Original Article

Identification and Antifungal Drug Susceptibility Pattern of *Candida auris* in India

Smita Deshkar, Niranjan Patil, Shraddha Amberkar, Ashish Lad, Farozan Siddiqui[1], Swati Sharan[2]

Departments of Infectious Diseases, [1]Microbiology and [2]Molecular Biology, Metropolis Healthcare Limited, Global Reference Laboratory, Mumbai, Maharashtra, India

Abstract

Introduction: *Candida auris* has turned up as a multidrug-resistant nosocomial agent with outbreaks reported worldwide. The present study was conducted to evaluate the antifungal drug susceptibility pattern of *C. auris*. **Methods:** Isolates of *C. auris* were obtained from clinically suspected cases of candidemia from January 2019 to June 2021. Identification was done with matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) and panfungal DNA polymerase chain reaction (PCR), followed by sequencing. Antifungal susceptibility testing was performed with broth microdilution method. **Results:** Out of 50 isolates *C. auris*, 49 were identified by MALDI-TOF and one isolate was identified with panfungal DNA PCR followed by sequencing. For fluconazole, 84% ($n = 42$) isolates were found to be resistant and 16% ($n = 8$) isolates were susceptible (minimum inhibitory concentrations [MICs] range 0.5–16). Posaconazole exhibited potent activity, followed by itraconazole. For amphotericin B, only 6% ($n = 3$) isolates were resistant with MICs ≥2 µg/mL. Only 4% ($n = 2$) isolates exhibited resistance to caspofungin. No resistance was noted for micafungin and anidulafungin. One (2%) isolate was found to be panazole resistant. One (2%) isolate was resistant to fluconazole, amphotericin B, and caspofungin. **Conclusion:** Correct identification of *C. auris* can be obtained with the use of MALDI-TOF and sequencing methods. A small percentage of fluconazole-sensitive isolates are present. Although elevated MICs for amphotericin B and echinocandins are not generally observed, the possibility of resistance with the irrational use of these antifungal drugs cannot be denied. Pan azole-resistant and pan drug-resistant strains of *C. auris* are on rise.

Keywords: Antifungal drug susceptibility, broth microdilution, *Candida auris,* identification, matrix-assisted laser desorption/ionization-time of flight, panfungal polymerase chain reaction

Introduction

Candida spp. infections are commonly associated with morbidity and mortality in critically ill patients.[1] A wide range of clinical manifestations, including bloodstream infections (BSIs), intra-abdominal candidiasis, deep-seated candidiasis, and superficial infections are caused by *Candida* spp.[1] Infections caused by *Candida* spp. have gradually increased over the last decades. This is associated with the increasing rate of invasive procedures, the extensive use of broad-spectrum antimicrobials agents as well as frequent immunocompromised status of critically ill patients.[2] Even though *Candida albicans* is a predominant cause of hospital-acquired fungal infection, several species of nonalbicans *Candida* namely *Candida tropicalis*, *Candida glabrata*, *Candida parapsilosis*, and *Candida krusei* account for increasing incidence of invasive fungal infections (IFIs) with high rates of therapeutic failure.[2] Since its isolation in the ear of a Japanese patient with external otitis in 2009, many reports of hospital outbreaks and IFI caused by *Candida auris* (*C. auris*) have been described in at least 44 countries across six continents.[3] Crude mortality rates are varied but have been reported to be as high as 66%.[4]

In 2016 and 2017, the Centers for Disease Control and Prevention (CDC) released a couple of clinical alerts to warn the emergence of *C. auris* infections.[3] In 2019, in the Report on Urgent Threats from the CDC, *C. auris* was

Access this article online

Quick Response Code:

Website: www.jgid.org

DOI: 10.4103/jgid.jgid_44_22

Address for correspondence: Dr. Smita Deshkar, Department of Infectious Diseases, Metropolis Healthcare Limited, Kohinoor City, Commercial Bldg. - A, 4th Floor, Off-L.B.S. Road, Vidyavihar (West), Mumbai - 400 070, Maharashtra, India.
E-mail: smitadeshkar@ymail.com

How to cite this article: Deshkar S, Patil N, Amberkar S, Lad A, Siddiqui F, Sharan S. Identification and antifungal drug susceptibility pattern of *Candida auris* in India. J Global Infect Dis 2022;14:131-5.

