

Susceptibility Pattern of Non-albicans *Candida* Isolates: Frozen or Mobile?

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Received: 11 Jun 2025/ Revised: 19 Aug 2025/ Accepted: 20 Oct 2025

ABSTRACT

Background: *Candida* sp. causes complicated infections with severe morbidity which warrant expensive treatment. Invasive candidiasis is caused by *Candida albicans* as well as non-albicans *Candida* sp. (NaC). Frequently isolated Non-albicans *Candida* are *Candida glabrata*, *C. tropicalis* and *C. parapsilosis*. This study aimed to isolate Non-albicans *Candida* sp. from clinical specimens and to determine their antifungal susceptibility pattern.

Methods: A Total of 615 clinical specimens were inoculated on Sabouraud's dextrose agar. Germ tube-negative isolates were tested with Hi-Chrome *Candida* agar. Antifungal susceptibility test was performed utilizing Mueller-Hinton agar with 2% glucose & 0.5µg/ml methylene blue by the disc diffusion method.

Results: A total of 60 non-duplicate Non-albicans *Candida* sp. were obtained from 615 clinical specimens. On Hi-chrome *Candida* agar *C. tropicalis* was the most frequently found one (58.33%) followed by *C. glabrata* (23.33%). 94.28% and 77.14% of *C. tropicalis* were susceptible to fluconazole and voriconazole, respectively.

Conclusion: Non-albicans *Candida* sp. had 30% resistance to fluconazole and 25% resistance to voriconazole. Multicentric studies with a larger number of isolates targeting antifungal resistance genes would shed more light on this global public health issue.

Key-words: Antifungal susceptibility, Hi-chrome *Candida* agar, Non-albicans *Candida* sp.

INTRODUCTION

The Global burden of fungal infections due to *Candida* species is increasing at a high rate. The incidence rates of invasive candidiasis are 25,000 to 29,000 per annum ^[1]. In India, it ranges from 0.24 to 34.30 patients per 1000 ICU admissions. The mortality owing to candidemia is 35-45% ^[2]. The spectrum varies from trivial intertriginous infection to fatal invasive candidiasis ^[2].

Concurrent complications such as infective endocarditis necessitate expensive treatment. They result in significant morbidity and mortality especially among high-risk patients. Healthcare-associated invasive infections due to *Candida* species are also increasing in a broad range ^[2].

At present, over 90-95% of invasive candidiasis is caused by species such as *Candida albicans* and non-albicans *Candida* sp. (NaC). Frequently isolated NaC are *Candida glabrata*, *C. tropicalis*, *C. parapsilosis* and *C. krusei*. ^[1,2] Less frequent NaC are *C. guilliermondii*, *C. dublinensis*, *C. haemulonii*, *C. famata*, *C. lusitaniae*, *C. rugosa*, *C. intermedia* and *C. kefyr*. ^[1,3] They are emerging opportunistic pathogens among vulnerable patients in critical care settings ^[3].

Virulence factors such as serine protease and biofilm formation enhance the pathogenicity of NaC strains.

How to cite this article

Subbiah G, Ganesan AT, Santhanakumarasamy PL. Susceptibility Pattern of Non-albicans *Candida* Isolates: Frozen or Mobile?. SSR Inst Int J Life Sci., 2025; 11(6): 8717-8724.



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Moreover, some species are inherently resistant to certain antifungals. Sub-optimal/inappropriate antifungal therapy may culminate in drug resistance and treatment failure.

In most of the developing countries, speciation of *Candida* isolates is often not performed owing to lack of sophistication in the laboratories [3]. Successful antifungal therapy requires properly performed speciation, antifungal susceptibility testing and high-quality reports. Hence, this study was undertaken to isolate *NaC* species from clinical specimens, speciate them with chrom-agar, and to determine their antifungal susceptibility pattern.

MATERIALS AND METHODS

Place of the study- This cross-sectional study was carried out in March and April 2024, in the department of Microbiology, Tirunelveli Medical College, Tirunelveli, Tamil Nadu, India.

