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Review

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Candida Auris — a New Pathogen of Nosocomial Infections

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ABSTRACT In recent years, the number of infectious diseases caused by fungi has been progressively increasing, which is a serious clinical problem. The literature review is devoted to *Candida auris* — a new causative agent of nosocomial fungal infections with multidrug resistance. This pathogen was first described in 2009. As of the beginning of 2021, the documented isolation of *Candida auris* was noted in 47 countries around the world. This pathogen can persist for a long time on various surfaces in hospitals, is resistant to antifungal drugs and traditional disinfectants, and causes invasive infections accompanied by high mortality. The study of *Candida auris* is important both for the development of approaches to the diagnosis and treatment of diseases caused by this pathogen, and for predicting the emergence of new pathogens in the future.

Keywords: Candida auris, fungal infection, nosocomial infections, infection control

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Over the past few decades, new high-tech methods for diagnosing and treating various diseases have been regularly introduced into clinical practice. This makes it possible to save the lives of patients in critical conditions, who until recently were considered hopeless. Many achievements of modern medicine have become possible thanks to the active use of new antimicrobial drugs with a wide spectrum of action in practical healthcare.

However, the widespread use of new technologies and drugs also has its downside. In recent years, the number of immunocompromised patients with a high risk of developing bacterial complications and invasive mycoses has increased. The unrestricted use of antimicrobial drugs has contributed to the growth of pathogen resistance to antibiotics, and an increase in the etiological role of bacteria and fungi that were not previously found in the structure of the main pathogens.

The last decade is marked by the emergence and worldwide spread of *Candida auris* – a multidrug-resistant yeast-like fungus. This pathogen was described and registered as a new species of the genus *Candida* in 2009 [1]. It got its name due to the fact that it was first isolated from the external auditory canal of a Japanese hospital patient. Phylogenetically, *C. auris* is similar to the species *Candida haemulonii*, *Candida pseudohaemulonii* and *Candida ruelliae*. Traditional diagnostic methods using manual test systems or automatic microbiological analyzers do not accurately identify *C. auris*. To date, it is possible to reliably distinguish the new species from other yeast-like fungi only using one of two laboratory diagnostic methods: matrix-assisted laser desorption- ionization (MALDI-TOF) or real-time polymerase chain reaction (PCR) with sequencing of the D1–D2 region of the 28S ribosomal DNA. Due to the complexity of identifying this pathogen, it is very difficult to assess the extent of its true prevalence.

PREVALENCE IN THE WORLD AND THEORIES OF EMERGENCE

Following the description of the new species of *C. auris* in 2009, retrospective analyzes of international and national yeast culture collections were conducted along with prospective studies to search for misidentified isolates. The earliest isolate was discovered in South Korea thanks to the re-identification of an unidentified in 1996 strain, the causative agent of fungemia in a one-year-old child [2]. No other *C. auris* isolates were found among previously unidentified cultures.

Since 2009, reports of *C. auris* isolation from patients in various countries began to appear. In South Korea, two cases of fungemia caused by *C. auris* [2] were recorded in 2009. In India, 12 C. auris isolates were identified in patients with bloodstream infections between 2009 and 2011 [3]. In South Africa, the first documented case of *Candida auris* also occurred in 2009; however, the pathogen was initially misidentified as *Candida haemulonii*. The presence of *C. auris* was only confirmed retrospectively in 2014, when four more cases of fungemia caused by the novel pathogen were described in South Africa [4].

In European countries, *C. auris* was first isolated in 2013 from 2 patients in the UK [5]. Subsequently, Europe's first documented outbreak of nosocomial infections due to *C. auris* occurred at a cardiothoracic center in London [6].

Between November 2015 and December 2017, *C. auris* was isolated from 7 patients treated in different hospitals in Germany [7]. Six of them were previously observed in medical centers in other countries and were transferred to Germany for further treatment.

In the United States, 7 cases of *C. auris* were reported between May 2013 and August 2016 in four states. Six of the seven cases were identified by retrospective analysis. Only one of these patients had left the United States and was transferred from a hospital in the Middle East [8].

