Oral Candidiasis: A brief Review

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***Abstract*: Oral Candidiasis or thrush is an infection of the oral cavity caused by Candida albicans. In healthy individuals, candida exists harmlessly in mucus membranes such as our tears, eyes, gastrointestinal tract, mouth, nose etc. It is known as "beneficial flora" and has a useful purpose. When an imbalance in the normal flora occurs it causes an overgrowth of candida albicans. The term is candidiasis or thrush. When this happens, it can create a widespread havoc to our overall health and well-being of our body. Oral Candidiasis is generally obtained secondary to immune suppression depending upon whether a patient's oral cavity has decreased immune function or if it is systemic.**

This article aims to illustrate the clinical significance of Candidiasis, its pathogenesis and how clinical and mycological findings can be used together to establish a proper diagnosis in order to provide a suitable treatment.

Index Terms: Oropharyngeal Candidiasis, Candida albicans, Oral Candidiasis, Oral lesions, CHROMagar Candida

INTRODUCTION

Oral candidiasis is a common opportunistic infection of the oral cavity caused by an overgrowth of *Candida* species, the commonest being *Candida albicans*. The incidence varies depending on age and certain predisposing factors. There are three broad groupings consisting of acute candidiasis, chronic candidiasis, and angular cheilitis. Risk factors include impaired salivary gland function, drugs, dentures, high carbohydrate diet, and extremes of life, smoking, diabetes mellitus, Cushing's syndrome, malignancies, and immunosuppressive conditions. Management involves taking a history, an examination, and appropriate antifungal treatment with a few requiring samples to be taken for laboratory analysis. In certain high risk groups antifungal prophylaxis reduces the incidence and severity of infections. The prognosis is good in the great majority of cases. **[1]**

ETIOLOGY

Candida albicans (*C. albicans*) accounts for around 80% of infections and can colonise the cavity, either alone or in combination with non-albican species, including *Candida glabrata* and *Candida tropicalis.* **[2]**

The additional important species isolated from clinical infections include, *C. glabrata, C. guilliermondii, C. krusei, C. lusitaniae, C. parapsilosis, C. pseudotropicalis, C. stellatoidea*, and *C. tropicalis* (Crist et al., 1996).

PATHOGENESIS

Although Candida is seen as a commensal oral flora, the occurrence of candidiasis occurs in selective individuals predominantly those with compromised immune response or those with prostheses coupled with poor oral hygiene habits etc. The conversion of this relatively innocuous commensal organism to the pathogenic stage therefore depends on an interaction between the organisms with the host environment and the resident bacteria present and can be triggered by serum, proline, N-acetyl glucosamine, and different carbon sources etc.

Role of Hyphae in Pathogenesis of Candida-Formation of hyphae results in the development of fungal biofilms which promotes adhesion to biotic or abiotic surfaces and tissue penetration leading to infection. These biofilms are relatively resistant to treatment with antifungal agents and thus pose clinical problems. **[3]**

EPIDEMIOLOGY

Oral candidiasis is frequent in the extremes of age. Approximately 5–7% of infants develop oral candidiasis.(Infants show initial signs and symptoms of immunosuppression with the presenting features of diarrhoea, rashes and hepatosplenomegaly) Its prevalence in AIDS patients is estimated to be 9–31% and close to 20% in cancer patients (Lalla et al., 2013). The oral carriage of candida organisms is reported to be 30–45% in the general healthy adult population. **[4]**

PREDISPOSING FACTORS

Impaired salivary gland function can predispose to oral candidiasis. Secretion of saliva causes a dilutional effect and removes organisms from the mucosa. Antimicrobial proteins in the saliva such as lactoferrin, lysozyme, histidine-rich polypeptides, and specific anti candida antibodies, interact with the oral mucosa and prevent overgrowth of candida. Therefore conditions such as Sjögren's syndrome, radiotherapy of the head and neck, or drugs that reduce salivary secretions can lead to an increased risk of oral candidiasis. **[5]**

Drugs such as inhaled steroids have been shown to increase the risk of oral candidiasis by possibly suppressing cellular immunity and phagocytosis. The local mucosal immunity reverts to normal on discontinuation of the inhaled steroids.

