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DENTURE STOMATITIS - A REVIEW

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ABSTRACT:

Denture stomatitis is the most predominant and long-standing problem in denture wearers. The etiopathogenesis of denture stomatitis is multiphase and complex to understand. The placement of denture produces suggestive changes in the oral environment and adversely affects the integrity of oral tissues. The combination of entrapment of yeast cells in irregularities in denture-base and denture relining materials, poor oral hygiene and several systemic factors is the most probable reason for the onset of this infectious disease. Candida species causes colonization and growth on prostheses. This article gives a comprehensive review of epidemiology, etiology, classification, clinical features, prevention and management of denture stomatitis.

KEYWORDS: Denture stomatitis; denture wearers; candida albicans

INTRODUCTION:

Denture stomatitis is a very known disorder affecting denture wearers. Denture stomatitis is described as erythema and inflammation of the oral mucosal regions filled by the denture[1-3]. Several studies implied up to two-thirds or more of individuals who wear removable complete dentures can suffer from denture stomatitis[3-6]. Although its frequency, denture stomatitis is most often asymptomatic; only a minority of sufferer's experience pain, itching, or burning sensation, and the disorder is primarily diagnosed during examination as presence of inflammation or swelling of mucosal tissues covered by the denture[2-7]. Indeed though its congruity, the etiology of denture stomatitis is inadequately understood. Associations of denture stomatitis have been resulted with mucosal trauma due to poor denture fit, increasing age of the denture user, increased age of dentures, bacterial and fungal (primarily Candida) infection, and poor denture hygiene;[1,4,5,8] however, no clear cause-and-effect relationships have been manifested for most associated etiologic factors. Actually, the current thinking is that the etiology of denture stomatitis is multifactorial. In many incidences it likely includes a pathogenic outcome to Candida infection, and primarily infection with C. albicans[5-7].

While access to dental care is enhancing, and persons are retaining their natural dentition for longer periods of their lives, the occurrence of edentulousness remains significant, particularly among the elderly. This review provides an update on the epidemiology, etiology, classification, clinical featues, prevention and treatment of denture stomatitis and the potential role of denture materials in this disorder. As denture matrices differ in the capability of oral bacteria and yeast to form biofilms and colonize them, they may reflect greater or lesser susceptibility for occurrence of denture stomatitis[9].

EPIDEMIOLOGY:

The prevalence of denture stomatitis has been reported in 11-67% of complete denture wearers and the mean age at which complaints started, nearly 49 years. Most investigators have concluded that denture stomatitis is more common in female[1].

CLASSIFICATION:

In 1962 Newton first to propose a classification of denture stomatitis. Based on Newton's original method, Budtz–Jorgensen and Bertram in 1970 and Bergendal and Isacson in 1983 proposed other classifications of denture stomatitis[10].

Newton classified denture stomatitis into three types based on the clinical findings of the lesion:

Punctiform hyperemia (Class I): Hyperemia signs on minor palatine salivary glands, presence of erythematous punctiform aspect, and small areas in palate may be affected.

Diffuse hyperemia (class II): Presence of erythematous aspect with smooth and atrophic mucosa under the denture. It is considered the most common aspect of Candida associated denture stomatitis.

Granular hyperemia (Class III): More common in dentures with suction chambers. Affect the central region of the palate, with rough and nodular appearance of the mucosa[11].

Newton's type I has been shown to be the result of trauma, whereas Newton's class III has a multivariable interaction phenomenon[12].

Based on the type of inflammation observed on the mucous membrane of the palate under a maxillary denture. According to Budtz–Jorgensen and Bertram classification,

• Simple localized inflammation - limited area

• Simple diffuse inflammation - the whole area covered by the denture

• Granular inflammation - often localized to the central part of the hard palate.

Bergendal and Isacsson followed Ostlund's classification.

Local inflammation – Presence of red spots found around the small palatal minor salivary glands; the lesion usually associated with trauma from the dentures.

Diffuse reddening referred to a diffuse hyperaemic, smooth and atrophic mucosa extending over the entire denture area and was associated with increased growth of yeasts[5].

ETIOLOGY:

MULTIFACTORIAL FINDINGS:

The etiology of denture stomatitis remains disputed as it is of multifactorial nature. Denture trauma, denture cleanliness, night time denture wearing, dietary factors, Candida infections and predisposing systemic conditions have been defined as associated factors in denture stomatitis[1,13,14].

