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Outbreak of *Candida auris* in Patients Infected with SARS-CoV-2 Experience in a Tertiary Care Center in Colombia

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Abstract

During the SARS-CoV-2 pandemic, healthcare systems faced an increased burden of secondary infections, including those caused by *Candida auris*, an emerging multidrug-resistant pathogen associated with hospital outbreaks worldwide. We aimed to describe the clinical and microbiological characteristics of a hospital outbreak of *Candida auris* fungemia in patients with COVID-19 in a tertiary care center. This retrospective observational study included 14 patients diagnosed between November 2020 and April 2021. Infection was initially identified using VITEK2® and confirmed using MALDI-TOF MS, with antifungal susceptibility determined using broth microdilution. All patients developed fungemia, were mechanically ventilated, and had central venous access and prolonged hospital stays; 64.2% had comorbidities. Resistance frequencies based on CDC tentative MIC thresholds were observed for fluconazole (64.2%) and amphotericin B (71.4%). In-hospital mortality was 50%; exploratory analyses of factors associated with mortality should be interpreted cautiously given the small sample size. These findings highlight the significant clinical impact of *Candida auris* in critically ill COVID-19 patients, particularly in the context of invasive care and antimicrobial resistance. The outbreak underscores the importance of early identification, appropriate antifungal therapy, and strict infection prevention and control measures. Further studies are needed to better define risk factors and optimize management strategies in similar settings.

Keywords: *Candida auris*; SARS CoV-2; outbreak

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1. Introduction

Severely ill patients (e.g., patients with chronic obstructive pulmonary disease, influenza, or coronavirus disease 2019 [COVID-19]) or those with conditions that cause

immunosuppression (neutropenia, hematologic malignancies, or solid organ transplantation), either due to the condition itself or treatment with corticosteroids, have a wide range of comorbidities and conditions that predispose them to the development of invasive fungal infections [1].

The SARS-CoV-2 (COVID-19) pandemic has presented a challenge to health systems worldwide. Immunological dysregulation, the presence of various comorbidities, prolonged hospital stays, and the overuse of antimicrobials facilitate the emergence of superinfections, mainly through multidrug-resistant (MDR) bacteria and fungi, in patients with COVID-19, increasing adverse events and even related mortality [1–3].

SARS-CoV-2 is strongly associated with invasive fungal infections, including filamentous fungi and yeasts, especially Mucor, Aspergillus, and Candida auris. All of these are present in different countries and continents around the world [2].

Countries such as Spain, Canada, the United States (specifically New York), China, and Mexico have reported data on hospital-acquired infections (HAIs), mainly in patients with severe COVID-19 which is usually associated with invasive devices [1,2,4]. In general, these superinfections occur as events related to mechanical ventilation (MV), urinary tract infections, and, to a lesser extent, bloodstream infections [5–7]. Fungal pathogens such as *Aspergillus spp. Mucor* and *C. auris.* are among the emerging fungal pathogens of greatest concern related to steroid use and immunosuppression associated with the administration of monoclonal antibodies such as Tocilizumab and Sarilumab, as well as immunomodulatory drugs like Anakinra [3].

Since the emergence of *Candida auris* in 2009, it has spread across several continents, generating different geographic clades [8]. Critically ill patients, particularly those with COVID-19, are increasingly recognized as being vulnerable to invasive fungal pathogens including *Candida auris* [3,5].

Candida auris is an emerging fungal pathogen that poses a significant global health threat due to its ability to colonize the skin and medical devices, its resistance to multiple antifungal agents, challenges in identification with certain automated systems, and difficulties in environmental eradication. These factors contribute to nosocomial outbreaks worldwide and are associated with increased mortality [6,8,9].

In 2016, the Pan American Health Organization (PAHO) and the World Health Organization (WHO) issued an epidemiological alert, emphasizing the need for early detection and reporting of *C. auris* cases, along with the implementation of preventive measures to control local spread in communities and health settings, considering the occurrence of outbreaks before COVID-19 [9,10]. By 1 February 2021, this alert was reissued due to an increase in cases occurring in patients with COVID-19 [11,12].

