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CASE REPORT

Candida fermentati as a Cause of Persistent Fungemia in a Preterm Neonate Successfully Treated by Combination Therapy with Amphotericin B and Caspofungin

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A case of persistent candidemia in a preterm neonate caused by *Candida fermentati*, identified by sequencing of the internally transcribed spacer region of ribosomal DNA (rDNA), is described. The neonate was treated for 30 days by combination therapy with amphotericin B (AmBisome) and caspofungin with a successful outcome, and no drug-related side effects were observed.

CASE REPORT

A 27-week-preterm male, weighing 1,040 g, was delivered to a Kuwaiti mother by emergency longitudinal cesarean section in a surgical intensive care unit. On day 0, the baby was diagnosed to have hyaline membrane disease and patent ductus arteriosus with Apgar scores of 7 and 9. Soon after delivery, an umbilical artery catheter (UAC), an umbilical venous catheter (UVC), and a peripheral cannula were placed to provide fluids and medication and the baby was also intubated. On day 5, he was empirically started on ampicillin and amikacin. On day 12, he developed signs and symptoms of sepsis requiring a change in treatment to cloxacillin and amikacin. Since his septic condition continued, the treatment was changed to vancomycin. On day 27, the blood culture grew a yeast (Kw3414/13), which was identified as *Candida famata* by the use of a Vitek 2 yeast identification (YST ID) card (bioMérieux, Marcy l'Étoile, France). While cultures of UVC and UAC tips were negative for growth, a culture from a long-line tip yielded yeast which was also identified as *C. famata*. The antifungal drug susceptibility was determined by Etest, and the isolate appeared susceptible to amphotericin B (Table 1). The baby was started on amphotericin B at 1.5 mg/kg of body weight. A blood culture repeated after 2 days of treatment again yielded *C. famata*, leading to a change of treatment to a lipid formulation of amphotericin B (AmBisome) (8.5 mg once daily). Despite treatment with amphotericin B for 5 days, the blood culture remained positive for *C. famata*, prompting addition of caspofungin (2.5 mg/kg). Although additional blood cultures performed on day 50, day 53, and day 59 yielded *C. famata* (identified by the use of a Vitek 2 yeast identification system), there was symptomatic improvement in the condition of the baby. A blood culture repeated on day 66 (after 30 days of combination therapy with amphotericin B and caspofungin) became negative for the yeast. There was no recurrence of infection during 3 weeks of follow-up, and the baby was subsequently discharged in healthy condition.

During 6 weeks of follow-up, 6 blood culture isolates were obtained and were characterized. All isolates formed whitish colonies on Sabouraud dextrose agar which became slightly tanned on aging. Microscopic examination revealed ovoid to elongated yeast cells of variable sizes (2 to 5.2 by 2.2 to 5.6 μm) occurring singly, in pairs, or in short chains. A Dalmau slide culture on cornmeal agar at 30°C showed well-branched pseudohyphae with clusters of budding yeast cells (Fig. 1). True hyphae were lacking. Growth on ascospore agar and malt extract agar, incubated at 25°C for 30 days, did not show ascospores. Based on the results of carbohydrate assimilation profiles obtained by ID32C and a Vitek 2 yeast identification system, the isolates were identified as *C. famata*. For molecular identification, the genomic DNA from the isolates was prepared as described previously (1). The internally transcribed spacer (ITS) region of ribosomal DNA (rDNA) was amplified and sequenced as described previously (2). Molecular fingerprinting of the isolates was carried out by PCR sequencing of the intergenic spacer region between the 28S rRNA and 5S rRNA genes by using IGS1F (5′-CGGAGTATTGTAAGCAGT AGA-3′) and IGS1R (5′-TAGTGGGAGACCATACGCGAA-3′) as PCR amplification and sequencing primers, essentially as described previously (3). PCR fingerprinting of the isolates was also carried out with microsatellite-based ($GACA_4$, 5′-GACAG ACAGACAGACA-3′) and minisatellite-based (M13-MIN, 5′-GAGGGTGGCGGTTCT-3′) primers as described previously (4). The ITS region sequences of our isolates were identical and showed complete (100%) identity with the corresponding sequences from reference *Candida fermentati* strains CBS 9966 (type strain) and CBS 2022 but showed 4 nucleotide differences from the sequence from *Candida guilliermondii* CBS 2030, thus establishing their identity as *C. fermentati* (5). The IGS region sequences of all the isolates were identical. PCR fingerprinting carried out with microsatellite-based and minisatellite-based

