LETTERS TO THE JOURNAL

Sir,

Recurrent Visual Loss Secondary to an Iris Microhaemangioma

A 55-year-old woman presented to our casualty department in August 1993 with a history of sudden loss of vision and mild discomfort in the right eye shortly after waking up. She described her vision as being roughly hand movements, followed by rapid improvement half an hour later. She had had three similar episodes in the previous 6 months, each occurring shortly after awakening. On two occasions she had presented to the casualty department some 5 hours later and a 'mild iritis' was diagnosed. However, in this particular episode she had also noticed 'blood in the eye' on inspection in the mirror. There was no history of trauma or associated cardiac or neurological symptoms. Her general health was good.

At the time of examination 2 hours later, her visual acuity had already improved to 6/6. There was a 1 mm hyphaema in the right eye with a blood clot at the 6 o'clock position of the pupillary margin (Fig. 1). Intra-ocular pressures measured 43 mmHg in the right eye and 18 mmHg in the left with open angles. Ocular and physical examination revealed no further abnormality.

After treatment with acetazolamide and a topical beta-blocker, her intraocular pressure stabilised with complete resolution of the hyphaema by the next day. At the site of the previous clot on the pupillary margin, a small tuft could be seen at high magnification. There was also a small iris remnant on the lens capsule, which disappeared over the following few days. A full blood count, clotting screen and urine analysis were normal.

It seems likely that the source of the hyphaema was an iris microhaemangioma at the pupillary margin. Larger vascular malformations of the iris have previously been documented by Fuchs.[1] Cobb *et al.*[2] described over 100 cases of 'vascular tufts' at the pupillary margin. They found an association with diabetes mellitus and myotonic dystrophy. These tufts occasionally bleed and because of rapid resolution, may mimic amaurosis fugax. They are thought to represent microhaemangiomas and fluorescein studies have revealed them to be more numerous than can be detected by direct inspection.[3]

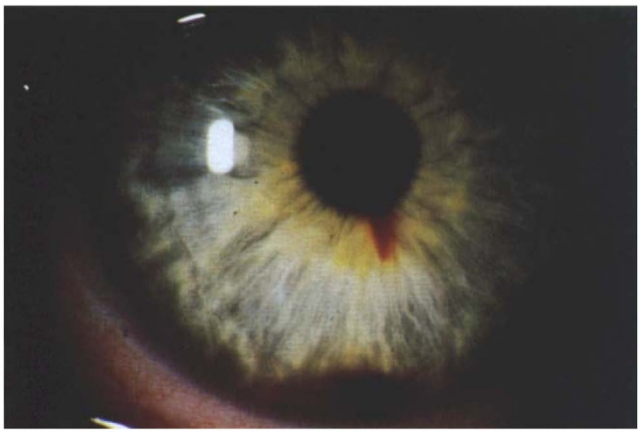

Fig. 1. *The right eye showing a 1 mm hyphaema and a blood clot at the 6 o'clock position of the pupillary margin.*

This particular case serves to remind us that in any patient who complains of transient loss of vision suggestive of amaurosis fugax but who is not examined very soon after, an iris microhaemangioma ought to be considered, especially if a few cells are later detected in the anterior chamber. Its recurrent nature shortly after awakening has not, to our knowledge, previously been described and we wonder whether the formation of a small posterior synechia coupled with pupillary dilatation following sleep miosis may account for this type of presentation. The patient may describe seeing 'blood in the eye' on looking in the mirror. A history of erythropsia or red desaturation may suggest the presence of blood in the anterior chamber. Gonioscopy may be useful in revealing a small resolving hyphaema. Unnecessary investigations and treatment of the carotid circulation may thus be avoided.

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Sir,

Scleritis Associated with Coxsackle B Type 5 Infection

A variety of ocular complications has recently been described as accompaniments, usually rare, of Coxsackie

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-