References

1 Kunavisarut P, Poopattanakul P, Intarated C, Pathanapitoon K. Accuracy and reliability of IOL master and A-scan immersion biometry in silicone oil-filled eyes. *Eye* 2012; **26**: 1344–1348.
2 Retzlaff JA, Sanders DR, Kraff MC. Development of the SRK/T intraocular lens implant power calculation formula. *J Cataract Refract Surg* 1990; **16**: 333–340; Erratum in: *J Cataract Refract Surg* 1990; **16**: 528.
3 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; **327**: 307–310.

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Sir,

Reply to Richard Symes

We appreciate Symes[1] for his interest and comment in our article 'Accuracy and reliability of IOL master and A-scan immersion biometry in silicone oil-filled eyes'.[2] Regarding his comment, the axial length measurements for silicone oil-filled eyes using IOL master showed a high correlation with the postoperative axial length measurement, while the postoperative refractive outcomes varied. We illustrated the plot described by Bland and Altman[3] (Figure 1) following his suggestion and found that few measurements were outside the 95% limits of agreement, resulting in a wide range of predictive postoperative refractive error (− 2.74 to + 2.33 D). But most measurements were in the range, resulting in high correlation (0.966) and a small mean predictive postoperative refractive error (0.60 ± 0.23 D).

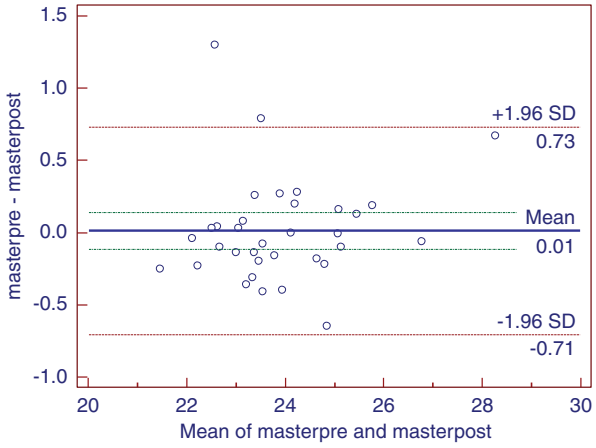

Figure 1 The plot described by Bland and Altman[3] demonstrates that few measurements were outside the 95% limits of agreement, resulting in a wide range of predictive postoperative refractive error, while most measurements were in the range, resulting in high correlation (0.966) and a small mean predictive postoperative refractive error (0.60 ± 0.23 D).

Conflict of interest

The author declares no conflict of interest.

References

1 Symes RJ. Accurate biometry in silicone oil filled eyes. *Eye* 2013; **27**(6): 778–779.
2 Kunavisarut P, Poopattanakul P, Intarated C, Pathanapitoon K. Accuracy and reliability of IOL master and A-scan immersion biometry in silicone oil-filled eyes. *Eye* 2012; **26**: 1344–1348.
3 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; **327**: 307–310.

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Sir,

Disappearance of central confluent soft drusen following vitrectomy and ILM peeling

We report a long-term effective treatment for drusenoid pigment epithelial detachment.

Case report

A 66-year-old woman presented with central confluent drusen, incipient cataract OU, and macular hole OD (Figure 1). BCVA was 20/200 OD and 20/30 OS. The patient underwent macular hole surgery including cataract surgery, vitrectomy, posterior vitreous detachment (PVD), peeling of epiretinal membrane plus internal limiting membrane (ILM) after ICG staining, and gas tamponade. Before ILM staining with ICG, the macular hole was covered with a small PFCL-bubble to avoid any contact of ICG with the RPE. Postoperatively, the macular hole was closed and the confluent drusen almost completely disappeared. Only few small drusen were present 12 months after surgery (BCVA 20/60). Comparison of pre- and postoperative photographs suggests that these drusen were newly formed. The drusen in the untreated left eye increased in size. During the further course of >6 years (75 months, Figure 1) BCVA OD stabilized at 20/25. New drusen developed slowly in a ring-shaped pattern almost completely sparing the center. No signs of atrophy occurred. In contrast, OS developed CNV (BCVA 20/100).

