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Case Report

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Late Reactivation of Scarred Choroidal Neovascular Membrane Secondary to Ocular Toxoplasmosis after Cataract Surgery

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Abstract

Ocular toxoplasmosis is a parasitic disease characterized by recurrent episodes of active chorioretinal, vitreous and anterior segment inflammation. It could leave chorioretinal scar. Reactivation of ocular toxoplasmosis or development of choroidal neovascularisation (CNV) could be triggered by various endogenous and exogenous factors including cataract surgery, both reactivation of the scar and CNV may cause visual loss. Intravitreal anti-vascular endothelial growth factor (VEGF) agent could be a treatment option for CNV, although there were other different treatment options for CNV including laser photocoagulation, photodynamic therapy and surgical intervention. These are rarely used nowadays. We report herewith, a case of late recurrence of CNV secondary to ocular toxoplasmosis after cataract surgery. The chorioretinal scar has been stable for 25 years prior to cataract surgery. The patient was treated with a single injection of intravitreal bevacizumab (IVB). He demonstrated progressive regression of CNV and complete resolution of CNV with no recurrence of CNV for three years. This case report highlights few important clinical points; firstly the patient should be warned about the recurrence of CNV after cataract surgery with guarded visual prognosis in spite of stability for 25 years, and secondly long term follow up is indicated prior to discharging the patient, in addition to the dramatic response to single injection of IVB.

Keywords: *Choroidal Neovascular Membrane; Ocular Toxoplasmosis; Phacoemulsification*

Introduction

Choroidal neovascularization reported to be associated with various ocular diseases [1]. Ocular toxoplasmosis is a parasitic ocular disease characterized by recurrent episodes of active retinal inflammation. Also, it may lead to CNV that may contribute to severe visual loss [2].

Many studies showed that cataract surgery might induce exacerbations in different types of intraocular inflammations [3]. Bosch-Driessen*et al* reported 36% reactivations of ocular toxoplasmosis following cataract extraction in 36% of their series [2]. However others reported that reactivation of infection is controversial following intraocular surgery [4].

There are different modalities for treating CNV associated with ocular toxoplasmosis. Intravitreal anti-VEGF therapy is a treatment that could provide a favourable visual outcome [5].

We report a case with late reactivation of CNV of historically stable ocular toxoplasmosis scar for 25 years following uncomplicated cataract surgery. The CNV showed regression and visual acuity was stable after a single dose of intravitreal injection of bevacizumab.

Case Report

A 69-year-old gentleman was complaining of gradual visual deterioration in his left eye for the last 6 months. The patient was taking medications to control his hypertension and hypercholesterolaemia. He was mildly myopic in both eyes with a history of a left longstanding chorioretinal scar secondary to ocular toxoplasmosis. The scar has been stable for the last 25 years. His best-corrected visual acuity (BCVA), using Snellen chart, was 6/7.5 in the right and 6/9 in the left. His anterior segment examination showed bilateral nuclear sclerotic cataract left more than the right with no evidence of inflammation. Dilated fundus examination was unremarkable in the right, however it showed juxtafoveal chorioretinal scar in his left eye.

The patient was very keen to go ahead with left cataract surgery. This was performed without complications. Two weeks post-operatively, his left BCVA was 6/5. The patient was very happy with the outcome of surgery and was discharged. Eleven months later, he was referred again due to reduced left BCVA from 6/5 to 3/60. Ocular examination showed signs suggestive of choroidal neovascular membrane in his left chorioretinal scar (Figure 1a) however, there was neither evidence of anterior chamber nor vitreous inflammation. Causes of uveitis were excluded with clinical examination and investigation.

Fundus fluorescein angiography confirmed active CNV (Figure1b). Optical coherence tomography (Figure1c) showed sub-foveal scar with intraretinal fluid. The patient was treated with a single injection of IVB [1.25 mg/0.05 ml prepared from a vial commercially available for intravitreal use (Avastin)].

The treatment led to a regression of the CNV with improvement of his visual acuity to 6/24. No further therapy was needed during three years of follow-up. Fortunately, there was no recurrence of the CNV in his left eye (Figure 1d).

