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A Two-Case Series of Oral Candidiasis as an Opportunistic Infection in HIV Patients

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Abstract

Background: Oral candidiasis remains one of the most common opportunistic infections in people living with HIV (PLHIV), particularly in patients with impaired cellular immunity. Pseudomembranous candidiasis is clinically relevant because it may indicate immune deterioration, low CD4+ T-cell count, or advanced HIV disease. **Case Presentation:** This case series describes two HIV-positive male patients who presented with extensive pseudomembranous oral candidiasis. The first patient was a 30-year-old male with known HIV infection and concomitant *Pneumocystis jirovecii* pneumonia. Intraoral examination showed removable whitish-yellow plaques on the tongue, vestibular mucosa, and lips, with an erythematous base after removal. His CD4+ T-cell count was 258 cells/µL. Antiretroviral therapy was initiated during hospitalization, followed by antifungal treatment using nystatin oral drops, ketoconazole, and adjunctive antiseptic mouthwash. The second patient was a 35-year-old ART-naïve male with systemic symptoms and widespread removable pseudomembranous plaques involving the tongue, buccal mucosa, gingiva, and palate. His CD4+ T-cell count was 11 cells/µL, indicating profound immunosuppression. **Conclusion:** Pseudomembranous oral candidiasis may serve as an important clinical marker of CD4+ T-cell depletion and immune deterioration in PLHIV. Dentists should recognize this lesion early because oral findings may support timely referral, HIV disease assessment, and comprehensive management.

Keywords: CD4 lymphocyte count; HIV infection; Opportunistic infections; Oral manifestations; Pseudomembranous candidiasis

Introduction

Oral candidiasis remains one of the most frequent and clinically significant opportunistic infections in people living with HIV (PLHIV). A meta-analysis published in 2021 reported a global prevalence of approximately 35% among HIV-infected individuals, highlighting the persistent burden of this condition[1]. Even in the era of antiretroviral therapy (ART), oral candidiasis continues to be prevalent, particularly among patients with severe immunosuppression or low CD4+ T-cell counts[2].

Recent studies have documented the emergence of antifungal-resistant Candida species in HIV-positive individuals, raising concerns regarding therapeutic challenges[3]. In addition, vitamin D deficiency has been implicated as a potential risk factor for oral candidiasis in HIV-infected patients, suggesting that host nutritional status contributes to disease susceptibility[4]. Epidemiological studies from sub-Saharan Africa have demonstrated that untreated HIV

infection is associated with higher rates of oral candidiasis and increased fungal burden, particularly when CD4+ T-cell counts decline below 200 cells/μL[5].

Clinically, pseudomembranous candidiasis remains the most commonly observed form of oral candidiasis in HIV-infected populations and is frequently associated with advanced immunodeficiency[6]. Beyond causing local discomfort and impaired oral function, oral candidiasis serves as an important clinical marker of immune deterioration and may indicate ART failure or poor immune reconstitution[7]. Therefore, early recognition and appropriate management of oral candidiasis are essential components of comprehensive HIV care.

This case series reports two cases of pseudomembranous oral candidiasis in HIV-positive patients, highlighting their clinical presentation, immunological status, and therapeutic considerations in the current ART era.

Case report

Case 1

A 30-year-old male presented to Prof. Dr. Margono Soekarjo Teaching Hospital, Purwokerto, with progressive shortness of breath, chronic cough, and painful oral lesions. He reported increasing discomfort in the oral cavity and tongue due to extensive fungal deposits. His medical history was significant for human immunodeficiency virus (HIV) infection and a previously diagnosed anal fistula.

On admission, the patient was diagnosed with HIV disease complicated by *Pneumocystis jirovecii* pneumonia based on clinical manifestations and radiological findings. Laboratory evaluation performed during hospitalization revealed a reactive HIV antibody test and a CD4+ T-lymphocyte count of 258 cells/μL, which indicated moderate immunosuppression.

