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Sir,

Comment on macular full-thickness and lamellar holes in association with type 2 idiopathic macular telangiectasia

The article by Charbel Issa *et al*[1] is an interesting report on the association of type 2 idiopathic macular telangiectasia (IMT) with both full-thickness and lamellar macular holes (MHs). The aetiology of IMT is unknown, but possibilities include chronic leakage from hyper-permeable capillaries and ischaemia. The authors hypothesise that MHs in IMT are caused by tissue loss consequential to these factors, with secondary draping of an as yet undefined membrane, possibly ILM, over these areas rather than the accepted concept of epiretinal tractional forces that result in idiopathic MHs.

We describe a case of IMT with ERM that showed visual improvement with vitrectomy, which is relevant to the aetiology of IMT and the evolution to a MH. A 66-year-old pseudophakic woman presented with a 3-year history of gradually reducing central vision. Fundoscopically she had the typical signs of IMT, which were confirmed on angiography. In addition, she had very fine surface ERM in both eyes, which was worse in the left eye. Vitrectomy was carried out on the left eye, with separation of an incompletely attached posterior hyaloid face and peeling of the ERM. Postoperatively her vision improved from 6/36 to 6/12. In view of the improvement in the left eye, a similar surgery was carried out on the right eye. Once again, visual acuity improved from 6/60 to 6/18. Her vision remained stable at follow-up 3 years later.

ERM formation can be seen in a number of situations in which chronic retinal vascular leakage and hypoxia occur, most notably in diabetic maculopathy. ERM formation in these situations is likely to be driven partly by hypoxic cytokine-driven tissue repair.[2–4] Perhaps in IMT, this also has a significant role in leading to a detrimental repair process causing ERM, and in some cases, traction. Vitrectomy increases oxygenation in the vitreous cavity, increasing the availability of oxygen to the retina and thus potentially dampening tissue repair processes, as well as improving retinal function.[5]

Vitrectomy may therefore both remove detrimental traction and improve retinal oxygenation. It is possible that patients with early symptomatic IMT, particularly those with epiretinal membranous changes, could actually benefit from vitrectomy and membrane peeling before atrophic MH formation occurs. Further study on this aspect is needed.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Sequential corneal infection with two genotypically distinct *Acanthamoebae* associated with renewed contact lens wear

Acanthamoeba keratitis (AK) is a rare infection that is estimated to occur in between 1–100 cases per million

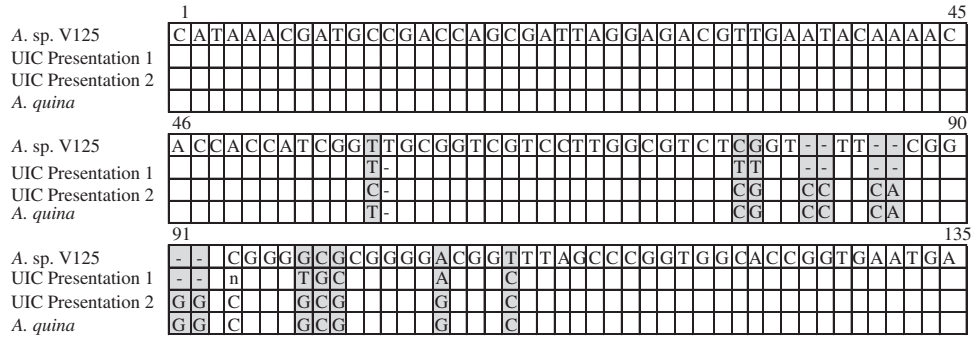

Figure 1 DNA sequence analyses for isolate identification of the Acanthamoebae from the patient's first keratitis (UIC Presentation 1) and second keratitis after restarting contact lens wear (UIC Presentation 2). Shaded sites indicate locations where the bases differ between UIC Presentation 1 and 2. Empty sites indicate that all sequences are the same as sequence 1, *A.* sp V125 (*Acanthamoeba* species V125). UIC Presentation 1 and UIC Presentation 2 each individually resembles previously sequenced isolates *A.* sp V125 and *A. quina*, respectively.

contact lens wearers per year, suggesting that the simple probability of re-infection is low.[1,2] Although recrudescence has been reported, we report a patient with two genotypically, geographically and temporally distinct AK infections.

