Further topical therapy was given and the right IOP fell to 32 mmHg, but the patient still complained of feeling unwell. The IOP in his left eye was checked and found to be 52 mmHg with an associated shallow anterior chamber. A diagnosis of acute angle closure glaucoma was made. Further systemic and topical treatment was given and the respective IOPs fell to 31 and 21 mmHg in the right and left eyes with associated relief of the symptoms. A left YAG peripheral iridotomy was subsequently performed.

Comment

The most likely cause of the acute glaucoma in the unoperated eye was the prolonged posturing in the face down position. Indeed, one provocative test for glaucoma is to place patients in the prone position.[2] The mechanism for this is the shifting of the lens-iris diaphragm anteriorly. This shallows the anterior chamber and narrows the angle. In our patient, the problem was compounded by the dilatation of the eye. Although it is unlikely that the episode of angle closure was solely precipitated by dilatation as both eyes had been dilated previously at vitreo-retinal clinic without incident. Also, gonioscopy had found a slightly narrow angle and the axial length was not particularly short. Biometry prior to the cataract surgery found axial lengths of 24 and 23.7 mm in the right and left eyes, respectively. Furthermore, it is unlikely that there was a phacomorphic component as there was no significant cataract in the left eye.

Raised IOPs can, therefore, be found in both the operated and unoperated eyes following pars plana vitreous surgery. Indeed for the unoperated eye this is not surprising, as vitreoretinal surgery often requires prone posturing and dilatation, both of which may precipitate angle closure glaucoma in those at risk.

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Sir,

A case of postoperative candida endophthalmitis

Candida endophthalmitis is a potentially devastating complication of cataract surgery.[1] It is an important opportunistic infection of intravenous (iv) drug abusers and debilitated patients[2] and it is the most common cause of endogenous endophthalmitis.[3] Generally, visual outcome from this disease is poor and candida endophthalmitis requires aggressive management. Therapy is controversial regarding whether surgery is required, choice of antifungal agent, administration route, frequency and what individual and total dosage can be given. We present a case of candida endophthalmitis in which clinical deterioration occurred despite intravitreal and iv amphotericin B, vitrectomy, and lens implant extraction. Subsequent improvement was noted after repeated multiple intracameral injection of amphotericin B.

Case report

An 85-year-old well-controlled noninsulin-dependent diabetic male on latanoprost 0. 005% nocte for open-angle glaucoma underwent uneventful left phacoemulsification with lens implant. A visual acuity of 6/6 was achieved. Vision remained at count fingers in the right eye secondary to glaucomatous damage. A left low grade, painless anterior uveitis subsequently developed and a latanoprost-induced uveitis was considered as the underlying cause. The latanoprost was withdrawn and the uveitis treated with topical dexamethasone hourly and atropine 1% bd. Despite treatment vision gradually dropped to 2/60 and a fibrinous anterior uveitis with a hypopyon developed. Anterior chamber aspiration was preformed and tissue plasminogen 25 μg/0.1 ml given intracamerally in an attempt to disintegrate the hypopyon. Negative Gram stain and culture of aqueous fluid was reported. Visual acuity improved to 6/24 but

then deteriorated to hand movements over 3 weeks. A progressive severe fibrinous uveitis with increased hypopyon developed and removal of the left intraocular lens implant, lens capsule and anterior vitrectomy was preformed. Gram stain and culture of aqueous demonstrated candida. On systemic investigation, candida was also grown from his dentures but, otherwise blood and urine culture and echocardiography was unremarkable. Intravenous liposomal amphotericin B 75 mg o.d. and i.v. flucytosine 2.5 mg q.d.s. was commenced and intravitreal amphotericin B 4 μg/0.1 ml given. After 1 day, an iris fungal lesion was noted and a second intravitreal amphotericin B dose given following a left anterior vitrectomy with partial iridectomy. The fungal colonies continued to develop and a further anterior vitrectomy with a broad iridectomy performed. Vitreous biopsy contained candida and Gram-positive cocci and topical vancomycin 2.5% hourly was added. Over the next 3 days the hypopyon increased and iris fungal seeding progressed. Vision was light projection and a third intravitreal amphotericin B dose given. After 2 days, exudates extended into the posterior chamber and corneal endothelium seeding was noted. A pars planar vitrectomy with excision of any iris remnant was preformed and fourth intravitreal amphotericin B given. There was no sign of chorioretinal involvement. Postoperatively acuity dropped to perception of light and anterior segment fungal colonies persisted (Figure 1). A treatment regime of intracameral amphotericin B injections administered according to the clinical response, usually on alternate days, was commenced. An initial 5 μg/0.5 ml was given followed by 11 subsequent 4 μg/0.4 ml doses. One injection of 2 μg/0.2 ml was given intrastromally into a dense fungal colony that had grown at the limbal incision site from previous surgery. The colonies responded to this treatment and the eye settled (Figure 2). After 3 months left visual acuity was 6/18 with aphakic correction. Left intraocular pressure was 8 mmHg without topical antiglaucoma treatment. There was no sign of corneal decompensation and he had a normal fundal appearance. The patient had remained systemically well throughout and the right eye had remained unaffected.

