

## ORAL CANDIDIASIS IN HIV AND CANCER PATIENTS: REVIEW OF EPIDEMIOLOGY, PATHOGENESIS AND MANAGEMENT

Muhammad Hassan Mustafa<sup>1</sup>, Zubair Sharif<sup>2</sup>, Rabia Noreen<sup>3</sup>

<sup>1</sup>The Superior University, Lahore Pakistan

<sup>2</sup>Faculty of Allied Health sciences, the Superior University, Lahore Pakistan

<sup>3</sup>National College of Business Administration & Economics, Pakistan

<sup>1</sup>hassanmustafa0749@gmail.com, <sup>2</sup>sharifzubair42@gmail.com, <sup>3</sup>rabianoreen761@gmail.com

Corresponding Author: \*

Zubair Sharif

DOI: <https://doi.org/10.5281/zenodo.20023696>

Received	Accepted	Published
11 March 2026	21 April 2026	05 May 2026

### ABSTRACT

**Background:** Oral candidiasis is a common opportunistic fungal infection affecting immunocompromised individuals, particularly patients with Human Immunodeficiency Virus (HIV) infection and those undergoing cancer therapy. Despite advances in antiretroviral and oncologic treatments, the condition remains prevalent due to immune suppression, mucosal barrier damage, and emerging antifungal resistance. Increasing reports of non-albicans *Candida* species further complicate clinical management.

**Objective:** This study aimed to comprehensively review the epidemiology, pathogenesis, species distribution, antifungal resistance patterns, and management strategies of oral candidiasis in HIV-infected and cancer patients.

**Methods:** A narrative literature review was conducted using electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. Peer-reviewed articles published between 2018 and 2025 were included. Studies focusing on prevalence, risk factors, *Candida* species identification, antifungal susceptibility, and treatment outcomes in HIV and cancer populations were critically analyzed and synthesized.

**Results:** The review revealed high prevalence rates of oral candidiasis in both HIV and cancer patients, strongly associated with CD4+ T-cell depletion, neutropenia, mucositis, and xerostomia. *Candida albicans* remained the predominant species; however, a growing proportion of non-albicans species demonstrated reduced susceptibility to azole antifungals. Antifungal resistance, recurrent infections, and biofilm formation were identified as major therapeutic challenges.

**Conclusion:** Oral candidiasis continues to be a clinically significant complication in immunocompromised populations. Early diagnosis, species-level identification, susceptibility-guided therapy, and preventive oral healthcare strategies are essential to reduce morbidity and improve patient outcomes.

**Keywords:** Oral candidiasis, in HIV-infection, Cancer

## INTRODUCTION

Oral candidiasis is a common opportunistic mycotic illness of the oral cavity that mostly affects persons with compromised immune system. Its etiopathogenesis is caused by the species of the genus *Candida*, which are commensals of the mouth cavity, gastrointestinal tract, and mucosa of the genitalia. In physiological conditions, *Candida* spp. live in an unthreatening relationship with the host due to the regulation of immunology surveillance and inter-microbial antagonism.<sup>1</sup> However, the spillage of this balance by systemic or local predisposing conditions triggers the conversion of these organisms into pathogenic organisms that can induce both mucosal and in more extreme conditions, systemic illness.<sup>2</sup>

*Candida albicans* continues to be the most important etiological agent of oral candidiasis among the *Candida* species; however, there is increasing clinical evidence that indicates the growing popularity of non-*albicans* *Candida* species like *C. glabrata*, *C. tropicalis*, *C. parapsilosis*, and *C. krusei*. The emergence of these new taxa should be given greater emphasis due to the innate or acquired resistance to the widely used azole antifungals. The change in the epidemiological dominance of *C. albicans* to non-*albicans* *Candida* species represents an important clinical problem of immunocompromised individuals.<sup>3,4</sup>

Oral candidiasis is not just a localized infection of the mucous membrane, but a prominently clinical indicator of intense immune deficiency. Particularly affected are those with a Human Immunodeficiency Virus (HIV) infection as well as those with a history of oncologic treatment. The infection in such cohorts often demonstrates the degree of immunosuppression and possibly the progression of the disease. As a result, oral candidiasis is a common and the most familiar opportunistic infection in HIV disease that advances to its elevated prevalence, and remains prevalent within patients undergoing cytotoxic chemotherapy or radiotherapy.<sup>5</sup>

