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CASE REPORT

Contact Lens-Associated *Serratia marcescens* Keratitis: A Case Report

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Abstract: *Serratia marcescens* (*S. marcescens*) keratitis is a rare, sight-threatening infection primarily seen in contact lens users. A 22-year-old female developed sudden ocular symptoms two days after contact lens use. Examination revealed significant conjunctival injection, anterior chamber inflammation, and a corneal abscess with an epithelial defect. Despite initial moxifloxacin treatment, her condition worsened, requiring a switch to fortified vancomycin, ceftriaxone, and voriconazole. Cultures confirmed *S. marcescens*, allowing treatment to be streamlined to fortified ceftriaxone. The infection gradually resolved, with visual acuity returning to 20/20 and minimal residual scarring noted at one month.

Keywords: *Serratia marcescens*, keratitis, contact lens, corneal abscess, microbial keratitis

Introduction

Contact lens-associated microbial keratitis (CLMK) is a severe ocular infection that can lead to significant vision loss if not promptly managed.[1] The incidence of severe CLMK is higher in users of extended-wear lenses, with a rate of 2.52 per 10000 users, compared to 0.62 per 10,000 for daily disposable lens users.[2]

Despite advances in lens hygiene and disinfection solutions, microbial keratitis remains a clinically significant problem, particularly in younger, healthy populations who frequently use contact lenses for refractive correction or cosmetic purposes.[3]

Serratia marcescens (*S. marcescens*), a gram-negative bacillus in the Enterobacteriaceae family, has emerged as an increasingly recognized cause of CLMK. Although traditionally associated with nosocomial infections, its ability to colonize contact lenses, storage cases, and lens care solutions has made it an opportunistic pathogen in community-acquired keratitis. Its pathogenicity is mediated by multiple virulence factors, including fimbrial adhesins, which facilitate epithelial adherence; extracellular proteases such as serralysin, which degrade stromal collagen; and biofilm formation on lens surfaces, which enhances survival in hostile environments and confers resistance to disinfection agents.[4,5]

Antimicrobial resistance is a growing concern with *S. marcescens*, particularly to fluoroquinolones, which are frequently used as first-line empirical therapy in bacterial keratitis. Mechanisms of resistance include the overexpression of efflux pumps, production of beta-lactamases, and alterations in target enzymes such as DNA gyrase.[6]

Previous studies have reported a growing trend in gram-negative organisms as causative agents in contact lens-related keratitis, with *S. marcescens* increasingly identified among them.[7]

This local knowledge gap underscores the importance of reporting individual cases to raise awareness of emerging patterns and guide empiric treatment choices in our setting.

In this case report, we present a previously healthy 22-year-old female who developed rapidly progressive *S. marcescens* keratitis shortly after contact lens use. The case exemplifies the clinical aggressiveness of the organism, the limitations of initial empirical therapy, and the importance of culture-directed management to preserve visual outcomes. It also highlights the need to incorporate regional data and clinical experience into the broader understanding of CLMK pathogenesis and treatment.

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Case Presentation

A 22-year-old female medical student with no significant past ocular, systemic illness, immunodeficiency, or use of immunosuppressive medications presented to the ophthalmology emergency department with a two-day history of ocular redness, purulent discharge, and blurred vision in her left eye, temporally associated with the recent application of soft contact lenses. The patient denied any history of trauma. She endorsed showering sometimes in her lenses, but she denied swimming in contacts, or using tap water for lens hygiene, sleeping a few hours during the day with her CL on her eyes, and she stated that they were monthly disposable lenses. Still, she exceeded the duration to 5 weeks.

On initial examination, the best-corrected visual acuity was measured at 20/32 in the affected eye, compared to 20/25 in the right eye. Slit-lamp biomicroscopy of the left eye revealed pronounced severe conjunctival and limbal injection, a fibrinous reaction with +3 cells within the anterior chamber, and a well-circumscribed, paracentral corneal abscess measuring approximately 1.5 mm × 1.5 mm, accompanied by an overlying epithelial defect, diffuse stromal haze, and positive fluorescein staining.

Given these findings, prompt diagnostic measures were undertaken, including corneal scraping and culture of the implicated contact lens. The patient was initiated on an intensive regimen of moxifloxacin (0.5%) ophthalmic solution administered hourly, alongside strict discontinuation of contact lens use.

The following day, the patient experienced increased ocular pain and further deterioration of vision; subsequent examination confirmed ongoing inflammatory signs with the emergence of a 0.5 mm hypopyon (Figure 1A).

