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Opinion Article

Pathophysiology of immune reconstitution inflammatory syndrome and its risk factors

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DESCRIPTION

Immune Reconstitution Inflammatory Syndrome (IRIS) is a hyperinflammatory response condition that usually occurs in the first six months of treatment in HIV/AIDS patients. This is a potential complication of the use of highly active antiretroviral therapy (HAART). The overall incidence of IRIS is still unknown. However, some studies report that up to 25 to 30% of HIV patients on antiretroviral therapy have IRIS. This activity outlines the risk factors, pathophysiology, and role of the interprofessional team in the management of immune reconstitution syndrome.

Pathophysiology

Pathogenesis of IRIS in HIV patients: Inflammatory immune reconstitution syndrome has been extensively studied in HIV patients receiving HAART. The terms “unmasking”, “dysregulated” and “paradoxical” are often used to describe the state of hyper-inflammatory response seen in IRIS. Patients who are HIV positive and have a low CD4 positive cell count usually begin to see a decrease in their viral load within the first 1 to 2 weeks after starting HAART. Improvement in CD4 count usually occurs in the first three to six months after starting HAART.

As mentioned above, low CD4 count and disseminated latent infection before initiation of HAART, rapid improvement in CD4 count and suppression of infection after initiation of HAART are all risk factors for IRIS. The underlying mechanism of IRIS is complex and is thought to be caused by an imbalance between anti-inflammatory cytokines and pro-inflammatory cytokines that occurs rapidly after recovery of immune function in HIV patients initiated on HAART.

CD4 count increases after initiation of HAART. This includes an initial rapid increase in the number of memory CD4 positive T cells due to reduced apoptosis and redistribution of lymphocytes from peripheral lymphoid tissues. This is followed by a slower increase in the number of naïve CD4 positive T cells. Later in the course of treatment, clonal proliferation of these CD4 positive cells occurs, causing a further increase in the number of cells. Along with CD4 positive T cells, there was also an improvement in CD8 positive T cells.

This dramatic improvement in the number of CD4-positive and CD8-positive T cells leads to improved cell- and antibody-mediated immunity. And it can lead to the following,

1. Excessive pathogen-specific cellular immune response.

2. Decreased ability of regulatory T cells to regulate and suppress inflammation.

3. Uncoupling of innate and acquired immunity.

All of this culminates in a state of hyper-inflammatory response against the underlying pathogens, culminating in the symptoms of IRIS.

Risk factors

Risk factors for IRIS in non-HIV patients are not fully understood. However, similar to HIV patients, a sudden transition from an anti-inflammatory to a pro-inflammatory state would cause an increased risk of IRIS. For example, patients on TNF-alpha blockers or high-dose steroids who are suddenly discontinued, or neutropenic patients who have had a sudden improvement in their white blood cell count after an allogeneic bone marrow or stem cell transplant.

For HIV patients receiving HAART based on previous observational studies, risk factors for IRIS are as follows,

1. Initiation of HAART at a younger age or in male patients has been shown to be associated with an increased risk of developing IRIS.

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2. CD4+T cell count less than 100 cells per microliter at the time of initiation of HAART.

3. Accelerated increase in CD4 count after HAART treatment.

4. Rapid viral suppression of HIV RNA within ninety days of HAART increases the risk of immune reconstitution syndrome.

5. Preexisting latent opportunistic infection with high antigen load increases the risk and severity of IRIS.

6. Initiation of HAART within a short time interval (30 days) after completion of treatment for an opportunistic infection.