Received: 26 February 2022 **Revised:** 04 April 2022
Accepted: 19 April 2022 **Published:** 01 November 2022

again categorized as one of the main urgent threats, together with other multidrug-resistant nosocomial organisms.[5] An advisory note was released by the Indian Institute of Medical Research (ICMR) to ensure active surveillance of this species in Indian hospitals.[6] Diagnosis and antifungal susceptibility testing (AFST) of *C. auris* are a challenge. *C. auris* can be misidentified as *Candida haemulonii*, *Candida duobushaemulonii, Candida lusitaniae*, *Candida sake,* and *Rhodotorula mucilaginosa* by commercial identification systems.[7] Automated susceptibility testing methods give misleading minimum inhibitory concentrations (MICs), thus overestimating resistance.[8] Existing breakpoints for the broth microdilution (BMD) method, which is recommended by the Clinical and Laboratory Standards Institute (CLSI), are defined based on expert opinion.[8] There are limited studies about the drug resistance profile of *C. auris* in India. Therefore, the present study was conducted to study the antifungal drug susceptibility pattern of *C. auris* by BMD method.

Methods

The present cross-sectional study was carried out with 50 isolates of *C. auris* obtained from clinically suspected cases of candidemia between January 2019 and July 2021. Approval from an independent ethics committee (Conscience Independent Ethics Committee; Protocol ID MHL/Mol/2021/05) was obtained with a waiver on the patient's consent. The study was conducted as per an approved study protocol.

Identification by culture methods

All *Candida spp*. isolates, from various clinical specimens (n = 5131) underwent standard mycological procedures, including isolation on Sabouraud dextrose agar with chloramphenicol (0.5 g/100 mL) and gentamicin (0.3 g/100 mL) (HiMedia, Mumbai, India), species identification on DifcoTM CHROMagar Candida medium (Becton Dickinson and Co., Baltimore, MD, USA). The results were read visually.

Identification by commercial systems

All *Candida* spp. isolates (n = 5131) underwent testing on VITEK MS v. 3.0 (bioMérieux, France), an automated mass spectrometry microbial identification system that uses matrix-assisted laser desorption/ionization-time-of-flight mass spectrometry (MALDI-TOF MS) technology either using the Food and Drug Administration (FDA)-approved IVD v3.2 or "research use only" libraries.

Target sequence analysis of the 28S rRNA gene and internal transcribed spacer region

Genomic DNA was extracted from test isolate using a QIAamp1 DNA Mini Kit (QIAGEN, Hilden, Germany). DNA was amplified and sequenced on 3500DX Analyser (ThermoFisher Scientific, Massachusetts, USA) using internal transcribed spacer (ITS) and 28S rRNA primers, which amplify the ITS region and the D1/D2 domain of the large ribosomal subunit of the 28S rRNA gene. The sequences were aligned and then run through GenBank Basic Local Alignment Search Tool searches for species identification.

Antifungal susceptibility testing

All isolates confirmed as *C. auris* were subjected to AFST by the CLSI-recommended BMD method for azoles (fluconazole, voriconazole, posaconazole, and itraconazole), amphotericin B, and echinocandins (caspofungin, micafungin, and anidulafungin). Breakpoints were defined based on expert opinion as released by the US CDC in October 2017 and modified in April 2019 as mentioned in table 1.[8,9] Quality control was performed as per standard operating protocol using *Candida parapsilosis* ATCC 22019 and *Candida krusei* ATCC 6258. The MIC distributions was spread over 11 dilutions (0.008, 0.015, 0.03, 0.06, 0.12, 0.25, 0.5, 1, 2, 4, 8) for fluconazole voriconazole, itraconazole, posaconazole, caspofungin, and micafungin. The MIC distributions spread over 10 dilutions for anidulafungin (0.015, 0.03, 0.06, 0.12, 0.25, 0.5, 1, 2, 4, 8) and 7 dilutions for amphotericin B (0.12, 0.25, 0.5, 1, 2, 4, 8).

