large and sited too anteriorly it presents a potential porthole for unwanted foreign bodies.[1,2] There have been numerous case reports of the consequences of injury from hymenopteran insects, caterpillar setae, and insect wings.[3,4] However, we understand that this is the first report of insect entrapment in the subconjunctival space following Sub-Tenon's anaesthesia. Following cataract surgery, patients are instructed not to rub their eyes. This may explain why the insect was not wiped away after initial entrapment. Subconjunctival haemorrhages are common in patients taking oral anticoagulants who undergo Sub-Tenon's anaesthesia, and this presents as an unusual precipitant. Care should be taken when administering Sub-Tenon's anaesthesia to ensure that the conjunctival incision is small and sited correctly so as to minimise the potential complication of foreign bodies as illustrated in this case.

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Sir,

Acute angle closure in Miller Fisher syndrome

Guillain–Barre syndrome is an acute inflammatory demyelinating polyneuropathy often triggered by a preceding infection,[1] of which Miller Fisher syndrome (MFS), characterized by the clinical triad of ophthalmoplegia, ataxia, and areflexia, is considered to be a variant.[2] Nearly half of patients with MFS demonstrate decreased pupillary reactions and mydriasis.[3] A literature search did not reveal any reports of an association between MFS and acute angle closure (AAC). We therefore describe the first case of MFS as the precipitating factor for an attack of AAC.

Case report

A 64-year-old man presented with a 5-day history of ataxia, and diplopia with a preceding malaise and general myalgia. Initial neurological examination demonstrated dysarthria, bilateral positive cerebellar signs, weak proximal muscle groups, hyporeflexia, and hypertonia, with reduced sensation to light touch, vibration, and joint position. Cardiovascular examination revealed autonomic instability with hypertension 210/120 mmHg and tachycardia (100 bpm). Abdominal and respiratory examinations were normal. Ocular movements were reduced in abduction bilaterally, and there was a left ptosis. Pupillary reactions were equal but poorly reactive to light and accommodation, remaining mid-dilated. A diagnosis of Miller–Fisher variant of Guillain–Barre syndrome was made and intravenous pooled immunoglobulins commenced.

At 4 days into his admission, he complained of a reduction in left visual acuity and was referred to the ophthalmology on-call team. Initial examination revealed a vision of hand movements in the left eye and 6/9 in the right eye without improvement, and bilateral sixth nerve palsies. There was mild conjunctival injection, left corneal oedema, bilateral mid-dilated pupils, and intraocular pressures were 20 mmHg on the right and 56 mmHg on the left eye. Gonioscopy revealed closed angles on the left and Schaffer grade 1 angle on the right; the anterior chambers were shallow centrally, the crystalline lens was clear. Treatment commenced with g-timolol BD, g-iopidine TDS, g-latanoprost nocte, and oral acetazolamide 250 mg QDS. The next day, his pressures were 13 mmHg in the right eye and 6 mmHg in the left eye. Bilateral YAG laser peripheral iridotomies were performed. Subsequently, his vision has improved to 6/6 OD and 6/12 OS, with refractive correction of $+1.25$DS/$+0.25$DC $\times 99^{\circ}$ OD and $+4.00$DS/$+0.25$DC $\times 51^{\circ}$ OS. His axial lengths were 22.68 mm in the right eye and 21.60 mm in the left eye with anterior chamber depths being 2.64 mm in the right eye and 2.59 mm in the left eye.

Comment

Primary angle closure results from peripheral iris apposition with the trabecular meshwork, creating an

increased resistance to the outflow of aqueous humour from the anterior chamber in anatomically predisposed eyes. Risk factors for the development of angle closure glaucoma include older age, female gender, narrow angles, Asian ethnicity, and shallow anterior chamber depth.[4] The risk of mydriasis resulting in an episode of AAC is approximately 0.03% after pharmacological dilatation with tropicamide 0.5% and phenylephrine 5%[5] in a normal Caucasian population. We suggest that the constant mydriasis due to pupillary sphincter paralysis in an at-risk, hyperopic eye, with a shallow anterior chamber, resulted in angle occlusion.

Clinicians should be aware that all patients suffering from an autonomic neuropathy, who complain of deterioration in vision, with or without a red eye, should have an ophthalmologic assessment to exclude AAC.

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Sir,

Dematiaceous fungal keratitis presented as a foreign body-like isolated pigmented corneal plaque: a case report

The clinical presentations of Dematiaceous fungal keratitis were not frequently reported.[1–5] Here we report an unusual case of dematiaceous fungal keratitis caused by *Wangiella dermatitidis* presenting as a foreign body-like corneal pigmented plaque.

Case report

A 75-year-old male received penetrating keratoplasty in his right eye in 2001. At 7 months postoperatively, a dust-like foreign body was found on the graft. The foreign body was removed and topical ciprofloxacin was instilled. Bacterial and fungal cultures from the corneal scrapings showed negative findings. After 2 months, the patient complained of foreign body sensation of his right eye. His visual acuity was 20/400 and IOP was 16 mmHg. Slit-lamp biomicroscopy revealed a brownish, elevated, well-defined, pasty, foreign body-like plaque on the cornea surface with surrounding stromal infiltrates and a 1+ anterior chamber reaction (Figure 1). The elevated corneal plaque was removed by a No. 64 Beaver blade and sent for microbial and pathological examination by laboratories of National Taiwan University Hospital. After 3 days, the microorganism grown on Sabouraud dextrose agar revealed yeast-like fungus. Pathologic examination showed numerous fungal hyphae with spore formation (Figure 2). After 2 weeks mold, which was proved to be *W. dermatitidis* by thermotolerance test and positive tyrosine decomposition test, was isolated. Initially, the patient was treated with topical amphotericin B and oral fluconazole. The medications were shifted to topical

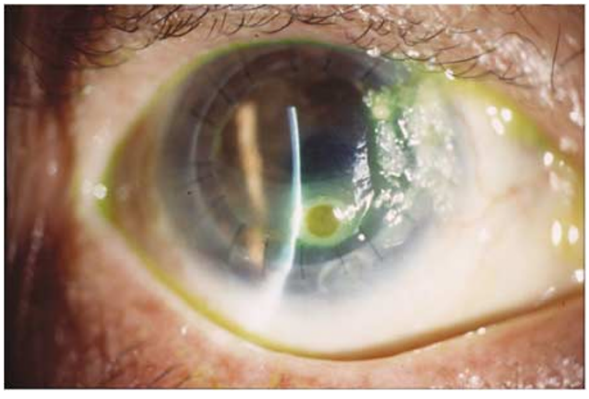

Figure 1 Brownish, elevated plaque with surrounding stromal infiltrates at the lower part of the graft was noted 2 months after a foreign body injury. The plaque was isolated, well defined, evenly coloured mimicking a pigmented foreign body.