Multiple variants of oral candidiasis as a predictor for HIV/AIDS diagnosis-A case report

Jayapriya Ramadurai[1], T.N. Uma Maheswari[2], Mukundh Chaithanya V[1]

[1]Post Graduate Student, [2]Professor, Department of Oral Medicine and Radiology, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

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Corresponding author: **T. N. Uma Maheswari.** Email: umasamsi@gmail.com

ABSTRACT

Oral candidiasis is the most common opportunistic infection in human immunodeficiency virus infection. History, clinical examination, investigation and diagnosis of oral candidiasis is important for early diagnosis and treatment of HIV infection. Pseudomembranous candidiasis and erythematous candidiasis are associated with immune compromised state and so it is of prognostic significance like reduction in the viral load and CD4 positive T lymphocytes. In this case report, we present a case of a 34-year-old physically challenged asymptomatic male who came for replacement of mandibular anterior tooth who was diagnosed with all four common variants of oral candidiasis which predicted HIV infection. Quality of life of the patient was improved after the diagnosis and treatment.

Keywords: Acquired immunodeficiency syndrome; human immunodeficiency virus; anti-retroviral therapy; oral candidiasis; opportunistic infection.

INTRODUCTION

*Candida albicans*is a fungal organism present as a commensal in the oral mucosa. It is a dimorphic fungus which grows both as yeast form and hyphal form (1). When the host immune system is defective, there is transformation of commensal state to the infective state (2, 3). During this process, there is a morphological alteration from yeast form to hyphal form, latter being more adherent and competes with other microflora for host binding site which leads to increased virulence of Candida species (4). Oral candidiasis is one of the HIV-defining lesions (5). Variety of oral mucosal changes like pseudomembranous candidiasis, erythematous candidiasis, angular cheilitis, hyperplastic candidiasis or candidial leukoplakia, linear gingival erythema, median rhomboid glossitis can occur (6). Treatment of oral candidiasis is by topical and systemic azoleanti fungal drugs (7, 8). The problem with oral candidiasis in HIV patients is the relapse of infection (9). In this case report, we present a case of oral candidiasis with multiple clinical variants which is rare and led to diagnosing HIV, its management and follow-up of the patient.

Case report

A 34 years' old physically challenged male patient was reported to the Department of Oral Medicine, Radiology and Special care dentistry for replacement of missing lower front tooth. History revealed that he had trauma 2 years back after which the lower front tooth was mobile and he visited a dentist and extraction of it was done. Past medical history revealed that during his childhood he had poliomyelitis and lower half of the body was paralyzed. Personal history revealed that he was a tobacco smoker, smokes about 2 cigarettes per day for 9 years. Six months back he quit his habit. Patient was married and his wife died due to tuberculosis 6 months back and does not have any

kids. He did not give any history of having unprotected sex.

General examination revealed muscle atrophy and weakness in lower limbs. Intraoral examination reveals ill-defined erythema on the right buccal mucosa and atrophy of filiform papilla on the dorsal surface of the tongue with wrinkling and fissuring in the left angle of the mouth (Fig.1,2) multiple raised irregularly shaped whitish plaque seen on the palatal mucosa (Fig.3). Left buccal mucosa revealed a non-scrapable white patch about 1x2 cm in size which was scrapped off leaving an erythematous base (Fig.4). The above clinical features led to a provisional diagnosis of chronic hyperplastic in palate, atrophic candidiasis in right buccal mucosa and Pseudomembranous candidiasis in the left buccal mucosa.

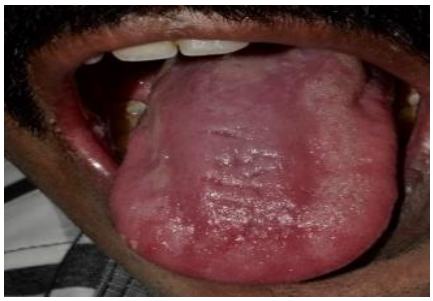

Fig. 1: Atrophic candidiasis in dorsal surface of tongue and angle of the mouth

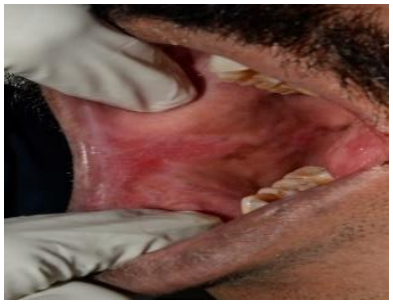

Fig. 2: Atrophic candidiasis in the right buccal mucosa

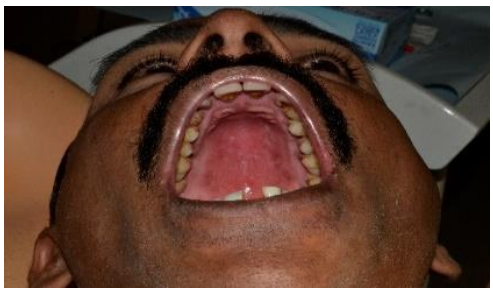

Fig. 3: Chronic hyperplastic candidiasis in palate

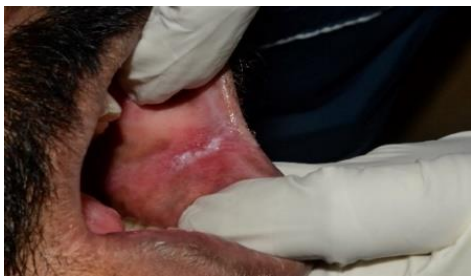

Fig. 4: Pseudomembranous candidiasis in left buccal mucosa

Suspecting HIV, Tri-Dot rapid fourth generation ELISA test which turned out to be positive (Fig.5) with routine hematological examination which showed raised ESR level of 57 mm/hour. Smear from all surface were taken which revealed candidial hyphae confirming the diagnosis (Fig.6).Thus, based on the clinical features and investigations final diagnosis of HIV induced oral candidiasis was made.

