angiotensin converting enzyme and treponemal serology were all normal and a chest radiograph revealed no abnormality. Two weeks later the uveitis had cleared and the drops were reduced over 2 weeks. She had suffered no similar episodes 9 months later.

Discussion

HTLV-1 infection is strongly linked to the subsequent development of adult T-cell leukaemia/lymphoma and HTLV-I associated myelopathy (HAM), which is also referred to as tropical spastic paraparesis (TSP). It is also associated with other inflammatory conditions but causes asymptomatic lifelong infection in the majority of individuals.[1–3] Ocular manifestations associated with HTLV-I infection have been documented, particularly in Japan, and include a steroid-resistant vasculitis[4] and a granulomatous or non-granulomatous uveitis.[5] However, the exact role of HTLV-I in the pathogenesis of these disorders remains uncertain.[6]

In keeping with previous reports, our patients' anterior uveitides resolved completely with topical steroids. However, the development in case 1 of HAM 3 months after the initial presentation is in contrast with the results of a recent study of 32 patients with HTLV-I associated uveitis in which no patient developed neurological disease. Anterior uveitis has been described in patients with *pre-existent* HAM/TSP.[7]

HTLV-I is endemic in Japan, the Caribbean, Melanesia, Central Africa and parts of Central and South America, and in peoples who have migrated from these regions. Thus amongst the Afro-Caribbean community in the United Kingdom, the prevalence of HTLV-I is 2%,[8] although its prevalence in those with uveitis is unknown. The infection may be acquired by vertical transmission, as in case 2, and this emphasises the importance of a family history in the diagnosis and counselling of patients found to be HTLV-I positive.

Although the cause of uveitis in these two cases was ascribed to HTLV-I infection without intraocular biopsy, they highlight the need to consider HTLV-I infection in patients belonging to at-risk groups who present with uveitis of unknown aetiology, and the need for further studies into the prevalence of positive HTLV-I serology in such patients.

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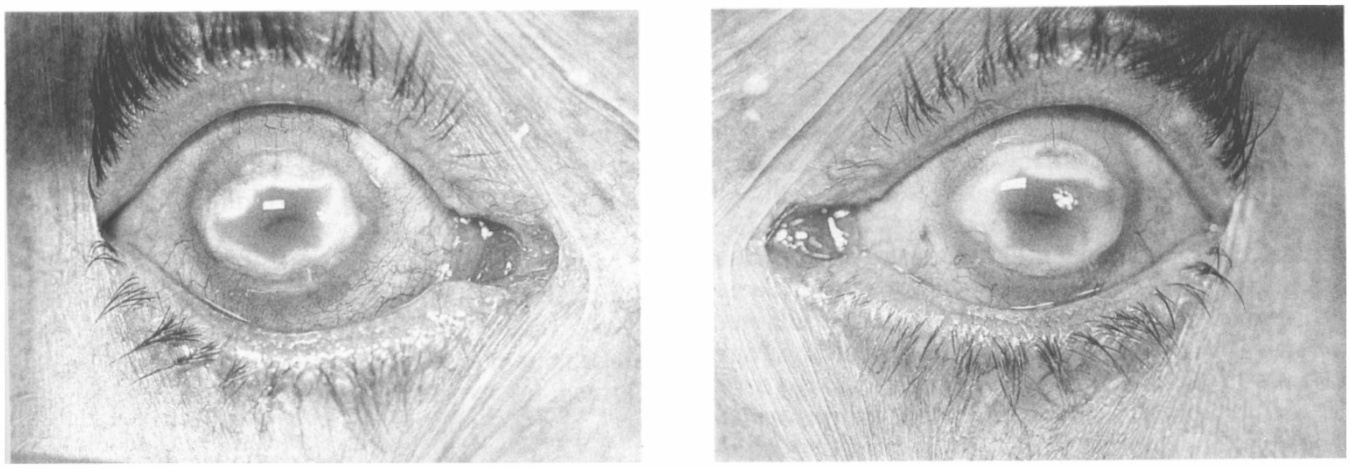
Sir,

Corneal Deposits and Penetrating Keratoplasty in a Patient with Hyperparathyroidism, Hyperlipidaemia and Multiple Myeloma