Results

Out of 5131 *Candida* spp. isolates, 50 (0.97%) isolates were of *C. auris*, 49 were identified by MALDI-TOF MS with 99.9 confidence value and one isolate was identified with panfungal DNA polymerase chain reaction (PCR) followed by sequencing (Gene bank accession number MH549191.1) with 99.25% identification. Out of 50 *C. auris* isolates, 43 (86%) were isolated from blood, 4 (8%) from urine, 2 (4%) from tissue, and 1 (2%) was isolated from pus specimen. All 50 *C. auris* isolates were tested for AFST by BMD against eight antifungals fluconazole, voriconazole, itraconazole, posaconazole, amphotericin B, caspofungin, micafungin, and anidulafungin. MIC distribution, Modal MICs, Geometric Mean (GM), MIC range, MIC 50, and MIC 90 are shown in tables 2 and 3. Among the azoles, posaconazole (GM 0.05 μg/mL) exhibited the most potent activity, followed by itraconazole (GM 0.14 μg/mL). Considerably, 84% (n = 42) of isolates exhibited resistance to fluconazole with MICs between 32 and ≥64 μg/mL. Significantly, 16% (n = 8) isolates were susceptible to fluconazole with MICs between 0.5 and

Table 1: Tentative minimum inhibitory concentration breakpoints of *Candida auris* by Centers for Disease Control and Prevention

Antifungal drug	Tentative MIC breakpoints (μg/mL)
Fluconazole	≥32
Voriconazole and other second generation triazoles	NA
Amphotericin B	≥2
Anidulafungin	≥4
Caspofungin	≥2
Micafungin	≥4

MIC: Minimum inhibitory concentration, NA: Not available

Table 2: Minimum inhibitory concentration distribution of *Candida auris* isolates against antifungal drugs tested (*n*=50)

Drug	≤0.015	0.032	0.064	0.125	0.25	0.5	1	2	4	8	16	32	≥64
Fluconazole						1	1	1	1	1	3	8	34
Voriconazole		1	3	14	10	6	5	3	3			3	2
Itraconazole		8	9	15	11	3	2	1				1	
Posaconazole	14	13	11	7	1	1	1		1			1	
Amphotericin B			1	3	16	25	2		2	1			
Caspofungin				9	7	7	25	1	1				
Micafungin	1	2	17	20	8	1	1						
Anidulafungin	1	1	4	19	10	14	1						

Table 3: Geometric mean and minimum inhibitory concentrations distribution of antifungal drugs for *Candida auris* (*n*=50)

Drug	*GM	Range	†MIC 50	†MIC 90
Fluconazole	42.81368	0.5-≥64	64	64
Voriconazole	0.50794	0.032-≥64	0.25	4
Itraconazole	0.14475	0.032-32	0.125	0.5
Posaconazole	0.05441	≤0.015-32	0.032	0.125
Amphotericin B	0.41774	0.064-8	0.5	0.5
Caspofungin	0.53589	0.125-4	1	1
Micafungin	0.10820	≤0.015-1	0.125	0.25
Anidulafungin	0.20429	≤0-1	0.125	0.5

*GM: Geometric mean, †MIC: Minimum inhibitory concentration

16 μg/mL. For itraconazole, a modal MIC of 0.125 μg/mL was noted and 8% (*n* = 4) had an MIC range of 1–32 μg/mL. For amphotericin B, 94% (*n* = 47) of isolates were susceptible and 6% (*n* = 3) isolates had MICs ≥2 μg/mL, indicating resistance. No resistance was observed for micafungin and anidulafungin. Micafungin and anidulafungin MICs distribution spanned within ± 3 2-fold dilution of the modal MIC (0.125 μg/mL). Among echinocandins, modal MICs of micafungin, anidulafungin, and caspofungin were 0.125, 0.125, and 1 μg/mL, respectively. Caspofungin resistance was observed in 4% (*n* = 2) isolates. One (2%) isolate was panazole resistant. One (2%) isolate was resistant to fluconazole, amphotericin B, and caspofungin (Table 4).

