

Endogenous Endophthalmitis Originating from Liver Abscess: An Uncommon but Severe Clinical Entity

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DOI: <https://doi.org/10.36347/sjmcr.2025.v13i10.065>

| Received: 29.08.2025 | Accepted: 16.10.2025 | Published: 23.10.2025

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Abstract

Case Report

Endogenous endophthalmitis (EE) is an uncommon but vision-threatening intraocular infection resulting from hematogenous spread of microorganisms from a distant systemic focus. It typically affects immunocompromised or diabetic patients and requires prompt recognition and treatment to prevent irreversible visual loss. A 47-year-old man with poorly controlled type 2 diabetes mellitus presented with diabetic ketoacidosis secondary to a pyogenic liver abscess. During hospitalization, he developed sudden, painless vision loss in the left eye. Ophthalmic examination revealed findings consistent with EE. Despite negative microbiological cultures, empirical treatment with intravitreal vancomycin and ceftazidime, in conjunction with fortified topical and systemic antibiotics, was promptly initiated. Percutaneous drainage of the liver abscess was also performed. Rapid initiation of combined ophthalmic and systemic therapy resulted in marked clinical improvement, with near-complete visual recovery one month after treatment. This case underscores the importance of early recognition and multidisciplinary management of EE in high-risk patients. Prompt intravitreal antibiotic administration and effective control of the primary infection source are critical to preserving visual function and improving prognosis.

Keywords: Endogenous endophthalmitis; Liver abscess; Diabetes mellitus; Hematogenous infection.

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INTRODUCTION

Endogenous endophthalmitis (EE) is a rare but potentially devastating intraocular infection that poses a serious threat to visual function and may indicate an underlying life-threatening systemic disease. Unlike exogenous endophthalmitis, which typically follows ocular surgery or trauma, EE arises from the hematogenous dissemination of infectious agents, which cross the blood–retina barrier and seed the intraocular structures [1]. The primary infectious source is often located in the gastrointestinal, genitourinary, or cardiovascular systems, although other foci, such as hepatic or pulmonary infections, may also be implicated. Although EE accounts for only 2–8% of all endophthalmitis cases [2], [3], it carries a high risk of permanent visual impairment if diagnosis and treatment are delayed. The condition predominantly affects immunocompromised individuals, including patients with diabetes mellitus, HIV infection, malignancies, or those receiving immunosuppressive therapy, all of which increase susceptibility to systemic and ocular infections [4]. The eye's status as an immune-privileged site further facilitates microbial proliferation once pathogens breach

the blood–retina barrier, often leading to rapid intraocular inflammation and tissue destruction. Among the various systemic infections leading to EE, liver abscesses represent a particularly serious and life-threatening source. Although uncommon in Western populations, pyogenic liver abscesses are increasingly recognized as an important cause of metastatic EE, particularly in patients with diabetes or other immunosuppressive conditions. The visual prognosis in EE secondary to liver abscess is frequently poor, highlighting the need for prompt recognition, systemic management of the primary infection, and immediate intravitreal therapy.

In this report, we present the case of a 47-year-old diabetic patient who developed EE complicating a liver abscess. Through this case, we aim to emphasize the importance of early diagnosis, multidisciplinary management, and timely intervention in improving both systemic and ocular outcomes in high-risk patients.

Citation: Charaf Bouabbadi, Rachid Zarrouk, Adil Elkhouyaali, Aissam Fiqhi, Yassine Mozari. Endogenous Endophthalmitis Originating from Liver Abscess: An Uncommon but Severe Clinical Entity. Sch J Med Case Rep, 2025 Oct 13(10): 2465-2469.

CASE PRESENTATION

A 47-year-old man with a history of poorly controlled type 2 diabetes mellitus and pseudophakia in

the left eye (OS) for the past eight years was admitted to the Emergency Department of Mohammed V Military Teaching Hospital, Rabat, for diabetic ketoacidosis secondary to a pyogenic liver abscess (**Fig. 1**).



