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ORIGINAL RESEARCH

Epidemiology, Antifungal Susceptibility, and in-Hospital Mortality of Candidemia in a Tertiary Hospital of China: A 10-Year Retrospective Analysis

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Purpose: In response to the increasing incidence of candidemia, this study aimed to investigate its epidemiology and risk factors for in-hospital mortality in an eastern Chinese tertiary hospital, thereby guiding clinical management.

Patients and Methods: We retrospectively enrolled 387 inpatients with candidemia (2015–2024). Species distribution, clinical characteristics, and antifungal susceptibility were analyzed. Cox regression model was used to identify independent risk factors for in-hospital mortality.

Results: The cumulative incidence of candidemia was 0.214 per 1,000 inpatients, with an overall in-hospital mortality of 33.85%. The intensive care unit (ICU) was the primary patient location (55.30%). *Candida albicans* remained the predominant pathogen (31.27%), followed by *Candida glabrata* (25.58%), *Candida parapsilosis* complex (21.44%), and *Candida tropicalis* (18.60%). Ward-specific analysis showed that *C. albicans* was most frequent in the ICU (34.11%) and infectious disease wards (50.00%), whereas *C. glabrata* predominated in urology (52.17%) and pancreatic surgery (41.18%) wards. In Hematology, *C. tropicalis* was the primary pathogen (66.67%). Most of *C. albicans* and *C. parapsilosis* were susceptible (>90%) to all nine antifungals; while *C. tropicalis* exhibited relatively high resistance/non-wild-type (18.06%–69.44%) to four azoles, with 27.78% cross-resistance to fluconazole and voriconazole. Age ≥63 years (*HR* = 1.530, 95% *CI*: 1.077–2.174, *P* = 0.018), ICU admission (*HR* = 2.005, 95% *CI*: 1.200–3.350, *P* = 0.008), and acute or chronic renal failure (*HR* = 1.849, 95% *CI*: 1.261–2.711, *P* = 0.002) were identified as independent risk factors, whereas *C. parapsilosis* complex infection (*HR* = 0.456, 95% *CI*: 0.275–0.756, *P* = 0.002) and antifungal therapy (*HR* = 0.313, 95% *CI*: 0.194–0.505, *P* = 0.001) were independently associated with lower risks.

Conclusion: *C. albicans* was the predominant cause of candidemia in our institute. Given its high incidence and mortality, it was imperative to develop tailored management and prevention strategies based on the local pathogen profile, antifungal susceptibility patterns, and specific mortality risk factors. Attention should focus on high-risk patients to improve clinical outcomes.

Keywords: candidemia, epidemiology, species distribution, drug resistance, in-hospital mortality, risk factors

Introduction

The *Candida* genus comprises approximately 200 distinct species, among which at least 30 are pathogenic to humans, with *Candida albicans, Candida glabrata, Candida parapsilosis* and *Candida tropicalis* being the most common species.[1,2] *Candida* infections can occur in virtually any anatomical site in the human body, with particular propensity to invade the bloodstream.[3] As a major pathogen of hospital-acquired bloodstream infections, *Candida* has consistently ranked among the top five causative agents of nosocomial bloodstream infections over the past two decades, with this trend being particularly prominent in patients admitted to intensive care unit (ICU).[4,5]

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Candidemia is a severe and life-threatening infection associated with high morbidity, prolonged hospitalization, and substantial healthcare costs.[6,7] A single-center study in China demonstrated a steady increase in the prevalence of candidemia over recent decades.[8] Despite improved understanding of risk factors, antifungal treatment options, and infection control measures, its mortality rate remains alarmingly high, up to 25%–50%.[7] Notably, multidrug-resistant *Candida* strains represented by *Candida auris* have emerged as a novel global public health threat due to their intrinsic antifungal resistance and high mortality rates,[9] which underscores the critical importance of early effective antifungal therapy and supportive care in improving the outcomes of patients with candidemia.

While blood culture remains the current gold standard for diagnosing candidemia,[10] the inherent slow growth of fungi renders conventional laboratory testing inadequate to meet clinical demands for rapid and precise diagnosis, necessitating empirical antifungal therapy as an essential approach.[9,11] The selection of such empirical treatment primarily relies on epidemiological data and antifungal susceptibility profiles, which exhibit significant regional variations.[4,12,13] Therefore, obtaining patient-based regional microbiological and clinical surveillance data is of paramount importance.

Current research on candidemia in China remains insufficient, particularly lacking systematic data regarding epidemiological characteristics, pathogen distribution, antifungal susceptibility profiles, as well as clinical features and prognostic factors. This study was conducted at a 4500-bed tertiary-care grade A hospital in Eastern China. Through a 10-year retrospective analysis, we aimed to partially elucidate the microbiological and clinical characteristics of candidemia inpatients in this region, while contributing to prognostic assessment and identification of disease-related risk factors.

Materials and Methods

Study Design

We conducted a retrospective analysis of electronic laboratory data from patients with blood culture-confirmed candidemia at the First Affiliated Hospital with Nanjing Medical University (Nanjing, China) between January 2015 and December 2024. This study was approved by the Research Ethics Committee of the First Affiliated Hospital with Nanjing Medical University (No. 2026-SR-297).

Patient Selection Criteria

A candidemia episode was defined as the isolation of any *Candida* spp. in at least one blood culture sample from a patient with corresponding clinical signs and symptoms.[3] The diagnostic criteria strictly adhered to the most recent expert consensus by the Chinese Adult Candidiasis Diagnosis and Management Expert Consensus Group (2020), while remaining consistent with the international guidelines issued by the European Society of Clinical Microbiology and Infectious Diseases and the Infectious Diseases Society of America Clinical Practice Guidelines for the Management of Candidiasis.[12,14] The inclusion criteria were as follows: **a**. hospitalized patients; **b**. blood culture-confirmed candidemia with available antifungal susceptibility and absolute neutrophil count results; **c**. only the first episode of candidemia per patient was included. Patients with incomplete clinical data or unclear discharge survival status were excluded.