Discussion

Correct identification of *C. auris* is a major diagnostic challenge. Due to close genetic relatedness with *Candida haemulonii* complex, *C. auris* is often misidentified as *Candida haemulonii* in routine diagnostic laboratories.[2] Commercially available biochemical-based tests, including API AUX 20C, VITEK-2 YST, BD Phoenix, and MicroScan also misidentify *C. auris* as a wide range of *Candida* spp.[10] Studies conducted by Kordalewska *et al*., Hata *et al*. and Kathuria *et al*. affirmed the suitability of MALDI-TOF MS as a more facile approach for *C. auris* confirmation.[11-13] Notably, the US FDA had approved the BRUKER MALDI Biotyper CA system and the bioMérieux Vitek MS for *C. auris* identification.[14] In the present study, we could correctly identify 49 isolates as *C. auris* by MALDI-TOF MS (bioMérieux, France); however, one isolate remained unidentified which was subjected to identification by panfungal DNA PCR and sequencing. Sequencing of genetic loci, including D1/D2, RPB1, RPB2, and ITS domains of the rRNA, remains the gold standard for the identification of *C. auris*, but it is not used as a routine diagnostic procedure and is unlikely to be available outside reference laboratories.[15] However, more recent development of PCR assays specific for *C. auris* and for *C. auris*-related species could prove useful, particularly in outbreak situations.[11]

Based on a recent review by Lockhart, resistance is the new norm for *C. auris* with a minority of clinical isolates displaying antifungal susceptibility.[16] Resistance has been observed across all main classes of antifungals, with a significantly high proportion of isolates being reported as multidrug-resistant. Epidemiological cut-off values for *C. auris* do not exist. Only the interim tentative breakpoints proposed by CDC are available.[4] Most notably, *C. auris* is frequently associated with high levels of fluconazole resistance, with multiple studies reporting over 90% of isolates nonsusceptible to fluconazole.[17-19] Studies from India had shown susceptibility of 9.1% (1/11) and 9.71% (34/350) for fluconazole.[8,20] However, regarding voriconazole, variable susceptibility patterns have been reported in previous studies in a limited number of isolates.[13,17-19,21-24] A study from Colombia observed that only 23% (4/17) isolates were nonsusceptible to voriconazole (MICs ≥2 μg/mL)[19] whereas all *C. auris* isolates reported from Spain (*n* = 8) and Venezuela (*n* = 18), had voriconazole MICs ≥2 μg/mL.[18,25] Of particular note, elevated MICs for itraconazole and posaconazole were not documented.[17,20,26] Consistent with the previous findings, in the present study, 84% (42/50) of isolates were resistant to fluconazole. For voriconazole, itraconazole, and posaconazole only 22% (11/50) isolates had MIC ≥2 μg/mL. It was suggested that high fluconazole resistance could be related to the fact that majority of tested isolates were from highly fluconazole-resistant South Asia clade, and significant number of fluconazole susceptible isolates are still present, especially within the East Asia and South American clades.[16,17,22] In contrast to earlier reports, recent reports from India have documented few isolates susceptible to fluconazole.[8,20,23] In the present study, we found 16% (*n* = 8) isolates susceptible to fluconazole (MICs Range 0.5–16). These significant

Table 4: Distribution of *Candida auris* isolates exhibiting multidrug resistance

Resistance type	Number of isolates (%)
Fluconazole + Amphotericin B	2 (4)
Fluconazole + Caspofungin	1 (2)
Fluconazole + Amphotericin B+Caspofungin	1 (2)
Fluconazole + Voriconazole	5 (10)
Pan Azole	1 (2)

fluconazole susceptible isolates could indicate that either resistance can be lost or that new clones are emerging.[16]

