Figure 2 Photomicrograph showing well-differentiated epithelium with glassy appearance and a central keratin-filled crater. Scattered neutrophils are present throughout the epithelium with focal microabscess formation (top left), and there is a dense inflammatory infiltrate in the subepithelium. Magnification: $\times 100$, counterstain: haematoxylin and eosin.

infiltrate at the base. Lesions may have invasive features but rarely extend deeper than the sweat coils, and mitoses should only be present in the outer-most layers.⁶ Invasion has been described⁷ and some have suggested that keratoacanthoma is a variant of squamous cell carcinoma. Our case emphasizes the need for a histological diagnosis to be made as the occurrence of keratoacanthoma on mucosal sites and in non-Caucasians is rare, and furthermore cannot be easily differentiated from malignant tumours such as squamous cell carcinoma clinically.

Unlike cutaneous lesions, the natural history of conjunctival keratoacanthoma is obscure because they are excised early. Whether these lesions would regress like their cutaneous counterparts or go on to become invasive is unknown. Of the six or so previously documented cases of definite conjunctival keratoacanthoma, there have been no reports of recurrence after excision with follow-up ranging from 4 to 24 months. However, a noteworthy case, described by Grossniklaus *et al*,⁸ of a rapidly growing limbal lesion with keratoacanthoma features was excised after 3 weeks with evidence of invasion on histology. There was subsequent rapid recurrence with intraocular invasion requiring enucleation. It is unclear whether the lesion in their report was a keratoacanthoma or a very rapidly growing squamous carcinoma, but clearly early excision of a suspected conjunctival keratoacanthoma is recommended in light of this particular case.

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Sir,

Proliferative vitreoretinopathy developed in HLA-B27-associated uveitis

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In Japanese, HLA-B27-associated uveitis is quite rare because of the low frequency of HLA-B27-positive individuals.¹ The main clinical feature is similarly anterior uveitis as that of Caucasian population. Its various lesions in the nonanterior segment include vitritis, papillitis, macular oedema, retinal vasculitis, disc neovascularization and so forth. This type of uveitis usually has a good therapeutic response to corticosteroid therapy.^{2,3} We now report a case of HLA-B27-associated

uveitis where very severe retinal vasculitis resulted in proliferative vitreoretinopathy (PVR). Ophthalmologists should note that a fulminant course can happen in HLA-B27-associated uveitis.

Case report

A 14-year-old Japanese boy noted a blurred vision for 3 weeks in the left eye and was referred to our clinic on 1 February 2001. His best-corrected visual acuity (BCVA) was 6/6 in the right eye and 6/300 in the left eye. Intraocular pressure was normal in both eyes. Heavy ciliary injection, fine keratic precipitates, 2+ cells, 2+ flare, peripheral anterior synechia (PAS) in the anterior chamber, as well as 2+ vitreous cells were noted in the left eye. Both ocular angles were wide open, except for the PAS, with no hypopyon. Fundoscopic examination disclosed ghost vessels in both eyes. Severe retinal vasculitis, pre-retinal haemorrhage, vitreous opacity and scattered exudate were noted in the left eye (Figure 1). Fluorescein angiography (FAG) revealed perivascular staining and leakage from the veins in the left eye. Erythrocyte sedimentation rate (ESR) was 20 mm/h. HLA typing revealed B27 haplotype. Purified-protein derivative skin test was barely positive, but not strong enough to indicate Eales disease. Results of other laboratory tests were normal. Rigorous examination in internal medicine, neurology and dermatology did not detect any symptoms or signs of Behcet's disease, sarcoidosis, systemic lupus erythematosus, and other autoimmune diseases. HLA-B27-associated uveitis was suspected. Both eyes were treated with topical 0.1% betamethasone. For the left eye, additional dexamethasone (6 mg) was subconjunctivally injected daily.

Figure 1 Panfundoscopic view of the left eye at the initial examination.

