Candida auris

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Purpose of review
To give an update on the recent emergence of *Candida auris*.

Recent findings
C. auris is a pathogen, that evades present therapeutic options, that is highly virulent, causes disease in all types of patients, and spreads easily in the environment and among patients, thereby posing an imminent threat to our patients. The fact that *C. auris*, in addition, is more resilient to environmental disinfection and frequently misclassified during microbiological diagnostics only heightens its potential as a 'perfect villain.'

Summary
Healthcare institutions, especially hospitals, need to ensure that their diagnostic and infection control policies to handle *C. auris* are in place.

Keywords
Candida auris, emerging, multidrug resistance, yeast

INTRODUCTION

Candida auris was first described in 2009 in Japan after being isolated from external ear discharge of a patient [1]. As of 2011, sporadic cases and clusters of *C. auris,* specifically fungemia, emerged in many different geographical regions [2▪]. Molecular typing of strains suggest isolates are highly related within a region but highly distinct between continents, showing clustering in four distinct clades, suggesting independent emergence [3▪▪,4,5▪–7▪,8]. On the basis of retrospective evaluation of isolate collections, the earliest known infection with *C. auris* occurred in South Korea in 1996 [9] followed by Pakistan in 2008 [10▪▪] and India in 2009 [11▪]. A review of the SENTRY collection with over 15 000 isolates from four continents between 2004 and 2015 did not reveal the presence of other misidentified *C. auris* from samples collected before 2009 [5▪]. A recent detailed search for *C. auris* in Taiwan of more than 5000 archived *Candida* isolates from the period 1999–2016 was negative [12▪]. Consequently, experts assume that the current emergence of *C. auris* has to be seen as the manifestation of an 'old bug' in new clinical settings, possibly because of increasing antifungal selection pressures in humans, animals, and the environment [4,5▪,13▪▪]. Above all, effective control may be hampered, by unknowns, such as the population prevalence, environmental niches, and the true mechanisms of spread [14▪].

CLINICAL PRESENTATION

Candida auris has been reported to cause bloodstream infections, wound infections, and otitis [9]. It has also been cultured from other sites and media including the respiratory tract and urine. *Candida auris* has been documented to cause infections in patients of all ages; however, with predominance reported for male patients and patients in the ICU [3▪▪,4,15▪,16▪]. In general, patients were found to have similar risk factors for infections as those patients with other *Candida spp.* infections, including: immunocompromising diseases, recent surgery, recent antibiotics, and presence of central venous catheters or urinary catheters [3▪▪,4,5▪,9,17,18▪,19▪,20,21]. Additionally, detection of *C. auris* has been reported, in patients receiving antifungals for infections with other *Candida spp.* [9]. Pathogenic potential of *C. auris* is almost the same as that of *Candida albicans* as shown in

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KEY POINTS

- Increasing global spread.
- Growing number of international outbreaks.
- Attention needed for early detection and identification.
- Intense infection control measures and attention to environmental cleaning should be given at first detection of *C. auris*.

animal models [22]. The time from hospital admission to onset of candidemia with *C. auris* has been reported to range between 9 and 62 days [4,5▪,15▪,18▪,23,24▪]. The delayed occurrence of candidemia suggests a nosocomial acquisition and spread [3▪▪,15▪,18▪,25▪▪].

The overall crude 30 day in-hospital mortality rate from *C. auris* candidemia in case series ($n > 1$) ranges from 33 to 72% [5▪,15▪,18▪,26▪]. Better survival likelihood was seen for neonates and infants and patients with immediate source control [9,18▪]. *Candida auris* attributable mortality cannot be established from those studies as underlying medical conditions are severe and because of the multidrug-resistant nature of *C. auris*. Research from the United Kingdom, however, showed no direct contribution of *C. auris* to death of patients [3▪▪].

ANTIFUNGAL RESISTANCE AND THERAPY

Although at the moment, no established minimum inhibitory concentration (MIC) breakpoints exist for *C. auris*, initial testing of an international collection of 54 isolates demonstrated that nearly all (93%) isolates were highly resistant to fluconazole based on breakpoints established for other *Candida spp.* [5▪]. In that study, more than half of *C. auris* isolates were resistant to voriconazole, around one-third (35%) were resistant to amphotericin B (MIC ≥2), and 7% were resistant to echinocandins [4]. Forty-one percentage of isolates were resistant to two antifungal classes and some (4%) isolates have demonstrated elevated MICs to all three major antifungal classes, including azoles, echinocandins, and polyenes, indicating that treatment options would be very limited [5▪]. Similar findings were reported from Kuwait showing 100% fluconazole, 73% voriconazole and 23% amphotericin B resistance among 56 isolates [27]. A large study with 350 isolates included (75% blood culture isolates) gave a less grim prospect of resistance percentages outside fluconazole. This study reported 90% of *C. auris* being fluconazole-resistant (MICs 32–64 mg/l), 8% amphotericin B-resistant (≥2 mg/l), 15% voriconazole resistant (>1 mg/l) and 2.5% resistant to echinocandins (16 mg/l) [11▪].

