Al Ameen J Med Sci 2026;19(3):224-229 ● US National Library of Medicine enlisted journal ● ISSN 0974-1143

ORIGINAL ARTICLE CODEN: AAJMBG

Oral candidiasis among cancer patients admitted to a tertiary care hospital: A prospective clinico-mycological study

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Received: 22nd April 2026; ***Accepted:*** 24th June 2026; ***Published:*** 01st July 2026

Abstract: *Introduction:* Oral candidiasis is a commonest opportunistic fungal infection in patients with malignancies. Prompt diagnosis with species identification of etiological agent and antifungal susceptibility testing guided therapy are crucial for effective management. *Aim:* To determine the occurrence of oral candidiasis among cancer patients, the distribution of Candida species, and their respective antifungal susceptibility patterns. *Materials and Methods:* Following informed consent, 300 cancer patients were screened for clinical signs of oral candidiasis. Oral swabs were collected, Candida isolates were identified to the species level and subjected to antifungal susceptibility testing. Demographic data and clinical histories were recorded for all participants. *Results:* Oral candidiasis was identified in 63 patients (21%). Candida albicans was the most frequent isolate (n=28; 44.4%), followed by C. tropicalis (n=21; 33.3%), C. glabrata (n=6), C. kefyr (n=3), C. krusei (n=3), and C. parapsilosis (n=2). High resistance rates were observed for ketoconazole (60%), fluconazole (43%), and miconazole (25%). Among C. albicans isolates, resistance was highest for ketoconazole (71%), while 32% demonstrated resistance to fluconazole. *Conclusion:* The study highlights a significant shift toward Non-albicans Candida species and rising drug resistance. The prevalence of species with intrinsic azole resistance, such as C. glabrata and C. krusei, underscores the necessity of routine identification and antifungal susceptibility testing.

Keywords: Oral Candidosis, Oral Thrush, Candida, Cancer.

Introduction

Oral candidiasis, commonly known as oral thrush, is a fungal infection caused by *Candida* species. These fungi generally reside as commensals in the oral cavity with a high carriage rate. Approximately 30% to 60% of healthy adults carry *Candida* as part of their normal flora [1]. However, these yeasts become invasive and pathogenic under certain conditions, when the local microbial balance is disturbed or when the host's immune defenses are compromised [2]. There has been a significant rise in the prevalence of fungal infections over the past two decades [3].

Oral candidiasis is common among immunocompromised individuals, including cancer patients [4]. Reported prevalence in cancer patients varies widely, from 7% to 52%, depending on geographic location, cancer type, treatment modality (e.g., chemotherapy or radiation), and the patient's immune status [5-6]. Oral candidiasis often leads to local discomfort, altered taste sensation, and dysphagia. If left untreated, it may leads to esophageal overgrowth, resulting in poor nutrition, delayed recovery, and prolonged hospitalization. Furthermore, any time they may leads to Invasive candidiasis which can end up in sepsis in cancer patients due to their compromised nature immunity. Hence oral candidiasis can serve as a clinical marker for systemic disease; in vulnerable patients, the infection may disseminate through the bloodstream or gastrointestinal tract, leading to severe systemic candidiasis with significant morbidity and mortality [4].

While *C. albicans* remains the most prevalent species involved in human colonization and infection, recent studies have reported an increasing incidence of Non-albicans *Candida* (NAC) species among patients with

malignancies [4]. This shift in etiology from *C. albicans* towards NAC species such as *C. krusei*, *C. tropicalis*, *C. glabrata*, and *C. parapsilosis* poses a significant clinical challenge. In addition to acquired drug resistance, several NAC species exhibit intrinsic resistance to commonly used azoles, complicating treatment protocols [4, 7].

Currently, there is a shortage of data regarding species distribution and antifungal resistance patterns in cancer patients. Such data are vital for guiding effective therapy and preventing fatal systemic spread. Therefore, the present study aims to determine the occurrence of oral candidiasis among cancer patients, identify the distribution pattern of various *Candida* species, and evaluate the antifungal susceptibility patterns of the clinical isolates. These data will help in management of oral candidiasis among cancer patients.

