Community-Acquired Fungemia Due to a Multiple-Azole-Resistant Strain of Candida tropicalis

Candida tropicalis is one of the more common causes of candidemia [1]. Recent reports describing clinical and in vitro resistance of Candida species associated with azole therapy have been of concern [2–4]. Azole-resistant Candida isolates have been identified, particularly in patients with advanced HIV disease. Resistance to fluconazole is infrequently described with regard to immunocompetent individuals [5]. Despite the increase in frequency of isolation, persistent candidemia due to azole-resistant C. tropicalis has not been previously reported. We report a patient from the community with C. tropicalis fungemia who remained candidemic despite high-dose fluconazole therapy.

A 46-year-old man with diabetes mellitus was admitted after being found unresponsive. His history included use of alcohol, injection drugs, and cigarettes. On admission he was unresponsive, with a blood pressure of 70 mm Hg over palpable, a pulse rate of 50/min, an oral temperature of 37.3°C, and a respiratory rate of 30 breaths/min. The remainder of his physical examination yielded nothing remarkable.

Laboratory values on admission included the following: serum glucose, 1215 mg/dL; bicarbonate, 4.1 mmol; and arterial pH, 6.99. After treatment with iv hydration and an insulin drip, he became more alert. Within 24 h his blood pressure normalized and his acidosis resolved. One day after admission a blood culture was reported positive for yeast, and fluconazole therapy at a dose of 400 mg/d was initiated. On day 3 the yeast was identified as C. tropicalis. In addition, a urine culture from the day of admission demonstrated the presence of C. tropicalis (>100,000 cfu/mL). Blood cultures performed on days 4, 8, 11, and 13 remained positive for C. tropicalis.

On day 13 the antifungal regimen was changed to administration of amphotericin B lipid complex at a dosage of 300 mg/d iv. An extensive evaluation was performed, and none of the studies demonstrated evidence of disseminated candidiasis. On day 28 the amphotericin was withdrawn and substituted with oral itraconazole (200 mg once daily for an additional 2 weeks). The patient recovered with no further medical problems and was discharged to home.

The identity of the C. tropicalis was established by means of the Yeast API 20C method (Analytab Products, Plainview, NY). In vitro susceptibilities were determined with use of the methodology of the National Committee for Clinical Laboratory Standards [6]. Strain delineation of the C. tropicalis isolates was established by electrophoretic karyotyping, which was performed according to the published method for contour-clamped homogeneous electric fields electrophoresis (CHEF) [7]. The results of in vitro susceptibility analysis of the isolates were identical and demonstrated in vitro cross-resistance (table 1). Karyotyping revealed that the 5 C. tropicalis isolates were of the same strain. The ATCC isolate, however, was of a different strain type.

To our knowledge, azole-resistant C. tropicalis has not been previously reported as a cause of community-acquired candidemia. There is, however, 1 report of a patient with leukemia who developed fungemia with C. tropicalis while receiving fluconazole. Afterward, the isolate was found to be resistant to fluconazole [8]. Reports of studies that have evaluated the resistance of bloodstream isolates of C. tropicalis to fluconazole either focus on nosocomial infections or do not describe the origin of the isolates.

There are conflicting reports in the literature regarding the prevalence of resistance to C. tropicalis. A large multinational observational study that included the United States, Canada, and South America revealed that 99.4%–100% of the C. tropicalis isolates were susceptible to fluconazole [9].

One possible explanation for resistance is that a susceptible strain might mutate and become resistant during therapy [10]. The patient described in this report presented to the hospital from the community with infection due to a strain of C. tropicalis.

Table 1. Results of in vitro susceptibility testing of Candida tropicalis isolates from a man with persistent community-acquired candidemia.

<table>
<thead>
<tr>
<th>Isolate no. (hospital day)</th>
<th>MIC, µg/mL</th>
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<tbody>
<tr>
<td>Clotrimazole</td>
<td>Itraconazole</td>
</tr>
<tr>
<td>1 (1)</td>
<td>4</td>
</tr>
<tr>
<td>2 (3)</td>
<td>4</td>
</tr>
<tr>
<td>3 (8)</td>
<td>4</td>
</tr>
<tr>
<td>4 (11)</td>
<td>4</td>
</tr>
<tr>
<td>5 (13)</td>
<td>4</td>
</tr>
<tr>
<td>ATCC 44508</td>
<td>0.03</td>
</tr>
</tbody>
</table>

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Clinical Infectious Diseases 1999;29:1583–4
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Detection of Human Herpesvirus 8 in Cervical Cells of Chinese Women with Abnormal Papanicolaou Smears

Human herpesvirus 8 (HHV-8), also known as Kaposi’s sarcoma (KS)–associated herpesvirus, is the first member of the genus *Rhadinovirus* known to infect humans [1]. HHV-8 has been strongly associated with KS in both immunocompromised and immunocompetent patients. HHV-8 has also been detected in high percentages of primary effusion lymphomas, cases of multicentric Castleman’s disease, and bone marrow dendritic cells of patients with myeloma. There is controversy about whether HHV-8 is ubiquitous and how the virus is transmitted in the healthy population. Reports on the frequency of virus shedding in the genital tract are being disputed, and the role of heterosexual transmission is also uncertain.

We have examined the presence of HHV-8 DNA in cervical scrapes from 404 consecutive Chinese women referred to a colposcopy clinic because of abnormal Papanicolaou smears. These patients were aged 16–88 years (mean, 40.6 years; SD, 11.7). None were commercial sex workers, 25% reported regular use of barrier contraception, 16% reported having >1 sexual partner, and 2% recalled having had sexually transmitted disease(s). None of the women were pregnant or had a history of cervical premalignant/malignant disease.

Colposcopy was performed for all the patients, and biopsies

References