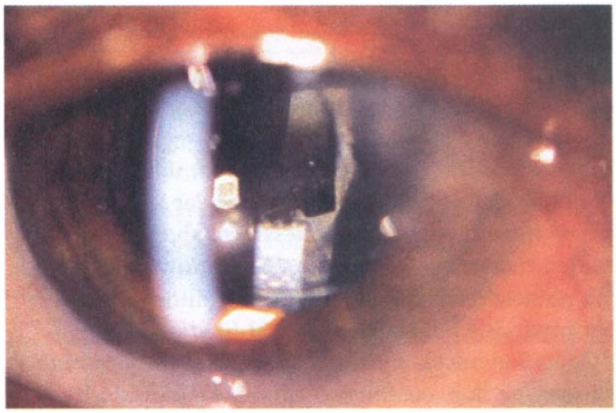

Fig. 2. *Digital photograph of the anterior segment after Nd:YAG laser capsulotomy. The depth of focus is unchanged from Fig. 1. Note that the posterior capsule has now returned to the normal position and both the posterior capsule and cornea are sharply focused within the same depth of field.*

Capsular block syndrome, or capsular bag distension syndrome, was first reported in 1990[1] and has subsequently been classified according to the time of onset: intraoperative, early post-operative and late post-operative.[2] In late-onset capsular block syndrome, residual lens epithelial cells left behind after cortical aspiration undergo metaplastic change and proliferate within the capsular bag. As well as causing posterior capsular opacification, the metaplastic cells cause the occlusion of the capsular opening by sealing off the gap between the anterior capsule and the anterior surface of the intraocular lens optic. A build-up of fluid, thought to be secreted by the lens cells, occurs within the capsular bag causing capsular distension with the subsequent anterior and posterior displacement of the intraocular lens and posterior capsule respectively. Capsulorhexis-related lacteocrumenesia[3] or liquefied after cataract[4] describes the accumulation of a whitish fluid within the capsular bag. This proteinaceous fluid is also thought to be secreted by lens epithelial cells. Capsular block syndrome, although first described after continuous curvilinear capsulorhexis and phacoemulsification, has also subsequently been described after 'beercan' capsulotomy in extracapsular cataract extraction,[5] and can be a subtle cause of change in refractive error after cataract surgery.

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Sir,

Sphenoid sinus mucocoele mimicking aneurysmal oculomotor nerve palsy

We report a case of painful oculomotor nerve palsy with pupillary involvement, which was caused by a sphenoid sinus mucocoele. Aneurysms, especially of the posterior communicating artery, are the commonest cause for a painful third cranial nerve palsy with pupillary involvement. Mucocoeles of the sphenoid sinus alone are uncommon and only a few cases causing isolated oculomotor nerve involvement have been reported. Magnetic resonance imaging (MRI) and magnetic resonance angiography proved to be non-invasive and efficient methods of investigation and prompt drainage of the mucocoele led to a complete recovery from the palsy. The optic nerve is the most frequently involved cranial nerve in sphenoid sinus mucocoeles and sudden blindness is possible.[1] A clinician must have this remote possibility in mind while dealing with painful third nerve palsies.

Case report

A 71-year-old man, previously fit and well, presented to the eye casualty of our hospital with a 3 day history of severe headache and vertical diplopia. He had also noticed that his right eyelid had begun to droop. There was no history of trauma and he was not a known diabetic but was a hypertensive on treatment.

On examination, he had a partial ptosis of the right eyelid with the eyeball turned down and out. He had a dilated and unresponsive right pupil. Fundus examination and examination of the other eye was unremarkable. Hess chart demonstrated underaction of the extraocular muscles innervated by the third cranial nerve. Routine blood evaluation did not reveal any abnormality.

MRI of the brain showed diffuse high signal intensity (in axial proton density weighted and T2-weighted images) within the sphenoid sinus, which was expanded anteriorly (Fig. 1). The same mass was isointense on T1-weighted images (Fig. 2). Magnetic resonance angiography did not reveal any aneurysm in the circle of Willis.

An endoscopic sphenoethmoidectomy was performed and clear fluid and mucoid material was aspirated. No solid mass was found in the sinus. Histopathological examination confirmed the mucous nature of the aspirate and ruled out any organoid tissue or malignancy.