Definitions of Clinical Variables

The data collected from patients with candidemia included sex, age, body temperature, underlying diseases, invasive procedures, medication exposures, antifungal treatment, and ICU admission. The key clinical variables analyzed in this study were explicitly defined as follows: fever was defined as a tympanic membrane temperature of ≥37.5°C recorded at the onset of candidemia; acute or chronic renal failure was clinically diagnosed and documented in medical records, consistent with the KDIGO (Kidney Disease: Improving Global Outcomes) clinical practice guidelines;[15] neutropenia was defined as an absolute neutrophil count $<0.5 \times 10^9$/L; antibiotic exposure referred to the receipt of any systemic antibacterial agents for ≥48 hours; and corticosteroid therapy was considered present at the administration of systemic corticosteroids at a dose of ≥0.3 mg/kg/day for ≥3 weeks in the past 60 days.[16] Survival time was calculated from candidemia diagnosis to discharge or death. The primary endpoint was in-hospital all-cause mortality, defined as death from any cause during the index hospitalization.

Microorganism Identification and Antifungal Susceptibility Testing

All clinically submitted blood specimens were cultured with the Bactec FX400 automated blood culture system (BD, United States), equipped with Bactec Plus/F resin aerobic bottles and Bactec Lytic/10 anaerobic bottles containing lytic agents. Upon positivity, Gram staining was performed for microscopic examination, and the cultures were manually subcultured onto both Columbia blood and chocolate agar plates (Autobio, Zhengzhou, China) for further analysis. An additional Sabouraud dextrose agar plate (Autobio, Zhengzhou, China) was inoculated if fungal spores were observed on the smear, followed by aerobic incubation at 37°C for 24–72 hours. The identification of *Candida* species was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS; Bio-Mérieux, France).

The susceptibility to 9 antifungal agents was determined by the broth microdilution methods according to Clinical and Laboratory Standards Institute (CLSI) guidelines, employing in-house prepared 96-well microplates.[17] The tested agents comprised 3 echinocandins (anidulafungin, micafungin, and caspofungin), 4 azoles (posaconazole, voriconazole, itraconazole, and fluconazole), 5-flucytosine, and amphotericin B. The antifungal susceptibility testing results were interpreted based on species-specific CLSI clinical breakpoints (CBPs) as defined in document M60,[18] or on epidemiological cutoff values (ECVs) from document M59 for which no CBPs are available,[19] to distinguish wild-type (WT) from non-wild-type (NWT) strains. The quality control strains were *C. parapsilosis* ATCC 22019 and *Candida krusei* ATCC 6258. Furthermore, the minimum inhibitory concentration (MIC) distribution for each *Candida* species was summarized to calculate MIC_{50} and MIC_{90}.

Statistical Analysis

All statistical analyses were conducted using SPSS software (version 26.0; IBM, Armonk, NY, United States). Age was dichotomized using the median age of the adult patients in this specific study population as the cutoff. The distribution of continuous variables was assessed with the Kolmogorov–Smirnov test. Normally distributed data were expressed as mean ± standard deviation, while non-normally distributed data were summarized as median and interquartile ranges [*M (P25, P75)*]. Intergroup comparisons involved the Mann–Whitney *U*-test for continuous variables. Categorical variables were described as percentages (%), and group differences were evaluated via the $\chi 2$ test or Fisher-Freeman-Halton exact test. Variables with a *P* value <0.1 in univariate Cox regression analysis were subsequently included in the multivariate Cox regression analysis. Hazard ratio (HR) and 95% confidence interval (CI) were calculated to identify risk factors for in-hospital mortality among candidemia patients. A two-sided *P*-value of less than 0.05 was considered statistically significant.

Results

Study Participants and Epidemiological Findings

A total of 387 patients including 380 adults and 7 minors with candidemia were enrolled in our study, and the median age of the adult patients was 63 (50, 73) years. Among these participants, 43.41% (168/387) were the elderly (≥66 years old), and 64.08% (248/387) were males. ICU was the most common patient location for *Candida* bloodstream isolates at the onset (214, 55.30%), followed by the departments of internal medicine (92, 23.77%) and general surgery (81, 20.93%). Further analysis revealed that the wards of hematology (36, 9.30%), urology (23, 5.94%), pancreatic surgery (17, 4.39%), and infectious diseases wards (10, 2.58%) were the primary contributing units within the internal medicine and general surgery systems (Figure 1A). There were 10 *Candida* species identified, among which *C. albicans* remained the most prevalent causative species, accounting for approximately one-third of all isolates (121, 31.27%), while non-albicans *Candida* species, including *C. glabrata* (99, 25.58%), *C. parapsilosis* complex (83, 21.44%) and *C. tropicalis* (72, 18.60%), were the predominant causative agents of candidemia. Other rare non-albicans *Candida* isolates accounted for less than 5% of all strains, including *C. krusei* (5, 1.29%), *Candida lusitaniae* (2, 0.52%), *Candida haemulonii* (2, 0.52%), *Candida famata* (1, 0.26%), *C. auris* (1, 0.26%), and *Candida norvegensis* (1, 0.26%) (Figure 1B). The distribution of pathogens in candidemia varied among patients with different underlying diseases, invasive procedures, and medication regimens, and detailed demographic and clinical data were shown in Table 1.

