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Candidiasis: Host Immune Response

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Abstract

This transition is heavily reliant on an impressive repertoire of virulence factors, most notably cell surface adhesins, proteolytic enzymes, morphologic switching, and the development of drug resistance. In the oral cavity, the co-adhesion of *C. albicans* with bacteria is crucial for its persistence, and a wide range of synergistic interactions with various oral species were described to enhance colonization in the host. As a frequent colonizer of the oral mucosa, the host immune response in the oral cavity is oriented toward a more tolerogenic state and, therefore, local innate immune defenses play a central role in maintaining Candida in its commensal state. Specifically, in addition to preventing Candida adherence to epithelial cells, saliva is enriched with anti-candida peptides, considered to be part of the host innate immunity. The T helper 17 (Th17)-type adaptive immune response is mainly involved in mucosal host defenses, controlling initial growth of Candida and inhibiting subsequent tissue invasion. Animal models, most notably the mouse model of oropharyngeal candidiasis and the rat model of denture stomatitis, are instrumental in our understanding of Candida virulence factors and the factors leading to host susceptibility to infections.

Keywords: Candida albicans; Immune response

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Introduction

Oral candidiasis (OC), commonly referred to as “thrush” encompasses infections of the tongue and other oral mucosal sites and is characterized by fungal overgrowth and invasion of superficial tissues [1]. The colloquial term “thrush” refers to the resemblance of the white flecks present in some forms of candidiasis with the breast of the bird of the same name.

In 1771, Rosen von Rosenstein defined an invasive form of thrush; however, in 1839, Lange beck was credited with first documenting a fungus associated with thrush in a patient with typhoid fever. In 1846, Berg presented observations that thrush was caused by a fungus, which was classified in 1847 by the French mycologist, Charles Philippe Robin as Oidium albicans, the first use of albicans which means “to whiten”. In 1923, Berkhout reclassified the fungus under the current genus Candida, a name derived from the Latin word toga candida, referring to the white toga (robe) worn by Roman senators of the ancient Roman republic, a probable reference to the whitish colonies on agar or white lesions [2]. However, it was not until 1954 that the binomial Candida albicans was formally endorsed. In the 1980s, there was a clear surge of interest in oral candidal infections largely due to the increased incidence of OC because of the escalation in the acquired immune deficiency syndrome (AIDS) epidemic, and, to date, OC remains the most common oral opportunistic infection in human immunodeficiency virus (HIV)-positive individuals and in individuals with weakened immune systems. Vaginal candidiasis presents with genital itching, burning, and a white "cottage cheese-like" discharge from the vagina. The penis is less commonly affected by a yeast infection and may present with an itchy rash. Yeast infections may spread to other parts of the body resulting in fevers along with other symptoms and become invasive rarely [3]. Oral candidiasis is one of the most common fungal infections, affecting the oral mucosa. The yeast Candida albicans causes these lesions. Candida albicans are among the components of normal oral micro flora, and around 30% to 50% of people carry this organism. The rate of carriage increases with the age of the patient. Candida albicans are recovered from 60% of dentate patients' mouths over the age of 60 years [4].

There are many forms of Candida species, which present in the oral cavity. Species of oral Candida include *C. albicans*, *C. glabrata*, *C. krusei*, *C. parapsilosis*, *C. pseudotropicalis*, *C. stellatoidea*, and *C. tropicalis*. Oral candidiasis may present as a variety of disease entities in both normal hosts and the immunocompromised. These include hyperplastic or atrophic (denture) candidiasis, pseudomembranous candidiasis (thrush), linear gingival erythema, median rhomboid glossitis, and angular cheilitis [5].

C. albicans is by far the main causative agent of OC accounting for up to 95% of cases. Although considered a pathogen, *C. albicans* is a ubiquitous commensal organism that commonly colonizes the oral mucosa and is readily isolated from the oral cavities of healthy individuals. In fact, up to 80% of the general population are asymptomatic carriers, and simple carriage does not predictably lead to infection. Similar to the oral cavity, *C. albicans* asymptomatically colonizes the gastrointestinal tract and reproductive tract of healthy individuals where its proliferation at these various sites is controlled by the host immune system, and other members of the microbiota. Uniquely, C. albicans is a highly versatile commensal organism that is well adapted to its human host, and any changes in the host microenvironment that favor its proliferation provide this pathogen with the opportunity to invade virtually any site [6].

Histopathology

Candidiasis sections present spongiotic changes in the epidermis with irregular acanthosis, mild spongiosis, and inflammatory changes. The distinguishing feature of the superficial epidermis is the presence of neutrophils in the stratum corneum and upper layers of the epidermis. A small collection of neutrophils (spongiform pustulation) may form, which resembles impetigo or psoriasis [7].