Figure 1: Contrast-enhanced computed tomography (CT) of the abdomen demonstrating a well-defined, necrotic abscess cavity in hepatic segment VI, consistent with a pyogenic liver abscess

During hospitalization, the patient reported a sudden decrease in visual acuity in the left eye, associated with redness and ocular pain evolving over the previous 24 hours. Ophthalmologic examination of the left eye revealed a visual acuity limited to hand motion, conjunctival hyperemia with a perilimbal flush, purulent

discharge, granulomatous keratic precipitates, and a 3+ anterior chamber cell reaction with hypopyon and a cyclitic membrane (**Fig. 2**). Intraocular pressure was within normal limits. Fundus visualization was impossible due to dense vitritis. Examination of the right eye (OD) was unremarkable.



Figure 2: Slit-lamp photograph of the anterior segment of the left eye demonstrating marked conjunctival hyperemia, hypopyon, intense anterior chamber reaction with Tyndall effect, and the presence of a cyclitic membrane

B-scan ultrasonography of the left eye demonstrated dense vitreous opacities without evidence of a chorioretinal mass (**Fig. 3**). These findings supported

the diagnosis of endogenous endophthalmitis of the left eye.

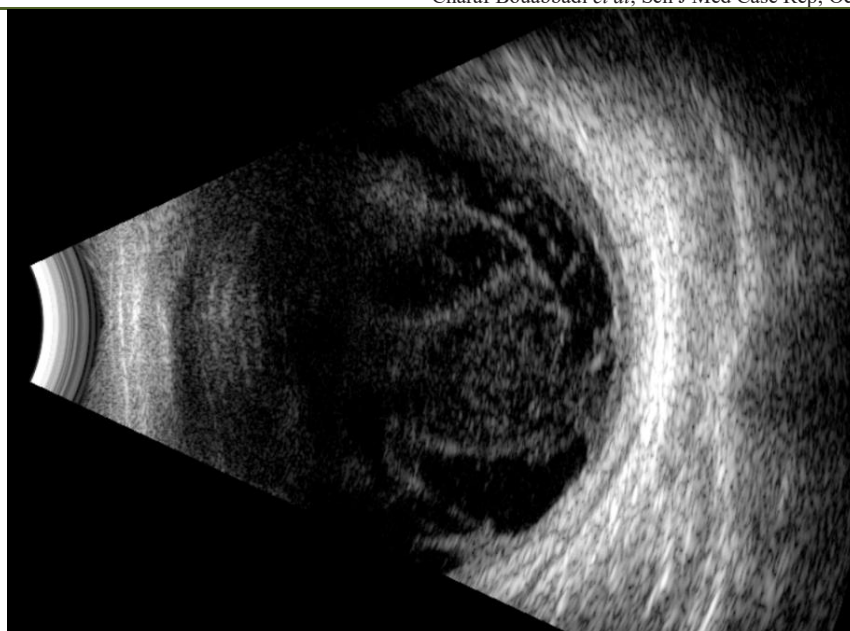


Figure 3: B-scan ultrasonography of the left eye demonstrating marked vitreous opacities consistent with vitritis; no chorioretinal mass or retinal detachment is observed

A systemic workup was performed to identify potential infectious foci other than the hepatic abscess. Urine cultures were sterile, transthoracic echocardiography excluded infective endocarditis, and chest radiography revealed no pulmonary involvement. Laboratory investigations showed a marked inflammatory response, with neutrophilic leukocytosis (15,500 cells/ μ L) and elevated C-reactive protein (CRP) level of 326 mg/L. Blood cultures were negative.

The patient underwent ultrasound-guided percutaneous drainage of the liver abscess and received systemic broad-spectrum intravenous antibiotic therapy consisting of imipenem (500 mg three times daily), levofloxacin (500 mg twice daily), and metronidazole (500 mg twice daily), along with correction of diabetic ketoacidosis.

Ocular management included intravitreal injections of vancomycin (1 mg/0.1 mL) and ceftazidime (2.25 mg/0.1 mL) administered on days 1, 3, and 6 (Fig. 4), in association with fortified topical antibiotics. Topical corticosteroids were introduced subsequently, after initial infection control. An anterior chamber paracentesis was performed prior to the first injection for microbiological analysis; however, both direct examination and culture results were negative.

The patient's systemic condition progressively improved. Serial ophthalmic evaluations demonstrated gradual resolution of ocular inflammation, with disappearance of discharge, regression of anterior chamber reaction, and clearing of vitritis. At one-month follow-up, the left eye showed near-complete visual recovery, with restoration of functional vision and no residual intraocular inflammation.