The first documented isolation of *Candida auris* in Russia occurred in Moscow in October 2016 in a 40-year-old patient from Central Asia. In the period 2016–2017, 49 cases of *C. auris* were detected in the intensive care unit of the same clinic [9].

In 2019, the European Center for Disease Prevention and Control (ECDC) reported 349 cases of C. auris isolation from 1 January 2018 to 31 May 2019 in European countries: Spain (n=291), UK (n=48), Germany (n=3), the Netherlands (n=2), Austria (n=1), France (n=1), Greece (n=1), Norway (n=1) and Poland (n=1). Bloodstream infections accounted for 84 cases (24.1%), other infections accounted for 7 (2.0%), and carriers accounted for 257 (73.6%). Among all reported cases, 324 (92.8%) were considered to be “local” infections; 19 (5.4%) were considered imported because patients were transferred from other countries with high incidence of *C. auris*. In 6 cases (1.7%) the site of infection was unknown [10].

As of February 2021, the US Centers for Disease Control and Prevention (CDC) reported the isolation of *Candida auris* in 47 countries worldwide [11]. The true spread of *C. auris* infection is currently unknown. This is due to the limitations of existing laboratory diagnostic methods in practical healthcare.

Whole-genome sequencing of *C. auris* isolates showed that the novel pathogen arose simultaneously and independently in different geographical areas of the world [12]. Phylogenetic analysis identified four major clades of *C. auris:* South Asian, East Asian, African and South American, or I, II, III and IV, respectively. Isolates from these clades differ genetically from each other by tens or hundreds of thousands of single nucleotide polymorphisms (SNPs).

To explain the emergence and spread of *C. auris*, scientists put forward several hypotheses. The simplest one suggests that *C. auris* was previously unrecognized using available laboratory methods and was misidentified as other *Candida* species. A reanalysis of 15,271 isolates collected as part of the SENTRY Antifungal Surveillance Program was conducted at 152 medical centers worldwide during 2004–2015. Only four putative *C. haemulonii* were reidentified as *C. auris* [13]. The absence of *C. auris* from historical culture collections and its rapid worldwide spread after 2009 suggest that this pathogen has only recently emerged as a human pathogen.

Another hypothesis suggests that the emergence of the novel pathogen was a natural biological response to the uncontrolled use of antifungal drugs in both medicine and agriculture. It was proven that the irrational use of antimicrobials creates selective pressure that promotes the selection, survival and proliferation of resistant strains of bacteria. This principle probably holds true for fungal pathogens as well.

Fungi, as causative agents of infections in humans, have been known since 1842 [14]. For a long time, only antiseptics and potassium iodide were used to treat fungal infections. And only in 1950 the antimycotic nystatin was isolated [15]. Significant advances in antifungal drug development occurred in the 70s of the 20th century. Azoles were synthesized and put into practice: clotrimazole (1969), miconazole and ketoconazole (1978), itraconazole (1980), fluconazole (1982). At the end of the 1970s, a novel class of antifungal drugs was discovered – echinocandins. Caspofungin (2001), micafungin (2005) and anidulafungin (2006) were introduced into clinical practice.

Since the 1970s, antifungal drugs, in addition to medical use, have become widely used in agriculture. By the early 1980s, about 10 different azole drugs were available for agricultural use. It was proven that azoles can penetrate into groundwater and accumulate in soil [12].

Although an environmental reservoir of *C. auris* has not yet been reported, the presence of some ecological niche for *C. auris* from which the pathogen spread cannot be ruled out. The high probability of this theory is confirmed by the fact that *C. auris* in laboratory conditions can grow on nutrient media at temperatures above 40°C. This distinguishes this pathogen from most other fungi, which do not survive at physiological temperatures of the human body (36.5–37.5 ° C) and, accordingly, cannot colonize people and cause infectious diseases. Global warming may have contributed to the evolution of *Candida auris* into a human pathogen [16].

