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Case report

Severe corneal ulcer with progression to endophthalmitis and high-grade bacteremia

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ABSTRACT

Purpose: Bacterial sepsis is a common consequence of many infectious processes. Here, we describe a case of a woman with a corneal ulcer who went on to develop group B streptococcal (GBS) endophthalmitis, bacteremia, and eventual loss of the eye.

Observations: A previously healthy, immunocompetent, middle aged, contact lens wearing female who, after freshwater boating in her contact lenses, developed a red, painful eye. She was initially prescribed an hourly topical steroid by an outside optometrist but worsening of her condition prompted her to present to our Emergency Department. Despite aggressive initial management, the patient went on to develop GBS endophthalmitis, sepsis with high-grade bacteremia, and eventual loss of the eye.

Conclusions and importance: Eye care providers should exercise caution when prescribing frequent, potent corticosteroids when an infectious etiology is in the differential diagnosis.

1. Introduction

Here we present a case of a previously healthy, immunocompetent, middle aged, contact lens wearing female who developed a corneal ulcer with a ring infiltrate in the setting of hourly topical Durezol (difluprednate 0.05%, Alcon, Ft. Worth, Texas) use and went on to develop GBS endophthalmitis, bacteremia, and eventual loss of the eye.

1.1. Case report

A healthy middle-aged woman with no significant past medical history presented to the Massachusetts Eye and Ear Infirmary (MEEI) Emergency Department with a painful red right eye. Two days prior she had developed vague right eye discomfort in the setting of wearing contact lenses while motor boating on a lake. The following day, she presented to an optometrist who started hourly difluprednate and twice daily Combigan (brimonidine tartrate 0.2%/timolol maleate 0.5%, Allergan, Irvine, California) for findings of a "corneal abrasion with inflammation and elevated intraocular pressure." The next day she presented to our institution with a vision of Hand Motion OD and 20/20 OS. Intraocular pressures were 26 mm Hg OD and 10 mm Hg OS. Exam findings are shown (Fig. 1a–b); the left eye was normal. The b-scan showed mild vitreal debris. Corneal scrapings were sent to microbiology and she was placed on hourly fortified vancomycin 25 mg/ml and tobramycin 14 mg/ml drops OD, oral ciprofloxacin 500 mg bid, Combigan bid OD and cyclopentolate 1% bid OD. Daily follow up exams and b-scans over the next 3 days showed slow progression (Images from day 2 are shown in Fig. 1c–d). An intravitreal tap and inject was considered but deferred as it was felt that the vitreal debris and choroidal thickening was primarily reactionary and not due to a frank endophthalmitis. Corneal scraping from the initial presentation grew abundant mixed gram-positive cocci and gram-negative rods without a single species predominating. On day 8, she developed malaise and a fever to 103.8F. She was admitted and found to have 4/4 blood culture bottles positive for *Streptococcus agalactiae*. Her anterior segment exam was stable but the significantly worsened vitreal debris (Fig. 2a) and the overall decline in clinical status prompted a vitreous tap and inject of vancomycin 1mg/0.1, ceftazidime 2.25mg/0.1 ml, and amphotericin 5mcg/0.1 ml; her vitreous culture grew the same streptococcal species.

Under the guidance of the MEEI/MGH Infectious Disease service an extensive inpatient workup was performed, including a transthoracic and transesophageal echocardiogram, computed

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[1] All authors attest that they meet the current ICMJE criteria for Authorship.

