

Risk factors and mortality of the newly emerging *Candida auris* in a university hospital in Saudi Arabia

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ABSTRACT

Candida auris presents a global health threat. We investigated risk factors and mortality of *Candida auris* infections in a retrospective study in Saudi Arabia. We included 27 patients ≥ 14 with invasive *Candida auris* from 2015–2022, with median age 58, and 66.7% males. All patients had indwelling devices. The most common infection sources were central line-associated bloodstream infection in 17 (63.0%), and urinary tract infections in four (12%). Fever and shock were observed in nine patients (33.3%) each, and 22 (81%) were admitted to the intensive care unit. Common comorbidities were diabetes and heart disease in 13 (48.1%) patients each. The median hospital stay was 78 days, and the median Charlson index was 4. The *C. auris* cultures were 100% susceptible to voriconazole, caspofungin, and amphotericin, while three were fully susceptible to fluconazole (11.1%). Despite treatment, 18 (66.7%) patients died. In conclusion, invasive *C. auris* infection had varied presentations. All patients had indwelling devices, and many had lengthy hospital stays. All isolates were susceptible to amphotericin and echinocandins, while few were fully susceptible to fluconazole.

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1. Introduction

Candida auris is an important yeast emerging as a pathogen that causes significant mortality, morbidity and hospital outbreaks worldwide. It was first isolated in 2009 from the ear of a Japanese patient, hence the name “auris”, since then many outbreaks of this pathogen have been reported (Arendrup and Patterson 2017; Clancy and Nguyen 2017; McCarthy and Walsh 2017; Jeffery-Smith et al. 2017; Vinuela-Sandoval et al. 2018). A retrospective analysis of *C. auris* infections worldwide from 2009 to 2020 found that the five risk factors most often found in patients were a history of broad-spectrum antibiotic treatment (55.9%), central venous catheter (55.1%), intensive care unit (48.9%), urinary catheter (38.0%), and surgery (37.1%) (Hu et al. 2021). *Candida auris* outbreaks are mainly ascribed to the ease of transmission and the persistence of the organism in the hospital environment (Lone and Ahmad 2019). Not surprisingly, risk factors for *C. auris* skin colonisation were being on a ventilator, receiving carbapenems or fluconazole in the prior

90 days, and an acute care hospital visit in the prior six months (Rossow et al. 2021). Several large outbreaks have been reported with treatment failure due to echinocandin and azole resistance and a high mortality rate (Borman et al. 2016). The coronavirus disease 2019 (COVID-19) pandemic contributed to the spread of *C. auris* as there was a sharp rise in the number of outbreaks reported worldwide (Chowdhary and Sharma 2020; Prestel et al. 2021), several of which were in tertiary hospitals in Saudi Arabia and intensive care units around the world (Alshamrani et al. 2021; Magnasco et al. 2021).

C. auris is difficult to treat as its antifungal resistance pattern is unfavourable, with reports of fluconazole resistance exceeding 90%, amphotericin resistance exceeding 35%, and combined resistance in over 40% (Morales-Lopez et al. 2017; Eyre et al. 2018). The organism can cause clinical disease such as bloodstream infections secondary to central line or abdominal infections, that can be complicated by infective endocarditis. There are several reports of

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catheter-associated and complicated urinary tract infections (UTI) caused by *C. auris* (Clancy and Nguyen 2017; McCarthy and Walsh 2017; Vinuela-Sandoval et al. 2018; Griffith and Danziger 2020). *C. auris* is also known to cause meningitis and otomycosis, although this last clinical presentation appears to mainly involve isolates from the East-Asian clade (Abastabar et al. 2019; Mirhendi et al. 2022). The prevalence of *C. auris* infections is probably underestimated due to both difficulties in microbiological identification and diverse clinical presentations (Abastabar et al. 2019; Mirhendi et al. 2022). The most dangerous clinical presentation is bloodstream infection which carries a very high crude mortality rate of 40% (Hori and Shibuya 2018; Chowdhary and Sharma 2020; Du et al. 2020; Griffith and Danziger 2020; Centers for Disease Control and Prevention 2021).

Except for some case reports, very little has been published about *C. auris* infections in Saudi Arabia, so knowledge about the clinical disease, risk factors, presentation, antifungal resistance, and predictors of mortality is lacking. The purpose of our study was to elucidate all information concerning *C. auris* infections in Saudi Arabia. Hereto we performed a retrospective analysis of *C. auris* infections at a large University Hospital in Saudi Arabia.

