Research Article

# Prevalence of non-albicans candidemia in a tertiary care hospital in Northeast India

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**Received:** August 12, 2017; **Accepted:** September 22, 2017

## ABSTRACT

**Background:** Candidemia is gaining significance worldwide and is the fourth most common cause of bloodstream infections (BSI) with mortality ranging from 5% to 71%. **Objectives:** To determine the prevalence of candidemia and characterize the non-albicans candida (NAC) causing BSI. **Materials and Methods:** The study was conducted in the department of microbiology for a period of 2 years (January 2014-December 2015). Blood samples received were cultured in brain-heart infusion broth. Subcultures were done in Sabouraud Dextrose Agar and Sabouraud Chloramphenicol Cycloheximide Agar. For identification of isolates, Gram stain, germ tube test, urea hydrolysis, cornmeal agar morphology, and sugar assimilation test were performed. Antifungal susceptibility testing was determined by Vitek 2 compact system using AST YS 06 Cards. **Results:** During the study period, a total of 4123 blood samples were received, of which 786 samples were positive. Among the 786 positive samples, 739 (94%) samples showed bacterial growth and 47 (6%) samples showed growth of *Candida* spp. The prevalence of NAC was 32 (70%) and rest 15 (30%) was *Candida albicans*. Most of the NAC species showed resistance to fluconazole, voriconazole, caspofungin, micafungin, and amphotericin B as compared to *C. albicans.* **Conclusion:** The study highlights the change in epidemiology in the species distribution of *Candida.* There is a rise in infections by NAC species as compared to those by *C. albicans.* Early and regular species identification and antifungal testing is necessary to decrease the mortality associated with it.

**KEY WORDS:** Non-albicans Candida; Bloodstream Infection; Candidemia; Antifungals

## INTRODUCTION

Bloodstream infections (BSI) caused by various *Candida* species have been reported from many countries worldwide and are a significant cause of morbidity and mortality in hospitalized patients. Candidemia has been associated with many risk factors such as long-term hospitalization, antibiotic therapy, use of intravascular catheters, and underlying diseases such as diabetes and malignancy. Early and prompt diagnosis, proper treatment, and prevention of candidemia pose a major challenge for microbiologists and clinicians worldwide.[1]

There is considerable regional variability, and therefore, local epidemiological knowledge is critical in the effective management of invasive candidiasis. Only a few studies from India have reported candidemia rates of 6-18%,[2,3] and an increase in isolation of non-albicans candida (NAC) species from blood samples[4,5] has been reported. Speciation and susceptibility testing of *Candida* is still routinely not done at most of the centers.

The susceptibility testing of fungi to antimycotic drugs, including that of yeasts, was until recently performed only rarely because of factors such as: The cases necessitating

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| **DOI:** 10.5455/ijmsph.2017.0823622092017 | |

systemic antifungal treatment were relatively rare; the number of antifungal drugs was limited; the incidence of resistance to the drugs was rare; and most importantly, correlation between *in vitro* and *in vivo* results was questionable. However, recently, there is a marked increase in the incidence of mycotic diseases, especially disseminated infections, which necessitates antifungal testing importance. Moreover, the number of available drugs has increased, and an innate or acquired resistance of certain yeast species to several drugs has been reported. Thus, susceptibility testing of yeasts has been standardized and published by Clinical Laboratory Standards Institute (M27-A3).[6]

Increased incidence of antifungal drug resistance has become a major cause of concern in the management of candidemia.[4] Therefore, species identification and determination of antifungal susceptibility pattern of the *Candida* isolates will help in early diagnosis and prompt therapeutic intervention.

## MATERIALS AND METHODS

This study was carried out in the department of microbiology in a tertiary care center in Northeast India for a period of 2 years from January 2014 to December 2015. Ethical clearance for the study was obtained from the Institutional Ethics Committee. Blood samples were collected aseptically in brain-heart infusion (BHI) broth from suspected cases of BSI. The BHI bottle was incubated at 37°C in the laboratory and observed daily for visible growth. Subculture was performed on Sabouraud agar, blood agar, and MacConkey agar. The yeast isolates were identified based on the colony morphology, gram stain, germ tube test, cornmeal agar morphology, sugar assimilation test, and CHROM agar. All isolates of *Candida* were characterized and subjected to antifungal susceptibility testing using the Vitek 2 system. AST YS 07 Cards were used as per to manufacturer's instructions, and results were evaluated for activity against fluconazole, voriconazole, caspofungin, micafungin, amphotericin B, and flucytosine.

