Clinics in Dermatology (2016) **34**, 487–494

ELSEVIER

Clinics in Dermatology

Oral candidiasis

Jillian W. Millsop, MD, MS, Nasim Fazel, MD, DDS*

Department of Dermatology, University of California at Davis School of Medicine, Sacramento, CA

Abstract Oral candidiasis (OC) is a common fungal disease encountered in dermatology, most commonly caused by an overgrowth of *Candida albicans* in the mouth. Although thrush is a well-recognized presentation of OC, it behooves clinicians to be aware of the many other presentations of this disease and how to accurately diagnose and manage these cases. The clinical presentations of OC can be broadly classified as white or erythematous candidiasis, with various subtypes in each category. The treatments include appropriate oral hygiene, topical agents, and systemic medications. This review focuses on the various clinical presentations of OC and treatment options.

Introduction

Candida is present in the normal oral flora of healthy individuals; it is estimated to be present in 45-65% of healthy infants and 30-55% of healthy adults.[1] A variety of both systemic and local factors can cause an overgrowth of *Candida* species in the oral mucosa, making oral candidiasis (OC) an important oral dermatologic entity. In humans, the most common *Candida* species found in both healthy oral mucosa and in OC is *C albicans* due to its adherence properties and greater level of pathogenicity.[2] *C albicans* is a dimorphic yeast, which may exist in both hyphal and yeast forms depending on the environment.[3] *C albicans* is isolated in more than 80% of oral lesions.[4,5] Other species that have been implicated include *C dubliniensis, C glabrata, C krusei, C kefyr, C parapsilosis, C stellatoidea*, and *C tropicalis*.[6,7]

A variety of predisposing local and systemic factors lead to the transition from commensal to pathogenic *Candida*. Local factors include the use of dentures, corticosteroid inhalers, and xerostomia, whereas systemic factors include immunosuppressed states, such as human immunodeficiency virus (HIV); leukemia; malnutrition; decreased immunity secondary to age; endocrine dysfunction such as diabetes; systemic chemotherapy; radiation therapy; and the use of systemic corticosteroids, immunomodulatory drugs, xerogenic drugs, and broad-spectrum antimicrobials.[8–16] Recently, psoriasis has been described as a predisposing factor for OC.[17]

Clinical presentations

OC presents in one of two forms: white or erythematous. White OC is characterized by lesions that are white, including pseudomembranous candidiasis and hyperplastic candidiasis. Erythematous OC is characterized by lesions that are red, including acute atrophic candidiasis, chronic atrophic candidiasis, median rhomboid glossitis, angular cheilitis, and linear gingival erythema. In addition, there are three forms of oral candidiasis that do not fit within these clinical categories:

* Corresponding author. Tel.: +1 916 734 6111; fax: +1 916 442 5702.
E-mail address: Nfazel@ucdavis.edu (N. Fazel).

http://dx.doi.org/10.1016/j.clindermatol.2016.02.022
0738-081X/

chronic mucocutaneous candidiasis, cheilocandidiasis, and chronic multifocal candidiasis.

White oral candidiasis

Pseudomembranous candidiasis: The classic presentation

Pseudomembranous candidiasis is the classic presentation of OC more commonly known as "thrush." One third or more of all cases of OC present as pseudomembranous candidiasis,[18] with confluent white plaques on the tongue, buccal mucosa, hard palate, soft palate, and oral pharynx (Figure 1).[19] Typically, patients are asymptomatic. Symptomatic patients may describe a burning oral sensation, changes in taste perception, a sour taste, or tendency for easy bleeding at the affected sites.[19] These lesions may be acute or chronic and are caused by the overgrowth of yeast in the oral mucosa with epithelial cell desquamation and accumulation of keratin, fibrin, necrotic tissue, and fungal hyphae.[20,21] A classic marker for this disease is the ability to easily wipe off the white plaques with gauze, leaving behind an erythematous surface.[3,13] This disease is more commonly found in individuals who are immunosuppressed, such as patients taking immunosuppressive medications, having had tissue or organ transplants, using corticosteroid inhalers, developing malignancies, and having immune diseases, such as HIV patients. Both the elderly and infants are susceptible.[20,22]

Hyperplastic candidiasis: Mimicker of leukoplakia

Hyperplastic candidiasis presents as well-circumscribed, slightly elevated, white plaques most commonly adherent to the buccal mucosa and that may involve the labial commissures (Figure 2).[19] The lesions may be small translucent to large opaque plaques.[13,14,23] The lesions may be nodular or speckled in appearance.[3,24] Hyperplastic candidiasis is less commonly found on the lateral tongue or the palate. It can be distinguished from pseudomembranous candidiasis because hyperplastic candidiasis cannot be easily wiped off. It is, however, difficult to distinguish hyperplastic candidiasis from leukoplakia. Hyperplastic candidiasis has been associated with increased malignant changes compared with noncandidal leukoplakia.[23] Fortunately, this form is an uncommon presentation of OC.

Fig. 1 Pseudomembranous candidiasis. Confluent white plaques on the dorsal tongue. (Courtesy Marc A. Silverstein, MD, Sacramento, CA.)

Fig. 2 Hyperplastic candidiasis. White plaque with fissuring involving the left labial commissure.

Erythematous oral candidiasis

Acute atrophic candidiasis: Erythematous oral patches

Acute atrophic candidiasis presents as erythematous patches, most commonly on the palate, especially in HIV patients.[18] Erythematous patches may also present on the buccal mucosa or dorsal tongue.[18] There may be accompanying atrophy of the lingual papillae. Patients may complain of a burning sensation in the mouth and soreness of the lip and tongue.[18,25] It is also referred to as antibiotic sore mouth because it may occur after use of broad-spectrum antimicrobials, which can increase the risk for overgrowth of *C albicans* by decreasing the normal bacterial flora in the mouth. Other predisposing factors for the development of acute atrophic candidiasis include use of corticosteroids, HIV disease, uncontrolled diabetes mellitus, iron deficiency anemia, and vitamin B_{12} deficiency.[14,26,27]

Chronic atrophic candidiasis: Denture stomatitis

Chronic atrophic candidiasis presents in patients with dentures. Poorly fitted dentures occlude the oral mucosa and inhibit salivary flow, leading to *Candida* overgrowth. Lesions are erythematous, edematous, and characteristically restricted to the oral mucosa in contact with the dentures.[28,29] Although often asymptomatic, patients may complain of a sore mouth or a burning sensation in the mouth. Often this condition presents with angular cheilitis.

There are three recognized clinical classifications of chronic atrophic candidiasis.[25,30] Type I is limited to local signs of disease with pinpoint petechial hemorrhage and local