Oral candidiasis and AIDS

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Abstract: Human immune deficiency virus [HIV] infected seropositive patient suffer from a lot of opportunistic infection because of defective immune response. The defective immune response in HIV seropositive patient is due to the depletion of CD4 lymphocytes with advancing stages of HIV infections. Oral candidiasis is commonly affecting opportunistic infections in patients. The predominant species affecting is candida albicans. Oral candidiasis is seen in increasing frequency in reduced CD4 counts. This article gives an attempt to an overview of oral candidiasis in HIV relationship of oral candidiasis with CD4 count, various modalities of diagnosis and treatment of oral candidiasis.

Keywords: candidiasis, candida albicans, fungal infection, HIV, opportunistic infection,

I. Introduction

Acquired immune deficiency syndrome (AIDS) a disease of the human immune system which is caused by the human immune deficiency virus (HIV) has emerged as a global crisis since its discovery in the summer of 1981 in the United States. Patients with defective cellular immunity associated with AIDS are under the risk for variety of opportunistic infection [1]. Oral candidiasis an opportunistic infection is a frequent and early manifestation of disease associated with the AIDS[2,3]. Candida albicans produces an opportunistic fungal infections which occurs in favorable conditions[1], including broad spectrum antibiotic therapy, xerostomia, presence of removable prosthesis, immune dysfunction such as diabetes or use of immune suppressant drugs[4].

II. Causative organism:

Among the fungal pathogens Candida species are the most predominant causes for the invasive infection[4] more than 95% of candidiasis is caused by 5 major species: Candida albicans, Candida glabrata, Candida parapsilosis, Candida tropicalis and Candida krusei [5,6,7,8]. Candida albicans is a dimorphic fungi which is typically present in the oral cavity in a nonpathogenic state in one half of healthy individual but they transform to pathogenic hyphal form under favorable environment[1]. Candida parapsilosis occur with high frequency in premature neonates and in patients with vascular catheters[9,10]. Candida glabrata occur common in elderly people and rarely in infant and children[11]. Candida tropicalis plays an important role as a cause of invasive disease in patients with hematological malignancy[12]. Candida glabrata and Candida krusei species have received attention due to their enhanced resistance to certain antifungal agents. Candida dubliniensis is recently identified pathogenic species [13].

III. Classification:

Samaranayake [14, 15] proposed a classification in which Group-1 (Primary oral candidiasis) is confined to lesions localized to the oral cavity with no involvement of skin or other oral mucosa and Group-2 (Secondary oral candidiasis) lesions are present on the oral as well as extra oral sites.

Classification Of Candidiasis

<table>
<thead>
<tr>
<th>Primary oral candidiasis</th>
<th>Secondary oral candidiasis (group-2)</th>
<th>Sub group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudo membranous(mainly acute)</td>
<td>Familial chronic mucocutaneous candidiasis</td>
<td>1</td>
</tr>
<tr>
<td>Erythematous (acute 'chronic)</td>
<td>Diffuse chronic mucocutaneous candidiasis</td>
<td></td>
</tr>
<tr>
<td>Hyperplastic(mainly chronic)</td>
<td>Candidiasis endocrinopathy syndrome</td>
<td></td>
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<tr>
<td>Plaque like</td>
<td>Familial mucocutaneous candidiasis</td>
<td>2</td>
</tr>
<tr>
<td>Ittomodular/speckled</td>
<td>Severe combined immunodeficiency</td>
<td>3</td>
</tr>
<tr>
<td>Candida associated lesion</td>
<td>DiGeorge syndrome</td>
<td>4</td>
</tr>
<tr>
<td>Denture stomatitis</td>
<td>Chronic granulomatous disease</td>
<td>5a</td>
</tr>
<tr>
<td>Angular chelitis</td>
<td>Acquired immune deficiency syndrome</td>
<td>5b</td>
</tr>
<tr>
<td>Median rhomboid glossitis</td>
<td>5c</td>
<td></td>
</tr>
<tr>
<td>Linear gingival erythema</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

Table.1[14, 15]
Oral candidiasis and AIDS

IV. Pathogenesis of Oral Candidiasis in HIV infected individual:

Transition of Candida from a harmless commensal to the pathogenic organism is complex and is related to the environmental changes which lead to the expression of various virulence factors [16]. The ability of Candida species persist on the oral mucosal surface of healthy individual is an important virulence factor [16]. This can be inhibited by saliva [17] and enhanced by dietary carbohydrates [18]. Increased frequency of Oral Candidiasis has been seen in patient with cell mediated immunodeficiency in an non HIV infected population also[19,20]. The effect of HIV on the immune system is depletion of CD4 lymphocytes, with disease progression there is drop in the absolute count and a reversal of CD4 counts ratio[21,22,23]. It has been found that HIV related oral candidiasis occurs in patient with CD4 count less than 300cells/mm³. Frequency of Oral Candidiasis also increased with reversal of CD4/CD8 cells ratio[24,25]. Severe Oral candidiasis is also found in the patient with the low CD4 counts with no HIV infection[26]. Torrssanderetal [24] noted a decreased number of CD4 counts in HIV infected patient with smears from the oral mucosa that showed pseudo mycelia forms of yeast, compared with other HIV infected patient with no pseudo mycelia forms of yeast. Sindrup et al[25] found HIV related mucocutanous lesions occurred twice as frequently in patients with a CD4 counts of less than 200/mm³.