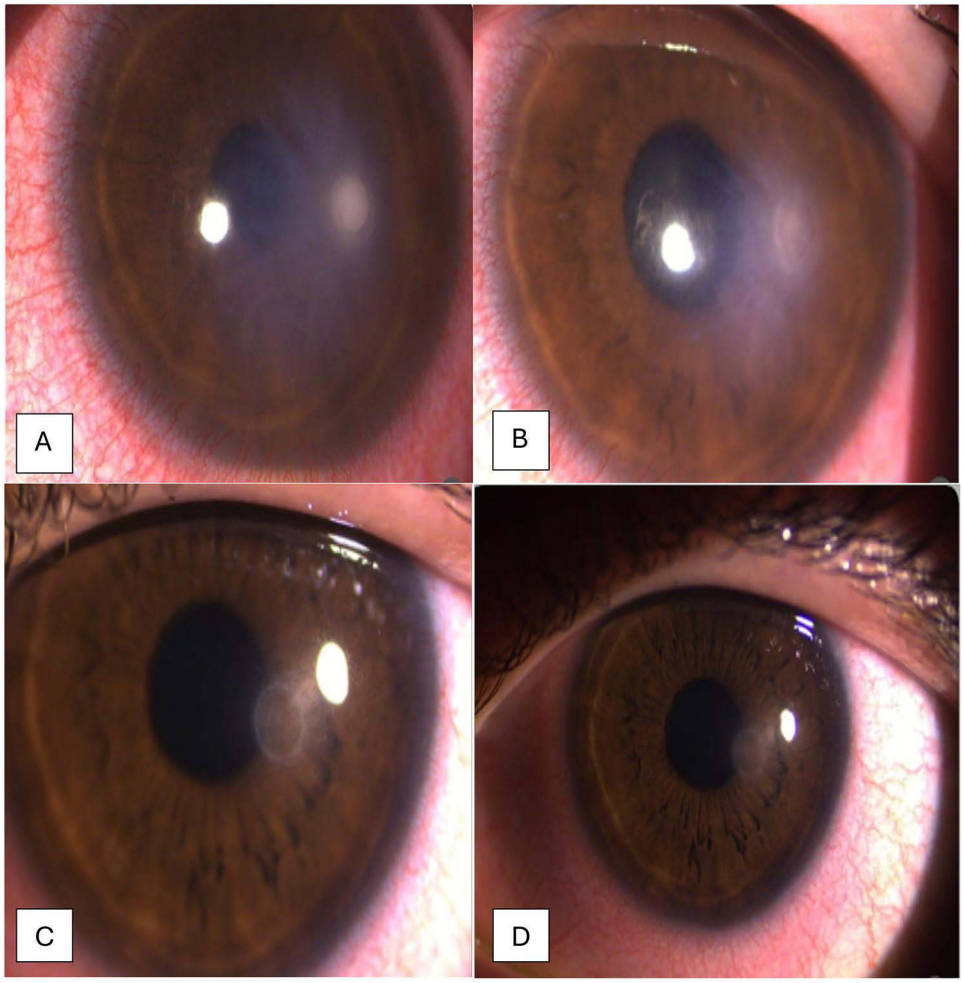

Figure 1 (**A**) On presentation cornea showed circular epithelial defect (1.5mm x1.5 mm) with dense stromal infiltrate surrounded by corneal haze, corneal oedema, and hypopyon (0.5 mm), (**B**) (Day 3) after starting fortified antibiotics, less stromal infiltrate and no hypopyon, (**C** and **D**) (Day 7) resolving infiltrate and haze with a residual circular scar and healed defect.

Consequently, the initial therapeutic approach was augmented to include fortified antibiotic regimens composed of vancomycin (50 mg/mL), ceftriaxone (50 mg/mL), and voriconazole (10 mg/mL) administered hourly.

Forty-eight hours following this intervention, although the visual acuity in the affected eye had declined to 20/200, clinical assessment revealed a slight reduction in the size of the epithelial abscess (now 0.75 mm × 0.75 mm), persistent hypopyon with +2 anterior chamber cells, and moderate conjunctival injection (Figure 1B).

Microbiological cultures from corneal scrapings and the contact lens subsequently yielded heavy growth of *S. marcescens*. Although this organism is known to produce a reddish-purple pigment (prodigiosin) under certain conditions, neither the corneal lesion nor the ocular discharge in this case exhibited any such pigmentation. Based on sensitivity testing demonstrating susceptibility to ceftriaxone, the therapeutic regimen was refined by discontinuing voriconazole and continuing treatment exclusively with fortified ceftriaxone (50 mg/mL).

Over the next week, the corneal ulcer exhibited significant resolution, with only minimal residual stromal infiltration, absence of further fluorescein staining, resolved conjunctival injection, and no cells in the anterior chamber. As the inflammatory process subsided and stromal haze remained as the predominant finding, adjunctive therapy with loteprednol etabonate 0.5% was introduced to mitigate potential scarring (Figure 1C and D).

At one-month follow-up, the patient's visual acuity returned to 20/20, with only minimal paracentral corneal scarring present, indicating a favorable clinical outcome.

