HIV Infection Tied to Acute Coronary Syndrome

BY ROBERT FINN San Francisco Bureau

SAN FRANCISCO — People infected with HIV are diagnosed with acute coronary syndromes an average of 11 years earlier than are their HIV-negative counterparts, Priscilla Hsue, M.D., reported at a meeting on HIV management sponsored by the University of California, San Francisco.

Moreover, atherosclerosis, as measured

by carotid intima-media thickness, progresses much faster in patients who are HIV-positive, and restenosis rates after percutaneous coronary intervention are significantly higher in HIV patients than in control subjects.

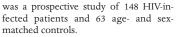
Although protease inhibitors and other components of highly active antiretroviral therapy may contribute to acute coronary syndromes in patients with HIV, this can't account for all of the differences between patients with HIV and noninfected controls. HIV may be an independent coronary risk factor, said Dr. Hsue, assistant professor of medicine at the university.

She reported on the results of two studies. One was a retrospective chart review of 68 acute coronary syndrome patients with HIV who were compared with 68 uninfected acute coronary syndrome patients. Acute coronary syndrome was defined by a diagnosis of acute myocardial infarction or unstable angina. The other



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The chart review showed that the average age of HIV-infected acute coronary syndrome patients was 50 years, compared with 61 years for the noninfected patients. HIV patients with acute coronary syndrome were significantly more likely to be male (90% vs. 62%), to be current cigarette smokers (68% vs. 41%), and to have low HDL cholesterol levels (35 mg/dL vs. 41 mg/dL). Results of this study were published last year (Circulation 2004;109:316-9).

HIV patients were significantly less likely to have diabetes (13% vs. 41%), and they had significantly less extensive coronary



The HIV-infected patients had significantly higher carotid intima-media thickness than did controls.

DR. HSUE

disease at angiography. An average of 1.3 vessels were involved in patients with HIV, compared with 1.9 vessels in controls.

Percutaneous coronary intervention was performed on 29 HIV patients and 21 controls. Restenosis occurred in 15 of the HIV patients (52%) and in 3 controls (14%), a significant difference.

In the prospective study, the HIV-infected patients had significantly higher carotid intima-media thickness (as measured by B-mode ultrasound) than did controls (0.91 mm vs. 0.74 mm). Investigators detected carotid plaques in 45% of the HIV patients and 24% of the control patients, a significant difference (Circulation 2004;109:1603-8).

In a multivariate analysis combining infected and uninfected patients, HIV infection proved to be an independent predictor of greater intima-media thickness, even after controlling for other classic coronary risk factors, including age, sex, smoking, hypertension, lipid abnormalities, and diabetes. Other independent predictors were age, LDL cholesterol, cigarette pack-years, and Hispanic race.

Investigators were able to obtain followup measurements at 1 year in 121 HIV patients and 27 controls. Among HIV patients, intima-media thickness increased a mean of 0.074 mm, while among control subjects intima-media thickness decreased by 0.006 mm, a significant difference.

Previous studies of noninfected patients suggest carotid intima-media thickness tends to increase at about 0.01 mm/year, a rate about sevenfold lower than that observed among HIV patients in this study.

These studies suggest that clinicians should engage in aggressive control of risk factors in patients with HIV. Smoking may be of particular importance because of its high prevalence in this population. Hypertension should be treated, LDL cholesterol should be reduced to low levels, and hypertriglyceridemia should be controlled, Dr. Hsue said.