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Sir,
Response to: 'Comment on Central retinal vein occlusion: modifying current treatment protocols'

We would like to thank Călugăru and Călugăru[1] for their comments on our paper, Central retinal vein occlusion: modifying current treatment protocols.

Although bevacizumab is widely used in common practice, there are no large-scale randomized control trials that have studied bevacizumab. Even the quoted paper[1] had a relatively small cohort of 57 patients. It is difficult to extrapolate recommendations especially with regard to long-term outcomes until larger studies have been conducted.

Although the 2 mg ranibizumab dose in the Relate study[2] did show a better anatomical response compared with the other doses, this was not mirrored in visual outcomes. In addition, with the absence of a commercially available 2.0 mg dose and in the context of a visual acuity guided strategy, it would be difficult to advocate quadrupling the dose of ranibizumab.

There have been no large studies that have looked into treat and extend for treating CRVO. However, we did not advocate this particular strategy. We proposed gradually extending the follow-up periods based on the data from HORIZON, which showed that in the second year patients followed up every 3 months post vision.[3] Hence with regard to certain patients following them closely would allow identification of early recurrences. Furthermore, we believe that the mandatory treatments during extension cycles typically reserved for age-related macular degeneration (AMD) is unnecessary in CRVO. In AMD, each recurrence is associated with a drop in final visual acuity as evidenced by a difference in final visual outcomes between monthly and PRN dosing regimens.[4] In diabetic macular edema and in CRVO, the pathology is quite different and as evidenced by the SHORE study, there is no difference between the patients treated using a PRN regimen and maximum monthly dose regimen.[5] Hence with regard to the treatment, PRN would seem to be the 'better' dosing option, and the standard Treat and extend would overtreat a significant number of patients.

The data from Călugăru and Călugăru[1] regarding the use of bevacizumab in cases of ischemic CRVO included 21 patients with ischemic CRVO/HRVO. These data are important, however, was not included because of the relatively small number. It would be interesting to study the effects of bevacizumab in ischemic CRVO on a larger scale.

Switching to aflibercept,[6] although still a relatively novel approach to treating resistant CRVO, has been gaining significant traction in real-world practice. It is a more appealing option than using steroids and there are mounting data that it might be a good option.[7,8] However, this has yet to be confirmed with larger studies conducted in a prospective manner.

Finally, we appreciate the in-depth analysis and the debate with regard to treating CRVO, and we acknowledge that this is a serious disease that requires aggressive and timely intervention to preserve vision.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Stability and safety of MA50 intraocular lens placed in the sulcus

We are grateful to the authors[1] for making this useful contribution to a limited literature, alluding to the fact that an ideal sulcus intraocular lens (IOL) option remains elusive, across a wide refractive range, for cases where posterior lens capsule support is lost.

Of particular interest is the data relating to the prevalence and description of glaucoma in this cohort, with none of the patients in whom optic capture was achieved developing glaucoma. The inference is that of the 38 eyes that did not have optic capture, 9 of these (24%) developed either ocular hypertension alone, glaucoma, or UGH type syndrome (non-neovascular glaucoma cases). It does seem reasonable here to suggest that the risk of further morbidity in these patients is enhanced by the absence of a captured optic, where support was available, exposing them to a greater risk of developing secondary glaucoma.

We do believe that the mechanism of ocular hypertension/glaucoma in this context is primarily an outflow obstruction, stemming from pigment deposition at the trabecular meshwork. This clinical scenario is well described in the literature for both sulcus-placed single-piece acrylic and 3-piece acrylic-PMMA intraocular lenses.[2–4] Indeed, from our own experience, the sequelae here can be significant, requiring aqueous shunt surgery.

In this patient cohort, most notably those without optic capture in whom the IOL would be prone to lateral instability, which included those cases in which ocular hypertension/glaucoma was observed, were no unilateral angle morphology changes observed at the trabecular meshwork consistent with pigment dispersion? One assumes that the clinical phenotyping of these patients was comprehensive, including gonioscopic evaluation? With a relatively short median long-term follow-up period in this study, more cases of pigment dispersion glaucoma may emerge after a longer follow-up period, as demonstrated in other case series.[3] It may be appropriate to counsel patients of this risk.

We believe that the importance of optic capture is understated and ought to attract greater emphasis in the management of phacoemulsification complications. Indeed, this practice was a recommendation of the 2009 ASCRS Cataract Clinical Committee, with Chang *et al*[2] eloquently describing the technique and the mechanisms of advantage.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Response to Dr Sandhu and Dr Clarke

We thank Dr Sandhu and Dr Clarke for their correspondence in regard to our paper on the safety and stability of the MA50 intraocular lens when placed in the sulcus.[1]

In addressing the angle morphology of patients without optic capture of the intraocular lens in which ocular hypertension, glaucoma or iritis was observed, none were diagnosed with pigment dispersion syndrome based on clinical characteristics. Of the eight patients, three had iritis, one had iritis and open angle glaucoma, one had ocular hypertension alone, one had steroid-induced ocular hypertension, one had neovascular