












# Endogenous Endophthalmitis as a Triggering Factor for Fatal Bacterial Meningitis Caused by *Streptococcus pneumoniae*: A Case Report

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## Abstract

Endogenous endophthalmitis is a rare condition with a poor long-term visual prognosis and significant mortality, often associated with the hematogenous spread of intravitreal infections and subsequent disruption of the blood-ocular barrier. Its anatomical proximity to the central nervous system (CNS) poses a high risk of infection dissemination, although cases documented in the literature are rare, and endogenous endophthalmitis is typically described as secondary to neuroinfections. We report the case of an 82-year-old female patient with a history of hypertension who presented with fever, decreased visual acuity, severe headache, chemosis, and conjunctival injection. Endogenous endophthalmitis was diagnosed, and antimicrobial treatment was initiated alongside surgical intervention by the ophthalmology service. However, the patient's condition worsened neurologically, and *Streptococcus pneumoniae* was identified in cerebrospinal fluid cultures. Despite intensive medical treatment, the patient's clinical course was poor, leading to death.

## Keywords

Central Nervous System Infections, Meningitis, Infectious Disease Medicine, Endogenous Endophthalmitis

## 1. Introduction

Endophthalmitis refers to any inflammation within the eye, though in clinical practice, it typically refers to intraocular infections caused by bacterial or fungal agents [1] [2]. This condition can be classified into two major types: exogenous and endogenous endophthalmitis, depending on the route of infection [2] [3]. Exogenous endophthalmitis occurs when the infectious agent enters the eye through an external injury, such as penetrating trauma, surgical procedures, or the introduction of foreign bodies [4]. In contrast, endogenous endophthalmitis results from hematogenous spread, wherein microorganisms access the eye through the bloodstream, crossing the blood-ocular barrier [2]-[5].

Endogenous endophthalmitis is a rare condition, accounting for only 2% to 8% of all cases of endophthalmitis [2]. Despite its low incidence, it is a serious condition with a poor visual prognosis and a significant mortality rate [2] [4] [6]. The first case of endogenous endophthalmitis was described in 1856, and since then, it has been primarily associated with immunocompromised patients, including those with diabetes mellitus, chronic kidney disease, liver cirrhosis, immunosuppressive treatments, or malignancies [2]. Early intervention and rapid initiation of treatment are crucial for patient outcomes [6]. However, up to 25% of endogenous endophthalmitis cases may be misdiagnosed initially due to nonspecific clinical presentation or the absence of characteristic findings in the early stages [2]-[4] [7].

While the eye's anatomical proximity to the central nervous system presents a high risk of complications, cases in which neuroinfection precedes endophthalmitis are relatively common in the medical literature [2] [6]. Conversely, cases where neuroinfection is secondary to endophthalmitis are rarer, making the described case particularly unusual [7] [8]. It is hypothesized that severe intraocular inflammation and the potential for dissemination through structures adjacent to the CNS could be triggering factors for bacterial meningitis in certain vulnerable patients [7]-[9].

Bacterial meningitis is a severe inflammatory condition that affects the meninges, and the protective layers surrounding the brain and spinal cord [9] [10]. This infection can be caused by various pathogens, but *Streptococcus pneumoniae* is one of the most common agents, alongside *Neisseria meningitidis* and *Haemophilus influenzae* [10]. In immunocompromised patients, *Listeria monocytogenes* may also be implicated [9] [10]. Meningitis is a medical emergency associated with high morbidity and mortality [11] [12]. Immediate diagnosis and treatment are required to prevent severe complications, such as permanent neurological damage or death [10].

In the context of this case, the combination of endogenous endophthalmitis and bacterial meningitis caused by *Streptococcus pneumoniae* represents an extremely rare entity. A clinical approach emphasizing early diagnosis and timely intervention is crucial, as delayed treatment can result in devastating sequelae for both vision and neurological function [9] [10]. Despite advances in imaging and laboratory

techniques, the insidious nature of both diseases may hinder early identification, underscoring the importance of clinical vigilance and the use of appropriate empirical therapies when these life-threatening infections are suspected [2] [4] [9] [10].

This article aims to explore the relationship between endogenous endophthalmitis and bacterial meningitis, highlighting the pathophysiological mechanisms that might explain the spread of infection between these two anatomically close yet functionally distinct regions, and stressing the importance of early detection and aggressive treatment to improve patient prognosis.

