complexes with inadequate hemidesmosomes and anchoring fibrils. Debris can then build up under the overlying loose epithelium resulting in erosion.[2,7] A large proportion of patients with EBMD remain symptomatic of erosion and are more likely to require continued treatment with topical lubricants.[8]

The majority of patients with erosions will respond to ocular lubricants and topical antibiotic cover in the acute phase with the removal of loose epithelium. The 3 cases presented were unusual in that they developed bacterial keratitis with hypopyon, resulting from a breach in the epithelium, allowing bacteria access to the underlying stroma.

All 3 patients responded well to ciprofloxacin monotherapy with resolution of hypopyon keratitis. Bacterial keratitis can result from a break in the corneal epithelium allowing bacteria access to the corneal stroma, where they multiply and spread, sometimes releasing toxins and enzymes. In the cases presented organisms may already have been present in the conjunctival fornices, becoming pathogenic when an epithelial break was incurred.

Interestingly, recurrent erosion has been associated with lid margin disease. Oral tetracycline has been shown to be effective in the treatment of meibomian gland dysfunction and also in recalcitrant corneal erosion, significantly reducing the time for erosions to heal.[9,10] Its mechanism of action is not fully understood, but may be unrelated to its antibacterial action. Tetracycline may inhibit lipid synthesis in meibomian glands[11] and binds to conjunctiva and goblet cells.[12] It also has an anti-collagenolytic activity,[13,14] inhibits metalloproteinases and may suppress connective tissue breakdown.[15]

Ciprofloxacin is a potent fluoroquinolone with broad spectrum antimicrobial action and a low incidence of resistance.[16] However, with topical use a precipitate may form at the superficial portion of the corneal defect.[17] This may be troublesome in EBMD and could impair epithelialisation. However, a precipitate did not occur in any of the cases presented. Precipitation is unrelated to the sex of the patient, causative organism, size or depth of the lesion or, indeed, the time for the defect to heal. More importantly, it is most often observed in patients over 60 years of age.[17] Patients with EBMD tend to be in a younger age group, and so this problem should be less likely to arise.

The presence of a stromal infiltrate with hypopyon is related to the severity of corneal inflammation and is often associated with an infected corneal lesion, requiring full microbiological investigation. Although the majority of corneal erosions are not infective, this series of three cases emphasises the need to be alert to the possibility of underlying infection.

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Sir

Bilateral keratomalacia in a cachectic scleroderma patient

It is estimated that 124 million children world-wide are vitamin A deficient, making it the world's second most prevalent nutritional disorder after protein energy malnutrition. Of these, 5 million will develop xerophthalmia and between 250 000 and 500 000 will become blind.[1] In the more developed parts of the world vitamin A deficiency is rarely encountered. We report an unusual case of corneal melting (keratomalacia) caused by nutritional deficiency of vitamin A secondary to scleroderma (CREST type). In developed countries clinical xerophthalmia due to low vitamin intake has been reported in food faddists and psychiatric patients.[2] Deficiency has also been reported in cases of chronic

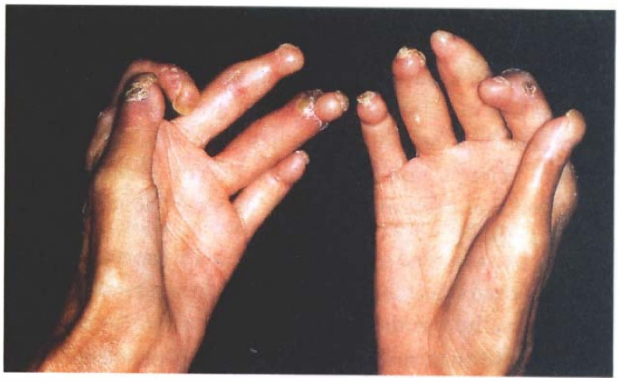

Fig. 1. *Hands of our patient demonstrating severe scleroderma.*

alcoholism, pancreatic diseases and lymphomas. However, most of the cases of xerosis observed in rich communities are due to conditions causing malabsorption, such as cystic fibrosis,[3] or surgical procedures involving jejuno-ileal bypass.[4] Xerophthalmia appears not to have been reported secondary to oesophageal motility problems prior to our report.