Material and Methods

This is a prospective study conducted for a period of six months in the Department of Microbiology, Yenepoya Medical College, Mangaluru. Ethical clearance for this study was obtained from the institutional ethical committee. Cancer patients admitted to Yenepoya Medical College Hospital were included in the study. Patients were informed about the study and consent was obtained from them. All the participants were screened for signs of oral candidiasis such as the presence of white plaque and erythematous lesions.

Patients on antifungal therapy currently or for the past two weeks were excluded from the study. Demographic data and clinical history were also collected. The samples were collected using sterile cotton swabs by swabbing over the lesions and then proceeded with Gram's stain (fig.1), culture using Sabouraud's dextrose agar and Hichrome *Candida* agar. Growth was identified by studying cultural characteristics (fig.2&3), Gram's stain, urease test, germ tube test, corn meal agar test, sugar assimilation tests and fermentation tests by using standard procedure [8-10].

Fig-1: *Candida* seen as Gram positive budding yeast cell

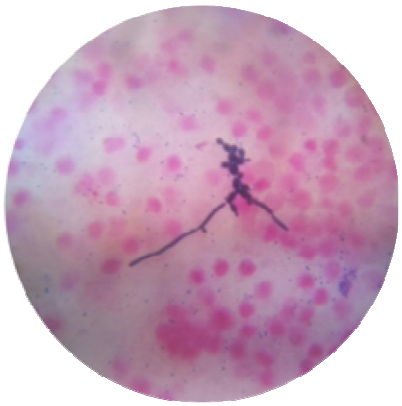


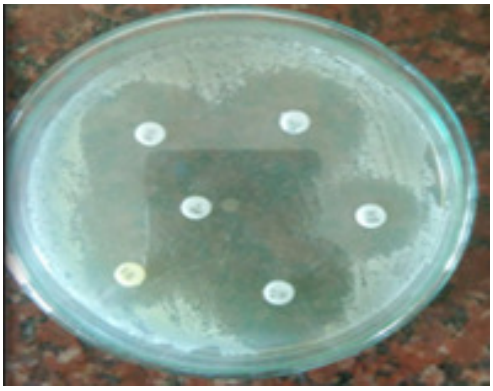
Fig-2: *Candida* on Sabouraud's dextrose agar

Fig-3: Hichrome Candida agar with *Candida species*

Antifungal susceptibility test was performed with fluconazole (25 µg), itraconazole (10 µg), ketoconazole (15 µg), miconazole (50 µg) and voriconazole (1 µg) by disk diffusion method as described by clinical and laboratory standards institute guidelines M44-A2 protocol (fig.4). Mueller Hinton Agar supplemented with 2% dextrose and 5 mg/ml methylene blue was used. The antifungal disks were procured from Himedia laboratories Pvt limited, Mumbai and were stored in desiccators at 4 °C. The zone diameters were interpreted as per the Clinical and Laboratory

Standards Institute approved M442-A2 guidelines. *C. albicans* ATCC 90028 was used as a control [11].

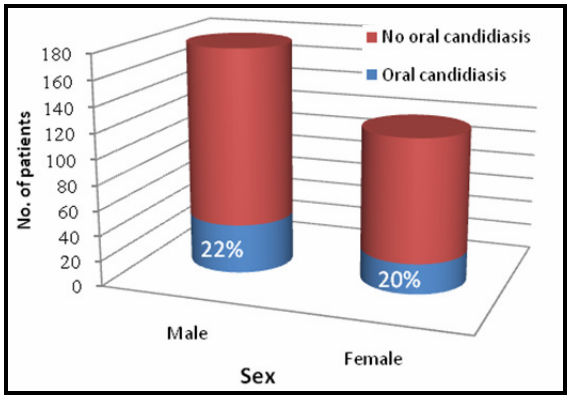
Fig-4: Antifungal susceptibility test by disk diffusion method