It is likely that in different parts of the world, the pressure of natural selection exerted by the widespread use of antibiotics and antimycotics in medicine, environmental pollution from pharmaceutical and agricultural waste, and global warming led to the emergence of *C. auris*. Subsequently, the novel pathogen colonized people with a disturbed natural microbiome, and adaptation of *C. auris* as a pathogen of nosocomial infections occurred.

ROUTES OF TRANSMISSION

Candida auris is successfully transmitted from person to person. This pathogen can colonize the skin of patients' axillae and groin asymptomatically for several weeks. Invasive infections may develop several months after colonization. Therefore, population migration, international travel and medical tourism have contributed to the transfer of *C. auris* to different countries with subsequent local spread.

The contaminated patient entering a medical facility becomes a source of contact transmission of the pathogen. An investigation into outbreaks of hospital-acquired infections caused by *C. auris* revealed contamination of floors, window sills, radiator grilles, medical equipment monitors, keypads, blood pressure monitors, and other surfaces in the rooms of the infected patients. Healthcare workers caring for the infected patients had temporary colonization of the hands, nostrils, and groin in 1% of cases. It was established that *C. auris* can exist in a viable state for up to 7 days on dry surfaces of environmental objects. Studies showed that contact with contaminated surfaces for 4 hours is sufficient to colonize hospital patients [6, 17].

A case of transmission of *C. auris* from donor to recipient during lung transplantation was described [18]. Initially, yeast-like fungi were isolated from the donor and identified as *Candida haemulonii*. Isolation of such a pathogen is not a contraindication for transplantation. And only after the development of the infectious process in the recipient was it possible to accurately identify the causative agent of the disease as *Candida auris*.

CLINICAL MANIFESTATIONS

In most cases, the clinical manifestations of infection caused by *C. auris* are nonspecific and do not differ from other systemic mycoses. This pathogen can cause fungemia, nosocomial

pneumonia, urinary tract infections, skin and soft tissue infections, meningitis, and otitis. Unlike other *Candida* species that are commensals of the human gastrointestinal tract, *C. auris* colonizes primarily the skin. The colonized patients are the main source of transmission of the pathogen to other people.

Risk factors for infection by *C. auris* are similar to those for other *Candida* species:

- immunosuppressive state;
- diabetes;
- chronic kidney diseases;
- previous therapy with broad-spectrum antibiotics or antifungal drugs;
- parenteral nutrition;
- central venous or urinary catheter insertion.

DIAGNOSIS

Laboratory diagnosis of *C. auris* using traditional phenotypic and biochemical methods is difficult due to frequent identification errors. When cultivated on chromogenic media, this pathogen can form polymorphic colonies of various colors (white, pink, purple). When using Vitek 2 (bioMérieux), Phoenix (BD), MicroScan (Beckman Coulter) automatic microbiological analyzers, *C. auris* isolates are mistakenly identified as other species: *C. haemulonii*, *C. famata*, *Rhodotorula glutinis* and others. When isolating such fungal species, it is currently recommended to perform further research in order to exclude *C. auris* [17, 19].

The cultivation temperature range for *C. auris* is 30–42°C. In addition, this pathogen is able to tolerate high concentrations of salt (>10% NaCl) [20]. These characteristics can help presumptively identify *C. auris*, but should not be used as the sole diagnostic methods.

For accurate identification of *Candida auris* in routine practice, the MALDI-TOF mass spectrometry (matrix-assisted laser desorption ionization) method is recommended. The "gold standard" for diagnosing *C. auris* infection is considered to be real-time polymerase chain reaction with sequencing of the D1–D2 region of the 28S ribosomal DNA, which has 100% specificity. Due to its high cost, this method is less common in widespread clinical practice.