We conducted a retrospective outbreak investigation describing the clinical, microbiological, and infection control characteristics of patients with *C. auris* fungemia identified during a COVID-19-associated pandemic. Although multiple COVID-19-era series have described *Candida auris* in critically ill patients, published outbreak investigations from Colombia that integrate clinical outcomes with modifiable, operational drivers of transmission (timing of isolation/cohorting, environmental decontamination performance, and time to species-level identification and targeted therapy) remain limited. Importantly, Colombia reported healthcare-associated *C. auris* outbreaks before the pandemic, indicating that the organism was already established and could be amplified when hospital systems are strained. By framing the present work as an outbreak investigation, rather than only a descriptive case series, our aim is to identify actionable failure points and response opportunities that are transferable to similar tertiary-care settings in Latin America, where pandemic surges can disrupt routine infection prevention practices and accelerate spread of highly transmissible, multidrug-resistant fungi.

2. Materials and Methods

This retrospective single-center outbreak investigation, with a descriptive case series component, included 14 hospitalized adult patients with RT-PCR-confirmed SARS-CoV-2 infection and bloodstream infection due to *Candida auris* during an outbreak surveillance period from November 2020 to April 2021 at a tertiary care center. The study was approved by the Research Ethics Committee of Hospital Universitario Erasmo Meoz (approval code 2023-136-015072-1).

A confirmed case was defined as any hospitalized adult patient ($\geq$18 years) with RT-PCR-confirmed SARS-CoV-2 infection and at least one blood culture positive for *Candida auris* obtained $\geq$48 h after hospital admission during the outbreak period (November 2020–April 2021). Patients with positive cultures obtained outside the study period or without microbiological confirmation of *C. auris* were excluded.

Microbiological methods: Blood cultures were processed using a continuous-monitoring blood culture system. Positive bottles were subjected to Gram staining and subcultured onto Sabouraud dextrose agar and blood agar according to standard microbiological procedures. Yeast isolates were initially identified using the VITEK 2® YST identification system (bioMérieux, Marcy-l'Étoile, France), which presumptively classified isolates within the *Candida haemulonii*/*Candida auris* complex. All suspected *Candida auris* isolates were subsequently referred to the National Reference Laboratory of the Instituto Nacional de Salud (INS), Colombia, for species confirmation by matrix-assisted laser desorption ionization–time-of-flight mass spectrometry (MALDI-TOF MS, Bruker Daltonics, Bremen, Germany), following national surveillance protocols and international recommendations.

Antifungal susceptibility testing was performed using the broth microdilution method according to the Clinical and Laboratory Standards Institute (CLSI) M27 guidelines. Minimum inhibitory concentrations (MICs) were determined after 24 h of incubation and are reported throughout the manuscript in μg/mL. Because CLSI clinical breakpoints for *Candida auris* have not been established, susceptibility classifications were interpreted using the Centers for Disease Control and Prevention (CDC) tentative MIC thresholds: fluconazole $\geq$ 32 μg/mL, amphotericin B $\geq$ 2 μg/mL, micafungin $\geq$ 4 μg/mL, and caspofungin $\geq$ 2 μg/mL. These thresholds represent provisional epidemiological criteria and should not be interpreted as established clinical breakpoints.

Where available, rapid molecular identification was performed directly from positive blood culture bottles using the BioFire FilmArray Blood Culture Identification 2 (BCID2) panel (bioMérieux, Marcy-l'Étoile, France), and results were communicated immediately to the treating physicians. The use of BCID2 was dependent on reagent availability and clinician request and was not applied systematically during the outbreak period.

Isolates were preserved at −70 °C in glycerol-supplemented brain–heart infusion broth at the reference laboratory. Molecular epidemiological analyses, including whole-genome sequencing, multilocus sequence typing (MLST), or other clonality assessments, were not performed.

Follow-up blood cultures were performed every 48–72 h after the initial positive culture until microbiological clearance was documented; however, adherence to this practice was inconsistent and reported as such. Single positive bottles in a single set with absence of clinical correlates and absence of growth on repeat sampling were classified as possible contamination. Catheter-related source investigations, including paired peripheral and central blood cultures or differential time-to-positivity analyses, were not performed systematically. Likewise, catheter-tip cultures were only available in cases in which catheter removal was clinically indicated. Given the recovery of *Candida auris* from blood cultures in critically ill patients with compatible clinical findings, all cases were considered true fungemia episodes.