Methodology- Non-duplicate isolates of *NaC* from all clinical specimens, such as blood, urine, pus and sputum from patients of all ages, genders and clinical diagnoses were studied. The sample size was 60 *NaC* isolates.

Inclusion criteria- Non-duplicate isolates of *NaC* from all clinical specimens such as blood, urine, pus and sputum from patients of all ages, gender and clinical diagnoses.

Exclusion criteria- *NaC* from environmental samples during the study period.

Data collection- From patients with *NaC* sp. culture positivity, socio-demographic details such as name, age, gender, OP/IP data, history of any significant relevant illness such as diabetes mellitus, hypertension, autoimmune disease and details of comorbid illnesses affecting liver, kidney & lungs were recorded using a structured proforma. Details of the present clinical diagnosis, treatment and other relevant investigations results were recorded. Due emphasize was provided for details such as malignancy, renal transplantation, recent major surgery, broad-spectrum antimicrobial therapy, fluconazole therapy, oral contraceptive intake, prolonged duration of central venous line/indwelling urinary catheter (>5 days), prolonged ICU stay (>14 days) and immunosuppressive therapy. Voluntary written informed consent was obtained from all the participants. Confidentiality was maintained.

Yeast isolates were obtained after processing the clinical specimens. By germ tube test, *C. albicans* isolates were identified and excluded. Germ tube negative, *NaC* were sub-cultured on Hi-chrome *Candida* agar for species identification. Antifungal susceptibility test was done utilizing Mueller-Hinton agar with 2% glucose & 0.5µg/ml methylene blue by disc diffusion method [4-6] for yeasts as per CLSI guidelines. Standard operating procedures were followed to ensure quality.

Statistical Analysis- The data obtained were entered in MS Excel and the results were analysed using SPSS. Descriptive statistics such as frequency and percentage of *NaC* were calculated. Chi-square test and Student t test were performed to find p-values for categorical and continuous variables, respectively. The association between drug-resistant *NaC* and various risk factors was studied.

Ethical Approval- Institutional ethics committee approval was obtained (20232858 /19.01.2024).

RESULTS

Out of 615 clinical specimens from patients with various clinical fungal infections, collected during the study period, 112 (18.21%) were *Candida* species. Among them, 52 (46.4%) were *Candida albicans* and 60 (53.5%) *NaC* sp. Females comprised 51.7% and the rest (48.3%) were males. The ages of the patients ranged from 1 to 93 years. The mean age of them was 53 years. The majority of the *NaC* were from 51-60 years age group. Overall, 83.3% were inpatients, whereas 16.6% were outpatients (Table 1).

NaC yield was predominantly from urine specimens (36/60, 60%) followed by sputum (10/60, 16.6%), while it was least from CSF specimens (1, 1.66%). On Hi-Chrome *Candida* agar *C. tropicalis* was the most frequently found one (35/60, 58.33%) followed by *C. glabrata* (23.33%). Sixty-six percent of *C. tropicalis* were from urine samples, followed by sputum (14.28%) and pus (11.42%). Similarly, the majority (36%) of *C. glabrata* were from urine specimens. Of the 6 *C. krusei* isolates 4 (67%) were from urine (Table 2).

