Enterobacter cloacae Keratitis in a Patient with Severe Dry Eye: A Rare Case Report and Review of the Literature

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Dear Editor,

Corneal ulcers are vision-threatening emergencies. Bacterial pathogens such as *Staphylococcus*, *Streptococcus,* and *Pseudomonas* are most frequently implicated in microbial keratitis.[1] The integrity of the corneal epithelium is critical in preventing infections, as it acts as a physical and immunological barrier.

Enterobacter cloacae is a Gram-negative rod in the Enterobacteriaceae family, commonly present in the environment and human gut microbiota. Although increasingly linked to nosocomial infections,[2] ocular involvement remains rare. The prevalence of *E. cloacae* keratitis ranges from 0.13% to 15.3%.[1,3] Most documented cases involve penetrating keratoplasty (PK), graft failure, chronic corneal edema, or topical corticosteroid use.[3,4,5,6]

Keywords: Corneal ulcer, *Enterobacter cloacae*, dry eye disease, topical corticosteroids

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Dry eye disease (DED) is a multifactorial disorder characterized by tear film instability, ocular surface inflammation, and neurosensory abnormalities.[7] Severe DED leads to disruption of the corneal epithelial barrier, impaired wound healing, and reduced resistance to pathogens. Topical corticosteroids or calcineurin inhibitors may exacerbate these effects.[8]

We present a rare case of *E. cloacae* keratitis that highlights the predisposing role of severe dry eye and topical corticosteroids and briefly review previously reported cases to contextualize the risk factors associated with this opportunistic infection.

A 77-year-old woman who did not use contact lenses presented with a 1-week history of pain, redness, and decreased vision in the right eye. Her best corrected visual acuity (BCVA) had declined from 20/25 to 20/32. She had a 3-year history of severe DED, with Schirmer test values of 4 and 3 mm, tear break-up time of 3 seconds, and grade II punctate keratitis in both eyes, together with meibomian gland dysfunction. No systemic predisposing conditions or previous ocular surgery were reported. The patient maintained adequate hygiene.

Treatment included warm compresses, lid hygiene, artificial tears (Thealoz Duo®; Laboratoires Théa, Clermont-Ferrand, France), and tear ointment (VitA-POS®; URSAPHARM Arzneimittel GmbH, Saarbrücken, Germany) at bedtime. Topical corticosteroid (dexamethasone 1 mg/mL, preservative-free; Dexafree®, Laboratoires Théa, Clermont-Ferrand, France) had been started 6 weeks earlier and was being tapered with a dose reduction every 10 days. At presentation, she was using fluorometholone 1 mg/mL (FML®, Allergan Pharmaceuticals, Westport, Ireland) once daily and cyclosporine A 0.05% eye drops (hospital-compounded formulation, Barraquer Ophthalmology Center Pharmacy, Barcelona, Spain) twice daily.

Slit-lamp examination showed mucous discharge, conjunctival hyperemia, and a 2-mm corneal ulcer with dense stromal infiltrate and surrounding calcium deposits in the inferior midperiphery (5 o'clock). No keratic precipitates were observed (Figure 1A).

Suspecting microbial keratitis, corneal scrapings were obtained for culture. Empirical treatment was begun with fortified cefazolin 50 mg/mL and amikacin 20 mg/mL eye drops (hospital-compounded formulation, Barraquer Ophthalmology Center Pharmacy, Barcelona, Spain) applied hourly while awake and ciprofloxacin 3 mg/g ointment (Oftacilox®, NTC Srl, Milan, Italy) at bedtime. Fluorometholone and cyclosporine A were discontinued.

Corneal cultures grew *E. cloacae* (Figure 1B) sensitive to amikacin, ciprofloxacin, and ceftazidime. After 1 week of treatment, the ulcer was controlled and the epithelial defect was closed. Thus, fortified cefazolin and amikacin were discontinued, and treatment was switched to ceftazidime 50 mg/mL (hospital-compounded formulation, Barraquer Ophthalmology Center Pharmacy, Barcelona, Spain) 5 times daily to reduce toxicity to the ocular surface, maintaining ciprofloxacin ointment every 12 hours. Medroxyprogesterone 20 mg/mL eye drops (hospital-compounded formulation, Barraquer Ophthalmology Center Pharmacy, Barcelona, Spain) were added to control inflammation.