Clinical and microbiological data, including demographics, comorbidities, antifungal therapy, and outcomes, were collected in a standardized database, and exported to JAMOVI version 2.6 software for further analysis. Variables collected included age, sex, comorbidities, invasive device use, microbiological findings, antifungal susceptibility profiles, antifungal therapy, and clinical outcomes.

The primary outcome was crude in-hospital mortality, defined as death occurring during the index hospitalization irrespective of the immediate cause of death. Given the coexistence of severe COVID-19 and multiple concurrent conditions, mortality attributable specifically to *Candida auris* infection could not be reliably determined.

3. Results

C. auris isolates were recorded in 14 patients diagnosed with COVID-19 during the *C. auris* outbreak that occurred between November 2020 and April 2021. Among them, 9/14 (64.2%) were male and 5/14 (35.7%) were female, with a mean age of 50.4 (SD:14,2) years. Nine patients (64.2%) had comorbidities, with the most common being hypertension and type II diabetes mellitus, affecting 42.8% (6/14) of the patients studied, followed by obesity in 35.7% (5/14) (Table 1).

Table 1. Clinical characteristics of the study population with SARS-CoV-2 pneumonia and *C. auris.*

Characteristics	No. =14 n (%)
Age	
• Mean (SD)	50.4 (14.2)
• Median (IQR)	49 (40.3:61.8)
Sex	
• Female	5 (35.7)
• Male	9 (64.3)
Comorbidities	
• HTN	6 (42.9)
• Obesity	5 (35.7)
• T2 DM	6 (42.9)
• Anemia	3 (21.4)
• Non-Hodgkin Lymphoma	1 (7.1) 1 (7.1)
Previous infection to detection of *C. auris*	
• Yes	9 (64.3)
• No	5 (35.7)
Days until *C. auris*	
• Mean (SD)	31.2 (22.7)
• Median (IQR)	20.5 (16.5:41)
• Rango (min-sup)	6:75
Days of steroid therapy [10]	
• Mean (SD)	17 (13.5)
• Median (IQR)	11 (10:23.5)
• Range (min-sup)	3:54
Previous antibiotic therapy	
• Yes	13 (92.9)
• No	1 (7.1)

Table 1. *Cont.*

Characteristics	No. =14 n (%)
Days until *C. auris*	
• Mean (SD)	31.2 (22.7)
• Median (IQR)	20.5 (16.5:41)
• Rango (min-sup)	6:75
Days of steroid therapy [10]	
• Mean (SD)	17 (13.5)
• Median (IQR)	11 (10:23.5)
• Range (min-sup)	3:54
Previous antibiotic therapy	
• Yes	13 (92.9)
• No	1 (7.1)
Days until bacterial infection	
• Mean (SD)	27.4 (22.3)
• Median (IQR)	19 (14.3:32.5)
• Range (min-sup)	5:76
Duration of antibiotic therapy	
• Mean (SD)	21.9 (20.7)
• Median (IQR)	16 (7.5: 26.5)
Antifungal therapy	
• Yes	12 (85.7)
• No	2 (14.3)
Duration of antifungal therapy	
• Mean (SD)	(n = 12)
• Median (IQR)	10.5 (6.3)
	10 (7.5:13.3)
ID consultation	
• No	5 (35.7)
• Yes	9 (64.2)
In-hospital mortality	
• Yes	7 (100)

Abbreviations: SD: standard deviation; IQR: interquartile range; HTN: hypertension; T2DM: type 2 diabetes mellitus. Continuous variables are expressed as mean (SD), median (IQR), or range (minimum–maximum), as appropriate. Categorical variables are expressed as number (percentage).

All patients required invasive devices such as central venous catheter (CVC), invasive mechanical ventilation (IMV), nasogastric tubes (NGTs) and parenteral nutrition (PN) during their hospital stays. All patients had previously received steroid therapy due to their initial SARS-CoV-2 viral diagnosis, and 50% (7/14) were administered prolonged regimens lasting more than 10 days.

