Annals of Parasitology 2025, 71, 157–169
doi: 10.17420/ap71.560

Review article

Medical case reports of *Candidozyma auris* (syn. *Candida auris*) infections in Europe – a systematic review

Jędrzej JANC[1], Natalia FELINIAK[1], Filip BIELEC[1], Katarzyna GÓRALSKA[2], Ewa BRZEZIAŃSKA-LASOTA[3], Dorota PASTUSZAK-LEWANDOSKA[1]

[1]Department of Microbiology and Laboratory Medical Immunology, Medical University of Lodz, Łódź, Poland
[2]Department of Biology, Parasitology and Medical Mycology, Medical University of Lodz, Łódź, Poland
[3]Department of Biomedicine and Genetics, Medical University of Lodz, Łódź, Poland

ABSTRACT

Candidozyma auris (syn. *Candida auris*) is a hazardous multi-drug resistant yeast that causes severe infections in hospitalized patients. Many uncertainties exist around its antifungal resistance, clinical presentation, dominant clade, and isolation sites in the European setting. Therefore, to properly assess these characteristics, we systematically reviewed case reports in Europe between the first case described in 2009 and November 2024.

We conducted this systematic review according to the PRISMA guidelines. Cases of both symptomatic and asymptomatic patients have been included. We extracted patients' demographics, *C. auris* isolation site, identified clade, clinical presentation, clinical outcome, as well as treatment. The case reports have been assessed for quality using standardized tools.

Out of 251 identified articles, 12 reports from 10 European countries describing 15 cases are included In the review. The youngest affected person was an infant and the oldest was 74 years old. Blood was the most common isolation site, reported in 26% of reported cases. The fatality rate could not have been reported due to the high number of asymptomatic patients and comorbidities or other infections in symptomatic patients. About 91% of reported isolates among the analyzed cases were resistant to fluconazole, 18% to amphotericin B and none was resistant to echinocandins. The quality assessment revealed that about 33% of case reports had a high risk of bias.

Our results show that any age group can be affected. The antimicrobials of choice in the European setting seem to be the echinocandins, although proper standardized susceptibility breakpoints are needed.

Keywords: *Candida*, *Candidozyma auri*s, systematic review, case study

Introduction

Candidozyma auris (Satoh & Makimura) Wang, Yurkov, Boekhout & Bai, 2024 – a fungus from the phylum Ascomycota was first classified as *Candida auris* Satoh & Makimura 2009 [1]. *C. auris* is an opportunistic pathogen that is a relatively new global threat, isolated for the first time at a hospital in Japan in 2009 from an ear (*auris* is Latin for "ear") of a female patient [2]. Initially it was classified in the *Candida* genus, with which it has a high degree of similarity [2]. The current taxonomic position was proposed by Liu et al. only in 2024, who assigned *C. auris* to the genus *Candidozyma* in the family Metschnikowiaceae [1]. Although it is possible that the pathogen appeared earlier, in Korea in 1996, but it was misidentified as *Candida haemulonii* (currently *Candidozyma haemuli*) and only later retrospectively correctly described [3, 1]. Currently, *C. auris* has been isolated in at least 40 countries across 6 continents, with the United States of America being the country with the highest number of reported cases. What seems remarkable is that *C. auris* has probably independently emerged in different locations across the globe. Whole genome sequencing has revealed 5 different clades, which have likely originated in South Asia (clade I), East Asia (clade II), South Africa (clade III), South America (clade IV), and one clade from Iran (clade V) [4]. Unfortunately, the true prevalence of this

specific yeast over the world remains partly unknown, as species identification can be challenging, especially in low-income countries [5]. Several outbreaks have been reported around the globe [6, 7], including the recent countrywide outbreak in Israel [8], proving that *C. auris* is still a potent epidemiological threat. In 2022 World Health Organization (WHO) has published the Fungal Priority Pathogens List, which classifies *C. auris* to the Critical group [9].

Risk factors for *C. auris* infection are not much different from infections caused by *Candida* spp. in general. These include immunosuppressed state, significant medical comorbidities, central venous catheters, urinary catheters, recent surgery, parenteral nutrition, exposure to broad-spectrum antimicrobials, diabetes mellitus, malignancies, intensive care unit admission, and specialized care residency [10].

C. auris appears to be well adapted to environmental challenges, it is highly resistant to higher temperatures in comparison to its close phylogenetic relatives. So much so that its ability to grow well even at 42°C is used to distinguish *C. auris* from other members of the closely related *C. haemulonii* complex [11]. This unusual thermal stability makes it particularly resilient to fever. One hypothesis states that this mechanism might have evolved as a response to the rising global temperatures caused by increased concentration of CO_2 in the atmosphere (a phenomenon known as global warming). The thermal tolerance of *C. auris* promoted its thriving in wetland ecosystem. The spread could have been then facilitated by animals, e.g. birds, to rural areas [12]. While the overuse of fungicides in agriculture might have exposed the environmental fungi to compounds similar to clinically used antifungals, which may explain the multi drug resistance of *C. auris* [13].

C. auris has an exceptional ability to survive starvation. It is able to remain viable for at least 14 days on a plastic healthcare surface, as measured by colony forming units (CFU) per unit area [14]. This species is able to form biofilms on synthetic sweat designed to mimic human skin conditions, similarly to other Candida species, which in practice, might made it less sensitive to disinfectants, such as chlorhexidine, povidone-iodine, hydrogen peroxide or sodium hypochlorite. However, *in vitro*, *C. auris* seems to be particularly susceptible to the anaerobic environment which might make it less likely to colonize the gastrointestinal tract, which would explain its predominant colonization of the skin, making it easily transmissible between patients [15]. In an invertebrate systemic infection model *C. auris* displayed a similar level of virulence to clinically common Candida albicans , despite the fact that *C. auris* formed neither hyphal nor pseudohyphal formations [16]. It displays multiple virulence traits with the most crucial being the production of extracellular hydrolase, e.g. secreted aspartyl proteinases (SAPS), hemolysins, lipases, and phospholipases which are involved in host tissue degradation and pathogen propagation [17].

C. auris shows a high level of resistance, including primary resistance, to all major antifungal drug classes, with a high proportion of isolates being multi-drug resistant (MDR). Multiple studies have reported isolates resistant to fluconazole, the most commonly prescribed antifungal agent. However, there are also reports of resistance to amphotericin B, and occasionally to echinocandins [18, 19]. Drug resistance to two or more antifungal drug classes is observed in ≥ 40% of isolates and resistance to all classes in approximately 4% of isolates [20]. At the same time, pan-resistant isolates are also observed among *C. auris* [9]. Additionally, *C. auris* has the ability to quickly develop resistance and because of that, it is crucial to identify susceptibility and use proper antifungal for the right number of days and in the correct dosage [21].

