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Clinical features, antibiotic susceptibility profiles, and outcomes of infectious keratitis caused by *Achromobacter xylosoxidans*

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Abstract

Purpose—Reports on *Achromobacter xylosoxidans* ocular infections are increasing, drawing attention to its emerging role in infectious keratitis. The purpose of this study is to report the clinical features, antibiotic sensitivities, and visual outcomes of infectious keratitis secondary to *A. xylosoxidans*.

Methods—A microbiology database and clinical chart review was performed in all patients diagnosed with *A. xylosoxidans* keratitis at the Bascom Palmer Eye Institute between the years 1987 and 2014. Initial presentation, antimicrobial susceptibilities, minimum inhibitory concentrations (MIC), treatment course and outcomes were recorded.

Results—Twenty eight patients were identified. The main risk factors were corneal graft (n=8, 28.6%) and contact lens wear (n=8, 28.6%). On presentation, visual acuity (VA) was 20/100 or worse in 20 (71.2%) patients. Hypopyon was present in 7 (25.0%) patients. In most cases, topical fluoroquinolones or tobramycin were the initial treatment, often accompanied by vancomycin. High susceptibility rates were found for piperacillin (100%, MIC₉₀=8) and ticarcillin (100%, MIC₉₀=16). Low susceptibility rates were documented for ciprofloxacin (46.7%, MIC₉₀=8), tobramycin (26.7%, MIC₉₀=16) and gentamicin (20%, MIC₉₀=16). One (3.6%) patient suffered endophthalmitis. Six (21.4%) patients underwent therapeutic penetrating keratoplasty and 2 (7.1%) patients had conjunctival flap surgery. VA at final follow up was 20/100 or worse in 16 (57.1%) patients.

Conclusion—Infectious keratitis caused by *A. Xylosoxidans* is associated with poor visual outcomes. Fluoroquinolones and aminoglycosides are not appropriate treatments for these ocular infections. Further studies are needed in order to define the clinical application of compound piperacillin and ticarcillin eye drops.

Keywords

infectious keratitis; *achromobacter xylosoxidans*; corneal transplantation; contact lens; topical corticosteroids

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Introduction

Approximately 1 million outpatient clinic visits for keratitis occur annually in the US, of which 76.5% are presumably infectious.¹ Infectious keratitis can result in endophthalmitis, corneal scarring and permanent vision loss. Gram positive bacteria followed by gram negative bacteria are the most common pathogens.^{2,3} In South Florida, *Pseudomonas aeruginosa* and *Serratia marcescens* were most commonly isolated in contact lens-associated keratitis.²

Achromobacter xylosoxidans is an aerobic, non-fermentative, gram-negative rod that is closely related to the *Pseudomonas* species and has been described in the general medical literature as an infection-causing pathogen in immunocompromised patients.⁴⁻⁵ In the past, this organism has rarely been associated with keratitis and endophthalmitis. Nevertheless, reports on ocular infections are increasing drawing attention to the emerging role of *A. xylosoxidans* in infectious keratitis.⁶⁻⁸ Reddy et al. described the largest series of *A. xylosoxidans*-associated ocular diseases with 8 cases of keratitis and 2 cases of postoperative endophthalmitis.⁶ They reported that the main risk factor for *A. xylosoxidans*-associated keratitis was a previous ocular surgery; none of their patients were contact lens wearers.⁶ In a report of corneal ulcers associated with therapeutic contact lens wear, *A. xylosoxidans* was isolated in less than 4% of cases.⁹ The literature is sparse regarding antibiotic susceptibility profiles in *A. xylosoxidans* ocular infections. This pathogen has been reported to be resistant to most aminoglycosides, ampicillin, aztreonam, and most cephalosporins.^{7, 10} Few reports demonstrate *A. xylosoxidans* susceptibility to piperacillin, ceftazidime, imipenem, and trimethoprim-sulfamethoxazole.¹¹⁻¹² As it is a gram-negative rod it is presumably susceptible to fluoroquinolones.