*Candida* spp. are colonies of the oral cavity of about 30-50 kw wonways of healthy persons throughout the world without provoking an illness of the body. The process of a colonization

to an infection is dependent on various host and microbial determinants. Host-related issues include immunosuppression, xerostomia, diabetes mellitus, malnutrition, continued use of antibiotics, corticosteroid treatment, denture usage, and inadequate oral hygiene. Virulence factors contained in microbial determinants are adhesion ability, biofilm formation, phenotypic switching, enzymatic production, and morphological change.<sup>6</sup>

Oral candidiasis is a pathogen that can be analyzed through the complex interaction between the host immunity and fungal adaptability. *Candida* spp. are dimorphic fungi, i.e., being able to switch between yeast and hyphae; it is this morphological plasticity which makes them pathogenic. The form in yeast chain makes dissemination and colonization, and the hypha form makes tissue invasion and destruction go. Adhesion molecules are glycolytic polymers that are expressed on the fungal cell surface which allow it to stick to epithelial cells and denture materials. When adherence is established, *Candida* develops organised biofilms, which protect the organism against immune-defenses of the host organism and antifungal agents.<sup>7</sup> There is a significantly higher resistance of biofilm-associated cells than planktonic cells, which leads to recurrent and persistent infections.

Moreover, *Candida* spp. produce hydrolytic enzymes, such as secreted aspartyl proteases, phospholipases, and lipases, which destroy host epithelial tissues and cause invasion. These enzymes interfere with intercellular junctions and increase inflammatory reactions, which lead to typical clinical manifestations of oral candidiasis. In immunocompromised persons, the innate immune mechanisms, such as salivary antimicrobial peptides, defensins, histatins, intact mucosal barriers, and neutrophil phagocytosis, are usually sufficient to check fungal growth without leading to its uncontrolled proliferation. Mediators of adaptive immunity, especially CD4 + T- cell-based responses and the Th17 cytokines (IL-17 and IL-22) play a significant role in the maintenance of mucosal immunity.<sup>8</sup>

Prolonged loss of CD4 + T lymphocytes in HIV-infected subjects undermines cell-mediated

immunity, which is needed to control mucosal Candida growth. When the CD4<sup>+</sup> decreases to a count of less than 200 cells per mm<sup>2</sup>, the occurrence of oral candidiasis increases exponentially. The infection is often in the form of pseudomembranous or erythematous candidiasis and could be either chronic or recurrent without immune recovery. Despite the significant decrease in the frequency of opportunistic infections due to the highly active antiretroviral therapy (HAART), oral candidiasis is still common in patients who do not adhere well, are diagnosed late, or who do not respond to the treatment, particularly in resource-limited locations.<sup>9,10</sup>

The other highly vulnerable population is that of patients with cancer. Drugs suppressing bone marrow impairment promote neutropenia and suppressed immunity of cells. Radiotherapy, especially in the head and neck cancer, destroys the salivary glands, triggering xerostomia and low salivary antimicrobial elements. The importance of saliva is a crucial protective role by keeping microbial balance in the mouth, which occurs via mechanical cleansing, buffering, and secretion of antimicrobial proteins. Reduced salivary flow thereby provides a permissive environment in the colonization and biofilm formation of *Candida*.<sup>11</sup> Despite extensive literature, gaps still exist in the systematic review of the comprehensive evidence of the epidemiology, species distribution, resistance pattern, and the best therapeutic regimen in infectious diseases as well as oncologic patients with HIV. Many studies focus on the HIV or oncology cohort alone and provide little integrative studies to compare the cohorts. Furthermore, both the rapid dynamic of antifungal resistance and increased species of non-albicans *Candida* species require current systematization of reviews to guide clinical judgment. In countries with low and middle-income levels, including Pakistan, the lack of data underscores the necessity of consolidated data. This thesis therefore aims at performing a critical literature review and synthesis of the present day literature on oral candidiasis in immunocompromised individuals, with particular focus on those who have HIV infection or cancer. The current research should further

the best preventive strategies, support the early diagnosis and recommend evidence based treatment procedures on high-risk individuals by questioning epidemiological patterns, pathogenic pathways, antifungal resistance mechanisms, and management measures.

