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Sir,

Congenital Tarsal Kink: A Rare Cause of Neonatal Corneal Ulcers

Congenital tarsal kink is a rare condition of unknown origin, in which a fold is present in the upper tarsal plate at birth. The bent edge of the tarsus may then traumatise the cornea causing ulceration.[1–3]

Case Report

A 1-week-old baby girl presented to the Eye Department with a history of sticky eyes and failure of eye opening since birth. Perinatal conjunctival swabs were negative.

The mother's obstetric history was unremarkable, and maternal microbiological investigations proved negative. Ocular examination revealed bilateral upper lid oedema, absent lid crease and conjunctival chemosis in the upper fornices (Fig. 1). Bilateral central corneal ulceration was also present, with the right eye more severely affected (Fig. 2).

The baby was treated with intensive topical penicillin and tetracycline and oral erythromycin, which resulted in slight improvement in the clinical signs. Further extensive microbiological investigations failed to detect a causative organism. The possibility of non-accidental injury was excluded.

The left ulcer slowly epithelialised following 2 weeks of treatment, but the right eye showed only minimal improvement. Lid oedema had now settled and it was apparent that the tarsus of both upper lids was rotated. Manipulation of the upper eyelids resulted in correction of the tarsal anomaly and the ulcer healed with residual corneal scarring.

Although corneal scarring was asymmetrical, the right eye being more affected than the left, vision as assessed by preferential looking following resolution of the ulcers was equal in both eyes and within normal limits for the stage of development.

Discussion

Congenital entropion of the upper eyelids is extremely rare.[4] Congenital tarsal kink represents a severe form of entropion whereby the tarsal plate is kinked along its horizontal length causing inversion of the eyelid and lashes.[1–3] It is characterised by blepharospasm and absence of the upper lid fold. Severe corneal ulceration may result from the inturned lids and lashes abrading the cornea. It is thought that mechanical trauma to the cornea occurs *in utero*.[2] The aetiology of the condition is unknown; a primary defect of the tarsus has been proposed as a possible underlying abnormality. Alternatively, overaction or malpositioning of orbicularis muscle fibres may result in an infolding of the tarsus *in utero*.[5]

Primary management of congenital tarsal kink requires manual unfolding of the tarsal plate. The lid may then be taped shut and a pressure dressing applied for 24–48 hours.[6] A bandage contact lens may aid corneal healing.

More severe cases require surgical management. A variety of surgical procedures have been described, including tarsal split,[1] tarsal wedge resection,[7] lamellar tarsoplasty[2] and repositioning of the anterior lamella.[5,8] As the condition may be corrected by altering the position of the orbicularis and creating a skin crease with a simple

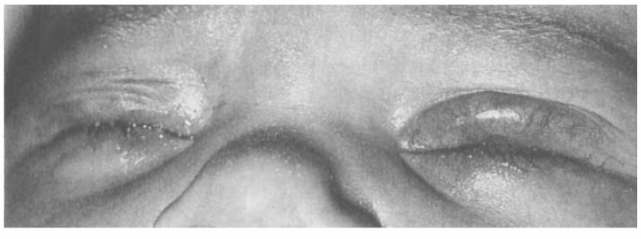

Fig. 1. *Eyelids showing tarsal plate anomaly.*

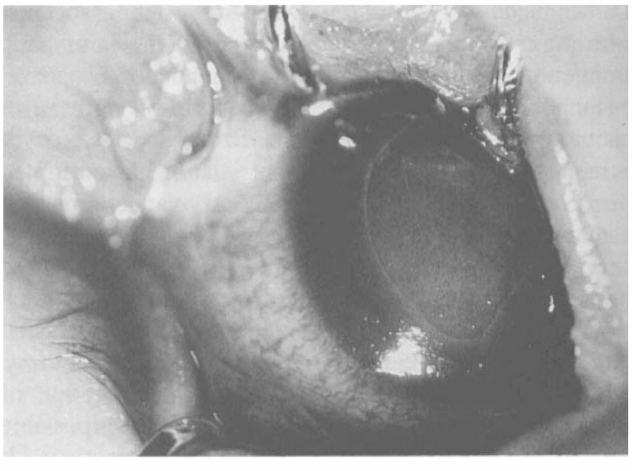

Fig. 2. *Right cornea showing central ulceration.*

anterior lamellar repositioning procedure, this would suggest that the primary lesion may be in the orbicularis muscle or in the formation of the lid crease.[5]

The rare occurrence of congenital tarsal kink often results in delay of diagnosis, and prolongs the course of corneal ulceration. It is important to consider congenital lid anomalies in the differential diagnosis of neonatal corneal ulcers, especially in those cases which do not respond to treatment.

