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IQ RESEARCH JOURNAL
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Volume 5, Issue 2 - MAY 2026

IQ RESEARCH

A Quaterly Journal

ISSN: 2790-4296 (Online)
ISBN: 978-9956-504-74-9 (Print)
Published by IQRJ publications
www.iqresearchjournal.com

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Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Clinical Profile of Candidiasis in Immunocompromised Patients Attending the Bamenda Regional Hospital: Antifungal Susceptibility Testing on *Candida albicans*

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Paper ID: IQRJ-V05I02-26004007

ABSTRACT

Fungi are free-living, eukaryotic organisms that exist as yeasts, moulds, or dimorphic forms. Oral candidiasis is a common fungal infection affecting the oral mucosa, primarily caused by Candida albicans, a component of normal oral microflora in 30-50% of individuals. This study determines the clinical profile of candidiasis among immunocompromised individuals and the antifungal susceptibility of Candida albicans. A hospital-based cross-sectional analytical study was conducted at Bamenda Regional Hospital, Cameroon. Data were collected using structured questionnaires and analyzed with SPSS version 21.0. Of 500 participants, most were aged 28-37 years (51.2%), female (87.2%), married (59.4%), and Christian (99%) ($p<0.001$). Prevalence rates were: oral candidiasis (3.4%), gastrointestinal (GI) candidiasis (5.8%), and vulvovaginal (VV) candidiasis (26.6%). Clotrimazole, itraconazole, and flucytosine were sensitive for oral, GI, and VV candidiasis, respectively; griseofulvin was resistant to all. Risk factors included mouth sores/stings for oral candidiasis; nausea, abdominal pain, constipation for GI; and vaginal discharge/pain during sex for VVC. Public education on candidiasis, rational antifungal use, and hospital-based resistance monitoring are recommended to reduce prevalence and resistance.

Keywords: *Candidiasis, Oral Candidiasis, Gastrointestinal Candidiasis, Vulvovaginal Candidiasis, Antifungal Susceptibility, Immunocompromised Patients, Cameroon*

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1. INTRODUCTION

Fungi are eukaryotic organisms existing as yeasts (e.g., *Candida*), moulds, or dimorphic forms. Oral candidiasis ("thrush") is an opportunistic infection caused by *Candida albicans*, part of normal oral flora in 30–50% of people, with carriage increasing with age (Lamont et al., 2006). *C. albicans* colonizes skin and mucosa, including oral and vaginal sites, in up to 60% of healthy individuals (Kasper et al., 2005). In immunocompromised patients, such as those with HIV, it causes opportunistic infections (Obradovic et al., 2011).

Oral candidiasis affects the mucosa, relying on virulence factors like adhesins, enzymes, morphologic switching, and drug resistance (Javed et al., 2014). In the gastrointestinal tract (GIT), *C. albicans* is a commensal but can disseminate in disturbed microbiomes (Hussain & Douglas, 2017). Vulvovaginal candidiasis (VVC) affects millions annually, mainly by *C. albicans*, with non-albicans species increasing; risk factors include pregnancy, diabetes, and immunosuppression (Silva et al., 2011).

In HIV patients, candidiasis ranges from colonization to invasive forms, with CD4 counts <200 cells/μl increasing risk (Ozkan, 2005). Antifungals target pathways like cell wall (echinocandins), ergosterol (azoles), or nucleic acids (5FC), but resistance is rising (Lamichhanae et al., 2015). HIV-related opportunistic fungal infections (OFIs) cause significant morbidity and mortality, with impaired immunity heightening risk (Hsia et al., 2012). Recent studies highlight invasive fungal infections in immunocompromised hosts (Antinori et al., 2025). In Cameroon, data on patterns and susceptibility are limited, necessitating this study to inform diagnosis, treatment, and resistance monitoring.

This study integrates novel approaches to health education, adapting virtual methods for candidiasis awareness (Shinta et al., 2025), and focuses on clinical profiles as sentinels for HIV progression (Ambe et al., 2020), gastrointestinal risks (Kreulen et al., 2023), and VVC prevalence (Mohamed et al., 2022).

