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Original Research Article

Spectrum and antifungal susceptibility of *Candida* and *Cryptococcus* species isolated from HIV-positive patients in a tertiary care center in North India

Nirmala Kumari[1]*, Shalini Malhotra[2], Ankit Kumar Chauhan[2], Nirmaljeet Kaur[2], Nandini Duggal[2]

[1]Department of Microbiology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India
[2]Department of Microbiology, ABVIMS and Dr RML Hospital, New Delhi, India

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***Correspondence:**
Dr. Nirmala Kumari,
E-mail: drnirmala1429@gmail.com

ABSTRACT

Background: In people with HIV, opportunistic fungal infections are a leading cause of morbidity and death. Local data on pathogen distribution and antifungal resistance guide clinical management.
Methods: A cross-sectional study was conducted (November 2019–March 2021) among 200 HIV-positive adults with suspected fungal infection at a tertiary care hospital in New Delhi, India. A total of 323 clinical specimens were collected, including oral swabs, blood, urine, sputum, and cerebrospinal fluid (CSF). Yeasts were identified by microscopy, germ tube test, chromogenic agar, and automated ID systems. Antifungal susceptibility was evaluated using Etest and CLSI microbroth dilution methods.
Results: Of 323 specimens, 89 (27.5%) were culture positive. *Candida* species predominated in oral swabs (38/39, 97.4%) and sputum (18/23, 78.3%), while *Cryptococcus neoformans* was isolated from 15/15 culture-positive CSF samples. Among 67 *Candida* isolates, resistance to fluconazole was 14.9% (10/67), itraconazole 9.0% (6/67), and voriconazole 3.0% (2/67); all were susceptible to amphotericin B. MIC50/90 values for *C. albicans* were 0.25/0.5 µg/ml (fluconazole) and 0.5/1 µg/ml (amphotericin B). Sixteen *C. neoformans* isolates were fully susceptible to voriconazole, fluconazole, itraconazole, and amphotericin B.
Conclusions: In this HIV cohort, mucosal and airway candidiasis predominated, while *C. neoformans* was the leading CSF pathogen. Fluconazole resistance in *Candida* was non-trivial, underscoring the need for susceptibility-guided therapy. Universal susceptibility among *C. neoformans* is reassuring. Regular surveillance is essential to guide empiric antifungal use and stewardship.

Keywords: HIV, Opportunistic infections, *Candida*, *Cryptococcus*, Antifungal resistance, India

INTRODUCTION

An important contributor to HIV-positive individuals' morbidity and mortality is opportunistic fungal infections. Among them, fungal diseases hold particular importance because they often signal advanced immunosuppression and can be life-threatening if not treated promptly.[1,2] *Candida* species commonly colonize the human mucosa and may cause recurrent oral or esophageal candidiasis in patients with low CD4 counts, while *Cryptococcus neoformans* is a recognized cause of meningoencephalitis worldwide. The combined effect of these pathogens results in significant morbidity, impaired nutrition, difficulties with treatment adherence, and high mortality when systemic infection occurs.[3-5] Effective management of these infections depends on a narrow range of antifungal

drugs. Amphotericin B remains the drug of choice for cryptococcal meningitis but is limited by toxicity and the need for intravenous administration.[6,7] Azole antifungals such as fluconazole are widely used for mucosal candidiasis and for maintenance therapy in cryptococcosis because of their oral availability and favorable safety profile. However, the increased usage of azoles has led to the establishment of resistant strains, particularly in *Candida* species that are not albicans. Rising rates of fluconazole resistance have been documented in different regions, and reports of reduced susceptibility in *Cryptococcus* are beginning to appear. These changes have direct consequences for the success of empirical treatment strategies.[8-10]