Intraoral examination revealed multiple pseudomembranous deposits with rounded, colony-like configurations consistent with fungal overgrowth. The lesions appeared as yellowish to whitish-yellow plaques with a soft, non-homogeneous surface that could be easily removed using gauze, leaving an erythematous mucosal base. The plaques were distributed over the dorsum and base of the tongue, the vestibular mucosa, and the inner surfaces of the lips, findings consistent with pseudomembranous oral candidiasis (Figure 1). Routine laboratory investigations, including complete blood count and biochemical parameters, were performed for systemic evaluation, and the results are presented in Table 1.

Antiretroviral therapy (ART) was initiated shortly after hospital admission using a once-daily fixed-dose combination regimen. Targeted antifungal therapy was started the following day, consisting of nystatin oral drops and ketoconazole 200 mg twice daily for 5 days. Due to persistent oral lesions, antifungal therapy was extended for an additional 5 days. Adjunctive antiseptic therapy with Minosep gargle was also administered three times daily to reduce the oral microbial load and support mucosal healing.

The combination of respiratory compromise, radiographic evidence of *P. jirovecii* pneumonia, moderate CD4+ T-cell depletion, and extensive pseudomembranous oral candidiasis represented a constellation of HIV-associated opportunistic infections occurring in the setting of progressive immunosuppression.

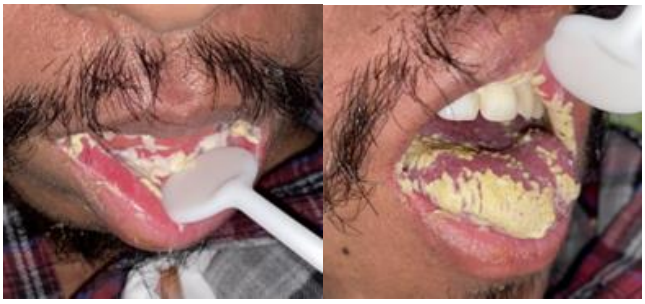

Figure 1. Intraoral photographs of patient 1 showing extensive pseudomembranous candidiasis characterized by removable whitish-yellow plaques on the tongue and oral mucosa

Table 1. Complete blood count of Patient 1

Parameter	Result	Interpre-tation	Unit	Reference Range
Hemoglobin	12.9	Low	g/dL	13.4–17.3
Erythrocytes	4.29	Low	$\times 10^6/\mu L$	4.74–6.32
Platelets	182,000	Low	/mm³	185,000–398,000
Mean corpuscular volume (MCV)	92	High	fL	73–91
Red cell distribution width (RDW)	17.3	High	%	11.3–14.6
Mean platelet volume (MPV)	10.4	High	fL	9.4–12.4
Band neutrophils (Stab cells)	0.6	Low	%	3.0–5.0
Lymphocytes	6.6	Low	%	20.4–44.6
Total lymphocyte count	560	Low	/µL	1,000–10,000
Neutrophil-to-lymphocyte ratio (NLR)	10.35	High	Ratio	0.78–3.53

Case 2

A 35-year-old male presented to Prof. Dr. Margono Soekarjo Teaching Hospital, Purwokerto, with progressive systemic and oral symptoms. His chief complaints included chest

pain, a burning sensation in the chest, nausea, diarrhea, generalized weakness, and intermittent fever. These symptoms had progressively worsened over several weeks.

The patient reported a prior hospitalization at Wiradadi Husada General Hospital one month earlier, during which he received three units of blood transfusion. An HIV antibody test performed during hospitalization was reactive. Subsequent immunological assessment revealed a CD4+ T-lymphocyte count of 11 cells/μL, which indicated severe immunosuppression. No prior antiretroviral therapy had been documented. Subsequent immunological assessment conducted on November 9, 2025, revealed a CD4+ T-lymphocyte count of 11 cells/μL (reference range: 500–1500 cells/μL), indicating severe immunosuppression. No prior antiretroviral therapy (ART) had been documented.

Comprehensive intraoral examination demonstrated widespread pseudomembranous lesions with irregular borders and a morphology suggestive of fungal overgrowth. The lesions appeared as white to yellowish-white plaques with a soft, thick, and non-homogeneous surface (Figure 2). The pseudomembranes were easily removed using gauze, revealing an underlying erythematous mucosa, occasionally accompanied by superficial erosions. The lesions were multifocal and involved the labial and buccal mucosa, vestibular mucosa, dorsum and ventral surfaces of the tongue, gingiva, and palatal mucosa.