Comment

Central confluent soft drusen, also called 'drusenoid pigment epithelial detachment' (DPED) has a poor prognosis and 42% of eyes progressed to advanced ARMD within 5 years.[1] No effective and overall accepted treatment of DPED is available so far. Regression of drusen has been shown after laser photocoagulation,[2] coincidental rhegmatogenous retinal detachment,[3] and intravitreal anti-VEGF therapy.[4–6] However, there is no evidence that laser photocoagulation reduces the risk of developing CNV, geographic atrophy, or visual acuity loss[2] and larger studies about intravitreal anti-VEGF treatment for DPED are desirable. The here reported patient with pronounced DPED showed remarkable resorption of almost all drusen without developing atrophic changes or CNV after surgery and with stable good vision for >6 years. Spontaneous resorption of drusen has been reported, however, not to the here shown extent and usually correlating with atrophic changes. While the drusen disappeared in the operated eye, DPED progressed OS. Resorption of drusen after macular hole surgery has been reported only once before.[7] The surgical procedure might have stimulated phagocytosis of drusen by macrophages. Stimulating factors might be the surgically induced PVD, peeling of epiretinal membranes, gas tamponade, and increased oxygenation following vitrectomy.[8] The additional ILM peeling in the here reported case might have enhanced the stimulation, resulting in an almost complete phagocytosis of drusen. Remarkably, postoperative new drusen formation spared the central area where compact drusen material had been present preoperatively.

Conflict of interest

The authors declare no conflict of interest.

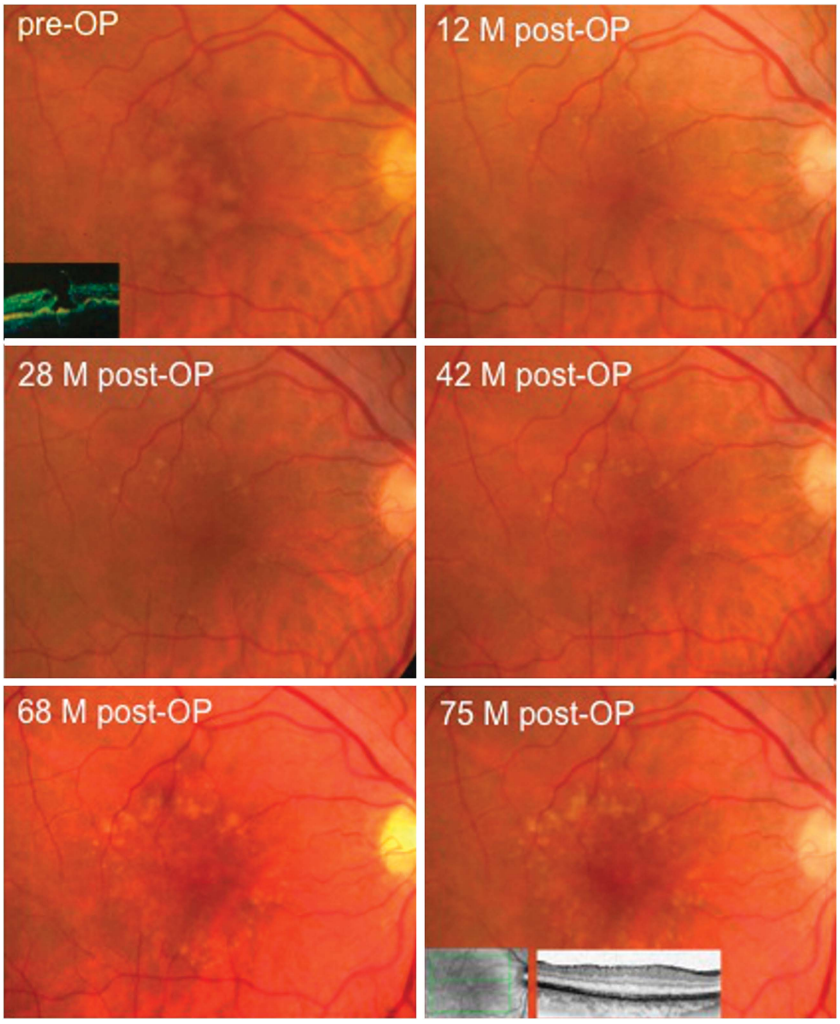

Figure 1 A 66-year-old patient with central confluent drusen and additional macular hole OD. The follow-up of 75 months demonstrate resorption of central confluent drusen postoperatively and slow new drusen formation in a ring-shaped pattern sparing the central area where compact drusen material had been present preoperatively. BCVA increased and stabilized from 20/200 preoperative to 20/25 75 months postoperative.

