B virus infections. These include chorioretinitis,[1] optic papillitis[2] and panuveitis.[3] We describe a case in which scleritis was associated with prolonged systemic upset. Coxsackie B5 was cultured from mucous membranes.

Case Report

A 32-year-old man presented in late summer with a 5 day history of headache, myalgia and fever of sudden onset. His eyes had become red and photophobic within 24 hours of the onset of symptoms. His general practitioner had prescribed erythromycin and co-dydramol. His past history was unremarkable, and he had not been abroad for over 1 year.

On examination he was pyrexial (38°C). There was generalised muscle ache and tenderness. Apart from sinus tachycardia (95 beats/min) the cardiovascular, respiratory and alimentary systems were normal. He was seen by an ophthalmologist who diagnosed uveitis and started treatment with topical steroids. Erythromycin was stopped.

Investigations

A full blood count revealed: haemoglobin 12 g/dl, white cell count 9.1×10^9/l (monocytosis 2.1×10^9/l), platelets 143×10^9/l. The erythrocyte sedimentation rate was >100 mm/hour. Serum chemistry was normal. The chest radiograph was normal. The angiotensin converting enzyme level was normal. Viral cultures of the pharynx, conjunctiva and perianal region all grew Coxsackie B type 5.

The patient's systemic symptoms gradually improved but his eyes remained painful. A further ophthalmic assessment 2 weeks after presentation showed sclerouveitis, the uveitic element being very mild. The vitreous was free of cells, but in each retina there was a single cotton wool spot, and a small peripheral haemorrhage in one. Acuity was 6/12 in each eye, but there was no disc or macular oedema. Treatment was begun with ibuprofen, 400 mg four times daily, and topical steroids continued. Symptoms and signs began to resolve rapidly. Acuity was 6/6 in each eye after a week. After 2 weeks the patient stopped the ibuprofen, but improvement continued until a mild relapse of scleritis occurred after another 11 days. Resolution again followed reintroduction of ibuprofen; this was gradually withdrawn with the topical steroids over the next 10 weeks. Repeat viral studies, blood count and erythrocyte sedimentation rate were normal after 1 month. The patient made a full clinical and ophthalmological recovery.

Discussion

Of the viruses well known to produce intraocular inflammation most belong to the herpes family[4] and are increasingly recognised against a background of immunosuppression. Coxsackie virus belongs to the enterovirus family, which includes Coxsackie A and B, poliovirus, echovirus and enterovirus serotypes 68–72. They are associated with a range of febrile illnesses, and enterovirus type 70 and Coxsackie virus A type 24 cause acute haemorrhagic conjunctivitis (AHC). This emerged in 1969 and has since reached pandemic proportions in many parts of the world.[5,6] Several outbreaks of severe uveitis associated with echovirus type 11 infection in infants have been reported from Russia.[7,8] The present case has many of the features of an enteroviral infection, including the systemic symptoms and occurrence in the summer months. We believe it is the first recorded association of Coxsackie B and scleritis. The almost simultaneous onset of ocular and systemic symptoms suggests that the scleritis resulted directly from the viraemia, presumably from induced vasculitis. Fluorescein angiography was not indicated in this case, but might have provided more evidence of vasculitis in the eye.

Although most enterovirus-related ocular disease has been hitherto sporadic, the epidemic enteroviral uveitis described in Russia was of a particularly severe pattern: in one series 43 of 56 children had permanent damage.[7] Enteroviruses tend to cause epidemics. New associations with various serotypes are continually being described, and often spread rapidly (cf. AHC). If enteroviral uveitis or scleritis causing long-term damage occurred in numbers similar to those of AHC there would be serious health care implications.

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Sir,

'Roller-Coaster Glaucoma': An Unusual Complication of Marfan's Syndrome

A 32-year-old man had been diagnosed as having Mar-

fan's syndrome at the age of 1 year when he was found to have bilateral subluxated lenses and a strong family history of the condition. At the age of 8 years a right intracapsular cataract extraction was performed due to pupil block glaucoma. Despite a freely mobile intact lens in the posterior segment, the left eye developed no other complications. He required no further surgical or medical treatment and remained symptom-free for many years.

He subsequently presented to the casualty department in May 1993 with a 4 day history of blurred vision and floaters in the left eye. The onset of his symptoms coincided precisely with a series of roller-coaster rides at a theme park. On examination the left visual acuity was 6/60 compared with 6/9 recorded 1 month previously. The eye was injected with corneal oedema and an intraocular pressure (IOP) of 46 mmHg. There was a moderate anterior and posterior uveitis with the lens lying inferiorly in the vitreous cavity. Ultrasound scan showed two distinct spherical masses, one at the extreme periphery at 7 o'clock and one at the equator more posteriorly. Leaning forward caused the more posterior mass to enter the anterior chamber. The impression was that the capsule was fixed inferiorly and separate from the mobile nucleus (Fig. 1). This compares with a scan taken at the last outpatient visit, 1 month previously, showing the intact lens to be adherent to the retina (Fig. 2). The patient was treated with topical steroids, timolol and oral acetazolamide, which resulted in the IOP returning to normal and the visual acuity improving to 6/9 within 24 hours. Over the course of the next 5 weeks the IOP fluctuated, despite continued timolol treatment, with a low-grade persistent iritis and vitritis. A vitrectomy was subsequently performed to remove the lens.

