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Sir,

Realignment of a previously dislocated intraocular lens with a secondary intraocular lens: a rare cause of severe myopia

An 88-year-old man, with a history of a retained right dislocated intraocular lens and secondary lens insertion, presented to our department with reduced vision. He had undergone uncomplicated phacoemulsification with posterior chamber lens insertion in 1996. Two years later a YAG capsulotomy was performed. In 2005, the vision in his right eye had suddenly decreased to 1/60 on Snellen visual acuity testing. Further examination revealed a complete dislocation of his intraocular lens into the vitreous inferiorly. There was no preceding trauma and he did not have pseudoexfoliation. The patient had a secondary sulcus intraocular lens inserted and the dislocated lens was not removed. He was happy with the improvement in his vision.

Three years later the patient re-presented with a 2-week history of reduced vision in his right eye. His right visual acuity was now counting fingers. The sulcus-fixated lens was well positioned, with no obvious evidence of the previously dislocated intraocular lens. Retinoscopy showed a high myopic refraction of approximately −20 D and visual acuity improved to 6/12 with correction. Autorefraction confirmed high myopia in his right eye. On further slit lamp examination it was difficult to identify a double lens. However, YAG lens pitting was visible behind the main lens implant. The pitting was not seen to be mobile with eye movement, suggesting that the original lens may have realigned and was now stable. Ultrasound imaging was equivocal and showed a lens in the vitreous adjacent to the sulcus lens (see Figure 1). The authors cannot fully

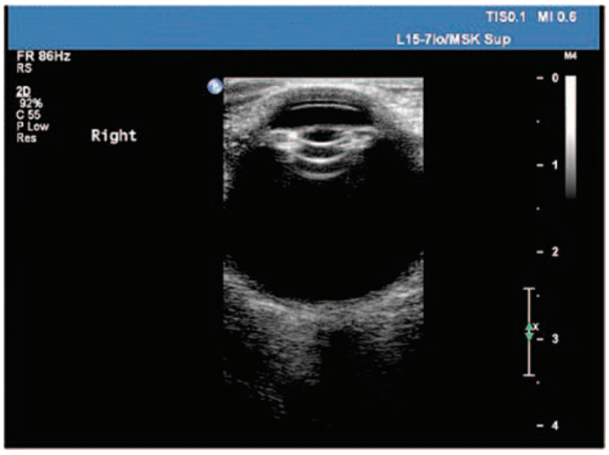

Figure 1 Ultrasound imaging showing a lens in the inferior vitreous.

account as to how the lens relocated and subsequently seemed stable. It is presumed that it lodged on or in the anterior vitreous. Re-dislocation of intraocular lenses and de-centration have been reported,[1] but to the authors' knowledge the realignment of a previously dislocated intraocular lens with a secondary intraocular lens resulting in severe myopia has not been described before.

At the time of investigation the patient was undergoing treatment for other co-morbidities, and a general anaesthetic for further surgery was contraindicated. He unfortunately died due to these co-morbidities, and further help for his unusual symptoms could not be offered.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

A case of diffuse fluorescein leakage not associated with a CNV in *Pseudoxanthoma elasticum*

Pseudoxanthoma elasticum (PXE) is an inherited multisystem disorder that is associated with accumulation of mineralised and fragmented elastic

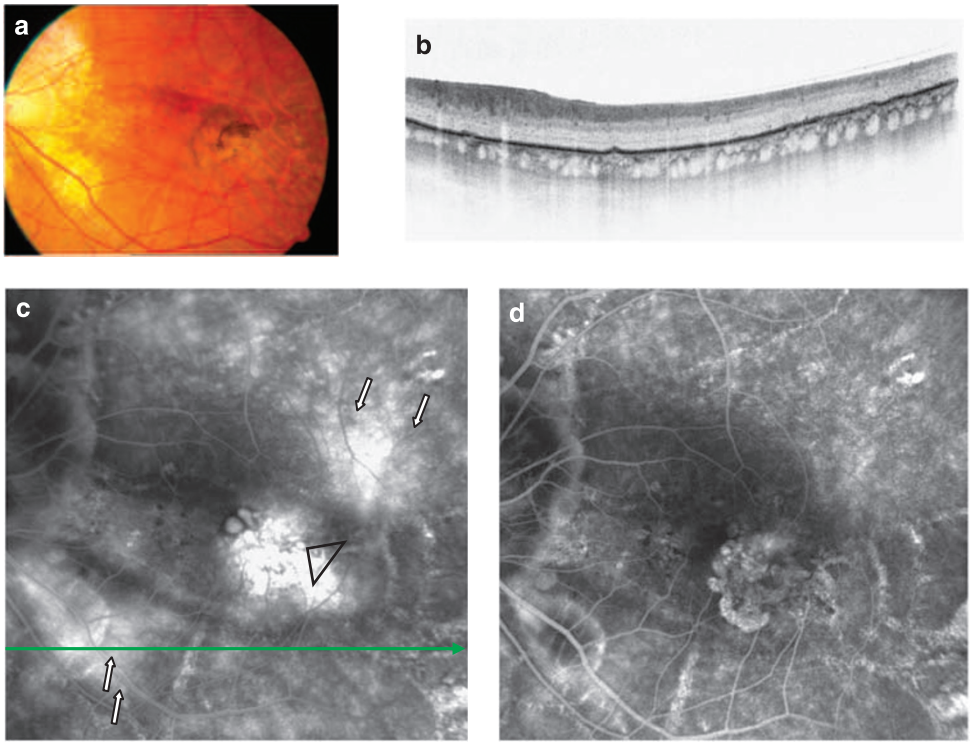

Figure 1 (a) Fundus photography of the patient, showing areas of atrophy and hyperpigmentation adjacent to the papilla and angioid streaks. (c) Late-phase fluorescein angiography shows localised leakage in the lower temporal macula adjacent to an area of atrophy that is visible in (a) (marked by the black arrow head), as well as diffuse leakage, especially in the area of the upper and lower vascular arcade (white arrows); a high-resolution OCT scan ((b), localisation of the scan indicated by the green arrow in (c)) through the lower area of diffuse leakage shows an intact RPE/Bruch's membrane layer and no signs of neovascularisation; (d) 1 month after intravitreal injection of ranibizumab, the localised as well as diffuse leakage is considerably reduced in late-phase angiography (both FAs late phase between 11–13 min).

