

A BRIEF OVERVIEW OF CANDIDIASIS

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Abstract: Candidiasis is generally provoked by candida species, which is a fungal disease impacting different mucosal surfaces in the human body. This review provide a brief explanation on candidiasis, encircling its epidemiology, pathogenesis, clinical indications, diagnoses, and treatment. Candida, especially *Candida albicans* inhabit symbiotic microorganisms in the oral cavity, gastrointestinal tract, and genitalia. However, disturbances in host immunity, modifications in the microbiota, or the use of broad-spectrum antibiotics can lead to excessive growth, inducing candidiasis. Its widespread occurrence is significant in immunocompromised people, such as those with HIV/AIDS, and in patients acquiring immunosuppressive antidotes. The pathogenesis of candidiasis encompasses adherence of candida to host cells, followed by aggression and tissue deterioration. Candida has virulence factors, comprising adhesins and enzymes, adding to its proficiency to elude the host immune response. The yeast-to-hypha metamorphosis is critical for tissue aggression, additionally underlining the complicatedness of its pathogenic mechanism. Clinical indications of candidiasis fluctuate based on the site of the disorder. Oral candidiasis is illustrated as white, adherent patches on the mucosal surfaces, while genital candidiasis is demonstrated as vulvovaginal irritation and discharge. Systemic candidiasis can lead to harsh, life-threatening infections, particularly in immunocompromised patients. Diagnosis depends on clinical assessment, microbiological culture, and more recently, molecular strategies.

Key Words: Candidiasis, Fungal disease, epidemiology, pathogenesis, Clinical Indication

INTRODUCTION

Out of more than 100'000 known fungal species, only about 300 cause disease in humans (Hamza et al., 2022). Our body temperature may provide a protective thermal barrier against the majority of species that grow best at ambient temperature (Hernandez & Martinez, 2018). The most common pathogens are *Candida*, *Aspergillus*, *Cryptococcus*, and *Pneumocystis* spp. causing more than 90% of reported deaths due to fungal disease (Schmiedel & Zimmerli, 2016). The top ten fungal infections are responsible for at least as many deaths as tuberculosis or malaria yet, on a global scale, fungal diseases are neglected (Banerjee et al., 2021). This is reflected by the lack of initiatives by the World Health Organization and the paucity of national surveillance programs. Although current trends show an overall increase in invasive fungal diseases (IFDs), their incidence is likely to be underestimated. IFDs are associated with high morbidity and mortality. Their diagnosis is challenging and their timely treatment often depends on a high level of clinical suspicion. Therefore, this review aims to give an overview of the current epidemiology, clinical presentation, diagnosis, and management of the four most common IFDs: invasive candidiasis, aspergillosis, cryptococcosis, and *Pneumocystis* pneumonia. Every year, *Candida*, *Aspergillus*, *Cryptococcus*, and *Pneumocystis* infect an estimated two million individuals worldwide (Denning, 2024)

Most are immunocompromised or critically ill. *Candida* is the most common fungal pathogen of the critically ill and of recipients of transplanted abdominal organs. In high-risk haemato-oncological patients, in contrast, the introduction of antifungal prophylaxis with fluconazole and later with mold-active posaconazole has led to a remarkable reduction of invasive candidiasis. It is likely to have a

similar effect on invasive aspergillosis. Invasive aspergillosis remains the dominant invasive fungal disease (IFD) of haemato-oncological patients and solid organ transplant recipients and is increasingly found in individuals with exacerbated chronic obstructive pulmonary disease on corticosteroids. In the developed world, owing to antiretroviral therapy *Pneumocystis pneumonia* and cryptococcosis have become rare in patients with human immunodeficiency virus (HIV) and are mainly found in solid-organ transplant recipients or immunocompromised patients (Teekaput et al., 2023). In the developing world, cryptococcosis remains a common and highly lethal disease of HIV-positive individuals with invasive candidiasis and invasive aspergillosis, timely diagnosis is the principal challenge.

The clinical presentation is nonspecific and current diagnostic tests lack sensitivity and specificity. The combination of several tests improves sensitivity, but not specificity. Standardized polymerase chain-reaction-based assays may be promising tools for more rapid and specific diagnosis of candidiasis and invasive aspergillosis (Felix et al., 2023). Nevertheless, initiation of treatment is often based solely on clinical suspicion. Empirical therapy, however, may lead to over-treatment of patients without IFD or it may miss its target in the case of resistance. Despite the success of antifungal prophylaxis in reducing the incidence of IFDs in haemato-oncological patients, there are a considerable number of breakthrough infections demonstrating not only fungal resistance but also the emergence of rare and often lethal fungal pathogens. Knowledge of the local epidemiology and antifungal resistance is therefore pivotal. Current trial-based guidelines leave major gaps in identifying those most at risk, who may benefit from prophylaxis. Ongoing searches for disease-associated genetic polymorphisms may contribute to the establishment of individual risk profiles and targeted prophylaxis (Schmiedel & Zimmerli, 2016).

