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Sir,

Vitamin A deficiency presenting with microbial keratitis in two patients in the UK

Vitamin A deficiency is a rare finding in the developed world. In the United Kingdom, corneal causes account for only 1.6% of cases of severe visual impairment in children.[1] We report one adult and one child who both presented with microbial keratitis as a consequence of the ocular surface features of vitamin A deficiency

Case 1

A 10-year-old boy of Afro-Caribbean descent presented in September 2003 with a 6-week history of a painful left eye with epiphora, which was not responding to chloramphenicol drops commenced by the local doctor. During this time, he had been unwell with anorexia, headaches, fevers and lethargy. Presentation to an emergency department 2 weeks previously had failed to reveal any specific systemic abnormality. Some years previously he had been investigated for haematuria for which no cause had been found. Otherwise there was no other history of significant systemic or ophthalmic problems, in particular, contact lens wear or trauma. Close questioning of the family revealed a 5-year history of poor dietary intake consisting of the following foods exclusively: rice, pasta, roast potatoes, hot chips, blackcurrant cordial, 'fairy cakes', deep-fried flour dumplings, chocolate biscuits, and prepacked strawberry/cherry-flavoured sweets. In particular, no fruit, vegetable, or meat was consumed.

Visual acuity was 6/9 right and 6/18 left. The left eye had periorbital oedema with an inferior left corneal infiltrate and overlying epithelial defect (approximately 2 mm horizontally by 1 mm vertically) and a small hypopyon (Figure 1). Widespread punctate epithelial erosions were present in both corneas. Both conjunctiva were dry looking and thickened with obscuration of normal vascularity, although no Bitot spots were noted. Both posterior segments were unremarkable.

A left corneal scrape was performed under general anaesthetic. Microscopy demonstrated large numbers of Gram-negative bacilli and a small number of polymorph neutrophils. Hourly by day and two hourly by night ofloxacin 0.3% drops were commenced, combined with cyclopentolate 1.0% twice daily. Culture grew *Pseudomonas aeruginosa*, sensitive to ciprofloxacin. The condition resolved over the coming 2 weeks of treatment leaving a small inferior corneal scar.

The clinical diagnosis of vitamin A deficiency was made. Vitamin A (retinol) blood assay confirmed a deficiency with blood concentration of <0.1 mg/l (reference range 0.2–0.8 mg/l). The paediatric unit reviewed the patient and were unable to find evidence of a malabsorption syndrome. In particular, his coeliac and inflammatory bowel disease markers were all within the normal range.

ERG demonstrated low amplitude in light- and dark-adapted conditions (Figure 2).

He took a short course of oral vitamin A supplementation (his diet had improved since discharge to include a broader range of foods). Systemically, there had been an improvement in his health. Subjectively, both eyes returned to normal; however, there was central punctate epithelial staining despite two to four times per day of polyvinyl alcohol lubricants. Visual acuity of the left eye was 6/9. At this stage, the ERG had shown recovery to a virtually normal level with light-adapted

Figure 1 Case 1 at presentation with corneal infiltrate, overlying epithelial defect and small hypopion.

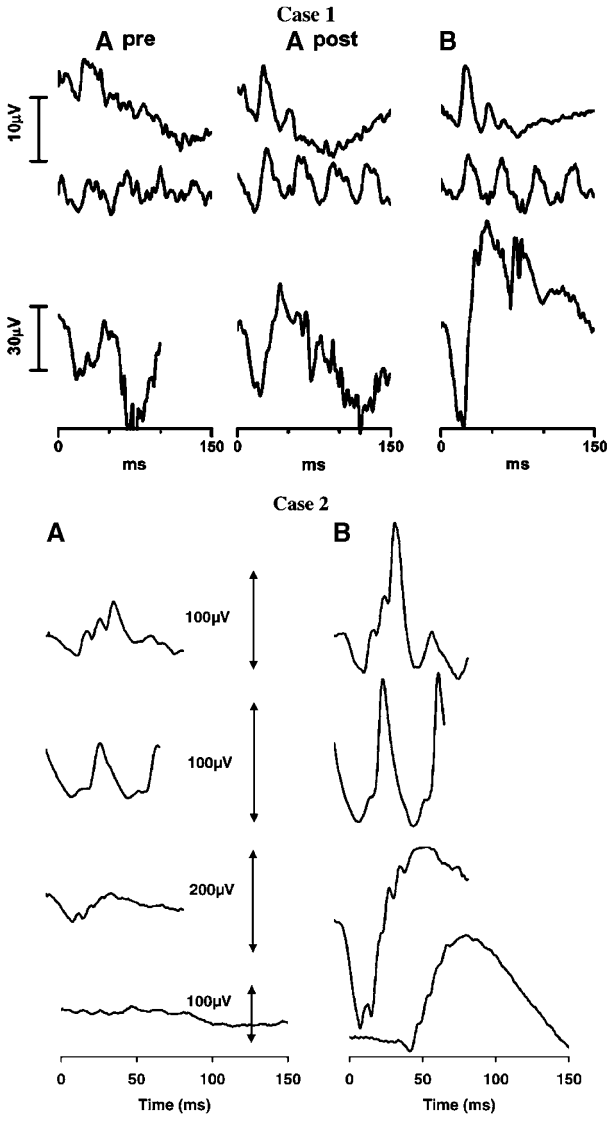

Figure 2 Photopic ERGs to single flash (top row), 30 Hz flicker stimulation (middle row), scotopic mixed response (bottom row) and rod response (very bottom row, Case 2 only) at presentation (A pre), and after vitamin A supplementation and dietary improvement (A post; note, no post-treatment data collected for Case 2). An age-matched normal ERG is shown in column B. ISCEV standards were adhered to where possible. In Case 1, the pupils were not dilated and an eyelid mounted lightweight skin electrode (Burden Neuroscience, Bristol) was used to aid compliance (subpalpebral DTL electrode used in Case 2). All data were from the left eye in Case 1 and right eye in Case 2.

flash and flicker stimulation, although the dark-adapted ERG was still attenuated.

