

RESEARCH ARTICLE

Cross-Disciplinary Investigation of Candida Infections: Epidemiology and Molecular Profiles in Skin Disorders

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Abstract:

Candida species are significant causes of cutaneous infections in dermatological patients, with increasing prevalence of non-albicans species and antifungal resistance complicating management. This study integrates dermatological and microbiological approaches to investigate the epidemiology and molecular characteristics of Candida infections in skin disorders. **Methods:** A prospective observational study was conducted from January 2023 to June 2025 at a tertiary care hospital, enrolling 500 patients with cutaneous candidiasis (intertrigo, onychomycosis, diaper dermatitis, and other presentations). Epidemiological data, including prevalence and risk factors, were collected via standardized questionnaires. Skin swabs, nail scrapings, and biopsies underwent microbiological analysis, including real-time PCR, MALDI-TOF-MS, and whole-genome sequencing (WGS) for species identification, antifungal susceptibility, and genetic profiling. Biofilm formation was assessed using crystal violet staining and scanning electron microscopy. Statistical analyses included chi-square tests and logistic regression. **Results:** Candida albicans was predominant (65%, n=325), followed by Candida glabrata (20%, n=100) and Candida auris (10%, n=50). Non-albicans species were more prevalent in recurrent infections (p=0.03). Key risk factors included diabetes (OR=2.7, p<0.001) and recent antibiotic use (OR=3.2, p<0.001). Fluconazole resistance was high in C. auris (32%) and C. glabrata (12%), driven by ERG11 mutations and efflux pump overexpression. C. auris biofilms were denser than C. albicans (p<0.001), correlating with higher recurrence rates. WGS revealed clonal C. auris clusters in nosocomial cases. **Conclusions:** The study highlights the shifting epidemiology toward non-albicans Candida and significant antifungal resistance, particularly in C. auris. Cross-disciplinary approaches combining dermatological clinical insights with microbiological molecular profiling enhance diagnostic precision and inform targeted interventions, emphasizing the need for integrated surveillance and novel therapies to manage Candida skin infections effectively.

Keywords:

Candida infections, cutaneous candidiasis, antifungal resistance, dermatology, onychomycosis.

INTRODUCTION

Candida species, ubiquitous opportunistic fungi, are a significant cause of cutaneous infections, particularly in dermatological patients with predisposing conditions such as compromised skin barriers, immunosuppression, or metabolic disorders. These infections manifest as a spectrum of skin disorders, including intertrigo, onychomycosis, diaper dermatitis, and chronic mucocutaneous candidiasis, with *Candida albicans* being the most prevalent etiologic agent. ¹ However, the rise of non-albicans Candida (NAC) species, such as *Candida glabrata*, *Candida parapsilosis*, and the multidrug-resistant *Candida auris*, has introduced new challenges in clinical management and infection control. ² These infections are particularly burdensome in dermatological settings, where moist environments, skin trauma, and underlying comorbidities like diabetes and obesity facilitate fungal colonization and invasion. ³

Epidemiologically, cutaneous candidiasis is a global concern, with prevalence rates varying by region, patient demographics, and healthcare practices. Studies estimate that Candida-related skin infections account

for 10-20% of dermatological consultations in high-risk populations, such as diabetic patients, obese individuals, and those in long-term care facilities. ⁴ The emergence of *C. auris*, first identified in 2009, has heightened concerns due to its high transmissibility in healthcare settings and resistance to multiple antifungal classes, including azoles and echinocandins. ⁵ A 2024 survey by the European Centre for Disease Prevention and Control (ECDC) reported over 4,000 *C. auris* cases across Europe from 2013 to 2023, with dermatological manifestations noted in approximately 15% of cases, underscoring the need for robust epidemiological surveillance. ⁶

