Review

Oral Candidiasis – A Review

Prasanna Kumar Rao

Reader Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Deralakatte, Nithyanandanagar Post, Mangalore, Karnataka, India. E-mail: drjpkrao@gmail.com

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Oral candidiasis, a frequent and important fungal condition of the oral cavity is caused by Candida species. There are few local factors that make the oral tissues susceptible to Candida infection. These factors include acid saliva, xerostomia, night use of prosthetic dentures, tobacco, carbohydrate rich-diets and patients that receive radiotherapy and chemotherapy in maxillofacial structures. Maintenance of oral hygiene and early diagnosis of this condition is very important.

KEY WORDS: Candidiasis, Candida albicans, oral thrush.

INTRODUCTION:

Oral candidiasis is one of the common fungal infection affecting the oral mucosa. These lesions are caused by the yeast Candida albicans. Candida albicans are one of the components of normal oral microflora and around 30% to 50% people carry this organism. Rate of carriage increases with age of the patient. Candida albicans are recovered from 60% of dentate patients mouth over the age of 60 years. There are nearly five types of candida species which are seen in the oral cavity (Gravina et al. 2007).

They are,

1. Candida albicans
2. Candida tropicalis
3. Candida krusei
4. Candida parapsilosis
5. Candida guilliermondii

Among these five types, Candida albicans are commonly seen in the oral cavity. Candida albicans is a dimorphic fungus that causes severe opportunistic infections in humans (Molero et al., 1998). An interesting feature of Candida albicans is its ability to grow in two different ways, reproduction by budding, forming an ellipsoid bud, and in hyphal form, which can periodically fragment and give rise to new mycelia, or yeast-like forms (Cutler, 2001). Yeast is innocuous and hyphal forms are associated with invasion of host tissue (Figure 1).

There are three general factors which helps the Candida albicans infection to develop in the patient’s body. They are:

1. Immune status of the patient
2. Oral mucosal environment
3. Strain of Candida albicans

The main factors which increase susceptibility of oral candidiasis are (Akpan and Morgan, 2002).

i. Immunosuppression
ii. Endocrinopathies
iii. Nutritional deficiencies
iv. Malignancies
v. Dental prosthesis
vi. Epithelial alteration
vii. High carbohydrate diet
viii. Infancy and old age
ix. Poor oral hygiene
x. Heavy smoking

Xerostomia: Saliva contains IgA which inhibits binding of Candida albicans to mucosal surfaces. It also provides a flushing action which removes Candida albicans from oral cavity. In case of xerostomia both these actions are absent because of lack of saliva production, so chances of candidiasis is more in oral cavity. Xerostomia is also
seen in case of anticancer treatment and irradiation which increases the proliferation of candidal cells and resistance of Candida cells to antifungal drugs. Xerostomia is also seen in case of Sjogren’s syndrome because of lymphocytic infiltration and destruction of salivary glands.

**Diabetes Mellitus:** Growth of Candida albicans thrives on increased levels of glucose in saliva which increases the ability of Candida albicans to adhere to oral mucous membranes.

**Medicines:** Prolonged use of antibiotics depletes normal oral flora and enables proliferation of Candida albicans in the oral cavity. In asthmatic patients due to use of steroid inhalers. Steroid aerosols interfere with the normal balance of microflora and favor the proliferation of candida albicans. Whereas systemic steroids cause suppression of the immune system.

**Classification:** Oral candidosis are mainly classified in to primary and secondary infections (Greenberg et al., 2008).

1. Primary oral candidosis
   a. Acute form
   i. Pseudomembranous
   ii. Erythematous

b. Chronic form
   i. Erythematous
   ii. Pseudomembranous
   iii. Plaque like
   iv. Nodular
   c. Candida associated lesions
   i. Denture stomatitis
   ii. Angular chealosis
   iii. Median rhomboid glossitis

2. Secondary oral candidosis
   a. Oral manifestation of systemic mucocutaneous candidosis
      i. Familial mucocutaneous candidiasis
      ii. Diffuse chronic mucocutaneous candidiasis
      iii. Familial mucocutaneous candidiasis
      iv. Chronic granulomatous disease
      v. Candidosis endocrinopathy syndrome
      vi. Acquired immune deficiency syndrome (AIDS)

Acute form of pseudomembranous candidiasis affects patients under antibiotics, immunosuppressant drugs etc. Clinically there will be scrapable pseudomembrane formation with inflamed mucosa below. Chronic form also referred to as atropic oral candidiasis There will be erythematous surface with increased vascularization. Plaque type was earlier known as candidal leukoplakia. Clinically there will be white plaque. Both the chronic plaque type and nodular type is associated with
malignant transformation.

