



# Diversity of *Candida* sp and Antifungal Susceptibility Patterns in Digestive Candidiasis among People Living with HIV in CHU of Libreville, Gabon

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## Research Article

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## Abstract

**Background:** Intestinal candidiasis is a common opportunistic condition in people living with HIV (PLHIV). Limited data are available in Africa on prevalence, species diversity, and antifungal susceptibility of intestinal *Candida*, particularly in asymptomatic or mildly symptomatic patients. This study assessed prevalence, species distribution, and resistance patterns of *Candida* sp isolated from stools of HIV-infected individuals in Libreville, Gabon.

**Methods:** A descriptive cross-sectional study was conducted from October to November 2021 at the University Hospital of Libreville and the Department of Parasitology, Mycology and Tropical Medicine. Adult PLHIV ( $\geq 18$  years), without recent antifungal therapy or other predisposing chronic diseases, were enrolled. Stool samples were cultured on chromogenic medium; species were identified by colony colour. Antifungal susceptibility (fluconazole, ketoconazole, clotrimazole, nystatin, miconazole, amphotericin B) was assessed by disc diffusion.

**Results:** Of 107 participants (median age 42 [35–49] years, sex ratio 2.3), median CD4 count was 185 [100–348] cells/mm<sup>3</sup>. *Candida* sp was isolated in 68.2% of samples. *C. albicans* (32.7%) and *C. glabrata* (31.8%) predominated; *C. krusei* accounted for 7.5%, while *C. tropicalis* was absent. Among 61 tested isolates, 19.7% were susceptible to all agents, 24.6% resistant to all, and 50.8% showed multidrug resistance. Resistance was highest to miconazole (75.4%) and fluconazole (70.5%).

**Conclusion:** Intestinal *Candida* colonisation is frequent in PLHIV in Libreville, with high prevalence of non-*albicans* species and substantial azole resistance, underscoring the need for enhanced antifungal resistance monitoring and adapted treatment strategies.

**Keywords:** Vulvovaginal Candidiasis; Suburbs; Senegal

## Introduction

Digestive candidiasis refers to fungal infections caused by colonisation and invasion of the gastrointestinal tract by yeasts of the genus *Candida*. These yeasts are usually commensal fungi of the digestive tract but can become opportunistic pathogens in situations of immunosuppression [1-4]. PLHIV are particularly at risk because of the profound decline in CD4 cell counts, making candidiasis one of the leading aetiologies of opportunistic fungal infections and an AIDS-defining condition [5-8].

The prevalence of oropharyngeal and/or intestinal candidiasis varies across countries and largely depends on patients' access to antiretroviral therapy (ART) [6]. Nevertheless, oropharyngeal candidiasis is generally more common in PLHIV compared with intestinal forms. Gastrointestinal T lymphocytes are a primary target of HIV infection, leading to increased permeability of the intestinal mucosa where these cells normally reside [4,6-8]. The reduction of gastrointestinal CD4+ T cells has been associated with dysbiosis, which may weaken the antagonistic effect of intestinal microbiota bacteria. Combined with immunosuppression, dysbiosis can promote fungal infections [8,9]. Interestingly, in some countries such as Cameroon, intestinal candidiasis has been reported to be more prevalent than oropharyngeal candidiasis [10].

Although *Candida albicans* remains the most frequently isolated species in digestive colonisations, recent studies highlight a shift in this profile, with an increasing proportion of non-*albicans* *Candida* species, particularly in cases of prolonged immunosuppression [6,11-13]. Frequent antifungal exposure among immunocompromised patients is thought to promote the emergence of non-*albicans* *Candida* species, which are often more resistant to azoles [6,14].

In Gabon, oropharyngeal candidiasis is highly prevalent among PLHIV and often represents one of the first clinical manifestations leading to HIV diagnosis [14]. However, available data on candidiasis in the context of AIDS are outdated (over 10 years old). Furthermore, very limited information is available on intestinal candidiasis in PLHIV, and no data exist regarding the resistance of *Candida* sp to azole antifungals, despite the widespread use of fluconazole prophylaxis.

This study was therefore designed to determine the prevalence of intestinal colonisation by *Candida* sp, describe the fungal diversity, and assess the antifungal susceptibility profiles of *Candida* sp isolated from stool samples of PLHIV in Libreville, Gabon.

