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# Corneal Infection by Enterobacter Cloacae post Penetrating Keratoplasty

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**Abstract**
*A 50-year-old woman underwent Penetrating Keratoplasty (PK) for Adherent Leukoma in her left eye. Three months later she reported with complaints of decrease in visual acuity, pain, redness and photophobia. She was given pulsed intravenous (IV) methyl prednisolone. There was reduction in symptoms however visual acuity did not improve. Ocular examination revealed visual acuity of 6/6 unaided in right eye and HMCF (Hand movements close to face) with accurate PR (Projection of rays).She developed a central corneal ulceration. Scraping of the corneal lesion revealed Gram-negative bacilli. Genus level identification was achieved using standard techniques and species level identification, revealing Enterobacter Cloacae, was aided by a VITEK 2 compact system. Broad-spectrum fortified antibiotics were initially started followed by species-sensitive fortified antibiotics. This case report, for the first time, highlights Enterobacter Cloacaeas a cause of an aetiological agent of corneal ulcer following PK.*
*Members of the genus Enterobacter are commensal organisms of the gastrointestinal tract and are considered pathogenic only for patients with lowered resistance to infection (e.g., chronic infection, cancer, or diabetes mellitus) or those with impaired immunity (congenital, acquired, or impaired immunity secondary to therapy). This organism has been implicated in endophthalmitis cases in previous case reports. However, to the best of our knowledge corneal infection caused by this organism has not been reported sofar.*
**Keywords:** *Penetrating Keratoplasty, Enterobacter Cloacae, Graft Failure.*

## Background

Enterobacter cause a plethora of infections that can include bacteremia, lower respiratory tract infections, skin and soft-tissue infections, urinary tract infections (UTIs), endocarditis, intra-abdominal infections, septic arthritis, osteomyelitis, CNS and ophthalmic infections. The infections can necessitate prolonged hospitalization, multiple and varied imaging studies and laboratory tests, various surgical and nonsurgical procedures, and powerful and expensive antimicrobial agents.

*Enterobacter cloacae*, an aetiological agent for post-op endophthalmitis has been sparingly described in the literature and, to the best of our knowledge, this report is the first of its kind to

describe corneal infection by *Enterobacter cloacae* in a postoperative setting in a patient who underwent PK.

**Case Presentation**

A 50-year-old woman non-diabetic and non-hypertensive underwent Penetrating Keratoplasty (PK) for Adherent Leukoma in her left eye. Three months later, she reported with complaints of decrease in visual acuity, pain, redness and photophobia in her left eye. She was diagnosed as a case of Graft rejection left eye. She was given pulsed intravenous (IV) methyl prednisolone followed by tapering doses of oral steroids. There was reduction in symptoms however, visual acuity did not improve. Ocular examination revealed visual acuity of 6/6 unaided in right eye and HMCF with accurate PR. Right eye examination was essentially within normal limits. Left eye examination showed oedema of upper and lower eyelids, circumciliary congestion, corneal epithelial defect with underlying multifocal infiltrates of varying size and 360° peripheral superficial vascularisation involving mainly the upper two quadrants (figure 1). Anterior chamber showed grade 1 flare and cells. The pupil was central, circular and sluggishly reacting to light. The lens was clear and fundus was unremarkable. Intraocular pressure as measured by applanation tonometer was 15 mm Hg in the right eye and digitally normal in the left eye.

**Investigations**

Scraping of the corneal lesion revealed Gram-negative bacilli on staining. Tryptone Soya Broth (TSB;Oxoid) or on MacConkey Agar (Oxoid). Double-strength Nutrient Broth (DSNB;Oxoid base) containing CaC1, 400 pg/ml was used for phage enrichments of sewage and slurry. Phages were propagated in Nutrient Broth (NB) with CaCl, 400pg/ml (CNB). Phage-typing agar (PTA) was composed of Nutrient Broth powder (Oxoid) 2% w/v, NaClO.5% w/v and agar (Oxoid) 0.7% w/v; CaCl, 0.24% w/v was added to the molten PTA at 50°C before pouring into petri dishes. All incubations with phages were at 32°C for 18 h. Colonies on Blood agar revealed large, smooth, flat colonies with entire margin without beta hemolysis[8].

Genus level identification was achieved using standard techniques and species level identification, revealing Enterobacter Cloacae, was aided by a VITEK 2 compact automated microbiology system.

**Treatment**

Broad-spectrum topical antimicrobial therapy in the form of Fortified E/d Ceftazidime 5% q1h, Fortified e/d Vancomycin 5% q1h, E/d Atropine 1% tidwas initially started. With the availability of an antibiotic sensitivity report through VITEK 2 compact automated microbiology system, fortified Ceftazidimewas continued, however, fortified Vancomycin was stopped. The isolate was found susceptible to all antimicrobials tested by the AST N-280 card, which included Beta lactams, Third generation Cephalosporins, Aztreonam, Carbapenems, Aminoglycosides, Quinolones, Colistin.

