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Original Research Article

Candida under the lens: species diversity and antifungal susceptibility trends

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ABSTRACT

Background: Non-*albicans Candida* (NAC) species are increasingly responsible for mucosal and invasive candidiasis and often exhibit reduced susceptibility to antifungal agents. This study assessed the species distribution of clinical *Candida* isolates and their antifungal susceptibility profiles.
Methods: In a prospective study conducted from 2015 to 2018 at a tertiary care center in Varanasi, India, 711 *Candida* isolates obtained from blood, urine, aspirates, and mucosal swabs were analyzed. Species identification was performed using PCR-restriction fragment length polymorphism (PCR-RFLP); unresolved isolates were further characterized by MALDI-TOF mass spectrometry and sequencing.
Results: Antifungal susceptibility to fluconazole, voriconazole, caspofungin, and amphotericin B was determined using the CLSI broth microdilution method. NAC species accounted for 63% of isolates, with *Candida tropicalis* being the most prevalent. PCR-RFLP identified 92.5% of isolates, while MALDI-TOF MS and sequencing enabled identification of rare species, including *C. mesorugosa*, *C. africana*, and *Pichia farinosa*. Fluconazole showed high activity against *C. albicans* (94.3%) and *C. glabrata* (100%) but reduced activity against *C. tropicalis* (77.9%). Voriconazole demonstrated broad activity across species, and caspofungin exhibited low MICs for most isolates, although resistance was observed in 10.6% of *C. glabrata*. Amphotericin B remained active against most species, with elevated MICs noted in *C. lusitaniae*.
Conclusions: These findings underscore the importance of accurate species-level identification and routine antifungal susceptibility testing to optimize therapy for Candida infections.

Keywords: Broth microdilution method, *C. albicans*, Fluconazole, Non-*albicans Candida*, Resistance

INTRODUCTION

Candida infections are on the rise globally, ranging from mild mucosal conditions such as oropharyngeal and vulvovaginal candidiasis to severe, life-threatening forms including candidemia and invasive candidiasis.[1] The genus *Candida* includes nearly 200 species, around 30 of which are implicated in various types of Candida infections. While *Candida albicans* continues to be the most prevalent, the incidence of non-albicans Candida (NAC) species- such as *C. tropicalis*, *C. glabrata*, *C. krusei*, *C. parapsilosis*, *C. lusitaniae*, and the multidrug-resistant *C. auris*- have risen significantly.[2]

The distribution of *Candida* species is influenced by factors such as geographic variation, patient-specific predisposing conditions, and local hospital epidemiology. Given the evolving patterns of *Candida* isolation from diverse clinical specimens, it is crucial to continuously evaluate the prevalence and species profiles in suspected cases of candidiasis.[3]

Majority laboratories still rely conventional approaches for the identification of *Candida* spp. which are not reliable due to overlapping morphologies and metabolism of closely related *Candida* spp. Alternative strategies like polymerase chain reaction (PCR) and matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) have been adapted to identify yeast species in clinical settings with better accuracy and less time when compared to conventional methods.[4,5] The widespread use of antifungal agents including their prophylactic use has led to the emergence of species with reduced susceptibility or resistance. Consequently, antifungal susceptibility testing has become paramount in the effective management of patients with *Candida* infections.[6] Hence, this study was carried out with the following objectives: a) to identify *Candida* spp. obtained from various clinical specimens using PCR, MALDI-TOF MS and sequencing, b) to observe the antibiogram of *Candida* spp. obtained from different clinical sources.

In many settings, data on the prevalence and antifungal susceptibility of *Candida* spp. across diverse clinical specimens remain limited. This study addressed an important knowledge gap by providing up-to-date, species-level epidemiological data on *Candida* isolates from a wide range of clinical specimens within a local healthcare setting. This study encompassed both *Candida albicans* and NAC species, enabling a more comprehensive understanding of this pathogen. Furthermore, by coupling species identification with antifungal susceptibility profiling, the study highlighted the evolving epidemiology and provides actionable insights for clinicians in tailoring antifungal therapy.