## Methods

### Study Design and Population

This was a descriptive cross-sectional study with prospective data collection conducted from October to November 2021 in the Infectious Diseases Ward of the Centre Hospitalier Universitaire de Libreville (IDW-CHUL) and the Department of Parasitology-Mycology and Tropical Medicine of the Université des Sciences de la Santé (USS). The study was nested within a prevalence survey of opportunistic infections among PLHIV. Participants included in the study, who agreed to provide a stool sample, met the following inclusion criteria: age  $\geq 18$  years, no antifungal therapy within the previous three months, provision of oral informed consent, and absence of any other chronic condition known to predispose to candidiasis.

### Study Procedures

Participants completed a questionnaire, and additional clinical information was retrieved from their medical records when missing. Collected variables included age, sex, date of HIV diagnosis, use of antiretroviral therapy (ART), CD4 cell count (when available), and WHO clinical stage of HIV infection. After the interview, each participant received a sterile stool container and instructions on proper sample collection to avoid contamination with urine. Samples were returned immediately after collection to the study staff.

### Laboratory Diagnosis

Each stool specimen was cultured on a selective and differential chromogenic medium (Chromatic™ *Candida*, LIOFILCHEM) for species identification. Plates were incubated at 37 °C for 18-24 hours. Only specimens yielding more than 10 colonies were considered positive. Colony colours were interpreted according to the manufacturer's recommendations: *C. albicans* (green), *C. glabrata* (beige), *C. krusei* (pink with pale edges), *C. tropicalis* (blue), and *C. parapsilosis* (pale pink-white) [5,6,15]. A 0.5 McFarland inoculum was prepared from a suspension of five colonies in 5 mL of physiological saline. The suspension was streaked on Mueller-Hinton (MH) agar using a sterile swab [11-13, 16-18].

### Antifungal Susceptibility Testing

Antifungal susceptibility was assessed using the disc diffusion method according to the manufacturer's instructions, with a 0.5 McFarland inoculum for each isolate. Quality control was ensured using *C. albicans* ATCC 90028 and *C. tropicalis* ATCC 750 strains for both

identification and susceptibility testing. The antifungal discs tested were fluconazole (25µg), ketoconazole (15µg), clotrimazole (10µg), nystatin (10µg), miconazole (10µg), and amphotericin B (10µg). Discs were placed on MH agar inoculated with *Candida* isolates and incubated at 37 °C for 24 hours [12,13,16,17]. For data analysis, isolates with intermediate susceptibility were considered resistant to the antifungal agent tested. Multidrug resistance was defined as resistance to at least three antifungal agents.

### Statistical Analysis

Data were entered into Microsoft Excel (Microsoft Corporation, Seattle, USA) and analysed using Statview software V5.0. Statistical significance was considered with  $p < 0.05$ . Qualitative variables were compared using Chi-square or Fisher's exact test as appropriate.

### Ethical Considerations

The study protocol was approved by the National Ethics Committee for Research (Protocol N° 0027/2022/CNER/SG/P). Research authorisations were also obtained from

CHUL authorities. Oral informed consent was obtained from each participant (or guardian when applicable) after study explanations. Confidentiality was maintained by anonymising participants with identification numbers. All study participants received medical care at the IDW-CHUL.

## Results

### Epidemiological Characteristics of the Study Population

A total of 107 PLHIV provided stool samples for analysis. The median age was 42 [35-49] years with a sex ratio of 2.3 (Table 1). More than three-quarters of participants were single, and 47.6% (n=51/107) had been diagnosed with HIV for 2-10 years. Only 13.1% (n=14/107) were not receiving ART. Among those with recent CD4 counts available, more than half were immunosuppressed, with a median CD4 count of 185 [100-348] cells/mm<sup>3</sup> (Table 1). The WHO clinical stage was reported for 106 participants; 41.5% (n=44/106) were in an advanced stage of disease. The mean interval between HIV diagnosis and inclusion in the study was 7.3 (±5.4) years.