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TABLE 1 Antifungal susceptibility of *C. fermentati* strains isolated from blood samples

Strain or parameter	Date of isolation	MIC by Etest (μg/ml)[a]							
		AP	FL	VO	POS	IT	FC	CS	MYC
Kw3414/13	30 September 2013	0.002	3	0.125	0.19	3	0.003	0.125	0.38
Kw3434/13	2 October 2013	0.047	3	0.125	0.19	4	0.006	0.19	0.25
Kw3476/13	7 October 2013	0.064	4	0.125	0.19	4	0.004	0.38	0.38
KW3556/13	21 October 2013	0.094	4	0.38	0.19	3	0.003	0.19	0.38
Kw3593/13	24 October 2013	0.094	3	0.19	0.25	4	0.012	0.38	0.25
Kw3644/13	29 October 2013	0.125	4	0.38	0.25	4	0.016	0.75	0.75
Geometric mean of MICs		0.043	3.46	0.194	0.208	3.634	0.005	0.280	0.370

[a] Abbreviations: AP, amphotericin B; FL, fluconazole; VO, voriconazole; IT, itraconazole; POS, posaconazole; FC, flucytosine; CS, caspofungin; MYC, micafungin.

primers also yielded identical amplified DNA fragment patterns from all the 6 isolates, indicating clonality.

A case of persistent fungemia caused by *C. fermentati* in a preterm neonate is described. All six *C. fermentati* isolates showed antifungal susceptibility profile consistent with the published studies (6–9), and there was no indication of acquisition of resistance during treatment (Table 1). A noteworthy feature of our case is the successful use of caspofungin in combination with amphotericin B for an extended period of 30 days without any evidence of side effects or toxicity. There appears to be no previous report of the combined use of these two drugs in the management of candidemia in preterm neonates. Presently, there is no recommended dose for caspofungin for the treatment of invasive candidiasis in neonates (10). However, based on limited clinical data, a dosage of 25 mg/m^2/day has been suggested, which is comparable to the dosages corresponding to the results of area under curve/pharmacokinetic studies carried out in adults (11). In some studies, caspofungin has been used in neonates who were either refractory to or intolerant of amphotericin B therapy without causing any adverse effects (12, 13).

Candida fermentati (teleomorph *Meyerozyma caribbica*) is another pathogenic species within the *C. guilliermondii* complex, representing about 9% of the isolates in this complex in the global collection originating from different geographic regions (6). There are only two accepted species in the genus *Myerozyma*, *M. caribbica* and *M. guilliermondii*, although some additional closely related taxa have been proposed upon further molecular analysis (14). These species are estimated to cause about 1% to 3% of the total number of cases of candidemia (6, 7). *Candida fermentati* was first described in 2005 by Vaughan-Martini et al. as an anamorphic species of *Pichia carribica* under the *Pichia guilliermondii* complex (15). It was later placed in the ascosporic genus *Meyerozyma*, as *M. caribbica*, along with *M. guilliermondii* (14). Members of the *C. guilliermondii* complex are indistinguishable by conventional phenotypic methods but appear genetically heterogeneous (16–18). Although *C. guilliermondii* is widely distributed in the environment and has been isolated from a variety of saprophytic sources, it plays an infrequent role in human diseases (19). So far, *C. fermentati* and *C. guilliermondii* are the known human pathogens in this complex. Some recent reports suggest that the incidence of *C. guilliermondii* or *C. fermentati* fungemia is highest in Latin America (7, 20), China (21, 22), and Taiwan (16), particularly in pediatric-care age groups (20–23). In a recent Chinese study, 39 of 238 (16.5%) patients had candidemia due to *C. guilliermondii* (22). A multivariate analysis of the risk factors in these patients revealed that preterm infants with low birth weight were at the higher risk of candidemia due to *C. guilliermondii* strains than non-*C. guilliermondii* strains (51.3% [20/39] versus 8.5% [17/199], respectively; $P = 0.001$). Other risk factors included intravenous nutrition and surgery (22). In an earlier series of 9 neonatal cases of *Candida* septicemia from China, 6 were caused by *C. guilliermondii* (21). The reasons for the increased rate of occurrence of *C. guilliermondii* candidemia in these two studies need further epidemiological investigations.