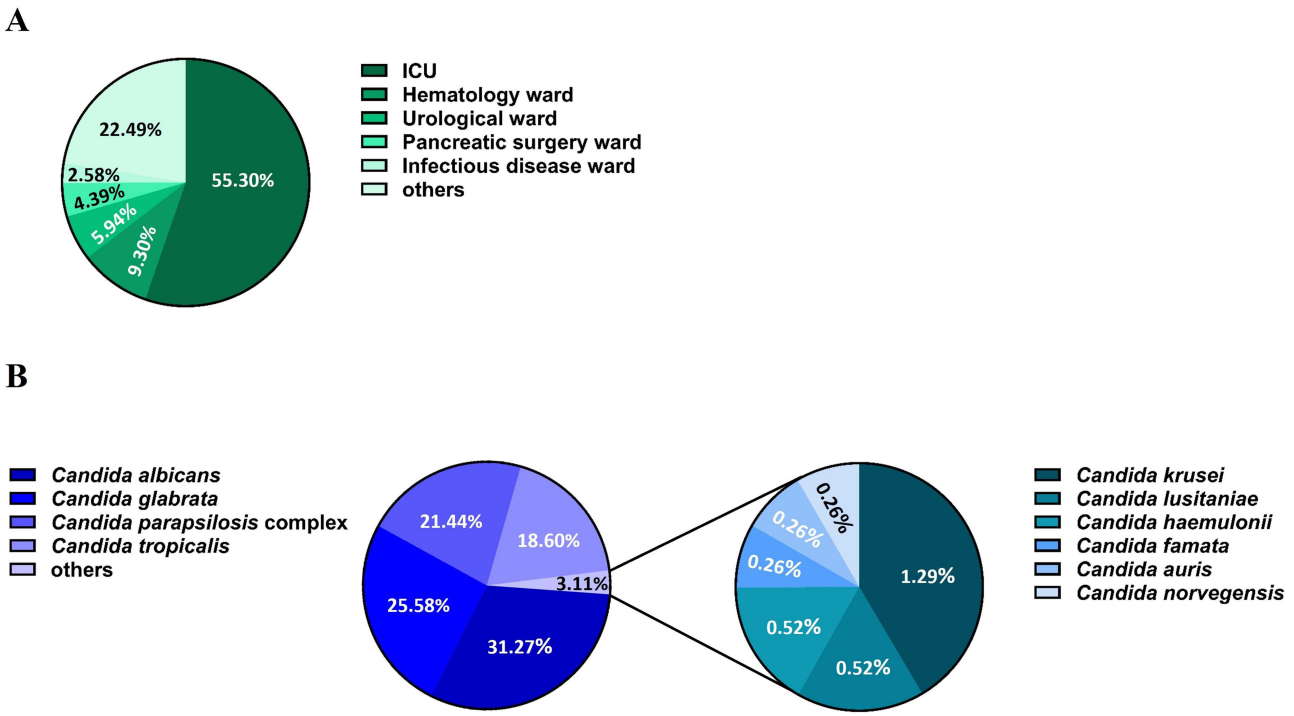

Figure 1 (A) Distribution of main clinical departments as sources of *Candida* spp.in candidemia. **(B)** Overall distribution of 10 confirmed *Candida* spp. during the study period.
Abbreviation: ICU, intensive care unit.

Trends in the Morbidity and Mortality Rates of Candidemia

Based on surveillance data during the period from January 2015 to December 2024, our study observed dynamic evolving trends in the morbidity and mortality of candidemia (Figure 2). The cumulative incidence was 0.214 per 1,000 admissions. It initially showed an upward trend, rising from 0.156 per 1,000 admissions in 2015 to a peak of 0.256 per 1,000 admissions in 2018 and subsequently stabilized and remained between 0.201 and 0.253 per 1,000 admissions from 2019 to 2024, exhibiting an overall fluctuating decline. Although the incidence decreased and stabilized after 2018, mortality did not show a corresponding reduction, consistently remaining at a high level with significant annual fluctuations. The overall mortality rate was 33.85% (131/387), and it even increased sharply to 53% in 2024.

Distribution of Pathogenic Microorganisms at the Onset of Candidemia

The distribution of the top five pathogens with the highest isolation proportion in candidemia cases was displayed in Figure 3A, along with epidemiological characteristics including age, gender, seasonality, and their distribution across primary clinical departments. The pathogens were arranged in descending order of isolation proportion from left to right along the horizontal axis, and clinical departments were ranked in descending order starting with ICU from up to down of the vertical coordinate. Among individuals under 18 years old, the *C. parapsilosis* complex accounted for the highest proportion, at 42.86%, while *C. tropicalis* was the most common in the 18–45 age group (29.31%). In other age groups, particularly among those aged ≥66 years, *C. albicans* showed the highest isolation proportion, reaching 38.10%. Overall, *C. albicans* accounted for the highest proportion in the first half of the year, exceeding 35%, while the incidence of the four common candidemia types was comparable in the second half of the year. Each department appeared to have its own distinct candidemia epidemiology. In our study, *C. albicans* bloodstream infections were most frequently observed in the ICU and infectious disease wards. In the urological wards and pancreatic surgery wards, *C. glabrata* was the most common causative agent of candidemia, whereas the hematology departments reported *C. tropicalis* as the predominant pathogen, with a notably high incidence of 66.67%.

Over the ten-year period, the proportion of *C. albicans* exhibited considerable fluctuation, ranging between 18.60% and 42.22%. In 2017, 2018, and 2020, the isolation proportion of the *C. parapsilosis* complex, *C. glabrata*, and

Table 1 Clinical characteristics of 387 patients with candidemia of different species

Characteristics	*Candida albicans* n=121 (%)	*Candida glabrata* n=99 (%)	*Candida parapsilosis* Complex n=83 (%)	*Candida tropicalis* n=72 (%)	Others n=12 (%)	χ^2	*P*
Male	78 (64.46)	56 (56.57)	58 (69.88)	48 (66.67)	8 (66.67)	3.893	0.421
0-17 years	1 (0.83)	1 (1.01)	3 (3.61)	1 (1.39)	1 (8.33)	/	0.185
18-45 years	14 (11.57)	12 (12.12)	14 (16.87)	17 (23.61)	1 (8.33)	6.597	0.159
46-65 years	42 (34.71)	42 (42.42)	33 (39.76)	32 (44.44)	5 (41.67)	2.258	0.688
≥66 years	64 (52.89)	44 (44.44)	33 (39.76)	22 (30.56)	5 (41.67)	9.780	0.044
Fever	76 (62.81)	63 (63.64)	54 (65.06)	52 (72.22)	9 (75.00)	2.467	0.651
Co-existent bacteremia	36 (29.75)	21 (21.21)	19 (22.89)	19 (26.39)	5 (41.67)	4.029	0.402
Underlying diseases							
Cardiovascular disease	79 (65.29)	51 (51.52)	54 (65.06)	32 (44.44)	7 (58.33)	11.428	0.022
Solid tumor	39 (32.23)	29 (29.29)	19 (22.89)	16 (22.22)	5 (41.67)	4.543	0.338
Leukemia and lymphoma	2 (1.65)	3 (3.03)	6 (7.23)	29 (40.28)	1 (8.33)	84.227	<0.001
Diabetes mellitus	34 (28.10)	37 (37.37)	21 (25.30)	14 (19.44)	3 (25.00)	7.252	0.123
Acute/Chronic renal failure	25 (20.66)	19 (19.19)	15 (18.07)	15 (20.83)	5 (41.67)	3.720	0.445
Hepatitis	6 (4.96)	4 (4.04)	8 (9.64)	8 (11.11)	3 (25.00)	/	0.044
Neutropenia	3 (2.48)	2 (2.02)	0 (0.00)	19 (26.39)	0 (0.00)	/	<0.001
Invasive procedure							
Mechanical ventilation	63 (52.07)	45 (45.45)	45 (54.22)	19 (26.39)	5 (41.67)	15.301	0.004
Body cavity drainage tube	54 (44.63)	53 (53.54)	34 (40.96)	21 (29.17)	5 (41.67)	10.372	0.035
Surgical operation	52 (42.98)	47 (47.47)	36 (43.37)	23 (31.94)	3 (25.00)	5.732	0.220
Indwelling urinary catheter	47 (38.84)	50 (50.51)	36 (43.37)	18 (25.00)	4 (33.33)	11.980	0.018
Central venous catheter	28 (23.14)	28 (28.28)	29 (34.94)	28 (38.89)	1 (8.33)	9.249	0.055
Total parenteral nutrition	32 (26.45)	27 (27.27)	34 (40.96)	11 (15.28)	5 (41.67)	13.927	0.008
Pre-Candidemia							
Antibiotic exposure	109 (90.08)	91 (91.92)	75 (90.36)	65 (90.28)	9 (75.00)	3.466	0.483
Blood product transfusion	97 (80.17)	69 (69.70)	55 (66.27)	57 (79.17)	8 (66.67)	7.239	0.124
Corticosteroid therapy	7 (5.79)	12 (12.12)	14 (16.87)	23 (31.94)	2 (16.67)	25.186	<0.001
Receipt of immunosuppressants	9 (7.44)	9 (9.09)	9 (10.84)	21 (29.17)	1 (8.33)	22.315	<0.001