Identifying the risk factors and the susceptibility patterns for *A. xylosoxidans* is important as prompt treatment can help prevent complications such as corneal scar, endophthalmitis and the need for therapeutic corneal transplantation.¹¹⁻¹² The purpose of this study is to report the clinical settings, antimicrobial susceptibilities, and outcomes of culture-proven *A. xylosoxidans* infectious keratitis.

Materials and methods

The Ocular Microbiology Laboratory Database at the Bascom Palmer Eye Institute was searched to identify patients with positive corneal cultures for *A. xylosoxidans* between the years 1987–2014. Cultures were obtained following corneal scrapings submitted to rule out microbial keratitis. Isolates were identified using standard microbiologic procedures by experienced laboratory personnel. A positive culture was considered when there was growth of more than 50 colonies on 1 solid media, 10 to 50 colonies on 2 or more media, or 1 to 10 colonies on 1 solid media and positivity on smears/Thioglycollate broth. All growth had to be on “C” streaks, thus any growth not on “C” streaks was considered a contaminant. Organisms were classified according to usual laboratory criteria. The Ocular Microbiology Laboratory Database was also searched for susceptibility profiles. E-strips (bioMérieux, Inc. Durham, NC) were used to determine the *in-vitro* minimum inhibitory concentration for 90% of isolates (MIC₉₀, in micrograms per milliliter) for each isolate. After analyzing the

microbiology records, the corresponding medical records of these patients were reviewed for demographics, predisposing factors, clinical presentation, presenting and final visual acuities (VA), as well as treatment course and outcomes.

This study was approved by the Institutional Review Board of the University of Miami Miller School of Medicine and complied with the principles outlined in the Declaration of Helsinki.

Results

The study included 28 patients, all of whom demonstrated unilateral keratitis. Twelve patients presented through the years 1987–2000 and 16 patients presented on 2001–2014.

PATIENT DEMOGRAPHICS AND RISK FACTORS

Female patients accounted for 16 (57.1%) of the total population, and right eyes accounted for 16 (57.1%) of the cases. The mean age at presentation was 60.4 ± 22.2 years (range 15–95). Among the predisposing factors, 8 (28.6%) patients had previous penetrating keratoplasty (PK) for which all except one were using topical corticosteroids. Eight (28.6%) patients were contact lens wearers, 3 (10.7%) presented with ocular trauma, 2 cases (7.1%) were secondary to HSV, 2 (7.1%) had a keratoepithelial defect (KED) as a predisposing factor, 2 (7.1%) had exposure keratopathy, 1 (3.6%) was on corticosteroid treatment due to filamentary keratitis, 1 (3.6%) had a neurotrophic corneal ulcer and in 1 (3.6%) patient the site of the infection was adjacent to a bleb.

MICROBIOLOGY AND ANTIMICROBIAL SUSCEPTIBILITIES

The antibiotic susceptibilities and MIC₉₀ values are presented in Table 1. Only 46.7% of the isolates were susceptible to ciprofloxacin. Susceptibility for ceftazidime was 70.0%. *A. xylosoxidans* is intrinsically resistant to aminoglycosides which is why high resistance rates and high MIC₉₀ values (suggesting resistance) were documented; only 26.7% and 20.0% of the isolates were sensitive to tobramycin and gentamycin, respectively. All (100%) of the isolates were susceptible to piperacillin and ticarcillin. MIC₉₀ values did not change over the study period, so there has been no change in the frequency of *A. xylosoxidans* resistance during this time. Concomitant pathogens were identified in 10 (35.7%) patients: 3 had *Serratia marcescens*, 2 had *P. aeruginosa*, 2 had *Staphylococcus aureus* and 3 showed a mix growth patterns of: *P. aeruginosa* and *Providencia rettgeri*; *S. aureus* and *Streptococcus mitis*; and *Acinetobacter woffii* and *Stenotrophomonas maltophilia* (Table 2).

CLINICAL PRESENTATION AND TREATMENT

Data for each patient is summarized in Table 2. Mean time from symptoms to presentation in the clinic was 13.3 ± 13.0 days (range: 1 to 42 days). Mean visual acuity (VA) on presentation was $20/730 \pm 20/210$ (range: 20/20 to no light perception). In 20 (71.4%) patients, VA was 20/100 or worse. Corneal infiltrate was central or paracentral in 23 (82.1%) patients at presentation and hypopyon was present in 7 (25.0%) patients. Fluoroquinolones were the initial treatment of choice, often accompanied by fortified vancomycin. Fortified tobramycin and polymyxin B/trimethoprim were other common treatments.

