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Review Article

Unraveling Candidiasis: Pathogenic Mechanisms and the Spectrum of Infections

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A B S T R A C T

Worldwide, fungal infections—especially those brought on by Candida species like Candida albicanscause a considerable amount of illness and mortality, particularly in people with weakened immune systems. Through morphological changes, adhesion, invasion, biofilm formation, and the release of hydrolytic enzymes, Candida albicans demonstrates complicated pathogenicity. Its pathogenicity is increased by its capacity to change phenotypes and adjust to shifting environmental conditions. Drug-resistant strains such as Candida auris are becoming more and more common, which emphasizes the urgent need for efficient therapies and a better comprehension of the mechanisms behind fungal pathogenicity. Oral and vulvovaginal infections are among the many forms of candidiasis caused by Candida species, especially Candida albicans. Whereas vulvovaginal candidiasis affects the female reproductive system, oral candidiasis presents as thrush. Hormonal fluctuations, antibiotic usage, and immunosuppression are risk factors. Antifungal drugs are one kind of treatment; recurrence is frequent in some groups. Candida species, including Candida albicans, are responsible for a number of illnesses, including invasive candidiasis, gastrointestinal infections, and skin infections. Immunocompromised people are more susceptible to these infections, which can range from localized skin and gastrointestinal colonization to potentially fatal systemic infections. Antifungal drugs such as amphotericin B, azoles, and echinocandins are used in treatment.

Key word: Candidiasis,Candida albicans, Virulence, Oral candidiasis, Vulvovaginal Candidiasis, Cutaneous candidiasis

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INTRODUCTION

Fungal infections cause around 1.7 million fatalities globally each year largely in immunocompromised patients with two or more chronic conditions (1). An estimated 1.5 to 5 million species make up the fungus kingdom, one of the biggest eukaryotic kingdoms. Their life cycles, metabolisms, morphogenesis, and ecologies, which include commensalism, parasitism, and mutualism with several living things make them a varied group. With their ability to break down a wide variety of biopolymers and other biological compounds in both dead and living hosts, as well as to synthesize a wide range of classes of biomolecules for human and other eukaryotic consumption, they are found in all temperature zones of the planet and have a profound and wide-ranging impact on the ecosystem (2).Currently, only approximately 15 of the more than 150 species of Candida are known to be common human pathogens. These include Candida albicans, Candida glabrata, Candida tropicalis, Candida parapsilosis, Candida krusei, Candida guilliermondii, Candida lusitaniae, Candida dubliniensis, Candida pelliculosa, Candida kefyr, Candida lipolytica, Candida famata, Candida inconspicua, Candida rugosa, and Candida norvegenis (3). The vast majority of fungal infections in humans are caused by members of the Candida species, which include the drug-resistant Candida glabrata, the new global public health threat Candida auris, the most common cause of opportunistic infections, Candida albicans, and other emerging species like Candida tropicalis, Candida parapsilosis, and Candida krusei (4).

Candida albicans is the primary cause of candidiasis, which has steadily raised morbidity and mortality rates over the past few decades. Invasive fungal diseases claim the lives of about 1.5 million people annually worldwide. Hospital-acquired opportunistic fungal infections, most frequently caused by

Candida species, account for over 80% of fungal sepsis-related mortality. Even Nevertheless, since 2009, the incidence rate of Candida auris has been steadily rising. In healthy people, Candida albicans often coexist as non-pathogenic symbiotic fungi that primarily colonize human mucosa surfaces, such as the skin, gastrointestinal tract, urogenital tract, and oral and pharyngeal areas.Candida albicans may quickly change from non-pathogenic to pathogenic fungus when the human immune system is weakened, leading to superficial or deep candidiasis, which includes candidemia and thrush. Every year, invasive candidiasis affects more than 1,565,000 people worldwide and causes almost 1,000,000 deaths. As a result, candidiasis becomes a global issue that demands careful attention because of its effects on human health (5, 6).According to taxonomy, Candida is a member of the genus Candida (7), order Cryptococcales, family Cryptococcaceae, class Blastomycetes, and phylum Ascomycetes. Mucosal candidiasis, cutaneous candidiasis, onychomycosis, and systemic candidiasis are among the various forms of candidiasis(8).