Candida albicans is a pathogenic yeast-like fungus that grows partly as yeast and partly as elongated cells resembling hyphae which form pseudo mycelium. Candida albicans can be identified from other candida species by growth characteristics, sugar assimilation, and fermentation tests. It produces germ tubes within two hours when incubated in human serum at 37-degree Celsius [8].

Host Immune Response

As *C. albicans* is a frequent commensal colonizer of the oral mucosa, the host immune response in the oral cavity is oriented toward a more tolerogenic state, to avoid an excessive inflammatory response that could be damaging to the oral tissue. However, the polysaccharide-rich cell wall makes *C. albicans* highly immunogenic and easily recognized by the host pattern recognition receptors (PRRs). Epithelial cells, upon Candida recognition, induce the secretion of several antimicrobial peptides with a direct killing effect on the fungal cell, which aid in controlling local colonization. Secretion of proinflammatory mediators such as cytokines and chemokines (G-CSF, GM-CSF, IL-1α, IL1β, IL-6, IL-8, and CCL5) by epithelial cells, signal the recruitment of phagocytic cells, including neutrophils, macrophages and dendritic cells (DCs) to the site of infection. Several comprehensive reviews on *C. albicans* and host cells and the immune response during *C. albicans* mucosal infection were recently published. Here, we focus on highlighting oral local innate immune defenses that play a crucial role in maintaining Candida in its commensal state in the oral cavity. Antibiotic usage is commonly associated with candidiasis. Cancer cytotoxic chemotherapy may result in fungemia caused by Candida albicans, which develop from fungal translocation through compromised mucosal barriers. Fungal commensals in the upper and lower GI tract can transform into opportunistic pathogens due to changes in endogenous bacterial population size or composition, as well as changes in the host environment [9]. Vaginal colonization increases in diabetes mellitus, pregnancy, and the use of oral contraceptives. Oral candidiasis is very closely associated with HIV patients. More than 90% of patients with HIV present with candidiasis.

Oral epithelial cells are the first line of defense against *C. albicans*, functioning as a physical barrier. However, the constant flow of saliva also acts as an important mechanical clearance mechanism by preventing adherence of Candida to the epithelial cells and, therefore, saliva secretion is important for maintenance of the commensal state of *C. albicans* in the mouth. Additionally, saliva is highly enriched in antimicrobial peptides (AMPs), which play a vital role in innate immunity and defense against microbial colonization. While most AMPs are produced by several cell types, the histatins, a family of 12 histidine-rich cationic peptides with broad-spectrum antimicrobial activity, are unique in that they are exclusively produced by the salivary glands. Among the members, histatin-5 specifically possesses potent antifungal activity, primarily against *C. albicans*. The anticandidal mechanism of histatin-5 is described to involve binding to specific receptors on the fungal cell wall and intracellular uptake where it targets the mitochondria, disrupting cell homeostasis [10].

Conclusion

The most important histological finding in this study is that an accumulation of inflammatory cells mainly composed of neutrophils was observed under the oral mucosal epithelium in the early process of formation of a pseudomembranous oral candidiasis model in mice. The neutrophil accumulation was formed at the sites into which Candida hyphae penetrated from 24 h after infection, and the area with neutrophils became more enlarged at 48 h after infection, reaching almost to the tip of the invading

The worst condition in the pathological process in experimental oral candidiasis was found 48 h after *C. albicans* inoculation. When the surface of the Candida-inoculated tongues was covered with Candida-hyphae, a dense accumulation of neutrophils was observed under the lesions and homogenates of the tongues contained increased levels of IL-12p70 and IFN-γ. These suggested that local pathological condition of Candida-infected tongues may be affected by neutrophils accumulation and increased levels of some cytokines. Pregnant women have higher chances of colonizing Candida in the vagina during pregnancy; vaginal candidiasis is among the common forms of fungal diseases frequently occurring in pregnant women which may lead to systemic infections in neonates, especially with low birth weight (LBW) and prematurity after delivery.

Intertrigo is a common inflammatory dermatosis affecting opposing skin surfaces that can result from Candida, under the effect of mechanical and environmental factors. It presents with pain and itching, which decreases the quality of life, leading to high morbidity. Predisposing factors, such as obesity, diabetes mellitus, and immunosuppressive conditions, facilitate both the incidence and recurrence of the disease. Candidal interring is

usually treated with topical application of NY statin and azole group antifungals.

Acknowledgement

None

Conflict of Interest

None

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