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Cardiovascular disease and HIV

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Different population studies have consistently shown that HIV-infected patients have approximately 2-fold higher incidence of coronary artery disease or myocardial infarction than non-HIV-infected persons.

However, all these studies have shown a higher prevalence of traditional cardiovascular risk factors (smoking, hypertension, diabetes mellitus, and dyslipidemia) in HIV+ relative to HIV-. These factors are the most common determinants of the cardiovascular risk in a given HIV-infected patient. They should be screenned for in any patient and agressively treated if estimated cardiovascular risk is moderate/high.

HIV infection itself additionally contributes to a higher risk of cardiovascular disease through inflammation, immune depression (low CD4 cell counts), and inmmune activation. There is a need to control HIV infection with antiretroviral therapy to decrease the cardiovascular risk associated with HIV infection. The overall effects of any effective antiretroviral therapy on cardiovascular disease are definitely more positive than not giving therapy at all. Antiretroviral therapy contributes to decrease the effect of HIV infection, although it may be unable to lower it to a level similar to that of uninfected persons.

Antiretroviral therapy may contribute to cardiovascular risk in a more modest way than that of traditional risk factors and uncontrolled HIV infection, through the induction of metabolic abnormalities (dyslipidemia and insulin resistance). This effect has been proven for protease inhibitors, and it remains controversial for thymidine nucleoside reverse transcriptase inhibitors. It should be beard in mind that not all protease inhibitors and not all patients receiving a protease inhibitor necessarily develop metabolic abnormalities. It is unclear whether antiretroviral therapy may have pathogenetic mechanisms other than metabolic abnormalities contributing for a higher cardiovascular risk.

Abacavir has been identified as a marker of cardiovascular disease, but its potential role as a causative agent is confounded by multiple factors that are impossible to adjust for completely in cohort studies. A plausible underlying mechanism is not known either. The results of a BICOMBO sub-study sugested that abacavir does not not cause inflammation, endothelial dysfunction, hypercoagulability, or insulin resistance in virologically suppressed HIV-infected patients. As of January 2010, health authorities in Europe and in the United States (EMEA and FDA, respectively) have concluded that there is no definitive information proving that abacavir may induce cardiovascular disease.

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