particularly in the right eye. Two months following laser photocoagulation and 6 weeks after commencement of warfarin, the disc new vessels had regressed. Six months into follow-up, his condition is stable.

Comment

Antiphospholipid antibodies were first detected as a result of disturbances they cause in routine biological tests. These effects include lupus anticoagulant in the augmented partial thromboplastin test (APTT) and false serological tests for syphilis in the VDRL test. The systemic clinical features are mild thrombocytopenia, chorea, heart valve disease, livedo reticularis and, most commonly, recurrent pregnancy loss. The mechanism by which they paradoxically cause thrombosis *in vivo* has been debated. A direct antigen–antibody interaction in the vascular wall seems to be an attractive concept.[10]

Symptomatic central and branch retinal arteriolar occlusions, ischaemic and non-ischaemic central retinal vein occlusions and ischaemic optic neuropathy have all been described in the primary and secondary APL syndromes.[5–9] Our patient was asymptomatic and even reluctant to undergo panretinal scatter photocoagulation. Castanon *et al.*[11] studied 17 patients with the primary syndrome and found vascular changes in all, although only 10 patients described visual symptoms. This study and our case report highlight the importance of ocular screening in such individuals.

Aggressive steroid therapy, aspirin and dipyridamole have not universally shown beneficial effects in controlling visual symptoms and preventing progression of vaso-occlusion.[6,8,9,12] Recently, in a randomised controlled clinical trial, low-dose aspirin (75 mg) together with heparin has been shown to be more effective than aspirin alone in improving outcome of pregnancy in recurrent fetal loss associated with this syndrome.[13] The patient described by Manzanares *et al.*[7] experienced a similar beneficial effect on switching from aspirin to acenocoumarol. Oral anticoagulants probably need to be continued for at least 6 months or, in the secondary syndrome, until the PTT becomes normal.

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Sir,

Endogenous *Candida* endophthalmitis with no apparent predisposing factors

Endogenous *Candida* endophthalmitis (ECE) is well recognised in patients with certain predisposing factors such as indwelling intravenous catheters, total parenteral nutrition, abdominal surgery, broad spectrum antibiotics, immunosuppressive therapy and intravenous drug use. The case recently reported in *Eye*[1] illustrates this well. However, occasionally no obvious predisposing factors exist and we report a patient who did not, apparently, have any of these risk factors. The diagnosis was suggested by clinical signs and confirmed at vitrectomy.

Case report

A 65-year-old man presented with a 2 week history of a red, aching right eye associated with reduced vision. He had no problems with his left eye and no past ocular history. In his past medical history he had eczema and had become asthmatic 2 years previously; the asthma initially required oral steroids but more recently had been controlled with inhaled steroids and a beta-agonist. Two years previously he had required surgery for an inguinal hernia. He was a retired gastroenterologist and his latest travel abroad had been to Egypt 2 years previously.

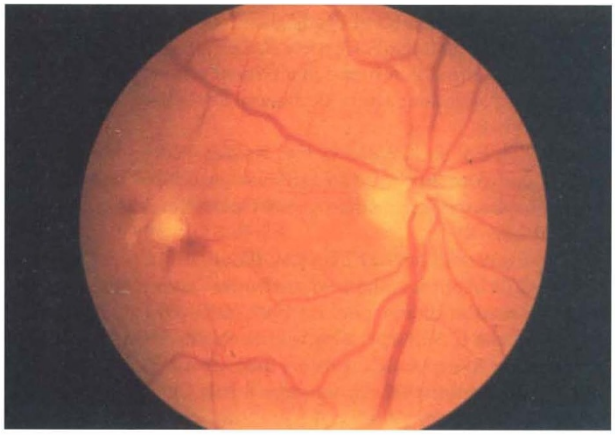

Fig. 1. *Fundus photograph of the right eye showing a fluffy, white lesion with associated haemorrhage at the macula.*

On examination his left eye saw 6/5 and was entirely normal. The right eye was injected and the visual acuity was count fingers with moderate anterior chamber activity and 2+ cells in the vitreous. The fundus (Fig. 1) showed a white, fluffy area at the macula associated with haemorrhage. The fluorescein angiogram (Fig. 2) showed masking by the haemorrhage with some leakage associated with the pale lesion. General examination revealed no abnormality. Investigations including FBC, ESR, CRP, U&E, LFT, glucose, ACE, blood cultures, *Toxoplasma* and *Histoplasma* titres, Mantoux and HIV tests and chest radiograph were all negative.

