

Nosocomial Fungal Infections

Epidemiology, Control Strategies, and Prevention of *Candida* and Other Yeasts



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KEYWORDS

- Nosocomial • Invasive candidiasis • *Candida* • Yeast
- Immunocompromised patients

KEY POINTS

- Invasive candidiasis is a frequent cause of hospital-acquired infections, commonly associated with invasive devices, immunosuppression, and environmental contamination.
- Conventional culture-based methods often lack sensitivity for diagnosing fungal infections, making advanced nonculture-based testing essential for accurate detection.
- Effective prevention and control of invasive fungal infections require a combination of traditional infection control practices and targeted antifungal prophylaxis for high-risk individuals.
- *Candida auris*, an emerging multidrug-resistant nosocomial pathogen, requires active surveillance and rigorous infection prevention and control to enable early detection and prevent healthcare-associated outbreaks transmission.

Invasive fungal infections remain a major source of morbidity and mortality among hospitalized and immunocompromised patients. This article reviews the current epidemiology of nosocomial fungal infections in adults, with a focus on invasive candidiasis (IC) and the escalating threat of *Candida auris*, an emerging multidrug-resistant pathogen. Emphasis is placed on active surveillance for *C. auris* as a critical strategy to identify colonized patients early and prevent transmission within health care settings. The discussion highlights the clinical impact of *C. auris* outbreaks and their implications for infection prevention and control, underscoring the need for robust containment measures and proactive surveillance. Published recommendations and guidelines for prevention and control are summarized.

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Abbreviations	
BCID	blood culture identification
BCs	blood cultures
BSIs	bloodstream infections
CHG	chlorhexidine gluconate
CLABSI	central line-associated bloodstream infections
CVC	central venous catheters
FDA	Food and Drug Administration
HAI	healthcare-associated infections
IC	invasive candidiasis
ICUs	intensive care units
NCT	nonculture-based tests
PCR	polymerase chain reaction
SOT	solid organ transplantation
T2MR	T2 Magnetic Resonance

EPIDEMIOLOGY OF NOSOCOMIAL FUNGAL INFECTIONS

The global prevalence of invasive fungal infections continues to rise, accompanied by a marked increase in fungal healthcare-associated infections (HAIs). This trend is predominantly driven by the widespread use of aggressive immunosuppressive therapies such as chemotherapy, hematopoietic stem cell transplantation, solid organ transplantation (SOT), and newer immunomodulatory agents (Table 1).^{1–3} Despite this

Table 1 Risk factors associated with invasive fungal infections			
Candida	Pneumocystis	Trichosporon	Malassezia
• Acute necrotizing pancreatitis • Abdominal surgery; anastomotic leak or repeat laparotomies • Broad-spectrum antibiotics • Central venous catheters • Hemodialysis • HSCT • Immunosuppression, including corticosteroids and chemotherapy • Malignancy • Mechanical ventilation >3 d • Multifocal candida colonization • Neutropenia • Prolonged ICU stay • Prolonged hospitalization • SOT (kidney and liver) • Total parenteral nutrition	• Advanced HIV disease • Allogeneic HSCT • Graft-versus-host disease • Hematologic malignancy • Immunosuppression, including biologics, calcineurin inhibitors • corticosteroids, and T-cell depleting agents • Hematologic malignancies • SOT (lung, heart, and kidney)	• Broad-spectrum antibiotics • Central venous catheter • Chemotherapy • Corticosteroids • Hematologic malignancies • HSCT • Neutropenia • Prolonged ICU stay • Severe burns • SOT	• Central venous catheter • Corticosteroids • Diabetes mellitus • Hematologic malignancy • HSCT • Corticosteroids • Lipid emulsions • Prematurity • Prolonged ICU stay • SOT • Total parenteral nutrition

Abbreviation: HSCT, hematopoietic stem cell transplantation.

rise, the true burden of fungal diseases is likely underestimated due to diagnostic limitations and underreporting. Current estimates indicate that fungal diseases affect more than 6.55 million individuals annually, resulting in approximately 2.55 million attributable deaths.⁴