2. Patients and methods

2.1. Patient selection and data collection

We conducted a retrospective study at the King Abdulaziz University Hospital in Jeddah, Arabia, which is a hospital with 1,000 beds. We reviewed all positive cultures that grew *C. auris* from 1 January 2015, until 31 December 2022. We included patients aged 14 years and older with clinical disease and one or more *C. auris* positive cultures from blood, body fluid, cerebrospinal fluid, tissue, or urine. We excluded patients under 14 years of age and patients with inconclusive evidence of infection, such as those with *C. auris* positive sputum or tracheal aspirate cultures, as these cultures were likely positive due to colonisation rather than true infection. We excluded asymptomatic patients with *C. auris* positive urine cultures as asymptomatic bacteriuria also more likely represented colonisation rather than infection. We extracted patient information from

the statuses, including patient demographics, comorbidities, human immunodeficiency virus (HIV) status, severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection status, length of hospital stay, intensive care unit (ICU) admissions, presence of an indwelling device, surgeries in the last three months prior to the positive culture, clinical signs and symptoms, culture results, presence of coinfection (with an organism other than *C. auris*), antifungal resistance of the *C. auris*, treatment, treatment duration, and mortality within three months after positive culture.

As outcomes, we analysed ICU admission, antifungal treatment duration, length of hospital stay, and death.

2.2. Ethics statement

The authors confirm that the ethical policies of the journal, as noted on the journal's author guidelines page, have been adhered to, and the appropriate ethical review committee approval has been received from the Unit of Biomedical Ethics, Research Ethics Committee at King Abdulaziz University.

2.3. Microbiological identification

The microbial species were identified by matrix-assisted laser desorption ionisation-time of flight mass spectrometry (MALDI-TOF; MS VITEK MS, bioMérieux, Marcy-l'Étoile, France) using VITEK MS v4.0 software. Antifungal susceptibility testing (AFST) was carried out according to the Clinical and Laboratory Standards Institute microdilution method using the Sensititre YeastOne panel (Thermo Scientific, Waltham, MA, USA); minimum inhibitory concentration (MIC) values were determined for azoles, echinocandins, and amphotericin B. Since no species-specific susceptibility breakpoints are currently available for *C. auris*, AFST results were interpreted according to the tentative breakpoints proposed by the US Centers for Disease Control and Prevention (Centers for Disease Control and Prevention 2022).

2.4. Statistical analysis

Data were checked for completeness and correctness. Categorical variables were presented as frequencies and percentages, and continuous variables were presented as means and standard deviations. Data were

checked for normality using the Shapiro-Wilk test and Kolmogorov-Smirnov test. The association between categorical variables was assessed with the Chi-square test. The Pearson correlation test assessed the correlation between age, length of hospital stay, and the Charlson weighted comorbidity index. The relationship between categorical variables such as gender and type of infection and numerical variables such as the Charlson weighted comorbidity index, length of hospital stay, and duration of antifungal therapy was established with the independent samples *t*-test. Data were entered and analysed in SPSS version 24 (IBM, Armonk, NY, USA) and were presented with 95% confidence intervals.

3. Results

3.1. Patients and clinical presentation

Based on the chart reviews, we identified 27 patients that were infected with *C. auris* in the period from 2015 to 2022. Their median age was 58, and eighteen of the patients were male (66.7%). Thirteen patients were tested for SARS-CoV-2, four (14.8%) were positive for SARS-CoV-2 and two of those had signs and symptoms of COVID-19, while nine patients were negative for SARS-CoV-2.

The most common source of infection was Central Line-associated Bloodstream Infection (CLABSI) in 17 (63.0%), and Catheter-Associated Urinary Tract Infection (CAUTI) in four (12%) (Table 1), followed by skin and soft tissue infections in two (7.4%), and intra-abdominal infections

in five (18%) patients. Of the five patients with intra-abdominal sources of infection, two had hepatobiliary infections, and one had a complicated urinary tract infection. The average length of hospital stay of the 27 patients was 78 days, and their average Charlson weighted comorbidity index was 4 (Table 1). Two patients (11%) with central line infections developed infective endocarditis.