Case 2

A 46-year-old White British lady presented in April 2004 to her local ophthalmologist with a 1-week history of a red, itchy left eye. She was diagnosed with left bacterial keratitis and a large right sterile corneal epithelial defect. The local ophthalmologist performed a left corneal scrape and commenced hourly ofloxacin to that eye. At follow-up 2 days later, the left cornea had perforated requiring further referral. Past ocular history consisted of 3 years of severe bilateral dry eyes and nyctalopia. She had been noncompliant with lubricants. There was a 3-year history of poor diet. Her intake consisted exclusively of white fish and beer. Her weight had fallen from 96 to 50 kg over the preceding year.

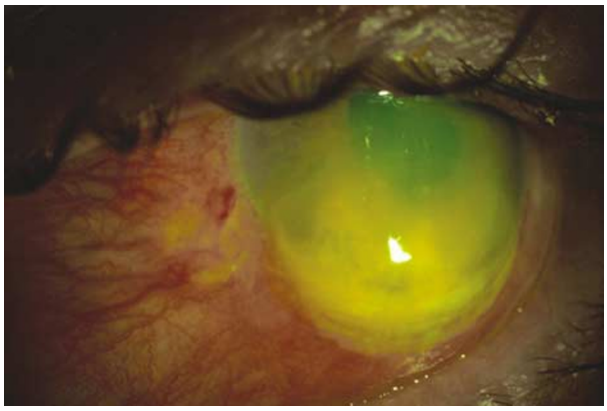

Figure 3 Case 2 at presentation with large crescent shaped infiltrate.

Visual acuity was count fingers bilaterally. The left eye had a large inferior crescent-shaped infiltrate (Figure 3). The anterior chamber was formed but shallow inferiorly, without any area of frank leak. There was a marked anterior chamber inflammatory reaction with posterior synechiae, but there was no obvious hypopyon. The right cornea had an almost complete epithelial defect without any focus of infection. Both conjunctiva appeared thickened with Bitot's spots and obscuration of blood vessels. The corneal scrape performed earlier demonstrated a light growth of *Streptococcus viridans*.

Ofloxacin 0.3% was continued two hourly to the left eye and four hourly to the right eye combined with bilateral half hourly preservative-free lubricants.
A clinical diagnosis of vitamin A deficiency was made. At referral, blood vitamin A levels were $<0.1\,\mu$mol/l (reference range 0.84–3 μmol/L). The ERG supported this diagnosis (Figure 2). Oral and intramuscular vitamin A supplements were commenced, with an oral intake programme supervised by a dietitian to minimize the risk of refeeding syndrome (a serious metabolic derangement, precipitated by too rapid nutrition reintroduction).

Subjectively, the patient made a rapid improvement as an inpatient over the following week. The left anterior chamber gradually deepened and the corneal epithelial defect had resolved by 1 month. The right corneal epithelial defect had resolved by day 6. On last review,

she had an inferior corneal scar sparing the visual axis and an early cataract. Final visual acuities were right 6/9 and left 6/12.

Comment

Worldwide, an estimated 260 000 children have severe visual impairment from corneal causes. This is the most important cause of avoidable worldwide childhood blindness.[2] Most of this occurs in 'low-income countries', in particular, those in Sub-Saharan Africa, India, and other parts of Asia There are, however, case reports of vitamin A deficiency presenting in developed communities. Inadequate diet is one mechanism. In one case a 7-year-old vegetarian British girl presented with Bitot's spots.[3] In another case, a 6-year-old child presented with xerophthalmia secondary to anorexia from severe gastro-oesophageal reflux and oesophagitis.[4] Malabsorption syndromes, particularly those affecting the terminal small intestine where vitamin A is absorbed, can also result in vitamin A deficiency. In one case, a child with short bowel syndrome presented with xerophthalmia.[5] Large intestine pathology is a less likely cause, although it can occur and has been reported in a patient who underwent a haemicolectomy.[6] In our cases, the patient's chosen diet was the cause of the deficiency. They presented with xerophthalmia complicated by microbial keratitis.

Both patients' ERG s showed evidence of significant retinal involvement with subsequent partial recovery after vitamin A supplementation. In keeping with other studies,[7] the rod-mediated ERG was more affected, but implicit times were unchanged. This reduction in ERG amplitude is said to correlate with reduced levels of the rod pigment, rhodopsin, resulting from the reduction in blood retinol.

It is important to consider vitamin A deficiency in the differential diagnosis of any 'ocular surface presentation' in developed communities with features of surface drying. In both cases, the patients presented with microbial keratitis, without any underlying ocular associations. Poor dietary history combined with conjunctival and corneal drying, in theses cases, suggested the diagnosis of vitamin A deficiency. Nyctalopia is usually the earliest manifestation of the disease, although it does not occur in all cases.[6] Prompt recognition and treatment of vitamin A deficiency with oral supplements combined with treatment for microbial keratitis brought about an effective recovery.

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Sir,

Suprachoroidal haemorrhage: a rare complication of cyclodiode laser therapy

Suprachoroidal haemorrhage (SCH) is a severe complication of intraocular surgery. We report the first case of delayed SCH following cyclodiode laser therapy for refractory glaucoma.

Case report

The subject was a 72-year-old man with bilateral aphakia and secondary glaucoma. He had undergone multiple penetrating keratoplasties and trabeculectomies in both eyes. Previous cyclodidode laser therapy in the left