Severe Retinal Necrosis Due to *Klebsiella pneumoniae* After Acute Prostatitis

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**Patient:** Male, 72-year-old  
**Final Diagnosis:** Endogenous endophthalmitis  
**Symptoms:** Vision loss  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** Ophthalmology

**Objective:** Unusual clinical course  
**Background:** We report a rare case of unilateral *Klebsiella pneumoniae* endogenous endophthalmitis with retinal necrosis secondary to acute prostatitis and its clinical management.  
**Case Report:** A 72-year-old immunocompetent male presented with high fever and gastrointestinal and genitourinary symptoms. He was diagnosed with acute prostatitis, hospitalized, and started on a systemic antibiotic. After 3 days, he experienced floaters in the right eye with subsequent loss of vision. He was referred to the ophthalmology department, where endophthalmitis was diagnosed. The patient underwent complete pars plana vitrectomy (PPV); vitreous samples were taken, and intravitreal antibiotics were injected. Intraoperatively, the retina appeared moderately ischemic, with signs of vasculitis and an area of infiltrated retina temporal to the central fovea. The microbiology results from the vitreous samples showed *Klebsiella pneumoniae* presence. After 9 days, rhegmatogenous retinal detachment ensued and the patient underwent phacoemulsification + intraocular lens implantation in the capsular bag, a second PPV, and silicone oil tamponade. Temporal to the fovea, a large area of retinal necrosis was observed. After a 10-month followup period, the silicone oil was removed, and subsequently, visual acuity improved, while the retina remained attached.  
**Conclusions:** *Klebsiella pneumoniae* can be an aggressive microorganism that can cause retinal necrosis and compromise visual function. Prompt PPV can lead to some preservation of vision. This case demonstrates that a second PPV can prove to be a good therapeutic solution and should not be delayed.

**Keywords:** Endophthalmitis • *Klebsiella pneumoniae* • Ophthalmology • Prostatitis • Retina  
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Background

Endophthalmitis is an uncommon but potentially sight-threatening condition characterized by inflammation of intraocular tissues and fluids caused by microorganisms [1]. According to the etiology or the way microorganisms are transmitted, this ocular pathology can be divided into 2 categories: exogenous and endogenous [2]. Exogenous endophthalmitis is caused by microorganisms accessing the ocular tissues through the external environment; most commonly, it occurs as a complication of ocular surgery, trauma, or intravitreal injections [3]. Endogenous endophthalmitis (EE) occurs through hematogenous spread of microorganisms from inflammatory lesions elsewhere in the body. EE occurs when pathogenic microorganisms enter the bloodstream, and, via a damaged blood-ocular barrier, enter the retina and the vitreous [4]. Both categories lead to intraocular inflammation and potentially severe visual loss [5]. Only 53% of patients with EE are initially examined by an ophthalmologist; 35% present to an internist, general practitioner, pediatrician, or emergency department physician [6]. Therefore, collaboration among members of a diverse medical team, including experts in intensive care and infectious diseases, is frequently required. There are no recommendations for treating EE since it is relatively rare [7]. Several microorganisms have been known to cause bacterial EE, such as S. aureus, B. cereus, and gram-negative organisms, including Escherichia coli, Neisseria meningitidis, and Klebsiella spp. [8,9-12]. Klebsiella pneumoniae is a species of bacteria that mostly affects people with compromised immune systems and causes hospital-acquired infections. A subgroup of aggressive K. pneumoniae serotypes with high capsule polysaccharide synthesis can infect previously healthy people and produce potentially fatal community-acquired diseases such as pyogenic liver abscess, meningitis, necrotizing fasciitis, endophthalmitis, and severe pneumonia [13]. The most common systemic risk factors for EE are diabetes mellitus, immunosuppression, immunodeficiency, pneumonia, urinary tract infection, malignancy such as lymphoma or leukemia, colon adenocarcinoma, dental infection, vertebral osteomyelitis, liver abscess, asplenia, hypogammaglobulinemia, cardiac or renal transplant, acquired immune deficiency syndrome, chronic alcoholism, intravenous catheters, and intravenous drug abuse [14-18]. Ocular inflammation may be the initial symptom, or EE may be secondary to an already identified systemic illness [7].

