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ART alone not sufficient to reduce arterial inflammation among HIV-infected patients, study finds

Published on May 26, 2016 at 5:40 AM

Initiating antiretroviral therapy (ART) soon after diagnosis of an HIV infection did not prevent the progression of significant arterial inflammation in a small group of previously untreated patients. The findings from a Massachusetts General Hospital (MGH)-based research team suggest that ART alone is not sufficient to reduce the elevated arterial inflammation that appears to contribute to the increased risk of cardiovascular disease in HIV-infected individuals.

"Our previous studies have found that persistent arterial inflammation may predispose people living with HIV to the development of high-risk coronary artery plaques that are potentially more likely to rupture and cause a heart attack," says Markella Zanni, MD, of the Program in Nutritional Metabolism in the MGH Neuroendocrine Unit, lead author of the report published in *JAMA Cardiology*. "Our findings suggest that additional strategies geared toward reducing arterial inflammation among HIV-infected patients receiving ART may be needed."

Evidence from a number of studies suggests that HIV-infected patients have a 50 to 75 percent increased risk of heart attack and stroke compared with uninfected individuals who have the same traditional risk factors. A 2012 *JAMA* study led by Steven Grinspoon, MD, director of the Program in Nutritional Metabolism and senior author of the current study, found levels of inflammation within the aortas of HIV-infected individuals that were comparable to those seen in patients with established cardiovascular disease. HIV-infected participants in that study had been receiving ART for at least three months when they entered the study, but it was not known whether inflammation had developed before or during ART treatment. Since ART reduces several measures of immune system activation, the current study was designed to investigate whether it could suppress or reduce arterial inflammation.

The study enrolled 12 previously untreated HIV-infected men, who were about to begin ART with a currently used combination of four antiviral medications an average of 11 months after their original diagnosis. Another group of 12 uninfected, age-matched control participants was recruited for comparison of immune system and inflammatory factors. None of the participants had a prior history of coronary artery disease or autoimmune or inflammatory disease other than HIV infection, and none had significant cardiovascular risk factors.

At the outset of the study and six months later, PET scans with a radiopharmaceutical called FDG, which accumulates at sites of inflammation, were conducted -- imaging the aortas, hearts, underarm lymph nodes, spleens and bone marrow of the HIV-infected participants. Those patients also had CT angiograms to look for coronary artery plaques and blood tests to analyze lipid and immune systems factors, along with HIV viral loads.

While ART suppressed viral loads and increased levels of CD4 T cells in HIV-infected participants, 80 percent of them developed increased inflammation of the aorta during the 6-month study period. Although inflammation was reduced significantly in lymph nodes and somewhat in the spleen, arterial inflammation had increased to levels similar to those seen in the earlier study of patients already receiving ART. At the outset of the current study, some level of coronary artery plaque was seen in 25 percent of HIV-infected participants, and six months later those plaques had all enlarged. An additional participant was found to have developed a new plaque during the study period.

"Further research is needed to better understand the relationship between persistent immune activation, arterial inflammation and plaque development in HIV infection," says Grinspoon, who is a professor of Medicine at Harvard Medical School. "We need to compare the effects of different antiretroviral therapy regimens on arterial inflammation over time. Moreover, we need to investigate the important question of whether administering immune-modulatory strategies along with ART can help suppress arterial inflammation, stabilize coronary plaques and reduce cardiovascular risk in HIV-infected patients."

Source:
Massachusetts General Hospital