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Research Article

Shadow Pathogen Unmasked: Species Distribution and Antifungal Susceptibility Profiles of Non-Albicans *Candida* (NAC) Isolated from Heterogeneous Clinical Samples at a Tertiary Care Teaching Hospital- a Prospective Observational Study

Running title: Non-albicans *Candida* species distribution and antifungal susceptibility

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Article Info

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ABSTRACT

Background: Invasive fungal infections due to non-albicans *Candida* (NAC) species are increasingly recognised globally, with evolving species distributions and antifungal resistance patterns. The present study characterised NAC species isolated from heterogeneous clinical specimens, with a focus on distribution patterns and antifungal susceptibility. **Methods:** This cross-sectional, descriptive study was conducted over 12 months with a total of 65 clinical isolates suspected to represent candidiasis. Germ tube test and CHROMagar *Candida* were used for preliminary differentiation of *C. albicans*. NAC isolates (n=38) were identified and antifungal susceptibility testing (AST) performed. **Results:** Of 65 total isolates, 38 (58.5%) were NAC species. *Candida tropicalis* predominated (60.5%), followed by *C. parapsilosis* (15.8%), *Meyerozyma guilliermondii* (13.2%), *Nakaseomyces glabrata* (7.9%), and *Candidozyma auris* (2.6%). Urine was the most frequent source (57.9%). Four *C. tropicalis* isolates (17.4%) demonstrated fluconazole resistance while remaining susceptible to echinocandins and amphotericin B. *M. guilliermondii* exhibited the most alarming resistance profile, with 2/5 isolates showing multidrug resistance including resistance to fluconazole and caspofungin, and one isolate resistant to all agents tested. The single *C. auris* isolate from urine was pan-resistant. *N. glabrata* remained susceptible to all agents tested. *C. parapsilosis* isolates were uniformly susceptible. **Conclusion:** A significant epidemiological shift toward NAC species, with *C. tropicalis* dominance and emerging multidrug resistance particularly in *M. guilliermondii* and *C. auris*, underscores the necessity for routine speciation and AST in all clinical laboratories to guide targeted antifungal therapy and stewardship.

Keywords: non-albicans *Candida*; *Candida tropicalis*; CHROMagar; VITEK 2; antifungal susceptibility; antifungal stewardship.

Introduction

Fungal infections constitute a substantial and largely underappreciated burden of global morbidity and mortality, disproportionately affecting immunocompromised and critically ill patients. Among fungal pathogens, *Candida* species remain the most clinically significant, being responsible for an estimated 700,000 cases of invasive candidiasis annually worldwide [1]. Candidaemia carries crude mortality rates ranging from 30% to 55%, with attributable mortality approaching 25 to 40%, making it one of the most lethal healthcare-associated bloodstream infections recognised today [2].

Historically, *Candida albicans* accounted for the majority of clinical *Candida* isolates. However, over the past two decades, an unambiguous epidemiological transition has been observed, with non-albicans *Candida* (NAC) species collectively overtaking *C. albicans* in many tertiary care settings globally [1,3]. Longitudinal surveillance programmes such as the China Hospital Invasive Fungal Surveillance Net (CHIF-NET) reported that NAC species, particularly *C. tropicalis, C. parapsilosis* and *Nakaseomyces glabrata* (formerly *C. glabrata*), now account for over 50% of invasive candidiasis episodes in several high-income countries. This study involving 11,679 isolates over 12 years confirmed that antifungal resistance rates are rising significantly among NAC species, with azole resistance reaching 42.5% in *C. tropicalis* isolates causing candidaemia [1].

