**Transconjunctival Orbital Invasion by Methicillin-Resistant *Staphylococcus aureus***

Methicillin-resistant *Staphylococcus aureus* (MRSA) has been reported to cause conjunctivitis, en-dophthalmitis, keratitis following refractive surgery, corneal ulcer, wound infection after clear-cornea cataract surgery, and dacryocystitis. We report a case of community-acquired MRSA conjunctivitis that progressed to periorbital soft tissue invasion and destruction.

**Report of a Case.** A healthy 31-year-old man was referred for nonresolving conjunctivitis in the right eye. One week earlier, he developed redness and swelling of the right lower eyelid with conjunctival injection. After 4 days, he was evaluated in a local emergency department and began receiving cephalexin and gatifloxacin (Zymar) 4 times daily. The eyelid swelling partially resolved but the eye remained red, and the patient developed heavy yellow mucopurulent discharge that repeatedly accumulated in the medial canthus. He denied diplopia or epiphora. He was afebrile and felt well otherwise. He had no risk factors for immunosuppression or colonization with antibiotic-resistant bacteria.

Visual acuity without correction was 20/25 OU. Ocular motility was normal. The left eye and adnexa were normal. The right lower eyelid showed minimal edema and mild redness, which was worse nasally (Figure, A). There was +3 large vessel bulbar conjunctival injection that was greater nasally. The inferior palpebral conjunctiva was intensely inflamed and had 2 areas of ulceration down to the tarsus (Figure, B). A narrow tract of deep tissue necrosis with a blind end originated inferior to the caruncle; it probed inferonasally to a depth of approximately 7 mm. The anterior segment was otherwise normal. An orbital computed tomographic scan showed preseptal and postseptal edema with inflammatory changes in the nasal extracanal fat, without discrete abscess. Culture of the drainage grew MRSA susceptible to vancomycin hydrochloride, rifampin, and clindamycin phosphate but resistant to penicillin and cefazolin sodium. The patient was treated for 7 days with intravenous vancomycin and tobramycin sulfate as well as oral rifampin and then for 7 days with oral trimethoprim sulfa double strength with clinical resolution. Nasal cultures for MRSA were negative.

**Comment.** We report an unusual case of a healthy young patient who developed necrotizing community-acquired MRSA conjunctivitis that caused palpebral conjunctival ulceration and destruction of postseptal soft tissue with invasion of extraconal fat. Despite the depth of infection, external signs of eyelid inflammation were modest. The inferior palpebral conjunctiva was intensely inflamed and had 2 areas of ulceration down to the tarsus (Figure, B). A narrow tract of deep tissue necrosis with a blind end originated inferior to the caruncle; it probed inferonasally to a depth of approximately 7 mm. The anterior segment was otherwise normal. An orbital computed tomographic scan showed preseptal and postseptal edema with inflammatory changes in the nasal extracanal fat, without discrete abscess. Culture of the drainage grew MRSA susceptible to vancomycin hydrochloride, rifampin, and clindamycin phosphate but resistant to penicillin and cefazolin sodium. The patient was treated for 7 days with intravenous vancomycin and tobramycin sulfate as well as oral rifampin and then for 7 days with oral trimethoprim sulfa double strength with clinical resolution. Nasal cultures for MRSA were negative.

Community-acquired MRSA can produce large abscesses or carbuncles, possibly through production of a specific skin necrotic cytotoxin. Culture with sensitivity is critical as these MRSA strains are resistant to all β-lactam antibiotics and may be resistant to clindamycin. The proportion of staphylococcal ocular surface infections showing methicillin resistance has increased from 4% in 1999 to 17% in 2006. Approximately 96% of MRSA ocular isolates are resistant to ciprofloxacin hydrochloride and 82% are resistant to levofloxacin. A typical empirical treatment for moderately severe conjunctivitis with preseptal inflammation is an oral cephalosporin combined with a topical fourth-generation quinolone; this is ineffective for MRSA.

Physicians should consider MRSA in patients with atypical conjunctivitis showing patchy necrosis of conjunctiva and should investigate any dimpled or eroded areas for evidence of deeper invasion. If invasion is found, the patient should be presumptively treated for MRSA with systemic vancomycin while awaiting the results of bacterial culture. An orbital MRSA abscess within fat is relatively sequestered from antibiotic penetration, may
require surgical débridement, and could lead to extensive necrotic damage to orbital structures such as extracocular muscles and the lacrimal sac.

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COMMENTS AND OPINIONS

Potential Causes of Altered Autofluorescence in Diabetic Persons

I read with interest the article by Field et al.1 The authors ascribe autofluorescence measured using a fundus camera system to flavoprotein autofluorescence (FA), but seemingly do not account for other possible sources of the fluorescence measured or for the effect macular pigments may have on recording autofluorescence originating from the outer retina and retinal pigment epithelium (RPE).

Autofluorescence imaging with a fundus camera has the inherent difficulty of recording autofluorescence from every structure in the light path, not just from a region of interest. In addition, many fluorophores within the eye have overlapping excitation and emission spectra. For example, both the cornea and lens display autofluorescence, and the amount of autofluorescence is increased in diabetic persons compared with nondiabetic persons; for the lens, the increase is proportional to the hemoglobin A1C levels.2,3 Cataract formation is associated with increasing autofluorescence, and cataracts are more prevalent in diabetic persons.4 These autofluorescence changes occur in the wavelengths the authors used.5-7 Diabetes causes signs of oxidative stress in the retina, with increases in oxidation products. Autofluorescence from the retina is increased under oxidative stress,8 and precursors to lipofuscin form in the retina, including A2PE-H2 (dihydro-N-retinylidene-N-retinylphosphatidylethanolamine), A2PE (N-retinylidene-N-retinylphosphatidylethanolamine), and A2-rhodopsin.6,7 Fluorescence from these precursors, as well as other molecules such as advanced glycation end products (AGEs)8 and reduced nicotinamide adeninenucleotide (NADH), would be detected with the excitation and emission wavelengths used in the authors’ study.9

The authors used excitation wavelengths that show strong attenuation by macular pigment. The amount of macular pigment varies by a factor of more than 10 in the general population.10 Reduction of the amount of macular pigment present allows a larger amount of excitation light to reach deeper retinal layers and the RPE, resulting in greater autofluorescence arising from those layers. Lipofuscin in the RPE is composed of at least 10 different fluorophores, and the autofluorescence of lipofuscin consequently has a very broad range and would easily be detected within the filters used by the authors.11 (Delori et al12 found that the barrier filter used by the authors is well within the full width at half maximum of the lipofuscin spectrum.) Diabetic persons have reduced macular pigment compared with nondiabetic persons.13 Interestingly, the serum levels of circulating carotenoids are correlated with the level of diabetic retinopathy present.14 Macular pigment is negatively associated with increasing obesity15 and smoking,16 and is decreased in diseases of the macula.17-19 Macular pigment shows little, if any, correlation with patient age.20 Diabetic persons, then, have the potential to have increased autofluorescence detected by the authors’ system arising from the cornea, lens, retina, and RPE from fluorophores other than flavoproteins. The authors provided no information necessary for the reader to assess lens status, any factors related to macular pigment present, or even whether the patients had previous treatment. As such, it is difficult to know what proportion of the autofluorescence measured actually arose from flavoproteins. The authors have not shown scientific evidence to justify the assumption that the entirety, or even most, of the signal they measured arises from flavoproteins.

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6. Liu J, Iyagi Y, Ben-Shabat S, Nakahisa K, Sparrow JR. The biosynthesis of A2E, a fluorophore of aging retina, involves the formation of the precursor,