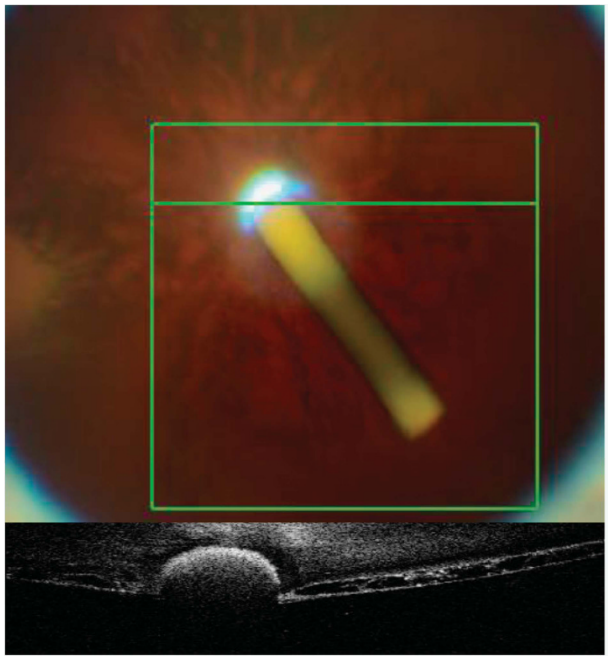

Figure 1 (Upper) Transillumination photograph showing posterior subcapsular cataract and intralenticular Ozurdex implant. (Lower) OCT scan of the crystalline lens showing posterior lens thickening and vacuolation, with the Ozurdex implant embedded within a posterior capsular defect.

to our clinic. The OCT scan confirmed an intralenticular location of the steroid implant with a posterior subcapsular cataract (Figure 1). The OCT scan of the lens confirmed the posterior capsular defects and extent of the cataract. Visual acuity was 2/60 and the OCT scan was unable to penetrate the cataract for macular evaluation. There was no fundus view, and the ultrasound scan showed no retinal breaks. On the basis of the significant cataract, inability to visualise the retina and evaluate the macular oedema, surgery was planned. Intraoperatively, the Ozurdex implant was adherent to the posterior capsule with entry and exit capsular defects present (Figure 1). After nucleus removal, a larger capsule rupture was noted around the Ozurdex implant impact site, and soft lens matter dropped into the vitreous. A complete 23-G vitrectomy was performed, and the Ozurdex implant was resited within the vitreous cavity and a sulcus lens implant inserted. Visual acuity 1 week post surgery was 6/24 with macular oedema, and the patient remains under follow-up at Moorfields. The case by Chhabra *et al*[1] and our report demonstrate two different scenarios for the management of such a rare complication. We believe that the clinical decision to observe or operate early should be based on the ability of the clinician to manage the primary underlying condition of macular oedema, and this requires relatively clear media.

Conflict of interest

The authors declare no conflict of interest.

Reference

1 Chhabra R, Kopsidas K, Mahmood S. Accidental insertion of dexamethasone implant into the crystalline lens—12 months follow-up. *Eye (Lond)* 2014; **28**: 624–625.

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Sir,
Reply to 'Vitreoretinal surgery for inadvertent intralenticular Ozurdex implant'

It is with great interest that we read another case of Ozurdex implant malpositioned in the crystalline lens, as reported by Chalioulias and Muqit.[1]

Although the complication is rare, with increasing use of intravitreal implants, the number of accidental malpositioning in the crystalline lens may also increase.

Cataract formation is evidently the major consideration in such cases, due to the active pharmacological ingredient being a steroid in close proximity to the lens matter in addition to the mechanical trauma. The two cases however suggest a variable pace of cataract progression underpinning relevant management decisions.

We fully agree with the authors that clinical management of each case should be individualized and based on concomitant findings and the development of any side effects.

The authors have taken an approach of early intervention prompted by rapid formation of dense cataract precluding fundal view. In our case, however, gradual cataract progression and media clarity for an extended period of time allowed for an approach of careful watch and wait.

During this course the therapeutic effects of the implant, albeit intralenticular, became quite obvious with resolution of CMO.

As an update on our case, we report no recurrence of CMO at 21 months follow-up with no need for any additional therapeutic intervention.

It was an engrossing case report and we commend the authors on a positive outcome.

Conflict of interest

The authors declare no conflict of interest.

Reference

1 Chalioulias K, Muqit MMK. Vitreoretinal surgery for inadvertent intralenticular Ozurdex implant. *Eye* 2014; **28**(12): 1523–1524.

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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

1 Ghasemi Falavarjani K, Hashemi M, Modarres M, Hadavand Khani A. Intrasilicone oil injection of bevacizumab at the end of retinal reattachment surgery for severe proliferative vitreoretinopathy. *Eye* 2014; **28**(5): 576–580.
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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.