Retinal necrosis is a consequence of severe chorioretinitis and can be caused by multiple diseases, including acute retinal necrosis caused by varicella zoster virus (VZV) or herpes simplex virus (HSV-1 or HSV-2). Retinal necrosis can also be caused by cytomegalovirus chorioretinitis, toxoplasma chorioretinitis, and endophthalmitis caused by K. pneumoniae. [19]. Here, we report a rare case of EE with retinal necrosis caused by K. pneumoniae.

Case Report

A 72-year-old immunocompetent male presented with severe high fever and gastrointestinal and genitourinary symptoms. At the urology department, he was diagnosed with acute prostatitis, hospitalized, and started on systemic antibiotic therapy. He received gentamycin 240 mg per 24 h i.v. empirically. From urine culture collected on admission, ampicillin-resistant K. pneumoniae was isolated in numbers greater than 100,000 CFU/ml. After 3 days, he experienced floaters in the right eye with consequent loss of vision, and was therefore referred to a local ophthalmologist. Since the examination showed intraocular inflammation, topical therapy with cycloplicics and corticosteroid eye drops was prescribed, with little benefit. Three days later, the patient presented to the Eye Hospital, University Medical Centre Ljubljana, Slovenia, where endogenous endophthalmitis was suspected. Best corrected visual acuity (BCVA) in the right eye was hand motion, and intraocular pressure was 12 mmHg. Dilated slit lamp eye examination showed marked exudation in the anterior chamber, poorly dilated, round pupil, fibrin on the anterior lens capsule, and opaque fundus view. The left eye showed a normal status. Right eye ultrasound showed a vitreous chamber with hyper-reflective signal compatible with abscess or hemorrhage. The patient underwent an urgent and complete pars plana vitrectomy (PPV) and vitreous biopsy with intravitreal injection of vancomycin 1 mg/0.1 ml + ceftazidime 2 mg/0.1 ml in the right eye. Intraoperatively, the retina appeared moderately ischemic, with signs of vasculitis and an area of infiltrated retina temporal to the central fovea. Vitreous samples underwent Gram staining and microbiological cultures for bacteria and fungi, which showed growth of K. pneumoniae. In the early days following the initiation of treatment, the inflammation slowly started to subside. The microbiological examinations showed that this strain of K. pneumoniae was resistant to ampicillin and sensitive to all other tested antibiotics. Three days after complete vitrectomy, the patient received a targeted injection of ceftazidime intravitreally, but on the same day he developed fever. Since sepsis was suspected, he was transferred to the infectious disease department and thereafter examined daily by an ophthalmologist. After 9 days, due to re-deterioration of the vision, the patient was again referred to the emergency eye clinic. Ophthalmoscopy examination revealed opacities in the vitreous and retinal detachment temporal to the central fovea with the presence of moderate cataract. The patient underwent phacoemulsification with in-the-bag intraocular lens (IOL) implantation, posterior capsulotomy, and PPV. Since the intraoperative visibility of the retina was reduced because of the cataract and it was expected to get even worse postoperatively, phacoemulsification with IOL implantation was performed. The intraoperative retinal examination showed the vitreous cavity to be highly infiltrated with dense inflammatory cells and fibrin (Video 1). Temporal to the fovea was a large...
retinal necrotic area of 4×5 optic disk diameters (Figure 1). Due to the extensive necrotic condition of the retina, no laser photocoagulation nor cryotherapy was performed. Since no holes were identified intraoperatively and a long-lasting tamponading agent (silicone oil) was chosen, a large barrage around the extensive necrotic area was deemed unnecessary at this stage. During the postoperative followup, scar tissue developed in the location of the necrotic retina. The retina was flat, and the inflammation completely disappeared. BCVA improved to 0.2 on the Snellen chart. After a 10-month followup period, the retina remained attached and without new changes; the silicone oil was therefore removed. Intraoperatively, laser photocoagulation around the edges of retinal defects was performed. During the followup period, visual function improved, and the retina remained attached. The optical coherence tomography scan (Figure 2) showed regular foveal contour despite the large temporal retinal defect (Figure 3). The final BCVA stabilized to 0.3 after 3 months.