Statistical analysis was performed using MedCalc for Windows, version 12.5 (MedCalc software, Ostend, Belgium), and the significance of *P* value was determined if it is <0.05. For comparison of antifungal drug resistance between *Candida albicans* and NAC, Chi-square test was used, and for comparison of drug resistance among different species of *Candida,* Kruskal–Wallis ANOVA test was applied.

## RESULTS

A total of 4123 blood samples were received during the study period, of which 786 (19.05%) samples were positive. Among the 786 positive samples, bacterial growth was seen in 739 (94%) samples and other 47 (6%) samples showed growth of *Candida* spp. The prevalence of NAC was 33 (70%) and rest 14 (30%) was *C. albicans* as shown in Table 1. Male preponderance was seen with male-to-female ratio 1.3:1. Maximum incidence was seen in old age group (>55 years) 29%.

Distribution of *Candida* species showed that maximum isolates were of *C. albicans* (*n* = 14, 30%), followed by *Candida tropicalis* (*n* = 10, 21%), *Candida glabrata* (*n* = 9, 19%)*, Candida lusitaniae* (*n* = 9, 19%), and *Candida parapsilosis* (*n* = 5, 11%) as depicted in Table 2.

### Antifungal Susceptibility

The sensitivity profiles of all *Candida* isolates are shown in Table 3. The susceptibility profile showed 89% sensitivity to fluconazole, 98% to voriconazole, 87% to caspofungin, 94% to micafungin, 77% to amphotericin B, and 81% to 5-flucytosine. *C. glabrata* showed higher resistance to fluconazole (*P* = 0.004) and caspofungin (*P* = 0.003) in comparison to *C. albicans*. *C. glabrata* showed higher resistance to micafungin in comparison to other *Candida* spp. (*P* < 0.0001) and flucytosine in comparison to *C. albicans* and *C. tropicalis* (*P* = 0.007). *C. glabrata* also showed higher resistance to amphotericin B in comparison to *C. albicans, C. tropicalis,* and *C. lusitaniae* (*P* = 0.0002).

NAC species showed more resistance to fluconazole, voriconazole, caspofungin, micafungin, and amphotericin B as compared to *C. albicans* (Table 4). Among the antifungal drugs tested, only amphotericin B showed statistically significant (*P* = 0.036) difference between NAC and *C. albicans.*

Minimum inhibitory concentration (MIC) profile of the *Candida* isolates as depicted in Table 5 showed MIC

**Table 1:** Distribution of *C. albicans* and NAC

| Species | *n* (%) |
|---|---|
| *C. albicans* | 14 (30) |
| NAC | 33 (70) |
| Total | 47 (100) |

NAC: Non-albicans candida, *C. albicans: Candida albicans*

**Table 2:** Distribution of *Candida* according to species

| Species | *n* (%) |
|---|---|
| *C. albicans* | 14 (30) |
| *C. tropicalis* | 10 (21) |
| *C. parapsilosis* | 5 (11) |
| *C. glabrata* | 9 (19) |
| *C. lusitaniae* | 9 (19) |

*C. albicans: Candida albicans, C. tropicalis: Candida tropicalis, C. parapsilosis: Candida parapsilosis, C. glabrata: Candida glabrata, C. lusitaniae: Candida lusitaniae*

**Table 3:** Antifungal susceptibility patterns of *Candida* isolates

| Species | Fluconazole (%) | Voriconazole (%) | Caspofungin (%) | Micafungin (%) | Amphotericin B (%) | Flucytosine (%) |
|---|---|---|---|---|---|---|
| *C. albicans* (14) | 100 | 100 | 100 | 100 | 100 | 50 |
| *C. tropicalis* (10) | 100 | 100 | 100 | 100 | 100 | 100 |
| *C. lusitaniae* (9) | 100 | 100 | 100 | 100 | 78 | 78 |
| *C. glabrata* (9) | 56 | 100 | 56 | 100 | 22 | 100 |
| *C. parapsilosis* (5) | 80 | 80 | 60 | 40 | 60 | 100 |
| Total (47) | 89 | 98 | 87 | 94 | 77 | 81 |