On 15 February, BCVA in the left eye had increased to 6/30. Retinal vasculitis remained severe and optic disc neovascularization was recognized. He started a daily dosage of 40 mg of prednisolone. On 1 March, retinal vasculitis subsided slightly. However, proliferative tissues along optic disc neovascularization remained unchanged. On 22 March, in the left eye, FAG revealed massive optic disc neovascularization and wide nonperfusion area became evident (Figure 2). Consequently, panretinal photocoagulation was immediately performed. However, on 16 April, traction detachment developed in the left eye. He received the first vitrectomy with silicone oil tamponade. Systemic prednisolone was tapered down to. However, on 6 June, PVR had relapsed and BCVA in the left eye was 6/60. There were no remarkable changes in the right eye. The second vitrectomy, reinjection of silicone oil, buckling and encircling were performed. Additionally, systemic prednisolone was increased to 40 mg. Since this operation, there has been no relapse and prednisolone had been tapered off over a period of 13 weeks. However, BCVA in the left eye has decreased and remained count finger.

Comment

The patient reported here could be one of the most fulminant manifestations developed in HLA-B27-associated uveitis. Systemic corticosteroid therapy could not terminate or decrease the development of neovascularization, resulting in PVR. Laser photocoagulation to the nonperfusion area did not yield any effects on the clinical outcome.

Figure 2 Fluorescein angiogram of the left eye on 22 March. Hyperfluorescence of the optic head and wide avascular area were seen.

Differential diagnosis included Behcet's disease, sarcoidosis, systemic lupus erythematosus, viral retinitis, and Eales disease. He did not have any systemic signs and symptoms such as oral aphtha, genital lesions, and skin lesions. The absence of these systemic symptoms and laboratory results indicative of autoimmune diseases excluded many of the aforementioned diseases, especially Behcet's disease. Viral retinitis will yield more severe exudates than preretinal or vitreous haemorrhage. It is uncommon in Eales disease that anterior uveitis and dense vitritis exist as seen in our case.⁴

This patient and his father have HLA-B27, which is possessed only in 0.8% of the Japanese population.¹ His father had a past history of unknown uveitis. It is quite possible that his disease was also related to the haplotype of HLA-B27. Furthermore, HLA-B27 AAU occurs in B27-positive members of the patients' families (approximately 10%) more frequently than in B27-positive healthy controls (1–2%).⁵ Posterior segment involvement in HLA-B27-associated uveitis occurs somewhere between 4 and 17.4% in Caucasians.^{2,6} Benitez del Castillo reported that the blood–aqueous barrier had been broken when HLA-B27-positive acute anterior uveitis was in active stage.⁷ However, these cases usually have good responses to corticosteroid or immunosuppressive therapy. HLA-B27-associated uveitis in Japanese have also good visual prognoses.⁸ However, there was no report of PVR despite intensive therapy. Our case, thus, verifies the fact that severe and refractory inflammation can happen in HLA-B27-associated uveitis that could lead to the development of PVR in a short period.

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Sir,

Iris microhaemangioma presenting with total hyphaema and elevated intraocular pressure
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Ah-Fat and Canning¹ have described a patient with recurrent visual loss related to spontaneous hyphaema. The source of the hyphaema was thought to be an iris microhaemangioma at the pupil margin and was preceded by ocular pain and amaurosis fugax. We describe a patient with spontaneous hyphaema from a similar vascular iris lesion but *without* any preceding ocular symptoms.

Case report

A 74-year-old lady presented with a 6-h history of sudden loss of vision in her right eye. There was no history of trauma. Visual acuity was 'hand movements'. There was a total hyphaema with no anterior chamber details visible. Applanation tonometry showed an intraocular pressure of 72 mmHg. The left eye was unremarkable. Her general health was good apart from systemic hypertension, which was well controlled on oral atenolol. She had no previous ocular complaints.

The lady was admitted and commenced on oral acetazolamide, topical beta-blockers and fluoromethalone. A full-blood count and clotting