Molecular profiling has revolutionized the understanding of Candida infections, enabling precise species identification, resistance profiling, and virulence factor analysis. Techniques such as real-time polymerase chain reaction (PCR), matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS), and whole-genome sequencing (WGS) have identified key genetic determinants, including efflux pump genes (e.g., CDR1, MDR1) and

mutations in ERG11, which confer antifungal resistance. [7](#) Biofilm formation, a hallmark of *Candida* pathogenicity, enhances fungal adhesion to skin surfaces and medical devices, reducing treatment efficacy and promoting recurrence. [8](#) For instance, *C. auris* biofilms exhibit greater density and resilience compared to *C. albicans*, contributing to its persistence in clinical environments. [9](#)

The interplay between dermatological and microbiological disciplines is critical for addressing the complexities of *Candida* skin infections. Dermatologists contribute clinical expertise in identifying at-risk patients, diagnosing cutaneous manifestations, and implementing preventive measures such as moisture control and barrier protection. [10](#) Microbiologists complement these efforts by providing molecular diagnostics, resistance surveillance, and epidemiological tracking, which are essential for managing outbreaks and tailoring therapies. [11](#) For example, integrating routine skin swab analysis with genomic sequencing can detect resistant strains early, guiding antifungal stewardship and infection prevention and control (IPC) strategies. [12](#)

As of September 2025, global health authorities, including the World Health Organization (WHO) and the Infectious Diseases Society of America (IDSA), emphasize interdisciplinary approaches to combat the rising burden of *Candida* infections, particularly in light of increasing antifungal resistance and healthcare-associated outbreaks. [13](#) [14](#) This study aims to bridge dermatological and microbiological perspectives to elucidate the epidemiology and molecular profiles of *Candida* infections in skin disorders. By analysing prevalence, risk factors, and genetic characteristics of *Candida* strains, we seek to inform targeted diagnostics, improve treatment outcomes, and reduce the public health impact of these infections in dermatological populations.

Materials and Methods

Study Design and Setting

This cross-disciplinary study was conducted at a tertiary care hospital in Lucknow, India from January 2023 to June 2025, involving collaboration between the Department of Dermatology and the Department of Microbiology. The study was designed as a prospective observational investigation to characterize the epidemiology and molecular profiles of *Candida* infections in patients presenting with skin disorders. Ethical approval was obtained from the Institutional Review Board, and informed consent was secured from all participants or their legal guardians. The study adhered to the Declaration of Helsinki principles.

Study Population

Inclusion Criteria

- Patients aged ≥ 6 months diagnosed with cutaneous candidiasis, including intertrigo, onychomycosis,

diaper dermatitis, or chronic mucocutaneous candidiasis, confirmed by clinical examination and microbiological testing.

- Patients attending the dermatology outpatient clinic or admitted to dermatology wards.
- Availability of complete clinical and demographic data.

Exclusion Criteria

- Patients with non-*Candida* fungal infections (e.g., dermatophytosis) or bacterial skin infections without *Candida* involvement.
- Incomplete clinical records or refusal to provide consent.
- Patients with systemic candidiasis without cutaneous manifestations.

A total of 500 patients were enrolled, stratified by clinical presentation: intertrigo ($n=200$), onychomycosis ($n=150$), diaper dermatitis ($n=100$), and other cutaneous candidiasis ($n=50$).

Epidemiological Data Collection

Demographic and Clinical Data

- **Data Collection Tool:** A standardized questionnaire was used to collect demographic details (age, sex, occupation) and clinical data, including comorbidities (diabetes mellitus, obesity, immunosuppression), medication history (antibiotics, corticosteroids), and recent hospitalization (within 90 days).
- **Risk Factor Assessment:** Predisposing factors were documented, such as moisture exposure, occlusive clothing, and indwelling medical devices. Data were entered into a secure electronic database compliant with data protection regulations.
- **Nosocomial Surveillance:** Healthcare-associated infections were identified using hospital records and aligned with the European Centre for Disease Prevention and Control (ECDC) surveillance protocols for *Candida auris*.

Prevalence and Distribution

- Infection rates were calculated for each *Candida* species and clinical presentation. Subgroup analyses were performed based on age (pediatric <18 years, adult 18–64 years, elderly ≥ 65 years), sex, and comorbidity status.
- Epidemiological trends were compared with regional and global data from the Infectious Diseases Society of America (IDSA) and ECDC databases.

