

Intravenous Catheter-Associated Fungemia Due to *Candida rugosa*

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We report a case of intravenous catheter-associated fungemia caused by *Candida rugosa*; this is the first report of such an infection in a human. Multiple cultures of blood taken over a 24-h period and of the intravenous catheter tip were positive for this unusual isolate. The patient was treated with intravenous amphotericin B and made an uneventful recovery. Intravenous cannulae and other intravascular devices are well recognized as potential sites of intravascular infection by a variety of microorganisms, including several *Candida* species; however, fungemia caused by *C. rugosa* has not been reported.

Fungemia is an infection that is usually nosocomial and that often develops in the setting of antimicrobial therapy, cancer chemotherapy, bowel surgery, or the use of intravenous plastic catheters. The majority of isolates are *Candida* spp., particularly *Candida albicans* and *C. tropicalis* (1, 4, 5, 9); although *C. rugosa* has been isolated from humans on rare occasions (2, 8), to our knowledge it has not been documented to cause severe disease in humans. We report the case of a patient who had *C. rugosa* fungemia related to the presence of an intravenous catheter.

A 54-year-old male was found comatose and was admitted to a local hospital on 26 August 1984. His past medical history included ethanol abuse and a surgically created portocaval shunt for management of portal hypertension. Computerized tomography of the head at the time of this admission revealed acute and chronic bilateral subdural hematomas, which were evacuated surgically. The hospital course was complicated by the development of aspiration pneumonia, for which the patient received a 2-week course of intravenous carbenicillin and amikacin. At the end of week 2 of hospitalization, a tracheostomy and a cut-down procedure over the left ankle for placement of an intravenous catheter were performed.

The patient was transferred to our hospital on 18 September 1984. Significant findings at that time were a temperature of 100°F (ca. 37.8°C), a grade II/VI holosystolic murmur with radiation to the axilla, and decubitus ulcers over the sacrum and both hips. A chest roentgenogram revealed a left-lower-lobe infiltrate, and sputum cultures grew *Citrobacter diversus* and *Pseudomonas aeruginosa*, for which carbenicillin and amikacin therapy was given for 9 days.

On 29 September 1984, the patient became febrile to 104°F (40°C); an examination revealed erythema, warmth, and tenderness over the area of the left ankle cut-down site. The leukocyte count was 3,600/mm³, with a differential of 72% polymorphonuclear leukocytes, 1% band forms, 16% lymphocytes, 10% monocytes, and 1% metamyelocytes. Three sets of blood cultures (brucella broth [GIBCO Laboratories, Chagrin Falls, Ohio] and Columbia broth [Difco Laboratories, Detroit, Mich.]) were done over a 24-h period. Empiric therapy with carbenicillin and gentamicin was instituted. A

two-dimensional echocardiogram did not reveal valvular vegetations; sonography of the abdomen and a radionuclide technetium liver-spleen scan were unremarkable.

On 1 October 1984, the vented bottles (Columbia broth) of two sets of blood cultures done 2 days previously were turbid; Gram staining of the broth revealed yeast cells with blastoconidia. The intravenous catheter was removed, and the tip was cultured semiquantitatively (6). On 3 October 1984, yeast cells with blastoconidia were growing in the remaining four blood culture bottles, and the culture of the catheter tip grew *C. rugosa*. Therapy with intravenous amphotericin B was begun, and the patient improved rapidly. Repeat blood cultures during therapy were sterile. Blood cultures at 2, 3, and 5 months after the completion of a course of amphotericin B (total dose, 500 mg) were also sterile.

The blood isolate was referred to the Fungus Reference Unit at the Centers for Disease Control, Atlanta, Ga., and was found to have the physiological and morphological characteristics of *C. rugosa*. The isolate was white to light tan on brain heart infusion and Sabouraud agars after 3 days of incubation at 30°C, and was negative for germ tube formation. The isolate produced typical blastoconidia and hyphae on cornmeal-Tween agar when inoculated by the Dalmau method. The physiological profile was identical to that of *C. rugosa* given by Van Uden and Buckley (12) and by Cooper and Silva-Hutner (3). The yeast was initially identified as *C. rugosa* with the API 20C yeast identification system.

Susceptibility testing of one of the isolates by a reference laboratory revealed that growth was inhibited by 0.156 µg of amphotericin B per ml, 0.39 µg of 5-fluorocytosine per ml, and 0.4 µg of ketoconazole per ml.

Persistent and sometimes fatal episodes of candidemia are often related to the presence of an intravascular catheter. One investigator noted that, of 69 patients with candidemia, 55 had infections that were associated with the presence of an intravenous catheter (11).

C. rugosa was originally isolated from human feces in 1917 by Anderson and called *Mycoderma rugosa* (12). It has subsequently been isolated from bovine droppings, stale butter, a margarine factory, and seawater (12). In a study of bovine mastitis in New York and Iowa, *C. rugosa* was the second most frequently isolated yeast (10). The organism has rarely been recovered from clinical samples from humans,

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however, and to our knowledge it has never been reported to cause fungemia in humans. The source of infection in our patient was obscure.

Our isolates of *C. rugosa* were accurately identified initially by the API 20C yeast identification system and then confirmed by the Wickerham assimilation and fermentation procedures at the Centers for Disease Control. Our isolates of *C. rugosa* were susceptible to all three antimycotic agents evaluated; in a study of the in vitro susceptibility of 18 species of yeasts isolated from infected bovine mammary glands, however, *C. rugosa* was the least susceptible of all the species evaluated (10). None of 13 *C. rugosa* isolates was susceptible to a 9.6- μ g amphotericin B disk or to a 45- μ g 5-fluorocytosine disk, and only 23% were inhibited by a 4.8- μ g miconazole disk; 100% were inhibited by a 1.0- μ g ketoconazole disk (7).

In summary, *C. rugosa* should be added to the list of opportunistic pathogens that may be associated with intravascular-device infections.

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