Inflammatory Immune Reconstitution Syndrome in HIV-Positive Patients

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Received 06 October 2021; **Accepted** 20 October 2021; **Published** 27 October 2021

Editorial

Antiretroviral therapy (ART) is becoming more widely available around the world. IRIS (immune reconstitution inflammatory syndrome) is a typical side effect after starting ART. We present an overview of the clinical and epidemiological aspects of HIV-associated IRIS, as well as current understanding of pathophysiological causes, accessible medication, and prevention methods in this review. The HIV-associated IRIS spectrum is discussed, with a focus on three major pathogen-associated forms: tuberculosis-associated IRIS, cryptococcal IRIS, and Kaposi's sarcoma IRIS. While clinical characteristics and epidemiology are well understood, there are significant gaps in our understanding of pathophysiology, resulting in inadequate treatment and prevention interventions. To reduce IRIS-related morbidity, the timing of ART introduction is crucial. Improved diagnostic techniques and better focused treatments will presumably result from a greater understanding of the pathophysiology of IRIS.

Antiretroviral therapy has significantly reduced HIV-related mortality, which has fallen from 2.3 million in 2005 to 1.6 million in 2012. 1-3 This represents a 10-fold increase in HIV-infected patients receiving ART in low- and middle-income countries, where the number of patients receiving ART has increased more than 30-fold and life expectancy is rising. International guidelines have been amended to reflect the fact that starting antiretroviral therapy (ART) early in HIV infection improves results. Tuberculosis (TB) is now considered an indication for ART, regardless of CD4 count, because ART reduces TB patient mortality.

However, there is a risk of problems while starting ART, especially in the first six months. IRIS (HIV-associated immune reconstitution inflammatory syndrome) has emerged as a significant early consequence of ART introduction, with significant morbidity and death, especially in patients who start ART with advanced immunosuppression. Immune recovery after starting ART is linked to a pathogenic inflammatory response, which is frequently directed at microbial antigens. Clinical worsening in the early weeks to months of ART, with indications of localised tissue inflammation with or without a systemic inflammatory response, is a crucial hallmark, despite considerable clinical and pathophysiology variation. We give an overview of the clinical and epidemiological features of HIV-associated IRIS, as well as current understanding of pathophysiological mechanisms, available therapy, and prevention strategies, with a focus on three important pathogen-associated forms: tuberculosis-associated IRIS (TB-IRIS), cryptococcal IRIS (C-IRIS), and Kaposi's sarcoma (KS) IRIS. While IRIS-related morbidity may be significant, ART is crucial for HIV survival, and the timing of ART commencement is critical. IRIS should only be used as a last resort to interrupt or stop ART. The scope of our discussion is limited to HIV-associated IRIS. IRIS has been reported after the reversal of different types of immunosuppression, such as iatrogenic immunosuppression in transplant recipients, bone marrow recovery after treatment for haematological malignancies, and termination of anti-tumor necrosis factor-therapy for rheumatoid arthritis.

Although global access to ART is improving, IRIS remains a prevalent consequence because many patients begin ART with low CD4 counts. IRIS is a complicated condition with a variety of case definitions. Some kinds of IRIS have well-defined clinical symptoms and epidemiology, but definitive diagnostic tests and evidence-based treatment options are missing. CNS IRIS is linked to a high rate of mortality, necessitating more effective therapies.

Cite this article: Michael Alumuku. "Inflammatory Immune Reconstitution Syndrome in HIV-Positive Patients". J HIV AIDS Res, 2021,3(3), 001-001.