Echinocandin use, therefore, has become more widespread and the go-to drug, although *C. auris* isolates with reduced susceptibility for this drug have been reported [5▪,28▪]. Fortunately, several new drugs with activity against *C. auris* are becoming available. The 1,3-β-D-glucan synthesis inhibitor SCY-078 has shown promising antifungal activity against all *C. auris* clades [13▪▪,29▪] as has the new drug APX001 (a GPI-anchored wall transfer protein 1) [30▪] and rezafungin (previously CD101) a long-acting echinocandin [28▪]. Unfortunately, the latter drug appears also to be inactive if the newly described substitution S639F in the FKS1 hotspot region is present [11▪,28▪]. Elevated echinocandin MICs were associated only with clinical failure if FKS1 mutations are present [31]. In-vitro combination of antifungal drugs against resistant *C. auris* provided some encouraging data [32▪].

EPIDEMIOLOGY

At present, *C. auris* infections, specifically fungemia, have been reported from South Korea [9], Japan [33], India [4], Pakistan [5▪], South Africa [34], Israel [35▪], Kuwait [23], Venezuela [18▪], Colombia [26▪,36], Panama [37▪], the United Kingdom [3▪▪], Spain [38▪], Oman [15▪,16▪], Canada [39▪], United Arab Emirates [40▪], Malaysia [41▪], the United States of America [42▪▪,43▪], Switzerland [44], and Germany [45]. *Candida auris* has also been identified in Austria, Belgium, China, and France [46,47]. Australia, Kenya, Norway, Russia, Saudi Arabia, Singapore, Thailand, and the Netherlands have reported cases, although detailed or published reports of these cases are not available [45,48▪▪,49]. Retrospectively it was found that the reported *C. auris* isolates from Brazil originated from Venezuela. A major outbreak has recently been described in Spain [50]. Figure 1 visualizes the worldwide report of *C. auris* infections. Moreover, official reports and publications on outbreaks will always be one step behind, making it harder to assess the true prevalence and threat of *C. auris* and consequently highlights the need for infection prevention guidelines.

Whilst isolated sporadic cases occur, there is a growing concern regarding the propensity of *C. auris* to cause widespread nosocomial outbreaks [19▪]. Clusters have been found around the globe [18▪,24▪,34,50–52]. In some regions *C. auris* is among the top-3 Candida species isolated from bloodcultures [53]. As of August, 2016, the CDC has been accumulating outbreak data in the United States of America. Within the first month, seven cases were recorded [24▪]. As of 2 March 2018, 215