(*Continued*)

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Table 1 (Continued).

Characteristics	*Candida albicans* n=121 (%)	*Candida glabrata* n=99 (%)	*Candida parapsilosis* Complex n=83 (%)	*Candida tropicalis* n=72 (%)	Others n=12 (%)	χ^2	*P*
Post-Candidemia							
Antifungal therapy	96 (79.34)	69 (69.70)	67 (80.72)	60 (83.33)	9 (75.00)	5.667	0.225
Antifungal regimen							
Fluconazole	13 (10.74)	15 (15.15)	10 (12.05)	9 (12.50)	4 (33.33)	5.346	0.254
Voriconazole	24 (19.83)	17 (17.17)	20 (24.10)	15 (20.83)	2 (16.67)	1.468	0.832
Caspofungin	39 (32.23)	24 (24.24)	18 (21.69)	11 (15.28)	2 (16.67)	8.015	0.091
Fluconazole + Caspofungin	8 (6.61)	5 (5.05)	7 (8.43)	6 (8.33)	3 (25.00)	/	0.201
Voriconazole + Caspofungin	12 (9.92)	5 (5.05)	12 (14.46)	8 (11.11)	0 (0.00)	/	0.210
Amphotericin B monotherapy	0 (0.00)	0 (0.00)	0 (0.00)	2 (2.78)	0 (0.00)	/	0.095
Amphotericin B combination therapy	0 (0.00)	2 (2.02)	0 (0.00)	8 (11.11)	0 (0.00)	/	<0.001
In-hospital mortality	45 (37.19)	35 (35.35)	19 (22.89)	28 (38.89)	4 (33.33)	5.972	0.201

Notes: /, Fisher-Freeman-Halton exact test was used if total sample size was < 40, any expected frequency < 1, or more than 20% of the expected frequencies < 5, and therefore no test statistic was available.

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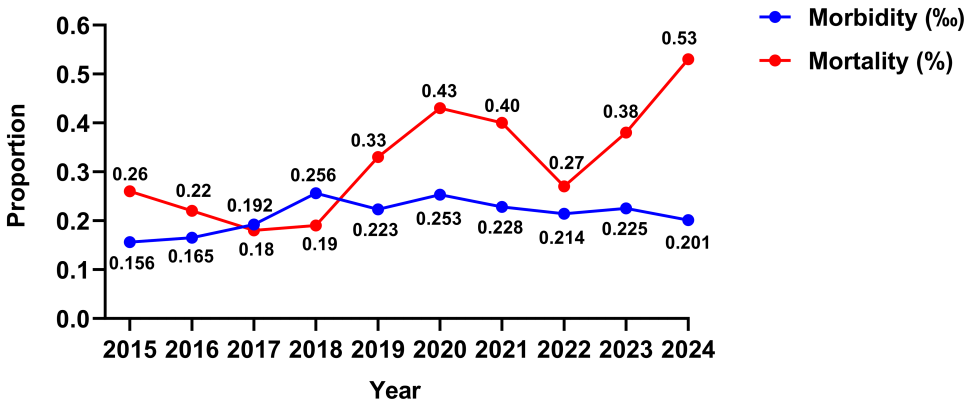

Figure 2 The tendency of candidemia morbidity and mortality rates during 2015–2024.

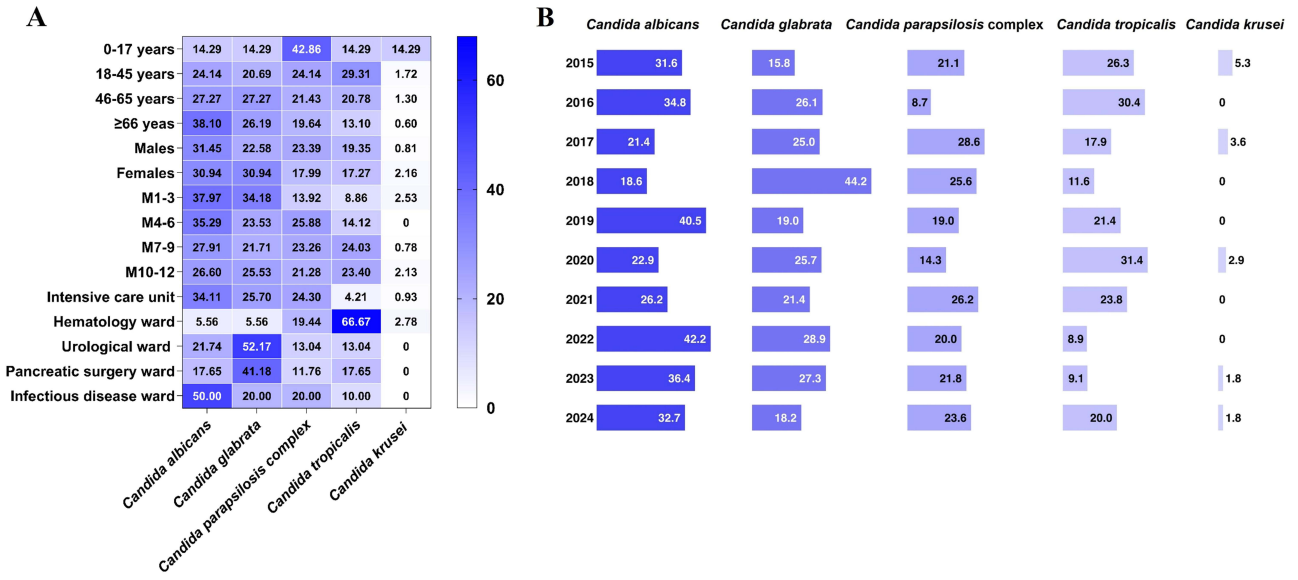

Figure 3 (A) Distribution of the top 5 *Candida* spp. and the top 5 departments of patients' sources of candidemia. **(B)** The evolution of the top 5 *Candida* spp. in candidemia (%) from 2015 to 2024.
Abbreviation: M, month.