Discussion

EE is a relatively rare but potentially devastating condition, accounting for only 2 to 16% of all endophthalmitis cases. The literature on EE mostly comprises case series or single case reports [6,20-22]. Due to the paucity of EE cases, unlike exogenous endophthalmitis, demographics, treatment options, and outcome measures in patients with EE have not been evaluated in large-scale studies [23].

*K. pneumoniae* EE is a rare secondary infection with poor visual prognosis, requiring early surgical intervention and aggressive antimicrobial treatment [9-12]. Prostatitis is a rare, but potentially fatal condition when associated with *K. pneumoniae*, requiring aggressive antimicrobial treatment along with surgical intervention. The condition requires adequate evaluation, while source investigation is crucial to avoid dissemination [24].
Risk factors like diabetes and conditions associated with immunosuppression may play a major role in the development of EE secondary to *K. pneumoniae* prostatitis [25,26]. Moreover, numerous cases of *K. pneumoniae* EE have been described as secondary to liver abscess hematogenous spread [27-29].

When bacterial endophthalmitis occurs, in the acute phase, microorganisms induce an inflammatory response that damages the retinal cells. Inflammation of the arterioles leads to occlusive events, favoring rapid necrosis of the retinal tissue [30]. In the late phase, contractile membranes may form on the surface of the retina. Both retinal necrosis and membrane proliferation can produce multiple retinal breaks and defects that may lead to retinal detachment [31,32]. A similar process occurs in acute retinal necrosis, in which retinal detachment has been described in up to 75% of eyes within 3 months of the onset of symptoms [33,34].

EE due to *K. pneumoniae* is a rare but devastating ocular infection with poor visual prognosis, requiring early surgical intervention and aggressive antimicrobial treatment [9-12]. Shields et al, in a case series of 12 eyes, reported very bad visual outcomes in most of the patients (with final visual acuity of light perception only or total blindness), and 5 of the 12 patients experienced enucleation or evisceration [12].

A devastating outcome in a case of EE due to *K. pneumoniae* was reported also by Zhou et al; despite PPV, the endophthalmitis deteriorated, eventually leading to corneal perforation [35]. A 10-year retrospective analysis of *K. pneumoniae* EE cases in western China showed a mortality rate of 10% of cases [36]. In their analysis, only 20% of patients with visual acuity better than hand movement at presentation achieved final BCVA of 0.1 or better on decimal scale [36].

Recent literature has reported an increase in the incidence of *K. pneumoniae* EE in Asia and Australia [37]. *K. pneumoniae* is now the most common cause of EE in Asia, with up to 9% prevalence in patients with *K. pneumoniae* liver abscess [38-40]. The incidence of metastatic spread is 10-fold higher in *K. pneumoniae* sepsis compared with sepsis caused by other organisms. The virulence of the K1 capsule serotype strain of *K. pneumoniae* has been regarded as a major risk factor for hematogenous complications [41,42].

The emergence of carbapenem-resistant *K. pneumoniae* in Asia, as well as resistance to third generation cephalosporins and fluoroquinolones is of great concern. For prompt diagnosis and correct treatment, the physician should be alerted to this issue [43].

In our scenario, the infection took 3 days to spread from the prostate to the eye. This is most likely because seeding through the bloodstream requires at least a few hours to take place. This period should alert the clinician to the clinical suspicion of endophthalmitis in the very first few days after the diagnosis of the primary infection, especially given the prevalence of multidrug-resistant bacteria. As indicated in our example, performing an early complete vitrectomy is critical for the functional prognosis in these patients.

Hereby, we report a rare case of *K. pneumoniae* EE with wide retinal necrosis in a previously healthy subject, and the treatment that led to a favorable outcome of the disease in a European setting.

**Conclusions**

EE is an intraocular inflammation with possible serious consequences for vision. *K. pneumoniae* can be a very aggressive microorganism that can cause retinal necrosis. The functional prognosis is poor, but prompt PPV can lead to some preservation of vision. This case demonstrates that a second PPV can prove to be a good therapeutic solution and should not be delayed.

