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.

Case report

A 26-year-old Caucasian woman was referred to the ophthalmology clinic by the haematologists with a 3 week history of deterioration of vision in her left eye. The patient had been diagnosed with acute myeloid leukaemia (AML) in April 1997. She underwent standard AML induction chemotherapy and eventually allogeneic bone marrow transplantation following administration of cyclophosphamide and total-body irradiation. Her condition relapsed in September 1997 and she was treated with further chemotherapy followed by allogeneic peripheral blood stem cell transplant. A few months later she developed sclerodermatous chronic cutaneous lesions affecting her skin and joints as a result of GvHD and she was commenced on oral steroids. Subsequently she developed foreign body sensation attributable to bilateral sicca syndrome, which was treated with topical lubricants. Six months later she experienced worsening of her chronic ocular irritation and further deterioration of vision in the left eye.

On examination, her corrected visual acuity was 6/9 and 6/18 in the right and the left eye respectively. A poor tear film break-up time and bilateral filamentary keratitis were indicative of severe dry eyes, with changes more marked on the left. A left central corneal perforation of 1.2 mm was noted which was plugged by iris tissue (Fig. 1). The Seidel test revealed slight intermittent leakage but the anterior chamber was quiet and well formed. There was no evidence of corneal infiltrate or infection. In addition there were bilateral moderate posterior subcapsular cataracts attributable to her prolonged steroid therapy. Fundoscopy was unremarkable.

Initial treatment included ofloxacin q.d.s. to the left eye and preservative-free lubricants every hour. Three days later there was no significant re-epithelialisation and a bandage contact lens was applied. The patient was reviewed twice weekly. Complete epithelial healing with a negative Seidel test was noted after 5 weeks and the contact lens was removed.

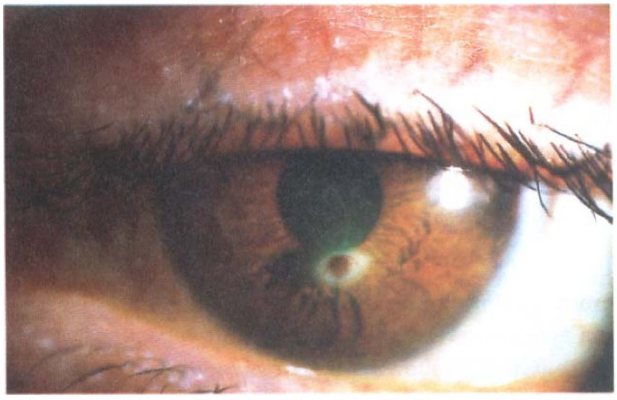

Fig. 1. *Photograph showing the central corneal perforation plugged by iris tissue.*

Comment

Ocular complications following bone marrow transplantation (BMT) have been well described. They are related to the underlying disease, total-body irradiation, systemic chemotherapy, GvHD and immunosuppression.[1]

Late-onset keratoconjunctivitis secondary to chronic GvHD is a frequent ocular complication of BMT. However, corneal perforation secondary to sicca syndrome without coexisting infective corneal ulcer constitutes a rare complication, and in the literature there are very few relevant reports.[2,3]

GvHD is a pleiotropic syndrome with variability in the time of onset, organ systems involved and rate of progression. The clinicopathological features resemble an overlap of several collagen vascular diseases with frequent involvement of the skin, liver, eyes, mouth, upper respiratory tract and oesophagus, and less frequent involvement of the serosal surfaces, lower gastrointestinal tract and skeletal muscles.[4] Patients with chronic GvHD show clinical and histological evidence of a dry eye condition in 60–100% of cases.[2,5] It has been postulated that the cause of dry eyes in GvHD is lacrimal gland stasis[6] or infiltration of the conjunctiva and the lacrimal gland by activated lymphocytes.[7] Jabs and colleagues have described a form of conjunctivitis that serves as a marker for severe GvHD. They classify the conjunctivitis into four stages of increasing severity: stage 1, conjunctival hyperaemia; stage 2, hyperaemia with chemosis or exudate; stage 3, pseudomembranous conjunctivitis; stage 4, corneal epithelial slough.[8] A pathognomonic sign for chronic GvHD of the conjunctiva is the fibrous-scarry Arlt lines of the tarsal conjunctiva.[9] The severity of these conjunctival lesions has been shown to correlate with the severity of GvHD. Other ocular findings in GvHD include cicatricial lagophthalmos, ectropion following lichenification of the skin of the lids, pseudomembranous and sterile conjunctivitis, persistent corneal epithelial defects, sterile corneal ulcers, iritis, and histological evidence of choroiditis.[3,10–12]

