

A Review on Denture Stomatitis

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ABSTRACT

Background: The most common adverse sequelae of wearing an ill fitting or a poorly maintained removable dental prosthesis is denture stomatitis. Denture stomatitis is an erythematous inflammatory reaction of the denture bearing mucosa, usually palatal mucosa. The prevalence of denture stomatitis is about 50% among complete denture wearers. The etiology is multi-factorial with a lot of predisposing factors. The commonly associated pathogenic organism is *Candida albicans*. This article reviews the etio-pathogenesis, diagnosis, prevention and treatment of denture stomatitis.

Keywords: Denture stomatitis, *Candida albicans*, predisposing factors, antifungal agents.

INTRODUCTION

Denture stomatitis refers to an inflammatory state of denture bearing mucosa, characterized by chronic erythema and edema of part or all the mucosa beneath dentures¹. It is called under several titles as denture induced stomatitis, denture sore mouth, denture stomatitis, inflammatory papillary hyperplasia and chronic atrophic candidosis². Denture stomatitis has been reported in 11 – 67% of complete denture wearers and is more common in women than men³. Palatal mucosa is the most common site of occurrence³. The principle causative agent of denture stomatitis is the *Candida* species, especially *Candida albicans*⁴. It has a multi-factorial etiology with the predisposing factors playing a significant role. Various systemic and local predisposing factors which may or may not be related to dentures, make the condition more susceptible to denture stomatitis. In majority of cases, antifungal therapy along with proper oral hygiene and denture hygiene maintenance effectively treats denture stomatitis³.

Classification:

Newton was the first to classify denture stomatitis in 1962⁵. According to his classification, three types of denture stomatitis can be distinguished based on clinical appearance.

Type I – a localized simple inflammation or a pinpoint hyperemia

Type II – an erythematous or generalized simple type seen as more diffuse erythema involving a part or entire denture covered mucosa.

Type III – a granular type (inflammatory papillary hyperplasia) commonly involving the central part of hard palate and the alveolar ridges.

Budtz – Jorgensen and Bertram in 1970 classified denture stomatitis based on the type of inflammation⁶.

Type I – simple localized inflammation

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Type II – simple diffuse (generalized) inflammation

Type III – granular inflammation

Bergendal and Issacson classified it into three types in 1982⁷.

Type 1 – local inflammation which appear as red erythematous found usually around the minor palatal salivary glands

Type 2 – diffuse reddening referred to a diffuse, hyperemic, smooth and atrophic mucosa extending over the entire denture area

Type 3 – granulated and characterized by hyperemic mucosa with a nodular appearance in centre part of palate.

All the three classifications explained the same types with slightly different terminologies.

Etiopathogenesis:

Denture stomatitis is multifactorial in nature. *Candida Albicans* of *Candida* species is the principle pathogen associated to denture stomatitis, though *C. Dubiliniensis*, *C. Parapsilosis*, *C. Krusei*, *C. Tropicalis* and *C. Glabrata* have been isolated from the inflammatory lesion⁸. The direct predisposing factor for *Candida* associated denture stomatitis is the presence of dentures in oral cavity². *Candida albicans* is a commensal in oral cavity of adults and children, without any clinical disease⁹. These microorganisms are encountered in the dentition, tongue, cheeks, palatal mucosa, restorative materials and oral prosthesis¹⁰. Certain local and systemic predisposing factors transform the commensal to pathogenic form, providing an oral environment adequate to adhesion of microorganisms to denture surface and mucosal epithelial cells¹¹. This is followed by cell multiplication, organization and secretion of extracellular matrix, resulting in the formation of biofilm, a three dimensional structure extremely organized¹². *Candida albicans* is found in two major forms, yeast form and hyphae form. Yeast is usually associated to commensal mucosa. The conversion of yeast to hyphae is commonly related to invasion of superficial cells of oral epithelium, leading to clinical infection¹³. In immunocompromised host cells, hyphae becomes active and starts secreting several hydrolytic enzymes such as proteinases and

phospholipases which help in their adherence to host cells and digesting their cell walls for nutrient supply to assist further invasion¹⁴

Predisposing factors:

Various systemic and local factors change the commensal to pathogenic form, favoring the development of the disease. The systemic factors influence the host defense mechanism, while the local factors provide an adequate oral environment¹⁵.

Systemic factors:

There are many systemic conditions that influence the development of *Candida* associated denture stomatitis. Old age², malnutrition², diabetes mellitus¹⁶, patients undergoing chemotherapy and radiation therapy¹⁷, hemophilia¹⁸, nutritional deficiencies (iron, folate or vitamin B12)², immunosuppression¹⁸ and use of broad spectrum antibiotics² can modulate the host parasite relationship and increase the susceptibility to *Candida* associated denture stomatitis.

Local factors:

Local factors can be denture related or non denture related factors.

Denture related local factors:

The presence of *Candida albicans* in oral cavity in patients with dentures was higher than in patients who do not use dentures¹⁹.

Poor denture hygiene:

It is a major factor in *Candida* associated denture stomatitis. Poor denture maintenance provides opportunities for food lodgment which favours biofilm development and *Candida* colonization²⁰.

Denture trauma:

Denture trauma may be due to ill fitting dentures or dentures fabricated with incorrect vertical and horizontal jaw relations²¹. Trauma plays a vital role in type I denture stomatitis and given least importance in type II and type III denture stomatitis²². Trauma could act as a co factor that favours the adhesion and penetration of the yeast, sustains phlogosis of the palate and increase the

permeability of the epithelium to toxins and soluble agents produced by *Candida*²³.

