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Oral Candidiasis: Fungal Infection in Mouth-A Review

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ABSTRACT: Candida albicans is regarded as the main pathogen responsible for oral candidiasis among the Candida species. This condition is characterized by habitual erythema and edema of part or all the mucosa beneath maxillary dentures. The etiology of denture stomatitis is considered multifactorial, denture shrine, trauma, candida, mislike, adverse systemic conditions, face texture, and permeability of the denture base and filling accouterments are considered as the major factors associated with the condition. However, with proper diagnosis and treatment, including antifungal therapy and improved oral hygiene, the symptoms of the condition can be effectively managed. Dental professionals should be aware of the role of Candida in this condition and take steps to educate their patients on the importance of good oral hygiene and regular dental check-ups.

KEYWORDS:Candida albicans, Mycosis, Glossitis, Atrophic, Denture stomatitis.

I. INTRODUCTION

An opportunistic infection of the oral cavity is oral candidiasis [3]. People have been known to suffer from thrush or candidiasis for more than 2000 years. Hippocrates, a renowned Greek physician, noted in his observations that it frequently manifests as superficial infections of the vaginal and oral mucosa [7]. The most significant fungal opportunistic pathogen is Candida albicans. It typically lives as a commensal in the oral and conjunctival flora, as well as the gastrointestinal and genitourinary tracts [2]. Almost half of healthy people have C. albicans, a dimorphic fungus organism, in their oral cavity in a non-pathogenic state. Under the right circumstances, the organism, which is typically present as yeast, has the capacity to change into a pathogenic one, frequent among the elderly and denture wearers [5]. Broad-spectrum antibiotic medication, xerostomia, immunological dysfunction, or the use of removable dentures or prosthetics can all contribute to the development of the illness [5]. Candida albicans is the species that is found the most frequently, but non-Albicans species have lately been reported to be prevalent [8]. It can also be a sign of systemic illnesses like diabetes mellitus and is a prevalent issue in those with impaired immune systems [3]. Around 65% of people who wear upper full dentures are affected by this lesion. Moreover, it is primarily seen in the palatine mucosa and is typically asymptomatic, though it might exhibit symptoms including discomfort, halitosis, itching, and burning [6].

II. CLASSIFICATION

A.ACUTE CANDIDIASIS
Pseudomembranous candidiasis (oral thrush)
Erythematous (atrophic) candidiasis

B. CHRONIC CANDIDIASIS
Erythematous (atrophic) candidiasis
Hyperplastic candidiasis (Candida leukoplakia)

C. CANDIDA-ASSOCIATED LESIONS IN THE ORAL CAVITY
Angular cheilitis
Denture-related stomatitis
Median rhomboid glossitis
Linear gingival erythema

A.ACUTE CANDIDIASIS
PSEUDOMEMBRANOUS CANDIDIASIS

This form of candidiasis classically presents as an acute infection, though the term habitual pseudomembranous candidiasis has been used to denote habitual rush cases. It's generally seen in axes of age, vulnerable-compromised cases especially in AIDS, diabetics, cases on corticosteroids, dragged broad-diapason antibiotic

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remedy, hematological, and other malice. They generally do as glutinous white pillars suggesting disgruntled milk or cabin rubbish on the face of the labial and buccal mucosa, hard and soft palates, periodontal tissue, and oropharynx. The membrane can be scrapped off with tar to expose the underpinning erythematous mucosa. It's frequently fluently diagnosed and is one of the commonest forms of oropharyngeal candidiasis accounting for nearly a third. The symptoms of the acute form are rather mild and the cases may complain only of a slight cutting sensation or foul taste, whereas, the habitual forms may involve the esophageal mucosa leading to dysphagia and casket pains. Many lesions mimicking pseudomembranous candidiasis could be white carpeted lingo, thermal and chemical becks, lichenoid responses, leukoplakia, secondary syphilis, and diphtheria.[5]

ERYTHEMATOUS (ATROPHIC) CANDIDIASIS

Atrophic or erythematous candidiasis is fairly rare and manifests in both acute and habitual forms. Preliminarily known as ‘antibiotic sore mouth', due to its association with dragged use of broad-spectrum antibiotics. This form is associated with pseudomembranous candidiasis. When the white shrine of pseudomembranous candidiasis is scrapped, frequently red atrophic and painful mucosa remains. likewise,the erythematous stomatitis and depapillation of lingo arise because of the repression of the normal bacterial foliage. The symptoms case frequently describes include vague pain or a burning sensation.[5]

