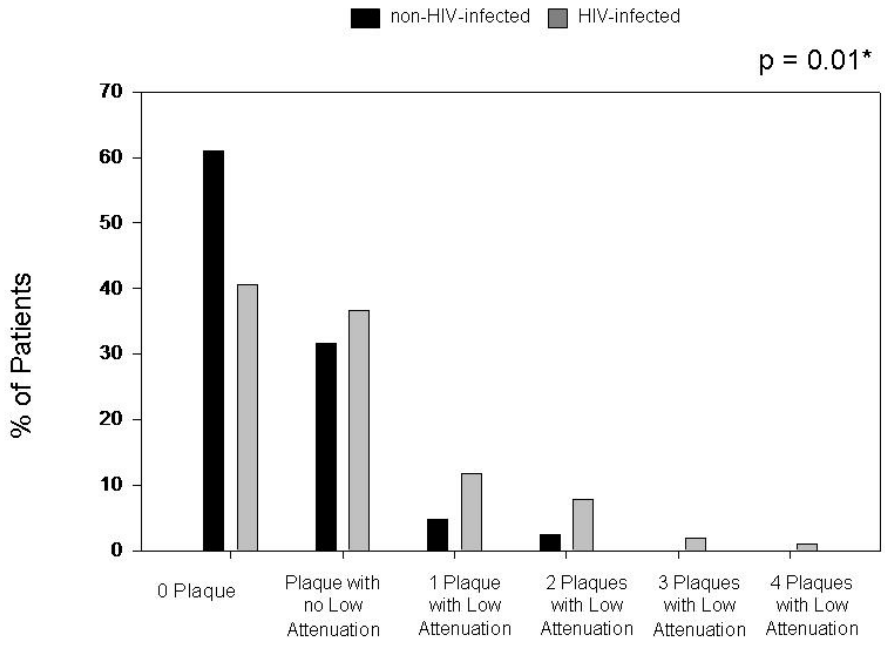
Supplement 1: Coronary CTA Imaging Methodology

A 64-slice CT scanner (Sensation 64; Siemens Medical Solutions, Forchheim, Germany) was used to obtain CT images. In order to obtain an optimal heart rate for CT scanning (</= 60 beats per minute), intravenous metoprolol was administered as need absent contraindications. Sublinguinal nitroglycerin was also administered to facilitate vasodilation and maximize image quality. Our protocol for scanning and methods for image reconstruction have been previously described in detail [8] [9]. Briefly, the images were obtained during inspiratory breath hold. 80-100 ml contrast agent (opamidol, Isovue, Bracco Diagnostics, Inc.) was administered intravenously at 5 ml/s to facilitate enhancement of the coronary arterial tree. Coronary CTA datasets were acquired with 64 x 0.6 mm slice collimation, a gantry rotation time of 330 ms, tube voltage of 120 kVp, and an effective tube current of 850 mAs using ECG-correlated tube current modulation when appropriate. Axial images were reconstructed with a slice thickness of 0.75 mm and increment of 0.4 mm using a half-scan algorithm with a temporal resolution of 165 ms. Images were initially reconstructed at 60, 65, 70, and 35% of the cardiac cycle. Additional reconstructions were performed to minimize motion artifacts, if necessary. All reconstructions were transferred to an offline work station for analysis (Leonardo; Siemens Medical Solutions).

