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Sir,

Spontaneous closure of Nd:YAG posterior capsulotomy in capsular blockage syndrome

We report the spontaneous closure of a posterior capsule 1 week following Nd:YAG central posterior capsulotomy for capsular blockage syndrome, which developed following routine cataract surgery.

Case report

A 68-year-old nondiabetic, highly myopic man (with no other risk factors) underwent uncomplicated phacoemulsification cataract surgery. An MA50BM (Alcon Ft Worth TX) three-piece acrylic lens was implanted into the capsular bag. Postoperatively, capsular blockage syndrome type 2 was diagnosed:[1] A small central Nd:Yag posterior capsulotomy was performed to allow the escape of fluid.

After 1 week later, the posterior capsule opening was found to have closed with recurrence of capsular blockage syndrome. A larger Nd:YAG posterior capsulotomy was created, and the opening confirmed at 1 week. The outcome capsular bag had deflated and the anterior chamber deepened, with posterior movement of the optic, which had become adherent to the posterior capsule. This outcome has not been described as a part of conventional management of capsular blockage syndrome. The opening was patent at 3 months (Figure 1) and fibrosis noted around the opening, with migration of lens epithelial cells.

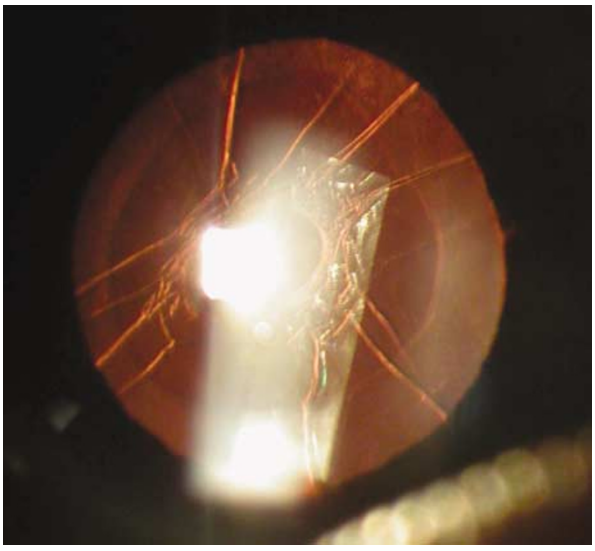

Figure 1 Photograph of lens capsule at 3 months after the capsulotomy.

Discussion

Capsular blockage syndrome is well described, and occurs when viscoelastic is trapped between the lens and the posterior capsule. Intraoperative capsule blockage syndrome is type 1 and postoperative capsule blockage syndrome may be early (type 2, within 1 month) or late (type 3).[1,2]

Masket first reported closure of a posterior capsulotomy with Elschnig pearls along its margin, thought to be due to excessive lens epithelial cell (LEC) proliferation.[3] Kato *et al*[4] reported string of pearls in 47.6% of patients within 1 year after Nd:YAG posterior capsulotomy, but found no increased rate in high myopes. Kurosaka *et al*[5] report a 77% rate of Elschnig pearls at 2 years after Nd:YAG posterior capsulotomy, 20% requiring repeat laser. McPherson *et al*[6] report a 0.7% incidence of re-opacification after Nd:YAG capsulotomy. All affected patients were younger than 50 years at the time of cataract surgery. De Groot *et al*[7] showed that LECs can proliferate on the basal lamina of the anterior vitreous face and close a posterior capsulotomy. Chatterjee *et al*[8] reported capsule re-opacification 8 months after Nd:YAG capsulotomy of a 48-year-old diabetic gentleman following routine phacoemulsification and posterior chamber intraocular single-piece polymethyl methacrylate lens implantation (requiring a repeat Nd:YAG capsulotomy). Oshika *et al*[9] reported the closure of a capsulotomy in the presence of a glistenings with hydrophobic acrylic lens. Surgical capsulotomy closure has also been reported in eyes at risk (uveitic, young adults).[10]

We report spontaneous closure of a Nd:YAG posterior capsulotomy for capsular blockage syndrome 1 week post-surgery, requiring repeat capsulotomy. This may be due to phimosis, but more likely from LEC migration. Our case differs from routine posterior capsulotomy in timing and anatomy. Capsulotomy was performed early when LECs were still stimulated following surgery, and the capsule was not adherent to the optic. It is our experience of human lens capsule culture that LECs will

rapidly migrate across the human capsule within 1 week.[11] Potentially, LECs migrate onto exposed capsule or along the anterior hyaloid face, closing the capsulotomy opening. We recommend that, if a posterior capsulotomy is required in the early postoperative period, consideration be given to perform a larger initial capsulotomy.

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Sir,

Red dots visual field test with blue on yellow & blue on red macula test grid

I read with interest the paper by FH Zaidi and colleagues titled 'The Amsler chart is of doubtful value in retinal screening for early laser therapy of subretinal membranes' (*Eye* 2004; **18**: 503–508).

Their work deserves commendation for bringing confirmation to our clinical observations and experience that 87% of threshold scotomas were not detected by black and white Amsler grid when the field defect is less than 6° (6 squares) on the grid. Authors report that a mere 29% of eyes with subretinal neovascular lesions were detected by high-contrast black-and-white Amsler grid testing. Unfortunately, it is not known what real visual deficits, both in terms of a scotoma as well as visual distortion, that is metamorphopsia, their 100 patients actually had at the time of presentation. In other words, the Amsler grid would detect vision deficit only if there is one, and there may not be anything to detect in some of their patients who passed the Amsler test for they were not checked with threshold perimetry or ideally with Fundus Scanning Laser Projection Perimetry. We also do not know the extent and severity of the organic lesions that was detected by the Amsler grid against the ones that passed the test. I believe it is important for this reason not to conclude that subjective vision tests with alternative macular test grids and colour-contrast tests would continue to be of limited usefulness in the future.

The red-on-black chart, the classical colour-contrast test version, of the Amsler grid is too difficult to be seen (very low contrast) by most patients and creates an unacceptably high false-alarm rate. Furthermore, none of the Amsler grid variations made available to date fully utilized the testing potential of their background at the same time as the foreground grid lines as the stimuli. Most retinal and macular lesions, such as macular degeneration or medication toxicity, cause quantifiable contrast sensitivity loss[1,2] and a blue–yellow defect, whereas optic nerve, chiasmal, and postchiasmal disorders (with the exception of dominantly inherited optic atrophy associated traditionally with a blue–yellow defect) cause a red–green defect.[3–7] Colour field test cards