
Bilateral Blindness From Orbital Cellulitis Caused by Community-Acquired Methicillin-Resistant Staphylococcus aureus

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PURPOSE: To describe bilateral blindness resulting from infection with community-acquired methicillin-resistant Staphylococcus aureus (MRSA).

DESIGN: Observational case report.

METHODS: A 44-year-old man developed proptosis, ptosis, ophthalmoplegia, and no light perception vision after attempting to lance a nasal pustule. A nasal culture grew MRSA. Imaging showed bilateral orbital cellulitis, pan-sinusitis, and cavernous sinus thrombosis. The right fundus showed severe ischemia, but the left fundus was essentially normal.

RESULTS: Despite initiation of appropriate antibiotics early in the course of infection, the patient lost sight in both eyes. Surgical drainage of the paranasal sinuses and use of intravenous corticosteroids and heparin led to the resolution of orbital cellulitis.

CONCLUSIONS: MRSA orbital cellulitis can progress to irreversible blindness despite antibiotic treatment. A new, community-acquired clone of this organism has exhibited increased potential for tissue invasion. (Am J Ophthalmol 2005;140:740–742. © 2005 by Elsevier Inc. All rights reserved.)

Accepted for publication Mar 29, 2005.
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This research was supported by an unrestricted grant from Research to Prevent Blindness, Inc., New York, New York.
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A VIRULENT ISOLATE OF METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS (MRSA) has been reported as the cause of abscesses among hospital-naïve populations, including football players, children attending daycare, and prisoners. In 2002, 928 cases of MRSA infection were reported among 165,000 inmates in the Los Angeles County Jail; 10 of 39 inmates initially hospitalized with skin infections subsequently developed bacteremia, osteomyelitis, or endocarditis. Community-acquired MRSA differs from hospital-acquired MRSA. Community-acquired strains are sensitive to multiple antibiotics outside the β-lactam class, whereas hospital-acquired strains are typically sensitive only to vancomycin and linezolid. Despite their broader antibiotic susceptibility, community-acquired strains can cause severe soft tissue infections and necrotizing pneumonia in healthy adults. We describe a case of bilateral blindness in a patient with orbital cellulitis, pansinusitis, and cavernous sinus thrombosis caused by community-acquired MRSA.

A 44-year-old previously healthy inmate developed fevers and chills 2 days after squeezing a pustule in his right naris. He had no history of HIV, diabetes, corticosteroid use, or hospitalization. Treatment was initiated with oral trimethoprim-sulfamethoxazole and rifampin. On day 4, the patient was admitted to a community hospital with swelling of the eyelids, temperature of 38.7°C, and a leukocyte count of 24×10⁹ cells/l. He received intravenous vancomycin, ceftriaxone, and metronidazole. Over the next 3 days, he developed massive proptosis, ophthalmoplegia, and complete bilateral visual loss. Orbital and brain magnetic resonance imaging revealed bilateral orbital cellulitis, pansinusitis, cavernous sinus enlargement, and tenting of the globes (Figure). A magnetic resonance venogram confirmed cavernous sinus thrombosis.

A nasal culture grew S aureus. It was resistant to oxacillin, ampicillin, cefazolin, and erythromycin; intermediate to levofloxacin; and sensitive to clindamycin, chloramphenicol, gentamicin, rifampin, trimethoprim-sulfamethoxazole, and vancomycin. This matched the sensitivity profile described for MRSA outbreaks among inmates and athletes. Blood and cerebrospinal fluid cultures were negative.

Upon transfer to our hospital’s intensive care unit on day 11, the patient had no light perception in either eye. Pupils were unreactive. Funduscopy and fluorescein angiography revealed disk edema and central retinal artery and vein occlusions in the right eye. Remarkably, the left eye had only mild congestion of the optic disk and major retinal veins (Figure). The paranasal sinuses were drained endoscopically; aerobic and anaerobic bacterial cultures were sterile, presumably because of prior treatment with antibiotics. Potassium hydroxide preparations, cultures, and tissue biopsies were negative for fungi. On day 13, treatment with heparin and intravenous methylprednisolone (250 mg every 6 hours) was started in an attempt
to salvage vision. The proptosis and orbital congestion improved, but the patient did not recover vision.

