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HIV INFECTION IS ASSOCIATED WITH PROGRESSION OF HIGH RISK CORONARY PLAQUE IN THE MACS

Clinical: (M) Cardiovascular Complications of HIV Infection and Antiretroviral Therapy

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Background: HIV infection is associated with coronary atherosclerosis, especially non-calcified plaque (NCP) and mixed plaque; however, development and progression of atherosclerosis associated with HIV has only been shown with coronary artery calcium scanning, which cannot detect more potentially unstable plaques. We prospectively evaluated the association between HIV serostatus and the progression and composition of coronary plaque among men in the Multicenter AIDS Cohort Study (MACS).

Methods: We performed baseline and follow-up coronary CT angiography in 409 men (253 HIV+, 156 HIV-; median interscan interval=4.5 yrs). Calcified and NCP volumes, including lipid-rich low attenuation plaque (LAP), were measured in each coronary segment. We used Poisson regression to test the association between HIV serostatus and incident plaque among men without baseline plaque, and generalized gamma regression to test the association with progression among men with baseline plaque, adjusting for time between scans, demographics and CVD risk factors. We also evaluated plaque progression differences between HIV- men and HIV+ men with suppressed viral load (<50 copies/mL, ≤ 1 "blip" <500 copies/mL) and those with viremia during the inter-scan interval.

Results: Mean age was 54 yrs (53 HIV+, 57 HIV-) and 32% were black (35% HIV+, 27% HIV-). 70% of HIV+ men were aviremic during the interval. There were 118 men (74 HIV+, 44 HIV-) with no baseline plaque. Incident plaque was seen in 36 (30%) men; 24 developed both NCP and calcified plaque (mixed plaque) and 12 developed only NCP. LAP developed in 27 men. HIV+ men had a greater adjusted incidence of NCP (IRR 2.13, p=0.03), LAP (IRR 2.84, p=0.05) and mixed plaque (IRR 3.09, P=0.01) than HIV- men. In addition, compared to HIV- men, the incidence of LAP was greatest among HIV+ men with viremia (IRR 5.4, p=0.009; aviremic men IRR 2.4, p=0.096). There were 291 men with baseline plaque (179 HIV+, 112 HIV-). Among men with the greatest NCP volume change, the adjusted increases were significantly greater among HIV+ compared to HIV- men (e.g. 80th %tile of change in NCP was 175 mm3 for HIV+ compared to 112 mm3 for HIV-, P=0.03), with similar trends for total plaque and LAP.

Conclusion: This is the first study to demonstrate that HIV infection is associated with an elevated incidence and progression of high risk coronary plaque and suggests the need for additional studies to determine the importance of controlling viremia to limit the excess burden of CVD events in this population.