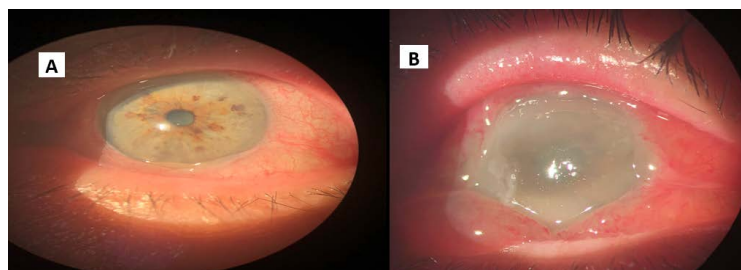
## 2. Case Presentation

An 82-year-old female patient with a history of well-controlled essential hypertension presented to the emergency department with a four-day history of asthenia, weakness, fever up to 39°C, and decreased visual acuity, associated with conjunctival injection and moderate headache (6/10 on the visual analog scale). Two weeks prior, the patient had been treated for community-acquired pneumonia with oral amoxicillin-clavulanic acid 1 gram every 12 hours for five days, without complications. There was no history of recent surgeries or ocular trauma.

On physical examination, the patient was tachycardic, with a heart rate of 105 beats per minute; other vital signs were within normal limits. Ophthalmological examination revealed a best-corrected visual acuity in the left eye of 20/40, with an intraocular pressure of 15 mmHg. The right eye had light perception only, with palpebral edema, erythema, preserved motility, 360° ciliary and conjunctival injection, and four-quadrant chemosis limiting eyelid closure. There was also corneal stromal edema, cloudiness, an intraocular pressure of 21 mmHg, and the fundus was unassessable due to opacification (**Figures 1-2**).

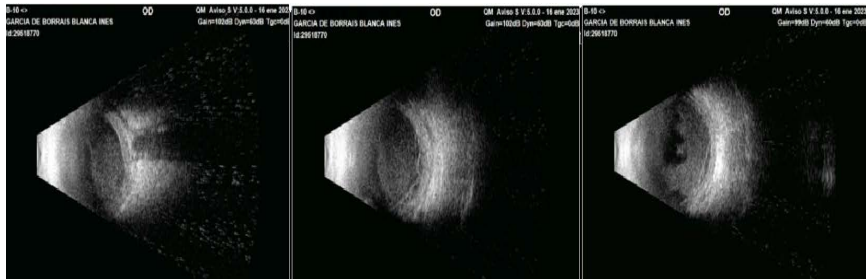


**Figure 1.** Patient admission photograph. Periorbital erythema, purulent discharge and alteration in ocular opening are evident.



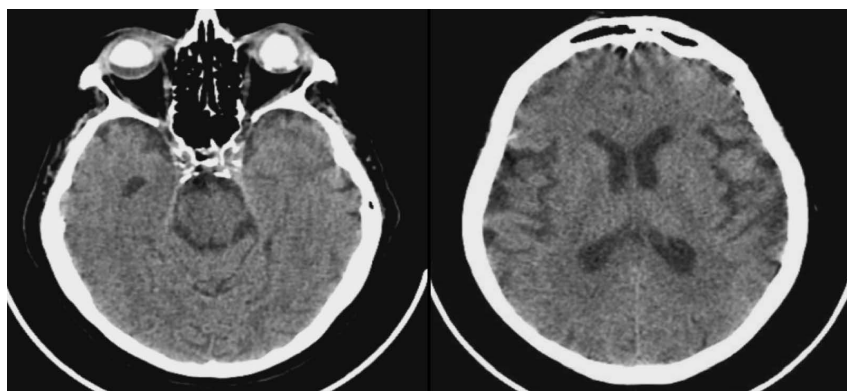
**Figure 2.** Left eye (A) and right eye (B).

Initial laboratory tests revealed leukocytosis (27,360 cells/ $\mu$ L) with 94% neutrophils, elevated C-reactive protein (31 mg/L), normal renal function, and normal urinalysis and chest X-ray. Specialized studies to investigate immunosuppressive conditions, including a normal hemoglobin A1c and negative HIV test, were performed. Ophthalmology initiated intravitreal vancomycin and ceftazidime, ordering an ocular ultrasound before the procedure (**Figure 3**). The patient later underwent vitrectomy, and antimicrobial therapy was continued.



**Figure 3.** Ocular ultrasound: Severe dense vitreous opacities, multiple images suggestive of choroidal detachment.

Despite optimal management, the patient's condition worsened, developing somnolence, bradycardia, and meningeal signs, including nuchal rigidity, positive Brudzinski's sign, and positive Kernig's sign. Systemic antimicrobial therapy with vancomycin and ceftriaxone was initiated, and additional studies, including a cranial computed tomography (CT) scan and lumbar puncture, were ordered. The CT scan showed chronic changes due to cortical atrophy (**Figure 4**), with increased density in both globes and no evidence of lesions or hemorrhage. Cerebrospinal fluid analysis revealed turbid fluid, hypoglycorrhachia, hyperproteinorrhachia, neutrophilic pleocytosis, and a positive culture for *Streptococcus pneumoniae*.



**Figure 4.** Cranial CT: Increased density in both globes, predominantly on the right. Mild cortical atrophy with white matter changes due to microangiopathy.

