

HIV Patient Care: An Overview On Management of Complications Arising from Highly Active Antiretroviral Therapy (HAART)

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Many advances have been made in understanding the pathogenesis and progression of HIV-1 disease that includes developing effective antiretroviral agents and regimens, learning how best to use these regimens for maximal and prolonged viral suppression, lowering the pill burden of regimens, and managing many acute adverse effects of treatment. The problems for many patients on long-term effective antiretroviral therapy are the resultant metabolic complications of treatment [1,2]. Much work remains to be done in identifying how best to avoid these complications and how to effectively treat them when they cannot be avoided. HIV-1-infected patients receiving highly active antiretroviral therapy (HAART) suffer from a number of metabolic complications, including lipid abnormalities, dysregulation of glucose metabolism, abnormal distribution of body fat, mitochondrial abnormalities, and bone abnormalities, as well as the sequelae of these disorders [2].

The etiology of these abnormalities remains largely undefined, and it is unclear whether they represent individual or multiple syndromes. The prospect of patients ultimately experiencing these abnormalities influences the timing of initiation of antiretroviral therapy, since the risk of long-term toxicity must be considered against the virological and immunological benefits of early treatment [3,4]. The high risk of these abnormalities should also influence the choice of initial therapy, and the selection should be done case-by-case basis based on risk factors considering the potential complications. The metabolic derangements have an impact on adherence to therapy, which threatens efficacy, and their presence may limit options in salvage therapy. Effective strategies to minimize the occurrence of these complications should be implemented, such as simplification of regimens, need to be developed to preserve the efficacy of HAART [5,6]. Lipo-dystrophy, including lipo-atrophy (wasting) and lipo-hypertrophy (deposition), has been observed in an estimated 40% to 50% of patients receiving long term HAART, and the morphologic changes have a significant impact on the quality of life of patient [7]. The exact etiology of the abnormalities remains still unknown, although it appears to be multi-factorial and could be influenced by specific antiretroviral drugs, host factors such as age and genetics, and HIV-1 disease stage. Research findings of previous studies demonstrate that substituting/switching antiretroviral drugs (substituting a protease inhibitor (PI) with a non-nucleoside reverse transcriptase inhibitor (NNRTI)) has been noted to be safe and could contribute to decrease in insulin resistance, reduce triglyceride levels, maintain total cholesterol and high-density lipoprotein (HDL) cholesterol levels and has posed reduced effects of either fat gain or wasting. Research results regarding the role of rosiglitazone in lipo-dystrophy indicate that it may not be effective in increasing subcutaneous fat in patients with lipo-atrophy alone but may elevate fat mass in those with both insulin resistance and lipo-atrophy. Treatment with this agent has been found to improve insulin sensitivity, hyper-insulinemia, and adiponectin levels, but may result in raised total and low-density lipoprotein (LDL) cholesterol levels [8].

Traditional cardiovascular risk factors have been noted to

contribute to Z (CAD) in HIV-1-infected population and these risk factors need to be identified and managed cautiously. HIV-1-infected patients appear to be at increased risk of coronary heart disease (CHD), as well as for diabetes and hypertension, both major risk factors for CAD. Antiretroviral therapy appears to accelerate the progression of insulin resistance and dyslipidemia. Recent research has observed that HIV-1 infection alone, prior to initiation of antiretroviral therapy, could be associated with elevated cholesterol levels and show an adverse effect on insulin sensitivity. HIV-1 infection mediated inflammation may also play a role in accelerating CHD. Treatment of risk factors for CHD is complicated by drug-drug and drug-disease interactions in HIV-1-infected population. Further studies are needed to understand the pathogenesis of dyslipidemia and cardiovascular disease in HIV-1-infected patients. Patients should be assessed for risk factors for metabolic complications of antiretroviral therapy prior to initiation of therapy and should be monitored for such complications every 3 to 6 months after starting treatment, at the time of switching therapy, and at least annually thereafter. Routine measurement of fasting glucose or glucose tolerance testing and routine monitoring of fasting lipids is recommended. Metabolic complications of long-term therapy threaten the clinical benefits of such effective treatment [9,10].

The disease course in HIV-1 infection appears to be highly complex more so after initiation of HAART and physicians involved in treating HIV-1 infected population should consider all factors that include the toxic side-effects of HAART for the effective management, improved patient outcome and better quality of life.

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