Although resistance to amphotericin B is not as common as fluconazole, it is reported variably from 0%–30%.[17,18,25] In accordance with these findings, we observed amphotericin B resistance rate of 6% (3/50) among our *C. auris* isolates. The higher MIC values to amphotericin B for *C. auris* are not generally observed as those are seen for other species in the Metschnikowiaceae family, with most resistant isolates being in the 2–4 μg/mL range.[16] It has been also suggested that amphotericin B resistance is inducible and transient; the MIC values of some isolates decrease following serial passage.[16] A widespread fluconazole resistance and variable amphotericin B resistance has been documented, but echinocandin resistance is not as common.[13,17,18,21,23,24] The first echinocandin-resistant isolates were reported in 2015[13] and then after few reports documenting echinocandin resistance were published.[17,20,24,27] In the present study, 4% (n = 2) isolates were found to be resistant to echinocandin caspofungin. Comparable levels of resistance (2% and 7%, respectively) were reported for *C. auris* isolates collected in India (10 hospitals) and by the CDC (from hospitals in Pakistan, India, South Africa, and Venezuela).[17,20] Although echinocandin-resistant isolates are relatively rare, they are a significant proportion of some populations of *C. auris* and there is a possibility that echinocandin resistance will rise in regions where they are available, as echinocandins are drug of choice for *C. auris* infections.[16] A combination therapy of an echinocandin and liposomal amphotericin B is widely advised in cases not responding to echinocandins, as synergistic interactions have a better efficacy.[28] Three cases of pan-resistant *C. auris* with resistance to all three classes of commonly prescribed antifungal drugs have been reported in the US.[29] Chowdhary *et al*. also reported 14 (4%) panazole-resistant *C. auris* isolates in India.[20] In the present study, we observed one (2%) panazole-resistant isolate and one (2%) isolate resistant to fluconazole, amphotericin B, and caspofungin. Although the overall numbers of pan-resistant cases reported so far are few, it is an alarming signal as *C. auris* infections are associated with limited treatment options, high mortality rates, and easy transmissibility in healthcare facilities. Even though *C. auris* is a multidrug resistant, all *C. auris* isolates should undergo AFST according to CLSI guidelines, as levels of antifungal resistance can vary widely across isolates.[9] AFST is therefore of utmost importance for in patients with clinically suspected or laboratory-confirmed IFI, when acquired resistance is suspected, or in patients with refractory, relapsing, or breakthrough fungal infection. Patients should be closely monitored and followed up, and microbiological culture-based reassessment is essential to detect the therapeutic failure and possible development of resistance by molecular methods if required.[30] The development of antifungal stewardship programs is essential for an adequate and early treatment and thus consequently to make the emergence of antifungal resistance less likely. With the alarming emergence of antifungal resistance, there is an increasing and urgent need for the development of new antifungal therapies.

The limitation of this study should be noted. We did not study *in vivo* clinical responses which limited reliability of *in vitro* antifungal susceptibility results as there might be a gap between *in vitro* susceptibility/resistance results and the clinical outcome. Molecular resistance determinant analysis of *C. auris* isolates was not performed, which could have helped in understanding the mechanisms of resistance.

Conclusion

Correct identification of *C. auris* is a major diagnostic challenge which can be overcome with the use of MALDI-TOF MS and sequencing methods. Although a high number of *C. auris* isolates are fluconazole resistant, a small percentage of fluconazole sensitive isolates are also present. Elevated MICs for amphotericin B and echinocandins were not noted in majority of isolates, the possibility of resistance with the irrational use of these antifungal drugs cannot be denied. Pan azole-resistant and pan drug-resistant strains are on rise and may continue to emerge independently, simultaneously, and rapidly throughout the world in coming years. Correct pathogen identification, continuous evaluation, and judicious use of antifungal drugs are of utmost importance to control the spread and improve diagnostic and therapeutic strategies of *C. auris*.

