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Ocular toxoplasmosis and retinal detachment: five case reports

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Abstract. – BACKGROUND: Ocular toxoplasmosis is a potentially blinding cause of posterior uveitis. Retinal detachment is rare complication of ocular toxoplasmosis.

AIM: To report the clinical course and prognosis of retinal breaks and detachments occurring in patients with ocular toxoplasmosis.

PATIENTS AND METHODS: This study was a retrospective, non-comparative case series of five patients with ocular toxoplasmosis who had consulted us with retinal detachment.

RESULTS: All of the participants had retinal detachment after severe and treatment resistant toxoplasmic retinochoroiditis, leaving one of them with decreased visual acuity to light perception in spite of treatment and final visual acuity was 20/100 or better in four patients.

CONCLUSIONS: The functional prognosis for the patients with retinal detachment was poor. Careful retinal examination in ocular toxoplasmosis is warranted, especially in patients with severe intraocular inflammation.

Key Words:
Ocular toxoplasmosis, Retinal detachment.

Introduction

Toxoplasmosis is the most common cause of posterior uveitis in the world. The seroprevalence of *Toxoplasma gondii* is different throughout the world. It is caused by the obligate intracellular protozoan *Toxoplasma gondii*[1] and infection is widespread in nature[2].

The cause of toxoplasmic retinochoroiditis may be a recurrence of congenital toxoplasmosis. However, acquired ocular disease is more common[3-7]. More than 82% of congenitally infected individuals not treated as infants will develop retinal lesions by the time they reach the adolescence[8]. It is estimated that about two-thirds of patients with toxoplasmosis have recurrent episodes of inflammation[3].

This disease typically affects the posterior pole of a single eye and the lesions can be solitary, multiple or satellite to a pigmented retinal scar be present[9-10]. In addition, the optic nerve head can also be involved in ocular toxoplasmosis[11].

The frequency and visual impact of retinal detachment (RD) in ocular toxoplasmosis (OT) are not well defined.In this study, clinical records of five patients with retinal detachment after ocular toxoplasmosis were reviewed.

Patients and Methods

We reviewed 1000 charts of patients followed at the Ocular Immunology and Uveitis Service of the Feiz Eye Hospital from 2003 to 2009 inclusively. 193 of patients had toxoplasmosis uveitis (82 males and 111 females). Among 193 patients 5 patients had retinal detachment. Clinical records of five patients with ocular toxoplasmosis and retinal detachment were reviewed for details of clinical presentation, ophthalmic history, complications and visual outcome at final follow up.

Case Reports

Case 1

A 29-year-old man presented to our Department with the complaint of decreased central vision in his right eye for 3 weeks and redness, photophobia and pain from 1 week ago. He reported no underlying systemic disease or recent health change or medication and no family history of unusual eye disease. On examination, his visual acuity was 20/60 in his right eye and 20/20 in his left eye. Intraocular pressure (IOP) was 10 mm Hg in his right eye and 12 mm Hg in his left eye. There was +1 reaction of anterior chamber and 2+ anterior vitreal cells (with more cells in the poste-

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rior vitreous) with some macular edema of right eye. Dilated fundoscopic examination revealed a 2 disc diameter (DD), elevated, creamy retinal lesion in the superior nasal right fundus adjacent to a darker area in right eye. His chest x-ray was normal. In addition, fluorescent treponemal antibody, complete blood cell count, C-reactive protein, erythrocyte sedimentation rate, Lyme and *Bartonella* assays were negative. The purified protein derivative (tuberculin) was negative. However, Toxoplasma IgM and IgG titers were remarkable only for an increased toxoplasma IgG titer: 3.6 (reference range, 0 to 0.92), indicating prior exposure to toxoplasmosis.

The *Toxoplasma* IgG results combined with our patient's history led us to suspect ocular toxoplasmosis. Although the diagnosis was not definitive, our suspicion was strong enough to merit immediate therapy. He was started on standard regime of toxoplasmosis (oral regimen of sulfadiazine 500 mg qid and pyrimethamine 50 mg and after 1 day, 20 mg of prednisone). In addition, he was prescribed topical glucocorticoids, folinic acid, and mydriatic agents. After six months the patient returned to our Service with decrease of visual acuity. On examination, his best corrected visual acuity was poor light perception in right eye and failed to improve with glasses or a pinhole aperture. The IOP were –12.2 and 17.3 mm of Hg in the right and left eyes respectively. Examination revealed a dense relative afferent pupillary defect (RAPD) in the right eye and posterior segment could not be visualized due to the mature cataract formation. On B-scan posterior segment was seen with total retinal detachments in a closed funnel configuration. A decision was made to attempt rehabilitation of one eye. After lensectomy and vitrectomy were performed on the right eye, uncorrectable retinal fibrosis with a total closed funnel traction retinal detachment with shortening and fibrosis were noted.

