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A Novel Density-Volume Calcium Score by Non-Contrast CT Predicts Coronary Plaque Burden on Coronary CT Angiography: Results from the MACS (Multicenter AIDS Cohort Study)

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Abstract

Background: The purpose of this study is to determine if a new score calculated with coronary artery calcium (CAC) density and volume is associated with total coronary artery plaque burden and composition on coronary CT angiography (CCTA) compared to the Agatston score (AS).

Methods: We identified 347 men enrolled in the Multicenter AIDS cohort study who underwent contrast and non-contrast CCTs, and had CAC>0. CAC densities (mean Hounsfield Units [HU]) per plaque) and volumes on non-contrast CCT were measured. A Density-Volume Calcium score was calculated by multiplying the plaque volume by a factor based on the mean HU of the plaque (4, 3, 2 and 1 for 130–199, 200–299, 300–399, and ≥400HU). Total Density-Volume Calcium score was determined by the sum of these individual scores. The semi-quantitative partially

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calcified and total plaque scores (PCPS and TPS) on CCTA were calculated. The associations between Density-Volume Calcium score, PCPS and TPS were examined.

Results: Overall, 2879 CAC plaques were assessed. Multivariable linear regression models demonstrated a stronger association between the log Density-Volume Calcium score and both the PCPS (β 0.99, 95%CI 0.80–1.19) and TPS (β 2.15, 95%CI 1.88–2.42) compared to the log of AS (PCPS: β 0.77, 95%CI 0.61–0.94; TPS: β 1.70, 95%CI 1.48–1.94). Similar results were observed for numbers of PC or TP segments.

Conclusion: The new CAC score weighted towards lower density demonstrated improved correlation with semi-quantitative PC and TP burden on CCTA compared to the traditional AS, which suggests it has utility as an alternative measure of atherosclerotic burden.

Keywords

Coronary artery calcium; Calcium density; Calcium volume; Coronary computed tomographic angiography; Human immunodeficiency virus

INTRODUCTION

Coronary artery calcium (CAC), detected on non-contrast computed tomography (CT), is a marker of coronary atherosclerosis which is associated with total coronary plaque burden[1]. CAC scanning is widely used to determine risk for coronary artery disease (CAD) events among asymptomatic individuals [2–6]. Agatston scoring is a simple method to estimate total CAC burden. It is determined by the sum of a lesion score, which is calculated by multiplying the maximal density factor by the plaque area for each plaque. The maximal density factor ranges between 1 and 4 (factor 1=130–199 Hounsfield Unit [HU], 2=200–299 HU, 3=300–399 HU, and 4 ≥400 HU) [7]. In this regard, Agatston scoring is heavily weighted towards high CAC score with greater CAC density.

Coronary computed tomographic angiography (CTA) enables both non-calcified and calcified plaques to be displayed, thus more accurately detecting the true overall atherosclerotic burden in the coronary arteries than with non-contrast CT imaging. However, coronary CTA requires the injection of intravenous contrast and often the administration of a beta blocker to slow the heart rate. Given the simpler protocol of CAC scanning, it is important to determine whether there are more accurate methods to utilize the coronary calcium density and volume measurements from non-contrast CT than the traditional Agatston CAC score to estimate total coronary plaque burden, including non-calcified components. We have developed a new method, the Density-Volume Calcium score. The purpose of this study was to examine if Density-Volume Calcium scores, obtained from non-contrast CT scans, are associated with the number of plaques (calcified, partially calcified and non-calcified), the number of partially calcified plaques, and the total plaque burden and partially calcified plaque burden which also incorporates a visual estimate of the plaque volume from coronary CTA.

Materials and Methods

Study population—The Multicenter AIDS Cohort Study (MACS) is a prospective cohort study enrolling homo- or bisexual men conducted in 4 sites in the U.S (Baltimore, Maryland; Chicago, Illinois; Pittsburgh, Pennsylvania; and Los Angeles, California), to assess the natural history of the infection causing acquired immunodeficiency syndrome [8]. As previously reported [9], the MACS cardiovascular sub-study was conducted to investigate coronary artery atherosclerosis by non-contrast and contrast coronary CT among HIV-infected and uninfected men. All men were aged between 40 and 70 years, weighing <136kg (300 pounds), and did not have a history of cardiac surgery or any coronary intervention. Among 1001 men who were enrolled in the study between January 2010 and August 2013, 759 underwent both CAC scanning and coronary CTA studies [9]. Of those, we identified 347 participants who had CAC Agatston score >0. Institutional Review Board approval was obtained at each institution. All participants provided informed consent.