CLINICAL OUTCOMES

Clinical outcomes for each patient are shown in Table 2. The mean time from the initial treatment until resolution and completion of treatment was 7.7 ± 5.5 weeks (range: 0.3 to 20 weeks). Mean VA at the last follow up visit was $20/600 \pm 20/280$ (range: 20/20 to no light perception) ($p=0.38$, when compared to VA at presentation). VA was 20/70 or better for 10 (35.7%) patients and 20/100 or worse for 16 (57.1%) patients. In 2 patients, VA at last follow up was not available. Intraocular pressure did not change significantly during follow up (13.5 ± 6.5 mmHg in presentation versus 14.9 ± 5.4 mmHg on last examination, $p=0.44$). In 16 (57.1%) patients the corneal ulcer resolved with varying degrees of stromal scarring and haze. Eight (28.6%) patients had a non-healing corneal ulcer. Six (21.4%) of them had a therapeutic PK and in 2 patients a conjunctival flap applied. One patient underwent 2 therapeutic PKs but the infection recurred and the eye was ultimately enucleated. In 1 patient an amniotic membrane was transplanted. Six (21.4%) patients were lost to follow-up before resolution of the infection and completion of treatment, so outcomes could not be fully assessed. The mean overall follow up time was 39.5 ± 56.0 weeks (range 0.3 to 240 weeks).

Discussion

In the current study we found that infectious keratitis resulting from *A. xylosoxidans* has poor VA outcomes with a significant number of patients requiring therapeutic PK or conjunctival flap. Important risk factors for *A. xylosoxidans* keratitis include corneal graft, contact lens wear and corticosteroid treatment. While a few case reports and small case series were published in the past,^{6, 11–17} to our knowledge this study represents the largest case series and review of culture-positive infectious keratitis caused by *A. xylosoxidans*. It was previously reported that the most common predisposing factor for *A. xylosoxidans* corneal ulcers is a prior PK.^{14, 18} Reddy et al. reported this factor to be present in 6 out of 8 patients with infectious keratitis.⁶ In a recent microbiology study, cases and lenses of patients with mild keratitis or corneal ulcer were tested using culture-independent methods. *A. xylosoxidans* was a predominant bacteria identified, pointing out its potential emerging role in infectious keratitis.⁸ Other studies have described immunosuppression therapy including topical corticosteroids as being a common predisposing factor.^{6, 12, 19} In the current study, prior PK and topical corticosteroids were found to be important risk factors. Additionally, the importance of contact lens wear in this setting was documented, as nearly one third of our patients were contact lens wearers.

The clinical diagnosis of microbial keratitis often relies on the history and the morphologic features of corneal inflammation.²⁰ On a large survey performed among ophthalmologists, most reported fourth generation fluoroquinolones as their first treatment of choice for presumed bacterial keratitis.²¹ Forster recommended fourth generation fluoroquinolones for milder forms of bacterial keratitis and fortified aminoglycosides as the empirical treatment for more aggressive keratitis.²² Park et al. suggested that ceftazidime and amikacin may best serve as first line treatment for suspected bacterial keratitis.⁷ In our institute, the treatment strategy was determined by the individual ophthalmologist, and no standardized protocol was followed. We found that fluoroquinolones followed by fortified aminoglycosides were the most common initial antibiotic regimen prior to microbial identification and

susceptibility results. However the *A. xylosoxidans* isolates were highly resistant to ciprofloxacin, gentamicin and tobramycin with high MIC₉₀ values of 8, 16 and 16 mcg/ml, respectively. Resistance of *A. xylosoxidans* to fluoroquinolones in ocular infections was previously reported.⁶ While others reported a 90% sensitivity rate, we found that only 70% of the isolates were susceptible to ceftazidime.⁶ All (100%) of the tested isolates were sensitive to the beta-lactams piperacillin and ticarcillin with MIC₉₀ values of 8 and 16 mcg/ml, which are in the limits of susceptibility. This study is the first to report the MIC₉₀ values for *A. xylosoxidans* keratitis.