B. CHRONIC CANDIDIASIS

ERYTHEMATOUS (ATROPHIC) CANDIDIASIS

The habitual form is generally seen in HIV cases involving the dorsum of the lingo and the palate and sometimes the buccal mucosa. Cases who wear dentures continuously day and night are most generally affected by the infection. This form of atrophiccandidiasis is also nominated as ‘Denture sore mouth’. The complaint is characterized by the conformation of asymptomatic erythema and inflammation of the entire denture-bearing mucosa of the palate.[5]

HYPERPLASTIC CANDIDIASIS (CANDIDA LEUKOPLAKIA)

Hyperplastic candidiasis is the least common of the trio of major clinical variants, with 5 of the cases. CHC can manifest in nodular form or as whitish pillars that cannot be attributed to any other complaint, don't detach upon rasping, and are generally located on the impertinence mucosa and lingo, and especially bilaterally at both lip retro-commissures. In this form of infection, the Candida hyphae aren't only set up at the epithelial face position but also foray into deeper situations where epithelial dysplasia can be observed, with the associated threat of malice. Hyperplastic candidiasis may persist for a time without any symptoms[5].

C. CANDIDA-ASSOCIATED LESIONS IN THE ORAL CAVITY

ANGULAR CHEILITIS

Angular cheilitis is an inflammation of one, or further generally both of the corners of the mouth. Originally, the corners of the mouth develop an argentine-white thickening and conterminous erythema. Latterly, the usual appearance is a roughly triangular area of erythema, edema (lump), and maceration at either corner of the mouth. Angular cheilitis can do spontaneously but more frequently develops in those who wear oral dentures and appliances, those who are needed to wear masks as part of their occupation, and in some small children, particularly those who slobber and use soporifics. Generally, the lesions give symptoms of soreness, pain, pruritus (itching) or burning, or a raw feeling after the stage. Angular cheilitis frequently represents an opportunistic infection of fungi and/ or bacteria, with multiple original and systemic prepping factors involved in the inauguration and continuity of the lesion.[5]

DENTURE-RELATED STOMATITIS

Denture-related stomatitis refers to a seditious state of the denture-bearing mucosa, characterized by habitual erythema and edema of part or all the mucosa beneath maxillary dentures. It's also the most generally encountered mucosal lesion with removable prostheses and affects one in every three complete denture wear and tear. The frequency of its development is 25 – 67, constantly seen among womanish cases, and frequency increases with age. Denture stomatitis is also known as denture sore mouth, seditious papillary hyperplasia, denture-convinced stomatitis, and habitual atrophic candidiasis. Although the etiology of denture stomatitis is considered multifactorial, denture shrine, trauma, candida albicans, mislike, adverse systemic conditions, face texture, and permeability of the denture base and filling accouterments are regarded as the major factors associated with the condition. In the maturity of the cases, elimination of denture faults, control of denture shrine, and discontinuing the wearing of dentures are sufficient.[5]

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MEDIAN RHOMBOID GLOSSITIS

Median rhomboid glossitis is a diamond-shaped, elevated, seditious lesion of the lingo, covered by smooth red mucosa. It's positioned anterior to the circumvallate papillae, at about the junction of the anterior two-thirds and posterior one-third of the lingo. It generally affects males while many studies showed womanish transcendence. The most common clinical donation of the complaint is an erythematous or white-erythematous area on the dorsal standard face of the lingo, incontinently previous to Region V of the circumvallate papilla(terminalgingiva nodular element is sometimes set up, or the organ can be lobulated. The texture may be analogous to the subjacent or firm part of the lingo, and its face is fairly soft.occasionally, soft palate erythema may be seen where the lesion of median rhomboid glossitis touches the palate. This erythematous area is nominated as a 'kissing lesion'. Generally, median rhomboid glossitis is asymptomatic. Still, in many cases, pain and ulceration have been reported [5].