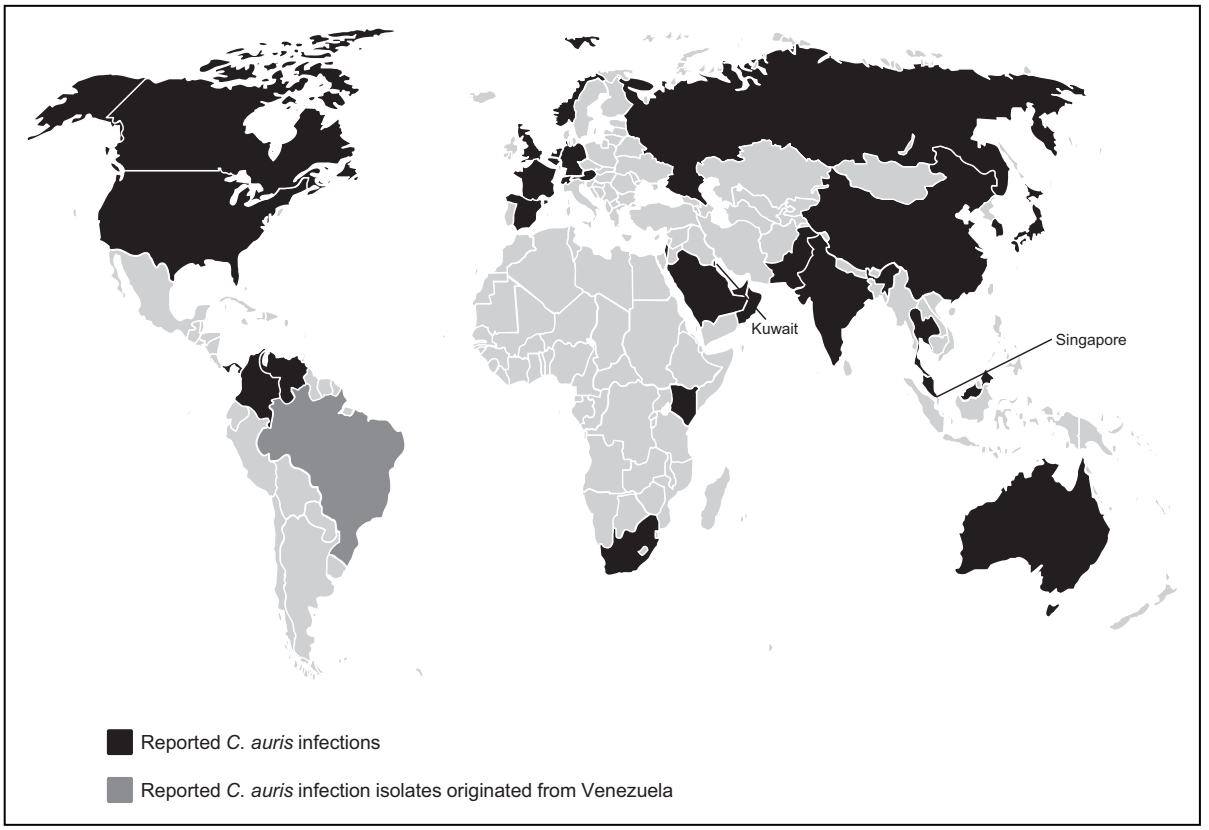

FIGURE 1. World map highlighting reported *Candida auris* infections including single case reports (published and unpublished).

confirmed infection cases were recorded and 347 colonized patients ia targeted screening [42▪▪].

Schelenz *et al.* [3▪▪] suggested that *C. auris* is capable of horizontal transmission in the healthcare setting causing potentially serious infections of a global concern. *Candida auris* poses a serious risk to critically ill patients. Therefore, immunosuppressed patients should not share facilities with colonized or infected patients [54▪]. Also, intensive care stay has been reported a major risk factor for *C. auris* [3▪▪,18▪,55]. Furthermore, patients diagnosed with a *C. auris* infection and who received treatment with antifungals to which the specific *C. auris* was susceptible, were not all cleared of the infection [24▪]. Acquisition of *C. auris* is suggested to be as little as 4 h, from either a patient or the environment [3▪▪]. Additionally, *C. auris* remains viable for at least 2 weeks up to 7 months on environmental hospital surfaces [3▪▪,24▪,56▪] and has shown pathogenicity with biofilm formation capability and a range of virulence factors aiding in nosocomial spread [13▪▪,57▪].

DIAGNOSTICS

Candida auris requires dedicated methods for identification because it is often misidentified as *Candida haemulonii, Candida duobushaemulonii,* hypopigmented *Rhodotorula glutinis* or *Saccharomyces cerevisiae* when using traditional biochemical methods [58]. Most clinical laboratories do not routinely perform molecular identification, which has led to underestimation of the prevalence of *C. auris* in the early days [19▪]. *Candida auris* isolates are ovoid without pseudohyphae on microscopic examination and may be difficult to distinguish from other species of *Candida* [16▪]. The organism appears pale purple to pink on CHROM agar and grows at 37–42°C. Phenotypic identification with API or automated systems such as VITEK-2 do not correctly speciate *C. auris* and give false names such as *C. haemulonii*, *Candida sake*, and *Rhodotorula mucilaginosa*. If microscopic examination would be used more often *C. auris* would not be mistaken with *C. haemulonii*, the latter form pseudohyphae with blastoconidia and does not grow at 42 °C, which certainly may aid in differentiating the two species. Currently, the most reliable method for speciation next to molecular based methods, are MALDI-TOF MS both the Bruker and the MS-VITEK (Biomerieux, Marcy l'Etoile, France) platforms but only if the databases are up to date [59].

At present, there is heightened awareness among microbiologists regarding misidentification of *C. auris* by commercial identification systems and their inclination for submitting *Candida* isolates for analysis by MALDI-TOF MS or sequencing.

Recently rapid, robust, easy-to-perform and interpret PCR and real-time PCR assays to identify *C. auris* and related species have been developed [60▪]. The performance of a *C. auris* real-time PCR assay was recently evaluated by using 623 surveillance samples, including 365 patient swabs and 258 environmental. PCR detected more positive samples and far more quicker than conventional culture-based surveillance [61▪]. This allows for accurate and rapid screening of *C. auris* and can increase effective control and prevention of this emerging multidrug-resistant fungal pathogen in healthcare facilities similar as we routinely do with screening for vancomycin-resistant *Enterococcus* (VRE), methicillin-resistant *Staphylococcus aureus* (MRSA) and *Clostridium difficile*.