Case report

A 51-year-old white woman presented to eye casualty with a red, watery, mildly irritable left eye for 2 weeks with visual acuity of 6/48 left eye. She had been using lubricating eye drops for mild dry eye symptoms since uncomplicated cataract surgery 7 years previously. She had suffered from scleroderma, of the CREST type, for 18 years (Fig. 1). She weighed only 24 kg at presentation with a height of 1.47 m (her body mass index was 11.1; normal 20–25) having lost 10–12 kg in the previous 4 months due to oesophageal motility problems.

She had normal lids and the conjunctiva were injected with good lid margin tear film. There was a left circumscribed paracentral corneal ulcer, 1.6 × 2.2 mm, to Descemet's membrane depth (Fig. 2). The edges of the ulcer were steep with no infiltration and the cornea was otherwise clear with a quiet anterior chamber. Right eye examination was unremarkable at this time. Corneal cultures for bacteria, viruses and fungi revealed no growth.

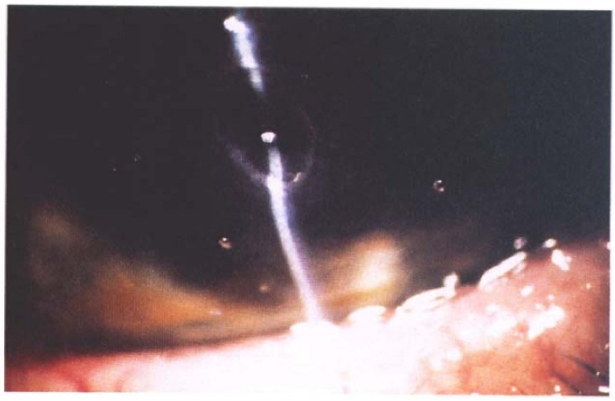

Fig. 2. *Left corneal ulcer at presentation.*

The patient was initially treated for bacterial keratitis with topical antibiotics (cefuroxime and ciprofloxacillin). Following the negative cultures and owing to the lack of clinical response the working diagnosis was modified to a sterile vasculitic ulcer. However, there was no improvement with hypromellose and prednisolone drops or with botulinum-induced ptosis. Hypromellose was the only therapy to her right eye at this time. Her medical therapy included nutritional support but she continued to lose weight.

One month after initial presentation she developed painless blurring of vision in her contralateral eye. Examination revealed a concentric paracentral corneal melt 1 × 1.5 mm, similar to that in the other eye. Corneal cultures again revealed no growth. Further biochemical investigation showed hypoalbuminaemia with serum albumin 23 g/l (normal 35–55 g/l). Vitamin A level was 0.07 mg/l (normal 0.2–0.6 mg/l) and serum beta-carotene was undetectable. The clinical diagnosis was revised to keratomalacia due to vitamin A deficiency. There was no clinical evidence of malabsorbtion, and clinically her weight loss and malnutrition was secondary to oeosphageal motility disorder.

In view of her deteriorating medical condition she was admitted for nasogastric and then intravenous feeding with vitamin A supplementation. At this stage her corneal ulceration began to heal. Her general health deteriorated further and she died 10 weeks later. Post-mortem examination showed no sign of malabsorption, but confirmed severe oesophageal involvement.

Comment

The differential diagnosis of keratomalacia includes severe sicca syndrome, infective, neuropathic and auto-immune ulcers and exposure. Our patient had a good tear film (an uncommon but recognised feature of vitamin A deficiency),[5] normal corneal sensation and good lid closure, her cultures grew no infective agent in either eye, and there was no clinical response to topical antibiotics. Auto-immune ulcers have not to our knowledge been reported in scleroderma[6] and there was no response to topical steroid. When the second eye became involved we recognised the classical picture of keratomalacia, a diagnosis supported by her markedly abnormal biochemical investigations. Further support for this diagnosis comes from her clinical response to vitamin A supplementation.