Increased use of invasive devices, particularly central venous catheters (CVC), has contributed to higher rates of *Candida*-related central line-associated bloodstream infections (CLABSI).^{1,3,5} The economic burden of fungal diseases in the United States is substantial, with estimated costs reaching approximately \$19.4 billion annually. *Candida* infections accounted for nearly 66% of this total.⁶

Candida spp. are significant causes of serious HAIs, particularly among patients in intensive care units (ICUs),^{1,3,5,7,8} and are among the most common pathogens responsible for nosocomial bloodstream infections (BSIs) in US hospitals.^{3,5,7} Data suggest that the global incidence of IC is rising, with rates of approximately 3 to 5 cases per 100,000 persons in the general population.⁸ IC affects an estimated 25,000 patients annually in the United States,⁷ with an incidence of about 7 cases per 100,000 persons and mortality rates ranging between 40% and 55%.^{7–9} IC imposes a considerable economic burden, with costs averaging \$48,487 to \$157,574 per episode in the United States.^{3,10} *C. auris*, a multidrug-resistant organism, is highly transmissible between patients via contact with contaminated surfaces or objects in health care facilities. Its ability to cause nosocomial outbreaks and persist in the environment has made it a significant public health concern globally and within the United States. Since its initial detection in the United States in 2013, the number of clinical and screening cases has increased each year, surpassing 33,000 in 2023.¹¹

The overall burden of disease from nosocomial fungal infections remains significant. Limitations in current diagnostic methods, the emergence of antifungal resistance, and high associated mortality underscore the critical need for effective prevention of fungal HAIs. The following sections address key risk factors, diagnosis and strategies for control and prevention.

COMMON NOSOCOMIAL INFECTIONS CAUSED BY YEASTS

Candida Species

Candida spp, particularly *Candida albicans*, is commensal flora of the gut and skin; therefore, most candidal infections are endogenous in origin. IC, including candidemia, disseminated hematogenous infections, and deep-seated infections, can occur in immunocompromised patients, such as those with neutropenia, as well as in critically ill patients.^{8,12,13} In individuals with chemotherapy-induced neutropenia and mucositis, candidemia often originates from the gastrointestinal tract.^{8,12,13} However, in critically ill patients, candidemia most often originates from a CVC colonized by *Candida* spp originating from either from the patient's endogenous microbiota or acquired from the health care environment.^{3,12–15} *Candida* spp. have been recovered from environmental surfaces in hospitals, including high-touch surfaces, floors, and sink drains.¹⁶ The propensity of *Candida* spp to cause CLABSI is largely attributed to their ability to form biofilms on catheter surfaces.¹⁷

Overall, *Candida* spp. accounted for 7.5% of 401,323 HAIs in adults reported to the National Health care Safety Network at the Centers for Disease Control and Prevention between 2018 and 2021.⁵ Notably, *Candida* spp. were the most common cause of CLABSI in the ICU and hospital wards, accounting for 28% and 17% of CLABSI, respectively.⁵ The rising proportion of candidemia in non-ICU settings is likely attributable to the increased use of long-term CVCs.³ Additional risk factors for nosocomial candidemia are listed in [Table 1](#).

A study of IC from 203 centers in the United States between 2009 and 2017 identified *C. albicans* (48%), *Candida glabrata* (24%), *Candida parapsilosis* (11%), and *Candida tropicalis* (7%) as the most common *Candida* pathogens. No significant adjusted annual percent change in incidence was observed for *C. albicans*, *C. glabrata*, *C. parapsilosis*, and *C. tropicalis*; however, the incidence of other *Candida* species as a whole increased significantly, with an annual rise of 7.2%.⁵ Geographic variation in *Candida* distribution is well recognized. While *C. albicans* remains the predominant species worldwide, US surveillance and multicenter studies over the past decade have documented a notable shift toward non-*albicans* species. Although *C. albicans* still accounts for nearly half of IC cases, infections caused by non-*albicans* *Candida* now represent the majority of candidemia episodes in certain US health care settings.^{5,8,18,19} Globally, *C. albicans* accounts for 40% to 60% of cases, with significant regional differences—remaining more than 50% to 70% in Northern and Middle Europe but dropping below 50% in Southern Europe, Latin America, and Australia. Non-*albicans* species have increased markedly: *C. glabrata* predominates in the United States, Australia, and Northern Europe; *C. parapsilosis* in Asia, Latin America, and Southern Europe; and *C. tropicalis* in Latin America and much of Asia.¹⁹

Although most cases of IC are endogenous, exogenous transmission of *Candida* spp. may occur. Characteristics of specific *Candida* spp. may influence the risk for exogenous transmission and nosocomial infections in certain patient populations.

