parafoveal atrophic hypoautofluorescent patches in both maculae (Figures 1c and d). Spectral domain optical coherence tomography (SD-OCT) appears in Figures 1e and f. Pattern electroretinograms (ERGs) were abnormal and multifocal ERG revealed parafoveal dysfunction (Figure 2b). Full-field ERGs (Figure 2a) and electrooculography were normal. The *PRPH2*/*RDS* gene showed a normal coding sequence.

Sanger sequencing of exons 1–22 of the *ANO5* gene showed homozygosity for c.191dupA, p.(N64KfsX15) in exon 5, which, if expressed would lead to a peptide missing the carboxyl 850 of its 913 amino acids.
The 78-year-old affected brother had VAs of 6/9 right, 6/6 left, with bilateral epiretinal membranes (Figures 1i and j). Autofluorescence was normal (Figures 1g and h).

Comment

The index case maculopathy is unlike that seen in age-related macular disease, or in known mendelian dystrophies and probably represents a novel late-onset macular dystrophy. *ANO5* is expressed in the human RPE/choroid[4] and although its exact function remains unknown, it is thought to encode a CaCC.[1,2] CaCCs have a physiological role in RPE, important for fluid and ion transport across the RPE.[1,5,6]

To the best of our knowledge this is the first report regarding this association.

Although the possibility of the coincidence of two rare disorders in the proband cannot be excluded, the data are consistent with the occurrence of retinal macular disease in *ANO5*-mediated muscular dystrophy in some, but not all, mutation carriers.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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Sir,
Secondary glaucoma due to chronic scleritis: trabeculectomy in scleromalacia: a case report

Secondary open-angle glaucoma is frequently refractory and difficult to manage.[1]

Glaucoma is uncommon in scleritis, but may develop due to permanent damage to the trabecular meshwork even in quiescent scleritis.[2] Posterior scleritis has been reported to cause angle-closure glaucoma.[3–6] Other mechanisms include secondary steroid-induced glaucoma, peripheral anterior synechiae, and angle neovascularisation.

Scleritis has been reported following surgical trabeculectomy.[7]

Case report

A 68-year-old Caucasian patient had been followed up for 14 years with recurrent bilateral sectoral anterior non-necrotising scleritis in the absence of underlying systemic disease.

The intraocular pressure (IOP) had been controlled with topical g.Timolol 0.5% BD for the previous 5 years. Five months prior to surgery, IOP increased to 30 mm Hg. Despite maximal medical treatment (g.Bimatoprost/Timolol (Ganfort), g.Brinzolamide (Azopt), and Acetazolamide SR 250 mg p.o. BD) her IOP remained at 39 mm Hg. This was associated with a cup-disc ratio of 0.85 and predominantly nasal superior and inferior visual field defects (Figure 1).

This patient was surgically challenging due to the presence of diffuse scleromalacia (Figures 2 and 3

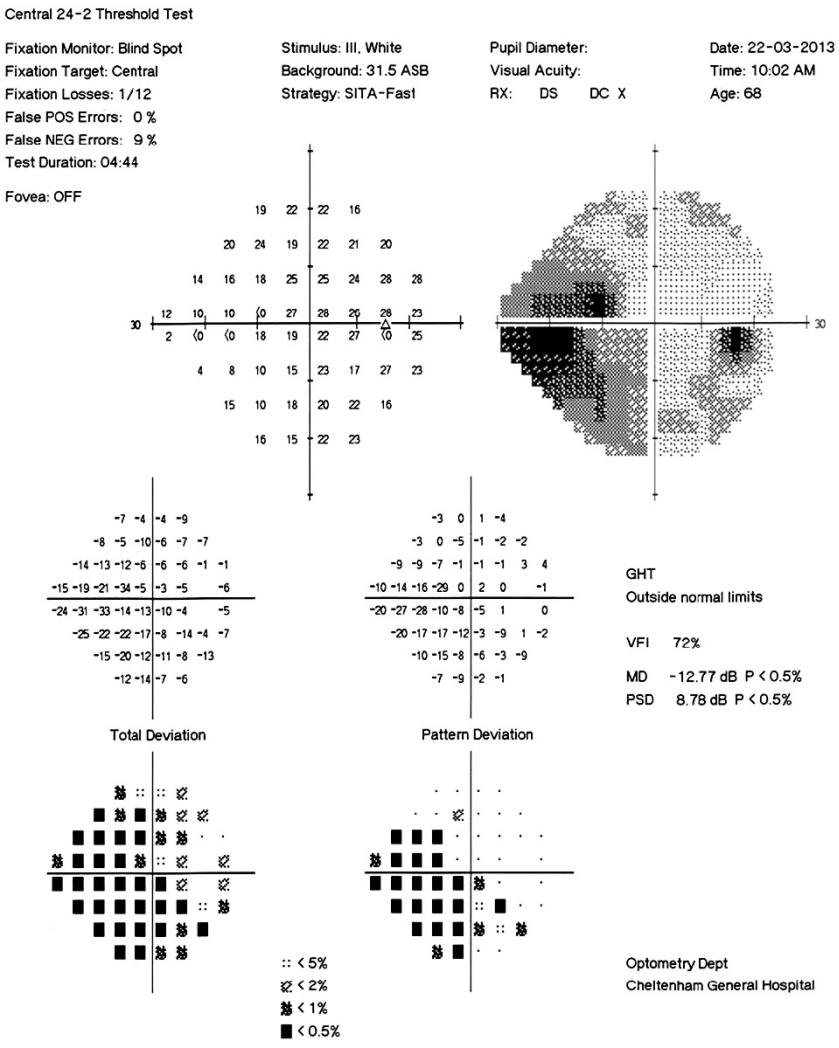

Figure 1 Visual fields.

