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Original Research Paper

Dentistry

OROPHARYNGEAL CANDIDIASIS :- A REVIEW

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ABSTRACT Oral Candida is opportunistic pathogen. Oral candidiasis is one of the common fungal infections, affecting the oral mucosa and is caused by Candida albicans. Candida pathogenicity is facilitated by a number of virulence factors, the most important of which are those for adherence to host tissues and medical devices, biofilm formation and secretion of hydrolytic enzymes. Assessment of predisposing factors plays a crucial role in the management of candidal infection. Carefully recording the medical history is important Topical antifungal therapy is the recommended first line treatment for uncomplicated oral candidiasis.

KEYWORDS : Candida, pathogenicity, antifungal

INTRODUCTION

Oropharyngeal *Candidiasis* is the most commonly reported opportunistic fungal infection in diabetic patients, occurring in an estimated 13.7-64% patients and lesions are asymptomatic in most cases.[1] Worsening of glycaemic control has been reported to have significant correlation with*Candida* colonization, however few studies failed to prove such relationship.[2,3,4]

OPC and other forms of Candidiasis are caused by Candida spp., eukaryotic diploid yeast like fungi belonging to the Ascomycetes phylum, order Saccharomycetales. The genus Candida is comprised of over 200 species and constitutes an extremely diverse yeast species that multiply by budding. The medically significant Candida species include: Candida albicans, Candida glabrata, Candida parapsilosis, Candida tropicalis, and Candida Krusei. Candida albicans has been consistently demonstrated as the most frequent species responsible for OPC (80-90%).[5,6]

Epidemiology:

Candida spp. are known commensals of humans and other warm blooded animals. Although many *Candida spp.* are considered normal flora, *Candida albicans* is the most common species recovered. The occurrence of *Candida albicans* in the oral cavity ranges from 75-86.5% and sites include the tongue, palate and the buccal mucosa. Variations regarding the presence of *Candida spp.* in healthy individuals may be a function of multiple factors including climate, diet and age.[1, 7] *Candida albicans* is also infrequently from water sources, soil, air and plants. Colonization of mucosal surfaces by *Candida* is assumed to occur at an early age, the organism being acquired from the mothers birth canal, nursing or from food sources. Although most *Candidal* infections are endogenous in origin, lateral transmission from health care workers has been known to occur.[5]

Kleinegger et al. (1996) studied the proportion of *Candida albicans* in the oral cavity of different age groups and revealed that it was 55% among subjects of 0.5 to 1.5 years, 32% in subjects of 5 to 7 years, 91% in 15 to 18 years, 84% in 30 to 45 years, and 90% in subjects of age above 60 years. Conversely, the proportions of yeast isolates which were non *Candida albicans* isolates in these age groups were 44%, 68%, 9%, 16% and 10% respectively.[8,9]

A study done **by Russell and Lay. (1995)** showed that only 5.7% of neonates carried *Candida albicans* in the oral cavity, and that count increased to 14.2% at the time of discharge from the hospital(usually 7 days after birth), and to 82% 4 weeks after birth. The frequency decreased to 50% at I year of age. They suggested that these changes in frequency may be due to physiological changes related to age, the changes in environment (hospital verses home residence) and diet (breast feeding verses formula milk feeding).[8]

Sharp et al. (1992) surveyed *Candida spp.* and *Candida albicans* biotypes in the mouth and rectum among 163 neonates present in the regional neonatal intensive care and surgical unit and showed that the *Candida* colonization rate was 28.2%. *Candida albicans* was the species most frequently isolated among them (80.4), followed by *Candida parapsilosis* (13-0%) and *Candida famata* (1%).[10]

Pathogenicity Of *Candida* Species

The pathogenicity of the c. albicans depends on different aspects such as enzymes of *Candida*, temperature variations, adhesion of *Candida* and switching phenomenon.

Enzymes Of *Candida:*

Candida albicans produces certain enzymes that facilitate penetration of the mucous membranes. *Candida* has ability to produce phospholipases at the tips of fungal hyphae in the vicinity of host cellular compartments where active invasion is occurring. These enzyme activities were found in most *Candida albicans* strains but not less virulent organisms such as *Candida glabrata, Candida tropicalis and Candida parapsilosis*.

Pirjo Parnanen et al showed that *Candida* has the ability to produce enzymes effective in tissue destruction. The ability of the different *Candida* yeast species to degrade human Lm-332, fibronectin, and E-Cad vary from strain to strain and aiding invasion into deeper tissue was found. *Candida albicans* yeast form seems to be related to superficial infections, and hyphal forms can apparently invade deeper tissues between the epithelial cells by degradation of E Cad.[11]

Temperature Variations:

The virulence of *Candida albicans* is associated with increased germ tube production by yeast grown at the lower temperature which enhances the adherence characteristics compared with the yeasts in the blastopore phase. Yeasts grown at room temperature are more resistant to destruction.

Adhesion Of *Candida:*

Kennedy et al. (1988) has reviewed the relationship between the adherence of *Candia albicans* to surfaces and its ability to colonize and cause the disease. The pathogenicity of the organism is due to its nonspecific affinity and binding to acrylic resins to dentures.

The initial yeast to epithelium contact may be due to nonspecific adhesion observed once *Candida albicans* is attached. It may be that the *Candida* cells bump into epithelial cells, initially binding reversibly and then physiological changes strengthen the adhesion. These changes could modify the epithelium by exposing the receptors which

stabilizes and strengthen the adhesion.