In total, 92.8% of the patients received broad-spectrum antibiotic therapies prior to the diagnosis of *C. auris* as part of the COVID-19 management scheme, with a median duration of 10 days. The most frequently used antibiotics are piperacillin/tazobactam, meropenem, and cefepime. Of these patients, 64.2% received antifungal therapy before the diagnosis of *C. auris* infection, and fluconazole was administered without prior detection of a proven invasive fungal infection.

C. auris fungemia was detected between days 6 and 75 after initial admission for SARS-CoV-2. Positive cultures of *C. auris* corresponded to fungemia in all cases, with funguria initially detected in three cases. The mean length of hospital stay was 35.2 days.

3.1. Bacterial Coinfection

All patients had bacterial coinfections; 57.1% had infections prior to the detection of fungemia, 28.5% developed infections after, and 14.2% had concomitant infections with the diagnosis of *C. auris*. The most common isolates were Gram-negative, non-fermenting bacteria, such as *Pseudomonas aeruginosa* (6/14) (42.8%), of which 3/6 exhibited a multidrug-resistant (MDR) pattern. Subsequently, *Acinetobacter baumannii* complex was identified in 5/14 (35.7%) patients, with 2/5 displaying an MDR pattern. Finally, *Klebsiella pneumoniae* with extended-spectrum beta-lactamase (ESBL) was noted. Among the Gram-positive bacteria, we found methicillin-resistant *Staphylococcus aureus* (MRSA) and Ampicillin-sensitive *Enterococcus faecium*. These bacterial isolates were predominantly obtained from pulmonary sources (64.2%) and bloodstream infections (35.7%) (Table 2).

Table 2. Clinical characteristics of 14 patients with pneumonia due to SARS-CoV-2 infection and disease caused by *C. auris*.

N° Patient	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Age/ Sex	39/ F	28/ M	39/ M	55/ M	52/ F	46/ M	52/ M	65/ M	64/ F	74/ M	31/ F	46/ M	44/ M	71/ F
Coinfections	Pa	A. bau MDR	E. faecium	Pa MDR	Kp ESBL	MRSA	Pa	A. bau	Pa	Pa MDR	A. bau	A. bau	AB MDR	Pa KPC, K. oxy
Leukocytes (×109 c/L)	12.3	20.3	9.8	17.9	19	12.2	21.2	13.4	18.8	16.8	15.4	12.6	NA	17,48
Lymphocytes (×109 c/L) (%)	31	25	5.1	31	15.0	23	25	31	25	5.1	31	15.0	NA	2,6
Neutrophils (×109 c/L) (%)	62	71	83.2	62.4	68.2	76	78	60	71	87.2	62.4	70.2	NA	91,2
Platelets (×109 c/L)	166	380	233	455	378	198	160	577	361	161	672	508	NA	291
Hemoglobin (gr/dL)	10.2	11.2	10.3	7.2	9.7	10.3	10.7	7	8	11.3	8.6	11.9	NA	NA
D-dimer (ng/mL)	3212	890	1390	2300	3212	3200	7880	3270	877	59,600	703	205	NA	NA
Ferritin (ng/mL)	2321	789	321	2100	2890	2100	3080	2000	1200	1146	367	2776	NA	NA
Invasive devices	IMV, CVC, NG, TT	IMV, CVC, NG	IMV, CVC	IMV, CVC, NG, TT	IMV, CVC, NG, TT	IMV, CVC, NG, TT	IMV, CVC, TT	IMV, CVC, NG, TT	IMV, CVC, NG	IMV, CVC, NG	IMV, CVC, NG, TT	IMV, CVC, NG, TT	IMV, CVC, NG	IMV, CVC, NG, TT

Abbreviations: Pa: *Pseudomonas aeruginosa*; A. bau: *Acinetobacter baumanii*; Kp ESBL: *Klebsiella pneumoniae* extended-spectrum beta-lactamase; CVC: central venous catheter; IMV: invasive mechanic ventilation; NA: not available; NG: nasogastric tube; TT: trach tube. Data unavailable for patients 13 and 14 due to incomplete records at time of chart review.

In total, 50% (7/14) of the patients died, and 4/7 (57.14%) had associated comorbidities (Table 1). Additionally, 100% of the deceased patients had invasive devices such as CVC, IMV and TPN and had received broad-spectrum antibiotic therapy prior to the detection of *C. auris* as part of the management of bacterial infections. Of these, 57.1% (4/7) received prolonged steroid therapy.