Case 2

A 16-year-old Iranian woman, presented to the Ocular Uveitis Service of the Feiz Eye Clinic with complaints of a decreased visual acuity and redness in her left eye of 5-day duration and an episode of intense pain in the left eye 2 weeks prior to the visit. The pain persisted for 1 week. She did not have any allergies. Her past medical history was unremarkable. The patient's visual acuity was 20/20 in the right eye and 20/50 in the left eye. There were keratic precipitates on the corneal endothelium, and 3 + cells in the anterior chamber of the left eye. Dilated examination of the fundus revealed 3 + cells in the vitreous and funduscopy showed a diffuse, elevated white lesion in the left retina 1 DD in size located half a DD temporal to the fovea. A focal condensation of inflammatory cells within the vitreous was seen overlying the area of active chorioretinitis. Toxoplasma IgG titer was 3.7 (reference range, 0 to 0.92), indicating prior exposure to toxoplasmosis. On the follow-up (2 weeks) visit the patient's visual acuity in the affected eye had decreased to 20/300. In spite of this therapy the patient developed retinal detachment after 2 weeks of therapy in temporal and infratemporal. A horseshoe tear was developed in the periphery of temporal retina. The retinal detachment treated with scleral buckeling and retina was attached. She had no reactivation of toxoplasmosis in the 4 years follow up.

Case 3

A 37-year-old Iranian man was referred for treatment of decreased visual acuity and 1-week duration of pain of his right eye to our Ocular Immunology and Uveitis Service. The patient did not have any significant past medical history or allergies, and was on no systemic medications. He was using topical prednisolone acetate 1% suspension hourly and scopolamine 0.25% solution twice daily in his right eye. His visual acuity was 20/400 in the right eye and 20/20 in the left eye. Slit-lamp examination revealed keratic precipitates on the corneal endothelium, 3+ cells in the anterior chamber and pigment deposits on the anterior capsule of the lens of the right eye. Dilated fundus examination showed an active toxoplasmic lesion infero-temporal to the macula, and 3+ vitreous cells. Toxoplasma IgG titer was 3.1 (reference range, 0 to 0.92), indicating prior exposure to toxoplasmosis. The patient was begun on toxoplasmosis treatment. One month after recovery the patient presented with worsening visual acuity in the affected eye (light perception with correct light projection) due to acutely symptomatic RD in his right eye. The retina was detached from 1 to 5 o'clock because a peripheral U-shaped tear at 1 o'clock. The macula was attached and corrected visual acuity was 20/25 in the right eye and 20/20 in the left. The RD was repaired on the day of presentation under general anaesthetic. Subretinal fluid was drained, cryotherapy was applied, and 20% sulfur hexafluoride gas was injected. A 276 segmental circumferential silicone explant was then sutured to the sclera in the supero-nasal quadrant. Early postop-

erative progress was satisfactory, with a flat retina and high indent. After 8 weeks the patient was presented with worsening of visual acuity in right eye. In examination a new and peripheral RD was found infero-temporally in the right eye due to peripheral U-shaped tear at the 7 o'clock position, 6 clock hours away from the sealed original tear adjusted to the old toxoplasmosis scar. A three-port pars plana vitrectomy was carried out, with internal drainage of subretinal fluid, 30% sulfur hexafluoride exchange, and indirect laser photocoagulation around the tear. The retina has since remained attached in 3 years follow up and the ocular toxoplasmosis does not recurrent.

Case 4

A 27-year-old Iranian man was referred for treatment of decreased visual acuity of his left eye of 1-week duration to Ocular Immunology and Uveitis Service. His visual acuity was 20/20 in the right eye and 20/50 in the left eye. The anterior segments were normal and the intraocular pressures were 16 mm Hg right eye, 14 mm Hg left eye. Funduscopy showed a diffuse, elevated white lesion in the left retina 2-3 disc diameter in size located in peripheral of disc. The vitreous of his left eye had 3+ anterior vitreal cells (with more cells in the posterior vitreous). Toxoplasma IgG titer was 3.00 (reference range, 0 to 0.92), indicating prior exposure to toxoplasmosis. The patient was begun on standard regime of toxoplasmosis treatment. After 3 weeks the patient presented with worsening visual acuity in the affected eye (light perception with correct light projection) due to acutely symptomatic RD in his left eye due to U-shaped tear. The RD was repaired with scleral buckeling and cryopexy. The retina has since remained attached in 3 years follow up with +1 to +2 vitritis with the active scar.