Table 1: Distribution of *NaC* study group n=60

Age group in years	Male no. (%)	Female no. (%)	Total no. (%)	Outpatient no. (%)	Inpatient no. (%)	Total no. (%)
1-10	02 (3.3)	0	02 (3.3)	0	02 (3.3)	02 (3.3)
11-20	0	02 (3.3)	02 (3.3)	0	02 (3.3)	02 (3.3)
21-30	0	04 (6.6)	04 (6.6)	0	04 (6.6)	04 (6.6)
31-40	03 (4.9)	01 (1.7)	04 (6.6)	01 (1.7)	03 (4.9)	04 (6.6)
41-50	03 (4.9)	05 (8.3)	08 (13.3)	02 (3.3)	06 (10)	08 (13.3)
51-60	08 (13.2)	10 (16.6)	18 (30)	04 (6.6)	14 (23.3)	18 (30)
61-70	06 (10)	06 (10)	12 (20)	02 (3.3)	10 (16.6)	12 (20)
71-80	06 (10)	03 (4.9)	09 (14.9)	01 (1.7)	08 (13.3)	09 (13.3)
81-90	0	0	0	0	0	0
91-100	01 (1.7)	0	01 (1.7)	0	01 (1.7)	01 (1.7)
Total	29 (48.3)	31(51.7)	60 (100)	10 (16.6)	50 (83.3)	-

Table 2: Specimen-wise isolation of *NaC* (n=60)

<i>Non-albicans Candida sp.</i>	Urine no. (%)	Sputum no. (%)	Pus no. (%)	Blood no. (%)	BAL no. (%)	CSF no. (%)	Total no. (%)
<i>C. tropicalis</i>	23 (66)	05 (14.3)	04 (11.4)	02 (5.7)	01 (2.9)	0	35 (58.3)
<i>C. glabrata</i>	05 (36)	04 (29)	02 (14)	01 (7)	01 (7)	01 (7)	14 (23.3)
<i>C. krusei</i>	04 (67)	0	01 (17)	01 (17)	0	0	06 (10)
<i>C. parapsilosis</i>	03 (100)	0	0	0	0	0	03 (05)
<i>C. kefyr</i>	01 (50)	01 (50)	0	0	0	0	02 (3.3)
Total	36 (60)	10 (16.66)	07 (12)	04 (6.66)	02 (3.33)	01(1.66)	60

Among the study subjects, a few risk factors favouring *NaC* were found. 1. Chronic antimicrobial therapy 83.33%, 2. Diabetes Mellitus 66.66%, 3. Neutropenia 41.66%, 4. Prolonged bladder catheterization 36.66%, 5. Chronic renal disease 23.33%, 6. Renal transplantation 1.66%.

Regarding the overall antifungal susceptibility pattern, voriconazole was found to have with highest susceptibility rate (75%) followed by fluconazole (70%). In contrast, only 45% *NaC* were susceptible to amphotericin B. The isolates had the highest resistance to itraconazole (53.3%) (Table 3).

Table 3: Overall antifungal susceptibility pattern of the *NaC* isolates (n=60)

Antifungal (µg)	Fluconazole (25)	Itraconazole (10)	Voriconazole (01)	Amphotericin B (50)	Caspofungin (05)	Micafungin (2.5)
Susceptible	42 (70%)	28 (46.66%)	45 (75%)	27 (45%)	33 (55%)	29 (48.33%)
SDD	0	0	0	03 (05%)	01 (01.66%)	03 (05%)
Resistant	18 (30%)	32 (53.33%)	15 (25%)	30 (50%)	26 (43.33%)	28 (46.66%)
Total	60	60	60	60	60	60

The antifungal susceptibility pattern of *C. tropicalis* (35/60) isolates was studied. Most of them (33/35, 94.28%) were susceptible to fluconazole, while 77.14% were susceptible to voriconazole. Fifty-seven percent of

C. tropicalis were susceptible to amphotericin B, whereas it was 54% for caspofungin and micafungin. Only 48.5% were susceptible to itraconazole (Table 4).

Table 4: Antifungal susceptibility pattern of *C. tropicalis* isolates n=35

Antifungal drug (µg)	Susceptible frequency (%)	Resistant frequency (%)
Fluconazole (25)	33 (94.28)	02 (05.71)
Voriconazole (01)	27 (77.14)	08 (22.85)
Amphotericin B (50)	20 (57.14)	15 (42.85)
Caspofungin (05)	19 (54.28)	16 (45.71)
Micafungin (2.5)	19 (54.28)	16 (45.71)
Itraconazole (10)	17 (48.57)	18 (51.42)

Seventy-one percent of *C. glabrata* (10, 71.42%) were susceptible to caspofungin as well as to voriconazole. Eight of *C. glabrata* were found to be susceptible to micafungin (57%), and 4 (28.57%) were susceptible to itraconazole. Only one (7.1%) isolate was susceptible to fluconazole. The highest resistance (92.85%) was found with fluconazole.