Diagnosis of *Candida*:

Beighton et al. (1995) studied the efficiency of CHROMagar isolation of clinically important yeasts composed to sabouraud dextrose agar. The recovery of yeasts on the medium was significant compared to the recovery on sabouraud dextrose agar among 450 individuals. The identities of green colonies on CHROMagar *Candida*, presumably identified as *Candida albicans* on the basis of the manufacturers instructions, were confirmed by testing for b-N-acetylgalactoseaminidase.[12]

Bouchhara et al studied efficiently of CHROMagar for routine investigation of clinical specimens. 6150 clinical samples of yeast collected from nostril, mouth, ear, vaginal swabs were plated on CHROMagar, of which 1673 yielded positive results. Simultaneous cultures of 366 samples on sabouraud dextrose agar and CHROMagar *Candida* showed 89.9% similarity. This shows that the 26CHROMagar medium is extremely helpful in routine clinical mythological examinations facilitating the detection of mixed cultures of yeasts.[13]

Frank C Odds et al studied 726 yeast isolates using CHROMagar. The specificity and sensitivity was proved to be 99% using this medium. In parallel cultures of 348 cases using SDA medium, 78 cases showed positivity for the two media suggesting the importance of CHROMagar in presumptive identification of mixed yeast cultures.[14]

Ramoj et al determined use of CHROMagar *Candida* as the primary isolation medium when utilizing the IDS RapID yeast plus system or the API 20C AUX system for Confirmatory identification of yeasts. They studied the IDS and API systems on 56 identified the species up to 95% and API system correctly identified the species up to 80%. This data supported the use of CHROMagar *Candida* ion conjugation with the IDS RapID yeast plus system and the API 20CV AUX yeast identification systems for the confirmatory identification of yeast.[15]

Clinical Appearance:-

Pseudo membranous form: also known as oral thrush. Thrush forms soft, friable, and creamy plaques on the mucosa that can be wiped off, leaving a red, raw or bleeding, and painful surface. The buccal mucosa, palate and tongue are common locations.[7]Discrete white pseudo membranous patches that may become confluent are seen and they comprise candidal elements, desquamated epithelial cells, fibrin, inflammatory cells and debris.[24] Diagnosis is usually based on clinical criteria. Burning sensation in the mouth is present which increases on taking spicy food. Direct smear microscopic examination with potassium hydroxide and culture are helpful.[16] The clinical presentation of acute and chronic pseudomembranous candidiasis are indistinguishable. The chronic form emerged as a result of human immunodeficiency virus (HIV) infections as patients with this disease may be affected by a pseudomembranous candida infection fora long period of time.[22] The infection has traditionally, been regarded as an acute condition, often affecting newborn babies where there is an immature immune system. In older individuals, acute pseudomembranous candidosis often occur when there is a nutritional limitation, local immune suppression (eg: steroid inhaler administration for treatment of asthma) or an underlying disease most notably HIV infection and AIDS.[18]

Erythematous form: also known as acute atrophic candidiasis. It appears as red, raw looking area which is tender.[10] It may arise as a consequence of persistent acute pseudomemebranous candidiasis. It is characterised by erythematous areas generally on the dorsum of tongue, palate or buccal mucosa. Lesions on the dorsum of tongue present as depapillated areas. Red areas are often seen in the palate in HIV disease.[18] Burning sensation can be present. A form of erythematous candidiasis that is especially common involves the hard palate and gingiva beneath a denture or removable partial denture.[17]

Hyperplastic candidiasis (chronic form):resembles leukoplakia, hence also known as candidal leukoplakia.[19] A white patch is present, that cannot be rubbed off and can affect any mucosal site.[24]

Angular Cheilitis:

There is crackling or ulcerations seen around the corners of mouth.[19]It manifests as erythematous fissures or macerations affecting both mucosa and skin at the corner of the mouth.[24] Both yeasts and bacteria (especially Staphylococcus aureus) are involved, as interacting, predisposing factors. However, it is very occasionally, an isolated and initial sign of anemia and vitamin deficiency, such as vitamin B12 deficiency, and resolves when the underlying disease has been treated.[21]

Median rhomboid glossitis: Midline glossitis, or glossal central papillary atrophy, is characterised by an area of papillary atrophy that is elliptical or rhomboid in shape, symmetrically placed centrally, at the midline of the tongue.

Treatment:-

some antifungal agents used in the treatment of oral candidiasis[23,24]Nystatin(cream, oral suspension) 1-2 mL 4-5 times/day,7-14 days Amphotericin B(Lozenges) 10 mg,14-21 days Clotrimazole (Lozenges) 10 mg, 14 days Fluconazole (tablets) 200 mg per day,7-14 daysKetoconazole(tablets) 200-400 mg daily,7-14 days Itraconazole (capsules) 100 mg per day, 7-14 days

CONCLUSION:-

Dental clinicians play an important role in the diagnosis and management of oral fungal diseases.[25] Carefully recording the medical history is important in identifying this clinical problem. Predisposing factors should be treated or eliminated where feasible.[18] In most of the cases, oral candidiasis is a cause of secondary superficial infection which can easily be resolved with antifungal therapy and proper oral hygiene maintenance.[20]

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