**Declaration of Figures’ Authenticity**

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

**References:**

1. Mamalis N. Endophthalmitis. J Cataract Refract Surg. 2002;28(5):729-30
2. Vaziri K, Schwartz SG, Kishor K, Flynn HW. Endophthalmitis: State of the art. Clin Ophthalmol. 2015;9:99-108
3. Kent M, Kampik A. Endophthalmitis: Pathogenesis, clinical presentation, management, and perspectives. Clin Ophthalmol. 2010;4:121-35
4. Connell PP, O’Neill EC, Fabinyi D, et al. Endogenous endophthalmitis: 10-year experience at a tertiary referral centre. Eye (Lond). 2011;25(1):66-72
5. Guniani B, Kaur K. Endogenous endophthalmitis. StatPearls Publishing; 2021; [cited 2022 Jun 5]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK576391/
6. Okada AA, Johnson RP, Liles WC, et al. Endogenous bacterial endophthalmitis. Report of a ten-year retrospective study. Ophthalmology. 1994;101(9):832-38
7. Danielescu C, Anton N, Stanca HT, Munteanu M. Endogenous endophthalmitis: A review of case series published between 2011 and 2020. J Ophthalmol. 2020;2020:8869590
8. Durand ML. Bacterial and fungal endophthalmitis. Clin Microbiol Rev. 2017;30(3):597-613
9. Ghiam BK, Israelson P, Wang A, et al. Klebsiella pneumoniae endogenous endophthalmitis presenting as orbital cellulitis. GMS Ophthalmol Cases. 2019;9:Doc30
10. Yin W, Zhou H, Li C. Endogenous Klebsiella pneumoniae endophthalmitis. Am J Emerg Med. 2014;32(10):1300.e3-5
11. Chung CY, Wong ES, Liu CCH, et al. Clinical features and prognostic factors of Klebsiella endophthalmitis – 10-year experience in an endemic region. Eye. 2017;31(11):1569-75
12. Shields RA, Smith SJ, Pan CK, Do DV. Endogenous Klebsiella pneumoniae endophthalmitis in northern california. Retina. 2019;39(3):614-20
13. Li B, Zhao Y, Liu C, et al. Molecular pathogenesis of Klebsiella pneumoniae. Future Microbiol. 2014;9(9):1071-81
14. Durand ML. Endophthalmitis. Clin Microbiol Infect. 2013;19(3):227-34
15. Keyashian K, Malani PN. Endophthalmitis associated with intravenous drug use. South Med J. 2007;100(12):1219-20
16. Cheng HH, Ding Y, Wu M, et al. Endogenous aspergillus endophthalmitis after kidney transplantation. Int J Ophthalmol. 2011;4(5):567-71
17. Wu ZHY, Chan RPS, Luk FJO, et al. Review of clinical features, microbiological spectrum, and treatment outcomes of endogenous endophthalmitis over an 8-year period. J Ophthalmol. 2012;2012:265078
18. Regan KA, Radhakrishnan NS, Hammer JD, et al. Endogenous endophthalmitis: Yield of the diagnostic evaluation. BMC Ophthalmol. 2020;20(1):138
19. Davis JL. Diagnostic dilemmas in retinitis and endophthalmitis. Eye (Lond). 2012;26(2):194-201
20. Greenwald MJ, Wohl LG, Sell CH. Metastatic bacterial endophthalmitis: A contemporary reappraisal. Surv Ophthalmol. 1986;31(2):81-101
21. Romero CF, Rai MK, Lowder CY, Adal KA. Endogenous endophthalmitis: Case report and brief review. Am Fam Physician. 1999;60(2):310-14
22. Malmin A, Syre H, Ushakova A, et al. Twenty years of endophthalmitis: Incidence, aetiology and clinical outcome. Acta Ophthalmol. 2021;99(1):e62-69
23. Sadiq MA, Hassan M, Agarwal A, et al. Endogenous endophthalmitis: Diagnosis, management, and prognosis. J Ophthalmic Inflamm Infect. 2015;5:32
24. Kim JH, Yang WJ, Kim TH. Klebsiella pneumonia-induced prostate abscess: How to work it up? Can Urol Assoc J. 2014;8(11-12):E841-44