CANDIDA ALBICANS

There are several morphological forms of Candida albicans, including hyphae, pseudohyphae, andblastospores. Yeast, another name for blastospores, divide asexually by budding.

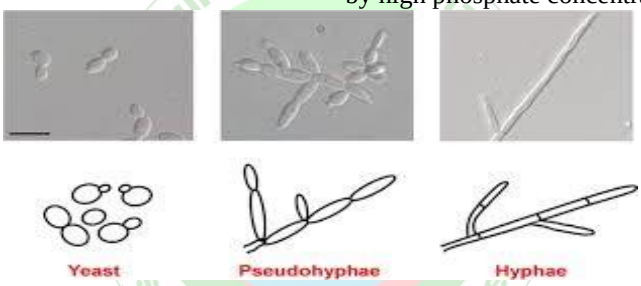

Figure 1: Morphologies of human fungal pathogens.

Carbohydrates make up 80–90% of Candida albicans' cell wall. The primary polysaccharides of the cell wall are represented by three fundamental components: (i) glucose polymers with branched b-1,3 and b-1,6 links (b-glucans); (ii) N-Acetyl-D-glucosamine (GlcNAc) polymers without branches that have b-1,4 bonds (chitin); and (iii) mannose (mannan) polymers covalently bound to proteins (glyco[manno]-proteins). Furthermore, proteins (6–25%) and trace quantities of fat (1–7%) are found in cell walls (12).

PHATHOGENICITY

Numerous virulence variables and fitness features enhance Candida albicans' capacity to infect such a wide variety of host habitats. Many characteristics are regarded as virulence factors (13), such as the morphological change from yeast to hyphal forms, the production of adhesins and invasins on the cell surface, thigmotropism, biofilm development, phenotypic switching, and the release of hydrolytic enzymes. Because of its features, Candida albicans is a useful model for studying the hyphal growth, pathogenicity, and virulence of fungi(14). One group of virulence factors initiates colonization, or the start of an infection, whereas another group aids in the illness's dissemination (fig.2) (9).

New cell material forms on the blastospore's surface during that procedure. The new bud, which develops from a tiny, carefully chosen blastospore, is often found far from the location of a birth scar, after which the development phase starts. Following the completion of the growth phase, the cells split, with the daughter cell forming a partition to separate from the parent cell [9]. Due to their tendency to become polarized, elongated, and end-to-end connected, pseudohyphae and hyphae are frequently referred to as the "filamentous" morphologies. Pseudohyphal cells often feature constrictions at the septal junctions and are ellipsoidal, meaning that their breadth is greater in the middle than at the ends. On the other hand, hyphal cells often exhibit equal width, parallel edges, and genuine septa devoid of constrictions. In contrast to pseudohyphae, hyphal cells feature cell-to-cell communication channels in their septa (fig.1) (10).

Numerous environmental factors can cause morphological transition from yeast to filamentous forms. With the addition of serum and a growth temperature of 37°C, hyphae may be consistently produced from unbudded yeast cells. Hyphae and pseudohyphae are also induced by a culture temperature over 35°C and a pH of neutral; little variations in these parameters affect the developmental result. According to recent reports, homogeneous pseudophyphal shape is induced by high phosphate concentrations (up to 600 mM) (11).