Despite timely interventions and appropriate antimicrobial coverage, 48 hours after identifying the neuroinfection, the patient progressed to septic shock, with

multiorgan failure leading to cardiopulmonary arrest and subsequent death. Endogenous endophthalmitis, possibly secondary to the previously documented infectious process, with advanced age being the only defined risk factor for this, triggering bacterial meningitis by proximity, with a fatal outcome for the patient.

### 3. Discussion

Endogenous endophthalmitis is a rare intraocular infection representing approximately 2% - 15% of all cases of endophthalmitis, that, despite its low incidence, represents a serious medical emergency due to its significant mortality and poor visual prognosis [2] [4] [7]. This condition results from hematogenous dissemination of microorganisms, which can be bacterial, fungal, or even mycobacterial, from a distant infectious focus to the eye, leading to severe inflammation and disruption of the blood-ocular barrier [13]-[17]. Although cases of endogenous endophthalmitis are infrequent, timely diagnosis is crucial for improving visual prognosis and reducing morbidity and mortality associated with the event [4].

The clinical presentation of endogenous endophthalmitis is often nonspecific, which may delay the correct diagnosis [4]. Patients may present with decreased visual acuity, ocular pain, redness, and eyelid edema, as seen in the case of the patient presented here [2] [6]. In terms of the order of symptom presentation, the most common is vision loss in up to 89% of cases, followed by pain in 48%, and fever, the most frequent systemic manifestation, occurring in 37% of cases. Regarding signs, the most common is hypopyon in 35% - 65% of cases [2] [6]. The main complications include glaucoma in 23% of cases and retinal detachment in 15% [2]. It is important to note that although endophthalmitis can occur in any individual, those with risk factors such as diabetes, immunosuppression, oncologic processes, or chronic diseases are more likely to develop it [2] [13] [15].

The relationship between endogenous endophthalmitis and neuroinfections, such as bacterial meningitis, underscores the importance of quickly suspecting and treating these infections [13] [14] [16] [17]. Although the anatomical risk of dissemination to the central nervous system is high due to the proximity of the eye to the meninges, neuroinfection secondary to endophthalmitis is a rare phenomenon [2]. In most reported cases, meningitis precedes endophthalmitis, while the case described here represents an unusual scenario in which neuroinfection is a secondary complication of ocular infection, with rapid progression and a fatal outcome, with few cases found in the medical literature of this sequence of events [5] [7] [8] [13] [14] [17].

In the case of bacterial meningitis, *Streptococcus pneumoniae* is the most common etiological agent in Colombia, which was involved in the case of our patient, possibly acquired weeks earlier during the respiratory process she experienced [10]. This agent is well known for its ability to cause bacterial meningitis; however, it is an uncommon etiological agent in cases of endogenous endophthalmitis, where gram-negative infections, specifically *Klebsiella pneumoniae*, followed by *Pseudomonas aeruginosa*, are more frequent, with *S. pneumoniae* ranking third

among gram-positive infections, after *Staphylococcus aureus* [2]. If not treated adequately, this infection can be fatal and is a major cause of morbidity and mortality globally, particularly in vulnerable populations such as the elderly, immunosuppressed individuals, or those with chronic diseases, as well as in geographically predisposed areas such as the meningitis belt in Africa [9] [11] [18].

In this context, early diagnosis of endogenous endophthalmitis and its association with neuroinfections is crucial [9] [10]. Ophthalmologists and primary care physicians must be alert to any signs of systemic dissemination in patients presenting with ocular symptoms. A delayed diagnosis of endophthalmitis may allow infection to spread to other areas, including the CNS, as occurred in this clinical case [4] [7].

The management of endogenous endophthalmitis includes a multidisciplinary approach that combines the administration of broad-spectrum antimicrobials, both intraocularly and systemically, as well as surgical interventions when necessary [2] [4] [6]. Vitrectomy is a common procedure in these cases, as it allows for the removal of purulent material from the vitreous and facilitates the direct delivery of drugs to the affected eye [4]. Furthermore, when neuroinfection is suspected, prompt use of complementary studies such as computed tomography and lumbar puncture is essential for proper diagnosis [4] [7].

It is important to emphasize that even when appropriate treatment is initiated early, the prognosis for patients with endogenous endophthalmitis and bacterial meningitis remains poor [2] [5] [6] [8]. As observed in this case, the patient experienced rapid clinical deterioration despite timely medical interventions. The mortality rate in these patients remains high, highlighting the need for further research and advances in the diagnosis and management of these infections [17].

#### 4. Conclusion

Endogenous endophthalmitis, although rare, is a potentially fatal infection that requires early diagnosis and prompt management. Its association with neuroinfections, such as bacterial meningitis, should be considered in the appropriate clinical context, especially in patients with risk factors. The case presented highlights the importance of clinical suspicion and rapid treatment to improve survival rates, although in some cases, like the one described, the disease progresses despite optimal interventions. This case also underscores the need for continued investigation into the underlying mechanisms of infection dissemination from the eye to the CNS, with the goal of developing more effective strategies to prevent these devastating complications.

#### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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