Variables	N	%
<b>Age group</b>		
< 55 years	94	87.9
≥ 55 years	13	12.1
<b>Gender</b>		
Male	32	29.9
Female	75	70.1
<b>Occupation</b>		
Manager	29	27.1
Worker	45	42
Unemployed	33	30.9
<b>Matrimonial status</b>		
Single	84	78.5
Married/widowed	23	21.5
Alcohol consumption	41	38.3
<b>Time since HIV diagnosis</b>		
1 year	22	20.5
2-5 years	27	25.2
6-10 years	24	22.4
> 10 years	34	31.8
ART	93	86.9
CD4 count measurement	96	89.7

<b>CD4 group (cell/mm3)</b>		
≤ 200	60	62,5
201-499	23	23,9
> 499	13	13,5
Antifungal therapy	42	39,3
<b>WHO Stage</b>		
I	52	48,6
II	10	9,3
III	42	39,3
IV	2	2,8
<b>Gastrointestinal symptoms</b>		
Diarrhoea	15	14,0
No diarrhoea	92	86,0

**Table 1:** General Characteristics of Study Population.

### Frequency of Intestinal Colonisation by *Candida* sp

*Candida* sp was isolated from 73 stool samples, representing a colonisation prevalence of 68.2%. Women

(79.7%) were significantly more frequently colonised than men ( $p=0.04$ ) (Table 2). No significant association was found between *Candida* colonisation and age, ART status, CD4 count, WHO stage, or the presence of diarrhoea (Table 2).

	<b>n</b>	<b>%</b>	<b>p</b>
<b>Age group</b>			0.93
< 55 years	64	68.1	
≥ 55 years	9	69.2	
<b>Sex</b>			0.04
Male	17	53.1	
Female	56	74.7	
<b>ART</b>			0.73
Yes	64	68.8	
No	9	64.3	
<b>CD4 group (cell/mm3)</b>			0.99
[0-200]	41	68.3	
[201-499]	16	69.6	
>499	9	69.3	
<b>OMS Stage</b>			0.3
I	38	73.1	
II	8	80	
III	26	61.9	
IV	1	50	

**Table 2:** Prevalence of Intestinal Candidiasis According to the Characteristics of the Study Population.



## Discussion

There are limited African data on intestinal colonisation by *Candida* spp in PLHIV, even though these yeasts are integral components of the intestinal mycobiome, which plays a crucial role in maintaining microbiota balance, metabolic stability, and host immune responses [3]. Within the gut mycobiome, *Candida albicans* has been associated with modulation of local mucosal immune responses, as well as innate and adaptive immunity [3]. Moreover, the onset of candidemia in immunocompromised individuals is often preceded by heavy intestinal colonisation with *C. albicans* detected in stool [3]. Alterations of the intestinal microbiota, including an overgrowth of *Candida* sp, have been implicated in progression to AIDS [1,19]. If the suggested links between intestinal colonisation, mycobiome alterations, AIDS progression, and the risk of invasive candidiasis are confirmed, assessing the diversity and density of intestinal fungal populations could serve as an indicator for invasive candidiasis or disease progression.

The prevalence of intestinal *Candida* colonisation was 68.2%, with more than three-quarters of cases being asymptomatic. This rate is considerably higher than those reported in India (9.7%) and Iran (0.05%) [15,20]. Only few studies have specifically assessed intestinal colonisation in PLHIV; most investigations of digestive candidiasis in this population have focused on oropharyngeal and oesophageal infections, which are recognised as major opportunistic infections. Nevertheless, intestinal colonisation rates comparable to ours have been reported in Ghana (75.3%), Chad (34%), Ethiopia (37.5%), and Cameroon (42%) [8,21-23]. The high prevalence observed in our study may be explained by the high proportion (>60%) of immunocompromised patients, since gastrointestinal CD4+ T lymphocytes are preferential HIV targets. Their depletion leads to mucosal barrier damage, disruption of the gut microbiota, and enhanced susceptibility to colonisation [24].

*Candida* colonisation was significantly more frequent in women (74.7%) than in men. This finding could reflect the greater vulnerability of women to HIV infection as well as their higher rates of healthcare attendance. Colonisation was not significantly associated with age, ART exposure, CD4 count, or WHO stage. The relatively small sample size and the predominance of older participants (>55 years), most of whom were on ART and had CD4 counts <500 cells/mm<sup>3</sup>, may have limited the statistical power to detect such associations. Previous studies have consistently shown a relationship between CD4 cell count and candidiasis, particularly oropharyngeal forms [7,8,19,25].

Esebelahie NO, et al. [5] reported that CD4 counts <200 cells/ $\mu$ L in ART-naïve patients were predictive of candidiasis.