Results

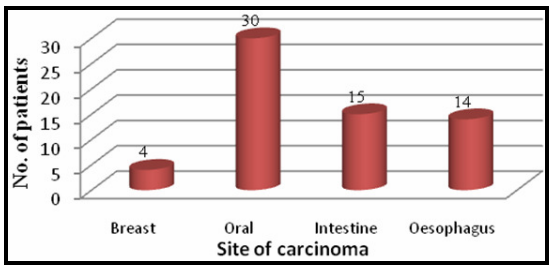
Among 300 patients with malignancy who were screened, 63 patients had oral candidiasis (21%). Oral candidiasis was seen more among male (62%). But the total number of male patients (179) enrolled is more than the female (121). Among 179 male cancer patients, 39 patients had oral candidiasis (22%). Among 121 female cancer patients, 24 patients had oral candidiasis (20%) (Graph-1). There was no statistically significant difference in the prevalence of oral candidiasis between genders (p=0. 68).

Graph-1: Gender distribution of cancer patients

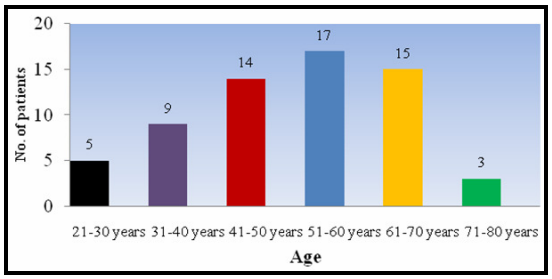


The patients enrolled in the study were identified with different types of carcinoma with oral as the commonest (30), followed by intestine (15), esophagus (14) and breast (4) (Graph-2). Eventhough the study group included individuals from age 4 to 83 years, oral candidiasis was seen among the age group of 26 to 80 years. It was seen more among the patients aged 51-60 years (27%) followed by 61-70 years (24%) and 41-50 years (22%) (Graph-3).

Graph-2: Type of carcinoma among patients with oral candidiasis

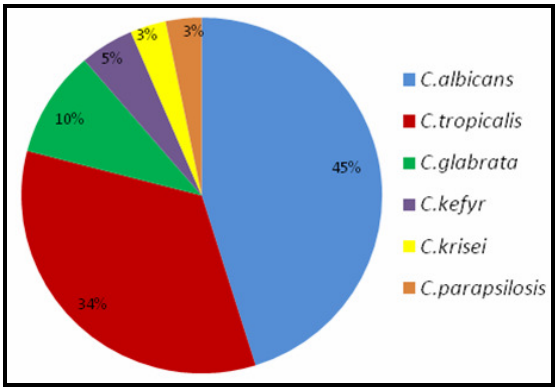


Graph-3: Age distribution of patients with oral candidiasis



Among the various species isolated, *C. albicans* was the most common species (28), followed by *C. tropicalis* (21). Other species include *C.glabrata* (6), *C.kefyr* (3), *C.krusei* (3) and *C. parapsilosis* (2) (Graph-4).

Graph-4: Species distribution of *Candida* among candidiasis



Antifungal susceptibility test was performed for all the isolates for flucoconazole, itraconazole, ketoconazole, miconazole, voriconazole (Table 1). Maximum resistance was seen with ketoconazole (60%) followed

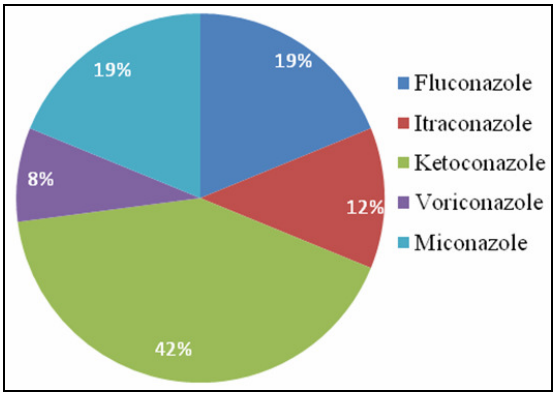
by fluconazole (43%) and miconazole (25%). Resistance was least with voriconazole (25%). In case of *C. albicans* (28), 68% were sensitive to fluconazole, 61% were sensitive to itraconazole, 29% were sensitive to ketoconazole, 86% were sensitive to voriconozole, 64% were sensitive to miconazole. In case of NAC (35) 46% were sensitive to fluconazole, 54% were sensitive to itraconazole, 40% were sensitive to ketoconazole, 63% were sensitive to voriconozole, 71% were sensitive to miconazole (Graph-5).

