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Prevalence and Pattern of Antifungal Drug Minimum Inhibitory Concentration (MIC) of Invasive Candidiasis and its Associated Risk Factors*

Angeli Joana Robillos[1,2] and Evelina Lagamayo[1]

[1]*St. Luke's Medical Center-Global City, Taguig, Philippines*
[2]*Armed Forces of the Philippines Medical Center, Quezon City, Philippines*

ABSTRACT

Background. Invasive candidiasis is defined by the growth of *Candida* species in the bloodstream or other internal organs. It is a global concern due to increasing multidrug resistance and high mortality rates. This study aimed to update prevalence data on *Candida* infections in the Philippines, analyzing demographic factors (age, sex), specimen sources, and associated risk factors. We compared antifungal resistance patterns against CLSI epidemiological cutoff values (ECVs) and clinical breakpoints and examined MIC variations by underlying disease to inform potential standardized empiric therapies.

Methodology. We conducted a retrospective analytical cross-sectional study (SLMC-IERC approval, minimal risk) reviewing one year of *Candida* speciation and susceptibility results from January 2024 to December 2024 at a private tertiary hospital. All aseptically collected samples that tested positive for *Candida* species were included. Respiratory and wound specimens required a Gram stain demonstrating yeasts and hyphae prior to culture, while urine cultures were included only if they yielded ≥100,000 CFU/mL. Identification and susceptibility testing were performed using the VITEK 2 system, with results interpreted using CLSI breakpoints and ECVs.

Results. Among 266 patients with *Candida* infections, invasive candidiasis predominated in those aged ≥60 years (66.4%). *Candida albicans* (21.7%) and *Candida tropicalis* (13.5%) were more frequent in females, while *Candida parapsilosis* (13.2%) and *Candida glabrata* (5.3%) were more common in males. Blood and CSF samples strongly correlated with invasive disease and underlying risk factors. *C. albicans* was linked to infection-related conditions (13.9%), malignancy (9.0%), and cardiovascular disease (6.8%). *C. parapsilosis* (23.3%) and *C. tropicalis* (20.7%) were frequently associated with infection, malignancy, and metabolic disorders. *C. glabrata* (7.5%), noted for antifungal resistance, was isolated in patients with direct infections (3.4%) and malignancies (1.9%). Among azoles, fluconazole demonstrated greater susceptibility against *Candida* species, requiring lower concentrations for inhibition, despite a higher resistance rate (13.22%) compared to voriconazole (8.95%). Among echinocandins, micafungin showed better susceptibility than caspofungin. Amphotericin B demonstrated the highest overall susceptibility (93–100%), though MICs approached ECV limits. Most susceptible MIC values were fluconazole 0.5 µg/mL for *C. albicans* and *C. parapsilosis*, 1.0 µg/mL for *C. tropicalis*; voriconazole and caspofungin 0.12 µg/mL; micafungin 0.06 µg/mL; amphotericin B 0.5 µg/mL; and flucytosine <1 µg/mL for all species.

Conclusion. These findings support a species-specific, risk-adapted approach to antifungal therapy, incorporating demographic and clinical variables. Continuous surveillance of invasive candidiasis prevalence and antifungal MIC trends, with periodic breakpoint updates, is crucial to preserve therapeutic efficacy. Effective management of multidrug-resistant *Candida* infections also requires close collaboration between clinicians and pharmacists, as well as the development of new dosing strategies based on pharmacokinetic/pharmacodynamic (PK/PD) principles.

Key words: candidiasis, antifungal agents, drug resistance, Candida, azoles

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Corresponding author: Angeli Joana F. Robillos, MD
E-mail: ajfrobillos@st.lukes.com.ph
ORCiD: https://orcid.org/0009-0000-2996-6333

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INTRODUCTION

Invasive candidiasis occurs among hospitalized patients when *Candida* infects the internal organs (such as the kidney or brain) or the bloodstream.[1] This infection typically happens when *Candida* species breach the damaged organ barrier, which can be caused by various factors such as cancer chemotherapy, surgery, bacterial toxins, and local vascular perfusion disorders (endogenous route). This primarily applies to species such as *C. albicans, Candida tropicalis, Candida glabrata (Nakaseomyces glabrata), Candida lusitaniae (Clavispora lusitaniae), Candida parapsilosis* and, to a lesser extent, *Candida krusei (Pichia kudriavzeveii).* Infections can also occur via an exogenous route, such as through healthcare workers' hands or central line colonization and subsequent spread into the blood. This is especially true for *Candida auris, C. parapsilosis, Candida lusitaniae (C. lusitaniae)* and *Candida haemulonii*.[2]

Besides colonization and immunosuppression, factors known to be associated with increased risk of invasive candidiasis (IC) are broad spectrum antibiotic use, total parenteral nutrition (TPN), central venous catheter (CVC), surgery, renal replacement therapy, diabetes, prolonged mechanical ventilation, severe sepsis, and high Acute Physiology and Chronic Health Evaluation (APACHE) II score,[3] which classifies disease severity and predict mortality in intensive care unit (ICU) patients. There are other early diagnostic tools to assess the risk of invasive candidiasis early in patients admitted to ICU, such as the *Candida* Colonization Index (CCI) and the *Candida* score. The use of these is debated and not validated for all populations. While the risk scoring showed sensitivity and specificity for IC of 81% and 74%, respectively, the population tested mostly included surgical ICU patients, with only 35% of admissions for medical reasons. Additionally, the tools may be less reliable for patients with nonsurgical reasons for ICU admission. Furthermore, considering these, the key management in improving patient outcome is prompt source control (i.e., the elimination of the focus of infection) through early initiation of antifungal therapy to reduce hospital mortality.[4]

The prevalence of candidiasis has been on a growing trend, which is often related to the immunological status of patients.[5] It remains one of the top 5 healthcare-associated bloodstream infections in the world and causes high mortality rates. In the Philippines, candidiasis constitutes to about 80.40% of the fungal infections in 2016.[6] Utilization of the health budget has improved over the years, but the governance and implementation challenges persist due to the fragmented nature of the system. Moreover, the country has a mixed health system with an expanding private sector. The regional and socioeconomic disparities, access to effective treatment from the subsidy of the government or availability of resources are prominent concerns especially to the marginalized communities.[7]

At present, antifungal agents are limited to three major classes: the polyenes, which bind fungal cell membrane ergosterol leading to cell lysis; azoles that inhibit ergosterol biosynthesis; and echinocandins that inhibit fungal (1,3)-β-D-glucan cell wall biosynthesis.[8] Due to indiscriminate use of antifungal prophylaxis, limited choice of appropriate antifungal drugs, delay in obtaining antifungal susceptibility testing (AFST) results and the lack of pharmacodynamic correlation between minimal inhibitory concentration (MIC) values and risk factors has caused emergence of rising numbers of multi-drug resistant microorganisms. Due to this emergence of acquired antifungal resistance, reassessing the pattern and discrepancy in clinical breakpoints (CBPs) of antifungal agents made it more complex. These problems are important to consider in updating the appropriate antifungal selection.[9]

Antifungals are associated with greater uncertainty. This is partly due to the relatively low prevalence of invasive fungal infections (mold infections) and some specific biological characteristics of fungal pathogens like the possibility of biofilm formation (especially for *Candida* species).[9]

Gaps in the overall improvements in health systems and ICU care in the last few decades, as well as the development of different antifungals and microbiological techniques, mortality rates in invasive candidiasis have not substantially improved. One of them is that early diagnosis of candidemia and deep-seated candidiasis remain a challenge due to the prolonged time to positivity of blood cultures, which can take up to 5 days to become positive, and due to the low yield of culture diagnostic tests for deep-seated candidiasis (50%). Guidelines may have existed to guide the choice of antifungal therapy, however, patients affected by invasive candidiasis need a tailored approach due to heterogeneous host factors and significant geographical variation in species distribution and antifungal drugs resistance rates. Also, the value of different treatment strategies remains to be clarified.[4]

Globally, the incidence rate of invasive candidiasis had been 3–5 per 100,000 persons in the general population, 1%–2% of all ICU admissions and 750,000 cases per year. *Candida albicans* is the most common cause of invasive candidiasis. Currently, non-*albicans Candida* species account for an increasing proportion of cases. It was known that deep-seated candidiasis, arising from direct inoculation or hematogenous dissemination of *Candida* into normally sterile body sites, is often difficult to diagnose and may affect a population as large as that of candidemia.[10]