TRAUMA:

According to Nyquist;[15] trauma caused by dentures was the controlling factor in denture stomatitis. Cawson;[16] concluded that the trauma and candidal infection are main causes of denture stomatitis. According to some recent proof, nocturnal wear of dentures and smoking are suggested as other significant risk factors for denture stomatitis[17,18].

MICRO ORGANISMS:

But some earlier investigators linked denture stomatitis with trauma or bacterial infection, others had isolated the strains of the genus Candida, in particular Candida albicans from the mouths of patients with this condition[5,6,19-24].

Denture induced stomatitis or chronic atrophic Candidiasis is the general form of oral Candidiasis and is current in 24-60 percent of denture wearers. Denture stomatitis has been associated with angular cheilitis, acute pseudomembranous Candidiasis, atrophic glossitis and chronic hyperplastic Candidiasis, and has been found to be more common in females than males[1].

DENTURE LINING MATERIAL:

Denture lining materials are most frequently used in association with the mandibular denture.

Candidal growth has been connected with mandibular dentures relined with soft liner. The most commonly identified yeasts were strains of the genus Candida, in specific C. albicans, C.glabrata and C. tropicalis[25].

DENTAL PLAQUE:

Denture Plaque Poor denture hygiene is found to be one of the etiologic factors for denture stomatitis[26].

SURFACE TEXTURE AND PERMEABILITY OF DENTURE BASE:

The tissue surface of the dentures generally shows micropits and microporosities. Microorganisms harboring in these areas are difficult to eliminate mechanically or by chemical cleansing. Surface roughness may facilitate microbial retention and infection[27].

ALLERGIC CONDITION/REACTION:

Toxicity is usually manifested by the release a lot of chemical constituents from the material, which can cause an allergic response in terms of localized or generalized stomatitis/ dermatitis, serious toxicological reactions or carcinogenic/ mutagenic effects. Several forms of allergies inclusive of type IV hypersensitivity, urticaria, allergic stomatitis, dermatitis and psoriasis have been reported in literature from different polymer components[28.]

SYSTEMIC FACTORS:

In cases that fail to respond to the usual treatments, consider the role of systemic disease and its effect on oral function. Certain systemic conditions such as diabetes mellitus (iron, folate, or vitamin B12), hypothyroidism, nutritional deficiencies, Immunocompromised conditions (HIV infection), malignancies (acute leukemia, agranulocytosis), iatrogenic immune-suppressive drugs, e.g. Corticosteroids, may also predispose the host to candida-associated denture stomatitis[26,20].

CLINICAL FEATURES:

The extent of inflammation has been correspond with the presence of yeast colonising the denture surface.

1. Clinical presentation of erythema and edema in palatal mucous.
2. Fungal infection in the form of white surface colonies or plaques may be detected on the mucosal surface[29].
3. Variably intense erythema, which may also be associated with scattered petechiae, is diffused over the mucosa covered by the base of the denture but not beyond.
4. Palpation of the involved mucosa discloses no tenderness or tissue friability Intense erythema is the most common finding.
5. Stomatitis unlikely develops under a lower denture. The affected mucosa is often sharply described, in the shape of the covering denture[30].

PREVENTION:

It is compulsory to include denture stomatitis averting in oral health care programmes. Dental professionals working with geriatric patients must encourage these preventive programmes among all health care workers, home caregivers, members of the patient's family and, of course, the patients themselves. A preventive programme should include[31]:

A ordinary basis inspection of the oral cavity for screening for this disorder, even when the lesions are asymptomatic. Proper denture sanitization and execute good oral hygiene. Proper denture-wearing habits, instructing the patient to take his/her denture out of the mouth for 6-8 hours each day. Patients with partial dentures should endure periodic professional plaque control procedures[32].

MANAGEMENT OF DENTURE STOMATITIS:

Elimination of predisposing factors is considered the first and most essential step[33].The therapeutic management are included.

CORRECTION OF ILL-FITTING DENTURE:

Ill-fitting dentures were considered to be the main lead factor for the occurrence of denture stomatitis. Therefore, improving adaptation of the denture should be contemplate for the management of denture stomatitis[34]. Correction of ill-fitting denture is important for the treatment of denture stomatitis. Discontinuous denture wearing are also observed important for the treatment of denture stomatitis[8].

ANTIFUNGAL AGENTS:

Antifungal agents are either polyenes (nystatin and am-photericin B) or azoles which are classified into: imidazoles (fenticonazole, clotrimazole, isoconazole, econazole, , ketocon-azole, miconazole, sulconazole, tioconazole); and triazoles (fluconazole, itraconazole)[35]. These act by inhibiting pathways (enzymes, substrates, transport) required for cell membrane synthesis or altering the permeability of the cell membrane (polyenes) of the fungal cell. It may further alter RNA and DNA metabolism or an intracellular accumulation of peroxide that is toxic to the fungal cell. The impact of the antifungal agent depends on its concentration, susceptibility of the strain and the source of the mucosal surface[5]. Many of these drugs are used topically, while remaining are used in systemic form.

