal lesions and large areas of retinal necrosis or retinochoroiditis are seen in atypical cases of ocular toxoplasmosis.[3] Therapy must target both the parasite and host immunity. Typical regimens include Pyrimethamine/Sulfadiazine or Trimethoprim-Sulfamethoxazole and Prednisolone as triple therapy, with the addition of Clindamycin in quadruple treatment for at least 4 weeks–6 weeks . A few authors [4] have reported oral Prednisolone dosages ranging from 1.0 mg/kg/day to 1.5 mg/kg/day, up to a daily maximum of 20 mg/day. However, corticosteroid therapy in patients with ocular toxoplasmosis varies from not using them at all to starting them within three days or a week after anti-parasitic treatment.[5]. In non responsive cases, other treatment modalities like intravitreal clindamycin injection with or without dexamethasone implantation and pars plana vitrectomy with silicone oil tamponade can be considered.[6]

II. CASE REPORT

A 44 year old male with no known co morbidities presented with complaints of blurring of vision and floaters in the right eye for 1week.He consumes mixed diet and has a history of contact with stray cats. His best corrected visual acuity was 20/30 in right eye and 20/20 in left eye. Slit lamp biomicroscopy revealed circumcorneal congestion with 2+ cells in anterior chamber and cells in retrolental space in his right eye with left eye being quiet.Fundoscopic examination of right eye showed a well demarcated atrophic hyperpigmented lesion of size 3DD which is about 4DD away from inferonasal margin of disc, suggestive of a healed toxoplasmosis scar. A new retinochoroidal lesion of size 3/4DD was noted 1/2DD away from nasal margin of scar with adjacent vessel sheathing suggestive of reactivation of toxoplasma infection. He was treated with Sulfamethoxazole and trimethoprim combination ,oral clindamycin, oral methyl prednisolone and topical steroids. The serology tests identified anti-toxoplasma IgG and IgM. After 1 month of treatment his vision improved to 20/20 regression of the new lesion and vasculitis.

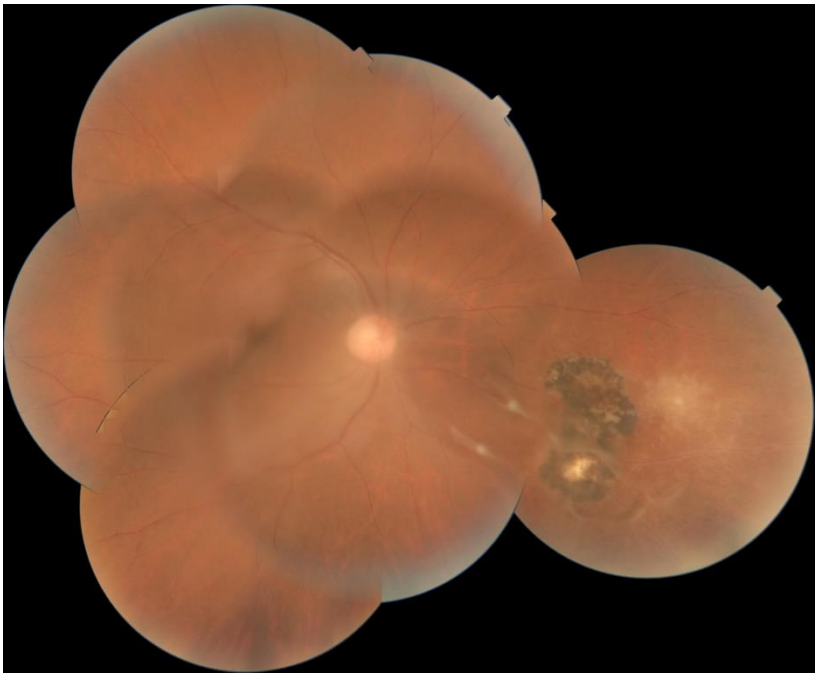

Figure 1: Fundus photo of right eye at presentation showing a well demarcated atrophic hyperpigmented healed toxoplasmosis scar with an adjacent new retinochoroidal lesion with mild focal vasculitis suggestive of reactivation of toxoplasma infection.