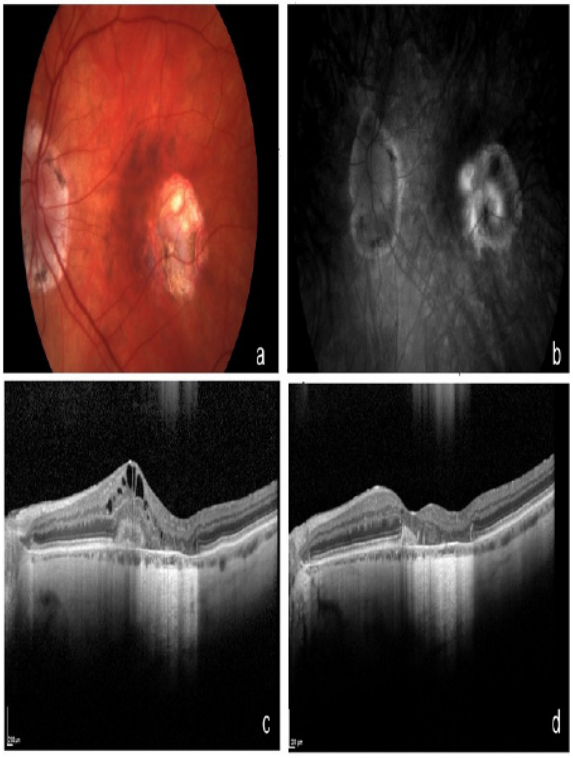

Figure 1: (a) Colour fundus photograph of the left eye shows signs suggestive of choroidal neovascular membrane (CNV)
(b) Fundus fluorescein angiography confirmed active CNV
(c) Optical coherence tomography (OCT) revealed sub-foveal scar with intraretinal fluid
(d) OCT showed regressed CNV 11 months after treatment

Discussion

In this report, we discuss a case of late reactivation of CNV secondary to toxoplasmosis after cataract surgery that responded well to single injection of IVB. Reactivation of CNV has been mentioned in previous studies as a late complication of ocular toxoplasmosis, mostly occurring in healed, inactive lesions and may threaten vision, especially in young patients [6,7]. Reported one patient developed CNV at 3 months postvitrectomy and epiretinal membrane removal. In our case, CNV appeared 11 months after surgical intervention.

Choroidal neovascular membrane may mimic active toxoplasmic retinochoroiditis and vice-versa. A thorough ophthalmic, serological, and immunological examination (in ocular fluids) may help in the differential diagnosis allowing for proper therapeutic decision-making [8]. In our patient, there is no evidence of reactivation of inflammation.

Many authors tried to postulate the reason of repeated re-activations in ocular toxoplasmosis. These included host-related factors and parasite-related factors [9]. In addition, exogenous factors such as trauma and stress have been also reported [10]. It was suggested that mechanical trauma might reactivate ocular toxoplasmosis. However, no adequate pathogenic mechanism was reported. It was also suggested that toxoplasmic cyst wall would lose its elasticity by aging and might fracture as a result of blunt trauma. This could be a responsible factor of recurrences of ocular toxoplasmosis following cataract surgery [3]. In this case, it could be explained that surgical trauma could

play a role in the development of CNV [11]. Bosch-Driessen*et al* suggested that the energy given to the eye during phacoemulsification and the psychological stress of the surgery might contribute to the development of recurrences [2]. Other authors postulated that ultrasound waves in the eye might stimulate the release of free radicals that can act as inflammatory mediators or otherwise that the cysts might be sensitive to the sonic waves induced [12]. However, we cannot rectify the causes of late recurrence in our case.

Multiple treatment modalities have been described to treat CNV secondary to ocular toxoplasmosis, including laser photocoagulation, photodynamic therapy and surgical intervention [3]. In this case we felt that we need a rescue treatment to halt its progression. Recently, few studies showed the efficacy of antivascular endothelial growth factor (anti-VEGF) therapy in treating this sight threatening complication [3]. Benevento., *et al*. suggested that the combination therapy with intravitreal ranibizumab and anti-parasitic agents would give efficient results in the treatment of CNV secondary to ocular toxoplasmosis [13]. Nevertheless, in this case there is no evidence of activity of ocular infection that needs antiparasitic treatment pre or post operatively. Rishi., *et al*. reported that IVB augmented with photodynamic therapy was also effective [3]. In Bosch-Driessen., *et al*. the authors suggested that prophylactic treatment with antiparasitic drugs during and after the cataract surgery might be beneficial for patients at risk of visual loss [2]. On the other hand, in this patient, there was no active vitreous reaction suggesting active retinochoroiditis. Therefore, we did not add anti-parasitic treatment. Other studies suggested the use of concomitant antitoxoplasma as a treatment prophylaxis [13]. In this case, a single IVB was effective in regressing the CNV for 3 years. It is difficult to predict future reactivation of CNV. Overall the results suggested a possible beneficial effect of anti-VEGF on CNV, but the long-term effect of the treatment is still uncertain.

This case report highlights few important clinical points; firstly it is worthwhile warning the patient about the recurrence of CNV with guarded visual prognosis in spite of stability for 25 years, and secondly long term follow up is indicated prior to discharging the patient. We report herewith, a case of late reactivation of CNV in a patient with stable toxoplasmosis scar for 25 years. It also showed regression after treatment with bevacizumab and improvement of his visual acuity. No further therapy was needed for 3 years follow-up. Fortunately, there was no recurrence of the CNV in his eye.

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