After 1 month, infection was controlled but the calcium deposit enlarged (Figure 1C), causing a new epithelial defect. Conservative management with daily fluorometholone, tobramycin 3 mg/g ointment (Tobrex®, Novartis Pharma, Barcelona, Spain), and an eye patch was started. As no improvement occurred after 3 days, corneal curettage was performed, leaving 387 µm of residual stroma on anterior segment tomography (Figure 1D). The same regimen achieved complete epithelial closure within 1 week.

Maintenance treatment for severe DED was then resumed, including 50% autologous serum drops hourly, fluorometholone 1 mg/mL once daily, and cyclosporine A 0.05% twice daily to control chronic ocular surface inflammation.

At 7-month follow-up, stromal thinning and corneal haze persisted (Figure 1E), but BCVA improved to 20/25. Maintenance therapy continued with 6-month follow-up.

E. cloacae keratitis is rare; only 13 cases have been reported (Table 1). Most occurred in patients with a history of PK, graft failure, or prolonged topical corticosteroid use, often associated with corneal bullae, irregular ocular surface, or neurotrophic keratopathy. Gross and Meyer[4] noted reduced tear production as a possible factor. Rajarajan et al.[3] reviewed 7,787 infectious keratitis cases and identified *E. cloacae* in only 10 (0.13%): 9 following PK and 1 associated with bullous keratopathy. They suggested that epithelial bullae and an irregular corneal surface from graft failure may compromise the epithelial barrier, particularly when combined with long-term corticosteroid use. The most recent case, reported by Al Rasheed et al.,[6] occurred after corneal cross-linking with epithelial debridement, further emphasizing epithelial disruption as a major predisposing factor. Additionally, Feizi et al.[9] reported *E. cloacae* contamination in 7.02% of therapeutic bandage contact lenses, suggesting possible environmental exposure.

In our case, the absence of prior surgery, corneal decompensation, systemic disease, or contact lens use suggests that alternative risk factors must be considered. Severe DED is widely recognized to compromise epithelial integrity, impair tear film antimicrobial defense, and reduce corneal sensitivity, factors that collectively reduce ocular immunity.[7]

Interestingly, as shown in Table 1, no previously reported case of *E. cloacae* keratitis identified corticosteroid use as the sole risk factor. In all cases, topical corticosteroids were associated with epithelial barrier disruption such as post-surgical status, corneal bullae, graft failure, or chronic surface disease. This reinforces the idea that both epithelial compromise and local immunosuppression are required to trigger opportunistic keratitis.

Corticosteroids, while reducing ocular surface inflammation, further impair local defense mechanisms and increase susceptibility to opportunistic infections in eyes with already compromised epithelial integrity.[8] Our patient was using fluorometholone at presentation. Following TFOS DEWS III, medroxyprogesterone and autologous serum were used as corticosteroid-sparing therapy. Both provide regenerative and anti-inflammatory effects without relevant immunosuppression, making them appropriate for high-risk corneas.[7]

An additional complication was the formation of a calcium deposit at the ulcer site, delaying epithelial healing and requiring curettage. This was likely related to phosphate-containing fluorometholone (FML, Allergan). In compromised ocular surfaces, phosphate buffers may precipitate with calcium in the tear film, forming hydroxyapatite crystals.[10] Popiela and Hawksworth[10] noted this calcification can occur regardless of dosage or duration and advised avoiding phosphate-based drops in chronic ocular surface disease. This case highlights the need to critically evaluate the use of corticosteroids in DED.

In conclusion, this case suggests that severe DED and topical immunosuppression may suffice to trigger infection by an opportunistic pathogen such as *E. cloacae,* even without classical risk factors like PK or graft failure.

