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MYCOBACTERIUM FORTUITUM KERATITIS

C Sanghvi

Abstract

We report a case of mycobacterial keratitis characterized by apparently spontaneous onset, delayed diagnosis, and eventually necessitating evisceration inspite of systemic antibiotics and repeated corneal grafts.

Key words: Corneal grafts, evisceration, keratitis, non-tuberculous mycobacteria

Non-tuberculous mycobacteria (NTM) are opportunistic pathogens implicated in indolent infections that are difficult to eradicate. *M. fortuitum* and *M. chelonae* are the commonest NTM causing ocular infection. They are ubiquitous in soil and water and can cause infection when surgical instruments or clinical devices are exposed to contaminated water, ice or other solutions. These pathogens are resistant to nutritional deprivation, temperature extremes and chemical disinfectants such as chlorine, which contributes to their occurrence in keratitis after surgical procedures.

Case Report

A 78-year-old man developed a sore right eye while on holiday in New Zealand. There was no history of any antecedent injury. At presentation, his vision was 6/60 and he had a hypopyon and nasal corneal infiltrate, which failed to respond to topical antibiotics (ciprofloxacin two hourly) or yield an organism on scraping and subsequently perforated. On his immediate return to UK, the leak was glued and a bandage contact lens applied. Postoperative treatment consisted of chloramphenicol ointment six hourly and artificial tears two hourly. The anterior chamber (AC) reformed, but a raised, vascularised conjunctival lesion at the nasal limbus was noted and thought to be responsible for a dellen effect resulting in corneal thinning, consequent infection and perforation. Excision of this lesion and a 4-mm amniotic membrane graft was undertaken after a week once the eye was stable. Histopathology revealed an excessively vascularised pingueculum.

He re-presented 3 months later with a central corneal abscess (Fig. 1), 5 × 4 mm in size, with feathery satellite infiltrates and hypopyon. Two repeat corneal scrapings for culture of bacteria, fungi and viruses were negative. Topical gentamicin, cefuroxime, chloramphenicol, ofloxac cin and prednisolone acetate for 3 weeks were ineffective. Finally, corneal biopsy revealed acid-fast bacilli (AFB) with Ziehl Neelsen (ZN) staining (Fig. 2). Treatment was changed to topical amikacin (20 mg/mL) and systemic ciprofloxacin (750 mg bd), but the cornea perforated. A 7-mm therapeutic penetrating keratoplasty (Fig. 3) and AC washout with 2 mg/mL unpreserved amikacin was undertaken. The corneal button was divided for microbiologic and histopathologic examination. Postoperative treatment consisted of topical amikacin (20 mg/mL), topical prednisolone 0.5% qds and oral ciprofloxacin 500 mg bd.

*Mycobacterium fortuitum* susceptible to amikacin, azithromycin and clarithromycin and resistant to ciprofloxacin, gentamicin and cefuroxime was isolated from the host cornea after prolonged chocolate agar incubation. Histopathological examination of biopsy material revealed the presence of NTM. On histopathology, the ulcer extended into the posterior third of the stroma with an acute inflammatory infiltrate at all levels. Oral ciprofloxacin was substituted for oral azithromycin 500 mg bd.

Two weeks later, discrete infiltrates developed near the graft host junction (Fig. 4). Despite systemic azithromycin (500 mg bd) and topical amikacin (20 mg/mL), a hypopyon appeared and the graft perforated. One month after the initial graft, the patient underwent regrafting with a large 9.5-mm donor cornea combined with intracameral irrigation of amikacin (4 mg/mL). Topical and intracameral anaesthesia was necessary for the procedure since azithromycin had interacted with his warfarin treatment for cardiac arrhythmia resulting in a high international normalized ratio of 5.3, thus posing a risk of bleeding with injectable anaesthesia. Postoperatively, azithromycin was stopped after 3 days due to deteriorating renal function, but topical amikacin and prednisolone continued. However, he re-presented months later with infiltrates around two sutures resulting in a wound gape and AC leak. A corneal scraping confirmed recrudescence of the same infection. After two failed grafts and systemic contraindications to azithromycin, further surgery to restore ocular integrity seemed to hold little prospect of clearing the infection and he underwent uneventful evisceration.

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A long latent period is typical of mycobacterium keratitis, reported to have developed 2 years after radial keratotomy. The slowly progressing keratitis may mimic the indolent course of fungal or herpetic infection. Lack of response to conventional antibacterials or antivirals should raise the possibility of NTM infection. Often the patient receives corticosteroids prior to identification of infection and this may prolong the course of the disease.

Mycobacteria appear as red rods on ZN staining and require prolonged incubation on chocolate agar, which may not be included in the routine microbiologic workup for keratitis. Culture from scrapings may be negative because the infection is deep in the cornea. A corneal biopsy yielded the organism in this case.

Nearly all reported cases of this rare opportunistic infection have followed corneal trauma. Foreign bodies, contact lens wear, pterygium surgery and penetrating keratoplasty have all been implicated. Mycobacterium interface keratitis after laser in situ keratomileusis (LASIK) is being increasingly reported.

Infection causes multiple foci of disease at various levels and the organism is often sequestered deep in the cornea. Definitive treatment may thus require complete excision of the infected tissue and has in most post-LASIK cases necessitated flap removal. In this patient, recrudescence of infection occurred in both grafts, necessitating complete elimination of infected tissue by evisceration.

Medical treatment of mycobacterial keratitis is prolonged and difficult due to deep-seated infection, poor drug penetration, slow growth of mycobacterium and survival within keratocytes and macrophages. In vitro susceptibilities and clinical response to treatment may poorly correlate.
addition, long-term antibiotics may be potentially toxic. In one series, 64% did not respond to medical treatment and required surgery.\textsuperscript{12}

This case emphasizes the indolent nature of the infection and its unresponsiveness to conventional diagnostic and treatment methods. The creation of an interface on the cornea either after LASIK as discussed above or in the form of an amniotic membrane graft, as in our case, seems to perpetuate the problem. The occurrence of keratitis after the creation of an interface on the cornea should therefore require a low threshold of suspicion for NTM. The virulent nature of this organism, which in this patient led to evisceration in spite of vigorous diagnostic and therapeutic measures, should be borne in mind when treating this infection.

References


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