



Novel *Candida* sequence types and Antifungal profiles in a Northern Brazil Hematology Center

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Abstract

Introduction: Invasive fungal infections, particularly candidiasis, remain a major cause of morbidity and mortality in patients with hematologic malignancies, complicated by emerging antifungal resistance.

Methods: Here we characterized *Candida* species isolated from hematologic patients at a tertiary referral center in Northern Brazil using antifungal susceptibility testing and multilocus sequence typing.

Results: We identified *C. albicans*, *C. krusei*, and *C. tropicalis* isolates, including fluconazole and amphotericin B resistant *C. krusei* strains. Molecular analysis revealed three novel *C. albicans* sequence types and a new *C. krusei* sequence type, phylogenetically linked to global circulating clones. These findings indicate local revolution and potential clonal dissemination within the hospital environment.

Conclusion: Our study underscores the importance of ongoing molecular surveillance and antifungal susceptibility monitoring to inform infection control and optimize treatment strategies in immunocompromised hematologic patients. This is the first report of novel *Candida* sequence types in a hematologic reference center in the Amazon region.

Keywords: Molecular epidemiology, invasive fungal infection, oncohematological patient, *Candida* infections, Hematologic malignancies, antifungal resistance

Introduction

Candida albicans is a commensal yeast that is part of the normal human microflora. This species can also act as an infectious agent, causing invasive disease, especially in oncohematological patients such as those with acute lymphocytic leukemia (ALL), acute myeloid leukemia (AML), or those undergoing chemotherapy [1-5]. Invasive candidiasis remains a significant concern in hospitals worldwide, with its incidence increasing in recent years [5,6]. The epidemiological profile of candidemia varies among cancer patients, particularly those with oncohematologic malignancies [5]. Invasive fungal infection (IFI) poses a significant threat to oncohematological patients and is one of the main causes of morbidity and mortality [1,7]. Clinical signs are often nonspecific because of the lack of characteristic symptoms. Furthermore, resistance to standard antifungal treatments leads to longer hospital stays, treatment difficulties, and higher healthcare costs [1,7]. Among the yeasts, *Candida albicans* (*C. albicans*) and non-*Candida albicans* (NAC) species – including *Candida glabrata* (*C. glabrata*), *Candida tropicalis* (*C.*

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tropicalis), *Candida parapsilosis* (*C. parapsilosis*), and *Candida krusei* (*C. krusei*) – account for more than 90% of invasive candidiasis cases [4]. The reported incidence ranges from 0,15% to 1,5% in hospitalized patients with underlying malignancies [6]. In recent years, emerging infections caused by NAC species have become an increasing clinical concern [8].

The incidence and mortality rates of IFIs vary by region. In Iran, the estimated mortality from *Candida* infections ranges from 30% to 80%, while in Taiwan it is approximately 5.9%. In India, the prevalence among AML and ALL patients was 62.5% (15/24) and 37.5% (9/24), respectively [9]. Vargas-Espindola *et al.* [6] reported a mortality rate of 50% in a multicenter study conducted at five cancer treatment centers in Bogota, which is similar to the global average found in various studies [10-12]. In Brazil, Camplesi Jr. *et al.* [1] identified invasive fungal infections in 16.2% (n = 19) of hematologic patients, with 6 cases caused by yeasts and 13 by filamentous fungi. In Manaus (Amazonas, Brazil), mycological diagnostic records from the Bacteriology Laboratory of the HEMOAM Hospital Foundation (January–August 2022) showed that fungal species isolated from patients with hematologic diseases included: *C. albicans* (2.5%), *C. guilliermondii* (21.0%), *C. tropicalis* (5.3%), *C. pelliculosa* (5.3%), *C. famata* (5.3%), *C. krusei* (8.0%), *C. haemulonii* (7.7%), *C. parapsilosis* (26.4%), *Cryptococcus laurentii* (13.2%), and *Fusarium* spp. (5.3%). Even with various therapeutic options available, the prevalence of fungal infections has increased, possibly due to the use of antifungal drugs as prophylaxis, which may encourage the development of resistance mechanisms in these microorganisms. Additionally, current medications, such as polyenes (amphotericin B), azoles (itraconazole, fluconazole, voriconazole), and echinocandins (caspofungin, micafungin, and anidulafungin), still have drawbacks related to effectiveness, selectivity, toxicity, resistance, and spectrum of activity [13-15].

