posterior staphylomata and must be differentiated from exudative detachment of the macula.

The macular lesion in our case was a small, round, hole-like lesion with a yellow base. Fluorescein angiography showed hypofluorescence in the centre and hyperfluorescence surrounding it, and this was typical for bull's eye maculopathy. Considering the macular lesions in pathological myopia, this lesion may be a sequela of haemorrhage (Fuchs' spot?) that blocked fluorescence in the centre. There is an RPE atrophy that enables the choroidal fluorescence surrounding the lesion. However, biomicroscopy and OCT scans did not support these findings. Horizontal and vertical optical coherence tomography images obtained through the fovea demonstrated a full thickness defect of the highly reflective band corresponding to the RPE and choriocapillaris. An area of increased reflectivity was observed beneath this defect. The overlying retinal layers were normal. According to this appearance on OCT, our interpretation was a colobomatous defect of RPE and choriocapillaris in the foveal area. Fluorescein angiography findings were in accordance with this lesion. The central hypofluorescent area corresponds to the area of choriocapillaris and RPE atrophy, while the surrounding hyperfluorescence corresponds to the RPE defect only (window defect). If this lesion were a Fuchs' spot, on OCT scans, we would expect to find an irregular RPE and choriocapillaris layer with RPE atrophy surrounding it.

With these findings, we conclude that this is an unusual atrophic macular lesion of pathological myopia having different features with different diagnostic examinations. We intend to follow up this patient in order to evaluate the prognosis.

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Sir,

Late onset group B streptococcus endophthalmitis associated with conjunctival filtration bleb

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Endophthalmitis is a well-recognised late complication of glaucoma filtration surgery. The causative organisms differ from those of acute postoperative endophthalmitis. We present the first case description of late bleb-related endophthalmitis caused by *Streptococcus agalactiae* or group B Streptococcus (GBS).

Case report

A nondiabetic 86-year-old woman presented to the emergency eye service in 2001 with a 1-day history of painful, inflamed left eye associated with marked reduction in visual acuity. She was known to have bilateral advanced primary open-angle glaucoma, which had been treated with bilateral trabeculectomies without antifibrotic agents (right 1998, left 1999), and she had been bilaterally pseudophakic for 12 months. Recently, she had undergone treatment for right cystoid macular oedema. At 2 weeks before presentation, her visual acuities were 6/36 (right) and 6/24 (left), and a slow leak was detected in her left conjunctival surgical bleb. The anterior chambers were deep.

At presentation, her visual acuities were counting fingers (right) and perception of light (left). The left conjunctiva was severely injected. The bleb was opaque with marked corneal oedema and intense fibrino-cellular reaction in the anterior chamber. The intraocular

pressures were 12 mmHg (right) and 43 mmHg (left). A diagnosis of left bleb-related endophthalmitis was made. Vitreous humour was sampled from this eye and ceftazidime 2 mg and vancomycin 1 mg were injected intravitreally. Topical postoperative treatment consisted of cefuroxime 5% hourly, gentamicin 1.5% hourly, atropine 1% tds, timolol 0.5% b.i.d., and prednisolone acetate 1% 2-hourly. Oral ciprofloxacin 750 mg b.i.d. and acetazolamide SR 250 mg qd were also prescribed. Vitrectomy was not performed because of its uncertain benefit in bleb-related endophthalmitis.

After 24 h, GBS was isolated from the vitreous fluid. Topical gentamicin and oral ciprofloxacin were replaced by topical penicillin hourly and intravenous imipenem 1 g t.i.d. However, after 2 days there was no improvement and the vitreous tap was repeated. Vancomycin 1 mg, ceftazidime 2 mg, and dexamethasone 0.4 mg were injected intravitreally. This second sampling yielded GBS on culture again.

At 4 days after presentation, the left eye had no perception of light. The conjunctival bleb had perforated and the cornea was opaque. After 1 month, the eye was phthisical.

Comment

GBS is a well-documented neonatal pathogen, although invasive adult GBS disease is also increasing in frequency. However, GBS remains an uncommon cause of intraocular infections. Endogenous GBS endophthalmitis in adults is usually secondary to meningitis or endocarditis,[1] while endophthalmitis in neonates results from septicaemia.[2] Exogenous intraocular infection with GBS is uncommon and occurs mostly in patients with severely damaged corneal surfaces.[3] A few cases of early postoperative endophthalmitis (postpenetrating keratoplasty or cataract surgery) because of GBS have also been reported.[3,4] Delayed onset endophthalmitis is a well-recognised complication of conjunctival filtration blebs. Although most series report a preponderance of streptococcal species as the causative organisms, these are usually viridans streptococci or *Streptococcus pneumoniae*.[5] To our knowledge, this is the first description of late onset GBS infection associated with a conjunctival filtration bleb. In this case, there was no other focus of infection with GBS and blood cultures were negative.

