Therapeutic options to prevent further vascular occlusion are varied and may involve antithrombotic agents alone or in combination with steroid immunosuppression to modulate the prothrombotic effect of the antibodies, and aggressive therapy with both modalities has been recommended for younger patients.[2–4] It is recommended that anticoagulation be continued until 6 months after the disappearance of the antibody.[2]

Given the lack of a consistent response to a specific therapy in the antiphospholipid syndrome and the possibility of future pregnancy, this patient was initially treated with oral prednisolone and with aspirin rather than intensive warfarin treatment (INR>3).[3,4,10] During 14 months of treatment she has had one further BRAO. In the light of experience with this case we would now anticoagulate with warfarin all patients with antiphospholipid syndrome and recurrent retinal vascular occlusion, and we would continue treatment indefinitely.

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Sir,

Bilateral anterior ischaemic optic neuropathy following amiodarone

Amiodarone, initially used in Europe as a coronary vasodilator and anti-anginal agent, is now widely used as an anti-arrhythmic for supraventricular and ventricular tachyarrhythmias. However, the drug has numerous pulmonary, hepatic, thyroid and neurotoxic adverse effects. Corneal microdeposits develop in nearly all patients. These do not significantly affect vision and are reversible on discontinuing the medication. Rarely, more profound effects on vision can develop with the drug.[1]

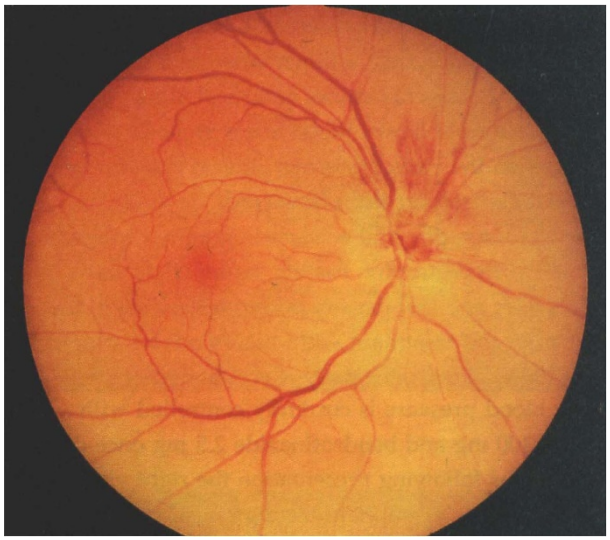
Case report

A 58-year-old man presented with a 5 day history of acute blurring of the lower half of the visual field in his left eye. He had no headache, jaw claudication, scalp tenderness or polymyalgia rheumatica. He had been taking amiodarone 200 mg b.d., bumetanide 1 mg o.d. and captopril 25 mg for the previous 2 months, having been diagnosed as having hypertrophic cardiomyopathy.

On examination, the visual acuity was 6/5 in the right eye and 6/5 in the left eye. A left relative afferent pupillary defect was present. The anterior segments showed vortex keratopathy and the intraocular pressures were normal. The superior half of the left optic disc was oedematous and pale, with a few splinter haemorrhages. The right disc was normal. Visual fields confirmed an inferior altitudinal defect in the left eye. He was generally fit and well. The blood pressure was 140/90 mmHg and he was in sinus rhythm. Haematological investigations, liver function tests, tests for syphilis, autoantibody screening and the coagulation profile including thrombophilia screening were all normal. The erythrocyte sedimentation rate was 5 mm/h (Westergren) and the C-reactive protein level < 10 g/l.

Amiodarone was discontinued immediately in consultation with the cardiologist. Six weeks later, the patient presented with a similar picture affecting the right optic disc (Fig. 1), with an inferior altitudinal field loss. There was pallor of the superior half of the left disc. Fundus fluorescein angiography showed the typical appearance of fresh anterior ischaemic optic neuropathy in the right eye and disc staining in the left. He had had no episodes of feeling unwell since his last visit and continued to be in sinus rhythm. A CT scan of the orbits and brain was normal. Oral prednisolone 60 mg was commenced and tapered gradually over 4 weeks. Unfortunately, there was no visual recovery in either eye.