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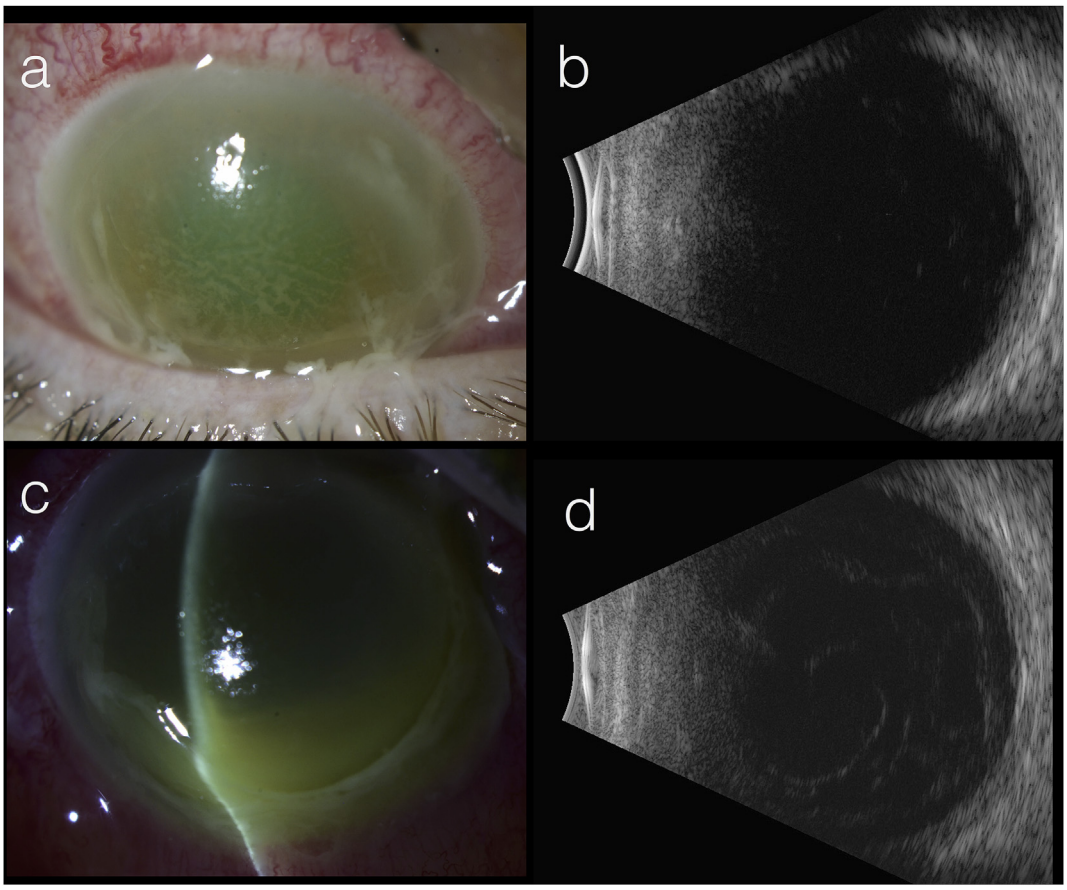

Fig. 1. (a) Near total epithelial defect with a large ring infiltrate from 1:00 to 8:00, diffuse endothelial caking with a 3 mm hypopyon (b) Mild vitreous debris and choroidal thickening (c) Mild central thinning, consolidating anterior chamber debris (d) Slight progression in vitreal debris.

tomograph (CT) of the orbits, chest, abdomen, and pelvis, colonoscopy, pelvic exam, and dental examination with orthopentogram, but no alternative sources of infection were identified except for the eye. She underwent several additional tap and injects that were sterile. Her course was further complicated by a decline in vision to No Light Perception and *Clostridium difficile* colitis. Given the blind painful eye with poor cosmesis (Fig. 2b), she underwent evisceration three months later. Histopathological findings of the eye are shown (Fig. 2c–d).

2. Discussion

Infectious keratitis progressing to endophthalmitis is an uncommon event, occurring in less than 1% of cultured corneal ulcers.[1] In the largest published series, 76% of patients had been on topical steroids before the diagnosis was made, fungus was isolated as the responsible microbe in 53% of cases, and 35% of patients progressed to NLP vision. As this case illustrates, it is important to remain vigilant to the rare possibility of a corneal ulcer progressing to endophthalmitis and if vitreal debris is worsening, to consider a tap and inject.

We are not aware of a prior report in the literature in which a corneal ulcer led to endophthalmitis and bacteremia. Although it is possible that there was a nidus of infection elsewhere in the body and the eye was merely seeded secondarily, an exhaustive search failed to reveal any such alternative sources. Since primary bacteremia without an identifiable source comprises up to 40% of invasive GBS episodes and no blood cultures were collected at the time of the patient's initial presentation, it is possible that the eye was seeded hematogenously.[2,3] However, it seems unlikely that the patient could have had a bloodstream infection, selectively seeding the anterior segment causing an epithelial defect with a ring infiltrate, and remained without constitutional symptoms or endocarditis for more than a week.

In summary, we describe a previously healthy contact lens user who presented with a corneal epithelial defect and ring infiltrate in the setting of hourly difluprednate use and then developed endophthalmitis and septicemia. Eye care providers should exercise caution when prescribing potent corticosteroids when an infectious etiology is in the differential diagnosis.