METHODS

The present prospective study was conducted in the department of microbiology, Institute of Medical Sciences (IMS), Banaras Hindu University (BHU), Varanasi, and its associated tertiary care hospital over a three-year period from June 2015 to May 2018. The study received approval from the Institutional Ethics Committee (ECR/526/Inst/UP/2014). All the *Candida* species isolated from the patient samples received from both inpatient and outpatient departments of IMS, BHU and the associated tertiary care hospital, Varanasi in the duration from June 2015 to May 2018 were included in the present study. Total 711 isolates from different clinical sources were isolated. Duplicate isolates from the same patient during the same infectious episode, mixed cultures that could not be purified, non-viable isolates, and isolates with incomplete laboratory records were excluded.

Identification of Candida spp.

Fungal growth on BHI, blood agar and SDA were processed using standard mycological procedures. *Candida* spp. were identified based on PCR- restriction fragment length polymorphism (PCR- RFLP) using MspI restriction enzyme. Isolates that could not be identified by PCR-RFLP were analysed using MALDI-TOF MS, and those remaining unidentified were further subjected to sequencing.

Identification by PCR-RFLP

All the isolated strains were further identified by PCR-RFLP using *Moraxella* species restriction endonuclease I (MspI) restriction enzyme. The PCR-RFLP identification was done according to the method described previously with little modifications. Genomic DNA from *Candida* spp. was extracted using the CTAB method.[7,8] Briefly, yeast cells were lysed by grinding with glass beads, followed by incubation with CTAB extraction buffer at 65°C for 30 min. DNA was purified using phenol: chloroform:isoamyl alcohol (25:24:1), precipitated with chilled isopropanol, and dissolved in TE buffer (pH 8.6). DNA quantity and quality were assessed by nanodrop spectrophotometry. The ITS1-5.8S-ITS2 region was amplified using universal fungal primers ITS1 (5′-TCC GTA GGT GAA CCT GCG G-3′) and ITS4 (5′-TCC TCC GCT TAT TGA TAT GC-3′) in a 25 µl PCR reaction. Cycling conditions included initial denaturation at 95°C for 5 minutes, followed by 30 cycles of 95°C for 30 seconds, 55°C for 30 seconds, 72°C for 1 minute, and a final extension at 72°C for 5 minutes. Amplicons were visualized on 1% agarose gel under UV illumination. For RFLP analysis, 5 µl of PCR product was digested with MspI in a 15 µl reaction (1 µl enzyme, 2 µl NEB buffer) at 37°C for 2 hours, and fragments were resolved on 2% agarose gel for interpretation.[7]

Identification by MALDI TOF MS

The isolates, not identified by PCR-RFLP were further identified by MALDI TOF mass spectrometry at PGIMER, Chandigarh. For this, the on-plate formic acid extraction method was used.[9] Briefly, a loop-full of fresh Candida colonies (2-3 colonies) were emulsified in 1 ml sterile double-distilled water in a microcentrifuge tube and vortexed vigorously. The suspension was then centrifuged at 13,000 rpm for 2 minutes, and the pellet was collected. This step was repeated two times and the pellet was re-suspended in 50 µl sterile double distilled water. One microliter of this suspension was spotted on the MALDI target plate and air-dried. Subsequently, the spot was overlaid with 0.5 µl formic acid (98%) and again left for air drying. Finally, 0.8 µl of matrix solution (100 µl of matrix solution containing: 50 µl acetonitrile, 2.5 µl trifluoroacetic acid, 1 mg α-cyano-4-hydroxycinnamic acid and 47.5 µl sterile double dis- tilled water) was added on each spot and air dried. MALDI-TOF MS analysis was performed on MALDI Microflex LT mass spectrometer (Bruker Daltonik GmbH, Bremen, Germany). The results were analyzed on the basis of score values recommended by the manufacturer that was as: ≥2 log score for species identification; 1.7-1.99 for genus identification; and <1.7 as unreliable. Confirmed isolates were stored in glycerol broth at −20°C for further analysis.

Antifungal susceptibility testing

Antifungal susceptibility testing of amphotericin B, caspofungin, fluconazole and voriconazole against n=401 isolates obtained from critically ill patients were executed by broth microdilution method as described in the CLSI M-27 A3 guidelines.[10] The minimum inhibitory concentration (MIC) of antifungal agents was determined using the broth microdilution method in round-bottom 96-well microtiter plates. Each well contained 100 µl of serially diluted antifungal drug solutions prepared in the appropriate growth medium. Standardized fungal inocula were added, and the plates were incubated at 35°C for 24 hours. Following incubation, wells were examined visually to assess fungal growth. The MIC was defined as the lowest drug concentration that completely inhibited visible growth for amphotericin B, while for azoles and caspofungin, it corresponded to the concentration causing a marked reduction in growth (≥50 %) relative to the drug-free control.[10]

RESULTS

A total of 711 *Candida* species were isolated in the 3 years duration and included for the identification by PCR RFLP assay. Distribution of isolates sample wise is shown in Figure 1. The distribution profile of *Candida* species identified by PCR RFLP is given in Table 1.

