reported subspecies outside United States.[1] Mosquitoes are the biological vectors in transmitting the parasite from animals to human. Sometimes they can penetrate the tissue and spread systemically into internal organs.[2] Pampiglione *et al*[3] reported a case series of dirofilarial infections in which 30% had ocular involvement. Ocular infection can be subconjunctival, periorbital, orbital, or intraocular. Orbital infections can result in proptosis, ptosis, or diplopia.[4]

Systemic features such as fever, lymphadenopathy, and reactive arthritis are occasionally seen. Only 25% of subcutaneous cases has peripheral eosinophilia.[4] The diagnosis is made on the basis of histological and morphological identification. Serology and molecular biology techniques may provide supportive information, but the significance of them being the definitive diagnostic tool has not yet been established.[3,5]

In our case, the acquisition of the parasite may have occurred in Hong Kong or in China. Neither the time of transmission nor the route of entry into the lacrimal gland can be determined. Direct inoculation into lacrimal gland is probably not possible. One possibility is that, following an innocuous mosquito bite in the periorbital region, the microfilaria developed in the subcutaneous tissue and finally burrowed into the lacrimal gland. Surgical excision is the recommended treatment for dirofilariasis and this can also provide histological diagnosis. Unlike other filarial infections such as onchocerciasis and loiasis, microfilaraemia is unusual and antihelminthic agents are generally not indicated.

To the best of our knowledge, this is the only report in the English literature that dirofilariasis presented as a lacrimal mass. The initial clinical and radiological findings were all suggestive of a benign tumour. The deceptive feature led to a diagnostic challenge. One should consider this as an unusual differential diagnosis especially for those who have travelled in the past to endemic areas.

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Sir,

Giant retinal pigment epithelium rip secondary to subretinal proliferative vitreoretinopathy

Retinal pigment epithelial (RPE) rips occur relatively uncommonly and are usually associated with serous detachments of the pigment epithelium, secondary to choroidal neovascularization. These typically occur at the macular and cause rapid visual loss. Once the pigment epithelial tear occurs, the RPE retracts from the outer portion of Bruch's membrane and scrolls up. Fluorescein angiography typically demonstrates a well-demarcated hyperfluorescence in the early phase of the angiogram. We describe a case of RPE tear, unusual in terms of its pathogenesis and size.

Case report

An 81-year-old lady was referred with deteriorating vision in her right eye over a period of weeks. Her left eye was blind due to an inoperable retinal detachment 60 years previously. Examination revealed acuity of 6/9 in her right eye and hand movements in her left. On fundoscopy, the left eye had a chronic inferior macular off retinal detachment. Above the superior arcade, there

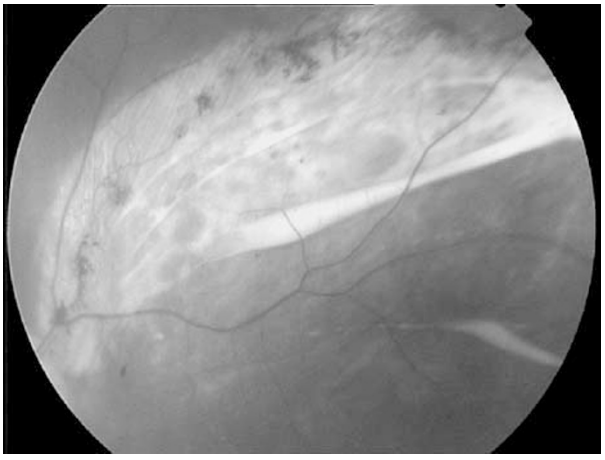

Figure 1 A retracted flap of RPE lay adjacent to a hypopigmented region of denuded Bruch's membrane.