Patient consent

Consent to publish this case report was not obtained, as no personal information that could lead to the identification of the patient was used.

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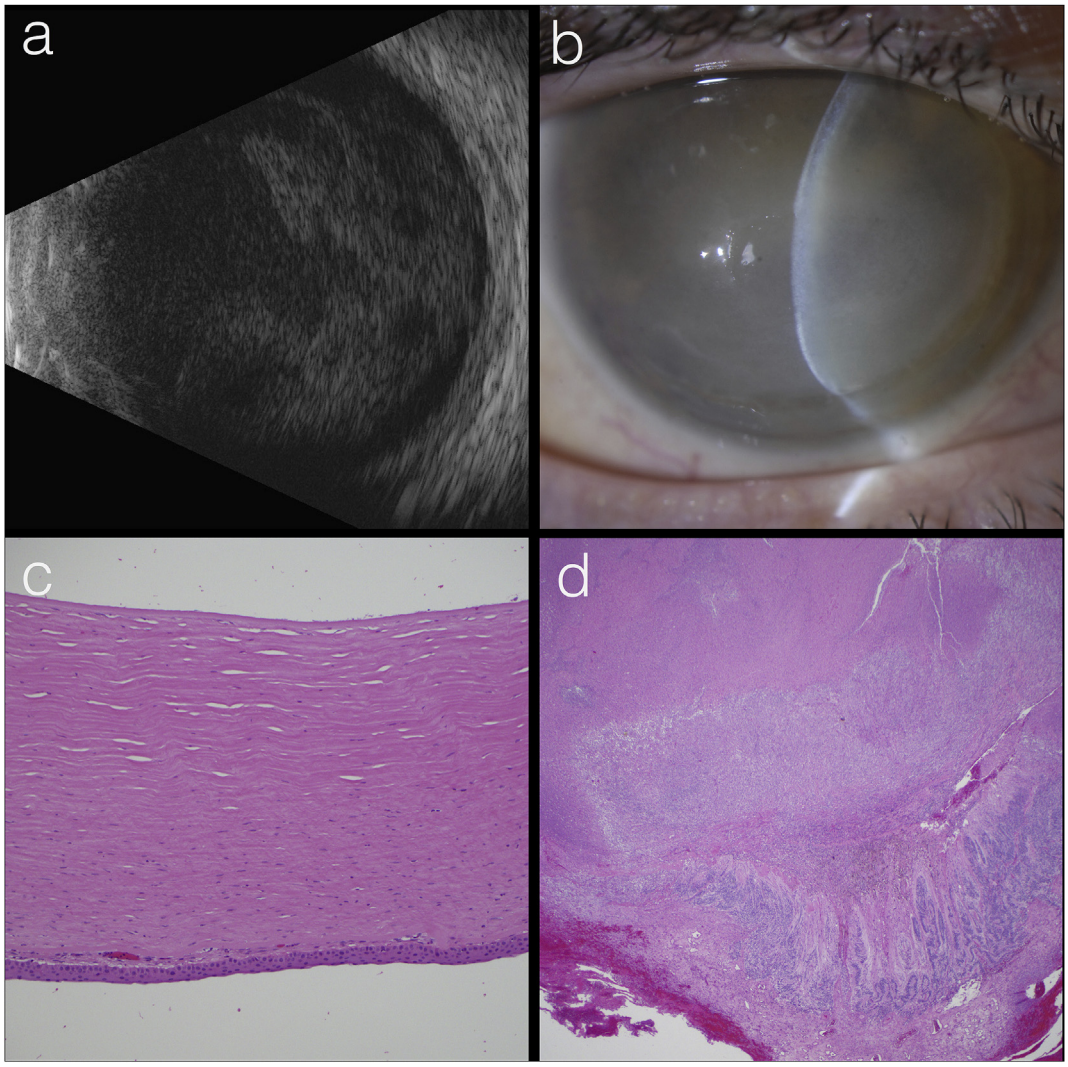

Fig. 2. (a) Progression in vitreal debris (b) White and quiet eye with corneal scarring (c) hematoxylin and eosin (H&E) micrograph (100×) showing subepithelial neovascularization and a hypercellular anterior stroma (d) H&E micrograph (20×) showing profound leukocytic infiltrate in the vitreous cavity and a severely necrotic retina.

Conflicts of interest

The authors do not have any financial interest in the subject matter of this paper.

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