relapsing course punctuated by frequent remissions and exacerbations. It is a self-limiting disorder, extending over 1–2 years with remission. The underlying process is believed to be an immunological reaction, leading to small vessel vasculitis[4] causing micro infarcts in the retina, brain and the apical turn of the cochlea. The important differentials would be multiple sclerosis, aseptic meningitis, systemic lupus erythematosus, Bechet's and complicated migraines. The other vasculitides like sarcoidosis, tuberculosis, syphilis and lymphomas have to be ruled out.

A high index of suspicion, leading to early recognition of this syndrome is important because treatment with immunosuppression may minimise permanent cognitive, audiologic and visual sequelae. This syndrome has a good prognosis when treated early. In patients whom early diagnosis has led to early administration of immunosuppressive therapy, recovery can be almost complete.

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Sir,

Cocaine-associated central retinal artery occlusion in a young man

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Retinal vascular occlusions in young adults are seen very infrequently and are generally associated with systemic disorders.[1] With the increasing abuse of cocaine in Western countries, reports of medical complications are increasing. Besides the more common vascular complications described, such as myocardial infarction[2] and cerebrovascular events,[3] only one case of central retinal artery occlusion (CRAO) in an intravenous cocaine abuser, had until recently been reported.[4] There have now also been two case reports of CRAO and central retinal vein occlusion in intranasal cocaine abusers.[5,6]

Case report

A 42-year-old man presented to casualty with a sudden painless loss of vision in his right eye 9 hours previously. He was not on any medication and was otherwise entirely fit and well with no history of migraine. He was a smoker of ten cigarettes a day for the past 20 years. He admitted to smoking 'crack' cocaine (more potent alkaloid form of cocaine) twice a week for the last 4 years, although he denied use of intranasal or intravenous abuse. He had smoked cocaine the previous evening. Visual acuity in the right eye was counting fingers at one metre, left eye 6/4. There was a right relative afferent pupillary defect. Examination of the anterior segments was unremarkable. Examination of the right fundus revealed diffuse retinal whitening and oedema in the posterior pole, with a foveal cherry-red spot and vascular attenuation, with sludging and segmentation of the blood column, consistent with a CRAO (Figure 1). No arterial emboli were seen. The left fundus was unremarkable (Figure 2).

Immediate treatment was instituted comprising 500 mg of acetazolamide intravenously, intermittent ocular massage and re-breathing into a paper bag for half an hour. Cardiovascular examination was normal with no carotid bruits, a regular pulse and no murmur audible.

Blood pressure, echocardiography, carotid ultrasonography, full blood count, erythrocyte sedimentation rate, fasting lipids and glucose, autoantibody screen including anti-cardiolipin antibody, protein S and C levels, antithrombin III level, Factor V Leiden, and blood homocysteine levels, were all normal. He was found to be sickle cell trait positive.

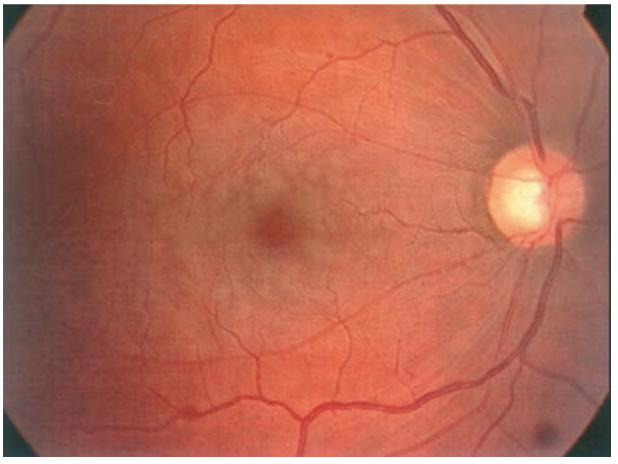

Figure 1 Fundus photograph of the right eye, showing retinal whitening and oedema in the posterior pole, with a foveal cherry-red spot and vascular attenuation, consistent with a CRAO. No arterial emboli are seen.

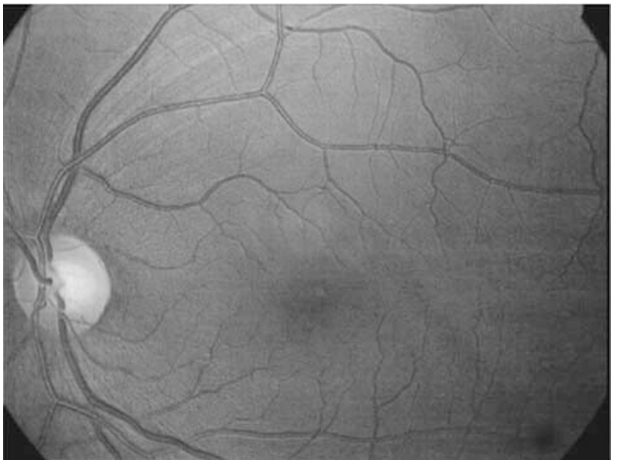

Figure 2 Fundus photograph of the left eye for comparison.

He has been reviewed 6 weeks later with no change in visual acuity. He has also been reviewed 2 months later and his visual acuity has improved to 6/18. His optic disc now appears pale. He has been strongly advised to stop smoking, both tobacco and in particular cocaine.

