Eikenella corrodens keratitis: case report

LISA KELLY AND JOSEPH ELIASON

From Stanford University Division of Ophthalmology, Stanford, California, USA

SUMMARY Eikenella corrodens is a Gram-negative facultative anaerobe which is part of the normal human oropharyngeal flora and an opportunistic pathogen of mucous membrane tissues. We report a case of secondary E. corrodens ulcerative keratitis with hypopyon in a 39-year-old male with herpes simplex keratitis. To the best of our knowledge this is the first reported case of E. corrodens as a pathogen in bacterial keratitis.

In 1948 Henriksen described a previously unreported anaerobic Gram-negative bacillus that grew in an unusual pitting pattern on agar.\(^1\) Rather than growing on the surface agar colonies of this organism formed depressions in the media. Because of its growth pattern Holm gave this newly discovered organism the name ‘corroding bacillus’.\(^2\) It was not until 1958 that Eiken\(^1\) did a thorough study of the organism, recording the colonial growth patterns and further defining its biochemical properties. From this research he proposed that the bacteria be placed in the genus Bacteroides and gave it the name Bacteroides corrodens. Further work by Henriksen\(^3\) and Jackson et al.\(^4\) revealed that a subgroup of B. corrodens was in fact not strictly anaerobic. Immunological and nucleotide base pair analysis of this subgroup uncovered properties not consistent with the characteristics of Bacteroides. Subsequently this group was removed from the Bacteroides genus and renamed Eikenella corrodens in the family Brucellaceae.\(^5,6\)

Eikenella corrodens appears to be part of the normal flora of the human oropharynx. Khairat\(^7\) examined 100 blood cultures one minute after a tooth extraction. He isolated Eikenella from 16 of these cultures; in three cases Eikenella was the sole isolate. Brooks et al.\(^8\) isolated Eikenella from 13 out of 40 gingival cultures in clinically uninfected people. These results suggest that Eikenella frequently colonises the human upper respiratory tract and that it is capable of invading the blood stream. The first work to document the pathogenic capabilities of this organism was done by Marsden and Hyde,\(^9\) who reported six cases of Eikenella corrodens infection in children, including two cases of brain abscess secondary to sinusitis, two gastrointestinal abscesses, a human bite, and a case of sinusitis. In another study at Boston City Hospital retrospective analysis revealed 72 cultures positive for Eikenella during 1971–2.\(^10\) Of these isolates 46 were obtained from sputum of bronchial washings, 11 throat or oropharyngeal cultures, 8 various wounds, 3 abscesses, 2 human bites, a middle ear aspirate, and a tooth socket. This review suggests that Eikenella, in addition to frequently being part of the normal flora of the human respiratory tract, can act as an opportunistic pathogen with a predilection for mucous membranes of the respiratory system and gastrointestinal tract.

In the ophthalmic literature Schwartz et al. have reported two cases of Eikenella infection in orbital cellulitis.\(^11\) In one case the cellulitis was secondary to sinusitis and in the other case there was an infection of an ocular prosthesis after orbital exenteration. Jones and Robinson,\(^12\) in a review of anaerobic ocular infections, reported a case of endophthalmitis following penetrating keratoplasty with positive cultures for Staphylococcus epidermidis and Bacteroides corrodens (sic), and also a case of chronic dacryocystitis with a mixed culture that included Eikenella corrodens. In an unreported case we have also treated a 72-year-old female with chronic dacryocystitis with cultures positive for two strains of Eikenella in mixed flora. To the best of our knowledge this is the first reported case of Eikenella corrodens keratitis.

Case report

A 39-year-old male with a history of recurrent herpes simplex keratitis of his left eye since 1967 presented to his ophthalmologist on 20 February 1986 with a
complaint of discomfort in the left eye. He was diagnosed as having a dendritic lesion on his left cornea and given topical trifluridine every three hours and scopolamine 0.25% three times a day; no antibiotics were given. The patient returned to the doctor three days later complaining of a marked increase in pain and decreased visual acuity. He was noted to have developed a corneal ulcer with hypopyon and was referred to the Palo Alto Veterans Administration Hospital for treatment.