While *C. albicans* remains the most prevalent species in many parts of the world, the past decade has seen a notable rise in non-*albicans Candida* species. For instance, *C. glabrata*, particularly among elderly patients and solid organ transplant recipients, is the second most common species in the United States, northwestern Europe, and Canada. In contrast, *C. parapsilosis* and *C. tropicalis* are more frequently reported in Southern Europe, South America, India, and Pakistan.[4] In the Philippines, a prospective study conducted in Metro Manila in 2012 reviewed 39 candidemia cases with isolated species. *Candida tropicalis* (35.9%) was the most prevalent, followed by *C. parapsilosis* (30.8%) and *C. albicans* (28.2%). The majority of the isolates were susceptible to fluconazole. The top 3 underlying conditions that lead to candidemia were cancer (28.2%), neurologic disease (20.5%) and both solid tumor and renal disease (17.9%). The most common risk factors were orotracheal intubation (33%) and intraabdominal surgery (33%).[11] In a retrospective study done in Bacolod City in

2018, 184 *Candida species* were isolated. The most frequent among these isolates were *C. albicans* (62%), *C. tropicalis* (15%) and *C. ciferrii complex* (10%). The samples submitted were 69.02% from respiratory specimens (sputum and tracheal aspirate), 20.65% from urine specimens, 7.61% from blood, and 2.72% from vaginal discharge.[6]

A study on risk prediction for invasive candidiasis by Ahmed et al. demonstrated that identifying patients at increased risk led to a meaningful reduction in fungal infections through the use of antifungal prophylaxis, as well as a significant decrease in overall mortality. This can be achieved by incorporating microbiological parameters to stratify high-risk groups, rather than relying solely on clinical indicators. One approach involves determining the MICs for a clinical isolate and interpreting susceptibility according to established CBPs, which is essential for selecting the most appropriate antifungal therapy.[12]

In this study, VITEK 2® system (bioMerieux, France), a commercial automated method that allows rapid and accurate species identification by comparison of the biochemical profile with an extensive database. Its system incorporates the YS08 antifungal susceptibility testing (AFST) cards with expanded role in yeast susceptibility test that determines *Candida* growth by attenuation of light measured through optical scanner, performing fully automated testing of susceptibility to flucytosine, amphotericin B, fluconazole, and voriconazole.[13] It has a miniaturized and automated version of the doubling dilution technique for MICs determined by microdilution method. It followed the breakpoints from Global Clinical and Laboratory Standards Institute-based (CLSI M27M44S 3rd Edition, August 2022) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) (version 2020) guideline for the *Candida* species.

Therefore, this study aims to review the current prevalence of invasive candidiasis and help clinicians improve patient outcomes through knowing the MIC values of antifungal drugs against isolated *Candida* species and determine risk factors affecting it. This will further help clinicians make the choice of empiric antifungal drugs by preventing side effects as well as evolution to drug resistance. Additionally, there is no available data for some low- and middle-income countries due to the absence of hospital infrastructures for blood culture analyses and further AFST workups.[4]

METHODOLOGY

The study design was retrospective, analytical cross-sectional, performed in the Microbiology section of a private tertiary hospital in Taguig City, Metro Manila. Random data were gathered retrospectively from January 2024 to December 2024. Reference materials used were the retrieved compilation of *Candida* speciation printed records of samples (blood, body fluids, urine, sputum, and body tissues) collected aseptically in sterile appropriate containers. The archive results from VITEK 2 identified specimens with *Candida* infection and measured the Anti-susceptibility Testing (AST). This programmed machine utilized the standard procedure of the Global Clinical and Laboratory Standards Institute (CLSI) M27M44S for antifungal susceptibility testing (AFST) and derivation of minimum inhibitory concentration (MIC) values. The researchers sought assistance from the medical technology staff in the Microbiology section for the retrieval of *Candida* speciation test results upon approval of the conduct of this study. Consequently, retrospective data collection, analysis of data in Excel worksheet and statistical computations were done in a timely manner.

Inclusion criteria

Culture results dated between January 2024 and December 2024 were collected. Patients with *Candida* species as the causative agent of their infection were included in the study. Data acquisition included only the patients' accession number, age, sex, underlying risk factor of disease or diagnosis, kind or source of specimen, date of laboratory submission, identified isolates, and its susceptibility test results. The timings of reading were noted to evaluate consistency of the MIC determination. From the institutional database, all patients with *Candida* speciation requests to include Antimicrobial Sensitivity test results with MIC of each antifungal agent, were collected.

To increase the data collection, all one-year data with positive *Candida* infection was scrutinized and were considered relevant. All samples (sterile and non-sterile) were included in the study. For respiratory specimens included, gram stain results revealed presence of yeasts and hyphae. While urine cultures included were with colony growth of at least 100,000 colony-forming units (CFU) per milliliter of *Candida* species. These were processed by the medical technology staff in the Microbiology section for the specific organism identification and susceptibility testing using the VITEK 2 machine with AST-YS08 biochemical cards. The workflows were all based on the procedures of the laboratory.

Exclusion criteria

This study excluded patients with incomplete demographics and medical assessment. Also, if the causative agent was not *Candida* species it was disregarded and if the anti-susceptibility testing was terminated run in the VITEK 2 machine.

Sample size estimation

Sample sizes were obtained using the Krejcie-Morgan equation considering the essential measure of a level of accuracy as well as the required confidence level based on the existing population available for this study. The study size had a confidence level of 95% and error rate of 5%. This was due to 80% prevalence of Candidiasis in the Philippines.[6] The formula was as follows:

$$n = X^2NP1\text{-}P[e2\ N\text{-}1+X^2\ P1\text{-}P]$$

Where:

N = population size
X^2 = chi-square value at 95% confidence level with a degree of freedom of 1
P = proportion of the population
e = margin of error
n = 3.8412450.81-0.8[0.052245-1+3.8410.81-0.8]
n = 150.567 1.22
n = 123.42

Therefore, this study required at least 123 samples based on the calculation being made by the researcher and had a final 266 samples during the collection process from January 2024 to December 2024.

Data analysis

All data were analyzed using statistical tool SPSS version 27. Descriptive and exploratory data analyses were performed using absolute frequencies (n), relative frequencies (%), Measures of Central Tendency (mean and median) and had variance (standard deviation). Analysis of Variance was used to compare the antifungal drug resistance pattern of identified *Candida* species based on the Global CLSI ECVs and breakpoints. Furthermore, the differences of the Antifungal MIC of isolated *Candida* species according to risk factors of underlying disease using Analysis of Variance. The significance among identified variables was calculated based on the standard p-value of <0.05.

Ethical considerations

The study abided by the Principles of the Declaration of Helsinki (2013) and conducted in accordance with the Guidelines of the International Conference on Harmonization-Good Clinical Practice (ICH-GCP), E6 (R2) and other ICH-GCP 6 (as amended); National Ethical Guidelines for Health and Health-Related Research (NEG HHRR), 2017, and the Philippine Data Privacy Act of 2012. This protocol was approved by the SLMC Institutional Ethics Review Committee. Patient confidentiality was respected by ensuring anonymity of patient records. This research had no direct involvement with any vulnerable subjects that may breach ethical codes (e.g., children, prisoners, pregnant women, mentally challenged, educationally and economically disadvantaged), with no consent necessary due to the use only of laboratory information systems and printed records.

RESULTS

From January 2024 to December 2024, the tertiary hospital had a total of 447 requests of *Candida* speciation with susceptibility testing done in the hospital, giving an overall *Candida* infection prevalence rate of 59.51 % for a year. Of those 181 were excluded because identified were other filamentous fungi or susceptibility testing data was incomplete. A total of 266 patients with candidiasis were included for analysis. It was observed that the majority consists of admitted patients (98%) who had stayed in the hospital for at least 35 days and longer.

Candida species frequency is presented in Figure 1 by age group (0–18, 19–59, ≥60 years) to show the distribution of the species. The most common species was *C. albicans* (41.0%), and it was most prominent in the ≥60 years age group (26.7%), which may suggest that this species was more common in older people. The second most common species, *C. parapsilosis* (23.3%), also occurred most frequently in older adults (12.4%) and, to a lesser extent, in those 19–59 years of age (10.2%). This data suggests that these species may be more familiar with advancing age due to age-related changes in the host immune system or the presence of other diseases. On the other hand, uncommon species such as *C. glabrata* and *C. tropicalis* exhibited distinct age-related distribution. *Candida tropicalis* was present in 14.7% of patients who were ≥60 years old, whereas *C. glabrata* was rare and isolated from older adults (6.8%). Rare species like *C. krusei*, *C. lusitaniae*, and *C. orthopsilosis* were also seen in small numbers, in the older groups, consistent with their rarity. The table further supports the idea that age should be considered as an element in diagnosing and treating *Candida* infections because some species were more frequent in certain age groups.

