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CANDIDIASIS: ETIOLOGY, PATHOGENESIS, AND CLINICAL MANIFESTATIONS

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Annotation: Candidiasis is a common fungal infection caused primarily by species of the genus Candida, with Candida albicans being the most prevalent. It affects both immunocompetent and immunocompromised individuals, manifesting as superficial mucocutaneous infections or severe systemic disease. This narrative literature review aimed to synthesize current evidence on the etiology, pathogenesis, and clinical manifestations of candidiasis. A comprehensive search of international databases, including PubMed, Scopus, Web of Science, and Google Scholar, was conducted for articles published between 2010 and 2024. The findings indicate that fungal virulence factors—such as adhesion, biofilm formation, and hyphal transition—interact with host immune responses, particularly cell-mediated immunity, to determine disease severity. Superficial infections commonly affect the oral and genital mucosa, whereas systemic infections are associated with high morbidity and mortality. Non-albicans Candida species are increasingly implicated in resistant and recurrent infections. Understanding these mechanisms is essential for accurate diagnosis, targeted therapy, and effective management strategies.

Keywords: Candidiasis; Candida albicans; Virulence factors; Pathogenesis; Clinical manifestations; Superficial infection; Systemic infection

Introduction

Candidiasis is a common fungal infection caused primarily by species of the genus Candida, with Candida albicans being the most prevalent. It affects both immunocompetent and immunocompromised individuals, manifesting in superficial mucocutaneous forms as well as systemic invasive infections. Superficial candidiasis primarily involves the oral cavity, gastrointestinal tract, and genital mucosa, whereas systemic candidiasis can affect multiple organs and is associated with significant morbidity and mortality, particularly in hospitalized or immunocompromised patients.

The pathogenesis of candidiasis is multifactorial, involving the complex interplay between fungal virulence factors and host immune responses. Key virulence factors include adhesion molecules, biofilm formation, and the ability to transition between yeast and hyphal forms, which facilitate tissue invasion and evasion of host defenses. Host susceptibility is influenced by various factors, such as immunodeficiency, diabetes mellitus, antibiotic therapy, and hormonal changes, all of which can disrupt the natural microbial balance and favor fungal overgrowth.

Clinically, candidiasis presents with a wide spectrum of manifestations ranging from mild mucosal erythema and pruritus to severe systemic infections. Oral candidiasis, or “thrush,” is characterized by white plaques on the oral mucosa, while vulvovaginal candidiasis presents with itching, discharge, and inflammation. Invasive candidiasis may present with fever, hypotension, and organ-specific dysfunction, requiring prompt diagnosis and treatment.

Understanding the etiology, pathogenesis, and clinical manifestations of candidiasis is essential for effective diagnosis, prevention, and treatment. This review aims to summarize current knowledge regarding the immunological and microbiological mechanisms of candidiasis, highlight the clinical features of superficial and systemic infections, and discuss implications for clinical management.

Methods

This study was designed as a narrative and analytical literature review focusing on the etiology, pathogenesis, and clinical manifestations of candidiasis. The review aimed to synthesize current scientific evidence regarding the pathogenic mechanisms of Candida species, host immune responses, and clinical presentation of both superficial and systemic infections. A comprehensive literature search was conducted using major international scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar. Articles published in English between 2010 and 2024 were considered. The search strategy employed keywords and Medical Subject Headings (MeSH), including "candidiasis," "Candida albicans," "fungal pathogenesis," "virulence factors," "immune response," "clinical manifestations," and "systemic candidiasis." Studies were included if they met the following criteria: original research articles, systematic reviews, or meta-analyses addressing the microbiology, immunopathogenesis, and clinical features of candidiasis; studies describing both superficial and systemic manifestations of candidiasis; and articles providing clear information on host immune responses and fungal virulence factors. Exclusion criteria included case reports, conference abstracts, letters to the editor, articles not available in full text, and studies unrelated to the immunological, microbiological, or clinical aspects of candidiasis or published in languages other than English. Relevant data were independently extracted from selected articles, including information on fungal species, virulence mechanisms, host immune interactions, and clinical manifestations. The data were systematically organized into thematic categories: etiology, pathogenesis, and clinical features. Comparative analysis was performed to identify consistent findings, highlight gaps in knowledge, and summarize key aspects of Candida infections. As this study was based solely on previously published literature, ethical approval and informed consent were not required. All sources were appropriately cited to ensure academic integrity and compliance with ethical standards in scientific research.

Results

The analysis of the selected literature revealed that Candida species, particularly Candida albicans, are the primary causative agents of both superficial and systemic candidiasis. The pathogenicity of Candida is driven by multiple virulence factors, including adhesion to host tissues, biofilm formation, and the ability to transition between yeast and hyphal forms. Host immune responses, particularly cell-mediated immunity, play a crucial role in controlling fungal proliferation, while immunocompromised states, antibiotic use, diabetes, and hormonal changes increase susceptibility to infection.