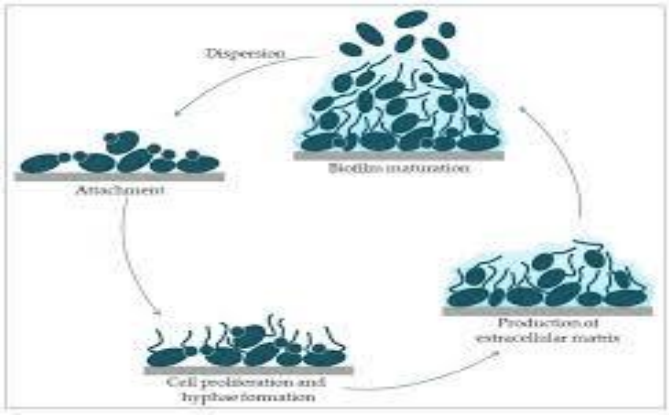

Figure 2: Phases of C. albicans biofilm formation

The most crucial stage in tissue penetration is the yeasts' capacity to stick to the epithelium, which is a powerful inducer of the hyphal metamorphosis. Compared to yeasts, C. albicans hyphae have a greater epithelial adherence. The inability to form hyphae prevents more aggressive species of Candida albicans from adhering to epithelium. Fungal invasion causes the physical barrier to break down, allowing Candida albicans to spread to vascular tissues underneath and eventually to distant organs. Although C. albicans's ability to convert into the hypha form is a crucial virulence factor for

both phagocyte attachment and epithelial penetration, the yeast form is necessary for the establishment of systemic infection and dissemination (7).

Yeast forms may easily spread throughout the host tissues, whereas filamentous shapes with a higher capacity for adhesion aid in the invasion of the host tissues. Adhesion proteins found on the fungal cell surface and immobilized ligands (cadherins, integrins, or other microorganisms) are the first ways that Candida species bind to the host cell. The adherence is followed by fungal cell invasion of the tissue. Pathogenicity is defined as the invasion and destruction of the epithelium. Depending on the kind of host cell, it may happen by either endocytosis or active penetration(15). Calcium channel-mediated extracellular calcium intake controls hyphae thigmotropism in Candida albicans. This process is crucial for increasing the pathogenicity of Candida species. Thigmotropism helps disseminate in the host tissue and form a biofilm on abiotic surfaces (9). Body warmth and EC contact are two of the many cues that cause hyphal development. After the invasion process begins, Candida albicans develops early virulence factors. The expression of the GPI-linked cell surface proteins Hwp1 and the HSP70 family member Als3 or Ssa1 are two examples. The transcription factor Bcr1, which controls the production of biofilms, controls Hwp1, which is necessary for mucosal pathogenicity and serves as a substrate for epithelial transglutaminases (16).

It has been suggested that the "phenotypic switching" phenomenon adds to the organism's flexibility. Although phenotypic flipping is also seen in clinical isolates, studies on the phenomenon have concentrated on a single switch system, the white–opaque switch, which describes the colony color and shape of each switch phenotype present in a single strain (WO-1). The variations in colony phenotype are accompanied by modifications in tissue affinities, antigen expression, and adhesion characteristics (17).The host invasion process is aided by a number of hydrolytic enzymes that Candida albicans secretes, including lipases, phospholipases, hemolysins, and proteases. Factors linked to Candida albicans pathogenicity include secreted aspartic proteases (Saps), which are expressed by a family of genes (SAP1 to SAP10). Hemoglobin, albumin, keratin, collagen, laminin, fibronectin, mucin, and almost all immunoglobulins are among the human proteins they break down. Sap9 and Sap10 stay attached to the cell surface and play a significant role in C. albicans' ability to produce biofilms (1).

Yeasts are no exception to the complicated, multifaceted process of aging, which is accompanied by a variety of alterations in a cell's physiology. The host's age may also have an impact on how an infection turns out. Cell functions change with age, and for humans, this also means that the immune system is compromised. In addition to having several underlying illnesses, elderly persons are more likely to get opportunistic infections overall. The mortality rate for candidemia is greater in this age group. Fitness qualities also include metabolic flexibility, strong food acquisition mechanisms, a robust stress response machine, and quick adaptability to changes in ambient pH(13).

TYPE OF CANDIDIASIS

Candida species are found in around 50% of the population in this form and typically live as commensal organisms as part of an individual's regular microflora. However, Candida species frequently turn pathogenic if the immune system is weakened or the equilibrium of the natural flora is upset (19). Clinical signs of Candida sp. infections range from minor mucocutaneous conditions to invasive infections that impact many organs (9).