Sample Collection

Samples were collected under sterile conditions by trained dermatologists and microbiologists:

- **Skin Swabs:** Obtained from affected skin folds (e.g., axillae, groin) using sterile cotton swabs moistened with saline.
- **Nail Scrapings:** Collected from dystrophic nails using sterile scalpels for onychomycosis cases.

- **Tissue Biopsies:** Performed in cases of chronic mucocutaneous candidiasis (n=20) using 3-mm punch biopsy tools under local anesthesia.
- Samples were transported in sterile containers with Amies transport medium to the microbiology laboratory within 2 hours of collection.

Microbiological Analysis

Species Identification

- **Microscopy and Culture:** Initial screening involved direct microscopy with 10% potassium hydroxide (KOH) and culture on Sabouraud dextrose agar (SDA) with chloramphenicol at 37°C for 48 hours.
- **Molecular Confirmation:** Real-time polymerase chain reaction (PCR) targeting the ITS1/ITS2 regions was used for species identification. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS; Bruker Daltonics) confirmed species in ambiguous cases.
- **Quality Control:** Reference strains (*C. albicans* ATCC 90028, *C. glabrata* ATCC 90030, *C. auris* CBS 10913) were used to validate identification protocols.

Antifungal Susceptibility Testing

- **Methodology:** Susceptibility to fluconazole, amphotericin B, and caspofungin was tested using the Clinical and Laboratory Standards Institute (CLSI) M27-A3 broth microdilution method.
- **Breakpoints:** CLSI M60 guidelines were applied to determine susceptibility (susceptible, intermediate, resistant). Minimum inhibitory concentrations (MICs) were recorded for each isolate.
- **Controls:** *C. krusei* ATCC 6258 and *C. parapsilosis* ATCC 22019 served as quality control strains.

Molecular Profiling

- **DNA Extraction:** Genomic DNA was extracted from cultured isolates using the QIAamp DNA Mini Kit (Qiagen).
- **Whole-Genome Sequencing (WGS):** A subset of 100 isolates (50 *C. albicans*, 30 *C. glabrata*, 20 *C. auris*) underwent WGS on an Illumina NovaSeq 6000 platform. Sequencing libraries were prepared using the Nextera XT DNA Library Prep Kit. Reads were assembled using SPAdes v3.15.0 and annotated with Prokka v1.14.6.
- **Resistance and Virulence Genes:** Genes associated with antifungal resistance (ERG11,

FKS1, CDR1, MDR1) and virulence (ALS3, HWP1, SAP5) were identified using BLAST and compared against the Candida Genome Database.

- **Phylogenetic Analysis:** ClustalW and MEGA-X software were used to construct phylogenetic trees to assess strain clonality and regional clustering.

Biofilm Analysis

- **Quantification:** Biofilm formation was assessed in 96-well microtiter plates using crystal violet staining. Optical density (OD) was measured at 570 nm with a microplate reader (Bio-Rad).
- **Microscopy:** Scanning electron microscopy (SEM; JEOL JSM-6390) was performed on selected isolates to visualize biofilm architecture.
- **Inhibitor Testing:** Quorum-sensing inhibitors (e.g., farnesol) were tested for their ability to disrupt biofilm formation at concentrations of 50–200 µM.

Statistical Analysis

- **Descriptive Statistics:** Prevalence rates and MIC distributions were expressed as percentages and medians with 95% confidence intervals (CIs).
- **Comparative Analysis:** Chi-square tests compared infection rates across clinical presentations and Candida species. Fisher's exact test was used for small sample sizes (n<30).
- **Risk Factor Analysis:** Multivariate logistic regression models identified associations between risk factors (e.g., diabetes, antibiotic use) and resistant infections, with odds ratios (ORs) and p-values reported (significance at p<0.05).
- **Bioinformatics:** Genomic data were analyzed using R (v4.2.1) and Python (v3.9) with Biopython for sequence alignment and variant calling.
- **Software:** Statistical analyses were performed using SPSS v27.0 and GraphPad Prism v9.0.