*C. albicans: Candida albicans, C. tropicalis: Candida tropicalis, C. parapsilosis: Candida parapsilosis, C. glabrata: Candida glabrata, C. lusitaniae: Candida lusitaniae*

**Table 4:** Comparison of antifungal susceptibility between NAC and *C. albicans*

| Species | Fluconazole | Voriconazole | Caspofungin | Micafungin | Amphotericin B | Flucytosine |
|---|---|---|---|---|---|---|
| *C. albicans* (%) | 100 | 100 | 100 | 100 | 100 | 50 |
| NAC (%) | 85% | 97 | 82 | 91 | 67 | 61 |
| *P* | 0.306 | 0.655 | 0.218 | 0.607 | 0.036 | 0.879 |

NAC: Non-albicans candida, *C. albicans: Candida albicans*

**Table 5:** MIC profile of *Candida* isolates

| Species | Fluconazole (%) | Voriconazole (%) | Caspofungin (%) | Micafungin (%) | Amphotericin B (%) | Flucytosine (%) |
|---|---|---|---|---|---|---|
| *C. albicans* | ≤1 (100) | ≤0.12 (100) | ≤0.25 (100) | ≤0.06 (100) | 0.5 (100) | ≤1 (50) |
| *C. tropicalis* | ≤1 (100) | ≤0.12 (100) | ≤0.25 (100) | ≤0.06 (100) | 0.5 (60) | ≤1 (100) |
| *C. lusitaniae* | ≤1 (100) | ≤0.12 (100) | ≤0.25 (100) | ≤0.06 (100) | 0.5 (22) | ≤1 (78) |
| *C. glabrata* | 2 (56) | 1 (78) | ≤0.25 (56) | ≤0.06 (67) | 0.5 (22) | ≤1 (100) |
| *C. parapsilosis* | 4 (80) | ≤0.12 (80) | 1 (60) | ≤0.06 (40) | 0.5 (60) | ≤1 (100) |

*C. albicans: Candida albicans, C. tropicalis: Candida tropicalis, C. parapsilosis: Candida parapsilosis, C. glabrata: Candida glabrata, C. lusitaniae: Candida lusitaniae,* MIC: Minimum inhibitory concentration

of <1 μg/ml for fluconazole in *C. albicans*, *C. tropicalis*, and *C. lusitaniae,* and higher MICs were seen in *C. glabrata* and *C. parapsilosis* of 2 and 4 μg/ml, respectively. Voriconazole showed MIC of ≤0.12 μg/ml for all except *C. glabrata.* Caspofungin showed MIC of ≤0.25 μg/ml for most of the *Candida* spp. except *C. parapsilosis* while micafungin showed MIC of ≤0.06 μg/ml for most of the isolates. Amphotericin B showed MIC of 0.5 μg/ml for *C. albicans,* and higher MICs were seen in NAC. Flucytosine showed MIC of ≤1 μg/ml for *C. tropicalis, C. glabrata, and C. parapsilosis*.

## DISCUSSION

The present study was a retrospective study conducted in the department of microbiology in a tertiary care center in Northeast India for a period of 2 years from January 2014 to December 2015. The main objective of the study was to determine the prevalence of candidemia and characterize the *Candida* isolates from blood culture sample received from patients admitted in various intensive care units (ICUs) and wards. Our study showed a prevalence rate of 6% candidemia with predominance of NAC species (70%). The most common isolate was *C. albicans* (30%) followed by *C. tropicalis* (21%). The most common age group was >55 years with male-to-female ratio of 1.3:1. Antifungal susceptibility showed higher resistance in NAC species which was statistically not significant except for amphotericin B which was significant ($P < 0.05$), and *C. glabrata* showed high resistance to most of the antifungals, especially to amphotericin B.