Discussion

The ocular abnormalities of Marfan's syndrome are well known.[1] Lens subluxation, which is usually bilateral, symmetrical, non-progressive and upward, occurs in up to 80% of cases. A patient with ectopia lentis may be followed for many years without significant ocular problems.

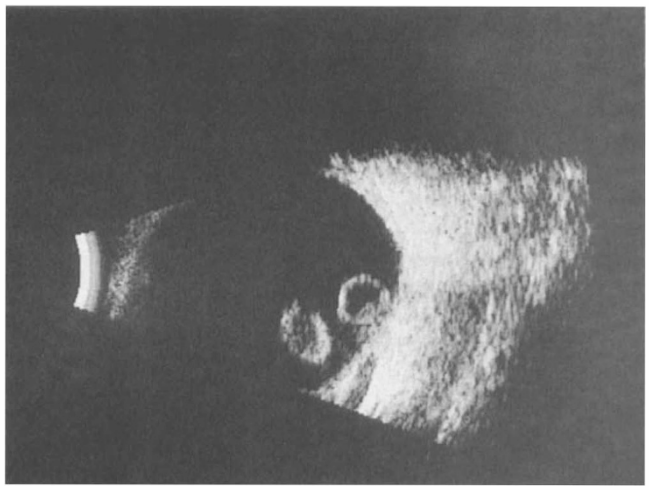

Fig. 1. *B-mode ultrasound scan of the left eye shows the fixed lens capsule separate from the nucleus which was mobile.*

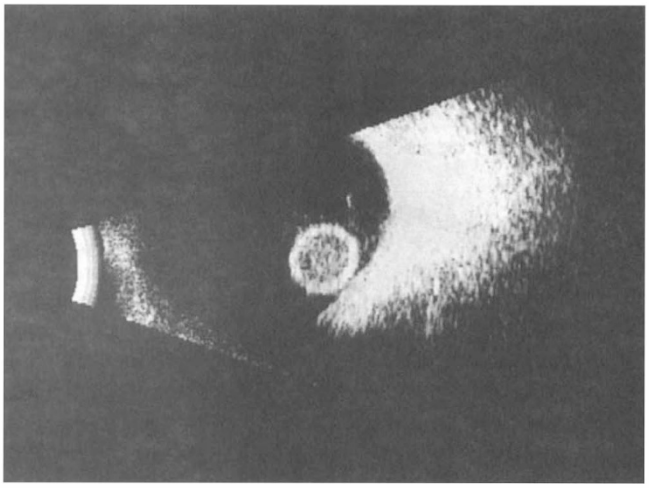

Fig. 2. *Previous B-mode ultrasound scan of the left eye shows the intact lens dislocated into the inferior vitreous cavity.*

However, ocular complications associated with displacement of the lens are unfortunately frequent and often serious.[2] The associated abnormalities include amblyopia, uveitis, glaucoma, cataract formation and retinal detachment.

Uveitis may occur in ectopia lentis by two different mechanisms. Iridocyclitis may result from contact irritation of the ciliary body or iris.[3] This type of uveitis is often acute, recurrent and is usually responsive to topical steroids. A posterior dislocation is usually well tolerated for years provided the lens capsule does not become permeable or rupture, allowing lens protein to escape.[4] If this occurs, a phacolytic uveitis with or without a secondary glaucoma may result, requiring a cataract extraction.[5,6]

The mechanism of glaucoma may also vary. It may result from outflow obstruction by chronic inflammatory cells from a lens-induced uveitis,[7] macrophages from a phacolytic glaucoma,[5,8] or vitreous forced into the anterior chamber angle.[9] It may also occur from pupillary block, or by dislocation of the lens into the anterior chamber. Iridectomy is often the treatment of choice; it can prevent or cure the pupil block and simultaneously provide a clear optical area.

In this interesting case, the patient remained symptom-free for years despite the presence of his lens in the vitreous cavity. During the course of the roller-coaster rides he was subjected to forces of acceleration of up to 3.5 *G*, which effectively acted as a mechanical 'centrifuge' with regard to the lens within the vitreous space. This resulted in disruption of the vitreous gel and also damage to the lens capsule, giving rise to separation of the nucleus from the capsule. The secondary rise in IOP was inflammatory in nature, most probably with an additional phacolytic element as well.