Non-contrast and contrast CT Image Acquisition Protocol—As described previously [10], all participants underwent ≥64-slice coronary CTA (Lightspeed VCT, GE Healthcare, Milwaukee, WI; Somatom Sensation and Definition CT, Siemens, Forchheim, Germany; Aquilion One, Toshiba, Otawara, Japan). Participants received oral and/or intravenous beta-blockade to achieve a target heart rate <65 beats/min, as well as sublingual nitroglycerin for coronary artery dilatation, unless contra-indicated. Participants underwent non-contrast CT to measure CAC before contrast coronary CTA scanning. Prospective ECG-triggering CT acquisition was used for non-contrast CT. Scan parameters were obtained as follows: 2.5mm or 3mm slice thickness, 30–35 mm field of view, 512×512 matrix size, and peak tube voltage of 120 kVp. Images were analyzed on our custom software (Density-Volume CAC software, Los Angeles Biomedical Research Institute at Harbor UCLA Medical Center, Torrance, California, USA). CAC density measurements including mean of density (HU) and SD for each lesion were performed by a CT core laboratory (LA Biomedical Research Institute at Harbor UCLA Medical Center, Torrance, California, USA) using custom software to quantify new Density-Volume Calcium score as well as the CAC score using the Agatston method [7]. The CAC volume was measured by calculating the plaque area multiplied by the slice spacing. A Density-Volume Calcium score was calculated by multiplying the plaque volume by a factor based on the mean HU of the plaque using the following categories, with a greater factor assigned for lower density plaques: 4 for 130–199 HU, 3 for 200–299 HU, 2 for 300–399 HU, and 1 for ≥400 HU. Total Density-Volume Calcium score was determined by the sum of these lesion scores.

The coronary CTA scanning protocols have been previously reported [9, 10]. Oral and/or intravenous beta-blocker was administered to reach target heart rate <65 beats/min. immediately before scanning, sublingual nitroglycerin was administrated. Participants underwent prospective electrocardiogram-triggering study, except if the HR remains >65 beats/min.

Coronary CTA image analysis for coronary plaque type—Coronary CTA data sets were assessed using a 15-segment American Heart Association coronary tree model in accordance with Society of Cardiovascular Computed Tomography guidelines [11] by the CT

core laboratory (Los Angeles Biomedical Research Institute at Harbor UCLA Medical Center, Torrance, California, USA) in a blinded manner. Coronary plaque was identified as any hyper- or hypodense structure distinct from the lumen and >1mm^2 in size. A plaque without calcification was defined as a plaque which was more hypodense than the lumen and more hypodense than epicardial fat, and usually Hounsfield Units>−30. Plaque type was categorized into three groups: non-calcified (contains no calcified plaque), partially calcified plaque (includes both non-calcified and calcified plaque) and calcified plaque. The numbers of total or partially calcified plaques (PCP) were defined as the sum of the segments with each specific plaque type.

The total plaque score (TPS) was semi-quantitatively assessed by straight view of curved multi planar reconstruction (Figure 1A–C) and cross-sectional images (Figure 1D–F). Using a 15-segment American Heart Association coronary tree model, TPS was calculated by summing the plaque size score (1: mild, 2: moderate and 3: severe) with either calcified (CP), non-calcified (NCP), or PCP per segment, to a maximum score of 45 [12]. Mild, moderate and severe TPSs were defined as when a plaque extends within one-third of the segment (Figure 1A and D), between 1/3 and 2/3 of the segment (Figure 1B and E) and with > two-third of the segment, respectively (Figure 1C and F).

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation (SD) or median (interquartile range) in the descriptive statistics. A two-sided p value of <0.05 was set as significance level. We assessed the association between the Density-Volume Calcium score and the Agatston CAC score with the number of PCP and the number of total plaques, the PCP score (PCPS), and the TPS, using both unadjusted linear regression and multivariable linear regression accounting for potential confounders. We made the modeling assumption that the count variable for number of plaques was properly approximated as a continuous measure, permitting the use of linear regression models. To compare the two versions of CAC score, we focused on using r^2 to estimate the goodness of fit of the linear models.

The CAC density-score and Agatston scores were both log transformed using a natural log (CAC Score +1) approach to account for a high degree of skew in these variables. Linear regression models were adjusted for age, systolic blood pressure, BMI, diabetes, total cholesterol, HDL-cholesterol, glucose, cumulative pack-years smoking, African-American race/ethnicity, statin use, and HIV serostatus. Correlation matrices between the log of the Density-Volume Calcium score based on mean Hounsfield units, log of the traditional Agatston score, TPS, and PCPS were also examined to look at the degree of correlation between the Density-Volume/ Agatston CAC score and relevant plaque scores. These correlations stratified by statin use and HIV status were also assessed. The correlations between the log-transformed Density-Volume Calcium score, log-transformed Agatston score, total plaque burden score, and the partially calcified plaque score were calculated using Pearson correlations. All statistical calculations were performed using SAS version 9.4 for Windows.

