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Clinical Features, Antibiotic Susceptibility Profile, and Outcomes of Infectious Keratitis Caused by *Stenotrophomonas maltophilia*

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Abstract

Purpose—*Stenotrophomonas maltophilia*, an uncommon cause of infectious keratitis, is difficult to treat due to its resistance to multiple antibiotics. The purpose of this study is to describe the clinical features, antibiotic susceptibility profile, and outcomes of *S. maltophilia* keratitis.

Methods—A retrospective review of records from 1987–2014 identified 26 eyes of 26 patients who were treated at the Bascom Palmer Eye Institute for a *S. maltophilia* corneal ulcer. Clinical data was analyzed as to predisposing factors, clinical presentation, antibiotic susceptibility, treatment selection and clinical outcomes.

Results—Median age at presentation was 64.9 years (range, 16.0 – 97.7). Twelve patients were using topical corticosteroids, 8 patients had a history of penetrating keratoplasty and 9 were contact lens wearers. All patients received topical antibiotics, two required a therapeutic penetrating keratoplasty, and one was enucleated. At presentation, 57.7% (15/26) of the patients had visual acuity 20/400 or worse. At the final visit only 30.4% (7/23) of the patients had visual acuity worse than 20/400, while 65.2% (15/23) of the patients were at 20/100 or better. Almost all isolates (25/26, 96.2%) were susceptible to fluoroquinolones and 77.3% (17/22) of them to polymyxin B/trimethoprim. Only 33.3% (5/15) of the tested isolates were susceptible to aminoglycosides and 58.3% (7/12) to cephalosporins.

Conclusion—Infectious keratitis due to *S. maltophilia* presents a treatment challenge due to its resistance to aminoglycosides and cephalosporins, which are typically used for empiric broad-spectrum gram-negative coverage as fortified solutions. Fluoroquinolones and polymyxin B/trimethoprim should be considered instead in cases of *S. maltophilia* infection.

Keywords

antimicrobial susceptibility; infectious keratitis; *Stenotrophomonas maltophilia*; *Pseudomonas aeruginosa*; fluoroquinolones; aminoglycosides

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Introduction

Stenotrophomonas maltophilia is an aerobic, motile gram-negative bacillus that is ubiquitous in nature.¹ It has been isolated from plants, animals, soil and even nutrient-poor aqueous environments.² In recent years it has emerged as an opportunistic pathogen due to its intrinsic resistance to extended-spectrum cephalosporins, carbapenems and aminoglycosides.³ The inherent expression of at least two inducible beta-lactamases is responsible for *S. maltophilia*'s resistance to beta-lactam agents and its high lethality rate among hospitalized patients.⁴

S. maltophilia primarily causes infection in immunocompromised patients such as meningitis, endocarditis, upper respiratory tract infections, urinary tract infections, and skin/soft tissue infections.⁵ It is an uncommon keratitis pathogen with the total number of cases reported in the literature less than 40.^{6–16} The purpose of this study is to report the clinical characteristics, antibiotic susceptibilities, treatment course and outcomes of culture-proven *S. maltophilia* infectious keratitis.

Methods

A search of the database of the Ocular Microbiology Laboratory at the Bascom Palmer Eye Institute was performed for patients with corneal cultures positive for *S. maltophilia* between the years 1987 and 2016. Patient records were retrospectively reviewed for demographic information, medical and ocular history, systemic and local predisposing factors, presenting signs and symptoms, presenting and final visual acuity, treatment course, and outcomes. Corneal scrapings were routinely cultured on chocolate, 5% sheep blood, and Sabouraud with gentamicin agars and thioglycolate broth. Plates were incubated in CO₂ (chocolate, blood, thioglycolate) and or non-CO₂ (sabouraud) at 35°C for up to 7 days. Isolates were identified using the Vitek2 Gram Negative Identification (GNI) cards (BioMerieux, Durham, NC), commercial kits (API NE -nonfermenter Enterobacteriaceae- BioMerieux, Durham, NC; Remel NF plus -nonfermenter- San Diego, CA) and or conventional microbiologic procedures (aerobic/anaerobic growth, oxidase, motility, nitrate) by experienced laboratory personnel. A culture was considered positive if there was growth of more than 50 colonies on one solid media (3+, heavy growth), 10–50 colonies on 2 or more media (2+, moderate growth), or 1–10 colonies on one solid media and positivity on smears and/or thioglycolate broth (1+, light growth). Antimicrobial susceptibility was evaluated using a combination of disk diffusion, E-tests and or Vitek 2 Gram Negative susceptibility cards. Results were interpreted according to the most current Clinical Laboratory Standards Institute (CLSI) guidelines.

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

Twenty-six patients (58% female) with unilateral *S. maltophilia* keratitis were identified during the study period. The median follow-up time was 4.0 months (range, 1–120 months).