Discussion

Extended-wear contact lenses have been associated with a higher risk of infection than daily disposables, a disparity linked to reduced oxygen permeability, prolonged lens use, and suboptimal hygiene practices that favor microbial colonization. The rapid onset of symptoms following contact lens use underscores the potential for severe infection even in a healthy young adult.[3]

In this case, the patient's rapid clinical deterioration despite empiric treatment with moxifloxacin underscores the organism's potential for early aggressive tissue invasion. This progression may be explained, in part, by biofilm formation on the contact lens surface, a well-documented virulence factor of *S. marcescens* that confers protection from both host immune defenses and antibiotic penetration.[8]

S. marcescens is well recognized for its multifaceted virulence, which includes strong adherence capabilities and the formation of biofilms on abiotic surfaces such as contact lenses.[9] *S. marcescens* produces fimbrial adhesins and other surface structures that facilitate adherence, while biofilm formation protects the bacteria from antimicrobial agents and the host's immune response.[10] In addition to these mechanisms, the bacterium secretes several extracellular proteolytic enzymes that degrade the corneal stroma, contributing to tissue invasion and abscess formation. The robust inflammatory response elicited by *S. marcescens*, including the production of proinflammatory cytokines, further exacerbates tissue damage and may lead to the accumulation of inflammatory cells in the anterior chamber (hypopyon), as observed in this patient.[11]

In the clinical management of bacterial keratitis, initial empirical treatment with fourth-generation fluoroquinolones like moxifloxacin may be inadequate due to emerging resistance among causative pathogens.[12]

Although moxifloxacin is commonly used as first-line therapy in bacterial keratitis, the patient's lack of improvement raised suspicion for resistance. In this case, the *S. marcescens* isolate was confirmed to be resistant to moxifloxacin, which directly explains the poor initial therapeutic response. This finding aligns with broader reports of fluoroquinolone resistance among *S. marcescens* isolates, often mediated by overexpression of efflux pumps and mutations in DNA gyrase.[4,13]

Studies have demonstrated that prior use of fluoroquinolones is associated with increased antibiotic resistance in bacterial keratitis cases, potentially leading to suboptimal treatment outcomes. Fluoroquinolones were associated with a 2.01-fold increase in the minimum inhibitory concentration for bacterial isolates, indicating higher resistance level. Therefore, they may not be suitable for initial monotherapy in severe cases.[14] Resistance to fluoroquinolones was observed in 23.1% of gram-positive and 2.8% of gram-negative bacteria in a study from Seville, suggesting that fluoroquinolones may not be suitable for initial monotherapy in severe case.[15]

The increased resistance to fluoroquinolones can lead to treatment failures and prolonged recovery times, as seen in a meta-analysis where fluoroquinolones had a shorter time to cure compared to fortified antibiotics, but similar cure rates.[16]

However, such resistance trends can vary geographically. While studies from Seville and global meta-analyses have identified increasing resistance patterns, the extrapolation of such data may not fully reflect our regional antimicrobial landscape. Unfortunately, local susceptibility data for ocular isolates are limited in our setting, highlighting a key gap in evidence-based empiric treatment selection and the need for more robust regional surveillance.

In CLMK cases where initial therapy fails, escalation to fortified antibiotics such as vancomycin and ceftriaxone is recommended to broaden antimicrobial coverage, especially in severe infections or when a hypopyon is[4] present. Once pathogen identification and susceptibility profiles are available, therapy should be tailored accordingly to enhance efficacy and minimize resistance development.[17]

The adjunctive use of topical corticosteroids in bacterial keratitis remains a topic of debate. While some evidence suggests that corticosteroids may help reduce inflammation, corneal scarring, and neovascularization, their overall effectiveness and safety are not conclusively established.[18] Initiating corticosteroid therapy only after confirming infection control is a cautious approach aimed at mitigating immune-mediated damage without compromising antimicrobial efficacy.[19]

Future research should focus on molecular characterization of *S. marcescens* isolates from contact lens-associated infections. Enhanced understanding of its virulence determinants could inform the development of novel therapeutic agents or contact lens disinfection technologies. These insights would contribute to reducing the incidence and severity of such infections, ultimately improving clinical outcomes for contact lens users.

Conclusion

Prompt recognition and culture-directed management of contact lens-associated microbial keratitis caused by *Serratia marcescens* can lead to excellent visual outcomes. In this case, early intervention, escalation to targeted antimicrobial therapy based on sensitivity results, and the careful introduction of corticosteroids helped control inflammation and minimize stromal scarring. While *S. marcescens* is known to produce a reddish-purple pigment (prodigiosin) under specific environmental conditions, neither the corneal lesion nor the ocular discharge in this patient demonstrated such pigmentation.

Ethics and Consent Statement

Formal institutional review board approval was not required to publish the case details at our institution. However, written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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Disclosure

The authors declare no potential conflicts of interest for this work.

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