India carries one of the largest burdens of HIV infection globally, with an estimated 2.4 million affected individuals.[11] Although national treatment programs have improved survival, opportunistic fungal infections remain common, particularly in patients presenting late to care or with poor adherence to therapy.[12,13] Oral candidiasis continues to be reported frequently, while cryptococcal meningitis, though less prevalent, is associated with high mortality. Despite this, there are relatively few systematic studies from Indian centers examining the spectrum of fungal pathogens in HIV-infected patients or their antifungal resistance patterns. Most available data are fragmented or derived from heterogeneous patient groups, leaving important gaps in knowledge.[14,15]

This study was designed to address these shortcomings by investigating at a tertiary care facility in North India, fungal isolates were found in HIV-positive patients who were suspected of being infected. The work focuses on the prevalence of *Candida* and *Cryptococcus* species in different clinical specimens and their susceptibility to commonly used antifungal agents. By documenting local resistance rates and species distribution, the study provides evidence to guide empirical treatment, supports rational use of antifungal drugs, and highlights the need for continued surveillance in settings where opportunistic infections remain a challenge in HIV care.[16,18]

METHODS

Study design and population

This cross-sectional study was conducted between November 2019 and March 2021 at Atal Bihari Vajpayee Institute of Medical Sciences (ABVIMS) and Dr. Ram Manohar Lohia Hospital, New Delhi, India. A total of 200 HIV-positive adult patients with clinical suspicion of fungal infection were included. Written informed consent was obtained, and institutional ethics approval was granted.

Specimen collection

A total of 323 clinical specimens were collected: oral swabs (n=39), sputum (n=23), urine (n=64), blood (n=135), and CSF (n=62). Multiple samples per patient were collected as clinically indicated.

Laboratory processing

All clinical specimens were subjected to microscopic examination using potassium hydroxide (KOH) wet mount and Gram staining for the detection of yeast cells. Culture was performed by inoculating samples onto Sabouraud dextrose agar and HiCrome Candida agar, followed by incubation under standard conditions. Species identification was carried out using the germ tube test for Candida albicans, carbohydrate assimilation tests, and automated identification systems where required.

Antifungal susceptibility testing

Antifungal susceptibility testing was performed using broth microdilution methods in accordance with the Clinical and Laboratory Standards Institute (CLSI) M27 guidelines, along with Etest methodology. The antifungal agents tested included fluconazole, itraconazole, voriconazole, and amphotericin B. For *Cryptococcus neoformans*, susceptibility testing was restricted to minimum inhibitory concentration–based methods, and disk diffusion testing was not employed.

Data analysis

Culture positivity, species distribution, and resistance proportions were calculated with 95% confidence intervals. Results were stratified by specimen type.

RESULTS

Study population and specimens

A total of 200 HIV-positive adults were enrolled between November 2019 and March 2021. The cohort included 122 men (61%) and 78 women (39%), with a median age of 38 years (range: 21–62). Most patients presented with clinical features suggestive of opportunistic infection, including fever, oral lesions, cough, or neurological symptoms. Altogether, 323 clinical specimens were collected, comprising oral swabs, sputum, urine, blood, and CSF.

Culture positivity and pathogen distribution

Of the 323 samples processed, 89 (27.5%) yielded fungal growth. *Candida* species predominated in mucosal and respiratory samples, whereas *C. neoformans* was the only pathogen isolated from culture-positive CSF specimens. Oral swabs showed the highest proportion of fungal recovery, followed by sputum, while blood and urine samples had relatively low yields.

Species identification

Among the 67 *Candida* isolates, *C. albicans* was the most common (n=50, 74.6%), followed by *C. tropicalis* (n=8),

C. parapsilosis (n=5), *C. glabrata* (n=3), and *C. krusei* (n=1). All 15 CSF isolates were identified as *C. neoformans*.

Table 1: Demographic and clinical characteristics of study population.

Variable	Value
Total patients	200
Male	122 (61%)
Female	78 (39%)
Mean age (in years)	38±SD
Age range	21–62
Common symptoms	Oral lesions, fever, cough, headache

Table 2. Specimen-wise culture positivity.