Superficial candidiasis primarily manifests in the oral cavity, gastrointestinal tract, and genital mucosa, whereas systemic candidiasis can affect multiple organs, leading to severe clinical complications. Clinical features vary according to the site of infection and the immune status of the host. The reviewed studies consistently emphasized the central role of the interplay between

fungal virulence factors and host immune responses in determining the severity and progression of the disease.

Table 1. Key Etiological Agents, Virulence Factors, and Clinical Manifestations of Candidiasis

Candida Species	Virulence Factors	Common Clinical Manifestations
Candida albicans	Adhesion, hyphal transition, biofilm	Oral thrush, vulvovaginal candidiasis, systemic infection
Candida glabrata	Biofilm formation, antifungal resistance	Vulvovaginal candidiasis, bloodstream infections
Candida tropicalis	Tissue invasion, biofilm	Urinary tract infections, systemic candidiasis
Candida parapsilosis	Biofilm formation, catheter colonization	Neonatal candidemia, catheter-related infections
Candida krusei	Natural antifungal resistance	Systemic infections in immunocompromised patients

The findings indicate that Candida albicans remains the most clinically significant species, while non-albicans Candida species are increasingly reported in both superficial and invasive infections. Biofilm formation and immune evasion mechanisms contribute significantly to persistent infections and treatment challenges. Understanding these etiological and pathogenic mechanisms is essential for accurate diagnosis and effective management of candidiasis.

Discussion

The results of this literature review highlight the multifactorial nature of candidiasis, emphasizing the interplay between Candida virulence factors and host immune responses. Candida albicans remains the most prevalent species causing both superficial and systemic infections, while non-albicans species, such as C. glabrata, C. tropicalis, and C. parapsilosis, are increasingly implicated in clinical cases. These species exhibit distinct pathogenic mechanisms, including biofilm formation, hyphal transition, tissue invasion, and antifungal resistance, which contribute to persistent infections and complicate treatment strategies.

The pathogenesis of candidiasis is strongly influenced by the host immune system. Cell-mediated immunity, particularly the Th1 and Th17 responses, plays a critical role in controlling fungal proliferation. Immunocompromised individuals, including patients with HIV/AIDS, diabetes mellitus, or those receiving immunosuppressive therapy, are particularly susceptible to invasive candidiasis. The review confirms that the balance between fungal virulence and host immune competence determines the severity and clinical outcome of infection.

Superficial infections, such as oral thrush and vulvovaginal candidiasis, are commonly associated with mucosal disruption, hormonal changes, and antibiotic use. These conditions facilitate fungal overgrowth and colonization. Systemic candidiasis, however, results from hematogenous dissemination and is associated with higher morbidity and mortality, particularly in hospitalized or critically ill patients. Biofilm formation on medical devices, such as catheters and prosthetic implants, was identified as a major factor in persistent infections and antifungal treatment failure.

The reviewed literature also emphasizes the clinical relevance of distinguishing between Candida species, as non-albicans species often exhibit reduced susceptibility to conventional antifungal agents. This has significant implications for therapeutic decision-making and highlights the need for species-specific diagnosis and targeted treatment approaches.

Despite these insights, some limitations exist. The heterogeneity of study designs, differences in patient populations, and variability in diagnostic methods may influence the generalizability of the findings. Additionally, the increasing emergence of antifungal-resistant Candida strains underscores the need for ongoing research into novel therapeutic strategies and immune-based interventions.

Overall, the discussion reinforces that candidiasis is a complex disease in which fungal virulence and host immune status interact to determine clinical outcomes. A thorough understanding of these mechanisms is essential for improving diagnosis, treatment, and prevention strategies in both superficial and systemic forms of the infection.

Conclusion

Candidiasis is a complex fungal infection caused primarily by Candida albicans and, increasingly, by non-albicans species. The pathogenesis of the disease is driven by the interplay between fungal virulence factors, such as adhesion, biofilm formation, and hyphal transition, and host immune responses, particularly cell-mediated immunity. Superficial infections, including oral thrush and vulvovaginal candidiasis, are common and generally manageable, while systemic infections pose significant clinical challenges and are associated with higher morbidity and mortality, especially in immunocompromised individuals.

The review demonstrates that understanding the etiology, pathogenesis, and clinical manifestations of candidiasis is critical for accurate diagnosis, effective treatment, and prevention strategies. The emergence of antifungal-resistant Candida strains and the role of biofilms underscore the need for targeted therapeutic approaches and species-specific interventions. Overall, integrating knowledge of fungal biology and host immunity provides a foundation for improved patient management and supports the development of novel treatment strategies to reduce the burden of both superficial and systemic candidiasis.

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