Table 1: Distribution profile of *Candida* species after identification with PCR RFLP.

Candida species	No. of isolates	Percentage
C. albicans	263	37.0
C. tropicalis	238	33.4
C. glabrata	82	11.5
C. krusei	32	4.5
C. parapsilosis	30	4.2
C. kefyr	5	0.7
C. famata	3	0.4
C. lusitaniae	3	0.4
C. rugosa	2	0.3
Unidentified	53	7.5
Total	711	100

A total of fifty-three (7.5%) out of 711 *Candida* isolates, remained unidentified by PCR RFLP assay, which were subjected to MALDI TOF MS at PGIMER, Chandigarh, India. It identified 47/53 (88.68%) isolates and 11.32% (6/53) remained unidentified which were finally confirmed by Sangers Sequencing at PGIMER Chandigarh, India. The distribution profile of these Candida species identified by MALDI TOF MS is given in Figure 2.

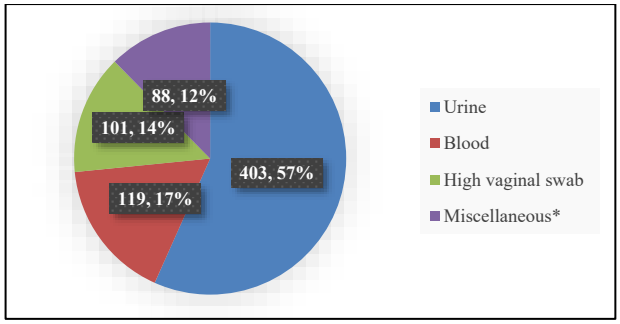

Figure 1: Distribution of *Candida* isolates according to clinical sources.

*88 (misc -pus, BAL, skin scraping, oral and ear swabs, endotracheal tubes, umbilical catheter tips, CSF).

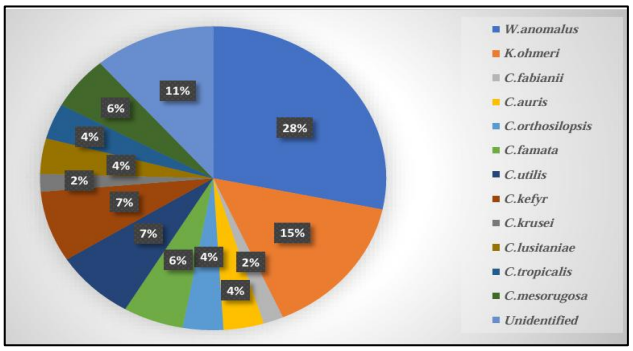

Figure 2: Distribution of PCR-RFLP-unidentified *Candida* isolates characterized by MALDI-TOF MS (n=53).

Table 2: Overall distribution profile of *Candida* species after identification with PCR RFLP, MALDI-TOF and sequencing.

Candida species	No of isolates	Percentage
C. albicans	263	37.0
C. tropicalis	240	33.8
C. glabrata	82	11.5
C. krusei	33	4.6
C. parapsilosis	30	4.2
C. kefyr	9	1.3
C. famata	6	0.8
C. lusitaniae	5	0.7
C. rugosa	5	0.7
C. mesorugosa*	4	0.6
W. anomalus	15	2.1
K. ohmeri	8	1.1
C. utilis	4	0.6
C. auris	2	0.3
C. orthosilopsis	2	0.3
C. africana*	1	0.1
C. fabianii	1	0.1
P. farinosa*	1	0.1
Total	711	100

*These isolates were identified using sequencing.

Table 3: AFST of *Candida* species to voriconazole (V), fluconazole (F), amphotericin B (A) and caspofungin (C) with MIC values, MIC ranges, MIC_{50} and MIC_{90}.