Calcium deposition in the cornea is usually the result of long-standing ocular disease but may also be associated with underlying hypercalcaemia. Occasionally, corneal deposits may be the presenting sign of an underlying disorder of calcium and phosphate metabolism which may occur with hyperparathyroidism, sarcoidosis, chronic renal failure and hypervitaminosis D.[1,2]

Lipid deposition occurs in scarred vascular corneas; however, primary lipid keratopathy may be a manifestation of underlying hyperlipidaemia.[3,4] Crystalline corneal deposits have been described in association with multiple myeloma[5] and streptococcal infections of the cornea.[6]

A case in which corneal deposits were the presenting sign of both hyperparathyroidism and hyperlipidaemia is discussed. The recurrence and



(a) (b)

Fig. 1. *Corneal deposits prior to surgery. (a) Right eye. (b) Left eye.*

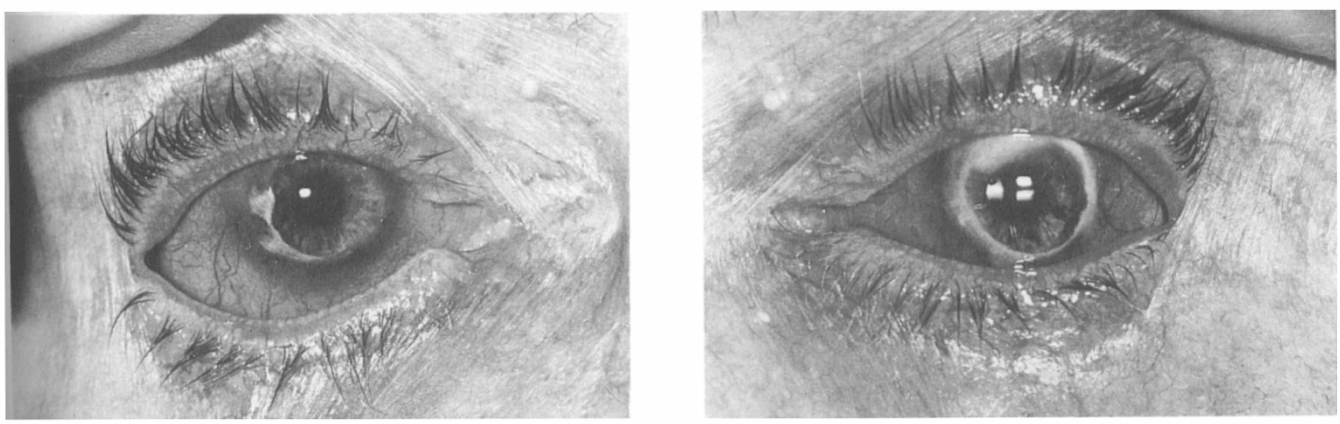
progression of lipid keratopathy bilaterally in corneal grafts, following a diagnosis of multiple myeloma in the same patient, is described.

Case Report

A 25-year-old woman presented to the ophthalmic department in 1951, complaining of bilateral chronic ocular irritation. Visual acuity in both eyes was recorded as 6/6 and no specific ocular lesion was identified. She presented again in 1963 and peripheral corneal degeneration was evident; she subsequently re-attended with recurrent episodes of blepharoconjunctivitis. By 1970, peripheral calcium deposition was noted and she was treated with topical EDTA 4% and cortisone 1%. Her serum calcium was elevated and primary hyperparathyroidism was diagnosed. She underwent parathyroidectomy and histological examination confirmed a parathyroid adenoma. She was subsequently lost to follow-up until 1982, aged 56 years, when she presented with bilateral vascularised peripheral corneal opacities with lipid deposition. Visual acuities were now 6/24 in the right eye and 6/18 in the left. Serum lipids and cholesterol were elevated, consistent with a type IIb hyperlipidaemia. Investigations for multiple endocrine neoplasia proved negative.