The resistance to environmental stresses combined with the low susceptibility to antifungal drugs helps to understand the high bloodstream infection-associated mortality rates ranging from around 28% to 66% [22]. While in cases of invasive *C. auris* candidiasis, the mortality rate is estimated at 29-53% [9]. The key characteristics of *C. auris* could probably explain *C. auris* ability to thrive in a nosocomial setting and cause outbreaks (Table 1) [23].

The correct recognition and treatment of *C. auris* infection have been particularly important during the Coronavirus disease 2019 (COVID-19) pandemic, because one of the complications of critically ill COVID-19 patients is invasive fungal infection, including infection by *C. auris*, especially after prior anti-fungal treatment. COVID-19-related *C. auris* outbreaks have been associated with even higher fatality rates, ranging from 30% to 89% [24].

The proper understanding of this pathogen is of utmost importance especially during patients' treatment. Unfortunately, there are still many uncertainties surrounding *C. auris* in Europe, therefore we conducted a systematic review of case

Table 1. Key characteristics of *Candidozyma auris*

No.	Key characteristics of *C. auris*	References
1	Ability to survive in high temperatures exceeding 40°C	[11]
2	Ability to thrive in nosocomial setting and cause outbreaks	[14, 15], [23]
3	Predominant colonization of the skin	[15]
4	Susceptible to an anaerobic environment	[15]
5	Multidrug resistance in many isolates, especially to fluconazole	[18, 19]
6	The rapid development of resistance while treating the patient	[21]
7	High bloodstream infection-associated mortality rates (28–66%)	[22]

reports to adequately assess the isolation site, dominant clade, patients' demographics, clinical presentation, outcome, and drug resistance with a strong emphasis on MIC of antifungals in the context of *C. auris* isolated from the European patients. This information might then be used to improve clinical outcomes.

Methods

Criteria for Considering Studies for Review

This systematic review follows the preferred reporting items for systematic reviews and meta−analyses (PRISMA) 2020 guidelines [25]. Only case reports were included in this review, irrespective of their language. The case reports were considered for inclusion if they presented *C. auris* isolation identified in Europe between the first case in 2009 and November 2024.

Search Methods for Identification of Studies

The search was conducted up to November 2024 in the following databases with no restriction on publication year, or publication status:

- PubMed (https://pubmed.ncbi.nlm.nih.gov/), (Access on 24 November 2024),
- Scopus (https://www.scopus.com/ (Access on 24 November 2024),
- Web of Science (https://www.webofscience.com/wos/woscc/basic-search), (Access on 24 November 2024).

The following search strategy was used for the databases searches: "*Candida auris* AND case report". We used "*Candida*" as a keyword because the species was classified as *Candidozyma* only in 2024 [1], so earlier case reports included the previous genus name.

There was no restriction on the language. If studies in languages other than English or Polish had been found, the authors sought an initial translation of the abstract to apply the inclusion criteria.

The authors also scanned the references of all relevant articles.

The titles and abstracts identified through the search process were reviewed by the authors. Following this, full texts of the selected articles were retrieved and assessed for eligibility.

Currently, there are no specific Minimal inhibitory concentration (MIC) breakpoints that are established for *C. auris*, so the interpretation was conducted using non-ideal Centers for Disease Control and Prevention (CDC) tentative breakpoints [26].

The quality of the studies has been assessed by authors using The Joanna Briggs Institute (JBI) Critical Appraisal tools for case reports which consist of eight yes/no/unclear/not applicable questions [27]. To summarize the overall risk of bias of case reports, they have been grouped into the following categories:

1. Low risk of bias (studies that met at least 75% of the quality criteria),
2. Moderate risk of bias (studies that met at least 50% and less than 75% of the quality criteria),
3. High risk of bias (studies that met less than 50% of the quality criteria).

Results

The search results are summarized in the diagram in Figure 1. Out of 570 articles found in the selected databases, 319 articles were excluded due to being a duplicate. Among the 251 publications identified, 12 were potentially appropriate for this systematic review. 237 were excluded due to not presenting a case from a European country or not being a case report, and 2 due to not being a peer-reviewed article. The characteristics of the studies included in this systematic review are summarized in Table 2.

The final analysis included 12 publications yielding data on *C. auris* infections in Europe, taken from 15 reported cases of *C. auris* isolation in 10 European countries: Italy (n=3), Germany (n=2), The Netherlands (n=2), Denmark (n=2), Poland (n=1), Austria (n=1), Spain (n=1), Switzerland (n=1), Portugal (n=1) and Greece (n=1) (Figure 2). Our results showed that any age group could be