Patient Demographics and Risk Factors

Median age at presentation was 64.9 years (range, 16 to 98). A total of 9 ulcers (34.6%) occurred in the right eye and 17 (65.4%) in the left eye. In 25 of the 26 patients a risk factor for infectious keratitis was present and in 13 (50.0%) patients there were multiple causes. Eight (30.8%) patients had a history of penetrating keratoplasty, and 9 patients (34.6%) were contact lens wearers. Three (11.5%) patients had history of a non-healing epithelial defect, two (7.7%) had recent surgery (EDTA chelation and LASIK), two (7.7%) had history of LASIK with evidence of epithelial ingrowth, 1 (3.8%) had a Boston keratoprosthesis and was on topical corticosteroids and 1 (3.8%) had history of trauma and was also HIV positive.

Microbiology and Antibiotic Susceptibilities

Light to moderate growth of isolates was recovered from 80% of patient samples. Isolates were identified using a combination of commercial kits (API NE, Rapid NF), Vitek 2 GNI cards and/or supplementary conventional tests. Identification probabilities were accepted at 90% for Vitek or at least 85% for both commercial kit systems.

The antibiotic susceptibilities of the *S. maltophilia* isolates are summarized in Table 1. In general, isolates were susceptible to the fluoroquinolones, with greater than 90% for both ciprofloxacin and levofloxacin. However, susceptibilities were less than 80% for polymyxin B and trimethoprim/sulfamethoxazole (17/22; 77.3%). Only 35.7 % of the tested isolates were susceptible to gentamicin, 33.3% to tobramycin, and 27.3% to amikacin. Susceptibility of the isolates to the cephalosporins was below 60%.

Concomitant pathogens (14/40, 35.0%) were recovered in 10 (38.5%) patients. (See Table, Supplemental Digital Content 1, which reviews information on concomitant pathogens, treatment course and outcomes for each patient included in this study). Three patients (30.0%) had multiple pathogens while seven (70.0%) had a single co-pathogen. Fifty-seven percent of the copathogens (8/14; 28.5% each) were recovered among contact lens wearers (4 copathogens) and patients with history of penetrating keratoplasty (4 copathogens). One patient was both a contact lens wearer and had history of penetrating keratoplasty. Multiple organisms were recovered from one patient with no identified risk factor. Most attendant pathogens were gram negative (7/14, 50.0%)

Clinical Presentation and Treatment

A summary of each patient's course is included in Supplemental Digital Content 1. Median time of symptom duration prior to presentation was 5 days (range, 1–90 days). At presentation 57.7% (15/26) of the patients had visual acuity 20/400 or worse. In 19 (73%) patients, vision at presentation was 20/100 or worse. Sixteen (61.5%) patients had a central or paracentral infiltrate at presentation, and 5 (19.2%) had hypopyon. The initial empirical treatment was fortified vancomycin and tobramycin in 10 (38.5%) patients and fortified cefazolin and tobramycin in 5 (19.2%) patients. The addition of a fluoroquinolone or polymyxin B/trimethoprim was done for most of these patients once culture results were available (see Supplemental Digital Content 1, Table, which reviews the treatment course for each patient), for those not originally presenting with that treatment (10/15 or 66.7% of patients). Seven eyes (26.9%) did not receive either a fluoroquinolone or polymyxin B/

trimethoprim (cases 1, 3, 4, 12, 15, 16, and 18). For the patients whose isolate was sensitive to the empiric antibiotic therapy (Table 2) (cases 1, 3, 15, 16), no change was made to the treatment regimen. Case 12 was lost to follow-up once empiric treatment was started. Cases 4 and 18 were improving clinically on the antibiotic regimen they were receiving – amikacin and tobramycin respectively– and, thus, no change was made.

Clinical Outcomes

The treatment outcome for each patient is also shown in Supplemental Digital Content 1. The median duration of treatment with antibiotics was 29 days (range, 4 – 105 days). At the final visit only 30.4% (7/23) of the patients had visual acuity worse than 20/400, while 65.2% (15/23) of the patients were at 20/100 or better. For three patients final visual acuity was not available.

In 13 (50.0%) patients the corneal infection resolved with limited stromal scarring and in 5 (19.2%) patients with significant scarring and thinning. One patient (3.8%) with exposure keratopathy required a tarsorrhaphy for the epithelial defect to heal. Two patients (7.7%) required a therapeutic penetrating keratoplasty, one (3.8%) underwent enucleation of a painful blind eye with recurrence of the infection, and one (3.8%) had LASIK flap amputation. Three patients (11.5%) were lost to follow up.