Research quality and ethics statement

This study was approved by the Independent Ethics Committee (Conscience Independent Ethics committee DGCI Reg Number ECR/233/Indt/GJ/2015/RR-21). The authors followed applicable EQUATOR Network guidelines (http://www.equator-network.org/) during the conduct of this research project.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Kullberg BJ, Arendrup MC. Invasive candidiasis. N Engl J Med 2015;373:1445-56.
2. Cortegiani A, Misseri G, Fasciana T, Giammanco A, Giarratano A, ChowdharyA. Epidemiology, clinical characteristics, resistance, and treatment of infections by *Candida auris*. J Intensive Care 2018;6:69.
3. Fan S, Zhan P, Bing J, Jiang N, Huang Y, Chen D, *et al*. A biological and genomic comparison of a drug-resistant and a drug-susceptible strain of *Candida auris* isolated from Beijing, China. Virulence 2021;12:1388-99.

4. Kean R, Ramage G. Combined antifungal resistance and biofilm tolerance: The global threat of *Candida auris*. mSphere 2019;4:e00458-19.
5. Garcia-Bustos V, Cabanero-Navalon MD, Ruiz-Saurí A, Ruiz-Gaitán AC, Salavert M, Tormo MÁ, *et al*. What do we know about *Candida auris*? State of the art, knowledge, gaps, and future directions. Microorganisms 2021;9:2177.
6. Indian Council of Medical Research (ICMR). *Candida Auris* in Healthcare Settings – India. New Delhi, India: ICMR; 2017. [Last accessed on 2020 Mar 30].
7. Forsberg K, Woodworth K, Walters M, Berkow EL, Jackson B, Chiller T, *et al. Candida auris*: The recent emergence of a multidrug-resistant fungal pathogen. Med Mycol 2019;57:1-12.
8. Ninan MM, Sahni RD, Chacko B, Balaji V, Michael JS. *Candida auris*: Clinical profile, diagnostic challenge and susceptibility pattern: Experience from a tertiary-care centre in South India. J Glob Antimicrob Resist 2020;21:181-5.
9. Centers for Disease Control and Prevention (CDC) *Candida Auris*. Antifungal Susceptibility Testing and Interpretation. Atlanta, GA: CDC; 2020. Available from: https://www.cdc.gov/fungal/candida-auris/c-auris-antifungal.html.
10. Centers for Disease Control and Prevention (CDC) *Candida auris* Identification. Atlanta, GA: CDC; 2018. Available from: https://www.cdc.gov/fungal/candida-auris/recommendations.html.
11. Kordalewska M, Perlin DS. Identification of drug resistant *Candida auris*. Front Microbiol 2019;10:1918.
12. Hata DJ, Humphries R, Lockhart SR**;** College of American Pathologists Microbiology Committee. *Candida auris*: An emerging yeast pathogen posing distinct challenges for laboratory diagnostics, treatment, and infection prevention. Arch Pathol Lab Med 2020;144:107-14.
13. Kathuria S, Singh PK, Sharma C, Prakash A, Masih A, Kumar A, *et al.* Multidrug resistant *Candida auris* misidentified as *Candida* haemulonii: Characterization by matrix assisted laser desorption time of flight mass spectrometry and DNA sequencing and its antifungal susceptibility profile variabilbity by Vitek 2, CLSI broth microdilution, and Etest method. J Clin Microbiol 2015;53:1823-30.
14. Zhu Y, O'Brien B, Leach L, Clarke A, Bates M, Adams E, *et al.* Laboratory analysis of an outbreak of *Candida auris* in New York from 2016 to 2018: Impact and lessons learned. J Clin Microbiol 2020;58:e01503-19.