DISCUSSION

Endogenous endophthalmitis (EE) is a rare but severe intraocular infection resulting from hematogenous dissemination of microorganisms from a distant septic focus. Its incidence among all endophthalmitis cases is generally low, ranging from 0.04% to 0.5% [5], though higher proportions have been reported in certain populations.

EE may present unilaterally or bilaterally, with some studies suggesting a left-eye predominance [6], [7]. In Chinese series, trauma (71%) and ocular surgery (18%) are the most common causes of endophthalmitis, whereas EE accounts for approximately 7.8% of cases [8]. Incidence varies geographically, with reported rates of 7.4% in India, 16.5% in Thailand, and up to 23.5% in the United States [9-11]. The eye, as an immune-privileged site, provides an environment conducive to microbial proliferation once the blood-retina barrier is breached [7]. Jackson et al. identified diabetes, intravenous drug use, and malignancies as major risk factors for EE [1]. Primary infectious foci include endocarditis (46% of cases), followed by gastrointestinal, genitourinary, dental, hepatic, meningeal, and pulmonary infections; in approximately 10% of cases, the primary source remains unidentified [12].

EE complicating a liver abscess is uncommon but carries significant morbidity. Reported incidence ranges from 0.84% to 6.9% among patients with liver abscesses [13]. While liver abscesses remain relatively rare in Western populations, they are associated with high morbidity and potential mortality [14].

A shift in predominant pathogens has been observed in East Asia, with *Klebsiella pneumoniae* emerging as the leading causative organism in pyogenic liver abscesses and metastatic infections, including EE [15,16]. Hypervirulent K1 and K2 strains exhibit enhanced invasive potential and dissemination. Diabetic patients appear particularly susceptible, likely due to impaired neutrophil function and altered immune responses [2,14]. In a recent Korean cohort, larger abscess size (>5 cm), portal or hepatic vein thrombophlebitis, and cholangitis were identified as independent risk factors for metastatic EE [3].

Microbiological confirmation is crucial for EE diagnosis, with positive blood or vitreous cultures reported in 80–96% of cases [7]. Nevertheless, cultures may be negative if antibiotics are administered prior to sampling or if the sample volume is insufficient, as observed in our patient.

No definitive international guidelines exist regarding vitrectomy in EE; management generally involves prompt intravitreal and systemic broad-spectrum antibiotics [1]. Prognostic determinants include microbial virulence, host immune status, and rapid initiation of therapy.

Intravitreal vancomycin and ceftazidime are widely recommended to cover Gram-positive and Gram-negative bacteria [19]. Early intravitreal injection has been shown to significantly increase visual preservation and reduce enucleation rates [7]. Delays of more than 48 hours in initiating therapy are associated with poor visual outcomes [20]. Baseline visual acuity is also predictive: eyes with counting-fingers vision or better at presentation tend to have more favorable recovery [21,22].

In our case, treatment was initiated within 48 hours of symptom onset, correlating with near-complete visual recovery at one month. Recent reviews (2025) of *Klebsiella*-associated EE indicate that early combined systemic and intravitreal therapy is associated with improved visual outcomes, particularly in patients with diabetes or other immunocompromising conditions [16].

Despite aggressive management, many EE cases progress to limited visual recovery, phthisis, or enucleation, particularly if diagnosis or treatment is delayed [13,14,7]. Close ophthalmic follow-up is essential to monitor for late complications, including retinal detachment, recurrent infection, or fibrovascular membrane formation.

CONCLUSION

Endogenous endophthalmitis, although uncommon, represents a severe and vision-threatening complication of liver abscess. Its management requires prompt recognition and a multidisciplinary approach, involving both infectious disease specialists and

ophthalmologists. Early initiation of combined systemic and intravitreal antibiotic therapy—typically vancomycin and ceftazidime—alongside timely drainage of the hepatic abscess, is essential for preserving visual function, particularly in patients with poorly controlled diabetes mellitus. Careful monitoring and rapid intervention remain critical to optimizing both ocular and systemic outcomes, underscoring the importance of vigilance in high-risk populations.

Conflict of Interest: The authors declare that they do not have any conflict of interest.

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