In recent years DNA sequence-based methods have helped to confirm taxonomic relationships within the genus and have been used to confirm that both sexual and nonsexual *Candida* species are ascomycetes (Hagen et al., 2023). Molecular methods have shown that many of the medically important *Candida* species belong to a phylogenetic subgroup known as the CTG clade, a group of largely commensal yeast species that translate CTG as serine instead of leucine. Most women suffer from vulvovaginal candidiasis (VVC) at least once in life, with as many as 8% experiencing regular recurrent infections. VVC is primarily caused by *Candida albicans*, while *C. glabrata* is the second most common cause of this infection (kan et al., 2023). *Candida* species are an important component of the normal flora of the human oral cavity, and if given the opportunity, these can overgrow and cause oropharyngeal candidiasis (OPC). The introduction of highly active antiretroviral therapy (HAART) in the mid-1990s has led to a marked decrease in the incidence of OPC in HIV-infected individuals (Gonçalves et al., 2016). In general, the majority of *C. albicans* isolates are fully susceptible to all major classes of antifungal agents, including azoles, echinocandins, and polyenes. *Candida parapsilosis* is frequently isolated from physical surfaces in the hospital environment, making it unique among *Candida* species. The remaining *Candida* species associated with human disease are only rarely detected, and therefore relatively little is known about the etiology or the epidemiology of the diseases they cause (Moran et al., 2011).

Microbes are very small living things that the human eye is not capable of seeing but are only visible under a microscope. *Candida* is a fungal contamination in the body as it is a type of yeast (Tamo., 2020). Candidiasis is called the yeast contagion, due to the infecting agent a yeast, *Candida*. *Candida albicans* are pathogens that are of much significance and are present everywhere and mostly reside alongside plentiful bacteria near the mouth, gastrointestinal tract, and vagina. Wilkinson was the first person to describe candidiasis in 1849. Candidiasis of the mouth is termed as Thrush. Those areas of the skin where there is very little ventilation and are uncommonly wet develop infection of the skin called Cutaneous candidiasis (Pappas & Bergamo., 2007). When Fungi invade inside the body, enter the bloodstream, and spread everywhere inside it is called deep Candidiasis. Treatment methodologies are different for each case. Natural prevention is mandatory to save yourself from being a victim of the disease (Qadir et al., 2020).

The most prevalent type of *Candida*, *Candida albicans*, can cause invasive candidiasis, a serious fungal illness linked to health care. However, the incidence of these organisms varies greatly depending on geographic region. Invasive candidiasis can cause anything from mildly symptomatic

candidaemia to fulminant sepsis, which has a death rate of more than 70% (Bow et al., 2010). Common commensal organisms in the skin and gut microbiota are *Candida* spp., and invasive illness is encouraged by changes in the cutaneous and gastrointestinal barriers (e.g., gastrointestinal perforation).

Developing early intervention tactics and vaccine candidates has benefited greatly from a clearer understanding of the genetic basis of host susceptibility, host immunological response, and unique virulence features of *Candida* spp. The development of quick molecular diagnostics could enhance the capacity to act quickly and possibly lower mortality in cases with invasive candidiasis. Early diagnosis is difficult but essential to effective care (Pappas et al., 2020). Furthermore some species of fungi can also affect the human by effecting food while the only way to secure food from fungal contamination is to use preservatives. There is big risk that fungus can effect perishable foods e.g., meat, so that's why to improve the shelf life of this meat -18C temperature is required to save it from spoilage (Samad et al., 2024). Secondly, preservatives also have ability to stop the microbial growth (Talib et al., 2024). The purpose of this review is to explore the management and techniques against the fungal infection especially candidiasis. Moreover this review also explains candidiasis comprehensively.