TREATMENT

Treatment of infections caused by *C. auris* is similar to the treatment of other fungal diseases, but must take into account the high level of resistance of the pathogen to existing antimycotics. A significant proportion of *C. auris* strains isolated worldwide are resistant to several, and sometimes even all, available antifungal drugs [21]. The majority of *C. auris* isolates are resistant to the two main classes of antifungal drugs (azoles and polyenes). The least resistance is observed to echinocandins. Drugs of this group in standard therapeutic dosages are recommended as initial therapy for infections caused by *C. auris*. The patients should be closely monitored during treatment. If there is no significant clinical effect from treatment with echinocandins, or fungemia persists for more than 5 days, it is recommended to change therapy to liposomal amphotericin B (5 mg/kg per day) or a combination of echinocandins with liposomal amphotericin B [22, 23].

The effectiveness of combination antifungal therapy is being studied. In vitro studies have shown that the combination of micafungin with voriconazole has a synergistic effect against multidrug-resistant *C. auris* strains [24].

Active research is currently underway to develop new antifungal drugs, including those active against *C. auris*.

INFECTION CONTROL AND PREVENTION

Recommendations for infection control and prevention of infections caused by *C. auris* developed in many countries were adapted from infection control strategies for other infectious diseases [22, 25, 26].

It has now been proven that outbreaks of nosocomial infections are most often associated with pathogen transmission through hands and contaminated surfaces [6, 17, 27]. Therefore, the main infection control measures include contact precautions (hand hygiene, use of disposable gloves and gowns, high-quality cleaning of instruments, use of disposable care items, isolation of sick patients).

Health care personnel who come into contact with *C. auris*-infected patients should follow

standard hand hygiene principles. In case of severe contamination, hands should be washed with soap and water. The preferred hand disinfectants are chlorhexidine or alcohol-based products. The use of gloves is not a substitute for hand hygiene.

C. auris can survive in health care settings on a variety of surfaces for up to 14 days in a viable state [20]. On hospital surfaces, *C. auris* not only withstands drying, but also resists quaternary ammonium disinfectants, peracetic acid, standard concentrations of sodium hypochlorite, and standard UV cycle times [22, 25]. Experts recommend using strong chlorine disinfectants, hydrogen peroxide with silver nitrate, or phenol to disinfect the environment [22, 25, 26].

If a patient infected with *C. auris* is identified, it is recommended that he be isolated in a separate room with a limited circle of interacting medical personnel and strict adherence to contact precautions.

Studies have shown that colonization of patients by *C. auris* after hospital discharge can persist from 1 month to 3 years with the development of subsequent invasive infection [6, 20, 28]. It is recommended to examine the colonized patients once a week for at least 3 months.

Preliminary screening of patients for *C. auris* carriage is recommended when transferring patients from health care facilities with proven cases of such infections, and in cases of known contact with infected or colonized individuals [22, 25, 26]. Recommended screening sites: groin, axilla, urine, nasal cavity, perineal and rectal smears. If a patient is identified as colonized with *C. auris*, it is recommended that infection control measures be taken the same as for infected patients.

One of the factors contributing to the emergence and spread of *C. auris* is considered to be the uncontrolled use of antibiotics and antifungal drugs. Therefore, in hospitals faced with the development of *C. auris* infections, it is necessary to reconsider approaches to antimicrobial therapy. Rational antimicrobial therapy will not only achieve therapeutic and cost effectiveness of treatment, but also minimize the unintended consequences of the use of antimicrobial drugs, such as the spread of multidrug-resistant strains of bacteria and fungi.

CONCLUSION

Candida auris is a novel multidrug-resistant pathogen. It spread to hospitals around the world over the past ten years due to its ability to colonize human skin, persist for a long time on all types of surfaces, and its resistance to standard disinfection regimes. Difficulties in routine microbiological diagnosis, and violations of generally accepted infection control requirements in hospitals contributed to the fact that *Candida auris* became a serious problem for clinicians and microbiologists throughout the world.

Studying the causes for the infection's occurrence, as well as the mechanisms of development and the routes of spread of *C. auris* will allow scientists to predict the emergence of novel pathogens in the future, and develop measures to prevent the spread of multidrug-resistant pathogens.

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