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Case report

# Crystalline lens dislocation secondary to bacterial endogenous endophthalmitis

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ABSTRACT

*Purpose:* To present an unusual case of endogenous endophthalmitis secondary to Group A *streptococcus* (GAS) that resulted in dislocation of the crystalline lens.

*Observations:* An immunocompetent 51-year-old man presented to the emergency room (ER) with upper respiratory infection (URI) symptoms and painful right eye. He was diagnosed with URI and viral conjunctivitis and discharged on oral azithromycin and polytrim eyedrops. He returned to the ER 30 h later with sepsis and findings consistent with endophthalmitis, including light perception only vision. Ophthalmology was consulted at this time and an emergent vitreous tap and injection was performed. Both blood and vitreous cultures grew an atypical non-hemolytic variant of GAS (*Streptococcus pyogenes*). The primary source of infection was presumed to be secondary to pharyngitis or cutaneous dissemination. Final vision in the affected eye was no light perception, likely from a combination of anterior segment scarring, posterior segment damage, and hypotony. Interestingly, head computed tomography (CT) at the initial ER presentation showed normal lens position, but repeat CT at re-presentation revealed posterior dislocation of the lens.

*Conclusions and importance:* Endophthalmitis secondary to GAS has been sparsely reported in the literature, and this case highlights a unique clinical presentation. We suspect that this atypical non-hemolytic strain may have evaded detection on initial pharyngeal cultures. Additionally, we hypothesize that GAS-mediated protease release resulted in breakdown of the zonular fibers and subsequent lens dislocation. Ophthalmologists should be aware of GAS and its devastating intraocular manifestations.



## 1. Introduction

Endogenous endophthalmitis (EE) is defined by hematogenous intraocular seeding by microorganisms from a distant focus. Compared to fungal pathogens, bacterial agents are the culprit of EE in a minority of cases, and bacterial EE represents 2–8% of all cases of endophthalmitis.[1] A recent large study found the most common sources of infection to be the liver, lung, and endocardium.[2] Conditions predisposing to bacterial EE include diabetes, immunosuppression, intravenous drug abuse, and indwelling catheters among others.[3] In the US, nearly half of all cases of bacterial EE are due to gram positive organisms, most commonly *Staphylococcus aureus* and *group B Streptococcus*.[2,4–8] Group A *Streptococcus* (GAS) as the etiologic agent for EE is an exceedingly rare presentation, reported only a handful of times.[13–15] Furthermore, to our knowledge, lens dislocation has never been reported as a sequela of EE.

## 2. Case report

A 51-year-old immunocompetent male with quiescent diverticulitis and no past ocular history presented to the emergency room (ER) with two weeks of upper respiratory infection (URI) symptoms (cough, congestion, and sore throat), as well as one day of subjective fevers and a red, painful right eye. ER examination revealed conjunctival injection of the right eye. Ophthalmology was not consulted during this visit, thus a complete eye exam was not performed. Visual acuity was presumably grossly normal. Rapid streptococcal antigen and throat culture were negative. Head computed tomography (CT) was ordered due to 8 out of 10 eye pain

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and revealed normal orbital and intraocular anatomy (Fig. 1A). White blood cell count (12.9k/μL) and serum lactate were elevated (2.2 mmol/L), but the patient was afebrile (37.5C). He was diagnosed with URI and conjunctivitis, and sent home on oral azithromycin and polytrim eyedrops with instructions for outpatient follow-up with his internist.

The patient returned to the ER 30 h after initial presentation with decreased vision in the right eye, increasing eye pain with painful eye movements, and back pain. He was febrile (39.4C), tachycardic (129 beats per minute), and had an elevated white blood cell count (12.8k/μL) indicative of sepsis. Ophthalmologic examination of the right eye revealed marked lid swelling, severe conjunctival injection with chemosis, complete corneal opacification with Descemet's membrane folds, and an inferior hypopyon. Anterior and posterior segment evaluation was significantly limited by the corneal opacification. Vision was light perception only and intraocular pressure was 34 by tonopen. Exam of the left eye was unremarkable with 20/20 snellen acuity and intraocular pressure of 19 by tonopen. B-scan ultrasonography of the right eye (Fig. 2) revealed vitreous inflammatory debris as well as posterior dislocation of the crystalline lens. This latter finding was also seen on repeat head CT, which was ordered by the ER to evaluate for orbital cellulitis (Fig. 1B and C).

