Candida pseudotropicalis Fungemia and Invasive Disease in an Immunocompromised Patient

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A case of *Candida pseudotropicalis* fungemia and invasive disease in an immunocompromised patient is reported. Multiple blood cultures taken over a 2-week period were positive, and histopathological slides of postmortem spleen and kidney tissue showed tissue invasion by the organism. The source of the yeast infection was determined to be the urinary tract. This is the first report of *C. pseudotropicalis* fungemia documented by culture.

Fungemia and invasive disease caused by *Candida pseudotropicalis* are extremely rare. Four reports of deep or systemic disease caused by this yeast have appeared in the literature to date; however, these cases were detected postmortem, and blood cultures were not attempted (2, 8-10, 13). The present case is significant because it emphasizes the potential of this yeast to act as an opportunistic pathogen and because it provides what appears to be the first report of recovery of *C. pseudotropicalis* from blood.

Case report. A 58-year-old woman was hospitalized on 27 December 1978 after she had experienced hematuria for 1.5 weeks. She had a history of adenocarcinoma of the breast and had undergone bilateral mastectomy, radiotherapy, and chemotherapy in 1969. In 1975 she developed a pelvic mass which included the bladder and was proven to be metastatic adenocarcinoma. She received radiation and chemotherapy, which resulted in the apparent resolution of the pelvic mass.

On this admission, laboratory studies disclosed that she had a hemoglobin level of 6.4 g/dl, leukocyte count of 5,200/mm³, platelet count of 58,000/mm³, and changes in intravascular coagulation and fibrinolysis. A urine culture showed *Escherichia coli* (>10⁵ bacteria per ml) and *Klebsiella pneumoniae* (10⁴ to 10⁵ bacteria per ml), for which a 10-day course of oral ampicillin (250 mg four times daily) was given. During the time that the patient was hospitalized, she received several units of packed erythrocytes and several units of platelets for anemia secondary to hematuria and for thrombocytopenia of unknown etiology. Although the hematological values fluctuated, the leukocyte count never dropped below 1,500/mm³.

We believed that the hematuria was due to invasion of the bladder wall by a metastatic tumor. Treatment included radiotherapy for a total of 15 treatments throughout January 1979, prednisone (40 mg/day), and one dose of doxorubicin hydrochloride (Adriamycin) (20 mg/ml) weekly. An indwelling urethral catheter was occasionally used during the patient's hospitalization. A midstream urine culture taken on 28 January 1979 grew *C. pseudotropicalis* (colony count of 10⁴ to 10⁵ yeasts per ml). This yeast was also recovered from urine that was cultured on 4 and 5 February (colony count of 10⁴ to 10⁵ yeasts per ml). The patient became febrile on 4 February (100.5°F [ca. 38.0°C]), and cultures of blood and a total parenteral nutrition line were negative for bacteria and fungi. The patient remained febrile for the rest of her

hospitalization, with daily temperature spikes to 103.6°F (ca. 39.8°C).

A cystectomy with right ureteral diversion and ileal conduit construction was performed on 7 February 1979. Histopathological examination of bladder tissue confirmed the presence of a metastatic tumor. Two days after surgery, the patient developed difficulty in breathing, and her respiratory status continued to decline over the next 5 days. Chest roentgenograms showed increasing interstitial pulmonary infiltrates in spite of empirical treatment with cephalothin. On 13 February the laboratory reported that *C. pseudotropicalis* had been recovered from the ureteral fluid cultured during surgery and from blood cultured on 8 February 1979. Amphotericin B therapy was initiated, but blood cultures remained positive for *C. pseudotropicalis* until the death of the patient on 19 February 1979. Death was attributed to fungemia, respiratory failure, and renal and hepatic insufficiency.

Postmortem examination demonstrated widespread adenocarcinoma involving the vertebrae, ribs, uterus, cervix, ovaries, soft tissue of the pelvis, stomach, serosal surfaces of the abdomen, spleen, liver, and skin. The lungs showed diffuse pulmonary consolidation. The brain was normal in shape and mass, but it had a patchy subarachnoid hemorrhage in the left temporal and occipital lobes and multiple areas of coagulative necrosis typical of recent infarcts. Postmortem cultures were not done, but hyphae and budding yeasts typical of *Candida* species were observed in histopathological tissue slides of the spleen and kidneys.

C. pseudotropicalis has a comparatively restricted natural distribution. This yeast has been recovered from cattle (1) and dairy products (6) but has not been reported to occur in other mammals, primates, birds, or food products or in nature (1, 5, 6). Although the etiological agents of candidiasis occur in several habitats, the most important for human disease are the indigenous flora of humans, of which *C. pseudotropicalis* is a component. *C. pseudotropicalis* has been reported to colonize the oral cavity (4, 5, 7, 9, 12), rectum and feces (11), and vagina (11), but the degree of colonization is extremely low compared with colonization by other *Candida* species. Published surveys of yeast colonization in hospitalized and nonhospitalized subjects indicate that *C. pseudotropicalis* is present in less than 1% of the samples tested (4, 5, 9, 11, 12).

C. pseudotropicalis grows well on routine mycological media, including those containing cycloheximide. The fungal blood culture system at Mayo Clinic in 1979 used a perma-

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nently vented biphasic brain-heart infusion bottle (3). The bottle was incubated at 30°C for 30 days; *C. pseudotropicalis* was recovered after a mean time of 2.6 days of incubation.

Colonies of this yeast are initially white and shiny and become dull and wrinkled with aging. Biochemical characterization by carbohydrate assimilation is necessary to definitively identify the organism to species level. The carbohydrates used include dextrose, galactose, sucrose, cellobiose, raffinose, L-arabinose, and lactose. Also, the pseudomycelium formed on cornmeal-Tween 80 agar is often characteristic, with the formation of very elongate cells which readily fall apart and lie parallel. In some instances pseudohyphae produce chains of blastoconidia in a verticillate arrangement.

The patient described here was predisposed to fungal infection by several factors, including antibiotic therapy, neoplasia, corticosteroids and other immunosuppressive agents, and indwelling intravenous and urinary catheters. The portal of entry for fungemia was most likely genitourinary tract, since yeast was recovered from urine cultures on three different occasions and urine cultures were positive immediately before fever developed and blood cultures became positive for *C. pseudotropicalis*. Antibiotic therapy is known to upset the normal equilibrium among the microbial inhabitants of the small intestine and can allow *Candida* species to proliferate as normal intestinal flora. *Candida* from the intestinal tract could have colonized the urethra or urethral catheter and subsequently infected the bladder. Unabated, the infection could have ascended to the kidneys and become the source of fungemia.

The information presented here supports previous reports that *C. pseudotropicalis* occasionally appears as indigenous flora in humans and as a rare cause of funguria, fungemia, and disseminated disease. Although the incidence of disseminated infection is extremely rare, *C. pseudotropicalis* can occur as an opportunistic pathogen in immunocompromised hosts.

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