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Socio-Demographic Determinants and Risk Factors for Candidiasis in Immunocompromised Patients: Evidence from Northwest Cameroon

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ABSTRACT

Background: Candidiasis, primarily caused by *Candida albicans*, represents a significant opportunistic infection in immunocompromised individuals, particularly those living with HIV. Understanding socio-demographic determinants and risk factors is essential for developing targeted public health interventions.

Objective: To investigate socio-demographic determinants and risk factors associated with oral, gastrointestinal (GI), and vulvovaginal candidiasis (VVC) among immunocompromised patients in Northwest Cameroon.

Methods: A cross-sectional study was conducted at Bamenda Regional Hospital involving 500 immunocompromised patients. Data were collected through structured questionnaires assessing socio-demographic characteristics, candidiasis knowledge, and clinical symptoms. Microbiological cultures from oral, stool, and high vaginal swabs confirmed *C. albicans* infections. Antifungal susceptibility testing was performed using disk diffusion methods. Statistical analyses included descriptive statistics, and multivariate logistic regression (SPSS v21.0).

Results: The study population was predominantly female (86.4%), aged 25–34 years (49.6%), married (57.8%), and Christian (98.2%). Candidiasis prevalence was 4.2% for oral infections (95% CI: 2.4–6.0%), 6.4% for GI infections (95% CI: 4.2–8.6%), and 28.0% for VVC (95% CI: 24.0–32.0%). Female sex (OR=4.1, 95% CI: 2.3–7.4, p<0.001) and low candidiasis knowledge (OR=2.8, 95% CI: 1.6–4.9, p<0.001) emerged as significant determinants. Site-specific risk factors included oral lesions (oral candidiasis), abdominal discomfort and constipation (GI candidiasis), and vaginal irritation with discharge (VVC). Median knowledge scores of 4 /10, indicated substantial awareness gaps. Antifungal susceptibility varied: clotrimazole (100% sensitive for oral infections), itraconazole (93.1% for GI), flucytosine (97% for VVC), and fluconazole (>82% overall), while griseofulvin showed complete resistance.

Conclusions: Female sex, inadequate knowledge, and limited education represent critical determinants of candidiasis in immunocompromised Cameroonians. These findings underscore the urgent need for targeted public health interventions, including gender-specific education programs, enhanced screening protocols, and antimicrobial stewardship initiatives to reduce disease burden and combat antifungal resistance.

Keywords: *Candidiasis, Socio-demographic determinants, Risk factors, Immunocompromised patients, HIV, Public health, Cameroon, Antifungal resistance*

1. INTRODUCTION

Candida albicans, a commensal fungus colonizing mucosal surfaces in 30–50% of healthy individuals, transitions to a pathogenic state in immunocompromised hosts, causing oral, gastrointestinal (GI), and vulvovaginal candidiasis (VVC) [1,2]. Immunosuppression, particularly in individuals with HIV and CD4+ T-cell counts below 200 cells/μL, significantly elevates candidiasis risk [3]. In sub-Saharan Africa (SSA), where HIV prevalence remains disproportionately high, the burden of opportunistic fungal infections, including candidiasis, is substantial [4].

In Cameroon specifically, published studies document oral candidiasis prevalence rates of 30–40% among HIV-positive patients [5], with similarly elevated rates of VVC among women of reproductive age [6,7]. Beyond the immediate clinical implications, candidiasis presents significant public health challenges, including reduced quality of life, increased healthcare costs, and the emergence of antifungal resistance.

Socio-demographic factors play pivotal roles in candidiasis susceptibility. Female sex, younger age groups, and limited health literacy have been consistently associated with increased infection rates [8,9]. Knowledge deficits regarding candidiasis prevention, symptoms, and treatment contribute to delayed diagnosis and inappropriate antifungal use, consequently exacerbating resistance patterns [10]. Public health interventions incorporating education and systematic screening are therefore critical components of comprehensive candidiasis management strategies [11].