All deceased patients had bacterial coinfections, with the site of origin being pulmonary in 57.1% (4/7) due to ventilator-associated pneumonia (VAP) and bacteremia in 42.8% (3/7), and the most frequently isolated microorganism was *Pseudomonas aeruginosa* (57.1%, 4/7) (Table 2). In total, 5/7 (71.4%) of the deceased patients were not assessed by the infectious disease department.

The BioFire FilmArray BCID2 panel was not performed in any of the patients who died during hospitalization. Among patients who underwent molecular testing, antifungal

therapy was adjusted to caspofungin within 24 h of organism identification. In contrast, patients managed using conventional culture-based identification experienced a mean delay of approximately 120 h before targeted antifungal therapy was initiated. Although these observations suggest that rapid molecular diagnostics may facilitate earlier optimization of antifungal treatment, the small sample size and observational design preclude any conclusions regarding their impact on clinical outcomes.

3.1.1. Antifungal Susceptibility

Antifungal susceptibility results were interpreted using CDC tentative MIC thresholds because CLSI clinical breakpoints for *Candida auris* have not been established. Using these provisional criteria, 64.2% (9/14) of isolates exceeded the CDC tentative MIC threshold for fluconazole (MIC $\geq$ 32 µg/mL), while 71.4% (10/14) exceeded the tentative threshold for amphotericin B (MIC $\geq$ 2 µg/mL). In addition, 28.5% (4/14) of isolates exceeded the tentative MIC thresholds for both fluconazole and voriconazole. Six isolates (42.8%) exceeded the tentative MIC thresholds across multiple antifungal classes, including azoles and polyenes, and were therefore classified as multidrug-resistant according to current CDC surveillance criteria. The MIC values for echinocandins remained below CDC tentative thresholds in most isolates; however, two isolates demonstrated elevated micafungin MICs. Because species-specific clinical breakpoints for *C. auris* are unavailable, these findings should be interpreted as microbiological classifications based on provisional epidemiological thresholds rather than as definitive predictors of clinical treatment failure (Table 3).

Table 3. Antifungal minimum inhibitory concentrations (MICs) for the *Candida auris* isolates obtained during the outbreak.

Patient No.	MIC (µg/mL)				
	FLC	VRC	MFG	AMB	CAS
1	64	1	0.12	0.5	0.25
2	16	0.25	4	2	0.25
3	64	0.25	4	0.5	0.25
4	16	0.25	0.12	2	0.25
5	64	1	0.12	4	0.25
6	32	0.25	0.12	0.5	0.25
7	64	1	0.12	2	0.25
8	16	0.25	0.12	2	0.12
9	32	0.25	0.12	4	0.12
10	16	0.25	0.12	0.5	0.25
11	16	0.25	0.12	2	0.25
12	32	1	0.12	2	0.25
13	64	0.25	0.12	2	0.25
14	64	0.25	0.12	2	0.25

Abbreviations: FLC: fluconazole; VRC: voriconazole; MFG: micafungin; AMB: amphotericin B; CAS: caspofungin. MICs are expressed in µg/mL and were determined by broth microdilution per CLSI M27. Susceptibility categorization uses the CDC tentative MIC thresholds for *Candida auris* (fluconazole $\geq$ 32 µg/mL, amphotericin B $\geq$ 2 µg/mL, micafungin $\geq$ 4 µg/mL, and caspofungin $\geq$ 2 µg/mL); these thresholds are provisional and are NOT equivalent to established CLSI clinical breakpoints, which do not exist for *C. auris*.

A total of 85.7% (12/14) of patients received fluconazole empirically, and only 35.7% (5/14) of the detected isolates underwent Filmarray (BCID panel 2.0 Biomerieux®, North Ryde, NSW, Australia). All five (100%) of those tested had their antifungal coverage adjusted to caspofungin within the 24 h following the molecular report (on day 3 of fluconazole). None of the deceased patients (7/14) had a BCID 2.0 panel®, while adjustments

to therapy for the remaining patients were made after 7 days of empirical fluconazole treatment based on final culture reports.