Candida species distribution is presented in Figure 2 by sex, which shows the differences in species prevalence in male and female patients. The most common species remained *C. albicans* (41.0%), with slightly more in females (21.7%) than in males (19.5%). The second most common species, *C. tropicalis*, was also more prevalent in females (13.5%) than males (7.1%). *Candida glabrata*, on the other hand, presented a male prevalence, with 5.3% of the cases in males and 2.3% in females. There were minimal differences in the incidence of *C. krusei* and *C. orthopsilosis* between the two groups because these were rare in the study population. Interestingly, some species, such as *C. lipolytica* and *C. famata*, were only or mainly found in males, but they were very few. These findings highlight the need to consider the patient's sex when dealing with *Candida* infections because some species may have a different prevalence in males and females and thus require different

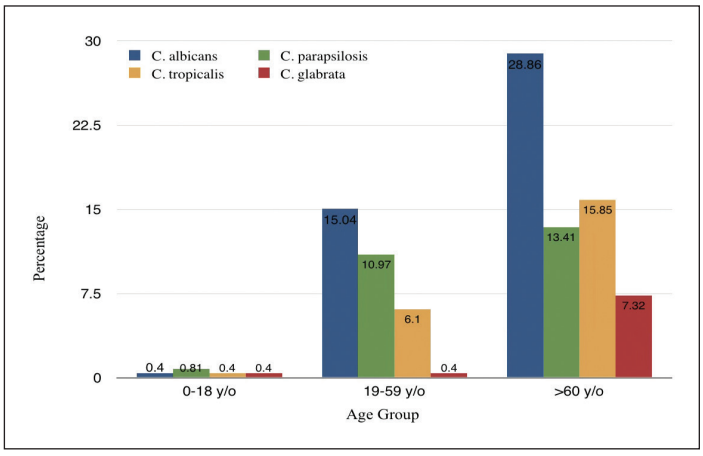

Figure 1. Age distribution of top 4 *Candida* species from January 2024 to December 2024 in a private tertiary hospital.

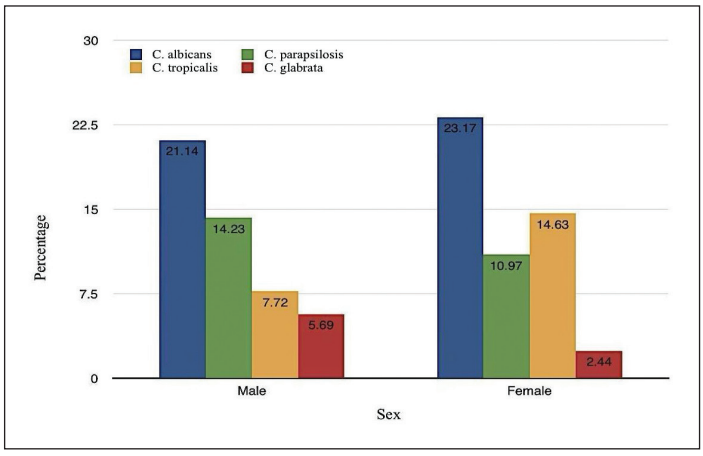

Figure 2. Sex distribution of top 4 *Candida* species from January 2024 to December 2024 in a private tertiary hospital.

approaches to diagnosis and treatment. Hence, association to anatomical, physiologic, and hormonal characteristics in relation to *Candida* growth is suggestive for further investigation.

Figure 3 shows the frequency distribution of *Candida* species according to the source of the samples and reveals considerable differences in prevalence among different sample types. The most common species was *C. albicans* (41.0%), which was most frequently found in cerebrospinal fluid (CSF, 10.2%), and in blood (9.0%). The second most common species, C. *parapsilosis* (23.3%), was strongly associated with sepsis and was isolated from blood in 18.8% of the cases. *C. tropicalis* (20.7%) also mainly recovered from CSF (8.6%).

Notably, this study identified the presence of *C. albicans* (16.8%) and *C. tropicalis* (8.6%) in respiratory specimens. These findings were included to explore the observations by Barantsevich et al., who reported strong associations between *Candida* isolated from multiple specimen types and invasive disease, supporting the Infectious Diseases Society of America (IDSA) recommendation for non-invasive diagnostics and empiric antifungal therapy guided by clinical risk, surrogate markers, and culture data.[14]

Although *Candida* species are often considered innocent colonizers of the respiratory tract, components like beta-glucan in their cell walls can act as lung pro-inflammatory agents, leading to macrophage and neutrophil dysfunction.[6] Colonization studies suggest that disruption of the bacterial flora and factors facilitating fungal translocation into the bloodstream increase the risk of invasive candidiasis. Immune status also plays a critical role, influencing both the clearance of fungal organisms and the risk of organ involvement.[1]

According to Meena and Kumar, diagnosing *Candida* pneumonia remains challenging due to nonspecific imaging findings and the lack of definitive diagnostic tools. The clinical significance of *Candida* in respiratory samples, particularly in ventilator-associated pneumonia (VAP) and community-acquired pneumonia (CAP), remains uncertain. Nonetheless, despite limited clinical evidence, the potential roles of biofilm formation, advanced genetic techniques, and fungal-bacterial crosstalk should not be overlooked.[15]

The rare species such as *C. krusei*, *C. lipolytica*, and *C. auris* were isolated from different sample types but to a minimal level in this study population. These findings also support the idea that the sample source is critical in determining the clinical relevance of *Candida* species since some pathogens are more associated with harboring areas that allow it, which helps in selecting the appropriate diagnostic and therapeutic measures.

Figure 4 highlights the distribution of *Candida* species across various risk factors, demonstrating significant associations with underlying health conditions. *Candida albicans* (41.0%) was the most prevalent species, frequently linked to infection-related conditions (13.9%), malignancy (9.0%), and cardiovascular disease (6.8%), suggesting its opportunistic nature in immunocompromised patients. Similarly, *C. parapsilosis* (23.3%) and *C. tropicalis* (20.7%) were commonly associated with infections, malignancies, and metabolic conditions, reinforcing their role in hospital-acquired and systemic fungal infections. *Candida glabrata* (7.5%), known for its antifungal resistance, was primarily isolated in patients with infections (3.4%) and malignancies (1.9%), while *C. krusei* (1.1%) was found in a smaller subset of individuals, emphasizing the clinical relevance of species identification in high-risk populations.

The data further illustrates that metabolic disorders, particularly diabetes, were strongly associated with *C. albicans* (3.4%) and *C. tropicalis* (2.3%), aligning with the known impact of hyperglycemia on fungal growth. Chronic kidney

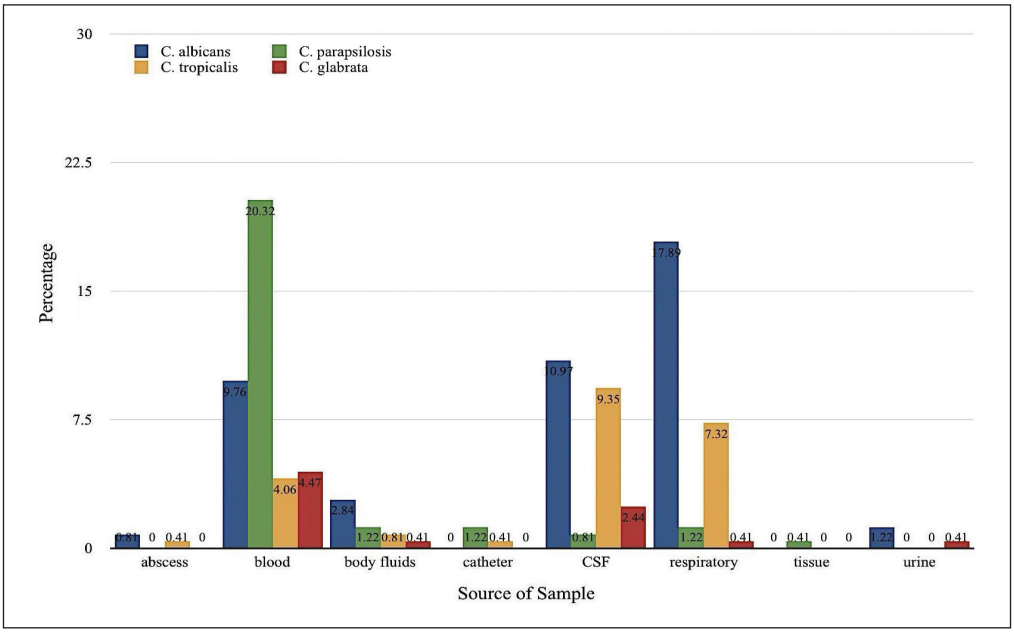

Figure 3. Distribution of sample source of top 4 *Candida* species from January 2024 to December 2024 in a private tertiary hospital.

