Unilateral vitritis in an immunocompetent individual – A rare presentation of ocular toxoplasmosis

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SUMMARY

Ocular toxoplasmosis is the leading cause of infectious retinochoroiditis in both adults and children. It is caused by the obligate intracellular parasite, *Toxoplasma gondii*. It is a common cause of posterior uveitis and focal retinitis, typically seen in immunocompetent individuals as a primary infection or in immunocompromised individuals as reactivation of latent infection. Here, we report a rare case of a 29-year-old immunocompetent female presenting with gradual, painless diminution of vision in the left eye associated with headache for over one month. She had a history of hypertension but no other significant medical history. On ocular examination anterior segment was unremarkable and fundus examination of left eye showed "Headlight in fog appearance" suggestive of vitritis. Serological evaluation revealed positive for Toxoplasma IgG antibodies. The patient was treated with cotrimoxazole, oral prednisolone, and topical steroids for 4-6 weeks. Following treatment, her visual acuity improved to 6/6 in left eye. This case highlights the importance of considering ocular toxoplasmosis in the differential diagnosis of unilateral vitritis, even in immunocompetent patients.

KEYWORDS:
Toxoplasma gondii, Vitritis, Trimethoprim and Sulfamethoxazole

INTRODUCTION

Ocular toxoplasmosis, caused by the intracellular protozoan parasite *Toxoplasma gondii*, is the leading cause of infectious retinochoroiditis in both adults and children.[1] The usual manifestation involves localized retinochoroiditis near a pigmented chorioretinal scar, accompanied by inflammation in the vitreous. Beyond this typical presentation, other atypical manifestations such as scleritis, rhegmatogenous and serous retinal detachment, retinal vasculitis, retinal vascular occlusion, optic neuropathy and punctuate outer retinal toxoplasmosis have been reported. Toxoplasmosis of the central nervous system typically occurs in individuals with severe immunosuppression. However, ocular toxoplasmosis can occur in immunocompetent individuals.

In this report, we present a rare case of unilateral vitritis in a young immunocompetent patient who was ultimately diagnosed with ocular toxoplasmosis. This case highlights the atypical presentation and importance of comprehensive diagnostic evaluation.

CASE PRESENTATION

A 29-year-old woman complained of progressive, painless loss of vision in her left eye associated with a headache over the course of a month. History of exposure to pets one month after her recent trip. History of intermittent fever initially for 7 days. She had a one-year history of systemic hypertension, but no history of diabetes mellitus, steroid use, or other systemic disorders. No history of photophobia, double vision, seizures, vomiting, rashes, neck stiffness, cough, or breathlessness.

On examination, the best-corrected visual acuity was 6/6 in the right eye and 4/60 in the left eye. Examination of the anterior segment revealed no bilateral abnormalities, and the intraocular pressure was within the normal range. However, visualization of the posterior segment was hindered due to dense vitritis in left eye, resulting in a "Headlight in fog appearance." B-scan ultrasonography was performed to rule out retinal detachment and other retinal pathologies. OCT of the macula was performed and found to be within normal limits.

The complete blood count and liver function test results were normal. The results of the Venereal Disease Research Laboratory, Treponema Pallidum Hemagglutination, HSV-1 IgG, HSV-2 IgG, CMV IgG, and Mantoux tests were negative. High-resolution computed tomography of the chest and magnetic resonance imaging of the brain and orbit revealed normal findings. Serological evaluation revealed positivity for Toxoplasma IgG, confirming the diagnosis of ocular toxoplasmosis. Serology for HIV and HBsAg was negative.

The patient received treatment with a combination of trimethoprim 160 mg/sulfamethoxazole 800 mg (TMP-S) twice daily for 6 weeks, Oral Prednisolone 60 mg once daily for 1 week, and was gradually tapered over a period of 6 weeks. Topical Prednisolone 1% eye drops were started with a frequency of 3rd hourly and gradually tapered over a period of 6 weeks, and IOP monitoring was performed in subsequent follow-up. Over the course of the three-month follow-up, the visual acuity of the left eye improved to 6/6, and the vitreous cavity was quiet and clear. There were no signs of recurrence.

