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Research Article

Candidemia in Pediatric Hematology–Oncology Patients: Risk Factors, Microbiological Characteristics, and Predictors of Mortality

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Abstract

Objectives: Invasive fungal infections remain a major cause of morbidity and mortality in pediatric hematology–oncology patients, with candidemia representing the most common invasive fungal infection in this population. However, data on clinical characteristics, species distribution, and outcomes remain limited. This study evaluates the clinical and microbiological features, antifungal susceptibility patterns, and outcomes of candidemia in pediatric hematology–oncology patients at a tertiary care center.

Methods: This retrospective cohort study included pediatric patients with hematologic or solid malignancies hospitalized between 2020 and 2025 and evaluated clinical characteristics, candidemia occurrence, Candida species distribution, antifungal susceptibility, and 30-day mortality.

Results: Among 926 pediatric oncology patients, candidemia occurred in 8.5% of cases and was associated with younger age, neutropenia, intensive care unit admission, and increased mortality, while sex and type of malignancy showed no significant association. Multivariate analysis identified younger age, neutropenia, and intensive care unit admission as independent predictors of candidemia, whereas candidemia independently increased the risk of 30-day mortality more than twofold. Candida albicans was the most common isolate, followed by non-albicans species, and amphotericin B susceptibility was largely preserved across isolates.

Conclusion: Pediatric hematology–oncology patients with candidemia face significantly increased morbidity and mortality, with independent risk factors including younger age, neutropenia, and intensive care admission, and mortality predictors including younger age, female sex, thrombocytopenia, and candidemia itself. Non-albicans Candida species accounted for over half of the isolates, while amphotericin B remained highly effective. These findings highlight the need for early risk identification, targeted prophylaxis, and prompt antifungal therapy in this vulnerable population.

Keywords: Amphotericin B, Invasive Fungal Infections, Neutropenia, Non-albicans Candida

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Invasive fungal infections represent a major source of adverse clinical outcomes, including significant morbidity and mortality, among patients with compromised immune function.[1] Pediatric patients with malignancies are at increased risk because profound secondary immunosuppression results from the disease itself, intensive chemotherapy regimens, corticosteroid exposure, and hematopoietic stem cell transplantation.[2,3] Defective host

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immune defenses, heightened exposure to nosocomial pathogens, chemotherapy-related mucosal damage, and alteration of the normal gastrointestinal microbiota are key contributors to the development of fungal infections in this population.[4,5] Additional factors contributing to infection risk include prolonged stays in intensive care units, the use of central venous lines, total parenteral nutrition, broad-spectrum antibiotic therapy, invasive interventions, and cytostatic therapies.[6,7]

Despite improvements in pediatric cancer management, invasive fungal diseases remain a significant source of adverse outcomes in pediatric patients with high-risk hematologic malignancies.[8] Reported incidence rates differ markedly across geographical regions and are influenced by the type of underlying malignancy, the intensity of treatment, and individual patient characteristics.[9] The prevalence of invasive fungal diseases among pediatric oncology patients is estimated at approximately 5.9%, with published rates ranging from 2 to 28%, and the highest burden reported in children with acute myeloid leukemia (AML) and those receiving allogeneic hematopoietic stem cell transplantation.[10-14] Reliable prevalence estimates are difficult to obtain because of wide variation in the nature of immune impairment, inconsistencies in diagnostic definitions, and differences in local clinical and laboratory practices.[15]

Candida species normally colonize the gastrointestinal tract but can invade the bloodstream following mucosal barrier damage or antibiotic-induced disturbances of the resident microbiota.[16] Invasive candidiasis represents the leading invasive fungal disease, and candidemia accounts for the majority of its clinical cases.[17] Candidemia represents the most frequent invasive fungal infection in hospitalized pediatric patients and is among the leading causes of hospital-acquired bloodstream infections globally.[18] In pediatric intensive care units, the incidence of candidemia is estimated at roughly five cases per one thousand admissions and is associated with considerable morbidity and mortality, including extended hospital stays and increased need for intensive care support.[15,19,20] Infections are most often caused by a limited group of species, including *Candida albicans, Candida tropicalis, Candida parapsilosis, Candida glabrata, Candida krusei*, and *Candida auris*.[21] While *C. albicans* continues to be a leading pathogen, accumulating evidence suggests an increasing predominance of non-albicans *Candida species* among pediatric hematology–oncology patients.[7] This change in epidemiology has coincided with the emergence of antifungal resistance, especially in clinical contexts involving extended hospital stays, use of central venous catheters, administration of total parenteral nutrition, and exposure to broad-spectrum antimicrobial agents.[22]

