chromosome be active in a sufficient proportion of cells in a tissue in which the gene is expressed then a female may manifest the disease in that tissue. If cells of only parts of that tissue are affected then a mosaic pattern may be demonstrated. The X-inactivation ratio determines the degree to which the tissue is affected and in our patient could explain the laterality of involvement.

It might be argued that these changes are due to other causes. Age-related macular degeneration is a possibility; however, the relatively early age of onset, paucity of drusen and marked asymmetry of the macular findings are somewhat atypical. Serpiginous choroiditis commencing at and involving solely the macular region has been described.[14,15] Involvement is bilateral, although its extent is frequently asymmetrical.[14,16] Our patient's condition has remained essentially unilateral for more than 4 years now, with no signs of acute inflammation at any stage. Geographical atrophy of the macula is also described in Stargardt's disease,[17] a heredo-macular disorder. Onset is usually bilateral and in the teenage years.[18,19] Fundus flavimaculatus, a variant, has onset in the fourth or fifth decade. Retinal flecks are not always present,[17,20] but the visual prognosis is generally good. Other fundal dystrophies have been reported to give a similar appearance to choroideremia;[12,21] these include gyrate atrophy, diffuse choriocapillaris atrophy, central areolar choroidal dystrophy and retinitis pigmentosa.

We feel the family history and carrier status of our patient are important when considering the cause of her eye disease. Mechanisms by which an obligate heterozygote for an X-linked condition may manifest disease uniocularly and to varying degree within the same eye have been postulated. We believe manifest choroideremia is the most likely cause of our patient's visual disability, and would be interested to hear of any similarly affected female carriers.

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Sir,

Darkening of eyelashes in a patient treated with latanoprost

Latanoprost, a prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) analogue, is the first in a new class of drugs for the treatment of open angle glaucoma. Its primary mode of action is by increasing uveoscleral aqueous outflow. Iris pigmentation is a well-documented association with topical latanoprost treatment.[1,2] We report a case of darkening of the eyelashes in a patient treated with latanoprost.

Case report

A 78-year-old woman attended the Birmingham and Midland Eye Centre with a diagnosis of pseudoexfoliative glaucoma in her right eye. Her right

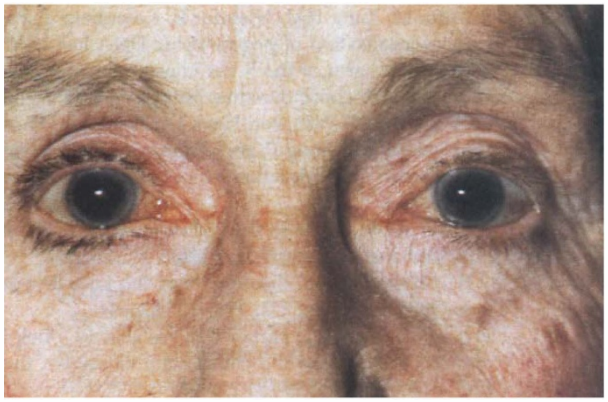

Fig. 1. En face *photograph of the patient after 12 months of treatment with topical latanoprost 0.005% drops to her right eye, showing thicker, darker and denser eyelashes of this eye. The left eye is untreated.*

Snellen visual acuity was 6/9 and intraocular pressure (IOP) measured 32 mmHg. She had a grey-brown iris, a vertical cup:disc ratio of 0.5 and Humphrey 24–2 visual fields demonstrated a nasal scotoma. The left eye showed changes of pseudoexfoliation with normal acuity, IOP, optic disc and field. Topical ß-blocker therapy was contraindicated as she had chronic bronchitis. Her IOP was inadequately controlled at 30 mmHg with dipivefrine 0.1% b.d. and in March 1996 she was enrolled in a long-term open safety study of latanoprost 0.005% as adjunctive therapy.

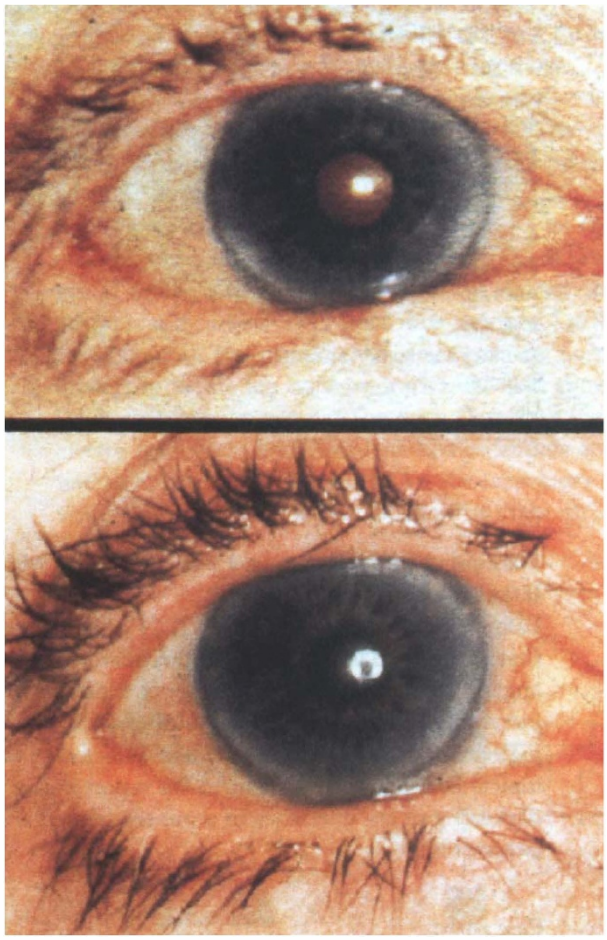

Fig. 2 *The eyelash colour and density in the right eye at baseline (top) and after 12 months of treatment with topical latanoprost 0.005% (bottom).*

When reviewed 4 months following treatment, she reported that her grand-daughter had asked her why she wore mascara on her affected eye, although she never wore any eye make-up. Examination revealed that she had darker-coloured eyelashes on her right eye, the eye treated with latanoprost (Figs. 1, 2), as compared with her left untreated eye which was normal. Her right visual acuity was 6/9 and her IOP was adequately controlled at 15 mmHg. The eyelashes of her right eye have continued to darken over the past 12 months and she has not developed increased iris pigmentation.