ORAL CANDIDIASIS

Fungal proliferation and invasion of superficial tissues are hallmarks of oral candidiasis (OC), sometimes known as "thrush," which includes infections of the tongue and other oral mucosal sites (20). Between 30 and 60 percent of healthy people are thought to have Candida species in their mouths. Instead of being a pathogenic condition, commensal colonization is the way that the great majority of these microbes exist (21). An estimated 45% of neonates, 45%–65% of healthy children, 30%–45% of healthy adults, 50%–65% of people wearing removable dentures, 65%–88% of patients in acute and long-term care facilities, 90% of patients with acute leukemia receiving chemotherapy, and 95% of patients with HIV have been found to have C albicans isolated from the oral cavity (22).

Thrush, also known as oral candidiasis, is characterized by large white pseudomembranes made of fibrin, fungal hyphae, and desquamated epithelial cells. The tongue, periodontal tissues, hard and soft palate, labial and buccal mucosa, and oropharynx are all affected by these white spots (22). For most clinical types of oral candidiasis, the tongue dorsum is the initial site of infection, serving as the main reservoir for oral Candida carriage. One such condition is oropharyngeal candidiasis, which frequently develops as an extension of OC and is defined by invasion of the oropharyngeal cell lining. For OC, there are several clinical manifestations and categorization schemes (23).

Classification of Oral Candidosis

Primary oral candidosis (Group I)

- Acute
- Pseudomembranous
- Erythematous
- Chronic
- Erythematous
- Pseudomembranous
- Hyperplastic
- Nodular
- Plaque-like
- Candida-associated lesions
- Angular cheilitis
- Denture stomatitis
- Median rhomboid glossitis
- Keratinized primary lesions superinfected with Candida
- Leukoplakia
- Lichen planus
- Lupus erythematosus.
- Secondary oral candidoses (Group II)
- Oral manifestations of Systemic mucocutaneous(24-26)

Although the exact cause of this infection is still unknown, a number of systemic (such as immunosuppression or endocrine disorders) and local (such as decreased salivary flow, wearing dentures, or eating a high-sugar diet) factors

have been linked to an overabundance of Candida species, with Candida albicans being the species most frequently linked to oral lesions. Numerous predisposing variables change the environment to encourage the growth of Candida, causing it to go from commensal to pathogenic, which can manifest as clinical symptoms of oral candidiasis (27). Oral infections caused by fungi (Candida spp.) have become more common recently. Additionally, it is a result of the growing use of antibiotics and immunodeficiency disorders linked to HIV infection. A few investigations have revealed that candidiasis is an asymptomatic oral colonization that can lead to widespread infections or the advancement of oral lesions (28). One risk factor was One risk factor for oral candidiasis is impaired salivary gland function. Histidine-rich polypeptides, lactoferrin, sialoperoxidase, lysozyme, and some anti-candida antibodies are examples of salivary antimicrobial proteins that interact with the oral mucosa to inhibit the development of candida (24). By potentially inhibiting phagocytosis and cellular immunity, medications such inhaled steroids have been demonstrated to raise the risk of oral candidiasis (22).

Because a rupture in the oral epithelium provides a portal of entry for Candida, prolonged denture use, poor denture cleanliness, and mucosal trauma are significant local variables that lead to the development of OC. People who smoke cigarettes are known to have much greater oral candidal carriage levels, which puts them at a higher risk of acquiring OC (20). Systemic variables like advanced age might make people more susceptible to candidiasis because of reduced or immature immunity. Iron is the most prevalent critical micronutrient deficit among the nutritional deficiency states that has been linked to candida colonization. Long-term use of systemic medications, such as immune-suppressants, broad-spectrum antibiotics, and medications with xerostomic side effects, might change the local oral flora, damage the mucosal surface, or decrease salivary flow, all of which can foster the growth of candida (22, 29).

