CASE REPORT OF *CRYPTOCOCCUS ALBIDUS* PERITONITIS IN A PERITONEAL DIALYSIS PATIENT AND A REVIEW OF THE LITERATURE

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***Cryptococcus albidus* is a saprophytic yeast linked to just 26 reports of human infection in the world literature. Here, we report the first case of *C. albidus* peritonitis, in a patient with end-stage renal disease and hepatitis C-associated cirrhosis who is on peritoneal dialysis. The patient was treated successfully with a week-long course of amphotericin B. Non-*neoformans* cryptococcal infections present a clinical challenge, because they are difficult to diagnose and lack established guidelines for treatment. We present a review of the literature on *C. albidus* infections and their treatment.**

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In April 2012, a 57-year-old man with hepatitis C-associated cirrhosis, hypertension, type 2 diabetes, and end-stage renal disease on peritoneal dialysis (PD) for more than 4 years presented initially at an outside hospital with abdominal pain. He was diagnosed with fungal peritonitis by effluent culture and was discharged on oral fluconazole. His PD catheter was not removed, and speciation of the fungus was pending at discharge.

One week later, the man presented to Mount Sinai Hospital with 4 days of persistent severe generalized abdominal pain associated with nausea, vomiting, and inability to tolerate oral intake. His hepatitis C was genotype 1B, and he had no decompensated liver disease in the form of portal hypertension, varices, portal-systemic encephalopathy, bleeding, or muscle wasting. Additionally, he had no prior history of ascites or

peritonitis, and his peritoneal membrane had no impairment to ultrafiltration. The patient had never received treatment for hepatitis C. He reported no long-term exposure to pigeons.

On admission, the patient was afebrile and hemodynamically stable. His abdomen was exquisitely tender to palpation. The PD catheter site was intact, without erythema or exudates. Peritoneal fluid was turbid, and return of dialysate from the catheter was poor, leading to concern for blockage.

Laboratory studies revealed blood glucose 86 mg/dL, blood urea nitrogen 37 mEq/L, creatinine 12.7 mg/dL, albumin 2.1 g/dL, white blood cell count $6.1 \times 10^3/\text{mm}^3$, platelet count $202 \times 10^3/\text{mm}^3$, and international normalized ratio 1.1. Blood urea nitrogen, creatinine, and platelet count were not significantly changed from this patient's baseline.

Analysis of peritoneal effluent revealed a white blood cell count of $350/\text{mm}^3$, including a polymorphonuclear cell count of $66/\text{mm}^3$, and a red blood cell count of $20/\text{mm}^3$, with rare yeast noted. Those findings were consistent with peritonitis. Staining with calcofluor white was negative. Abdominal radiography revealed no obstruction and no perforation. An ELISA assay for HIV-1 and -2 was negative.

Peritoneal lavage was performed until the returning fluid was clear, with vancomycin 2 g added to one bag of dialysate with dwell time of 6 hours. The causative agent was considered most likely to be *Candida albicans*, and the patient was continued on fluconazole 200 mg daily with no further intraperitoneal antibiotics.

On day 2 of the patient's hospitalization, his symptoms persisted, raising concern for infection with an azole-resistant candidal infection. For that reason, he was switched to intravenous (IV) caspofungin 70 mg daily, which was reduced to 50 mg daily IV after 1 day.

On day 5 of hospitalization, the PD catheter was removed, and the patient received a dual-lumen central venous catheter, through which hemodialysis was started.

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On day 7 of hospitalization, fungal culture of the effluent grew *Cryptococcus albidus* [identification by the Vitek 2 system (bioMérieux, Marcy l'Etoile, France) for rapid microbial identification]. Serum cryptococcal antigen was negative. At this time, it was confirmed that an effluent culture from the outside hospital was also growing *C. albidus*, and there the organism had been identified after 3 days' growth.

Although the chance of misidentification of fungal isolates is not insignificant, the fact that the same organism was independently identified at two separate institutions made the possibility of false identification in our case highly unlikely, and the patient was diagnosed with *C. albidus* peritonitis. He was started on IV amphotericin B, liposomal formulation, 400 mg daily, subsequently completing a 7-day course. Susceptibilities could not be obtained for the organism because of poor growth on the testing media.