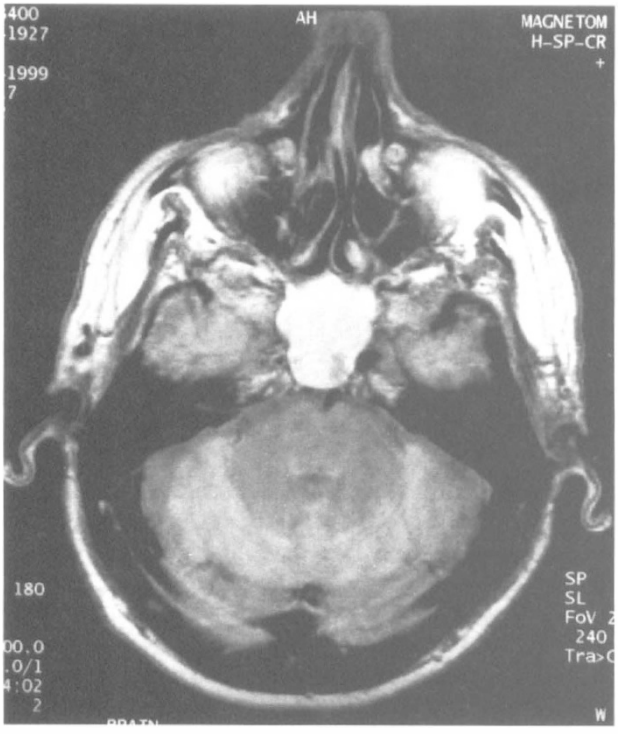

Fig. 1. *Coronal T1-weighted MRI scan showing an isointense mass occupying the sphenoid sinus.*

The patient showed good signs of recovery from the palsy soon after the procedure. By the second postoperative day, ptosis had resolved though there was some residual medial rectus weakness and the pupil was weakly reactive. He had fully recovered when he was assessed in the eye clinic 5 weeks later.

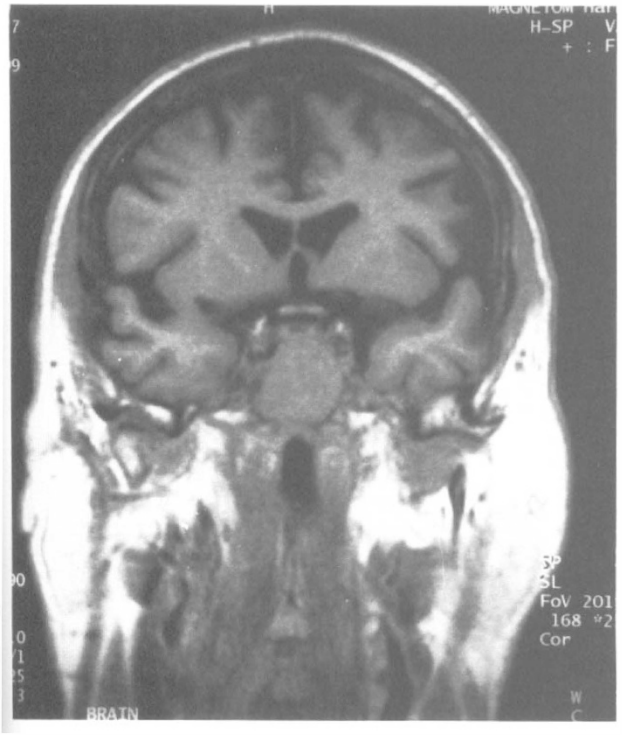

Fig. 2. *Axial proton density weighted MRI scan showing diffuse high signal intensity within the sinus with expansion anteriorly.*

Comment

Sphenoid sinus mucocoeles are rare and isolated involvement represents 1% of all paranasal sinus mucocoeles.[2] Patients are usually asymptomatic or have non-specific symptoms.[1] The most common symptom is headache, which is typically retro-orbital although it may be frontal or diffuse. The pain is worse when the patient is supine.[3] Cranial nerves II, III, IV and VI may be involved. The optic nerve is the most frequently involved cranial nerve.[3] It is important to treat mucocoeles early because advanced cases causing optic neuropathy can lead to blindness. Sometimes the initial presentation can itself be blindness.[4] In cases of optic nerve involvement, there is a poor prognosis for vision if the mucocoele is not treated within 7–10 days.[5] Endocrine abnormalities due to pituitary involvement have been reported.[6]

Pupillomotor fibres are present on the periphery of the oculomotor nerve. Therefore compressive lesions (typically aneurysms) involve the pupil whereas ischaemic microangiopathy (typically diabetes) tends to result in motility dysfunction but spares the pupil. Sphenoid sinus mucocoeles can present in both ways.

The secretions accumulated within the sinus have no exit and a gradual expansion of the cavity occurs due to the release of osteolytic prostaglandins and mediators with bony remodelling and erosion.[7] In our case, there was expansion of the sinus by the mucocoele anteriorly at the level of the anterior clinoid process where the oculomotor nerve bears the closest relationship to the sinus, explaining its isolated involvement.

MRI has been shown to be a useful non-invasive diagnostic tool in oculomotor nerve palsy for patients who do not have clear symptoms or signs of subarachnoid haemorrhage.[8] Had the MRI examination been normal, we would have proceeded to do a cerebral angiography, which is an invasive procedure with a certain amount of risk of morbidity and mortality but nevertheless conclusive in ruling out a cerebral aneurysm.

Ophthalmologists should be aware of this rare, sight-threatening condition as it may present with eye signs alone with little clue to its actual aetiology.