Comment

To the best of our knowledge this is the first case reported of CRAO associated with smoking cocaine in a previously healthy relatively young man. Previous case reports describe vascular occlusion following intravenous and intranasal cocaine abuse.

Inhibition of noradrenaline re-uptake by adrenergic nerve endings, direct vasoconstriction of vascular smooth muscle[7] and increased platelet aggregation[8] are among the invoked mechanisms of cocaine-related vascular complications. Systemically cocaine results in increased vascular tone, increased heart rate and blood pressure, and increased myocardial contractility.

CRAO, like cerebral infarctions, are typically found in older individuals and are often associated with systemic arterial hypertension.[9] Arterial occlusions in the retina in older adults commonly occur secondary to embolisation from atheromatous plaques of the carotid artery.[9] Arterial occlusions in younger patients are more commonly associated with migraine, trauma, cardiac embolic sources and various hypercoagulable states leading to thrombosis.[1] The high prevalence of underlying systemic disease in these young patients necessitates a thorough evaluation to rule out potential life-threatening embolic and hypercoagulable conditions, thereby minimising additional systemic and ocular morbidity.[1]

Vasospasm may play a role in arterial occlusions in young adults. CRAO associated with migraine-induced vasospasm has been described. Myocardial infarctions in young adults after cocaine use have been attributed to vascular spasm and the dramatic increase in sympathetic tone.[2] Arterial spasm may be induced by cocaine[7] and may thus result in decreased ocular perfusion. In addition it is possible that severe hypertension from cocaine use could also have led to CRAO by causing fibrinoid necrosis within the vessels or haemorrhage under an atheromatous plaque within the central retinal artery.[10]

Hypercoagulable states including antiphospholipid antibody syndrome, Factor V Leiden, protein S and C deficiencies, homocystinuria and hyperhomocysteinemia have all been excluded in our case. The only possible risk factor that has been established is his sickle cell trait status. We propose that smoking cocaine and his underlying sickle cell trait status have been the causative factors in this man's CRAO.

In conclusion, cocaine abuse should be considered as part of the differential diagnosis for retinal vascular occlusion, especially in relatively young, otherwise healthy patients, in whom CRAO is extremely rare.

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Sir,

Acute painful third nerve palsy: the sole presenting sign of a pituitary adenoma

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Painful third nerve palsy is a well known presenting sign of a posterior communicating artery aneurysm.[1] However it is unusual for a pituitary adenoma to present as a third nerve palsy. This report describes a patient who presented with an acute painful pupil involving third nerve palsy, which on neuroimaging was found to be due to a pituitary adenoma with parasellar extension.

Case report

A 43-year-old man presented with a 3-week history of headaches, intermittent drooping of right upper lid and diplopia. These had largely settled on antibiotics prescribed by his GP for frontal sinusitis. He was seen in the eye Casualty and apart from tenderness in the right supratrochlear region and mild ptosis, the ocular examination was normal. A month later he represented to the eye clinic with a sudden onset of severe occipital headache, diplopia and right upper lid ptosis. On examination the unaided visual acuity was 6/6 in the right eye and 6/5 in the left eye. There was right partial ptosis with anisocoria that increased in the bright light. The pupils measured 4.5 mm on the right, with sluggish reaction to direct light, and 3 mm on the left, with a normal reaction to light. A cover test revealed exotropia with a mild restriction of adduction, moderate restriction of depression and elevation in the right eye. The Hess chart was consistent with the diagnosis of right third nerve palsy (Figure 1). Fundoscopy showed well-defined disc margins with preserved spontaneous venous pulsations. The patient was X-linked red-green colour blind. Visual fields were normal.

The remaining cranial nerves were intact and there was no evidence of sensory or motor weakness. A diagnosis of a right painful pupil involving third nerve palsy was made. An aneurysm of the posterior communicating artery was suspected. An MRI scan of head and sellar region showed a large mass centred on pituitary fossa with invasion of both cavernous sinuses and bilateral parasellar extension, more marked on the right (Figure 2). There was no evidence of intracerebral aneurysm and this was confirmed on cerebral angiography. Haematological and endocrinal investigations were normal. The patient subsequently underwent transphenoidal resection of the tumour. Histology confirmed the diagnosis of pituitary adenoma. Postoperatively his visual acuity was 6/12 in the right eye and 6/5 in the left. Over the next 6 months the patient's ocular motility and ptosis improved and at the last follow-up visit his right third nerve palsy had resolved completely.

Comment

The most common ophthalmic presentation of pituitary tumours is with visual field defects.[1] Painful third nerve palsy is an unusual presentation of pituitary adenoma. Saul *et al*[1] reported five cases of third nerve palsy as the sole presenting sign of pituitary adenoma. Out of these in only one patient the third nerve palsy was painful. Cano *et al*[2] reported one case of intermittent third nerve palsy secondary to pituitary adenoma. This was accompanied by short nocturnal attacks of retro-orbital pain, rhinorrhoea and lacrimation.

Pituitary tumours cause third nerve palsy by several mechanisms. It may occur slowly secondary to mechanical compression against the interclinoid ligament, or by compression and invasion of the cavernous sinus by the tumour. Secondly it may occur rapidly, associated with headache due to compressive