She was treated with topical dexamethasone 0.1% q.i.d. and oral co-trimoxazole 960 mg b.d. but the latter drug was stopped after only 2 days as she experienced side-effects. After discussion with the patient it was decided not to use any alternative anti-parasitic drugs or systemic corticosteroid. The topical dexamethasone 0.1% was discontinued after 3 weeks, as the anterior chamber was quiet. Over the next year the lesion healed and became an atrophic scar. The visual acuity improved to 6/18 with residual vitreous opacities.

Comment

Ocular toxoplasmosis in the elderly is rare. A recent report[1] of 7 patients (3 with acquired disease) aged 69–82 years showed the necrotising retinitis to be atypical and severe, which is in contrast to our patient. Ronday *et al*.[2] previously reported 8 patients aged 42–75 years with focal chorioretinitis presumably caused by acquired ocular toxoplasmosis.

To the best of our knowledge this is the oldest patient with ocular toxoplasmosis who has been reported. The infection was presumed to be acquired despite the ELISA IgM being negative, probably because the test was performed 5 months after the symptoms began. The ISAGA IgM was positive and this is a more sensitive test and can remain positive up to 18 months from the initial infection.[3,4]

A diagnosis of toxoplasmosis should still be considered in an elderly patient in good general health with a necrotising retinitis.

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Sir,

Severe bilateral panuveitis in a patient with asymptomatic Epstein–Barr virus infection

There are many reports that suggest that Epstein–Barr virus (EBV) infection may lead to dry eye syndrome, conjunctivitis and keratitis.[1,2] EBV-associated anterior or posterior uveitis is also occasionally seen. Uveitis has been associated with infectious mononucleosis (IM),[3–5] but also with chronic active infections.[6,7] We describe an unusual case with asymptomatic EBV infection and with severe bilateral acute panuveitis.

Case report

A 58-year-old male teacher developed bilateral visual loss, pain and redness. Athough treated with cycloplegics and topical corticosteroids, his vision decreased within the next 2 days, and he was referred to our clinic. On initial examination, his vision was CF/1 m in both eyes. He had bilateral haemorrhagic conjunctivitis, mild superficial punctate keratopathy, numerous fine keratic precipitates, heavy cellular infiltration and mild fibrin formation in the anterior chamber, bilateral anterior chamber flattening, iris hyperaemia, clear lenses and dense vitritis (Fig. 1). The intraocular pressure was 8 mmHg in the right and 10 mmHg in the left eye. Other than disc swelling, no further details of the fundus were visible because of the vitreous opacities. Conventional ultrasound imaging disclosed bilateral diffuse uveal effusion syndrome, advancing from the ciliary body to the optic nerve head, as well as diffuse serous retinal detachment that was more pronounced in the inferior part of the globe.

The detailed ophthalmic and systemic medical history was unremarkable. The patient was immunocompetent. Extensive laboratory tests, X-ray films of the chest and sinuses were done. Neurology, ENT and internal medicine specialists were consulted. An anterior chamber tap from the right eye was performed in order to determine VZV, HSV, EBV and CMV DNA by the polymerase chain reaction (PCR) method.[8]

Intravenous corticosteroids were instituted with 500 mg prednisolone acetate daily for 3 days, and this

Fig. 1. *Patient with Epstein–Barr virus infection and with anterior chamber inflammation and vitritis.*

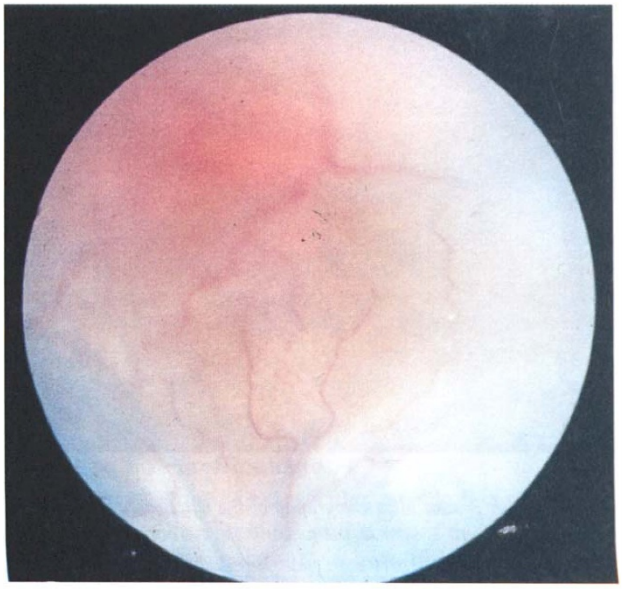

Fig. 2. *Patient with Epstein–Barr virus infection and bilateral panuveitis. There is uveal effusion and serous retinal detachment.*

was followed by oral prednisolone, starting with 1 mg/kg daily. This was combined with 2 g intravenous cefacoline (Kefzol, Lilly) daily. Under this regimen, the pain disappeared promptly. Within 1 week the vision improved to 0.1 and 0.2 in the right and left eye, respectively. Conjunctival haemorrhages resolved, cells in the anterior chamber and vitreous decreased, and the uveal effusion and retinal detachment that were visible now by ophthalmoscopy, markedly flattened (Fig. 2).

At this time, the results of the diagnostic tests were available. The anti-EBV antibody titres were increased: EBV IgM 1:160, EBV IgG 1:1.320, EBNA-specific IgG 1:320 (as detected by ELISA method; normal: negative), but titres for viral capsid antigen (VCA) and Epstein–Barr diffuse components (EA-D) were negative. ANA on Hep-2 cells was increased with 1:160 (normal: <1:80). Blood cell counts and the results of all other tests and consultations were unremarkable. The PCR analysis of the aqueous humour was positive for EBV DNA, but negative for the other herpes viruses.

The antibiotic therapy was discontinued after 7 days, and prednisolone was tapered off within 6 weeks. The vision improved to 0.8 in both eyes, the signs of intraocular inflammation disappeared biomicroscopically and ophthalmoscopically, no uveal or retinal abnormalities were detected by ultrasound imaging, and the intraocular pressure returned to normal levels (14–16 mmHg). Only mild opacities remained in the vitreous body. Serum EBV IgM was negative, while VCA IgG and EBNA IgG were positive.

Comment

Our case demonstrates that EBV infection should be added to the differential diagnosis in patients with severe bilateral panuveitis even when the clinical signs of EBV infections are not present. The increased titres of IgM and IgG antibodies directed against EBV and EBNA suggest recurrence or reinfection of EBV infection. It has been suggested previously that vitritis, retinal vasculitis, retinal haemorrhages and disc swelling might be associated with acute EBV infection.[5,9] Other authors have suggested that multifocal choroiditis and panuveitis may also occur in patients with chronic EBV infections.[6,7] In contrast to our patient, however, the acute lesions appeared as grey-to-yellow retinal infiltrates with a size between 50 and 500 μm, and the lesions healed with discrete punched-out chorioretinal atrophic scars.[7] Punctate outer retinal involvement has also been seen with acute IM.[5] Like our case, most of these patients had no clinical signs of IM.

This case report suggests that an analysis of aqueous humour by the PCR method appears to be helpful in detecting EBV infection in the eye.[8] This may be by direct invasion of the ocular tissue, or via circulating B-cells that had been infected with EBV and infiltrate the eye during inflammation.

EBV-induced uveitis is mostly self-limited, and treatment is not indicated in most cases. However, we suggest that high-dose corticosteroids should be used to treat severe panuveitis cases in order to avoid secondary complications.[6,7]

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