DISCUSSION

Ocular toxoplasmosis is a well-documented manifestation of *Toxoplasma gondii* infection that often presents with

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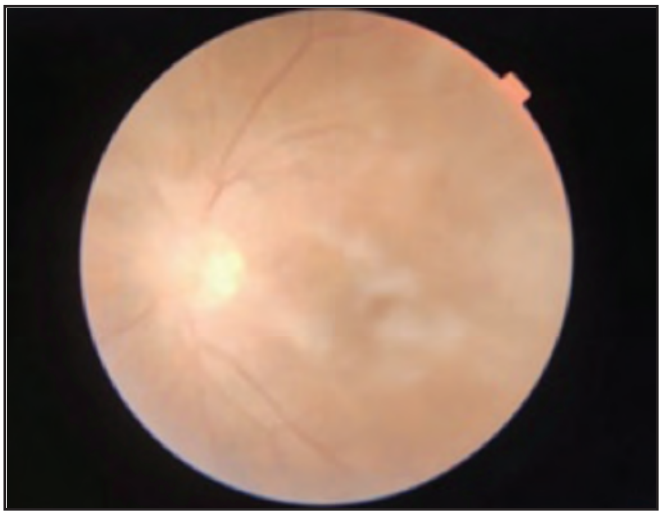

Fig. 1: Showing dense vitritis in left eye. (suggestive of "headlight in fog appearance")

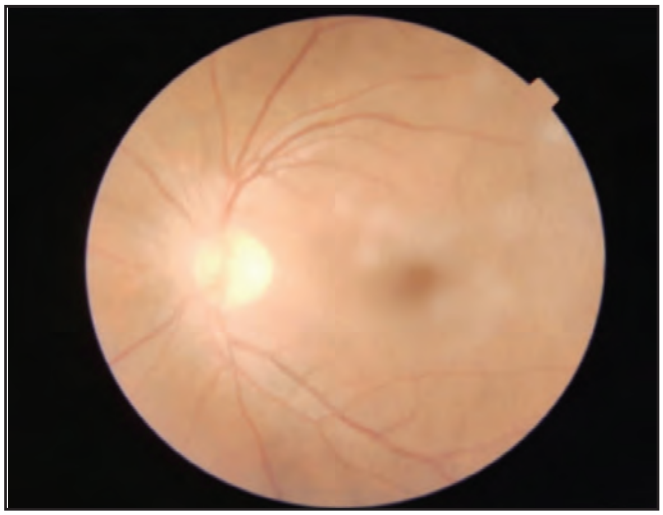

Fig. 2: Showing clear vitreous and macula in left eye following the treatment

retinochoroiditis. In most cases, patients will have some degree of vitritis and/or anterior inflammation, together with retinitis at the border of an inactive chorioretinal scar. As a result, the look of the bright white, focused retinitis viewed through vitreous haze is commonly described as a "headlight in the fog." Vasculitis frequently coexists with retinitis and can manifest as "Kyrieleis plaques," which are segmental lesions that are yellow-white in the retinal arteries. Because of Werner Kyrieleis's objectionable ideological beliefs, these plaques are now known as segmental retinal arteritis.[2] Numerous chorioretinal lesions and recurring disease are more common in female patients. According to certain studies that imply a gender difference in the patient population.[3] Acquired toxoplasmosis commonly leads to unilateral lesions, while congenital cases often exhibit bilateral involvement in approximately 75% of the patients, with a tendency to affect the macula.

It is primarily diagnosed on the basis of clinical presentation, although additional tests can help confirm the diagnosis and monitor progress. Ultrawidefield color fundus imaging provides objective measures to observe improvements in vitritis and retinitis. Fluorescein angiography can differentiate between active (leaking) and inactive (staining without leaking) lesions, and can also identify areas of retinal vessel occlusion caused by lesions. Optical coherence tomography of the macula helps rule out cystoid macular edema and aids in grading vitreous cells. Raster scans through lesions can confirm retinitis and assess lesion depth by detecting full-thickness hyper-reflectivity.[4]