The distribution pattern, ease of plaque removal, and presence of an erythematous base were highly consistent with pseudomembranous candidiasis, an opportunistic infection commonly observed in individuals with advanced HIV disease and profound cellular immunodeficiency. Routine laboratory investigations, including complete blood count and biochemical parameters, were performed for systemic evaluation, and the results are presented in Table 2.

In view of the patient's profound immunosuppression, antiretroviral therapy (ART) was initiated during hospitalization using a once-daily fixed-dose combination tablet. The initiation of ART aimed to restore immune function and reduce the risk of further opportunistic infections. The initiation of ART aimed to restore immune function and reduce the risk of further opportunistic infections.

The combination of systemic manifestations, an extremely low CD4+ T-cell count, and extensive pseudomembranous oral candidiasis indicated advanced HIV-associated immunosuppression, placing the patient at high risk for additional opportunistic infections and systemic complications.

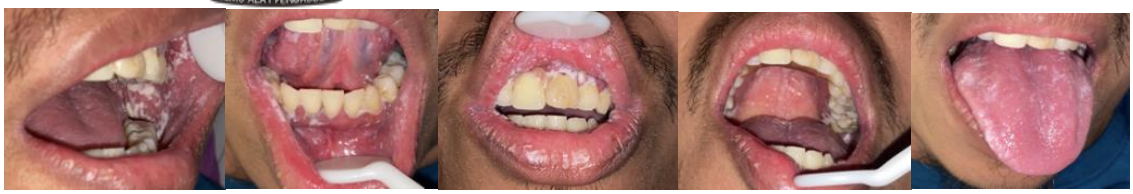

Figure 2. Intraoral photograph of Patient 2 demonstrating white to yellowish-white plaques characterized by a soft, thick, and non-homogeneous surface

Table 2. Complete blood count of Patient 2

Parameter	Result	Interpretation	Unit	Reference Range
Hemoglobin	11.1	Low	g/dL	13.4–17.3
Leukocytes	4280	Low	/mm³	5070-11100
Hematocrit	35	Low	%	40-51
Erythrocytes	4.18	Low	106/uL	4.74-6.32
RDW	19.2	High	%	11.3-14.6
MPV	7.5	Low	fL	9.4–12.4
Eosinophils	0.1	Low	%	0.7 - 5.4
Stem	0.4	Low	%	3.0 - 5.0
Calcium	7.7	Low	mg/dL	8.6-10.3
Natrium	130	Low	mmol/L	136-145
Potassium	2.9	Low	mmol/L	3.5-5.1
Chloride	97	Low	mmol/L	97-107

Discussion

Oral candidiasis is one of the most frequent opportunistic infections in people living with HIV and is strongly associated with impaired cell-mediated immunity. In HIV infection, progressive depletion of CD4+ T lymphocytes reduces the host's ability to control fungal colonization, particularly *Candida albicans*, which normally exists as a commensal organism in the oral cavity. When immune surveillance decreases, *Candida* can shift from a commensal to a pathogenic state through epithelial adhesion, hyphal transformation, enzymatic tissue invasion, and biofilm formation. These mechanisms explain why pseudomembranous candidiasis commonly appears in patients with advanced immunosuppression.[5,12,13]

The two cases in this report demonstrated typical clinical features of pseudomembranous oral candidiasis, including removable whitish-yellow plaques and an erythematous mucosal base after removal. These findings are clinically important because pseudomembranous candidiasis is frequently associated with reduced CD4+ T-cell count and may reflect deterioration of systemic immune status. In the first case, the CD4+ T-cell count was 258 cells/µL, indicating moderate immunosuppression. The presence of concomitant *Pneumocystis*

jirovecii pneumonia suggested that the oral lesion occurred as part of a broader spectrum of HIV-related opportunistic infection. In the second case, the CD4+ T-cell count was markedly lower, at 11 cells/μL, and the oral lesions were more extensive, involving multiple mucosal sites. This comparison supports the clinical relationship between the severity of oral candidiasis, lesion distribution, and the degree of immunosuppression.

