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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

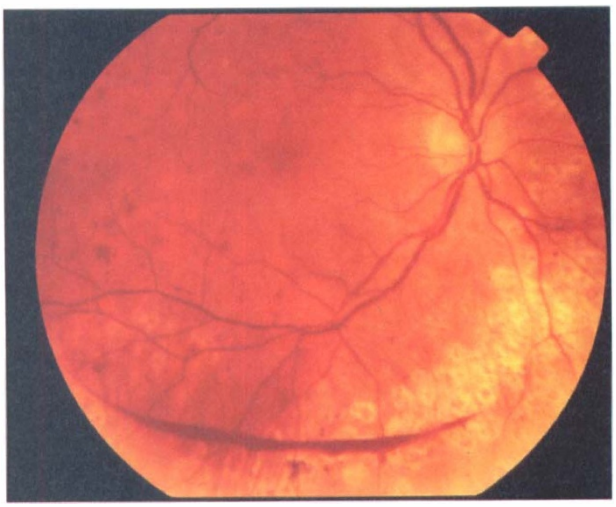

Fig. 1. *Fundus picture of right eye showing preretinal haemorrhage and panretinal laser photocoagulation scars.*

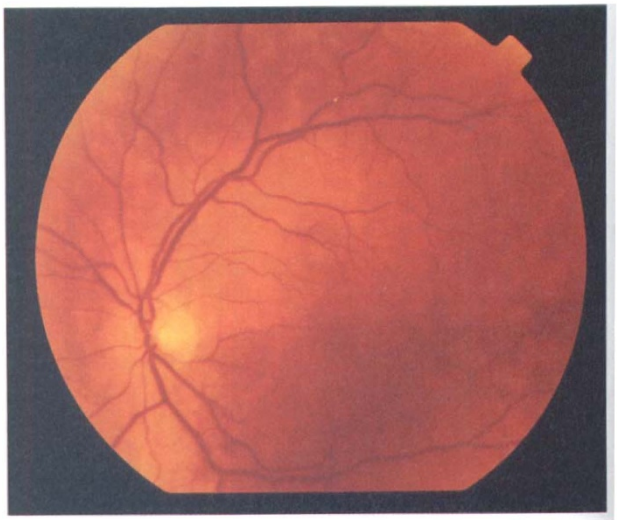

Fig. 2. *Fundus picture of left eye showing mild background diabetic retinopathy. Note the cholesterol embolus along the superior temporal arteriole.*

neurovascular clinic. Carotid Doppler/duplex scans, showed a 90% stenosis in the right internal carotid artery and 80% in the left. This was later confirmed on a magnetic resonance angiogram. There was also a 'moderate' stenosis of the left external carotid artery. Her resting blood pressure was 150/74 mmHg, her electrocardiogram was normal, her cholesterol was marginally raised at 5.6 mmol/l.

In view of the lack of symptoms of cerebral transient ischaemic attack and given the degree of stenosis, it was decided to defer carotid endarterectomy. Panretinal laser photocoagulation was carried out. Eight weeks following the laser treatment to the right eye there was marked regression of the new vessels. The right eye has remained stable. In November 1999 she collapsed at home following an episode of light-headedness. A magnetic resonance angiogram confirmed a 90% stenosis of the right internal carotid and 80% of the left side. She underwent successful right carotid endarterectomy and is currently asymptomatic. There has been no progression of the diabetic retinopathy in her left eye during her 2 year follow-up.

Comment

Asymmetric diabetic retinopathy has been described in association with a number of risk factors including previous branch retinal vein occlusion, carotid artery disease, complicated cataract surgery, trauma, asteroid hyalosis and uveitis, whereas high myopia, chorioretinal and optic atrophy are considered 'protective' factors.[1,3–7] Carotid artery stenosis has usually been reported as being a protective factor against the development of proliferative diabetic retinopathy[1,3,8,9] with isolated reports of it acting as a risk factor.[3] It presumably protects the eye from proliferative retinopathy by lowering retinal perfusion pressure. Venous stasis retinopathy or low-flow retinopathy may develop with a tightly stenosed internal carotid artery. The ocular ischaemic syndrome is found with severe ischaemia associated with ipsilateral common carotid artery stenosis or severe bilateral obstruction of the internal carotid arteries.[10] In our case, there was no suggestion of the ocular ischaemic syndrome either clinically or on angiography to explain the development of proliferative diabetic retinopathy on the same side as the severe stenosis. The other symptoms mentioned in the history of this patient, i.e. the transient unilateral visual loss in bright light (hemeralopia) and of feeling dizzy on first opening the curtains and seeing light in the morning are important symptoms suggestive of critical ischaemia of the eyes and/or brain. The aetiology is believed to be due to metabolic impairment of photopigment regeneration due to hypoxia.[11] This may be of importance to this case in suggesting that a more global picture of ischaemia may be predictive of the likelihood of worsening of diabetic retinopathy. While a certain degree of carotid occlusion is protective, any further compromise may be a risk factor.

This case highlights the risk of severe carotid artery stenosis leading to, rather than preventing, the development of ipsilateral proliferative diabetic retinopathy, particularly in patients with evidence of widespread ischaemia.

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Sir,

Corneal perforation in chronic graft-versus-host disease

Sicca syndrome related to chronic graft-versus-host disease (GvHD) following bone marrow transplantation has been well described. However, dry eyes leading to corneal melting with subsequent perforation in the absence of infection is a rare complication of GvHD.