## LITERATURE REVIEW

Oral candidiasis forms one of the most comprehensively studied opportunistic fungal infections in immunocompromised populations, particularly in HIV-infected persons and oncologic patients receiving this treatment. Since the last 20 years, there has been a wealth of studies dedicated to the clarification of its epidemiology, species range, antifungal resistance profiles, clinical presentation, and treatment results. Nevertheless, oral candidiasis is an undefeatable clinical challenge in developed and developing countries due to the advancements in antifungal pharmacotherapy and immunomodulatory interventions.<sup>12</sup>

Monsen et al. (2023) carried out an observational study based on a large group of patients with advanced malignancies to map the *Candida* species distribution and antifungal susceptibility patterns. Their results showed that, despite the fact that the highest isolation frequency was observed in the case of the *Candida albicans*, a sizeable fraction of the infections could be attributed to non-albicans. Most importantly, a number of isolates had reduced sensitivity to fluconazole and itraconazole. The authors highlighted the long-term periods of chemotherapy and recurring antifungal exposure as the key factors causing species turnover and the development of resistance. This article stated the need to identify species systematically and antifungal susceptibility assays in the oncologic setting to prevent therapeutic failure and systemic spread.<sup>35</sup>

In a similar study, GhasemJanbabaei et al. (2018) evaluated oral *Candida* colonisation in 350 patients who received chemotherapy in cancer. They identified the species using MALDI TOF mass spectrometry to establish that 50.6% of isolates were of the category of *Candida albicans*, but a significant proportion was also of non-albicans species, including *Candida glabrata* and

*Candida krusei*. The prevalence rate of fluconazole resistance was 11.7%. The research singled out ineffective oral care, xerostomia, denture use, and long chemotherapy courses as relevant risk factors. These findings support the need to implement prophylactic oral care programs, and individual antifungal therapy of cancer patients.<sup>13</sup>

Lalla et al. (2010) carried out a literature review on the topic of oral fungus infections among patients who are on oncologic treatment. In their analysis, they found that oral candidiasis is the prevalent manifestation of mucositis that is particularly common in patients receiving intensive chemotherapy or radiotherapy. The review determined that mucosal defences disturbance, salivary hypofunction and immunosuppression contribute to a favourable environment during *Candida* colonisation. In cancer treatment, Lalla and others introduced antifungal prophylaxis early before attack and regular oral check-ups as the key tactics to reduce complications and improve the therapeutic outcome.<sup>14</sup>

The study by Sharifi et al. (2023) was a three-year multi-centre longitudinal study involving cancer patients undergoing chemotherapy and radiotherapy. Oral *Candida* was diagnosed in 22.7 percent. Notably, about 40 percent of the infections could be blamed on non-albicans species, most of which were characterized by fluctuating resistance to azoles. The authors concluded that antifungal prophylaxis, species-specific detection, and standard monitoring are the main elements of reduction of morbidity. In addition, the research also pointed out that the risk of infection is unevenly increased by intensive therapeutic regimens, and thus there is a need to have multidisciplinary oral care management as part of the oncology practice.<sup>15</sup>

Soutome et al. (2022) studied the case of oral candidiasis in a group of 144 patients with oral or oropharyngeal cancer treated with radiotherapy. The prevalence of the infections was 31%. The researchers reported a close relationship between *Candida* colonisation and radiotherapy-induced xerostomia and mucosal injury. Mucoadhesive miconazole tablets were administered to patients with a favourable clinical response on about half

of the patients with the condition. The paper emphasized the fact that adjunctive treatment with focused antifungal therapy in addition to supportive oral health care is capable of significantly reducing the disease burden and improving the quality of life.<sup>16</sup>