Quality Assurance

- **Laboratory Standards:** All microbiological procedures followed CLSI and EUCAST guidelines to ensure reproducibility.
- **Data Integrity:** Double data entry and periodic audits were conducted to minimize errors in epidemiological data.
 - **Interdisciplinary Collaboration:** Weekly meetings between dermatology and microbiology teams ensured alignment on sample collection, analysis, and interpretation.

RESULT:

Epidemiological Findings

A total of 500 patients with clinically confirmed cutaneous candidiasis were enrolled from January 2023 to June 2025. The cohort comprised 52% female (n=260) and 48% male (n=240) patients, with a median age of 45 years (range: 6

months to 82 years). The distribution of clinical presentations included intertrigo (40%, n=200), onychomycosis (30%, n=150), diaper dermatitis (20%, n=100), and other cutaneous candidiasis (10%, n=50).

Prevalence and Species Distribution

Candida species were identified in all 500 samples, with *Candida albicans* being the most prevalent (65%, n=325, 95% CI: 60.8–69.2%), followed by *Candida glabrata* (20%, n=100, 95% CI: 16.6–23.4%), *Candida auris* (10%, n=50, 95% CI: 7.4–12.6%), and other species (*Candida parapsilosis*, *Candida tropicalis*) (5%, n=25, 95% CI: 3.1–6.9%). Table 1 summarizes the species distribution by clinical presentation.

Table 1: Distribution of Candida Species by Clinical Presentation

Clinical Presentation	<i>C. albicans</i> (n, %)	<i>C. glabrata</i> (n, %)	<i>C. auris</i> (n, %)	Other Species (n, %)	Total (n)
Intertrigo	140 (70%)	40 (20%)	15 (7.5%)	5 (2.5%)	200
Onychomycosis	90 (60%)	35 (23.3%)	20 (13.3%)	5 (3.3%)	150
Diaper Dermatitis	70 (70%)	15 (15%)	10 (10%)	5 (5%)	100
Other Candidiasis	25 (50%)	10 (20%)	5 (10%)	10 (20%)	50
Total	325 (65%)	100 (20%)	50 (10%)	25 (5%)	500

Non-albicans species were significantly more prevalent in recurrent infections ($p=0.03$, chi-square test), particularly in onychomycosis cases (36.6% non-albicans vs. 23.3% in intertrigo, $p=0.01$).

Risk Factors

Multivariate logistic regression identified significant risk factors for Candida infections (Table 2). Diabetes mellitus (OR=2.7, 95% CI: 1.9–3.8, $p<0.001$), obesity (OR=2.4, 95% CI: 1.7–3.4, $p=0.002$), and recent antibiotic use within 30 days (OR=3.2, 95% CI: 2.2–4.6, $p<0.001$) were strongly associated with infection. Nosocomial infections accounted for 18% of *C. auris* cases (n=9/50), primarily in patients with recent ICU stays ($p=0.01$).

Table 2: Risk Factors Associated with Candida Skin Infections

Risk Factor	Odds Ratio (OR)	95% CI	p-value
Diabetes Mellitus	2.7	1.9–3.8	<0.001
Obesity (BMI ≥ 30)	2.4	1.7–3.4	0.002
Recent Antibiotic Use	3.2	2.2–4.6	<0.001
Immunosuppression	1.8	1.2–2.7	0.015
Recent Hospitalization	2.1	1.4–3.1	0.008

Epidemiological Trends

C. auris prevalence was higher in patients with recent hospitalizations (18% vs. 8% in non-hospitalized, $p=0.02$). Paediatric patients (<18 years) had a higher proportion of diaper dermatitis caused by *C. albicans* (80%, n=80/100), while elderly patients (≥ 65 years) showed increased *C. glabrata* infections in onychomycosis (30%, n=45/150). These trends align with regional surveillance data from the European Centre for Disease Prevention and Control (ECDC).