C. tropicalis exceeded that of *C. albicans*, becoming the most frequently isolated species in candidemia cases for those years. However, no statistically significant difference was observed in the annual proportion or changing trends between *C. albicans* and non-albicans *Candida* species ($P = 0.213$), as shown in Figure 3B.

In vitro Antifungal Susceptibility Test

The results of antifungal susceptibility testing for 373 *Candida* bloodstream isolates of the four major *Candida* species were summarized in Table 2. Overall, echinocandins exhibited potent in vitro activity against the four major *Candida* species, with susceptibility rates >98.5% (except for a 9.09% intermediate rate to caspofungin observed in *C. glabrata*); but the MIC_{90} of *C. parapsilosis sensu stricto* exhibited 3-6-fold higher MIC_{90} values than those of the other three *Candida* species. Similarly, for amphotericin B, the NWT rate was only 3.70% for *C. parapsilosis sensu stricto*, while the rates for the other three *Candida* species were all 0%. Although none of these four *Candida* species have established CBPs or ECVs for 5-flucytosine, they have relatively low MICs with $MIC_{90} \leq 0.5$ μg/mL. In contrast, susceptibility to azoles varied markedly by species. Both *C. albicans* and *C. parapsilosis sensu stricto* isolates remained susceptible or had WT MICs to voriconazole, fluconazole, and posaconazole (with rates >90%). However, *C. glabrata* generally

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Table 2 Ten-Year Susceptibility Trends of Four Major *Candida* Species Bloodstream Isolates to Nine Antifungal Agents

Candida spp.	Antifungal Agent	MIC (μg/mL)			Antifungal Susceptibility		
		Ranges	MIC_{50}	MIC_{90}	S/WT (n, %)	SDD/I (n, %)	R/NWT (n, %)
Candida albicans (N = 121)	Anidulafungin	0.015–0.25	0.03	0.06	121 (100)	0	0
	Micafungin	0.008–0.06	0.015	0.015	121 (100)	0	0
	Caspofungin	0.015–0.5	0.03	0.12	120 (99.17)	1 (0.83)	0
	Posaconazole	0.015–2	0.03	0.12	109 (90.08)	-	12 (9.92)
	Voriconazole	0.008–8	0.008	0.125	112 (92.56)	6 (4.96)	3 (2.48)
	Itraconazole	0.03–16	0.06	0.125	-	-	-
	Fluconazole	0.03–256	0.5	2	113 (93.39)	3 (2.48)	5 (4.13)
	5- Flucytosine	0.06–64	0.06	0.25	-	-	-
	Amphotericin B	0.25–2	0.5	1	121 (100)	-	0
Candida glabrata (N = 99)	Anidulafungin	0.015–0.12	0.03	0.06	99 (100)	0	0
	Micafungin	0.008–0.03	0.015	0.015	99 (100)	0	0
	Caspofungin	0.008–0.25	0.06	0.12	90 (90.91)	9 (9.09)	0
	Posaconazole	0.06–8	1	2	62 (62.63)	-	37 (37.37)
	Voriconazole	0.03–4	0.25	1	49 (49.49)	-	50 (50.51)
	Itraconazole	0.12–16	0.5	1	97 (97.98)	-	2 (2.02)
	Fluconazole	1–256	16	64	-	89 (89.90)	10 (10.10)
	5- Flucytosine	0.06–0.5	0.06	0.06	-	-	-
	Amphotericin B	0.25-2	1	1	99 (100)	-	0
Candida parapsilosis sensu stricto (N = 81)	Anidulafungin	0.03-4	1	2	80 (98.77)	1 (1.23)	0
	Micafungin	0.015-2	1	2	81 (100)	0	0
	Caspofungin	0.06–2	0.5	1	81 (100)	0	0
	Posaconazole	0.008–0.5	0.03	0.12	80 (98.77)	-	1 (1.23)
	Voriconazole	0.008–1	0.015	0.12	79 (97.53)	0	2 (2.47)
	Itraconazole	0.015–0.5	0.06	0.12	81 (100)	-	0
	Fluconazole	0.25–128	0.5	2	74 (91.36)	2 (2.47)	5 (6.17)
	5- Flucytosine	0.06–1	0.06	0.5	-	-	-
	Amphotericin B	0.25–2	0.5	1	78 (96.30)	-	3 (3.70)
Candida tropicalis (N = 72)	Anidulafungin	0.03–0.25	0.12	0.25	72 (100)	0	0
	Micafungin	0.015–0.06	0.03	0.06	72 (100)	0	0
	Caspofungin	0.015–0.5	0.06	0.25	71 (98.61)	1 (1.39)	0
	Posaconazole	0.03–2	0.25	1	22 (30.56)	-	50 (69.44)
	Voriconazole	0.015–8	0.25	8	36 (50.00)	15 (20.83)	21 (29.17)
	Itraconazole	0.06–16	0.25	1	59 (81.94)	-	13 (18.06)
	Fluconazole	0.12–256	2	256	40 (55.56)	8 (11.11)	24 (33.33)
	5- Flucytosine	0.06–2	0.06	0.12	-	-	-
	Amphotericin B	0.12–1	1	2	72 (100)	-	0

Notes: -: No species-specific clinical breakpoints (CBPs) or epidemiological cutoff values (ECVs) available.