Of the 27 patients, nine (33.3%) had a fever, and nine (33.3%) had a shock. All patients had indwelling devices (either a catheter or a central line), and 22 (81%) were admitted to the ICU. The most common comorbidities were diabetes mellitus and heart disease, as they were each present in 13 (48.1%) patients. The clinical features and comorbidities are presented in Table 2. Despite treatment, 18 (66.7%) patients died within 90 days of diagnosis.

3.2. Culture results and antimicrobial resistance

Multiple microbial specimens were identified in samples from nineteen patients (70.4%), whereas in samples from eight (29.6%) patients, only *C. auris* was identified. *C. auris* culture found 100% susceptibility to the antifungal agents voriconazole, caspofungin, and amphotericin. However, only three *C. auris* specimens were fully susceptible to fluconazole (11.1%), while 12 were intermediate (44.4%), and 12 were fully resistant (44.4%) (Table S1).

Table 1. Demographics and clinical data.

Nominal variables	Attributes	n	Percentage (%)
Gender	Male	18	66.7
Culture site	Blood	17	63.0
	Other	10	37.0
Source of infection	CAUTI	3	11.1
	CLABSI	17	63.0
	Other	7	25.9
Numerical variables			
	Mean (range)	Median	SD
Age, in years	58.07 (18–94)	58.00	17.57
Length of hospital stay (d)	83.19 (3–209)	78.00	58.80
Antifungal treatment duration (d)	23.04 (2–57)	18.00	15.66
Charlson weighted comorbidity index	4.56 (0–10)	4.00	2.22
White blood cell count (10 ³ /μL)	13.39	13.00	9.14
Platelet count (10 ³ /μL)	251.26	231.00	162.91
AST (U/L)	77.63	36.00	173.17
Bilirubin (μmol/L)	14.48	8.00	15.08
INR	1.41	1.20	1.01
Creatinine (μmol/L)	125.04	95.00	100.47

AST, aspartate aminotransferase; CLABSI, central line-associated bloodstream infection; CAUTI, catheter-associated urinary tract infections; INR, international normalised ratio; n, number; SD, standard deviation.

Table 2. Distribution of clinical features and comorbidities.

Variables	Present, n (%)
Fever	9 (33.3)
Shock	9 (33.3)
Hypothermia	0 (0.0)
Indwelling device	27 (100.0)
ICU admission	22 (81.5)
Diabetes mellitus	16 (59.3)
Hypertension	13 (48.1)
Renal impairment	11 (40.7)
Hemodialysis	5 (18.5)
Heart disease	13 (48.1)
Cerebrovascular disease	10 (37.0)
Chronic liver disease	10 (37.0)
Lung disease	2 (7.4)
Malignancy	6 (22.2)
Immunosuppressive therapy	10 (37.0)
Recent surgical history	11 (40.7)
HIV	2 (7.4)
COVID-19	2 (7.4)

HIV, human immunodeficiency virus; ICU, intensive care unit; n, number; COVID-19, coronavirus disease 2019.

3.3. Associations between variables and outcomes

Although the number of patients was small, we performed statistical analyses to identify potential associations between various variables and outcomes. Patients' sex and type of infection (poly- or monomicrobial) were not significantly associated with mortality or ICU admission ($P > 0.05$) (Table 3). Analysis of associations between outcomes showed a modest

positive correlation between length of hospital stay and antifungal treatment duration, $r = 0.549$, $P = 0.003$ (Table 4).

Analysis of an association between gender and the Charlson weighted comorbidity index, or the outcomes length of hospital stay and antifungal treatment duration, revealed no statistically significant results (all $P > 0.05$) (Table 5). However, when the relationship between the type of infection and the Charlson weighted comorbidity index, or the outcomes length of hospital stay, and duration of antifungal treatment duration were assessed, a statistically significant association was found between length of hospital stay and type of infection ($P = 0.043$, $t = -2.127$, 95% CI = -97.37 to -1.58) (Table 5).

4. Discussion

We studied the clinical characteristics and outcomes in patients with invasive *C. auris* infections at a single centre in Saudi Arabia. We found a wide range of clinical presentations, and the majority of infections were secondary to central line infection. The rest had urinary tract infections, intra-abdominal infections,

Table 3. Analyses of association between outcomes and sex or type of infection.

