second culture of the discharge revealed the same organisms. We subsequently started oral ampicillin together with topical chloramphenicol. The patient showed significant improvement in the first week, and the canaliculitis gradually resolved (Fig. 2). In January 2005, 2 years after the canaliculitis, there had been no recurrence.

Comments

*Lactococcus lactis cremoris* is one of the lactic acid bacteria. It is associated with dairy food products. *L. lactis cremoris* is classified as nonpathogenic and possibly even beneficial to health.

In recent years, however, its pathogenic potential is increasingly being understood. Human infections due to *L. lactis cremoris* (*Streptococcus lactis*), include septicaemia, cerebellar abscess, and periodontitis.

*Eikenella corrodens* is part of the normal flora in the human oral cavity. It is increasingly being recognised as a human pathogen. It has been reported in canaliculitis, dacryocystitis and even endophthalmitis.

For an opportunistic infection to occur with these commensals, first, there should be a substantial increase in the number of the opportunistic bacteria. Second, the host defence should be depressed. Both factors could have been responsible for the canaliculitis in our patient: First, our patient had periodontal disease, which might have resulted in an overgrowth of the two organisms in the oropharyngeal area. Second, an immunocompromised state related to her diabetes mellitus might have precipitated an opportunistic infection by both *L. lactis cremoris* and *E. corrodens*.

In any chronic canaliculitis not responsive to conventional treatment, and especially in an immunocompromised individual, we should have a high index of suspicion for unusual organisms, or even a mixed infection, as in our case. With prompt and appropriate antibiotics, the clinical outcome appeared favourable.

As far as we are aware, this is the first reported human ocular infection by *L. lactis cremoris*. This organism should be added to the list of etiological agents causing canaliculitis.

**Key Words:** canaliculitis, *Lactococcus lactis cremoris*

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Received: May 8, 2005 / Accepted: September 12, 2005

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DOI 10.1007/s10384-005-0318-5

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Bilateral *Mycobacterium chelonae* Keratitis After Laser in Situ Keratomileusis

Post-laser in situ keratomileusis (-LASIK) infectious keratitis, a rare but vision-threatening complication of the LASIK procedure, has aroused attention recently with the increase in the number of LASIK procedures being performed. Nontuberculous *Mycobacterium* is an important pathogen causing post-LASIK keratitis because of the difficulty in its diagnosis and management.¹ ³ We report a case of bilateral *Mycobacterium chelonae* keratitis after LASIK that was successfully treated with fortified topical antibiotics as an adjunct to flap amputation.

Case Report

A 25-year-old man underwent uneventful bilateral simultaneous LASIK surgery on December 8, 2001, with the right eye operated on first. Two days after the surgery, he began to experience bilateral progressive blurred vision and ocular pain. Flap lifting with antibiotic irrigation was repeatedly performed, and topical ciprofloxacin 0.3% was prescribed for both eyes. Because of the aggravated symptoms and signs, the patient was referred to our hospital.

The initial slit-lamp examination showed diffuse multifocal milary stromal infiltration at the interface of the LASIK flap with stromal edema in his right eye (Fig. 1A), and localized stromal infiltration over the lower nasal area in his left eye (Fig. 1B). Flap lifting, scraping of the stromal infiltrates, and amikacin (10mg/ml) irrigation were performed in both eyes. The scraping smear revealed numerous acid-fast bacilli. Bacteriological cultures grew *Mycobacterium chelonae*. The medication was changed to topical amikacin 2.5% and clarithromycin 1%, based on the in vitro sensitivity test.

Despite the intensive antibiotic treatment, the stromal infiltrates became confluent with progressive flap melting and total epithelial defects. The corneal flap of his right eye was amputated on January 23, 2002. The keratitis in his left eye was controlled with amikacin and clarithromycin. These medications were continued and tapered over the following
3 months. Six months after diagnosis, the corneal surface was smooth with a grade II haze in the right eye (Fig. 2A) and a 3+ scar at the interface in the left eye (Fig. 2B). The uncorrected visual acuity of the right eye and left eye was 20/25 and 20/60, respectively.

**Comments**

Post-LASIK mycobacterial keratitis has been noted with increasing frequency recently.1-3 Eyelashes, conjunctiva, irrigating solution, the surrounding atmosphere, and all the instruments used during LASIK could be likely sources of mycobacterial infection. The mycobacterial keratitis in our case was more severe in the right eye than in the left eye. Because the surgery was performed using the same instrument with the right eye operated on first, it is possible that more pathogens were inoculated into the right eye than into the left. A previous report4 and our case show that the risk of bilateral infection after bilateral simultaneous LASIK should not be overlooked.

The treatment of mycobacterial keratitis is difficult because of its indolent clinical course and delayed diagnosis, and the inadequate corneal penetration of topical antibiotics.5 Immediate flap lifting to obtain specimens for smear and culture is important for accurate diagnosis and further treatment. Surgical management is often necessary to eradicate the infection.5 Amputation of the corneal flap, another form of therapeutic lamellar keratectomy, should be considered for post-LASIK mycobacterial keratitis that is refractory to medical treatment. In this patient, we used this effective therapeutic modality to eradicate mycobacterial keratitis successfully.

Another interesting finding of this case is the visual prognosis of the patient. The visual acuity of the right eye after flap amputation was even better than that of the left eye. The interface scar of the left eye was denser and caused more irregular astigmatism than that of the right eye, which healed smoothly with less scarring after flap amputation. This case suggests that flap amputation before destruction of the stromal bed not only helps control the