The optimal therapy for ocular infections with GBS remains uncertain. Intravitreal administration of ceftazidime and vancomycin achieves antibiotic levels that exceeds the minimum inhibitory concentrations for the organism. The Endophthalmitis Vitrectomy Study concluded that treatment with systemic antibiotics conferred no additional benefit in the management of postcataract endophthalmitis,[6] although there was a role for vitrectomy. However, the extrapolation of these results to other categories of endophthalmitis remains a topic of debate.[7] Intravitreal antibiotics may only maintain effective local concentrations for periods of 36–48 h[8] and leakage of aqueous humour through the perforated conjunctival bleb in this patient may have contributed to persistence of the organism in samples taken 48 h following initial intravitreal therapy. GBS remains universally susceptible to penicillin.[9] However, most penicillins penetrate poorly into the vitreous humour. Similarly, intraocular levels of systemic vancomycin[10] and cephalosporins are variable. Although oral ciprofloxacin achieves adequate levels in vitreous humour for the treatment of Gram-negative infections, the concentrations achieved may be subinhibitory for most Gram-positive organisms.[11] Systemic imipenem therapy achieves effective penetration into ocular fluids.[12] Hence it was added to this patient's therapy.

The outcome of GBS endophthalmitis appears to be poor. Late infection in this patient resulted in rapid and complete loss of vision. In earlier reported case studies,[3] three out of the four cases of early postoperative GBS endophthalmitis resulted in a blind eye. Similarly, a case series of endogenous GBS endophthalmitis also reported poor outcomes.[1] Perhaps, bleb revision immediately upon detection of a leak would prevent endophthalmitis in some exogenous cases.

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Sir,

Ciliary body arteriovenous malformation?

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High-frequency ultrasound biomicroscopy (UBM) has been proven useful in the diagnosis and management of anterior segment pathology.[1,2] A case of probable arteriovenous malformation (AVM) of the ciliary body that was imaged with UBM is described.

Case report

A 33-year-old male presented with abnormal redness in the left eye associated with symptoms of itch and discomfort for 5 years. He was most disturbed by the cosmetic appearance of persistent dilated vessels (Figure 1). There was no history of orbital trauma or family history of vascular disorders. Visual acuity (VA) unaided was 20/20 in both eyes. Eye position was neutral with no proptosis. Pupils were isocoric and round, with full extraocular movements. Slit-lamp examination revealed dilated tortuous conjunctival and episcleral vessels in the nasal aspect of the left eye. Intraocular pressures were normal. There was no abnormal iris pigmentation or mass. Fundus examination showed normal retinal vessels. A provisional diagnosis of AVM over the left nasal conjunctiva was made and was initially managed expectantly.

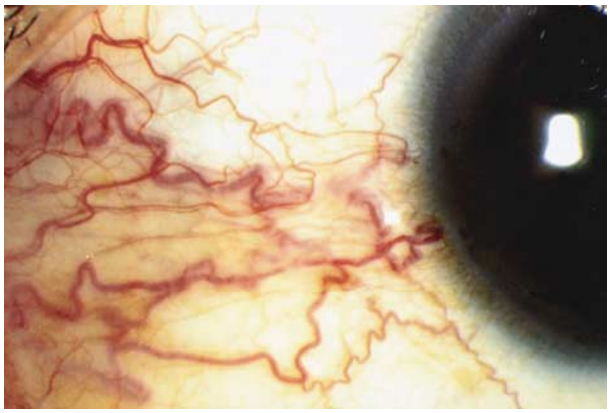

Figure 1 Nasal aspect of left eye.

UBM revealed hypoechoic 'sheets' and tubular structures (Figure 2, arrows) in the ciliary body corresponding to the position of the overlying dilated conjunctival and episcleral vessels and with possible communications between the deep and superficial portions. These hypoechoic areas were shown to communicate. The iris configuration was normal. There was no ciliary body mass or swelling and the ciliary body processes appeared normal. The patient elected for surgical excision for cosmesis, and this was performed. At the time of surgery, the dilated, tortuous vessels were cauterised and excised without difficulty and a free conjunctival autograft was obtained from the superotemporal bulbar conjunctiva and sutured over the nasal defect. The eye was treated with topical antibiotics and steroids postoperatively.

VA was unchanged postoperatively. The patient was happy with the cosmetic result and no recurrence of dilated vessels occurred. UBM repeated 2 weeks postoperatively showed no change in the extent or configuration of the ciliary body hypoechoic areas and no ciliary body swelling or mass.

Comment

An AVM is a dysplastic vascular lesion with the normal capillary bed being replaced by a network of abnormally connected arterial and venous channels. It is a congenital lesion that develops during the late somite stage in the fourth week of embryonic life.[3] Isolated intraorbital AVMs have been described and they usually present with symptoms and signs of proptosis, restricted extraocular movements, limbal chemosis, headache and secondary glaucoma, and dilatation of the veins of the retina, conjunctiva and eyelids. Intraocular AVMs of the anterior segment are rare. They have been described in the iris[4,5] but no reports exist of such vascular abnormalities of ciliary body. Intraocular tumour is an