Most cases of sicca syndrome respond well to topical lubricants and antibiotics, bandage contact lenses, tarsorrhaphy, conjunctival flaps or conjunctival homografts. Ocular problems frequently resolve with improvement or resolution of GvHD. Recent studies illustrate the possible benefit of topical cyclosporin A 1% (CsA) as an adjunct in managing ocular surface abnormalities in cases of GvHD refractory to conventional therapy.[3]

Our case emphasises the need for frequent follow-up after BMT even in the absence of severe subjective symptoms. The course of sicca syndrome may be insidious, leading to corneal perforation without significant symptoms. In addition, all patients who have undergone BMT for leukaemia need to be monitored closely to estimate the level of GvHD they are in. This applies especially to those patients who are treated according to the regimen of adaptive immune therapy. Close cooperation between oncologists and

ophthalmologists can provide important information regarding the prognosis of GvHD by assessing the conjunctival morphological changes and prevent the development of severe ocular complications.[9]

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Sir,

Sporadic Burkitt's lymphoma presenting as solely orbital disease in a 78-year-old

Orbital involvement in sporadic Burkitt's lymphoma is extremely rare and only 5 cases have previously been reported. We present a case of primary orbital Burkitt's lymphoma in a 78-year-old white woman and outline the clinical features, histological findings and management.

Case report

A 78-year-old white woman was referred from another unit to the orbital clinic at Moorfields Eye Hospital. She had a 3 week history of increasing swelling at the upper outer quadrant of her left orbit. At the time of attendance she had experienced rapid increase in the swelling associated with severe pain over the previous 24 h. She also described fluctuations in her vision. She had no previous ophthalmic history. She was hypertensive and had suffered a stroke 12 years previously which had left her with no motor deficits. Her medications included amlodipine and aspirin.

On examination her visual acuities were 6/9 in the right eye and 6/12 in the left. Pupillary responses and colour vision were normal. She had a swelling at the superotemporal margin of her left orbit measuring 1.5 cm by 2 cm. This was associated with proptosis of 1 mm and inferonasal displacement of the left globe (Fig. 1). The anterior segment examination was normal. Examination of the posterior pole revealed choroidal folds affecting the superotemporal quadrant. CT scanning showed a left superotemporal orbital mass in the region of the lacrimal gland causing nasal displacement of the globe (Fig. 2). A differential diagnosis of dacroadenitis, lymphoma or malignant mixed tumour of the lacrimal gland was made on the basis of the clinical history and the CT scan findings and she was listed for orbital biopsy to elucidate the nature of the lesion. An anterior orbitotomy and biopsy of the lesion was conducted through a skin crease incision. The orbital lobe of the left lacrimal gland was found to be smoothly enlarged and firm in texture.

Histological investigation established the diagnosis of a high-grade B cell lymphoma with appearances consistent with Burkitt's lymphoma (Fig. 3). Immunohistochemical markers were positive for CD10 and CD20 (Fig. 4). A full blood count was normal apart from a mild eosinophilia, and plasma protein electrophoresis was normal.

She was referred to the radiotherapy unit at St Bartholomew's Hospital. Staging revealed no evidence of systemic disease and for this reason she was treated with primary local radiotherapy. She received a total of 30 Gy in 15 fractions over a 21 day period between 19 November 1998 and 11 December 1998. Treatment was given using 6 MV photons via a three-field plan. One

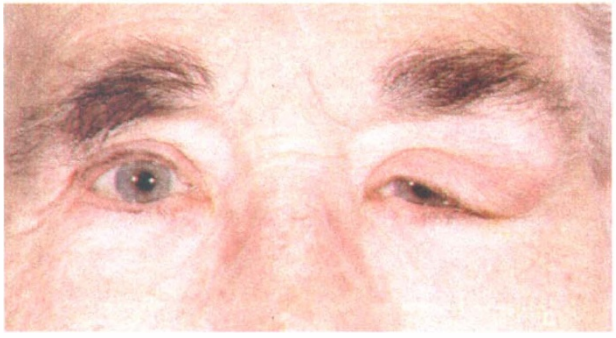

Fig. 1. *Clinical photograph of the patient at presentation showing inferonasal displacement of the left globe by the tumour associated with mild proptosis.*