DISCUSSION

As infections with *NaC* are increasing, rapid identification of them is crucial. This study determined the species distribution and antifungal susceptibility pattern of them so that early & appropriate antifungal therapy can be administered.

In the present study, *C. albicans* isolation was 46.4% which was comparable to the study of Bhattacharjee (48.57%).^[7] and Costa de Oliveira *et al.*^[8,9] (43%). This could possibly be explained by the changing host factors determining the distribution of *C. albicans* that happens in the modern era, owing to the co-existing predisposing factors for fungal infections.

The isolation rate of *NaC* sp. in this study was 53.57% which agreed with the results of Bhattacharjee (51.42%)^[7] and Resende *et al.* (49%)^[10]. However, Negri *et al.*, Kothalawala *et al.*, Singh *et al.*, and Shukla *et al.* found the same 61%, 69%, 71.7% and 75% respectively^[2,11-13]. This could possibly be explained by the geographical, spatial and temporal variations. The higher rate of *NaC* could be explained by improved laboratory diagnostic tools for *NaC*, prolonged use of antifungals and cytotoxic therapy.

This study found that 51.7% of patients with *NaC* were females, which was like the findings of Bhattacharjee (52.77%) and Jayalakshmi *et al.* (54.28%)^[7,13]. The male: female ratio in the present study was 1:1.06. The gender difference could be attributed to hormonal influences such as oestrogen. Higher oestrogen with greater glycogen serves as a good carbon source and disrupts the relationship between the *NaC* sp. and the hosts^[14].

Predominant age group associated with *NaC* isolates was 51- 60 years (30%) in the current study, which was in concordance with the finding by Sabhapandit *et al.* (29%)^[15] and Priyavalli *et al.*^[16]. Bhattacharjee too found that infections with *NaC* were common among 51-60 years group^[7]. This might be due to senile immunodeficiency and health-seeking behaviour of the elderly at this setting, as this is a tertiary care public hospital. The highest age at which the *NaC* was detected in this study was 93 years, which correlates with that of Urvashi *et al.* (92 years)^[17].

This study obtained 60% isolates of *NaC* from urine samples, similar to Urvashi *et al.* (60.71%) and Negri *et al.* (55.81%)^[17,11]. But Priyavalli *et al.*^[16] isolated 85% of the same from urine samples. The highest number of *NaC* (67.5%) was obtained from urine samples by Singh M *et al.*, also^[18]. This could possibly be explained by the fact that the urinary system is the preferred niche of *NaC* colonization owing to the expression of virulence factors like adhesins and biofilms by *NaC*. Moreover, prolonged urinary bladder catheterization, which was found in 36.66% of this study group, favour urinary tract infection

and hence the highest level of isolation was from urine samples.

Many of the authors, including Edula *et al.*, Jayalakshmi *et al.* and Priyavalli *et al.* reported *C. tropicalis* as the commonest isolate of *NaC*, which was like the present study ^[4,13,16]. *C. tropicalis* isolation was 58.3% in this study, while it was 61.33% in the study by Shukla *et al.* ^[2], 62% by Capoor *et al.*, ^[19] 62.12% by Agarwal *et al.* ^[20] 62.96% by Edula *et al.* ^[4] and 54% by Adhikary *et al.* ^[21]. Chakrabarti *et al.* ^[22] and Bhattacharjee ^[7] also found high *C. tropicalis* isolation among their samples. This could be explained by the irrational use of fluconazole for common fungal lesions, which exerts selection pressure among the *Candida* sp. and shifts towards drug-resistant *C. tropicalis*. Long admission in IMCU, neutropenia and total parenteral nutrition are also risk factors for the same ^[23].