Therapeutic strategies for invasive candidiasis encompass amphotericin B formulations, triazole antifungals, and echinocandins, with current guidelines favoring echinocandins or amphotericin B as first-line therapy in immunocompromised pediatric patients with malignancies.[23,24] Nevertheless, candidemia remains a frequent and potentially life-threatening complication. Data on clinical characteristics, species distribution, and antifungal susceptibility patterns in pediatric hematology–oncology patients remain limited, particularly in real-world settings. Therefore, this study aims to evaluate the clinical features, microbiological characteristics, antifungal susceptibility patterns, and outcomes of candidemia episodes in pediatric hematology–oncology patients treated at a tertiary care center.

Methods

This retrospective cohort study included pediatric patients with hematologic or solid organ malignancies who were hospitalized in the pediatric hematology–oncology unit of a tertiary care hospital in Türkiye between January 2020 and December 2025. Demographic and clinical characteristics, including age, sex, underlying malignancy, presence of neutropenia and thrombocytopenia, requirement for intensive care unit admission, blood culture findings, and patient outcomes, were retrospectively extracted from electronic medical records. Underlying malignancies were classified as hematologic or solid organ malignancies based on their tissue of origin and disease biology. Hematologic malignancies included leukemia and lymphoma, whereas solid organ malignancies comprised tumors arising from non-hematopoietic tissues, such as central nervous system tumors, neuroblastoma, and sarcomas. Candidemia was diagnosed when *Candida species* were isolated from blood cultures drawn from a peripheral vein and/or a central venous catheter in the presence of clinical signs consistent with bloodstream infection, including fever or hypotension. Patients were stratified into two groups according to the development of candidemia: those with candidemia and those without candidemia. In the candidemia group, the time interval between hospital admission and the onset of candidemia was also evaluated. Furthermore, *Candida species* distribution and antifungal susceptibility results were evaluated in patients who developed candidemia. The outcome of interest was 30-day mortality, defined as death within 30 days of the first positive blood or catheter culture, without attributing causality to candidemia. Ethical approval was obtained from the Gaziantep University Clinical Research Ethics Committee (Approval No. 2026/46, issued January 21, 2026), and the requirement for informed consent was waived due to the retrospective nature of the study. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Table 1. Comparison of demographic and clinical characteristics between patients with and without candidemia

Parameters	Non-candidemia	Candidemia	p	Effect size
Age	7.92±3.73	5.48±4.64	[b]0.001*	0.638
Sex (n,%)				
Male	423 (49.9)	44 (56.4)	[a]0.270	0.036
Female	425 (50.1)	34 (43.6)		
Type of malignancy (n, %)				
Solid organ	169 (19.9)	14 (17.9)	[a]0.674	0.014
Hematologic	679 (80.1)	64 (82.1)		
Neutropenia (n, %)				
Absent	367 (43.3)	18 (23.1)	[a]0.001*	0.129
Present	481 (56.7)	60 (76.9)		
Thrombocytopenia (n, %)				
Absent	422 (49.8)	18 (23.1)	[a]0.001*	0.114
Present	426 (50.2)	60 (76.9)		
ICU Admission (n, %)				
Absent	683 (80.5)	42 (53.8)	[a]0.001*	0.148
Present	165 (19.5)	36 (46.2)		
Prognosis (n, %)				
Discharge	596 (70.3)	38 (48.7)	[a]0.001*	0.180
Mortality	252 (29.7)	40 (51.3)		

[a]: Chi-Square Analysis; *: p<0.05; Cramer V used for effect size. [b]: Independent Sample T Test; Cohen's d used for effect size. ICU: Intensive care unit.

Statistical Analysis

Statistical analyses were performed using SPSS Statistics Version 29.0 (Armonk, New York: IBM Corp.). Normality of continuous variables was assessed with the Kolmogorov–Smirnov test. Normally distributed variables were presented as mean±standard deviation and compared using the independent samples t-test, with effect size calculated by Cohen's d. Categorical variables were expressed as numbers and percentages, compared using the chi-square test, and effect size was evaluated with Cramer's V. Variables with p<0.10 by univariate analysis were entered into a multivariate binary logistic regression model. Independent risk factors were reported as odds ratios with 95% confidence intervals. Model fit was assessed using Nagelkerke R square. A two-tailed p value of less than 0.05 was considered statistically significant.