The patient has been followed up for 18 months. He has a visual acuity of 6/6 in both eyes, atrophy of the upper halves of both optic discs and bilateral non-progressive inferior altitudinal field defects.



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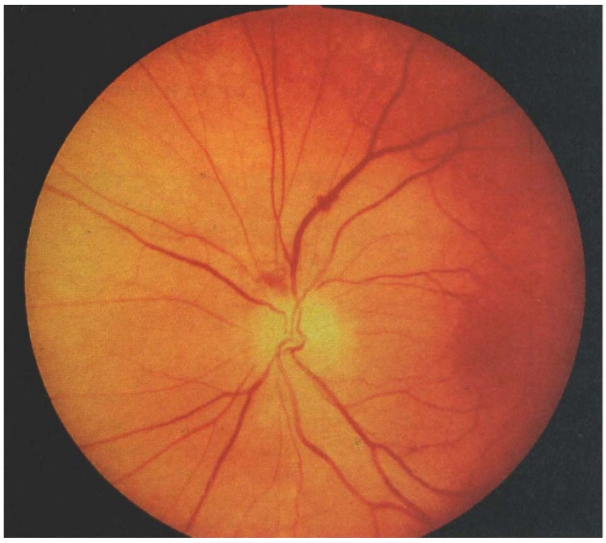

(b)

Fig. 1. *Fundus photographs of right (a) and left (b) eyes. Note the marked pallor of the upper half of the left disc and the resolving peripapillary haemorrhages.*

Discussion

The clinical picture, including angiographic changes, is one of bilateral anterior ischaemic optic neuropathy (AION). The age of the patient, the lack of systemic symptoms, the relatively mild visual loss and the normal erythrocyte sedimentation rate and C-reactive protein level exclude an arteritic form of AION. The non-arteritic form of AION can occur in diabetes mellitus, uraemia, following cataract extraction, following acute blood loss and in the collagen disorders. None of these situations applies to our patient. We could find no reports of AION following bumetanide or captopril.

Papilloedema as a result of pseudotumour cerebri and optic neuropathy' has been reported[2,3] in patients taking amiodarone. The features of amiodarone-induced optic neuropathy are: relatively well preserved visual function, reversibility on discontinuation and the absence of the characteristic crowding of the optic disc that predisposes to non-arteritic AION. Multi-lamellar inclusion bodies, typical of genetic and drug-induced lipidosis, have been demonstrated in the large axons of the retrobulbar optic nerve from an asymptomatic patient.[4] Amiodarone also generates free radicals *in vitro* and *in vivo*,[5] which again may result in neurotoxicity. A leucocytoclastic vasculitis and/or endothelial injury[6,7] affecting the posterior ciliary circulation may result in AION rather than an optic neuropathy. Sedwick[1] described a 62-year-old woman with hypertension and diabetes mellitus, taking amiodarone, who developed sequential non-arteritic AION, the visual loss continuing despite stopping the drug. Our patient has fairly similar features (including the presence of hypertrophic cardiomyopathy), although the visual acuity has been well maintained with no stepwise or gradual deterioration as seen in classic non-arteritic AION.

The patient described by Sedwick had an episode of 'fibrillation' soon after discontinuation of amiodarone. A resultant embolic phenomenon was felt to have contributed to the visual loss in the second eye. Our patient, however, had no symptoms other than visual loss and was in sinus rhythm. The half-life of amiodarone is approximately 40 days (range 20–107 days) and that of its desethyl derivative even longer.[8,9] Could this explain the visual loss in the second eye despite discontinuing the drug immediately? A serum amiodarone level would have ruled out toxicity although it does not have a predictive power for the occurrence of side effects.[9]

It is not clear which patients are more susceptible to optic nerve damage following use of amiodarone. Advanced age, renal failure, diabetes mellitus and alcoholism may be risk factors.[10] There appears to be no correlation with either the duration of therapy, daily dose or blood levels of amiodarone.[3] Vitamin E (alpha-tocopherol) was able to reduce amiodarone and desethylamiodarone toxicity in human cell cultures.[11] This raises the hope that the side-effects of amiodarone can be prevented or reversed by the administration of alpha-tocopherol.

The cause–effect relationship between amiodarone and AION remains debatable. In a study of 447 patients taking amiodarone, Feiner *et al*.[12] calculated the incidence of amiodarone-induced AION to be 1.76% during the preceding 10 years versus 0.3% in patients over 50 years not taking amiodarone.