This study found *C. glabrata* as the second most common isolate from the samples, which was in concordance with Deorukhkar *et al.* ^[23]. The rate of isolation was 23.33% which was comparable with that of Badiie *et al.* (25.80%) and Jayalakshmi *et al.* (27.77%). ^[24,13]

In the current study, 83.33% of the study group had chronic antimicrobial therapy. This was comparable to Xess *et al.* (71%) and Kothalawala *et al.* (73.7%). ^[25,3] Capoor *et al.* had found the rate as 100% ^[19]. In addition, Chakrabarthi *et al.* also observed a higher rate of *NaC* infections in those patients on antimicrobials for >7days and receiving three or more antimicrobials ^[22]. Administration of broad-spectrum antibiotics suppresses the endogenous microflora, permitting fungal overgrowth and impairment of mucosal immunity.

Sixty-six percent of the present study group had diabetes mellitus (DM), which was comparable to the finding of Shukla *et al.* (46%) ^[2]. Fungal infections in diabetic patients might be due to uncontrolled hyperglycemia, which results in immune dysfunction, such as defective oxidative burst, thereby increasing the susceptibility to infections.

Neutropenia was found in 42% of this study group, while the same was 87% as studied by Capoor *et al.* ^[19]. Chronic renal disease was found in 23% in the present study, which was like Kothalawala *et al.* (29.7%), Capoor *et al.* (17.7%) and Shukla *et al.* (11%) studies ^[2,3,19].

Overall antifungal susceptibility pattern analysis revealed increased rates of resistance among *NaC* isolates. This

was comparable with the observation of many other authors. Gandhi *et al.* ^[26] had noted 25%, 23%, 36% and 20% resistance to fluconazole, voriconazole, itraconazole and miconazole, respectively, among blood culture isolates of *Candida* sp. in Ahmedabad in 2012. Jayachandran *et al.* found higher (35%) resistance to fluconazole and lower (29%) resistance to itraconazole among *Candida* isolates in 2018 at Chennai ^[27]. On the other hand, Kothalawala *et al.* and Capoor *et al.* documented 63% and 100% overall resistance to azoles, respectively, among *NaC*. ^[3,19] Discordantly, Tankhiwale *et al.* ^[28] found only 14.28% resistance to fluconazole among *Candida* isolates. Priyavalli *et al.* found 6.52% and 26.08% resistance rates to caspofungin and voriconazole, respectively, among *NaC* ^[16].

Susceptibility to voriconazole among *NaC* was highest (75%) in the present study, which was comparable with Urvashi *et al.* (86%), Shukla *et al.* (82.6%) and Gandhi *et al.* (77%) studies ^[17,2,26]. Discordant to this, Edula *et al.* ^[4] and Kumar *et al.* ^[29] found the susceptibility as 92.59% and 100% respectively. This might indicate that there are no enzyme/target modifications among their study isolates.

Fluconazole susceptibility in this study was 70% and it was in good conjunction with the results of Kumar D *et al.* (72.7%), Negri *et al.* (73%), Adhikary *et al.* (75%), and Verma *et al.* (79%) ^[29,11,21,30].

Resistance to fluconazole was 30% in this study and it was 30.8% in the study by Giri *et al.* ^[5]. It was comparable with the results of Jayalakshmi *et al.* (34.2%) and Kothari *et al.* (36%) ^[13,31]. On the other hand, it was reported as 17.2% by Kumar *et al.* in 2005 ^[32]. This implies about 2-fold rise in fluconazole resistance among *NaC* during recent times, owing to molecular mechanisms of drug resistance in them, a rise in susceptible hosts, and favourable environment.