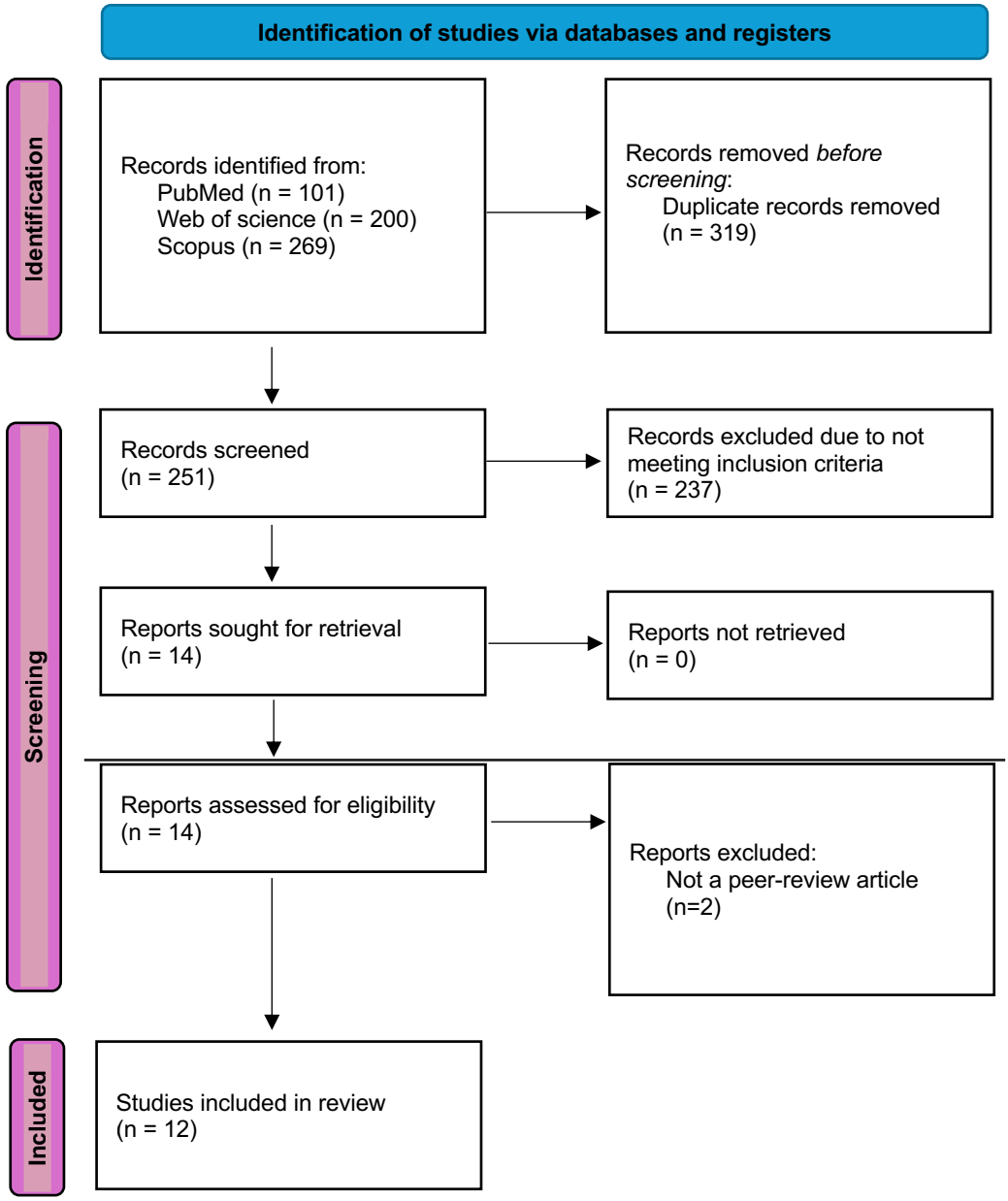

Figure 1. PRISMA Flow Diagram acc. Page et al. (2021).

infected [28, 29]. Around 85% (n=11) of adult patients were male and 15% (n=2) were female. One case report of 2 patients did not include their genders (Table 2) [30].

Out of 15 reported patients 5 have died, which would imply 33% case fatality, although it cannot be reliably assessed from these case reports due to a high proportion of patients being asymptomatic, lack of proper follow-up in some cases, as well as a high number of comorbidities and other infections in the symptomatic patients that might manifest in a similar way to the *C. auris* infection (Table 2).

Matrix-assisted laser desorption/ionization time-of-flight (MALDI TOF) mass spectrometry, polymerase chain reaction, and sequencing were the methods of choice for the identification of *C. auris*. 7 case reports properly determined the clade of the isolates and most of them belonged to the South Asian (I) clade [31-35, 38] and one belonged to South African (III) Clade [39].

During an active symptomatic infection, *C. auris* was most frequently isolated from the blood (n = 4). However, isolation from the sputum (n = 1), urine (n = 2), wound (n = 1), bronchoalveolar lavage (n = 1)

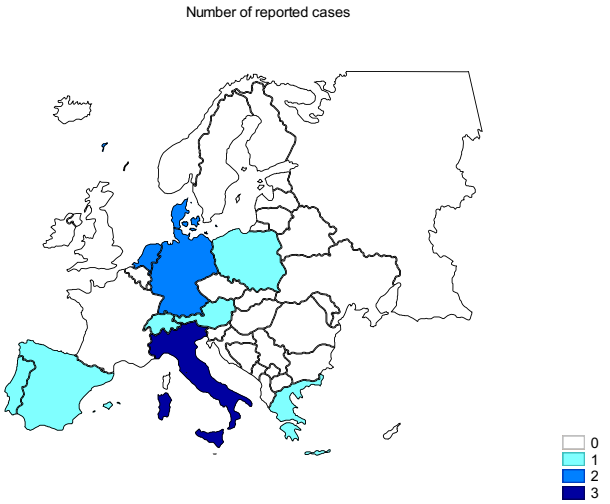

Figure 2. Location of *C. auris* cases in Europe

and ear (n = 1) was also reported. Colonization was detected on various body sites, including the nares, groin, axilla and rectum (Table 2). The clinical symptoms of *C. auris* infection were usually nonspecific and hard to distinguish from other infections, with fever being the most commonly described. The way the patient had become infected was undetermined in most cases. However, one case presented a vertical colonization of an infant by the mother, confirmed by a positive vaginal swab and colonization in the infant's axilla, skin, eyes, and ears, although the child did not present any symptoms of infection [28].

The antifungal susceptibility showed that around 91% (n=10) of *C. auris* isolates were resistant to fluconazole, 18% (n=2) to amphotericin B and none showed resistance to micafungin, anidulafungin, and caspofungin (Table 3). Isolate from the first case presented by Theut, M. et al. [30] showed susceptibility to echinocandins and amphotericin B, but resistance to fluconazole. And the isolate from the second case by Theut, M. et al. [30] showed decreased sensitivity to fluconazole and anidulafungin, but susceptibility to amphotericin B. Two case reports that together described 3 patients did not include the minimal inhibitory concentrations (MIC) of antimycotics to the isolates. MIC of tested antimycotics was reported in 10 out of 12 studies in 11 isolates and is presented with The Centers for Disease Control and Prevention (CDC) tentative breakpoints in Table 3 [26]. In reviewed studies the most often used antifungals were echinocandins, with five case reports including them in their treatment protocols [31, 35, 36, 38, 39], which resulted in the recovery of two out of five patients. Two reports used amphotericin B [30, 36], one study used an oral suspension of nystatin [37] and one used oral posaconazole [32]. One of the patients described by Reque, J. et al. [36] had improved after administration of intravenous amphotericin B 100 mg daily for three weeks and two weeks of intravenous anidulafungin 100 mg daily. Unfortunately, most of the included studies neither described adverse events of their treatment nor stated the lack of them. Only one study reported liver toxicity after starting caspofungin [36].

The quality of the presented studies, with the overall risk of bias, have been presented in Table 4. None of the included twelve case reports met all eight questions, however seven have managed to satisfy them sufficiently to be considered low risk of bias. One were considered moderate risk of bias. While four cases fulfilled less than half of the criteria and were considered as high risk of bias.

Discussion

We performed a systematic review of case reports of the incidence of *C. auris* in Europe between the first case and November 2024. *C. auris* is a hazardous MDR yeast that is an emerging global health problem. Our results show that any age group can be affected as previously reported in the literature [40]. However, preterm infants as well as geriatrics are known to be highly at−risk patients due to their weaker immune systems, resulting in high risk of death upon being infected with *C. auris* [41].