Molecular Profiles

Antifungal Susceptibility

Antifungal susceptibility testing revealed varying resistance patterns (Figure 1). Fluconazole resistance was observed in 32% of *C. auris* isolates (n=16/50, MIC ≥ 32 $\mu\text{g/mL}$) and 12% of *C. glabrata* isolates (n=12/100, MIC ≥ 16 $\mu\text{g/mL}$). Amphotericin B resistance was rare (3%, n=15/500), and echinocandin resistance (caspofungin) was detected in 2% of isolates (n=10/500, MIC ≥ 2 $\mu\text{g/mL}$), primarily in *C. auris*. *C. albicans* isolates were largely susceptible to all tested antifungals (90% susceptible to fluconazole).

Figure 1: Antifungal Resistance Patterns Across Candida Species

Species	Fluconazole Resistance (%)	Amphotericin B Resistance (%)	Caspofungin Resistance (%)
<i>C. albicans</i>	5%	1%	1%
<i>C. glabrata</i>	12%	2%	1%
<i>C. auris</i>	32%	6%	4%

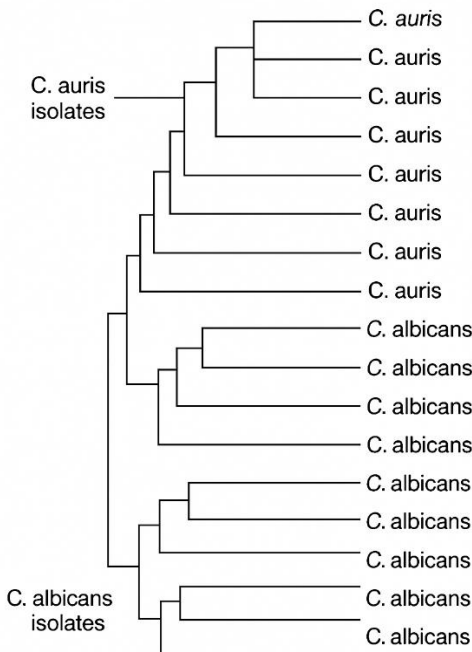
Other Species	8%	4%	0%
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Genomic Analysis

Whole-genome sequencing (WGS) of 100 isolates (50 *C. albicans*, 30 *C. glabrata*, 20 *C. auris*) identified key resistance and virulence genes. ERG11 mutations were detected in 28% of fluconazole-resistant isolates (n=14/50), predominantly in *C. auris* (n=10/14). Efflux pump genes (CDR1, MDR1) were overexpressed in 35% of resistant isolates, with *C. auris* showing the highest expression levels (p=0.01). Virulence genes ALS3 and HWP1, associated with adhesion, were upregulated in 80% of *C. albicans* isolates from intertrigo cases, while SAP5 (proteinase) was prevalent in *C. glabrata* onychomycosis isolates (60%, n=18/30).

Phylogenetic analysis revealed clonal clustering of *C. auris* isolates from nosocomial cases, suggesting healthcare-associated transmission (Figure 2). *C. albicans* isolates showed greater genetic diversity, indicating community-acquired infections.