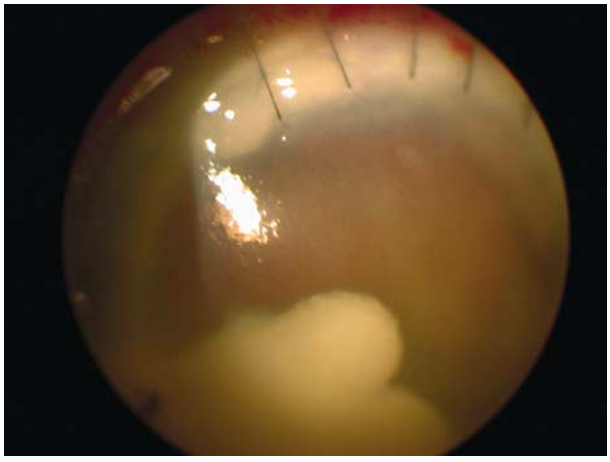

Figure 1 Appearance of eye with candida colonies in anterior segment before commencing intracameral amphotericin B.

Discussion

Candida endophthalmitis is a potentially devastating complication of cataract surgery, the features of which can be misleading. Unlike bacterial endophthalmitis, ocular pain and redness are not typical features of fungal endophthalmitis. At least 50% of patients at the time of presentation with candida endophthalmitis will have a vitreous abscess. The infiltrate in exogenous fungal endophthalmitis is localised frequently, to the anterior chamber, papillary space or anterior vitreous.[4] Treatment with prolonged topical steroids for a painless postoperative low-grade anterior uveitis secondary to fungal endophthalmitis is not untypical.[5,6]

Amphotericin B has low penetration as a topical agent and in patients without signs or symptoms of nonocular candidiasis withholding systemic amphotericin can be considered in view of potential side effects. Our patient's only seeing eye was involved thus initial treatment included systemic antifungal agents. Excision of visibly involved tissue that can act as a reservoir for recurrent infection has been recommended;[2] however, in our case despite iridectomy, lens implant and capsule removal, and pars planar vitrectomy, growth of iris fungal colonies persisted and subsequent secondary colonisation of the corneal wound was noted. Amphotericin B has been used via the intracameral route in severe cases of exogenous candida endophthalmitis[2] and its potential as an intracameral agent has been reported with encouraging results in severe keratomycosis.[5] In a rabbit

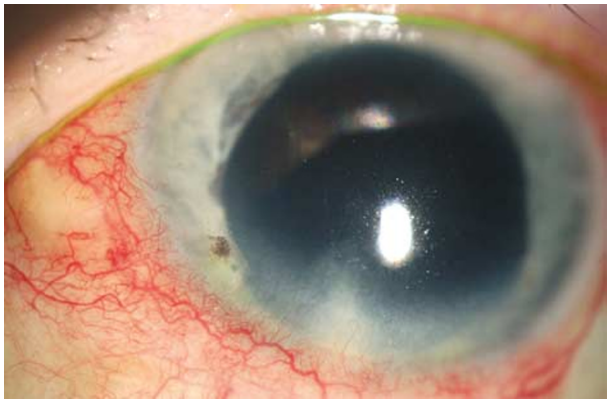

Figure 2 Appearance of eye with resolution of colonies after course of intracameral amphotericin B.

model, as much as 50 μg of amphotericin has been shown not to cause corneal or lenticular toxicity, and liposome incorporation of amphotericin reduces ocular toxicity in the rhesus monkey.[7] In the three cases of severe keratomycosis treated with intracameral amphotericin B the total dose varied from 10 to 25 μg with no clinical evidence of corneal decompensation.[5]

The presence of corneal involvement has been shown to be the single most independent predictor of poor final outcome[8] in exogenous fungal endophthalmitis. In this case, we aggressively managed the patient and a total of 67 μg amphotericin B was given (49 μg intracamerally; 16 μg intravitreally, and 2 μg intrastromally). No visible adverse effects on the cornea or retina were noted and a good visual outcome was achieved. The exact source of the infection is unknown, although colonisation at the time of cataract surgery, endogenous seeding or postoperative self-inoculation by the patient may have occurred. This illustrates a difficult case of candida endophthalmitis that was successfully treated with repeated intracameral amphotericin B.

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Sir,

Atypical manifestation of neurofibromatosis type 2 in a boy

Patients with neurofibromatosis type 2 (NF-2), a rare autosomal dominant disease caused by mutations in the NF-2 tumour-suppressor gene on chromosome 22q12,[1] are predisposed to central nervous system tumours. Paediatric presentation is not yet well defined and differs from adult onset as the diagnostic criteria[2] are often not met at initial clinical manifestation.[3]

This report describes a boy with a trigeminal and a plexiform schwannoma and juvenile cataract without bilateral vestibular schwannomas and lacking a positive family history, who was diagnosed with NF-2.

Case report

A 10-year-old boy was referred by the paediatric ward with a 3-week history of recurrent vomiting, lack of appetite, weight loss, frontal headaches, and inattentiveness at school. Psychiatric assessment indicated an onset of anorexia/bulimia with functional headaches possibly due to social and school problems.

Pre-existing diagnoses were posterior subcapsular lens opacity in the right eye (Figure 1) with amblyopia, esotropia, and dissociated vertical divergence. Strabismus surgery had been performed at 7 years of age.

Figure 1 The left eye presents with a posterior subcapsular lens opacity.