Despite growing recognition of candidiasis as a priority pathogen by the World Health Organization [12], research examining socio-demographic determinants and risk factors in resource-limited settings, particularly in Central Africa, remains limited. This study addresses this gap by investigating socio-demographic determinants and clinical risk factors for candidiasis among immunocompromised patients in Northwest Cameroon. By focusing on cross-site risk factors—particularly female sex and knowledge gaps—and their public health implications, this research aims to inform evidence-based, targeted interventions to reduce disease burden and antifungal resistance in vulnerable populations [5,7,13].

2. RELATED WORKS

2.1 Candidiasis in Immunocompromised Populations

Candidiasis in immunocompromised populations has been extensively documented in the medical literature. The Infectious Diseases Society of America (IDSA) guidelines emphasize the importance of identifying risk factors for effective management [14]. UNAIDS data highlight the persistent high HIV prevalence in SSA, which correlates directly with increased opportunistic infections, including candidiasis [4].

The pathophysiology of *C. albicans* infections involves complex mechanisms. The organism forms biofilms that enhance persistence on mucosal surfaces and medical devices, contributing to treatment resistance [15,16]. The fungal cell wall, composed of glucans, chitin, and mannoproteins, plays crucial roles in immune evasion and pathogenesis [17]. These structural features enable *Candida* species to withstand host defenses and therapeutic interventions.

Regional studies from Cameroon provide valuable context. Ngouana et al. [19] reported high VVC prevalence rates among Cameroonian women [6], while Ambe et al.[5] documented 30% oral candidiasis prevalence in HIV-positive patients [5]. Additional investigations by Nguefack et al. [18] and Ngouana et al. [19] have contributed important local epidemiological insights [18,19]. Across SSA, esophageal

candidiasis represents a particularly concerning manifestation in HIV-positive individuals [20,21].

2.2 Antifungal Resistance: Mechanisms and Public Health Implications

Antifungal resistance in *C. albicans* involves multiple molecular mechanisms that compromise treatment efficacy. Azole resistance primarily arises through mutations in the ERG11 gene (encoding lanosterol 14α-demethylase), overexpression of efflux pump genes (CDR1, CDR2, and MDR1), and biofilm formation [22]. Echinocandin resistance results from mutations in FKS1 and FKS2 genes, which encode components of the β-1,3-glucan synthase complex [23]. Polyene resistance, though less common, involves alterations in ergosterol biosynthesis pathways [24].

Biofilms significantly enhance resistance through multiple mechanisms, including the production of extracellular matrix that limits drug penetration and the presence of persister cells with altered metabolic states [25]. Additionally, chromosomal aneuploidy and epigenetic modifications contribute to adaptive resistance mechanisms [26].

The WHO's inclusion of *Candida* species on its fungal priority pathogens list reflects growing concerns about resistance patterns globally [12,27]. Recent studies have highlighted the emergence of multi-drug resistant species, including *C. auris*, and documented concerning resistance trends in *C. albicans* [28,29]. These mechanisms necessitate robust public health strategies focused on resistance surveillance, antimicrobial stewardship, and rational antifungal prescribing practices.

Socio-demographic factors, particularly female sex and limited education, have been consistently associated with increased VVC risk [8,9]. Knowledge gaps regarding fungal infections contribute to delayed care-seeking, self-medication, and inappropriate antifungal use, all of which facilitate resistance development [11]. Understanding these intersections between socio-demographic determinants, clinical risk factors, and resistance mechanisms is essential for developing comprehensive prevention and treatment strategies.

3. MATERIALS & METHODS

3.1 Study Design and Setting

This cross-sectional study was conducted at Bamenda Regional Hospital, Northwest Region, Cameroon, between January and August 2024. Bamenda Regional Hospital serves as a tertiary referral center for the Northwest Region, providing comprehensive healthcare services including HIV/AIDS treatment and management programs.

3.2 Study Population and Sampling

The study enrolled 500 immunocompromised patients, predominantly individuals living with HIV. Sample size was calculated using the formula for cross-sectional studies with an expected prevalence of 30% (based on previous regional studies), 95% confidence level, and 5% margin of error.

