

familial amyloidotic polyneuropathy caused by amyloidogenic transthyretin Y114C. *Ophthalmology* 2005; **112**: 2212–2218.

- 3 Kojima A, Ohno-Matsui K, Mitsuhashi T, Ichinose S, Nemoto T, Akashi T *et al.* Choroidal vascular lesions identified by ICG angiography in a case of familial amyloidotic polyneuropathy. *Jpn J Ophthalmol* 2003; **47**: 97–101.
- 4 Hattori T, Shimada H, Yuzawa M, Kinukawa N, Fukuda T, Yasuda N. Needle-shaped deposits on retinal surface in a case of ocular amyloidosis. *Eur J Ophthalmol* 2008; **18**: 473–475.
- 5 Anderson DH, Talaga KC, Rivest AJ, Barron E, Hageman GS, Johnson LV. Characterization of  $\beta$  amyloid assemblies in drusen: the deposits associated with aging and age-related macular degeneration. *Exp Eye Res* 2004; **78**: 243–256.

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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*<sup>1</sup> 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.<sup>2–4</sup> 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.<sup>5</sup>

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.

**References**

- 1 Charbel Issa P, Scholl HP, Gaudric A, Massin P, Kreiger AE, Schwartz S *et al.* Macular full-thickness and lamellar holes in association with type 2 idiopathic macular telangiectasia. *Eye* 2009; **23**: 435–441.
- 2 Jumper JM, Embabi SN, Toth CA, McCuen II BW, Hatchell DL. Electron immunocytochemical analysis of posterior hyaloids associated with diabetic macular edema. *Retina* 2000; **20**(1): 63–68.
- 3 Gandorfer A, Rohleder M, Grosselfinger S, Haritoglou C, Ulbig M, Kampik A. Epiretinal pathology of diffuse diabetic macular oedema associated with vitreomacular traction. *Am J Ophthalmol* 2005; **139**(4): 638–652.
- 4 Snead DR, James S, Snead MP. Pathological changes in the vitreoretinal junction 1: epiretinal membrane formation. *Eye* 2008; **22**(10): 1310–1317.
- 5 Williamson TH, Grewal J, Gupta B, Mokete B, Lim M, Fry CH. Measurement of PO<sub>2</sub> during vitrectomy for central retinal vein occlusion, a pilot study. *Graefes Arch Clin Exp Ophthalmol* 2009; **247**(8): 1019–1023.

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Sir,  
**Sequential corneal infection with two genotypically distinct *Acanthamoeba* 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