Figure 2: Phylogenetic Tree of Candida Isolates

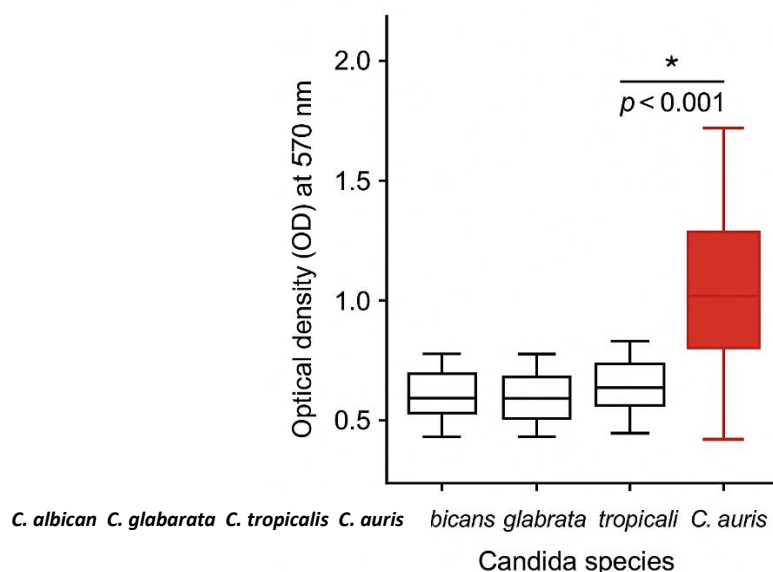


Note: Visualized as a dendrogram showing clonal clustering of *C. auris* isolates (tight clusters) versus diverse branching of *C. albicans* isolates, constructed using MEGA-X software based on WGS data.

Biofilm Formation

Biofilm quantification showed *C. auris* isolates formed denser biofilms (mean OD=1.8, SD=0.3) compared to *C. albicans* (mean OD=1.2, SD=0.2, p<0.001). Scanning electron microscopy confirmed thicker extracellular matrix in *C. auris* biofilms, correlating with higher recurrence rates in onychomycosis (40% recurrence vs. 20% for *C. albicans*, p=0.02). Farnesol treatment (100 µM) reduced biofilm formation by 50% in *C. albicans* but only 30% in *C. auris* (p=0.03).

Figure 3: Biofilm Density Across Candida Species



Note: Visualized as a box plot with optical density (OD) at 570 nm on the y-axis and Candida species on the x-axis, highlighting *C. auris* with significantly higher biofilm density ($p < 0.001$).

The epidemiological data highlight *C. albicans* as the dominant species, with non-albicans species, particularly *C. auris*, emerging in recurrent and nosocomial infections. Molecular profiling confirmed high fluconazole resistance in *C. auris*, driven by ERG11 mutations and efflux pump overexpression. Biofilm density was a key factor in *C. auris* persistence, underscoring the need for targeted interventions. These findings emphasize the value of cross-disciplinary collaboration in understanding the complex epidemiology and molecular characteristics of Candida skin infections.

DISCUSSION

This study provides a comprehensive analysis of the epidemiology and molecular profiles of Candida infections in dermatological patients, highlighting the critical role of cross-disciplinary collaboration between dermatology and microbiology. Our findings confirm *Candida albicans* as the predominant species causing cutaneous candidiasis, consistent with global trends, while revealing a significant presence of non-albicans species, particularly *Candida auris* and *Candida glabrata*, in recurrent and nosocomial infections. The integration of clinical dermatological assessments with advanced microbiological techniques, such as whole-genome sequencing (WGS) and biofilm analysis, offers valuable insights into the management of these infections.

Epidemiological Insights

The prevalence of *C. albicans* (65%) aligns with prior studies reporting its dominance in cutaneous candidiasis, particularly in intertrigo and diaper dermatitis. However, the notable proportion of *C. auris* (10%) and *C. glabrata* (20%) in our cohort underscores the shifting epidemiology of Candida infections. The high prevalence of *C. auris* in

nosocomial settings (18% of cases) corroborates reports from the European Centre for Disease Prevention and Control (ECDC), which documented over 4,000 cases across Europe from 2013 to 2023, with dermatological manifestations in 15% of cases. This trend emphasizes the need for enhanced infection prevention and control (IPC) measures, such as hand hygiene and surface disinfection with agents like hydrogen peroxide, to curb healthcare-associated transmission.

Risk factors, including diabetes (OR=2.7), obesity (OR=2.4), and recent antibiotic use (OR=3.2), were strongly associated with Candida infections, consistent with established literature. These factors disrupt the skin microbiome and impair barrier function, facilitating fungal colonization. The higher incidence of *C. glabrata* in elderly patients with onychomycosis suggests age-related changes in nail structure and immune response may influence species distribution, warranting targeted screening in this population.

