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...
Table 1. Summary of data for three patients with candiduria despite treatment with ABLC.

<table>
<thead>
<tr>
<th>Finding</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underlying disease</td>
<td>Gastric cancer</td>
<td>Acute lymphocytic leukemia</td>
<td>Cholangiocarcinoma</td>
</tr>
<tr>
<td>Foley catheter</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>BUN level (mg/dL):creatinine level (mg/dL) at initiation of ABLC treatment</td>
<td>94/2.5, nonoliguric</td>
<td>66/1.6, nonoliguric</td>
<td>56/2.2, nonoliguric</td>
</tr>
<tr>
<td>Hemodialysis</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Candida isolate</td>
<td>Candida glabrata</td>
<td>Candida albicans</td>
<td>No</td>
</tr>
<tr>
<td>ABLC dosage (mg/[kg · d])</td>
<td>2.5</td>
<td>5</td>
<td>Candida species</td>
</tr>
<tr>
<td>Susceptibility to amphotericin B (MIC)</td>
<td>Yes (0.12 μg/mL)</td>
<td>Yes (0.5 μg/mL)</td>
<td>2.5</td>
</tr>
<tr>
<td>Evidence for persistent candiduria</td>
<td>Yeast by UA on days 11 and 15 and positive cultures on days 2, 5, 8, 12, and 17 of ABLC therapy</td>
<td>Yeast by UA and culture on day 17 of ABLC therapy</td>
<td>Yeast by UA on days 12 and 23 of ABLC therapy</td>
</tr>
<tr>
<td>Outcome</td>
<td>Alive</td>
<td>Died</td>
<td>Died</td>
</tr>
</tbody>
</table>

NOTE. ABLC = amphotericin B lipid complex; BUN = blood urea nitrogen; ND = not determined; UA = urinalysis.

start of ABLC therapy. On the basis of results of imaging studies, none of the patients had structural urinary tract abnormalities. Isolates from two patients who had received ≥2 weeks of ABLC therapy at the time of the positive culture did not reveal evidence of amphotericin B resistance (MIC, ≤0.5 μg/mL; Mycology Laboratory, Wadsworth Center, New York State Department of Health, Albany, NY).

Our results contrast with the well-documented efficacy of amphotericin B for treatment of candiduria. In a study by Leu and Huang [1], even a single intravenous dose of 15 mg of amphotericin B deoxycholate was highly successful therapy for candiduria. Insufficient urinary levels of amphotericin B in patients receiving the ABLC formulation may explain our observations. The relatively large-sized particles of the lipid amphotericin B preparations [2] may not be as readily filtered by the kidney, with preferential uptake of these particles by the reticuloendothelial system. No reported studies exist on the urinary levels of amphotericin B in patients receiving ABLC treatment.

Our findings, while anecdotal, suggest that certain lipid prepa-
rations of amphotericin B may not be efficacious in treating candiduria, at least in patients with renal insufficiency. Additional pharmacokinetic and clinical studies with this class of drugs are warranted.

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References

Serum Levels of Nitrite and Nitrate in Patients with Systemic Inflammatory Response Syndrome

Systemic inflammatory response syndrome (SIRS) is an inflammatory process independent of its cause [1]. SIRS is seen in association with a large number of clinical infective and non-infective conditions. Nitrosoyl (NO), a potent vasodilator and cytotoxic molecule, has emerged as a candidate for the final common mediator leading to vasodilatation in shock [2]. Increased plasma levels of the stable by-products of NO, nitrite and nitrate, have been described in patients with sepsis and septic shock [3]. In contrast, several studies have demonstrated that endothelium-derived production of NO is significantly reduced during endotoxemia [4]. In this study, we determined serum levels of NO, detected by its end-stable products nitrate and nitrate, in patients with infectious and noninfectious SIRS. We evaluated 45 patients (mean age ± SD, 53.9 ± 21.0 years; 26 males and 19 females) negative for HIV type 1 infection who met clinical criteria for SIRS [1]. Twenty healthy subjects (mean age ± SD, 42.1 ± 4.7 years) served as controls. Thirty-eight patients (mean age ± SD, 52.5 ± 21.5 years) had infective SIRS caused by gram-positive bacteria (7 patients), gram-negative bacteria (6), several organisms (4), protozoa and mycobacteria (5), and an unknown etiology (16). NO levels were

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