small graft may have been repopulated by endothelial cells from the periphery of the host cornea of a young recipient. Experimental studies on rabbits have revealed endothelial cell migration across the host–graft junction.[10,11] In a study on rabbits using sex chromatin marker, spread of corneal endothelial cells on to the recipient side with absent endothelial cells has been demonstrated.[11] In a recent experimental study on rabbits, migration of host endothelial cells onto the endothelial-deficient graft was observed after 4 days.[12]

This concept of endothelial cell migration has also been proposed by Ohguro *et al.*,[13] who observed that in transplanted corneas the peripheral recipient endothelial cells may migrate and primarily contribute to resurfacing the endothelial defects caused by cataract surgery. They concluded that cataract surgery in eyes with transplanted corneas is safe in cases where the peripheral recipient endothelium has sufficient cell density and normal morphology. Several authors have reported lower success rates in achieving clear grafts in bullous keratoplasty in comparison with eyes in which the recipient cornea has healthy endothelium, viz., corneas with keratoconus, herpetic keratitis, localised leucomatous opacities, etc.[14,15] Rao and Aquavella[16] have observed that in such eyes the peripheral recipient endothelium has a higher cell density and more normal morphology than donor endothelium. Matsubara *et al.*[15] reported through a life-table analysis that the success rate of penetrating grafts in bulbous keratopathy decreased to about 30% over an observation period of 4 years, whereas the rate in keratoconus was maintained at 97.5%. They showed that graft endothelial cells continued to enlarge in bullous keratopathy much more rapidly than in keratoconus.

A young recipient, such as our patient, with a small localised lesion, can also be expected to have a higher endothelial cell density in the rest of the cornea, and these peripheral endothelial cells could have migrated towards and repopulated the grafted cornea resulting in it remaining clear.

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Sir,

Bilateral poliosis and granulomatous anterior uveitis associated with latanoprost use and apparent hypotrichosis on its withdrawal

Latanoprost is an effective long-acting prostaglandin $F_{2\alpha}$ analogue which lowers intraocular pressure by increasing uveoscleral outflow. Since its widespread clinical use in the medical treatment of glaucoma several side effects have surfaced including skin rash,[1] hypertrichosis, hyperpigmentation of eyelashes,[2,3] corneal psudodendrites,[3] iris hyperpigmentation, anterior uveitis,[5] choroidal effusions and cystoid macular oedema.[6] We report a case of bilateral poliosis and granulomatous anterior uveitis during treatment with latanoprost and apparent hypotrichosis on its withdrawal.

Case report

A 77-year-old woman was diagnosed with primary open angle glaucoma in October 1995. Her vision was 6/9 in each eye. The intraocular pressures (IOPs) were 28 mmHg in the right eye and 26 mmHg in the left. The right optic disc and visual fields were essentially normal but the left eye had a cupped disc and a dense superior visual field defect.

Initially the IOPs were satisfactorily lowered by g. timolol 0.25% b.d., but 8 months later g. dorzolamide also was necessary. After several months the patient developed progressive lower eyelid skin erythema, conjunctival hyperaemia and thickening and eventually medial ectropion. The changes did not resolve completely on stopping the dorzolamide drops and surgery was necessary to reposition the puncta. The patient was then treated with g. alphagan b.d. in addition to g. timolol 0.25% b.d. However, her eyes became red, watery and itchy. Therefore the alphagan drops were stopped and instead latanoprost drops were commenced once at night. Two months later the patient complained of discomfort, watering and blurring of vision in both eyes. Her visual acuities were 6/24 on the right and 6/18 on the left. The IOPs were 21 mmHg in the right and 24 mmHg in the left. The conjunctiva was hyperaemic and chemosed with papillary hypertrophy. Mutton-fat keratic precipitates were diffusely scattered on the corneal endothelium, the anterior chamber showed 2+ cells and there were early posterior synechiae. The vitreous was quiet and there were no chorioretinal abnormalities. Additionally it was noted that the patient had bilateral segmental poliosis, particularly on the right lower lid (Fig. 1). There was no history of uveitis, no previous intraocular surgery or trauma and no medical condition associated with uveitis.