Ectopia lentis continues to be a difficult management problem for the ophthalmologist. Emphasis should be placed on preservation of sight and the globe by prevention and amelioration of complications. Trauma may precede the migration of a previously subluxated lens into the anterior chamber or vitreous, and these patients are also

more likely to develop retinal detachments following trauma. They should therefore be advised against participation in contact sports,[10] and in the light of this report they should also be aware of the potential risks of other vigorous activities such as high-speed fairground rides.

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Sir,

Management of Intraretinal Metallic Foreign Bodies Without Retinopexy

Foreign bodies embedded in the retina and choroid comprise a distinct subtype of retained intraocular foreign body (IOFB). The management of these foreign bodies necessitates the use of vitrectomy surgical instruments and techniques in conjunction with IOFB forceps or an intraocular rare earth magnet.[1-3] Retinopexy using laser photocoagulation or cryo around the retinal break caused by the foreign body is often assumed to be necessary.[1,2]

Retinopexy can be difficult or dangerous because of surrounding retinal, subretinal or choroidal haemorrhage, or the proximity of the IOFB to the optic disc and macula. I describe here the management of five retained metallic IOFBs which were embedded in the retina and choroid but without retinal detachment. These cases were managed by vitrectomy techniques without retinopexy around the retinal break caused by the foreign body.

Case Reports

The 5 patients were all male, ranging in age from 10 to 37 years, and all the IOFBs were caused by the use of a hammer on a chisel or metal. All the patients had corneal entrance sites with traumatic cataract and the corneal lacerations were repaired prior to referral. The time between occurrence of the trauma and surgery ranged from 5 to 12 days.

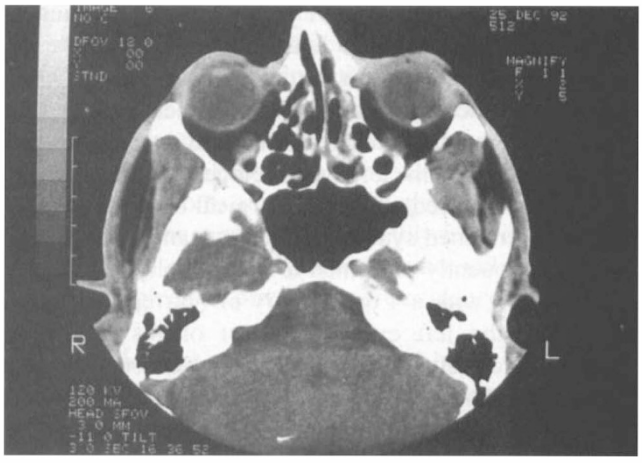

Fig. 1. *Computed axial tomogram showing an intraocular foreign body embedded in the posterior eye wall.*

All eyes had standard radiographs of the orbit to confirm the presence of the foreign body. Computed tomographic scans demonstrated IOFBs embedded in the posterior eye wall (Fig. 1). B-scan ultrasonography demonstrated flat retina in all 5 cases.

A pars plana lensectomy and vitrectomy were performed. The preretinal blood was evacuated using a Charles flute needle and the embedded foreign bodies were seen surrounded by a grey-white capsule. The IOFBs were located posteriorly outside the arcade vessels in 3 eyes, inside the arcade vessel in 1 eye and along the upper temporal artery one disc diameter from the optic disc in 1

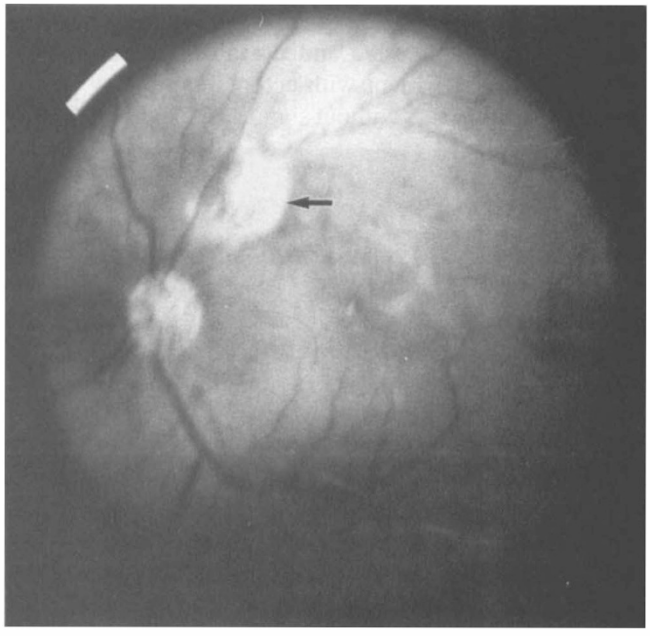

Fig. 2. *Chorioretinal scar at the foreign body impaction site on the upper temporal artery (arrow).*