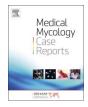
Contents lists available at ScienceDirect





Medical Mycology Case Reports

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

C. dubliniensis in an immunocompetent patient with metal lingual frenulum piercing



Gary Ventolini*, Peihsuan Tsai, Lee David Moore

Texas Tech University Health Sciences Center at the Permian Basin, 701 W 5th Street, Odessa, TX 79763, USA

A R T I C L E I N F O

Keywords: Candidas Dubliniensis Frenulum Lingual Piercing ABSTRACT

Candida spp. are opportunistic unicellular fungi, known to cause oral, vaginal, lung and occasionally systemic infections. Characteristically, they colonize the oral cavity, the mucosal surfaces of the cheek, palate, and tongue. Usually harmless, oral Candidas may become pathogenic under immunosuppressive conditions, dentures presence, or salivary flow impairment. Accurate species identification is important because *C. dubliniensis* can rapidly develop fluconazole resistance. We report *C. dubliniensis* in an immunocompetent patient with a metal lingual frenulum piercing.

1. Introduction

Candida spp. are opportunistic unicellular fungi known to cause to oral, vaginal, lung, and occasionally systemic infections. Characteristically, they colonize the oral cavity, the mucosal surfaces of the cheek, palate, and tongue. Oral Candida species are usually harmless, but they may become pathogenic under immunosuppressive conditions, with the presence of dentures, or with impaired salivary flow. Candida species of clinical interest include: *C. albicans, C. tropicalis, C. krusei, C. parapsilosis, C. dubliniensis, C. glabrata, and C. lusitaniae* [5].

Accurate identification of the species is important for appropriate treatment.

According to our literature search, this is the first reported case of *C. dubliniensis* in an immunocompetent patient with a metal lingual frenulum piercing.

Reviewing and reporting a de-identified photograph was not considered clinical research by the Texas Tech University Health Sciences Center Internal Review Board, therefore IRB approval was not required by TTUHSC.

2. Case

The patient, a 20-year-old, healthy, Caucasian woman in a stable exclusive lesbian relationship, consulted the gynecologic clinic with her partner for recurrent vaginal fungal infections on day 0. The patient also complained of under the tongue redness with a thick white plaque, stomatopyrosis (mouth burning), and soreness for 10 days. On +30 d, both their vagina cultures returned positive for *C. dubliniensis*. She had

her tongue frenulum pierced more than a year ago (Figs. 1-3).

Both their fresh normal saline wet-mount microphotographs demonstrated an abundance of chlamydospores in pairs, chains, clusters, and short pseudo hyphae typical of *C. dubliniensis*. Microbiologic culture on classical Sabouraud was done and colony morphology type in Sabouraud-triphenyltetrazolium agar confirmed *C. dubliniensis* on day +30. The patient removed her piercing on day 0 and her symptoms regressed. She was treated with topical vaginal boric acid and oral nystatin suspension beginning on day 0 for two weeks. Her symptoms improved after two weeks and her test of cure returned negative on day +60.

3. Discussion

C. dubliniensis was described in 1995 in HIV positive patients in Dublin, Ireland. Is is dimorphic yeast of the genus Candida, phenotypically similar but genotypically distinct to *C. albicans* with a distinct phylogenetic cluster in DNA fingerprinting. These fungi form dark green colonies on chromogenic Candida agar plates and are identified by Bichro-Dubli latex agglutination test. Additionally *C. dubliniensis* will not survive when cultured above 42 °C [10].

C. dubliniensis has a decreased ability to form hyphae but will form chlamydospores (in pairs, chains, and clusters). *C. dubliniensis* has the ability to rapidly develop resistance to fluconazole. This resistance is mediated by a multidrug transporter that is rapidly mobilized in vitro after fluconazole exposure. Retrospective studies revealed that *C. dubliniensis* had been commonly misidentified as *C. albicans*; therefore proper identification is mandatory in patents with oral candidiasis [11].

E-mail address: gary.ventolini@ttuhsc.edu (G. Ventolini).

http://dx.doi.org/10.1016/j.mmcr.2016.11.003

Received 10 November 2016; Received in revised form 23 November 2016; Accepted 24 November 2016 Available online 26 November 2016

2211-7539/ © 2016 The Author(s). Published by Elsevier B.V. on behalf of International Society for Human and Animal Mycology. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/BY-NC-ND/4.0/).

^{*} Corresponding author.



Fig. 1. : Tongue frenulum pierced, whitish dense exudate present.



Fig. 2.: wet-mount microphotographs showing chlamydospores in pairs, chains, clusters, and short pseudo hyphae typical of C. dubliniensis. 400X.

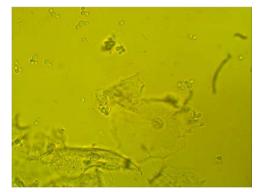


Fig. 3. : wet-mount microphotographs demonstrated an abundance of chlamydospores in pairs, chains and clusters typical of C. dubliniensis.200X.

The surface of the tongue is considered to be an ideal environment for candida colonization because of the humidity, temperature, and existence of hidden niches between the papillae of the tongue [2]. This finding was supported by a study which concluded that the tongue is the primary oral reservoir for candida species [12]. As the most common oral candidiasis typically affects the surface of the tongue, it can be surmised that the tongue may harbor many of the potentially pathogenic candida species responsible for oral candida infection [13].

The stratified squamous epithelium of the buccal mucosa was found to be less ideal for candida colonization compared to the papillary surface of the tongue. A possible reason for this is the continuous shedding by exfoliation of the buccal mucosa, as buccal epithelial cells have an estimated turnover rate of 5-6 days [14].

