SCIENTIFIC REPORT

Moraxella keratitis: predisposing factors and clinical review of 95 cases

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Aim: To analyse the clinical presentation, identify predisposing risk factors and evaluate the outcome of treatment of *Moraxella* keratitis.

Methods: A retrospective analysis was carried out of culture-proved cases of *Moraxella* keratitis from hospital records during a 10-year period (from December 1995 to November 2005) at the Corneal Unit of the Royal Victorian Eye and Ear Hospital, Melbourne, Australia.

Results: 95 episodes of *Moraxella* keratitis were identified in 92 patients. 3 (3.2%) patients had recurrent keratitis. The mean age of the patients was 70 (range 17–93) years. Multiple predisposing factors were identified in 23 (24%) eyes, including corneal graft (n = 15), previous herpes keratitis (n = 15) and eye lid diseases (n = 15). Adjunctive procedures were carried out in 42 eyes. These included botulinum toxin injection (n = 17), tarsorraphy (n = 12), penetrating keratoplasty (n = 8), enucleation (n = 3), tissue adhesive and bandage contact lens (n = 4), and conjunctival flap (n = 5). Polymicrobial infection was present in 17 eyes. Final visual acuity was counting finger or less in 25 (26%) eyes.

Conclusions: Local ocular predisposing factors play a major role in *Moraxella* keratitis. This infection has a poor visual outcome attributable to both the nature of the infection and the predisposing factors.

Bacterial keratitis is a serious ocular infectious disease which can lead to considerable loss of vision.[1] The severity and outcome of corneal infection usually depend on the underlying condition of the cornea and the virulence of the organism. Bacterial keratitis is rare in the absence of predisposing factors.[2 3] *Moraxella* sp are well-known causative agents of bacterial keratitis and often occur in people who are chronic alcoholics or otherwise immuno-compromised.[4]

Although it is often a less virulent opportunist organism, *Moraxella* can cause severe vision-threatening complications. *Moraxella* ocular infections range in severity from angular blepharoconjunctivitis[5] to severe stromal involvement with corneal perforation.[6] The pathogenesis of these ulcers is related to the production of proteases and endotoxin.[7 8]

Moraxella is an aerobic, non-motile, large thick Gram-negative rod. It has a tendency to resist decolourisation during Gram staining. In smears from tissues, the organisms are short and stout and typically appear in pairs. Occasionally, they may appear in short chains. Growth is slow on blood agar and very poor on chocolate agar. Incubation at 37℃ in a moist atmosphere improves growth. Colonies on blood agar at 24 h are tiny circular, raised, grey and translucent. With further incubation, they become somewhat larger and more opaque.

Our experience with a large case series has prompted us to retrospectively review the predisposing risk factors, clinical presentation and outcome of treatment of *Moraxella* keratitis.

MATERIALS AND METHODS

The clinical records of all culture-proved cases of *Moraxella* keratitis seen at the Royal Victorian Eye and Ear Hospital (Melbourne, Victoria, Australia) between December 1995 and November 2005 were retrospectively reviewed. The following data were collected from each record: age, sex, predisposing factors (ocular and systemic), clinical presentation, mode of management and outcome of treatment.

A detailed microbiological investigation was carried out on each patient after slit-lamp examination. All ulcers were routinely scraped for Gram and Blankophor smears, and for plating on blood agar, chocolate agar, Sabouraud's dextrose agar and thioglycolate broth. Swabs were taken for herpes simplex virus where there was a clinical suspicion of associated viral infection. The decision to admit patients was influenced by the severity of the infection based on overall clinical impression and the ability of the patient to instil drops intensively. Intensive medical treatment was started in all patients. The initial intensive antibiotic instillation protocol was 1 drop/h for 48 h. The antibiotic drops were progressively tapered or modified according to the clinical response and bacterial susceptibility. Adjunctive procedures such as tissue adhesive application, tarsorraphy, botulinum injection and therapeutic penetrating keratoplasty were carried out as required. The indications for adjunctive procedures were corneal thinning and perforation, persistent epithelial defect, and eye lid disorders such as ectropion and lagophthalmos.

RESULTS

Ninety five episodes of *Moraxella* infection in 92 patients were reviewed; three (3.2%) patients had recurrent infection. Over this period, 3378 patients with bacterial keratitis were treated at the Royal Victorian Eye and Ear Hospital. The age of the patients ranged from 17 to 93 years (mean age 70 years) with 74 (77.8%) patients >60 years old. Most of the 92 patients were women (M:F 44:48). Seventy of them were treated as inpatients during intensive medical treatment.

An ocular predisposing condition was identified in 78 eyes. Multiple predisposing factors were present in 23 (24%) eyes (table 1). Eighteen patients were using steroid drops at the time of presentation. The duration of symptoms varied from 1 day to 40 days before presentation.