3.1.2. Validity of Blood Cultures and Control of Fungemia Clearance

Fungaemia was not treated in two patients because it was assumed to be contaminated; both patients subsequently died. Blood culture control was performed in only 9 of the 14 (64.2%) patients, emphasizing the inadequate therapeutic approach for establishing the duration of therapy.

3.1.3. Cleaning, Environment Control and Cohortization

Contact isolation from admission was implemented in 7 of 14 patients, whereas the remaining seven were placed under contact precautions only after the outbreak was formally recognized. The outbreak was declared after identification of the seventh case (approximately day 80 of the surveillance period), and an institutional response was initiated within 24 h by the Hospital Infection Control Committee.

Control measures included patient cohorting in dedicated isolation areas, assignment of dedicated healthcare personnel, reinforcement of contact precautions, restriction of non-essential staff access, and intensified environmental cleaning and terminal disinfection procedures. Environmental decontamination was performed using sodium dichloroisocyanurate at a concentration equivalent to 4000 ppm of available chlorine, with routine cleaning conducted twice daily and additional terminal disinfection after patient discharge or transfer.

Visitor access was restricted, and screening cultures were obtained from epidemiologically linked contacts. No additional cases were identified through contact screening. Whenever feasible, medical equipment was dedicated to individual patients; shared devices underwent cleaning and disinfection after each use.

Of the 14 outbreak-associated cases, seven occurred before the implementation of outbreak-control measures and seven occurred during the intervention period. Environmental cultures, healthcare-worker screening cultures, and molecular typing of isolates were not performed.

4. Discussion

We describe a retrospective outbreak investigation of *Candida auris* fungemia involving 14 critically ill patients with SARS-CoV-2 infection during a period of substantial healthcare-system strain. The COVID-19 pandemic created unprecedented challenges for infection prevention and control, particularly in intensive care units, where overcrowding, prolonged hospitalization, extensive antimicrobial exposure, and shortages of healthcare resources may have facilitated the emergence and transmission of multidrug-resistant pathogens, including *C. auris* [12–14].

The characteristics of this outbreak are broadly consistent with reports from other countries describing *Candida auris* transmission among critically ill patients with COVID-19 [15]. Similar outbreaks have been reported in Brazil, Mexico, India, and Lebanon, where prolonged intensive care unit stays, mechanical ventilation, central venous catheters, corticosteroid exposure, and extensive antimicrobial use were common predisposing factors [3,5,16–19]. The recurrence of these risk factors across geographically distinct healthcare settings suggests that the convergence of critical illness, invasive supportive care, and healthcare-system strain during the COVID-19 pandemic created favorable conditions for the emergence and spread of *C. auris*.

Several observations suggest that infection prevention and control challenges may have contributed to transmission within our institution. Only half of the patients were

isolated upon admission, while the remainder were cohorted after recognition of the outbreak. Furthermore, multiple patients shared clinical areas before the implementation of enhanced control measures. Although environmental cultures and molecular typing were not performed, limiting confirmation of transmission pathways, the temporal and spatial clustering of cases is compatible with nosocomial spread. Similar findings have been described in outbreaks from Latin America, Asia, and the Middle East during the COVID-19 pandemic [16,17,20]. Therefore, our observations support the importance of timely implementation of contact precautions, cohorting strategies, and environmental disinfection as core components of outbreak control [11,21–25].

In-hospital mortality reached 50%, comparable to rates reported in other cohorts of critically ill patients with COVID-19 and *C. auris* infection [15,16,21]. Interpretation of mortality-associated findings should be approached cautiously given the small sample size and the presence of multiple potential confounders, including baseline severity of illness, bacterial coinfections, prolonged intensive care exposure, and healthcare-system strain. Most survivors received infectious diseases consultation during their hospitalization, whereas specialist involvement was less frequent among non-survivors. Because of the small cohort size and the absence of a formal adjusted analysis, no conclusions can be drawn regarding the relationship between infectious diseases consultation and mortality. Nevertheless, early specialist involvement remains an important component of candidemia management and outbreak response [26,27].

