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Macromolecule Translocation through Apoptotic Leaks and Transcytosis across the Intestinal Mucosa of HIV-infected Patients

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Introduction

HIV, the Human Immunodeficiency Virus, remains a global health challenge, affecting millions of people worldwide. While much progress has been made in understanding the virus and developing antiretroviral therapies, there are still many aspects of HIV infection that require further investigation. One such aspect is the impact of HIV on the intestinal mucosa and its role in macromolecule translocation through apoptotic leaks and transcytosis. With the advent of effective antiretroviral therapy, the life expectancy of PLWH has substantially improved. However, this population now faces new challenges, including an increased risk of developing certain types of cancers. This section introduces the topic and highlights the importance of understanding the evolution of cancer incidence in different periods of antiretroviral treatment [1,2]. The gastrointestinal tract plays a pivotal role in HIV pathogenesis. The gut-associated lymphoid tissue (GALT) houses a substantial portion of the body's immune cells, including CD4+ T cells, which are the primary target of HIV. As a result, the gut mucosa is a major site of HIV replication and immune system damage. This article delves into the mechanisms and consequences of macromolecule translocation across the intestinal mucosa in HIV-infected patients [3].

Description

To comprehend the significance of macromolecule translocation in HIV, it is essential to understand the gut-immune axis. The intestinal mucosa is not only a physical barrier but also a critical site for immune surveillance and regulation. The mucosal immune system is highly specialized and tightly regulated to balance the protection against pathogens and tolerance towards commensal microorganisms. In HIV infection, the virus predominantly targets CD4+ T cells, which are abundantly present in the gut-associated lymphoid tissue. This targeting leads to massive CD4+ T cell depletion, local inflammation, and mucosal damage [4]. The loss of gut mucosal integrity is a hallmark of HIV infection, contributing to chronic immune activation and disease progression. Apoptosis, or programmed cell death, plays a crucial role in maintaining tissue homeostasis and eliminating damaged or infected cells. In the context of HIV infection, apoptosis takes on added significance. Infected CD4+ T cells often undergo apoptosis as a result of viral replication and immune responses. Apoptotic cells are typically cleared by phagocytes, such as macrophages, before they can release cellular debris or contents into the surrounding tissue. However, in the setting of HIV infection, this clearance process may be impaired due to the high volume of apoptotic cells. As a result, apoptotic cells can release their contents, including macromolecules, into the intestinal mucosa [5,6].

Conclusion

The translocation of macromolecules through apoptotic leaks and transcytosis across the intestinal mucosa represents a complex and multifaceted aspect of HIV pathogenesis. It contributes to chronic inflammation, immune activation, and microbial translocation, all of which have adverse effects on the health of HIV-infected individuals. Understanding these mechanisms is crucial for developing novel therapeutic strategies to complement existing antiretroviral therapies and improve the overall well-being of those living with HIV. Ongoing research in this field holds promise for reducing the burden of HIV-related complications and improving the quality of life for affected individuals. Targeting viral entry and transcytosis mechanisms could potentially disrupt HIV's ability to establish and maintain infection in the gut mucosa. These approaches may complement existing antiretroviral therapies.

Acknowledgement

None.

Conflict of Interest

None.

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