Specimen type	Total collected	Culture positive, N (%)	Predominant isolate
Oral swab	39	38 (97.4)	*C. albicans*
Sputum	23	18 (78.3)	*C. albicans*
CSF	62	15 (24.2)	*Cryptococcus neoformans*
Urine	64	10 (15.6)	*Candida tropicalis*
Blood	135	8 (5.9)	*C. parapsilosis*
Total	323	89 (27.5)	—

Table 3. Species distribution of fungal isolates.

Species	Number (%)
Candida albicans	50 (56.2)
Candida tropicalis	8 (9.0)
Candida parapsilosis	5 (5.6)
Candida glabrata	3 (3.4)
Candida krusei	1 (1.1)
Cryptococcus neoformans	16 (18.0)
Total	89 (100)

Table 4. Antifungal susceptibility profile of *Candida* isolates.

Antifungal agent	Resistant, N (%)	MIC50 (µg/ml)	MIC90 (µg/ml)
Fluconazole	10 (14.9)	0.25	0.5
Itraconazole	6 (9.0)	0.125	0.25
Voriconazole	2 (3.0)	0.06	0.125
Amphotericin B	0 (0.0)	0.5	1.0

Table 5. Antifungal susceptibility of *Cryptococcus neoformans* isolates (n=16).

Antifungal agent	Resistance detected	MIC range (µg/ml)
Fluconazole	None	0.25–2.0
Itraconazole	None	0.06–0.5
Voriconazole	None	0.03–0.25
Amphotericin B	None	0.25–1.0

Table 6: Minimum inhibitory concentration (E strip & MBD) of *Candida* species.

S. no	Fluconazole			Voriconazole			Itraconazole			Amphotericin B		
	E Test	MBD	Result	E Test	MBD	Result	E Test	MBD	Result	E Test	MBD	Result
1	1	1	S	0.064	0.0625	S	-	0.125	S	0.25	0.25	S
2	0.19	0.25	S	0.064	0.0625	S	-	0.125	S	4	4	S
3	0.5	0.5	S	0.25	0.25	S	-	0.5	S	0.75	1	S
4	0.5	0.5	S	0.125	0.125	S	-	0.0625	S	0.5	0.5	S
5	0.19	0.25	S	0.047	0.0625	S	-	0.125	S	0.25	0.25	S
6	0.125	0.125	S	1	0.25	S	-	0.5	S	0.5	0.125	S
7	0.125	0.125	S	1	0.25	S	-	0.5	S	0.5	0.625	S
8	0.38	0.5	S	0.38	0.5	S	-	0.5	S	0.25	0.25	S
9	0.047	0.0625	S	0.19	0.25	S	-	0.5	S	0.5	0.5	S
10	No zone	8	R	0.25	0.25	S	-	0.0625	S	0.25	0.25	S
11	0.25	0.25	S	0.25	0.25	S	-	0.5	S	0.19	0.25	S
12	0.5	0.5	S	0.125	0.125	S	-	0.25	S	0.38	0.5	S
13	0.5	0.5	S	0.094	0.125	S	-	0.25	S	0.064	0.0625	S
14	No zone	16	R	0.19	0.25	S	-	0.5	S	0.25	0.25	S
15	0.5	0.5	S	0.094	0.125	S	-	0.25	S	0.5	0.5	S
16	No zone	8	R	0.5	0.5	S	-	1	R	1	1	S
17	No zone	16	R	0.094	0.125	S	-	0.25	S	0.38	0.5	S
18	No zone	8	R	0.25	0.25	S	-	0.5	S	0.5	0.5	S
19	0.38	0.5	S	0.125	0.125	S	-	0.25	S	0.064	0.0625	S
20	0.19	0.25	S	0.5	0.5	S	-	2	R	0.38	0.5	S

Continued.