Analysis	Variables	Attributes	Alive, n (%)	Dead, n (%)	<i>P</i> value	Odds ratio (95% CI)
Analysis of association with mortality	Sex	Male	7 (38.9)	11 (61.1)	0.667	2.23 (0.36–13.96)
		Female	2 (22.2)	7 (77.8)		
	Type of infection	Monomicrobial	6 (31.6)	13 (68.4)	0.999	0.77 (0.14–4.33)
		Polymicrobial	3 (37.5)	5 (62.5)		
Analysis of association with ICU admission	Sex	Male	14 (77.8)	4 (22.2)	0.636	2.29 (0.22–24.14)
		Female	8 (88.9)	1 (11.1)		
	Type of infection	Monomicrobial	16 (84.2)	3 (15.8)	0.616	0.56 (0.08–4.24)
		Polymicrobial	6 (75.0)	2 (25.0)		

ICU, intensive care unit; n, number. Statistical analyses were performed with Chi-square tests.

Table 4. Correlation between continuous variables.

Variable		Length of hospital stay	Charlson weighted comorbidity index	Antifungal treatment duration
Age	Correlation coefficient	-0.197	-0.016	-0.409*
	<i>P</i> value (2-tailed)	0.325	0.937	0.034
Length of hospital stay	Correlation coefficient		-0.043	0.549**
	<i>P</i> value (2-tailed)		0.832	0.003
Charlson weighted comorbidity index	Correlation coefficient			0.015
	<i>P</i> value (2-tailed)			0.941

*, correlation is significant at the 0.05 level (2-tailed); **, correlation is significant at the 0.01 level (2-tailed). Correlations were assessed with Pearson correlation tests.

Table 5. Relationship between gender or infection type and Charlson index, length of hospital stay, and antifungal treatment duration.

Analysis Variable	t	95% confidence interval		P value
		Lower	Upper	
Analysis of association between gender and ...				
Charlson weighted comorbidity index	1.503	-0.49336	3.16003	0.145
Length of hospital stay	0.504	-37.88530	62.44086	0.619
Antifungal treatment duration	0.679	-8.91748	17.69526	0.503
Analysis of association between infection type and ...				
Charlson weighted comorbidity index	0.456	-1.52686	2.39529	0.652
Length of hospital stay	-2.127	-97.36789	-1.57948	0.043
Antifungal treatment duration	-1.027	-20.60815	6.50289	0.294

Statistical analyses were performed with independent samples *t*-tests.

and skin and soft tissue infections. A similar distribution of infection sources was observed in a study in Pakistan (Sayeed et al. 2019).

The mortality among our patients was 66.7% which was higher than the reported mortality rate of up to 42% for bloodstream infections with *Candida* in general (Brown et al. 2012). In studies of *C. auris*, the mortality rate was 35%–59%, and the majority of patients died within 30 days of acquiring the infection (Lockhart et al. 2017; Morales-Lopez et al. 2017; Sayeed et al. 2019). Our patients with candidemia had high mortality regardless of the appropriateness of therapy. All of our patients received antifungal therapy within 24–48 hours of having the culture results, while if needed, sometimes the therapy was started empirically before the culture results were available. They received antifungals for prolonged durations, but that did not change the outcome. This high mortality despite prolonged treatment was also observed in other studies (Todd 2017; Sayeed et al. 2019; Chowdhary and Sharma 2020). Due to the small sample size, it was not possible to detect statistically significant differences among risk factors, but we observed that most patients that died were of older age, and had heart disease, diabetes, malignancy, or used immunosuppressive therapy. This is also seen in other studies (Schelenz et al. 2016; Ruiz Gaitan et al. 2017; Chowdhary and Sharma 2020). A recent systematic review and meta-analysis of the global prevalence of COVID-19-associated *C. auris* infections reported that hypertension was the most prevalent comorbidity (59.4%) followed by diabetes mellitus (52.9%) and cardiovascular disease (31.4%). It also reported that men were three times more likely to be infected with *C. auris* than women, and that the prevalence of *C. auris* infections decreased during the COVID-19 pandemic (Vaseghi et al. 2022).

We also observed that the number of invasive *C. auris* cases increased in our institution year by year, but unfortunately, we did not collect data about the number of colonised versus invasive disease cases, so we cannot determine whether the burden of disease or its implications changed. The study points to the possibility that outbreaks have occurred over the last two years that have gone unnoticed and unstudied as the COVID-19 pandemic and its overwhelming burden on hospital resources required a focus elsewhere (Lockhart et al. 2017; Lowe et al. 2021). One of the findings in our study was that all cases of invasive *C. auris* disease were seen in our hospital in 2021 and 2022, although the study stretches from 2015 to 2022. So, no cases were found during the pre-COVID-19 era. For unknown reasons we only started seeing *C. auris* after the (start of the) COVID-19 pandemic. This may be due to an overall increase in the risk factors for *C. auris* infection, which in our patients we found to be prolonged hospitalisation, use of broad-spectrum antibiotics, and presence of indwelling devices.