C. parapsilosis candidemia is frequently observed in neonates and transplant recipients. Its common isolation from the hands of health care workers and molecular epidemiologic evidence of outbreaks suggest horizontal transmission to neonates. The organism's ability to form biofilm and thrive in glucose-rich total parenteral nutrition solutions likely contribute to its propensity to cause outbreaks associated with CVC.²⁰ Hence, the frequent isolation of *C. parapsilosis* should prompt measures to enhance hand hygiene and appropriate care of CVCs.

Emerging *Candida* spp. that are relatively resistant to fluconazole, such as *C. auris*,^{8,14,15,21–28} *Candida guilliermondii*,²⁹ and *Candida rugosa*³⁰ have also been associated with nosocomial outbreaks, some involving CVCs. *C. auris*, first isolated from the external ear canal of a hospitalized patient in Japan in 2009, has since been associated with nosocomial outbreaks in health care facilities globally.^{8,14,15,21–27} Its ability to persistently colonize patients and survive on surfaces for months has contributed to outbreaks with potential for patient-to-patient transmission.^{8,14,22–26}

The first *C. auris* case in the United States was identified in New York in 2013.^{25,26} From 2016 through 2023, a total of 10,788 clinical cases and 22,931 screening cases have been reported in the United States; most confirmed cases occurred in New York, Illinois, California, Florida, and Nevada.¹¹ The coronavirus disease 2019 (COVID-19) pandemic markedly accelerated the emergence and spread of *C. auris* in critical care settings. Prepandemic, *C. auris* outbreaks were sporadic; however, during and after COVID-19 surges, incidence rose sharply worldwide. Critically ill patients in ICUs were disproportionately affected, largely due to prolonged hospitalization, mechanical ventilation, CVCs, and extensive use of broad-spectrum antimicrobials and immunomodulators.^{14,15,24,26–28} Outbreaks have involved up to 180 patients and led to ward closures. Molecular epidemiology has been essential for understanding transmission. While early studies using amplified fragment length polymorphism (AFLP) and micro-satellite typing confirmed clonal relationships, whole-genome sequencing now provides high-resolution evidence of patient-to-patient transmission and environmental reservoirs.²⁶

Risk factors for *C. auris* infection mirror those associated with other invasive *Candida* spp and include extended ICU stay, recent surgical procedures, prior antibiotic or

antifungal exposure, presence of CVCs or indwelling urinary catheters, total parenteral nutrition, hematologic malignancies, SOT, hematopoietic stem cell transplantation, and immunosuppression.^{12,14,22,23,25–28}

C. auris has been misidentified as *Candida haemulonii* when using conventional diagnostic methods.^{11,22,25–28} It has been isolated from blood, urine, wounds, and various body fluids, including cerebrospinal, pleural, pericardial, biliary, and peritoneal fluids.^{11,22,25,28} While BSIs represent the majority of reported cases, other invasive manifestations such as myocarditis, pericarditis, meningitis, hepatosplenic candidiasis, osteomyelitis, endophthalmitis, and otitis have also been documented.^{11,22,25,28} *C. auris* is often resistant to one or more classes of antifungal agents.^{11,14,22–25,27,28} In the United States, approximately 90% of isolates have been resistant to fluconazole, about 30% to amphotericin B, and fewer than 2% to echinocandins, which is the drug of choice in clinical infections.^{11,28} Despite geographic variability, overall mortality rates range from 30% to 60%.^{23,27}