S. no	Fluconazole			Voriconazole			Itraconazole			Amphotericin B		
21	0.38	0.5	S	0.064	0.0625	S	-	0.25	S	0.19	0.25	S
22	0.094	0.125	S	0.5	0.5	S	-	0.5	S	0.75	1	S
23	0.064	0.0625	S	0.047	0.0625	S	-	0.25	S	0.094	2	S
24	0.19	0.25	S	0.094	0.125	S	-	0.125	S	0.5	0.5	S
25	0.094	0.125	S	0.5	0.5	S	-	2	R	0.38	0.5	S
26	0.19	0.25	S	0.047	0.0625	S	-	0.0625	S	0.38	0.5	S
27	No zone	NA	R	4	NA	R	-	NA			NA	
28	No zone	NA	R	No zone	NA	R	-	NA			NA	
29	0.38	0.5	S	0.125	0.125	S	-	0.0625	S	0.5	0.5	S
30	0.5	0.5	S	0.125	0.125	S	-	0.125	S	0.047	≤0.06 25	S
31	0.19	0.25	S	0.38	0.5	S	-	0.125	S	4	4	S
32	No zone	16	R	0.125	0.125	S	-	0.0625	S	0.5	0.5	S
33	0.38	0.5	S	0.094	0.125	S	-	0.125	S	0.064	0.0625	S
34	0.38	0.5	S	0.125	0.125	S	-	0.125	S	0.75	1	S
35	0.19	0.25	S	0.064	0.0625	S	-	0.0625	S	0.38	0.5	S
36	0.125	0.125	S	0.5	0.5	S	-	2	R	0.5	0.5	S
37	0.19	0.25	S	0.064	0.0625	S	-	0.0625	S	0.38	0.5	S
38	0.47	0.5	S	0.094	0.125	S	-	0.25	S	0.47	0.5	S
39	No zone	8	R	0.047	0.0625	S	-	0.125	S	0.38	0.5	S
40	0.125	0.125	S	0.125	0.5	S	-	2	R	0.38	0.5	S
41	0.19	0.25	S	0.064	0.0625	S	-	0.0625	S	0.5	0.5	S
42	0.47	0.5	S	0.125	0.125	S	-	0.0625	S	0.38	0.5	S
43	0.5	0.5	S	0.094	0.125	S	-	0.125	S	0.75	1	S
44	0.47	0.5	S	0.125	0.125	S	-	0.125	S	0.38	0.5	S
45	0.38	0.5	S	0.094	0.125	S	-	0.0625	S	1	1	S
46	0.38	0.5	S	0.125	0.125	S	-	0.125	S	0.125	0.125	S
47	No zone	16	R	0.125	0.125	S	-	0.125	S	1	1	S
48	0.19	0.25	S	0.047	0.0625	S	-	0.125	S	0.094	0.125	S
49	0.38	0.5	S	0.125	0.125	S	-	0.25	S	0.5	0.5	S
50	0.38	0.5	S	0.125	0.125	S	-	0.25	S	0.19	0.25	S
51	0.047	0.0625	S	0.19	0.25	S	-	2	R	0.25	0.25	S
52	0.19	0.25	S	0.047	0.0625	S	-	0.125	S	0.094	0.125	S
53	0.38	0.5	S	0.125	0.125	S	-	0.25	S	0.5	0.5	S
54	0.094	0.125	S	0.047	0.0625	S	-	0.0625	S	0.064	0.0625	S
55	0.5	0.5	S	0.19	0.25	S	-	0.5	S	1	1	S
56	0.38	0.5	S	0.125	0.125	S	-	0.25	S	0.5	0.5	S
57	0.19	0.25	S	0.094	0.125	S	-	0.25	S	0.38	0.5	S
58	0.5	0.5	S	0.25	0.25	S	-	0.5	S	2	2	S
59	0.25	0.25	S	0.094	0.125	S	-	0.25	S	0.5	0.5	S
60	0.38	0.5	S	0.064	0.0625	S	-	0.25	S	0.75	1	S
61	0.38	0.5	S	0.25	0.25	S	-	0.5	S	0.19	0.25	S
62	0.38	0.5	S	0.094	0.125	S	-	0.125	S	0.094	0.125	S
63	0.5	0.5	S	0.094	0.125	S	-	0.125	S	1	1	S
64	0.5	0.5	S	0.125	0.125	S	-	0.125	S	0.38	0.5	S
65	0.19	0.25	S	0.047	0.0625	S	-	0.125	S	0.38	0.5	S
66	0.38	0.5	S	0.094	0.125	S	-	0.0625	S	0.125	0.125	S
67	0.38	0.5	S	0.125	0.125	S	-	0.25	S	0.38	0.5	S