Computed tomography imaging revealed perisplenic and perihepatic ascites, but showed no evidence of abscess or focal contained collection. On hospital day 11, the patient developed *Clostridium difficile* diarrhea and was started on IV metronidazole 500 mg. Blood cultures remained negative throughout his hospital stay.

On hospital day 12, he was discharged to home on oral metronidazole 500 mg. His peritonitis has resolved, and he currently remains on thrice-weekly hemodialysis.

DISCUSSION

The genus *Cryptococcus*, a basidiomycetous yeast, comprises more than 30 species and is classically divided into two categories: *C. neoformans* and non-*neoformans* species (1). Whereas *C. neoformans* is a well-known human pathogen, non-*neoformans* species are generally saprophytic and rarely cause infection in humans (2). Since the early 1970s, the incidence of infection with non-*neoformans* cryptococci has been increasing, with more than 80% of cases involving *C. albidus* and *C. laurentii* (3).

To our knowledge, just 26 cases of infection with *C. albidus* have been reported in the world literature [Table 1 (1,4–23)]. The present case is unusual both because peritonitis with *C. albidus* has never before been reported, and because cryptococcal peritonitis is in general very rare, with only 4 cases having been reported in patients on PD (24). Here, we review the characteristics of *C. albidus* infections and cryptococcal peritonitis to develop a better understanding of the management of *C. albidus* peritonitis.

The environmental reservoirs of non-*neoformans* cryptococci are highly varied; the organisms are found

in air, water, wood, soil, pigeon excreta, cheese, fruits, pork products, beans, and wine (3). *C. albidus* has also been isolated from the skin of healthy patients (10). *C. albidus* can be differentiated from other cryptococci by features such as nitrate assimilation, variable growth at 37°C, fermentation of maltose and sucrose, and variable fermentation of galactose, melibiose, and erythritol (7). Although *C. albidus* shares several capsular antigens with *C. neoformans*, the latex agglutination test is specific for polysaccharide antigens found on *C. neoformans*, and thus a negative serum *Cryptococcus* assay does not rule out cryptococcal infection by non-*neoformans* species (16). Because only fungal culture or India ink preparation can detect the presence of this organism, it constitutes a difficult diagnostic challenge (12).

In only 2 of the 26 reported cases of *C. albidus* infection did the subject have a known exposure to pigeons: Burnik *et al.* (4) reported a patient who dealt with pigeons as a hobby, and da Cunha and Lusins reported a patient who worked as an air-conditioner repairman with exposure to pigeon excreta (5). Our patient reported no unusual exposure to pigeons. Given the nearly ubiquitous nature of this yeast, it is not possible to determine the source of his infection.

Of the 26 reported cases of *C. albidus* infection, 8 were cases of central nervous system infection (5–7,10,22); 8, of fungemia (8,9,11,12,15,16,19,23); 3, of cutaneous disease (1,13,20); 3, of ocular disease (14,17,21); 3, of pulmonary disease (4,6); and 1, of vulvovaginitis (18). Notably, the related organism *C. neoformans* has a propensity to invade the central nervous system, and furthermore, meningeal involvement with that fungus may be asymptomatic (8). Given the high number of reports of *C. albidus* infection involving the central nervous system, a recommendation was previously made that when this organism is isolated from another source, the cerebrospinal fluid should also be assayed for the same organism (6).

A recent review noted that 48% of non-*neoformans* cryptococcal infections involved patients with impaired cell-mediated immunity and that 16% involved patients with comorbid HIV infection (3). Among patients with *C. albidus* infection, 16 were noted to have a concomitant condition that might cause immune suppression: 6 were receiving immunosuppressive therapy (1,4,7,15,19,23) and 3 had acquired immunodeficiency syndrome (10,11,14). Although our patient was negative for HIV, immune suppression is likely to have been caused by uremia of end-stage renal disease and by liver disease. Uremia is known to cause impaired activation of T lymphocytes, reductions in the number of B lymphocytes, and altered function of antigen-presenting cells (25).