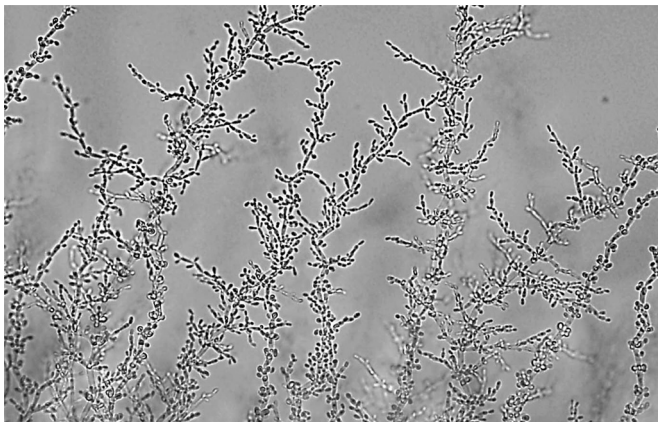

FIG 1 Dalmau slide culture on cornmeal agar showing branched pseudohyphae with budding yeast cells.

C. fermentati/*C. guilliermondii* complex isolates are known to exhibit reduced *in vitro* susceptibility to azoles and echinocandins (7–9, 19, 24, 25), which may have therapeutic implications. In addition, the possibility of a gradual increase in their incidence as a result of selection pressure also exists. *C. guilliermondii* complex isolates resistant to amphotericin B and azoles have been isolated from clinical specimens (26, 27). In one study conducted under the auspices of the ARTEMIS DISK Antifungal Susceptibility Program, 10.8% and 4.9% isolates of *C. guilliermondii* were resistant to fluconazole and voriconazole, respectively (7). Likewise, *C. guilliermondii* strains are found to be 2-fold to 16-fold less susceptible than other *Candida* species (excluding *C. parapsilosis*) to caspofungin, anidulafungin, and micafungin (7, 28). In a multicenter study, Pfaller et al. (9) examined 234 *C. guilliermondii* strains to determine epidemiologic cutoff values (ECVs) for anidulafungin and micafungin. MICs for ECVs for anidulafungin

and micafungin covering 97.5% of the strains were 8 and 2 μg/ml, respectively. Similarly, based on wild-type MIC distributions, the ECVs for *C. guilliermondii* ($n = 373$) for fluconazole, posaconazole, and voriconazole were 8, 0.5, and 0.12 μg/ml, respectively, in another study (10). Interstrain differences in susceptibility to azoles depending upon the source of isolation have also been noted among *C. guilliermondii* isolates (9). For example, 85% of bloodstream isolates of *C. guilliermondii* were susceptible to fluconazole, whereas only 67.7% from skin and soft tissues were susceptible. In contrast, 93.4% of isolates of *C. guilliermondii* originating from blood, skin, and soft tissues were susceptible to voriconazole whereas only 80.4% of isolates from the urinary tract were susceptible to this antifungal drug (7).

Here, it is pertinent to mention that most of the antifungal susceptibility data on *C. fermentati* reported in the literature are derived from isolates representing the *C. guilliermondii* complex (29). This is because the available phenotypic methods alone are insufficient to provide unequivocal identification of the species included in the complex (30). The importance of correct species identification is underscored by species-specific interpretive criteria. Limited *in vitro* susceptibility data suggest that there are differences between the antifungal susceptibility profiles of *C. fermentati* and *C. guilliermondii*. While *C. guilliermondii* isolates tend to show higher micafungin MICs (0.57 versus 0.35 μg/ml, $P = 0.0001$), *C. fermentati* isolates, on the other hand, have higher fluconazole (6.13 versus 3.17 μg/ml, $P = 0.079$) and amphotericin B (0.31 versus 0.18 μg/ml, $P = 0.088$) MICs (6). To what extent these differences in antifungal susceptibilities between the two species impact therapeutic outcome is unclear.

Yeast species with similar phenotypic characteristics have always posed identification problems (5, 30). Conventional identification methods often yield erroneous species identification. *C. famata* is often wrongly identified by conventional identification methods such the use of Vitek 2 (5, 6, 16, 18, 30, 31). Matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) platforms cannot be used for rapid identification of culture isolates since *C. fermentati* is not currently included in their database. Hence, any *C. famata* isolation from clinical specimens should be confirmed by sequencing of rDNA.