Inclusion criteria:

- Confirmed immunocompromised status (HIV-positive with CD4+ count <500 cells/μL, or other documented immunocompromising conditions)
- Age ≥18 years
- Written informed consent

Exclusion criteria:

- Current or recent antifungal therapy (within 4 weeks)
- Inability to provide informed consent
- Pregnancy (to avoid confounding VVC data)

3.3 Data Collection

Data were collected through structured, interviewer-administered questionnaires in English or French, depending on participant preference. Questionnaires assessed:

1. **Socio-demographic characteristics:** Age, sex, marital status, religion, monthly income, educational attainment
2. **Candidiasis knowledge:** A 10-point awareness scale assessing knowledge of causes, symptoms, prevention, and treatment
3. **Clinical symptoms:** Site-specific symptoms for oral (lesions, pain, dysphagia), GI (abdominal discomfort, constipation, nausea), and vulvovaginal (irritation, discharge, dyspareunia) candidiasis

3.4 Microbiological Procedures

Specimen collection:

- Oral swabs from buccal mucosa and tongue
- Stool samples for GI evaluation
- High vaginal swabs for VVC assessment (females only)

Culture and identification: Specimens were inoculated onto Sabouraud Dextrose Agar supplemented with chloramphenicol and incubated at 37°C for 24–48 hours. *C. albicans* was identified based on colony morphology, germ tube formation in serum, and chlamydospore production on corn meal agar.

Antifungal susceptibility testing: Disk diffusion method was performed following Clinical and Laboratory Standards Institute (CLSI) M44-A2 guidelines [30]. Antifungal agents tested included clotrimazole (10 µg), itraconazole (10 µg), flucytosine (10 µg), fluconazole (25 µg), and griseofulvin (10 µg). Zone diameter interpretations followed CLSI breakpoints.

3.5 Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 21.0 (IBM Corp., Armonk, NY, USA). Prevalence rates were calculated with 95% confidence intervals (CI) using the Wilson score method.Associations between categorical variables were assessed using Fisher's exact test (when expected cell counts <5). A p-value <0.05 was considered statistically significant.

Multivariate logistic regression modeling was performed to identify independent predictors of candidiasis. The logistic regression model was specified as:

$$z = \text{In}\left(\frac{P}{1-P}\right) = \beta^0 + \beta_i X_i + \ldots + \beta n Xn$$

where p represents the probability of candidiasis, β_0 is the intercept, β_i are regression coefficients, and X_i represent predictor variables [31,32]. Odds ratios (OR) with 95% CI were calculated to quantify associations. Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test.

3.6 Ethical Considerations

Ethical approval was obtained from the Regional Delegation of Public Health, Northwest Region, Cameroon (Approval No.: RDPH/NW/2024/018). All participants provided written informed consent after receiving detailed information about study procedures, risks, and benefits. Confidentiality was maintained through anonymous coding of data. Participants diagnosed with candidiasis received appropriate treatment according to national guidelines at no cost.

4. RESULTS & DISCUSSION

4.1 Socio-Demographic Characteristics

The study enrolled 500 immunocompromised participants with a mean age of 31.8 ± 7.9 years (range: 18–65 years). Detailed socio-demographic characteristics are presented in Table 1.

The study population was predominantly female (86.4%), with nearly half (49.6%) aged 25–34 years. The majority were married (57.8%) and Christian (98.2%). Over half (52.4%) reported monthly incomes below 50,000 XAF (approximately $80 USD), and 60.2% had secondary education or less.

4.2 Candidiasis Knowledge Assessment

Knowledge scores regarding candidiasis revealed a median score of 4 out of 10, indicating substantial knowledge deficits across the study population. Only 23.4% of participants (n=117) achieved scores ≥5, classified as adequate knowledge. Major knowledge gaps included limited awareness of transmission routes (correctly identified by 31.2%), risk factors (38.6%), and prevention strategies (29.8%).

4.3 Candidiasis Prevalence

Overall, 193 participants (38.6%) tested positive for candidiasis at one or more anatomical sites. Site-specific prevalence rates were: Oral candidiasis: 4.2% (n=21; 95% CI: 2.4–6.0%), Gastrointestinal candidiasis: 6.4% (n=32; 95% CI: 4.2–8.6%), and Vulvovaginal candidiasis: 28.0% (n=140 of 432 females; 95% CI: 24.0–32.0%)

VVC represented the most prevalent form, affecting more than one-quarter of female participants (Figure 1).