Therefore, due to the ongoing global reports of therapeutic failures in fungal infections caused by resistant strains and their potential link to higher mortality rates, the World Health Organization (WHO) published a list of priority fungal pathogens, including *C. albicans* in the critical group; *C. tropicalis* and *C. parapsilosis* in the high priority group [16]. There is an urgent need to implement susceptibility testing methods for fungi in hospitals, both to gather data for a better understanding of the issue and to identify the most effective treatments for patients [4]. In addition to resistance profiles, molecular epidemiology is crucial for understanding the evolutionary dynamics of *Candida* species and mapping the geographic distribution of resistant and non-resistant clonal groups. Several studies have identified dominant sequence types (STs), such as ST69 and ST90 for *C. albicans* and

ST256 for *C. krusei*, which have been isolated from various human and animal biological samples across continents, including Australia, Asia, Europe, North America, and South America (including Brazil) [2, 17, 18, 27]. Therefore, this study aims to investigate the infectious processes caused by different *Candida* species in hematological patients admitted to a reference hematology and hemotherapy hospital in northern Brazil. The study will evaluate the antifungal susceptibility profiles and molecular epidemiology of the isolates to determine their origin and evolutionary relationships among circulating sequence types. The findings may help develop strategies to prevent outbreaks, limit the spread of resistance, and improve clinical outcomes and prognosis for patients affected by this condition.

Methods

Study design and Patients

This is a qualitative, observational, and exploratory study with experimental elements, based on the analysis of five cases of infections caused by *Candida* spp. The study used data from diagnostic and therapeutic procedures, electronic medical records, and laboratory records from the HEMOAM Hospital Foundation. The cases involved hematological patients of both genders and all age groups who were treated or hospitalized with suspected or confirmed fungal infections between January 2022 and December 2023. The cases described refer to patients diagnosed and treated at FHEMOAM, originating from the capital city of Manaus and rural municipalities in the State of Amazonas, including Eirunepé, Fonte Boa, São Gabriel da Cachoeira, Manacapuru, São Sebastião de Uatumã, and Santa Isabel do Rio Negro. These patients had previously been diagnosed with hematological cancers, including B-lineage acute lymphoblastic leukemia (ALL-B), acute myeloid leukemia (AML), diffuse large B-cell lymphoma, and high-risk Burkitt lymphoma. All patients were hospitalized for chemotherapy and developed neutropenia during treatment, along with clinical suspicion of infection. Blood and urine cultures were ordered, and microbiological tests identified *C. albicans*, *C. tropicalis*, and *C. krusei*. After identifying the fungal pathogens, antifungal therapy with fluconazole was started, leading to favorable outcomes in patients with large cell diffuse lymphoma, AML, and Philadelphia chromosome-positive ALL-B. The patients diagnosed with ALL-B and high-risk Burkitt lymphoma, however, died.

Microbiological Diagnosis

The samples were collected during routine procedures at the FHEMOAM Clinical Microbiology Laboratory. The biological samples sent to the bacteriology laboratory included urine and blood. Urine was cultured on URILAB-CLED/MacConkey slide culture media (Laborclin- Biokar Diagnostics, Allonne, France) for primary seeding. After 24