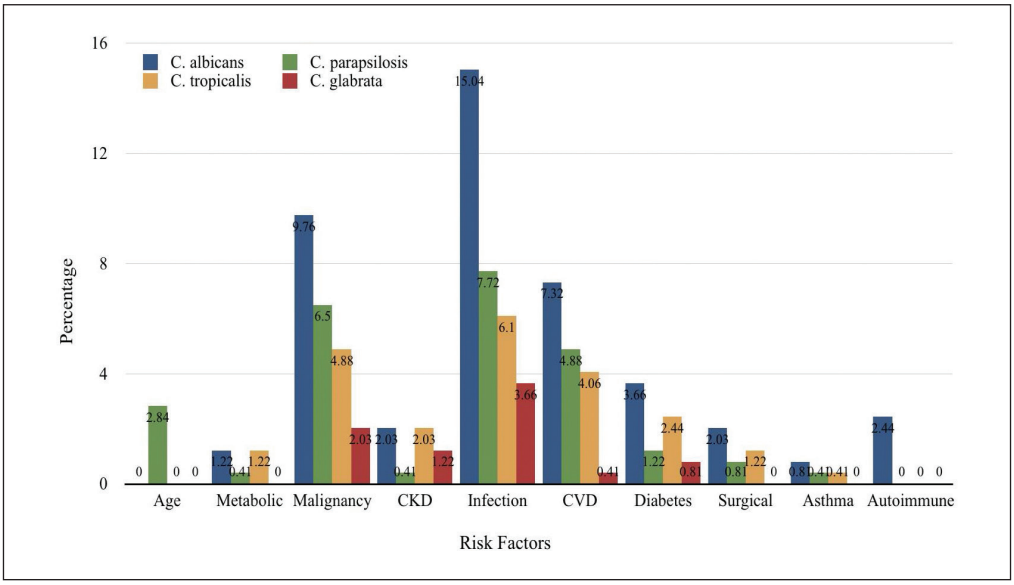

Figure 4. Distribution of risk factors for top 4 *Candida* species from January 2024 to December 2024 in a private tertiary hospital.

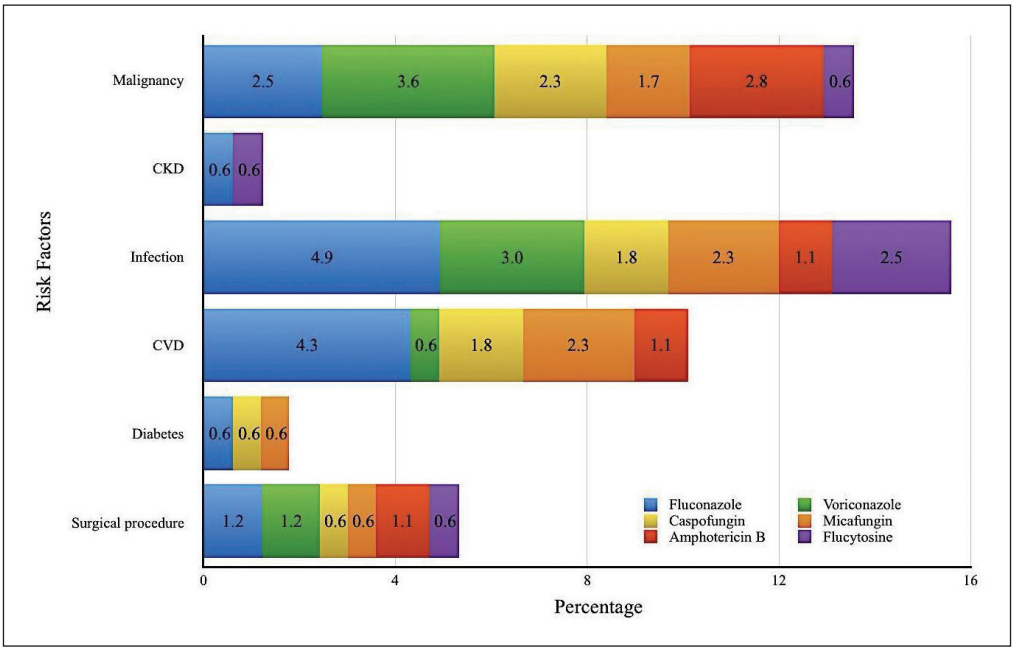

Figure 5. Antifungal drug resistance pattern according to risk factors from January 2024 to December 2024 in a private tertiary hospital.

disease (CKD) was linked to *C. albicans* and *C. tropicalis* (both at 1.9%), highlighting renal dysfunction as a potential predisposing factor. While surgical procedures and autoimmune diseases had lower associations with *Candida* infections, their presence suggests that immunosuppression or prolonged hospitalization may still contribute to fungal colonization. These findings underscore the need for targeted surveillance and early antifungal intervention in high-risk groups, particularly those with malignancies, diabetes, or CKD, to prevent complications associated with resistant *Candida* strains.

Figure 5 presents the distribution of antifungal drug resistance patterns according to risk factors. Among the six antifungal agents used in the study, the azole antifungal group had the highest number of drug resistance (13.22%) particularly fluconazole. The results indicated that fluconazole resistance was increased in infection-related (3.0%) causes collectively (such as pneumonia, cellulitis, ascites, pleural effusions, tendonitis, and sacral ulcers), cardiovascular diseases (CVD) collectively (such as hypertension, infarct and aneurysms) (2.6%) and malignancy (1.5%). Voriconazole resistance (8.95%) showed secondly in this study and was seen in patients with malignancy (2.3%) and infection (1.5%). Micafungin resistance (6.39%) and caspofungin resistance (5.98%) were seen mostly in patients with malignancy, CVD, and infection (1.37%, average). Amphotericin B resistance (5.7%) and

flucytosine resistance (5.96%) were commonly seen in malignancy (1.9%) and infection (1.5%), respectively.

Table 1 shows the MIC of the six antifungals for the AST of the top 4 *Candida* species. Fluconazole predominantly exhibited a MIC value of 0.5 ug/mL for the (69) susceptible *C. albicans* (40.66%) and the (22) susceptible *C. parapsilosis* (10.3%). While fluconazole predominantly exhibited a minimum inhibitory concentration (MIC) value of 1.0 ug/mL for the (40) *C. tropicalis* (18.79%) identified which were four dilutions away from the breakpoint (CLSI 8 ug/mL for *C. albicans*, *C. parapsilosis* and *C. tropicalis*). Voriconazole for

Table 1. Summary distribution antifungal minimum inhibitory concentration (MIC) of isolated Top 4 *Candida* species

Antifungal Agent MIC, ug/mL	Interpretation	*C. albicans*	Percentage %	*C. glabrata*	Percentage %	*C. parapsilosis*	Percentage %	*C. tropicalis*	Percentage %
Fluconazole									
0.5	S	67	40.66%	Not reported	Not reported	17	10.3%	6	3.64%
1	S	5	3.03%	Not reported	Not reported	4	2.42%	31	18.79%
2	S	4	2.42%	Not reported	Not reported	7	4.24%	5	3.03%
4	SDD	3	1.82%	Not reported	Not reported	0	0%	1	0.61%
8	R	9	5.45%	Not reported	Not reported	1	0.61%	1	0.61%
16	R	0	0%	Not reported	Not reported	0	0%	0	0%
32	R	4	2.42%	Not reported	Not reported	0	0%	0	0%
64	R	0	0%	Not reported	Not reported	0	0%	0	0%
	Total N=165	92				29		44	
Voriconazole									
0.008	S	0	0%	Not reported	Not reported	0	0%		0%
0.12	S	77	45.83%	Not reported	Not reported	28	16.67%	46	27.38%
0.25	I	2	1.19%	Not reported	Not reported	1	0.6%	0	0%
0.5	I	2	1.19%	Not reported	Not reported	0	0%	0	0%
1	R	4	2.38%	Not reported	Not reported	0	0%	0	0%
2	R	0	0%	Not reported	Not reported	0	0%	0	0%
4	R	4	2.38%	Not reported	Not reported	0	0%	1	0.6%
8	R	3	1.78%	Not reported	Not reported	0	0%	0	0%
	Total N=168	92				29		47	
Caspofungin									
0.008	S	0	0%	0	0%	0	0%	0	0%
0.12	S	81	46.55%	6	3.45%	3	1.72%	44	25.29%
0.25	I	3	1.72%	1	0.57%	20	11.49%	0	0%
0.5	I	0	0%	1	0.57%	5	2.87%	0	0%
1	R	0	0%	0	0%	0	0%	0	0%
2	R	0	0%	0	0%	0	0%	0	0%
8	R	8	4.6%	0	0%	0	0%	2	1.15%
	Total N=174	92		8		28		46	
Micafungin									
0.008	S	0	0%	0	0%	0	0%	0	0%
0.06	S	81	46.02%	9	5.11%	1	0.57%	44	25%
0.12	S	0	0%	0	0%	1	0.57%	0	0%
0.25	S	0	0%	0	0%	0	0%	0	0%
0.5	S	1	0.57%	0	0%	21	11.93%	0	0%
1	S	0	0%	0	0%	3	1.7%	0	0%
2	R	0	0%	1	0.57%	0	0%	0	0%
4	R	2	1.14%	0	0%	0	0%	2	1.14%
8	R	8	4.54%	0	0%	1	0.57%	1	0.57%
	Total N=176	92		10		27		47	
Amphotericin B									
0.12	S	0	0%	0	0%	0	0%	0	0%
0.25	S	13	7.18%	2	1.1%	7	3.87%	26	14.36%
0.5	S	50	27.62%	8	4.42%	20	11.05%	21	11.6%
1	S	23	12.71%	2	1.1%	1	0.55%	0	0%
2	R	1	0.55%	0	0%	0	0%	0	0%
4	R	1	0.55%	0	0%	1	0.55%	0	0%
8	R	2	1.1%	0	0%	0	0%	0	0%
16	R	2	1.1%	0	0%	0	0%	1	0.55%
	Total N=181	92		12		29		48	
Flucytosine									
0.006	S	0	0%	0	0%	0	0%	0	0%
<1	S	79	73.15%	12	11.11%	27	25%	46	43.52%
2	S	3	2.78%	0	0%	0	0%	0	0%
4	S	1	0.92%	0	0%	0	0%	0	0%
8	I	2	1.85%	0	0%	0	0%	0	0%
16	R	1	0.92%	0	0%	0	0%	0	0%
32	R	0	0%	0	0%	0	0%	0	0%
64	R	6	5.55%	0	0%	1	0.92%	1	0.92%
	Total N=108	92		12		28		47	

