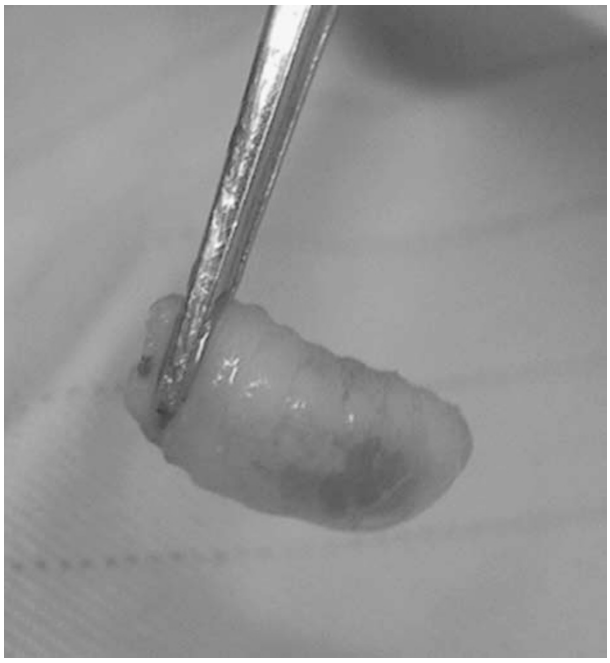

Figure 2 The extracted Tumbu fly larva.

Infestation of humans in areas where the fly is endemic is not uncommon, but the lesions are usually on nonexposed parts of the body. The larvae need air to develop and so asphyxiation is an effective, although slow, treatment. Paraffin, petroleum jelly, or sticking plasters have been used to occlude the central pore.[2] Toxic agents (eg insecticide) may also be applied, or the larvae can be extracted taking care to remove it in one piece. Complications of the infestation are unusual, although rupture may lead to a severe inflammatory response.[3]

Basic sanitation helps to prevent infestations, by covering or removing areas of soil contaminated with urine or faeces. Ironing of clothes or bedding that has been dried outside is also believed to help by killing eggs within the fabrics.

Other flies that cause furuncular myiasis include *Cordylobia rhodaini* (Lund fly) and *Dermatobia hominis* (Human Botfly), which are found in tropical Africa and southern America, respectively. Cutaneous myiasis of any cause is uncommon in the UK and infestations on the face are even more unusual. They may be confused with other causes of abscess, but larval infestations should be considered in patients returning from endemic areas.

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Sir,

Pseudophakic cystoid macular oedema: 30 months after latanoprost challenge

Latanoprost-induced cystoid macular oedema (CMO) is well documented, and stopping the drops before or after cataract surgery has been used to reduce this risk.[1] What is not clear is whether cataract surgery can increase the risk of prostaglandin-induced CMO, following recovery from cataract surgery. In this context, we report a case of latanoprost-induced CMO developing 30 months after an uneventful cataract surgery.

Case report

An 83-year-old male patient with primary open-angle glaucoma on latanoprost (0.005%) and brinzolamide (1%) eye drops in both eyes underwent phacoemulsification with posterior chamber intraocular lens implanted in the left eye and achieved best-corrected visual acuity of 6/6. Latanoprost eye drops were continued before and after the surgery. After 3 months, the visual acuity in the operated eye dropped to 6/18 and cystoid macular oedema (CMO) was seen clinically. He was in good general health and did not suffer from diabetes mellitus. Latanoprost drops were immediately withdrawn and subtenon's triamcinolone injection was administered.

The CMO resolved and the visual acuity improved to 6/9. After 4 months, a trabeculectomy with mitomycin-C (0.4 mg/ml) was carried out in the same eye. The postoperative course was uneventful, and all the antiglaucoma medications in that eye were stopped. After 5 months, needling of the bleb with 5-fluorouracil (5 mg/0.1 ml) was performed. The IOP remained high and the patient was instructed to use brinzolamide eye drops in both eyes and latanoprost eye drops in the right, nonoperated, eye.