OUTBREAK OF CANDIDIASIS

Zhu et al. (2020) described that *Candida auris* is a multidrug-resistant yeast that has emerged in healthcare facilities worldwide; however, little is known about identification methods, patient colonization, environmental survival, spread, and drug resistance. Colonization on both biotic (patients) and abiotic (health care objects) surfaces, along with travel, appears to be the major factors for the spread of this pathogen across the globe. In this investigation, we present laboratory findings from an ongoing *C. auris* outbreak in New York (NY) from August 2016 through 2018. A total of 540 clinical isolates, 11, 0.5 patient surveillance specimens, and 3,672 environmental surveillance samples were analyzed. Laboratory methods included matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) for yeast isolate identification, real-time PCR for rapid surveillance sample screening, culture on selective/nonselective media for recovery of *C. auris* and other yeasts from surveillance samples, antifungal susceptibility testing to determine the *C. auris* resistance profile, and Sanger sequencing of the internal transcribed spacer (ITS) and D1/D2 regions of the ribosomal gene for *C. auris* genotyping. Results included (a) identification and confirmation of *C. auris* in 413 clinical isolates and 931 patient surveillance isolate as well as identification of 277 clinical cases and 350 colonized cases from 151 healthcare facilities, including 59 hospitals, 92 nursing homes, 1 long-term acute care hospital (LTACH), and 2 hospices, (b) successful utilization of an in-house developed *C. auris* real-time PCR assay for the rapid screening of patient and environmental surveillance samples, (c) demonstration of relatively heavier colonization of *C. auris* in nares than in the axilla/groin, and (d) predominance of the South Asia clade I with intrinsic resistance to fluconazole and elevated MIC to voriconazole (81%), amphotericin B (61%), flucytosine (5FC) (3%), and echinocandins (1%). These findings reflect greater regional prevalence and incidence of *C. auris* and the deployment of better detection tools in an unprecedented outbreak.

SIGNS AND SYMPTOMS

Odds et al. (1988) discussed that among 106 women harboring yeasts in the vagina and with other causes of genital pathology excluded, there was a statistically significant association between numbers of yeasts recovered semi-quantitatively from vaginal swabs and symptoms of pruritus and signs of abnormal vaginal discharge but no association between yeast numbers and other individual symptoms or signs of vaginal candidosis, including patients' subjective assessment of abnormal vaginal discharge. The presence of yeasts detectable by direct microscopic examination was statistically associated with pruritus, discharge, and vaginitis. There was no relationship between the number of vaginal yeasts and histories of antibiotic or oral contraceptive usage or the stage of the menstrual cycle. Distributions of *Candida* species and *Candida albicans* biotypes were not statistically related to any symptoms, signs, or other factors. The results of this study suggest that vaginal pathology caused by *Candida* species may be related to the quantity of the fungus in the

vagina and that only pruritus and objectively assessed vaginal discharge are firm clinical indicators of Candida infection. (Sobel, J.2005) demonstrated that genital candidiasis is a worldwide problem affecting millions of women. It is not a traditional STI but has a complex etiology in which genetic, biological, and behavioural factors interact to cause symptomatic or asymptomatic infection. Clinical signs and symptoms are nonspecific and diagnosis is often difficult, with a tendency to both under-diagnosis and over-diagnosis. Simple, reliable, and rapid diagnostic tests remain elusive. Management has been simplified by classifying infection into uncomplicated (90%) and complicated (10%) diseases. Uncomplicated candidiasis is effectively treated with short-course antifungal therapy regardless of the route of administration. In contrast, complicated disease requires more prolonged therapy, including long-term weekly fluconazole for recurrent disease. *Candida albicans* resistant to azoles remains extremely rare, but vaginitis caused by *C. glabrata* responds less well to azoles and is best treated with topical boric acid or 17% flucytosine (Mendling., 2015). The single most important challenge to successful treatment of genital candidiasis remains the availability of a rapid, inexpensive, reliable point-of-care test to increase diagnostic accuracy.

EPIDEMIOLOGY OF CANDIDIASIS

Pfaller, M. (1995) described that the increase in infections due to *Candida* over the past decade is significant. This is particularly true for hospitalized patients where the rate of blood-stream infection due to *Candida* spp. has increased by almost 500% over the decade of the 1980s. A significant excess mortality and a prolonged length of stay in the hospital accompany this increase. This trend continued into the 1990s when in the US *Candida* spp. remains the fourth most common blood-stream pathogen, accounting for 8% of all hospital-acquired blood-stream infections (Giri & Kindo., 2012). Notably, more than one-third of candidal bloodstream infections are caused by species other than *C. albicans*. The majority of these infections arise from an endogenous focus of colonization; however, the documentation of nosocomial transmission or ‘cross-infection’ and the recognition of resistance to antifungal agents pose new and significant problems. Recent studies indicate that *Candida* may be isolated from the hands of 15–54% of healthcare workers in the intensive care unit setting and that the strain of *Candida* carried on the hands may be shared by infected patients (Barac et al., 2020). These studies are facilitated by molecular typing and careful epidemiological investigation and suggest that cross-infection is an important and preventable feature of candidal blood-stream infection. Both endogenous and exogenous sources of infection are now well-documented and such information should help direct measures to prevent infections in high-risk individuals. (Yaper, N.2014) demonstrated that the number of immunosuppressive patients has increased significantly in recent years. These patients are at risk for opportunistic infections, especially fungal infections. Candidiasis is one of the most frequent fungal infections determined in these immunosuppressive patients and its epidemiology has changed over the last two decades. Recently, new antifungal agents and new therapy strategies such as antifungal prophylaxis, secondary prophylaxis, and preemptive therapy have come into use (Groll & Tragiannidis., 2009). These changes resulted in the alteration of *Candida* species causing invasive infections. The incidence of *Candida albicans* was decreased in many countries, especially among patients with immunosuppressive disorders, while the incidence of species other than *C. albicans* was increased (Ruhnke., 2006).