Antifungal susceptibility

Susceptibility testing of *Candida* spp. revealed resistance rates of 14.9% to fluconazole, 9.0% to itraconazole, and 3.0% to voriconazole. All *Candida* isolates were susceptible to amphotericin B. The MIC50 and MIC90 values for *C. albicans* were 0.25 µg/ml and 0.5 µg/ml for fluconazole, and 0.5 µg/ml and 1 µg/ml for amphotericin B, respectively. All 16 isolates of *C. neoformans* were susceptible to the antifungal agents tested, with low MICs across fluconazole, itraconazole, voriconazole, and amphotericin B.

Table 7: Minimum inhibitory concentration (E strip & MBD) of *Cryptococcus neoformans*.

S. no.	Fluconazole			Voriconazole			Itraconazole			Amphotericine B		
	E Test	MBD	Result	E Test	MBD	Result	E Test	MBD	Result	E Test	MBD	Result
1	2	2	S	0.047	0.0625	S	-	0.0625	S	0.125	0.125	S
2	0.5	0.5	S	0.064	0.0625	S	-	0.0625	S	0.047	0.0625	S
3	0.75	1	S	0.047	0.0625	S	-	0.125	S	0.38	0.5	S
4	2	2	S	0.064	0.0625	S	-	0.0625	S	0.5	0.5	S
5	1.5	2	S	0.125	0.125	S	-	0.0625	S	0.047	0.0625	S
6	0.5	0.5	S	0.047	0.0625	S	-	0.0625	S	0.047	0.0625	S
7	2	2	S	0.032	≤0.0625	S	-	≤0.0625	S	0.125	0.125	S
8	4	4	S	0.064	0.0625	S	-	0.125	S	0.125	0.125	S
9	2	2	S	0.032	≤0.0625	S	-	≤0.0625	S	0.38	0.5	S
10	4	4	S	0.064	0.0625	S	-	≤0.0625	S	0.25	0.25	S
11	1	1	S	0.047	0.0625	S	-	≤0.0625	S	0.125	0.125	S
12	4	4	S	0.064	0.0625	S	-	≤0.0625	S	0.38	0.5	S
13	1	1	S	0.094	0.125	S	-	0.125	S	0.25	0.25	S
14	2	2	S	0.064	0.0625	S	-	0.0625	S	0.047	1	S
15	2	2	S	0.047	0.0625	S	-	0.125	S	0.5	0.5	S
16	0.5	0.5	S	0.032	≤0.0625	S	-	0.0625	S	0.5	0.5	S

All *C. neoformans* isolates were susceptible to fluconazole, itraconazole, voriconazole, and amphotericin B. The narrow MIC ranges indicate preserved antifungal activity against C. neoformans in this setting, supporting the continued use of current treatment regimens for cryptococcal meningitis (Table 5).

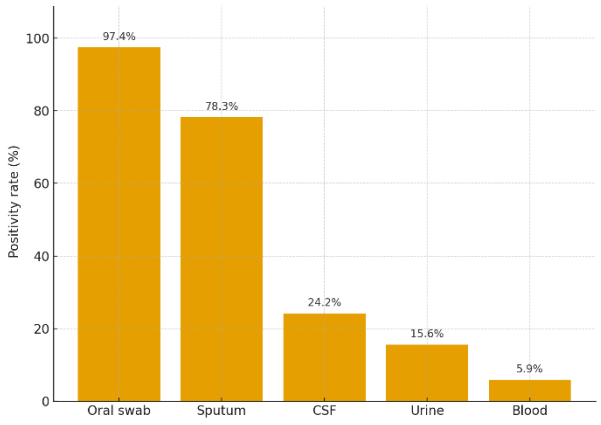

Figure 1: Specimen positivity rates.