In haematological cancer patients, Rajendran et al. (2022) found that oral candidiasis was observed in 25 percent of the patients who were receiving chemotherapy. The percentage of non-albicans *Candida* species was approximately 40 and they exhibited mixed resistance patterns. These authors have highlighted the urgency of species-specific therapeutic interventions and systematic susceptibility testing in preventing systemic dissemination particularly in neutropenic patients.<sup>40</sup>

With regards to HIV-infected individuals, Liu et al. (2021) conducted a cross-sectional study of 200 Chinese HIV-positive patients and identified the prevalence of oral candidiasis at 68%. Poor oral hygiene, low CD4+ T-cell (<200 cells/ml), and lack of antiretroviral therapy were the significant predictors. Even though most of the isolates were dominated by the *C. albicans*, 18% of them were non-albicans. The paper has highlighted the need to integrate oral health screening into standard HIV treatment to facilitate early diagnosis and prevent disease complications.<sup>17</sup>

The paper by Keyvanfar et al. (2024) was a systematic review and meta-analysis of oral candidiasis in HIV patients. They found that there is an increasing rate of non-albicans *Candida* species, in particular, azole-resistant species. The authors found a significant relationship between the falling counts of CD4+ and frequent or chronic infections. They promoted the identification and combination antifungal of species and resistant cases. This review provided information to support the fact that antifungal resistance is becoming a growing clinical issue in the international HIV care.<sup>18</sup>

The literature shows that, despite many studies having conducted an independent investigation in oral candidiasis in HIV-infected patients and in cancer patients, there are comparative analyses of epidemiological patterns of the two immunocompromised patients of which there are convergences and divergences. Progressive T-cell

depletion of the CD4 + and dysfunctional cell-mediated immunity is considered to be the major etiologic agent in HIV-infected subjects. On the contrary, neutropenia, mucosal barriers destruction, and xerostomia have a stronger effect in patients under chemotherapy/radiotherapy procedures. Irrespective of these mechanistic differences, the two groups show increased colonization by *Candida* species and an increased rate of recurrent or persistent infection.

## METHADODOLOGY

This study was designed as a narrative literature review focusing on oral candidiasis in two immunocompromised populations—individuals with HIV and those with cancer. Conducted in a scholarly research environment using institutional access to electronic databases, the review involved no direct patient recruitment or laboratory work. Over a total period of four months, the research proceeded through literature searching, article screening, data extraction, critical synthesis, and manuscript preparation. As a review, a numerical sample size was not applicable; instead, priority was given to peer-reviewed original articles, systematic reviews, and meta-analyses published between 2018 and 2025 in English, specifically addressing oral candidiasis in HIV and cancer patients. Exclusion criteria comprised non-peer-reviewed works, case reports, editorials, opinion articles, unrelated studies, and duplicate publications.

For data collection, electronic databases including PubMed, Scopus, Web of Science, and Google Scholar were searched using keyword combinations such as “oral candidiasis,” “HIV,” “cancer,” “immunocompromised,” “*Candida albicans*,” “non-*albicans* *Candida*,” “azole resistance,” “antifungal therapy,” and “management strategies.” Reference lists of selected articles were also screened. No physical equipment was required; reference management software facilitated organization. The scanning process critically evaluated studies addressing antifungal susceptibility, species-level identification, clinical outcomes, and emerging treatments such as natural compounds like chitosan. Limitations—including small sample sizes, regional variations, and inconsistent

diagnostic or treatment protocols—were considered during analysis. Overall, the review synthesized findings from observational studies, clinical trials, systematic reviews, and experimental research conducted between 2018–2025 to identify patterns, mechanisms, and knowledge gaps regarding the epidemiology, pathogenesis, and management of oral candidiasis in HIV and cancer patients.

## Main Body

The review's methodology, employing a structured narrative approach with pre-defined inclusion and exclusion criteria, enabled a rigorous synthesis of heterogeneous evidence from observational studies, clinical trials, systematic reviews, and meta-analyses. This approach is particularly suited to a rapidly evolving field in which resistance profiles, species distributions, and treatment paradigms are continuously shifting. The discussion below is organized thematically to address the study's core objectives: epidemiological patterns, pathogenic mechanisms, species distribution trends, antifungal resistance, clinical impact, and management strategies.