Abbreviations: MIC, minimum inhibitory concentration; MIC_{50}, 50% minimum inhibitory concentration; MIC_{90}, 90% minimum inhibitory concentration; S, susceptible; SDD, susceptible-dose dependent; I, intermediate; R, resistant; WT, wild type; NWT, non-wild-type.

exhibited low susceptibility to azoles, with 37.37% and 50.51% of isolates showing NWT MICs for posaconazole and voriconazole, respectively; for fluconazole, no CBPs or ECVs were available, and its MIC_{90} reached as high as 64 μg/mL. *C. tropicalis* showed the most pronounced trend of resistance or NWT MICs to all four azole agents, with rates of 69.44%, 29.17%, 18.06%, and 33.33% to posaconazole, voriconazole, itraconazole, and fluconazole, respectively. Moreover, 27.78% (20/72) of the *C. tropicalis* isolates exhibited cross-resistance to fluconazole and voriconazole.

Analysis of Risk Factors for in-Hospital Mortality of Candidemia Patients

Among the study subjects, 65.63% (254/387) presented with fever at the time of onset, and 56.33% (218/387) had a history of ICU admission before discharge or death. The vast majority of patients had underlying diseases, with cardiovascular disease being the most common (57.62%), followed by malignant tumors (38.50%), in which solid tumors were predominant (27.91%). Invasive therapeutic interventions were frequently employed, including mechanical ventilation (45.74%), body cavity drainage tube (43.15%), surgical operation (41.60%), indwelling urinary catheter (40.05%), central venous catheterization (29.46%), and total parenteral nutrition (28.17%). Concurrent bacteremia was documented in 100 patients (25.84%), while antibiotic therapy had been administered to 90.18% (349/387) of patients preceding the onset of candidemia. Following confirmation, 301 patients (77.78%) received guideline-directed antifungal therapy, primarily comprising azoles (fluconazole or voriconazole), echinocandins (caspofungin), and amphotericin B. Among these, 76 patients (25.25%) received combination therapy based on complex clinical scenarios, encompassing unsatisfactory antifungal monotherapy response, persistent candidemia, *Candida* endocarditis or central nervous system involvement. The remaining 86 patients received no antifungal therapy, of whom 31 died in-hospital due to poor baseline conditions prior to treatment initiation; the other 55 were discharged alive (39 transferred to local hospitals and 16 against medical advice). The Clinical characteristics of surviving and non-surviving patients with candidemia were listed in Table 3.

Patients who survived were often those with *C. parapsilosis* complex infection, and voriconazole monotherapy. In contrast, the nonsurvivor group was typically older, had an ICU admission history, and more frequently presented with fever, cardiovascular disease, acute or chronic renal failure, mechanical ventilation, total parenteral nutrition, blood product

Table 3 Comparison of Clinical Characteristics Between Survivor and Non-Survivor Groups in Candidemia Patients

Characteristics	Total	Survivor	Nonsurvivor	$\chi 2$	*P*
	N = 387 (%)	n = 256 (%)	n = 131 (%)		
Male	248 (64.08)	161 (62.89)	87 (66.41)	0.467	0.494
≥63 years	195 (50.39)	115 (44.92)	80 (61.07)	9.038	0.003*
Fever	254 (65.63)	159 (62.11)	95 (72.52)	4.163	0.041*
ICU admission	218 (56.33)	115 (44.92)	103 (78.63)	40.017	<0.001*
Co-existent bacteremia	100 (25.84)	64 (25.00)	36 (27.48)	0.278	0.598
Causative pathogens of candidemia					
Candida albicans	121 (31.27)	76 (29.69)	45 (34.35)	0.877	0.349
Candida glabrata	99 (25.58)	64 (25.00)	35 (26.72)	0.134	0.714
Candida parapsilosis complex	83 (21.44)	64 (25.00)	19 (14.50)	5.667	0.017*
Candida tropicalis	72 (18.60)	44 (17.19)	28 (21.37)	1.003	0.317
Others	12 (3.11)	8 (3.13)	4 (3.05)	0.001	0.969
Underlying diseases					
Cardiovascular disease	223 (57.62)	131 (51.17)	92 (70.23)	12.889	<0.001*
Solid tumor	108 (27.91)	71 (27.73)	37 (28.24)	0.011	0.916
Leukemia and lymphoma	41 (10.59)	26 (10.16)	15 (11.45)	0.153	0.695
Diabetes mellitus	109 (28.17)	70 (27.34)	39 (29.77)	0.252	0.615
Acute/Chronic renal failure	79 (20.41)	35 (13.67)	44 (33.59)	21.157	<0.001*
Hepatitis	29 (7.49)	16 (6.25)	13 (9.92)	1.687	0.194
Neutropenia	24 (6.20)	15 (5.86)	9 (6.87)	0.152	0.696
Invasive procedure					
Mechanical ventilation	177 (45.74)	90 (35.16)	87 (66.41)	34.111	<0.001*
Body cavity drainage tube	167 (43.15)	111 (43.36)	56 (42.75)	0.013	0.908
Surgical operation	161 (41.60)	115 (44.92)	46 (35.11)	3.431	0.064
Indwelling urinary catheter	155 (40.05)	97 (37.89)	58 (44.27)	1.471	0.225
Central venous catheter	114 (29.46)	69 (26.95)	45 (34.35)	0.282	0.131
Total parenteral nutrition	109 (28.17)	60 (23.44)	49 (37.40)	8.355	0.004*

(*Continued*)

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Table 3 (Continued).

Characteristics	Total	Survivor	Nonsurvivor	$\chi 2$	*P*
	N = 387 (%)	n = 256 (%)	n = 131 (%)		
Pre-Candidemia					
Antibiotic exposure	349 (90.18)	227 (88.67)	122 (93.13)	1.945	0.163
Blood product transfusion	286 (73.90)	172 (67.19)	114 (87.02)	17.677	<0.001*
Corticosteroid therapy	58 (14.99)	40 (15.63)	18 (13.74)	0.242	0.623
Receipt of immunosuppressants	49 (12.66)	33 (12.89)	16 (12.21)	0.036	0.850
Post-Candidemia					
Antifungal therapy	301 (77.78)	201 (78.52)	100 (76.34)	0.238	0.625
Antifungal regimen					
Fluconazole	51 (13.18)	37 (14.45)	14 (10.69)	1.074	0.300
Voriconazole	78 (20.16)	59 (23.05)	19 (14.50)	3.930	0.047*
Caspofungin	94 (24.29)	57 (22.27)	37 (28.24)	1.684	0.194
Fluconazole + Caspofungin	29 (7.49)	13 (5.08)	16 (12.21)	6.365	0.012*
Voriconazole + Caspofungin	37 (9.56)	26 (10.16)	11 (8.40)	0.310	0.578
Amphotericin B monotherapy	2 (0.52)	2 (0.78)	0 (0.00)	1.029	0.310
Amphotericin B combination therapy	10 (2.58)	5 (1.95)	5 (3.82)	1.196	0.274