Pneumocystis Jirovecii

Opportunistic pneumonia caused by *Pneumocystis jirovecii* has been traditionally attributed to reactivation of latent infection during periods of severe T-cell-mediated immunosuppression, particularly in transplant recipients and patients with advanced HIV disease. However, recent data suggest possible person-to-person transmission or a common environmental source as a potential mode of transmission.^{31–33} Molecular evidence has also identified nosocomial person-to-person transmission of *P. jirovecii* to be the likely cause of outbreaks of *Pneumocystis* pneumonia, particularly among SOT recipients.^{31–33} In a meta-analysis of 30 outbreaks, more than 80% occurred among renal transplant recipients. None of the patients received appropriate antipneumocystis prophylaxis.³¹ Molecular typing methods, including whole-genome sequencing, have been used to confirm outbreaks of *P. jirovecii*.^{34–36}

Other Yeasts

Malassezia spp are lipophilic yeasts that are frequent skin colonizers and the cause of pityriasis. Outbreaks of *Malassezia* fungemia have been reported in premature neonates and immunocompromised patients.³⁴ Prolonged use of CVC and total parenteral nutrition were important predisposing conditions.^{37,38}

Trichosporon spp fungemia has been reported in patients with hematologic malignancies and hematopoietic stem cell transplantation and SOT recipients. Systemic infections have also been reported in premature neonates, individuals with diabetes, and patients with solid tumors. Common risk factors in cases of nosocomial trichosporonosis are the presence of a CVC and exposure to prior antibiotics.³⁹ Waterborne outbreaks have also been reported.⁴⁰ The reported mortality rate ranges from 53% to 76%.³⁹

STRATEGIES FOR PREVENTION OF NOSOCOMIAL CANDIDIASIS

Prevention of Intravascular Catheter-Related Candidemia

Most outbreaks of nosocomial IC and invasive infections with other yeasts have been associated with CVCs. Evidence-based guidelines for preventing CLABSI have been published.^{41,42} Key infection prevention strategies include improving adherence to hand hygiene practices, such as avoiding long or artificial nails, following established guidelines for CVC insertion and maintenance, and promptly removing catheters that are no longer needed.^{41,42}

Although randomized clinical trials have shown that daily 2% chlorhexidine gluconate (CHG) bathing of ICU patients decreases the incidence of nosocomial

multidrug-resistant bacterial BSI, few studies have evaluated the impact on candidemia.^{43,44} A meta-analysis of 5 randomized controlled trials evaluating the efficacy of CHG bathing in reducing BSIs showed that CHG had no effect on fungal BSI.⁴⁴ Conversely, a more recent meta-analysis of 26 randomized and non-randomized studies reported a significant reduction in BSIs, including those caused by *Candida* spp.⁴³ However, emerging evidence suggests CHG has limited efficacy in reducing *Candida* skin colonization. In a cohort of 675 ICU patients (988 swabs), CHG did not significantly lower fungal burden at any time point, with only a transient decline during CHG use followed by rebound after discontinuation. High colony counts, particularly *C. albicans*, were common.⁴⁵ Similarly, *C. auris* exhibits resistance to benzalkonium chloride and variable susceptibility to chlorine and CHG, even at high concentrations.⁴⁶ Despite these limitations, daily CHG bathing remains a simple and effective strategy to reduce overall primary BSI rates, but additional or alternative agents are needed to achieve sustained *Candida* decolonization.

Additionally, the use of disinfection caps, which include alcohol or alcohol/CHG combinations, placed on needleless connectors or access ports, has demonstrated decreased CLABSI rates.^{47,48}

Unlike other *Candida* spp, *C. auris* spreads rapidly in health care facilities, causing difficult-to-control outbreaks. Patients can remain colonized for prolonged periods, shedding the organism into the environment, where it survives on diverse surfaces for months, facilitating patient-to-patient transmission.^{7,14,22–26} Even a single case warrants immediate infection control measures and investigation. Suspected or confirmed cases should be placed in a single room under contact precautions with strict hand hygiene and disinfection of shared equipment. Additional measures include daily and terminal cleaning with Environmental Protection Agency-registered disinfectants effective against *C. auris*, active surveillance of high-risk patients, contact tracing and testing, prospective laboratory surveillance for at least 1 month, and interfacility communication during transfers.^{11,49}