During the period between 2013 and 2021, 1812 instances of *C. auris* were reported in the EU (the UK is not included). The countries that reported the highest number of cases in that period were Spain (1,377) and Italy (292). Currently, Spain is the only European Union (EU) country where there is a reported regional endemicity [42]. Surveillance datasets provide aggregated epidemiological information (e.g., incidence, geographic distribution, and outbreak trends), but they typically lack nuance in clinical, microbiological and outcome-level details, so although the number of epidemiological cases in the European surveillance systems is higher than in our review, we intentionally focused exclusively on published case reports in peer-reviewed journals to better answer our research questions. Our review found the highest number of case reports from Italy, Germany, the Netherlands and Denmark. Only one case report

Table 2. Characteristics of reports included in the systematic review

No.	Reference	Age [years]	Sex	Country	Isolation site	Presentation	Clinical outcome	Coinfection	Colonization by other pathogens	Antifungal treatment	Clade
1	[28]	Preterm infant	Female	Italy	Vagina, axilla, skin, eyes and ears	Asymptomatic	Death	None	nd	nd	nd
2	[29]	74	Female	Switzerland	Tracheal aspirates	Probably asymptomatic	Death	Septic shock of unknown origin	nd	nd	nd
3	[30]	Case 1: 65. Case 2: 64	nd	Denmark	Case 1: drainage fluid and urine. Case 2: tracheal secretions, urine, as well as an inoculation from the axilla/groin and nose	Case 1: Asymptomatic. Case 2: Septic	Case 1: nd. Case 2: nd	Case 1: nd. Case 2: None	Case 1: nd. Case 2: None	Case 1: None. Case 2: Liposomal amphotericin B (dosage and duration of treatment were not reported)	nd
4	[31]	Mid-70s	Male	Italy	Blood	Fever, respiratory symptoms	Stable condition	None	nd	*Caspofungin* until 14 days after the first negative blood cultures	South Asian
5	[32]	20s	Male	Greece	Sputum	Cough and sputum production	Recovery	*Aspergillus fumigatus*, *A. terreus*, *Pseudomonas aeruginosa*, *Alcaligenes denitrificans*	nd	300 mg oral posaconazole (three 100 mg delayed release tablets), twice daily on the first day and once daily afterward	South Asian (I)
6	[33]	Case 1: middle-aged. Case 2: middle-aged	Case 1: Male. Case 2: Male	Case1: Germany. Case 2: Germany	Case 1: Wound, urine. Case 2: Urine	Case 1: nd. Case 2: nd	Case 1: Recovery. Case 2: Death	CMV pneumonia and recurrent septic episodes with *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*	*Klebsiella* pneumoniae with extended spectrum beta-lactamase phenotype and resistance to quinolones, vancomycin-resistant *Enterococcus faecium*, carbapenem-resistant *Pseudomonas aeruginosa*, *Candida albicans*	Case 1: None. Case 2: nd	South Asian (I)
7	[34]	Case 1: middle-aged. Case 2: middle-aged	Case 1: Male. Case 2: Male	Case 1: The Netherlands. Case 2: The Netherlands	Case 1: Central Venous Catheter Groin. Case 2: Urine	Case 1: Asymptomatic. Case 2: low-grade fever	Case 1: Recovery. Case 2: nd	None	Case 1: Multi drug resistant *Enterobacterales*, producing OXA 48 and NDM. Case 2: OXA and NDM-positive *Escherichia coli* and NDM positive *Pseudomonas aeruginosa*	Case 1: None. Case 2: None	South Asian (I)
8	[35]	18	Male	Poland	Blood, necrotic chronic wounds, and stumps	Impossible to assess	nd	meningococcal septicemia	nd	Micafungin 100 mg per day	South Asian (I)
9	[36]	57	Male	Spain	Blood	Fever	Recovery	Impossible to assess	None	Three weeks of intravenous amphotericin B 100 mg every day and two weeks of intravenous anidulafungin 100 mg every day	nd
10	[37]	22	Male	Austria	Ear	Otitis externa	Full recovery	None	None	Oral suspension of nystatin twice weekly for 3 weeks	nd
11	[38]	80	Male	Italy	Blood	Respiratory failure	Death	NDM-producing *Klebsiella pneumoniae*	nd	--caspofungin treatment	South Asian (I)
12	[39]	54	Male	Portugal	Bronchoalveolar lavage	Pneumoniae	Death	*Acinetobacter baumanii* MDR	Colonization on multiple sites (skin, rectum) by *Klebsiella pneumoniae* NDM and *Acinetobacter baumannni* carbapenemase-resistant	colistin for 16 days and caspofungin for 10 days	South African (III)

nd – not determin

Table 3. Susceptibility of analyzed *C. auris* according to CDC-suggested tentative MIC breakpoints in mg/L.

Study	AMB	FZ	FC	VRC	POS	ISA	ITA	MCF	ANF	CAF
CDC tentative	≥2	≥32	nd	nd	nd	nd	nd	≥4	≥4	≥2
Crea et al, 2019	2, R	>256, R	0.5	4	0.25	nd	0.5	0.12, S	0.25, S	0.12, S
Mesini et al, 2021	1, S	>256, R	nd	2	0.12	nd	2	0.12, S	0.25, S	0.12, S
Riat et al. 2018	1, S	256, R	nd	4	Nd	nd	nd	0.06, S	0.12, S	0.06, S
Stathi et al. 2019	0.25, S	>128, R	0.06	>8	>8	8	>4	0.01, S	0.03, S	0.12, S
Steinmann et al.	1, S	64, R	nd	1	≤0.016	≤0.016	nd	nd	0.25, S	nd
Steinmann et al.	2, R	>64, R	nd	16	0.25	8	nd	2, S	2, S	nd
Vogelzang et al.,	0.5–1, S	>64, R	nd	4	nd	Nd	nd	0.063, S	<0.016–0.063, S	nd
Pekard-Amenits	0.5–1, S	0.25–0.5, S	≤0.064	0.008–0.016	≤0.008–0.032	0.002	≤0.03	0.064–0.125, S	0.012–0.125, S	0.032–0.125, S
Prazynska et al.,	1, S	>256, R	nd	0.25	nd	nd	nd	0.064, S	0.047, S	0.25, S
Rimoldi et al., 2024	0.5, S	>32, R	32	0.06	≥8	nd	>4	0.06, S	0.03, S	0.125, S
Henriques et al.,	0.25, S	>128, R	<0.0625	2	0.25	nd	>4	0.0625, S	0.25, S	0.25, S

Abbreviations: AMB – amphotericin B; FZ – fluconazole; FC – flucytosine; VRC – voriconazole POS – posaconazole; ISA – isavuconazole; ITA – itraconazole; MCF – micafungin; ANF – anidulafungin; CAF – caspofungin; nd – not determined; S – susceptible; R – resistant.

each has been published from Poland, Spain, Austria, Portugal and Greece. The number of reported cases almost doubled between 2020 (335 cases reported by eight countries) and 2021 (655 cases reported by 13 countries), which was markedly more than in previous years. 1758 of these cases (97%) could not have been classified as imported or locally acquired. Forty-four (2.4%) cases were reported as imported and 10 (0.6%) as locally acquired. Unfortunately, the limited data available precluded any thorough examination of the origin of imported cases. For the few cases that did include the information, countries in Africa, Asia and Middle East were mentioned [43]. Our results showed that the most common clade in Europe is South Asian (I) clade, which might imply that South Asia may be the place of origin.