DIAGNOSIS OF CANDIDIASIS

Ellepola Arjuna et al. (2005) recounted that invasive candidiasis is associated with high morbidity and mortality. Clinical diagnosis is complicated by a lack of specific clinical signs and symptoms of the disease. Laboratory diagnosis is also complex because circulating antibodies to *Candida* species may occur in normal individuals as the result of commensal colonization of mucosal surfaces thereby reducing the usefulness of antibody detection for the diagnosis of this disease. In addition, *Candida* species antigens are often rapidly cleared from circulation so antigen detection tests often lack the desired level of sensitivity. Microbiological confirmation is difficult because blood cultures can be negative in up to 50% of autopsy-proven cases of deep-seated candidiasis or may only become positive late in the infection (Barantsevich & Barantsevich., 2022). Positive cultures from urine or mucosal surfaces do not necessarily indicate invasive disease although can occur during systemic

infection. Furthermore, differences in the virulence and the susceptibility of the various *Candida* species to antifungal drugs make identification at the species level important for clinical management. Newer molecular biological tests have generated interest but are not yet standardized or readily available in most clinical laboratory settings nor have they been validated in large clinical trials. Laboratory surveillance of at-risk patients could result in earlier initiation of antifungal therapy if sensitive and specific diagnostic tests, which are also cost-effective, become available (Pfaller & Castanheira., 2016). This review will compare diagnostic tests currently in use as well as those under development by describing their assets and limitations for the diagnosis of invasive candidiasis. (Martins et al., 2014) discussed that the most prevalent opportunistic yeast infection is candidiasis. Although other species of *Candida* as well as other microbes are implicated in this complex fungal illness, *Candida albicans* remains the most common. Over the last 20 years, aberrant overgrowth in the respiratory, gastrointestinal, and urinary tracts has been reported in both immunocompromised patients and healthy individuals. Since a yeast infection can be caused by a wide range of circumstances, candidiasis is an excellent illustration of a multifactorial condition (Ciurea et al., 2020). Numerous research have been conducted on this topic because of the sharp rise in the occurrence of these illnesses. Treatment and, more importantly, prevention of those consequences have been the center of attention lately. A candidiasis diagnosis might be very difficult to make. The best "treatment" is prevention, far more so than using antifungal medications to eradicate the yeast. The daily regimen can offer strength protection in several ways that need to be taken into account (Martins et al., 2014). Once the infection is established, though, a therapeutic approach is required, thus other options should be investigated.

PREVENTIVE MEASURES FOR CANDIDIASIS

True. (2020) described that invasive candida infections are the most important cause of nosocomial infections in intensive care units and risky groups such as immunosuppressed patients. These infections lead to undesirable consequences such as increased morbidity and mortality in patients, prolongation of hospital stay, and increased hospital costs. In recent years, the incidence of non-*albicans* *Candida* spp. has increased. Unfortunately, some of these species are naturally resistant to first-line antifungals. In addition, biofilm formation on the central venous catheter and invasive devices may cause treatment failure. The age of the patients, co-morbid diseases, the units where they are treated, the antibiotics and antifungals that are used for the treatment, and invasive devices are risk factors for invasive candida infections (Pieterse ., 2020) Some of these risk factors can be reduced by the behaviour of healthcare workers. The most important goal is to take precautions before the occurrence of invasive candida infections. Infection control measures to prevent hospital transmission of candida are very important. Compliance with hand hygiene before and after contact with the patient is the most important step to prevent the spreading of *Candida* spp. Observation of maximal barrier precautions during invasive catheterization is another important clause of this aim. Avoiding unnecessary invasive devices, antibiotics, and parenteral nutrition is also important to reduce the colonization of candida (Kabir et al., 2013). Infections caused by *Candida* species have increased dramatically worldwide due to the increase in immunocompromised patients. For the prevention and cure of candidiasis, several strategies have been adopted at the clinical level. *Candida*-infected patients are commonly treated with a variety of antifungal drugs such as fluconazole, amphotericin B, nystatin, and flucytosine. Moreover, early detection and speciation of the fungal agents will play a crucial role in administering appropriate drugs for antifungal therapy. Many modern technologies like MALDI-TOF-MS, real-time PCR, and DNA microarray are being applied for accurate and fast detection of the strains. However, during prolonged use of these drugs, many fungal pathogens become resistant, and antifungal therapy suffers. In this regard, a combination of two or more antifungal drugs is thought to be an alternative to counter the rising drug resistance. Also, many inhibitors of efflux pumps have been designed and tested in different models to effectively treat candidiasis (Holmes et al., 2020). However, most of the synthetic drugs have side effects and biomedicines like antibodies and polysaccharide-peptide conjugates could be better alternatives and safe options to prevent and cure the diseases. Furthermore, the availability of genome sequences of *Candida albicans* and other non-*albicans* strains has made it feasible to analyze the genes for their roles in adherence, penetration, and establishment of diseases. Understanding the

biology of *Candida* species by applying different modern and advanced technologies will help us in preventing and curing the diseases caused by fungal pathogens (Ball et al., 2020).

