a risk-free procedure. It illustrates the importance of careful technique in the hands of experienced surgeons and the need for careful supervision for trainee surgeons.

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Sir,

A case of recurrent infectious crystalline keratopathy secondary to *Haemophilus influenzae*

A 50-year-old man presented to our hospital with a 3-day history of blurred vision in his right, only eye. He had a history of bilateral congenital glaucoma leading to a left enucleation at the age of 3 years. He had undergone a right extra capsular cataract extraction without lens implant and two trabeculectomies.

There was a history of two right penetrating keratoplasties. The first was in 1991 for bullous keratopathy. In 1994, an episode of crystalline keratopathy occurred inferotemporally in the graft host junction. A corneal scrape grew *Haemophilus influenzae*, sensitive to penicillin, chloramphenicol, erythromycin, and tetracycline. After an initially poor clinical response to penicillin, the lesion responded to cefuroxime and ticarcillin. The second penetrating keratoplasty was in 1994 for corneal opacification secondary to this infection.

In the second presentation, his right visual acuity was 6/60. The cornea was clear and the anterior chamber had a moderate amount of flare and cells. He was taking the following medications: prednisolone acetate 1% drops twice daily, timolol 0.25% twice daily, pilocarpine gel nocte, latanoprost nocte, and polyvinyl alcohol as required. A provisional diagnosis of corneal graft rejection was made and the frequency of prednisolone acetate 1% drops was increased to six times per day.

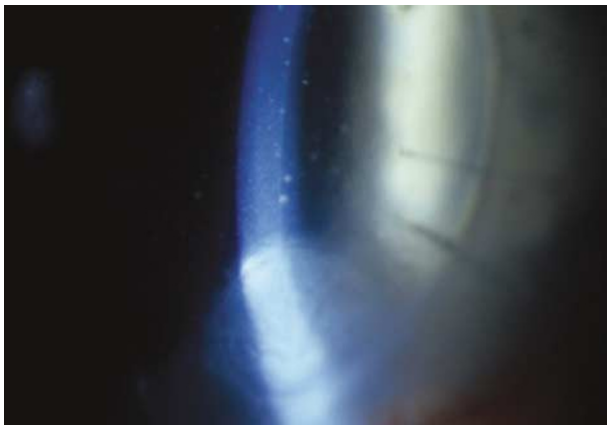

Figure 1 Slit-lamp examination demonstrating infiltrate with front-like processes.

He returned 3 days later without any subjective improvement. A corneal infiltrate with frond-like processes spreading into the surrounding stroma and 1.8 mm diameter overlying epithelial defect had developed in the peripheral donor inferonsasally (Figure 1). The stroma was thinned to 40% of normal. The corneal scrape stained with Gram stain demonstrating no neutrophils or organisms. Moderate growth of *Haemophilus influenzae* was noted on Chocolate agar plates, 24 h after inoculation. The organism was sensitive to chloramphenicol, ciprofloxacin, ofloxacin, gentamicin, and resistant to cefuroxime. The infiltrate healed over a period of 2 months with hourly ofloxacin 0.3% drops for 3 days, then reduced to 2 hourly and stopped after 2 weeks. Chloramphenicol 0.5% four times per day was then commenced for a duration of 2 months. A bandage contact lens was used from 2 weeks post presentation until resolution of the corneal epithelial defect at 2 months. This resulted in a vascularised stromal scar and localised thinning.

Discussion

To our knowledge, this is the first reported case of crystalline keratopathy secondary to *Haemophilus influenzae*. Crystalline keratopathy is an indolent corneal stromal infection characterised by a sharply demarcated white infiltrate with arborescent branching processes often in the absence of significant stromal or anterior chamber inflammation or epithelial defect.

This occurs most commonly in patients post corneal graft receiving topical steroids. The responsible organism can be hard to culture and the condition difficult to treat.[1]

The commonest causative agent is Streptococcus viridans.[2] Other aetiological agents include other Gram-positive and -negative organisms,[3] Candida species,[3] and *Haemophilus aphrophilus*.[4] In this previously reported case of *Haemophilus* crystalline keratopathy, the presentation was a similar chronic course, although there was a history of preceding chronic contact lens use and there was no anterior chamber activity.

The condition has been reported in two successive grafts in one patient,[5] where Streptococcus viridans was the cause in the first graft and *Candida albicans* the cause in the second.

Unusually, in our patient, the only sign at presentation was increased anterior chamber inflammation without corneal infiltrate or epithelial defect. A corneal infiltrate only became evident 3 days later. In another case report,[6] the patient's initial signs were nonspecific stromal thickening and descemet membrane folds without significant anterior chamber inflammation or corneal infiltrate which progressed to graft failure. Histology at regrafting identified multiple peripheral intrastromal Gram-positive cocci with minimal posterior stromal neutrophils. This patient had no clinical infiltrate or histological discrete focus of organisms.

Electron microscopy study of this condition has demonstrated the importance of biofilm formation in its pathogenesis where bacteria are surrounded by a polysaccharide- and glycoprotein-rich matrix presumably secreted by the organism.[7] This matrix sequests the organism, limiting the host inflammatory response. The branching appearance of infiltrate is a consequence of the collagenous lamellar architecture.[8]

Clinical isolates of *Haemophilus influenzae* from cases of otitis media in children and lower respiratory tract infection in adults with chronic obstructive pulmonary disease show marked variation in their ability to produce biofilm, depending on the strain.[9] These differences are reproducible when confirmed under repeated testing. The ability to produce biofilm relates to the type of lipooligosaccharide[9,10] expressed and presence or absence of pili.[9] The type of outer membrane protein expressed[9] was not found to influence biofilm formation. The relevance of these findings to corneal infections is unknown.

This report adds a further organism to the list of those capable of producing infectious crystalline keratopathy, and was unusual in that the same organism resulted in a recurrent episode after an interval of 10 years.

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