LINEAR GINGIVAL ERYTHEMA

Linear gingival erythema (LGE), which was formally appertained as HIV- gingivitis, is the most common form of HIV- associated periodontal complaint in HIV- the infected population. It's considered resistant to the conventional shrine-junking curatives, being considered, currently, a lesion of fungal etiology. It's characterized by a fired, direct band 2 to 3 mm wide on the borderline gingival accompanied by petechiae- suchlike or verbose red lesions on the attached gingival and oral mucosa, and may be accompanied by bleeding. The frequency of this lesion varies extensively in different studies, ranging from 0 to 48 presumably because in numerous of them, LGE was misdiagnosed as gingivitis.[5]

III. RISK FACTORS

PATHOGEN

Pathogens have peculiar traits that, in the appropriate environment, increase their rate of contagiousness. They are eukaryotic, non-photosynthetic organisms with a cell wall that is external to the plasma membrane, just like another fungus. The nuclear membrane contains a nuclear pore complex. Most of the sterols in the plasma membrane are typically ergosterol. The macroscopic and microscopic cultural traits of the various candida species are comparable, with a few variations. They are capable of metabolizing carbohydrates both aerobically and anaerobically. Higher temperatures, like those found in their potential host at 37°C, impact their growth by fostering the development of pseudohyphae. For their development, they need sources of fixed carbon in the atmosphere. Mycelium and hyphae exhibit filamentous growth, as well as apical expansion of the filament and the development of lateral branches, while yeasts exhibit single-cell division. Certain components of fungal cell walls, such as mannose, C3d receptors, mannoprotein, and saccharins, facilitate the adhesion of candida to epithelial cell walls, an essential step in the initiation of infection. It has also been suggested that the degree of hydrophobicity and the capacity to bind to host fibronectin are significant in the early phases of infection. Additionally, phenotypic switching—the capacity of some strains of Candida albicans to transition between various morphologic phenotypes—has been connected.[3]

HOST FACTORS

Host factors could be local and/or systemic

A.LOCAL FACTORS

Wearing dentures, having problems with your salivary glands, inhaling steroids, and having mouth cancer are all risk factors for local factors.

TRAUMA

Nyquist believed that traumas, rather than microbial communities or the existence of dentures, were the primary factor in determining Candida-associated denture stomatitis. Cawson later demonstrated that the etiology of denture stomatitis is caused by both traumas and Candida infection. According to the most recent research, trauma may be the cause of localized denture stomatitis rather than causing generalized denture stomatitis. Candida albicans, on the other hand, play the primary pathogenetic function in generalized forms. Furthermore, Candida colonization and tissue penetration are more likely to occur as a result of mechanical stress from ill-fitting dentures. [1]

SALIVA

It is still debatable how saliva affects Candida albicans growth. Lysozyme, nystatin, lactoferrin, calprotectin, and IgA are innate defense molecules found in saliva that have a physical cleaning impact. However, it has been demonstrated that salivary proteins like mucines and statherins, which are found in the mannoproteins found in the Candida species, may function as binding receptors. When a person with a xerostomy has less saliva or none at all, the usual microbial communities change and become unbalanced, which encourages the growth of

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bacteria like Staphylococcus aureus and prevents the commensals from adapting normally. A low pH and high oxygen tension also inhibit the development of some commensals while promoting the growth of Candida species, Streptococcus mutants, and Lactobacillus [1].

AGE OF THE DENTURE

According to a study, aging of the denture and the release of leftover monomer over time causes a poorer fit, which affects the denture's contamination. Furthermore, Candida infection rather than the intensity of inflammation was inversely correlated with denture age. Only 25% of people who had worn dentures for more than five years had the illness. Additionally, a denture's age and bad quality are both significant factors.[1]

ALLERGY

The denture base material's allergic and primary irritant responses also come into play. [1]

UNINTERRUPTED DENTURE WEARING

Wearing dentures at night should be taken into consideration as one of the risk factors.[1]

SMOKING

Additionally, there is still disagreement over whether tobacco use should be regarded as one of the predisposing variables. A rise in oral Candida colonization or the emergence of oral candidosis has all been linked to tobacco use in a number of earlier studies, either on its own or in conjunction with other local or systemic factors. Specifically, it has been shown that cigarette smoke interferes with Streptococcus mutans and Candida albicans adhesion, resulting in biofilm formation on dental restoration materials, which suggests that cigarette smokers are more susceptible to life-threatening oral infections including candidiasis. However, this association has not been found in other research. Smoking also looked to make erythematous candidiasis more common in patients with candidal leukoplakia and denture wearers. The density of Candida development in oral rinse cultures correlated positively but was only weakly significant with daily cigarette consumption. As a result, cigarette smoke can be classified as a substance that fosters infection.[1]

B. SYSTEMIC FACTORS

Extremes of age, smoking, diabetes mellitus, Cushing's syndrome, immunosuppression, malignancies, nutritional deficits, and antibiotic use are risk factors for systemic.

