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Sir,

Bilateral hypopyon and vitritis associated with rifabutin therapy in an immunocompetent patient taking itraconazole

Uveitis is a rare complication of rifabutin therapy when given in conjunction with fluconazole and clarithromycin in patients with mycobacterium avium complex infection.[1,2,3] Typically, these patients are immunocompromised and present with anterior uveitis, hypopyon, and occasionally, vitritis.[4] The pathophysiology of this condition is unknown. We describe a case of bilateral, sequential rifabutin-associated uveitis (RAU) with severe vitritis in an immunocompetent patient taking rifabutin, itraconazole, and clarithromycin.

Case report

A 73-year-old man with chronic obstructive pulmonary disease complicated by mycobacterium avium complex infection and aspergillosis presented with painless, blurred vision OD. Current drugs included clarithromycin 500 mg b.d., rifabutin 300 mg o.d., and itraconazole 200 mg. These drugs were started at the beginning of November 2006.

BCVA was CF OD, 6/6 OS. There was 1 mm hypopyon, fine keratitic precipitates, and severe vitritis. Retinal detail was not visible. The left eye was unremarkable. Endogenous endophthalmitis was suspected and vitreous biopsy with intravitreal voriconazole 100 mcg/0.1 ml and ceftazidine 2 mg/0.1 ml were performed. Postoperative drops were prednisolone 1% q.d.s. and cyclopentolate 1% b.d. No organisms were seen on microscopy or culture. BCVA improved from CF to 6/6 over 8 weeks. All signs of inflammation resolved. There was no evidence of any retinal or choroidal inflammation at any stage.

One day following discharge, the patient presented with an identical history and findings in the left eye as in the right eye and was managed in the same way. The outcome was the same.

Considering the history, a retrospective diagnosis of RAU was made.

Plasma rifabutin levels were elevated (Predose level 0.16 mg/l, 3-h postdose levels 0.44 mg/l and 0.73 mg/l on two separate occasions). Autoimmune markers were negative. Rifabutin and itraconazole administrations were discontinued. There was no recurrence of uveitis at 6 months.

Comment

RAU is well recognised in immunocompromised patients taking fluconazole and clarithromycin, but unusual in an immunocompetent patient taking itraconazole and clarithromycin. RAU responds well to topical steroids and/or cessation of rifabutin therapy. RAU should be considered in immunocompetent as well as immunocompromised patients with an appropriate pharmacological history. Itraconazole as well as fluconazole can be a predisposing feature in this condition.

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Sir,

UBM study in spontaneous bleb formation and blebitis after cataract surgery in Werner syndrome

Werner syndrome is a rare disorder characterized by premature ageing. It is associated with bilateral cataract. Impaired wound healing is a feature of this syndrome.[1]

We reported a female who had presented with this rare disorder and undertook lensectomy and subsequent bleb formation and blebitis several years later. Ultrasound biomicroscopy (UBM) was carried out to evaluate the aetiology of bleb formation.

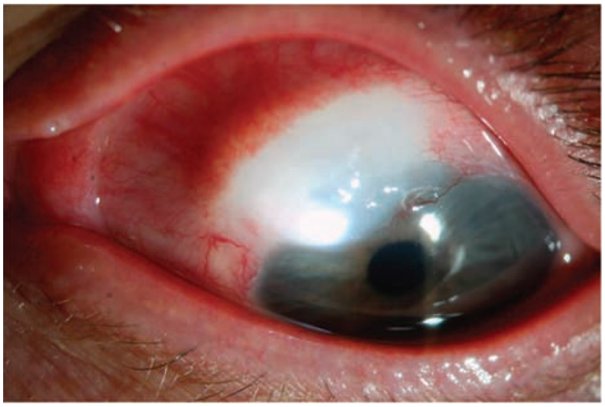

Figure 1 Slit lamp photograph showing a thin cystic white elevated conjunctival bleb with surrounding conjunctival injection.

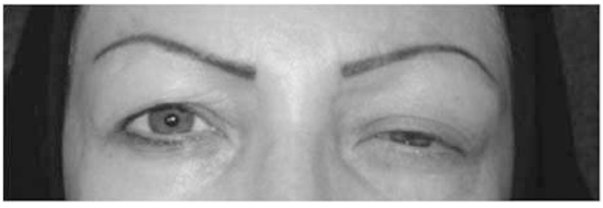

Figure 2 Characteristic atrophic skin, wrinkles and characteristic bird facies of the patient.

Case report

A 28-year-old woman presented with a complaint of a red eye. She had a history of bilateral lensectomy through the scleral tunnel 10 years ago.

Best-corrected visual acuity (BCVA) was 20/200 in the involved eye, with an intra-ocular pressure (IOP) of 4 mm Hg. During slit-lamp examination it was discovered that there was a thin, cystic, white elevated conjunctival bleb with surrounding conjunctival injection (Figure 1). A diagnosis of spontaneous bleb formation after lensectomy with subsequent blebitis was made.

She had a characteristic dermatological pathology (atrophic skin and subcutaneous atrophy) and characteristic bird-like facies of Werner syndrome (Figure 2). Her scalp hairs were thin and she had a high-pitched voice.

Ultrasound biomicroscopy was carried out and it showed the internal and external openings of the fistula and its tract (Figure 3). Blebitis was resolved with topical antibiotics.