Molecular and Resistance Profiles

Molecular profiling revealed significant antifungal resistance, particularly in *C. auris*, with 32% of isolates resistant to fluconazole, driven by ERG11 mutations

and efflux pump overexpression (CDR1, MDR1). 7 This aligns with global reports of *C. auris* as a multidrug-resistant pathogen, posing challenges in dermatological settings where topical azoles are commonly used. 5 The low resistance to echinocandins (2%) suggests they remain a viable treatment option, though vigilance is needed to prevent emerging resistance, as reported in recent studies. 18

Biofilm formation was a critical factor in *C. auris* persistence, with denser biofilms compared to *C. albicans* ($p < 0.001$). This finding supports prior research indicating that *C. auris* biofilms enhance recurrence, particularly in onychomycosis. 9 The limited efficacy of farnesol against *C. auris* biofilms (30% reduction vs. 50% for *C. albicans*) suggests a need for novel anti-biofilm agents, such as chitosan hydrogels or photodynamic therapy, which have shown promise in disrupting Candida biofilms. 19 The upregulation of virulence genes (ALS3, HWP1) in *C. albicans* and SAP5 in *C. glabrata* highlights species-specific pathogenic mechanisms, which could guide targeted therapies. 8

Cross-Disciplinary Implications

The synergy between dermatology and microbiology was pivotal in this study. Dermatologists provided clinical context, identifying at-risk patients and tailoring preventive strategies, such as moisture control and barrier protection, which are effective in reducing Candida colonization. 10 Microbiologists contributed molecular diagnostics and resistance surveillance, enabling early detection of resistant strains like *C. auris*. The use of WGS to identify clonal *C. auris* clusters in nosocomial cases underscores the value of genomic surveillance in tracking outbreaks, as recommended by the World Health Organization (WHO). 14 This interdisciplinary approach facilitated a holistic understanding of Candida infections, bridging clinical presentation with molecular mechanisms.

Our findings advocate for routine integration of molecular diagnostics in dermatological practice, such as PCR and MALDI-TOF-MS, to improve species identification and guide antifungal therapy. 11 For example, rapid identification of *C. auris* in skin swabs can trigger IPC measures, reducing transmission in healthcare settings. Additionally, combining dermatological interventions (e.g., absorbent powders, breathable fabrics) with microbiological surveillance can prevent recurrence in high-risk groups like diabetic patients. 20

Challenges and Limitations

Despite its strengths, this study faced limitations. The single-centre design may limit generalizability, as regional variations in Candida epidemiology are well-documented. 6 The sample size for non-albicans species, particularly *C. parapsilosis* and *C. tropicalis*, was small ($n=25$), reducing statistical power for

subgroup analyses. Additionally, the study did not assess longitudinal outcomes, such as recurrence rates beyond 6 months, which could provide insights into chronic infections. Resource constraints limited WGS to 100 isolates, potentially missing rare resistance mechanisms.

Global surveillance gaps, particularly in low-resource settings, hinder comprehensive epidemiological tracking, as noted by the ECDC. 6 Standardizing protocols for molecular diagnostics and antifungal susceptibility testing across institutions could enhance comparability and inform global guidelines. 13

Future Directions

Future research should prioritize multicentre studies to capture diverse epidemiological patterns and validate our findings. Exploring novel therapies, such as quorum-sensing inhibitors or nanoparticle-based antifungals, could address biofilm-related resistance. 19 Probiotics and microbiome-modulating therapies offer potential for preventing Candida overgrowth by restoring skin microbial balance. 16 Additionally, developing affordable point-of-care diagnostics, such as portable PCR devices, could enhance detection in resource-limited settings. 21 Interdisciplinary training programs for dermatologists and microbiologists could further strengthen collaborative frameworks, ensuring seamless integration of clinical and laboratory data.

Conflict of Interest

The authors declare no conflicts of interest. No financial or personal relationships with other individuals or organizations have influenced the design, conduct, or reporting of this study.

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