**Outcome and follow-up**

Patient kept on maintenance steroid therapy. Planned for re-grafting 6 months after complete resolution

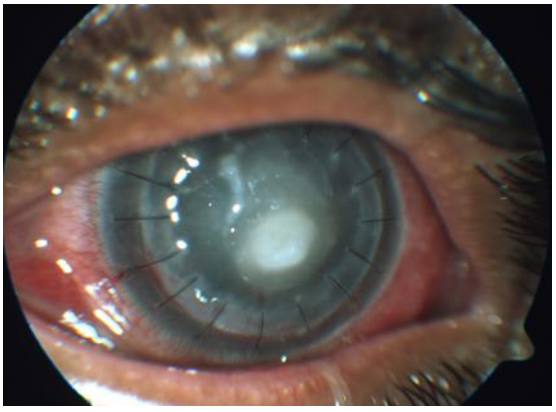

**Figure 1:** Slit Lamp Photograph showing ocular surface infection on initial evaluation

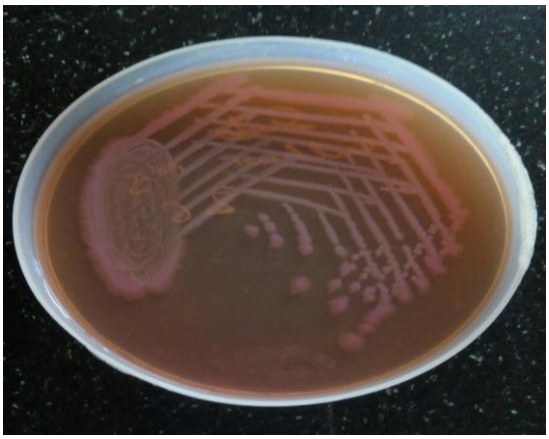

**Figure 2:** Enterobacter Cloacae growth on Mac Conkey medium

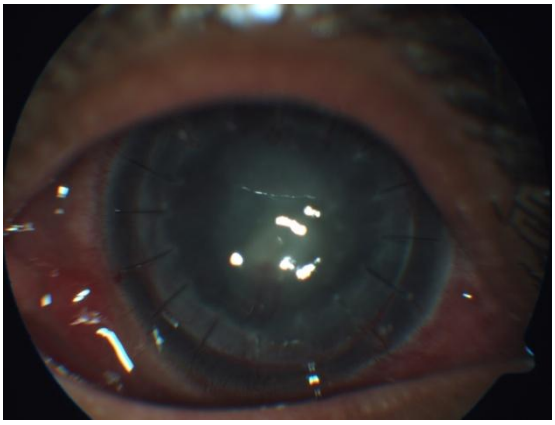

**Figure 3:** Slit Lamp Photograph showing reduction in signs post steroid therapy

**Discussion**

Microbial keratitis is a significant cause of ocular morbidity in cornea transplant patients and constitutes an important cause for graft failure [1,2].It necessitates a prompt diagnosis, thorough microbial investigations, and an intense medical therapy. The16-18.5% of cases of culture proven post-surgical endophthalmitis and 30% of post-traumatic cases in rural areas are primarily caused by Gram-negative organisms. The most common include Pseudomonas, H.Influenzae, Proteus spp., E. coli, and Klebsiella[3,4,5].

Enterobacter is a genus of a common Gram-negative, facultative anaerobic, rod-shaped, non-spore-forming bacteria belonging to the family Enterobacteriaceae. Enterobacter-aerogenes and E. cloacae are nosocomial pathogens that can cause a range of infections such as bacteremia, lower respiratory tract infection, skin and soft tissue infections, urinary tract infections, endocarditis, intra-abdominal infections, septic arthritis, osteomyelitis, and ophthalmic infections[6]. Higher incidence of post-operative endophthalmitis secondary to infection with gram-negative organisms is seen in diabetic patients than non- diabetics than patients who are not diabetic[7]. E. cloaca has been implicated in post-traumatic endophthalmitis as part of a mixed infection[8]. The earliest case reports of postsurgical endophthalmitis caused by Enterobacter cloacae were published in 1966 and 1975[9,10]. Be it any etiology, the clinical picture at presentation is that of a severe, rapidly progressive disease with acute onset resulting in poor final visual outcome and even loss of the eye. The dissemination of Enterobacter sp. is associated with the presence of redundant regulatory cascades that efficiently control the membrane permeability ensuring the bacterial protection and the expression of detoxifying enzymes involved in antibiotic degradation/inactivation. In addition, these bacterial species are able to acquire numerous genetic mobile elements that strongly contribute to antibiotic resistance[10]. The organism's multiple antibiotic resistance further complicates the management of these infections. Enterobacter spp. is resistant to several antibiotics especially β-lactams[11].A recent study demonstrated the remarkable dissemination and variety of carbapenemase genes in E. cloacae complex[12,13]. Enterobacter infections do not produce a unique enough clinical presentation to differentiate them clinically from other acute bacterial infections.

**Competing interests:** None declared. Patient consent: Obtained.

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