HIV Study Notes More Cardiovascular Disease in Long-Term Nonprogressors

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Long-term nonprogressors (LTNPs)—people living with HIV for several years without requiring antiretroviral (ARV) therapy—face a higher risk of cardiovascular disease (CVD) compared with HIV-negative people, according to a new study reported at the 50th Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC) on Sunday, September 12, in Boston. These findings, the authors suggest, indicate that HIV infection itself can lead to higher rates of CVD, independent of ARV treatment.

About 1 in 100 people living with HIV are LTNPs, in that they're able to maintain low viral loads and higher CD4 cell counts without taking ARVs. An even smaller number of people—about 1 in 300—are considered "elite controllers." These rare individuals have undetectable viral loads and normal CD4 counts in the absence of therapy.

Because LTNPs experience slow HIV disease progression—or do not progress at all, even after several decades of living with HIV—they are significantly less likely to experience hallmark AIDS-related manifestations, such as opportunistic infections. However, there has been some evidence that they are more likely than people not infected with HIV to develop certain non-AIDS-related diseases, such as cancer and CVD. This is likely because HIV is still active in their bodies and, as a result, they remain susceptible to health problems caused by underlying inflammation.

Because many LTNPs and elite controllers do not take ARV therapy to maintain their health, researchers have been keen on studying them to learn more about untreated HIV's affects on the human body, notably the risk and severity of non-AIDS-related diseases not typically tied to CD4 cell loss.

One such study, reported at ICAAC by Hector Bonilla, MD, of Summa Health System in Akron, Ohio, and his colleagues, compared markers of CVD in 13 LTNPs (including four elite controllers) and 13 HIV-negative subjects. Evaluations of carotid intima-media thickness (IMT) —ultrasound measurements of the walls of the arteries in the neck channeling oxygenated blood to the brain—and various blood markers were conducted on all patients in the study.

Among the 13 patients in the LTNP group, four were women and six were African American. The LTNPs had been infected with HIV for about 13.5 years upon taking part in the study and averaged 44 years old. The average CD4 count was 440 cells. Most LTNPs had viral loads below 2,000 copies at study entry.

According to Bonilla's report, carotid IMT in the LTNPs was similar to that in the HIV-negative

patients. However, there was some evidence of increased IMT of the carotid bulb—the point where the artery splits into the internal and external carotid arteries—in the LTNPs, compared with the HIV-negative study subjects. Yet the observed difference was shy of statistical significance, meaning that it could have been due to chance.

Only one blood marker of inflammation—soluble tumor necrosis factor receptor type 2 (sTNF-RII)—was found to be significantly higher in the HIV-positive LTNPs compared with the HIV-negative patients. However, Bonilla's group noted that "good" HDL cholesterol levels tended to be much lower among LTNPs than the HIV-negative study subjects. Eight of the 13 LTNPs had HDL levels below 40 milligrams per deciliter (mg/dL), an established risk factor for CVD.

Bonilla's team also suggested that another marker, soluble vascular adhesion molecule (sVCAM), was significantly correlated with increased carotid IMT in LTNPs.

"Endothelial markers are significantly elevated in long-term nonprogressors compared to healthy controls, and correlate with carotid IMT measurements," Bonilla's team concluded. The study also underscores the need for more research focusing on the effects of HIV infection itself on the increased risk of CVD in people living with HIV.

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