Screening for colonization using a composite axilla-groin swab is critical and may follow prevention-based or response-based strategies. When a new case is identified, broad screening via point prevalence surveys is recommended, as targeted approaches may miss colonized patients.^{11,49} The Centers for Disease Control and Prevention does not recommend screening health care workers or rescreening previously colonized patients, as colonization can persist for months.^{11,49} Although culture remains an option, real-time polymerase chain reaction (PCR) is the preferred method for detecting colonization.^{11,50} Despite the importance of early detection in limiting spread, significant evidence gaps remain, and uptake of screening practices remains low.^{51,52}

A recent study evaluated expanded admission screening for all patients from skilled nursing facilities. Between January 2022 and September 2023, 591 patients were screened, identifying 14 cases (2.4%). Broader screening enabled earlier detection and prevention of nosocomial transmission.⁵³

Prevention of invasive candidiasis

Clinical guidelines recommend antifungal chemoprophylaxis during the pre-graftment period of neutropenia and mucositis to prevent dissemination of endogenous *Candida* spp from the gastrointestinal tract in high-risk patients.^{3,54,55}

Diagnosis of invasive candidiasis

Although blood cultures (BCs) are positive in only about 50% of patients with IC, they are considered the gold standard for the diagnosis of candidiasis.^{3,13,56,57} Thus, the

true incidence of nosocomial candidemia is often underestimated. Nonculture-based tests (NCT) are employed to detect IC, including candidemia, particularly in patients with suspected IC when BCs may yield negative results. However, the use of these assays for routine surveillance of nosocomial IC is yet to be defined. Fungal biomarkers, including 1,3 β -D-glucan, and PCR-based assays are some of these tests used to aid in the diagnosis of IC.^{3,13,56–59} Although β -D glucan has high false-positive rates and is not specific, it has been shown to have a high negative predictive value in the ICU setting.^{3,59,60} PCR-based assays provide species-level identification but remain limited in clinical practice. No Food and Drug Administration (FDA)-cleared PCR tests exist for IC, despite widespread use of commercial and in-house platforms.^{3,8,56–58} A meta-analysis of 54 studies involving almost 5000 patients, including 963 with proven, probable, or possible IC, reported pooled sensitivity of 95% and specificity of 92% for blood-based PCR testing in patients suspected IC. Higher sensitivity was observed with whole blood samples, species-specific targets, and assays with low detection limits.^{56–58} Commercial multiplex panels typically cover the 5 most common *Candida* spp., yet none have undergone multicenter validation or received FDA clearance for US clinical use.^{56,57}

Recently, the Simplexa *C. auris* Direct assay (DiaSorin, Saluggia, Italy), a real-time PCR test authorized through the FDA De Novo pathway, was introduced for direct detection of *C. auris* from axilla and groin swabs, making it well-suited for colonization screening in health care settings.⁶¹

The T2Candida Panel (T2 Biosystems, Lexington, MA), once FDA-approved and recognized for its direct-from-whole-blood detection using T2 Magnetic Resonance (T2MR) technology, is no longer commercially available, thereby eliminating its role in current clinical practice.⁶²

Several molecular panels have been developed to rapidly identify pathogens from positive BCs. Among these, the cobas ePlex blood culture identification (BCID) panels (Roche Diagnostics, Basel, Switzerland), the first FDA-approved multiplex molecular panels, provide broad coverage of bacterial and fungal pathogens along with key resistance markers,⁶³ and the BioFire BCID2 panel (bioMérieux, Salt Lake City, UT) offers comprehensive detection of bacterial and yeast pathogens with resistance genes.⁶⁴ Compared to traditional culture, these assays deliver actionable results within 60 to 120 minutes following growth detection in BCs.

C. auris, a nationally reportable pathogen, is difficult to identify and has been often misidentified as *C. haemulonii*, *Candida famata*, *Candida sake*, *Rhodotorula glutinis*, *Rhodotorula mucilaginosa*, and *Saccharomyces*, or less commonly as *Candida catenulata*, *Candida lusitanae*, *C. guilliermondii*, or *C. parapsilosis* or only to the *Candida* spp. level when using standard laboratory methods.^{11,22,25–28} If the above species are identified, or species identity cannot be determined, the Centers for Disease Control and Prevention recommends further characterization using alternative methodology, including matrix-assisted laser desorption/ionization time-of-flight or molecular-based methods. The cobas ePlex BCID Panels Panel detects 16 fungal targets, including *C. auris*, from positive BC with a high sensitivity and specificity.⁶³ Various PCR methods have been developed for the detection of *C. auris*.