Case 5

A 26-year-old Iranian man was referred to our Department for an acute decrease in his right-eye vision, persisting for 4 weeks, after several days of metamorphopsia. His best-corrected visual acuity was 20/400 right eye and 20/20 in left eye. Slit-lamp examination revealed motton fat keratic precipitates on the corneal endothelium, 3+ cells in the anterior chamber and 330 degree of posterior synechiae and pigment deposits on the anterior capsule of the lens of the right eye. The posterior segment could not be visualized. The left fundus was normal. The patient was begun on oral and topical steroid and topical and mydriatic for one week. The patient was not respond to our treatment. On B-scan posterior segment was seen with retinal detachment. We considered deep vitrectomy. A diffuse, elevated white lesion in the right retina 4 disc diameter in size located superior to the fovea with tractional retinal detachment was seen. There was an area of associated perivascular sheathing. An active right retinochoroiditis with associated retinal vasculitis was diagnosed. The patient underwent right deep vitrectomy with silicone oil tamponade and 360° endophotocoagulation.

The patient had undergone an evaluation that disclosed an antitoxoplasmic IgG antibody level of 2.2 ELISA units (normal range 0-0.9).

Results

We have described five patients with retinal detachment and ocular toxoplasmosis. The clinical features are summarized in Table I. All patients were Iranian. All participants had a severe inflammation. One patient had a prolonged course. All patients had permanent decrease in their best-corrected visual acuity. One patient had a decrease in visual acuity to light perception in the affected eye due to uncorrectable retinal fibrosis with a total closed funnel traction retinal detachment with shortening and fibrosis. Interestingly, in one case the retina has since remained attached in 3 years follow up with +1 to +2 vitritis with the active scar.

Discussion

In this study, we identified 5 patients for retinal detachment among 193 patients with ocular toxoplasmosis. The occurrence of retinal detachment (RD) and retinal breaks (RBs) was significantly associated with severe attacks of ocular toxoplasmosis (OT) and preceding (diagnostic) vitrectomy.

Rhegmatogenous retinal detachment occurs when retinal break (RB) allows liquid vitreous access to subretinal space. Incidence is about 1 in 10,000/year; retinal breaks can be found in 97% of cases. Most tears are located superiorly between 10 and 2 o'clock positions. Risk factors are age, history of retinal detachment in the fellow eye (15%), high myopia/axial length (7%), family history, lattice degeneration, trauma, cataract surgery, diabetes, and Nd:YAG laser posterior capsulotomy[12]. The frequency and visu-

Table I. Brief summary of the course of ocular toxoplasmosis in five patients.

Characteristic	Case 1	Case 2	Case 3	Case 4	Case 5
Age (years)	29	16	37	27	26
Sex	Male	Female	Male	Male	Male
Affected eye/area of involvement	Right eye/macula-threatening lesion along the superonasal arcade	Left eye/ supero-temporal to the macula	Right eye/ infro-temporal to the macula	Left eye/ peripheral to disc	Right eye/macula-threatening lesion superior to fovea
Visual acuity at flare-up	20/60	20/50	20/400	20/50	20/400
Duration of inflammation to retinal detachment	6 months	2 weeks	1 month	3 weeks	4 weeks
Final visual acuity	Light perception	20/40	20/100	20/30	20/100
Type of RD	Unknown	Rhegmatogenous	Rhegmatogenous	Rhegmatogenous	Tractional
Location of retinal break	Tractional	Temporal upper quadrant	Upper nasal quadrant	Temporal quadrant	Tractional
Severe vitreitis during OT attack preceding RD/RB	Yes	Yes	Yes	Yes	Yes
Systemic treatment of OT attack Preceding RD/RB	Antiparasitics with corticosteroids	Antiparasitics with corticosteroids	Antiparasitics with corticosteroids	Antiparasitics with corticosteroids	None
Treatment RD/RB	Vitrectomy 1 lensectomy	Scleral buckling	Cryopexia, scleral buckling	Scleral buckling	Vitrectomy 1 silicone oil
Complications	Atrophic eye	None	Recurrence of RD	None	None

RB = retinal break; RD = retinal detachment; OT = ocular toxoplasmosis.

al impact of RD in OT are not well defined. Organization of vitreous infiltrates may lead to tractional detachment. A rhegmatogenous detachment can start from a retinal hole at the site of a former toxoplasmosis scar where the adhesion to the pigment epithelium and sclera was strongest.