In conclusion, a case of persistent candidemia due to *C. fermentati*, identified by sequencing of the ITS region of rDNA, is described. The neonate was treated with amphotericin B and caspofungin combination therapy for 30 days with no drug-related side effects. To the best of our knowledge, this is the first report of the successful use of amphotericin B and caspofungin in combination in a preterm neonate for the treatment of persistent candidemia. Additionally, the report highlights the use of molecular methods in the identification of rare or cryptic *Candida* species causing human infections.

Nucleotide sequence accession numbers. Nucleotide sequences determined in this work have been deposited in the EMBL database under accession no. HG970745, HG970748, LK392385 to LK392391, and LN651284 to LN651286.

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REFERENCES

1. **Ahmad S, Mustafa AS, Khan Z, Al-Rifayi AI, Khan ZU.** 2004. PCR-enzyme immunoassay of rDNA in the diagnosis of candidemia and comparison with amplicon detection by agarose gel electrophoresis. Int J Med Microbiol **294:**45–51. http://dx.doi.org/10.1016/j.ijmm.2004.01.002.
2. **Khan ZU, Ahmad S, Hagen F, Fell JW, Kowshik T, Chandy R, Boekhout T.** 2010. *Cryptococccus randhawai* sp. nov., a novel anamorphic basidiomycetous yeast isolated from tree trunk hollow of *Ficus religiosa* (peepal tree) from New Delhi, India. Antonie van Leeuwenhoek **97:**253–259. http://dx.doi.org/10.1007/s10482-009-9406-8.
3. **Asadzadeh M, Ahmad S, Al-Sweih N, Khan ZU.** 2009. Rapid molecular differentiation and genotypic heterogeneity among *Candida parapsilosis* and *Candida orthopsilosis* strains isolated from clinical specimens in Kuwait. J Med Microbiol **58:**745–752. http://dx.doi.org/10.1099/jmm.0.008235-0.
4. **Ahmad S, Khan Z, Mustafa AS, Khan ZU.** 2003. Epidemiology of *Candida* colonization in an intensive care unit of a teaching hospital in Kuwait. Med Mycol **41:**487–493. http://dx.doi.org/10.1080/1369378031000147458.
5. **Desnos-Ollivier M, Ragon M, Robert V, Raoux D, Gantier JC, Dromer F.** 2008. *Debaryomyces hansenii* (*Candida famata*), a rare human fungal pathogen often misidentified as *Pichia guilliermondii* (*Candida guilliermondii*). J Clin Microbiol **46:**3237–3242. http://dx.doi.org/10.1128/JCM.01451-08.
6. **Lockhart SR, Messer SA, Pfaller MA, Diekema DJ.** 2009. Identification and susceptibility profile of *Candida fermentati* from a worldwide collection of *Candida guilliermondii* clinical isolates. J Clin Microbiol **47:**242–244. http://dx.doi.org/10.1128/JCM.01889-08.
7. **Pfaller MA, Diekema DJ, Mendez M, Kibbler C, Erzsebet P, Chang SC, Gibbs DL, Newell VA.** 2006. *Candida guilliermondii*, an opportunistic fungal pathogen with decreased susceptibility to fluconazole: geographic and temporal trends from the ARTEMIS DISK antifungal surveillance program. J Clin Microbiol **44:**3551–3556. http://dx.doi.org/10.1128/JCM.00865-06.