25. Ebisuno S, Miyai M. A case of Klebsiella pneumoniae endophthalmitis metastasized from prostateitis. Hinyokika Kiyo. 1994;40(7):625-27
26. Crane AB, Diaz MCA, Jiang Y, Pergament KM. Rare case of endogenous Klebsiella endophthalmitis associated with emphysematous prostatic abscess in a patient with diabetes, cirrhosis and COVID-19. BMJ Case Rep. 2021;14(4):e240425
27. David M, Poncecy AL, Kerwat R, Habal S. Klebsiella pneumoniae liver abscess with endophthalmitis in a diabetic man with gallstones. BMJ Case Rep. 2021;14(2):e239833
28. Pentecost GS, Kesterson J, Pyogenic liver abscess and endogenous endophthalmitis secondary to Klebsiella pneumoniae. Am J Emerg Med. 2021;41:264.e1-e3
29. Hussain I, Ishrat S, Ho DCW, et al. Endogenous endophthalmitis in Klebsiella pneumoniae pyogenic liver abscess: Systematic review and meta-analysis. Int J Infect Dis. 2020;101:259-68
30. Vallejo-Garcia IL, Asencio-Duran M, Pastorra-Salvador N, et al. Role of inflammation in endophthalmitis. Mediators Inflamm. 2012;2012:196094
31. Haider NB, Cruz NM, Allocca M, Yuan J. Pathobiology of the outer retina: Genetic and nongenetic causes of disease. In: McManus LM, Mitchell RN, editors. Pathobiology of human disease. San Diego: Academic Press; 2014; 2084-114
32. Wang T, Moinuddin Q, Abuzaitoun R, et al. Retinal detachment after endophthalmitis: Risk factors and outcomes. Clin Ophthalmol. 2021;15:1529-37
33. Bergstrom R, Tripathy K. Acute retinal necrosis. StatPearls Publishing; 2022; [cited 2022 Jun 8]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470588/
34. Garty DS, Spalton DJ, Tilzey A, Hykin PG. Acute retinal necrosis syndrome. Br J Ophthalmol. 1991;75(5):292-97
35. Zhou Y, Wang X, Shen J, et al. Endogenous endophthalmitis caused by carbapenem-resistant hypervirulent Klebsiella pneumoniae: A case report and literature review. Ocul Immunol Inflamm. 2019;27(7):1099-104
36. Yang G, Huang X, Jiang S, Xu Z. Endogenous endophthalmitis caused by Klebsiella pneumoniae: A ten-year retrospective study in Western China. Ophthalmic Res. 2020;63(5):507-16
37. Oudouard C, Dng D, Shah PR, et al. Rising trends of endogenous Klebsiella pneumoniae endophthalmitis in Australia. Clin Exp Ophthalmol. 2017;45(2):135-42
38. Chavada R, Ng J, Maley M, Descallar J. Emergence of Klebsiella pneumoniae liver abscesses in South-Western Sydney. Infection. 2014;42(3):595-96
39. Park IH, Jun CH, Wi JW, et al. Prevalence of and risk factors for endogenous endophthalmitis in patients with pyogenic liver abscesses. Korean J Intern Med. 2015;30(4):453-59
40. Kashani AH, Elliott D. The emergence of Klebsiella pneumoniae endogenous endophthalmitis in the USA: Basic and clinical advances. J Ophthalmic Inflamm Infect. 2013;3(1):28
41. Shon AS, Baiwa RPS, Russo TA. Hypervirulent (hypermucoviscous) Klebsiella pneumoniae. Virulence. 2013;4(2):107-18
42. Liu Y, Wang J, Jiang W. An increasing prominent disease of Klebsiella pneumoniae liver abscess: Etiology, diagnosis, and treatment. Gastroenterol Pract. 2013;2013:258514
43. Chiu S-K, Wu T-L, Chuang Y-C, et al. National surveillance study on carbapenem-resistant hypervirulent Klebsiella pneumoniae in Taiwan: The emergence and rapid dissemination of KPC-2 carbapenemase. PLoS One. 2013;8(7):e69428