Table-1: Antifungal susceptibility pattern of Candida species

Antifungal Susceptibility Test															
Species of *candida*	**Fluconazole (No.)**			**Itraconazole (No.)**			**Ketoconazole (No.)**			**Voriconozole (No.)**			**Miconazole (No.)**		
	S	**I**	**R**	**S**	**I**	**R**	**S**	**I**	**R**	**S**	**I**	**R**	**S**	**I**	**R**
C.albicans (28)	19	-	9	17	5	6	8	-	20	24	-	4	18	1	9
C.tropicalis (21)	13	-	8	12	3	6	7	3	11	13	-	8	15	2	4
C.glabrata (6)	-	1	5	2	1	3	4	-	2	5	1		5	-	1
C.kefyr (3)	1	-	2	2	-	1	1	-	2	1	-	2	2	-	1
C.krusei (3)	-	-	3	2	-	1	1	-	2	2	-	1	2	1	-
C.parapsilosis (2)	2	-	-	1	1	-	1	-	1	1	-	1	1	-	1
Total N (%)	**35 (56)**	**1 (2)**	**27 (43)**	**36 (57)**	**10 (16)**	**17 (27)**	**22 (35)**	**3 (3)**	**38 (60)**	**46 (73)**	**1 (2)**	**16 (25)**	**43 (68)**	**4 (6)**	**16 (25)**

S- Sensitive, I-Intermediate, R-Resistant

Graph-5: Azole resistance among *C. albicans*



Discussion

Oral candidiasis is frequently seen in cancer patients and is generally considered a clinical marker of impaired local or systemic defense mechanisms [4-5]. Its significance is that *Candida* infections in patients with malignancies can progress to life-threatening invasive disease, where early detection and treatment are paramount. However, the selection of therapeutic agents must be handled with care as *Candida* species often exhibit both intrinsic and acquired drug resistance. In clinical settings, patients are frequently prescribed antifungal agents based on a provisional clinical diagnosis without further microbiological confirmation. There is a significant lack of data regarding the specific *Candida* species causing oral candidiasis among cancer patients and their corresponding drug resistance patterns in our region. This information is crucial for effective empirical therapy. Therefore, this study was designed to know the species distribution and antifungal susceptibility pattern of *Candida* isolates in this vulnerable population.

Among the 300 patients with malignancy screened in this study, 63 were diagnosed with oral candidiasis, with a prevalence of 21%. This result is consistent with existing literature, where prevalence rates have been reported ranging from 7% to 52% in cancer settings [4, 12-14]. These wide range in prevalence are likely due to geographical variations, differences in sample collection method and interpretation techniques, and specific study population characteristics including type of cancer, the intensity of chemotherapy, and age demographics [14]. In addition, differential diagnosis is complicated by the fact that radiation-induced oral mucositis often presents similarly to, or is superimposed by, oral candidiasis [14].

In the present study, the majority of participants were with oral carcinoma (n = 30), which is reflective of the high enrollment of oral cancer patients in our study. Consistent with previous studies, we found no significant correlation between the primary site of cancer

and the prevalence of oral candidiasis. While the infection was more common among males (62%), this was due to the higher number of male participants (n = 179) compared to females (n = 121). When analyzed within genders, no statistically significant association was found between gender and the prevalence of infection ($p>0.05$), aligning with the findings of Pagheh et al. [4].

