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OPINION

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Immunology and the Pathophysiology of HIV/AIDS

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DESCRIPTION

HIV is typically spread through unprotected sexual contact, blood transfusions, hypodermic needles, and mother-to-child transmission. Once the virus is internalized, it multiplies and kills T helper cells, which are necessary for almost all adaptive immune responses.

An initial stage of influenza-like disease is followed by a latent, asymptomatic stage. When the CD4 lymphocyte count drops to under 200 cells per milliliter of blood, the HIV host has advanced to AIDS, a condition marked by a lack of cell-mediated immunity and the ensuing increased susceptibility to opportunistic infections and some cancers.

Immunology

Once the virus has entered the body, it undergoes a phase of high viral replication, which causes an overabundance of virus in the peripheral circulation. Several million viral particles per milliliter of blood are possible during primary infection.

The quantity of circulating CD4+ T lymphocytes significantly decreases in conjunction with this response. The activation of CD8+ T cells, which kill HIV-infected cells, and subsequent antibody generation, or seroconversion, is almost always associated with this acute viremia in patients. Virus levels peak and then begin to drop as CD4+ T cell counts increase, and it is believed that the CD8+ T cell response plays a crucial role in regulating these levels. Despite the fact that it does not completely eradicate the virus, a strong CD8+ T cell response has been associated with a slower disease progression and a better prognosis.

Apoptosis may potentially play a role in the acute phase CD4+ T cell depletion, however HIV-induced cell lysis and the death of infected cells by cytotoxic T cells are the main causes. The repercussions of widespread immunological activation and the progressive loss of the immune system's capacity to produce new T cells during the chronic phase seem to be responsible for the steady decrease in CD4+ T cell counts. The majority of CD4+ T cell loss happens in the early weeks of infection, particularly in the intestinal mucosa, which houses the majority of the lymphocytes found in the body, even if immunodeficiency symptoms do not manifest for years after a person is infected.

Mucosal CD4+ T cells are lost preferentially because they express the C-C chemokine receptor type 5 (CCR5) in greater numbers than CD4+ T cells in the circulation, which are only a small percentage of all CD4+ T cells. During an acute infection, HIV seeks for and kills CD4+ cells that express CCR5. The infection is eventually brought under control by an active immune response, which also starts the clinically latent period. However, CD4+ T lymphocytes in mucosal tissues continue to decline during the illness, Although CD4+ T cells were initially sufficient to fend off life-threatening infections The chronic phase of HIV infection is characterized by ongoing widespread immunological activation. The activity of multiple HIV gene products and the immune system's reaction to continued HIV replication cause immunological activation, which is manifested by immune cells, enhanced state of activation and the release of proinflammatory cytokines. Depletion of mucosal CD4+ T cells during the acute stage of illness is another factor that contributes to the collapse of the immune surveillance system of the mucosal barrier.

Since the mucosal immune system normally regulates the normal flora of the gut in a healthy person, this causes the immune system to be exposed to microbial components of the gut's normal flora throughout the body. Immune activation creates new HIV infection targets by causing T cells to multiply and become activat-

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ed. Though just 0.01-0.10% of CD4+ T cells in the blood are infected, direct death by HIV alone cannot explain the observed loss of CD4+ T cells. The increased sensitivity of CD4+ T cells to apoptosis when the immune system is still active appears to be a primary factor in CD4+ T cell depletion. Although the thymus regularly produces new T cells to replace the ones that are lost, direct HIV infection of its thymocytes slowly impairs the thymus' ability to regenerate. Eventually, the minimum number of CD4+ T cells required to maintain an adequate immunological response is eliminated, resulting in AIDS.