and collagenous fibres in the skin, vascular walls, and Bruch's membrane in the eye.[1,2] Besides cardiovascular and skin pathologies, patients show characteristic lesions of the posterior segment of the eye, including peau d'orange, angioid streaks, chorioretinal atrophies such as comet tail lesions, and choroidal neovascularisation (CNV). There is yet no causal therapy for the gene defect (*ABCC6* gene on chromosome 16p13.1) and its presumed metabolic consequences.[1] However, the frequently occurring secondary CNVs can now be effectively treated with intravitreally administered anti-VEGF agents.[3]

A 44-year-old female patient suffering from PXE (confirmed by skin biopsy and genetic analysis) complained about gradually increasing blurred vision for the past 3–4 months in both eyes. Her best corrected visual acuity was 20/32 in the right eye and 20/63 in the left eye. An active CNV in the left eye was confirmed on fluorescein angiography (FA) and was treated with an intravitreal injection of 0.5 mg ranibizumab. At follow-up 1 month later, the CNV was inactive and visual acuity had increased to 20/50. Notably, a diffuse leakage at the posterior pole extending just beyond the vascular arcades of the left eye and not associated with the CNV had as well disappeared (Figure 1). On an indocyanin green angiography at baseline, no signs of an occult neovascularisation had been observed within the area of diffuse leakage on FA.

The diffuse VEGF-dependent leakage provides further evidence for a generalised alteration of the Bruch's membrane–retinal pigment epithelium (RPE) complex in patients with PXE. The barrier function of the RPE was shown to be VEGF dependent.[4] Possibly, PXE-associated alterations of Bruch's membrane render the overlying RPE more vulnerable to VEGF effects, resulting in diffuse leakage because of increased VEGF levels.[5] The abnormal VEGF level involved in the development of the CNV may therefore have further increased the RPE-layer permeability in this case.

In conclusion, diffuse fluorescein leakage in the absence of vascular pathology may indicate alterations in the barrier function of the RPE in patients with PXE.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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Sir,

Cystoid macular oedema after selective laser trabeculoplasty

We report a case of cystoid macular oedema (CMO) after selective laser trabeculoplasty (SLT). An 89-year-old Caucasian woman with a previous complicated left cataract surgery was referred to our practice. Visual acuity was 6/12 in the right eye and 6/36 in the left eye. There was right cataract, a left posterior chamber intraocular lens with an absent posterior capsule, and left CMO (later confirmed on optical coherence tomography (OCT)).

She underwent right cataract surgery, while the left eye was treated with topical dexamethasone 0.1% q.i.d. and ketorolac q.i.d. Over 2 years, acuity in the left eye improved to 6/9; however, intraocular pressure (IOP) increased to 30 mm Hg. The topical steroid was ceased and topical brinzolamide (0.1% b.i.d.) commenced. On account of persisting elevated IOP and questionable compliance with brinzolamide, SLT was offered. Pre-laser acuity was 6/9 and IOP was 20 mm Hg on brinzolamide. A 180-degree inferior treatment was performed with 57 applications of 0.7 mJ and total energy of 40 mJ. There was no post-treatment uveitis.

The patient noticed decreasing vision and at 4 weeks acuity was reduced to 6/18. The IOP had improved to 10 mm Hg. Clinically, there was CMO, confirmed on OCT. Ketorolac drops q.i.d. were commenced and brinzolamide continued. Three weeks later acuity had improved to 6/6 with marked resolution of the macula both clinically and on OCT. The IOP remained at 12 mm Hg.

To our knowledge, this is the first reported case of CMO after SLT, which is considered a relatively safe and innocuous procedure.[1–3] As inflammatory processes may be inherent to its mechanism of action,[3] we postulate that upregulation of inflammatory pathways could have triggered a recurrence of the CMO, especially with an absent posterior capsule, allowing greater access of inflammatory mediators to the posterior segment. Other inflammatory reactions to SLT are well recognised, such as ciliary injection and anterior chamber inflammation,[1,2] and there is one report of choroidal effusion associated with severe inflammation.[4]

We recommend caution in using SLT in eyes with a tendency for developing CMO, especially if CMO has been prolonged or was associated with complicated cataract surgery. If SLT is required in such cases, one might consider pre-treating with a non-steroidal anti-inflammatory agent.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Simultaneous bilateral cataract surgery: a further advantage

We welcome the article by Nassiri *et al*.[1] Their paper lends further evidence to the growing body in support of simultaneous bilateral cataract surgery (SBCS).

As Nassiri *et al*. suggest, the main cited objection to SBCS is the very rarely reported (four cases in the literature) risk of bilateral simultaneous infectious endophthalmitis. Conversely, the advantages of SBCS have been repeated numerous times. We hypothesise a further advantage.

If we assume that a patient undergoing SBCS would have a clinic appointment, a biometry and assessment