Note: * $P < 0.05$.
Abbreviation: ICU, intensive care unit.

transfusion, and combination therapy with fluconazole and caspofungin (all $P < 0.05$). In the multivariate Cox regression model, age ≥63 years (*HR* = 1.530, 95% *CI*: 1.077–2.174, $P = 0.018$), ICU admission history (*HR* = 2.005, 95% *CI*: 1.200–3.350, $P = 0.008$), and acute or chronic renal failure (*HR* = 1.849, 95% *CI*: 1.261–2.711, $P = 0.002$) were identified as independent risk factors that significantly increased the risk of in-hospital mortality in patients with candidemia. In contrast, *C. parapsilosis* complex infection (*HR* = 0.456, 95% *CI*: 0.275–0.756, $P = 0.002$) and antifungal therapy (*HR* = 0.313, 95% *CI*: 0.194–0.505, $P = 0.001$) were independently associated with significantly reduced risks (Figure 4).

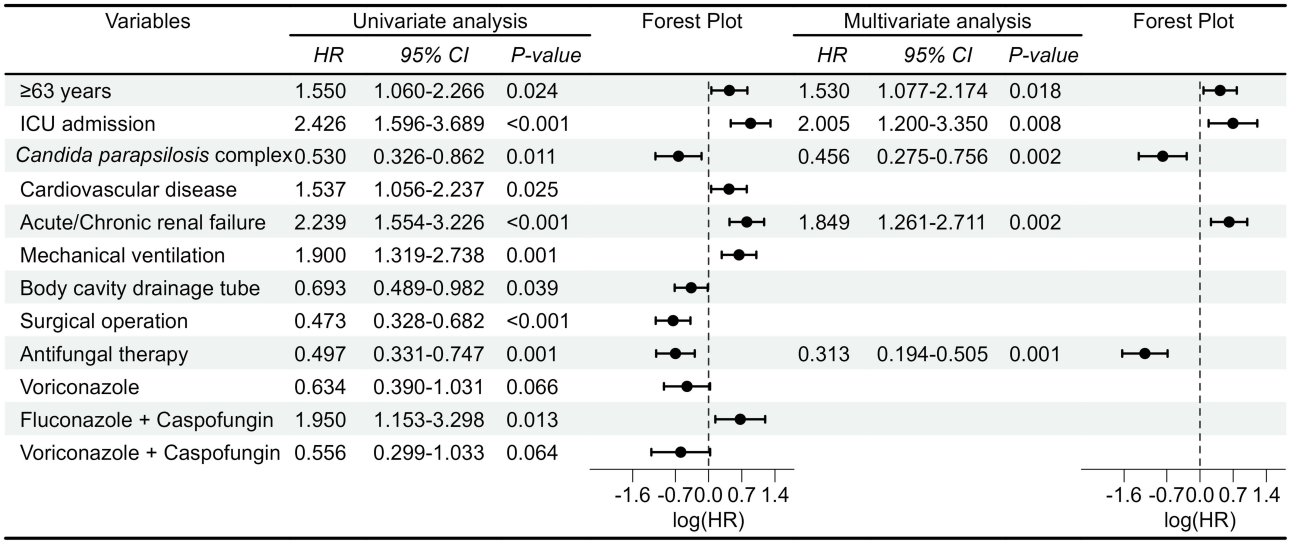

Figure 4 Univariate and multivariate Cox regression analysis of in-hospital mortality in patients with candidemia.
Abbreviations: ICU, intensive care unit; HR, hazard ratio; CI, confidence interval.

Discussion

In our retrospective study of 387 candidemia patients over the past decade, the overall in-hospital mortality rate was 33.85%. We report a distinct epidemiological profile, clinical characteristics, and antifungal susceptibility patterns for this cohort. Finally, we identified advanced age, ICU admission, renal dysfunction, *C. parapsilosis* complex infection, and antifungal agent administration as five independent predictors for in-hospital mortality.

Compared to other domestic regions, the cumulative incidence of candidemia in our hospital was lower than the 0.33/1,000 admissions reported by a teaching hospital in Beijing from 2015 to 2022, but the mortality rate is similar (32.8%).[20] Meanwhile, a single-center study in Shanghai from 2008 to 2018 showed an incidence of 0.39/1,000 admissions and a mortality rate of 28.5%.[21] In addition, a multicenter study carried out in Southwest China from 2016 to 2021 reported a lower incidence of 0.15/1,000 admissions, along with a correspondingly lower mortality rate of 20.2%.[22] These differences may be attributed to variations in medical resource allocation, infection control measures, laboratory diagnostic capacity, and antimicrobial use strategies across institutions. In addition, we observed a significant increase in mortality from 2019 to 2022. This trend might be associated with the widespread use of immunosuppression and invasive ventilation, and prolonged hospitalization in critically ill COVID-19 patients against the condition of the global and domestic pandemic, consistent with other studies.[23] Notably, the mortality rate further rose to 53% in 2024. We attributed this primarily to limited antifungal options, poorer baseline status, and compromised immune competence in these patients, as evidenced by a high-risk profile characterized by elderly age (34/55, 61.82%), ≥2 underlying diseases (37/55, 67.27%), and frequent ICU admission (42/55, 76.36%).

Overall, *C. albicans* remained the most common pathogen causing candidemia. Although the proportion of non-albicans *Candida* species in our study was nearly 70%, higher than that reported in other literature and consistent with the global tendency,[9,24] their annual variation trend was not statistically significant ($P = 0.213$). According to data from CHIF-NET 2015–2017, *C. parapsilosis* complex was the most common species among non-albicans *Candida* bloodstream infections;[9] however, *C. glabrata* was the dominant non-albicans *Candida* species in this study, accounting for 25.58%, which was consistent with the species distribution reported in regions of Beijing and Western countries.[20,25,26] Despite the first isolation of *C. auris* at our institution in 2024, which accounted for a minimal proportion (0.26%) of candidemia episodes, it still warranted attention. As a multidrug-resistant pathogen, *C. auris* exhibits high-level resistance to commonly used azole antifungals such as fluconazole and voriconazole, with a propensity for nosocomial transmission.[27] Therefore, even a single identified case necessitates enhanced active surveillance and optimized infection control strategies to mitigate its potential spread among high-risk populations, particularly critically ill patients.