STRATEGIES FOR CONTROL OF INFECTION

Rodrigues et al. (2016) demonstrated that in recent years, there has been a significant increase in the incidence of human fungal infections. The increase in cases of infection caused by *Candida* species, and the consequent excessive use of antimicrobials, has favoured the emergence of resistance to conventional antifungal agents over the past decades. Consequently, *Candida* infections morbidity and mortality are also increasing. Therefore, new approaches are needed to improve the outcome of patients suffering from *Candida* infections, because it seems unlikely that the established standard treatments will drastically lower the morbidity of mucocutaneous *Candida* infections and the high mortality associated with invasive candidiasis. This review aims to present the latest advances in traditional antifungal therapy and present an overview of novel strategies that are being explored for the treatment of *Candida* infections, with a special focus on combined antifungal agents, antifungal therapies with alternative compounds (plant extracts and essential oils), adjuvant immunotherapy, photodynamic therapy, and laser therapy. (Polke et al., 2015) narrated that only a few *Candida* species, e.g., *Candida albicans*, *Candida glabrata*, *Candida dubliniensis*, and *Candida parapsilosis*, are successful colonizers of a human host. Under certain circumstances, these species can cause infections ranging from superficial to life-threatening disseminated candidiasis. The success of *C. albicans*, the most prevalent and best studied *Candida* species, as both commensal and human pathogen depends on its genetic, biochemical, and morphological flexibility which facilitates adaptation to a wide range of host niches. In addition, the formation of biofilms provides additional protection from adverse environmental conditions. Furthermore, in many host niches, *Candida* cells coexist with members of the human microbiome. The resulting fungal–bacterial interactions have a major influence on the success of *C. albicans* as commensal and also influence disease development and outcomes (Braunsdorf & LeibundGut-Landmann., 2018).

CLINICAL TRIALS FOR THE TREATMENT OF CANDIDIASIS

PETERSEN et al. (1970) they discussed that twelve patients with chronic mucocutaneous candidiasis were assigned by random allocation to a 6-month course of treatment with ketoconazole or placebo in a double-blind trial. All six recipients of ketoconazole had remission of symptoms and virtually complete regression of mucosal, skin, and nail lesions, whereas only two of the six receiving placebo had even temporary mucosal clearing, and none had improvement of skin or nail disease. The clinical outcome in the ketoconazole-treated group was significantly more favourable ($p < 0.001$) than in the placebo-treated group. The six patients receiving a placebo in the controlled trial were then treated with ketoconazole in an open trial, and all responded favourably. Hepatitis, probably drug-induced, developed in one patient after 6 months of treatment but proved to be mild and reversible. Oral ketoconazole is an effective treatment for chronic mucocutaneous candidiasis (Horsburgh & Kirkpatrick 1983).

MANAGEMENT FOR CONTROL OF INFECTION

Stevens et al. (2014) narrated that guidelines for the management of patients with invasive candidiasis and mucosal candidiasis were prepared by an Expert Panel of the Infectious Diseases Society of America. These updated guidelines replace the previous guidelines published in the 15 January 2004 issue of *Clinical Infectious Diseases* and are intended for use by healthcare providers who care for patients who either have or are at risk of these infections. Since 2004, several new antifungal agents have become available, and several new studies have been published relating to the treatment of candidemia, other forms of invasive candidiasis, and mucosal disease, including oropharyngeal and oesophageal candidiasis. There are also recent prospective data on the prevention of invasive candidiasis in high-risk neonates and adults and on the empiric treatment of suspected invasive candidiasis in adults. (Cruciani & Serpillon., 2008) they narrated that the epidemiology of *Candida* infection in intensive care units (ICUs) and the management strategies for such infections in non-neutropenic intensive care patients are discussed in this review. *Candida* species are one of the leading causes of nosocomial bloodstream infections and a significant cause of morbidity in

patients admitted to the ICU. Prophylactic, pre-emptive, and empiric treatment strategies for Candida infections have been explored in ICU patients. Routine prophylaxis should not be administered to the whole population of ICU patients, because the concerns about the selection of azole-resistant Candida strains or the induction of resistance are justified. Treatment of fungal infections is now possible with newer antifungal agents, including newer azoles (e.g., voriconazole, posaconazole) and echinocandins (e.g., micafungin, anidulafungin). However, there is a critical need for improvement in the diagnosis of invasive Candida infection to provide clinicians the opportunity to intervene earlier in the disease course (Clancy & Nguyen., 2013).

