surgery. It has been suggested that the 25-gauge system may reduce the risk of endophthalmitis owing to the smaller incision size, reduced operating time, lack of foreign-body suture material, and reduced conjunctival manipulation. However, the unsutured sclerostomy wounds may provide a conduit for bacterial ingress and the lower flow-rates of the 25-gauge system (reduced by approximately 6 times[1]) allow bacteria an increased opportunity to gain a foothold perioperatively.

We believe that this case emphasises that postoperative endophthalmitis is still a complication, albeit rare, of this form of vitrectomy surgery.

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Sir,

Anterior capsular phimosis with complete occlusion of the capsulorhexis opening

We describe a case of anterior capsular phimosis with complete occlusion of the capsulorhexis opening following routine phacoemulsification and implantation with an AcrysofÂ® intraocular lens (IOL) (Alcon laboratories, Fort Worth, TX, USA). The patient had no relevant predisposing ocular pathology. Histologically, the occluding membrane was composed of proliferated fibrocytic cells, derived from residual lens epithelial cells within the capsular bag. The extent of this exaggerated response is very unusual in the presence of a hydrophobic acrylic IOL.

Case report

A 90-year-old lady was admitted for daycase cataract surgery. She underwent routine left phacoemulsification and lens implantation with a 22.5 Dioptre AcrysofÂ® MA30 lens. She was noted to have a small pupil of 5 mm diameter, but did not require iris manipulation to carry out the capsulorhexis. Her capsulorhexis was thus slightly smaller than 5 mm but was sufficient to continue with uneventful surgery. Her visual acuity at 1 week was 6/18, which improved to 6/12 after a refraction at 1 month. She had dry age-related macular changes.

At 2 months after surgery she presented for right cataract surgery and was noted to have a marked deterioration in vision in her previously operated left eye. She was only able to see 1/60. Examination of her left eye revealed a markedly thickened anterior capsule with impressive capsular contraction and complete occlusion of the capsulorhexis opening (Figure 1). Arrangements were made to clear the visual axis with a surgical capsulotomy.

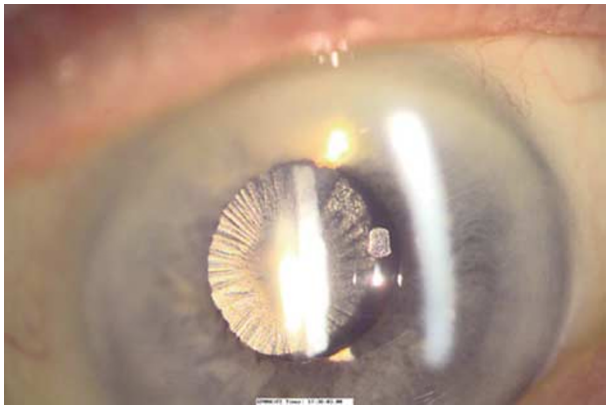

Figure 1 Capsular phimosis with central occluding membrane.

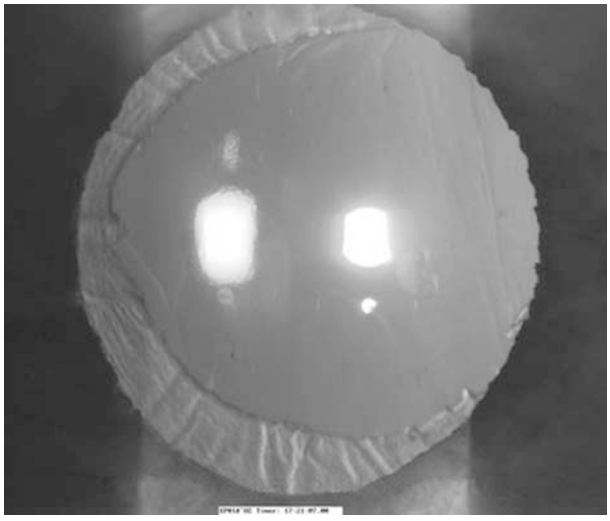

Figure 2 Postoperative appearance after surgical capsulotomy.