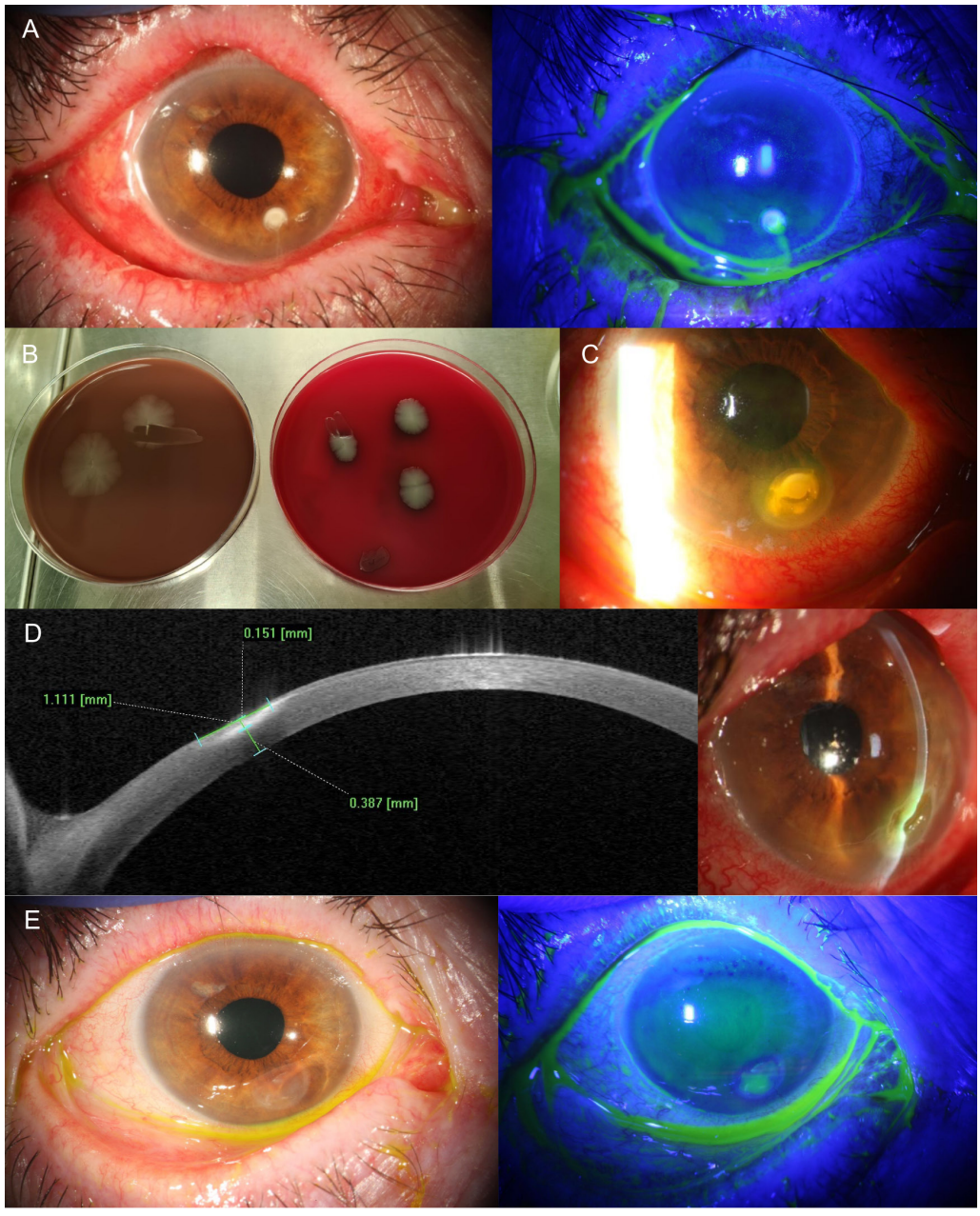

Figure 1. A) Slit-lamp exam at presentation showing corneal ulcer in the inferior midperiphery with dense infiltrates, calcium deposits and positive fluorescein staining. B) *Enterobacter cloacae* growth on chocolate agar (left) and blood agar (right). C) Slit-lamp photograph showing progressive calcium deposition hindering epithelial closure two months after initial treatment. D) Slit-lamp image (left) and anterior segment tomography (right) demonstrating focal corneal thinning. E) Slit-lamp exam at final follow-up showing no epithelial defect and fluorescein pooling due to residual surface irregularity

Table 1. Reported cases of *Enterobacter cloacae* keratitis in the literature and proposed risk factors

