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Sir,
Comment on 'Intrasilicone oil injection of bevacizumab at the end of retinal reattachment surgery for severe proliferative vitreoretinopathy'

We read with great interest the article titled 'Intrasilicone oil injection of bevacizumab at the end of retinal reattachment surgery for severe proliferative vitreoretinopathy' by Ghasemi Falavarjani *et al*.[1] We beg to differ on some of the points though.

Proliferative vitreoretinopathy (PVR) is associated with elevated levels of many pro-inflammatory cytokines and growth factors including, vascular endothelial growth factor (VEGF).[2] All patients were treated with oral steroids and sub-tenon triamcinolone injections. However, the role of oral steroids in preventing PVR changes in an eye with rhegmatogenous retinal detachment has not been proven conclusively.[2] There is no correlation between the levels of inflammatory mediators or growth factors and the severity of PVR and hence an association between them is difficult to prove.[2] Improper injections of anti-VEGF agents can worsen tractional retinal detachment in an eye with fibrovascular membranes.[3] Similarly, inadequate understanding of the role of VEGF in formation of PVR and thereby the role of anti-VEGF agents in the prevention of PVR can prove detrimental. Bevacizumab injection was given before the closure of inflow sclerotomy in this study. We believe that such a practice might result in the leakage of the injected drug through the open port and hence suggest injecting the drug after closure of all the sclerotomies. The use of encerclage, meticulous dissection of all membranes, adequate vitrectomy, and use of Perfluorocarbon liquids and silicone oil are some of the methods to reduce the chances of retinal redetachment. We appreciate the reporting of the results by the authors though the results were contrary to the hypothesis.

Conflict of interest

The authors declare no conflict of interest.

References

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Sir,
Proliferative vitreoretinopathy and antivascular endothelial growth factor treatment

We thank Radke *et al*[1] for their interest in our manuscript.[2] Recent studies have shown a strong role for growth factors in the pathogenesis of proliferative vitreoretinopathy (PVR).[3,4] Vascular endothelial cell growth factor (VEGF) A has been reported to be able to activate the platelet-derived growth factor (PDGF) receptor α, a receptor tyrosine kinase that is key to pathogenesis of PVR.[3] Interestingly, Pennock *et al*[4] reported that ranibizumab protected the rabbits from developing PVR. In contrast to these findings, our results showed that intrasilicone injection of bevacizumab does not eliminate the risk of subsequent PVR and may be associated with subretinal proliferation.[2]

We generally close the eyes after silicone injection with an intraocular pressure (IOP) of around 20 mm Hg. To avoid an increase in IOP after bevacizumab injection, we injected bevacizumab before closure of inflow sclerotomy. Considering that the fluid is heavier than silicone oil and the injections were made in the mid-vitreous cavity, we did not expect to have drug regurgitation.

Several preclinical and clinical studies reported promising results of corticosteroid therapy via systemic, periocular and intraocular routes for prevention of PVR.[5–7] Although the effect is still controversial, we consider corticosteroid therapy as an available and easy-to-use pharmacologic modality in high-risk patients to reduce the rate of subsequent PVR.

We agree with Radke *et al* about the reported detrimental effect from the injection of anti-VEGF agents on 'fibrovascular' membranes. However, such membranes are usually encountered in retinovascular diseases such as proliferative diabetic retinopathy (as depicted in their reference 3). In proliferative vitreoretinopathy the membranes are fibroglial and not fibrovascular.[3] We did not find any previous study indicating detrimental effects from anti-VEGF agents on PVR. Actually this is the exact point that makes our study so unique. We look forward to future studies by other investigators to further elucidate the role of anti-VEGF agents in the management of proliferative vitreoretinopathy.