Dentures predispose to infection with candida in as many as 65% of elderly people wearing full upper dentures. Wearing of dentures produces a microenvironment conducive to the growth of candida with low oxygen, low pH, and an anaerobic environment. This may be due to enhanced adherence of *Candida* species to acrylic, reduced saliva flow under the surfaces of the denture fittings, improperly fitted dentures, or poor oral hygiene.

Other factors are smoking, diabetes, Cushing's syndrome, immunosuppressive conditions such as HIV infection, malignancies such as leukemia, and nutritional deficiencies—vitamin B deficiencies have been particularly implicated. Ninane found that 15%–60% of people with malignancies will develop oral candidiasis while they are immunosuppressed. **[6]**

CLASSIFICATION

Proposed revised classification of oral Candidiasis **[7]**

Primary oral candidosis (Group I)

- Acute
  - Pseudomembranous
  - Erythematous
- Chronic
  - Erythematous
  - Pseudomembranous
  - Hyperplastic
  - Nodular
  - plaque-like
- Candida-associated lesions
  - Angular cheilitis
  - Denture stomatitis
  - Median rhomboid glossitis
- keratinized primary lesions superinfected with Candida
  - Leukoplakia
  - Lichen planus
  - Lupus erythematosus.

Secondary oral candidiasis (Group II)

- Oral manifestations of Systemic mucocutaneous.
- Candidosis (due to diseases such as thymic aplasia and candidiasis endocrinopathy syndrome).

CLINICAL FEATURES

Oropharyngeal candidiasis manifests as creamy white lesions, usually on the tongue or inner cheeks. Sometimes it may spread to the roof of the mouth, gums or tonsils, or the back of the throat. Candida albicans is the most commonly implicated organism in this condition. The prevalence of oropharyngeal candidiasis remains very high in immunocompromised patients, and three clinical forms are generally encountered among which pseudomembranous, atrophic erythematosus, and hyperplastic candidiasis. **[8]**

The spectrum of oral lesions of candidiasis varies from large white plaques of pseudomembranous candidiasis on the buccal mucosa to erythematous lesions of chronic atrophic candidiasis on the palate. **[9]**

HISTOPATHOLOGY

In candidiasis, sections show predominantly spongiotic changes in the epidermis with irregular acanthosis, mild spongiosis and inflammatory changes. In the superficial epidermis, the characteristic feature is the presence of neutrophils in the stratum corneum and upper layers of the epidermis. The neutrophils may form small collections (spongiform postulation) which resembles impetigo or psoriasis. **[10]**

CLINICAL AND MICROBIOLOGICAL DIAGNOSIS

The diagnosis of oral candidiasis is fundamentally clinical. Microbiological techniques are used when the clinical diagnosis needs to be confirmed, for establishing a differential diagnosis with other diseases, and in cases characterized by resistance to antifungal drugs. Biopsies in turn are indicated in patients with hyperplastic candidiasis. Staining (10% KOH) and culture (Sabouraud dextrose agar) are the methods most commonly used for diagnosing primary candidiasis. Identification of the individual species of Candida is usually carried out with CHROMagar Candida®. For the diagnosis of invasive candidiasis, and in cases requiring differentiation between C. albicans and C. dubliniensis, use is made of immunological and genetic techniques such as ELISA and PCR. **[11]**

Conventional oral specimens for recovery of yeasts are swabs and smears. Oral rinses and imprint/impression cultures can also be used. Yeasts grow well at room temperature and may multiply in specimens under transport. Direct smears examined for blastospores, hyphae, and inflammatory cells ensure rapid presumptive diagnosis. Fungal identification requires culture, preferably on different media and at different temperatures to ensure recognition of all species present. YM agar supplemented with 0.01% aniline enables detection of Candida albicans and C. parapsilosis on primary plates through fluorescence. Mycologic findings should be interpreted together with clinical findings. **[12]**

TREATMENT

Depending on its virulence, location and type of candidiasis, appropriate treatment is provided. First has been the use of conservative measures before starting drug treatment, promoting good oral hygiene along with removing the dentures at night, thereby it will benefit the removal of the biofilm layer generated in the prosthetic surface. Dentists should also correct the predisposing factors and underlying diseases and try to promote the use of oral antiseptic and antibacterial rinses such as Chlorhexidine or Hexetidine. Regarding the pharmacological treatment of candidiasis, it can be distinguished between two procedures. Topical drugs, which are applied to the affected area and treat superficial infections and systemic drugs that are prescribed when the infection is more widespread and has not been enough with the topical therapy. **[13]**

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