Among other diagnostic techniques, the microscopic detection of Candida in oral samples and/or its isolation in culture can be used to validate the diagnosis of any of the types of oral candidiasis, which is primarily a clinical one (30). Although a fungal smear or culture, as well as sometimes an oral biopsy, may be necessary, the diagnosis is typically made clinically. To rule out leukoplakia and keep an eye out for dysplastic characteristics, it's critical to follow up on chronic hyperplastic candidiasis, including serial biopsy if needed (31).In patients with persistent erythematous candidiasis, oral candidiasis is treated by controlling the recognized risk factors, such as proper denture cleaning with 1% sodium hypochlorite preparations or chlorhexidine solution (0.2% w/v) (32). Itraconazole, fluconazole, and ketoconazole are examples of systemic medications that have been widely utilized to treat oral candidiasis. The growing number of reports of resistance to these medications, especially fluconazole, is a serious issue, especially for individuals who need ongoing or repeated treatment. The rising incidence of more resistant strains of Candida albicans requires the development of prevention and management strategies (26).

Antifungal medications used topically to the interior of the oral cavity for seven to fourteen days are often used to treat mild to severe oral infections brought on by Candida albicans. These include the antifungal medications nystatin, miconazole, and clotrimazole. The most widely used antifungal medication for severe infections is fluconazole, which may be administered intravenously or orally [9].For simple cases of oral candidiasis, topical antifungals and good oral hygiene are typically sufficient. The majority of oral candidiasis infections in denture wearers can be avoided with regular dental and oral hygiene practices and routine oral exams (22). The majority of people with oral candidosis have a favorable prognosis, and the condition is curable. For patients with oral candidosis to get effective therapy, a comprehensive medical history and the right workup are essential. Maintenance of effective oral hygiene and removal of risk factors can lower the chance of acquiring oral candidosis (23). Washing the mouth with water or mouthwash after using an inhaler may help people who take inhaled corticosteroids lower their chance of acquiring thrush (29).

VULVOVAGINAL CANDIDIASIS

A very frequent mucosal infection of the lower female reproductive tract (FRT), vulvovaginal candidiasis (VVC) is mostly brought on by the polymorphic opportunistic fungus Candida albicans. Candida albicans is a frequent asymptomatic colonizer of the vaginal lumen and a part of the typical human microbiota [33]. 70% to 75% of all women in their reproductive years will experience at least one episode of vaginitis, which is the second most common cause of vaginitis globally after bacterial vaginosis (BV) (34). A Candida yeast infection results in vulvovaginal candidiasis, a symptomatic vaginitis (inflammation of the vulva and/or vagina). According to reports, 10% of women are asymptomatic (35). Many women with vaginal yeast colonization have varied degrees of vaginal itching, which is the symptom most unique to VVC, even though the majority are asymptomatic. Additionally, some individuals may have increased discharge, dyspareunia, dysuria, vaginal discomfort, or edema (36). The most frequent cause of VVC, Candida albicans, is a natural component of the vaginal microbiota. C. glabrata, isolated in 7–16% of cases, is the second most prevalent pathogen found in women with VVC. When the equilibrium between the invading microbes and the host is upset, clinical inflammation results (37). Although there has been evidence linking the pathophysiology of vulvovaginal candidiasis to neutrophil infiltration against C. albicans infection, the role of the neutrophil infiltrate is still up for debate. Therefore, understanding the pathogenic mechanism of vulvovaginal candidiasis and the vaginal immune responses against Candida species infection may help manage this prevalentillness(38).

Although symptomatic recurrences are prevalent throughout pregnancy, the prevalence of clinical episodes' peaks in the third trimester. There is evidence of a yeast cytosol receptor or binding mechanism for female reproductive hormones, and estrogens increase the avidity of vaginal epithelial cells for Candida adhesion. Estrogens also promote the transformation of yeast into mycelial cells (39). Broad-spectrum antibiotic usage is also thought to result in Candida colonization, which has been linked to a reduction in Lactobacillus colonization. This is most likely due to the disruption of epithelial binding sites (40). Many reasons contribute to the high prevalence of

VVC in women with diabetes, including increased vaginal mucosa from high glycogen accumulation in vaginal tissue (41).