Our findings also highlight the importance of maintaining robust laboratory and infection prevention capacity during periods of healthcare-system stress. Accurate species identification remains essential because *C. auris* may be misidentified by conventional phenotypic methods, potentially delaying both appropriate antifungal therapy and implementation of infection-control measures [28]. Rapid molecular diagnostics were available for a subset of patients and facilitated earlier species identification and antifungal treatment adjustment. However, the observational design and limited sample size preclude conclusions regarding their effect on survival. The apparent association between molecular testing and more timely antifungal optimization should therefore be interpreted cautiously and viewed as hypothesis-generating rather than confirmatory [28,29].

Antifungal susceptibility testing demonstrated that a substantial proportion of isolates exceeded CDC tentative MIC thresholds for fluconazole and amphotericin B, consistent with previous reports from Colombia [10] and other countries in the region [19]. Importantly, because CLSI clinical breakpoints for *C. auris* have not been established, susceptibility classifications in this study were based on CDC tentative epidemiological thresholds and should not be interpreted as definitive predictors of clinical response [30]. Most isolates demonstrated MIC values below CDC tentative thresholds for echinocandins, supporting their continued role as first-line therapy [31]. Two isolates exhibited elevated micafungin MICs; however, the clinical significance of this finding remains uncertain and warrants continued surveillance.

This study has several limitations. First, its retrospective design and small sample size limited statistical power and prevented robust assessment of independent predictors of mortality. Consequently, all comparative analyses should be interpreted as exploratory. Second, important severity indicators, including SOFA scores, vasopressor requirements, renal replacement therapy, and oxygenation parameters, were not available. Third, environmental cultures, healthcare-worker screening cultures, and genomic typing were not performed, preventing confirmation of environmental reservoirs, transmission pathways, or clonal relatedness of isolates. Finally, incomplete follow-up blood culture data limited assessment of microbiological clearance and treatment response.

Despite these limitations, this outbreak investigation provides clinically relevant information regarding the epidemiological, microbiological, and infection-control characteristics of *C. auris* fungemia among critically ill patients with COVID-19. Our findings reinforce the importance of early recognition, accurate laboratory identification, prompt implementation of infection prevention measures, and appropriate antifungal management in controlling healthcare-associated *C. auris* outbreaks.

5. Conclusions

This outbreak investigation describes 14 cases of *Candida auris* fungemia among critically ill patients with SARS-CoV-2 infection in a tertiary-care hospital during the COVID-19 pandemic. All patients had prolonged hospitalization, invasive devices, and extensive exposure to broad-spectrum antimicrobials, highlighting the vulnerability of critically ill populations to healthcare-associated fungal infections. A substantial proportion of isolates exceeded CDC tentative MIC thresholds for fluconazole and amphotericin B, underscoring the therapeutic challenges posed by *C. auris*.

The temporal clustering of cases and delayed implementation of infection-control measures are consistent with nosocomial transmission, although environmental cultures and molecular typing were not available to confirm transmission pathways. These findings emphasize the importance of early recognition, accurate laboratory identification, prompt infection prevention interventions, and appropriate antifungal management during suspected *C. auris* outbreaks. Given the small sample size and retrospective design, all comparative observations should be considered exploratory. Further multicenter studies incorporating environmental sampling and genomic epidemiology are needed to better characterize transmission dynamics and optimize outbreak-control strategies.

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Informed Consent Statement: Because this research involved retrospective analysis of de-identified existing data, individual informed consent was not required by the Ethics Committee in accordance with institutional and applicable regulatory guidelines.

Data Availability Statement: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

SARS-CoV-2	Severe Acute Respiratory Syndrome-Coronavirus 2
INS	National Institute of Health
PICCS	Peripherally Inserted Central Lines

MDR:	Multidrug-Resistant
HAIs	Hospital-Acquired Infections
MV	Mechanical Ventilation
PAHO	Pan American Health Organization
WHO	World Health Organization
CLSI	Clinical and Laboratory Standards Institute
CDC	Centers for Disease Control and Prevention
MIC	Minimum Inhibitory Concentration
CVC	Central Venous Catheter
IMV	Invasive Mechanical Ventilation
NGT	Nasogastric Tube
PN	Parenteral Nutrition
ESBL	Extended-Spectrum Beta-Lactamase
MRSA	Methicillin-Resistant Staphylococcus Aureus
VAP	Ventilator-Associated Pneumonia
BCID	Blood Culture Identification Panel

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