Candidemia is becoming a common occurrence in patients admitted in ICUs. Intense therapy, invasive procedures, and various underlying diseases lead to BSI leading to sepsis with various bacterial and fungal agents. Our study showed prevalence rate of 6% candidemia during the study period. The result of our study was comparable to Xess et al. from AIIMS, New Delhi, where 6% prevalence rate was found during 5-year study (2001-2005),[3] Sahni et al. from Maulana Azad Medical College, New Delhi, found 6.9% prevalence for *Candida* species in BSI,[7] and Kumar et al.[8] reported 5.7% prevalence rate. However, some studies showed higher prevalence of 18% for *Candida* species among blood culture isolates,[9] and another study from Rohtak, North India, reported an isolation rate of 8.1% for *Candida* species from cases of neonatal septicemia.[10] The prevalence of *Candida* species in BSI has increased worldwide in the last decades. In the last few years, occurrence of NAC species

is steadily increasing. In the present study, NAC accounted for 70% similar to studies by Oberoi et al.,[11] 83.2% and Bhatt et al.,[12] 85.3%. Over the past 20 years, a shift toward NAC species has been reported previously from the USA, Europe, and Australia although the precise pattern of causative species varies across countries.[13] We observed a significant predominance of NAC species (70%), with *C. albicans* being the most common isolate (30%), followed by *C. tropicalis* (21%). Traditionally, *C. tropicalis* has been the second most common *Candida* species recovered from blood.[14] Similarly, as previously reported at a hospital in China, *C. albicans* (57.8%) continued to play a dominant role in candidemia, followed by *C. tropicalis* (12.8%).[15] In many countries, *C. tropicalis* and *C. parapsilosis* have become the most common *Candida* species to cause BSI.[16] In India, *C. tropicalis* is now the most common cause of nosocomial candidemia, accounting for 67-90% of cases of candidemia which has been shown by many epidemiological studies.[9,17,18] The increased use of fluconazole has been determined to be the major cause of predominance of NAC, especially *C. tropicalis* over *C. albicans*. In our study, *C. tropicalis* was the predominant isolate among the NAC accounting for 21% which was similar to studies by Shivprakasha et al. from South India where *C. tropicalis* was the most prevalent species of *Candida* (35.6%).[4] and *C. albicans* was only 3.4% of the cases. Similar reports have also been documented by Adhikary and Joshi (39.7%),[19] Xess et al. from AIIMS, New Delhi,[3] and Verma et al. (33%).[17] Our study showed that older age group patients of >55 years were more prone to developing candidemia which also reported by Guzman et al.[20] (<7 days and >61 years) and Ma et al.[21] (>65 years) which shows that immunity is lower at early and extreme of age wherein high-risk area like ICUs. In our study, male-to-female ratio was 1.3:1 which shows that there is no significant gender preponderance in the development of candidemia.