There was 50% overall susceptibility to Amphotericin B in this study, which agreed with Urvashi *et al.* (40%) ^[17]. But Bhattacharjee found amphotericin B resistance as 30.5% which was remarkably lower ^[7]. Kumar *et al.* reported 0% resistance to amphotericin B. ^[29] The varying levels of resistance could be attributed to heterogeneity in study population dynamics. Mechanism of resistance to amphotericin B is a reduction in the ergosterol content of the plasma membrane of the yeast cell following mutation in ERG 2,3,5,6,11 genes ^[33]. This leads to a lower affinity of amphotericin B to the plasma

membrane. Reduced rate of intracellular activation of drug also results in resistance. This occurs commonly among immunocompromised patients who have antifungal prophylaxis with azoles and amphotericin B [20].

Overall susceptibility of *NaC* to caspofungin in the present study was 56.6% which was significantly lower than the study by Shukla *et al* (89.3%, 2016-17) [2].

This study found 94.28% susceptibility rate to fluconazole among *C. tropicalis* isolates, which agreed with that of Tankhiwale *et al.* (96.29%) [28].

In the present study, susceptibility to voriconazole among *C. tropicalis* was 77.14% which was higher than Kothalawala *et al.* (50%) and lower than the observation of Edula *et al.* (94.11%) [3,4].

Though echinocandin drugs such as caspofungin and micafungin were highly effective, they are very expensive. Caspofungin susceptibility among *C. tropicalis* isolates of the present study was 54.28% which was lower than the result by Shukla *et al.* (89.1%) [2]. As with caspofungin, micafungin susceptibility was 54.28% in this study, lower than that reported by Shukla *et al.* (89.1%) [2] for *C. tropicalis* isolates. This also implies the emerging echinocandin resistance among the *NaC* of this setting.

The resistance to fluconazole was 92.85% among *C. glabrata* isolates of this study, which was comparable to that of Edula *et al.* (75%), Shukla *et al.* (67%). [4,2]. This might possibly happen due to high-level efflux pumps because of mutations in CDR1 and MDR1, PDR1 genes, changes in transport proteins, ploidy changes in chromosomes and deficient intracellular uptake of the drug [34,35].

Multi-drug resistance (MDR) was found in 51.66% of *NaC* of the present study, which was in congruence with Kothalawala *et al.* (50%) and Colombo *et al.* (>30%) [3,35]. The p-value for infection with multi-drug resistant *NaC* and gender was .037 and the association was statistically significant. This was comparable to Mohamed *et al.* [36], who reported that the rate of MDR among *NaC* isolates from females was 28.75%, which was higher than that in males (15%). Antifungal prophylaxis was commonly used in patients with specific risk factors like malignancies, neutropenia and transplantation. But overuse of them without appropriate monitoring may lead to MDR [20,32].

CONCLUSIONS

Non-albicans Candida sp. had 30% resistance to fluconazole and 25% resistance to voriconazole. Resistance to caspofungin was 43%. Routine speciation and antifungal susceptibility testing of *Candida* isolates would aid in choosing appropriate targeted antifungal therapy. Effective antifungal stewardship combined with the establishment of up-to-date local antifungal treatment guidelines is needed at the hour.

Strengths of the study

- ❖ Isolation and relatively rapid speciation of common *non-albicans Candida* sp. employing conventional and Hi Chrome differential media from various clinical samples.
- ❖ Antifungal susceptibility testing of the isolates using the disk diffusion method as per CLSI guidelines.

LIMITATIONS

- ❖ Single-centre study, limiting external validity.
- ❖ Small sample size, reducing statistical power.
- ❖ Short-term, cross-sectional design without patient follow-up.
- ❖ Lack of outcome assessment for patients undergoing antifungal therapy.
- ❖ Absence of MIC-based testing (microbroth dilution, agar dilution, E-test) due to cost constraints.
- ❖ No molecular characterization of resistant isolates (advanced PCR, gene sequencing).
- ❖ Unavailability of automated yeast identification and susceptibility systems (VITEK-2), limiting diagnostic detail.

