normal (fig. 1b) and remained so at further review 3 months later and again 1 year later.

The pathogenesis of circumscribed palmar or plantar hypokeratosis is uncertain. The cases of Perez *et al*.[1] had remained unchanged for years and had no history of preceding trauma. Although the authors proposed that the lesions represent an epidermal malformation this seems doubtful, particularly considering that all their cases except one presented late, either in middle or old age. The history of progressive enlargement, seen clearly in our patient and in another published case,[2] and the superficial resemblance to porokeratosis, known to be caused by an abnormal clone of peripherally expanding epidermal cells,[7,8] raises the question whether this condition too is caused by an abnormal clone of keratinocytes, the abnormality in this case being the production of an unusually thin stratum corneum. Although the possibility of a clonal basis was considered by Perez *et al*., it was discounted in favour of an epidermal malformation.[1] We think the clonal explanation is more plausible. Our patient's response to cryotherapy was presumably due to destruction of the abnormal clone followed by repopulation with normal epidermal cells. To our knowledge, cryotherapy treatment for this condition has not been reported previously and, in view of our result, we suggest is worth considering for similar cases. A recent report suggests that topical calcipotriol may also be helpful.[6] The history of trauma in our case is intriguing, suggesting that the condition may be triggered by trauma. A less clear-cut history of trauma was also given in another case[3] that occurred at the site of an old burn.

The morphological features of circumscribed palmar or plantar hypokeratosis appear to be distinctive and unique. The pink colour is presumably caused by the dermal vasculature showing through the thin stratum corneum. The abnormally thin stratum corneum probably also explains the slight tenderness noted in our patient and in one other published case.[3]

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Periocular pigmentation associated with use of travoprost for the treatment of alopecia areata of the eyelashes

Editor

Prostaglandin analogues are intraocular pressure (IOP)-lowering drugs for use in patients with glaucoma and ocular hypertension. Travoprost (AL-6221) (Travatan™, Alcon, Ft Worth, TX, USA), a PGF2a analogue, is an isopropyl ester of the more active enantiomer(+) of fluprostenol, a selective FP prostanoid receptor agonist.[1] It is structurally similar to other prostaglandin F2a analogues such as latanoprost.

Since the introduction of prostaglandin analogues in 1996, several adverse effects have been reported, including increased eyelash growth, darkening of the iris and periocular skin colour change.[2]

Travoprost induces growth of lashes and ancillary hairs around the eyelids. Manifestations include greater thickness and length of lashes, additional lash rows, and conversion of vellus to terminal hairs in canthal areas as well as in regions adjacent to lash rows. In conjunction with increased growth, increased pigmentation occurs. Vellus hairs of the lower eyelids also undergo increased growth and pigmentation.

Eyelash hypertrichosis has recently been reported in 75% of patients in clinical trials evaluating efficacy of the PGF2a analogue travoprost in the treatment of ocular

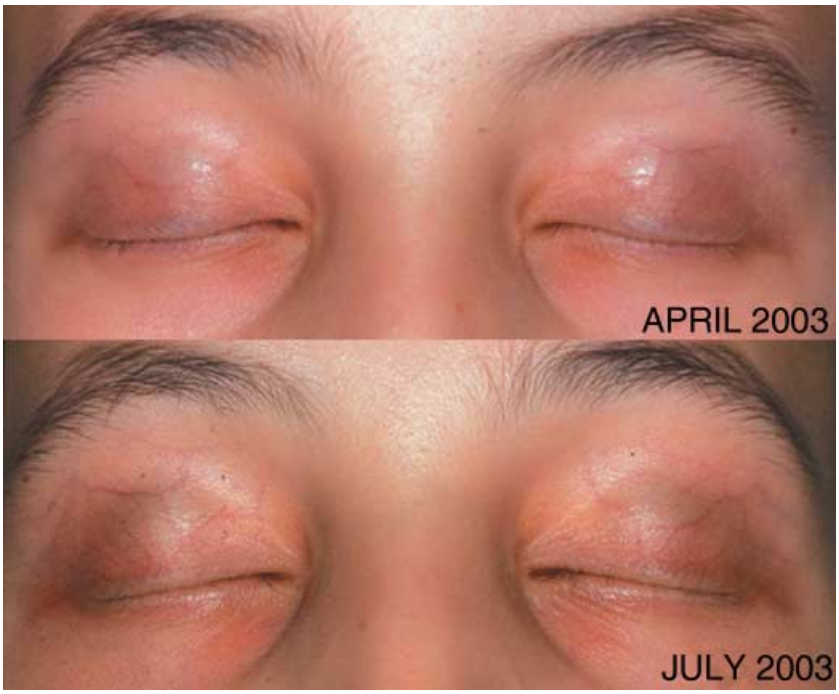

fig. 1 Case 1. Periocular pigmentation before start of therapy, and after 3 months.

hypertension.[3] Prostaglandin receptors are present in the dermal papilla and in the outer root sheath of the hair follicle and appear to be involved in the development and regrowth of the hair follicle in mice. Prostaglandin analogues are believed to prolong the anagen phase of eyelashes.[4] Experimental studies indicate that hair growth stimulation by minoxidil might also be mediated by prostaglandin production.[5] Travoprost has therefore been proposed as a possible treatment for alopecia areata involving the eyelashes. We report three cases of periocular skin pigmentation, which developed during treatment with topical travoprost for alopecia areata involving the eyelashes.