The South Asian epidemiological context is particularly salient. *Candida tropicalis* has emerged as the dominant uropathogen in diabetic and immunocompromised populations across this region, driven by its capacity to form biofilms, adapt metabolically under glycosuric conditions, and upregulate resistance determinants including ERG11, CDR1, MDR1 and FKS1 [4]. Diabetes mellitus and chronic kidney disease (CKD) represent major risk factors for candiduria, with glycosuria providing a nutrient-rich substrate that promotes fungal colonisation of the urinary tract [5]. A five-year retrospective study of *Candida* urinary tract infections (UTIs) confirmed that prolonged hospitalisation, urinary catheterisation and broad-spectrum antibiotic exposure are independent risk factors for candidial UTI [6].

Beyond *C. tropicalis*, the recognition of *Meyerozyma guilliermondii* (formerly *C. guilliermondii*) as an intrinsically multidrug-resistant species warrants heightened attention. This organism, phylogenetically reclassified in the *Meyerozyma* genus, is known to exhibit reduced susceptibility to azoles through overexpression of efflux pumps and alterations in ergosterol biosynthesis, and may also develop acquired resistance to echinocandins [7]. Identification laboratories that do not perform species-level speciation risk misclassifying *M. guilliermondii* as *C. albicans* or other susceptible species, leading to therapeutic failure.

Perhaps the most significant emerging threat is *Candidozyma auris* (formerly *C. auris*), classified by the World Health Organization (WHO) as a critical priority pathogen in 2022. This halophilic yeast, first identified in Japan in 2009, now causes multidrug-resistant outbreaks on six continents. Its propensity for nosocomial transmission, frequent misidentification by conventional phenotypic methods, and pan-resistance in many clinical strains render it uniquely dangerous [8]. Whole-genome sequencing studies have revealed that healthcare-associated transmission clusters are common, with ERG11 and FKS1 mutations as primary resistance determinants [9].

Despite this global context, local epidemiological data remain indispensable for guiding antifungal policy. Regional species distributions, resistance rates, and clinical associations differ substantially between settings, and empirical treatment decisions must be underpinned by locally generated surveillance data [10]. National and international guidelines increasingly emphasise the need for antifungal stewardship programmes

anchored in institution-specific resistance profiles [11].

Tertiary care hospitals in South India serve populations with high burdens of diabetes, CKD, and indwelling device use, creating a permissive environment for NAC colonisation and infection. Despite this imperative, data characterising NAC distribution and susceptibility from tertiary institutions in South India remain sparse, and few studies have comprehensively reported antifungal susceptibility profiles across all major NAC species using standardised automated methods [12] (Aruna and Jahappriya, 2025). The present study was therefore designed to fill this evidence gap by characterising the species-level distribution of NAC isolates recovered from heterogeneous clinical specimens at a tertiary care teaching hospital, and by evaluating their antifungal susceptibility profiles using VITEK 2, an internationally validated automated system, with interpretation against current CLSI M27/M44S 2022 breakpoints. The findings provide institution-specific data to inform empirical and targeted antifungal therapy and to support the development of evidence-based antifungal stewardship frameworks in this setting.

Materials and Methods

This was a cross-sectional, observational and descriptive study conducted in the Department of Microbiology at a tertiary care teaching hospital, Tamil Nadu, India, over a period of 12 months. The study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [13] (Von et al., 2008). Ethical clearance was obtained from the Institutional Ethics Committee (Ref: 504/ TSRMMCH&RC/ME-1/2024 – IEC No. 091 dated 04.05.2024) prior to commencement. As the clinical samples were collected for observational study, obtaining informed consent was waived by the IEC. All consecutive samples received in the Microbiology laboratory with a clinical suspicion of candidiasis were eligible for inclusion.

The study population comprised of patients in all ages and both genders attending the tertiary care hospital whose clinical specimens were received for mycological investigations. All clinically suspected samples were included (urine, blood, sputum, wound swab, throat swab, high vaginal swab (HVS) and endotracheal (ET) secretions). Samples from patients with a documented history of antifungal therapy within the preceding six months were excluded to minimise the confounding effect of prior antifungal exposure on susceptibility profiles.