hours of incubation and a colony count of 1,000,000 CFU/mL, a subculture was performed on 5% sheep blood agar, Mueller-Hinton agar, MacConkey agar, and Sabouraud agar (Himedia, Hexasystems, Mumbai, India), followed by another 24 hours of incubation at 35.4°C. A pure colony grown on Sabouraud agar was selected for subsequent tests and Gram staining. The positive blood sample was cultured on 5% sheep blood agar, chocolate agar, Mueller-Hinton agar, MacConkey agar, and Sabouraud agar at 35.4°C for 24 hours. After incubation, a pure colony from Sabouraud agar was chosen for further testing and Gram staining. Using standard microbiological procedures, phenotypic identification and susceptibility testing (including antifungal susceptibility) with respective MIC values were performed using automated equipment (VITEK-2, bioMérieux, Craponne, France), following the manufacturer's instructions and the 2024 guidelines from the Brazilian Committee on Antimicrobial Susceptibility Testing (BrCAST) and the Clinical & Laboratory Standards Institute (CLSI). Isolates of *C. albicans*, *C. tropicalis*, and *C. krusei* demonstrating resistance in antifungal susceptibility testing (antifungigram) were preserved at -80°C in cystine tryptic soy broth supplemented with 20% glycerol at the Bacteriology Laboratory of the HEMOAM Foundation.

Molecular Epidemiology

Genomic DNA from *Candida* spp. isolates was extracted using the PureLink Genomic DNA Mini Kit (Invitrogen, CA, USA) following the manufacturer's protocol, with minor modifications. Polymerase chain reaction (PCR) was performed to amplify multilocus sequence typing (MLST) genes specific to *C. albicans*, *C. tropicalis*, and *C. krusei*, following protocols described in previous studies [19-21]. PCR products were sequenced using the BigDye Terminator v3.1 Cycle Sequencing Kit (Thermo Fisher Scientific) and analyzed with the ABI PRISM 3130 XL Genetic Analyzer (Applied Biosystems, Foster City, CA), according to the manufacturer's instructions. Sequence analysis of each MLST gene for *C. albicans*, *C. tropicalis*, and *C. krusei* was conducted following protocols available on the PubMLST website (<https://pubmlst.org/organisms>) [22]. Sequence fragments were mapped, trimmed, and aligned using Geneious v.2025 software with reference sequences from the PubMLST database. Sequence typing was performed by comparing allele combinations with database entries to determine the sequence type (ST). Newly identified STs for *C. albicans* and *C. krusei* were uploaded to the MLST database. Phylogenetic models of the newly identified STs of *C. albicans* were constructed using allele profile data (AAT1a, ACC1, ADP1, SYA1, VPS13, and ZWF1b) from 3,252 entries, and for *C. krusei* using 427 entries (HIS3, LEU2, NMT1, TRP1, LYS2D), obtained from various sources (Table 1) and regions (Graphic 1 and Graphic 2). Notably, the only record for *C. krusei* in the PubMLST database corresponds to

the strain described in this study (Graphic 2). Phylogenetic trees were generated using Phyloviz v2.2 software, available at <https://www.phyloviz.net/goeburst/> and <https://www.phyloviz.net/> [23,24].

Table 1: Quantification of human biological samples used on the eBURST organogram