Despite the rising global prevalence of Non-albicans *Candida* (NAC), *C. albicans* remains the predominant species isolated in this study (44%), which is consistent with several previous reports [4, 15-16]. Some studies, such as that by Monsen et al., have reported even higher *C. albicans* prevalence rates of up to 72% [17]. Among the NAC species, *C. tropicalis* (n = 21) was the most frequent isolate in our study, echoing many prior findings [4, 14-17]. In contrast, some researchers have identified *C. glabrata* as the most common NAC species in cancer patients [18].

These variations may stem from different epidemiological factors, species identification methods, and patient-specific variables, including prior exposure to antifungal therapy. Although isolated in smaller numbers, the presence of *C. glabrata* (n = 6) and *C. krusei* (n = 3) is clinically significant, as these species often exhibit intrinsic resistance to commonly used azoles, complicating the selection of effective treatment.

Antifungal susceptibility pattern revealed concerningly high resistance rates to ketoconazole (60%) and fluconazole (43%). There is significant variation in azole susceptibility reported in the literature [4, 17-21]. For *C. albicans*, the most common isolate in our study, 32% were resistant to fluconazole. This is comparable to the findings of Vidyut et al., who reported a similar resistance profile [14]. However, other studies, such as those by Monsen and Astvad et al., have reported fluconazole resistance as low as 2% in *C. albicans* isolates [17, 21].

Notably, the highest resistance in our study was observed with ketoconazole, even among *C. albicans* strains. This increasing trend in azole resistance underscores the necessity of regular antifungal susceptibility monitoring to guide clinical management. Moreover, the emergence of multidrug resistance among both *C. albicans* and NAC species poses a significant global health threat, as it severely limits the remaining therapeutic options.

A limitation of this study is the small patient sample size which makes it difficult to categorise and compare the patients with different types of cancer, stage of cancer and treatment modalities used. A future prospective with multicentric studies and drug resistance detection by determining minimum inhibitory concentration for all the azoles could yield further knowledge for establishing guidelines for diagnosis and management of oral candidiasis in cancer patients.

Conclusion

This study demonstrates that although there is a rising prevalence of non-albicans *Candida* (NAC), *C. albicans* remains the predominant pathogen. Increased isolation rate of NAC species with intrinsic azole resistance such as *C. glabrata* and *C. krusei* underscores the critical need for precise species identification. Furthermore, the high level of azole resistance observed in *Candida* species highlights that antifungal susceptibility testing should be performed alongside species identification as a routine practice for all cancer patients with oral candidiasis.

Financial Support and sponsorship: Nil

Conflicts of interest: There are no conflicts of interest.

References

1. Taylor M, Brizuela M, Raja A. Oral Candidiasis. In: StatPearls [Internet]. Treasure Island (FL): *Stat Pearls Publishing*. 2025 Jan [cited 2025 Feb 24]. Available from: *https://www.ncbi.nlm.nih.gov/books/NBK545282*.
2. Hellstein JW, Marek CL. Candidiasis: Red and White Manifestations in the Oral Cavity. *Head Neck Pathol.* 2019; 13(1):25-32.
3. Seagle EE, Williams SL, Chiller TM. Recent Trends in the Epidemiology of Fungal Infections. *Infect Dis Clin North Am.* 2021; 35(2):237-260.