Discussion

S. maltophilia has emerged as a global opportunistic pathogen responsible for multi-drug resistant nosocomial infections in debilitated hosts.¹ The prevalence rates of *S. maltophilia* from systemic sources have increased from 0.8 – 1.4% during 1997–2003 to 1.3–1.68% during 2007–2012.^{17–23} Though the rate of systemic *S. maltophilia* systemic infections has been slowly rising, the rate of *S. maltophilia* keratitis cases has remained stable at our institute for the periods 1989–2001 (6/774, 0.8%) and 2002–2014 (16/2475, 0.65%). The rate for 2015–2016 is 0.8 % (4/503). *S. maltophilia* has also been reported to cause conjunctivitis,²⁴ endophthalmitis,²⁵ infantile dacryocystitis,⁷ and preseptal cellulitis.⁷

This study is the largest case series and review of culture-positive corneal ulcers caused by *S. maltophilia*. Almost half the patients (12/26; 46.2%) in our series were on chronic topical corticosteroids either because they had history of a penetrating keratoplasty (8/12; 66.7%) or Boston keratoprosthesis (1/12; 8.3%) or because of recent ocular surgery (2/12; 16.7%) or recurrent ocular surface inflammation with a chronic non-healing epithelial defect (1/12; 8.3%). In the second largest study to date on *S. maltophilia* keratitis more than 75% of the patients (16/21) were on chronic corticosteroids because of history of corneal graft surgery (9/16; 56.3%), herpes virus keratitis (4/16; 25.0%), exposure keratitis (2/16; 12.5%) and Stevens-Johnson syndrome (1/16; 6.3%). Other than chronic corticosteroid use and ocular surface compromise, we found contact lens wear (9/26; 34.6%) to be an additional predisposing factor for keratitis in our series. Similarly, in the study by Wu et al. 9 out of 21 patients (42.9%) were contact lens wearers. *S. maltophilia*, along with *Achromobacter xylosoxidans* and *Delftia acidovorans*, has emerged as one of the predominant organisms in biofilms isolated from contact lens cases²⁶ and lens care systems,^{27,28} while its resistance to specific multipurpose disinfecting solutions has been well demonstrated *in vitro*.^{28,29}

Sequencing of the *S. maltophilia* K279a genome revealed several genes that can explain the high-level resistance observed to a variety of chemically different antibiotics.³⁰ Intrinsic expression of beta-lactamases, aminoglycoside-modifying enzymes and multidrug-efflux pumps, the presence of a low permeability outer membrane and the capability of this bacterium to acquire additional resistance genes through incorporation of plasmid and transposon DNA render *S. maltophilia* immune to beta-lactam antibiotics, aminoglycosides, quinolones, and disinfectants.^{31,32}

Worldwide surveillance projects and multicenter studies on systemic infections show that the susceptibility of *S. maltophilia* to ceftazidime has decreased globally from 47–75% during 1997–1999 to 30.5–36.8% during 2009–2012.^{17,19,33,34} During the same time period, susceptibility to trimethoprim/sulfamethoxazole remains stable with resistance rates that are generally less than 10%.^{17,19,33–37} Sensitivity of *S. maltophilia* to levofloxacin has decreased from 83.4% during 2003–2008 to 77.3% in 2011.^{17,33} Nonetheless, two studies on moxifloxacin showed low MIC₅₀ (0.5mg/L) and MIC₉₀ (8 and 4 mg/L) values, suggesting that moxifloxacin remains a good therapeutic option.^{38,39}

In our series, only 33.3% of the tested isolates were sensitive to the aminoglycosides and 58.3% to the cephalosporins. This finding is consistent with the inherent resistance of *S. maltophilia* to these classes of antibiotic agents and to the global trend of decreasing susceptibility of *S. maltophilia* to cephalosporins. In contrast, all isolates except for one were sensitive to the fluoroquinolones and 72.2% of them were sensitive to trimethoprim/sulfamethoxazole. The single isolate that showed resistance to the fluoroquinolones was sensitive to Polymyxin B. The respective patient who was initially on Ofloxacin showed improvement once Trimethoprim/Polymyxin B was added to his treatment regimen. Similarly, in the study by Wu et al all 21 *S. maltophilia* isolates were sensitive to the fluoroquinolones and 66.7% of them to trimethoprim/sulfamethoxazole.

All patients received medical therapy as per the culture results and therapy was tapered based on clinical response. In 50% of them (n=13), the infection did not result in significant stromal scarring or thinning. Nine of these 13 patients had received empirical antibiotic therapy prior to the culture results to which *S. maltophilia* was proved to be sensitive to. In 5 patients (19.2%) the infection resolved with topical antibiotics albeit with significant stromal scarring. Four patients (15.4%) required therapeutic surgery, either penetrating keratoplasty (n=2), LASIK flap amputation (n=1) or enucleation (n=1). Similarly, Wu et al. reported that close to one fifth of their patients required therapeutic surgery for resolution of the infection while in the others the *S. maltophilia* keratitis resolved with medical therapy alone.¹⁶