Laboratory tests are useful when the diagnosis of toxoplasmosis is uncertain, based on clinical presentation and imaging. Polymerase chain reaction testing of aqueous or vitreous samples is effective, with some studies indicating higher sensitivity in vitreous samples.[5] Serological testing for *Toxoplasma gondii* IgG and IgM can evaluate past or recent systemic infections and is mainly useful for ruling out toxoplasmosis when IgG is negative.[6]

Most cases of toxoplasmosis can be effectively treated with systemic medications that target both the parasite and associated inflammatory response. Prior to 2015, the standard regimen was "triple therapy," consisting of pyrimethamine (100 mg for 2 days, then 25 mg daily), sulfadiazine (2 g) and folinic acid (5 mg). Subsequently, new treatment protocols have incorporated additional antimicrobials, such as azithromycin, clindamycin, and TMP-S, used both in conjunction with pyrimethamine and as monotherapies. While these medications have shown efficacy in multiple studies, few prospective randomized trials have directly compared their effectiveness.[7]

Yanxia Zhang, MD, of Sun Yat-sen University in China, and colleagues conducted a study in which they compared the results with systemic pyrimethamine-sulfadiazine, clindamycin, azithromycin and TMP-S using network meta-analysis. They discovered that the most significant increases in vitreous inflammation resolution and visual acuity were associated with clindamycin. In the same trial, TMP-S was shown to have fewer adverse effects and a reduced recurrence rate. When there are no contraindications, TMP-S is usually prescribed as first-line treatment.[8] And, patients with TMP-S should undergo regular renal function tests. Patients who are contraindicated for systemic therapy, for example, in the first trimester of pregnancy, or who have retinal lesions that require a quicker response might benefit from intravitreal clindamycin (1 mg/0.1 ml).

Systemic steroids are typically employed to manage severe inflammatory responses caused by Toxoplasma infections, especially severe vitritis. Depending on the severity of inflammation, oral prednisone may be started at up to 1 mg/kg and gradually decreased over weeks to months, or until vitritis clears up and retinitis seems dormant. Intravitreal dexamethasone also demonstrated good efficacy when administered in conjunction with intravitreal clindamycin. Topical steroids and cycloplegics may be administered in cases where anterior inflammation is evident. In our patient, a TMP-S regimen was selected

because of its favorable side effect profile and effectiveness in reducing recurrence rates.

In one similar case report, the patient had hypertensive non-granulomatous panuveitis, retinal vasculitis with focal retinochoroiditis with pigmented central area suggestive of ocular toxoplasmosis.[9] In another case report, the patient's anterior segment was unremarkable with funduscopic examination showing active lesions of whitish foci of chorioretinitis with surrounding oedema along the superonasal vessels and retinal vasculitis with perivascular sheathing in right eye.[10] The above two case reports were the different presentation of ocular toxoplasmosis. However, in this case, the patient was immunocompetent and had unilateral vitritis without accompanying retinochoroiditis, which was an unusual presentation. The patient tolerated the TMP-S regimen well, and the recovery period was short compared to the above case reports. This underscores the importance of considering ocular toxoplasmosis in the differential diagnosis of vitritis, even in the absence of typical retinal lesions and in non-immunosuppressed patients.

Ocular toxoplasmosis-related complications include cataracts, scleritis, retinal gliosis, cystoid macular edema, optic atrophy, secondary glaucoma, band keratopathy, vascular occlusions, tractional retinal detachment, and the formation of choroidal neovascular membranes.

The prognosis of ocular toxoplasmosis varies, with potential complications, including recurrent inflammation, retinal scarring, and vision loss. Regular follow-up is essential to monitor the treatment response and early detection of recurrence. In our case, the patient responded well to treatment, with resolution of vitritis and no recurrence during follow-up.

CONCLUSION

This case report underscores the significance of considering ocular toxoplasmosis as a potential cause of unilateral vitritis, even in immunocompetent individuals. Prompt recognition and comprehensive diagnostic work-up, including advanced imaging techniques, laboratory tests, and treatment, are crucial in averting vision loss and complications associated with the condition. In summary, this case presented an unusual presentation of ocular toxoplasmosis, characterized by unilateral dense vitritis that resolved following appropriate therapy.

DECLARATION

The authors declare no conflict of interest.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report.

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