ACKNOWLEDGEMENT

The authors are thankful to the Tamil Nadu State Research Committee, King Institute of Preventive Medicine and Research, Chennai, for financial support.

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REFERENCES

- [1] Husni R, Bou Zerdan M, Samaha N, Helou M, Mahfouz Y, et al. Characterization and susceptibility of non-albicans *Candida* isolated from various clinical specimens in Lebanese hospitals. *Front Public Health*, 2023; 11 (10): 11–55.
- [2] Shukla R, Gopal Reddy S, Kumar Bilolika A. A study of *Candida albicans* and non-albicans *Candida* species isolated from various clinical samples and their antifungal susceptibility pattern. *J Med Sci Res.*, 2020; 8 (1): 01–11.
- [3] Kothalawala M, Jayaweera JAAS, Arunan S, Jayathilake A. The emergence of non-albicans candidemia and evaluation of HiChrome *Candida* agar and VITEK2 YST platform. *BMC Microbiol.*, 2019; 19 (1): 36.
- [4] Edula AR. Antifungal susceptibility of clinically significant *Candida* species by disk diffusion method. *IP Int J Med Microbiol Trop Dis.*, 2021; 7 (2): 77–80.
- [5] Giri S, Kindo AJ. Evaluation of antifungal susceptibility testing in *Candida* isolates by Candifast and disk-diffusion method. *Indian J Pathol Microbiol.*, 2014; 57: 595–97.
- [6] CLSI. Method for antifungal disc diffusion susceptibility testing of yeasts; approved guideline, 2nd ed. CLSI M44-A2. Clinical and Laboratory Standards Institute, 2009.
- [7] Bhattacharjee P. Epidemiology and antifungal susceptibility of *Candida* species in a tertiary care hospital, Kolkata, India. *Curr Med Mycol.*, 2016; 2 (2): 20–27.
- [8] Costa-de-Oliveira S, Pina-Vaz C, Mendonça D, et al. A Portuguese epidemiological survey of fungaemia. *Eur J Clin Microbiol Infect Dis.*, 2008; 27: 65–74.
- [9] Falagas ME, Roussos N, Vardakas KZ. Relative frequency of albicans and non-albicans *Candida* spp among candidemia isolates worldwide. *Int J Infect Dis.*, 2010; 14 (11): e54–66.
- [10] Resende JCP, De Resende MA, Saliba JL. Prevalence of *Candida* spp. in hospitalized patients and risk factors. *Mycoses*, 2002; 45 (7–8): 06–12.
- [11] Negri M, Henriques M, Svidzinski TI, Paula CR, Oliveira R. Correlation between E-test, disk diffusion and microdilution for *Candida* susceptibility testing. *J Clin Lab Anal.*, 2009; 23 (5): 24–30.
- [12] Singh T, Kashyap AK, Ahluwalia G, Chinna D, Sidhu SS. Epidemiology of fungal infections in critical care. *J Clin Sci Res.*, 2014; 3 (1): 14–25.
- [13] Jayalakshmi L, Ratna Kumari G, Samson SH. Isolation, speciation and antifungal susceptibility of *Candida* in a tertiary hospital. *Sch J App Med Sci.*, 2014; 2 (6E): 93–98.
- [14] Babic M, Hukic M. *Candida albicans* and non-albicans species causing vaginitis in women. *Bosn J Basic Med Sci.*, 2010; 10 (1): 89–97.
- [15] Sabhapandit D, Lyngdoh WV, Bora I, Prasad A, Debnath K, et al. Prevalence of non-albicans candidemia in Northeast India. *Int J Med Sci Public Health*, 2017; 6 (11): 20–25.
- [16] Priyavalli S, Sudha Rani V. Speciation, biofilm production and antifungal susceptibility of non-albicans *Candida*. *IOSR J Dent Med Sci.*, 2023; 22 (1): 34–42.
- [17] Chongtham U, Athokpam D, Singh R. Isolation and antifungal susceptibility of *Candida* species in Manipur, India. *J Clin Diagn Res.*, 2022; 16: 86.
- [18] Singh M, Chakraborty A. Antifungal drug resistance among *Candida* isolates from a tertiary centre at Allahabad. *J Antimicrob Agents*, 2017; 3 (4): 50.
- [19] Capoor M, Nair D, Deb M, Verma P, Srivastava L, et al. Emergence of non-albicans *Candida* and antifungal resistance. *Jpn J Infect Dis.*, 2006; 58 (6): 44–48.
- [20] Agarwal S, Manchanda V, Verma N, Bhalla P. Yeast identification in routine microbiology lab and relevance. *Indian J Med Microbiol.*, 2011; 29 (2): 72–77.
- [21] Adhikary R, Joshi S. Species distribution and antifungal susceptibility of candidemia in southern India. *Indian J Med Microbiol.*, 2011; 29 (3): 09–11.
- [22] Chakrabarti A, Chatterjee SS, Rao KL, Zameer MM, Shivaprakash MR, et al. Experience with fungaemia and azole resistance. *Scand J Infect Dis.*, 2009; 41 (4): 75–84.