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Sir,

Ipsilateral proliferative diabetic retinopathy in carotid stenosis

Asymmetric diabetic retinopathy is not common, with reports of incidence varying from 5% to 10%.[1] Although there is no universally specified classification to define when asymmetry exists, the Diabetic Retinopathy Study Group used the criteria of proliferative diabetic retinopathy in one eye, with neither proliferative nor preproliferative changes in the fellow eye. Other authors[2] have stressed that such asymmetry should be present for 2 years to exclude patients with diabetic retinopathy which develops at a slower rate in one eye to eventually become symmetrical. Carotid stenosis is one of a number of factors implicated in the development of asymmetric diabetic retinopathy, the eye with the worse retinopathy being on the side contralateral to the more significant stenosis. This phenomenon has been explained as being due to the protective influence of the carotid stenosis on the development and progression of diabetic retinopathy. We report a more unusual case of unilateral proliferative diabetic retinopathy on the side ipsilateral to the more severe carotid stenosis.

Case report

A 65-year-old woman with a 2 year history of non-insulin-dependent diabetes presented to the eye clinic in April 1998 with a history of slightly blurred vision in the right eye for the past month. On further questioning she also complained of a wavy line developing across her vision on first getting up in the morning on opening her curtains. She also described two non-specific episodes of 'not seeing things properly' out of the right-hand side of her vision. She denied any periocular pain or discomfort. There was no history supportive of amaurosis fugax or transient ischaemic attacks. She had a 15 year history of hypertension for which she was being treated with bendrofluazide and amlodipine. She regularly attended the hospital diabetic clinic and described her current control as 'up and down'. She had recently been advised to lose weight and to cut down her smoking from 20 cigarettes a day.

On examination, her uncorrected visual acuity was 6/12, N10 in the right eye and 6/9, N10 in the left eye, distance and near respectively, improving to 6/6, N5 either eye with +1.00 Dsph for distance and +3.00 Dsph for near. There was no pupillary defect and examination of the anterior segments revealed bilateral early lens opacities. There was no corneal oedema, rubeosis or anterior chamber flare. The intraocular pressures were 18 mmHg either eye. Fundal examination showed proliferative diabetic retinopathy in the right fundus, with disc new vessels and a fresh preretinal haemorrhage inferiorly (Fig. 1). There were splinter haemorrhages near the disc, but no peripheral or mid-peripheral dot or blot haemorrhages. In addition there were features of hypertensive retinopathy (arteriovenous nipping and venous beading) in both eyes. The left fundus revealed mild background diabetic retinopathy with a small cholesterol embolus superotemporally (Fig. 2). There was no evidence of previous retinal vein occlusions or chorioretinal scarring in either eye. Bilateral carotid bruits were detected, louder on the right than the left side. She was commenced on aspirin 75 mg daily, and fundus fluorescein angiography was carried out which confirmed right-sided retinal ischaemia. The arm to retina time was 14 s in both eyes. Choroidal perfusion was normal. The patient was referred to the

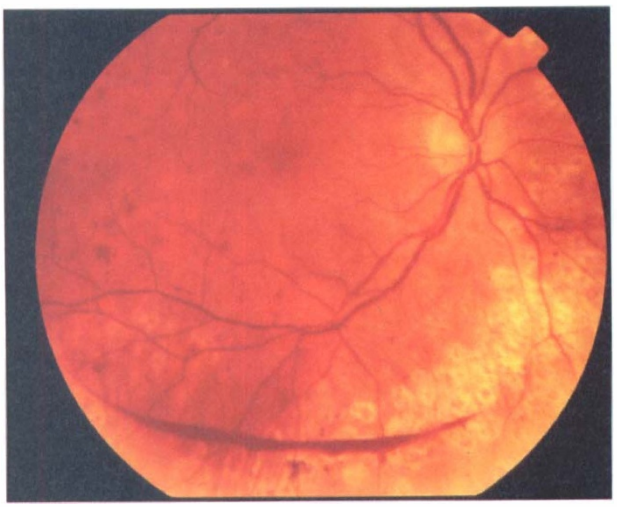

Fig. 1. *Fundus picture of right eye showing preretinal haemorrhage and panretinal laser photocoagulation scars.*

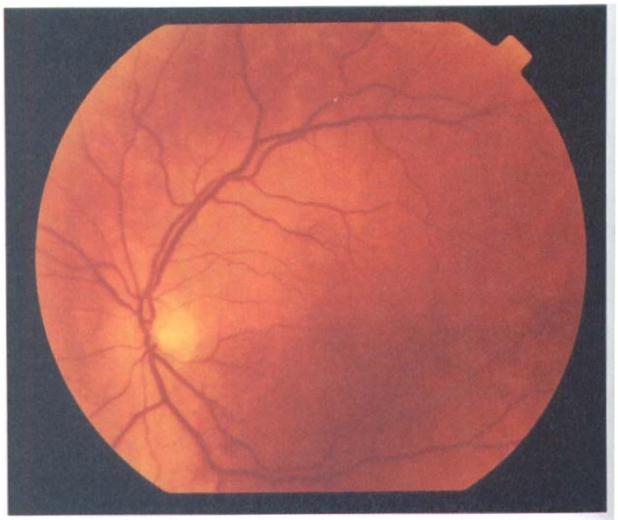

Fig. 2. *Fundus picture of left eye showing mild background diabetic retinopathy. Note the cholesterol embolus along the superior temporal arteriole.*