The latanoprost drops were discontinued immediately. No topical steroids were prescribed. Within 2 weeks the uveitis had completely resolved; the poliosis began to improve and 10 months later was barely noticeable. However, hypotrichosis was evident at 1 month and progressed over the following 10 months (Fig. 2). Meanwhile, the patient continued to experience watery eyes using g. timolol 0.25% twice daily, and this, therefore, was stopped. In view of the glaucomatous changes in the left eye a left trabeculectomy with 5-fluorouracil was performed and no further treatment has been necessary. The IOP in the right eye has been successfully controlled, and the eye has remained symptom-free on unpreserved pilocarpine drops.

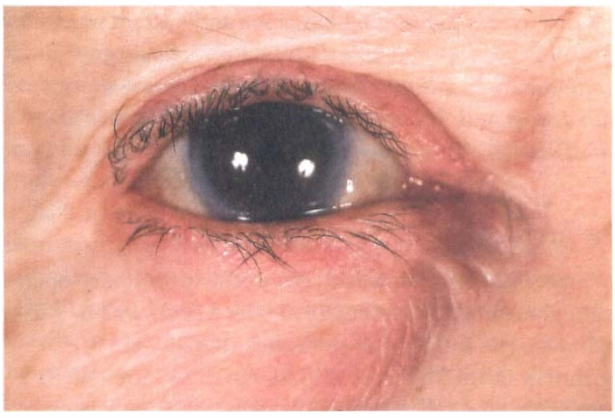

Fig. 1. *Latanoprost-treated right eye. Note the poliosis on the lower eyelid.*

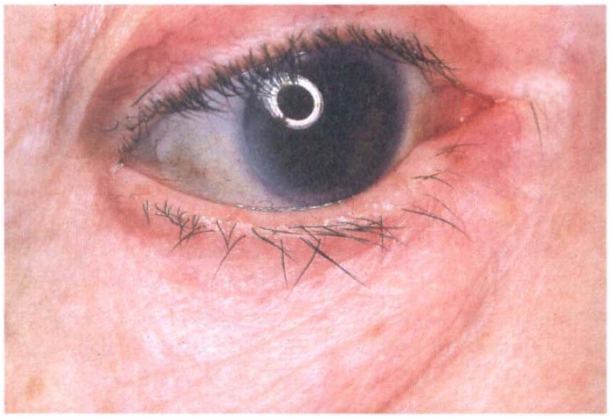

Fig. 2. *Right eye showing hypotrichosis and barely noticeable poliosis 10 months following discontinuation of latanoprost.*

Comment

It is generally accepted that prostaglandins and their analogues play a perpetuating role in ocular inflammation. Uveitis as an adverse affect of latanoprost has been reported many times in the literature.[5,7] Reports by Watson and Stjernschantz[8] and Camras[9] contradict this view, and the latter claims that 'intraocular inflammatory effects due to latanoprost do not occur'. In evaluation of drug-induced toxicity Naranjo *et al.*[10] have highlighted the following criteria: (a) the reaction is a frequently described event; (b) recovery occurs on withdrawal of the drug; (c) other possible causes of reaction have been excluded; (d) objective signs of reaction were documented; (e) signs return on re-challenging with the same drug. That our patient fulfilled four of these criteria (the fifth having not been tested) strongly supports latanoprost as the cause of her uveitis.

Our patient also developed poliosis and on withdrawal of the drug demonstrated apparent hypotrichosis. Poliosis has been described as an adverse effect with drugs such as cyclosporin A[11] and chloroquine.[12] It can be explained on the basis of immune complex activation against the hair follicle melanosomes from focal drug toxicity. This may involve either anti-drug antibodies or anti-drug plus protein antibodies against the melanosomes.[13] While the poliosis may have been induced by latanoprost in pre-existing lashes, it is possible that it occurred only in those lashes which grew in response to latanoprost. The latter would explain the segmental nature of the poliosis.

