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DYSLIPIDEMIA DUE TO HIV INFECTION AND ITS THERAPY

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Recent reports of hyperlipidemia, central obesity and insulin resistance in HIV infected patients on Protease Inhibitor (PI) therapy have pointed towards increased risk of coronary artery disease (CAD). However, changes in lipoproteins were previously reported due to HIV infection. To understand the risk of CAD it is important to review all changes in lipid metabolism that occur. Early in HIV infection there are decreases in HDL cholesterol levels. Low HDL is by itself a significant risk factor for CAD. Later in HIV infection LDL levels decrease, but the LDL is small and dense, which is associated with increased atherogenesis. Subsequently, at the time of transition to AIDS, triglyceride levels rise, due to an increase in VLDL. Changes in triglycerides correlates with circulating levels of interferon- α , the host response to viral infected cells. VLDL cholesterol also contributes to CAD. Genetics influences the degree of change. The contribution of body composition to lipid changes needs more investigation.

AZT treatment of therapy naïve patients with advanced HIV disease leads to decreased interferon and triglyceride levels. In contrast, treatment of HIV infected patients with HIV Protease Inhibitors (PI) induces a different profile. Triglyceride levels increase, the opposite of what was seen with AZT. LDL cholesterol levels increase to normal. HDL is not significantly changed. Using epidemiological data, the major risk of CAD is from the low level of HDL seen in HIV infection. PI induced increases in LDL and VLDL cholesterol also predict an increased risk of CAD events, but the predicted event rate is insignificant compared to the proven benefit of PI's in decreasing death and complications in AIDS. However, the extension of life provided by Highly Active Antiretroviral Therapy means that CAD will be increasingly seen. A patient's genetic background (family history) and other risk factors for CAD will help predict the need for hypolipidemic therapy.

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