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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.

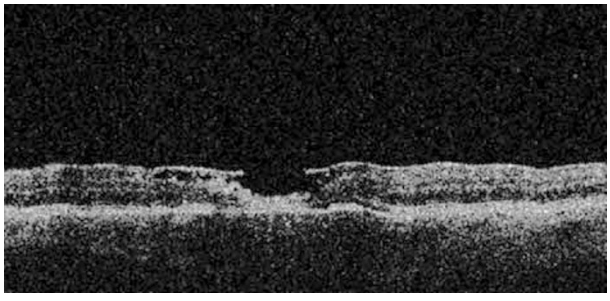

Figure 1 OCT scan of left macula showing a disrupted fovea.

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

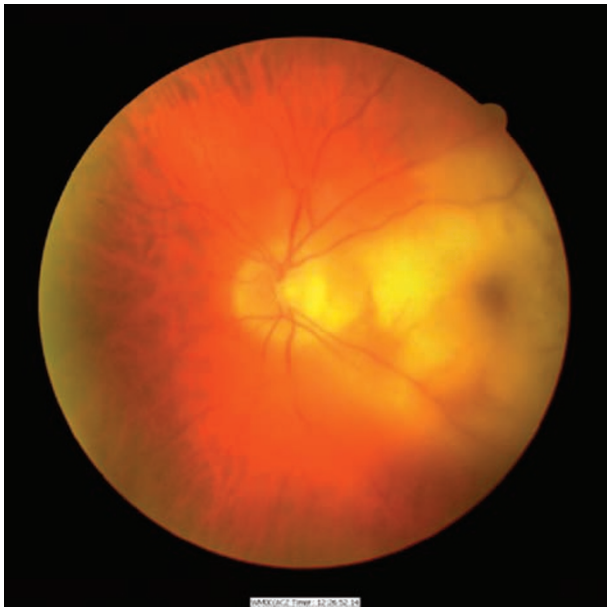

Figure 2 Fundus photograph of left macula with an enlarging area of focal and diffuse arteritis.

anti-toxoplasma therapy. In four of the six cases, improvement was noted after steroid injection and in two cases deterioration was noted.[8]

Our patient was given oral corticosteroid initially with clinical improvement. A decision for 4 mg IVTA was made to attempt to reduce the inflammatory response and reduce macula damage. This was covered by anti-toxoplasma medication following a positive serology 1 month later. There was further clinical improvement followed by a fulminant, rapid reaction leading to a vitrectomy with silicone oil to help preserve the eye cosmetically rather than for any visual recovery. In the past, histological examination has been undertaken in toxoplasmosis-affected eyes treated with corticosteroids alone. Necrosis was seen in areas where there were numerous free parasites without an inflammatory reaction.[2,5]

There is clearly a role for corticosteroids in the treatment of ocular toxoplasmosis under the cover of antiparasitic drugs, especially in the elderly or those with evidence of a primary infection.[9] However, we feel that the administration of low dose IVTA in our patient was detrimental to the control of the ocular toxoplasmosis and urge extreme caution in taking this approach.

Acknowledgements

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Sir,

Intraoperative floppy-iris syndrome associated with chronic use of chlorpromazine

I would like to comment on the above correspondence by Ünal *et al*.[1] The authors conclude that 'discontinuation of chlorpromazine might be a wise course of action before cataract surgery to avoid the possibility of IFIS'. I feel that such advice should not be given on the basis of a single anecdotal report.

Intraoperative floppy iris syndrome (IFIS) is characterised by subnormal preoperative pupil dilation, repeated intraoperative prolapse of a billowing, floppy iris, and progressive intraoperative miosis.[2] It was originally suggested that this was specific to patients on tamsulosin and not to other alpha blockers, however this and other reported cases suggest that IFIS may occur with all commercially available alpha-blockers (alfuzosin, doxazosin, tamsulosin and terazosin).[3] Osher[4] suggests that IFIS is a form of iris dystonia which can result from many different causes, one of which is flomax (tamsulosin), and can occur in both sexes with a highly variable degree of susceptibility and severity. IFIS may even occur without any identifiable causative factor.

Here, many questions spring to mind: why did this patient present with cataract at age 48. Had there been other significant disease, trauma or treatment? Was this a first or second eye operation? What was the experience with the other eye? Why did the patient require a general anaesthetic? Were there other factors expected to cause difficulties? What other medications had the patient used? Having been diagnosed with schizophrenia over 29 years ago the patient must have been exposed to various medications linkable to IFIS.

In conclusion I do not think it is wise to advocate cessation of chlorpromazine before cataract surgery. There is no mention of the risks attached to stopping a medication especially when it is used in controlling mental illness beyond most ophthalmologists area of expertise. Chlorpromazine is not an uncommon drug and has been in use for many years without prior report of this effect.

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