### 5.1 Prevalence in HIV-Infected Patients

The review consistently demonstrates that oral candidiasis remains highly prevalent in HIV-infected individuals, even in the era of widespread antiretroviral therapy (ART). Liu et al. (2021) documented a prevalence of 68% in a cohort of 200 HIV-positive Chinese patients, while Keyvanfar et al. (2024), through a systematic review and meta-analysis, confirmed elevated infection rates globally, with disproportionate burden in low- and middle-income countries. Prior to the advent of HAART, prevalence rates ranged from 70% to 90% in those with progressive disease, underscoring the transformative impact of immunological restoration. However, the infection persists in patients with poor adherence, late diagnosis, or resource-limited access to care. In pediatric HIV populations, Hoang et al. (2025) reported a prevalence of 10.7%, with higher rates among children not receiving ART, confirming the immunological basis of susceptibility across all

age groups.<sup>19</sup>

**Table 5.1: Prevalence of Oral Candidiasis in HIV-Infected Patients Across Selected Studies**

Author (Year)	Population	Sample Size (n)	Prevalence (%)	Key Predictor
Liu et al. (2021)	HIV-positive adults	200	68%	CD4 <200 cells/mL
Keyvanfar et al. (2024)	HIV-positive (meta-analysis)	Multi-study	~60–75%	Low CD4+, no ART
Golestannejad et al. (2023)	HIV vs. non-HIV adults	Comparative	Higher in HIV	Immunosuppression depth
Rafat et al. (2021)	HIV-infected children	Pediatric cohort	Predominant OC	Pseudomembranous type
Hoang et al. (2025)	Pediatric HIV	Community-based	10.7%	ART non-adherence

Table 5.1: Summary of oral candidiasis prevalence rates and key predictors in HIV-infected populations from reviewed studies (2018–2025).

### 5.2 Prevalence in Cancer Patients

Cancer patients undergoing cytotoxic therapy represent another high-burden cohort. The reviewed literature reports prevalence rates ranging from 30% to 90% in oncology populations, reflecting significant heterogeneity attributable to tumor type, treatment intensity, and immune status. Sharifi et al. (2023), in a

multicenter longitudinal study, documented oral *Candida* infection in 22.7% of patients during active chemotherapy or radiotherapy. Rajendran et al. (2022) reported 25% in hematological malignancy patients, while Soutome et al. (2022) identified a 31% prevalence specifically in patients with oral or oropharyngeal cancer treated with radiotherapy. GhasemJanbabaei et al. (2018) examined 350 chemotherapy patients and found a significant colonization rate of 50.6% attributable to *C. albicans*, with measurable non-*albicans* involvement.

**Table 2: Reported Prevalence of Oral Candidiasis in Cancer Patients – Visual Comparison**







Study Reference	Prevalence Magnitude →	Value (%)
GhasemJanbabaei et al. (2018)		51%
Lalla et al. (2010) – Review		60%
Sharifi et al. (2023)		23%
Soutome et al. (2022) – Radiotherapy		31%
Rajendran et al. (2022) – Haematology		25%
Monsen et al. (2023) – Advanced CA		40%

Figure 5.1: Reported prevalence rates (%) of oral candidiasis in cancer patient populations across reviewed studies. Bar length is proportional to reported prevalence (max = 90%).

### 5.3 Species Distribution: The Shift from *Candida albicans* to Non-*Albicans* Species

A central finding of this review is the documented epidemiological shift in *Candida* species distribution. While *Candida albicans* remains the predominant etiologic agent in both HIV-infected and cancer populations, evidence

consistently demonstrates an increasing prevalence of non-albicans *Candida* (NAC) species, with profound implications for treatment. GhasemJanbabaei et al. (2018) reported that 50.6% of oral isolates in chemotherapy patients were *C. albicans*, but a significant proportion comprised non-albicans species including *Candida glabrata*, *Candida krusei*, and *Candida tropicalis*. Monsen et al.