Thirty months after the cataract surgery, the patient noticed reduction of visual acuity in the left eye (6/36 now), and CMO was diagnosed that was confirmed on optical coherence tomography (Figure 1). The patient admitted using latanoprost eye drops in both eyes in the preceding 1 month because of an error in the repeat prescription issued by the General Practitioner. Latanoprost eye drops were stopped immediately in the left eye and a repeat posterior subtenon's triamcinolone was given. After 1 week, the CMO started to resolve (Figure 2) and the visual acuity improved to 6/9 over the next 1 month.

Comment

Many antiglaucoma drops have been shown to cause CMO, especially in aphakic and pseudophakic eyes. Latanoprost-associated CMO in pseudophakic patients is thought to occur because of breakdown of the blood–retinal barrier (BRB).

In a retrospective review of 145 patients after an uneventful cataract surgery with lens implant, CMO was identified in four (3%) cases using latanoprost.[2] None of the patients who were taken off the latanoprost before surgery developed postoperative CMO in this cohort. Miyake and Nobuhiro[3] reported that latanoprost affects the wound healing process of lens epithelial cells, resulting in the biosynthesis of prostaglandins and other mediators that eventually lead to angiographic CMO. Another study by Miyake *et al*[4] suggested that the main cause of CMO induced by various eye drops was the added preservative, benzalkonium chloride. The preservative is thought to cause CMO in the same way as the eye drops themselves do.

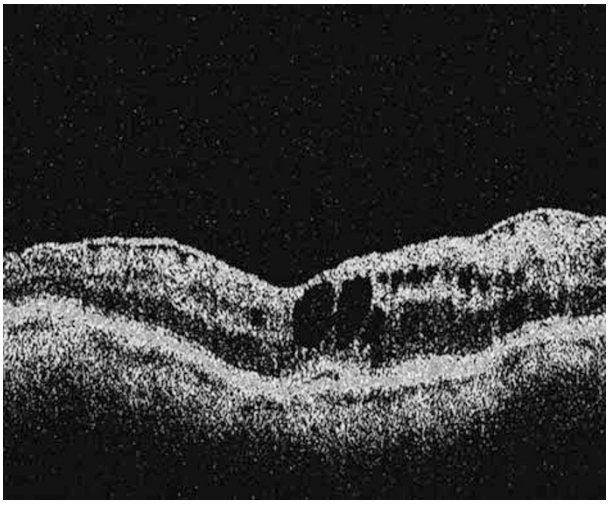

Figure 1 Optical coherence tomography showing gross CMO.

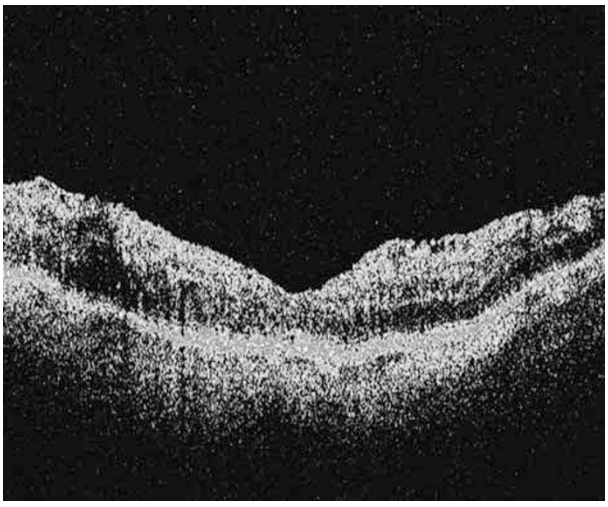

Figure 2 Reduction in CMO after latanoprost withdrawl.