Cases	Gender/ age	Previous surgeries	Risk factors	Presenting VA	Ulcer size and morphology	Sensitive antibiotic	Final VA	Outcome/ duration (d)
Gross and Meyer[4] (1985)	F/70	-Extracapsular cataract extraction -PK for pseudophakic bullous keratopathy	-Chronic edema after graft rejection -Topical corticosteroid therapy -Tear insufficiency -Poor toilet hygiene and inadequate hand washing	HM at 15 cm	3.0x1.2 mm Dense, white stromal infiltrate	Gentamicin and sulfacetamide	NM	Residual edema secondary to previous graft rejection/7
Sharma et al.[5] (2020)	F/50	PK	NM except for surgery	HM close to face	Multifocal infiltrates of varying size	Ceftazidime	NM	Regraft/NM
Rajarajan et al.[3] (2021) (Case 1)	F/62	PK	-Failed PK -Corneal bullae and irregular ocular surface -Topical corticosteroid therapy	CF at 10 cm	6 mm^2 Patchy yellow	Ciprofloxacin	CF at 10 cm	Resolved/45
Rajarajan et al.[3] (Case 2)	M/45	PK	-PK -Corneal bullae and irregular ocular surface -Topical corticosteroid therapy -Neurotrophic keratopathy	CF at 10 cm	2.7 mm^2 Dense white	Ofloxacin	CF at 10 cm	Resolved/30
Rajarajan et al.[3] (Case 3)	M/41	PK	-Failed PK -Loose sutures -Topical corticosteroid therapy -Neurotrophic keratopathy	20/50	2.7 mm^2 Patchy white	Gentamicin, imipenem and chloramphenicol	20/100	Resolved/40
Rajarajan et al.[3] (Case 4)	M/58	PK	-Failed PK -Corneal bullae and irregular ocular surface -Topical corticosteroid therapy -Neurotrophic keratopathy	CF at 10 cm	1 mm^2 Patchy white	Ofloxacin	CF at 10 cm	Resolved/9
Rajarajan et al.[3] (Case 5)	M/55	PK	-Failed PK -Corneal bullae and irregular ocular surface -Topical corticosteroid therapy	HM	64 mm^2 Dense yellow	Amikacin	HM	Graft edema/ NM
Rajarajan et al.[3] (Case 6)	F/70	PK		CF at 10 cm	20.8 mm^2 Dense yellow	Chloramphenicol	NLP	Phthisis/120
Rajarajan et al.[3] (Case 7)	M/50	PK	-Failed PK -Topical corticosteroid therapy	Light perception	27.9 mm^2 Dense yellow	Gentamicin	HM	Scar/110
Rajarajan et al.[3] (Case 8a)	M/50	PK	-Failed PK -Corneal bullae and irregular ocular surface -Topical corticosteroid therapy	20/400	27.44 mm^2 Dense yellow	Azithromycin	CF at 10 cm	Regraft/60
Rajarajan et al.[3] (Case 8b)	M/50	PK		CF at 10 cm	9.5 mm^2 Dense yellow	Azithromycin	CF at 10 cm	Failed PK/45
Rajarajan et al.[3] (Case 9)	M/61	NM	-Corneal bullae and irregular ocular surface -Topical corticosteroid therapy	20/600	5.25 mm^2 Patchy white	Gatifloxacin	20/60	Scar/45
Al Rasheed et al.[6] (2023)	F/19	Corneal cross-linking for keratoconus	-Non-adherence to post-procedure medications -Corneal epithelium debridement	20/200	7.8 mm^2 Ring-shaped	Amikacin and moxifloxacin	20/40	Stromal haze and subepithelial scar/30
Cintas et al. (present case)	F/77	None	-Severe DED -Topical corticosteroid therapy	20/32	2 mm^2 Dense white with calcium deposits	Amikacin, ciprofloxacin and ceftazidime	20/25	Stromal thinning and corneal haze/70

CF: Counting fingers, d: Days, DED: Dry eye disease, F: Female, HM: Hand motion, M: Male, NLP: No light perception, NM: Not mentioned, PK: Penetrating keratoplasty, VA: Visual acuity

Evidence indicates that both epithelial disruption and corticosteroid use are usually required for the development of *E. cloacae* keratitis. Close monitoring of severe DED patients may prevent such infections.

Ethics

Informed Consent: Written informed consent was obtained from the patient for publication of the case and accompanying images.

Declarations

Authorship Contributions

Surgical and Medical Practices: V.C., Concept: N.C., E.R.P., G.J., V.C., Design: N.C., E.R.P., G.J., V.C., Data Collection or Processing: V.C., Analysis or Interpretation: N.C., E.R.P., G.J., V.C., Literature Search: N.C., E.R.P., G.J., V.C., Writing: N.C., E.R.P.

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