TABLE 1
Published Cases of Infection with *Cryptococcus albidus*

Reference	Age	Sex	Cause of immune suppression	Type of infection	Treatment	Outcome
Krumholz, 1972 in Burnik <i>et al.</i> , 2007 (4) ^a	68	Male	None	Pneumonitis	AmB 1.0 g total over 5 months	Resolved
da Cunha and Lusins 1973 (5)	45	Male	None	Meningitis	AmB 1.5 g total over 2 months	Resolved
Wieser, 1973 in Horowitz <i>et al.</i> , 1993 (6) ^a	75	Male	Lung cancer on autopsy	CSF infection	None (organism grew postmortem)	Death
	73	Female	Polycythemia vera	CSF infection	None (organism grew postmortem)	Death
	48	Male	None	CSF infection	None	Resolved
	20	Male	None	CSF infection	None	Resolved
Melo <i>et al.</i> , 1980 (7)	29	Male	Juvenile rheumatoid arthritis, on corticosteroids, alcoholic liver disease	Meningitis	AmB	Death
Gluck <i>et al.</i> , 1987 (8)	65	Female	Acute myeloid leukemia, on chemotherapy	Fungemia	AmB 235 mg total, FC 150 mg/kg daily	Death
Lin <i>et al.</i> , 1989 in Horowitz <i>et al.</i> , 1993 (6) ^a	45	Male	Pemphigus foliaceus, on corticosteroids, cyclophosphamide	Fungemia	KE oral	Resolved
Horowitz <i>et al.</i> , 1993 (6)	37	Male	End-stage renal disease, on hemodialysis	Empyema	AmB 50 mg every other day for 3 months, 1.9 g total	Resolved
Taylor <i>et al.</i> , 1994 (9)	NA	NA	NA	Fungemia	NA	NA
Loison <i>et al.</i> , 1996 (10)	38	Male	AIDS	Meningitis, fungemia (recurrence)	Oral FL 600 mg daily, oral IT 400 mg daily (recurrence)	Death
Kordossis <i>et al.</i> , 1998 (11)	47	Female	AIDS	Fungemia	AmB 20 mg daily, FC 5 g daily	Death
Wells <i>et al.</i> , 1998 (12)	4	Female	Acute lymphocytic leukemia	Fungemia	AmB 1 mg/kg daily	Resolved
Narayan <i>et al.</i> , 2000 (13)	70	Male	Sézary syndrome	Cutaneous	Oral FL 200 mg daily for 2 weeks	Resolved

TABLE 1 (cont'd)

Reference	Age	Sex	Cause of immune suppression	Type of infection	Treatment	Outcome
Garelick <i>et al.</i> , 2004 (14)	16	Female	AIDS	Scleral ulceration	AmB topical, IV 1 mg/kg daily for 5 days, then IT 100 mg twice daily	Resolved
Lee <i>et al.</i> , 2004 (15)	23	Male	Renal graft, on cyclosporine, prednisolone	Fungemia	IV FL for 10 days, then oral 200 mg daily for 12 months	Resolved
Ramchandren and Gladstone, 2004 (16)	51	Male	T-cell lymphoma, acute myeloid leukemia, multiple cycles of chemotherapy	Fungemia	AmB 0.6 mg/kg for 2 weeks, oral IT for 6 weeks	Resolved
de Castro <i>et al.</i> , 2005 (17)	69	Female	None	Keratitis	Gatifloxacin topical	Resolved
Jackson <i>et al.</i> , 2005 (18)	NA	NA	Vulvovaginitis	NA	NA	NA
Burnik <i>et al.</i> , 2007 (4)	44	Male	Still's disease, on low-dose steroids, methotrexate	Pneumonia, ARDS	AmB 400 mg daily	Death
Hoang and Burruss, 2007 (1)	14	Male	Psoriasis, on etanercept	Cutaneous scalp infection	FL 100 mg twice daily	Resolved
Cleveland <i>et al.</i> , 2013 (19)	55	Male	Liver graft, on sirolimus, mycophenolate mofetil, lymphocyte immune globulin	Fungemia	Oral posaconazole 200 mg 3 times daily	Resolved
Endo <i>et al.</i> , 2011 (20)	NA	NA	Palmopustular psoriasis	NA	NA	NA
Drancourt <i>et al.</i> , 2008 (21)	NA	NA	Uveitis	NA	NA	NA
Dorneanu <i>et al.</i> , 2008 (22)	NA	NA	Meningitis	NA	NA	NA
Present case	57	Male	End-stage renal disease, hepatitis C cirrhosis	Peritonitis	Oral FL 200 mg daily for 3 days, CF 70 mg for 1 day, then 50 mg daily for 2 days, then AmB 400 mg daily for 7 days	Resolved

AmB = amphotericin B; CSF = cerebrospinal fluid; FC = 5-fluorocytosine; KE = ketoconazole; NA = not available; FL = fluconazole; IT = itraconazole; IV = intravenous; ARDS = adult respiratory distress syndrome; CF = caspofungin.