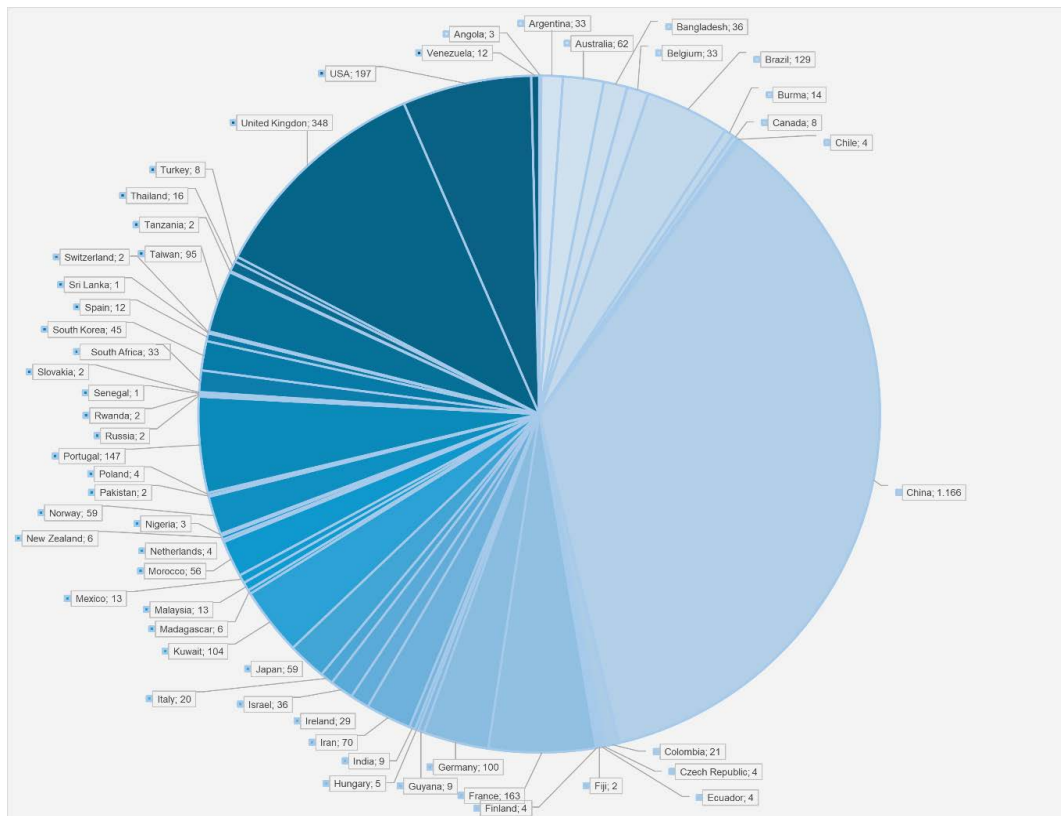
Source	<i>C. albicans</i>		<i>C. krusei</i>	
	n	%	n	%
Blood	1,295	39,8	154	56,4
Catheter	47	1,4	-	-
Feces	135	4,2	-	-
Bronchial lavage	25	0,8	-	-
Other sterile sites	-	-	28	10,3
Other superficial source	-	-	43	15,8
Oropharynx	579	17,8	-	-
Oropharynx/sputum	-	-	39	14,3
Skin	65	2,0	-	-
Sputum	148	4,6	-	-
Urine	209	6,4	-	-
SWAB vaginal	726	22,3	11	4
Wound	23	0,7	-	-
Not informed	-	-	152	55,7