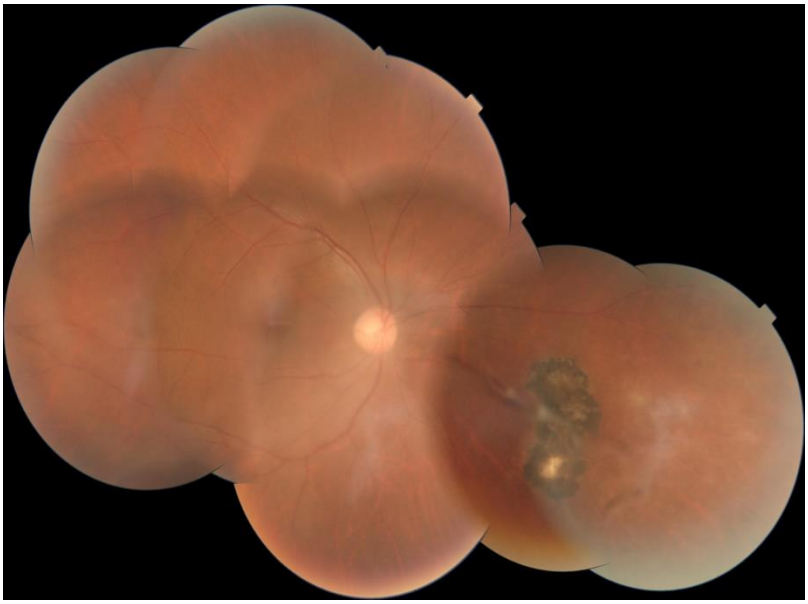

Figure 2: Fundus photo of right eye after antiparasite treatment combined with prednisolone showing regression of retinochoridal lesion and focal vasculitis .

III. DISCUSSION

Toxoplasmosis is caused by a protozoan,Toxoplasma gondii an obligatory parasite of cat. Infection can be acquired by ingestion of oocysts in cat faecal matter or bradyzoites in undercooked meat. Our patient had history of contact with cats and consumption of undercooked meat. His source of infection is most likely to be acquired by ingestion of oocysts in cat faecal matter or bradyzoits in undercooked meat.

Ocular toxoplasmosis may not require immunosuppression to occur. It is thought to represent either a reactivation of congenital infection or an acquired infection by a parasite.[7] Unilateral lesions are more common with acquired toxoplasmosis. In contrast, congenital disease is bilateral in three quarters of patients and has a predisposition to involve the macular region. Typical Ocular toxoplasmosis findings include white focal retinitis with overlying vitreous inflammation (headlight in the fog appearance) , adjacent pigmented retinochoroidal scar, vitreous inflammation (mild, moderate or severe), secondary non-granulomatous iridocyclitis,

granulomatous and stellate keratic precipitates, inflammatory ocular hypertension and retinal vasculitis (usually near the focus of retinochoroiditis). Atypical features, which can present with retinochoroiditis, could include papillitis, neuroretinitis, retrobulbar neuritis, scleritis, retinal detachment, punctate outer retinitis,branch retinal artery occlusion, frosted branch angiitis, Coats'-type response, Fuchs-like anterior uveitis or multifocal diffuse necrotising retinitis.

Recurrence in an old, healed, ocular lesion is the most common cause of active infection in healthy individuals and typically occurs adjacent to an old scar. The cysts remain inactive at the borders of the scar for years and may rupture causing recurrence. Toxoplasmic retinochoroiditis is a recurrent disease in two-thirds of patients.[8] The recurrences occur between the age of 10 and 35 years with an average age of 25 years and is greater during the 1 st year after an acute infection than during subsequent years.[9] The active lesions of ocular toxoplasmosis are classically adjacent to or at the border of an old inactive pigmented scar (satellite lesion). Typical symptoms are blurred vision, floaters and metamorphopsia. It typically affects the posterior pole of the fundus and usually presents as focal necrotizing retinitis involving the inner retinal layers appearing as a circular whitish fluffy lesion with surrounding retinal edema, localized or diffuse vitritis and a granulomatous anterior uveitis. The size varies from one-tenth to five disc diameters. The severe vitritis gives a "headlight in the fog" appearance.[10] The choroid and sclera may become involved secondarily.[11] Hypersensitivity reaction to the antigen is responsible for the anterior uveitis. Sheathing of the retinal vasculature, vascular occlusions and periarterial exudates (kyrieleis arterialitis) at or away from the foci of retinitis may be seen. In healthy patients, the retinitis heals within 1-4 months of treatment and is replaced with a sharply demarcated atrophic scar with pigmented borders.

When the lesion is characteristic, the demonstration of IgG serum antibody gives a presumptive diagnosis and allows initiation of specific therapy.[12] In doubtful cases, it is possible to detect the parasite DNA by vitreous puncture and PCR for T. gondii. Classic therapy is sulfadiazine and pyrimethamine. Folinate must be given in order to prevent pyrimethamine-induced pancytopenia and steroids can be given for sight-threatening lesions in order to control inflammation.Some authorities advocate the addition of clindamycin to the protocol. Clindamycin is also an alternative therapy when sulfa drugs are not tolerated. Other effective drugs are spiramycin, atavaquone or azithromycin with or without pyrimethamine. Recent studies show that co-trimoxazole is also an effective therapy.[13] Our patient responded to treatment with Sulfamethoxazole and trimethoprim combination, oral clindamycin, oral methyl prednisolone and topical steroids.

IV. CONCLUSION

Awareness of risk factors for acquired ocular toxoplasmosis is crucial to enable prevention of initial infection. Proper hand washing after contact with cats and strict food hygiene are important. A thorough history focusing on known risk factors such as immunosuppression and potential routes of infection can be crucial in the design of ophthalmic differential diagnoses. PCR of intraocular fluids , serology of anti- toxoplasma antibodies and response to adequate antiparasite treatment combined with prednisolone will confirm the definite diagnosis.

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