Limitations of this study include its retrospective nature and the small sample size. Moreover, the presence of several polymicrobial cases is a well-known issue in any microbiological study and attests to the opportunistic nature of the organism and its affinity for compromised tissue. Though we cannot be sure that *S. maltophilia* was the main pathogen in each polymicrobial case, all patients responded well to the antibiotics that *S. maltophilia* was shown to be susceptible to *in vitro*. In the second largest on *S. maltophilia* keratitis by Wu et al, there were 12 polymicrobial cases (12/21; 57.1%)¹⁶ and in our series 10 cases (10/26; 38.5%). The issue of colonization versus infection has been raised for

systemic *S. maltophilia* infections as well.³ *In vitro* studies have elucidated the role of *S. maltophilia* as an indirect pathogen in polymicrobial infections. Beta-lactamase leaking from *S. maltophilia* may encourage the growth of other β -lactam susceptible Gram-negative bacteria such as *Serratia marcescens* and *Pseudomonas aeruginosa* even in the presence of high levels of imipenem or ceftazidime.⁴⁰ We, thus, recommend treating *S. maltophilia* in polymicrobial ocular infections in patients with a compromised ocular surface.

Though related genetically to *Pseudomonas aeruginosa*, infectious keratitis due to *S. maltophilia* is slowly progressive with smoldering symptoms. The patients in this study sought medical attention on average 14 days after their first symptoms. In contrast, patients with *P. aeruginosa* keratitis usually present much earlier due to rapid evolution of the disease process.⁴¹ Future studies comparing *S. maltophilia* to a more common ocular pathogen such as *Pseudomonas aeruginosa* in terms of clinical signs and symptoms, antibiotic susceptibility profiles and clinical outcomes, will enable a better understanding of the pathogenicity of this peculiar organism. *S. maltophilia* keratitis should be suspected in any patient with a gram-negative infection in a compromised ocular surface who does not respond to fortified aminoglycoside or cephalosporin antibiotics. Fluoroquinolones and polymyxin/trimethoprim are the treatment of choice for *S. maltophilia* infectious keratitis.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1 Antibiotic Susceptibility of *Stenotrophomonas maltophilia* Isolates *

Antibiotic	Samples Tested	Susceptible (%)
Gentamicin	14	5 (35.7)
Tobramycin	15	5 (33.3)
Amikacin	11	3 (27.3)
Imipenem	5	1 (20.0)
Ciprofloxacin	12	11 (91.7)
Levofloxacin	18	18 (100.0)
Trimethoprim/Sulfamethoxazole	18	13 (72.2)
Polymyxin B	10	8 (80.0)
Ceftazidime/Ceftriaxone	12	7 (58.3)

* Percent susceptible based on a combination of disk diffusion zone sizes (millimeter), Etests (µg/ml) and or Vitek 2 automated breakpoints (µg/ml) and interpretative guidelines in accordance with the Clinical Laboratory Standard Institute (CLSI) guidelines. Low numbers (<30 isolates) may be limited in predicting percent susceptible.

Table 2

Antibiotic Susceptibility of *Stenotrophomonas maltophilia* Isolates

N	Gentamicin	Tobramycin	Amikacin	Imipenem	Ciprofloxacin	Levofloxacin	Sulfamethoxazole	Trimethoprim	Polymyxin B	Ceftazidime/Ceftriaxone
1	S	I	-	-	-	-	-	-	-	-
2	R	R	R	R	S	-	-	-	-	R
3	S	S	S	S	S	-	-	-	-	R
4	R	R	-	-	-	-	S	-	R	-
5	S	S	S	-	S	-	R	-	-	R
6	R	R	R	R	S	-	R	S	S	S
7	R	R	R	R	R	-	R	S	S	R
8	-	-	-	-	-	S	S	-	-	S
9	-	-	-	-	-	S	S	-	-	-
10	R	R	R	-	S	S	S	R	-	-
11	R	R	R	-	S	S	-	S	S	S
12	R	R	R	-	S	S	-	S	S	S
13	R	R	R	R	S	S	-	-	-	-
14	S	S	S	-	I	-	S	S	S	S
15	S	S	-	-	S	S	-	-	S	I
16	R	R	R	-	S	S	-	-	S	I
17	-	-	-	-	-	S	S	-	-	-
18	-	-	-	-	-	S	S	-	-	R
19	-	-	-	-	-	S	S	-	-	-
20	-	-	-	-	-	S	S	-	-	-
21	-	-	-	-	-	S	S	-	-	-
22	-	-	-	-	-	S	S	-	-	-
23	-	-	-	-	-	S	R	-	-	-
24	-	-	-	-	-	S	S	-	-	-
25	-	R	-	-	-	S	S	-	-	-
26	-	-	-	-	-	S	R	S	S	-

S, sensitive; I, intermediate; R, resistant