ART-mediated immune reconstitution, through CD4 recovery, is known to reduce the incidence of candidiasis [1]. In our study, intestinal colonisation was comparable between ART users and non-users, which could be partly explained by poor adherence to ART a factor not directly assessed here. Barriers such as financial constraints, lack of psychosocial support, limited awareness of the need for regular follow-up, adverse drug effects, stigma, and asymptomatic disease progression are known to affect ART adherence and thus favour opportunistic infections [14,26].

The predominance of non-*albicans* *Candida* species is another important finding. *C. glabrata* (47.9%) was more frequent than *C. albicans* (43.7%). *C. glabrata* has been associated with more severe and treatment-refractory infections compared to *C. albicans* [27]. While *C. albicans* remains the most common cause of candidiasis, increasing rates of non-*albicans* species in digestive infections among PLHIV and other immunocompromised populations have been increasingly documented [6,10,15]. Selective antifungal pressure, particularly from fluconazole widely used for both treatment and prophylaxis of cryptococcal meningitis may contribute to the emergence of non-*albicans* species [28]. This selective pressure is especially relevant for *C. krusei* and *C. glabrata*, which are intrinsically resistant or dose-dependent susceptible to fluconazole, respectively [29,30]. Co-infections, also observed in our cohort, may further enhance azole resistance [31].

More than 80% of isolates were resistant to at least one antifungal, with resistance being more common to azoles than to polyenes. The highest resistance rates were observed to miconazole (75.2%), fluconazole (70.5%), and ketoconazole (41.0%). Comparable findings have been reported in Uganda, where resistance to fluconazole (77.1%) and amphotericin B (94.1%) was observed in PLHIV [32]. Conversely, an Ethiopian study reported fluconazole susceptibility in 85.6% of isolates [30]. High resistance rates to azoles may be attributed to their frequent use as therapeutic alternatives to amphotericin B [33]. Fluconazole, is widely used in Gabon due to its affordability, availability, oral formulation, and lower toxicity. High resistance to miconazole has also been reported in Brazil (65.6%) [34]. Cross-resistance among azoles, due to shared mechanisms of action, likely explains the high rates of resistance observed in our setting, even though miconazole use is limited. Amphotericin B is the second most commonly used antifungal in hospital settings, which may account for the higher resistance rates observed compared to literature reports, where susceptibility often exceeds 95% [8,34]. By contrast, most isolates remained susceptible to nystatin, which is rarely used in clinical practice. This suggests that nystatin could be considered as an alternative in settings where azole efficacy continues to decline.

Species-specific resistance patterns were also evident. All five *C. krusei* isolates were resistant to at least two antifungals, including fluconazole and miconazole, reflecting their intrinsic resistance to azoles and reduced susceptibility to other antifungals, including amphotericin B [35]. Prolonged fluconazole use may also explain the progressive replacement of *C. albicans* by resistant non-*albicans* species. Among *C. glabrata* isolates, all strains resistant to miconazole also exhibited multidrug resistance involving fluconazole; nearly 50% (n=9/19) were resistant to at least five antifungals, compared with less than 30% (n=7/25) of *C. albicans* isolates. Profiles of multidrug resistance involving 4-6 antifungals have rarely been reported in sub-Saharan Africa [36]. Lackner M, et al. [37] highlighted that pan-azole and amphotericin B resistance, though uncommon, is not rare [37]. Such resistance is linked to mutations in *ERG2*, *ERG3*, and *ERG5* genes, particularly in *C. glabrata* and *C. parapsilosis*. The first case of pan-azole and polyene resistance was reported by Sida H, et al. [7].

This study has some limitations. First, it was a monocentric study, conducted among PLHIV with advanced disease, which limits the generalisability of findings to other populations. Second, budgetary constraints precluded the use of more advanced identification methods such as MALDI-TOF, PCR, or sequencing. Nonetheless, our study provides valuable preliminary insights into the diversity and resistance profile of intestinal *Candida* sp in PLHIV in Gabon, a field with limited data both locally and globally. Further studies including molecular characterisation of isolates, larger and more diverse samples (including asymptomatic PLHIV, HIV-negative individuals, children, and pregnant women) are needed.

## Conclusion

Intestinal colonisation by *Candida* sp is highly prevalent among PLHIV in Gabon, with an increasing emergence of non-*albicans* species. Most isolates were resistant to fluconazole, the most widely used antifungal agent in both outpatient and inpatient settings. These findings underscore the urgent need to expand epidemiological and resistance surveillance of candidiasis in PLHIV and to update therapeutic guidelines for the management of these infections.

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