Direct microscopic examination was performed following Gram staining, with particular attention to the identification of budding yeast cells, pseudohyphae, or true hyphae. For culture, specimens were inoculated in 5% sheep blood agar (SBA) and Sabouraud dextrose agar (SDA) supplemented with chloramphenicol (0.05 mg/mL) to inhibit bacterial contaminants. All culture plates were incubated at 37ºC and examined daily for five days. Colonies displaying characteristics consistent with *Candida* species, including cream to off-white, pasty, smooth colonies with a yeasty odour, were selected for further identification.

Preliminary differentiation of *Candida albicans* from NAC species was performed using two complementary phenotypic methods. The germ tube test (GTT) was conducted by inoculating a single yeast colony into 0.5 mL of human serum and incubating at 37ºC for two to three hours; formation of a germ tube confirmed *C. albicans*, which was then excluded from further NAC characterisation. CHROMagar *Candida* (HiCrome *Candida* Differential Agar, HiMedia Laboratories) was used for chromogenic differentiation. Colonies were incubated at 37ºC for 48 hours, and colour reactions were interpreted according to the manufacturer's guidelines: *C. albicans* as light green, *C. tropicalis* as metallic blue, *C. parapsilosis* as

white to cream, and *C. guilliermondii* as pink to purple. *C. glabrata* (now *N. glabrata*) appeared as cream to white. Quality control strains used included *C. albicans* ATCC 10231, *C. tropicalis* ATCC 750, *C. parapsilosis* ATCC 22019 and *C. guilliermondii* ATCC 6260.

Definitive species-level identification of all NAC isolates was performed using the VITEK 2 automated microbiology system (BioMerieux, Inc., Durham, NC, USA) with the YST identification card (REF 21343). Yeast suspensions were prepared by emulsifying a pure 24 hour subculture colony in 3 mL of sterile saline to a turbidity of 2.0 McFarland units, as recommended by the manufacturer. The VITEK 2 system employs fluorescence-based technology and biochemical mini-reactions to generate species identification with a probability score; identifications with a probability of 95% or above were accepted as definitive. Contemporary taxonomic nomenclature was applied in reporting: *Meyerozyma guilliermondii* (formerly *C. guilliermondii*), *Nakaseomyces glabrata* (formerly *C. glabrata*) and *Candidozyma auris* (formerly *C. auris*).

Antifungal susceptibility testing (AST) was performed for all NAC isolates using the VITEK 2 AST-YS09 card (REF 4233331, bioMerieux). The card evaluates minimum inhibitory concentrations (MICs) for the following antifungal agents: fluconazole, voriconazole, caspofungin, micafungin, amphotericin B and flucytosine. MIC results were interpreted using Clinical and Laboratory Standards Institute (CLSI) M27-Ed4 and M44S 2022 species-specific breakpoints. Isolates were categorised as susceptible (S), intermediate (I) or resistant (R) based on these breakpoints. CLSI quality control reference strains were tested concurrently with each batch to ensure assay validity.

Categorical variables, including species distribution across sample types and antifungal susceptibility categories were summarised as frequencies and proportions. Descriptive statistics were employed throughout. The chi-square test as appropriate, was used to compare resistance rates between species where cell counts permitted. A p-value of less than 0.05 was considered statistically significant. All statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 65 clinical isolates from heterogeneous samples were processed during the study period. Of these, 38 (58.5%) were identified as non-albicans *Candida* species and constituted the primary study cohort. The remaining 27 (41.5%) were identified as *C. albicans* by germ tube positivity and were excluded. The overall distribution of clinical samples and isolate yield is summarised in Table 1.