*C. auri*s appears to stand out in its capacity to spread between patients and cause outbreaks of disease in healthcare facilities, and several molecular studies confirm intra- or interhospital transmission. *C. auris* can be transmitted by contact with infected individuals or surfaces that have been contaminated [44, 45]. The study from Denmark showed one possible indirect case of *C. auris* transmission in a hospital setting, in a patient who stayed in a room in which the previous occupant showed positive *C. auris* colonization. Even though the room had been cleaned. The patient that contracted the infection developed sepsis with positive *C. auris* blood cultures about one month later, after being treated with echinocandins for Nakaseomyces glabratus (syn. *Candida glabrata*) esophagitis [30]. While the study in Italy proved

Table 4. JBI Critical Appraisal Checklist for Case reports with overall risk of bias

Author	Were the patient's demographic characteristics clearly described?	Was the patient's history clearly described and presented as a timeline?	Was the current clinical condition of the patient on presentation clearly described?	Were diagnostic tests or assessment methods and the results clearly described?	Was the intervention(s) or treatment procedure(s) clearly described?	Was the post-intervention clinical condition clearly described?	Were adverse events (harms) or unanticipated events identified and described?	Does the case report provide takeaway lessons?	The overall risk of bias
Crea et al., 2019	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Low
Mesini et al, 2021	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Low
Reque et al., 2022	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Low
Riat et al. 2018	No	No	No	Yes	No	Yes	No	Yes	High
Stathi et al. 2019	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Low
Steinmann et al. 2021	No	Unclear	No	Yes	Yes	No	No	Yes	High
Theut et al. 2022	No	No	No	Yes	No	No	No	Yes	High
Vogelzang et al., 2019	No	No	Yes	Yes	Not applicable	Not applicable	Not applicable	Yes	Moderate
Pekard-Amenitsch et al., 2018	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Low
Prazynska et al., 2022	No	Yes	No	Yes	No	No	No	Yes	High
Rimoldi et al., 2024	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Low
Henriques et al., 2023	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Low

that vertical transmission from mother to child is possible, it is the way that is rarely mentioned in the literature [28]. To correctly counteract the spreading of *C. auris* in a hospital setting, environmental cleaning should be performed regularly, preferably 3-5 times a week using ideally sodium hypochlorite or hospital-grade sporicidal disinfectants. In patients with a high risk of candidemia, additional skin and mucosal decontamination with the use of chlorhexidine should be considered [46].

Our results show that during active infection *C. auris* is most commonly isolated from the blood. During the outbreak in the United Kingdom, which affected 50 people, 22 patients required antifungal treatment and 9 out of those patients had confirmed candidemia (which accounts for a total of 18% of reported cases) which was the most common isolation site among symptomatic patients. It seems to align with our results [47]. Studies have shown that the mortality due to the Candida infection in the bloodstream is around 30–40%, even for patients treated with antifungal drugs [48]. Although our results showed the similar rate of 33%, it was limited by the lack of proper follow-up in some of the reviewed studies.

C. auris is reportedly difficult to identify and is often mistaken for some Candida species, which is a major problem with infection management [41]. Matrix-assisted laser desorption/ionization time-of-flight (MALDI TOF) mass spectrometry and polymerase chain reaction were the methods of choice for the identification of *C. auris* in reviewed

case reports, which is in line with the current diagnostic standards and should be used in clinical practice with the potential to more in-depth identification down to lineages within the species [23, 49]. Proper identification is important as *C. auris* is known for a high level of antimicrobial resistance and requires adequate treatment [18, 19]. This means that clinical decisions should be based on microbial identification and antimicrobial susceptibility tests [50].

In most cases, the clinical presentation of *C. auris* infection is non-specific, and it is often difficult to differentiate between other types of systemic infections [51], which is in line with our results. The clinical presentation of *C. auris* infected patients is similar around the world [52]. Given its high fatality, antifungal resistance [22], and the fact that early initiation of antifungal therapy of invasive candidiasis reduces mortality [53], screening should be considered in a healthcare setting. Especially taking into account that *C. auris* supposedly has the ability to cause low-grade disease years after colonization [14]. Heath et al. described a case of osteomyelitis of the sternum caused by *C. auris* in a patient who was colonized by *C. auris* 3 years prior to clinical disease manifestation [54].

According to The European Centre for Disease Prevention and Control (ECDC), the detection of a case of *C. auris* should trigger an investigation including a detailed case review and screening of close contact patients for *C. auris* carriage. Screening for *C. auris* colonization is achieved by performing a composite swab of the patient's bilateral axilla and groin. Other sites (urine, wounds, catheter exit sites, throat, etc.) can be sampled, if clinically relevant or indicated. Effective and quick response with epidemiological investigation, complemented by a cross-sectional screening of patients for *C. auris* carriage, is useful to establish the source of the outbreak and thus prevents further cases. Although environmental sampling or screening of healthcare workers are not routinely recommended [44]. CDC has released a similar recommendation, with the addition of screening patients colonized with carbapenemase-producing Gram-negative bacteria. As *C. auris* co-colonization with these organisms has been observed regularly [55]. Unfortunately, the lack of a rapid, point-of-care test makes such screening difficult [56]. All confirmed identifications of *C. auris* should be reported to local or national public health authorities, and infection control practices to prevent transmission should be implemented at facilities where the patients reside [38]. We also suggest screening of infants born from mothers colonized with *C. auris* as a case of vertical transmission has been observed [28].

Currently, *C. auris* is known for its resistance to standard antifungal drugs [18, 19]. Our results showed that azoles are an ineffective therapeutic strategy, with even 91% of isolates being resistant to fluconazole. Similarly, 18% of isolates were resistant to amphotericin B. Those results are based on a small sample size (n=10) and should not be used for clinical judgment. Resistance to fluconazole and amphotericin B appear to be more prevalent in our results than previously reported values of around 45% and 15% of isolates being resistant to fluconazole and amphotericin B, respectively [41, 42, 57]. It may imply higher resistance to these two drugs in Europe than in other regions, although our results do not differ from WHO estimates (WHO priority list) [9]. Although in general, when dealing with candidiasis, such choice should be based on local epidemiology and drug-drug interactions in the individual patient [50]. High azole resistance in Europe may be explained by the rise of triazole fungicide usage in agriculture, particularly in the western Europe which accounts for 37% of its world consumption; this mechanism was hypothesized to be the reason for azole resistance in *Aspergillus fumigatus* Fresen., 1863 and could also be applicable here, as the basis for further research. These triazole fungicides (for example, difenoconazole, epoxiconazole, propiconazole and tebuconazole) are structurally similar to clinically used first-line medical triazoles (isavuconazole, itraconazole, posaconazole and voriconazole), but are characterized by long degradation half-life, which allows them to last longer in the environment [13]. The mechanisms of resistance are still being studied, although *C. auris* demonstrated high ATP-binding cassette (ABC) and major facilitator superfamily (MFS) efflux pump activity, which together with mutations in the ERG11 gene mutations explain the high azole resistance. Whereas mutations in the FKS1 gene cause echinocandin resistance and single nucleotide polymorphism (SNP) at various genomic loci are associated with resistance to polyenes [17].

For identification of susceptibility CDC tentative breakpoints were used as neither the European Committee on Antimicrobial Susceptibility Testing (EUCAST) nor the Clinical and Laboratory Standards Institute (CLSI) has released antimicrobial's MIC in the context of *C. auris* [56].