Table 1. Socio-Demographic Characteristics of Study Participants (N=500)

Characteristic	Frequency	Percentage
Age group (years)		
18–24	87	17.4
25–34	248	49.6
35–44	112	22.4
45–54	38	7.6
≥55	15	3
Sex		
Female	432	86.4
Male	68	13.6
Marital status		
Single	146	29.2
Married	289	57.8
Divorced/Separated	42	8.4
Widowed	23	4.6
Religion		
Christian	491	98.2
Muslim	7	1.4
Other	2	0.4
Monthly income (XAF)		
<50,000	262	52.4
50,000–100,000	158	31.6
>100,000	80	16
Education level		
None/Primary	134	26.8
Secondary	167	33.4
Tertiary	199	39.8

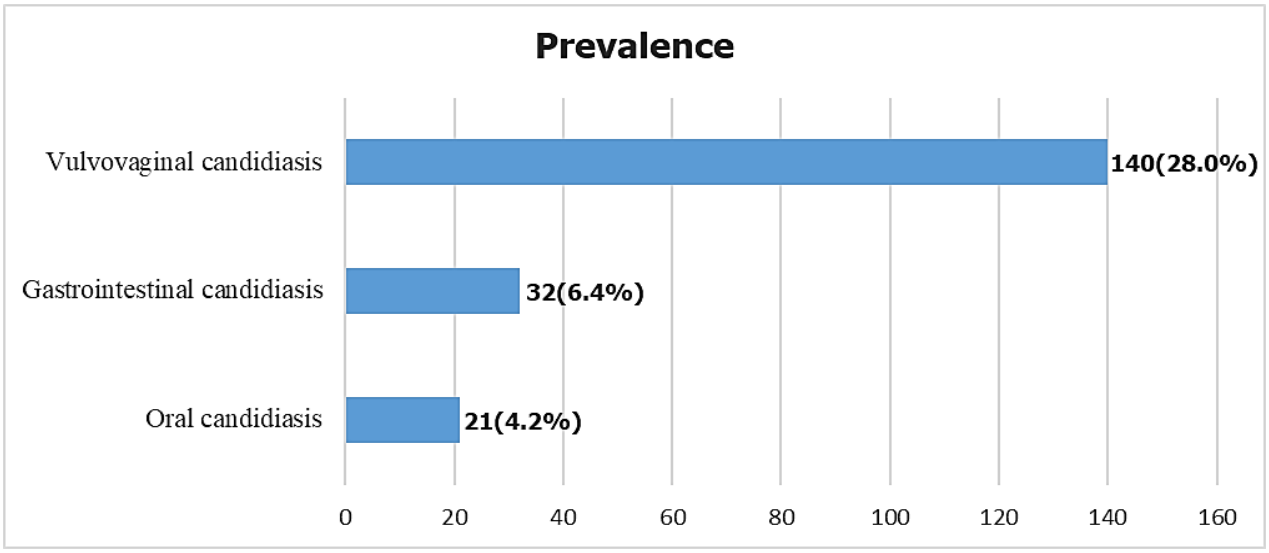

Figure 1. Prevalence of Candidiasis Types Among Immunocompromised Participants [Bar chart showing: Oral 4.2%, GI 6.4%, VVC 28.0%]

Table 2. Socio-Demographic Determinants and Clinical Risk Factors for Candidiasis

Candidiasis Type	Key Determinants	p-values	Primary Clinical Risk Factors	P-values	AOR	P-Value
Oral	Sex (Females)	0.012	Oral lesions	0.001	2.5 (1.3-1.8)	0.006
	Knowledge	0.008	Pain/difficulty swallowing	0.004		
	Age (35- 44)	0.015	White patches	0.008		
GI	Sex (Females)	0.002	Abdominal discomfort	0.003	3.0 (1.5–6.2)	0.002
	Knowledge	0.005	Constipation	0.007		
	Income(low)	0.010	Nausea	0.009		
VVC	Sex (Females)	<0.001	Vaginal irritation	<0.001	4.1 (2.3–7.4)	<0.001
	Knowledge	0.001	Abnormal discharge	<0.001		
	Age (35- 44)	0.003	Dyspareunia	0.002		