RESULTS

Overall, 57.6% (n=200) of participants were HIV infected. The baseline characteristics of participants in the cohort are listed in Table 1. Mean age was 58±6 years and 71% of participants were white. Almost half of participants were former smokers and statin was prescribed in 45% (Table 1). Table 2 lists non-contrast and contrast coronary CTA findings, including number of plaques of each type, plaque scores, and CAC scores. Median Density-Volume Calcium score was higher than Agatston CAC score. With respect to contrast CT findings, greater mean number of plaques was shown in calcified, followed by non-calcified and then partially calcified. A similar trend was observed with the plaque score.

In univariate models, the Density-Volume Calcium score was associated with the number of total plaques or partially calcified plaques, as well as total plaque scores or partially calcified plaque scores (Table 3). The log-transformed Density-Volume Calcium score showed better goodness of fit, as measured by the r^2 parameter from the unadjusted linear regression model, as compared to the log Agatston score (r^2=0.40 versus r^2=0.36) when using the score to estimate the number of total plaques present. There was a similar improvement in the goodness of fit parameter when the endpoint was the number of partially calcified plaques (r^2=044 versus r^2=0.40) in the unadjusted linear models.

Multivariable linear regression analysis was performed to determine if the log-transformed Density-Volume Calcium score was associated with the number of total plaques or partially calcified plaques after adjusting for potential confounders (including age, systolic blood pressure, BMI, diabetes, total cholesterol, HDL, glucose, cumulative pack-years smoking, race/ethnicity, body mass index, statin use and HIV serostatus). In this linear regression model, a one unit change in the log-transformed density-volume Calcium score was associated with an increase in the number of total plaques (β 1.13, 95% CI 0.98 to 1.29, p<0.001) and the number of PCP (β 0.48, 95% CI 0.38–0.59, p<0.001) (Table 3). A one unit change in the log-transformed version of the traditional CAC Agatston score also showed a significant, but weaker association, with the number of total plaques (β 0.91, 95% CI 0.77 to 1.04, p<0.001) and the number of PCP (β 0.37, 95% CI 0.29–0.46, p<0.001) (Table 3).

We also assessed the relationship of semi-quantitative plaque burden as measured by the TPS, and the PCPS to the log-transformed CAC scores. A one unit change in the log-transformed Density-Volume Calcium score was associated with a greater TPS (β 2.15, 95% CI 1.88–2.42, p<0.001) as compared to the log-transformed Agatston score (β 1.70, 95% CI 1.48–1.94, p<0.001). A one unit change in the log-transformed Density-Volume Calcium score was associated with a higher PCPS (β 0.99, 95% CI 0.80 to 1.19, p<0.001) as compared with the log-transformed Agatston score (β 0.77, 95% CI 0.61–0.94, p<0.001) (Table 4). Sensitivity analysis showed associations were similar when stratified by HIV status and/or statin use (supplemental tables 1 and 2).

Table 5 lists Pearson correlations between the log-transformed Density-Volume Calcium score, log-transformed Agatston score, total plaque burden score, and the partially calcified plaque score. The log-transformed Density-Volume Calcium score correlates very well with the log-transformed Agatston score, as we would expect as both are proxy measures of

underlying atherosclerotic disease. Both CAC scores show modest correlations with the TPS and the PCPS.

DISCUSSION

This is the first study to examine the relationship between a new score by CAC density/volume, and the total or partially non-calcified plaque burden on coronary CTA, independent of traditional CAD risk factors. We developed the new Density-Volume Calcium score that is derived from non-contrast CT scanning. The technical manner to measure CAC density is easy and similar to that for a well-established traditional CAC score; however, this method takes into account further important information regarding density and volume of calcified plaque by properly weighting plaque density to more accurately represent the potentially more vulnerable plaques that may increase risk for CVD events [13]. We demonstrated that participants with lower CAC density and greater volume have greater total plaque and partially calcified plaque scores on coronary CTA.

Most plaque ruptures causing acute coronary syndromes occur at the sites with non-calcified plaque components [14] and thus heavily calcified plaques, presented as higher calcium density on CT, may be more stable. This fact is in direct contrast to the well-established findings that greater Agatston score is associated with CAD events. Criqui et al. recently demonstrated that CAC density is inversely associated with CAD event risk in 3398 asymptomatic participants from the Multi-Ethnic Study of Atherosclerosis (MESA), after controlling for CAC volume [13]. In addition, while prevalence was low, prior studies found that vascular inflammation was confirmed by fluorodeoxyglucose positron emission tomography imaging at the sites of vascular calcifications on CT [15, 16]. These prior findings indicate that the Agatston CAC score may not be able to capture all of the important information with respect to plaque composition or burden in the presence of active inflammation that can be found on more detailed plaque imaging.