The use of newer NCT could impact the reporting of CLABSIs to the National Healthcare Safety Network. A positive NCT result may meet the laboratory-confirmed BSI criteria regardless of the BC result. In response to concerns from reporting health care institutions, National Healthcare Safety Network revised reporting criteria starting January 1, 2020: If an NCT is positive but the corresponding BC is negative for the same organism 2 days before, the day of, or 1 day after, the NCT result is disregarded. However, if no BC is collected within this timeframe, the NCT result is used for

laboratory-confirmed BSI surveillance determination and will be reported as a CLABSI if criteria are met.⁶⁵

Prevention of *Pneumocystis Pneumonia*

Pneumocystis pneumonia prophylaxis is recommended for hematopoietic stem cell transplantation and SOT recipients during high-risk periods of immunosuppression, especially the first 100 days after transplantation.^{31,66} Although there is potential for person-to-person transmission of *P jirovecii* leading to nosocomial outbreaks, current guidelines do not recommend specific isolation measures for the care of these hospitalized patients.³⁰ However, they do recommend avoiding cohorting infected patients with patients who are immunocompromised.³²

Diagnosis of *Pneumocystis Pneumonia*

P.jirovecii cannot be cultured, and microscopic visualization of cysts or trophozoites in lower respiratory samples, such as induced-sputum and bronchoalveolar lavage fluid, remains the traditional gold standard for diagnosis, despite limited sensitivity and specificity.^{31,33} In recent decades, molecular diagnostics such as PCR have become widely used for detection of *P jirovecii* in clinical respiratory samples; although sensitivity and specificity are generally high, false negatives can occur in lower respiratory samples.^{31,33} Quantitative PCR is preferred over qualitative PCR to help distinguish colonization from probable disease; however, no universally accepted threshold for positivity has been established.³³

COVID-19-Associated Candidiasis

Candidemia has been increasingly reported in critically ill patients with COVID-19, a condition termed COVID-19-associated candidiasis (CAC).^{67–71} To date, no specific COVID-19-related immunologic defect predisposing to IC has been identified. Recent multicenter studies and systematic reviews report CAC incidence ranging from approximately 0.7% to 24%, varying by geographic region and ICU practices.^{67–71} The median time from hospital or ICU admission to CAC diagnosis was 10 to 15 days, indicating that CAC is likely an HAI. Overall 30-day mortality frequently exceeded 50%.^{67,69,70} Non-albicans spp, including *C auris*, were commonly isolated, and infections with non-albicans species were associated with worse outcomes.⁶⁷ Reported risk factors include prolonged ICU stay, cardiovascular disease, diabetes mellitus, mechanical ventilation, hemodialysis, prolonged CVC dwell time, and immunomodulatory therapy such as tocilizumab.^{67–71} Traditional risk factors such as chemotherapy, neutropenia, or transplantation were uncommon.

These findings suggest CAC predominantly results from CVC-related infection, and its increased incidence reflects the unique challenges of managing critically ill patients during the pandemic. Strict adherence to infection prevention and control measures in ICUs remains essential to reduce CLABSIs in COVID-19.

SUMMARY

Nosocomial fungal infections, particularly IC, are associated with substantial morbidity and mortality in critically ill and severely immunocompromised patients. Adherence to recommended infection prevention strategies is essential to prevent catheter-related candidemia. Emerging NCT diagnostic methods offer the potential to improve early detection and management of these infections. In select patient populations at elevated risk for invasive fungal disease, antifungal prophylaxis should be considered during periods of profound immunosuppression.

CLINICS CARE POINTS

- Active surveillance is essential for early detection and interruption of *Candida auris* transmission within health care facilities.
- Integration of newer nonculture-based diagnostics and molecular panels improves sensitivity and accelerates identification of invasive fungal infections, complementing traditional culture methods.
- Strict adherence to evidence-based infection prevention and control practices, including environmental controls and hand hygiene, is essential to reduce the incidence of healthcare-associated fungal infections.

DECLARATION OF AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the author(s) used M365 Copilot in order to proofread the content. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

DISCLOSURE

The authors have nothing to disclose.

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