Table 1. Distribution of clinical samples and NAC isolate yield

Parameter	N=100 (%)
C. albicans isolates (excluded)	27 (41.5%)
NAC isolates identified	38 (58.5%)
Urine cultures	22 (57.9%)
Sputum	4 (10.5%)
Wound swab/pus	4 (10.5%)
Endotracheal culture	2 (5.3%)

Blood culture	2 (5.3%)
Throat swab	2 (5.3%)
High vaginal swab	2 (5.3%)

Among the 38 NAC isolates, *Candida tropicalis* was the most prevalent species, accounting for 23 isolates (60.5%), followed by *C. parapsilosis* with 6 isolates (15.8%). The detailed species distribution is shown in Table 2. Urine samples yielded the highest proportion of NAC isolates across nearly all species, constituting 22/38 (57.9%) of the total NAC cohort. Within the urinary isolates, *C. tropicalis* accounted for 16 of 22 (72.7%), reflecting a strong predilection of this species for the urogenital tract.

Table 2. Species distribution of non-albicans Candida isolates (n=38)

NAC Species	N (%)	Predominant Sample
Candida tropicalis	23 (60.5)	Urine (16)
Candida parapsilosis	6 (15.8)	Urine (3)
Meyerozyma guilliermondii	5 (13.2)	HVS (2)
Nakaseomyces glabrata	3 (7.9)	Urine, Throat and sputum (1 each)
Candidozyma auris	1 (2.6)	Urine (1)

The antifungal susceptibility profiles of all five NAC species against major antifungal agents are detailed in Table 3. *C. tropicalis* (n=23) demonstrated selective azole resistance, with 4 isolates (17.4%) resistant to fluconazole; all remaining isolates were susceptible to voriconazole, echinocandins (caspofungin, micafungin), amphotericin B, and flucytosine. The fluconazole resistance rate in *C. tropicalis* isolates was the highest among the azole-susceptible major NAC species in this cohort. *C. parapsilosis* (n=6) exhibited complete susceptibility to all antifungal agents tested, including fluconazole, caspofungin, and amphotericin B, representing the most favourable resistance profile in this series.

M. guilliermondii (n=5) demonstrated the most clinically concerning resistance pattern among NAC species. Two of five isolates (40.0%) were resistant to fluconazole, and two isolates (40.0%) were resistant to caspofungin, with one isolate demonstrating pan-resistance to all agents tested including fluconazole, caspofungin, and amphotericin B, and a second isolate resistant to fluconazole, caspofungin, and amphotericin B simultaneously.

The multidrug resistance rate for *M. guilliermondii* (2/5, 40%) was significantly higher than for *C. tropicalis* (0/23, 0%; $p<0.05$, Fisher exact test). *N. glabrata* (n=3) remained susceptible to all antifungal agents tested, including fluconazole, caspofungin, and amphotericin B. The single *C. auris* isolate recovered from a urine sample was resistant to all antifungal agents tested, including fluconazole, caspofungin, voriconazole, and amphotericin B, confirming a pan-resistant profile.

Table 3. Antifungal susceptibility of NAC species tested by VITEK 2 AST-YS09 (n=38), interpreted per CLSI M27/M44S 2022 breakpoints

Species and Antifungal Agent	Susceptible	Intermediate	Resistant	Resistance Rate
Candida tropicalis (n=23)				
Fluconazole	19	0	4	17.4%
Meyerozyma guilliermondii (n=5)				
Fluconazole	3	0	2	40%
Caspofungin	3	0	2	40%
Amphotericin B	4	0	1	20%
Candidozyma auris (n=1)				
Fluconazole	0	0	1	100%
Caspofungin	0	0	1	100%
Amphotericin B	0	0	1	100%
Voriconazole	0	0	1	100%

Discussion

The present study provides contemporary, institution-specific data on NAC species distribution and antifungal resistance from a tertiary care teaching hospital in South India, a region characterised by high burdens of diabetes, CKD and healthcare-associated infections. Our principal findings collectively demonstrate a dominant epidemiological role of *C. tropicalis*, concerning multidrug resistance in *M. guilliermondii*, pan-resistance in the first locally identified *C. auris* isolate, and a preserved susceptibility profile in *C. parapsilosis* and *N. glabrata*. These findings have direct implications for empirical antifungal selection and infection control policy.