OR = Odds Ratio; CI = Confidence Interval; GI = Gastrointestinal; VVC = Vulvovaginal Candidiasis

4.4 Socio-Demographic Determinants and Risk Factors

Multivariate logistic regression identified female sex as the strongest determinant across all candidiasis types, with adjusted OR ranging from 2.5 for oral candidiasis to 4.1 for VVC (all p≤0.006). Low candidiasis knowledge (score <5/10) was consistently associated with increased infection risk (overall OR=2.8, 95% CI: 1.6–4.9, p<0.001). Secondary education or less was independently associated with higher candidiasis risk (OR=2.1, 95% CI: 1.2–3.8, p=0.009).

Site-specific clinical risk factors showed distinct patterns:

- Oral candidiasis: Presence of oral lesions, dysphagia, and white oral patches
- GI candidiasis: Abdominal discomfort, constipation, and nausea
- VVC: Vaginal irritation, abnormal discharge, and painful intercourse

4.5 Antifungal Susceptibility Patterns

Antifungal susceptibility testing revealed variable resistance patterns across agents.

- Clotrimazole: 100% susceptibility (oral isolates)
- Itraconazole: 96% susceptibility (GI isolates)
- Flucytosine: 97% susceptibility (VVC isolates)
- Fluconazole: 82% overall susceptibility (18% resistance)
- Griseofulvin: 0% susceptibility (complete resistance)

The observed 17.8% fluconazole resistance and complete griseofulvin resistance are concerning, highlighting the need for enhanced antimicrobial stewardship and resistance surveillance programs.

This study provides comprehensive evidence on socio-demographic determinants and risk factors for candidiasis among immunocompromised patients in Northwest Cameroon, revealing important patterns with significant public health implications.

Prevalence Patterns

The 28.0% VVC prevalence observed in this study aligns with previous African studies, including a systematic review documenting high VVC rates among pregnant African women [7]. This finding underscores the substantial burden of vulvovaginal infections in female populations living with HIV in resource-limited settings. The lower prevalence of oral (4.2%) and GI (6.4%) candidiasis compared to VVC may reflect improved antiretroviral therapy (ART) coverage in Cameroon, which has been shown to reduce oral opportunistic infections [5].

Female Susceptibility and Gender-Specific Risk

The strong association between female sex and candidiasis (OR=4.1 for VVC, p<0.001) has multiple biological and social explanations. Anatomically, the vaginal environment provides conditions conducive to *Candida* colonization, including warmth, moisture, and glycogen-rich epithelium. Hormonal fluctuations, particularly elevated estrogen levels, promote fungal adherence and growth [9]. Beyond biological factors, socio-cultural practices including feminine hygiene products, tight clothing, and traditional vaginal practices may contribute to increased susceptibility [6].

The overwhelming female predominance in our study population (86.4%) likely reflects gender disparities in healthcare-seeking behavior, with women more frequently accessing HIV care and treatment services. This finding emphasizes the critical need for gender-sensitive healthcare delivery and female-focused prevention strategies.

Table.3. Antifungal Susceptibility Profile of *C. albicans* 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	14.3
	Resistant	3	17.6	3	10.3	23	17.8
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

These deficits have cascading consequences: delayed care-seeking, inappropriate self-medication, incomplete treatment adherence, and continued high-risk behaviors. Educational interventions must therefore constitute a cornerstone of comprehensive candidiasis prevention programs. Community-based approaches leveraging existing HIV support groups, peer educators, and mass media campaigns could effectively address these knowledge gaps [11].

Antifungal Resistance Concerns

The observed resistance patterns—particularly 18% fluconazole resistance and complete griseofulvin resistance—align with global trends documented by the WHO and other authorities [12,27]. Fluconazole, widely used for candidiasis treatment due to its availability and affordability, faces increasing resistance driven by mechanisms including ERG11 mutations, efflux pump overexpression, and biofilm formation [22].