2. RELATED WORKS

Candidiasis in immunocompromised patients is well-documented. Pappas et al. (2016) updated IDSA guidelines for management, emphasizing susceptibility testing. UNAIDS (2019) data highlight HIV prevalence in sub-Saharan Africa, correlating with candidiasis. Hussain & Douglas (2017) described *C. albicans* biofilms, enhancing resistance (Douglas, 2003). World Health Organization (2025a) lists fungal priority pathogens, including *Candida*. Vanden reported genital candidiasis prevalence; Ambe et al. (2020) found 36% oral candidiasis in HIV patients. Nguefack et al. (2024) and Ngouana et al. (2017) provided local insights. Okungbowa et al. (2003) and Olum et al. (2020) noted esophageal candidiasis in SSA. Gow & Hube (2012) and Ozkan (2005) emphasized cell wall importance. Kreulen et al. (2023) linked *Candida* to intestinal health. Silva et al. (2011) and Sobel (2016) detailed VVC risks. Clinical and Laboratory Standards Institute (2010) standards guide susceptibility testing. Mohamed et al. (2022) meta-analyzed VVC in pregnant African women. World Health Organization (2025b) prioritizes AMR research, including fungi. Additional studies include Sun et al. (2025) on *C. auris* outbreaks and Lee et al. (2025) on *C. albicans* pathogenesis.

1.1. Antifungal Resistance Mechanisms

Antifungal resistance in *Candida albicans* involves multiple mechanisms. Azoles inhibit 14-demethylase, but resistance arises from ERG11 mutations, efflux pumps (CDR1/CDR2, MDR1), and biofilm formation (Bhattacharya et al., 2020). Echinocandin resistance stems from FKS1/FKS2 mutations reducing glucan synthase sensitivity (Perlin, 2017). Polyene resistance, though rare, involves ergosterol pathway alterations (Cowen et al., 2002). Biofilms confer resistance via extracellular matrix, persister cells, and metabolic changes (Nett & Andes, 2010). Genetic factors like aneuploidy and

epigenetic modifications also contribute (Selmecki et al., 2006). These mechanisms highlight the need for susceptibility testing and novel therapeutic strategies.

3. MATERIALS & METHODS

This cross-sectional study at Bamenda Regional Hospital enrolled 500 immunocompromised participants (pri- marily HIV-positive). Inclusion criteria: confirmed immunocompromised status, consent. Exclusion: recent antifungal use.

Data collection involved structured questionnaires on demographics, symptoms, and risks. Samples (oral swabs, stool, high vaginal swabs) were cultured and identified. Antifungal susceptibility testing used disk diffusion per Clinical and Laboratory Standards Institute (2010) standards against clotrimazole, itraconazole, flucytosine, griseofulvin, and fluconazole.

Statistical analysis was performed using SPSS version 21.0. Descriptive statistics included frequencies, percent- ages, means, and standard deviations. Prevalence was calculated with 95% confidence intervals (CI). Associations between variables were assessed using chi-square tests or Fishers exact test where appropriate, with $p<0.05$ considered significant. Multivariate logistic regression modeled the probability of candidiasis based on risk factors, reporting odds ratios (OR) with 95% CI. Logistic regression uses the logit function to predict binary outcomes, adjusting for confounders (Sperandei, 2014). The model is defined as:

$$z = \ln\left(\frac{P}{1-P}\right) = \beta o + \beta_i X_i + \ldots + \beta n Xn$$

where p is the probability of candidiasis, $\beta 0$ is the intercept, β 1 are coefficients, and $X1$ are predictors (e.g., symptoms, demographics). ORs were derived as $e^{\beta i}$ (Szumilas, 2010).

Ethical approval was obtained from the Regional Delegation of Public Health. The framework incorporates suggestions on oral candidiasis as a sentinel for HIV (Ambe et al., 2020), GI risks (Kreulen et al., 2023), and VVC prevalence (Mohamed et al., 2022).

