Medical Mycology Case Reports 21 (2018) 41–43

Contents lists available at ScienceDirect

Medical Mycology Case Reports

journal homepage: www.elsevier.com/locate/mmcr

Candida dubliniensis abscess: A clinical case and a review of the literature

Eric Y. Chang[a,*], Shaheen Fatima[a], Shuba Balan[a], Kshama Bhyravabhotla[a], Marc Erickson[a], Austin Chan[a,b], Chinedu Ivonye[a], Cinnamon Bradley[a]

[a] Department of Internal Medicine, Morehouse School of Medicine, 720 Westview Dr. S.W., Atlanta, GA 30310, USA
[b] Division of Infectious Diseases, Department of Internal Medicine, Morehouse School of Medicine, 720 Westview Dr. S.W., Atlanta, GA 30310, USA

ARTICLE INFO

Keywords:
Candida dubliniensis
Diabetes
Fungal soft tissue infection

ABSTRACT

Candida dubliniensis infections are rare in the absence of prolonged immunocompromised status or intravenous drug abuse. We present a case of a *C. dubliniensis* soft tissue abscess in a patient with uncontrolled diabetes as his only immunocompromising risk factor, treated with surgical drainage and medical management.

1. Introduction

Fungal skin and soft tissue infections are associated with significant morbidity if not identified and treated early. *Candida dubliniensis,* a lesser known cousin of the infamous *Candida albicans*, is an opportunistic fungal pathogen most commonly found in the oropharynx of immunocompromised patients or individuals with a pre-existing nidus for infection [1]. However, its presence in patients without these predisposing factors is rare, especially as an isolated abscess. We report a rare case of a *C. dubliniensis* finger abscess without concomitant fungemia or *C. albicans* co-infection in a patient with uncontrolled diabetes as his only predisposing risk factor.

2. Case

A 56 year-old man with a history of uncontrolled diabetes mellitus type 2 on home insulin presented via emergency medical services after being found down on the street by a bystander. On presentation (Day 0), the patient would open his eyes to voice but was not oriented or able to provide any collateral history. His vital signs were significant for a temperature of 93.2 F, heart rate of 105, and blood pressure of 143/105. Physical exam was limited by the patient's altered mentation and lack of participation but revealed a 2-cm laceration over the dorsum of the right index finger with an associated area of fluctuance that was draining serosanguinous fluid. There were no obvious signs of recent trauma. Labs were significant for a blood glucose of 1600, lactic acid of 6.4, leukocytosis of 17.7, and a significant anion gap metabolic acidosis (44). Urine drug screen, ethanol level, and HIV screening was negative. Urinalysis was significant for 3 + glucose and 1 + ketones. CT head without contrast was negative for any acute intracranial abnormalities. X ray of the right hand was negative for any acute fractures or osseous abnormalities. The patient was immediately transferred to the intensive care unit where he was initiated on early goal directed sepsis protocol and insulin drip for hyperosmolar hyperglycemic syndrome. Blood cultures were obtained and vancomycin and piperacillin-tazobactam were initiated. Orthopedic surgery evaluated the patient and performed a bedside incision and drainage with plans to perform a debridement in the operating room pending medical stability.

On day + 2, the patient's mentation remained without improvement. He was taken to the operating room on day + 3 for debridement of the skin and subcutaneous tissue of the right hand abscess due to concerns for systemic infection and to pursue source control and function optimization. Pus and necrotic tissue were evident in the wound with tracking in a dorsal to volar direction through the first and second web spaces. The nonviable tissue was debrided and two sets of tissue were sent for analysis via matrix assisted laser desorption ionization – time of flight mass spectrometry (MALDI-TOF MS), which grew *C. dubliniensis* susceptible to fluconazole, caspofungin, and voriconazole. However, blood cultures from admission were negative for bacteremia and fungemia.

Oral fluconazole (200 mg daily) was initiated but the patient continued to remain lethargic and have persistent purulent drainage from the finger. Repeat debridement was performed in the operating room on day + 10 for further wound assessment and to decrease the risk of secondary infection. Repeat wound cultures were obtained given concerns for possible contamination of the initial cultures but were positive for *C. dubliniensis*. However, the purulent drainage from the wound ceased. A final debridment and wound closure was performed on day + 15. By this time, the patient was alert, oriented, and back to baseline per his family. The patient was prescribed two additional weeks of

* Corresponding author.
E-mail address: eric.young.chang@gmail.com (E.Y. Chang).

https://doi.org/10.1016/j.mmcr.2018.04.003
Received 25 February 2018; Received in revised form 16 March 2018; Accepted 16 April 2018
Available online 19 April 2018

fluconazole on discharge with an outpatient appointment for further evaluation to determine if he required additional medication. However, the patient never presented for his follow-up appointment and multiple attempts to contact the patient were unsuccessful.

3. Discussion

Candida dubliniensis is a dimorphic yeast that was first isolated in Dublin, Ireland in 1995 by Sullivan et al. who described unusual isolates of oral *Candida* from HIV/AIDS patient with recurrent oral candida infections [2]. It is very closely related to its more famous cousin, *Candida albicans*, and shares many phenotypical characteristics including the ability to produce hyphae and chlamydospores [1]. However, *C. dubliniensis* is rarely found in the oral flora of healthy individuals and is significantly less likely to result in candidemia than *C. albicans* [3–5].

