D Shukla and S Chakraborty

Retina-Vitreous Service, Aravind Eye Hospital and Postgraduate Institute of Ophthalmology, Madurai, Tamil Nadu, India
E-mail: daksh66@gmail.com

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Sir,

Simple method to reduce iatrogenic retinal trauma during vitreous surgery

Posterior iatrogenic retinal trauma is a known complication of vitreous surgery.[1] We have had two cases where the endoillumination pipe has caused direct retinal trauma during epiretinal or internal-limiting membrane peel. This is a delicate procedure that demands intense concentration, often using a viewing lens that greatly restricts the visible field.

To reduce the likelihood of trauma during this stage of the procedure, we employed the simple use of a length of butterfly tubing (Figures 1 and 2) threaded along the illumination pipe to limit the depth to which the illumination instrument can be inserted into the eye. This butterfly tubing can be adjusted in length as required. This has also been employed during a complex four-port diabetic delamination surgery, where an assistant holds the light pipe. The technique should be particularly useful for trainee retinal surgeons.

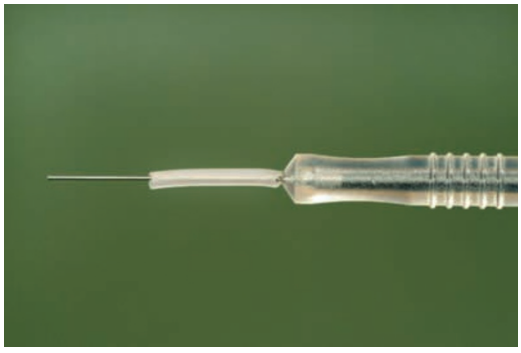

Figure 1 Butterfly tubing cut to desired length and threaded along endoillumination pipe.

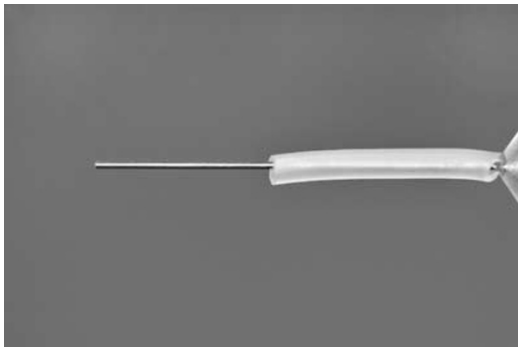

Figure 2 Butterfly tubing cut to desired length and threaded along endoillumination pipe.

Thankfully, we have not had any similar iatrogenic breaks after employing this device.

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Reference

1 Michels RG, Wilkinson CP, Rice TA. *Retinal Detachment*. C V Mosby: St Louis, 1990, p869.

C Goldsmith[1], D Gupta[1] and RL Burton[2]

[1]James Paget University Hospital, Gorleston, UK
[2]Norfolk and Norwich University Hospital, Norwich, UK
E-mail: craig.goldsmith@jpaget.nhs.uk

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Sir,

Intravitreal triamcinolone acetonide as an adjunct in the treatment of severe ocular toxoplasmosis

Treatment of ocular toxoplasmosis is highly controversial. Results of such treatment depend on host factors, such as age and immune status, as well as on parasite factors.[1] The use of corticosteroids is even more debatable. Although most uveitis specialists agree that corticosteroid therapy without the concurrent use of antimicrobial agents can lead to severe ocular tissue destruction, patients who did well treated with corticosteroids alone have been seen.[1]

Aggressive cases, such as the one reported by Backhouse *et al*, have also been reported. It is important to note, however, that clinical deterioration did not occur immediately after the introduction of oral or intravitreal corticosteroids. On the contrary, in these two circumstances it appears that some improvement was initially observed. Of note, the antimicrobial agent was introduced 1 month following intravitreal triamcinolone acetonide. Taking into account that clinical picture worsening occurred only 2 weeks thereafter (6 weeks following intraocular injection), the temporal relationship strongly suggests that the intravitreal triamcinolone acetonide injection was not directly related to the outcome.

In our opinion, intravitreal corticosteroids should be used with caution in active ocular toxoplasmosis. Clinicians should avoid their use on recalcitrant, rapid worsening cases or those with questionable diagnosis. Intravitreal steroids without appropriate antimicrobial drugs should also be discouraged. On the other hand, there may be a role for them in patients with relatively controlled infection, as those we reported previously on this journal.[2]

References

1 Holland GN LX Edward Jackson Lecture Ocular toxoplasmosis: a global reassessment Part II: disease

manifestations and management. *Am J Ophthalmol* 2004; **137**: 1–17.
2 Aggio FB, Muccioli C, Belfort Jr R. Intravitreal triamcinolone acetonide as an adjunct in the treatment of severe ocular toxoplasmosis. *Eye* 2006; **20**: 1080–1082.