Comment

Werner syndrome is a rare autosomal recessive disorder characterized by many features of premature ageing, including scleroderma-like skin changes, premature greying of the scalp hair, bilateral cataracts, and some endocrinologic disorders. The syndrome is most common in patients of Japanese origin.[2]

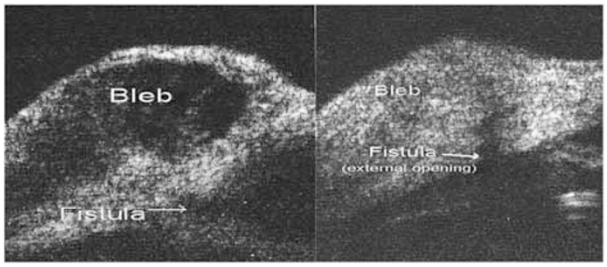

Figure 3 Ultrasound biomicroscopy of patients showing internal and external opening of the fistula and its tract.

Cataract surgery in Werner syndrome may be complicated by wound dehiscence or corneal endothelial decompensation. There are some reports of unplanned filtering bleb in these patients.[3,4]

Two of 18 eyes from patients with Werner syndrome in Jonas's[4] study were complicated with unplanned filtering bleb. He used extracapsular cataract surgery. However, Kocabora[3] showed that Werner's syndrome cataracts can be safely managed with the current phacoemulsification and temporal clear corneal small-incision surgery techniques.

Ultrasound biomicroscopy has been shown to be useful in showing an aetiology of a spontaneous filtering bleb, such as Terrien's marginal degeneration and Axenfeld syndrome, after cyclophotocoagulation or sutured posterior chamber intraocular lenses.[5] UBM might be helpful in differentiating a filtering bleb from a possible conjunctival cyst. It is also useful for surgical planning if deemed necessary.

Ophthalmologists should be careful with the creation of well-constructed wounds in Werner syndrome. By reporting this case, we also highlight the use of UBM to visualize and evaluate the fistula. We recommend the use of UBM for the early diagnosis of the aetiology of bleb and the position of the fistula in this syndrome. Moreover, emergent diagnosis and treatment of presumed blebitis is to be kept in mind.

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Sir,

Transient amaurosis with intracameral lidocaine

Intracameral lidocaine is often used to augment topical anesthesia during ocular surgery. This is a safe and effective method of anesthesia that eliminates discomfort caused by tissue manipulation and thus improves patient cooperation. Although rare, complications can occur. We describe a case of a rare complication with intracameral lidocaine.

Case report

We report a patient who underwent peripheral iridectomy under topical anaesthesia, augmented by intracameral lidocaine 1%. He had vitrectomy for retinal detachment years ago, and vision was 20/70. He underwent a recent repeat vitrectomy with secondary anterior chamber intraocular lens inserted for dislocation of posterior chamber intraocular lens. The surgery was uneventful, but when the dressing was removed an hour later, he noted complete loss of vision. Vision was perception of light with the presence of a relative afferent pupillary defect (APD). Anterior and posterior segment was normal, with no evidence of disc swelling or cherry red spot. Carotid examination revealed no bruit. He reported gradual return of vision over 4 h. Within 20 h, vision improved to counting fingers at 3 m with resolution of APD. Two weeks later, vision recovered to 20/30 with no residual defects.

Comment

Intracameral lidocaine (1%) can result in transient visual loss.[1,2] The recovery period here was similar to the case of Lincoff *et al*,[3] where inadvertent intraocular injection of lidocaine showed improvement in retinal function 4 h later and recovery in 16 h. This is rare but can occur especially in cases of communication with the posterior segment, for example, ruptured posterior capsule and aphakia, where anaesthesia can diffuse readily into the vitreous cavity coming into direct contact with the retina and optic nerve. Visual recovery is complete with no apparent functional damage. Electrophysiological studies in animals injected with intraocular lidocaine[4] show b-waves demonstrated a decrease in the amplitude and an increase in the implicit time. Electroretinogram responses recovered within 24 h.

In the absence of a definite anaesthesia history, only after adequate investigation to rule out vascular or neurological complications, one can attribute amaurosis to intracameral lidocaine.

This case illustrates that intracameral lidocaine 1% is safe to use to augment topical anaesthesia, even when posterior capsule is not intact. The surgeon should be aware that transient amaurosis may occur. Patients can be reassured that it is reversible, although this may take up to several hours to days. In patients with a deficient capsule, or one-eyed patients, other alternatives such as subtenon or peribulbar anaesthesia can be considered.

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Sir,

New fundus findings in a case of Kabuki syndrome

Kabuki syndrome is a multiple congenital anomalies/mental retardation syndrome of unknown cause. Its five cardinal manifestations are characteristic facies, skeletal anomalies, dermatoglyphic anomalies, mental retardation, and short stature.[1–4]

We report a case of tortuous retinal vessels and prepapillary gliosis in Kabuki syndrome.

Case report

A 21-year-old man diagnosed with Kabuki syndrome by the medical geneticist was referred with peculiar optic discs and macula irregularities.

He was born by caesarean section following fetal distress. He was noted to have cleft soft palate, micrognathia (Figure 1), and umbilical hernia. He developed jaundice after birth and was treated with phototherapy. He also had a hypoglycaemic seizure in