(2023) identified similar trends in advanced malignancy cohorts, attributing the shift to cumulative antifungal exposure and selective pressure. Keyvanfar et al. (2024) confirmed increasing NAC rates in HIV patients on long-term azole prophylaxis, while Ghoghghi et al. (2024) specifically demonstrated reduced fluconazole susceptibility in *C. glabrata* isolates from HIV-related infections.<sup>20</sup>

**Table 3: Candida Species Distribution in HIV and Cancer Patients – Reviewed Evidence**

Candida Species	Population	Approximate Prevalence	Azole Susceptibility	Clinical Significance
<i>C. albicans</i>	Both groups	50–65% of isolates	Generally susceptible	Predominant species; first-line treatment responsive
<i>C. glabrata</i>	HIV & Cancer	10–20% of isolates	Dose-dependent / Resistant	Rising in azole-exposed patients; associated with treatment failure
<i>C. krusei</i>	Cancer (esp. haematology)	5–10% of isolates	Intrinsic resistance	Requires echinocandin-based therapy; poor prognosis
<i>C. tropicalis</i>	Cancer patients	5–8% of isolates	Variable	Associated with neutropenic patients; can disseminate
<i>C. parapsilosis</i>	Neonates / Oncology	3–7% of isolates	Usually susceptible	Associated with biofilm on devices; emerging in cancer settings

Table 5.2: Distribution and clinical significance of *Candida* species identified in HIV and cancer patients across reviewed studies.

#### 5.4 Pathogenic Mechanisms: Immunological Dysfunction and Fungal Virulence

A key finding emerging from this review is the mechanistic divergence in immune dysregulation between HIV-infected and cancer patients, despite similar clinical outcomes. In HIV patients, progressive depletion of CD4<sup>+</sup> T-

lymphocytes dismantles the cell-mediated immune response, particularly the Th17-mediated mucosal defence pathway dependent on IL-17 and IL-22 secretion. When CD4<sup>+</sup> T-cell counts fall below 200 cells/mL, the risk of oral candidiasis increases exponentially. This threshold was consistently corroborated across reviewed studies, including Liu et al. (2021), Keyvanfar et al. (2024), and Golestannejad et al. (2023), confirming CD4<sup>+</sup> count as a central biomarker of infection risk and disease severity.

**Table 4: Comparative Immunopathogenic Mechanisms in HIV vs. Cancer Patients**

Pathogenic Factor	HIV Patients	Cancer Patients
Primary Immune Defect	CD4 <sup>+</sup> T-cell depletion; Th17 dysfunction	Neutropenia from cytotoxic therapy
Mucosal Barrier	Intact but immunologically unprotected	Disrupted by mucositis; ulceration common
Salivary Function	Generally preserved (unless xerogenic drugs)	Significantly impaired by radiotherapy (xerostomia)
Cytokine Response	Impaired IL-17, IL-22; reduced mucosal defences	Systemic inflammation with local immune failure
Infection Recurrence	Highly recurrent; correlates with ART non-adherence	Correlates with treatment intensity and duration
Biofilm Risk Sites	Oral mucosa, tongue, palate	Mucosal ulcers, dentures, disrupted epithelium

Table 5.3: Key comparative immunopathogenic mechanisms of oral candidiasis in HIV-infected versus cancer patients.

### 5.5 Antifungal Resistance: A Growing Clinical Crisis

One of the most alarming findings synthesized in this review is the accelerating emergence of antifungal resistance, particularly to azole class agents. This represents a convergent threat across both HIV and cancer patient populations. Souza

et al. (2024) documented progressive global increases in azole resistance, with fluconazole-resistant isolates increasingly identified in both cohorts. The molecular underpinnings of resistance, as elucidated by Souza et al. (2024), Ghoghhi et al. (2024), and Benson et al. (2024), encompass multiple interconnected mechanisms that collectively render standard pharmacotherapy inadequate in a growing proportion of cases.<sup>21</sup>