Argon laser was applied to the feeder corneal vessels in both eyes using a 200 μm spot size, 0.1 second duration and a power setting of 300 mW, and the keratopathy partially regressed. By 1985, visual acuities had deteriorated to 6/36 in the right eye and 6/60 in the left eye (in which there was also evidence of cataract formation), and corneal deposits persisted (Fig. 1). She underwent left intracapsular cataract extraction with iris-fixated lens implant (IOLAB 1036 severin (suture) PC optic) and, as a separate procedure, penetrating keratoplasty was undertaken 5 months later, using an 8 mm donor button on a 7.5 mm recipient bed and sutured with continuous 10-0 nylon. Post-operative corrected visual acuity was 6/9. One year later, lipid degeneration had recurred at the graft margins; however, the visual axis remained clear.

In 1989 she underwent right extracapsular cataract extraction with posterior chamber implant (Cooper



(a) (b)

Fig. 2. *Deposits at graft margins. (a) Right eye (3 years post-keratoplasty). (b) Left eye (7 years post-keratoplasty).*

vision style 865.06) combined with penetrating keratoplasty. Again, an 8 mm donor button was sutured to a 7.5 mm recipient bed using continuous 10-0 nylon, and she subsequently achieved a corrected acuity of 6/12 in this eye. Histopathological examination of the host cornea showed infiltration of foamy cells at the edge of the cornea, with adjacent corneal degeneration and cholesterol deposits, consistent with lipid keratopathy.

In 1990, bilateral fatty corneal deposits were noted following a diagnosis of multiple myeloma, and systemic melphelan and prednisolone were commenced. Lipid deposition, however, progressed at the periphery of both corneal grafts, and she underwent further argon laser treatment to the feeder corneal vessels, resulting in regression of the keratopathy. Two years later, the central cornea remained clear in both eyes, but the patient continued to suffer with recurrent blepharoconjunctivitis associated with seborrhoeic dermatitis resulting in a chronic limbitis (Fig. 2). To date, the visual axis remains unaffected in both eyes.

Discussion

Chronic ocular disease, in particular uveitis, old keratitis and phthisis bulbi as well as underlying hypercalcaemia, may result in calcium deposition in the cornea. Changes are usually first seen in the periphery; with time, the deposits may coalesce to form a horizontal band of calcium deposits in the interpalpebral area.

Hypercalcaemia due to underlying hyperparathyroidism is known to cause corneal calcification[3] and, in a few instances, corneal involvement may be the presenting sign.[1,2] In the case presented, ocular irritation was noted 12 years before corneal calcium deposition was observed and underlying hypercalcaemia detected. In contrast to band keratopathy, in which calcium is deposited extracellularly, with hyperparathyroidism, calcium deposition is intracellular.[7]

Lipid keratopathy is mainly secondary to corneal inflammation, often associated with herpes simplex, herpes zoster and disciform keratitis. It may, however, be a sign of underlying hyperlipidaemia, excess lipid in the blood being deposited in the cornea. Lipid deposits may also occur as a result of defective fat metabolism or lipid processing within corneal cells.[4] In those cases of lipid keratopathy which fail to respond to topical steroid treatment, argon laser may be used to occlude the feeder corneal blood vessels and so prevent further lipid deposition.[8] Complications include transient haemorrhage into the cornea, iris atrophy, and corneal thinning following the resorption of lipid.[9] None of these complications occurred in this instance. It has also been shown that subsequent penetrating keratoplasty in these patients is not compromised by previous argon laser treatment.[9] In our patient, primary lipid keratopathy was a presenting sign of underlying systemic abnormality. The lipid deposits initially responded to argon laser occlusion of corneal blood vessels, but subsequent penetrating keratoplasty was required and resulted in good visual outcome.

Multiple myeloma is a malignant proliferation of plasma cells which infiltrate bone marrow and result in excessive production of a distinct immunoglobulin or fragment of an immunoglobulin cell clone. Corneal involvement in multiple myeloma is uncommon and possibly the first clinical sign of systemic disease.[5] Characteristically, crystalline deposits occur throughout the corneal stroma,[10] although several varieties have been described.[11] Proteinaceous deposits have also been documented.[12] There have been numerous reports as to the content of the crystals. Some have suggested they contain cholesterol,[10] while others believe they are composed of immunoglobulin.[5] Klintworth *et al.*[5] described two patients with corneal and conjunctival intraepithelial crystalline deposits. By immunofluorescence and immunoperoxidase techniques, they showed that the crystals reacted positively for immunoglobulin and, in particular, IgG kappa chains. They proposed that the crystals were either the result of an infiltration of plasma cells with crystalline inclusions, the spontaneous crystallisation of immunoglobulin, or a combination of both mechanisms. Some crystals may disappear following chemotherapy.[5] It may therefore be advisable to defer penetrating keratoplasty in such patients until they have received chemotherapy. Corneal deposits have been reported to recur in patients who have been grafted, and may result in visual impairment.[13]

