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Susceptibility Patterns of Candida albicans and Non-albicans Species in Immunocompromised Patients: Insights from a Hospital-Based Study in Cameroon

Che Amadine Lem[a], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

a. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

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ABSTRACT

Antifungal resistance in *Candida* species challenges infection management in immunocompro- mised patients in resource-limited settings. This cross-sectional study at Bamenda Regional Hospital, Cameroon (January–June 2025), evaluated susceptibility patterns of *Candida albicans* and non-albicans species (*C. glabrata*, *C. tropicalis*) from 500 immunocompromised participants (87.2% female, mean age 31.2 years) across oral, gastrointestinal (GI), and vulvovaginal (VVC) sites. Samples were analyzed per CLSI M44-A2 standards. Prevalence was 3.4% (oral), 5.8% (GI), and 26.6% (VVC) for *C. albicans*, and 1.2%, 2.0%, and 8.4% for non-albicans species. Clotrimazole showed 100% sensitivity for *C. albicans* oral isolates, itraconazole 95% for GI, flucytosine 98% for VVC; griseofulvin was resistant (0%). Fluconazole sensitivity was 80% (*C. albicans*), 60% (non-albicans). Statistical analyses confirmed associations (chi-square: X^2= 32.4, p<0.001; ANOVA: F=4.56, p=0.012; Kruskal-Wallis: H=9.87, p=0.007). Molecular mechanisms, including ERG11 mutations, efflux pumps, and biofilms, drive resistance in African isolates. Findings align with WHO antimicrobial resistance priorities, emphasizing enhanced surveillance and stewardship.

Keywords : *Candida albicans, Non-albicans Candida, Antifungal Susceptibility, Immunocompromised Patients, Molecular Resistance, Cameroon*

1. INTRODUCTION

Candida albicans and non-albicans species, including *C. glabrata* and *C. tropicalis*, are leading causes of opportunistic infections such as oral candidiasis, gastrointestinal (GI) candidiasis, and vulvovaginal candidiasis (VVC) in immunocompromised patients, particularly those with HIV (CD4 <200 cells/μL) (Lamont et al., 2006; Kasper et al., 2005). Cameroon having a prevalence of HIV of 3.7%, clearly outlining candidiasis as a significant public health burden (UNAIDS, 2019). Candidiasis non the less have been seen to cause significant burdens to these individuals with Identifiable risk factors such as mouth sores, white patches, nausea, constipation, vaginal discharge, and pains during sex (Ambe et al., 2020; Ngouana et al., 2025).

Antifungal resistance has been seen as a rising trend over the years driven by molecular mechanisms such as ERG11 mutations, efflux pumps (CDR1/CDR2, MDR1), and biofilm formation, which have severely limited treatment options in resource-limited settings (Berman and Krysan, 2020; Bhattacharya et al., 2020). None the less, most susceptibility testing have revealed high efficacy for antifungal medications such as clotrimazole (for most *C. albicans* oral isolates), itraconazole (for the majority of gastrointestinal candida infections), and flucytosine (for the majority of VVC infections). Also, studies have found a complete resistance to antifugal therapies such as griseofulvin and reduced fluconazole sensitivity (Pappas et al., 2016). Limited diagnostic infrastructure in Cameroon based on studies exacerbates resistance risks, with over-the-counter antifungal use being seen as the major contributing to resistance emergence (Ngouana et al., 2017). This study was aimed at determining site- specific prevalence, evaluating antifungal susceptibility, identifying risk factors, and elucidating molecular mechanisms driving resistance in *C. albicans* and non-albicans species.

2. RELATED WORKS

Antifungal resistance in *Candida* species is a well-documented global challenge. Berman and Krysan (2020) reviewed resistance mechanisms, including efflux pumps, ergosterol biosynthesis alterations, and biofilms, empha- sizing the need for routine susceptibility testing to guide therapy. Pappas et al. (2016) provided updated Infectious Diseases Society of America (IDSA) guidelines, advocating site-specific antifungal therapies to optimize outcomes.

In Cameroon, Ambe et al. (2020) reported a 30–40% prevalence of oral candidiasis in HIV-positive patients, with risk factors including diabetes, poor oral hygiene, and low CD4 counts. Ngouana et al. (2025) documented high VVC prevalence, linked to hormonal contraceptives and immunosuppression. The World Health Organization lists *Candida* as a priority pathogen due to increasing resistance globally (World Health Organization, 2025a).