Although *Candida* species in general are thought to be urinary tract colonisers rather than pathogens that cause urinary tract infections, we found several patients with symptomatic *C. auris* infections in the urinary tract. These had positive urine cultures as well as signs and symptoms of urinary tract infections such as pyelonephritis and ureteric stent infections. Similar cases have only been observed sporadically in cases (Centers for Disease Control and Prevention 2019; Griffith and Danziger 2020).

All of our patients had some sort of indwelling device, the majority of which were central lines. These probably contributed strongly to acquiring *C. auris* infections. High numbers of indwelling lines (88%) were also observed in a study in Pakistan (Sayeed et al. 2019).

We observed that all of the *C. auris* specimens cultured from the patients were 100% susceptible to amphotericin and echinocandins. Susceptibility for fluconazole was intermediate for 44% susceptible and complete for only 11%. This is promising news as previous studies from around the globe showed high resistance to echinocandins and amphotericin (Morales-Lopez et al. 2017; Eyre et al. 2018; Bing et al. 2022), but so far, we have not observed this yet.

The strength of our study is that it is a comprehensive overview of patients with invasive *C. auris* infections over the last seven years in Saudi Arabia, as so far only case reports and outbreak management reports have been available (Almaghrabi et al. 2020).

We found that despite the fact that the patients received appropriate therapy, the vast majority died. As treatment appears not to be effective enough, it should be emphasised that good infection control measures are needed to prevent these infections. Hand hygiene remains the most important key factor in breaking the chain of infection. Another prevention method we had not been practicing in our hospital at the time, but that was implemented recently, is placing patients in contact isolation once they have a positive *C. auris* culture. In addition, we should be screening patients to determine whether they are colonised with this particular strain to place them in isolation to avoid any possible outbreaks and provide all physicians with practical training in preventive measures. Given that all of our patients had indwelling devices, another measure would be to remove central lines or other unneeded devices soon after use to prevent infection with *C. auris* and other multidrug-resistant organisms.

Almost all the patients received antifungal monotherapy that was appropriate based on the *C. auris* susceptibility profiles as determined by culture. Source control was also not the issue as most of our patients had their indwelling devices removed. Nonetheless, 66.7% of the patients died. These results, as well as similar results observed in other studies, do make one wonder whether a combination of antifungals or multimodal therapy might be required. The best treatment for invasive *C. auris* disease is not yet established; prospective studies that compare therapies are needed to address this issue.

There are several limitations to our study. First and most important is that due to the small sample size, the factors responsible for mortality could not be

assessed by regression analysis. Therefore, we recommend that further studies involving multiple medical facilities are performed to investigate the factors responsible for deaths in *C. auris* infected patients. The second is that it is a retrospective study in nature which can have the potential for information bias. The third is that we did not have data on colonisation before developing invasive disease that would allow us to assess the incidence and prevalence of the disease in our hospital. We are currently not screening patients that are admitted to the hospital for colonisation due to limitations to our resources.

5. Conclusion

C. auris is an important fungal pathogen that causes a spectrum of invasive clinical diseases. The problem is that *C. auris* does not respond well to therapy and hence has a high mortality rate. Prevention remains an important tool to control the spread of this fungal infection and should consist of good infection control measures and removing indwelling devices as soon as they are no longer needed. Further studies are needed in this field to control the outbreaks and identify effective treatments for these patients.

Ethical statement

The authors confirm that the ethical policies of the journal have been adhered to and that ethical review committee approval has been received.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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References

- Abastabar M, Haghani I, Ahangarkani F, Rezai MS, Taghizadeh Armaki M, Roodgari S, Kiakojuri K, Al-Hatmi AMS, Meis JF, Badali H. 2019. *Candida auris* otomycosis in Iran and review of recent literature. *Mycoses*. 62(2):101–105. doi:10.1111/myc.12886.
- Almaghrabi RS, Albalawi R, Mutabagani M, Atienza E, Aljumaah S, Gade L, Forsberg K, Litvintseva A, Althawadi S. 2020. Molecular characterisation and clinical outcomes of