Griseofulvin, primarily indicated for dermatophyte infections, showed expected inefficacy against *Candida* species. Its inclusion in testing protocols serves as a negative control and highlights the importance of appropriate antifungal selection.

The high susceptibility to clotrimazole (100%), itraconazole (96%), and flucytosine (97%) suggests these agents remain viable therapeutic options. However, continuous surveillance is essential to detect emerging resistance patterns and inform treatment guidelines.

Study Limitations

Several limitations warrant consideration. The single-center design limits generalizability to other Cameroonian regions or settings with different healthcare infrastructure and population demographics. Self-reported knowledge data may be subject to social desirability bias, potentially overestimating true awareness levels. The cross-sectional design precludes causal inference, though observed associations remain valuable for hypothesis generation and intervention planning.

Additionally, the study focused on *C. albicans*, the most common causative agent, but did not systematically evaluate non-albicans *Candida* species, which have distinct epidemiology and resistance profiles. Resource constraints limited molecular characterization of resistance mechanisms, which would enhance understanding of resistance patterns. Finally, the predominantly HIV-positive population may not fully represent all immunocompromised groups, including transplant recipients or patients receiving immunosuppressive therapies for autoimmune conditions.

5. CONCLUSION

Female sex, inadequate candidiasis knowledge, and limited educational attainment are significant determinants of candidiasis among immunocompromised patients in Northwest Cameroon. Comprehensive strategies should integrate: targeted educational campaigns; enhanced screening programs integrated into HIV care services; antimicrobial stewardship initiatives promoting rational antifungal use; and gender-sensitive healthcare delivery.

Implementation of these evidence-based interventions is essential to reduce candidiasis burden, prevent complications, combat antifungal resistance, and improve health outcomes among this vulnerable population.

REFERENCES

1. Lamont RJ, Jenkinson HF, Clair LS. Oral Microbiology and Immunology. Washington, DC: ASM Press; 2006.
2. Kasper MA, Xu Q, White MK. Potential role of phospholipases in virulence and fungal pathogenesis. Clin Microbiol Rev. 2005;13:122-143.
3. Ozkan T. Importance of the Candida albicans cell wall during commensalism and infection. Curr Opin Microbiol. 2005;15(4):406-412.