Candida species*	No. of isolates	Anti-fungal agents	No. of isolates with MIC (µg/mL)											MIC_{50}	MIC_{90}	Range
			≤0.06	0.12	0.25	0.5	1	2	4	8	16	32	≥64			
C. tropicalis	145	V	75	36	23	7	2		2					0.06	0.25	0.01-4
		F		6	24	25	29	29	22	4	1	1	4	1	4	0.12-128
		A	3	2	15	64	58	3						0.5	1	0.06-2
		C	125	17		1		2						0.03	0.12	0.008-2
C. albicans	106	V	83	14	3	4	2							0.01	0.12	0.01-1
		F		23	18	23	22	14	5	1				0.5	2	0.12-8
		A	7	4	12	47	36							0.5	1	0.03-1
		C	96	7	2	1								0.03	0.06	0.008-0.5
C. glabrata	47	V	19	11	5	8	2	2						0.12	0.5	0.01-2
		F		2	5	7	9	13	6	3	2			1	4	0.12-16
		A		1	4	15	26	1						1	1	0.12-2
		C	36	3	3	5								0.03	0.25	0.008-0.5
C. parapsilosis	27	V	18	3	2	4								0.06	0.5	0.01-0.5
		F		2	5	2	5	8	5					1	4	0.12-4
		A		1	1	11	13	1						1	1	0.12-2
		C	7	10	6	4								0.12	0.5	0.01-0.5
C. krusei	26	V	12	3	4	5	2							0.12	0.5	0.01-1
		F		1	7		6	2	7	3				1	8	0.12-8
		A		2	2	8	14							1	1	0.12-1
		C		23	1	2								0.01	0.12	0.008-0.25
W. anomalus	15	V	1	2	6	6								0.25	0.5	0.06-0.5
		F						2	8	5				4	8	2-8
		A	6		3	3	2	1						0.25	1	0.01-2
		C	15											0.03	0.06	0.008-0.06
K. ohmeri	8	V	3	2	3									0.12	0.25	≤0.06-0.025
		F						4	2	2				4	8	2-8
		A			1	2	3	2						1	2	0.25-2
		C	6	1		1								0.03	0.5	0.01-0.5
C. rugosa/ C. mesorugosa	7	V	2	4	1									0.12	0.25	0.06-0.25
		F				1	2	4						2	2	0.5-2
		A				4	3							0.5	1	0.5-1
		C	7											0.03	0.03	0.01-0.03
C. lusitaniae	5	V	2	2		1								0.12	0.5	≤0.06-0.5
		F					2	3						2	2	1-2
		A					2		3					4	4	1-4
		C	4	1										0.06	0.12	≤0.06-0.12
C. famata	4	V	2	2										0.12	0.12	≤0.06-0.12
		F			1	1		1	1					2	4	1-4
		A				2	2							1	1	0.05-1
		C	4											0.06	0.12	≤0.06-0.12
C. kefyr	3	V	2	1										0.06	0.12	0.06-0.12
		F				3								0.5	0.5	0.5-0.5
		A				2	1							0.5	1	0.5-1
		C	3											0.01	0.03	0.01-0.03

*Isolates for which MIC breakpoints were unavailable at the time of the study were excluded from the analysis (n=8).

The overall distribution of isolated *Candida* species, identified after executing three different methods i.e. PCR RFLP, MALDI-TOF MS and Sangers sequencing has been given in Table 2.

Out of a total of 711 *Candida* isolates, 63% (n=448) were identified as non-*albicans Candida* (NAC) species, while 37% (n=263) were *C. albicans*. Among the NAC group, *C. tropicalis* was the most prevalent species (33.7%), followed by *C. glabrata* (11.5%), *C. krusei* (4.6%), *C. parapsilosis* (4.2%), and the uncommon

Wickerhamomyces anomalus (2.1%). Analysis of species distribution across clinical specimens revealed that *C. tropicalis* predominated in both blood (n=29) and urine (n=172) samples. In bloodstream infections, *C. tropicalis* remained the most frequent isolate, followed by *C. parapsilosis* (15.1%) and *W. anomalus* (12.6%), indicating a diverse species profile. In cases of vulvovaginal candidiasis, *C. albicans* (49/101) was the predominant species, with *C. tropicalis* (20/101) being the next most common isolate.