- Candida auris* infection: single-centre experience in Saudi Arabia. *Mycoses*. 63(5):452–460. doi:10.1111/myc.13065.
- Alshamrani MM, El-Saed A, Mohammed A, Algoribi MF, Al Johani SM, Cabanalan H, Balkhy HH. 2021. Management of *Candida auris* outbreak in a tertiary-care setting in Saudi Arabia. *Infect Control Hosp Epidemiol*. 42(2):149–155. doi:10.1017/ice.2020.414.
- Arendrup MC, Patterson TF. 2017. Multidrug-resistant *Candida*: epidemiology, molecular mechanisms, and treatment. *J Infect Dis*. 216(suppl_3):S445–S451. doi:10.1093/infdis/jix131.
- Bing J, Wang S, Xu H, Fan S, Du H, Nobile CJ, Huang G. 2022. A case of *Candida auris* candidemia in Xiamen, China, and a comparative analysis of clinical isolates in China. *Mycology*. 13(1):68–75. doi:10.1080/21501203.2021.1994479.
- Borman AM, Szekely A, Johnson EM, Mitchell AP. 2016. Comparative pathogenicity of United Kingdom isolates of the emerging pathogen *candida auris* and other key pathogenic *Candida* species. *mSphere*. 1(4):e00189–16. doi:10.1128/mSphere.00189-16.
- Brown GD, Denning DW, Gow NA, Levitz SM, Netea MG, White TC. 2012. Hidden killers: human fungal infections. *Sci Transl Med*. 4(165):165rv113. doi:10.1126/scitranslmed.3004404.
- Centers for Disease Control and Prevention. 2019. *Candida auris*: General information about *Candida auris*. [Atlanta (GA)]: CDC; [accessed 2023 Mar 24]. <https://www.cdc.gov/fungal/candida-auris/candida-auris-qanda.html>.
- Centers for Disease Control and Prevention. 2021. *Candida auris*: Information for laboratorians and health professionals. [Atlanta (GA)]: CDC; [accessed 2023 Mar 24]. <https://www.cdc.gov/fungal/candida-auris/health-professionals.html>.
- Centers for Disease Control and Prevention. 2022. *Candida auris*: Identification of *Candida auris*. [Atlanta (GA)]: CDC; [accessed 2023 Mar 24]. <https://www.cdc.gov/fungal/diseases/candidiasis/recommendations.html>.
- Chowdhary A, Sharma A. 2020. The lurking scourge of multidrug resistant *Candida auris* in times of COVID-19 pandemic. *J Glob Antimicrob Resist*. 22:175–176. doi:10.1016/j.jgar.2020.06.003.
- Clancy CJ, Nguyen MH. 2017. Emergence of *Candida auris*: an International Call to Arms. *Clin Infect Dis*. 64(2):141–143. doi:10.1093/cid/ciw696.
- Du H, Bing J, Hu T, Ennis CL, Nobile CJ, Huang G, Xue C. 2020. *Candida auris*: epidemiology, biology, antifungal resistance, and virulence. *PLoS Pathog*. 16(10):e1008921. doi:10.1371/journal.ppat.1008921.
- Eyre DW, Sheppard AE, Madder H, Moir I, Moroney R, Quan TP, Griffiths D, George S, Butcher L, Morgan M, et al. 2018. A *candida auris* outbreak and its control in an intensive care setting. *N Engl J Med*. 379(14):1322–1331. doi:10.1056/NEJMoa1714373
- Griffith N, Danziger L. 2020. *Candida auris* urinary tract infections and possible treatment. *Antibiotics (Basel)*. 9(12):898. doi:10.3390/antibiotics9120898.
- Hori Y, Shibuya K. 2018. Role of *FKS* gene in the susceptibility of pathogenic fungi to echinocandins. *Med Mycol J*. 59(2):E31–E40. doi:10.3314/mmj.18.004.
- Hu S, Zhu F, Jiang W, Wang Y, Quan Y, Zhang G, Gu F, Yang Y. 2021. Retrospective analysis of the clinical characteristics of *Candida auris* infection worldwide from 2009 to 2020. *Front Microbiol*. 12:658329.
- Jeffery-Smith A, Taori SK, Schelenz S, Jeffery K, Johnson EM, Borman A, *Candida Auris* Incident Management T, Manuel R, Brown CS. 2017. *Candida auris*: a review of the literature. *Clin Microbiol Rev*. 31(1):e00029–17.
- Lockhart SR, Etienne KA, Vallabhaneni S, Farooqi J, Chowdhary A, Govender NP, Colombo AL, Calvo B, Cuomo CA, Desjardins CA, et al. 2017. Simultaneous emergence of multidrug-resistant *Candida auris* on 3 continents confirmed by whole-genome sequencing and epidemiological analyses. *Clin Infect Dis*. 64(2):134–140.
- Lone SA, Ahmad A. 2019. *Candida auris*-the growing menace to global health. *Mycoses*. 62(8):620–637.
- Lowe H, Woodd S, Lange IL, Janjanin S, Barnett J, Graham W. 2021. Challenges and opportunities for infection prevention and control in hospitals in conflict-affected settings: a qualitative study. *Confl Health*. 15(1):94.
- Magnasco L, Mikulska M, Giacobbe DR, Taramasso L, Vena A, Dentone C, Dettori S, Tutino S, Labate L, Di Pilato V, et al. 2021. Spread of carbapenem-resistant gram-negatives and *Candida auris* during the COVID-19 pandemic in critically ill patients: one step back in antimicrobial stewardship? *Microorganisms*. 9(1):95.
- McCarthy MW, Walsh TJ. 2017. Containment strategies to address the expanding threat of multidrug-resistant *Candida auris*. *Expert Rev Anti Infect Ther*. 15(12):1095–1099.
- Mirhendi H, Charsizadeh A, Aboutalebian S, Mohammadpour M, Nikmanesh B, de Groot T, Meis JF, Badali H. 2022. South Asian (Clade I) *Candida auris* meningitis in a paediatric patient in Iran with a review of the literature. *Mycoses*. 65(2):134–139.
- Morales-Lopez SE, Parra-Giraldo CM, Ceballos-Garzon A, Martinez HP, Rodriguez GJ, Alvarez-Moreno CA, Rodriguez JY. 2017. Invasive infections with multidrug-resistant yeast *Candida auris*. Colombia. *Emerg Infect Dis*. 23(1):162–164.
- Prestel C, Anderson E, Forsberg K, Lyman M, de Perio MA, Kuhar D, Edwards K, Rivera M, Shugart A, Walters M, et al. 2021. *Candida auris* outbreak in a COVID-19 specialty care unit. *MMWR Morb Mortal Wkly Rep*. 70(2):56–57.
- Rossow J, Ostrowsky B, Adams E, Greenko J, McDonald R, Vallabhaneni S, Forsberg K, Perez S, Lucas T, Alroy KA, et al. 2021. Factors associated with *Candida auris* colonization and transmission in skilled nursing facilities with ventilator units. *Clin Infect Dis*. 72(11):e753–e760.
- Ruiz Gaitan AC, Moret A, Lopez Hontangas JL, Molina JM, Alexandre Lopez AI, Cabezas AH, Mollar Maseres J, Arcas RC, Gomez Ruiz MD, Chiveli MA, et al. 2017. Nosocomial fungemia by *Candida auris*: first four reported cases in continental Europe. *Rev Iberoam Micol*. 34(1):23–27.