Improper denture usage:

Nocturnal and continuous 24 hours of denture usage⁽²⁴⁾ is associated with an increase in frequency and density of *Candida albicans* on the fitting surface of maxillary dentures compared to that in an intermittent denture wearer⁷. Continuous wearing of dentures reduces the protective covering of saliva; prevent proper oxygenation of palatal mucosa leading to local trauma to the mucosa²⁵.

Denture age:

As age advances, the dentures become ill fitting due to residual ridge resorption. Poorly fitting denture helps in accumulation of plaque and microbial pathogen²⁶.

Denture lining materials:

Soft denture liners and tissue conditioners are used in management of traumatized oral mucosa to provide a cushion between the hard denture base and supporting tissue⁽²⁷⁾. Various studies show overgrowth of fitting surface by *Candida albicans* hypae even though lining materials have tissue tolerance. This growth destroys the surface properties of liner and leads to trauma to oral tissues⁽²⁸⁾.

Surface texture and permeability of denture base:

Irregularities like micro-pits and micro-porosities in the tissue surface of dentures help in colonization of *Candida* species. Substrate surface properties as surface charge, surface free energy, hydrophobicity and roughness have all been reported to influence the initial adhesion of microorganism²⁹.

Non denture related local factors:

Xerostomia, smoking, carbohydrate rich diet, environment pH are some of the non denture related local factors². Factors like irradiation and smoking lead to xerostomia which reduces the ability of cleaning and buffering of saliva. Carbohydrate rich diet, environment pH and xerostomia induce changes in normal microbial communities, favouring proliferation of bacteria,

which inhibit the normal adaptation of commensal fungi⁴. These modulate the biofilm development on denture surface by affecting both structural features and virulence factor in *Candida albicans* biofilm³⁷.

Clinical manifestations:

Clinical characteristics may range from no symptoms to severe pain and difficulty in swallowing⁹. Most common signs include changes in colour and texture of mucosa, drymouth, painful symptamatology and erthymatous aspect³⁰. It has been estimated that between 28 – 70 % of patients with denture stomatitis have oral complaints³¹. If symptoms are present, the condition is most frequently associated with mucosal bleeding, swelling, burning or painful sensation, halitosis or an unpleasant taste and dryness in the mouth³¹. Denture stomatitis has a frequent association with angular cheilitis which is present in 33 – 82 % of the cases³¹. Other associated lesions are atrophic glossitis, median rhomboid glossitis, acute pseudomembranous candidiasis and candidal leukoplakia^{31, 32, 33}.

Diagnosis:

Diagnosis of denture stomatitis is confirmed histopathologically by the finding of mycelia or pseudo-hypae in a direct smear or isolation of *Candida* species with ≥ 50 colonies from the lesion².

Prevention and treatment:

Institution of efficient oral hygiene and denture hygiene is the basic preventive measure. Correction of denture wearing habits is mandatory. Patient should be instructed to take out the dentures for 6 – 8 hours/ day. Correction of ill fitting dentures and achieving occlusal balance is a must to avoid trauma³⁴. Dentures should be brushed in warm soapy water and soaked overnight in an antiseptic solution³⁵. When the dentures were immersed in antiseptic solutions like 2% chlorhexidine or sodium hypochlorite, the results were more encouraging²³. If necessary, new dentures must be made. Local factors which enhance denture stomatitis like smoking should be discouraged.

Anti fungal therapy is recommended only if diagnosis is confirmed histopathologically or in patients whom the infection has spread to other sites of oral cavity or pharynx or in patients with

increased risk of systemic mycotic infection². Topical application is preferred to systemic therapy as resistance occurs to systemic drugs. Topical application is available in many forms like pastilles, trouches, creams, ointments and oral suspensions³⁶. The topical antifungal suspensions are based on nystatin, amphotericin B, miconazole and fluconazole. 2% miconazole has been successfully used. It is recommended in the dosage of 2 to 3 times a day for one or two weeks, according to patient's response.⁵ 1% clotrimazole cream is also used topically, because of its gastrointestinal and neurological toxicity when used systemically.

When the topical antifungal therapy does not lead to clinical improvement of the patient, the use of systemic antifungal agent is recommended especially in immune-compromised patients³⁷. Among the systemic antifungal agents, fluconazole has been extensively used due to its lower toxicity, high bioavailability in oral formulations and less expensive in relation to other antifungal agents³⁸. It is recommended in a single and daily dose, first dose of 400mg and subsequent daily doses of 100 mg for one or two weeks³⁹. Other systemic antifungal therapy are with ketaconazole, 200 to 400mg, orally once daily or itraconazole, 100mg capsules once daily⁴⁰.

In cases of type III denture stomatitis, surgical elimination of deep crypt formation with laser beam⁴¹, cryosurgery⁽⁴²⁾, electro-surgery and scalpel surgery is required in prior for effective mucosal hygiene. In both type II and type III denture stomatitis, about 1mm of tissue surface of dentures penetrated by microorganisms should be removed and relined with a soft tissue conditioner. A new denture should be provided when the mucosa is healed and the patient is able to maintain good oral and denture hygiene².

CONCLUSION

In clinical practice, it is very important to have a thorough knowledge about candida associated denture stomatitis. As denture stomatitis is mostly asymptomatic, the practitioner should examine the patients wearing dentures periodically to check for this condition and institute effective oral and denture hygiene. If any pathological reactions are found associated with denture bearing mucosa as in denture stomatitis, the etiology along with the

predisposing factor should be found out to aid in proper treatment of the patient.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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