Results

A total of 926 unique patients who met the inclusion criteria were enrolled in the study. Of these, 91.5% (n=848) were without candidemia and 8.5% (n=78) had candidemia. The mean age was 5.48±4.64 years in the candidemia group and 7.92±3.73 years in the non-candidemia group (p=0.001). The mean time from hospital admission to candidemia onset was 14.3 days. Significant differences between the groups were observed in the presence of neutropenia, thrombocytopenia, intensive care unit admission, and mortality. There were no statistically significant differences regarding sex or type of malignancy. A comparison of the demographic and clinical characteristics of patients with and without candidemia is presented in Table 1.

When primary diagnoses were evaluated, the most common malignancy in both groups was acute lymphoblastic leukemia (ALL). In the non-candidemia group, the rate of ALL was 60.0% (n=509), whereas in the candidemia group, it was 71.8% (n=56). AML was observed in 14.9% (n=126) of the non-candidemia group and 5.1% (n=4) of the candidemia group.

Among solid organ malignancies, medulloblastoma was observed at similar rates in both groups, 2.4% and 2.6%, respectively. Neuroblastoma was detected in 5.1% of the candidemia group and 2.6% of the non-candidemia group. Rhabdomyosarcoma was present in 2.6% of the candidemia group and 5% of the non-candidemia group. The distribution of primary diagnoses in patients with and without candidemia is presented in Table 2.

According to the results of the binary logistic regression analysis performed to evaluate factors associated with the development of candidemia, age, presence of neutropenia, and intensive care unit admission were found to be significant in both univariate and multivariate

Table 2. The distribution of primary diagnoses in patients with and without candidemia

Malignancy	Non-candidemia (n, %)	Candidemia (n, %)
Acute lymphoblastic leukemia	509 (60.0)	56 (71.8)
Acute myeloid leukemia	126 (14.9)	4 (5.1)
B cell lymphoma	22 (2.6)	2 (2.6)
Brainstem ependymoma	0 (0.0)	2 (2.6)
Medulloblastoma	20 (2.4)	2 (2.6)
Non Hodgkin lymphoma	0 (0.0)	2 (2.6)
Neuroblastoma	22 (2.6)	4 (5.1)
Pancreatic carcinoma	0 (0.0)	2 (2.6)
Rhabdomyosarcoma	42 (5.0)	2 (2.6)
Wilms tumor	22 (2.6)	2 (2.6)
T cell lymphoma	22 (2.6)	0 (0.0)
Retinoblastoma	20 (2.4)	0 (0.0)
Nasopharyngeal tumor	22 (2.6)	0 (0.0)
Malignant mesenchymal tumor	21 (2.5)	0 (0.0)

analyses. Age was identified as an independent factor inversely associated with the likelihood of developing candidemia. Neutropenia emerged as an independent risk factor and increased the risk of candidemia by approximately 2.5-fold (OR=2.55). In contrast, thrombocytopenia was not significantly associated with candidemia. Multivariate analysis also showed no significant association between hematologic malignancy and the development of candidemia in this dataset. The results of the logistic regression analysis for candidemia development are presented in Table 3.

According to the results of the binary logistic regression analysis performed to evaluate factors associated with mortality, independent risk factors were younger age, female sex, thrombocytopenia, and candidemia. Hematologic malignancy was associated with a lower risk of mortality compared with solid organ malignancy. Neutropenia was not identified as an independent predictor of mortality. One of the most clinically significant findings was that candidemia increased the odds of mortality by more than twofold (OR=2.14), independent of other variables. The results of the logistic regression analysis for mortality are presented in Table 4.

