BRIEF OBSERVATIONS

Fungemia with Saccharomyces cerevisiae after Treatment with Saccharomyces boulardii

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Usually considered a nonpathogenic yeast, Saccharomyces boulardii has been used to prevent antibacterial-associated diarrhea and to treat recurrent Clostridium difficile colitis and other diarrheal illnesses (1). It has been available in Europe in standardized lyophilised form since 1962. S boulardii is registered under the name S cerevisiae Hansen CBS 5926, but the manufacturer states that S boulardii is not the same as baker’s yeast (S cerevisiae) (2). We present a patient with S cerevisiae fungemia after treatment with S boulardii.

CASE REPORT

A 51-year-old woman was transferred with fever, bilateral knee arthritis, livedo reticularis, Raynaud’s phenomenon, and renal failure. Biopsy showed leukocytoclastic vasculitis. A diagnosis of polyarteritis nodosa was made, and immunosuppressive treatment with steroids (starting with 1 g intravenous methylprednisolone and tapering to 7.5 mg oral prednisone daily) and cyclophosphamide was begun. During the following weeks, the patient was treated with several antimicrobials agents, including amoxicillin and metronidazole for eradication of Helicobacter pylori, amoxicillin/clavulanic acid for fever of uncertain origin, cotrimoxazole followed by trimethoprim/dapsone and then atovaquone for Pneumocystis carinii pneumonia, ganciclovir for cytomegalovirus disease, and clarithromycin for erysipelas. An episode of toxin-positive C difficile colitis was treated with oral metronidazole (500 mg 3 times a day) for 10 days. A toxin-positive relapse was treated with the same dose of metronidazole and oral S boulardii (Perenterol 500 mg twice a day). At that time, the patient had a central venous line. After a 18-day course of therapy, the patient again became febrile (39°C), and blood cultures yielded S cerevisiae. Amphotericin B therapy was started, and the fever resolved within 24 hours.

The biochemical characteristics of the yeast isolated from the blood and those of yeast cultured from Per-enterol capsules were identical, and matched those for S cerevisiae according to a commercial identification system (ID32C, bioMérieux, Marcy-l’Etoile, France; Auxac-dor, Diagnostics Pasteur, Marnes-la-Coquette, France) and to the microbiology manual (3). Further studies with pulsed field gel electrophoresis showed identical pattern of both isolates with different enzymes whereas 12 unrelated S cerevisiae control strains were different (Figure).

DISCUSSION

Our patient had fungemia with S boulardii as an adverse consequence of treatment with this yeast. S boulardii and S cerevisiae can not be differentiated as, according to biochemical and molecular biologic studies, both strains are identical.

S cerevisiae is an important industrial yeast used in baking and brewing. It is a common colonizer of human mucosal surfaces. Although this fungus is generally believed to be nonpathogenic, there have been reports of infections with S cerevisiae, including fungemia, endocarditis, pneumonia, liver abscess, peritonitis, vaginitis, and urinary tract infections in elderly or immunocompro-
REFERENCES


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Mised patients (4). S boulardii (S cerevisiae Hansen CBS 5926) is the only yeast that has been tested in double-blind studies (1). The manufacturer claims that S boulardii is not identical to S cerevisiae (2). However, some authors consider S cerevisiae to be synonymous with S boulardii, and have successfully treated patients with baker’s yeast for C difficile diarrhea (5).

Treatment with S boulardii has previously been shown to cause invasive fungal disease in 7 patients (6–10). All of these cases occurred in debilitated or immunosuppressed patients who had central venous lines. All patients, like ours, recovered with systemic antifungal treatment.

CASE REPORT

A 45-year-old previously healthy Japanese woman was hospitalized with a 1-week history of high-grade fever, nonproductive cough, and dyspnea. Velcro rales were audible over both lungs. There were no skin rashes, subcutaneous nodules, palpable hepatosplenomegaly, lymphadenopathy, or enlargement of the thyroid gland. Chest roentgenograms revealed a reticulonodular infiltrate in the bilateral lower lobes and confluent opacities in both lung bases (Figure, panel A). Computed tomogram of the chest showed a massive parenchymal infiltrate in the lower lobes (Figure, panel B). Analysis of arterial blood showed that the partial pressure of oxygen was 54 mm Hg, partial pressure of carbon dioxide was 47 mm Hg. The white cell count was 5,900/μL, and the serum C-reactive protein level was 2.6 mg/dL. The values for urea nitrogen, creatinine, calcium, phosphorus, electrolytes, aspartate aminotransferase, and alanine aminotransferase were normal. The serum total protein level was 9.6 g/dL and M-peak was recognized on electrophoresis. Serum levels of immunoglobulins were as follows: IgG 1,268 mg/dL, IgA 3,115 mg/dL, IgM 82 mg/dL, IgE 15.5 U/mL.
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