Beyond geographical factors, the distribution of *Candida* species is also influenced by patients' age, underlying diseases, hospital-specific factors, and even climatic conditions.[28] Our clinical data indicated that *C. albicans* predominated in patients over 46 years of age, those admitted in the first half of the year, and inpatients in the ICU and department of infectious diseases. The distribution of non-albicans *Candida* species, however, showed distinct variations. *C. tropicalis* exhibited the highest detection rate in the hematology ward (66.67%), which was highly consistent with previous findings.[29,30] Further mechanistic studies attributed this phenomenon to endogenous infection by *C. tropicalis*. The proposed mechanism was that in hematologic malignancy patients, especially those high-risk populations with concurrent mucosal inflammation or chemotherapy-induced neutropenia and other immunosuppressive states, virulent *C. tropicalis* from the gut microbiota could translocate into the bloodstream, resulting in invasive infection.[22,31] In contrast, the urology and pancreatic surgery wards were predominantly characterized by *C. glabrata*. This pattern was considered attributable to the higher proportion of elderly patients in these units, who often exhibited impaired skin and mucosal barrier function and frequently underwent invasive procedures such as central venous catheterization and urinary catheterization, thereby creating favorable conditions for colonization and infection by *C. glabrata*.[32] Furthermore, infections caused by the *C. parapsilosis* complex in this study were most prevalent among adolescent patients, consistent with literature reports.[33] Infections with this species are mostly exogenous and closely associated with the widespread use of total parenteral nutrition and suboptimal hand hygiene practices.[33,34]

According to CHIF-NET surveillance data, antifungal agents other than azoles tested exhibited high susceptibility against common *Candida* species, while azole-resistant *C. tropicalis* showed a continuous and rapid upward trend, with

resistance rates rising from 6%–20% during 2009–2014 to 30% in 2015–2017.[35] These data underscore that azole resistance has already become a critical issue of global concern. In our hospital, *C. tropicalis* showed relatively high resistance to four azoles, with 29.17%, 33.33%, 69.44% and 18.06% being resistant or having NWT MICs to voriconazole, fluconazole, posaconazole, and itraconazole, respectively. The pattern was in line with CHIF-NET data and the reports from other studies in China.[20,36] Additionally, we found higher MIC_{90} values for voriconazole (8 μg/mL) and fluconazole (256 μg/mL) against *C. tropicalis* relative to a previous investigation.[36] The increasing resistance in *C. tropicalis* might be driven by the favorable safety profile, cost-effectiveness, and excellent tissue penetration of azoles, which has led to their widespread use for empirical and prophylactic treatment by clinicians.[37] Furthermore, we observed notable cross-resistance between voriconazole and fluconazole in 27.78% of *C. tropicalis* isolates in our study, which might be associated with modifications in the azole target Erg11p or increased efflux pump activity.[38] These findings suggested that voriconazole or fluconazole might be suboptimal for empirical therapy of suspected *C. tropicalis* infections, which was consistent with reports linking azole usage to higher initial failure rates in such infections.[29,39] According to the IDSA 2016 guidelines, echinocandins were recommended as first-line agents for invasive candidiasis (including candidemia), whereas amphotericin B and voriconazole were more frequently used in subsequent targeted therapy;[12] and this recommendation has been strongly reaffirmed in the recent global guideline.[40] Therefore, combined with the clinical data from this study, our findings could serve as a reference for clinicians in selecting empirical antifungal therapy for different types of candidemia.

Candidemia is associated with high morbidity and mortality, particularly in critically ill or immunocompromised patients.[41] Previous studies have shown that age, renal impairment, and ICU admission are independent predictors of mortality from candidemia,[7,42] these findings were consistent with our results. ICU patients were often older, immunocompromised, subjected to invasive procedures, and suffered from severe underlying conditions such as renal insufficiency. Therefore, heightened vigilance in clinical practice is necessary to mitigate the risk of candidemia in elderly ICU patients. We also noted a considerably lower case-fatality rate among patients with *C. parapsilosis* complex candidemia in our study, a finding corroborated by research from Tadeja Matos et al.[43] The younger age of patients with *C. parapsilosis* complex might partially explain this difference; however, other unmeasured confounding factors, such as differing rates of catheter management, underlying illness severity, or timing of effective therapy, could also contribute significantly. Thus, the observed association likely represents a multifactorial relationship rather than solely reflecting the pathogen's intrinsic virulence. Additionally, our study revealed that antifungal therapy was independently associated with significantly reduced in-hospital mortality. This finding underscored the critical importance of timely and appropriate antifungals administration in improving outcomes for candidemia patients. Previous studies have consistently shown that delayed initiation of effective antifungal therapy was associated with increased mortality.[44,45] For patients with highly suspected or confirmed candidemia, especially those with risk factors, local epidemiological trends and antifungal resistance profiles can guide clinicians in delivering a rapid empirical antifungal regimen.

This study has several limitations that should be acknowledged. First, its retrospective and single-center design might restrict the generalizability of our findings to other institutions and regions. Second, due to the constraints of the retrospective data, the precise timing of antifungal treatment initiation could not be reliably extracted and analyzed for all patients. Third, our analysis was confined to phenotypic antifungal susceptibility testing and could provide guiding significance for clinical practice; however, molecular investigations to elucidate the underlying resistance mechanisms were not performed.

Conclusion

The epidemiological landscape of candidemia in our institution was characterized by the predominance of non-albicans *Candida* species, high resistance rates in *C. tropicalis*, and the emergence of *C. auris*. The urgent medical circumstances of the COVID-19 pandemic might be closely associated with the observed rise in mortality during that period. These findings underscore the necessity of tailoring empirical antifungal strategies based on local pathogen distribution and susceptibility data. Targeted interventions should be prioritized for high-risk patients, including those with advanced age, renal failure, or admitted to the ICU, to improve clinical outcomes.

Data Sharing Statement

The raw data supporting the conclusions of this article will be made available by Jue Wang, without undue reservation.

Ethics Statement

This study was reviewed and approved by the Research Ethics Committee of the First Affiliated Hospital with Nanjing Medical University (No. 2026-SR-297) and complied with the Declaration of Helsinki. This retrospective study utilized only objective data from patients' routine clinical care, which met our institution's policy for granting an exemption from informed consent. The exemption applied to all participants, including parents/guardians of minors, as the study involved no more than minimal risk and did not adversely affect patients' rights and welfare.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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