Sir,
Topiramate maculopathy secondary to dose titration: first reported case

Isolated topiramate maculopathy is a rare phenomenon.[1,2] We believe this is the first reported case of a 'topiramate maculopathy' secondary to dose titration.

Case series

A 27-year-old male presented to eye casualty with a one week history of bilateral reduced vision. He was a known migraine sufferer and had seen his neurologist 10 days earlier. It later transpired the neurologist had increased his topiramate from 75 to 100 mg. The patient reported no vision symptoms on the lower dose of topiramate. He was on no other medication and his past medical and ophthalmic history were unremarkable.

On examination his best-corrected visual acuity (BCVA) was 6/36 right and 6/24 left, with no improvement with pin hole. His intraocular pressure (IOP) was 13 in both eyes. The colour pictures reveal bilateral macular striae (Figure 1) which were confirmed by Topcon OCT (Figure 2) and red free (Figure 3). The fluorescein angiogram was normal. He was reviewed two weeks later and admitted discontinuing his topiramate of his own volition after the onset of his visual symptoms. His BCVA was now 6/9

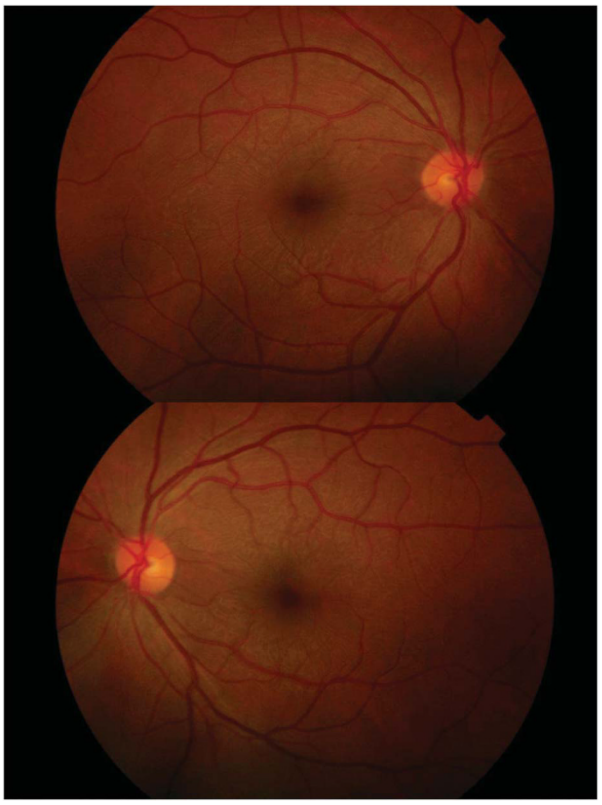

Figure 1 Fundi reveal bilateral macular striae on presentation.

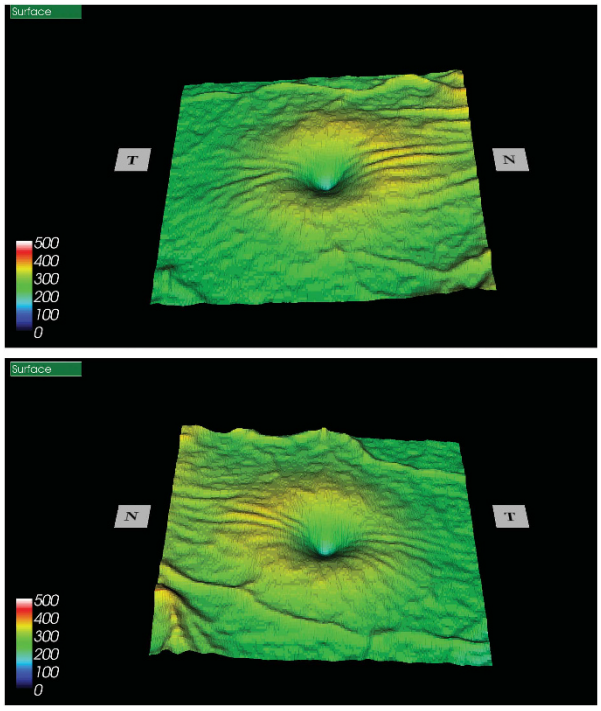

Figure 2 Striae confirmed by Topcon OCT on presentation.

