LETTERS TO THE EDITOR

Central-Line–Associated Bloodstream Infection Caused by *Sporobolomyces salmonicolor*

To the Editor—Sporobolomyces salmonicolor is one of the Basidiomycetous yeasts, which include *Cryptococcus* spp., *Malassezia* spp., *Rhodotorula glutinis*, and *Trichosporon asahii*.[1] It has long been isolated from environmental sources, such as freshwater, marine water, air, tree leaves, and orange peels.[2] However, *Sporobolomyces* species causing human infections have rarely been reported,[2–7] and most of the reported cases developed in AIDS patients.[5,6] To best of our knowledge, central-line–associated bloodstream infection caused by *S. salmonicolor* has not been previously reported.

A 65-year-old woman with poorly controlled diabetes mellitus was admitted for evaluation of a breast mass of approximately 9×9 cm. After serial examinations, local advanced breast cancer was diagnosed. Ten days later, she received central-line insertion due to poor venous access. However, she developed a fever after 3 weeks of hospitalization. Physical examinations were unremarkable except for the breast mass. Laboratory examination results were as follows: white blood cell count (WBC), 29,400/mm^3 (94.1% neutrophils) and C-reactive protein, 297.2 mg/L (normal reference, <3 mg/L). Neither pyuria, bacteria, nor funguria was detected by urinalysis. Chest radiography did not show any active lung lesions. An empirical antibiotic with ciprofloxacin (400 mg every 8 hours) was prescribed, but in vain. Three days later, blood cultures from the central venous catheter (CVC) and peripheral blood yielded a yeast-like organism. Therefore, intravenous micafungin was added to the regimen to cover fungemia. However, no other specimens, including sputum, or urine grew any yeast. Finally, the pathogen was further identified as *S. salmonicolor*. The antifungal treatment was shifted to voriconazole, and the CVC was removed. Thereafter, her fever abated and repeated blood culture did not yield fungus.

S. salmonicolor rarely infects humans, and the infections reported include endophalmitis, meninigitis, lymphadenitis, and dermitis.[2,3,6,7] Herein, we present the first reported case of a central-line–associated bloodstream infection caused by *S. salmonicolor*. Our finding indicates that *S. salmonicolor* can cause catheter-associated infection and further expands the clinical spectrum of *S. salmonicolor* infections.

Most reported cases have shown that *Sporobolomyces* spp. develops in immunocompromised patients, especially patients with AIDS.[5,6] Although the patient in the present case did not have AIDS, the possible risk factor may be the underlying breast cancer and poorly controlled diabetes mellitus. In contrast, *Sporobolomyces* spp. infection is known to develop among immunocompetent patients, as in 1 reported case with endogenous endophalmitis[2] and another case with possible meningitis.[4] All of these findings suggest that clinicians should consider *Sporobolomyces* spp. as a possible pathogen for immunocompromised patients, such as HIV-infected patients, cancer and diabetic patients, and even rarely for immunocompetent patients.

The clinical outcomes of patients with *S. salmonicolor* fungemia have not been well defined because of the limited number of cases. In the present report, the patient had favorable outcomes after antifungal treatment and removal of catheters. However, further large-scale studies are needed to better understand the clinical manifestations and prognosis of *S. salmonicolor* fungemia.

Because the studies and the reported case regarding *S. salmonicolor* infections are limited, the drug of choice remains unclear. A previous in vitro study[1] showed that the minimum inhibitory concentration needed to kill 90% of the organisms (MIC_{90}) of the tested azoles, including albaconzaole, voriconazole, itraconazole, ravuconazole, ranged only from 0.06 to 0.12 μg/mL. In contrast, the MIC range and MIC_{90} of the micafungin were as high as 128 and 128 μg/mL, respectively. Based on the aforementioned in vitro studies, azoles may be considered the drugs of choice to treat *S. salmonicolor* infection.

In conclusion, *S. salmonicolor* is an emerging pathogen that can cause invasive infections, and it may play an important role in the clinical setting of central-line–associated bloodstream infections. Healthcare-associated catheter-associated bloodstream infections due to that pathogen may respond well to an appropriate antifungal agent plus catheter removal.