8. **Espinel-Ingroff A, Pfaller MA, Bustamante B, Canton E, Fothergill A, Fuller J, Gonzalez GM, Lass-Flörl C, Lockhart SR, Martin-Mazuelos E, Meis JF, Melhem MS, Ostrosky-Zeichner L, Pelaez T, Szeszs MW, St-Germain G, Bonfietti LX, Guarro J, Turnidge J.** 2014. A multilaboratory study of epidemiological cutoff values for azole-resistance detection of eight *Candida* spp. to fluconazole, posaconazole, and voriconazole. Antimicrob Agents Chemother **58:**2006–2012. http://dx.doi.org/10.1128/AAC.02615-13.
9. **Pfaller MA, Espinel-Ingroff A, Bustamante B, Canton E, Diekema DJ, Fothergill A, Fuller J, Gonzalez GM, Guarro J, Lass-Flörl C, Lockhart SR, Martin-Mazuelos E, Meis JF, Ostrosky-Zeichner L, Pelaez T, St-Germain G, Turnidge J.** 2014. Multicenter study of anidulafungin and micafungin MIC distributions and epidemiological cutoff values for eight *Candida* species and the CLSI M27-A3 broth microdilution method. Antimicrob Agents Chemother **58:**916–922. http://dx.doi.org/10.1128/AAC.02020-13.
10. **Hope WW, Castagnola E, Groll AH, Roilides E, Akova M, Arendrup MC, Arikan-Akdagli S, Bassetti M, Bille J, Cornely OA, Cuenca-Estrella M, Donnelly JP, Garbino J, Herbrecht R, Jensen HE, Kullberg BJ, Lass-Flörl C, Lortholary O, Meersseman W, Petrikkos G, Richardson MD, Verweij PE, Viscoli C, Ullmann AJ, ESCMID Fungal Infection Study Group.** 2012. ESCMID* guideline for the diagnosis and management of *Candida* diseases 2012: prevention and management of invasive infections in neonates and children caused by *Candida* spp. Clin Microbiol Infect **18**(Suppl 7)**:**38–52. http://dx.doi.org/10.1111/1469-0691.12040.
11. **Sáez-Llorens X, Macias M, Maiya P, Pineros J, Jafri HS, Chatterjee A, Ruiz G, Raghavan J, Bradshaw SK, Kartsonis NA, Sun P, Strohmaier KM, Fallon M, Bi S, Stone JA, Chow JW.** 2009. Pharmacokinetics and safety of caspofungin in neonates and infants less than 3 months of age. Antimicrob Agents Chemother **53:**869–875. http://dx.doi.org/10.1128/AAC.00868-08.
12. **Odio CM, Araya R, Pinto LE, Castro CE, Vasquez S, Alfaro B, Sàenz A, Herrera ML, Walsh TJ.** 2004. Caspofungin therapy of neonates with invasive candidiasis. Pediatr Infect Dis J **23:**1093–1097.
13. **Manzar S, Kamat M, Pyati S.** 2006. Caspofungin for refractory candidemia in neonates. Pediatr Infect Dis J **25:**282–283. http://dx.doi.org/10.1097/01.inf.0000200141.46957.71.
14. **Kurtzman CP, Suzuki M.** 2011. *Meyerozyma*, p 621–624. *In* Kurtzman CP, Fell JW, Boekhout T (ed), The yeasts, a taxonomic study, 5th ed. Elsevier, London, United Kingdom.
15. **Vaughan-Martini A, Kurtzman CP, Meyer SA, O'Neill EB.** 2005. Two new species in the *Pichia guilliermondii* clade: *Pichia caribbica* sp. nov., the ascosporic state of *Candida fermentati*, and *Candida carpophila* comb.