ENDOCRINE DISORDER

Comparable studies by Manfredi et al. and Al-Karaawi et al. present contradictory results with lower rates of candidal carriage in diabetic patients compared with healthy controls, despite some studies showing increased oral colonization of Candida albicans in diabetic subjects. Since poor control can lower salivary flow and pH and raise salivary glucose levels, it appears that glycaemic control is a more important component than the disease itself. These elements encourage the colonization and spread of oral candida. In vitro studies have shown that diabetics' saliva promotes the development of Candida albicans, and it has been demonstrated that diabetics' denture surfaces have higher colony counts of the yeast than non-diabetic subjects do. Therefore, patients with diabetes are more prone than non-diabetic patients to develop oral candidal infections.[1]

IMMUNOLOGICAL DISORDER

Frequently, when the host defense system suffers because of any alterations, like immunodeficiency, Candida albicans become virulent and generate candidiasis, which can be manifested through various clinical forms, involving one or more oral sites, up to affect the whole oral cavity and disseminate into invasive forms. More than 60% of HIV-positive individuals and more than 80% of AIDS patients develop candidiasis. Additionally, a number of other immunodeficiencies, such as hereditary myeloperoxidase deficiency, Chediak-Higashi syndrome, severe combined immunodeficiency syndrome, and DiGeorge syndrome frequently present as candidosis.[1]

DEFICIENCY OF NUTRITIONAL FACTORS

Reduced host defenses and a loss of mucosal integrity, as well as protein-energy malnutrition, high cholesterol, iron, folate, vitamin C, and vitamin B12, and probably vitamin A deficiency, according to some authors, may make the body more vulnerable to hyphal invasion and infection. On the other hand, diets high in carbohydrates might be a risk factor that makes it more likely that Candida species will attach to epithelial cells [1].

DRUG THERAPY

Oral candidosis is predisposed by a number of groups of pharmacologic agents, including xerogenic drugs, immunomodulatory drugs, and broad-spectrum antibiotics. Corticosteroids, antidepressants, antipsychotics,

anticholinergics, antihypertensives, and antiadrenergic are just a few of the prescription medications that can cause xerostomic adverse effects [1].

IV. PATHOGENESIS

Poor oral hygiene, practices similar as failure to remove the denture whilst sleeping, and poor denture sanctification allow the accumulation of biofilm, which is defined as a structured microbial community that's attached to a face, conforming to further than 1011 microorganisms per gram of dry weight and girdled by a tone-produced extracellular matrix. For illustration, streptococcus mutans biofilm, which is considered one of the primary pioneers of oral biofilm, is heavily involved in the original stages of biofilm conformation, producing redundant cellular matrix polysaccharides and easing the attachment of other microorganisms similar to candida albicans. The proliferation of bacteria and fungi can beget bad breath, acrylic resin saturation, staining, the conformation of math deposits, and the development of habitual atrophic candidiasis, also known as denture stomatitis. Numerous factors affect the adhesion and biofilm conformation of candida on acrylic shells, similar to face roughness of the inner face of the prosthesis, salivary pellicle, hydrophobic and electrostatic relations, and receptor-ligand list. There are factors that hinder attachment because the faces of both candida and epithelial cells are negatively charged. A bilayer of provocations, origin tubes, and young hyphae with an extracellular polymeric material matrix makes up the candida biofilm community. Hawser & Douglas were the first to demonstrate that candida biofilms flaunt antifungal resistance, and numerous other experimenters have confirmed this. As biofilms on acrylic shells exposed to sugars demonstrated advanced candida numbers, phospholipase exertion, and enhanced production of extracellular matrix substance, healthy habits may also affect the resistance of fungus in biofilms to antifungal drugs [1].