IMPORTANCE OF CANDIDIASIS DISEASE

Harriott et al. (2011) said that *Candida albicans* is the most prevalent human fungal pathogen, with an ability to inhabit diverse host niches and cause disease in both immunocompetent and immunocompromised individuals. *C. albicans* also readily forms biofilms on indwelling medical devices and mucosal tissues, which serve as an infectious reservoir that is difficult to eradicate and can lead to lethal systemic infections. Biofilm formation occurs within a complex milieu of host factors and other members of the human microbiota. Polymicrobial interactions will probably dictate the cellular and biochemical composition of the biofilm, as well as influence clinically relevant outcomes, such as drug and host resistance and virulence. (Coleman et al.,1998) discussed that *Candida* species other than *C. albicans* have become a significant cause of infection in humans. Several of the more commonly isolated of these species are less susceptible to commonly used azole antifungal drugs, a factor that poses significant difficulties for effective treatment. The modern mycology laboratory has an important role to play in several aspects relating to these organisms, including therapy, detection, identification, and epidemiological analysis. The application of molecular techniques and phylogenetic analysis has led to the identification of a new species of *Candida* associated with mucosal candidiasis in HIV-infected individuals named *Candida dubliniensis*, the clinical significance of which is currently under investigation. Molecular techniques are also being applied to the analysis of determinants involved in the pathogenicity of species such as *Candida glabrata* (Dadar et al., 2018). These approaches should lead to a better understanding of these organisms and their ability to cause disease and should also provide more effective treatment.

CONCLUSION

Many species of *Candida* cause candidiasis, which affects mucosal surfaces and can occasionally result in serious systemic infections, especially in immunocompromised people. It is still a major worldwide health problem. Aspects of candidiasis such as epidemiology, etiology, clinical manifestations, diagnosis, therapy, and preventative strategies are all included in the study. The distribution of *Candida* species has changed over time, and antifungal drug resistance has increased, resulting in changes to the epidemiology of candidiasis. Comprehending the epidemiological patterns is essential for proficient illness management and containment.

Due to the ambiguous clinical presentations and limits of available diagnostic tools, diagnosing candidiasis can be difficult. But improvements in early detection and prompt treatment start are possible because to advances in molecular diagnostics. Antifungal drugs are the mainstay of treatment for candidiasis, while newer drugs may be an option in situations of resistance. In addition, controlling candidiasis requires taking preventative steps including infection control procedures and lowering risk factors. Novel therapeutic strategies, including as immunotherapy, plant extracts and essential oils, and combination antifungal medications, are the subject of emerging study. Moreover, knowledge of the genetic underpinnings of *Candida* species' virulence traits and host vulnerability aids in the creation of focused treatments. To sum up, there are a lot of difficulties in managing candidiasis in clinical settings and with public health. To lessen the impact of candidiasis and enhance patient outcomes, more research must be done in addition to thorough preventative and control measures being put in place.