Culture has remained the gold standard for diagnosing vaginal fungal infections, despite the fact that clinical suspicion and microscopy have been employed to identify VVC for decades. All three diagnostic techniques are useful tools, but they have limitations. Yeast cultures can cause a delay in diagnosis and therapy, because both clinical diagnosis and microscopy have low sensitivity (36). Various therapy methods are currently available to treat VVC, depending on whether the presentation is simple or complex. Prescription oral dose forms, over-the-counter topical treatments, and vaginal suppositories are the major modes of treatment (33). Treatment for complex VVC instances must be ongoing.Local azoles can be applied daily for at least one week, or oral fluconazole can be administered three times with a 72-hour interval (37) Because of their quick symptom alleviation, few side effects, and limited systemic absorption, intravaginal creams and suppositories are frequently used to treat VVC (32).

GASTROINTESTINAL CANDIDIASIS

With colonization rates between 30% and 70% in healthy people, Candida species are common gastrointestinal (GI) tract occupants (42). In the mouth cavity and other areas of the gastrointestinal system, it may exist as a temporary or persistent colonizer. This fungus, which resembles yeast, is thought to be an opportunistic microbe that can harm every area of the digestive system (43). Each person has a different gut flora, which is generally consistent in healthy individuals. It varies from person to person based on age and dietary or hygiene practices, and its composition varies along the digestive system (9). The yeast A common, but erratic, component of the human gut mycobiota, Candida albicans is found all throughout the world. However, little is currently known about how Candida albicans interacts with the gut microbiota in general and the enteric yeast population in particular (44). A colonization of the gut itself can be regarded as typical as Candida albicans colonizes the majority of the human population. On the other hand, intestinal diseases have occasionally been linked to C. albicans colonization (45).

Gastrointestinal candidiasis, which is an infection of the stomach and small and large intestines, can result from persistence in the GI tract. More significantly, GI colonization and infection put patients at risk for systemic candidiasis, often referred to as candidiasis of endogenous origin, because of GI tract expansion (42). Disseminated candidosis requires at least one of four things to occur: direct penetration of blood capillaries and arteries by epithelial cells (ECs), indirect translocation of C. albicans cells that are phagocytosed by host immune cells, direct damage to mucosal barriers, and dissemination from fungal biofilms (9).

CUTANEOUS CANDIDIASIS

There have been several reports of Candida colonization in GI tract illness patients. Candida colonization was more common in patients with various GI tract disorders than in control subjects (46).Patients who have intra-abdominal infections and risk factors for candidiasis, such as necrotizing pancreatitis, anastomotic leaking, or recent abdominal surgery, should be treated with antifungal medication. Controlling the source of infections should also be incorporated. Fluconazole is an alternate treatment, and echinocandin is advised as the first line of treatment. If Candida species that are resistant to azoles are not the source of the illness, then azoles are employed (9).Bacteria, viruses, fungus, and archaea all live in the skin, which is a dynamic and diverse ecosystem. Numerous illnesses, ranging from milder and more superficial clinical presentations to life-threatening conditions in immunocompromised people, are caused by Candida albicans. To become pathogenic and infiltrate host tissues, this microbe may develop in a variety of morphological forms, including unicellular budding yeast, pseudohyphae, and genuine hyphae (47). About 1% of all outpatient visits and 7% of all inpatient admissions to dermatological clinics are caused by cutaneous candidiasis, a prevalent condition that affects people of all ages (48).

Although cutaneous infections have a low fatality rate, they can lead to systemic and invasive candidiasis, which has a 25–50% mortality risk, if they are left undiagnosed or untreated for an extended period of time (49). In people who are susceptible, cutaneous candidiasis typically occurs as a secondary infection of the skin and nails (body folds). It manifests as a chronic or subacute infection. Localized or widespread disease involvement of the skin or nails is possible. Diaper rash, intertrigo candidiasis, Candida folliculitis, otomycosis, onychia, and paronychia are among the several forms of cutaneous candidiasis (50).The clinical manifestations of mucocutaneous candidiasis are diverse. The patient's age group, the place of the injury, and other risk factors all play a role. Intertriginous zones, such as the submammary, inguinal folds, intergluteal creases, and pannus folds in overweight patients, are the most often involved locations (9). Numerous topical antifungal medications, including clotrimazole, econazole, miconazole, ketoconazole, ciclopiroxolamine, sulconazole, and oxiconazole, can be used to treat cutaneous candidiasis infections. The preferred medication for pulse treatment is oral itraconazole (50).