Antifungal MIC of fluconazole, voriconazole, amphotericin B and caspofungin was determined according to recommended guidelines. The MIC's, MIC ranges, MIC50 and MIC90 obtained upon antifungal susceptibility testing against the isolated *Candida* spp. are summarized in Table 3 for all 401 species.

DISCUSSION

Identification of *Candida* species by conventional methods- including the germ tube test, growth on *Candida* CHROMagar, sugar assimilation and fermentation tests, and growth on corn meal agar- are widely employed. These techniques are time-consuming, labour-intensive, require multiple culture-based assays and expert interpretation. Collectively, these delay accurate diagnosis and initiation of appropriate antifungal therapy. RFLP offers a practical, low-cost alternative, while MALDI-TOF MS provides rapid, accurate, and reproducible results, making it the emerging gold standard for yeast identification in clinical settings.[4]

Using RFLP, we successfully identified 92.5% (658/711) of *Candida* isolates, while 53 remained unidentified. Karimi et al, reported a high discriminatory power of 97.31% (832/855) for RFLP in yeast identification, highlighting its overall robustness.[4] Limitations in identifying particular *Candida* species using RFLP have been noted by other researchers.[11,12] While MALDI-TOF successfully resolved most of the isolates that RFLP could not identify, it still failed to identify six isolates, highlighting that even advanced methods have certain limitations. In a similar study, MALDI-TOF achieved 98.3% correct identification of clinical isolates at the species level underscoring its reliability in routine fungal diagnostics.[13] In our study, sequencing allowed identification of *C. mesorugosa* (n=4), *C. africana* and *Pichia farinosa* (n=1 each) that remained unresolved after PCR-RFLP and MALDI-TOF MS. Sequencing of the D1/D2 and ITS regions is widely recognized as the reference standard for yeast identification.[14]

In the present study, NAC species and *Candida albicans* constituted 63% and 37% isolates respectively. The preponderance of NAC species over *C. albicans* is increasingly being observed.[15,16] Our findings reveal, *Candida tropicalis* as the predominant NAC isolate, reaffirming its epidemiological significance in Indian setting, where its prevalence has been attributed to high virulence and increasing resistance to azole antifungals.[17,18] This pattern contrasts with the reports from United States and Australia where *C. glabrata* predominates, and Latin America where *C. albicans* still overshadows NAC species prevalence.[16,19,20] The evolution of different NAC species highlights the need for surveillance and species-level identification to guide effective management.

Treatment of Candida infections relies on antifungal agents that target the synthesis and integrity of the fungal cell membrane and wall comprising of azoles, which inhibit ergosterol biosynthesis; polyenes, which bind to ergosterol, compromising membrane stability; and echinocandins, which disrupt β-(1,3)-D-glucan synthesis, leading to cell wall destabilization.[21]

Fluconazole maintained favourable activity against the majority of *Candida* species, with MIC values ≤4 µg/ml for *C. albicans, C. tropicalis,* and *C. parapsilosis*. In contrast, elevated MICs were observed in *C. glabrata, C. krusei, W. anomalus,* and uncommon isolates such as *K. ohmeri* and *C. famata*. In our study, fluconazole susceptibility profiles were: *C. albicans* (S=94.3%, SDD=4.7%, R=0.9%), *C. tropicalis* (S=77.9%, SDD=15.2%, R=6.9%), *C. glabrata* (S=100%), and *C. parapsilosis* (S=81.5%, SDD=18.5%, R=0). These findings are consistent with data from the Asia-Pacific region, and, in agreement with earlier studies, further highlight the increasing trend of elevated fluconazole MICs among *C. tropicalis*.[22-24]

Voriconazole demonstrated strong activity against most *Candida* species, with elevated MICs observed only in single isolates of *C. glabrata* and *C. tropicalis* (MIC ≥2 µg/ml). These findings parallel earlier reports of higher voriconazole MICs in NAC species by Cuenca-Estrella et al.[25] Both *C. albicans* and *C. tropicalis* were highly susceptible (91.5% and 76.6%, respectively), while *C. krusei*, despite its intrinsic resistance to fluconazole, showed excellent susceptibility to voriconazole (S=92.3%, SDD=7.7%, R=Nil). Among rare yeasts, activity remained broad, with resistance detected in only one isolate. Clinically, these results underscore voriconazole as a valuable alternative, particularly for fluconazole-resistant *Candida* infections.