^a Original publication could not be accessed, information gathered from reference provided.

Patients with liver disease have an increased risk of infection because of impaired phagocyte function, decreased complement levels, dysimmune regulation,

and potentially, gastrointestinal bleeding, which can cause translocation of bacteria from the gastrointestinal tract to the blood (26).

Because no guidelines for the treatment of *C. albidus* infections have been established, a review of the literature on the susceptibility of this organism to antifungal agents is informative (Table 2). Amphotericin B was the only antifungal agent to which *C. albidus* consistently demonstrated susceptibility (2,4,10,11,13,14,27). In our patient, treatment with that agent was successful. It is important to note that *C. albidus* also exhibits susceptibility to flucytosine, posaconazole, fluconazole, voriconazole, itraconazole, and miconazole in some cases (10,11,13,19,27–29). Because of these variations in *C. albidus* antifungal susceptibility, treatment should ideally be tailored to the individual disease-causing organism. However, as demonstrated in the present case, fastidious growth requirements might make such an approach impossible.

Lessons learned from the more general issue of cryptococcal peritonitis provide further guidance on therapeutic choices for *C. albidus* peritonitis, given that *C. albidus* and *C. neoformans* (the more common culprit in cryptococcal peritonitis) share susceptibility to amphotericin B at similar minimum inhibitory concentrations (2,27,30). The mainstays of fungal peritonitis management are rapid peritoneal fluid exchanges to minimize gross turbidity and the risk of adhesions, early removal of the catheter, and concurrent parenteral antifungal treatment (24). In *C. neoformans* peritonitis, amphotericin B is the antifungal agent of choice, followed by oral fluconazole (26,31), which is known to achieve a peritoneal penetration of more than 60% (24). There are additionally

2 cases in the literature of *C. laurentii* peritonitis, both of which occurred in patients on PD (32). Both patients were treated successfully, one with 60 days of amphotericin B, and one solely with removal of the peritoneal catheter and peritoneal irrigation using saline solution (3). Intraperitoneal amphotericin B is contraindicated, because it may cause peritoneal fibrosis (24).

Given our patient's history of hepatitis C cirrhosis, it is worthwhile noting that liver disease and cryptococcal peritonitis have a known association (26). Cirrhosis has also been found to be an independent predictor of mortality in patients with cryptococcosis (31). Proposed reasons for those observations are that cirrhotic ascites is typically deficient in complement concentration and opsonin activity (31).

Given the diagnostic challenge presented by cryptococcal infections, the importance of heightened clinical suspicion for these infections is underscored, so as to expedite time to treatment.

SUMMARY

From our case of *C. albidus* peritonitis and from earlier reports of infection with *C. albidus*, we can conclude that, in the absence of susceptibility data, amphotericin B is the antifungal agent of choice for treatment; however, the paucity of cases of cryptococcal peritonitis precludes determination of a dosing regimen. As with any case of fungal peritonitis in the context of PD, early catheter removal and peritoneal irrigation are essential

TABLE 2
Cryptococcus albidus Antifungal Susceptibility

Antifungal	MIC ₉₀ (27) (n=10)	MIC ₉₀ (2) (n=10)	Dose (mg/L) MIC ₅₀ (28) (n=5)	MIC (20) (n=1)	MIC (11) (n=1)
Amphotericin B	0.5	1		0.5	0.5
Flucytosine		>64.0	0.5	64	1.5
Fluconazole	64	>64.0		128	4
Voriconazole	32	8		16	
Itraconazole	1	>8		16	0.5
Miconazole					
Posaconazole		>8		16	
Ketoconazole					0.25
Ravuconazole	32	>8			
Albaconazole	32				
Terbinafine	32				
Caspofungin		>16		32	
Anidulafungin		>16			
Micafungin	128	>16			

MIC = minimum inhibitory concentration.

components of therapy. Examination of the cerebrospinal fluid should be considered, given the propensity of this organism for central nervous system infection. Finally, we advise increased clinical suspicion for cryptococcal infection in patients with liver disease presenting with peritonitis, so as to shorten the delay until administration of adequate antifungal therapy.

DISCLOSURES

No financial conflict of interest exists.

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