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Sir,

Microsporidial Keratoconjunctivitis Treated Successfully with a Short Course of Fumagillin

Several case reports have recently described the successful treatment of microsporidial infection in the cornea and conjunctiva of HIV-infected patients with fumagillin. To date the most efficacious dosing regimen has not been determined. We describe a case of microsporidia treated successfully with a short course of Fumidil B.

Case Report

A 43-year-old HIV-positive white man with a CD4 count of 20 cells/mm^3 presented with a 4-month history of ocular irritation, fluctuating visual acuity, and burning sensation in both his eyes. He was thought to have herpes zoster keratitis, and was treated with oral acyclovir 4000 mg a day for 2 weeks, but failed to improve. He was referred for further evaluation when corneal epithelial opacities were noted.

On initial examination, his best corrected visual acuity was 6/12 right eye and 6/18 left eye. There was a moderate mixed papillary and follicular reaction of the palpebral conjunctiva in both eyes. The corneas were covered with diffuse punctate epithelial opacities with irregular surface fluorescein staining. There was also mild fluorescein staining of the bulbar conjunctiva. The remainder of the examination was unremarkable.

Numerous intracytoplasmic oval, dark-staining organisms consistent in morphology with microsporidia[1,2] were found in Giemsa-staining corneal scrapings (Fig. 1). Culture, Giemsa, and direct fluorescent antibody stain for herpes simplex and fluorescent antibody for herpes zoster were negative.

Treatment of the keratoconjunctivitis with propamidine isethionate 0.1% six times daily[3] was commenced in the right eye, using the left eye as an untreated control. There was little change after 10 days of therapy. Treatment with Fumidil B (Mid-Con) 5.2 mg/ml (0.11 mg/ml active fumagillin)[4,5] in an artificial tear preparation was then commenced in the left eye at a frequency of one drop every 3 hours. After 48 hours the patient reported marked improvement in symptoms and a decrease in cornea epithelial staining in the left eye was observed. The propamidine isethionate 0.1% was stopped and fumagillin was begun in the right eye. The patient stopped the fumagillin due to complete resolution of symptoms after only 3 days of therapy.

Ocular examination demonstrated a best corrected visual acuity of 6/6 in each eye. There was a mild papillary reaction and the corneas had an irregular epithelium with no fluorescein staining. The patient has remained free of recurrences for over 16 months.

The clinical findings and laboratory investigation of the

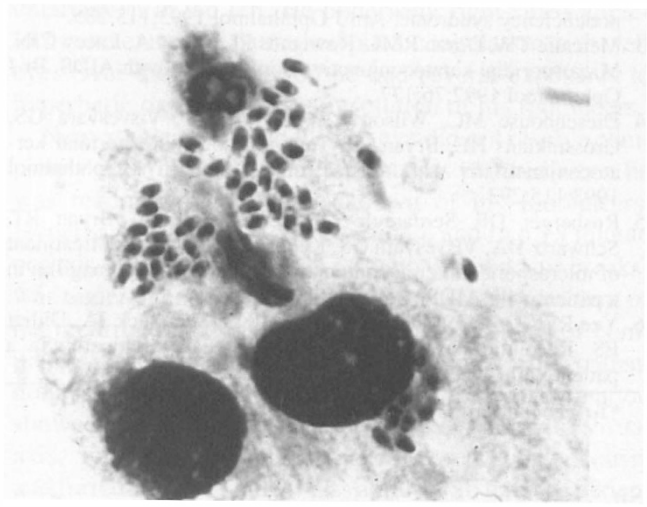

Fig. 1. *Giemsa-stained smear of human corneal epithelium demonstrating the small oval intracellular microsporidial spores. (×1000)*