TOPICAL ANTIFUNGALS:

Topical antifungal therapy residue the corner stone of treatment in mild localized cases of candidoses in healthy patients. They are accessivle in many forms like pastilles, troches, creams, ointments and oral suspenstions[36]. The antifungal treatments more used are antifungal suspensions based on nystatin[5] amphotericin-B,[37,33] miconazole[11,38] and fluconazole[5]. In addition, Clotrimazole[39] is usually presented in a cream or solution form; the cream form also has an antistaphylococcal activity. About all drugs generally produce a complete remission of symptoms within 12-14 days[40]. A nystatin suspension 100,000 unit per ml is recommended. Clotrimazole (1% cream) is only

used topically, due to gastrointestinal and neurological toxicity; Econazole available in topical form only; miconazole (2-4% cream) can be used topically[34].

Amorolfine related to a new class of chemical antifungal. Its fungistatic and fungicide effect is based on the modification of the fungal cell membranes, in particular at the level of the sterol's biosynthesis. Likewise, the content of ergosterol is reduced, and at the same time not usual planar sterols accumulate[41].

SYSTEMIC ANTIFUNGALS:

Systemic antifungal agents have been prescribed for patients with poor compliance such as patients with special needs. They are further recommended for immunocompromised patients[42]. Among systemic antifungal drugs, fluconazole and itraconazole have been the most widely studied and proven as efficient antifungal drugs. Fluconazole is commonly used in the form of 50 - 100 mg capsules, and itraconazole in the form of 100mg capsules. ketoconazole is given 200-400 mg, orally once daily[33].

PRESERVATIVE AND DISINFECTANT AGENTS:

Large positive results are obtained when the dentures are immersing into 2% chlorhexidine as aid to topical therapy. Further antiseptic substance used is sodium hypochlorite. It is demonstrated that by diving the denture in a solution of 0.02% sodium hypochlorite, the number of Candida and bacteria amount on the denture surface effectively decrease. Unfortunately, sodium hypochlorite may not be used for an undefined period of time according to its ability to damage the prosthetic handiwork[40].

MICROWAVE IRRADIATION:

Irradiation with microwave has been proposed as a soon effective and cheap method for the denture disinfection. In vitro the exposure to the microwaves are root for the cell death of Candida albicans[40]. There are many proof showing a new-alternatives, such as the use of microwave irradiation at a specified setting and exposure time, are bactericidal and fungicidal[5]. Thomas and Webb manifested that microwaving of dentures at medium setting (350 W, 2450 MHz) for six minutes caused minimal change which was considered to be harmless in the long-term[43].

SURGICAL TREATMENT:

In mild cases of inflammatory papillary hyperplasia antifungal treatment without surgery might be an substitute before the dentures are relined or replaced. In severe papillary hyperplasia of palate, cryosurgery or excision can be considered[34].

CONCLUSION:

Denture stomatitis affects a maximum percentage of persons wearing removable complete dentures. It has a multifactorial etiology. Key factors that can dramatically increase the risk of denture stomatitis are poor denture fit, poor denture hygiene, and colonization of the denture surface and oral mucosa, primarily mucosa in contact with denture fitting surfaces, with C. albicans. Poor denture care and hygienic maintenance leads to rapid place of a biofilm and collection of denture plaque. Since this provides the means for denture colonization by Candida strains, the correlation between lack of hygiene and propensity for Candida infection is clear. Denture materials themselves can contribute to the risk for denture stomatitis, as regions of surface roughness and the hydrophobicity of denture surfaces can upgrade attachment of microorganisms and progress of the biofilm.

It is important to reduce risk for development of denture stomatitis. Good quality prostheses combined with clear instructions to denture wearers by dentists and prosthodontists on the importance of diligent maintenance and application of a daily cleaning regimen are required. Denture wearers must practice appropriate denture hygiene.

In addition to, denture wearers should remove their dentures at night. Routine follow-up visits to evaluate that the prostheses maintain proper fit and function, and that users are maintaining denture hygiene is of extreme importance in reducing risk for developing stomatitis. Finally, treatment of stomatitis appears to rely on stringent cleaning or replacement of dentures, together with suitable antifungal therapy.

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