Table 6 presents the minimum inhibitory concentration values obtained using both E-test and broth microdilution methods for *Candida* isolates. Overall, MIC values obtained by the two methods showed good concordance. Elevated MICs and resistance were most frequently observed for fluconazole, particularly among non-albicans *Candida* species, whereas amphotericin B consistently demonstrated low MIC values across isolates.

Table 7 demonstrates the MIC distribution of Cryptococcus neoformans isolates tested by E-test and broth microdilution. All isolates showed low MIC values for azoles and amphotericin B, indicating uniform susceptibility. The agreement between testing methods further supports the reliability of the susceptibility results.

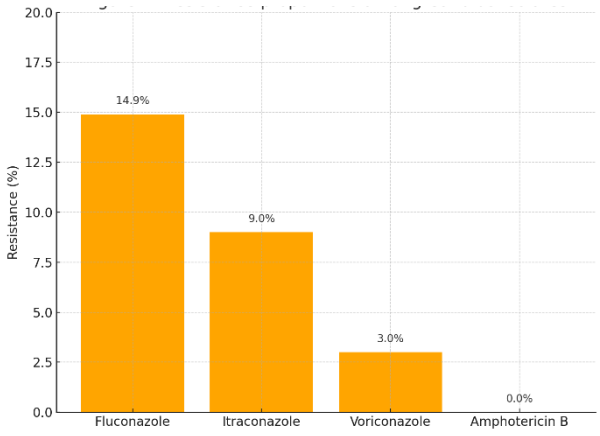

Figure 2: Resistance proportions among *Candida* isolates.

Figure 1 illustrates the specimen-wise distribution of fungal culture positivity. Oral swabs showed the highest positivity rate, followed by sputum and cerebrospinal fluid samples, while blood samples demonstrated minimal fungal recovery. This pattern highlights the predominance of mucosal and respiratory fungal infections in HIV-positive patients.

As shown in Figure 2, fluconazole resistance was the most commonly observed among *Candida* isolates, whereas resistance to itraconazole and voriconazole was comparatively lower. No resistance to amphotericin B was

detected. These findings emphasize the emerging challenge of azole resistance, particularly to fluconazole, in the management of candidiasis.

DISCUSSION

This investigation highlights that opportunistic fungal pathogens continue to play a prominent role in HIV-associated illness in North India. Nearly one-third of clinical specimens yielded fungi, with *Candida* species dominating mucosal and respiratory sites, and *Cryptococcus neoformans* accounting for all positive cerebrospinal fluid cultures. These observations are consistent with the clinical impression that oropharyngeal candidiasis and cryptococcal meningitis remain the most frequent fungal presentations in advanced HIV infection. What this study adds is contemporary, locally relevant microbiological data that can guide therapy in an era of emerging antifungal resistance.[5,13]

The predominance of *Candida albicans* among isolates in this cohort is not surprising, yet the consistent recovery of non-albicans species deserves attention. The identification of *C. tropicalis*, *C. parapsilosis*, and *C. glabrata* reflects a shift that has been reported internationally, where non-albicans strains are increasingly recognized as important opportunists. These species matter clinically because they often exhibit reduced susceptibility to azoles. For example, *C. parapsilosis* is associated with bloodstream infections linked to intravascular devices, and its biofilm-forming ability complicates eradication. *C. glabrata* is known to acquire resistance during therapy, particularly to fluconazole, and requires careful management. While *C. krusei* was rare in this study, its intrinsic fluconazole resistance highlights why precise species identification cannot be overlooked.[17,18]