Prostaglandin $F_{2\alpha}$ is capable of inducing DNA replication and stimulation of cell division and growth in a number of tissues *in vitro*. Latanoprost retains the functional group of prostaglandins that confer the ability to act as a mitogen or growth factor and hence may cause hypertrichosis. Hypotrichosis on stopping the drug can also be explained on this basis. However, it is possible that the apparent hypotrichosis in this patient may have represented a return to the pre-latanoprost eyelash population, rather than a true retardation of eyelash growth.

To the best of our knowledge granulomatous anterior uveitis and poliosis on latanoprost use and hypotrichosis on its withdrawal have not been previously described in

the literature. We wish to bring this observation to the notice of ophthalmologists prescribing latanoprost 0.005% so that patients undergoing treatment may be informed of its new side effects.

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Sir,

Intralenticular abscess caused by *Stenotrophomonas maltophilia*

The non-fermentative Gram-negative bacillus *Stenotrophomonas maltophilia* has increased in profile in recent years as a significant nosocomial pathogen with rapidly rising clinical importance.[1] We describe a case of intralenticular abscess resulting from this organism, which has not, to our knowledge, been previously reported.

Case report

A 27-year-old woman was referred from a provinicial ophthalmology department with right uveitis for further assessment and management. She had first presented to that centre 16 months earlier with right iritis, after which initial investigations and treatment were never completed, the symptoms resolved, and she did not re-present for follow-up. Four weeks previously she had given birth to a normal-term infant. She had lived in a rural setting for 4 years.

Two weeks before tertiary referral, there had been a rapid onset of photophobia, right eye ache and redness. She was admitted to the provincial hospital for 2 days, treated with dexamethasone 1% eyedrops 2-hourly and atropine 1% eyedrops 8-hourly. The patient stated that she initially improved but then worsened. Betamethasone was injected subconjunctivally.

Anterior segment activity was recorded as increasing despite treatment, and a dense exudative and inflammatory membrane had formed across the pupil. Iris bombe had developed and YAG iridotomy was attempted. Erythrocyte sedimentation rate at the provincial centre was 46 mm/h.

On examination at admission to Royal Brisbane Hospital, the Snellen visual acuity was light perception in the right eye and 6/5 in the left. There was a right relative afferent pupillary defect. In the right eye there was 3+ conjunctival injection, and mild corneal epithelial oedema. The anterior chamber was very shallow with areas of iris–endothelial contact. There was a fibrinous exudate across the anterior chamber forming a pupillary membrane. Mild iris bombe was present. Intraocular pressure in the right eye was 22 mmHg. No red reflex was visible and there was no fundal view. B-scan ultrasonography showed clear vitreous and no detectable retinal detachment. Pathology tests screening for causes of uveitis were performed showing no positive results. On examining the left eye, no abnormalities were noted.

Treatment was commenced with prednisolone 1%/phenylephrine 0.12% eyedrops half-hourly and atropine 1% eyedrops 6-hourly with oral prednisone 80 mg daily. There was decreased pain and decreased redness but a fibrin plaque persisted with leakage of lens material into the anterior chamber. Blood vessel growth was noted on the anterior surface of the lens.

Surgery was performed with removal of the dense, fibrinous pupillary membrane, and an anterior capsulotomy was made with aspiration of liquid pus. No lens fragments were identified. Intravenous vancomycin and ceftazidime were commenced. Microscopy of the lens aspirate showed Gram-negative rods.

A three-port pars plana vitrectomy was performed the next day, with debulking of a fibrinous exudate on the posterior hyaloid face. Three peripheral retinal tears were noted, and there were scattered satellite lesions on the retinal surface. A 360° scleral buckle was placed and endolaser performed. Amikacin 400 μg and vancomycin 2 mg were given intravitreally.