The pathogenesis of pseudomembranous candidiasis in HIV-positive patients is closely related to CD4+ T-cell depletion. CD4+ T cells are essential for coordinating antifungal immune responses through cytokine-mediated activation of epithelial defense, neutrophil recruitment, and macrophage function. When CD4+ T-cell levels decline, mucosal defense becomes insufficient, allowing *Candida* to proliferate and invade superficial epithelial layers. The detachable white plaque represents fungal hyphae, desquamated epithelial cells, fibrin, necrotic debris, and inflammatory cells. Removal of the pseudomembrane exposes an erythematous or erosive mucosal surface because of epithelial disruption and local inflammation. This mechanism explains the clinical appearance observed in both patients.

The different CD4+ T-cell counts in these cases may also explain the difference in lesion severity. The first patient had moderate immunosuppression and presented with multifocal lesions involving the tongue, vestibular mucosa, and lips. The second patient had profound immunosuppression and showed more widespread involvement of the tongue, buccal mucosa, gingiva, and palate. This finding is consistent with the concept that lower CD4+ T-cell counts increase susceptibility to more extensive oral opportunistic infections. Therefore, oral pseudomembranous candidiasis should not be viewed only as a local fungal infection, but also as a visible clinical marker of systemic immune deterioration in PLHIV.

Dentists have an important role in the early detection of immune deterioration in PLHIV because oral manifestations may be the first visible sign of advanced HIV disease or poor immune control. Recognition of extensive pseudomembranous candidiasis, especially when lesions are multifocal, recurrent, persistent, or associated with systemic symptoms, should prompt clinicians to consider underlying immunosuppression. Dentists should perform careful oral examination, document lesion distribution and severity, assess risk factors, and refer patients for medical evaluation when HIV status is unknown or when immune deterioration is suspected. In patients with established HIV infection, dentists can contribute to monitoring disease progression by identifying oral lesions that may indicate low CD4+ T-cell count, inadequate ART response, poor adherence, or the need for further systemic assessment.[7,8,9]

Management of oral candidiasis in HIV-positive patients should include both local infection control and systemic immune optimization. Current therapeutic approaches include topical antifungal agents for mild disease and systemic antifungal therapy for more extensive or severe cases. Fluconazole is generally recommended as first-line systemic therapy for oropharyngeal candidiasis, while nystatin, clotrimazole, or miconazole may be considered for selected mild cases. In recurrent or refractory cases, clinicians should evaluate ART adherence, immune status, possible non-albicans *Candida* species, and antifungal resistance. Adjunctive oral hygiene instruction, antiseptic mouthwash when indicated, nutritional support, and regular dental follow-up are also important to reduce recurrence and improve oral comfort.[7,9,15]

These two cases emphasize that pseudomembranous oral candidiasis remains clinically relevant in the ART era. The lesion may reflect the degree of immunosuppression and provide dentists with an opportunity to support early diagnosis, referral, and interdisciplinary management of PLHIV. Early recognition of oral candidiasis is particularly important when lesions are extensive, persistent, or accompanied by systemic manifestations, because these findings may indicate advanced immune dysfunction and increased risk of other opportunistic infections.

Conclusion

Pseudomembranous oral candidiasis may serve as an important clinical indicator of immune deterioration in people living with HIV. In these two cases, the presence and extent of removable whitish-yellow oral plaques were consistent with reduced CD4+ T-cell counts and impaired cellular immunity. The patient with the lowest CD4+ T-cell count showed more widespread oral involvement, supporting the relationship between pseudomembranous candidiasis and the severity of immunosuppression. Dentists play an important role in recognizing these oral manifestations, documenting lesion severity, and referring patients for systemic evaluation or HIV disease monitoring when immune deterioration is suspected. Early detection, appropriate antifungal therapy, optimization of ART, and regular oral follow-up are essential to reduce morbidity and prevent recurrence.

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