C. albicans (45.83%), *C. tropicalis* (27.38%) and *C. parapsilosis* (16.67%) predominantly exhibited a minimum inhibitory concentration (MIC) value of 0.12 ug/mL, which is three dilutions away from its breakpoint of 1.0 ug/mL. Among the azole group, *Candida* species in this study showed greater susceptibility to fluconazole, as it required lower concentrations relative to its clinical breakpoint. Despite rising resistance rates of 13.22% for fluconazole and 8.95% for voriconazole, fluconazole remains the preferred azole due to its excellent bioavailability and more favorable safety profile. In contrast, voriconazole is being linked to a higher risk of adverse effects and significant drug–drug interactions. However, resistance to fluconazole can develop through prolonged exposure, drug efflux mechanisms, and genetic mutations. A previous study comparing fluconazole monotherapy with a combination of fluconazole and amphotericin B demonstrated that the combination therapy achieved a higher overall treatment success rate and a lower incidence of persistent bloodstream infection.[10]

For *C. glabrata*, in accordance with the updated CLSI guidelines, modifications to the fluconazole formulation in the VITEK 2 AST-YS08 card have led to the exclusion of fluconazole from routine testing.[16] Heteroresistance to fluconazole is frequently observed in *C. glabrata* and is associated with the upregulation of ABC-type drug transporters. This phenomenon can be detected and quantified using population analysis profiling, a method not routinely employed in clinical microbiology laboratories.[10] Additionally, voriconazole was not requested for reporting by clinicians. Consequently, susceptibility results for these azole antifungals are not included in this study, although both agents are known to have resistance issues with *C. glabrata*.[17] Meanwhile, MIC values and interpretations for echinocandins indicate higher susceptibility to micafungin (90%) compared to caspofungin (75%). Susceptibility to amphotericin B and flucytosine was also tested, and the epidemiological cutoff values (ECVs) for these agents suggest that *C. glabrata* does not currently harbor known antifungal resistance mechanisms against them.

Two *C. dubliniensis* were identified in this study, with similar susceptible MIC value of 0.5 ug/mL in both azole drug, while one (0.4%) was identified to be resistant to fluconazole with MIC value of 32 ug/mL based on EUCAST breakpoint of >2 ug/mL. *Candida krusei* (0.8%) identified is known to be intrinsically resistant (IR) to fluconazole and no breakpoint was reported. Most of the (79) susceptible *C. albicans*, (42) C. *tropicalis*, (22) *C. parapsilosis* and (8) *C. glabrata*, the MIC value recorded were 0.06 ug/mL for micafungin and 0.12 ug/mL for caspofungin. Micafungin shows more susceptibility because C. *albicans, C. tropicalis* and *C. parapsilosis* were four dilutions away from the CLSI breakpoint. While, in caspofungin, *C.albicans, C. tropicalis* and *C. glabrata* were only two dilutions away from the CLSI breakpoint. Seen in this is a slight increase in resistance particularly of C. albicans to both echinocardins in (8) patients (3%) with malignancy, CVD, and infection (1.1%, 1.1% and 1.5%, respectively). Amphotericin B predominantly exhibited a minimum inhibitory concentration (MIC) value of 0.5 ug/mL for the top 4 *Candida*. However, most *C. albicans* (27.62%), *C. tropicalis* (11.6%) and *C. glabrata* (4.42%) were two dilutions away from the CLSI ECV of 2 ug/mL, while *C. parapsilosis* (11.05%) was one dilution away from the ECV value of 1.0 ug/mL. So, increasing resistance was noted in the usage of Amphotericin B, especially in malignant patients infected with *C. albicans* (2.3%). The three identified rare species such as *C. dubliniensis* and *C. lusitaniae* were both susceptible to Amphotericin B with MIC range value of 0.12 ug/mL to 1.0 ug/mL. Flucytosine, a combination drug to increase effectiveness of other drugs was not suppressed from the analysis in this study to verify its effectiveness to treat severe systemic IC. Thus, it showed that the predominant susceptible MIC value was <1 ug/mL. Among these susceptible results were *C. albicans* (28.6%), *C. tropicalis* (14.7%), *C. parapsilosis* (6.4%), and *C. glabrata* (3%). Only 1.9% of *C. albicans* were identified with resistance to flucytosine especially in infection-related causes. Despite minimal clinical data, the breakpoint (>32 ug/mL) used was based on the previous CLSI M27-S3: Reference Method for Broth Dilution Antifungal Susceptibility Testing of Yeasts, Third Informational Supplement, 2008. The results may outweigh the in vitro variability, and patients may benefit from this drug if proper dosing is monitored (LD_{50} = >15 gm/kg).

In addition to above findings, Appendices 6A-D show concordance with the MIC^{50}, MIC^{90} and percent of susceptible isolates of the top 4 *Candida* species according to 2022 Clinical and Laboratory Standards Institute (CLSI) Standards. Against all these top 4 *Candida* species, amphotericin B (93-100%) shows the highest susceptibility rate. *Candida* species growth is still inhibited by the lowest concentration of azole (fluconazole and voriconazole), but the MIC value (MIC^{90}) is higher than the susceptible breakpoint. *Candida parapsilosis* has the lowest susceptibility rate of 11% in caspofungin and signals the need for alternative agents in patients with persistent infection[10] due to increased intermediate growth (89%).

Moreover, Appendix 7 provides a summary of the antifungal drug resistance patterns observed among identified *Candida* species, highlighting variations in susceptibility across different antifungal agents. Candida albicans, the most prevalent species, demonstrated high susceptibility rates to antifungal drugs such as amphotericin B (30.8%), caspofungin (29.7%), and micafungin (29.7%). However, a small subset of *C. albicans* isolates exhibited resistance, notably to fluconazole (5.6%) and voriconazole (4.9%).

Candida parapsilosis, the second most common species, showed similar susceptibility patterns, with high susceptibility to amphotericin B and caspofungin but lower rates of resistance compared to *C. albicans*. Conversely, *C. glabrata* and *C. tropicalis* exhibited more concerning trends, with reduced susceptibility and occasional resistance across multiple drug classes. Notably, *C. glabrata* displayed resistance to caspofungin and fluconazole, drugs commonly used in clinical settings. Meanwhile, rare species like *C. krusei* demonstrated intrinsic resistance to fluconazole, underscoring the importance of species-level identification and tailored antifungal therapy.

Interestingly in this study, 2.63% of patients with invasive candidiasis particularly due to *C. tropicalis* (2 patients) and *C. albicans* (5 patients) were identified to be resistant to all these six antifungals. Other *Candida* species comprising

Table 2. Summary statistics of the differences in MIC of Antifungal drugs according to risk factors

Variables	Value	*p*-value
Fluconazole Risk Factor	110.93	0.005
Voriconazole Risk factor	133.62	<0.001
Caspofungin Risk factor	179.48	<0.001
Micafungin Risk factor	185.32	0.006
Amphotericin B Risk factor	176.68	<0.001
Flucytosine Risk factor	534.31	0.164

0.4% each were also identified using the VITEK 2 identification cards. These species were *C. hemolunii*, *C. lipolytica*, *C. auris*, *C. viswanath*, and *C. famanata*, however, MIC was not identified due to no reportable AST database.