Case reports

The patients were applying a drop of the drug by using a cotton-fioc on the eyelid border twice a day; clinical examination was performed, and external photographs were taken.

Case 1

A 12-year-old girl developed bilateral increased eyelid skin pigmentation 6 months after beginning treatment with topical travoprost in both eyes. The therapy did not result in regrowth of lashes so was discontinued. Periodic examination revealed that the eyelid skin pigmentation gradually diminished 1 month after cessation of the drug, and the decrease in pigmentation continued over 4 months of follow-up (fig. 1).

Case 2

A 45 year-old woman experienced regrowth of the upper eyelashes after 7 months of therapy but blue-grey increased pigmentation was observed in both upper and lower eyelids of both eyes; pigmentation was more evident in both lower lids. Pigmentation gradually diminished in 3 months after cessation of therapy. The patient continued to show further loss of darkening on both eyelids, although minimal brownish coloration remained along the lower eyelid folds of both eyes at 4 months. Hair regrowth was maintained despite travoprost interruption.

Case 3

A 32-year-old woman with universal alopecia areata noticed increased eyelid pigmentation without growth of lashes after 6 months of therapy and travoprost was discontinued. A gradual disappearing of pigmentation was noticed during the following months.

Pigmentation of the periorbital skin can be caused by many factors (Table 1). An increase in eyelid skin pigmentation as a possible complication of topical travoprost therapy has been reported and it appears to be a reversible change. In fact, cessation of the drug usually results in loss

Table 1 Causes of pigmentation of the periorbital skin

Cause	Colour	Associated features
Mercurial or silver preparation	Blue-grey	Pigmentation in the nasolabial and neck folds
Chrysiasis	Mauve	Slate blue discoloration in sun-exposed skin
Eczema (dermatitis)	Brown	Typical eczema signs
Familiar	Brown	None
Hormone-induced melanosis	Brown	Hypermelanosis of the face
Hypohidrotic dysplasia	Dark	Frontal bossing, saddle nose, prominent supraorbital ridge, hypohidrosis, dystrophy of the teeth[6]
Lichen planus	Violet, brown	Typical lichen planus signs
Minocycline	Grey	Blue-grey pigmentation within areas of inflammation or scarring, of the legs and of the forearms
Nevus of Ota	Blue-grey	Involvement of ocular structures, nasopharynx, auricular mucosa, tymphanic membrane and dura
Post-traumatic	Brown	None
Postinflammatory	Grey-black	None
Psoralens	Brown	Sunburn, nausea and vomiting, itching

of induced pigmentation. Most of the patients reported in the literature were Japanese or oriental and a racial influence may be hypothesized. In our patients we have excluded familial periocular hyperpigmentation. However, all the patients were dark skinned and this feature could be a possible predisposing factor to this adverse drug effect.

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A case of lymphangioma on the earlobe

Editor

Cutaneous lymphangioma or lymphatic malformation commonly develops as a tumoural change of the skin, and is composed of dilated ectatic lymphatic channels. Although cutaneous 'lymphangiomas' of the skin have been classified into four categories according to classic pathological findings, it is now considered as a malformation

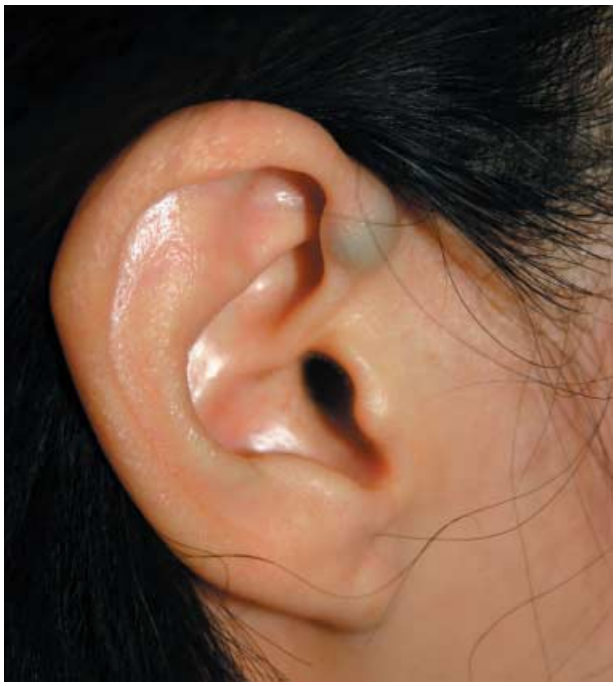

fig. 1 Solitary, tender, 7 × 8 mm-sized, blue-to-black-coloured cystic nodule on the earlobe.