Bodanowitz et al[13] reported retinal tear in retinitis associated with toxoplasmosis. Retinal tears or rhegmatogenous retinal detachment are rare complications of toxoplasmic retinochoroiditis. However, a tear may occur due to vitreoretinal traction following post inflammatory structural alteration of the vitreous.

It is noteworthy that the attacks of active OT preceding the manifestation of RD or RB were in most patients, characterized by severe intraocular inflammation. Fulminant vitritis preceding RD in OT was also described in previous case reports[14-15]. Lafaul et al[16] suggested that the presence of peripheral hypertrophy of the chorioretinal scar was a sign of congenital toxoplasmosis with choroidal vascularization. Proliferate protrusion of the sensory retinal layer and a secondary scar caused by congenital toxoplasmosis might be retinal tear in active ocular toxoplasmosis initiated with severe intraocular inflammation.

Such a severe inflammation of the vitreous may lead to vitreous traction causing not only the tractional but also a rhegmatogenous retinal detachment. Therefore, one would expect that the vitrectomy may protect severely inflamed eyes against the development of RD. These interventions may also have contributed to the later development of a detachment. Although retinal detachment can occur as a complication of pars plana vitrectomy, it is to be expected that in most cases this may occur shortly after the vitrectomy has been performed.

Ocular toxoplasmosis therapy includes antimicrobial drugs with or without the presence of corticosteroids. Several drugs have been proposed including pyrimethamine, sulfadiazine, spiramycin, clindamycin and trimethoprim-sulfamethoxazol[17,18].

The use of corticosteroids in OT is controversial; these drugs are mainly used to alleviate severe inflammatory reaction. Therefore, corticosteroids may influence the development of vitreoretinal traction and preretinal membranes.

Four patients with OT who had RD or RB used systemic corticosteroids with antiparasitics agents for the treatment of OT before the onset of RD. Corticosteroid monotherapy may induce extremely severe inflammation resulting in fulminant ocular disease; its use is, therefore, not recommended[19].

It is feasible that it is not the corticosteroids themselves but the severe retinitis that may be related to the development of RD or RB. Incidence of rhegmatogenous RD increases above the age of 50 years. However, patients with myopia over 8 Diopters tend to develop RDs when they are young[20].

The mean age of our patients with RD and RBs was 32 years, but none of these patients had myopia exceeding 4 Diopters.

Most breaks in our patients were located in the superior quadrants. This is similar to the cases of RBs in patients with OT reported earlier and to the location of retinal breaks in retinal detachment in a general population[14,21].

However, Byer[18] found 61% of asymptomatic retinal breaks to be located in the inferior quadrants. Visual prognosis of RD complicating OT was poor, because half of the patients who experienced RD became legally blind (visual acuity ≤20/200). The patients with attached RBs retained their visual potential. This may be because these RBs were diagnosed during ophthalmologic examinations needed for an active attack of OT and were, therefore, treated early. It was reported that in noninflamed eyes, asymptomatic breaks did not proceed toward retinal detachment[22]. We believe that the laser treatment of (asymptomatic) attached RB in an eye with recurrent intraocular inflammation is justified. First, there is an additional risk factor consisting of vitreous traction, and second, the visual prognosis after surgical repair for RD in OT was poor.

We have not an absolute explanation for the high frequency of RD after severe attacks of OT. We believe that this high frequency and poor visual prognosis warrant the careful retinal examination during active OT, especially in patients with additional risk factors for RD such as myopia and severe intraocular inflammation.

In summary, the timing of toxoplasma infection leading to ocular disease is rarely known. However, current evidence suggests that many more people are affected by postnatal than by prenatal toxoplasmosis. This has major public health implications. Considerable expertise and expense is concentrated on screening and health information to reduce the risks of toxoplasmosis due to prenatally acquired infection, principally to reduce the risks of ocular morbidity in the long term. Primary preventive strategies should include children and adults at risk of ocular disease as a result of postnatal infection and should not be confined to pregnant women.

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