The pathogenetic theory in denture stomatitis states that, the presence of bacteria as Streptococci and Actinomycetes induce the organism to produce proteases as (gA) and enzymes as amino-peptidases, hyaluronidase, chondroitinases and neuraminidases, enable to degrade the oral epithelium. Presence of these products, storing in close contact with the oral mucosa, determine an increase in the inflammatory exudate that favors the bacterial colonization and also the yeast proliferation since Candida colonizes more easily the mucosa in contact with the denture surface as compared to the rest of the buccal mucosa. The proteases can increase the pathogenic potential of the bacterial substances, leading to the destruction of the salivary immunoglobulins. A delayed hypersensitivity reaction towards Candida albicans contributes to the inflammatory reaction and the exfoliation of the epithelial cells, leading to the epithelial atrophy. It is a typical feature of the denture stomatitis. Denture stomatitis are devided in to three clinical types, they are (Salerno et al., 2011).

1. Type I  Localized pinpoint hyperaemia
2. Type II  Erythematous lesion involving denture covered mucosa
3. Type III Papillary type involving central part of hard palate & alveolar mucosa

These conditions are treated by antifungal drugs (Nystatin, Amphotericin-B, Miconazole and Fluconazole) and disinfectants (0.2% Chlorhexidine gluconate mouth wash 3 or 4 times a day), Microwave irradiation (exposure to the microwaves was able to cause the cell death of Candida albicans) and scrupulous removal of denture plaque. Even though denture stomatitis is asymptomatic, it should be treated as it may act as reservoir for infections which can become extensive and lead to the resorption of the alveolar bone. The most effective treatment is the eradication and control of the microbial plaque (Webb et al., 1998, Agha-Hosseini, 2006).

**Diagnosis:** Oral candidiasis is mainly diagnosed based on clinical sign and symptoms. Additional tests includes (Agha-Hosseini, 2006),
1. Exfoliative cytology
2. Culture (Figure 2)
3. Tissue biopsy

**ID – reaction:** It is a secondary skin response which is a localised or generalized sterile Vesicullopapular rash. These rashes resolve with treatment

**Management**

Always continue the treatment for 2 weeks after resolution of the lesions. When the topical therapy fails then one has to start systemic therapy because failure of drug response is the initial sign of underlying systemic disease. Follow-up appointment after 3 to 7 days is important to check the effect of drugs. Main Goals of treatment are (Parihar, 20111),
1. To identify & eliminate possible contributory factors
2. To prevent systemic dissemination
3. To eliminate any associated discomfort
4. To reduce load of candida

Treatment options are mainly categorized in to two lines, primary & secondary line of treatment

**Primary line of treatment:**

Nystatin is the drug of choice as a primary line of treatment. Usually for the mild and localized candidiasis this primary line of treatment is used other drugs includes Clotrimazole which can be taken as is Lozenges and Amphotericin B as oral suspension (Pappas, 2004).

**Nystatin:** It is another drug which can be used as a primary line of treatment. It is available as cream & oral suspensions. It is to be applied four times a day and allowed to act approximately for two minutes in the oral cavity and then it is to be swallowed. There is no significant drug interaction or side effects. (Figure 3)

**Amphotericin B:** This drug is available as Lozenge (Fungilin 10mg) and oral suspension 100mg/ml which is to be applied 3 to 4 times daily. Amphotericin-B inhibits the adhesion of Candida to epithelial cells. The side effect of the drug is nephrotoxicity.

**Clotrimazol:** This drug decreases fungal growth by inhibiting the synthesis of ergosterol. It is not indicated for systemic infection. This drug is available in Creams and Lozenge 10mg. main side effects of this drug is unpleasant mouth sensation, increases liver enzyme levels, nausea and vomiting.

**Second line of treatment:**

The second lines of treatment are used for severe, localized, immunosuppressed patients and patients who respond poorly to primary line of treatment. Drugs mainly used in second line of treatment are, (Pappas, 2004).

1. Ketoconazole
2. Fluconazole
3. Itraconazole

**Ketoconazole:** It blocks ergosterol synthesis in fungal cell membrane and is absorbed from the gastro intestinal tract and metabolized in the liver. The dosage is 200-400 mg tablets once or twice daily for 2 weeks. Side effects are nausea, vomiting, liver damage and also it interacts with anticoagulants.

**Fluconazole:** This drug inhibits fungal cytochrome P450 sterol C-14 alpha demethylation. It is used in oropharyngial candidosis and dosage is 50 – 100mg
capsule once a day for 2-3 weeks. Main side effects are nausea, vomiting and headache. It interacts with anticoagulants and this drug is contraindicated in pregnancy, liver & renal disease.

**Itraconazole:** It is one of the broad spectrum antifungal agents and contraindicated in pregnancy & liver disease. The dosage of the drug is 100 mg capsule once a day for 2 weeks. The main side effects are nausea, neuropathy & rashes.

**CONCLUSION**

The prognosis of oral candidiasis is good when the predisposing factors associated with this infection are eliminated. When the systemic predisposing factors arise even patient with primary candidiasis are also at risk. In most of the cases oral candidiasis is a cause of secondary superficial infection which can easily be resolved with antifungal therapy.

**REFERENCES:**