Case report

A 16-year-old male soft contact lens wearer (phemfilcon) using an unspecifed Renu branded solution and AMO Complete MoisturePlus (AMO-CMP) presented to the University of Illinois Eye and Ear Infirmary with keratitis refractory to topical and systemic antivirals. Visual acuity of the affected right eye was 20/70. Examination revealed an epitheliitis with radial keratoneuritis. Epithelial debridement was performed, followed by topical propamidine isethionate (Brolene, Sanofi-Aventis, Paris, France) and chlorhexidine gluconate 0.02% hourly, which was tapered over 4 months. The patient was refit with soft contact lenses (lotrafilcon B) using AMO-CMP by another practitioner after clinic discharge.

One year later, he again presented with a 2-week history of severe pain and decreased vision in the same eye without interim symptoms. He reported recently swimming in a lake with his contact lenses, near his new home, 900 miles away. Visual acuity was 20/40, with an anterior stromal keratitis and identical treatment initiated.

Confocal microscopy, smear, and culture were positive at each presentation. The final spectacle corrected vision was 20/20 after each treatment. Sequence analyses indicated genetically distinct isolates with 13/135 base pair differences of the 18S rRNA gene (Figure 1).

Comment

Recrudescent and bilateral AK are considered to be due to incomplete treatment and contemporaneous environmental exposure, respectively.[3] While initial infection involving two or more strains is possible, an asymptomatic interval of greater than a year and isolation of two genetically distinct species suggest unique infections. A cornea compromised by past infection, poor hygiene, and solution use were likely contributing factors.[4]

Patients are often eager to return to contact lens wear, notwithstanding animal studies suggesting a lack of protection from prior infection and the possibility of differential individual susceptibility.[5] Our case illustrates that, despite the rarity of AK, this can manifest in humans and, without any modification in risk factors, AK patients persisting with contact lens wear sustain demonstrable risk for re-infection.

Conflict of interest

The authors declare no conflict of interest.

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The subject matter was previously presented at the meeting of American Society of Cataract and Refractive Surgery, 2008.

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Sir,

Virtual assessment and glaucoma shared care

We read with interest the report by Bourne *et al*.[1] Our attention was particularly drawn to the use of a virtual glaucoma assessment to support the community optometrists and provide quality assurance.

Many glaucoma management decisions are based on clinical findings, optic discs, visual fields, corneal thicknesses, and intraocular pressures (IOP) that are straightforward. Expert opinion is required however for a significant minority of cases. Unfortunately, deciding which patients require this additional input may be challenging. We also use a virtual clinic assessment to augment the triage of new patients who have been seen by 'in-house' ophthalmic nurse practitioners.[2] The decision-making remains the responsibility of the consultant, who reviews the clinical data, in the absence of the patient and oversees initial management. Our feasibility study has confirmed virtual assessment to be a reliable method of obtaining an accurate working diagnosis showing good agreement with experienced ophthalmologists.

In common with Bourne *et al*[1] we found issues with disc image interpretation in some patients. For us this was largely due to disc size (for both small and large discs), extensive peripapillary atrophy, and abnormalities of shape (tilting). Such disc images may be difficult to interpret in isolation, but this need not mean they cannot be reviewed on a virtual basis through serial images over time. We too found subtle variations in IOP around 21 mm Hg between practitioners that would tend to suggest the allocation of patients to different parts of the glaucoma care pathway. However, we would argue that this variation reflects not the accuracy of the optometrists, but rather the need to base IOP-governed decisions on a series of values. Given the demands of the recent NICE guidelines for glaucoma and ocular hypertension,[3] schemes for referral refinement and review of patients deemed stable, which utilise resources in the professions allied to medicine, are timely and appropriate. The use of a virtual clinic environment to allow expert advice and quality assurance also has much to recommend it.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Immunohistochemical analysis of internal limiting membrane by confocal microscopy in a case of stage 4 idiopathic macular hole

There are only rare reports showing the presence of α-smooth muscle actin (α-SMA)staining-positive cells in internal limiting membrane (ILM) specimens removed during idiopathic macular hole surgery, and in these studies, specimens have been evaluated by means of light or scanning electron microscopy.[1–3] To the best of the authors' knowledge, identification and localisation of α-SMA microfilaments by confocal microscopy in the ILM surrounding the borders of a macular hole have not been previously described.

Case report

A 68-year-old woman underwent vitrectomy for stage 4 idiopathic macular hole. Preoperative evaluation of the macular hole biomicroscopically and imaging with optical coherence tomography showed the absence of an epiretinal membrane (Figure 1b). Triamcinolone–acetonide was used for visualising the posterior cortical