Letter to the Editor

Bilateral anterior uveitis associated with travoprost

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Editor,

P rostaglandin analogues are known to be closely related to anterior uveitis. The association of uveitis and latanoprost has been confirmed in previous studies (Fechtner et al. 1998; Smith et al. 1999, Warwar et al. 1998). Bimatoprost is also reported to have caused anterior uveitis (Packer et al. 2003; Parentin 2003).

Travoprost has twice been reported to be associated with uveitis (Faulkner & Burk 2003; Kumarasamy & Desai 2004). We report a patient who developed bilateral anterior uveitis as a possible complication of travoprost treatment for glaucoma.

A 76-year-old woman had a history of asthma and hypertension. She had also had capsular glaucoma for several years and was routinely followed up in our clinic. She had undergone bilateral laser trabeculoplasty in December 2001 and unilateral laser trabeculoplasty in the right eye in January 2002. She had no history of anterior uveitis.

At a regular follow-up visit in September 2004, her best corrected visual acuity (VA) was 20/30 RE and 15/20 LE. Her intraocular pressure (IOP) was 30 mmHg RE and 26 mmHg LE. At this point, she had used timolol 5 mg/ml (Blocanol; MSD, Mirabel, France) for over a year and brinzolamide 10 mg/ml (Azopt; Alcon, Buurs, Belgium) twice daily for 1 month. During the visit travoprost (Travatan; Alcon) once daily bilaterally was added to her glaucoma medication.

Five days after the initiation of travoprost treatment, the patient experienced discomfort, redness and lacrimation in her right eye. Two days after the onset of symptoms



Fig. 1. Colour photograph of the right eye showing conjunctival injection and posterior synechiae.

she presented at our clinic and was examined. Her best corrected VA was now 20/80 RE and 15/20 LE. She had photophobia and conjunctival injection in the right eye. The cornea was intact. Moderate cells and flare were noted in the anterior chamber. Posterior synechiae from iris to lens were present (Fig. 1). Intraocular pressure was 32 mmHg. Fundus examination revealed no sign of macular oedema.

The patient had milder conjunctival injection and no photophobia in the left eye. Few cells and mild flare were noted in the anterior chamber. Intraocular pressure was 20 mmHg. Again, there was no sign of macular oedema on fundus examination.

Travoprost was discontinued immediately. As a treatment for uveitis, the patient was started on dexametha sone (Oftan Dexa; Santen, Tampere, Finland) 1 mg/ml drops every hour and dexamethasone ointment at night. Scopolaminehydrobromide (Oftan Scopolamin; Santen) 2.5 mg/ml was begun in both eyes for mydriasis.

The treatment for uveitis continued for 8 weeks in the right eye and 6 weeks in the left eye. Oral prednisolone (Prednisolon; Leiras, Turku, Finland) 40 mg daily was begun and tapered down over a week. The synechiae in the right eye were resolved with mydriatic drops.

At the last follow-up visit for uveitis, the patient's best corrected VA had increased to 20/30 RE and 20/20 LE. Her IOP was 24 mmHg in both eyes. She continued glaucoma treatment of timolol twice daily and brinzolamide twice daily in both eyes. To the best of our knowledge, only two case reports concerning the association of anterior uveitis and travoprost have been published. Faulkner & Burk (2003) reported a case of bilateral uveitis and corneal oedema with diffuse corneal folds. Kumarasamy & Desai (2004) described a case of unilateral anterior uveitis during travoprost medication.

Ours is the first reported case of bilateral anterior uveitis with synechiae formation associated with travoprost. Further studies with bigger patient populations and longer follow-up periods should be conducted to establish the incidence of anterior uveitis in travoprost-treated glaucoma patients.

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