15. Jeffery-Smith A, Taori SK, Schelenz S, Jeffery K, Johnson EM, Borman A, *et al. Candida auris*: A review of the literature. Clin Microbiol Rev 2018;31:e00029-17.
16. Lockhart SR. *Candida auris* and multidrug resistance: Defining the new normal. Fungal Genet Biol 2019;131:103243.
17. Lockhart SR, Etienne KA, Vallabhaneni S, Farooqi J, Chowdhary A, Govender NP, *et al.* Simultaneous emergence of multidrug-resistant *Candida auris* on 3 continents confirmed by whole-genome sequencing and epidemiological analyses. Clin Infect Dis 2017;64:134-40.
18. Calvo B, Melo AS, Perozo-Mena A, Hernandez M, Francisco EC, Hagen F, *et al.* First report of *Candida auris* in America: Clinical and microbiological aspects of 18 episodes of candidemia. J Infect 2016;73:369-74.
19. Ruiz-Gaitán AC, Cantón E, Fernández-Rivero ME, Ramírez P, Pemán J. Outbreak of *Candida auris* in Spain: A comparison of antifungal activity by three methods with published data. Int J Antimicrob Agents 2019;53:541-6.
20. Chowdhary A, Prakash A, Sharma C, Kordalewska M, Kumar A, Sarma S, *et al.* A multicentre study of antifungal susceptibility patterns among 350 *Candida auris* isolates (2009-17) in India: Role of the ERG11 and FKS1 genes in azole and echinocandin resistance. J Antimicrob Chemother 2018;73:891-9.
21. Prakash A, Sharma C, Singh A, Kumar Singh P, Kumar A, Hagen F, *et al.* Evidence of genotypic diversity among *Candida auris* isolates by multilocus sequence typing, matrix-assisted laser desorption ionization time-of-flight mass spectrometry and amplified fragment length polymorphism. Clin Microbiol Infect 2016;22: 9.e1-9.
22. Chowdhary A, Anil Kumar V, Sharma C, Prakash A, Agarwal K, Babu R, *et al.* Multidrug-resistant endemic clonal strain of *Candida auris* in India. Eur J Clin Microbiol Infect Dis 2014;33:919-26.
23. Rudramurthy SM, Chakrabarti A, Paul RA, Sood P, Kaur H, Capoor MR, *et al. Candida auris* candidaemia in Indian ICUs: Analysis of risk factors. J Antimicrob Chemother 2017;72:1794-801.
24. Vallabhaneni S, Kallen A, Tsay S, Chow N, Welsh R, Kerins J, *et al.* Investigation of the first seven reported cases of *Candida auris*, a globally emerging invasive, multidrug resistant fungus – United States, May 2013-August 2016. MMWR Morb Mortal Wkly Rep 2016;65:1234-7.
25. Morales-López SE, Parra-Giraldo CM, Ceballos-Garzón A, Martínez HP, Rodríguez GJ, Álvarez-Moreno CA, *et al.* Invasive infections with multidrug-resistant yeast *Candida auris*, Colombia. Emerg Infect Dis 2017;23:162-4.
26. Magobo RE, Corcoran C, Seetharam S, Govender NP. *Candida auris*-associated candidemia, South Africa. Emerg Infect Dis 2014;20:1250-1.
27. Kordalewska M, Lee A, Park S, Berrio I, Chowdhary A, ZhaoY, *et al.* Understanding echinocandin resistance in the emerging pathogen *Candida auris*. Antimicrob Agents Chemother 2018;62:e00238-18.
28. Ademe M, Girma F. *Candida auris*: From multidrug resistance to pan-resistant strains. Infect Drug Resist 2020;13:1287-94.
29. Ostrowsky B, Greenko J, Adams E, Quinn M, O'Brien B, Chaturvedi V, *et al*. *Candida auris* isolates resistant to three classes of antifungal medications – New York, 2019. MMWR Morb Mortal Wkly Rep 2020;69:6-9.
30. Durand C, Maubon D, Cornet M, Wang Y, Aldebert D, Garnaud C. Can we improve antifungal susceptibility testing? Front Cell Infect Microbiol 2021;11:720609.