She underwent clearance of the visual axis which was achieved using retinal microscissors and a cystotome (Figure 2). The central occluding membrane was particularly thick and difficult to peel away from the capsular bag, although it was not particularly adherent to the anterior surface of the lens implant. The membrane was sent for histology. Following this procedure vision improved to 6/18. She maintained a capsulorhexis opening of 4.5 mm at a 1-month follow-up.

Her second eye was also noted to have a small pupil, which dilated poorly prior to cataract surgery. She underwent successful cataract surgery in this eye and was also implanted with an AcrysofÂ® lens. She was reviewed 2 months after surgery to this eye and a similar anterior capsular phimosis was noted in this eye, although the opening had not become occluded. The capsulorhexis opening had reduced down to 2 mm and was becoming visually significant. Her postoperative visual acuity in this second eye (right) had reduced from 6/12 to 6/24 in a period of 7 weeks.

Pathological findings—Haematoxylin and Eosin staining of the central occluding membrane showed a mass of proliferating cells derived from residual metaplastic lens epithelial cells, seen above and below the lens capsule remnants (Figure 3). They have a fibrocytic appearance and stain positive with cytokeratin (AE 1-3) and smooth muscle actin (SMA) stains (Figures 4 and 5). SMA and cytokeratin stains highlight the cytoskeleton of cells.

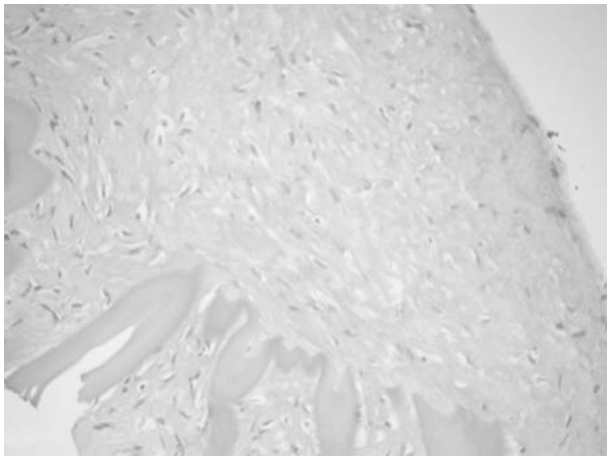

Figure 3 Haematoxylin and eosin staining of occluding membrane (Mag $\times 150$).

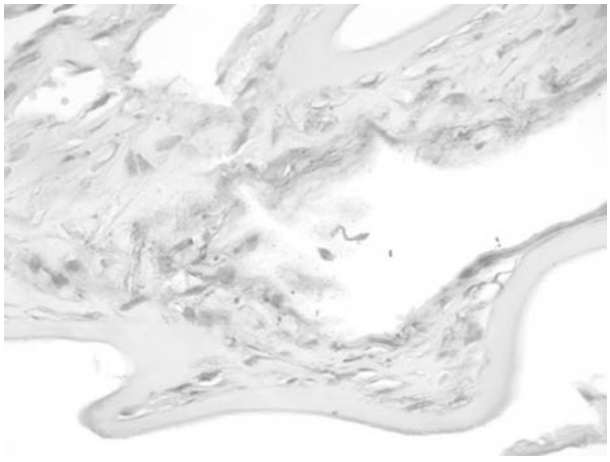

Figure 4 Cytokeratin stain (Mag $\times 400$).