Although both *A. xylosoxidans* and *P. aeruginosa* are closely related genotypically and microbiologically, their clinical presentation differs. For example, in cystic fibrosis patients, *A. xylosoxidans* pneumonia is a smoldering disease when compared to *P. aeruginosa* that elicits more rapid lung damage.^{23, 24} In our study, infection with *A. xylosoxidans* was subacute with existence of symptoms for an average of 2 weeks prior to presentation, mainly with mild pain and discomfort. This is as opposed to *P. aeruginosa* keratitis where the clinical course is rapid and symptoms have been reported to appear at an average of 4 days before the patient presents.²⁵ The poor VA outcomes and necessity for therapeutic corneal transplantation or conjunctival flap in 6 patients reported in the current study can be partially explained by the fact that corneal infection by *A. xylosoxidans* is insidious in onset, resulting in initially subclinical symptomatology, leading patients to seek medical attention relatively late in the course of infection.⁸ In addition, the initial treatment usually included fluoroquinolones or aminoglycosides which was, in many instances, ineffective. Finally, many patients had a history of a compromised pre-infection ocular surface, resulting in lower initial visual acuities.

Nine patients were treated with fluoroquinolones and/or aminoglycosides. Only in 3 of them (patients no. 12, 17, 25; Table 2), the infection resolved without significant scarring and without the need for surgical intervention such as therapeutic keratoplasty. In 2 of these patients concomitant pathogens were identified. It is possible that *A. xylosoxidans* was not the primary pathogen in these cases which may explain the fact that the keratitis responded well to the fluoroquinolone treatment. In the third patient (no. 17; Table 2), the infection was mild with good presenting and final visual acuities. Topical treatment in this case was short course fourth generation fluoroquinolone. Susceptibility testing of a fourth generation fluoroquinolone was not done in our study. Although ciprofloxacin is surrogate for fluoroquinolones, we cannot rule out that in *A. xylosoxidans* keratitis fourth generation fluoroquinolones has a better therapeutic effect than the second generation fluoroquinolones.

The limitations of our study include its retrospective design and the use of positive corneal scraping cultures as an inclusion criteria for the study, which might have excluded cases with false-negative culture results. Since our institute is a tertiary referral center, few patients continued follow up with their local ophthalmologist, so their full clinical course was not available for review. Despite these limitations, this study provides important data from a relatively large case-series regarding the clinical characteristics, antimicrobial susceptibility patterns and MIC₉₀ data for *A. xylosoxidans* infectious keratitis. In conclusion, *A. xylosoxidans* infection should be considered in patients with slowly progressive gram-negative keratitis and smoldering symptoms who are known to be contact lens wearers, have

a corneal graft or use topical corticosteroids. For many ophthalmologists, fluoroquinolones and aminoglycosides are the first empirical treatment of choice for presumed bacterial keratitis. It is important to know that these antimicrobials are not an appropriate treatment for *A. xylosoxidans* keratitis. Instead, compound piperacillin or ticarcillin eye drops should be considered. However as the topical regimen of these antibiotics was not applied, further studies are needed in order to define their clinical application.

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Table 1

Antimicrobial Susceptibility and Minimum Inhibitory Concentrations for *Achromobacter xylosoxidans* Keratitis Isolates

Antibiotic	Samples tested	Sensitive (%)	MIC ₉₀ mcg/ml (range)
Ampicillin	12	1 (8.3)	32 (4–32)
Cefazolin	12	0 (0)	64 (32–64)
Ceftazidime	10	7 (70.0)	16 (2–16)
Ciprofloxacin	15	7 (46.7)	8 (0.5–32)
Gentamicin	15	3 (20.0)	16 (2–16)
Imipenem	13	9 (69.2)	8 (4–16)
Piperacillin	10	10 (100.0)	8 (4–8)
Ticarcillin	12	12 (100.0)	16 (16–16)
Tobramycin	15	4 (26.7)	16 (4–16)
Trimethoprim/sulfamethoxazole	14	11 (78.6)	10 (0.032–320)