The unique qualities of the squamous epithelium of the palate, which is keratinized and is also less permeable compared to buccal mucosa, may be why the palate was found to have a lower percentage of candida occurrences compared to the tongue and buccal mucosa [15]. Furthermore, as different oral sites have different cell surface receptors, it is also a possibility that the cell surface receptors of the palate favor the cell surface adhesions of bacterial species over candida species [15].

Hennequin-Hoenderdos et al. systemically searched the literature for case reports concerning adverse effects associated with oral and peri-oral piercings regarding general and oral health [6]. They concluded that oral and peri-oral piercings were not risk free [1,3]. Common side effects include: swelling, pain, infection, hypersalivation, nerve damage, allergic reaction to the jewelry, and prolonged bleeding [7]. The procedure of oral piercing is done without anesthesia. A sharp needle is used for piercing followed by insertion of the jewelry. A person receiving oral piercing must take diligent care to prevent infection. In addition, there are risks attributable to the fact that the body piercing industry is minimally regulated. Many body piercers lack proper training as they often learn the practice via videos, books, observation, and trial-and-error procedures [7]. Piercers are also often unlicensed and lack an adequate amount of relevant medical and anatomical knowledge, which put their clients at higher risk for severe complications [7,4].

In our case, the patient's oral piercing had been uninfected and uncomplicated for a year prior to her visit to the clinic. With recurrent vaginal candidiasis, proper identification of the genus is mandatory in order to provide optimal treatment [9]. Similarly, diagnostic efforts for recurrent or coexistant oropharyngeal candidiasis should also be genus specific. According to Sullivan et al., epidemiological data on the prevalence of *C. dubliniensis* suggested that it is relatively rare in the normal oral flora of immunocompetent individuals, but is much more common in immunocompromised patients, especially in those infected with HIV [8]. Our patient was otherwise healthy and in an exclusive lesbian relationship. Therefore, we believe that oral piercings, especially frenulum tongue piercings, may be an independent risk factor for developing non-albicans oropharyngeal candidiasis. In order to provide the best treatment for these patients, clinicians should strive to identify the specific candida species.

Conflicts of interest

There are none.

Acknowledgements

Thank you to Melissa Waggoner, Senior Editor at TTUHSC at the Permian Basin, for editing this report.

References

- M.Y.1 Herskovitz, D. Goldsher, R. Finkelstein, Y. Bar-Vai, M. Constantinescu, G. Telman, Multiple brain abscesses associated with tongue piercing, Arch. Neurol. 66 (10) (2009) 1292.
- [2] Y.1 Zadik, S. Burnstein, E. Derazne, V. Sandler, C. Ianculovici, T. Halperin, Colonization of Candida: prevalence among tongue-pierced and non-pierced immunocompetent adults, Oral. Dis. 16 (2) (2010) 172–175.
- [3] D.1 Siebolz, E. Hornecker, R.F. Mausberg, Microbiological findings at tongue piercing sites: implications to oral health, Int J. Dent. Hyg. 7 (4) (2009) 256–262.
 [4] A. Kraytem, P.Y. Uldry, J.V. Lopez-Liuchi, Tattoos, body piercing and thrush: a
- [4] A. Kaytein, F.T. Oldy, J.V. Edgez-Intelli, Fattoos, body pieteing and intustical lesson on the harmful effects of lost objectivity, Mayo Clin. Proc. 74 (8) (1999) 844.
 [5] R.A. Zahir, W.H. Himratul-Aznita. Distribution of Candida in the oral cavity and its
- [5] R.A. Zahir, W.H. Himratul-Aznita, Distribution of Candida in the oral cavity and its differentiation based on the internally transcribed spacer (its) regions of rDNA, Yeast 30 (1) (2013) 13–23.
- [6] N.L.1 Hennequin-Hoenderdos, D.E. Slot, G.A. Van der Weijden, Complications of oral and peri-oral piercings: a summary of case reports, Int. J. Dent. Hyg. 9 (2) (2011) 101–109.
- [7] K.M. Janssen, B.R. Cooper, Oral piercing: an overview, Internet J. Allied Health Sci. Pract. 6 (3) (2008) 6.
- [8] 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 years Res. 4 (4–5) (2004) 369–376.
- [9] J.D. Sobel, Recurrent vulvovaginal candidiasis, Am. J. Obstet. Gynecol. 214 (1) (2016) 15–21.
- [10] G.D. Gilfillan, D.J. Sullivan, K. Haynes, T. Parkinson, D.C. Coleman, N.A. Gow,

Candida dubliniensis: phylogeny and putative virulence factors, Microbiol 144 (pt 4) (1998) 829–838.

- [11] L.M. Mesa, N. Arcaya, O. Cañas, Y. Machado, B. Calvo, Phenotypic evaluation to differentiate Candida albicans from Candida dubliniensis, Rev. Ibero. Micol. 21 (3) (2004) 135–138.
- [12] T.M. Arendorf, D.M. Walker, The prevalence and intra-oral distribution of Candida albicans in man, Arch. Oral. Biol. 25 (1) (1980) 1–10.
- [13] Darwazeh AM1, P.J. Lamey, L.P. Samaranayake, T.W. MacFarlane, B.M. Fisher,

S.M. Macrury, et al., The relationship between colonization, secretor status and invitro adhesion of Candida albicans to buccal epithelial cells from diabetics, J. Med. Microbiol. 33 (1) (1990) 43–49.

- [14] D.1 Harris, J.R. Robinson, Drug delivery via the mucous membranes of the oral cavity, J. Pharm. Sci. 81 (1) (1992) 1–10.
- [15] R.J.I Gibbons, Bacterial adhesion to oral tissues: a model for infectious diseases, J. Dent. Res. 68 (5) (1989) 750–756.