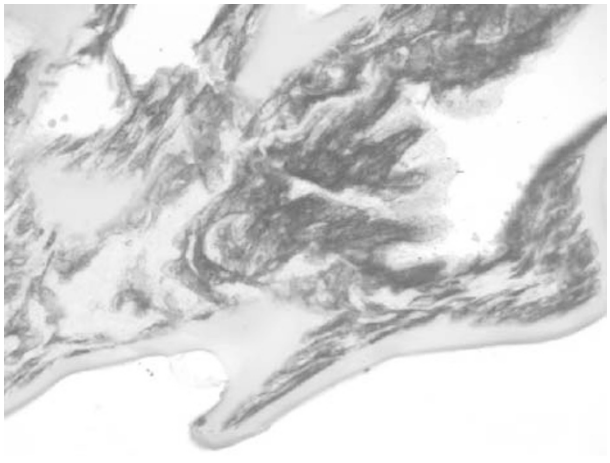

Figure 5 Smooth muscle actin stain (Mag $\times 400$).

Comment

Capsular contraction syndrome is an exaggerated fibrotic response reducing the size of the anterior capsulectomy and capsular bag diameter following extracapsular cataract surgery. It tends not to occur with can-opener style capsulectomies but is often seen following capsulorhexis.[1]

Capsular contraction syndrome has been described following implantation with a variety of types of intraocular lenses (IOLs) including silicone,[2] PMMA,[2,3] and more rarely AcrysofÂ® (hydrophobic acrylic) lenses.[4] It typically occurs in patients with conditions such as pseudoexfoliation,[1,5] uveitis,[1,3] myotonic dystrophy,[3,6,7] retinitis pigmentosa, and factors causing a weakness in the zonular fibres.[3] Its effects include extreme reduction and distortion of the capsulectomy opening, reduction in equatorial capsular diameter, and displacement of the IOL.[1] These effects are thought to be more exaggerated when the capsulorhexis is small. Following capsulorhexis, capsular contraction syndrome may become manifest as proportionally more anterior lens epithelium cells migrate and proliferate over both the anterior and posterior capsules. They then undergo fibrous metaplasia and act to reduce the opening size of the capsule.[3,4] One report analysed the composition of the occluding membrane and determined that it is composed of subcapsular fibrous tissue interspersed with cells. Morphologically and histologically these cells resemble fibrocytes with elongated nuclei and represent metaplasia of the lens epithelial cells or LECs.[3,4] Capsular contraction syndrome probably consists of two mechanisms. The first involves shrinkage of the capsulorhexis, probably due to actin filaments contained within residual LECs. The second mechanism of capsulorhexis closure appears to occur following the proliferation and metaplasia of these residual LECs.

Maximal rate of contraction appears to occur within the first 6 weeks following surgery and tends to be more pronounced with silicone lens implants.[2] The rate of anterior capsular opacification is lowest with acrylic lenses and higher with plate- haptic silicone lenses.[8] Aiming for a capsulorhexis size of between 5.5 and 6 mm along with careful clearance of cortical lens matter is thought necessary to preserve the pupillary zone thus preventing progressive shrinkage of the capsular opening.[9]

This case is unusual in that anterior capsular fibrosis with complete occlusion of the capsulorhexis opening occurred using an AcrysofÂ® lens in a patient with no predisposing pathology. Her possible risk factors for this may have been her smaller-than-ideal-sized capsulorhexis, resulting from a small pupil at the time of surgery and her advanced age. The latter would predispose her to zonular weakness which itself can contribute to anterior capsular phimosis.[3]

Complete occlusion of the capsulorhexis opening is extremely rare and has been previously reported using PMMA lenses in patients with pre-existing ocular pathologies.[3,10] Capsular phimosis without complete occlusion of the capsulorhexis opening has been reported using an AcrysofÂ® lens.[4] Complete occlusion of the capsulorhexis opening in the presence of an AcrysofÂ® lens has been reported once in a patient with pseudoexfoliation.[5] As far as we are aware, this is the first time this phenomenon has been described in the above-described clinical setting. IOL material and design are significant factors in development of anterior capsular opacification and also influence the clinical presentation of capsular shrinkage.[8] It is also possible that capsular phimosis may occur in the presence of smaller capsulorhexes regardless of the IOL material. The phimosed capsule may be cut safely with YAG laser[1,4,5,10,11] or surgically with the use of microscissors[3] as in this case where a histopathological diagnosis was sought.