Infiltrate sizes at presentation were divided into three groups (<2, 2–5 and >5 mm). The infiltrate size was <2 mm in 11 (12%) eyes, 2–5 mm in 50 (53%) eyes and >5 mm in 34 (36%) eyes. Hypopyon was present in 44 (46%) eyes at the initial presentation.

Seventeen eyes had polymicrobial infection. These included *Staphylococcus aureus* (n = 4), *S epidermidis* (n = 3), *Corynebacterium* sp (n = 5), *Streptococcus pneumoniae* (n = 1),

Table 1 Risk factors for *Moraxella* keratitis

	n (%)
Ocular risk factors	
Penetrating keratoplasty	15 (15.7)
Herpes simplex keratitis	15 (15.7)
Glaucoma	12 (12.6)
Blepharitis	12 (12.6)
Dry eye	8 (8.4)
Eye-lid margin disorder	8 (8.4)
Lagophthalmos	7 (7.3)
Trauma	3 (3.1)
Bullous keratopathy	5 (5.2)
Contact lens wear	2 (2.1)
Thyroid eye disease	2 (2.1)
Blind eye	3 (3.1)
Others	18 (18.9)
Systemic risk factors	
Diabetes mellitus	7 (7.3)
Rheumatoid arthritis	5 (5.2)
Leprosy	1 (1.0)

Table 3 Management procedures for *Moraxella* keratitis

	n (%)
Medical management	
Ciprofloxacin	50 (52.6)
Ofloxacin	25 (26.3)
Moxifloxacin	7 (7.3)
Fortified tobramycin and cefazolin	6 (6.3)
Fortified tobramycin alone	2 (2.1)
Fortified cephazolin alone	2 (2.1)
Tobramycin	2 (2.1)
Neosporin	1 (1.0)
Surgical management	
Botulinum toxin injection	17 (17.8)
Tarsorraphy	12 (12.6)
Penetrating keratoplasty	8 (8.4)
Glue and bandage contact lens	4 (4.2)
Conjunctival flap	5 (5.2)
Enucleation	3 (3.1)
Others	7 (7.3)

coagulase-negative *staphylococcus* (n = 3) and *Serratia liquifaciens* (n = 1). In 11 patients, polymerase chain reaction was positive for herpes simplex virus in addition to *Moraxella* infection during the presentation. None of the isolates were resistant to ciprofloxacin. Cefazolin resistance was seen only in two isolates (table 2).

Forty two eyes required an adjunctive procedure (table 3). Topical ciprofloxacin was used in 50 eyes and ofloxacin in 25 eyes. Topical moxifloxacin and a combination of fortified tobramycin and cefazolin was used in the remaining eyes. The time taken for complete closure of the epithelial defect ranged from 4 to 102 days. The mean healing time was 35 days.

Final visual acuity was available in 70 eyes. Among them, 29 eyes had best-corrected visual acuity better than or equal to 6/18. Vision was counting finger or less in 25 (26%) eyes. The eyes that had undergone penetrating keratoplasty for corneal perforation had a poor visual outcome; four (50%) eyes had a final visual acuity of counting finger or less.

DISCUSSION

In the late 19th century, Morax[9] and Axenfeld[10] described *M lacunata* as the cause of subacute conjunctivitis or angular blepharoconjunctivitis. Later, Petit[11] found *M liquifaciens* as an important cause of hypopyon corneal ulcer.

Moraxella was originally discovered as an ocular pathogen. Its reservoir is the mucous membranes of the mouth, upper respiratory tract and genitourinary tract of humans. It has also been isolated from the skin. The corneal infection characteristically has an indolent paracentral or peripheral ulcer that usually is oval in shape and localised, with an undermined necrotising edge. The ulcers are often painless, usually associated with a hypopyon and occasionally a hyphema. In our series half had a hypopyon and none had a hyphema. The ulcer may progress deep into the stroma over days or weeks, and untreated ulcers may perforate. The organism persists in the depth of the crater.

Table 2 Result of in vitro antimicrobial susceptibility

	No of isolates		
Antibiotic	Sensitive	Resistant	Sensitivity test not done
Cefazolin	58	2	35
Ciprofloxacin	87	0	8
Ofloxacin	83	0	12
Chloramphenicol	94	0	1
Neomycin	41	0	54
Tobramycin	46	0	49

Moraxella can cause corneal infection in immunocompromised hosts, particularly in patients who are alcoholic, diabetic or debilitated. Pyridoxine deficiencies in patients who are nutritionally debilitated may have a role in initiating surface infection.[12] Many patients have a history of trauma. Marioneaux *et al*[13] have reported alcohol misuse in 50% of their patients. However, it can occur in patients who are not alcoholic or in those without any history of trauma. Cobo *et al*[14] have reported *Moraxella* keratitis in a group of healthy people. They found chronic epithelial abnormality in seven of eight patients before the onset of keratitis. Although large proportions of our patients belong to the elderly population, we had only one patient who had history of alcohol misuse.