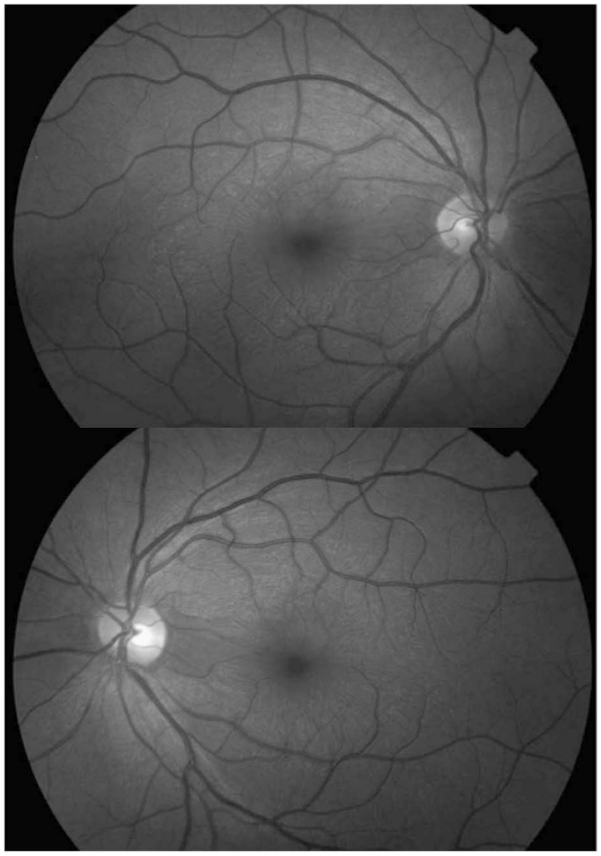

Figure 3 Red free images on presentation.

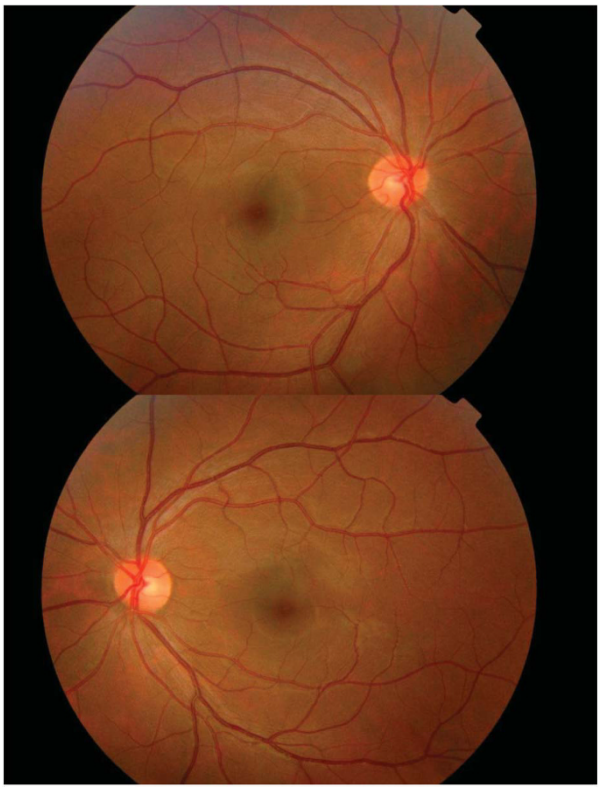

Figure 4 Normalisation after drug cessation.