The ophthalmic findings of a unilateral, relatively severe panuveitis associated with a pale, fluffy retinal focus in the presence of normal retinal vessels suggested an endogenous endophthalmitis. A diagnostic vitreous biopsy was performed. At surgery intravitreal amphotericin, vancomycin and ceftazidine were given. Microscopy of the vitreous specimen showed a small number of pus cells and no organisms but culture revealed scanty growth of *Candida albicans*, sensitive to fluconazole. The patient was commenced on oral fluconazole and flucytosine. The inflammation subsided although the central vision remained reduced due to a macular scar.

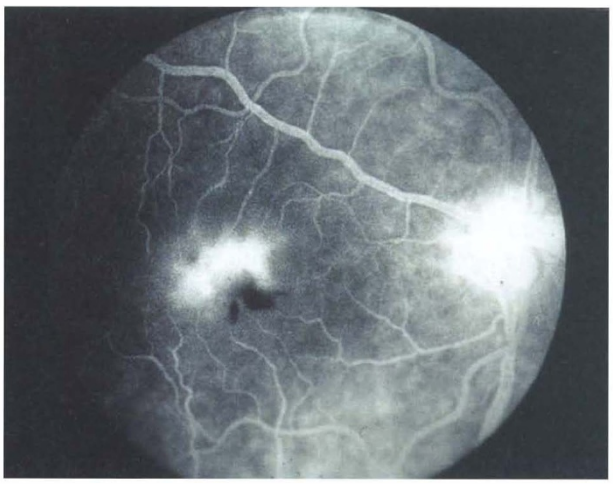

Fig. 2. *Fluorescein angiogram of the right eye showing masking by the haemorrhage and slight leakage associated with the pale lesion.*

Discussion

In a review of 14 patients with ECE over a 10 year period at St Thomas' Hospital[2] all had a predisposing factor for the development of ECE; 10 were in patients with intravenous infusions and 4 were intravenous drug abusers. There are a few case reports of patients with ECE who acquire the infection from more unusual sources. Four patients in Illinois developed ECE from contamination of the intravenous anaesthetic agent propofol.[3] One patient with onychomycosis and vaginal candidiasis developed ECE; the strain of *Candida* in the nail and the eye was the same,[4] Lithotripsy for renal calculi in the presence of a fungal urinary tract infection has been reported as the source of *Candida* in 3 cases of CEC[5] and in 1 patient the only risk factor appeared to be the use of anabolic steroids,[6] possibly causing a modification of his immune response.

The source of infection in this case has never been found. There are two possibilities. The patient's last intravenous infusion was during his herniorraphy 2 years previously, at a time when he required oral steroids for his asthma. It is possible that *Candida* gained access at this time and had then lain dormant for 2 years. The other possibility is that he developed asymptomatic oropharyngeal candidiasis as a result of his inhaled steroids. Thrush has been reported in 5–13% of adults on inhaled corticosteroids.[7] From this site it would need to gain access to the circulation. However, HIV-positive patients (with no history of intravenous drug abuse) frequently have oral, pharyngeal and oesophageal thrush but rarely develop *Candida* septicaemia, which makes this possibility less likely.

This case illustrates that *Candida* should be considered a possible organism in cases of endogenous endophthalmitis or recalcitrant uveitis in the absence of retinal vascular disease. When the usual predisposing factors are absent a history of apparent minor surgery may be relevant and in such cases the vitreous specimen should be examined for fungi.