References

1 Cukras C, Agrón E, Klein ML, Ferris 3rd, FL, Chew EY, Gensler G *et al*. Natural history of drusenoid pigment epithelial detachment in age-related macular degeneration: Age-Related Eye Disease Study Report No. 28. *Ophthalmology* 2010; **117**: 489–499.
2 Parodi MB, Virgili G, Evans JR. Laser treatment of drusen to prevent progression to advanced age-related macular degeneration. *Cochrane Database Syst Rev* 2009; Issue 3: (Art. No. CD006537)10.1002/14651858.CD006537.pub2).
3 Margolis R, Ober MD, Freund KB. Disappearance of drusen after rhegmatogenous retinal detachment. *Retinal Cases & Brief Reports* 2010; **4**: 254–256.
4 Gallego-Pinazo R, Suelves-Cogollos AM, Dolz-Marco R, Arevalo JF, García-Delpech S, Mullor JL *et al*. Intravitreal ranibizumab for symptomatic drusenoid pigment epithelial detachment without choroidal neovascularization in age-related macular degeneration. *Clin Ophthalmol* 2011; **5**: 161–165.
5 Kishore K, Jain S, Sharma YR, Kashyap B. Disappearance of drusen after intravitreal anti-VEGF injections for sub-macular hemorrhage (SMH) secondary to neovascular macular degeneration. *IOVS* 2012; **53**, (ARVO e abstract 2912).
6 Krishnan R, Lochhead J. Regression of soft drusen and drusenoid pigment epithelial detachment following intravitreal anti-vascular endothelial growth factor therapy. *Can J Ophthalmol* 2010; **45**(1): 83–84.
7 Holz FG, Staudt S. Disappearance of soft drusen following macular hole surgery. *Retina* 2001; **21**: 184–186.
8 Stefánsson E. Physiology of vitreous surgery. *Graefes Arch Clin Exp Ophthalmol* 2009; **247**(2): 147–163.

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Sir,

Acute cilio-choroidal effusion due to acetazolamide: unusual posterior involvement (OCT aspects)

Acetazolamide, a sulphonamide-derived medication, frequently used in glaucoma and after cataract surgery, can very rarely cause idiosyncratic reaction, and few reports are present in literature.[1–3]

We report ocular coherence tomography (OCT) scans of the posterior pole in a case of ciliary body oedema after the drug administration causing bilateral angle-closure glaucoma (ACG). In our case we found a massive choroidal effusion with posterior retinal folds and papillary oedema, never described before in literature.

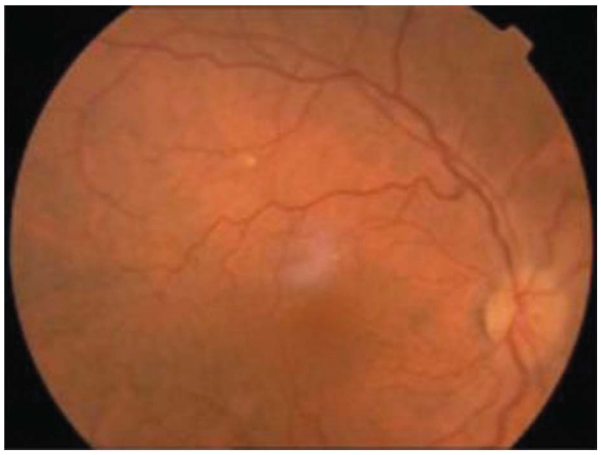

Figure 1 Fundus photo showing evident disc swelling and retinal folds.

Case report

The drug reaction occurred in a 71-year-old white man who was prescribed a single oral dose of acetazolamide (250 mg) after cataract surgery and IOL implantation under local anesthesia.

On examination, both eyes showed congestion and oedema of the inferior bulbar conjunctiva, heavy cloudy cornea, very shallow anterior chamber. IOL was shifted forward. Intra ocular pressure was 52 mm Hg in right eye and 60 mm Hg in left eye. A diagnosis of ACG was made. Fundus examination was characterized by bilateral peripheral choroidal detachment and papillary swelling (Figure 1). Posterior OCT scans confirmed papillary oedema together with retinal folds (Figure 2) and nerve fiber layer thickening.

Acetazolamide has been suspected to be the cause of the secondary ACG, and after it was discontinued the effusion receded rapidly.

Comment

Few cases of acute secondary ACG with choroidal effusion and anterior shift of the iris-lens diaphragm have been associated with acetazolamide compared with other sulphonamides.[1–3]

With regard to the posterior involvement there are only few reports of retinal folds attributed to topiramate and hydrochlorothiazide.[4,5]

Papillary oedema has never been associated with sulpha drugs. Posterior involvement with retinal folds and papillary oedema due to acetazolamide has never been described before.

OCT was able to document this effusion caused by the absence of any barrier in the prelaminar region that could inhibit the diffusion of fluid from the choroid into the papilla and peripapillary region.

Retinal folds were caused by the choroidal effusion contained by the barrier of retinal pigment epithelium and the inextensible scleral coat.

OCT shows the extensive posterior pole involvement and the resolution of the rare adverse reaction