- Sayeed MA, Farooqi J, Jabeen K, Awan S, Mahmood SF. 2019. Clinical spectrum and factors impacting outcome of *Candida auris*: a single center study from Pakistan. BMC Infect Dis. 19 (1):384.
- Schelenz S, Hagen F, Rhodes JL, Abdolrasouli A, Chowdhary A, Hall A, Ryan L, Shackleton J, Trimlett R, Meis JF, et al. 2016. First hospital outbreak of the globally emerging *Candida auris* in a European hospital. Antimicrob Resist Infect Control. 5:35.
- Todd B. 2017. Clinical alert: *candida auris*. Am J Nurs. 117(4):53–55.
- Vaseghi N, Sharifisooraki J, Khodadadi H, Nami S, Safari F, Ahangarkani F, Meis JF, Badali H, Morovati H. 2022. Global prevalence and subgroup analyses of coronavirus disease (COVID-19) associated *Candida auris* infections (CACa): a systematic review and meta-analysis. Mycoses. 65(7):683–703.
- Vinuela-Sandoval L, Falces-Romero I, Garcia-Rodriguez J, Eiros-Bouza JM. 2018. Candidemia and colonization by *Candida auris*, a diagnostic challenge. Enferm Infecc Microbiol Clin (Engl Ed). 36(4):253–255.