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Case Report

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# Corneal Endotheliitis Associated with a Methicillin Resistant Pyogenic Liver Abscess

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## Abstract

*Endotheliitis is the inflammation of the corneal endothelium resulting in edema and subsequent loss of vision. Bacterial causes of corneal inflammation primarily of the epithelium with subsequent, secondary involvement of the endothelium have previously been described. Notably, however, there are no reports of isolated endotheliitis related to a bacterial pathogen. We report, for the first time, a case of corneal endotheliitis associated with a pyogenic liver abscess caused by methicillin resistant Staphylococcus aureus (MRSA). Treatment targeting the underlying source of infection led to visual recovery in our patient.*

**Keywords:** Corneal Endotheliitis, Pyogenic Liver Abscess, Sepsis, Cornea, Bacteria, Immunology

## Introduction

The corneal endothelium regulates corneal hydration, which is critical for maintaining ocular surface transparency and clarity of vision. Inflammation of the endothelium, also known as endotheliitis, disrupts the function of this corneal sub-layer, resulting in corneal edema and subsequent vision impairment [1, 2]. Endotheliitis classically presents with corneal edema accompanied by inflammatory cellular deposits or keratic precipitates (KPs) [2]. Various causes of endotheliitis have been previously reported including microbial, autoimmune, drug-induced, and environmental exposures. Microbial etiologies may be viral, as commonly seen with herpes simplex virus (HSV), cytomegalovirus, and mumps virus [3-5]. Less common cases associated with fungal infection have also been documented [6, 7]. Aside from these pathogens, however, there are no reports of a bacterial etiology of endotheliitis. We report, for the first time, a case of isolated endotheliitis associated with a pyogenic liver abscess caused by methicillin resistant *Staphylococcus aureus* (MRSA).

## Case Presentation

A 65-year-old Caucasian woman presented to the emergency department with sepsis and blurry vision of the left eye. The patient was afebrile, intermittently tachycardic, and normotensive. Physical examination was significant for hypoactive bowel sounds and moderate abdominal distension without organomegaly. On ophthalmic examination, best-corrected visual acuity was count fingers at face in the right eye (unchanged from her baseline secondary to mature cataract and advanced glaucoma) and 20/400 in the left eye (markedly reduced from her baseline of 20/40). Slit lamp biomicroscopy showed mild conjunctival injection in both eyes. Notably, the cornea of the left eye exhibited a well-demarcated, central disciform area of ground glass deep stromal haze with underlying KPs and overlying stromal edema with Descemet folds (Figure 1). Slit lamp examination of the anterior chamber showed a hazy view. Cells were not appreciated and there was no hypopyon. Fundoscopic examination of the left eye revealed clear vitreous without inflammation, cup-to-disc ratio of 0.4, no hemorrhages or exudate, normal vessels, and normal macula reflex. Fundoscopic examination of the right eye was limited due to mature cataract. Tonometry measured an elevated intraocular pressure of 25 mm Hg in the left eye. Laboratory results were significant for leukocytosis with left shift (23.19 x $10^3$ cells/μL), transaminitis (aspartate aminotransferase 91 U/L, alanine aminotransferase 79 U/L), and elevated bilirubin (4.1 mg/dl). Urinalysis showed elevated urobilinogen (≥4.0 mg/dL). Additionally, computed tomography (CT) of the abdomen and pelvis showed right-sided colitis with left hepatic 3.7 x 3.6 cm hypoattenuating mass suspicious for a liver abscess, stable mild to moderate intrahepatic and extrahepatic biliary ductal dilatation with potential intraluminal filling defect near the distal common bile duct, nonspecific moderate gallbladder distention with associated sludge, and equivocal findings of small bowel malrotation (Figure 2). Orbital CT scan of the left eye was unremarkable with no signs of optic neuropathy.

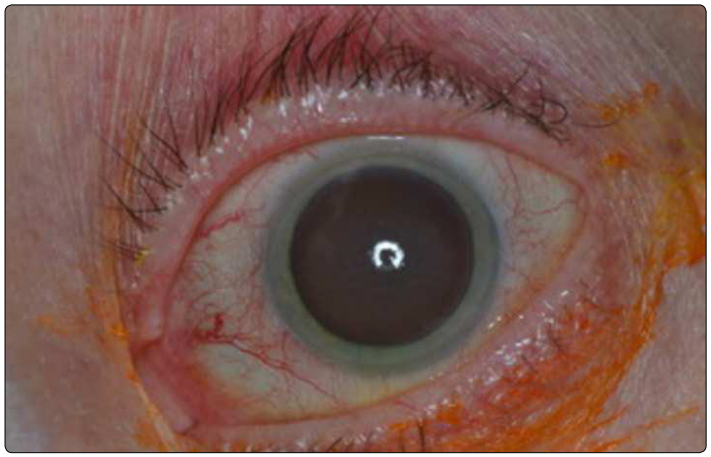

**Figure 1:** Slit lamp biomicroscopy showing disc-shaped pattern of keratic precipitates with overlying edematous lesions located centrally in the cornea.