Comment

An increase in pigmentation of the iris has been described in patients on topical latanoprost and appears during the first 6 months of treatment.[3] Mixed-colour irides, such as blue-brown, grey-brown, green-brown or yellow-brown, are more susceptible to developing increased pigmentation.[3] Neither naevi nor freckles of the iris have been affected, and though no further darkening occurs after discontinuation of treatment, the change in iris colour may be permanent.[4] The darkening of the iris appears to be due to increased melanogenesis, particularly with increased production of eumelanin;[3] this occurs without proliferation of iris stromal cells.[3] Cultured dermal and uveal melanocytes as well as uveal melanoma cells show no increase in cell proliferation when treated *in vitro* with latanoprost.[1]

Recently, similar cases of eyelash darkening in patients using latanoprost have been described.[5,6] This indicates that increased melanogenesis may occur elsewhere than in the iris. Skin, conjunctiva, hair and eyelash follicles contain melanocytes, and some arachidonic acid metabolites have been suggested to stimulate melanogenesis.[7] PGE_1 and PGE_2 have been reported to exert a melanogenic effect in cutaneous or hair bulb melanocytes *in vitro*.[8] When our patient attended for follow-up 16 months after commencing treatment, eyelashes were epilated from each eye. Microscopically, there was a 50% increase in diameter at the hair bulb end of the lashes of the treated eye, although the lashes of both eyes tapered to the same diameter at the end. Eyelashes from both eyes demonstrated pigmentation along the centre of the lash shaft, but there was a wider distribution of pigment throughout the centre and periphery of the treated eye's lash shaft. During eyelash growth, there is migration of pigmented melanosomes from the lash bulb along the keratinocytes within the shaft.[9] This differs from the iris melanocytes, which remain static rather than deliver melanosomes to surrounding cells. The significance of this finding, and the exact mechanism for the increased pigmentation, has yet to be elucidated.

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

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Sir,

Corneal endothelial precipitates in HIV- and CMV-positive patients without concomitant ocular disease

Human immunodeficiency virus (HIV) and associated opportunistic infections are well known to have ocular manifestations. The rate of eye involvement exceeds 70%.[1] Recently a new manifestation of corneal involvement was reported in the form of corneal endothelial precipitates in association with cytomegalovirus (CMV) retinitis.[1–3] We report two cases of HIV-positive patients with similar corneal endothelial deposits without the presence of CMV retinitis or other ocular diseases.

Case 1

A 28-year-old, HIV-positive white man, with a history of AIDS-defining infections including *Mycobacterium avium intracellulare*, *Pneumocystis* pneumonia and toxoplasmosis, presented with a complaint of blurred vision. Best corrected visual acuity was 6/7.5 in both eyes. External and visual field examinations were normal. Pupils were equal, round and reactive. Anterior segment evaluation was pertinent for bilateral reticular non-pigmented endothelial precipitates, which formed a 360° ring around the corneal periphery and were scarcely distributed throughout the rest of the endothelial surface (Fig. 1). The corneal epithelium and stroma were normal. The anterior chambers were quiet. No other signs of uveitis were noted. Fundoscopic evaluation of both eyes by a vitreoretinal specialist was unremarkable.

The patient was treated first with fluorometholone 0.1% and then with prednisolone 1% without improvement. He was a participant in an oral ganciclovir treatment trial. The trial evaluated oral ganciclovir versus placebo as a prophylactic agent against CMV disease.[4] Upon entering the trial the patient underwent screening for opportunistic infections including a CD4 count, an antibody test and urine culture for CMV, blood count and serum chemistry. All patients included in this trial had CMV infection proven by a positive blood titre or urine culture, but had no active CMV disease. This patient was in the ganciclovir group. The corneal precipitates were first noted at 28 months into the trial, at which point the patient was referred to our institution.

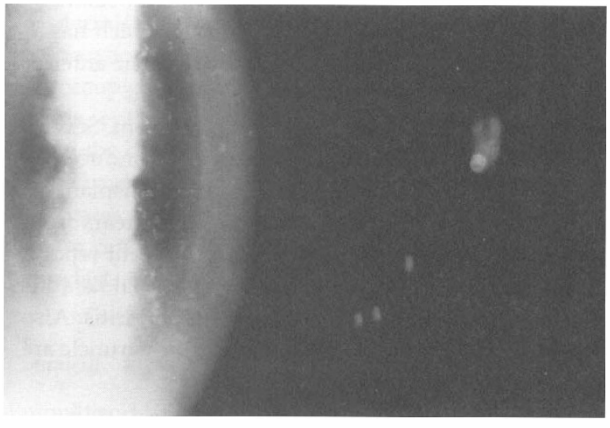

FIg. 1. *Case 1. A narrow beam slit-lamp photograph of the right eye showing non-pigmented diffuse reticular precipitates on the corneal endothelium.*