Our results revealed that in most of the isolates MIC of fluconazole was much higher and MIC of amphotericin B was slightly lower than CDC tentative breakpoint, and every isolate had much lower MICs of micafungin, anidulafungin and caspofungin which matched observations from outbreak that happened in Italy [59].

Consequently, the treatment options for *C. auris* infections remain limited and empirical treatment with an echinocandin drug is recommended until availability of susceptibility testing results as stated by ECDC [44, 60]. The CDC has issued recommendations for the treatment of *C. auris* infections in adults and children over 2 months of age, including the echinocandins: anidulafungin, caspofungin and micafungin [61]. According to WHO echinocandins are the most common antifungals used against *C. auris* [9]. The novel drug ibrexafungerp (formerly SCY-078) is the first compound of the enfumafungin-derived triterpenoid class of (1→3)-β-D-glucan synthase inhibitors (GSIs) that has showed activity against *C. auris* in early clinical studies in humans, providing hope for a possible new drug in the arsenal against this yeast [62].

The quality assessment of included case reports was performed using the Joanna Briggs Institute Critical Appraisal Checklist for Case Reports and none of the included articles met all 8 criteria. The most often overlooked part was the adverse events of the treatment used. Only one study reported liver toxicity after starting therapy with caspofungin [36]. Unanticipated events, if any that may yield new or useful information, should be identified and clearly described [27, 63]. In some of the included reports, antifungal therapy was administered to patients in critical conditions (e.g. septic shock or multiorgan failure), in which clinical and laboratory abnormalities are difficult to distinguish from drug-related adverse events. Consequently, possible cases of toxicity were frequently not reported, leading to a high risk of bias rating according to JBI criteria. This finding likely points to a limitation inherent to case report methodology in critically ill populations rather than selective underreporting.

Conclusion

The results of this systematic review suggest that *C. auris* can infect individuals of all age groups and can spread indirectly in hospital settings, as well as directly from person to person, including vertically from mother to child. In the case of newborn babies born by colonized or infected mothers, screening for colonization might be considered. The symptoms of *C. auris* infection are non-specific and similar to other infections. Clinical decisions should be based on microbial identification and antimicrobial susceptibility tests. European isolates have demonstrated high resistance to fluconazole, meaning that using it as empirical treatment or prophylaxis is not recommended in patients with a high risk of or recognized *C. auris* infection. Amphotericin B may also be ineffective, while none of the examined isolates showed resistance to echinocandins; ergo, they might be the best option for the treatment and prophylaxis. Currently, only the CDC has set tentative breakpoints, which may not be accurate. In our opinion, it is justified to develop novel MIC breakpoints of drugs against *C. auris*. The clinical presentations and case descriptions lacked information about adverse events of the treatment used. There is still much uncertainty surrounding *C. auris* and further research is needed to properly understand its emergence, biology, spread and resistance.

Limitations of the study

Our systematic review has several strengths; we conducted extensive literature search, did not impose restrictions based on language or time of publication, and assessed the reported cases according to predefined criteria. However, there are also several limitations. Firstly, some of the assessed papers did not report a proper follow-up, which made it difficult to properly estimate the case fatality rate. Secondly, due to the low number of published case reports in Europe, the review focused mainly on adults, with only one exception of a study describing *C. auris* isolation from an infant. The presentation and treatment of *C. auris* in the pediatric population is still poorly understood as a result of an insufficient number of articles published about the mentioned age group [64]. Finally, we did not incorporate patients from reports that were not presented as peer-review papers.

Conflict of interest

The authors declare that there are no conflicts of interest.

Acknowledgment

Alicja Walczak provided editorial assistance in the finalization and submission of this manuscript.

References

[1] Liu F., Hu Z.D., Zhao X.M., Zhao W.N., Feng Z.X., Yurkov A. et al. 2024. Phylogenomic analysis of the *Candida auris–Candida haemuli* clade and related taxa in the Metschnikowiaceae, and proposal of thirteen new genera, fifty-five new combinations and nine new species. Persoonia 52: 22–43. doi.org/10.3767/persoonia.2024.52.02

[2] Satoh K., Makimura K., Hasumi Y., Nishiyama Y., Uchida K., Yamaguchi H. 2009. *Candida auris* sp. nov., a novel ascomycetous yeast isolated from the external ear canal of an inpatient in a Japanese hospital. Microbiology and Immunology 53(1): 41–44. doi:10.1111/j.1348-0421.2008.00083.x

[3] Lee W.G., Shin J.H., Uh Y., Kang M.G., Kim S.H., Park K.H. et al. 2011. First three reported cases of nosocomial fungemia caused by *Candida auris*. J Clin Microbiol. 49(9): 3139–42. doi:10.1128/JCM.00319-11

[4] Ahmad S., Alfouzan W. 2021. *Candida auris*: epidemiology, diagnosis, pathogenesis, antifungal susceptibility, and infection control measures to combat the spread of infections in healthcare facilities. Micro-organisms 9(4). doi:10.3390/microorganisms9040807

[5] Desoubeaux G., Coste A.T., Imbert C., Hennequin C. 2022. Overview about *Candida auris*: What's up 12 years after its first description? J Mycol Med. 32(2): 101248. doi:10.1016/j.mycmed.2022.101248

[6] Al Maani A., Paul H., Al-Rashdi A., Al Wahaibi A., Al-Jardani A., Al Abri A.M.A. et al. 2019. Ongoing challenges with healthcare-associated *Candida auris* outbreaks in Oman. Journal of Fungi 5(4). doi: 10.3390/jof5040101

[7] Garcia C.S., Palop N.T., Bayona J.V.M., Garcia M.M., Rodriguez D.N., Alvarez M.B. et al. 2020. *Candida auris*: report of an outbreak. Enfermedades Infecciosas Y Microbiologia Clinica 38: 39–44. doi:10.1016/j.eimc.2020.02.007

[8] Biran R., Cohen R., Finn T., Brosh-Nissimov T., Rahav G., Yahav D. et al. 2023. Nationwide outbreak of *Candida auris* infections driven by COVID-19 hospitalizations, Israel, 2021–2022. Emerg Infect Dis. 29(7):1297–1301. doi:10.3201/eid2907.221888

[9] WHO. 2022. WHO fungal priority pathogens list to guide research, development and public health action. Geneva: World Health Organization. Available from: https://www.who.int/publications/i/item/9789240060241

[10] Bidaud AL, Chowdhary A, Dannaoui E.2018. *Candida auris*: An emerging drug resistant yeast – A mini-review. J Mycol Med. 28(3): 568–573. doi:10.1016/j.mycmed.2018.06.007

[11] Spivak E.S., Hanson K.E. 2018. *Candida auris*: an Emerging Fungal Pathogen. J Clin Microbiol. 56(2): e01588-17. doi:10.1128/JCM.01588-17

[12] Casadevall A., Kontoyiannis D.P., Robert V. 2019. On the emergence of *Candida auris*: Climate change, azoles, swamps, and birds. mBio 10(4): e01397-19. doi:10.1128/mBio.01397-19

[13] Jensen R.H. 2016. Resistance in human pathogenic yeasts and filamentous fungi: prevalence, underlying molecular mechanisms and link to the use of antifungals in humans and the environment. Dan Med J. 63(10): B5288.

