

HIV-1 INFECTION AND ITS INFLUENCE ON LIPID METABOLISM AND CARDIOVASCULAR DISEASE

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It is important to recognize the fact that human immunodeficiency virus (HIV-1) infection by itself can have its effect on lipid metabolism irrespective of initiation of highly active antiretroviral therapy (HAART). For example, it has long been recognized that triglyceride level increases markedly with HIV-1 disease progression,

likely reflecting persistence of an inflammatory state as well as wasting. A previous research has showed an increase in mean triglyceride value from 91 mg/dL in HIV-1 negative individuals to 166 mg/dL among HIV-1 infected group and 231 mg/dL in people living with AIDS. These results indicate an increase in triglyceride level

occurring in half of the HIV-1-infected patients. Total cholesterol value was noted to decrease from 190 mg/dL in non HIV-1 infected population to 157 mg/dL among patients with AIDS. Data from a Multicenter AIDS Cohort Study (MACS) cohort indicate that low-density lipoprotein cholesterol (LDL-C), total cholesterol, and HDL-C levels decrease with HIV-1 infection. Initiation of antiretroviral therapy is associated with increases in LDL-C and total cholesterol values and persistence of reduced HDL-C level. Antiretroviral agents can affect triglyceride and other lipid levels. In a study assessing changes in lipid levels in non-HIV-1 infected subjects, 5 days of administration of ritonavir-boosted lopinavir increased triglyceride value significantly (177 mg/dL) compared with atazanavir (131 mg/dL) or placebo (124 mg/dL), with no significant differences in total cholesterol, LDL-C, or HDL-C being observed. Another research study observed a group of patients that

included HIV-1 infected patients who were receiving a combination containing stavudine/ lamivudine with either nelfinavir or atazanavir. This study has noted that among those patients receiving nelfinavir there was increased activities of total cholesterol, LDL-C, and triglyceride values as compared to baseline levels. Study had also observed returning of activities of these parameters to near baseline values after patients were switched to open-label atazanavir at 72 weeks. In another experience, 162 patients with hyperlipidemia on other antiretroviral therapy regimens (34% receiving lopinavir/ritonavir) were crossed to ritonavir- boosted atazanavir as part of an early access program. After 6 months, total cholesterol activities were reduced by 12%, LDL-C by 10%, and triglyceride by 18%, and HDL-C was increased by 3% (all statistically significant changes). Almost one-third of patients who were receiving lipid-lowering therapy were able to discontinue such therapy

after the switch to ritonavir boosted atazanavir. Many HIV-1-infected patients have dyslipidemia and other cardiovascular risk factors prior to acquiring infection. Both HIV-1 infection by itself and initiation of antiretroviral therapy can cause or worsen lipid abnormalities. Management of dyslipidemia in the HIV-1-infected population requires awareness of the effects of antiretroviral agents on lipid profiles, including potential of sex, race and individual related effects, and interactions between lipid-modifying agents and antiretroviral agents.¹⁻¹³

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