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Sir,

Posterior scleritis mimicking orbital cellulitis

Posterior scleritis is a common cause of diagnostic confusion because of its variable clinical signs and symptoms.[1] We discuss a case of posterior scleritis that presented with classical signs and symptoms of orbital cellulitis. To our knowledge this clinical picture has not been previously reported.

Case report

A 63-year-old lady presented with a 7-day history of a painful, red left eye and periorbital oedema. There was no history of trauma, precipitating lid lesions or sinusitis. On examination her visual acuity was 6/9 OD, 6/12 OS. She had periorbital oedema and conjunctival injection with chemosis (Figure 1). Left eye movements were restricted horizontally and on downgaze. There was no proptosis. Intraocular pressures and fundal examination were normal, and she was apyrexial. A clinical diagnosis of orbital cellulitis was made and sinusitis was excluded by the otolaryngologist. The patient was admitted and started on intravenous antibiotics.

The following day the chemosis had worsened and the anterior chamber was shallow. Intraocular pressure was 25 mmHg OS and fundal examination showed 360° choroidal effusions. Ultrasound scan showed a choroidal ring detachment and scleral thickening posteriorly (Figure 2). The diagnosis was revised to one of posterior scleritis and the patient was started on oral anti-inflammatories, topical steroids, and mydriatics. Within 24 h the lid oedema and conjunctival chemosis resolved. Systemic investigations showed no abnormality.

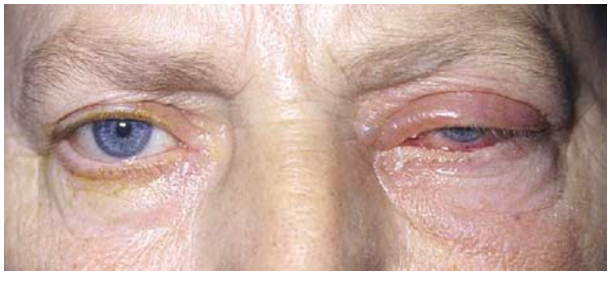

Figure 1 Left eye showing upper lid erythema, oedema, and conjunctival injection with chemosis.

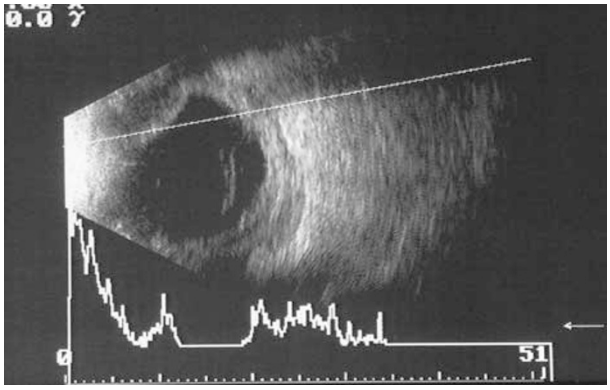

Figure 2 Ultrasound scan showing posterior scleral thickening and an anterior choroidal detachment.

Comment

Orbital cellulitis and posterior scleritis are both potentially life-threatening conditions that require urgent management. The patient described appeared clinically to have orbital cellulitis but was apyrexial with no obvious infective source. This illustrates that caution should be exercised when making a diagnosis of orbital infection in the absence of any obvious cause for or indicators of infection.

Posterior scleritis often presents a diagnostic challenge as it can frequently mimic other pathologies[1–5] and is almost certainly an underdiagnosed condition. It is commonly misdiagnosed because the presenting signs and symptoms are determined by the location and severity of the inflammation and its relationship to surrounding structures.[5] The inflammation appears to have spread anteriorly, involving the upper lid structures causing lid swelling and simulating cellulitis.

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