MIC₉₀= Minimum Inhibitory Concentration for 90% of Isolates

Table 2

Visual Acuity, Treatment Strategies, Follow Up and Outcomes in Patients with *Achromobacter xylosoxidans* Keratitis

N	Visual acuity at presentation	Concomitant pathogen	Treatment	Visual acuity at last follow up examination	Antibiotic treatment duration (weeks)	Outcomes
1	20/70		Moxifloxacin, polymyxin B/trimethoprim	20/25	2	Resolved
2	2/200		Gatifloxacin, polymyxin B/trimethoprim, erythromycin	20/300	8	Therapeutic PK
3	20/400		Fortified vancomycin, fortified tobramycin	3/200	7	Lost to follow up
4	20/40		Moxifloxacin, fortified vancomycin	20/20	8	Resolved
5	NLP		Gatifloxacin, erythromycin, polymyxin B	NLP	12	Resolved; blind eye secondary to glaucoma
6	LP		Gatifloxacin	LP	13	Therapeutic PK
7	HM		Polymyxin B/trimethoprim, fortified ceftazidime	HM	12	Therapeutic PK
8	HM		Ofloxacin	LP	4	Anniotic membrane transplant; resolved with significant scarred corneal graft
9	20/200	Serratia Marcescens	Fortified tobramycin, fortified cefazolin, ceftazidime	HM	6	Resolved with significant scar
10	20/100	Serratia Marcescens	Ofloxacin, polymyxin B/trimethoprim	20/70	1.5	Lost to follow up
11	HM	Staphylococcus aureus	Ciprofloxacin, fortified tobramycin, polymyxin B/trimethoprim	HM	16	Two therapeutic PK; enucleation
12	20/20	Serratia Marcescens	Ofloxacin	20/20	4	Resolved
13	20/800		Ofloxacin, fortified vancomycin, fortified tobramycin, fortified ceftazidime, polymyxin B/trimethoprim, imipenem	20/400	20	Therapeutic PK
14	LP	Pseudomonas aeruginosa	Ofloxacin	LP	8	Therapeutic PK
15	20/50		Fortified tobramycin, ceftazidime	20/25	8	Resolved
16	20/200		Levofloxacin, fortified tobramycin, moxifloxacin	CF	12	Lost to follow up
17	20/40		Moxifloxacin	20/20	1.1	Resolved
18	20/40		Ofloxacin, fortified tobramycin, fortified cefazolin	20/20	0.3	Lost to follow up
19	CF		Fortified cefazolin, fortified tobramycin	N/A	N/A	Lost to follow up
20	20/20	Staphylococcus aureus	Fortified cefazolin, gentamicin	N/A	N/A	Lost to follow up
21	HM		Fortified vancomycin, moxifloxacin	20/25	2	Resolved
22	20/200		Polymyxin B/trimethoprim, fortified cefazolin	20/200	20	Conjunctival flap performed
23	CF	Pseudomonas aeruginosa	Moxifloxacin, fortified tobramycin	HM	6	Conjunctival flap performed

N	Visual acuity at presentation	Concomitant pathogen	Treatment	Visual acuity at last follow up examination	Antibiotic treatment duration (weeks)	Outcomes
24	LP	<i>Pseudomonas aeruginosa</i> , <i>Providencia rettgeri</i>	Fortified tobramycin	LP	6	Resolved with significant scarred corneal graft
25	20/25	<i>Acinetobacter lwoffii</i> , <i>Stenotrophomonas maltophilia</i>	Ofloxacin	20/30	0.5	Resolved
26	6/400		Fortified vancomycin, fortified tobramycin, moxifloxacin	20/60	12	Resolved
27	CF		Vancomycin, fortified tobramycin, fortified ceftazidime	20/400	3	Compromised with blebitis and endophthalmitis
28	6/400	<i>Staphylococcus aureus</i> , <i>Streptococcus mitis</i>	Moxifloxacin, fortified tobramycin, polymyxin B/trimethoprim	20/200	8	Resolved with significant scar

NLP=No light perception; LP=Light perception; HM=Hand motion; CF=Counting fingers; PK=Penetrating keratoplasty