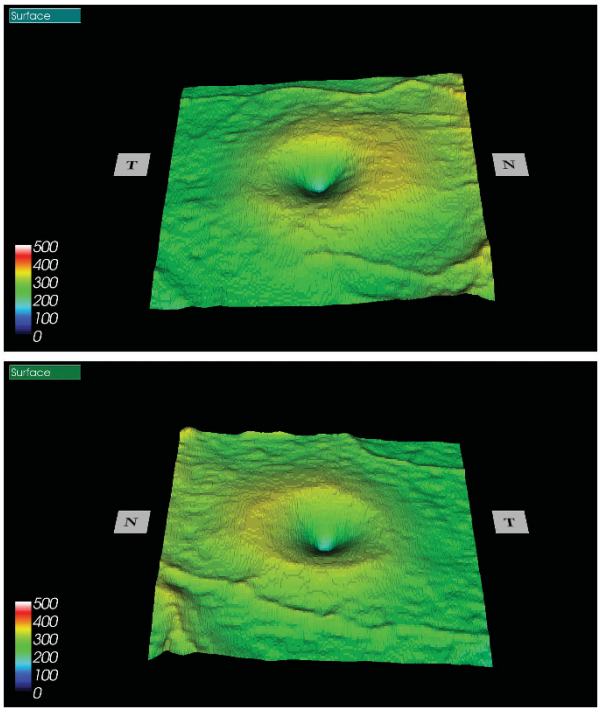

Figure 5 Improvement of OCT after drug cessation.

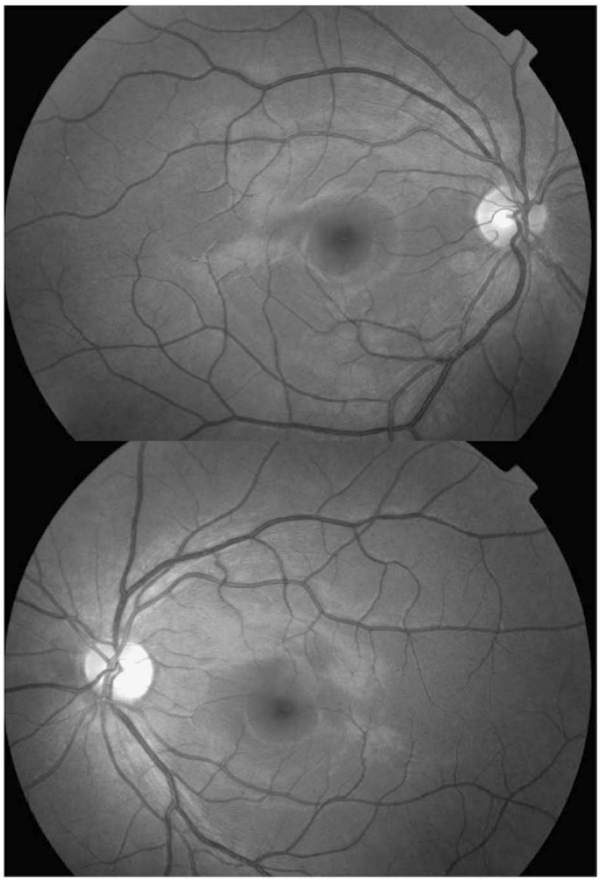

Figure 6 Red free images after recovery.

right and 6/5 left. The repeat images (Figures 4–6) confirm the resolution of his retinal striae.

Comment

Topiramate is a sulphamate-substituted mono saccharide derived from D-fructose. It is becoming increasingly popular for the management of epilepsy, migraine, trigeminal neuralgia, and depression.[3,4] The anterior segment ocular side effects have been extensively reported but the documentation and mechanism of a pure topiramate maculopathy is less well understood. This is highlighted by the omission of any reference of a pure maculopathy in the RCOphth guidelines.[5] However, the guidelines precede the initial case report and will hopefully be amended in the revision which were expected in October 2013.

We advise taking a detailed drug history including any recent change in dosage when faced with a similar clinical scenario. It is imperative the under lying diagnosis behind the use of topiramate is established and changes in dosage or discontinuation must be carried out in consultation with the patient's GP and/or neurologist. Topiramate maculopathy is not a life-threatening condition whereas status epilepticus is.

Conflict of interest

The authors declare no conflict of interest.