C. tropicalis dominated our NAC cohort (60.5%), a pattern consistent with surveillance data from South and Southeast Asian settings. A comprehensive review synthesising epidemiological, mechanistic, and genomic evidence identified *C. tropicalis* as the predominant *Candida* uropathogen in diabetic and immunocompromised populations across South Asia, substantially exceeding the dominance of *C. albicans* in these patients [4]. The predilection of *C. tropicalis* for the urinary tract is explained by its ability to exploit glycosuric conditions prevalent in diabetics, upregulate biofilm production under hypoxic and glucose-rich environments, and overexpress efflux pump genes including ERG11 and MDR1 under antifungal selection pressure [4]. In our cohort, *C. tropicalis* isolates (69.6%) originated from urine cultures, and the clinical profile of urinary isolates from patients with diabetes mellitus and CKD was consistent with the vulnerability described in prior literature [4,6].

Fluconazole resistance rate of 17.4% observed in *C. tropicalis* is clinically noteworthy. A five-year retrospective analysis of 229 *Candida* UTI cases from China confirmed that *C. tropicalis* exhibits significantly greater triazole resistance compared to *C. albicans* ($p<0.05$), with rising non-albicans proportions over the study period [6]. The CHIF-NET longitudinal study corroborated this finding, reporting that azole resistance in *C. tropicalis* causing candidaemia reached 42.5% for fluconazole in the most recent surveillance period [1]. Our relatively lower rate may reflect

differences in antifungal prescribing practices, patient demographics, or the inclusion of non-invasive specimens in our cohort; however, even a 17.4% resistance rate is sufficient to question the reliability of empirical fluconazole monotherapy in this setting. Importantly, all fluconazole-resistant *C. tropicalis* isolates in our study remained susceptible to echinocandins and amphotericin B, supporting the use of these agents when azole resistance is suspected or confirmed.

C. parapsilosis demonstrated complete antifungal susceptibility in all six isolates, a finding broadly consistent with literature. Studies from pediatric populations in southern Spain reported that *C. parapsilosis* accounted for 46.5% of candidaemia episodes, with fluconazole resistance remaining below 1% [14]. In Brazil, *C. parapsilosis* was the most frequently identified species in neonatal *Candida* infections, and while biofilm production was notable, antifungal susceptibility was generally preserved for azoles and echinocandins [15].

The consistently low resistance rates of *C. parapsilosis* to azoles and polyenes make this species relatively amenable to standard antifungal therapy, although surveillance for echinocandin resistance via FKS1 mutations warrants continued vigilance, particularly as this species forms robust biofilms [16]. In our dataset, all six *C. parapsilosis* isolates recovered from urinary and wound sources were susceptible, providing reassurance regarding local treatment options.

The resistance profile of *M. guilliermondii* is the most alarming finding of this study. Two of five isolates demonstrated multidrug resistance, with one isolate pan-resistant to all agents tested. The phylogenomic reclassification, possesses intrinsically elevated MICs for azoles compared to *C. albicans* due to constitutive overexpression of efflux transporter genes CDR1 and MDR1 [7,17]. A reference study characterising 20 clinical *M. guilliermondii* isolates using EUCAST-recommended broth microdilution found azole MIC ranges of 2-64 mg/L for fluconazole, with one isolate demonstrating a non-wild-type phenotype for all azoles tested, and confirmed that resistance to 5-fluorocytosine should also be considered in this species [7]. Clinically, the co-resistance to caspofungin observed in two of our *M. guilliermondii* isolates is particularly concerning given that echinocandins represent the first-line recommendation for most invasive candidiasis.

Resistance to both azoles and echinocandins simultaneously leaves clinicians with limited therapeutic options, essentially confining treatment to amphotericin B-based regimens or investigational agents. Our observation that one isolate was also resistant to amphotericin B renders the therapeutic landscape for this patient potentially treatment-refractory. These data firmly support the need for routine AST for all *M. guilliermondii* isolates and the avoidance of empirical fluconazole when this species is suspected.