Classic xerophthalmic ulcers are sharply punched out, partial or full-thickness corneal defects that progress to localised stromal destruction with liquefactive necrosis (keratomalacia). Ultimately this leads to perforation.[7] Although the role of vitamin A deficiency in xerophthalmia is not in doubt, the mechanisms are unclear. Vitamin A is involved in corneal metabolism and specific retinol binding proteins are present in the epithelium, keratocytes and endothelium.[8,9] Inflammatory cells are visibly involved in keratomalacic corneas and are known to release proteases such as collagenase. It is rare for vitamin A deficiency to be

encountered in isolation. Usually it is part of generalised protein energy malnutrition which may lead to reduction in retinol levels both directly and by impaired conversion of beta-carotene. In addition protein status may influence vitamin A metabolism at the level of the target cell.[10]

In poor communities in developing countries ocular complications arising from the nutritional deficiency of vitamin A are relatively common, anticipated and readily diagnosed. In affluent societies better nutritional standards make such complications rare, and testing for such a deficiency may be overlooked. As many more patients who were once regarded as terminally ill now have a longer life expectancy, the likelihood of developing vitamin A deficiency should be considered. Deficiency lesions are reversible if they are recognised early and vitamin A replacement undertaken promptly. Failure both in diagnosis and treatment may lead to unnecessary blindness, and also increases the risk of severe intercurrent systemic infection and death.

We thank Mr Cappin, FRCS, for encouraging publication of this unusual case.

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Sir,

***Acanthamoeba* keratitis following optical keratoplasty**

Acanthamoeba infection of the corneal graft is a rare entity and to date only one such case has been reported in the literature.[1] Here a case of graft infection by *Acanthamoeba* is reported that presented initially as a persistent corneal epithelial defect.

Case report

A 42-year-old man underwent uneventful penetrating keratoplasty for leucomatous corneal opacity subsequent to healed viral keratitis. Post-operatively betamethasone drop 2 hourly and ciprofloxacin eye drop q.i.d. were administered along with frequent artificial tears. The findings at 8 weeks post-operatively were a graft clarity of 4+ and visual acuity of 6/6 with −0.75 dioptre sphere.

One week later the patient presented with diminution of vision. Examination by slit-lamp disclosed a central epithelial defect with a loose suture at the 2 o'clock position. The suture was removed and the area cleaned with betadine solution. The eye was patched for 48 h after applying ciprofloxacin ointment. As the eye patching for 48 h did not show complete epithelial healing, a bandage contact lens was applied. The patient used to remove the bandage contact lens after every 2 weeks and clean it with multipurpose solution followed by heat sterilisation. He used to pour contact lens solution containing the bandage contact lens, into the contact lens case before applying. Each time the contact lens case was washed with tap water.

The patient was comfortable with the bandage contact lens for 2 months. Thereafter he developed pain, photophobia, watering and diminution of vision in the same eye. On examination, the visual acuity was counting fingers close to the face with a central corneal ulcer measuring 7.5 × 7.55 mm (Fig. 1). The bandage contact lens was removed and sent for microbiological investigation.

Light microscopic examination of a Gram-stained slide made after corneal scraping revealed the presence of numerous Gram-negative bacilli with no polymorphs. A KOH wet mount preparation failed to identify any organisms. Culture examination of the scraping material,

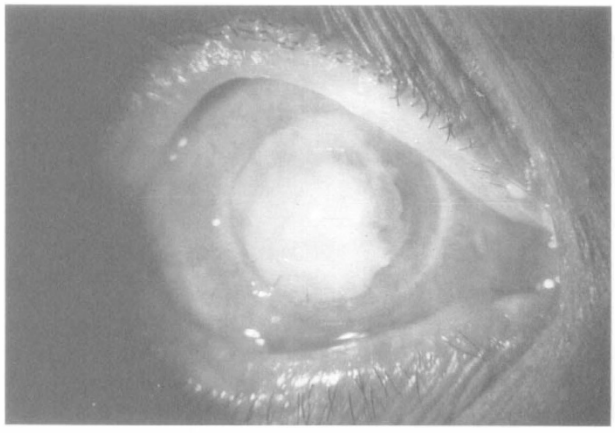

Fig. 1. *The appearance of the eye showing the central corneal ulcer.*