References

1 Gualtieri W, Janula J. Topiramate maculopathy. *Int Ophthalmol* 2013; **33**(1): 103–106.
2 Beyenburg S, Weyland C, Reuber M. Presumed topiramate-induced maculopathy. *Epilepsy Behav* 2009; **14**(3): 556–559.
3 Privitera MD, Brodie MJ, Mattson RH, Chadwick DW, Neto W, Wang S. Topiramate, carbamazepine and valproate monotherapy: double-blind comparison in newly diagnosed epilepsy. *Acta Neurol Scand* 2003; **107**(3): 165–175.
4 D'Amico D, Grazzi L, Usai S, Moschiano F, Bussone G. Topiramate in migraine prophylaxis. *Neurol Sci* 2005; **26**(Suppl 2): s130–s133.
5 Watts P. A review of the ocular side effects of topiramate. *RCOphth reference*: 2010/PROF/122.

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Sir,
Intraocular lens calcification following endothelial keratoplasty: a message for all cataract surgeons

We read with interest the UK case series recently reported in *Eye* detailing four cases of intraocular lens (IOL) opacification following Descemet's stripping automated endothelial keratoplasty (DSAEK).[1] This is a serious complication that causes visual loss and may necessitate IOL exchange that can adversely affect the long-term survival the corneal transplant.

All of the cases in the report involved Rayner (Hove, UK) hydrophilic acrylic IOL's and all involved rebubbling (repeat injection of intracameral air to achieve graft attachment). This series supports previous observations that such opacification appears to be almost unique to hydrophilic acrylic IOL's, and furthermore, having air or gas in the anterior chamber appears to be a risk factor.[2,3]

However, it is certainly possible for this complication to occur without rebubbling. Although all four cases had rebubbling in this series,[1] in our recently published UK series only two of the five cases had rebubbling.[4] In our series, all five cases were also hydrophilic acrylic IOL's, although only one was a Rayner implant highlighting that this problem relates to hydrophilic acrylic material regardless of the manufacturer (other IOL's that opacified included Zeiss, STABIBAG; Lenstec, LH 3000; Bausch and Lomb, MI60 and Bausch and Lomb, Akreos).[4]

We agree with the authors that patients requiring cataract surgery who are at risk of corneal endothelial failure (typically those with Fuchs' endothelial dystrophy) should not have a hydrophilic acrylic IOL inserted regardless of the manufacturer, in order to avoid the risk of IOL opacification. Although rebubbling may be a risk factor, our series demonstrates the complication can occur after DSAEK without rebubbling.

The number of corneal transplants in the UK has increased significantly in the last decade (2206 in 2002 rising to 3455 in 2011).[5] The proportion of endothelial transplants has risen markedly (0% in 2002 to 33% in 2011) and this is now almost as common as penetrating keratoplasty (33% *vs* 38%, respectively).[5] In the future, a significant number of patients may develop the serious complication of IOL opacification following DSAEK, and this could be reduced if cataract surgeons avoid hydrophilic acrylic IOL insertion in patients at risk of corneal endothelial failure.

Conflict of interest

The authors declare no conflict of interest.

References

1 De Cock R, Fajgenbaum MAP. Calcification of Rayner hydrophilic acrylic intra-ocular lenses after Descemet's stripping automated endothelial keratoplasty. *Eye* 2014; **28**: 1383–1384.
2 Neuhann IM, Neuhann TF, Rohrbach JM. Intraocular lens calcification after keratoplasty. *Cornea* 2013; **32**: e6–e10.
3 Dhital A, Spalton DJ, Goyal S, Werner L. Calcification in hydrophilic intraocular lenses associated with injection of intraocular gas. *Am J Ophthalmol* 2012; **153**(6): 1154–1160.
4 Park JC, Habib NE, Moate RM. Intraocular lens opacification after corneal endothelial keratoplasty: Electron microscopy and x-ray element spectroscopy analysis. *J Cataract Refract Surg* 2015; **41**: 140–145.
5 UK Transplant Registry. NHS Blood and Transplant authority, 2002 to 2011.

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Sir,
Reply to 'Intraocular lens calcification following endothelial keratoplasty: a message for all cataract surgeons'

We thank Park *et al* for their interest in our correspondence regarding the calcification of Rayner hydrophilic acrylic intraocular lens (IOL) implants following Descemet's stripping endothelial keratoplasty (DSAEK).[1]