On our initial examination the patient had a visual acuity of 20/20 OD, with adnexa and anterior and posterior segments of the right eye completely within normal limits. His left eye had an acuity of counting fingers at 30 cm. Slit-lamp examination revealed 1+ conjunctival injection and chemosis; the cornea had a 2.5x2.75 mm paracentral epithelial defect, with dense underlying stromal white infiltrate extending to mid stroma, overlying the site of the original dendritic lesion. The anterior chamber had a 1 mm hypopyon. There was no vitreous reaction, and funduscopic examination showed a normal appearance. The patient was admitted to hospital. Prior to antibiotic therapy corneal scrapings were obtained and plated on chocolate, blood, and Sabouraud's agar and thioglycollate broth media. Empirical therapy of topical cefazolin 100 mg/ml and tobramycin every half hour, and parenteral cefazolin 1 g every eight hours was initiated. On the patient's fourth day in hospital the cultures grew on blood agar a single organism identified as *Eikenella corrodens*. It was sensitive to amikacin, ampicillin, cephamandole, cefoxitin, cephalothin, chloramphenicol, gentamicin, nitrofurantoin, tetracycline, tobramycin, trimethoprim-sulphamethxazole, and ticarcillin. On this regimen the epithelial defect healed by hospital day eight. On day 10 in hospital the topical cefazolin and parenteral cefazolin were discontinued, and the patient was discharged from the hospital on topical tobramycin. By day 16 of treatment the infiltrate had resolved, and 56 days after the patient's initial presentation he had only a faint central stromal scar. Visual acuity had returned to 20/20. He did not receive steroids at any time during treatment.

**Discussion**

In recent years the role of anaerobic organisms in ocular infection has come under increased investigation. Jones and Robinson in a review of anaerobic ocular infections report five cases of keratitis. The anaerobic isolates were, in the first case, *Propionibacterium avidum*, in the second case *Actinomyces viscosus*, and in the third case both *Propionibacterium acnes* and *Peptococcus vanabilis*. Cases 4 and 5 both grew *Propionibacterium acnes*. Four of the five patients had pre-existing epithelial ulceration and stromal inflammation predisposing to opportunistic infection. The antecedent ocular disease in the first case was pseudomonas keratitis, the second case had both herpes zoster and herpes simplex keratitis, the third case had herpes simplex stromal keratitis, the fourth case had had a penetrating keratoplasty, and the fifth had cicatricial pephigoid. It is of note that two of Jones and Robinson's patients had pre-existing herpes simplex keratitis,14 as did our patient. *Eikenella corrodens* is a Gram-negative bacillus and facultative anaerobe that grows reliably only on blood agar. The optimal growth of the organism is dependent on a haemin concentration of 50 to 250 µg/l of media. Primary isolation is enhanced by a 5–10% carbon dioxide atmosphere *Eikenella* does not grow well in liquid media unless they are enriched with sheep serum or cholesterol.8

During the past two decades the role of *E. corrodens* as an opportunistic pathogen in infections of the upper respiratory and gastrointestinal mucous membranes has been well documented.15 There is no evidence that *Eikenella* is a part of the normal conjunctival flora. Perkins et al.16 cultured, both aerobically and anaerobically the conjunctivae of 273 eyes with conjunctivitis and of 96 normal persons and did not record any *Eikenella* isolates. However, this organism typically is slow growing and frequently requires 24 to 48 hours of incubation before the characteristic pitting colony pattern can be identified. This long period of incubation could cause *Eikenella* in a mixed flora to be overlooked.

This case reinforces the need to consider facultative anaerobic pathogens in keratitis cases, particularly in patients with compromised corneas that may be predisposed to opportunistic infection.

**References**

Lisa Kelly and Joseph Eliason


Accepted for publication 1 October 1987.