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Sir,

Reversible anterior ischaemic optic neuropathy in accelerated hypertension

Anterior ischaemic optic neuropathy secondary to previously undiagnosed accelerated hypertension can present to the ophthalmologist. We report such a case in which the anterior ischaemic optic neuropathy reversed following prompt treatment of the hypertension.

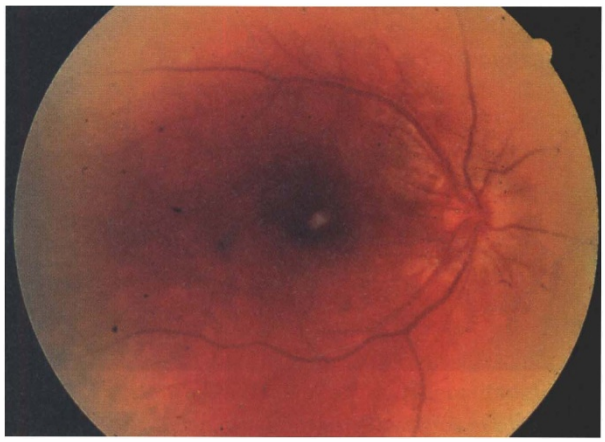
Case report

A 41-year-old man presented to casualty with a 2 h history of reduced vision in the right eye. There were no associated symptoms and no past medical history of note. There was a history of moderate alcohol consumption and a cigarette smoking habit of 30 per day.

Examination revealed a right visual acuity of hand movements, a right relative afferent pupillary defect and the presence of right optic disc oedema. The left visual acuity was 6/6 and fundoscopy revealed a cotton wool spot with a nerve fibre layer haemorrhage (Fig. 1). There was marked bilateral arteriolar narrowing. The blood pressure was recorded at 220/150 mmHg and the advice of a physician was sought for further investigation and management.

Following treatment with sublingual nifedipine 10 mg the blood pressure fell to 160/110 mmHg. The following morning the right visual acuity had improved to 6/9. Full blood count, mean cell volume, urea and electrolytes, erythrocyte sedimentation rate, vitamin B_{12}, folate, urinary catecholamines and renal ultrasound were all within normal limits. Renal artery stenosis was excluded on the basis of normal clinical examination, urea and electrolytes and ultrasound. Electrocardiography and echocardiography revealed left ventricular hypertrophy. Fasting serum cholesterol, triglycerides and low density lipoproteins were in the 'at risk' range and treatment with dietary advice and atorvastatin 20 mg once daily was commenced.

The blood pressure is currently controlled with atenolol 100 mg and bendrofluazide 2.5 mg once daily. Two months following presentation the right visual acuity was 6/6. Visual field testing revealed a non-specific pattern of field loss and retinal photography 5 months after initial presentation (Fig. 2) confirmed resolution of the fundal changes with pallor of the right optic disc. Unfortunately the patient continues to smoke 20 cigarettes a day and his alcohol consumption remains unchanged.



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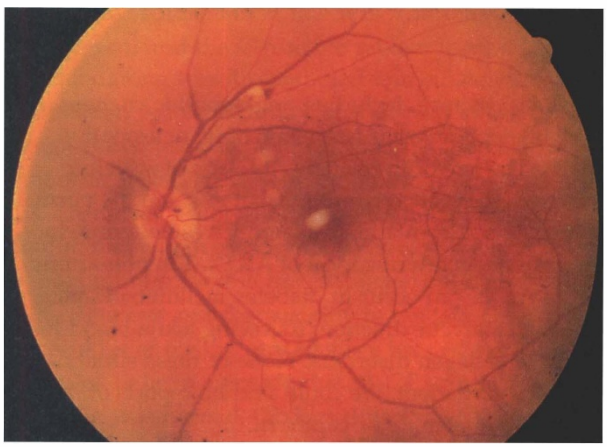

(b)

Fig. 1. *At presentation. (a) The right optic disc is swollen and there is widespread arteriolar narrowing. Other 'abnormalities' are artefact. (b) A cotton wool spot, nerve fibre layer haemorrhage and arteriolar narrowing are visible in the left fundus.*