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Immuno-Struggle of Trichomonas Vaginalis to Live in the Human Body

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Mini Review

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Abstract

Trichomonas vaginalis is a urogenital flagellate, the etiological agent of human trichomoniasis. It is a worldwide curable sexually transmitted infection. It is often passed on by an asymptomatic carrier. Trichomonas induce pro-inflammatory and immunosuppressive cytokines in infected subjects. The parasite has also different immune evasion mechanisms to establish infection.

Keywords: Trichomonas vaginalis; Flagellate; Trichomoniasis; Immune Evasion

Introduction

Trichomonas vaginalis is a flagellated protist belonging to the Parabasalia that causes human trichomoniasis [1]. It is the third most common vaginal infection with an estimated incidence of 248 million new cases per year [2]. The infection occurs in the female and male urogenital tract and humans are the only natural host for the parasite [3]. It is principally spread via unprotected intercourse with an infected partner [1].

In women, the disease is ranging from asymptomatic state to severe inflammation [4]. In infected female patients, symptoms include: vaginal discharge, vulvar pruritis, dysuria, and dyspareunia. Classical green, frothy, foul-smelling discharge occurs in 10% of the women, and the red granular 'strawberry' cervix can be identified by the naked eye in less than 2% of patients [5].

Moreover, re-infection may occur by sexual contact with the man who acts as an asymptomatic carrier [6]. This significant heterogeneity in the clinical manifestations is due to genetic variability among Trichomonas isolates and the host immune response [7].

Complications of T. vaginalis infection can occur in untreated women and include endometritis, infertility [8] and cervical erosion [9]. Other complications caused by T. vaginalis include atypical pelvic inflammatory disease, prolonged carriage of human papilloma virus [10]. It also increases the risk of cervical cancer [11]. Its complications in pregnant women include: post abortion infection, post cesarean infection, low birth weight infants preterm labor and premature rupture of the membranes [12]. Untreated T. vaginalis infection in men could lead to chronic prostatitis, urethral strictures, epididymitis and/or infertility [13]. This infection increases the predisposition to human immunodeficiency virus (HIV) sero-conversion [14].

Host Immune Reaction

Minimal data exist to detail the human immune response to infection with T. vaginalis. The natural infection of T. vaginalis seems to produce immunity that is only partially protective [11]. Infection with T. vaginalis elicits cellular, humoral and secretory immune responses. However, these responses do not protect patients against re-infection, so repeated infections in women are commonly encountered in clinical settings [9]. The Innate immunity is an important first response by the host. Trophozoites attach to different mucosal surfaces of the lower urogenital tract and they are able to survive for long periods of time in the environment despite adverse conditions, such as acidic pH, and a robust immune response [11]. The vaginal epithelial cells are the first physical barrier that produces antimicrobial molecules and cytokines that can recruit other innate immune effector cells [15].

The cell surface lipophosphoglycan triggers parasite-specific immunoglobulin G (IgG), IgA, cytokines, leukotrienes and reactive nitrogen intermediates. It also induces nitricoxide synthase; priming of helper T cells and promotes transmigration of neutrophils across the endothelium [16].

IL-8 is a major chemokine responsible for neutrophil recruitment to sites of tissue insult and aggravation of the vaginal inflammation seen in symptomatic patient. IL-8 produced by monocytes and neutrophils stimulated by live trophozoites or undefined T. vaginalis membrane fragments [7]. These explain the abundance of neutrophils on wet mounts [17].

The host cells produce specific immunoglobulins (IgG and IgA) as a response to parasite antigens. This response may protect target cells from parasite-mediated cytotoxicity. However, there is a little evidence that such antibodies contribute to parasite clearance. Furthermore, persistent T. vaginalis infections suggest that humoral immune responses are not broadly protective [18].

Antibodies and complement components, present in serum and genital fluids of some individuals with trichomoniasis, can bind the surface of T. vaginalis and lyse them. They stimulate the neutrophil respiratory burst facilitating parasite killing via classical and alternative complement pathways [19].

Immune Evasion

Additionally, T. vaginalis has the ability to evade the host's immune system through complement mediated destruction, molecular mimicry [16]. T. vaginalis can coat itself with host plasma proteins. Thus, the host immune system does not recognize the parasite as foreign. This parasite also secretes copious amounts of highly immunogenic soluble antigens in a continuous way to neutralize antibody or cytotoxic T lymphocytes [20].

Moreover, T. vaginalis produces immuno-suppressive cytokines (as IL-10) which causes caspase-mediated apoptosis in T-cells, macrophages and dendritic cells [21]. Cytotoxic effects of macrophages on T. vaginalis have an important role in host innate immunity, but it is reported that T. vaginalis inhibits pro-inflammatory cytokine production in macrophages to evade the immune system [22]. Another evasion way is phenotypic variation mechanism which detect by expression or non-expression of 270kDa protein. T. vaginalis are described as positive or negative P270. T. vaginalis which is positive cannot express protein hence doesn't have the ability of adhesion [23]. The role of the human immune system in trichomoniasis is important in order to develop targeted intervention strategies to control the disease.

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