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Emergence of a Novel Binary Toxin–Positive Strain of *Clostridium difficile* Associated With Severe Diarrhea That Was Not Ribotype 027 and 078 in China

To the Editor—Clostridium difficile is a frequent cause of antibiotic-associated diarrhea and healthcare-associated infections.[1,2] Epidemics of *C. difficile* infection (CDI) have occurred in North America and Europe over recent decades. In particular, epidemic *C. difficile* (sequence type [ST] 1/027/NAP1) has rapidly emerged in the past decade as the leading cause of *C. difficile*–associated diarrhea worldwide, resulting in high morbidity and mortality in hospitalized patients. Very little is known about the epidemiology of *C. difficile*–associated diarrhea outside of North America and Europe. However, the hypertoxigenic strain *C. difficile* RT027 has rarely been detected in Asia to date, specifically not in China before 2013.[3] In addition, cases of *C. difficile* RT078 have not been reported in China. We report a novel binary toxin–positive non-027, non-078 *C. difficile* associated with severe diarrhea recently identified in China.

A 65-year-old man was admitted to Xiangya Hospital in Changsha, China, with fever, headache, diarrhea, and impaired consciousness. He had been diagnosed with a central nervous system infection 9 days earlier in a local hospital, presumptively diagnosed as suppurative or tubercular meningitis. Antimicrobial therapy was initiated with ceftriaxone over the next 4 days in that local hospital. The patient's condition did not improve; he developed a pulmonary infection, with *Acinetobacter baumannii* isolated from the sputum, for which he was intravenously treated with cefepime for 5 days. The consciousness level of the patient deteriorated, and he developed severe diarrhea and experienced bouts of vomiting. Therefore, on February 8, 2014, he was transferred to Xiangya Hospital. Stool specimens collected during hospitalization tested positive for *C. difficile* toxin B by polymerase chain reaction assay, and *C. difficile* was confirmed by culture and biochemical characteristics. Enteral vancomycin was prescribed for the patient, but symptoms further deteriorated and he died on day 4 of hospitalization.

Stool specimens from the patient were cultured for anaerobic bacteria, and *C. difficile* was isolated and identified. The toxin genes *tcdA*, *tcdB*, *cdtA*, and *cdtB* were detected by polymerase chain reaction assay.[4] The isolate (LC693) was positive for toxin A, toxin B, and binary toxin. DNA sequence analysis of the toxin gene to the genome sequence of CD630 (GenBank accession No. AM180355.1) revealed an 18–base pair deletion (nucleotides 330-347) located in *tcdC* (682 base pairs, GenBank accession No. KM609431.1) of LC693. It did not express a variation at nucleotide position (nt) 117 (associated with RT027). However, there was a point mutation at nt 184, resulting in the introduction of a premature stop codon (TAA) in the putative TcdC protein (Figure 1). Multilocus sequence typing indicated the isolate was ST201. Alleles for the profile were *adk-1*, *atpA-6*, *dx-4*, *glyA-7*, *recA-2*, *sod-8,* and *tpi-31*, making the Xiangya hospital clinical isolate LC693 unique from the RT027 (ST1, R20291, GenBank accession No. FN545816.1) and RT078 (ST11, M120, GenBank accession No. FN665653.1) clones. Whole genome sequencing of *C. difficile* LC693 was performed using a MiSeq (Illumina) by PE300 strategy. Approximately 424 Mb clean data were obtained, with a mean read length of 300 base pairs, 100 times coverage of the approximately 4.07 Mb genome. hylogenomic analysis showed that the LC693 was more closely related to the RT027 (CD196, R20291) cluster than to the RT078 (M120) (Figure 2). Between LC693 and R20291, the single-nucleotide polymorphisms were 35,505; between LC693 and M120, the single-nucleotide polymorphisms were 87,672.

The incidence and associated mortality of CDI have been increasing. This changing epidemiology has coincided with the emergence and rapid spread of *C. difficile* RT027/078, involved in several large outbreaks of severe CDI in North America and Europe. Furthermore, hypervirulent RT027 and RT078 have been associated with more severe disease because of the production of higher amounts of toxin A and B due to *tcdC* deletion.[5,6] Sequence analysis of *tcdC* of the isolate LC693 showed an 18–base pair deletion and a premature stop codon (TAA), compatible with the genotype of a hypertoxigenic strain. However, it also differed from RT027 and RT078, indicating possible existence of a new hypervirulent strain in China. Our findings were similar to those of Lim et al,[7] who identified and characterized a *C. difficile* strain associated with a severe clinical phenotype that genetic analysis showed to be ST41/RT244 that was also different from RT027 and RT078 in Melbourne, Australia. There were 10,803 single-nucleotide polymorphisms between ST41/RT244 and RT027. Further tests are required to determine the ribotype and toxin production of the isolate LC693, and study of the epidemiology of the