The deposition of copper in Descemet's membrane has also been described in association with IgG myeloma or monoclonal gammopathy.[14,15] It is thought that copper binds to the abnormal immunoglobulin to form complexes which are deposited in the cornea and lens capsule.

Although our patient did not present with the classic crystalline changes, it is interesting that, shortly after the diagnosis of myeloma, there was progression of lipid deposition in the grafts.

Corneal deposits may occur in advance of other detectable clinical signs and in our patient corneal changes preceded the systemic diagnosis. The detection of corneal deposits in this case proved a useful aid in the diagnosis of underlying systemic disease.

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Sir,

Retinal Vascular Abnormalities in Aortic Coarctation

In the elderly population central retinal vein occlusion (CRVO) is often associated with systemic conditions such as arteriosclerosis, hypertension, diabetes mellitus, hyperviscosity and hypercoagulability states, and connective tissue disorders.[1] The prevalence of hypertension in patients with CRVO is reported as approximately 60%, which is twice as common as for an age-matched population.[1–5] CRVO is uncommon in the younger age group but has important associations including head injury, use of oestrogen-containing compounds, connective tissue disorders, hyperlipidaemia, hyperviscosity and cryofibrinogenaemia.[1,6,7] In young adults, hypertension is a less frequent association of retinal vein occlusion and is often secondary to renal disease, endocrine disorders, drugs, toxaemia of pregnancy and collagen disorders. Coarctation of the aorta is an uncommon cause of hypertension in adults. It is frequently accompanied by retinal vascular abnormalities,[8] which together with chronic hypertension can contribute to increased risk of retinal vascular occlusion.

A case of a previously healthy 23-year-old man with bilateral retinal vascular tortuosity, left CRVO and hypertension secondary to aortic coarctation is reported.

Case Report

A 23-year-old male office worker presented to the emergency eye clinic with a 3 day history of painless loss of vision in the left eye. He had been healthy in the past and had suffered no significant systemic or eye conditions. Corrected visual acuity was 6/6 right eye and 6/60 left eye. Anterior segment examination was unremarkable and the intraocular pressures were 17 mmHg in each eye. The right retinal vessels were tortuous (Fig. 1) and there was a left CRVO with marked oedema and superficial haemorrhages (Fig. 2). Left brachial arterial pressure was 170/100 mmHg, femoral pulses were diminished and labora-

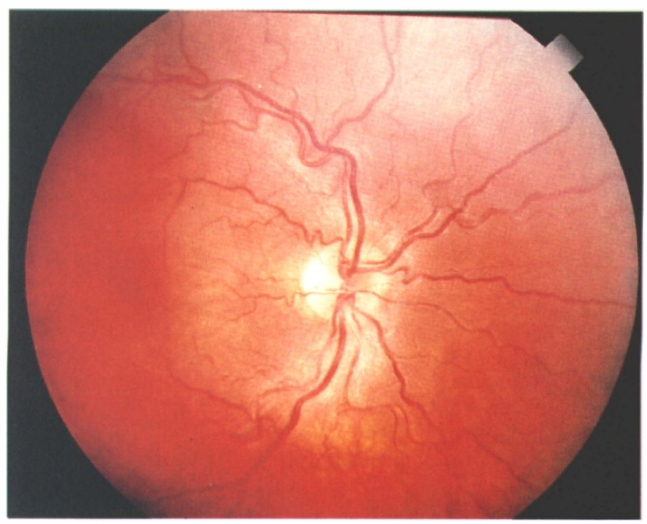

Fig. 1. *Right retinal arteriolar tortuosity associated with aortic coarctation.*