Given their similarities, distinguishing between these two closely related species has historically been difficult. However, multiple recent studies and advances in technology have led to increased diagnostic yield [6,7]. A recent study noted that 128 of 128 *C. dubliniensis* isolates all produced hyphal fringes after 48–72 h incubation on Pal's agar whereas 124 of 124 *C. albicans* isolates produced smooth colonies under the same conditions [8]. In addition, species specific polymerase chain reaction (PCR) and MALDI-TOF MS usage has provided more definitive, and rapid, identification methods [3,9,10]. These improvements have likely led to an increase in *C. dubliniensis* recognition and awareness.

There have been multiple, invasive *C. dubliniensis* infections described in the literature, especially in immunocompromised hosts [11,12] or IV drug abusers [13–15]. *C. dubliniensis* fungemia has been noted in allogeneic hematopoietic stem cell transplant patients as well as those who are post cytotoxic chemotherapy [11]. Disseminated *C. dubliniensis* fungemia with biopsy proven multiorgan involvement in an HIV patient has been described [16]. Other reported cases include *C. dubliniensis* spondylodiscitis in both HIV-1 patients [12] and intravenous (IV) drug abusers [15], spinal abscess in an IV drug user [12], and healthcare-associated catheter-related blood stream infections [17]. In addition, other complications include endocarditis in IV drug abusers [12] and endophthalmitis in both intravenous drug users and patients with no such history [18,19].

In general, fungal soft tissue infections are uncommon [20]. If present, *C. albicans* is the most common organism but polymicrobial infections are often seen, with staphylococcus and streptococcus the most frequently isolated pathogens [20]. However, per literature review, there has been an increase in reported *C. dubliniensis* infections, especially in immunocompromised individuals, most likely due to an improvement in identification methods via PCR and MALDI-TOF MS [3,9,10]. In the absence of an immunocompromised state, other reported risk factors include a history of surgery, the presence of indwelling catheters [16,17], or even frenulum piercings [11]. Given that necrotizing soft tissue infections have the potential to be fatal, high index of clinical suspicion and timely intervention is critical.

The use of MALDI-TOF MS for fungal pathogen identification has been gaining increasing awareness due to its ability to rapidly distinguish between closely related species such as *C. dubliniensis* and *C. albicans* [10]. Conventional methods (CI) for identification include analysis of phenotypic properties such as morphological and biochemical properties related to assimilation and fermentation of substrates, and may take up to five days for identification [10]. Given that these methods are based off of phenotypic characteristics, the results may be vague, leading to subsequent time-consuming gene analysis for confirmation. In contrast, identification through MALDI-TOF MS can be performed within 2 h from a pure culture [10]. More importantly, a recent analysis of MALDI-TOF MS vs CI revealed that when a discordance of *C. dubliniensis* identification was revealed between these two methods, confirmatory studies with PCR and gene sequencing showed that MALDI-TOF MS was able to correctly identify *C. dubliniensis* in all samples (11/11) while CI was not able to identify any (0/11), highlighting the powerful potential of MALDI-TOF MS to rapidly distinguish between these two closely related species [10].

Management of patients suspected to have *C. dubliniensis* soft tissue or skin infection begins with timely clinical judgment in addition to obtaining multiple blood cultures and initiating broad spectrum antibiotics, antifungals, and supportive management to control fungal infection spread while preventing secondary bacterial infection. Once the patient is hemodynamically stabilized, source control should be prioritized with surgical involvement for debridement as appropriate. In our patient's case, multiple debridement sessions were required for proper source control and he was treated with fluconazole per Infectious Disease Society of America (IDSA) treatment guidelines for Candida skin and soft tissue abscess treatment. A lack of improvement may signal fluconazole resistance [18], which has been reported in the literature. Other treatment options include intravenous caspofungin, anidulafungin, and other azoles.

4. Conclusion

This patient is the first reported case, to our knowledge, of an isolated *C. dubliniensis* abscess without fungemia in a patient with uncontrolled diabetes mellitus as his only immunocompromising factor, who was treated effectively with surgical debridement and fluconazole. *C. dubliniensis* should be considered an emerging threat, rather than being dismissed as a colonizer or contaminant. Proper management of patients suspected of fungal infections requires both medical and surgical management to optimize source control and prevent secondary infections.

Acknowledgements

The authors do not have any other individuals to acknowledge.

Conflict of interest

The authors have no conflicts of interest to declare.