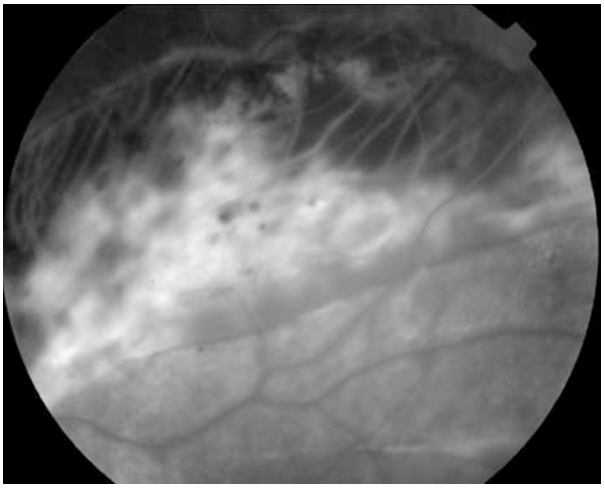

Figure 2 Fluorescein angiography demonstrated hypofluorescence over the flap adjacent to diffuse hyperfluorescence, with a characteristic demarcation line between the two.

was a large area of absent RPE, exposing the underlying choriocapillaris. A retracted flap of RPE lay adjacent to a hypopigmented region of denuded Bruch's membrane (Figure 1). Inferior to the rip was an area of subretinal scarring consistent with subretinal proliferative vitreoretinopathy (PVR). Fluorescein angiography demonstrated hypofluorescence over the flap adjacent to diffuse hyperfluorescence (Figure 2), with a characteristic demarcation line between the two.

Comment

Tears in the RPE were first described in 1981 by Hoskins *et al*.[1] They are usually associated with progressive serous pigment epithelium detachments in AMD, or with laser photocoagulation or photodynamic therapy.[1–3] RPE rips, however, may also occur in patients with central serous chorioretinopathy, lupus, polypoidal vasculopathy, chorioretinal scarring, and presumed ocular histoplasmosis syndrome.[4–7] In each of these conditions, tears may be associated with increased hydrostatic pressure generated by damaged choriocapillaris.

In contrast, Gass[5] proposed that in AMD choroidal, neovascularization directly separates the RPE from Bruch's membrane and contractile forces of the choroidal neovascular membrane tears the RPE.[4] Supporting this, others have noted CNV in the bed of RPE rips, as well as at the site of the scrolled PRE through angiographic and histologic examination.[8,9,10]

The RPE tear in our case has occured due to traction from a proliferative vitreoretinopathy subretinal membrane and not due to hydrostatic or other mechanisms. Relatively small rips in RPE have been reported in other cases with PVR.[4] Our case was unusual in that it covered 4 clock hours of mid-peripheral retina—being the first reported with a 'giant RPE rip'.

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Sir,

Spontaneous reattachment of extensive Descemet's membrane detachment following uneventful phacoemulsification surgery

Menezo *et al*[1] presented a case of extensive nonplanar Descemet's membrane detachment following 'routine' phacoemulsification. The detachment progressed over the postoperative period, and surgical intervention with anterior chamber air tamponade was required to restore normal anatomy. We have recently managed a similar case, which provides further insight into this complication of cataract surgery. A 78-year-old woman underwent routine right temporal clear corneal phacoemulsification and lens implantation (Acrysof MA 60). She had no other ocular problems and at a 1-week postoperative review achieved 6/6 with a small myopic correction. No corneal abnrmalities were noted. At 6 weeks postoperatively, she presented to the eye department complaining of a gradual decrease in acuity in the operated eye. On examination, she was found to have corneal oedema extending from the temporal section to the visual axis, with underlying planar detachment of Descemet's membrane. The eye was quiet and the intraocular pressure normal. The patient was offered surgical intervention but declined because she was flying on holiday the following day for 1 week. She was reviewed 10 days later on her return. She stated that her vision had returned. On examination she achieved 6/6 and the cornea was clear with no Descemet's detachment. There was a tidemark visible in Descemet's delineating the area of previously detached membrane. At 6 months follow-up, the corneal appearance remains unchanged.

It is likely that this patient had a small peripheral Descemet's detachment immediately following her surgery. This then extended in a manner similar to that described by Menezo *et al*. In our case, however, the delicate balance of forces across Descemet's membrane shifted to allow the fluid flow generated by the endothelium to reappose Descemet's to the underlying stroma without surgical intervention. The temporal location of the corneal section may have allowed gravity to assist in changing this balance in favour of reattachment. Despite the good outcome of conservative management in this case, we would still offer prompt surgical intervention to similar patients in the future.

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