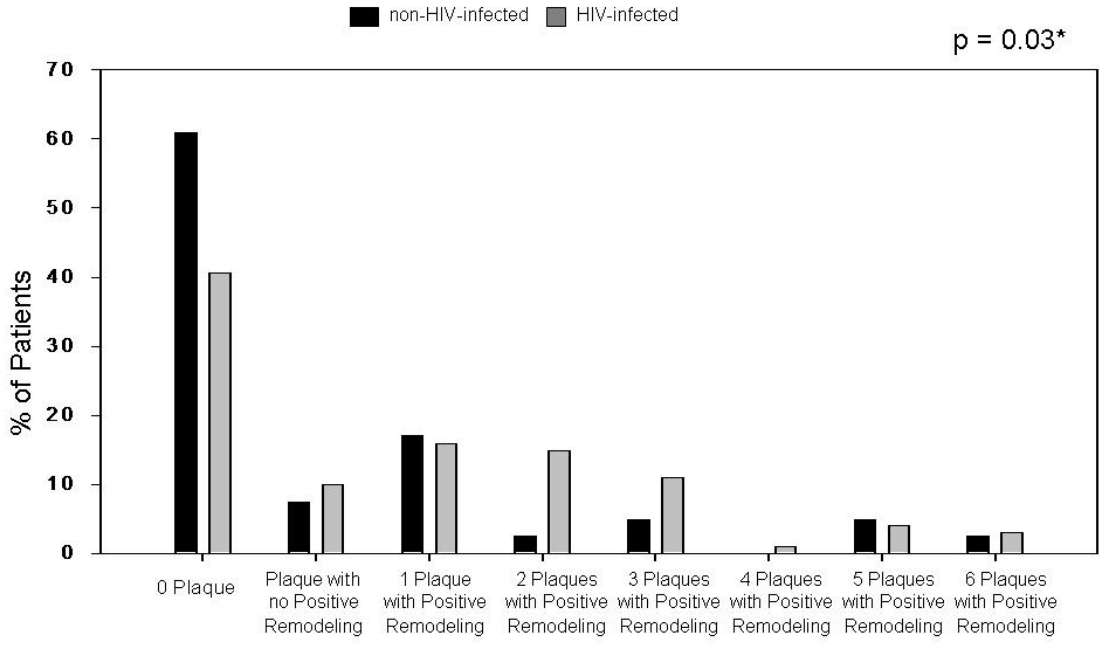
Supplement 2: Between Group Comparison (HIV versus non) of the Number of Plaque Segments with Vulnerability Features Among all Segments

Among the total arterial segments studied - 18 segments per study subject x 142 subjects for a total of 2556 segments - 11.3% of the 1818 segments among the HIV-infected group were noted to have plaque versus 8.1% of the 738 segments among controls (Conditional Logistic Regression Analysis Odds Ratio (OR) Point Estimate 1.5, 95% Wald Confidence Limits [1.1-2.1], p=0.01). Among the proximal arterial segments studied - 4 segment per study subject x 142 subjects for a total of 568 segments - 23.0% of the 404 segments among the HIV-infected group had plaque versus 15.9% of the 164 segments among controls (OR 1.6 [1.0-2.7], p=0.05). In addition, among all segments, there was a higher percentage of low attenuation plaque segments in the HIV-infected group versus the non-HIV-infected group (2.1% of 1818 segments versus 0.54% of 738 segments; OR 4.0 [1.4 - 11.4], p=0.009) and of positively remodeled segments (6.7% of 1818 segments versus 4.2% of 738 segments; OR 1.7 [1.1-2.6], p=0.01) in the HIV-infected group versus the non-HIV-infected group. There were not a higher percentage of spottily calcified segments in the HIV-infected group versus the non-HIV-infected group (3.4% of 1818 segments versus 2.9% of 738 segments; OR 1.2 [0.7 - 2.0], p=0.46).

Supplemental Figure 1a: Percentage distribution of the number of low attenuation plaques per subject among all subjects by HIV status. P value is for overall difference between HIV-infected and non-HIV-infected groups in number of low attenuation plaques per subject by Wilcoxon test.



Supplemental Figure 1b: Percentage distribution of the number of positively remodeled plaques per subject among all subjects by HIV status. P value is for overall difference between HIV-infected and non-HIV-infected groups in number of positively remodeled plaques per subject by Wilcoxon test.



Supplemental Table 1

Multivariate Model for Number of Low Attenuation Plaques Per Subject Among Entire Cohort of HIV-infected and Non-HIV-infected Subjects

Overall R^2 0.14 for model, p = 0.02

Parameter	Estimate	Standard Error	p value
Age	0.02	0.01	0.23
Family history of premature CHD	-0.17	0.11	0.13
Current smoking	0.02	0.09	0.79
Current diabetes	-0.12	0.22	0.58
Systolic blood pressure	0.009	0.007	0.20
LDL cholesterol	0.005	0.003	0.10
HIV status	-0.20	0.09	0.03*
Hepatitis C status	0.03	0.12	0.79