Results

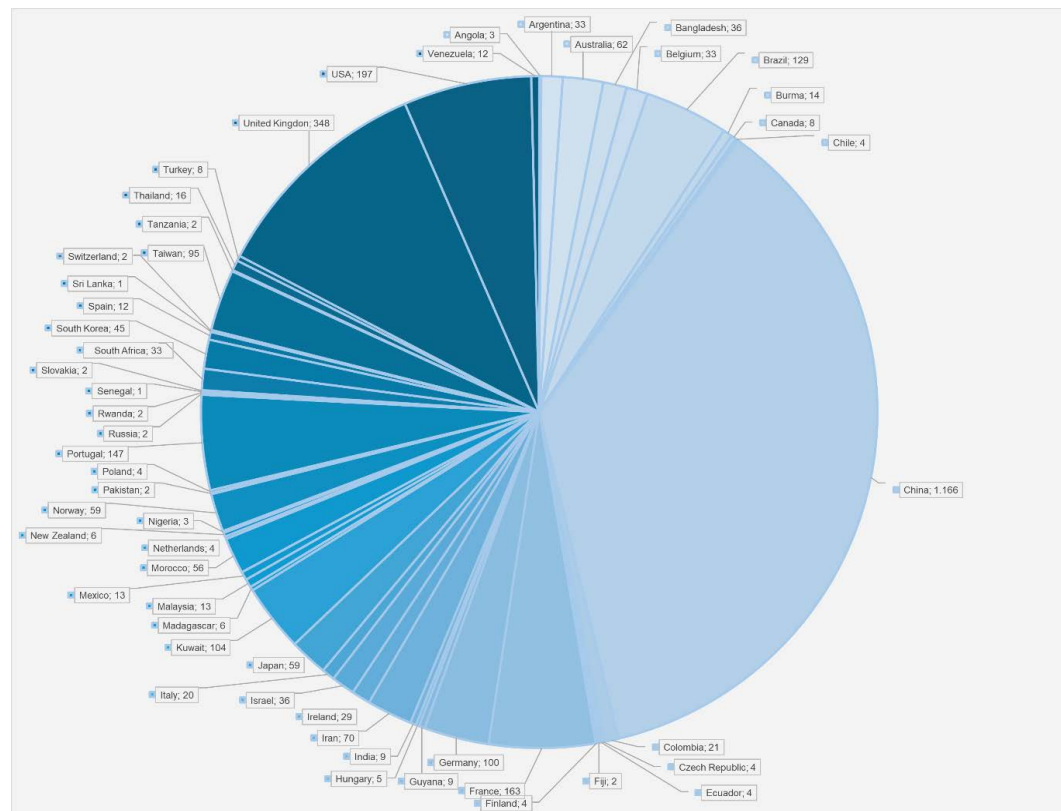
Microbiological analyses identified three (3) isolates of *C. albicans*, one (1) *C. tropicalis*, and two (2) *C. krusei*. The results of antifungal susceptibility testing (AST), including minimum inhibitory concentrations (MICs) and intermediate susceptibility phenotypes (i.e., susceptible, increased exposure), are shown in Table 3. Among the isolates, *C. krusei* showed resistance to fluconazole (MIC = 8 µg/mL) and amphotericin B (MIC=2 µg/mL). *C. albicans* exhibited an intermediate phenotype to voriconazole (MIC ≤ 0.12 µg/mL), while *C. tropicalis* was susceptible to all tested antifungal agents. Susceptibility profiles of *C. albicans* to the echinocandins caspofungin and micafungin could not be classified because clinical breakpoint values for these agents have not yet been established by the European Committee on Antimicrobial Susceptibility Testing (EUCAST v10.0) or the Clinical and Laboratory Standards Institute (CLSI, 2024) (Table 2).

Multi-Locus Sequence Typing

Molecular epidemiology analysis based on MLST genes for *C. albicans* identified three new sequence types (STs): ST4220, ST4278, and ST4279. Additionally, a new allele for the AAT1a gene (allele 215) was registered in the PubMLST database, originating from sample 126. For *C. krusei*, a new sequence type, ST303, was identified as circulating



Graphic 1: Quantitative of samples by countries used on *C. albicans* eBURST organogram



Graphic 2: Quantitative of samples by countries used on *C. krusei* eBURST organogram

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Table 2: *Candida* spp. antimicrobial susceptibility test

Isolates	id	Antifungal	MIC	Interpretation
<i>Candida albicans</i>	10	Fluconazole	≤0.5	S
		Voriconazole	≤0.12	I
		Caspofungin	≤0.12	-
		Micafungin	≤0.06	-
		Amphotericin B	1	S
	68	Fluconazole	1	S
		Voriconazole	≤0.12	I
		Caspofungin	≤0.12	-
		Micafungin	≤0.06	-
		Amphotericin B	1	S
	126	Fluconazole	2	S
		Voriconazole	≤0.12	I
		Caspofungin	≤0.12	-
		Micafungin	≤0.06	-
		Amphotericin B	0,5	S
<i>Candida krusei</i>	58	Fluconazole	8	R
		Voriconazole	≤0.12	IE
		Caspofungin	0.25	-
		Micafungin	0.12	IE
		Amphotericin B	2	R
<i>Candida tropicalis</i>	60	Fluconazole	1	S
		Voriconazole	≤0.12	S
		Caspofungin	≤0.12	-
		Micafungin	≤0.06	-
		Amphotericin B	≤0.25	S
	127	Fluconazole	≤0.5	S
		Voriconazole	≤0.12	S
		Caspofungin	≤0.12	-
		Micafungin	≤0.06	-
		Amphotericin B	≤0.25	S

MIC: Minimum Inhibitory Concentration. EUCAST v.10.0. S: Sensible. R: Resistant. I: Susceptible, increased exposure. IE: indicates that there is insufficient evidence that the species in question is a good target for therapy with the drug.