References

[1] G. Ventolini, P. Tsai, L.D. Moore, *C. Dubliniensis* in an immunocompetent patient with metal lingual frenulum piercing, Med. Mycol. Case Rep. 14 (2016) 27–29.
[2] D.J. Sullivan, T.J. Westerneng, K.A. Haynes, D.E. Bennett, D.C. Coleman, *Candida dubliniensis* sp. nov.: phenotypic and molecular characterization of a novel species associated with oral candidosis in HIV-infected individuals, Microbiology 141 (Pt7) (1995) 1507–1521.
[3] M.A. Jabra-Rizk, J.K. Johnson, G. Forrest, K. Mankes, T.F. Meiller, R.A. Venezia, Prevalence of Candida dubliniensis fungemia at a large teaching hospital, Clin. Infect. Dis. 41 (7) (2005) 1064–1067.
[4] D.J. Sullivan, G.P. Moran, E. Pinjon, A. Al-Mosaid, C. Stokes, C. Vaughan, et al., Comparison of the epidemiology, drug resistance, mechanisms, and virulence of Candida dubliniensis and Candida albicans, FEMS Yeast Res. 4 (4–5) (2004) 369–376.
[5] M.E. Brandt, L.H. Harrison, M. Pass, A.N. Sofair, S. Huie, R.K. Li, et al., Candida dubliniensis fungemia: the first four cases in North America, Emerg. Infect. Dis. 6 (1) (2000) 46–49.
[6] M. Muthig, A. Hebestreit, U. Ziegler, M. Seidler, F.M. Muller, Persistence of *Candida* species in the respiratory tract of cystic fibrosis patients, Med. Mycol. 48 (1) (2010) 56–63.
[7] S.H. Chotirmall, E. O'Donoghue, K. Bennett, C. Gunaratnam, S.J. O'Neill, N.G. Mc Elvaney, Sputum Candida albicans presages FEV1 decline and hospitalized treated exacerbations in cystic fibrosis, Chest 138(5) (138) (2010) 1185–1195.
[8] A. Al-Mosaid, D.J. Sullivan, D.C. Coleman, Differentiation of Candida dubliniensis from Candida albicans on Pal's Agar, J. Clin. Microbiol. 41 (10) (2003) 4787–4789.
[9] A. Sebti, T.E. Kiehn, D. Perlin, V. Chaturvedi, M. Wong, A. Doney, et al., Candida dubliniensis at a cancer center, Clin. Infect. Dis. 32 (7) (2001) 1034–1038.
[10] B. Sendid, P. Ducoroy, N. Francois, G. Lucchi, S. Spinali, O. Vagner, et al., Evaluation of MALDI-TOF mass spectrometry for the identification of medically-important yeasts in the clinical laboratories of Dijon and Lille hospitals, Med. Mycol. 51 (1) (2013) 25–32.
[11] J.F. Meis, M. Ruhnke, B.E. De Pauw, F.C. Odds, W. Siegert, P.E. Verweij, Candida dubliniensis candidemia in patients with chemotherapy-induced neutropenia and bone marrow transplantation, Emerg. Infect. Dis. 5 (1) (1999&;;) 150–153.

[12] H.J. Salzer, T. Rolling, E.M. Klupp, S. Schmiedel, Hematogenous dissemination of Candida dubliniensis causing spondylodiscitis and spinal abscess in a HIV-1 and HCV-coinfected patient, Med. Mycol. Case Rep. 8 (2015) 17–20.
[13] D. Sullivan, D. Coleman, Candida dubliniensis: characteristics and Identification, J. Clin. Microbiol 36 (2) (1998) 329–334.
[14] M.J. Carr, S. Clarke, F. O'Connell, D.J. Sullivan, D.C. Coleman, B. OConnell, First reported case of endocarditis caused by candida dubliniensis, J. Clin. Microbiol. 43 (6) (2005) 3023–3026.
[15] J. Oksi, T. Finnila, U. Hohenthal, K. Rantakokko-Jalava, Candida dubliniensis spondylodiscitis in an immunocompetent patient, Case Report. Rev. Lit. Med. Mycol. Case Rep. 3 (2013) 4–7.
[16] K.M. Chan-Tack, Fatal Candida dubliniensis Septicemia in a Patient with AIDS, Clin. Infect. Dis. 40 (8) (2005) 1209–1210.
[17] C.C. Lai, H.Y. Tsai, T.C. Chang, P.R. Hsueh, Catheter-related fungemia caused by Candida dubliniensis, J. Microbiol. Immunol. Infect. 46 (4) (2013) 306–308.
[18] G.P. Moran, D.J. Sullivan, M.C. Henman, C.E. McCreary, B.J. Harrington, D.B. Shanley, et al., Antifungal drug susceptibilities of oral Candida dubliniensis isolates from human immunodeficiency virus (HIV)-infected and non-HIV-infected subjects and generation of stable fluconazole-resistant derivatives in vitro, Antimicrob. Agents Chemother. 41 (3) (1997) 617–623.
[19] D.G. Espinosa-Heidmann, B.D. McMillan, P.R. Lasala, J. Stanley, C.R. Larzo, Candida dubliniensis endophthalmitis: first case in North America, Int. Ophthalmol. 32 (1) (2012) 41–45.
[20] P.J. Buchanan, B.A. Mast, L. Lottenberg, T. Kim, P.A. Efron, D.N. Ang, Candida albicans necrotizing soft tissue infection: a case report and literature review of fungal necrotizing soft tissue infections, Ann. Plast. Surg. 70 (6) (2013) 739–741.