Figure 1. A. Central cotton wool spots and beginning peripheral progressive outer retinal necrosis in an HIV-infected patient before treatment with cidofovir. B. After treatment with cidofovir, active inflammation ceased, central ischemia disappeared, and there is retinal scarring at the sides of the previous necrosis.

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Staphylococcus saprophyticus as an Unusual Agent of Nosocomial Pneumonia

Staphylococcus saprophyticus is a well-known and frequent etiologic agent of urinary tract infections [1]. It causes infections in young women, elderly men, and children [2, 3]. Rare cases of sepsis following pyelonephritis have been documented [4]. It is described as a bacterium of zoonotic origin, which has been isolated from pigs, cattle, goats, and other animals [5, 6]. Recently, we cloned and characterized an adhesive and autolytic surface protein of S. saprophyticus (AAS), which is a major adhesion factor for the organism [7]. Herein, we report a case of S. saprophyticus pneumonia after neurosurgical intervention. To our knowledge, no cases of nosocomial infection by S. saprophyticus have been reported, nor has S. saprophyticus been described yet as a pulmonary pathogen.

A 61-year-old man presented with intracerebellar hemorrhage caused by a hypertensive crisis during anticoagulative therapy (international normalized ratio, 3.55; prothrombin time, 17%). The patient underwent emergency craniotomy and was extubated soon after neurosurgical intervention. Three days later, his pulmonary condition deteriorated, and mechanical ventilation had to be used again. Seven days after craniotomy, he had mild hyperthermia (temperature, 38.3°C) and purulent bronchial secretion. One day later, the inflammatory parameters (C-reactive protein level and leukocytic elastase level) were elevated. A chest roentgenogram showed a new infiltrate in the left lower lobe. The patient was given intravenous therapy with ceftazidime (2 g q12h); the patient had no signs of urinary tract infection.

Bronchoalveolar lavage (BAL) was performed, and quantitative bacteriologic culture of the BAL fluid was done. Gram staining of the BAL sediment showed high counts of gram-positive cocci and

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Three days later, the patient's condition improved clinically, and the inflammatory parameters declined 10 days after treatment with penicillin G was started. At that time, the radiographic signs had nearly disappeared.

Coagulase-negative staphylococci are not normally considered to be pulmonary pathogens [8]. S. saprophyticus has never been described as an etiologic agent of pneumonia. In this case, no underlying immunodeficiency was known, but we believe that this intensive care unit patient's history characterized by prolonged mechanical ventilation (possibly because of intracerebellar hemorrhage) predisposed him to nosocomial pneumonia. We are well aware that it is important to interpret our findings of S. saprophyticus as a pulmonary isolate cautiously. However, we think that the result of quantitative culture of BAL fluid and the prompt improvement during penicillin G therapy corroborate S. saprophyticus as the infectious agent. Therefore, S. saprophyticus seems to have the potential to cause pulmonary infections.

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References

Failure of a Lipid Amphotericin B Preparation to Eradicate Candiduria: Preliminary Findings Based on Three Cases

For >30 years, amphotericin B deoxycholate has been the drug of choice for many serious fungal infections. Its use, however, is limited by a low therapeutic index and frequent adverse effects. The search for a less toxic but equally efficacious antifungal agent led to the development of lipid carrier systems for amphotericin B that limit the availability of free amphotericin B. Amphotericin B lipid complex (ABLC; Abelcet, Liposome Company, Princeton, NJ), one of three U.S. Food and Drug Administration–approved lipid amphotericin B preparations, consists of amphotericin B complexed with two phospholipids in a 1:1 drug-to-lipid molar ratio.

We describe three critically ill patients with impaired renal function who had candiduria despite receiving empirical treatment with ABLC for fevers (table 1). The patients were all being treated for neoplastic diseases; none of the patients was neutropenic at the

Figure 1. Quantitative culture of bronchoalveolar lavage fluid from a patient with nosocomial pneumonia; the fluid was plated on Columbia agar, and >10⁵ cfu of Staphylococcus saprophyticus/mL are shown.