REFERENCES

1. Ball, B., Langille, M., & Geddes-McAlister, J. (2020). Fun (gi) omics: Advanced and diverse technologies to explore emerging fungal pathogens and define mechanisms of antifungal resistance. *MBio*, 11(5), 10-1128.
2. Banerjee, S., Denning, D. W., & Chakrabarti, A. (2021). One Health aspects & priority roadmap for fungal diseases: A mini-review. *Indian Journal of Medical Research*, 153(3), 311-319.
3. Barac, A., Cevik, M., Colovic, N., Lekovic, D., Stevanovic, G., Micic, J., & Rubino, S. (2020). Investigation of a healthcare-associated *Candida tropicalis* candidiasis cluster in a haematology unit and a systematic review of nosocomial outbreaks. *Mycoses*, 63(4), 326-333.
4. Barantsevich, N., & Barantsevich, E. (2022). Diagnosis and treatment of invasive candidiasis. *Antibiotics*, 11(6), 718.
5. Bow, E. J., Evans, G., Fuller, J., Laverdiere, M., Rotstein, C., Rennie, R., ... & Vinh, D. C. (2010). Canadian clinical practice guidelines for invasive candidiasis in adults. *Canadian Journal of Infectious Diseases and Medical Microbiology*, 21, e122-e150.
6. Braunsdorf, C., & LeibundGut-Landmann, S. (2018). Modulation of the fungal-host interaction by the intra-species diversity of *C. albicans*. *Pathogens*, 7(1), 11.
7. Ciurea, C. N., Kosovski, I. B., Mare, A. D., Toma, F., Pinteas-Simon, I. A., & Man, A. (2020). *Candida* and candidiasis—opportunism versus pathogenicity: a review of the virulence traits. *Microorganisms*, 8(6), 857.
8. Clancy, C. J., & Nguyen, M. H. (2013). Finding the “missing 50%” of invasive candidiasis: how nonculture diagnostics will improve understanding of disease spectrum and transform patient care. *Clinical infectious diseases*, 56(9), 1284-1292.
9. Coleman, D. C., Rinaldi, M. G., Haynes, K. A., Rex, J. H., Summerbell, R. C., Anaissie, E. J., ... & Sullivan, D. J. (1998). Importance of *Candida* species other than *Candida albicans* as opportunistic pathogens. *Medical Mycology*, 36, 156-165.
10. Cruciani, M., & Serpelloni, G. (2008). Management of *Candida* infections in the adult intensive care unit. *Expert Opinion on Pharmacotherapy*, 9(2), 175-191.
11. Dadar, M., Tiwari, R., Karthik, K., Chakraborty, S., Shahali, Y., & Dhama, K. (2018). *Candida albicans*-Biology, molecular characterization, pathogenicity, and advances in diagnosis and control—An update. *Microbial pathogenesis*, 117, 128-138.
12. Denning, D. W. (2024). Global incidence and mortality of severe fungal disease. *The Lancet Infectious Diseases*.
13. Ellepola Arjuna, N. B., & Morrison Christine, J. (2005). Laboratory diagnosis of invasive candidiasis. *Journal of microbiology*, 43(spc1), 65-84.
14. Felix, G. N., de Freitas, V. L., da Silva Junior, A. R., Magri, M. M., Rossi, F., Sejas, O. N., ... & Del Negro, G. M. (2023). Performance of a Real-Time PCR Assay for the Detection of Five *Candida* Species in Blood Samples from ICU Patients at Risk of Candidemia. *Journal of Fungi*, 9(6), 635.
15. Giri, S., & Kindo, A. J. (2012). A review of *Candida* species causing blood stream infection. *Indian journal of medical microbiology*, 30(3), 270-278.
16. Gonçalves, B., Ferreira, C., Alves, C. T., Henriques, M., Azeredo, J., & Silva, S. (2016). Vulvovaginal candidiasis: Epidemiology, microbiology and risk factors. *Critical reviews in microbiology*, 42(6), 905-927.
17. Groll, A. H., & Tragiannidis, A. (2009, July). Recent advances in antifungal prevention and treatment. In *Seminars in hematology* (Vol. 46, No. 3, pp. 212-229). WB Saunders.
18. Hagen, F., Walther, G., Houbraken, J., Scott, J., Summerbell, R., & Boekhout, T. (2023). Molecular Taxonomy. In *Diagnosis and Treatment of Fungal Infections* (pp. 31-60). Cham: Springer International Publishing.
19. Hamza, M., Samad, A., Ahmer, A., Muazzam, A., Tariq, S., Hussain, K., & Waqas, M. U. (2022, July). Overview of Aspergillosis a fungal disease in poultry and its effect on Poultry Business. In *Proceedings of the 1st International Conference on Social Science (ICSS)* (Vol. 1, No. 1, pp. 81-87).
20. Harriott, M. M., & Noverr, M. C. (2011). Importance of *Candida*-bacterial polymicrobial biofilms in disease. *Trends in microbiology*, 19(11), 557-563.