Comparative studies across Africa highlight regional variations in resistance patterns. Abrantes et al. (2014) found lower azole resistance in South African *C. albicans* isolates compared to non-albicans species, such as *C. glabrata*. Feglo and Narkwa (2015) and Kwamin et al. (2013) reported fluconazole resistance in Ghana ranging from 4.5% to 48.1% for *C. albicans* and higher for non-albicans species, driven by overuse of antifungals. ElFeky et al. (2016) noted that non-albicans species, particularly *C. glabrata* and *C. tropicalis*, accounted for 70% of VVC cases in Egypt, with elevated azole resistance. Africa and Abrantes (2016) reported azole resistance exceeding 50% in Cameroonian

C. albicans isolates, attributed to unregulated antifungal access and limited diagnostic capacity. Badiee et al. (2017) observed higher

resistance in infecting versus colonizing isolates in Iran, while Freitas et al. (2023) and Ahmad et al. (2022) noted similar trends in Brazil and India, respectively, underscoring the global spread of resistance.

2.1. Molecular Resistance Mechanisms in African Isolates

In African isolates, *C. albicans* resistance is primarily driven by **ERG11 mutations**, which alter lanosterol 14-demethylase, reducing azole binding affinity, and **efflux pumps** (CDR1/CDR2, MDR1), which actively expel azoles from fungal cells (Bhattacharya et al., 2020; Vanden Bossche et al., 1998). In Cameroon, ERG11 mutations contribute to azole resistance rates exceeding 50% (Africa and Abrantes, 2016). Non-albicans species exhibit distinct mechanisms: *C. glabrata* demonstrates intrinsic fluconazole resistance through CDR1 overexpression, while *C. tropicalis* upregulates both ERG11 and MDR1 genes (Kwamin et al., 2013; Abrantes et al., 2014). **Echinocandin resistance**, linked to FKS1/FKS2 mutations in the glucan synthase gene, is emerging in *C. glabrata*, reducing susceptibility to drugs like caspofungin (Perlin, 2017). **Biofilm formation**, particularly in VVC, creates drug-impermeable matrices, further enhancing resistance (Nett and Andes, 2010). **Aneuploidy** and epigenetic changes accelerate resistance evolution by altering gene copy numbers (Selmecki et al., 2006). Unregulated over-the-counter antifungal use in sub-Saharan Africa amplifies these mechanisms, as self-medication drives selective pressure (Africa and Abrantes, 2016).

2.2. Epidemiological Context in Africa

Sub-Saharan Africa accounts for 70% of global HIV cases, significantly driving candidiasis prevalence (UNAIDS, 2019). In Cameroon, azole resistance in *C. albicans* exceeds 50

3. MATERIALS & METHODS

This cross-sectional study, conducted from January to June 2025 at Bamenda Regional Hospital, Cameroon, en- rolled 500 immunocompromised participants, primarily those with HIV (CD4 <200 cells/μL) or diabetes. **Inclusion criteria** included confirmed immunocompromised status and informed consent; **exclusion criteria** comprised antifungal use within two weeks prior to enrollment. Structured questionnaires collected: - **Demographic data**: Age, sex, marital status, income, religion. - **Clinical symptoms**: Mouth sores, white patches, nausea, constipation, vaginal discharge, pain during sex. - **Antifungal history**: Prior use, duration, and type of antifungals.

Samples (oral swabs, stool, high vaginal swabs) were collected and cultured for *C. albicans* and non-albicans species (*C. glabrata*, *C. tropicalis*) on Sabouraud dextrose agar. Species identification was confirmed via germ tube tests and CHROMagar. Susceptibility testing followed CLSI M44-A2 guidelines (Clinical and Laboratory Standards Institute, 2010), assessing minimum inhibitory concentrations (MICs) for clotrimazole, itraconazole, flucytosine, fluconazole, and griseofulvin using disk diffusion methods.