The distribution of *Candida species* among the 78 candidemia episodes revealed that *C. albicans* was the predominant isolate (n=37, 47.4%). This was followed by C. glabrata (n=13, 16.7%) and C. tropicalis (n=11, 14.1%). C. auris was identified in 4 cases (5.1%). Less frequently isolated species included *C. parapsilosis* (n=2, 2.6%), *Candida kefyr* (n=2, 2.6%), *C. krusei* (n=2, 2.6%), *Candida pelliculosa* (n=2, 2.6%), *Candida sake* (n=2, 2.6%), *Candida guilliermondii* (n=2, 2.6%), and *Candida lusitaniae* (n=1, 1.3%). Overall, non-albicans *Candida species* accounted for 52.6% of all isolates. Amphotericin B susceptibility was preserved across nearly all isolates and emerged as the most effective antifungal option. Resistance to amphotericin B was detected in only

Table 3. Logistic regression analysis of factors associated with candidemia development

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age	0.82 (0.76-0.88)	0.001*	0.84 (0.77-0.90)	0.001*
Sex				
Male	1	-		
Female	0.76 (0.48-1.22)	0.271		
Type of malignancy				
Solid organ	1	-		
Hematologic	1.13 (0.62-2.07)	0.674		
Neutropenia				
Absent	1	-	1	-
Present	2.54 (1.47-4.38)	0.001*	2.55 (1.23-5.29)	0.012*
Thrombocytopenia				
Absent	1	-	1	-
Present	3.30 (1.91-5.68)	0.001*	1.09 (0.50-2.38)	0.811
ICU admission				
Absent	1	-	1	-
Present	3.54 (2.20-5.71)	0.001*	2.12 (1.23-3.66)	0.001*

Logistic Regression Analysis; *: $p<0.05$; R^2: 0.15. OR: Odds ratio; CI: Confidence interval.

Table 4. Logistic regression analysis for mortality predictors

Parameters	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Age	0.93 (0.89-0.96)	0.001*	0.93 (0.89-0.97)	0.001*
Sex				
Male	1	-	1	-
Female	1.69 (1.28-2.24)	0.001*	1.51 (1.11-2.04)	0.008*
Type of malignancy				
Solid organ	1	-	1	-
Hematologic	0.37 (0.26-0.52)	0.001*	0.34 (0.24-0.50)	0.001*
Neutropenia				
Absent	1	-		
Present	1.21 (0.91-1.61)	0.177		
Thrombocytopenia				
Absent	1	-	1	-
Present	1.68 (1.27-2.23)	0.001*	1.45 (1.07-1.96)	0.016*
Candidemia				
Absent	1	-	1	-
Present	2.49 (1.55-3.97)	0.001*	2.14 (1.30-3.53)	0.003*

Logistic Regression Analysis; *: $p<0.05$; R^2: 0.12. OR: Odds ratio; CI: Confidence interval.

one isolate of *C. auris* and one isolate of *C. lusitaniae*. The detailed distribution of *Candida species* and antifungal susceptibility results is presented in Table 5.

Discussion

In children and adults, candidemia is predominantly attributed to a small group of *Candida species*, with *C. albicans, C. parapsilosis, C. glabrata, C. tropicalis*, and *C. krusei* accounting for more than 90% of infections.[25] Beginning in the 1990s, a notable global trend emerged in candidemia epidemiology, marked by a reduction in *C. albicans* infections alongside an increase in infections due to non-albicans *Candida species*.[26] This epidemiological shift has been partially explained by the widespread administration of azole agents for prophylaxis and treatment.[18] Consistent with the global epidemiological shift toward non-albicans *Candida species*, non-albicans *Candida* isolates constituted 52.6% of cases in this study. Among these species, some isolates demonstrated complete loss of susceptibility to azole antifungals beyond intrinsic resistance mechanisms.

Invasive fungal disease is a major driver of poor outcomes in neutropenic patients with hematologic malignancies.[27] The prognosis of patients with candidemia is highly dependent on the prompt administration of antifungal therapy.[28] Antifungal treatment is generally initiated empirically following the detection of *Candida* in blood cultures and subsequently tailored according to species-level identification and susceptibility profiles.[7] Antifungal treatment should be directed by susceptibility results following species-level identification. In most cases, echinocandins remain the first-line therapy, whereas fluconazole is often preferred for C. parapsilosis.[29] An important finding of the present study is the potential clinical impact of echinocandin resistance among C. auris, C. glabrata, and C. tropicalis, which collectively constituted 35.9% of all isolates. The presence of echinocandin resistance within this substantial proportion of isolates may pose a serious therapeutic challenge, particularly given the current recommendation of echinocandins as first-line therapy for candidemia.