Surveillance cultures can best be taken from clinical sites where other multiresistant microorganisms are cultured from: nose/throat, groin, axilla, rectum/peri-anal, central venous, and urinary catheters and clinical material such as urine, stool samples, and wound swabs even though the most adequate places for culturing of potential carriers of *C. auris* are still not conclusive [3▪▪,25▪▪,56▪,62,63].

INFECTION CONTROL AND PREVENTION

In essence, the infection control and prevention measures for *C. auris* are similar to those taken for other highly resistant microorganisms these are standard precautions including hand hygiene, adequate use of personal protective equipment, contact isolation in a single person isolation room (with ante-room and if available with airlock control system), and meticulous environmental cleaning [19▪].

Candida auris is a nosocomial disease-causing pathogen as shown by Schelenz *et al.* where prospective screening of new patients showed *C. auris* in 0.04% (1/2246) of patients in 1 year [3▪▪]. Therefore, prevention of spread starts with proper hand hygiene by healthcare workers. Schelenz *et al.* [3▪▪] did not find colonization with *C. auris* of healthcare workers (only transient carriage) who work closely with colonized or infected patients whereas Biswal *et al.* [25▪▪] found 3% (4/145) healthcare workers to be colonized on their hands. Biswal *et al.* [25▪▪] also showed that proper use of hand hygiene measures as per protocol eliminates *C. auris* thereby limiting spread and highlighting the importance of proper hand hygiene.

All contact patients of an infected patient should be traced and placed into (cohort) isolation until proven free off *C. auris*. All *C. auris* patients should be 'flagged' to ensure isolation measures and use of barrier precautions as well as screening at any readmission to the healthcare facility [62,64▪▪]. When patients move department within their institution or get transferred to another healthcare facility, extra caution and solid precaution measures should be taken. Family and visitors need to be instructed by the department of adequate use of isolation measures, such as personal protective equipment (PPE) use and hand hygiene. In addition, they should not visit other patients after seeing the *C. auris* patient.

Research into decolonization strategies are urgently needed, as decolonization strategies, with the possible exception of povidone iodine, are non-conclusive [25▪▪,65▪]. Biswal *et al.* [25▪▪] found the axilla to be the most heavily colonized site in patients possibly because of the use of colonized monitoring equipment near this body side. Unfortunately, the use of chlorhexidine washes has no proven consistent decolonization effect [25▪▪,66▪▪]. Abdolrasouli *et al.* [66▪▪] suggest possible recolonization from the environment and Biswal *et al.* [25▪▪] suggest that the ineffectiveness is possibly because of the use of less than recommended contact time. For oral decolonization nystatin has been found effective *in vivo* [3▪▪,25▪▪], however, *in vitro*, these effects are not supported for oral or skin eradication [11▪]. Sertaconazole is suggested as an effective antifungal with use as skin decontamination and topical management [11▪].

Environment contamination is of great importance for the spread and consequently the control of *C. auris*. Patients may shed *C. auris* for weeks to months from their skin and other body sites [3▪▪,25▪▪]. Consequently, the environment will be heavily colonized where even linen and mattresses have been shown to be colonized for up to 7 days [3▪▪,24▪,25▪▪,55]. However, timely and adequate precaution measures can possibly reduce the environmental contamination and spread within a healthcare setting [62,67▪]. Also wet and dry surfaces have been found to contain viable *C. auris* including temperature probes and echocardiogram leads [25▪▪,66▪▪]. Therefore, profound cleaning of the room and surrounding and especially of multiple-use equipment is essential. Where possible single use equipment should be used to avoid spread via inadequately disinfected equipment. Local waste and soiled linen policies for multiresistant microorganisms should be followed. At present, the best disinfection method is not yet conclusively known. Currently, more data is published on the effectiveness of environmental cleaning agents for *C. auris*, specifically [3▪▪,25▪▪,66▪▪]. Studies suggest

that high-strength chlorine-based agents, hydrogen peroxide (with silver nitrate) vaporization, and phenol are effective when used as per manufacturer's directions and protocol [3▪▪,24▪,25▪▪]. In addition, technology solutions such as UV-C disinfection may be helpful [68▪] (JF Meis and A Voss, unpublished data].

CONCLUSION

Candida auris is getting more and more attention and more and more is known about its characteristics. Additionally, the global awareness has prompted microbiologists to use different identification methods or send samples for analyses elsewhere making identification quicker. New antifungals are being tested and slowly more is known about this deadly yeast. The serious threat posed by *C. auris* has prompted various Centers for Disease Control and countries to rightfully issue alerts and guidelines for actively identifying and reporting *C. auris* to prevent its transmission in hospitals which with globally increasing numbers of reports is definitely needed [19▪,64▪▪].

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