- [23]Deorukhkar S, Saini S. Non-albicans Candida: epidemiology and resistance. *Pravara Med Rev.* 2015; 7: 07–15.
- [24]Badiie P, Alborzi A. Susceptibility of clinical Candida isolates by E-test in Iran. *Iran J Microbiol.*, 2011; 3 (4): 83–88.
- [25]Xess I, Jain N, Hasan F, Mandal P, Banerjee U. Epidemiology of candidemia: 5-year study. *Infect.*, 2007; 35 (4): 56–59.
- [26]Gandhi V, Patel M. Prevalence and antifungal susceptibility of Candida from blood culture. *Int J Curr Microbiol App Sci.*, 2017; 6 (6): 84–92.
- [27]Jayachandran AL, Katragadda R, Ravinder T, Vajravelu L, Manorajan L, et al. Antifungal susceptibility of Candida: disc diffusion vs broth dilution. *J Microbiol Infect Dis.*, 2018; 8 (3): 97–102.
- [28]Tankhiwale S, Gajbhiye S, Powar R. Fluconazole susceptibility of Candida: disc diffusion vs agar dilution. *J Evol Med Dent Sci.*, 2012; 1 (4): 27–31.
- [29]Kumar D, Bhattacharyya S, Gupta P, Banerjee G, Singh M. Disc diffusion vs E-test vs broth dilution for Candida susceptibility. *J Clin Diagn Res.*, 2015; 9 (11): 01–04.
- [30]Verma R, Pradhan DB, Hasan Z, Singh H, Jain AK, et al. Distribution and antifungal resistance of Candida species: systematic review. *Med Mycol.* 2021; 59 (12): 65–65.
- [31]Kothari A, Sagar V. Epidemiology of Candida bloodstream infections in India. *Indian J Med Microbiol.*, 2009; 27 (2): 71–72.
- [32]Kumar CP, Sundararajan T, Menon T, Venkatesikal M. Candidosis in children with onco-hematological diseases. *Jpn J Infect Dis.*, 2005; 58 (4): 18–21.
- [33]Arendrup MC, Patterson TF. Multidrug-resistant Candida: mechanisms and treatment. *J Infect Dis.*, 2017; 216 (3): S45–51.
- [34]Berkow EL, Lockhart SR. Fluconazole resistance in Candida: perspective. *Infect Drug Resist.*, 2017; 31: 37–45.
- [35]Colombo AL, De Almeida JN Jr, Guinea J. Emerging multidrug-resistant Candida species. *Curr Opin Infect Dis.*, 2017; 30 (6): 28–38.
- [36]Osman MA, Suliman MM, Abdelrahman HM, Fatahalrahman AI. Antifungal resistance and ERG11 mutations in Sudan. *F1000 Res.*, 2020; 26 (9): 50.

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