Given the severity of the exam and high clinical suspicion for endophthalmitis, an intravitreal tap and injection was performed with 0.1mL each of 1mg/0.1mL vancomycin, 2.25mg/0.1mL ceftazidime, and 1mg/0.1mL dexamethasone. This vitreous fluid grew out group A non-hemolytic *Streptococcus pyogenes*. Blood cultures grew the same group A *Streptococcus*. Abdominal imaging was negative, however spinal MRI revealed an L3-L4 epidural abscess presumably caused by the same bacteria. Transthoracic and transesophageal echocardiograms were negative for infective endocarditis. There were no identifiable risk factors for spontaneous lens dislocation, including connective tissue or metabolic disease. The patient completed a six-week course of intravenous penicillin and topical antibiotics and steroids, with improvement in his ocular pain. Visual acuity remained at light perception initially, but declined to no light perception at three month follow-up. Intraocular pressure declined over the first two weeks and measured zero after two months. The microbial insult and secondary inflammatory response led to extensive corneal opacification, anterior synechiae, and retinal toxicity, resulting in hypotony and early phthisical changes (Fig. 3). His ocular pain eventually recurred despite topical steroids and atropine. The patient is currently considering enucleation.

## 3. Discussion

This is an unusual case of disseminated GAS bacteremia in an immunocompetent individual. GAS may normally reside in the respiratory tract and as part of skin flora. It characteristically has a zone of beta (clear) hemolysis on blood agar due to expression of Streptolysin S exotoxin. However, non-hemolytic strains of GAS have infrequently been identified in cases of fatal septicemia,[9] severe soft tissue infections,[10] and an epidemic of pharyngitis and rheumatic fever.[11]

This patient lacked predisposing risk factors for invasive infection, including history of intravenous drug use or an indwelling catheter. Additionally, workup for infective endocarditis was negative. The primary source for his infection was not conclusively determined, but given his clinical presentation, it is likely that streptococcal infection of the pharynx preceded dissemination. This is despite a negative swab and culture in the ER. Rapid streptococcal antigen testing on pharyngeal swabs is falsely negative in up to 14% of samples,[12] and for this reason follow-up culture is routinely performed. However, pharyngeal cultures for GAS are

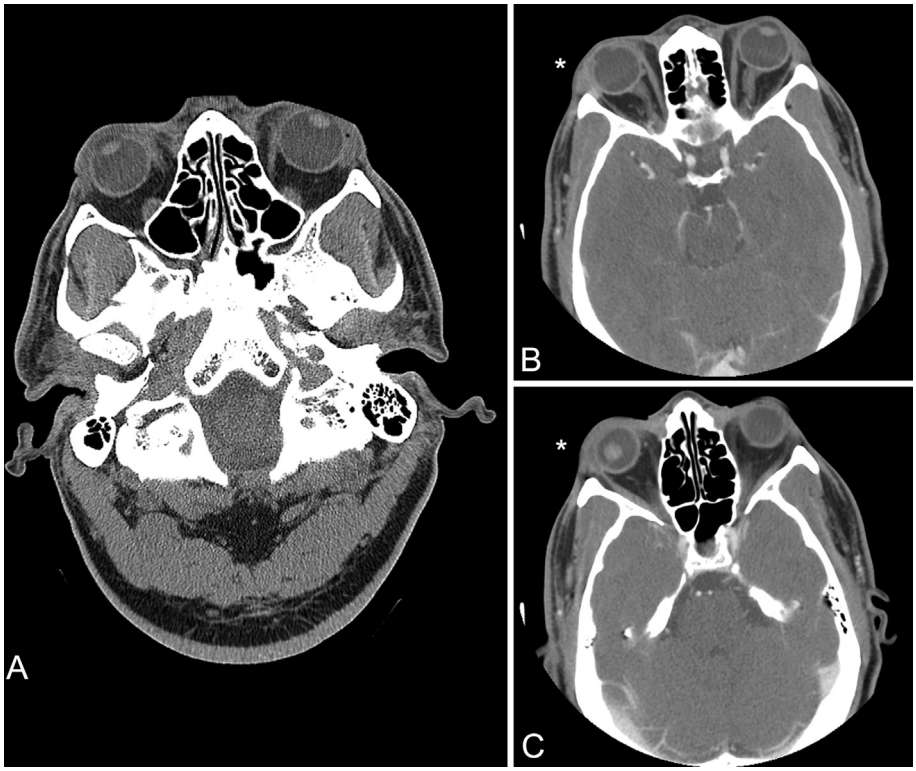


**Fig. 1.** Computed tomography (CT) head without contrast displaying crystalline lenses in physiologic location at initial presentation (a). Two days later CT (b) shows absence of right lens (asterisk) and inferior section (c) reveals posterior dislocation into the vitreous cavity (asterisk).