V. CANDIDA ALBICANS

Infection of the oral cavity caused by oral candida is called oral candidiasis. Elderly people frequently experience it and underdiagnose it, especially if they wear dentures. A good oral hygiene routine can often prevent it. It is a typical issue for those who are immune-compromised and can also be a sign of systemic disorders like diabetes mellitus. An oral cavity infection or overgrowth of the yeast-like fungus candida, which causes oral candidiasis, is to blame[3]. Candida albicans is regarded as the main pathogen responsible for oral candidiasis among the Candida species. This is a result of its strong capacity to cling to denture surfaces and oral tissues, which leads to the creation of biofilm. The majority (70–80%) of isolates from oral mucosal lesions come from C. albicans, the most virulent pathogenic Candida species [4].More than 400,000 fungal infections are caused each year by Candida, making it one of the most widespread causes of fungal infections globally. Due to aging demographics and an increase in patients with impaired immune systems, candidiasis has become more common recently [4]. If left untreated, this may result in malnutrition and a drawn-out recovery. When it spreads, it can be lethal in severe situations [3].

VI. DENTURE STOMATITIS

The multi-factorial etiology of denture-related stomatitis is linked to the usage of dentures, and the disease's appearance is primarily influenced by host variables and external factors, such as the introduction of aprosthesis. [9]It has been discovered that wearing dentures frequently cause denture stomatitis. The wearer's inappropriate maintenance regimen or an ill-fitting complete denture prosthesis that causes stress to the underlying mucosa are the likely causes of this. Moreover, the likelihood that the normally occurring oral commensal, Candida, would transform into a pathogen and produce Candida-associated-denture stomatitis increases if the patient has weakened immunity, such as is the case with diabetes or HIV [8].

In 1-16 different locations in the cosmos, CADS has been found to affect 65 to 70% of people who wear dentures. This is brought on by strain alterations that alter the phenotype of the bacterium or the emergence of treatment resistance. Rapid species-level identification is crucial in a clinical setting as it significantly influences therapy choices. Denture stomatitis was found in 27 of the 55 patients with complete dentures who were surveyed, or 50% of those who wore dentures. This finding supports the widespread occurrence of CADS in long-term complete denture wearers [8]. Males, older ages, and complete dentures were found to be associated with a higher incidence of CAC [9].

VII. TREATMENT

Potential pharmacological strategies include the use of

- New formulations of antifungals, such as liposomal amphotericin B, amphotericin B

lipid complex, amphotericin B colloidal dispersion, amphotericin B into a lipid nanosphere formulation, itraconazole, and β-cyclodextrin itraconazole (or)

- Combination therapies of one or more antifungal compounds, for example, amphotericin B + flucytosine, fluconazole + flucytosine, amphotericin B + fluconazole, caspofungin + liposomal amphotericin B, and caspofungin + fluconazole. New oral triazole albaconazole has good tolerability, distinctive pharmacokinetics, and broad-spectrum antifungal activity. In reality, for the treatment of acute vulvovaginal candidiasis, a single dose of albaconazole at 40 mg was more effective than 150 mg of fluconazole.[2]

Lack of preventive hygiene programs, improper cleaning of oral prostheses, and wearing dentures all day rather than taking them out at night are all factors in denture stomatitis. Several mechanical and chemical methods, either isolated or associated, can be used to clean the dental prosthesis, such as:

- Brushing the prosthesis in running water;
- Brushing the prosthesis using toothpaste;
- Brushing using ordinary soap;
- Cleaning with sodium hypochlorite (1.5-2.0%);
- Immersion in sodium hypochlorite (0.10-0.20%);
- Immersion in baking soda;
- Immersion in alkaline peroxide;
- Immersion in mouthwashes; and
- Use of effervescent tablets. But when it comes to cleaning dentures, a mix of mechanical and chemical techniques is thought to be the best option because it offers both mechanical removals of biofilm and antimicrobial action.[6]

The antifungals miconazole, fluconazole, itraconazole, nystatin, amphotericin B, ketoconazole, clotrimazole, and chlorhexidine are most frequently used to treat prosthetic stomatitis. The antibiotic that has been used most successfully is miconazole gel, 2%. The previously cleaned prosthesis serves as a "tray" by providing the medication more contact time with the lesions, improving response and hastening to heal. The medication is applied immediately to the prosthesis.[6]

VIII. CONCLUSION

In conclusion, Candida albicans are a major contributor to the development of denture stomatitis. The presence of this yeast in the oral cavity, combined with other factors such as poor oral hygiene and prolonged denture wear, can lead to the development of this condition. However, with proper diagnosis and treatment, including antifungal therapy and improved oral hygiene, the symptoms of denture stomatitis can be effectively managed. Dental professionals should be aware of the role of Candida albicans in this condition and take steps to educate their patients on the importance of good oral hygiene and regular dental check-ups.

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