Table 2 presents the statistical analysis of the differences in minimum inhibitory concentrations (MICs) of antifungal drugs according to risk factors. The results indicate significant variability in antifungal susceptibility based on patient-related factors, as shown by the p-values for fluconazole ($p = 0.005$), voriconazole ($p < 0.001$), caspofungin ($p < 0.001$), micafungin ($p = 0.006$), and amphotericin B ($p < 0.001$). These findings suggest that antifungal efficacy is influenced by specific risk factors such as underlying metabolic conditions, malignancy, and chronic kidney disease (CKD). Notably, flucytosine ($p = 0.164$) did not show statistical significance, indicating that its MIC distribution may be less affected by these clinical conditions.

These results underscore the importance of individualized antifungal treatment strategies, particularly for patients with malignancies, chronic kidney disease (CKD), and metabolic disorders, as these underlying conditions may influence antifungal susceptibility. The statistical significance observed in azole and echinocandin antifungals, especially fluconazole and caspofungin, highlights the potential for resistance development in high-risk populations. These findings highlight the critical importance of routine antifungal susceptibility testing (AFST) and tailored treatment strategies to enhance patient outcomes and curb the development of drug-resistant *Candida* infections.

DISCUSSION

The findings of this study provide significant insights into the distribution of *Candida* species, their demographic associations, and antifungal susceptibility patterns. *Candida albicans* emerged as the predominant species across all groups in this study, consistent with the meta-analysis by Yamin et al., on bloodstream infections in Southeast Asia.[18] *Candida parapsilosis* was the second most common species, notably associated with bloodstream infections and showing increased intermediate susceptibility to caspofungin. Similarly, Franconi et al., identified *C. parapsilosis* as the second leading cause of candidemia in regions including Europe, Latin America, South Africa, and Asia, while noting that echinocandin resistance remains less frequent than azole resistance. The differing susceptibility pattern observed in the present study underscores the need for molecular investigations of mutations linked to echinocandin tolerance.[19] In a trial comparing standard-dose and high-dose caspofungin regimens, the response rate for *C. parapsilosis* was lower in the standard-dose group (61%), though the difference was not statistically significant. Despite this, echinocandins remain the preferred treatment for *C. parapsilosis* infections. However, alternative agents should be considered in cases of persistent infection despite echinocandin therapy.[10]

The observed contrasting prevalence patterns underscore the need for continuous surveillance to accurately monitor the evolving burden of invasive candidiasis. A study by Yamin et al. reported prevalence rates of *C. albicans* (28.4%), *C. tropicalis* (29.2%), *C. parapsilosis* (19.1%), and *C. glabrata* (14.0%). Additionally, a multicenter study across six Southeast Asian countries identified *C. tropicalis* as the most prevalent species in hematology-oncology wards and tropical regions, likely influenced by variations in healthcare infrastructure, patient demographics, and associated risk factors.[1] These findings align with the observations of Cortes and Corrales, highlighting a global shift in *Candida* species distribution.

The detection of less common species like *C. krusei* and *C. glabrata*, though infrequent, highlights the diversity of *Candida* infections and the importance of accurate species identification for effective treatment.

Also, observed in this study, most direct wound infections occurred in outpatients, posed minimal health risks, and responded well to antifungal therapy. In contrast, hospitalized patients with risk factors and prolonged stays (>35 days) were more likely to develop systemic candidiasis and exhibit antifungal resistance. This is further complicated by the ability of *Candida* species to form biofilms, considered one of their most pathogenic traits, which can lead to treatment failure and recurrent infections. Additionally, certain species can transition into a filamentous form that enhances tissue invasion; however, this morphological shift is influenced by both environmental conditions and the specific *Candida* species involved.[6] Similarly, *Candida* isolated from respiratory and urinary specimens should be interpreted with caution, as it may represent colonization rather than true infection. In critically ill patients, concurrent candidemia originating from these sites has also been observed. Therefore, antifungal therapy should be reserved for immunocompromised patients[4,15] with clinical signs of septic shock and no alternative etiology.[20]

Similarly, Soriano et al., emphasized the importance of prompt antifungal therapy and the use of tools like MIC values for tailoring treatment strategies. Despite advances in diagnostic techniques, invasive candidiasis continues to pose a significant burden, particularly due to delayed diagnosis and the limited sensitivity of culture-based methods.

Notable resistance to azole-based treatments, including fluconazole and voriconazole, was observed. These azoles showed effectiveness but not of the lowest dose, thus,

warrant limited access for empirical therapy and somehow, becomes a challenge to patient management because this is a recommendable orally administered antifungal drug. Similar to other studies, antifungal susceptibility analysis revealed high overall effectiveness of drugs like amphotericin B and micafungin, particularly against *C. albicans* and *C. parapsilosis*. This high susceptibility shows increased chance of therapeutic concentration and effective clinical outcome. However, as amphotericin B in this study shows that MIC value (dilution away) is already near to its breakpoint. It will be for further investigation and for new treatment guidelines especially it is being recommended by Infectious Diseases Society of America (IDSA) as an alternative drug if azole or echinocandins fail to treat invasive candidiasis. Thus, this evolving resistance highlights the growing challenge of antifungal therapy, necessitating routine susceptibility testing and tailored treatment strategies like dosage adjustment based on therapeutic drug monitoring (TDM) could be needed when dealing with *Candida* strains having higher MICs near to the clinical breakpoint.[21] Furthermore, the observed variability in MICs based on risk factors underscores the complex interplay of host characteristics and fungal resistance. These findings advocate for an individualized, species-specific approach to antifungal therapy, integrating demographic and clinical factors to optimize outcomes in *Candida* infections.

CONCLUSION

This study highlights the significant prevalence of *Candida* infections and the variability in antifungal resistance patterns across different species. *Candida albicans* remains the most predominant species; however, the increasing prevalence of non-*albicans* species such as *C. parapsilosis, C. tropicalis* and *C. glabrata* underscores the need for vigilant surveillance and species-specific diagnostic approaches. The observed resistance to commonly used antifungal agents, particularly azoles like fluconazole and voriconazole, emphasizes the growing challenge of antifungal resistance, necessitating routine antifungal susceptibility testing (AFST) for effective treatment planning. Echinocandins especially micafungin is still the recommendable safest drug that shows high effectiveness against invasive candidiasis. Clinicians should consider this as their first choice of empiric treatment if needed, especially in areas where susceptibility testing is not available. Moreover, further research on amphotericin B MIC shifts is recommended due to its emerging resistance.

The findings also underscore the importance of considering patient-related factors, such as age, sex, and underlying conditions, in the management of *Candida* infections and may warrant periodic surveillance on these demographics to improve species-specific therapy. Minimum inhibitory concentration (MIC) values proved crucial in identifying resistance patterns and guiding appropriate antifungal therapy. To combat the rising threat of drug resistance, clinicians must adopt individualized, evidence-based strategies while integrating regional and local data on species distribution and antifungal susceptibility. Furthermore, the importance of knowing the MICs with different antifungal agents is to guide the infectious disease specialist, clinical pharmacologists, and pharmacists for a collaborative approach in computing for the pharmacokinetics/ pharmacodynamics (PK/PD) analysis to adjust the dosage of the antifungal drugs.

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APPENDICES

Appendix 1. Summary distribution of the proportion of *Candida* species according to age

Candida Species	Age (years)			Total (N = 266)
	0 – 18	19 – 59	≥60	
Candida albicans	1 (0.4%)	37 (13.9%)	71 (26.7%)	109 (41.0%)
Candida parapsilosis	2 (0.8%)	27 (10.2%)	33 (12.4%)	62 (23.3%)
Candida glabrata	1 (0.4%)	1 (0.4%)	18 (6.8%)	20 (7.5%)
Candida tropicalis	1 (0.4%)	15 (5.6%)	39 (14.7%)	55 (20.7%)
Candida krusei	0 (0.0%)	1 (0.4%)	2 (0.8%)	3 (1.1%)
Candida lipolytica	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)
Candida famata	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)
Candida lusitaniae	0 (0.0%)	2 (0.8%)	3 (1.1%)	5 (1.9%)
Candida orthopsilosis	0 (0.0%)	0 (0.0%)	5 (1.9%)	5 (1.9%)
Candida auris	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)
Candida dubliniensis	0 (0.0%)	1 (0.4%)	2 (0.8%)	3 (1.1%)
Candida duobushaemulonis	0 (0.0%)	1 (0.4%)	0 (0.0%)	1 (0.4%)

Appendix 2. Summary distribution of the proportion of *Candida* species according to sex