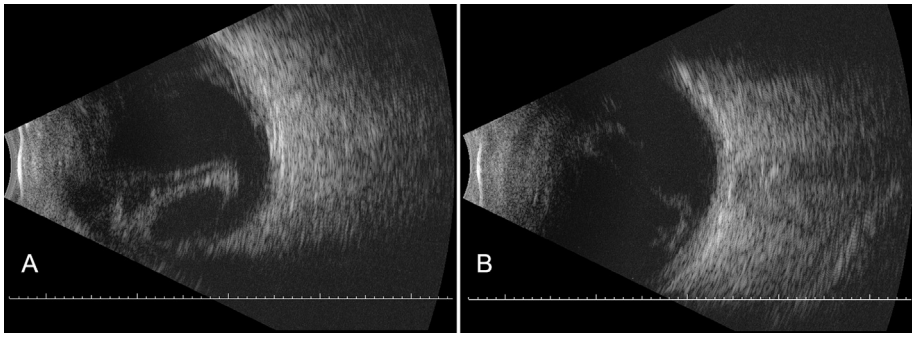


**Fig. 2.** B-scan ultrasound showing posteriorly dislocated lens (a) with sheets of vitritis (b) consistent with endophthalmitis.

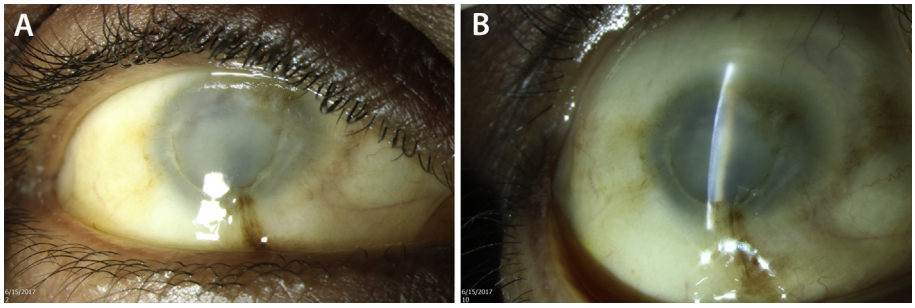


**Fig. 3.** Slit-lamp photographs of the affected eye seven months after presentation showing (a) corneal opacification and scarring of the anterior segment with (b) collapse of the anterior chamber and lens dislocation.

based on detection of beta-hemolytic colonies. Thus, our patient's pharyngeal culture was likely negative due to the atypical non-hemolytic nature of this isolate. This cannot be confirmed as the culture plate was discarded. Another possibility is that the skin served as a portal of entry with subsequent dissemination, as our patient was noted to have multiple abrasions on his limbs from occupational trauma.

Literature search reveals that GAS-mediated endophthalmitis has been sparsely reported. These cases have occurred in middle-aged females thought to be secondary to ipsilateral upper extremity erysipelas in one case,[13] after routine hysteroscopy in another,[14] and after perforated otitis media in a third.[15] Visual loss was severe and irreversible in these cases. In contrast, our patient was male and did not share any of these presenting features.

GAS is microbiologically noteworthy due to several potent virulence factors, exotoxins, and proteases that can cause a spectrum of disease and result in significant morbidity and mortality.[16] Fibrillin, a ubiquitous connective tissue glycoprotein that makes up the zonular support for the lens, could be susceptible to injury from GAS. We hypothesize that GAS-mediated protease release resulted in fibrillin degradation, leading to zonular dehiscence and subsequent lens dislocation. This finding has never been reported in the literature to the best of our knowledge.

## 4. Conclusions

GAS infection and its intraocular manifestations should be taken seriously, as they overwhelmingly cause irreversible and rapid vision loss. Unfortunately, the diagnosis of bacterial EE is frequently delayed or initially missed in 26% of cases, with a mean duration of delay of 3.2 days.[2] Such delay can lead to a grave visual prognosis. Our patient highlights the virulence of this organism in the eye, as he progressed rapidly despite prompt intraocular and systemic therapy. Ophthalmologists should be aware of this rare but devastating form of endophthalmitis.

## Patient consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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