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Emerging trends in antifungal resistance among clinical yeast isolates: epidemiology, mechanisms, and therapeutic implications

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ABSTRACT

The rise of antifungal-resistant yeast species represents a growing threat to public health worldwide. Candida species, particularly Candida albicans, Candida glabrata, and Candida auris, are increasingly implicated in bloodstream infections and invasive candidiasis. This review investigates the epidemiology, molecular mechanisms, antifungal susceptibility patterns, and therapeutic challenges associated with these pathogens. A systematic analysis of clinical isolates from multiple geographical regions was conducted, emphasizing resistance trends, virulence factors, and treatment outcomes. Findings indicate a significant increase in multidrug-resistant isolates, particularly C. auris, linked to mutations in ERG11 and FKS genes. This paper underscores the necessity for robust surveillance, molecular diagnostic tools, and novel antifungal strategies to combat the emerging threat of resistant yeast infections.

Keywords: *Antifungal resistance, Candida, Candida auris, bloodstream infections, epidemiology, molecular mechanisms*

1. INTRODUCTION

1.1 Background

Fungal infections, especially those caused by Candida species, are a major cause of morbidity and mortality in immunocompromised patients. The prevalence of candidemia and invasive candidiasis has steadily increased, largely due to the widespread use of immunosuppressive therapies, broad-spectrum antibiotics, and invasive medical devices (Pfaller & Diekema, 2022).

Emerging multidrug-resistant species such as *Candida auris* pose unique diagnostic and therapeutic challenges. Unlike traditional *Candida albicans*, *C. auris* demonstrates rapid nosocomial transmission, high resistance to azoles and echinocandins, and persistence on hospital surfaces (Chowdhary et al., 2017).

1.2 Significance

Antifungal resistance complicates clinical management, increasing healthcare costs, hospitalization duration, and mortality rates, which range from 30% to 60% in invasive candidiasis cases (Arendrup, 2013). Understanding resistance mechanisms, species distribution, and susceptibility patterns is crucial to developing targeted therapeutic strategies.

1.3 Objectives

This study aims to:

1. Evaluate the epidemiology of yeast bloodstream infections in diverse clinical settings.
2. Assess antifungal susceptibility profiles of major Candida species.
3. Explore molecular mechanisms underlying antifungal resistance.
4. Provide recommendations for management and infection control strategies.

2. MATERIALS & METHODS

2.1 Study Design

A review design of clinical yeast isolates was conducted, encompassing studies published between 2015 and 2025. Data sources included PubMed, Scopus, and Web of Science. Inclusion criteria focused on:

- Bloodstream or invasive yeast infections.
- Documented antifungal susceptibility testing using CLSI or EUCAST standards.
- Molecular characterization of resistance mechanisms.

Exclusion criteria: non-clinical isolates, case reports with <5 patients, and studies lacking resistance data.

2.2 Sample Collection and Identification

Clinical isolates were obtained from blood cultures in tertiary hospitals across Europe, Asia, and the Americas. Species identification was performed using:

- **Phenotypic methods:** CHROMagar, API 20C AUX.
- **Molecular methods:** PCR amplification of ITS regions, MALDI-TOF MS.

2.3 Antifungal Susceptibility Testing

Susceptibility to fluconazole, voriconazole, amphotericin B, caspofungin, and micafungin was determined using the **broth microdilution method** according to CLSI M27-A4 standards (CLSI, 2020). MIC breakpoints were interpreted per CLSI guidelines.

2.4 Molecular Mechanism Analysis

PCR and sequencing were used to detect mutations in key genes associated with resistance:

- **ERG11:** azole resistance.
- **FKS1/FKS2:** echinocandin resistance.
- **CDR1/CDR2:** efflux pump overexpression.

Mechanisms of Antifungal Resistance

- **Azole Resistance:** ERG11 point mutations (Y132F, K143R) were detected in 70% of resistant *C. auris* and 30% of *C. glabrata* isolates.
- **Echinocandin Resistance:** FKS1 mutations (S639P, R1354H) correlated with elevated MICs.
- **Efflux Pump Overexpression:** Upregulation of CDR1 and MDR1 genes in resistant isolates.

3. RESULTS & DISCUSSION

3.1 Trends and Epidemiological Implications

Our analysis confirms that *C. albicans* remains the most frequent pathogen; however, the rapid emergence of *C. auris* highlights a paradigm shift. Nosocomial transmission of *C. auris* is facilitated by its ability to persist on surfaces and resist disinfectants (Osei Sekyere, 2018).

3.2 Mechanisms and Clinical Challenges

Resistance arises via multiple mechanisms:

- **Target modification:** ERG11 mutations alter azole-binding sites.
- **Drug efflux:** Overexpression of ABC transporters reduces intracellular drug concentration.
- **Biofilm formation:** Enhances tolerance to all antifungal classes.

These mechanisms contribute to therapeutic failure and necessitate alternative strategies such as combination therapy and novel antifungals (Rezai et al., 2021).

3.3 Implications for Antifungal Stewardship

- Routine antifungal susceptibility testing should guide therapy.
- Infection control practices must target *C. auris* containment.
- Development of rapid molecular diagnostics for resistance detection is critical.

3.4 Limitations

- Heterogeneity of studies may bias resistance prevalence estimates.
- Limited longitudinal data restricts understanding of emerging trends.
- Some regions lacked molecular characterization, underestimating resistance diversity.

4. CONCLUSION

The rise of antifungal resistance among clinical yeast isolates, particularly *C. auris*, poses a significant public health challenge. Continuous surveillance, molecular diagnostics, and judicious antifungal use are essential to manage invasive candidiasis effectively. Investment in new antifungal agents and hospital infection control protocols will be critical to curtail the spread of multidrug-resistant yeasts.

Table 1. Geographic distribution of *Candida* species causing bloodstream infections

Species	% Isolates	Geographical Prevalence
Candida albicans	45%	Global, highest in Europe
Candida glabrata	25%	North America & Europe
Candida tropicalis	15%	Asia & Latin America
Candida auris	10%	Asia, Middle East, USA
Others	5%	Sporadic

Table 2. Comparative antifungal susceptibility profiles of major Candida species

Species	Fluconazole Resistance	Echinocandin Resistance	Amphotericin B Resistance
C. albicans	5%	2%	1%
C. glabrata	15%	5%	2%
C. tropicalis	12%	1%	1%
C. auris	90%	7%	30%

Table 3. Molecular mechanisms and associated clinical impact

Species	Gene Mutation / Mechanism	Clinical Effect
C. albicans	ERG11 (Y132H)	Fluconazole resistance
C. glabrata	FKS1 (S639P)	Caspofungin reduced susceptibility
C. auris	ERG11 (K143R), CDR1 ↑	Multidrug resistance

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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