21. Hernandez, H., & Martinez, L. R. (2018). Relationship of environmental disturbances and the infectious potential of fungi. *Microbiology*, 164(3), 233-241.
22. Holmes, A. R., Cardno, T. S., Strouse, J. J., Ivnitiski-Steele, I., Keniya, M. V., Lackovic, K., ... & Cannon, R. D. (2016). Targeting efflux pumps to overcome antifungal drug resistance. *Future Medicinal Chemistry*, 8(12), 1485-1501.
23. Horsburgh Jr, C. R., & Kirkpatrick, C. H. (1983). Long-term therapy of chronic mucocutaneous candidiasis with ketoconazole: experience with twenty-one patients. *The American journal of medicine*, 74(1), 23-29.
24. Kabir, M. A., & Ahmad, Z. (2013). Candida infections and their prevention. *International Scholarly Research Notices*, 2013.
25. Kan, S., Song, N., Pang, Q., Mei, H., Zheng, H., Li, D., ... & Liu, W. (2023). In Vitro Antifungal Activity of Azoles and Other Antifungal Agents Against Pathogenic Yeasts from Vulvovaginal Candidiasis in China. *Mycopathologia*, 188(1), 99-109.
26. Martins, N., Ferreira, I. C., Barros, L., Silva, S., & Henriques, M. (2014). Candidiasis: predisposing factors, prevention, diagnosis and alternative treatment. *Mycopathologia*, 177, 223-240.
27. Mendling, W. (2015). Guideline: vulvovaginal candidosis (AWMF 015/072), S2k (excluding chronic mucocutaneous candidosis). *Mycoses*, 58, 1-15.
28. Moran, G., Coleman, D., & Sullivan, D. (2011). An introduction to the medically important *Candida* species. *Candida and candidiasis*, 9-25.
29. Odds, F. C., Webster, C. E., Mayuranathan, P., & Simmons, P. D. (1988). *Candida* concentrations in the vagina and their association with signs and symptoms of vaginal candidosis. *Journal of medical and veterinary mycology*, 26(5), 277-283.
30. Pappas, P. G., & Bergamo, B. (2007). Superficial and mucosal fungal infections. In *Diagnosis of Fungal Infections* (pp. 153-170). CRC Press.
31. Pappas, P. G., Lionakis, M. S., Arendrup, M. C., Ostrosky-Zeichner, L., & Kullberg, B. J. (2018). Invasive candidiasis. *Nature Reviews Disease Primers*, 4(1), 1-20.
32. PETERSEN, E. A., ALLING, D. W., & KIRKPATRICK, C. H. (1980). Treatment of chronic mucocutaneous candidiasis with ketoconazole: a controlled clinical trial. *Annals of Internal Medicine*, 93(6), 791-795.
33. Pfaller, M. A. (1995). Epidemiology of candidiasis. *Journal of Hospital Infection*, 30, 329-338.
34. Pfaller, M. A., & Castanheira, M. (2016). Nosocomial candidiasis: antifungal stewardship and the importance of rapid diagnosis. *Medical mycology*, 54(1), 1-22.
35. Pieterse, A. M. (2020). Antibiotic exposure as a risk factor for secondary *Candida* infections in a private hospital intensive care unit (Doctoral dissertation, North-West University (South Africa)).
36. Polke, M., Hube, B., & Jacobsen, I. D. (2015). *Candida* survival strategies. *Advances in Applied Microbiology*, 91, 139-235.
37. Qadir, M. I., & Asif, H. (2020). An overview to candidiasis-a *Candida* infection. *International Journal of Advanced Research in Microbiology and Immunology*, 2(1), 31-33.
38. Rodrigues, M. E., Silva, S., Azeredo, J., & Henriques, M. (2016). Novel strategies to fight *Candida* species infection. *Critical reviews in microbiology*, 42(4), 594-606.
39. Ruhnke, M. (2006). Epidemiology of *Candida albicans* infections and role of non-*Candida albicans* yeasts. *Current drug targets*, 7(4), 495-504.
40. Samad, A., Alam, A. M. M., Kumari, S., Hossain, M. J., Lee, E. Y., Hwang, Y. H., & Joo, S. T. (2024). Modern Concepts of Restructured Meat Production and Market Opportunities. *Food Science of Animal Resources*, 44(2), 284-298.
41. Schmiedel, Y., & Zimmerli, S. (2016). Common invasive fungal diseases: an overview of invasive candidiasis, aspergillosis, cryptococcosis, and *Pneumocystis pneumonia*. *Swiss medical weekly*, 146, w14281.
42. Sobel, J. D. (2005). Genital candidiasis. *Medicine*, 33(10), 62-65.
43. Stevens, D. L., Bisno, A. L., Chambers, H. F., Dellinger, E. P., Goldstein, E. J., Gorbach, S. L., ... & Wade, J. C. (2014). Practice guidelines for the diagnosis and management of

- skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clinical infectious diseases*, 59(2), e10-e52.
44. Talib, Ambreen, Abdul Samad, Md Jakir Hossain, Ayesha Muazzam, Bushra Anwar, Rameen Atique, Young-Hwa Hwang, and Seon-Tea Joo. "Modern trends and techniques for food preservation." *Food and Life* 2024, no. 1 (2024): 19-32.
 45. Tamo, S. B. (2020). Candida infections: clinical features, diagnosis and treatment. *Infect. Dis. Clin. Microbiol*, 2, 91-103.
 46. Teekaput, C., Yasri, S., & Chaiwarith, R. (2023). Cryptococcal meningitis: differences between patients with and without HIV-infection. *Pathogens*, 12(3), 427.
 47. Ture, Z., & Alp, E. (2018). Infection control measures to prevent hospital transmission of candida. *Hospital Practice*, 46(5), 253-257.
 48. Yapar, N. (2014). Epidemiology and risk factors for invasive candidiasis. *Therapeutics and clinical risk management*, 95-105.
 49. Zhu, Y., O'Brien, B., Leach, L., Clarke, A., Bates, M., Adams, E., ... & Chaturvedi, S. (2020). Laboratory analysis of an outbreak of *Candida auris* in New York from 2016 to 2018: impact and lessons learned. *Journal of clinical microbiology*, 58(4), 10-1128.