N. glabrata (formerly *C. glabrata*) maintained complete susceptibility to all agents tested in our cohort. This finding diverges somewhat from global trends, which have documented rising echinocandin resistance in this species particularly in healthcare settings with heavy echinocandin use. A phylogenomic study of 142 *N. glabrata* clinical isolates from across Canada identified that 43.7% were resistant to at least one antifungal, with echinocandin resistance linked to well-characterised FKS1/FKS2 hotspot variants. The WHO classified *N. glabrata* as a high-priority pathogen in 2022 based on this trajectory [18].

The preserved susceptibility in our isolates may reflect the relatively limited historical use of echinocandins in this setting, underscoring the importance of ongoing local surveillance as antifungal prescribing practices evolve. Recent immunological studies have further demonstrated that echinocandin resistance in *N. glabrata* is associated with cell wall remodelling that can dampen innate immune recognition, potentially

conferring additional virulence advantages and complicating clinical management [19].

The single *C. auris* isolate from our cohort exhibited pan-resistance to all agents tested, a finding that, while based on a single isolate, is consistent with published reports. A whole-genome sequencing study of 68 *C. auris* isolates at Johns Hopkins Hospital confirmed widespread resistance determinants including ERG11 mutations conferring fluconazole resistance and FKS1 mutations conferring echinocandin resistance, with evidence of healthcare-associated transmission clusters [9]. Studies from Pakistan, where *C. auris* was similarly identified in ICU and SCU patients, documented resistance to fluconazole, amphotericin B, and voriconazole, confirming multidrug resistance as the expected phenotype for this emerging pathogen in South Asian settings [20]. The presence of even a single pan-resistant *C. auris* isolate in our institution demands immediate implementation of enhanced infection prevention protocols, including environmental decontamination, contact precautions, and prospective surveillance. Misidentification of *C. auris* using conventional systems remains a documented concern; chromogenic media specifically CHROMagar identified *C. auris* correctly with 100% specificity in comparative studies, supporting its utility in resource-limited laboratories [21].

Several important clinical implications arise from our findings. First, empirical fluconazole monotherapy for suspected candidiasis in our institution carries significant risk of therapeutic failure given the 17.4% fluconazole resistance in *C. tropicalis* and the intrinsic multidrug resistance of *M. guilliermondii*. Echinocandins are an effective drug therapy for azole-resistant *Candida* species. Second, the predominance of urinary tract isolates in our cohort supports the need for proactive screening in high-risk patients with diabetes and CKD, particularly those with indwelling urinary catheters, where *Candida* UTI is independently associated with prolonged hospitalisation and higher mortality. Third, the detection of *C. auris* obligates hospitals in this region to develop *C. auris* response plans incorporating rapid species identification, whole-genome sequencing capability for outbreak investigation, and mandatory notification protocols, consistent with multinational studies [5,8,11]. The sample size of 38 NAC isolates, while sufficient to characterise species distribution, affords limited statistical power for subgroup comparisons, particularly for less prevalent species such as *M. guilliermondii* (n=5) and *C. auris* (n=1). Clinical data including comorbidities, prior antifungal therapy and patient outcomes were not systematically available for all isolates, limiting correlation of microbiological findings with clinical context. Longitudinal surveillance spanning multiple seasons and years would be needed to definitively characterise temporal trends.

Conclusion

This single-centric study establishes a compelling case for the mandatory introduction of routine fungal speciation and antifungal susceptibility testing in all clinical microbiology laboratories, to move clinical practice away from empirical, one-size-fits-all antifungal therapy and toward species-directed, stewardship-informed treatment strategies. A significant epidemiological shift toward NAC species, with *C. tropicalis* dominance and emerging multidrug resistance particularly in *M. guilliermondii* and *C. auris*, underscores the necessity for routine speciation and AST in all clinical laboratories to guide targeted antifungal therapy and stewardship.

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