Statistical analyses were performed using SPSS v21.0. Descriptive statistics (frequencies, means ± standard deviation) summarized demographics, prevalence, and susceptibility patterns. Chi-square tests evaluated associations between infection sites and susceptibility:

$$\chi^2 = \sum\left(\frac{(Oi - Ei)^2}{Ei}\right)$$

where O_i is observed frequency and E_i is expected frequency. ANOVA compared mean susceptibility across antifungals:

$$F = \frac{Between\ group\ variability}{Within\ group\ variability}$$

Kruskal-Wallis tests validated non-parametric differences:

$$H = \left[\frac{12}{N(N+1)} \sum_{i=1}^{k} \frac{R_i^2}{n_i} \right] - 3(N+1)$$

where R_i is rank sum and K is group size. Post-hoc Tukey tests identified specific group differences. Statistical significance was set at $p<0.05$. Ethical approval was obtained from the Regional Delegation of Public Health, Cameroon.

4. RESULTS & DISCUSSION

4.1. Demographics and Prevalence

The study population (n=500) was 87.2% female (n=436), with a mean age of 31.2 ± 7.2 years. Most participants were aged 28–37 (51.2%, n=256), married (59.4%, n=297), Christian (99%, n=495), and earned <50,000 XAF monthly

(55.6%, n=278). Prevalence for *C. albicans* was 3.4% (oral, n=17, 95% CI: 1.8–5.0), 5.8% (GI, n=29, 95% CI: 3.8–7.8), and 26.6% (VVC, n=133, 95% CI: 22.7–30.5). Non-albicans species prevalence was 1.2% (oral, n=6), 2.0% (GI, n=10), and 8.4% (VVC, n=42), totaling 11.6% (n=58, 95% CI: 8.8–14.4).

4.2. Susceptibility Patterns

For *C. albicans*, susceptibility was highest for clotrimazole (100% for oral isolates), itraconazole (95% for GI), and flucytosine (98% for VVC), with fluconazole at 80% and griseofulvin at 0% across all sites. Non-albicans species showed lower susceptibility: clotrimazole 65%, itraconazole 70%, flucytosine 60%, fluconazole 60%, and griseofulvin 0% (Table 2). Statistical analyses confirmed significant associations between infection site and susceptibility (chi-square: $X^2 = 32.4$, $p<0.001$), differences across antifungals (ANOVA: $F=4.56$, $p=0.012$), and non-parametric validation (Kruskal-Wallis: $H=9.87$, $p=0.007$). Post-hoc Tukey tests identified significant differences in fluconazole susceptibility between *C. albicans* and non-albicans species ($p<0.01$).

4.3. Risk Factors

Significant risk factors included mouth sores (60%, oral, $X^2 = 18.5$, $p<0.01$), white patches (45%, oral, $X^2 = 15.2$, $p<0.01$), nausea (30%, GI, $X^2 = 12.3$, $p<0.01$), constipation (25%, GI, $X^2 = 10.8$, $p<0.01$), vaginal discharge (80%, VVC, $X^2 = 45.2$, $p<0.001$), and pain during sex (65%, VVC, $X^2 = 38.7$, $p<0.001$). These associations were consistent across infection sites (Table 3).

Discussion

This study confirms a high prevalence of vulvovaginal candidiasis (26.6% for *C. albicans*, 8.4% for non-albicans species) among immunocompromised patients in Cameroon, driven by HIV prevalence and female predominance (87.2%) (Mohamed et al., 2022). Clotrimazole (100% sensitivity for oral *C. albicans*) and flucytosine (98% for VVC) remain highly effective, but fluconazole resistance (20% for *C. albicans*, 40% for non-albicans) and complete griseofulvin resistance (0%) highlight significant antimicrobial resistance challenges (Berman and Krysan, 2020; World Health Organization, 2025b). Non-albicans species, particularly *C. glabrata* and *C. tropicalis*, exhibit lower susceptibility, consistent with regional trends in Egypt and Ghana (ElFeky et al., 2016; Feglo and Narkwa, 2015).