[14] de Jong A.W., Hagen F. 2019. Attack, defend and persist: How the fungal pathogen *Candida auris* was able to emerge globally in healthcare environments. Mycopathologia 184(3): 353–365. doi:10.1007/s11046-019-00351-w

[15] Bravo Ruiz G, Lorenz A. 2021. What do we know about the biology of the emerging fungal pathogen of humans *Candida auris*? Microbiol Res. 242: 126621. doi:10.1016/j.micres.2020.126621

[16] Borman A.M, Szekely A., Johnson E.M. 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

[17] Chakrabarti A., Singh S. 2020. Multidrug-resistant *Candida auris*: an epidemiological review. Expert review of anti-infective therapy 18(6): 551–562. doi:10.1080/14787210.2020.1750368

[18] Kean R, Ramage G. 2019. Combined antifungal resistance and biofilm tolerance: the global threat of *Candida auris*. mSphere. 2019;4(4): e00458-19. doi:10.1128/mSphere.00458-19

[19] Rybak J.M., Muñoz J.F., Barker K.S., Parker J.E., Esquivel B.D., Berkow E.L. et al. 2020. Mutations in TAC1B: a novel genetic determinant of clinical fluconazole resistance in *Candida auris*. mBio 11(3): e00365-20. doi:10.1128/mBio.00365-20

[20] Fasciana T., Cortegiani A., Ippolito M., Giarratano A., Di Quattro O., Lipari D. et al. 2020. *Candida auris*: An overview of how to screen, detect, test and control this emerging pathogen. Antibiotics (Basel) 9(11): 778. doi:10.3390/antibiotics9110778

[21] Chybowska A.D., Childers D.S., Farrer R.A. 2020. Nine things genomics can tell us about *Candida auris*. Front Genet. 11: 351. doi:10.3389/fgene.2020.00351

[22] Sears D, Schwartz B.S. 2017. *Candida auris*: An emerging multidrug-resistant pathogen. Int J Infect Dis. 63:95-98. doi:10.1016/j.ijid.2017.08.017

[23] Sarma S., Upadhyay S. 2017. Current perspective on emergence, diagnosis and drug resistance in *Candida auris*. Infect Drug Resist. 10: 155–165. doi:10.2147/IDR.S116229

[24] Hoenigl M., Seidel D., Sprute R., Cunha C., Oliverio M., Goldman G.H. et al. 2022. COVID-19-associated fungal infections. Nat Microbiol. 7(8): 1127–1140. doi:10.1038/s41564-022-01172-2

[25] Page M.J., McKenzie J.E., Bossuyt P.M., Boutron I., Hoffmann T.C., Mulrow C.D. et al. 2021. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 372: n71. doi:10.1136/bmj.n71

[26] CDC. *Candida auris*: Antifungal Susceptibility Testing and Interpretation. Available from: https://www.cdc.gov/fungal/candida-auris/c-auris-antifungal.html (accessed on 21 February 2023)

[27] Moola S., Munn Z., Tufanaru C., Aromataris E., Sears K., Sfetcu R. et al. 2020. Chapter 7: Systematic reviews of etiology and risk. *In*: Aromataris E., Munn Z. (Editors). JBI Manual for Evidence Synthesis: JBI doi:10.46658/JBIMES-20-08

[28] Mesini A., Saffioti C., Mariani M., Florio A., Medici C., Moscatelli A. et al. 2021. First case of *Candida auris* colonization in a preterm, extremely low-birth-weight newborn after vaginal delivery. J Fungi (Basel) 7(8): 649. doi:10.3390/jof7080649

[29] Riat A., Neofytos D., Coste A., Harbarth S., Bizzini A., Grandbastien B. et al. 2018. First case of *Candida auris* in Switzerland: discussion about preventive strategies. Swiss Med Wkly 148: w14622. doi:10.4414/smw.2018.14622

[30] Theut M., Antsupova V., Andreasen A.S., Buhl D., Midttun M., Knudsen J.D. et al. 2022. The first two cases of *Candida auris* in Denmark. Ugeskrift for laeger 184(16): V10210768.

[31] Crea F., Codda G., Orsi A., Battaglini A., Giacobbe D.R., Delfino E. et al. 2019. Isolation of *Candida auris* from invasive and non-invasive samples of a patient suffering from vascular disease, Italy, July 2019. Euro Surveill. 24(37). doi:10.2807/1560-7917.ES.2019.24.37.1900549

[32] Stathi A., Loukou I., Kirikou H., Petrocheilou A., Moustaki M., Velegraki A. et al. 2019. Isolation of *Candida auris* from cystic fibrosis patient, Greece, April 2019. Euro Surveill. 24(29): 1900400. doi: 10.2807/1560-7917.ES.2019.24.29.1900400.

[33] Steinmann J., Schrauzer T., Kirchhoff L., Meis J.F., Rath P.M. 2021. Two *Candida auris* cases in Germany with no recent contact to foreign healthcare-epidemiological and microbiological investigations. J Fungi (Basel) 7(5): 380. doi:10.3390/jof7050380

[34] Vogelzang E.H., Weersink A.J.L., van Mansfeld R., Chow N.A., Meis J.F., van Dijk K. 2019. The first two cases of *Candida auris* in the Netherlands. J Fungi (Basel) 5(4): 91. doi:10.3390/jof5040091

[35] Prazynska M., Zalas-Wiecek P., Bogiel T., Wlodarczyk Z., Deptula A., Wozniak M. et al. 2023. *Candida auris* infection in a meningococcal septicemia survivor, Poland. Mycopathologia 188(1–2): 135–141. doi:10.1007/s11046-022-00697-8

[36] Reque J., Arlandis R., Panizo N., Pascual M.J., Perez-Alba A. 2022. *Candida auris* Invasive Infection after Kidney Transplantation. Case Rep Nephrol. 2022: 6007607. doi:10.1155/2022/6007607

[37] Pekard-Amenitsch S., Schriebl A., Posawetz W., Willinger B., Kölli B., Buzina W. 2018. Isolation of *Candida auris* from ear of otherwise healthy patient, Austria, 2018. Emerg Infect Dis. 24(8): 1596–1597. doi:10.3201/eid2408.180495

[38] Rimoldi S.G., Nodari R., Rizzo A., Tamoni A., Longobardi C., Pagani C., et al. 2024. First imported case of *Candida auris* infection in Milan, Italy: genomic characterisation. Infection, 52(4), 1633–1638. doi:10.1007/s15010-024-02232-x