Rowe *et al*[5] reported an eye that had undergone uneventful phacoemulsification and developed CMO at an early postoperative stage, and then 1 year after topical latanoprost application. Watanabe *et al*[6] reported a case in which latanoprost use induced CMO 5 years after a complicated phacoemulsification, suggesting that blood–ocular barrier remains fragile.

Our case developed CMO 30 months after an uneventful phacoemulsification. The role of a later trabeculectomy and needling in keeping the BRB fragile is not known and this may have contributed to the development of CMO after a later challenge with latanoprost. Our case raises the possibility that CMO can be induced even many months after the surgery and this possibility should be considered in the management of these patients.

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Sir,

Combined fungal and acanthamoeba keratitis: diagnosis by *in vivo* confocal microscopy

We report an interesting case of corneal ulcer due to combined fungal and acanthamoeba infection, which was diagnosed by in vivo confocal microscopy.

Case report

A 32-year-old male patient presented with a history of redness, pain photophobia, and blurred vision in the right eye of 1-week duration. He is an agriculturist by profession. There was no history of contact lens wear or trauma to the eye.

His best-corrected visual acuity was counting fingers close to face and 6/6 in the right and left eye. Slit-lamp examination of the right eye revealed a corneal ulcer involving the temporal cornea extending from 7 o'clock to 11 o'clock along with a hypopyon (Figure 1). The left eye was normal. *In vivo* confocal microscopy (Rostock cornea module with HRT II, Heidelberg Engineering, Heidelberg, Germany) revealed plenty of fungal filaments, a few acanthamoeba cysts and trophozites (Figure 2a and b). Corneal scraping for fungus revealed *Fusarium* species. Culture for bacteria and acanthamoeba were negative. He was started on hourly application of Natamycin 1%, propamidine, and polyhexamethylene biguanide (0.02%) eye drops. *In vivo* confocal microscopy was used to monitor the response of the treatment. Natamycin eye drops were tapered and eventually stopped after 40 days, whereas propamidine and polyhexamethylene biguanide were stopped after 3 months. At the third month follow-up, the ulcer has healed well with scarring and vascularisation. His best-corrected visual acuity in the right eye had improved to 6/9. Confocal microscopy showed plenty of dendritic cells at the area of the corneal ulcer. No fungal filaments, acanthamoeba cysts, or trophozoites were noted.

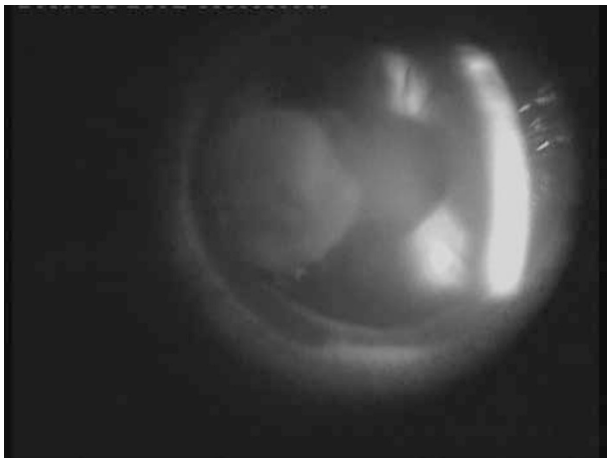

Figure 1 Slit-lamp photograph of the right eye showing corneal ulcer in the temporal cornea with hypopyon at presentation ($\times 10$).

Discussion

In vivo confocal microscopy using the principle of confocal microscopy, with an axial resolution of 5–10μ and lateral resolution of 1μ, enables us to understand the pathology at a cellular level.[1,2] The early detection of double-walled acanthamoeba cysts and trophozoites on confocal microscopy in this patient, with no growth on culture, completely alters our management and eventually a good prognosis.[2] Follow-up of these corneal ulcers with confocal microscopy helps us understand the changes occurring at the cellular level and moderate our treatment accordingly. We feel this tool can be used in our day-to-day practice in the management of combined corneal infections.