in the hospital during the study period. In contrast, *C. tropicalis* isolates were classified as ST1248 and ST327, previously reported in China and Taiwan, respectively, and identified from blood samples (Table 3).

e-BURST analysis of *C. albicans* sequence types showed that ST69 is the founding clone from which all the newly identified STs likely originated. This clone has been found in multiple regions and countries, as well as in various biological sources, including feces, oropharyngeal swabs, skin, blood, vaginal swabs, sputum, and urine (Table 2). ST4220 and ST4279 seem to cluster within the ST69 clonal complex

and are part of two separate subgroups formed by ST443, ST2904, and ST76. Both ST4220 and ST4279 are located as terminal nodes, indicating recent divergence and potential for future subgroup development. In contrast, ST4278 is part of a subgroup that traces back to ST1754 and shares ST90 as a common ancestor, also forming a distinct terminal node (Supplemental material- S1).

S1. Evolutionary relationship of *C. albicans* STs isolated from oncohematological patients at the FHE-MOAM in Manaus, Brazil.

Regarding *C. krusei*, the eBURST diagram (Figure 2) identified ST256, initially reported in China, as the ancestral clone. The new ST303 appears to have descended from ST69, a clone previously isolated from blood samples in the Czech Republic. Both are part of a clonal complex derived from ST19, a sequence type that circulates in the United Kingdom, Canada, Australia, and has also been detected in animal hosts [17] (Supplemental material- S2).

S2. Evolutionary relationship of *C. krusei* ST isolated from an oncohematological patient at the FHE-MOAM in Manaus, Brazil.

Discussion

Invasive fungal infections (IFIs) continue to pose a major challenge in hematology, with *Candida* species being the primary pathogens in this setting [29]. Candidiasis is among the most common invasive fungal diseases linked to bloodstream spread, especially in patients with hematological cancers, such as those treated at FHEMOAM [1,29].

In this series, three notable species—*C. albicans*, *C. tropicalis*, and *C. krusei*—were isolated from blood and urinary infections in hospitalized patients with oncohematological conditions. Notably, *C. albicans* and *C. tropicalis* are designated as WHO-priority fungal pathogens [16]. Similar findings have been reported by Chen et al. (China), Mitra et al. (India), Vargas-Espindola et al. (Colombia), and Diniz et al. (Brazil), all of whom identified *Candida* species associated with blood and urinary tract infections in neutropenic or immunocompromised patients [6,9,25,29].

Beyond the serious nature of these infections and the risks of morbidity and death, the circulating resistance of *C. krusei* to amphotericin B and fluconazole at FHEMOAM raises concerns and requires attention. While our *C. tropicalis* isolates were fully susceptible, previous studies in Thailand and Colombia reported fluconazole-resistant *C. tropicalis* strains [6,8]. In Brazil, Diniz et al. [25] noted that *C. albicans* and *C. tropicalis* remained sensitive to amphotericin B, fluconazole, and echinocandins, aligning with our results. These differences likely reflect variation in antifungal use and local environmental pressures influencing susceptibility patterns. Of particular interest is the resistance profile of *C.*

Table 3: Types of STs identified in *Candida* species isolated from oncohematological patients from Hemoam Foundation

