

Figure 1 Three-month post-op. The anterior and posterior capsules are fused at the IOL periphery, and the overall capsule is clear.



Figure 2 Twelve-month post-op. Note the clear posterior capsule centrally and peripherally. There is minimal fibrosis in the anterior capsule. The peppering on the anterior capsule inferiorly, over the IOL optic, was present at 3 months and remains unchanged.

reduction compared with the unoperated left eye of the patient.^{3,4}

Clinical photographs are shown at 3 months (Figure 1) and 12 months (Figure 2). An ultrasound biomicroscope (UBM) image at 12 months is shown (Figure 3).

Comment

The 'specialised' IOLs under development will require changes in surgical technique to effect complete evacuation of lens epithelial cells from the capsular bag to ensure low posterior capsule opacification levels and a flexible capsular bag.⁵

References

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Figure 3 UBM image at 12-month post-op. Note the fused peripheral anterior and posterior lens capsules with no inclusions.

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Diabetic macular oedema and erythropoietin

The study by Garcia-Arumi *et al*¹ was of engaging interest. The authors conclude that erythropoietin (EPO) has a neuroprotective role in diabetic macular oedema (DME). In addition to the limitations of this study, lucidly highlighted by the authors, we demur on some of the methodological constraints of this study.

Diabetes being a chronic multisystem disease with end-organ damage, it would be natural to expect raised serum EPO, especially with coexisting nephropathy and anaemia.² Earlier reports show that the correction of anaemia (and also supplementation of EPO) decrease the effects of retinopathy with structural improvement, possibly through the improved oxygenation of the macula.^{2,3} It would be fallacious then to compare the acute imbalance of retinal vein occlusion (RVO) cases, with DME. Also, EPO level in the DME group was found to be 430 mU/ml (41-3000), and in the RVO group, it was 91.3 mU/ml (30–417). It would have been interesting to note the near normal levels of EPO in some of the DME cases despite the presence of DME. This would probably imply the role of other factors in DME. In this regard, the authors may have overlooked important factors for the production of EPO, such as nephropathy status, Hb levels, and coexisting vascular disease.3

The authors have shown ingenuity in their study; however, we reckon that it would have been better served with a comprehensive outlook of the risk factors being studied.

References

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Sir, Erythropoietin and diabetic macular oedema

We appreciate the comments of Dr Uparkar regarding our article. We agree that serum erythropoietin (EPO) is expected to be raised in diabetic patients. However, we showed that vitreous EPO levels were raised because of intraocular production in our earlier article.¹ Dr Uparkar states that it would be fallacious to compare the acute imbalance or retinal vein occlusions with diabetic macular oedema (DME), a chronic condition. However, other vitreous factors, such as vascular endothelial growth factor (VEGF), are raised in both diseases;^{2,3} hence, we believe our study is not so fallacious. We agree that systemic factors are related with EPO production, but we remind Dr Uparkar that, as mentioned above, intraocular production was responsible for raised vitreous EPO levels.¹

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Sir,

Tonometer disinfection practice in the United Kingdom: the climate implications

We read with interest the article entitled 'Tonometer disinfection practice in the United Kingdom: a national survey,'1 and agree with the conclusion that guidelines for tonometer disinfection in the United Kingdom are necessary. We would like to comment on the article's surprising finding that 23% of ophthalmology units with training recognition have adopted exclusive use of disposable tonometer prisms despite evidence that there is still a risk of transmission of pathogens when using these,² and that they are less accurate than a Goldmann prism.³ In the absence of concrete evidence of reduced risk of transfer of viral and bacterial pathogens and controversy about the small theoretical risk of transmission of vCID,⁴ we must consider the financial and environmental costs associated with the use of disposable prisms. If we assume that an average ophthalmologist sees 4000 patients per annum, the cost of two non-disposable Goldmann prisms and 361 of 0.5% sodium hypochlorite solution would be £244 per annum. The cost for Tonosafe disposable prisms for this number of patients would be £2324 (personal communication, Haag Streit). This amounts to a cost per patient of 58.1 pence for the Tonosafe prism and 6.1 pence for the

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