Visual loss in the right eye was due to retinal and optic nerve infarction. The mechanism of blindness in the left eye was unclear. Potential etiologies include ischemic necrosis, bacterial invasion, stretching, and compression of the retrobulbar optic nerve. The patient's illness progressed despite prompt treatment with appropriate antibiotics, including trimethoprim-sulfamethoxazole, rifampin, and vancomycin. In retrospect, the patient might have
bacteria have acquired resistance to and cavernous sinuses. Facial skin or nasal cavity to the orbits, paranasal sinuses, ty-acquired MRSA infection can spread rapidly from thewith administration of appropriate antibiotics, communi-

deterioration occur with infection caused by mucormycosis and necrotizing strains of Streptococcus pyogenes. The case described here illustrates the potentially aggressive nature of community-acquired S aureus infection. Some of these bacteria have acquired resistance to β-lactam antibiotics and carry the gene for Panton-Valentine leukocidin. Even with administration of appropriate antibiotics, community-acquired MRSA infection can spread rapidly from the facial skin or nasal cavity to the orbits, paranasal sinuses, and cavernous sinuses.

REFERENCES


Penetration of Topically Applied Fluorescein Into Eyes With Avascular Filtering Bleb After Trabeculectomy
Hiroshi Matsuo, MD, Atsuo Tomidokoro, MD, and Makoto Araie, MD

PURPOSE: To evaluate intraocular penetration of topical fluorescein in eyes with avascular blebs after trabeculectomy.

DESIGN: Case control study.

METHODS: The study included 11 eyes with open-angle glaucoma and functioning avascular blebs, six of which were treated with topical antiglaucomatous medications and had no history of surgery, 15 with open-angle glaucoma, and untreated eyes suspected of having open-angle glaucoma. The fluorescein concentration in the superior peripheral and central corneal stroma and anterior chamber was determined 30 and 60 minutes after fluorescein instillation.

RESULTS: The fluorescein concentration in the superior cornea was significantly higher in eyes with blebs or those topically treated than in untreated eyes (P <.01); there was no significant difference in the central cornea. The fluorescein concentration in the anterior chamber was much higher in eyes with blebs than in those that were untreated or topically treated (P <.001).

CONCLUSION: The presence of avascular filtering blebs greatly enhances intraocular penetration of topically instilled fluorescein. (Am J Ophthalmol 2005;140:742–744. © 2005 by Elsevier Inc. All rights reserved.)

In eyes that underwent trabeculectomy with mitomycin C or 5-fluorouracil, there is increasing concern about late-onset bleb-related complications. Though corneal damage1 and increased epithelial permeability2 in the peripheral cornea adjacent to the filtering bleb have been reported, the effect of a filtering bleb on drug penetration into the anterior chamber has never been investigated.

The study included 11 eyes (11 patients) with open-angle glaucoma and functioning avascular blebs but without topical medications, a mean ± SD of 7.8 ± 2.9 (range 4 to 12) years after trabeculectomy (six with 5-fluorouracil and five with mitomycin C ; bleb group); six eyes (six patients) with open-angle glaucoma receiving topical antiglaucomatous medications for 6 or more months and no history of ocular surgeries (topical-medication group); and 15 eyes with open-angle glaucoma or those suspected of having open-angle glaucoma who were not receiving topical medication or had a history of surgery (untreated group). Eyes were excluded if they had lacrimal, conjunc-
tival, or corneal disorders including punctuate epithelial erosions or dellen formation, or Seidel-positive aqueous leakage on the bleb wall. Eyes with functioning blebs were defined as those with a postoperative intraocular pressure constantly lower than the preoperative values by 3 mm Hg or more without medication.

Accepted for publication Apr 4, 2005.
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