[39] Henriques J., Mixão V., Cabrita J., Duarte T.I., Sequeira T., Cardoso S. et al. 2023. *Candida auris* in intensive care setting: the first case reported in Portugal. J Fungi (Basel) 9(8): 837. doi:10.3390/jof9080837

[40] Caceres D.H., Forsberg K., Welsh R.M., Sexton D.J., Lockhart S.R., Jackson B.R. et al. 2019. *Candida auris*: A review of recommendations for detection and control in healthcare settings. J Fungi (Basel) 5(4): 111. doi:10.3390/jof5040111

[41] Osei Sekyere J. 2018. *Candida auris*: A systematic review and meta-analysis of current updates on an emerging multidrug-resistant pathogen. Microbiologyopen 7(4): e00578. doi:10.1002/mbo3.578

[42] Geremia N., Brugnaro P., Solinas M., Scarparo C., Panese S. 2023. *Candida auris* as an emergent public health problem: a current update on european outbreaks and cases. Healthcare (Basel) 11(3): 425. doi:10.3390/healthcare11030425

[43] Kohlenberg A., Monnet D.L., Plachouras D. 2022. Increasing number of cases and outbreaks caused by *Candida auris* in the EU/EEA, 2020 to 2021. Euro Surveill. 27(46): 2200846. doi:10.2807/1560-7917.ES.2022.27.46.2200846

[44] European Centre for Disease Prevention and control *Candida auris* in healthcare settings – Europe. Available from: https://www.ecdc.europa.eu/sites/default/files/documents/RRA-Candida-auris-European-Union-countries-first-update.pdf (accessed on 16 February 2023)

[45] Ahmad S., Asadzadeh M. 2023. Strategies to prevent transmission of *Candida auris* in healthcare settings. Curr Fungal Infect Rep. 17(1): 36–48. doi:10.1007/s12281-023-00451-7

[46] Garcia-Bustos V., Cabanero-Navalon M.D, Ruiz-Saurí A., Ruiz-Gaitán A.C., Salavert M., Tormo M. et al. 2021. What do we know about *Candida auris*? State of the art, knowledge gaps, and future directions. Microorganisms 9(10): 2177. doi:10.3390/microorganisms9102177

[47] Schelenz S., Hagen F., Rhodes J.L., Abdolrasouli A., Chowdhary A., Hall A. et al. 2016. First hospital outbreak of the globally emerging *Candida auris* in a European hospital. Antimicrobial Resistance and Infection Control 5: 35. doi:10.1186/s13756-016-0132-5

[48] Kullberg B.J., Arendrup M.C. 2015. Invasive candidiasis. N Engl J Med. 373(15): 1445–1456. doi: 10.1056/NEJMra1315399

[49] Jamalian A., Freeke J., Chowdhary A., de Hoog G.S., Stielow J.B., Meis J.F. 2023. Fast and accurate identification of *Candida auris* by High Resolution Mass Spectrometry. J Fungi (Basel) 9(2): 267. doi:10.3390/jof9020267

[50] Cornely O.A, Bassetti M., Calandra T., Garbino J., Kullberg B.J., Lortholary O. et al. 2012. ESCMID* guideline for the diagnosis and management of *Candida diseases* 2012: non-neutropenic adult patients. Clin Microbiol Infect. 18(Suppl 7): 19–37. doi:10.1111/1469-0691.12039

[51] Cortegiani A., Misseri G., Fasciana T., Giammanco A., Giarratano A., Chowdhary A. 2018. Epidemiology, clinical characteristics, resistance, and treatment of infections by *Candida auris*. J Intensive Care 6: 69. doi:10.1186/s40560-018-0342-4

[52] Navalkele B.D., Revankar S., Chandrasekar P. 2017. *Candida auris*: a worrisome, globally emerging pathogen. Expert Rev Anti Infect Ther. 15(9): 819–827. doi:10.1080/14787210.2017.1364992

[53] Aslam S., Rotstein C. 2019. *Candida* infections in solid organ transplantation: Guidelines from the American Society of Transplantation Infectious Diseases Community of Practice. Clin Transplant. 33(9): e13623. doi:10.1111/ctr.13623

[54] Heath C.H., Dyer J.R., Pang S., Coombs G.W., Gardam D.J. 2015. *Candida auris* Sternal Osteomyelitis in a Man from Kenya Visiting Australia, 2015. Emerg Infect Dis. 25(1): 192–194. doi:10.3201/eid2501.181321

[55] Centers for Disease Control and Prevention *Candida auris* Screening. Available from: https://www.cdc.gov/fungal/candida-auris/c-auris-screening.html (accessed on 16 February 2023)

[56] Chakrabarti A., Sood P. 2021. On the emergence, spread and resistance of *Candida auris*: host, pathogen and environmental tipping points. J Med Microbiol. 70(3): 001318. doi:10.1099/jmm.0.001318

[57] Gómez-Gaviria M., Martínez-Álvarez J.A., Chávez-Santiago J.O., Mora-Montes H.M. 2023. *Candida haemuloni*i complex and *Candida auris*: biology, virulence factors, immune response, and multidrug resistance. Infect Drug Resist. 16: 1455-1470. doi:10.2147/IDR.S402754

[58] Ninan M.M., Sahni R.D., Chacko B., Balaji V., Michael J.S. 2020. *Candida auris*: Clinical profile, diagnostic challenge and susceptibility pattern: Experience from a tertiary-care centre in South India. Journal of Global Antimicrobial Resistance 21: 181–185. doi:10.1016/j.jgar.2019.10.018

[59] Di Pilato V., Codda G., Ball L., Giacobbe D.R., Willison E, Mikulska M, et al. 2021. Molecular epidemiological investigation of a nosocomial cluster of *C. auris*: evidence of recent emergence in Italy and ease of transmission during the COVID-19 Pandemic. Journal of Fungi 7(2): 140. doi:10.3390/jof7020140

[60] *Candida auris* outbreak in healthcare facilities in northern Italy, 2019–2021. Available from: https://www.ecdc.europa.eu/sites/default/files/documents/RRA-candida-auris-Feb2022.pdf (accessed on 10 October 2023)

[61] Treatment and Management of *C. auris* Infections and Colonization. Available from: https://www.cdc.gov/fungal/candida-auris/c-auris-treatment.html (accessed on 10 October 2023)

[62] Ghannoum M., Arendrup M.C., Chaturvedi V.P., Lockhart S.R., McCormick T.S., Chaturvedi S. et al. 2020. Ibrexafungerp: A novel oral triterpenoid antifungal in development for the treatment of *Candida auris* infections. Antibiotics (Basel) 9(9): 539. doi:10.3390/antibiotics9090539

[63] Sahu R.K., Yadav R., Prasad P., Roy A., Chandrakar S. 2014. Adverse drug reactions monitoring: prospects and impending challenges for pharmacovigilance. Springerplus 3: 695.

[64] Warris A. 2018. *Candida auris*, what do paediatricians need to know? Arch Dis Child. 103(9): 891–894. doi:10.1186/2193-1801-3-695

Received 18 December 2024
Accepted 28 December 2025