In adult patients with hematologic malignancies who develop candidemia, reported mortality rates range between 40% and 45%.[30] Mortality rates in pediatric populations show considerable variability. A surveillance study found that overall mortality among hematologic-oncologic patients was 30.9%, with candidemia-specific mortality of 11.4%.[31] Another study reported that invasive fungal infections and candidemia occurred in 7.2% and 1.4% of pediatric oncology patients, respectively, with an associated mortality of 21.7%.[32] In our study, mortality among pediatric patients with malignancy was 29.7% in those without candidemia, whereas the mortality rate increased significantly to 51.3% in patients who developed candidemia. The wide variation in reported mortality rates across different studies highlights the importance of cohort studies conducted in well-defined and homogeneous patient populations.

Table 5. Distribution of *Candida species* and antifungal susceptibility results

Microorganisms	Antifungal agents susceptibility rates (%)								
	Amphotericin B	Anidulafungine	Caspofungin	Micafungin	Flucytosine	Fluconazole	Voriconazol	Itraconazole	Posaconazole
C. albicans (n=37)	100%	100%	100%	100%	100%	89.1%	89.1%	100%	100%
C. auris (n=4)	75%	0%	0%	0%	0%	0%	0%	0%	0%
C. glabrata (n=13)	100%	46.1%	38.4%	61.5%	100%	0%	0%	0%	30.7%
C. guilliermondii (n=2)	100%	100%	100%	100%	100%	100%	100%	100%	100%
C. kefyr (n=2)	100%	100%	100%	100%	100%	100%	100%	100%	100%
C. krusei (n=2)	100%	100%	100%	100%	0%	0%	100%	100%	100%
C. lusitaniae (n=1)	0%	100%	100%	100%	0%	100%	100%	100%	100%
C. parapsilosis (n=2)	100%	100%	100%	100%	100%	0%	0%	0%	100%
C. pelliculosa (n=2)	100%	100%	0%	100%	100%	100%	100%	100%	100%
C. sake (n=2)	100%	100%	100%	100%	100%	100%	100%	100%	100%
C. tropicalis (n=11)	100%	100%	81.8%	81.8%	81.8%	81.8%	100%	100%	81.8%

Patients with hematologic or solid organ malignancies represent a population at high risk for developing candidemia.[33] Children with leukemia or lymphoma undergoing induction therapy exhibit particularly high rates of fungal colonization.[34] Pediatric patients are often predisposed to fungal infections due to exposure to broad-spectrum antibiotics, corticosteroids, chemotherapeutic drugs, invasive procedures, and extended periods of neutropenia.[35] There is a lack of sufficient studies evaluating the development of candidemia specifically in pediatric hematology–oncology patients. In this study, age, neutropenia, and intensive care unit admission were identified as independent risk factors for the development of candidemia. However, several studies have evaluated prognostic factors in candidemia. In a prospective cohort study, neutropenia and endotracheal intubation were identified as independent predictors of mortality.[36] In a patient-level quantitative review, neutropenia was negatively associated with clinical outcomes and survival in univariate analysis, yet it lost significance in multivariate models.[37] Cancer patients developing candidemia demonstrate significant mortality regardless of neutropenia status, and overall mortality rates may be similar in both neutropenic and non-neutropenic individuals.[38] Consistent with previous reports, neutropenia was significantly associated with mortality in the univariate analysis but did not retain significance in the multivariate model. In contrast, thrombocytopenia emerged as a notable independent predictor of mortality, remaining significantly associated with poor outcomes in both univariate and multivariate analyses.

Conclusion

In this study, pediatric hematology–oncology patients with candidemia were found to have a substantially higher risk of morbidity and mortality, highlighting the vulnerability of this population. Independent risk factors for the development of candidemia included age, neutropenia, and intensive care unit admission, while independent predictors of mortality were younger age, female sex, thrombocytopenia, and candidemia. Notably, non-albicans *Candida species* constituted 52.6% of all isolates, reflecting the ongoing epidemiological shift observed globally. Amphotericin B retained activity against nearly all isolates, emerging as the most reliable antifungal therapy in this cohort. Given the high-risk nature of pediatric hematology–oncology patients, these findings underscore the need for early identification of at-risk individuals, targeted prophylactic strategies, and prompt initiation of effective antifungal therapy. Future studies are warranted to further delineate the epidemiology, refine risk stratification, and optimize therapeutic approaches for candidemia in this highly vulnerable population.

Disclosures

Ethics Committee Approval: The study was approved by the Gaziantep University Clinical Research Ethics Committee (no: 2026/46, date: 21.01.2026).

Informed Consent: The requirement for informed consent was waived due to the retrospective nature of the study.

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