nov. FEMS Yeast Res **5:**463–469. http://dx.doi.org/10.1016/j.femsyr.2004.10.008.

16. **Chen CY, Huang SY, Tang JL, Tsay W, Yao M, Ko BS, Chou WC, Tien HF, Hsueh PR.** 2013. Clinical features of patients with infections caused by *Candida guilliermondii* and *Candida fermentati* and antifungal susceptibility of the isolates at a medical centre in Taiwan, 2001–2010. J Antimicrob Chemother **68:**2632–2635. http://dx.doi.org/10.1093/jac/dkt214.
17. **Bai FY.** 1996. Separation of *Candida fermentati* comb. nov. from *Candida guilliermondii* by DNA base composition and electrophoretic karyotyping. Syst Appl Microbiol **19:**178–181. http://dx.doi.org/10.1016/S0723-2020(96)80043-9.
18. **Romi W, Keisam S, Ahmed G, Jeyaram K.** 2014. Reliable differentiation of *Meyerozyma guilliermondii* from *Meyerozyma caribbica* by internal transcribed spacer restriction fingerprinting. BMC Microbiol **14:**52. http://dx.doi.org/10.1186/1471-2180-14-52.
19. **Savini V, Catavitello C, Onofrillo D, Masciarelli G, Astolfi D, Balbinot A, Febbo F, D'Amario C, D'Antonio D.** 2011. What do we know about *Candida guilliermondii*? A voyage throughout past and current literature about this emerging yeast. Mycoses **54:**434–441. http://dx.doi.org/10.1111/j.1439-0507.2010.01960.x.
20. **Medeiros EA, Lott TJ, Colombo AL, Godoy P, Coutinho AP, Braga MS, Nucci M, Brandt ME.** 2007. Evidence for a pseudo-outbreak of *Candida guilliermondii* fungemia in a university hospital in Brazil. J Clin Microbiol **45:**942–947. http://dx.doi.org/10.1128/JCM.01878-06.
21. **Ma XL, Sun W, Liu T.** 2006. Clinical characteristics of *Candida* septicemia seen in a neonatal intensive care unit: analysis of 9 cases. Zhonghua Er Ke Za Zhi **44:**694–697. (In Chinese.)
22. **Wu Z, Liu Y, Feng X, Liu Y, Wang S, Zhu X, Chen Q, Pan S.** 28 February 2014, posting date. Candidemia: incidence rates, type of species, and risk factors at a tertiary care academic hospital in China. Int J Infect Dis http://dx.doi.org/10.1016/j.ijid.2013.11.011.
23. **Pemán J, Bosch M, Cantón E, Viudes A, Jarque I, Gómez-García M, García-Martínez JM, Gobernado M.** 2008. Fungemia due to *Candida guilliermondii* in a pediatric and adult population during a 12-year period. Diagn Microbiol Infect Dis **60:**109–112. http://dx.doi.org/10.1016/j.diagmicrobio.2007.07.014.
24. **Barchiesi F, Spreghini E, Tomassetti S, Della Vittoria A, Arzeni D, Manso E, Scalise G.** 2006. Effects of caspofungin against *Candida guilliermondii* and *Candida parapsilosis*. Antimicrob Agents Chemother **50:**2719–2727. http://dx.doi.org/10.1128/AAC.00111-06.
25. **Schmalreck AF, Willinger B, Haase G, Blum G, Lass-Flörl C, Fegeler W, Becker K, Antifungal Susceptibility Testing-AFST Study Group.** 2012. Species and susceptibility distribution of 1062 clinical yeast isolates to azoles, echinocandins, flucytosine and amphotericin B from a multicentre study. Mycoses **55:**e124–e137. http://dx.doi.org/10.1111/j.1439-0507.2011.02165.x.
26. **Dick JD, Rosengard BR, Merz WG, Stuart RK, Hutchins GM, Saral R.** 1985. Fatal disseminated candidiasis due to amphotericin-B-resistant *Candida guilliermondii*. Ann Intern Med **102:**67–68. http://dx.doi.org/10.7326/0003-4819-102-1-67.
27. **Savini V, Catavitello C, Di Marzio I, Masciarelli G, Astolfi D, Balbinot A, Bianco A, Pompilio A, Di Bonaventura G, D'Amario C, D'Antonio D.** 2010. Pan-azole-resistant *Candida guilliermondii* from a leukemia patient's silent funguria. Mycopathologia **169:**457–459. http://dx.doi.org/10.1007/s11046-010-9278-5.
28. **Girmenia C, Pizzarelli G, Cristini F, Barchiesi F, Spreghini E, Scalise G, Martino P.** 2006. *Candida guilliermondii* fungemia in patients with hematologic malignancies. J Clin Microbiol **44:**2458–2464. http://dx.doi.org/10.1128/JCM.00356-06.
29. **San Millán RM, Wu LC, Salkin IF, Lehmann PF.** 1997. Clinical isolates of *Candida guilliermondii* include *Candida fermentati*. Int J Syst Bacteriol **47:**385–393. http://dx.doi.org/10.1099/00207713-47-2-385.
30. **Castanheira M, Woosley LN, Diekema DJ, Jones RN, Pfaller MA.** 2013. *Candida guilliermondii* and other species of *Candida* misidentified as *Candida famata*: assessment by Vitek 2, DNA sequencing analysis, and matrix-assisted laser desorption ionization–time of flight mass spectrometry in two global antifungal surveillance programs. J Clin Microbiol **51:**117–124. http://dx.doi.org/10.1128/JCM.01686-12.
31. **Umamaheswari K, Menon T.** 2008. *Candida fermentati* from HIV patients in Chennai, South India. Int J Infect Dis **12:**e153–e154. http://dx.doi.org/10.1016/j.ijid.2008.04.007.