Figure 2 Pre-operative scleromalacia in the left eye.

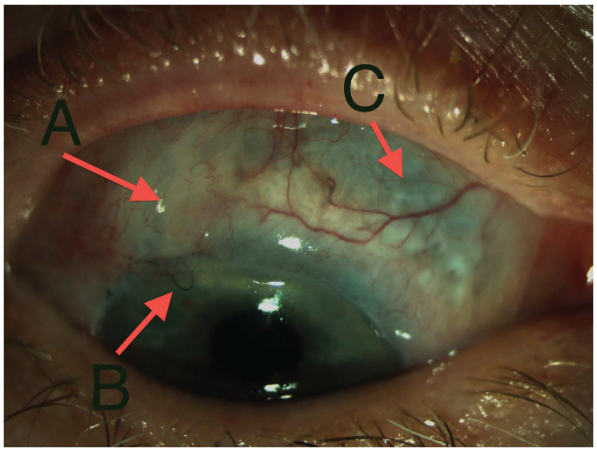

Figure 3 Post-operative trabeculectomy bleb (Arrow A).

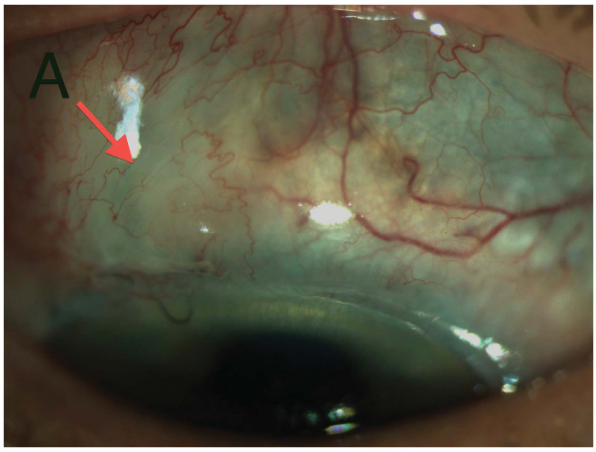

Figure 4 Post-operative trabeculectomy bleb (Arrow A).

(arrow C)), with one small island of peri-limbal white sclera supero-temporally. The sclera was too thin posteriorly for a tube or valve.

A trabeculectomy with Mitomicin C (0.2 mg/ml for 1 min) was performed without complication (Figures 3 (arrow A) and 4), using two releasable 10/0 nylon sutures to the scleral flap. Oral Prednisolone 40 mg/30/20/10 weekly taper, g.Prednisolone 1.0% 2 hourly, and g.Chloramphenicol q.d.s. were prescribed post-operatively. Subsequent IOP decreased to 6 mm Hg, and VA was 0.30. No recurrence of scleritis has occurred since the surgery, and the patient continues to use g.Prednisolone 1% o.d. 4 months post-operatively.

Comment

This patient's surgery was successful despite significant challenges and a known risk of scleritis resulting from the procedure itself. Oral Prednisolone was prescribed post-operatively, which was key in preventing the recurrence of scleritis in this patient.

Releasable sutures were used (Figure 3 (arrow B)), ensuring a low risk of hypotony immediately pre-operatively. The alternative of laser suture lysis would carry an increased risk of scleral perforation with such thin sclera.

Careful surgical planning, with judicious use of corticosteroids, can result in excellent surgical outcomes in patients with scleritis and secondary glaucoma.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Comment on: Pathogenic conjunctival bacteria associated with systemic co-morbidities of patients undergoing cataract surgery

I read with great interest Fernández-Rubio *et al*'s[1] study on colonization patterns in relation to systemic co-morbidities.

There appears to be, however, a misconception between the usage of intracameral cefuroxime and its perceived protective effect against PE secondary to enterococci. Cefuroxime is a second-generation cephalosporin with an intrinsic lack of activity against enterococci owing to the production of low-affinity penicillin binding proteins and L,D-transpeptidase.[2,3] This is commonly referred to as the 'enterococci-gap'.

Putting this into the broader picture of the quoted EVS[4] *vs* the Swedish National Cataract Register Study,[5] the *a priori* lack of efficiency of cefuroxime against enterococci explains the higher prevalence of enterococci-induced PE in the Swedish group (29.9% *vs* 2.2%) and is not surprising.

Given that enterococci-induced PE is a considerable problem in the era of intracameral cefuroxime, it seems counterintuitive that the authors' standard choice for topical preoperative prophylaxis is a combination of Polymyxin B and Trimethoprim, both of which have low effectiveness against enterococci. Topical Chloramphenicol would seem a superior choice in this context.

It would have been interesting to know which species of bacteria the cluster of four PEs in 2005/2006 belonged to and whether intracameral cefuroxime had been given.