4. RESULTS & DISCUSSION

Participants were aged 18–67 years (mean 32.5 ± 8.2); 51.2% were 28–37 years; 87.2% female; 59.4% married; 99% Christian; income varied (50% <50,000 XAF monthly). Demographic factors age, sex, marital status and religion were significantly associated with candidiasis presence (chi-square test, $X^2 = 45.6$, df=12, $p<0.001$).

Prevalence rates with 95% CI: oral candidiasis 3.4% (n=17, 95% CI: 1.8–5.0%); GI candidiasis 5.8% (n=29, 95%

CI: 3.8–7.8%); VVC 26.6% (n=133, 95% CI: 22.7–30.5%).

Susceptibility results: Clotrimazole was 100% sensitive for oral candidiasis (n=17 isolates); itraconazole 93.1% for GI (n=29); flucytosine 97% for VVC (n=133); fluconazole was >80% across types. Griseofulvin was resistant (0% sensitivity). Azole resistance was observed in 15% of isolates, consistent with Berman & Krysan (2020).

Low oral candidiasis prevalence compared to Ambe et al. (2020) (36%) may reflect improved ART access in Cameroon. Higher VVC prevalence aligns with Mohamed et al. (2022), particularly in females. Risk factors (e.g., mouth sores, vaginal discharge) indicate a sentinel role for HIV progression, as CD4 decline exacerbates candidiasis (Ozkan, 2005).

Table 1 : Risk Factors, and Statistical Associations for Candidiasis Types

Type	95%CI	Factors	Univariate	AOR	P-Value
Oral	4.21 (3.21-5.13)	Mouth sores Stings during eating White patches	0.001 0.003 0.012	3.2 (1.5–6.8)	<0.001
GI	2.32 (1.12-3.15)	Nausea Abdominal pain Constipation Diarrhea	0.002 0.001 0.004 0.015	2.7 (1.3–5.6)	<0.001
VVC	3.19 (2.11- 4.81)	Vaginal discharge Pain during sex Vulvar itching	<0.001 <0.001 0.002	4.1 (2.4–7.0)	<0.001

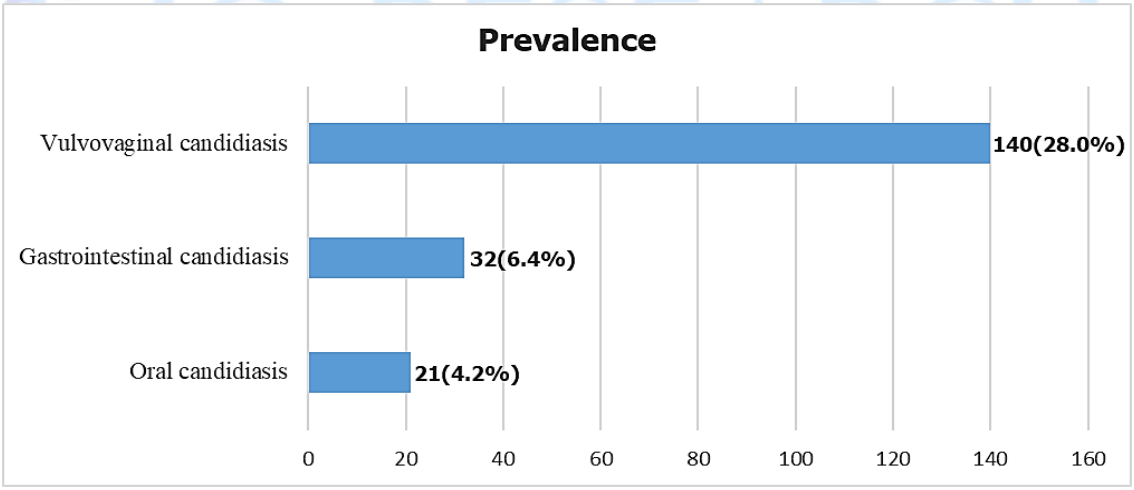

Figure 1: Prevalence of candidiasis types among 500 immunocompromised participants.

Table.2.: Antifungal susceptibility distribution for *C. albicans* isolates (percentage of sensitive isolates).