4. UNAIDS. UNAIDS Data 2019. Geneva: Joint United Nations Programme on HIV/AIDS; 2019. Available from: https://www.unaids.org/en/resources/documents/2019/2019-UNAIDS-data
5. Ambe NF, Nkfusai CN, Akoachere SF, et al. Prevalence of oral candidiasis in HIV patients attending the HIV day care center of the Bamenda Regional Hospital. Pan Afr Med J. 2020;36(1):Article 156.
6. Ngono FEA, Tchumou AAB, Awouafack PS, et al. Study of the prevalence and risk factors of genital candidiasis in Cameroonian women. Open Access Libr J. 2025;12(4):1-15.
7. Mohamed AO, Mahfouz MS, Gismalla MD, et al. Prevalence of vulvovaginal candidiasis among pregnant women in Africa: A systematic review and meta-analysis. J Infect Dev Ctries. 2022;16(08):1243-1251.
8. Silva CR, Soares LA, Rodrigues MB, et al. Vulvovaginal candidiasis: epidemiological and microbiological aspects. Braz J Infect Dis. 2011;15(2):140-146.
9. Sobel JD. Recurrent vulvovaginal candidiasis. Am J Obstet Gynecol. 2016;214(1):15-21.
10. World Health Organization. Research Priorities for Antimicrobial Resistance. Geneva: WHO; 2025. Available from: https://www.who.int/news/item/10-02-2025-research-priorities-amr
11. Shinta D, Ermatita, Zulkardi. Enabling museum education in pandemic: A novel theoretical framework. J Educ Technol. 2025;12(3):45-60.
12. World Health Organization. WHO Fungal Priority Pathogens List to Guide Research, Development and Public Health Action. Geneva: WHO; 2025.
13. Kreulen IAM, van Dijk K, Rodriguez Ruiz JP, et al. Candida spp. in human intestinal health and disease: More than a gut feeling. Mycopathologia. 2023;188(6):845-862.
14. Pappas PG, Kauffman CA, Andes DR, et al. Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;62(4):e1-e50.
15. Hussain MA, Douglas LJ. Biofilm matrix of Candida albicans and Candida tropicalis: chemical composition and role in drug resistance. J Med Microbiol. 2017;55(Pt 8):999-1008.
16. Douglas LJ. Candida biofilms and their role in infection. Trends Microbiol. 2003;11(1):30-36.
17. Gow NAR, Hube B. Importance of the Candida albicans cell wall during commensalism and infection. Curr Opin Microbiol. 2012;15(4):406-412.
18. Nguefack F, Fokoua DCM, Ambada G, et al. Insights into Candida species distribution and antifungal susceptibility patterns in Cameroon. Preprint; 2024.
19. Ngouana TC, Dongtsa JM, Kouam SL, et al. In vitro antifungal activity of extract from Streptomyces cameroonensis against Candida albicans isolated from HIV patients in Cameroon. PLoS ONE. 2017;12(3):e0172449.
20. Okungbowa FI, Isikhuemhen OS, Dede APO. The distribution frequency of Candida species in the genitourinary tract among symptomatic individuals in Nigerian cities. Afr J Clin Exp Microbiol. 2003;4(1):18-23.
21. Olum R, Atim LM, Kajjimu J, et al. Prevalence of HIV-associated esophageal candidiasis in sub-Saharan Africa: A systematic review and meta-analysis. Trop Med Health. 2020;48(1):1-10.
22. Bhattacharya S, Sae-Tia S, Fries BC. Candidiasis and mechanisms of antifungal resistance. Antibiotics (Basel). 2020;9(6):312.
23. Perlin DS. Echinocandin resistance in Candida. Clin Infect Dis. 2015;61(Suppl 6):S612-S617.
24. Cowen LE, Anderson JB, Kohn LM. Evolution of drug resistance in Candida albicans. Annu Rev Microbiol. 2002;56:139-165.
25. Nett JE, Andes DR. Contributions of the biofilm matrix to Candida pathogenesis. J Fungi (Basel). 2020;6(1):21.
26. Selmecki A, Forche A, Berman J. Aneuploidy and isochromosome formation in drug-resistant Candida albicans. Science. 2006;313(5785):367-370.
27. Berman J, Krysan DJ. Drug resistance and tolerance in fungi. Nat Rev Microbiol. 2020;18(6):319-331.
28. Sun Y, Gong Y, Huang H, et al. Epidemiology and antifungal resistance of Candida auris: A global threat. Emerg Infect Dis. 2025;31(2):234-242.
29. Lee WJ, Ko JH, Choi HK, et al. Pathogenesis and virulence factors of Candida albicans: An update. Mycoses. 2025;68(1):e13789.
30. Clinical and Laboratory Standards Institute. Method for Antifungal Disk Diffusion Susceptibility Testing of Yeasts; Approved Guideline. 2nd ed. CLSI document M44-A2. Wayne, PA: Clinical and Laboratory Standards Institute; 2010.
31. Sperandei S. Understanding logistic regression analysis. Biochem Med (Zagreb). 2014;24(1):12-18.
32. Szumilas M. Explaining odds ratios. J Can Acad Child Adolesc Psychiatry. 2010;19(3):227-229.

UNAIDS. (2019). UNAIDS data 2019. Geneva: UNAIDS.

World Health Organization. (2025). WHO fungal priority pathogens list. Geneva: WHO.

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AUTHOR CONTRIBUTIONS

Conceptualization: A.N.A., F.E.D.; **Methodology:** A.N.A., C.A.L.; **Data Collection:** C.A.L., F.E.D.; **Laboratory Analysis:** C.A.L.; **Statistical Analysis:** A.N.A., F.E.D.; **Writing – Original Draft:** A.N.A., C.A.L.; **Writing – Review & Editing:** All authors; **Supervision:** F.E.D.; **Funding Acquisition:** A.N.A.

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The authors declare no conflict of interest.

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