4. Pagheh AS, Kardan F, Ghojoghi A, Sebzari AR, Erfaninejad M, Askari P, et al. Exploring oral candidiasis among cancer patients undergoing chemotherapy in eastern Iran. *Iran J Microbiol.* 2024; 16(6):803-811.
5. Jayachandran AL, Katragadda R, Thyagarajan R, Vajravelu L, Manikesi S, Kaliappan S et al. Oral Candidiasis among Cancer Patients Attending a Tertiary Care Hospital in Chennai, South India: An Evaluation of Clinicomycological Association and Antifungal Susceptibility Pattern. *Can J Infect Dis Med Microbiol.* 2016; 2016:8758461.
6. Di Cosola M, Cazzolla AP, Charitos IA, Ballini A, Inchingolo F, Santacroce L. *Candida albicans* and Oral Carcinogenesis: A Brief Review. *J Fungi (Basel).* 2021; 7(6):476.
7. Lee Y, Puumala E, Robbins N, Cowen LE. Antifungal Drug Resistance: Molecular Mechanisms in *Candida albicans* and Beyond. *Chem Rev.* 2021; 121(6):3390-3411.
8. Fisher F, Cook N. Reagents, stains, media and methods. In: Fisher F, Cook N, editors. Fundamentals of diagnostic mycology. *Philadelphia: Saunders.* 1998; p. 196-225.
9. Odds FC, Bernaerts RI. CHROMagar Candida, a new differential isolation medium for presumptive identification of clinically important *Candida* species. *J Clin Microbiol.* 1994; 32(8):1923-1929.
10. World Health Organization. Laboratory manual for diagnosis of fungal opportunistic infections in HIV/AIDS patients. *Geneva: WHO.* 2009; p.73-74.
11. Clinical and Laboratory Standards Institute. Method for antifungal disk diffusion susceptibility testing of yeasts: Approved guideline M44-A2. *Wayne (PA): CLSI.* 2009; p.65-74.
12. Maheronnaghsh M, Fatahinia M, Dehghan P, Teimoori A. Identification of *Candida* species and antifungal susceptibility in cancer patients with oral lesions in Ahvaz, Southern West of Iran. *Adv Biomed Res.* 2020; 9:50.
13. Alnuaimi AD, Wiesenfeld D, O'Brien-Simpson NM, Reynolds EC, McCullough MJ. Oral *Candida* colonization in oral cancer patients and its relationship with traditional risk factors of oral cancer: a matched case-control study. *Oral Oncol.* 2015; 51(2):139-145.
14. Prakash V, Singh RK, Saurabh K, Kumar V, Kumari R, Kumar S, et al. Spectrum of chemo-radiotherapy induced fungal infection in head and neck cancer patients at tertiary care centre of Eastern India. *Oral Oncol Rep.* 2023; 6:100067.
15. Souza E Silva VC, Oliveira VC, Sousa ÁFL, Bim FL, Macedo AP, Andrade D et al. Prevalence and susceptibility profile of *Candida* spp. isolated from patients in cancer therapy. *Arch Oral Biol.* 2020; 119:104906.
16. Ghojoghi A, Khodavaisy S, Mahmoudabadi AZ, Nazar E, Fatahinia M. Exploring the diversity of uncommon oral yeast species and associated risk factors among substance abusers in southwestern Iran. *Sci Rep.* 2024; 14:1906.
17. Monsen RE, Kristoffersen AK, Gay CL et al. Identification and susceptibility testing of oral candidiasis in advanced cancer patients. *BMC Oral Health.* 2023; 23(1):223.
18. Ramirez-Amador V, Silverman S Jr, Mayer P, Tyler M, Quivey J. Candida colonization and oral candidiasis in patients undergoing oral and pharyngeal radiation therapy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997; 84(2):149-153.
19. Afraseyabi S, Afkhamzadeh A, Sabori H, Verdi F, Khaksar N, Mosavei B. Oral candidiasis amongst cancer patients at Qods hospitals in Sanandaj. *Afr J Clin Exp Microbiol.* 2011; 12(3):121-125.
20. Astvad K, Johansen HK, Høiby N, Steptoe P, Ishøy T. Oropharyngeal Candidiasis in Palliative Care Patients in Denmark. *J Palliat Med.* 2015; 18(11):940-944.
21. Shafi FT, Padmaraj SR, Mullessery NP. Species distribution and antifungal susceptibility pattern of *Candida* causing oral candidiasis among hospitalized patients. *Arch Med Health Sci.* 2015; 3:247-251.

Cite this article as: Jishitha K and Faseela TS. Oral candidiasis among cancer patients admitted to a tertiary care hospital: A prospective clinico-mycological study. *Al Ameen J Med Sci* 2026; 19(3): 224-229.

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