Nº	Species	ST	Continents/Regions	Sample	Comments
1	<i>C. albicans</i>	691	Africa, Asia, Australia, China, United Kingdom, United States of America, Brazil, France, Portugal, Austaly, New Zlandia, Norway, Ireland, Japan, Morocco,	feces, oropharynx, skin, blood, swab and urine, sputum	common ancestral of all ST
		4431	China	Blood, urine, oropharynx, vaginal swab	most recent common ancestor of ST2904
		4431	Taiwan	blood	most recent common ancestor of ST4279²
		791	China, United States, United Kingdom, Kuwait, Argentina, Colombia, Argentina, France, Japan, Portugal, Morocco, Portugal	urine, vaginal swab, blood, sputum	most recent common ancestor of ST4220²
		17541	China	oropharynx	most recent common ancestor of ST90
		901	South Africa, Kuwait, South Korea, Brazil, Guyana, China	blood, feces, oropharynx, urine, vaginal swab	most recent common ancestor of ST4278²
		42202	Brazil	blood, urine	-
		42782	Brazil	blood	-
		42792	Brazil	blood	-
		2641	China	Blood, other sterile sites	Common ancestral of all ST
2	<i>C. krusei</i>	19	United Kingdom, Canada, Australia	not informed	most recent common ancestor of ST24
		241	United Kingdom, Poland, Taiwan, France	blood	most recent common ancestor of ST69
		691	Czech	blood	common ancestor of ST303²
		3032	Brazil	blood	-

1: PubMLST data. 2: New ST (this study)

krusei, which has been documented across Asia, Europe, North America, and South America. Our findings agree with those of Domán et al. [17], who identified *C. krusei* strains with inherent fluconazole resistance or acquired amphotericin B resistance, often linked to mutations in the hotspot regions (HS1, HS2, HS3) of the conserved FKS1 gene, which encodes the target of echinocandins, underscoring the importance of ongoing antifungal monitoring [26].

Our molecular epidemiology results also reveal the genetic diversity and worldwide distribution of *Candida* clones. The ancestry analysis of new *C. albicans* sequence types (ST4220 and ST4279) showed that ST69, previously identified by Tian et al. [27] and McManus et al. [18], is a dominant global founder genotype. This clone is highly capable of colonizing humans and has been isolated from various anatomical sites and regions [18,28]. Although ST4278 is a novel *C. albicans* sequence type from our study, it shows a distinct evolutionary pathway with ST90 as it's closest ancestor. Despite being isolated from patients in the same facility, the three *C. albicans* types (ST4220, ST4279, and ST4278) occupy different branches in the eBURST diagram, indicating significant genomic variation. Similar genetic diversity has been observed in Kuwait, Germany, Taiwan, and Singapore, reflecting the complex population structure

of *C. albicans* [18,27,29]. To our knowledge, this is the **first report of novel STs of *C. albicans* and *C. krusei*** circulating in a Hematology Reference Center in Northern Brazil. The combination of antifungal susceptibility profiles and molecular typing underscores the importance of ongoing surveillance in immunocompromised populations [7,30]. Antifungal prophylaxis, when used judiciously, may help decrease morbidity and mortality in this setting [30,31].

Conclusion

This study identified new *Candida* STs 4220, 4279, and 4278 (*C. albicans*) and ST303 (*C. krusei*) at a Hematologic Reference Center in Brazil. These results highlight the circulation of previously unreported STs in this region. Amphotericin B- and fluconazole-resistant *C. krusei* is especially concerning due to the limited antifungal options for hematologic patients. Possibly influenced by local selective pressure, such as antifungal therapy, microevolutionary processes may promote the emergence of these resistant clones. While some of these STs share ancestry with globally widespread genotypes like ST69, their different evolutionary paths could support the idea of local adaptation and potential clonal spread within a hospital setting. Ongoing molecular surveillance, along with prudent antifungal use and informed treatment choices, may be importante for monitoring clonal

spread and guiding infection control strategies, as well as preventing outbreaks. These findings support the hypothesis that a new *Candida* ST maybe present in our hospital environment.

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Declarations

Ethics statement

This study was approved by the Foundation Human Research Ethical Committee (CEP/HEMOAM) under CAAE N° 68471223.5.0000.0009.

Competing interests

The authors declare no competing interests.

Author's Contributions

CMF, WAF co-conceptualized the study, undertook statistical analyses and wrote the original manuscript draft. CMF, WAF, FGN, GAVS, AMT, GMAF, JSC revised the manuscript. GMAF, LAG, ARP, JSC, AMT and FOC undertook sample collection and laboratory tests. VCS, FGN AND GAVS sequenced the isolates. CMF, WAF and GMAF supervised the work, vetted the results and critically reviewed the manuscript.

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Supplementary file

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