Enterobacter cloacae Postsurgical Endophthalmitis: Report of a Positive Outcome

Scott Butikofer^a Jason M. Dettori^b G. Atma Vemulakonda^b
Mark Slabaugh^b

^aUniversity of Washington School of Medicine, and ^bDepartment of Ophthalmology, University of Washington, Seattle, Wash., USA

Key Words

Endophthalmitis · Gram-negative · *Enterobacter cloacae* · Cataract surgery

Abstract

We report a positive outcome of postcataract endophthalmitis caused by *Enterobacter cloacae*, which has previously resulted in poor outcomes in endophthalmitis. A 67-year-old man underwent uncomplicated cataract surgery. On the morning of postoperative day (POD) #1, he had significant anterior chamber inflammation without pain, hypopyon, or vitritis but then rapidly developed hypopyon and worsening visual acuity. He underwent a tap and inject with vancomycin and ceftazidime and was prescribed topical steroids and antibiotics as well as oral levofloxacin. On POD #3, cultures of the vitreous and aqueous returned positive for *E. cloacae*. By POD #6, his hypopyon had resolved with improved vitritis, decreased inflammation, and visual acuity of 20/200. Two weeks after surgery, his best-corrected visual acuity was 20/60. Contrary to prior reports, we demonstrate that it is possible to achieve a good outcome in cases of *E. cloacae* endophthalmitis treated early with appropriate antibiotics and anti-inflammatory agents.

Introduction

Multiple studies have established risk factors for poor outcomes of postcataract endophthalmitis, including early onset, poor initial visual acuity, and etiologies other than coagulase-negative *Staphylococcus* [1]. Specifically, Gram-negative organisms have been associated with some of the poorest outcomes [2, 3]. *Enterobacter cloacae* is a part of the normal flora of the gastrointestinal tract in 40–80% of humans [4]. It is also prevalent in hospital

Mark Slabaugh, MD
Box 359608
325 9th Avenue
Seattle, WA 98104 (USA)
E-Mail mas12@uw.edu

settings, likely because of its resistance to disinfectants and antimicrobials [5]. It has historically been viewed as a harmless commensal organism, but as with other *Enterobacter* species, it has emerged as an important nosocomial infection [6]. Most commonly it is associated with the contamination of IV fluids, total parenteral nutrition solutions, prosthetic devices, and surgical equipment [7–9]. While it has become a common cause of other nosocomial infections, it has been identified in only 14 previously reported cases of endophthalmitis. In 13 of these cases, the visual outcome was hand motions or worse. We present a case where early treatment with broad-spectrum antibiotics and anti-inflammatory agents resulted in a good outcome.

Case Report

A 67-year-old man presented to our clinic with blurry vision and glare. His visual acuity was 20/60 in the right eye and 20/100 in the left eye. His past medical history was significant for diabetes mellitus and hypertension. He was found to have visually significant cataracts but an otherwise normal eye exam and underwent uncomplicated cataract surgery of his right eye. On the morning of postoperative day (POD) #1, he had a fibrinous reaction with plasmoid cells in the anterior chamber and mildly elevated intraocular pressure. His visual acuity was 20/100. There was minimal pain, no hypopyon, and no vitritis present. The patient was instructed to use prednisolone drops every hour and was examined again 6 h later. By this time, his vision was subjectively worsening and was measured to be 20/300. He continued to deny pain. Slit lamp exam revealed corneal edema and a 1-mm hypopyon. A tap and inject was performed consisting of vancomycin 1 mg and ceftazidime 2.25 mg, and the patient was instructed to use ofloxacin drops 4 times a day and continue prednisolone drops every hour while awake. He was also given a prescription for daily oral levofloxacin.

He did not return the following day but was seen again in the clinic on POD #3. His visual acuity had decreased to counting fingers at 3 feet. Cultures were positive with Gram-negative rods from both aqueous and vitreous samples. Slit lamp exam revealed a resolving hypopyon, improving corneal edema, and decreasing anterior chamber inflammation. The patient had not filled his prescription for oral levofloxacin and was again instructed to fill this prescription.

On POD #4, the patient's visual acuity had improved to the 200 'E' at 4 feet. His exam continued to improve and the culture results were positive for *E. cloacae* in both samples, resistant to ampicillin, but sensitive to ceftazidime. By POD #6, his hypopyon and corneal edema had resolved. The patient finished a 14-day course of levofloxacin, and on POD #17 had a best-corrected visual acuity of 20/60 in the right eye. Exam at that time revealed moderate vitreous debris but no retinitis.