Caspofungin exhibited uniformly low MICs (MIC_{90}≤0.5 µg/ml) across most *Candida* species, including rare isolates. Resistance was observed in 10.6% of *C. glabrata* strains, consistent with its recognized reduced susceptibility to echinocandins. Clinically, these results support current guidelines endorsing echinocandins as first-line empirical therapy for invasive candidiasis. Compared to our findings, Wisplinghoff et al reported a much higher resistance rate of 61.9% in *C. glabrata*, a discrepancy likely reflecting the limited use of caspofungin in resource-limited settings, where cost and availability constrain its widespread application.[26]

Amphotericin B exhibited consistent activity across most *Candida* species, with MIC_{90} values generally ≤1 µg/ml for *C. albicans, C. tropicalis, C. glabrata, C. parapsilosis,* and *C. krusei*. Elevated MICs were observed in *C. lusitaniae* (up to 4 µg/ml), reflecting its well-documented reduced susceptibility to polyenes, and in select rare species such as *K. ohmeri*. In our study, three *C. lusitaniae* isolates were resistant, a finding in line with results from Turkey.[27] Despite these exceptions, amphotericin B retains strong efficacy against the majority of species and remains a valuable option in multidrug-resistant infections or in cases of treatment failure with azoles and echinocandins.

A notable limitation of this study is its exclusive reliance on in vitro MIC determinations, without correlation to patients' clinical outcomes. Nevertheless, the findings underscore the critical importance of accurate species identification and antifungal susceptibility testing in guiding effective therapy and optimizing patient outcomes.

Although the dataset was collected earlier, it remains highly valuable as it offers a robust baseline for assessing temporal changes in species distribution and antifungal resistance patterns. In regions where, continuous surveillance data are limited, such datasets are critical for benchmarking current trends and understanding the trajectory of emerging resistance. The findings therefore retain strong clinical and epidemiological relevance, supporting ongoing efforts in antifungal stewardship and guiding evidence-based decision-making in the management of candidiasis.

CONCLUSION

Prompt and accurate identification of *Candida* species, alongside determination of MICs for antifungal agents, is imperative and allows early recognition of resistant strains and ensures effective antifungal therapy, improving clinical decision-making, and ultimately enhancing patient care in fungal infections.

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REFERENCES

1. Hani U, Shivakumar H, Vaghela R, Osmani MR, Shrivastava A. Candidiasis: a fungal infection- current challenges and progress in prevention and treatment. Infect Disord Drug Targets. 2015;15(1):42-52.
2. Arendrup MC, Patterson TF. Multidrug-resistant Candida: epidemiology, molecular mechanisms, and treatment. J Infect Dis. 2017;216(3):S445-51.
3. Guinea J. Global trends in the distribution of Candida species causing candidemia. Clin Microbiol Infect. 2014;20(6):5-10.
4. Karimi L, Mirhendi H, Khodadadi H, Mohammadi R. Molecular identification of uncommon clinical yeast species in Iran. Curr Med Mycol. 2015;1(2):1-6.
5. Pandey N, Gupta MK, Paul P, Tilak R. Necessity to identify candida species accurately with minimum inhibitory concentration determination in each case of bloodstream infections. J Infect Public Health. 2020;13(5):753-8.
6. Wiederhold NP. Antifungal resistance: current trends and future strategies to combat. Infect Drug Resist. 2017;10:249-59.
7. Mohammadi R, Mirhendi H, Rezaei-Matehkolaei A, Ghahri M, Shidfar MR, Jalalizand N, et al. Molecular identification and distribution profile of *Candida* species isolated from Iranian patients. Med Mycol. 2013;51(6):657-63.
8. Zhang YJ, Zhang S, Liu XZ, Wen HA, Wang M. A simple method of genomic DNA extraction suitable for analysis of bulk fungal strains: fungal DNA isolation by thermolysis. Lett Appl Microbiol. 2010;51(1):114-8.
9. Ghosh AK, Paul S, Sood P, Rudramurthy SM, Rajbanshi A, Jillwin TJ, et al. Matrix-assisted laser desorption ionization time-of-flight mass spectrometry for the rapid identification of yeasts causing bloodstream infections. Clin Microbiol Infect. 2015;21(4):372-8.
10. CLSI document. M27-A3: reference method for broth dilution antifungal susceptibility testing of yeasts; approved standard. Wayne; 2008.
11. Pfaller MA, Diekema DJ. Rare and emerging opportunistic fungal pathogens: concern for resistance beyond Candida albicans and Aspergillus fumigatus. J Clin Microbiol. 2004;42(10):4419-31.
12. Miceli MH, Díaz JA, Lee SA. Emerging opportunistic yeast infections. Lancet Infect Dis. 2011;11(2):142-51.
13. Lacroix C, Gicquel A, Sendid B, Meyer J, Accoceberry I, François N, et al. Evaluation of two matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS) systems for the identification of Candida species. Clin Microbiol Infect. 2014;20(2):153-8.
14. Slechta ES, Hohmann SL, Simmon K, Hanson KE. Internal transcribed spacer region sequence analysis using SmartGene IDNS software for the identification of unusual clinical yeast isolates. Med Mycol. 2012;50(5):458-66.
15. Singh T, Kashyap AK, Ahluwalia G, Chinna D, Sidhu SS. Epidemiology of fungal infections in critical care setting of a tertiary care teaching hospital in North