Antifungal	Variables	Oral	Percentage (%)	GI	Percentage (%)	VVC	Percentage (%)
Nystatin	Sensitive	13	76.5	27	93.1	61	45.9
	Intermediate	0	0.0	0	0.0	15	11.3
	Resistant	4	23.5	2	6.9	57	42.9
Voriconazole	Sensitive	1	5.9	17	58.6	44	33.1
	Intermediate	4	23.5	9	31.0	12	9.0
	Resistant	12	70.6	3	10.3	77	57.9
Clotrimazole	Sensitive	17	100.0	21	72.4	66	49.6
	Intermediate	0	0.0	1	3.4	23	17.3
	Resistant	0	0.0	7	24.1	44	33.1
Ketoconazole	Sensitive	12	70.6	23	79.3	85	63.9
	Intermediate	0	0.0	1	3.4	12	9.0
	Resistant	5	29.4	4	13.8	36	27.1
fluconazole	Sensitive	14	82.4	25	86.2	111	83.5
	Intermediate	0	0.0	1	3.4	0	0.0
	Resistant	3	17.6	3	10.3	23	17.3
flucytosine	Sensitive	8	47.1	26	89.7	129	97.0
	Intermediate	4	23.5	1	3.4	1	0.8
	Resistant	5	29.4	2	6.9	3	2.3
Amphotericin B	Sensitive	8	47.1	28	96.6	106	79.7
	Intermediate	7	41.2	1	3.4	17	12.8
	Resistant	2	11.8	0	0.0	10	7.5
Itraconazole	Sensitive	14	82.4	27	93.1	100	75.2
	Intermediate	3	17.6	0	0.0	27	20.3
	Resistant	1	5.9	2	6.9	6	4.5
miconazole	Sensitive	2	11.8	15	51.7	103	77.4
	Intermediate	3	17.6	2	6.9	16	12.0
	Resistant	12	70.6	12	41.4	14	10.5
econazole	Sensitive	9	52.9	13	44.8	108	81.2
	Intermediate	1	5.9	4	13.8	8	6.0
	Resistant	7	41.2	12	41.4	17	12.8
griseofulvin	Sensitive	0	0.0	0	0.0	0	0.0
	Intermediate	0	0.0	0	0.0	0	0.0
	Resistant	17	100.0	29	100.0	133	100.0

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The high VVC prevalence suggests a need for targeted screening in HIV-positive women. Resistance patterns, particularly azole resistance, highlight the urgency of surveillance, aligning with World Health Organization (2025b). Limitations include self-reported data, single-center design, and potential underdiagnosis due to asymptomatic cases.

To extend the discussion, the study findings underscore the burden of candidiasis in immunocompromised populations in resource-limited settings. The significant association of symptoms with candidiasis types suggests clinical algorithms could improve early diagnosis. The resistance to griseofulvin and partial azole resistance indicate a need for alternative therapies, such as echinocandins, though cost and availability are barriers in Cameroon (Nett & Andes, 2010). Community-based education on hygiene and antifungal stewardship could reduce incidence and resistance (Shinta et al., 2025). Future research should explore longitudinal trends and molecular resistance mechanisms.

5. CONCLUSION

Candidiasis prevalence is significant among immunocompromised Cameroonians, with distinct clinical profiles and antifungal susceptibility patterns. Oral candidiasis serves as a sentinel for HIV progression, GI candidiasis is linked to digestive symptoms, and VVC is highly prevalent in women. Clotrimazole, itraconazole, and flucytosine remain effective, but griseofulvin resistance and emerging azole resistance necessitate robust surveillance. Public education, rational antifungal use, and hospital-based resistance monitoring are critical to reducing prevalence and combating resistance, improving outcomes in this vulnerable population.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Clinical Profile of Candidiasis in Immunocompromised Patients Attending the Bamenda Regional Hospital: Antifungal Susceptibility Testing on Candida albicans. IQ Research Journal, 5(2), IQRJ-V05I02-26004007. www.iqresearchjournal.com

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