In contrast with systemic predisposing risk factors, ocular predisposing factors were present in a large number of eyes (84.2%) in our series. Most of the predisposing ocular risk factors include either the ocular surface or the eyelid. Multiple predisposing factors were present in 22 eyes. Garg *et al*[15] have found a predisposing systemic or ocular condition in 78% of eyes. Three of our patients had a further episode of *Moraxella* infection after an interval of 7 months, 8 months and 9 years, respectively. Of the three patients, one patient had undergone a corneal graft for herpetic corneal perforation. The other two patients also had a history of chronic herpes simplex keratitis. Cobo *et al*[14] were of the opinion that more corneal debilitation could provide an abnormal epithelial surface to which organisms could adhere more easily. Similar changes occur in the macerated lid-skin epithelium in angular blepharitis.

In our series, half the eyes required adjunctive procedures in addition to intensive medical treatment. Botulinum toxin injection was the most common procedure because of persistent epithelial defect, which was a major problem encountered in our series. *Moraxella* ulcers may be very slow to heal; it can take weeks before the epithelium is intact. In our series, healing time varied from 4 to 102 days. Eight eyes underwent penetrating keratoplasty and three blind eyes were enucleated. All cases in a series by Stern[6] and 50% of cases in a series by Marioneaux *et al*[13] required surgical intervention.

Various antibiotics have been recommended for treatment of *Moraxella* keratitis in the past. Although fluroquinolone eye

drops are used routinely as the preferred treatment for microbial keratitis in our centre, the combination of fortified cefazolin and tobramycin was used in few patients, as some of them were included in a randomised clinical treatment trial of antibiotics for bacterial keratitis. Garg *et al*[15] have noted resistance to cephazolin in 45% of isolates. In our series two isolates were resistant to cephazolin.

Our experience suggests that ocular predisposing factors play an important role in causation of *Moraxella* keratitis, which can lead to serious corneal infection and may need surgical intervention. Various modalities of management for delayed healing may be needed to prevent persistent epithelial defect.

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REFERENCES

1 **McLeod SD**, LaBree LD, Tayyanipur R, *et al*. The importance of initial management in the treatment of severe infectious corneal ulcers. *Ophthalmology* 1995;**102**:1943–8.
2 **Benson WH**, Lanier JD. Current diagnosis and treatment of corneal ulcers. *Curr Opin Ophthalmol* 1998;**9**:45–9.
3 **Musch DC**, Sugar A, Meyer RF. Demographic and predisposing factors in corneal ulceration. *Arch Ophthalmol* 1983;**101**:1545–8.
4 **Baum J**, Fedukowicz HB, Jordan A. A survey of Moraxella corneal ulcers in a derelict population. *Am J Ophthalmol* 1980;**90**:476–80.
5 **Schwartz B**, Harrison LH, Motter JS, *et al*. Investigation of an outbreak of Moraxella conjunctivitis at a Navajo boarding school. *Am J Ophthalmol* 1989;**107**:341–7.
6 **Stern GA**. Moraxella corneal ulcers: poor response to medical treatment. *Ann Ophthalmol* 1982;**14**:295–8.
7 **Frank SK**, Gerber JD. Hydrolytic enzymes of Moraxella bovis. *J Clin Microbiol* 1981;**13**:269–71.
8 **Davis RH**, Palazzolo AM. Evidence for the existence of endotoxic activity in three strains of Moraxella. *Can J Microbiol* 1975;**21**:668–74.
9 **Morax V**. Note sur un diplobacille pathogene pour la conjunctivite. *Ann Inst Pasteur* 1896;**10**:337–45.
10 **Axenfeld T**. Uber die chronische Diplobacille conjunctivitis. *Zentralbl Bakt* 1897;**21**:1–9.
11 **Petit P**. Sur une forme particuliere d'infection corneene a type serpigneux. *Ann Ocul* 1899;**140**:166–84.
12 **Van Bijesterveld OP**. Host parasite relationship and taxonomic position of moraxella and morphologically related organisms. *Am J Ophthalmol* 1973;**76**:545–54.
13 **Marioneaux SJ**, Cohen EJ, Arensten JJ, *et al*. Moraxella keratitis. *Cornea* 1991;**10**:21–4.
14 **Cobo LM**, Coster DJ, Peacock I. Moraxella keratitis in a non-alcoholic population. *Br J Ophthalmol* 1981;**65**:683–6.
15 **Garg P**, Mathur U, Athmanathan S, *et al*. Treatment outcome of Moraxella keratitis: our experience with 18 cases—a retrospective review. *Cornea* 1999;**18**:176–81.