The observation that every positive CSF culture grew *Cryptococcus neoformans* reinforces the singular role of this pathogen in HIV-related meningitis. Although the proportion of culture-positive CSF samples was smaller than for oral or sputum specimens, the clinical weight of cryptococcal disease is disproportionately high because of its high fatality rate. The exclusive recovery of *C. neoformans* also suggests that other fungal meningoencephalitides remain uncommon in this setting. This has practical value: when faced with an HIV-positive patient presenting with neurological symptoms, clinicians should prioritize cryptococcal antigen testing and empiric coverage for cryptococcosis while awaiting culture confirmation.[16,20]

Susceptibility testing of *Candida* isolates revealed a resistance rate of approximately 15% to fluconazole, with lower rates for itraconazole and voriconazole, and preserved activity of amphotericin B. These results mirror the growing body of Indian data indicating that fluconazole resistance among *Candida* spp. is no longer exceptional. The clinical implication is that reliance on fluconazole for empirical management of candidiasis may be increasingly unsafe without susceptibility confirmation, particularly in recurrent or refractory cases. Cross-resistance across azoles is also noteworthy, as itraconazole and voriconazole are often used as alternatives. The absence of resistance to amphotericin B is reassuring, but its toxicity profile and requirement for inpatient monitoring limit its role in routine mucosal disease. Nevertheless, for severe systemic candidiasis, amphotericin B remains an important option in this region.

In contrast to the mixed susceptibility profile of *Candida*, *Cryptococcus neoformans* demonstrated universal susceptibility to all agents tested, including fluconazole. This finding is reassuring and suggests that current treatment regimens for cryptococcal meningitis remain appropriate in North India. However, vigilance is needed, as reports from Africa and parts of Asia have described fluconazole resistance emerging in the context of widespread prophylactic or maintenance therapy. Even low-level resistance in *Cryptococcus* could have grave clinical consequences given the centrality of fluconazole in long-term suppression. The maintenance of amphotericin B and azole activity in this cohort should not lead to complacency but rather should motivate continued surveillance to detect early shifts in resistance.

The results must be interpreted with certain limitations. The study was cross-sectional, which restricts assessment of incidence or outcomes, and sample sizes for some species were small. Colonization and infection could not be reliably distinguished for oral and respiratory isolates, which means the true burden of invasive candidiasis may be lower than reported culture yields. In addition, susceptibility testing was limited to four antifungal agents; echinocandins, which are widely used in other contexts for invasive candidiasis, were not included. Molecular resistance mechanisms were not investigated, and clinical correlates such as CD4 count, ART adherence, and treatment outcomes were not systematically integrated with microbiological findings. These limitations temper the generalizability of the data but do not diminish their importance as a snapshot of local epidemiology.

Despite these constraints, the study offers valuable insights with practical implications. It demonstrates that fungal pathogens remain a considerable clinical challenge in HIV-positive patients and that resistance patterns are evolving. For clinicians, the key message is the need to obtain species-level identification and susceptibility data whenever possible, particularly before initiating prolonged antifungal therapy. For public health, the findings emphasize the need for antifungal stewardship programs to curb inappropriate azole use, which fuels resistance.

Future research should extend to multicenter surveillance, incorporation of rapid diagnostics such as cryptococcal antigen testing, and integration of laboratory findings with clinical outcomes. Only through sustained monitoring and rational prescribing can the effectiveness of the limited antifungal armamentarium be preserved.

CONCLUSION

In HIV-positive patients at a North Indian tertiary center, *Candida* species predominated in mucosal and airway sites, while *Cryptococcus neoformans* was the leading CSF pathogen. Fluconazole resistance among Candida spp. was notable, whereas all *C. neoformans* isolates were susceptible to first-line antifungals. These findings support routine species-level identification and susceptibility testing to guide therapy and reinforce the importance of antifungal stewardship.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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