India: a prospective surveillance study. J Clin Sci Res. 2014;3(1):14-25.
16. Lamoth F, Lockhart SR, Berkow EL, Calandra T. Changes in the epidemiological landscape of invasive candidiasis. J Antimicrob Chemother. 2018;73(1):i4-13.
17. Kaur R, Dhakad MS, Goyal R, Kumar R. Emergence of non-albicans *Candida* species and antifungal resistance in intensive care unit patients. Asian Pac J Trop Biomed. 2016;6(5):455-60.
18. Mohanraj H, Vinodhini VM, Vajravelu LK. Evaluation of epidemiological pattern of Candida species associated with candidemia from A tertiary care facility in South India. J Pure Appl Microbiol. 2024;18(3):1949-58.
19. Lockhart SR, Iqbal N, Cleveland AA, Farley MM, Harrison LH, Bolden CB, et al. Species identification and antifungal susceptibility testing of *Candida* bloodstream isolates from population-based surveillance studies in two US cities from 2008 to 2011. J Clin Microbiol. 2012;50(11):3435-42.
20. Matsumoto E, Boyken L, Tendolkar S, McDanel J, Castanheira M, Pfaller M, et al. Candidemia surveillance in Iowa: emergence of echinocandin resistance. Diagn Microbiol Infect Dis. 2014;79(2):205-8.
21. Ghannoum MA, Rice LB. Antifungal agents: mode of action, mechanisms of resistance, and correlation of these mechanisms with bacterial resistance. Clin Microbiol Rev. 1999;12(4):501-17.
22. Tan TY, Hsu LY, Alejandria MM, Chaiwarith R, Chinniah T, Chayakulkeeree M, et al. Antifungal susceptibility of invasive *Candida* bloodstream isolates from the Asia-Pacific region. Med Mycol. 2016;54(5):471-7.
23. Posteraro B, Spanu T, Fiori B, De Maio F, De Carolis E, Giaquinto A, et al. Antifungal susceptibility profiles of bloodstream yeast isolates by Sensititre YeastOne over nine years at a large Italian teaching hospital. Antimicrob Agents Chemother. 2015;59(7):3944-55.
24. Deorukhkar S, Saini S. Non *albicans Candida* species: its isolation pattern, species distribution, virulence factors and antifungal susceptibility profile. Int J Med Sci Public Health. 2013;2(3):533.
25. Cuenca-Estrella M, Gomez-Lopez A, Cuesta I, Zaragoza O, Mellado E, Rodriguez-Tudela JL, et al. Frequency of voriconazole resistance in vitro among Spanish clinical isolates of Candida spp. According to breakpoints established by the Antifungal Subcommittee of the European Committee on Antimicrobial Susceptibility Testing. Antimicrob Agents Chemother. 2011;55(4):1794-7.
26. Wisplinghoff H, Ebbers J, Geurtz L, Stefanik D, Major Y, Edmond MB, et al. Nosocomial bloodstream infections due to Candida spp. in the USA: species distribution, clinical features and antifungal susceptibilities. Int J Antimicrob Agents. 2014;43(1):78-81.
27. Dagi HT, Findik D, Senkeles C, Arslan U. Identification and antifungal susceptibility of Candida species isolated from bloodstream infections in Konya, Turkey. Ann Clin Microbiol Antimicrob. 2016;15(1):36.

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