Candida species	Sex		Total (N = 266)
	Male	Female	
Candida albicans	52 (19.5%)	57 (21.7%)	109 (41.0%)
Candida parapsilosis	35 (13.2%)	27 (10.2%)	62 (23.3%)
Candida glabrata	14 (5.3%)	6 (2.3%)	20 (7.5%)
Candida tropicalis	19 (7.1%)	36 (13.5%)	55 (20.7%)
Candida krusei	2 (0.8%)	1 (0.4%)	3 (1.1%)
Candida lipolytica	1 (0.4%)	0 (0.0%)	1 (0.4%)
Candida famata	1 (0.4%)	0 (0.0%)	1 (0.4%)
Candida lusitaniae	3 (1.1%)	2 (0.8%)	5 (1.9%)
Candida orthopsilosis	4 (1.5%)	1 (0.4%)	5 (1.9%)
Candida auris	1 (0.4%)	0 (0.0%)	1 (0.4%)
Candida dubliniensis	1 (0.4%)	2 (0.8%)	3 (1.1%)
Candida duobushaemulonis	1 (0.4%)	0 (0.0%)	1 (0.4%)

Appendix 3. Summary distribution of the proportion of *Candida* species according to source of sample

Candida Species	Source of Sample								Total (N = 266)
	Abscess	Blood	Body Fluids	Catheter	CSF	Respiratory Specimen	Tissue	Urine	
Candida albicans	2 (0.8%)	24 (9.0%)	7 (2.7%)	0 (0.0%)	27 (10.2%)	44 (16.5%)	0 (0.0%)	3 (1.1%)	109 (41.0%)
Candida parapsilosis	0 (0.0%)	50 (18.8%)	3 (1.1%)	3 (1.1%)	2 (0.8%)	3 (1.1%)	1 (0.4%)	0 (0.0%)	62 (23.3%)
Candida glabrata	0 (0.0%)	11 (4.1%)	1 (0.4%)	0 (0.0%)	6 (2.3%)	1 (0.4%)	0 (0.0%)	1 (0.4%)	20 (7.5%)
Candida tropicalis	1 (0.4%)	10 (3.8%)	2 (0.8%)	1 (0.4%)	23 (8.6%)	18 (6.8%)	0 (0.0%)	0 (0.0%)	55 (20.7%)
Candida krusei	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	3 (1.1%)
Candida lipolytica	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	1 (0.4%)
Candida famata	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)
Candida lusitaniae	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	4 (1.5%)	0 (0.0%)	0 (0.0%)	5 (1.9%)
Candida orthopsilosis	0 (0.0%)	5 (1.9%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	5 (1.9%)
Candida auris	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)
Candida dubliniensis	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	3 (1.1%)
Candida duobushaemulonis	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)

Appendix 4. Summary distribution of the proportion of *Candida* species according to risk factor

Candida Species	Risk Factors											Total (N = 266)
	Age	Metabolic	Malignancy	CKD	Infection	CVD	Diabetes	Surgical Procedure	Asthma	Anemia	Auto-immune	
Candida albicans	0 (0.0)	3 (1.1)	24 (9.0)	5 (1.9)	37 (13.9)	18 (6.8)	9 (3.4)	5 (1.9)	2 (0.8)	0 (0.0)	6 (2.3)	109 (41.0)
Candida parapsilosis	7 (2.6)	1 (0.4)	16 (6.0)	1 (0.4)	19 (7.1)	12 (4.5)	3 (1.1)	2 (0.8)	1 (0.4)	0 (0.0)	0 (0.0)	62 (23.3)
Candida glabrata	0 (0.0)	0 (0.0)	5 (1.9)	3 (1.1)	9 (3.4)	1 (0.4)	2 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	20 (7.5)
Candida tropicalis	0 (0.0)	3 (1.1)	12 (4.5)	5 (1.9)	15 (5.6)	10 (3.8)	6 (2.3)	3 (1.1)	1 (0.4)	0 (0.0)	0 (0.0)	55 (20.7)
Candida krusei	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	2 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (1.1)
Candida lipolytica	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)
Candida famata	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)
Candida lusitaniae	0 (0.0)	0 (0.0)	2 (0.8)	0 (0.0)	1 (0.4)	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	5 (1.9)
Candida orthopsilosis	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (1.1)	0 (0.0)	0 (0.0)	2 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)	5 (1.9)
Candida auris	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)
Candida dubliniensis	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (0.8)	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	3 (1.1)
Candida duobushaemulonis	0 (0.0)	0 (0.0)	1 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.4)

Appendix 5. Summary statistics of the antifungal drug resistance pattern according to risk factors

Anti-Fungal MIC Reading	Risk Factors												
	Age	Metabolic	Malignancy	CKD	Infection	CVD	Diabetes	Surgical Procedure	Asthma	Anemia	Auto-immune	X^2	*p*-value
Fluconazole													
Susceptible	0 (0.0%)	0 (0.0%)	31 (11.7%)	9 (3.4%)	36 (13.5%)	29 (10.9%)	17 (6.4%)	6 (2.3%)	2 (0.8%)	0 (0.0%)	5 (1.9%)		
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	0 (0.0%)		
Resistant	0 (0.0%)	0 (0.0%)	4 (1.5%)	1 (0.4%)	8 (3.0%)	7 (2.6%)	1 (0.4%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	73.354	<0.001
Intermediate	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
None	7 (2.6%)	7 (2.6%)	25 (9.4%)	4 (1.5%)	44 (16.5%)	7 (2.6%)	3 (1.1%)	4 (1.5%)	1 (0.4%)	1 (0.4%)	1 (0.4%)		
Voriconazole													
Susceptible	0 (0.0%)	0 (0.0%)	32 (12.0%)	11 (4.1%)	38 (14.3%)	31 (11.7%)	15 (5.6%)	6 (2.3%)	3 (1.1%)	0 (0.0%)	5 (1.9%)		
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
Resistant	0 (0.0%)	0 (0.0%)	6 (2.3%)	0 (0.0%)	5 (1.9%)	1 (0.4%)	0 (0.0%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	70.05	0.002
Intermediate	0 (0.0%)	0 (0.0%)	2 (0.8%)	0 (0.0%)	1 (0.4%)	3 (1.1%)	2 (0.8%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
None	7 (2.6%)	7 (2.6%)	22 (8.3%)	3 (1.1%)	44 (16.5%)	7 (2.6%)	3 (1.1%)	4 (1.5%)	1 (0.4%)	1 (0.4%)	1 (0.4%)		
Caspofungin													
Susceptible	0 (0.0%)	0 (0.0%)	37 (13.9%)	12 (4.5%)	41 (15.4%)	34 (12.8%)	17 (6.4%)	8 (3.0%)	3 (1.1%)	0 (0.0%)	5 (1.9%)		
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
Resistant	0 (0.0%)	0 (0.0%)	4 (1.5%)	0 (0.0%)	3 (1.1%)	3 (1.1%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	61.55	<0.001
Intermediate	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
None	7 (2.6%)	7 (2.6%)	21 (7.9%)	2 (0.8%)	43 (16.2%)	6 (2.3%)	2 (0.8%)	4 (1.5%)	1 (0.4%)	1 (0.4%)	1 (0.4%)		
Micafungin													
Susceptible	0 (0.0%)	0 (0.0%)	38 (14.3%)	13 (4.9%)	42 (15.8%)	33 (12.4%)	18 (6.8%)	8 (3.0%)	3 (1.1%)	0 (0.0%)	5 (1.9%)		
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
Resistant	0 (0.0%)	0 (0.0%)	3 (1.1%)	0 (0.0%)	4 (1.5%)	4 (1.5%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	58.01	<0.001
Intermediate	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
None	7 (2.6%)	7 (2.6%)	21 (7.9%)	1 (0.4%)	42 (15.8%)	6 (2.3%)	2 (0.8%)	2 (0.8%)	1 (0.4%)	1 (0.4%)	1 (0.4%)		
Amphotericin b													
Susceptible	0 (0.0%)	0 (0.0%)	41 (15.4%)	13 (4.9%)	44 (16.5%)	35 (13.2%)	19 (7.1%)	8 (3.0%)	3 (1.1%)	0 (0.0%)	5 (1.9%)		
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
Resistant	0 (0.0%)	0 (0.0%)	5 (1.9%)	0 (0.0%)	2 (0.8%)	2 (0.8%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	64.33	<0.001
Intermediate	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
None	7 (2.6%)	7 (2.6%)	16 (6.0%)	1 (0.4%)	42 (15.8%)	6 (2.3%)	2 (0.8%)	4 (1.5%)	1 (0.4%)	1 (0.4%)	1 (0.4%)		
Flucytosine													
Susceptible	0 (0.0%)	0 (0.0%)	40 (15.0%)	11 (4.1%)	37 (13.9%)	32 (12.0%)	16 (6.0%)	8 (3.0%)	3 (1.1%)	0 (0.0%)	4 (1.5%)		
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
Resistant	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)	4 (1.5%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	57.58	0.002
Intermediate	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)		
None	7 (2.6%)	7 (2.6%)	20 (7.5%)	2 (0.8%)	47 (17.7%)	9 (3.4%)	5 (19%)	4 (1.5%)	1 (0.4%)	1 (0.4%)	2 (0.8%)		

Appendix 6A. *Candida albicans*

Antifungal	Breakpoint (ug/mL)	MIC Range (ug/mL)	MIC^{50} (ug/mL)	MIC^{90} (ug/mL)	% Susceptible
Fluconazole	8	<0.5->64	0.5	8	83
Voriconazole	1	<0.008->8	0.12	1	84
Caspofungin	1	<0.008->8	0.12	0.25	88
Micafungin	1	<0.008->8	0.06	0.5	89
Amphotericin B	2 (ECV)	<0.12->16	0.5	1	93
Flucytosine	>32	<0.006->64	<1	4	90

MIC=minimum inhibitory concentration, ug/mL; MIC^{50} = MIC value at which growth was inhibited in 50% of isolates; MIC^{90} = MIC values at which growth was inhibited in 90% of isolates; According to CLSI M27 guidelines.