Fig. 5: Tri-dot ELISA test positive.

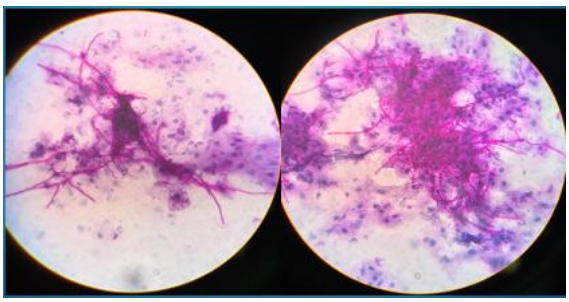

Fig. 6: Ex-foliative cytology showing candidial hyphae

Broad spectrum antifungal like topical clotrimazole1% mouth paint was prescribed daily for 2 weeks and he was informed about the condition and was referred to government hospital for initiation of HAART (Highly Active Anti-Retroviral Therapy). After 2 weeks patient reported with the western blot test report which was positive and CD4 count of 248 cells per cubic millimeter. Table 1 shows the timeline management, follow-up and CD4 cell count. With proper protocol oral prophylaxis and replacement of lower anterior teeth was done and oral hygiene practices were taught. Fig. 7 shows the clinical features after 3 months of follow-up.

Table 1: Timeline management, follow-up and CD4 count.

Timeline	Management	Clinical features	Cd4 cell count
At diagnosis	Topical clotrimazole 1% thrice daily over the affected tissues.	Asymptomatic HC in palate, EC in right buccal mucosa and tongue, PC in left buccal mucosa, AC in Left oral commisure	Not assessed
2 weeks follow up	Topical clotrimazole 1% thrice daily over the affected tissues. HAART	Same features no change in clinical features.	248 cells per cubic millimeter.
1 month follow up	Topical clotrimazole 1% thrice daily over the affected tissues. HAART	Reduction in the size of the lesion, completely healed AC.	323 cells per cubic millimeter.
3 months follow up	Topical clotrimazole 1% thrice daily over the affected tissues. HAART	Relapse of all the lesions, AC in right oral commisure.	352 cells per cubic millimeter.

**PC- Pseudomembranous Candidiasis, AC- Angular cheilitis, EC- Erythematous Candidiasis, HC- Hyperplastic Candidiasis, HAART- Highly Active Antiretroviral Therapy.

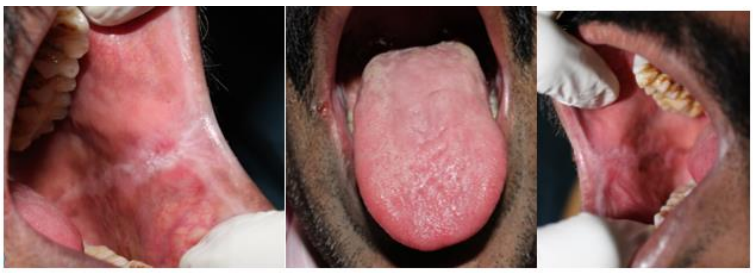

Fig. 7: After 3 months of follow-up

DISCUSSION

According to WHO July 2017 report, there are approximately 36.7 million people living with HIV globally (10). A study conducted with 151 HIV/AIDS patients revealed that the probability of immune failure in the presence of oral candidiasis was 91% men, 94% in females and 96% in intra venous drug users (11). Human immunodeficiency virus infection affects the host immune system by destruction of CD4positive T-Lymphocyte (12). When the T-Helper cells are low they fail to protect the individual and place the host at increased susceptibility to opportunistic infections. According to September 1992 consensus on classification of oral lesions associated with HIV infection oral candidiasis comes under group 1 where in lesions are strongly associated with HIV

infection (13). Four distinct clinical variety of oral candidiasis namely pseudomembranous candidiasis, erythematous candidiasis, hyperplastic candidiasis and angular cheilitis which rarely can coexist in a single patient (14). The term multifocal candidiasis is applied for either for the presentation of a single variant with multiple sites or different variants at two or more sites (15). A case report on oral mycosis in HIV infection by Samaranayake (1993) had similar clinical presentation to our case with all four variants in a single patient, such findings gives a clue to the clinician in diagnosing HIV infection (16). Highly active Anti-Retroviral Therapy first introduced in 1996 have changed the course of HIV infection and have reduced the incidence of HIV – induced oral lesions (17). WHO 2017 report shows only half of the HIV infected individuals are on therapy: 54% of adults and 43% children (10). A Greek study on the effect of HAART on the prevalence of oral candidiasis in HIV infected patients has shown that introduction of HAART was associated with a significant decrease in the prevalence of oral candidiasis with improved CD4 count (18, 19). Many HIV induced oral lesions reduce after HAART but oral candidiasis persists after the initiation of HAART and have higher tendency to recur or develop *de novo* which can be attributed to failure of HAART and multi-drug resistance (20). The increased susceptibility to oral candidiasis in HIV infection is due to two basic factors, one due to weakened systemic immune system and the other is due to increased virulence of candida species due to genetic switching, increased antifungal drug resistance, increased SAP production and increased candida adherence. Hence oral candidiasis can be used as a clinical marker for diagnosing HIV infection.

CONCLUSION

Oral candidiasis can be used as a predictive clinical marker for diagnosing HIV infection as it occurs at early stage of HIV infection, AIDS and during and after HAART. Dentists should be aware of HIV induced oral manifestations, different variants of oral candidiasis which will aid in immediate management thereby increasing the quality of life of the patient.

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