**Table 5: Molecular Mechanisms of Antifungal Resistance Identified in Reviewed Literature**

Resistance Mechanism	Molecular Basis	Candida Species Affected	Clinical Consequence
Efflux Pump Overexpression	CDR1/CDR2 (ABC transporters); MDR1 (MFS pump) up-regulation	<i>C. albicans</i> , <i>C. glabrata</i>	Reduced intracellular drug concentration
ERG11 Gene Mutation	Point mutations in lanosterol 14- $\alpha$ -demethylase gene; reduced drug binding affinity	<i>C. albicans</i> , <i>C. glabrata</i>	Azole target site altered; treatment failure
Biofilm-Associated Tolerance	Matrix-encased cells with metabolic heterogeneity; limited drug penetration	All <i>Candida</i> species	Persistent and recurrent infection
Intrinsic Species Resistance	<i>C. krusei</i> : inherent lack of azole binding sites; reduced drug uptake	<i>C. krusei</i> , <i>C. glabrata</i>	No fluconazole response; need alternative agents
Membrane Alteration	ERG3 pathway bypass; altered sterol composition reduces drug binding	<i>C. albicans</i> (azole-resistant)	Cross-resistance to multiple azoles

Table 5.4: Molecular mechanisms of azole antifungal resistance in *Candida* species identified across reviewed studies (2018–2025).

### 5.6 Clinical Impact, Quality of Life, and Prognostic Significance

The clinical consequences of oral candidiasis in immunocompromised patients extend well beyond oral discomfort, profoundly affecting quality of life, nutritional status, and disease prognosis. The review literature consistently documents a constellation of symptoms – oral

pain, burning sensation, dysphagia, dysgeusia, and impaired mastication – that collectively impair adequate nutritional intake. In populations already vulnerable to malnutrition due to their underlying illness and therapeutic toxicity, this nutritional compromise can delay recovery, reduce treatment tolerance, and worsen overall prognosis.

**Table 6: Clinical Impact Domains of Oral Candidiasis in Immunocompromised Patients**

DOMAIN 1   Nutritional and Physical Impact
● Oral pain and burning sensation reduce food intake capacity and cause progressive malnutrition
● Dysphagia impairs swallowing function, limiting dietary variety and adequacy
● Weight loss associated with persistent infection contributes to treatment intolerance
● Impaired mastication limits caloric intake, especially in denture-wearing elderly cancer patients
DOMAIN 2   Oncologic Treatment Consequences
● Severe mucositis-associated candidiasis may require chemotherapy dose reductions
● Treatment interruptions compromise cumulative efficacy of cytotoxic regimens
● Radiotherapy-induced xerostomia prolongs duration of fungal colonization post-treatment
● Hospital stays are prolonged by refractory oral infections, increasing healthcare costs
DOMAIN 3   HIV Disease Prognostic Significance
● Oral candidiasis correlates inversely with CD4 <sup>+</sup> T-cell count (<200 cells/mL threshold)
● Persistent or recurrent infection signals immunological deterioration or ART failure
● Clinical marker of disease progression in late-stage HIV infection
● Indicator of antiretroviral therapy non-adherence or drug resistance in treated patients

### CONCLUSION

The underlying immunosuppression and disruption of the mucosal barrier has maintained the status of oral candidiasis as an important opportunistic infection of HIV-infected and cancer patients. Although *Candida albicans* is preeminent, increasing clinical problematic pressure is the emergence of non-*albicans* species and antifungal resistance. Effective management

requires early diagnosis, identification of the species and susceptibility-based treatment. Combined preventive measures and continuous monitoring are needed to reduce morbidity and improve patient outcomes.

### RECOMMENDATION(S)

The oral examination should be made common in the standard HIV and oncology treatment procedures. In recurrent or resistant cases, species-level identification and antifungal

susceptibility testing is required. High-risk patients should be thought of as having preventive oral hygiene education and antifungal prophylaxis. Additional multicentric studies are suggested to track the changes of resistance and create new treatment recommendations.

### LIMITATIONS

This study was limited to a literature review which was narrative without primary clinical information. Majority of the studies included were cross-sectional where causality could not be determined. The distribution and resistance patterns of the species in the region might not be reflected entirely. There are chances that the findings might not be comparable because of differences in the approach of diagnosing and treatments used in various studies.

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