FB Aggio, C Muccioli and R Belfort Jr

Department of Ophthalmology, The Vision Institute, Federal University of São Paulo, São Paulo, Brazil
E-mail: aggio@oftalmo.epm.br

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Sir,

Intravitreal triamcinolone acetonide as an adjunct in the treatment of severe ocular toxoplasmosis

We read with interest the paper by Aggio *et al*[1] regarding the possible beneficial use of intravitreal triamcinolone acetonide (IVTA) injection for control of inflammation in severe ocular toxoplasmosis. We would like to add a word of caution to this approach.

Case report

A 49-year-old Caucasian male presented with a 5-day history of reducing acuity with metamorphopsia in the left eye. He was otherwise well. Snellen acuity was recorded as 6/5 OD and CF OS. A yellow macular lesion was seen surrounded by subretinal fluid. There was a trace of vitreous activity and mild peripheral retinal phlebitis. A fundus fluorescein angiogram revealed some early hyperfluorescence with mild late leakage. Blood investigations of FBC, CRP, U&Es, serum ACE, rheumatoid factor, ANA, ANCA, VDRL, serum electrophoresis as well as a Mantoux test and CXR were all normal. A toxoplasma titre remained outstanding. Three weeks following the first symptoms, oral prednisolone was started at 75 mg and rapidly reduced to 25 mg over 2 weeks. Partial resolution of the lesion was observed. An urgent referral was then made to the regional uveitis clinic.

In the uveitis clinic, 5 weeks following initial symptoms, vision had slightly improved to 6/60. An optical coherence topography scan (OCT) showed early foveal scar formation (Figure 1). Peripheral retinal phlebitis was confirmed. The working diagnosis was idiopathic granulomatous panuveitis. A decision was made to give 4 mg intravitreal triamcinolone to give the best chance of visual recovery. One month later azithromycin 500 mg was commenced as the toxoplasma dye test was positive (1000 IU) as was toxoplasma IgM. Two weeks later there was a dramatic worsening of symptoms. A large white lesion occupied the area within the macula arcades with overlying focal arteritis, multiple small peripheral lesions, and increased vitreous activity (Figure 2). Although the clinical findings were in keeping with fulminant ocular toxoplasmosis, a sample of anterior chamber fluid was sent for urgent polymerase chain reaction for CMV, HSV, VZV, EBV, tuberculosis and toxoplasmosis. All PCR results were negative except for a positive toxoplasmosis result.

Despite repeated courses of azithromycin and clindamycin, the inflammation continued to get worse and obscured fundal examination. An MRI scan of the orbits and brain showed involvement of the affected eye only. After 6 weeks, a vitrectomy was undertaken to remove the steroid and allow visualisation of the fundus. Unfortunately areas of the retina had become necrotic, with localised retinal detachment and so the eye was indented with a plomb, lasered, and filled with silicone oil. The retina remains flat but the left vision has deteriorated to hand movements secondary to macula subretinal fibrosis.

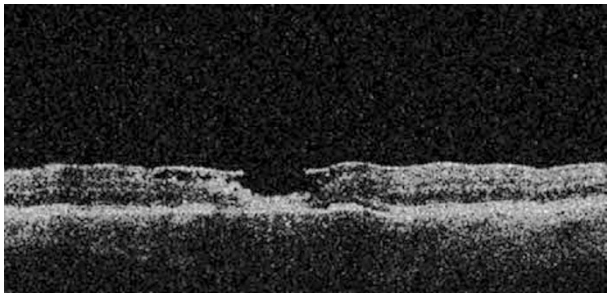

Figure 1 OCT scan of left macula showing a disrupted fovea.

Comments

Cases of fulminant, uncontrolled ocular toxoplasmosis as a result of a depot steroid injection have been reported, some with concurrent antiparasitic cover.[2–5] More recently a poor outcome was reported in those patients who received corticosteroids alone.[6,7] Following a survey, it was found that 9% of clinicians who responded had used periocular corticosteroids in combination with oral

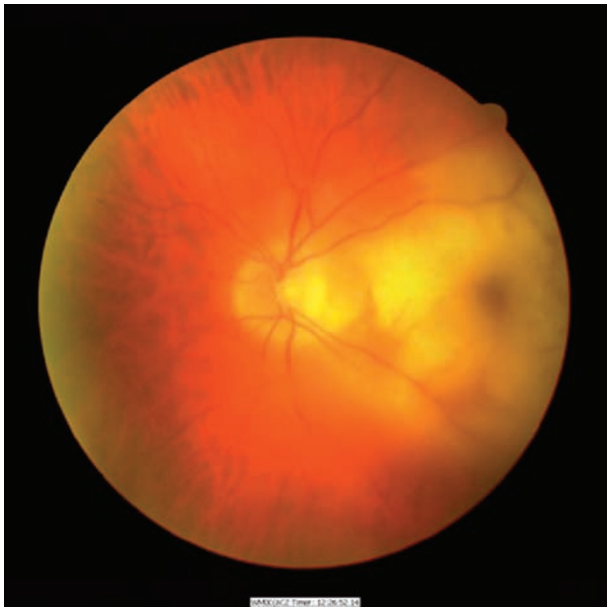

Figure 2 Fundus photograph of left macula with an enlarging area of focal and diffuse arteritis.