V. Clinical features in candidiasis infection:

Oral candidiasis infection has different clinical presentation which includes pseudo membranous candidiasis, erythematous candidiasis, hyperplastic candidiasis and atrophic candidiasis. Among the different clinical varities of Oral Candidiasis, erythematous type is most frequent, pseudo membranous candidiasis and angular chelitis are the next common type. While hyperplastic candidiasis are least common type [3, 18]

5.1 Pseudo membranous candidiasis:

Pseudo membranous candidiasis is commonly known as “thrush” and it is often seen in neonates and in people receiving corticosteroid therapy. It is usually present as the multiple white plaques resembling cottage cheese that can easily wiped away leaving bleeding spots. It is usually asymptomatic with minimal slight tingling sensation or foul taste [4]

5.2 Erythematous candidiasis:

Erythematous candidiasis, as the name implies clinically appears red or erythematous [4]. It appears as the mucosal erythema and depapillated patches on the dorsum surface of the tongue and may be painful. Erythematous candidiasis is the most frequent with approximately 50% of individuals with HIV infections. It may occurs independently or simultaneously with pseudo membranous types [29]

5.3 Hyperplastic candidiasis:

Hyperplastic candidiasis is the least common type[29] this form has been referred as “candidal leukoplakia” like leukoplakia,. hyperplastic candidiasis will present as a white plaque that cannot be wiped away by clinician[4]. It is often confused with hairy leukoplakia which is another lesions commonly seen in AIDS[29].

5.4 Angular chelitis:

The final clinical presentation of Oral Candidiasis infection is angular chelitis. This form presents as cracking, peeling or ulcerations involving the corner of the mouth. It is due to increased folding of soft tissue that is frequently seen at corners of the mouth [4].

VI. Diagnosis:

Diagnosis of Oral Candidiasis can often be made on the nature of clinical presenting features [26]. The various methods for isolating the Candida species are culture of whole saliva, swab, imprint culture, smear, concentrated oral rinse, biopsy, direct microscopy. Culture of whole saliva has the advantage of being sensitive and isolates the viable organism. Butt’s not site specific and there is problem with sample collection [30].

Biopsy is used in case of chronic hyperplastic candidiasis. Draw backs are invasive and it’s not suitable for other type of candidiasis [13].

In Direct microscopy a smear taken from the lesional site is fixed on to the microscopic slides and then stained either by gram stains or by periodic acid Schiff (PAS) technique [31]. Smear technique has the advantage of being simple while the disadvantage is viable cells cannot be identified [13].

In swab culture a sterile cotton swab is gently rubbed over the lesional tissue and then inoculated with primary isolation medium like SDA (sabouraud's dextrose agar) the advantage of swab is simple, site specific and viable cells are isolated. Drawback is difficult to standardize [13, 32].

Concentrated oral rinse can be used for isolation by asking the
Patient to hold the sterile phosphate buffer saline for 1 min in mouth and the solution is centrifuged and inoculated with agar medium. Advantage is viable cells are isolated while disadvantage are some people has difficulty to use rinse quantitative and it’s not site specific [33, 34].

In Imprint culture technique a Sterile foam pad is dipped in an liquid medium like sabourauds broth immediately before use then the pad is placed on the target site for 30 sec and transferred to agar for culture. Advantage of imprint culture are quantitative, viable cells are isolated and site specific. Draw backs with this are some sites are difficult to sample [34, 35].

Most commonly used primary isolation medium for Candida is SDA [36] which although permits the growth of Candida and suppress the growth of other oral bacteria due to low ph. [26]. Identification of Candida species is through morphological criteria [34], physiological criteria [37], serology [38], and molecular based identification method [34]

### VII. Management:

Wide variety of agents are available for the treatment of oral candidiasis [39] some of these antifungal drugs are used topically and others are used systemically either topical or ivroutines. Topical agents requires sufficient contact time between the drug and the oral mucosa as well as the presence of adequate saliva to dissolve the medication[40]. Several topical drugs contain sweetening agents sucrose or dextrose and long term use of these preparation may leads to increase in caries[40].

### Management of Candidiasis Infection

<table>
<thead>
<tr>
<th>Topical agents</th>
<th>Method of use</th>
</tr>
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<tbody>
<tr>
<td>Clotrimazol solution 1%</td>
<td>1 tea spoon (5ml) rinse and expectorate 4 times daily after meals at bed times</td>
</tr>
<tr>
<td>Clotrimazole (1%) cream</td>
<td>Apply to affected mucosa/skin twice daily</td>
</tr>
<tr>
<td>Nystatin(mycostatin):100000 ml</td>
<td>1 teaspoon (5ml) rinsed and swallowed 5 times daily</td>
</tr>
<tr>
<td>Ketoconazole (2%) cream</td>
<td>Apply to affected mucosa/skin after meals and bed time</td>
</tr>
<tr>
<td>Chlorhexidine (0.2% to 0.12%)</td>
<td>3 spoon rinse and expectorate twice daily after meals or bed times</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Systemic agents</th>
<th>Method of use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluconazole 50-100mg</td>
<td>2 tablets orally followed by 1</td>
</tr>
<tr>
<td>Ketoconazole 200-400</td>
<td>1 tablet once daily</td>
</tr>
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</table>

Table.2 [41]

### VIII. Conclusion:

In view of present global crisis of AIDS causing great amounts of mortality and morbidity where candidiasis infection in such AIDS individual has a high prevalence rate. It is imperative that there is regular monitoring of the patient suffering from AIDS for opportunistic infection like candidiasis which can serve as marker for progression of disease. Oral candidiasis in AIDS individual can be easily diagnosed using the technique described above. And should be treated to reduce the discomfort of the patients.

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