current lesions of hard consistency in the mucous membranes, which have a negative effect on homeostatic fibrinolysis because of mutations in the PLG gene. This disorder is usually detected in young children with recurrent conjunctivitis due to pseudomembranes that evolve to hard nodular masses of woodlike consistency. Ligneous conjunctivitis has an autosomal recessive pattern of transmission and several mutations in the PLG locus have been demonstrated in patients with this disorder. A mutation screening performed on the patient and her parents demonstrated a nonreported, homozygous, 14-base pair deletion in exon 5 of the PLG gene, which generates an early stop in the 5′ region of the gene. One recent report suggests a possible “hot spot” region around the Cys133-Cys157 in the PLG gene. We hypothesize that this homozygous mutation results in the synthesis of a truncated plasminogen molecule in the liver that is quickly degraded, a finding that correlates with the early onset and the severity of the eye abnormalities. Despite this physiologic defect, it is noticeable that the patient does not show involvement in other organs besides the eyes.

Melioidosis With Endophthalmitis

Melioidosis is an infectious disease caused by Burkholderia pseudomallei, a Gram-negative bacillus. It is endemic to southeast Asia and northern Australia as well as regions between 20° latitude north and south of the equator. The clinical manifestation varies from a latent infection with an incubation period of up to 29 years to fulminant sepsis with a high mortality rate. We report a case of endogenous endophthalmitis caused by B pseudomallei with a fulminant course.

Report of a Case. A 70-year-old Chinese male veteran living in southern Taiwan had a medical history of diabetes mellitus for more than 10 years and coronary artery disease after bypass graft surgery 2 years previously. Ocular history included herpetic keratouveitis in the right eye 4 years previously with residual corneal opacity, bilateral senile cataract, and bilateral nonproliferative diabetic retinopathy. Ocular trauma and operation history were not reported.

At initial examination on August 5, 2005, the patient had pain in the right eye and headache that had persisted for 1 day. Visual acuity of the right eye was decreased to light perception. Marked chemosis, corneal edema, hyphema, and elevated intraocular pressure were found. He was diagnosed with and treated for neovascular glaucoma. On day 11, hypopyon and a localized scleral suppuration, which disseminated over the following days (Figure 1), were noted. The vitreous echoes were heterogeneous with a fluffy retinal surface. Vitreous fluid was aspirated for smear and culture. Gram-staining smear disclosed numerous Gram-negative rods (Figure 2). The diagnosis of endogenous endophthalmitis was made. After immediate intravitreal injection of 1 mg of vancomycin hydrochloride, 0.4 mg of amikacin sulfate, and 0.4 mg of dexamethasone sodium phosphate, topical eyedrops consisting of vancomycin hydrochloride (50 mg/mL) and amikacin sulfate (25 mg/mL) were administered in combination with 4 g/d of intravenous ceftriaxone sodium. On day 16, both vitreous and blood cultures yielded B pseudomallei. Systemic cefazidine, cotrimoxazole, and granulocyte colony-stimulating factor were administered. However, the illness progressed to septic shock and multiple organ failure, and the patient died on day 18. Systemic survey

Figure 1. The right eye on day 18 showing disseminated multifocal scleral suppuration.

showed no definite focus of infection source.

Comment. Although an indigenous melioidosis outbreak occurred in late July 2005 shortly after flooding in southern Taiwan following Typhoon Haitang, the route of *B. pseudomallei* entry in this patient is uncertain because there was no wound in the skin or ocular surface and no apparent contact with contaminated soil and water, which is the most common way of acquisition in humans. Recrudescence of acute melioidosis from its latency in patients with decreased immunocompetence such as diabetes mellitus, the major risk factor for melioidosis, is an alternative explanation for this patient.

Ocular melioidosis is rarely described in the literature. To our knowledge, this is the first reported case of endogenous endophthalmitis with septicemic melioidosis. The initial appearance of hyphema with glaucoma could be due to a process of hemorrhagic necrosis in the anterior segment. The clinical findings of initial chemosis and subsequent scleral suppuration resemble the clinical evolutions in reported cases of corneal ulcer caused by *B. pseudomallei* infections, and these may be regarded as clinical indicators of ocular infection with this organism. Given the possible fulminant course and high mortality rate, ophthalmologists should remain ever vigilant for the disease. Systemic ceftazidime is the standard treatment for melioidosis in the acute phase. Intravitreal ceftazidime at an appropriate dosage may be required for cases of *B. pseudomallei* endophthalmitis.

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