Appendix 6B. *Candida glabrata*

Antifungal	Breakpoint (ug/mL)	MIC Range (ug/mL)	MIC^{50} (ug/mL)	MIC^{90} (ug/mL)	%Susceptible
Fluconazole	Not reported	<0.5->64	Not reported	Not reported	Not reported
Voriconazole	Not reported	<0.008->8	Not reported	Not reported	Not reported
Caspofungin	0.5	<0.008->8	0.12	0.25	75
Micafungin	0.25	<0.008->8	0.06	0.06	90
Amphotericin B	2 (ECV)	<0.12->16	0.5	0.5	100
Flucytosine	>32	<0.006->64	<1	<1	100

MIC = minimum inhibitory concentration, ug/mL; MIC^{50} = MIC value at which growth was inhibited in 50% of isolates; MIC^{90} = MIC values at which growth was inhibited in 90% of isolates; according to CLSI M27 guidelines.

Appendix 6C. *Candida parapsilosis*

Antifungal	Breakpoint (ug/mL)	MIC Range (ug/mL)	MIC^{50} (ug/mL)	MIC^{90} (ug/mL)	%Susceptible
Fluconazole	8	<0.5->64	0.5	2	96
Voriconazole	1	<0.008->8	0.12	0.12	96
Caspofungin	8	<0.008->8	0.25	0.5	11
Micafungin	8	<0.008->8	0.5	1	96
Amphotericin B	1 (ECV)	<0.12->16	0.5	0.5	96
Flucytosine	>32	<0.006->64	<1	<1	96

MIC = minimum inhibitory concentration, ug/mL; MIC^{50} = MIC value at which growth was inhibited in 50% of isolates; MIC^{90} = MIC values at which growth was inhibited in 90% of isolates; according to CLSI M27 guidelines. **Caspofungin- 89% intermediate to Candida parapsilosis

Appendix 6D. *Candida tropicalis*

Antifungal	Breakpoint (ug/mL)	MIC Range (ug/mL)	MIC^{50} (ug/mL)	MIC^{90} (ug/mL)	%Susceptible
Fluconazole	8	<0.5->64	1	2	95
Voriconazole	0.12	<0.008->8	0.12	0.12	98
Caspofungin	1	<0.008->8	0.12	0.12	96
Micafungin	0.25	<0.008->8	0.06	0.12	94
Amphotericin B	2 (ECV)	<0.12->16	0.25	0.5	98
Flucytosine	>32	<0.006->64	<1	<1	98

MIC = minimum inhibitory concentration, ug/mL; MIC^{50} = MIC value at which growth was inhibited in 50% of isolates; MIC^{90} = MIC values at which growth was inhibited in 90% of isolates; according to CLSI M27 guidelines.

Appendix 7. Summary statistics of the antifungal drug resistance pattern of identified *Candida* species

	Fluconazole (mean ± SD)	Voriconazole (mean ± SD)	Caspofungin (mean ± SD)	Micafungin (mean ± SD)	Amphotericin B (mean ± SD)	Flucocystine (mean ± SD)
Candida albicans	2.78 ± 6.85	0.67 ± 1.83	0.79 ± 2.49	0.58 ± 2.09	0.95 ± 2.41	4.64 ± 15.0
Susceptible	69 (25.9%)	67 (25.2%)	79 (29.7%)	79 (29.7%)	82 (30.8%)	76 (28.6%)
Susceptible dose dependent	3 (1.1%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	15 (5.6%)	13 (4.9%)	8 (3.0%)	8 (3.0%)	6 (2.3%)	5 (1.9%)
Intermediate/susceptible increase exposure	0 (0.0%)	6 (2.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	3 (1.1%)
None	22 (8.3%)	21 (7.9%)	22 (8.3%)	22 (8.3%)	21 (7.9%)	25 (9.4%)
Candida parapsilosis	0.43 ± 1.11	0.47 ± 0.62	0.11 ± 0.16	0.31 ± 1.03	0.22 ± 0.54	1.30 ± 8.11
Susceptible	22 (8.3%)	67 (25.2%)	79 (29.7%)	79 (29.7%)	82 (30.8%)	76 (28.6%)
Susceptible dose dependent	0 (0.0%)	2 (0.8%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	1 (0.4%)	13 (4.9%)	8 (3.0%)	8 (3.0%)	6 (2.3%)	5 (1.9%)
Intermediate/susceptible increase exposure	0 (0.0%)	6 (2.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	3 (1.1%)
None	39 (14.7%)	21 (7.9%)	22 (8.3%)	22 (8.3%)	21 (7.9%)	25 (9.4%)
Candida glabrata	0.80 ± 3.57	0.50 ± 0.15	0.05 ± 0.07	0.13 ± 0.44	0.28 ± 0.33	0.35 ± 0.45
Susceptible	0 (0.0%)	1 (0.4%)	6 (2.3%)	8 (3.0%)	10 (3.8%)	8 (3.0%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	1 (0.4%)	0 (0.0%)	1 (0.4%)	2 (0.8%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	19 (7.1%)	18 (6.8%)	12 (4.5%)	10 (3.8%)	10 (3.8%)	12 (4.5%)
Candida tropicalis	2.12 ± 5.11	0.17 ± 0.53	0.38 ± 1.49	0.26 ± 1.18	0.57 ± 2.13	1.79 ± 8.55
Susceptible	40 (15.0%)	44 (16.5%)	43 (16.2%)	43 (16.2%)	44 (16.5%)	39 (14.7%)
Susceptible dose dependent	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	4 (1.5%)	1 (0.4%)	2 (0.8%)	2 (0.8%)	1 (0.4%)	1 (0.4%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	10 (3.8%)	10 (3.8%)	10 (3.8%)	10 (3.8%)	10 (3.8%)	15 (5.6%)
Candida krusei	0.00 ± 0.00	0.08 ± 0.69	0.25 ± 0.25	0.08 ± 0.07	1.00 ± 1.00	23.3 ± 40.4
Susceptible	0 (0.0%)	44 (16.5%)	43 (16.2%)	43 (16.2%)	1 (0.4%)	1 (0.4%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)
Resistant	0 (0.0%)	1 (0.4%)	2 (0.8%)	2 (0.8%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	3 (1.1%)	10 (3.8%)	10 (3.8%)	10 (3.8%)	0 (0.0%)	2 (0.8%)
Candida lipolytica	8.00 ± 0.00	0.25 ± 0.00	0.12 ± 0.00	0.06 ± 0.00	0.25 ± 0.00	0.99 ± 0.00
Susceptible	0 (0.0%)	0 (0.0%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Candida famata	0.50 ± 0.00	0.12 ± 0.00	0.12 ± 0.00	0.06 ± 0.00	0.50 ± 0.00	0.48 ± 0.00
Susceptible	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Candida lusitaniae	0.20 ± 0.45	0.24 ± 0.54	1.60 ± 3.58	0.01 ± 0.03	0.45 ± 0.37	0.80 ± 0.45
Susceptible	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	1 (0.4%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	4 (1.5%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	4 (1.5%)	1 (0.4%)	4 (1.5%)
None	4 (1.5%)	4 (1.5%)	4 (1.5%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Candida orthopsilosis	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Susceptible	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	5 (1.9%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	5 (1.9%)	5 (1.9%)	0 (0.0%)	5 (1.9%)	5 (1.9%)	5 (1.9%)
Candida auris	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Susceptible	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)
Candida dubliniensis	11.17 ± 18.04	0.08 ± 0.06	0.08 ± 0.06	0.04 ± 0.03	0.46 ± 0.48	0.65 ± 0.51
Susceptible	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)
Candida duobushaemulonii	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	1.00 ± 0.00
Susceptible	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)
Susceptible dose dependent	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Resistant	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.4%)	0 (0.0%)
Intermediate/susceptible increase exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
None	1 (0.4%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	0 (0.0%)	0 (0.0%)