INVASIVE CANDIDIASIS

Invasive Candida Infections (ICIs), like many infectious illnesses, have experienced a number of noteworthy clinical and epidemiological alterations in recent decades. Furthermore, the incidence rate of ICIs has increased in recent decades (from around 2.18 cases per 100,000 people annually in the 1990s to 3.22 instances in the most recent decade for candidemia (51). Invasive candidiasis (IC) is a dangerous, frequently lethal, opportunistic infection linked to healthcare. IC includes deep-seated tissue candidiasis and candidemia, which are brought on by the spread of Candida species to ordinarily sterile areas (52). Patients may have a systemic Candida infection, particularly those who are very sick. A reservoir of organisms is provided by local infection, which poses an invasion risk and is especially important in patient groups with impaired immune systems (53). These infections usually happen when Candida organisms penetrate the compromised intestinal barrier, which can happen as a result of a number of things, including chemotherapy for cancer, surgery, bacterial toxins, and problems of local vascular perfusion (endogenous pathway) (51). Bloodstream infections brought on by Candida species are referred to as

invasive candidiasis. usually happen after crossing the intestinal barrier. Overgrowth of Candida albicans can weaken immunity, resulting in invasive candidiasis and other opportunistic infections in different organs (9). Deep-seated infections including osteomyelitis (bone infection), peritonitis (tissue covering the inner abdominal wall and organs), and intra-abdominal abscess are all considered forms of invasive candidiasis (IC), which can significantly affect any organ (15).

Both an increase in the fungal load and surface modification, whether it be to the skin or mucous membranes, are often necessary for the development of invasive illness. Every component of the immune system reacts as IC develops. While deficits in T-helper 17 cells have been linked to an increase in colonization and invasive illness, cell-mediated immunity via lymphocytes protects mucosal disease (54). Neutrophils and monocytes harm and eliminate blastospores and pseudohyphae. Thus, there is a high risk of developing candidemia and other types of IC in individuals with severe neutrophil dysfunction or leukopenia. Optimal opsonization and intracellular death of the organism depend on complement and immunoglobulins, and deficiencies in any of these substances may be linked to more complex or refractory illnesses (55). Since deep-seated candidiasis, or IC, is linked to a number of syndromes affecting organs such the liver, spleen, heart, eyes, peritoneum, kidney, bone, meninges, and lungs, with or without concurrent candidemia, it is a more challenging clinical entity to detect (56).Treatment recommendations have been influenced by the move away from Candida albicans, which is largely sensitive to all systemic antifungals, and toward species like Candida glabrata and Candida krusei that are more commonly resistant or tolerant to fluconazole (57). Three kinds of antifungal medications are available for the treatment of IC: amphotericin B-based regimens, azoles (fluconazole, voriconazole, itraconazole, posaconazole, isavuconazole), and echinocandins(anidulafungin, caspofugin or micafungin) (58).

CONCLUSION

The pathogenicity of Candida albicans, a primary cause of candidiasis, is attributed to its capacity to transition between filamentous and yeast forms. Its ability to penetrate tissues and cause infections, especially in immunocompromised persons, is made possible by virulence factors such adhesion, biofilm formation, and enzyme synthesis, underscoring its significance on world health.Common commensals, Candida species, including Candida albicans, can turn harmful in specific situations, resulting in illnesses including vulvovaginal and oral candidiasis. These infections range from minor mucosal problems to serious illnesses. Clinical examination and culture are required for diagnosis, and antifungal medications are available for both care and prevention. Although Candida species are frequent invaders of the gastrointestinal tract and skin, they can also result in illnesses such invasive candidiasis, cutaneous candidiasis, and gastrointestinal candidiasis. These infections are opportunistic, especially in those with weakened immune systems. Clinical evaluation is necessary for diagnosis, and antifungal medications are used for treatment. As resistance in some Candida species increases, management techniques change.

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