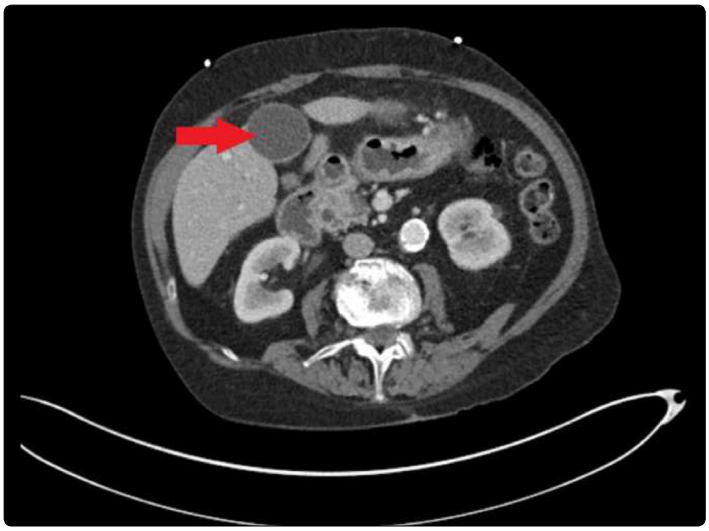

**Figure 2:** Abdominal computed tomography scan with intravenous contrast depicting the hepatic abscess (red arrow).

The patient was admitted to the hospital for inpatient management and was started on prednisolone acetate 1% topical drops four times daily. On hospital day two, the patient's abscess was drained. Her closed-suction drain put out purulent drainage for two days duration prior to removal. Cultures of drainage grew MRSA, and the patient received a 4-week course of intravenous vancomycin. Prednisolone acetate 1% drops were continued at four times daily dosing for a total of nine days and then tapered. Intraocular pressure normalized after treatment. Four weeks after hospitalization, the patient's cornea was clear and visual acuity returned to her baseline of 20/40.

## Discussion

Previous studies have documented bacterial causes of corneal inflammation primarily of the epithelium with subsequent, secondary involvement of the endothelium. Notably, however, there are no reports of isolated endotheliitis related to a bacterial pathogen. We present, for the first time, a case of corneal endotheliitis associated with a pyogenic liver abscess caused by MRSA. Endotheliitis can be classified into four categories according to the distribution patterns of corneal KPs and overlying stromal configuration: linear, sectoral, disciform, and diffuse [8]. Slit lamp biomicroscopy in our patient showed a disc-shaped pattern of KPs with overlying stromal edema located centrally in the cornea. This is consistent with the disciform subtype of endotheliitis. Notably, endotheliitis in our patient resolved following anti-biotic treatment of the hepatic infection. While disciform corneal endotheliitis is most commonly secondary to a viral cause, the temporal overlap between the onset of corneal pathology at the same time as onset of sepsis and the resolution of corneal pathology following treatment of the pyogenic liver abscess suggests a bacterial association with endotheliitis in our patient [9].

The pathophysiology of endotheliitis is poorly understood. Previous studies have suggested an autoimmune mechanism of disease referred to as anterior chamber-associated immune deviation (ACAID), which is triggered by intraocular microbial antigens [10, 11]. In this disease entity, the delayed-type hypersensitivity response is suppressed, whereas the humoral immune response remains intact or even enhanced. Similarly to our case, ACAID presents with mild to moderate anterior chamber reaction and without posterior segment involvement [10, 11]. In addition, ACAID may occur in immunocompetent patients and is responsive to topical anti-inflammatory treatment, which could explain our patient's response to topical corticosteroid therapy [10, 11]. Taken together, it is plausible that endotheliitis in our patient may have involved induction of ACAID-related disease by *S. aureus* antigens.

Alternative mechanisms of endotheliitis including direct microbial infection of endothelial cells have also been postulated [11]. The most severe septic complication of a pyogenic liver abscess is endogenous endophthalmitis, whereby hematogenous spread of pathogens or bacterial seeding leads to intraocular infection [12]. Endophthalmitis characteristically involves severe posterior chamber reaction and prognostic visual outcomes are often dismal [13, 14]. Vision is, however, salvageable with early detection and prompt initiation of therapy prior to progressing to severe disease stages [15-17]. Endotheliitis associated with early inflammatory changes preceding endophthalmitis would be unusual and far less likely in our case given the absence of vitritis or posterior segment involvement [13, 14].

Endotheliitis has a diverse array of etiologies and presentations. Considering a broad differential is important to distinguish this condition from alternative, similar appearing causes, since choice of therapy depends on the underlying etiology of the disease process. Endotheliitis is primarily a clinical diagnosis and further intervention was not indicated given our patient's improved clinical course as well as persistent asymptomatic state. Alternative causes including HSV are quite possible and cannot be ruled out with certainty. Nevertheless, the current article describes an atypical case of corneal endotheliitis associated with a pyogenic liver abscess and expands upon previously reported presentations of this disease entity. Corneal scraping or aqueous humor sampling may help to more definitively identify the infectious etiology and may be warranted in future studies.

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