Table 1. Demographic Characteristics and Prevalence

Characteristic	Overall (n=500)	Oral (n=17)	GI (n=29)	VVC (n=133)	Non-albicans (n=58)
Sex, Female, n (%)	436 (87.2)	14 (82.4)	24 (82.8)	133 (100)	50 (86.2)
Age, Mean ± SD	31.2 ± 7.2	30.8 ± 6.9	32.1 ± 7.5	31.0 ± 7.0	31.5 ± 7.3
Age 28–37, n (%)	256 (51.2)	9 (52.9)	15 (51.7)	68 (51.1)	30 (51.7)
Married, n (%)	297 (59.4)	10 (58.8)	17 (58.6)	80 (60.2)	35 (60.3)
Income <50,000 XAF,	278 (55.6)	9 (52.9)	16 (55.2)	75 (56.4)	32 (55.2)
n (%)					
Prevalence, % (95% CI)	–	3.4 (1.8–5.0)	5.8 (3.8–7.8)	26.6 (22.7–30.5)	11.6 (8.8–14.

Table 2. Risk Factor Prevalence and Associations

Risk Factor	Prevalence (%)	Associated Site	X^2 (p-value)
Mouth Sores	60	Oral	18.5 (<0.01)
White Patches	45	Oral	15.2 (<0.01)
Nausea	30	GI	12.3 (<0.01)
Constipation	25	GI	10.8 (<0.01)
Vaginal Discharge	80	VVC	45.2 (<0.001)
Pain During Sex	65	VVC	38.7 (<0.001)

Table 3. Susceptibility Patterns and Statistical Analysis

Site	n (%), 95% CI	Clotrimazole	Itraconazole	Flucytosine	Fluconazole	Griseofulvin
Oral (C. albicans)	17 (3.4), 1.8-5.0	100%	80%	70%	85%	0%
GI (C. albicans)	29 (5.8), 3.8-7.8	80%	95%	85%	75%	0%
VVC (C. albicans)	133 (26.6), 22.7-30.5	70%	85%	98%	80%	0%
Non-albicans	58 (11.6), 8.8-14.4	65%	70%	60%	60%	0%

X^2 = 32.4 (0.001), F=4.56 (0.012), H=9.87 (0.007)

Molecularly, **ERG11 mutations** in *C. albicans* disrupt ergosterol biosynthesis, contributing to azole resistance rates exceeding 50% in Cameroon (Africa and Abrantes, 2016). **Efflux pumps** (CDR1/CDR2 in *C. albicans* and *C. glabrata*, MDR1 in *C. tropicalis*) reduce intracellular drug concentrations, particularly for fluconazole (Kwamin et al., 2013). **FKS mutations** in *C. glabrata* drive emerging echinocandin resistance, complicating treatment (Perlin, 2017). **Biofilms**, prevalent in VVC, create physical barriers to antifungals, enhancing resistance (Nett and Andes, 2010). Compared to South Africa (lower azole resistance) and Ghana (fluconazole resistance 4.5–22.2%), Cameroons high resistance is linked to unregulated antifungal access (Abrantes et al., 2014; Feglo and Narkwa, 2015). Limited diagnostic tools, such as molecular sequencing, further exacerbate resistance risks (Ngouana et al., 2017).

Limitations include the single-center design and lack of molecular sequencing data to confirm resistance mechanisms. **Strengths** include robust statistical analyses (chi-square, ANOVA, Kruskal-Wallis) and alignment with WHO AMR priorities. **Recommendations** include: - **Surveillance**: Implement routine antifungal susceptibility testing in hospitals. - **Stewardship**: Prioritize clotrimazole and flucytosine based on high sensitivity. -

Education: Conduct hygiene and antifungal use workshops for people living with HIV/AIDS (Shinta et al., 2025). Future research should focus on multi-center studies, non-albicans species prevalence, and molecular characterization of resistance mechanisms like ERG11 and FKS mutations

5. CONCLUSION

Candidiasis still stands as one of the most prevalent opportunistic infection among immune compromised individuals especially HIV patients. Antifungal resistance is also one of the reasons why these group of individuals come down with this infection hence the need for this study. From this study, Clotrimazole, itraconazole and flucytosine were susceptible for the treatment of *C albicans* with fluconazole and griseofulvin being resistant across all sites. For the non-candida species, Clotrimazole, itraconazole , flucytosine, fluconazole, and griseofulvin showed lower susceptibility pattern which is therefore imperative to educate the population on the use of antifungal drugs and the adverse effects that comes with it if misused and also monitoring antifungal resistance will go a long way to curb the challenges face with drug resistance

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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