The antifungal drugs currently available for the treatment of invasive mycoses can be divided into four different classes on the basis of their mechanisms of action: (1) Alteration of membrane function (amphotericin B); (2) inhibition of DNA or RNA synthesis (flucytosine); (3) inhibition of ergosterol biosynthesis (azoles [fluconazole, itraconazole, and the newer agents voriconazole, posaconazole, and ravuconazole]); and (4) inhibition of glucan synthesis (echinocandins [caspofungin, micafungin, and anidulafungin]). Antifungal susceptibility testing of *Candida* isolates in our study was performed for all the different classes of antifungal drugs using Vitek 2 compact system which showed NAC had higher resistance to most of the antifungals when compared with *C. albicans* which was not statistically significant except for amphotericin B which was significant ($P < 0.05$) Among the NAC, *C. glabrata* showed high resistance to most of the antifungals, especially to amphotericin B similar to previous studies.[21-24] Azoles are the most commonly used group of antifungal agents for the treatment of candidemia. In our study, *C. albicans* showed 100% sensitivity to fluconazole whereas NAC showed 85% sensitivity. This finding was comparable to Oberoi et al.[11] and Pfaller et al.[16] where NAC showed higher resistance to fluconazole then *C. albicans*. Overall sensitivity to fluconazole was 89% which was similar to 88.3% by Xess et al.[3] and 75% by Adhikary and Joshi.[19] Fluconazole and the other triazoles have less activity against species of *Candida* such as *C. krusei* and *C. glabrata.* Fluconazole is the only antifungal agent for which considerable information regarding antifungal resistance trends and well-standardized guidelines for susceptibility testing are available. The other azole tested in our study was voriconazole which showed excellent sensitivity of 98% which was similar to findings by Kumar et al.,[8] Xess et al.,[3] and Ma et al.[21] However, another study by Kothari and Sagar 2008[9] showed a very high resistance of 56% to voriconazole. Echinocandins such as caspofungin and micafungin have shown considerable *in vitro* and *in vivo* efficacy in the treatment of invasive candidiasis and candidemia. Caspofungin got the food and drug administration approval for the treatment of candidemia in 2003. The emerging trend of resistance to fluconazole and other triazoles among *Candida* isolates from BSI has made echinocandins very important. Echinocandins have less drug-related toxicity compared to amphotericin B. However, the use of echinocandins is limited in developing countries like India due to its high cost and limited availability.[1] In our study, *C. albicans* showed 100% sensitivity to both caspofungin and micafungin similar to finding by Montagna et al.[25] where *C. albicans* showed sensitivity of 96% to micafungin and 97.2% to caspofungin. NAC showed 18% resistance to caspofungin in our study which correlated to studies by Farmakiotis et al.,[26] 10.3%, from the USA. *C. glabrata* showed high resistance of 44% which was similar to Montagna et al.[25] Micafungin showed 100% sensitivity for *C. albicans* and 91% for NAC which was similar to Pfaller et al.[19] from the USA and Montagna et al.[25] from Italy. Flucytosine has *in vitro* activity against many isolates of *Candida* species, but it is not commonly used because of drug toxicities as well as the frequent development of resistance when used as a single agent. The highest rates of primary resistance are found in *C. albicans* serotype B, *C. glabrata, C. krusei,* and *C. guilliermondii.* Secondary resistance in *C. albicans* is primarily due to a decrease in the activity of the uracil phosphoribosyltransferase.[27-29] Our study showed 81% sensitivity to flucytosine which was similar to 63% by Bhatt et al.[12] and 88.5% by Vinodkumar et al.[30] The incidence of amphotericin B-resistant *Candida* spp. in our study was 13% which is comparable to incidences reported in recent studies.[11,12] Amphotericin B in various formulations has been used for the treatment of disseminated candidiasis and candidemia. Although amphotericin B has a rapid cidal action against most strains of *Candida* species (especially *C. albicans*), it is not the first choice for treatment of cases of candidemia because of the nephrotoxicity associated with it.[31] Secondary resistance to amphotericin B also appears to be an uncommon development. There have been reports of some cases of disseminated infections due

to *C. glabrata, C. krusei,* and *C. albicans* isolates that developed amphotericin B resistance during treatment.[27] The mechanism of amphotericin B resistance appears to be an alteration or a decrease in the amount of ergosterol in the cell membrane.[32] Among the *Candida* species, *C. lusitaniae* accounting for 1-2% of candidemias has higher intrinsic resistance to amphotericin B.[33] This finding was similar to our study which showed high MIC seen in *C. lusitaniae*.

The present study being a retrospective one has not addressed specific risk factors, which plays a role in the selection of species causing fungemia as well as variable susceptibility patterns. Moreover, the sample size being small was another limiting factor in our study. Further studies with more clinical data and larger sample size will be more beneficial.

## CONCLUSION

In the present study, we identified the magnitude of candidemia from the blood culture sample received from patients in a tertiary care hospital and characterized the *Candida* isolates. Emergence of NAC and their resistance to antifungals is a matter of concern. The changing epidemiology of candidemia emphasizes the need for monitoring of distribution of *Candida* species and antifungal susceptibility testing to develop guidelines on empiric therapy for invasive candidiasis, based on the epidemiology of infection which will help us to recognize the emerging fungal pathogen and drug resistance.

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**How to cite this article:** Sabhapandit D, Lyngdoh WV, Bora I, Prasad A, Debnath K, Elantamilan D. Prevalence of non-albicans candidemia in a tertiary care hospital in Northeast India. Int J Med Sci Public Health 2017;6(11):1620-1625.

**Source of Support:** Nil, **Conflict of Interest:** None declared.