Discussion

We report a case of postsurgical *E. cloacae* endophthalmitis which was treated early, resulting in a positive outcome. The earliest case report of postsurgical *E. cloacae* endophthalmitis was reported in 1966 [10]. There have subsequently been 2 case reports and 2 case series of postsurgical *E. cloacae* endophthalmitis. Outcomes for these eyes have been uniformly poor. Of 14 cases reported in the literature, 1 culture-negative case included in a series based on history achieved useful vision, 1 achieved hand motions, 1 achieved light perception (LP); the other 11 had no LP (NLP). Okhravi et al. [11] reported 4 patients who

presented more than 3 days after surgery. They were treated with intravitreal injections of amikacin and vancomycin as well as oral ciprofloxacin and clarithromycin. One eye achieved hand motions; the other eyes resulted in NLP. Mirza et al. [12] reported 7 eyes in 6 patients who all underwent surgery on the same day and presented on POD #1 with endophthalmitis. They were initially treated with subconjunctival injections but then received intravitreal gentamicin and cefazolin as well as dexamethasone. Subsequent cultures revealed persistent growth of *E. cloacae*, which was resistant to these antibiotics. Repeat intravitreal injection with amikacin was completed on POD #6. Of these 7 eyes, 5 progressed to phthisis and 1 retained LP. The one eye that retained useful vision (0.9) did not undergo vitreous tap due to less severe presentation and thus never had a positive culture for *E. cloacae*. Milewski and Klevjer-Anderson [13] reported a case in which the patient presented on POD #3 with a 75% hypopyon and LP; she progressed to phthisis and NLP. Kroll [14] reported a case in 1975 in which the endophthalmitis was detected on the day the patient first became symptomatic, but in which the patient received no intravitreal antibiotics or vitrectomy. The patient was treated with systemic, topical, and sub-Tenon steroids and antibiotics, but her course eventually led to evisceration. Rose and Koch [10] reported the first case of postsurgical *E. cloacae* endophthalmitis as part of a case series, but the outcome of this patient was not discussed.

Our patient presented on the morning of POD #1 with concerning findings that rapidly progressed over the course of a few hours. The diagnosis of toxic anterior segment syndrome was entertained but endophthalmitis remained a high concern and we elected to treat with early injection of broad-spectrum antibiotics, frequent topical steroids, and oral antibiotics. Although he did not start his oral antibiotics until at least 3 days later, the intravitreal antibiotics and topical steroids were presumably effective in treating his infection and preventing further ocular injury. Given the relatively short follow-up period, it is possible that this patient had a relapse of his infection that went untreated; however, in the previously reported cases, there were no descriptions of meaningful short-term improvement. In 8 of the 14 cases that contain detailed time course descriptions, visual acuity had decreased to NLP within 3 weeks.

It is clear that early treatment in endophthalmitis is essential to achieve good outcomes. Had our patient waited for symptoms to develop or been seen 1 day later, he likely would have had a much poorer outcome. Our case implies that even with very virulent organisms such as *E. cloacae*, a good outcome can be achieved with effective, early treatment.

Acknowledgements

This research was supported in part by an unrestricted grant from Research to Prevent Blindness.

The authors have no financial interest or gain related to this research.

References

- 1 Lalwani GA, Flynn HW, Scott IU, et al: Acute-onset endophthalmitis after clear corneal cataract surgery. *Ophthalmology* 2008;115:473–476.
- 2 Durand M: Microbiologic factors and visual outcome in the Endophthalmitis Vitrectomy Study. *Am J Ophthalmol* 1997;124:127–130.
- 3 Pijl BJ, Theelen T, Tilanus MA, Rentenaar R, Crama N: Acute endophthalmitis after cataract surgery: 250 consecutive cases treated at a tertiary referral center in the Netherlands. *Am J Ophthalmol* 2010;149:482–487.

- 4 Keller R, Pedrosa MZ, Ritchmann R, Silva RM: Occurrence of virulence-associated properties in *Enterobacter cloacae*. Infect Immun 1998;66:645–649.
- 5 Ren Y, Ren Y, Zhou Z, et al: Complete genome sequence of *Enterobacter cloacae* subsp. *cloacae* type strain ATCC 13047. J Bacteriol 2010;192:2463–2464.
- 6 Gaston MA: *Enterobacter*: an emerging nosocomial pathogen. J Hosp Infect 1988;11:197–208.
- 7 Matsaniotis NS, Syriopoulou VP, Theodoridou MC, et al: *Enterobacter* sepsis in infants and children due to contaminated intravenous fluids. Infect Control 1984;5:471–477.
- 8 Archibald LK, Ramos M, Arduino MJ, et al: *Enterobacter cloacae* and *Pseudomonas aeruginosa* polymicrobial bloodstream infections traced to extrinsic contamination of a dextrose multidose vial. J Pediatr 1998;133:640–644.
- 9 Buxton AE, Anderson RL, Klimek J, Quintiliani R: Failure of disposable domes to prevent septicemia acquired from contaminated pressure transducers. Chest 1978;74:508–513.
- 10 Rose HD, Koch ML: Hospital-acquired *Aerobacter cloacae* infections. Arch Intern Med 1966;117:92–98.
- 11 Okhravi N, Ficker L, Matheson MM, Lightman S: *Enterobacter cloacae* endophthalmitis: report of four cases. J Clin Microbiol 1998;36:48–51.
- 12 Mirza GE, Karaküçük S, Doğanay M, Çağlayangil A: Postoperative endophthalmitis caused by an *Enterobacter* species. J Hosp Infect 1994;26:167–171.
- 13 Milewski SA, Klevjer-Anderson P: Endophthalmitis caused by *Enterobacter cloacae*. Ann Ophthalmol 1993;25:309–311.
- 14 Kroll M: Gram-negative endophthalmitis: case report. Ann Ophthalmol 1975;7:255–258.