We thank Professor D. Hay for discussion on the possible source of the *Candida* organism.

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Sir,

Vascular loop the loop

We report the case of a 34-year-old man in whom a thick epiretinal membrane was found in association with a branch retinal vein occlusion, distal to an arteriovenous crossing abnormality.

Case report

A 34-year-old man was referred to the vitreo-retinal clinic with a 4 year history of blurred vision and macropsia in the left eye. He was previously fit and well, with no relevant ophthalmic history.

On examination, the visual acuity in the right eye was 6/4 unaided, and in the left was 6/9. Both anterior segments were normal, as was the right fundus. However, the left fundus revealed a thick epiretinal membrane over the superotemporal arcade. Proximal to this, an arteriovenous crossing abnormality was noted; the vein completely looped around the artery (Fig. 1, arrow). A fluorescein angiogram confirmed the hypothesis of a branch retinal vein occlusion distal to the arteriovenous crossing abnormality. Fig. 1 shows the fluorescein angiogram taken in the early venous phase. Capillary drop-out and dilatation are revealed around the fovea, with straightening of the retinal blood vessels in this vicinity. Tortuosity of the superotemporal branch artery is apparent under the epiretinal membrane, which occupies approximately four disc areas above the fovea. There is collateral circulation involving branches of the superotemporal branch artery and vein, such that the veins accompanying the branch artery fill before the rest of the venous system (Fig. 1, labels 'a' and 'b').

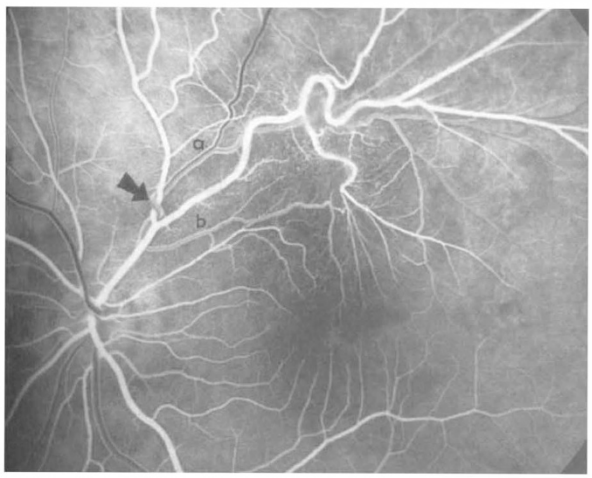

Fig. 1. *Fluorescein angiogram taken in the early venous phase. For details see text.*

The clinical observation of the epiretinal membrane and the findings of the fluorescein angiogram are consistent with a diagnosis of branch retinal vein occlusion. The patient was normotensive and no systemic predisposition to venous occlusive disease was identified.

Comment

Branch retinal vein occlusion is one of a number of causes of epiretinal membrane formation.[1] Others include surgical procedures such as cataract extraction, scleral buckling, retinal argon laser and cryopexy, together with chronic intraocular inflammation, diabetic retinopathy and blunt trauma. Approximately 60% of vein occlusions occur in the superotemporal quadrant,[2] with another 39% in the inferotemporal quadrant. They usually occur at arteriovenous crossings, and as there are more superotemporal crossings above the disc, the risk may be greater here. In addition, a greater proportion of these crossings have the artery passing over the vein, so-called artery-over-vein type.[3] Zhao *et al.*[4] Found that 99% of vein occlusions occurred at artery-over-vein crossings, suggesting that a mechanical obstruction of the vein may have a role to play in the formation of thrombus. Seitz[5] postulated that changes in the distension of the vein passing under the artery as opposed to over it resulted in haemodynamic differences between the two types of crossing. Turbulence secondary to reduced lumen diameter can cause intimal damage and initiate clotting. Klein[6] concluded that an increase in arteriovenous crossings, or congenital loop the loops in an otherwise normal fundus, could increase the risk of branch retinal vein occlusion. As yet, however, a complete loop the loop of vein and artery has not been described.

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