The high coronary plaque burden associated with greater CAC area or burden has been previously documented by several studies [1,17]. These studies highlighted the significant correlation between non-invasively measured CAC and pathologic features of overall coronary plaque area, confirming the concept of the predictive value of the traditional Agatston score by non-contrast CT to the overall underlying, and histologically significant, coronary plaque burden. Multiple studies also demonstrated the correlation between Agatston score and plaque burden by CTA. Nasir et al. demonstrated that an increase in Agatston CAC score was associated with a higher prevalence of the number of segments with any type of coronary plaque detected by coronary CTA and patients with CAC>100 possessed a 7-fold and 15-fold higher prevalence of ≥2 segments with calcified plaque and partially non-calcified plaque compared to those with CAC 1–10[18]. Recent investigations have demonstrated that regional CAC distribution in addition to Agatston CAC score also improves the correlation with total segment involvement scores (SIS), SIS with partially calcified plaque on coronary CTA, as well as cardiovascular events [19, 20]. These studies demonstrated the relation of higher Agatston CAC burden to coronary plaque components; especially, partially calcified plaque burden detected by contrast coronary CTA. While our findings are in line with these observations, we found a closer relationship between the

PCPS or TPS and a Density-Volume Calcium score when compared to the traditional Agatston CAC score. The underlying mechanism for why CAC density is associated with cardiovascular risks may be that CAC with lower density tends to be pathologically more unstable since progression of calcified plaque follows several pathways from inflammatory cells or apoptosis in plaque [21]. Prior autopsy studies showed that less calcification was observed in ruptured or vulnerable plaques compared to stable plaque among victims of sudden coronary death [21]. Another autopsy study showed an inverse correlation between extent of calcification and degree of cap inflammation [21, 22]. The findings of this study suggest that calcified plaque is more associated with a stable state of plaque and in this regard, should not be considered as being vulnerable [22]. A possible hypothesis of our findings showing a new score is more closely associated with total and partially calcified plaques is that higher calcium densities may be associated with an increased proportion of calcified plaque within a plaque, such as thickness or burden of calcified plaque. As such, on a per lesion or segment basis, a higher density calcified plaque may be less likely to contain non-calcified plaque components, which in turn suggests that lower density CAC is more associated with partially calcified or total plaque burden including more non-calcified plaque components.

In the aforementioned study, Criqui et al. raised the question of the integral role for the prognostic value of the calcium density and volume since the traditional Agatston CAC score is heavily weighted based upon greater calcium density and area [13] The authors demonstrated that the lower quartile density groups were more strongly associated with CVD risk. While these findings are very important, their method of calculating a density score, which divided the total CAC Agatston scores by the area of total plaques, may be limited because it does not take into account the vital information found within each CAC plaque, and does not allow integration into a single score. Thus, the fundamental question regarding the morphological information per an individual plaque remains unanswered. Compared to this method, our new method includes the volume and density in each individual plaque. Another potential role of the new CAC score may be that the score can be used as a novel marker to assess the therapeutic effect of statin on CAC. Since previous reports failed to show an independent connection of statin treatment to CAC regression overtime [23, 24], future studies are needed to examine if this novel Density-Volume Calcium score can predict CAC regression/progression on statin therapy.

Limitations

There are several limitations in the current study. We examined the current study among men who were enrolled in the MACS and included HIV infected and uninfected [9]. The application of our findings to women remains unknown. We used a semi-quantitative plaque score for the overall assessment of coronary atherosclerotic burden. Although it is time-consuming to manually measure all coronary plaque volumes, several newly developed plaque software are contributing more accurate evaluations of the coronary plaque burden[25, 26]. More data with respect to the association between Density-Volume Calcium score and quantitative total plaque burden will be required to confirm our concept and our group is currently continuing to examine such investigations. Given the limitation of the current study focusing on the relationship between CAC density-volume and plaque burden,

our results are only limited among patients with CAC, while our group recently reported the association between HIV status and non-calcified plaque among patients without CAC [27]. Lastly, although our new score demonstrates insights on potential underlying pathophysiology regarding the association between calcium density and plaque burden, a new scoring system built upon by predictive events, not by predicting plaque burden, is ideal. Based upon our current findings, a new CAC score may be required in the future. Additionally, the current score uses the mean density, whereas Agatston score uses the maximum density. The variability within a calcified plaque may provide more insights on calcium plaque stability.

Conclusion

The new Density-Volume Calcium score obtained from non-contrast CT demonstrated an improvement in estimating the number of partially calcified plaques and total plaque burden on coronary CTA relative to the traditional CAC Agatston score. Despite these promising results, the final determination of the optimal score to use will require follow-up events to demonstrate which score best represents cardiovascular risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

CAC	Coronary artery calcium
CCTA	Coronary computed tomographic angiography
CAD	Coronary artery disease
HIV	Human immunodeficiency virus

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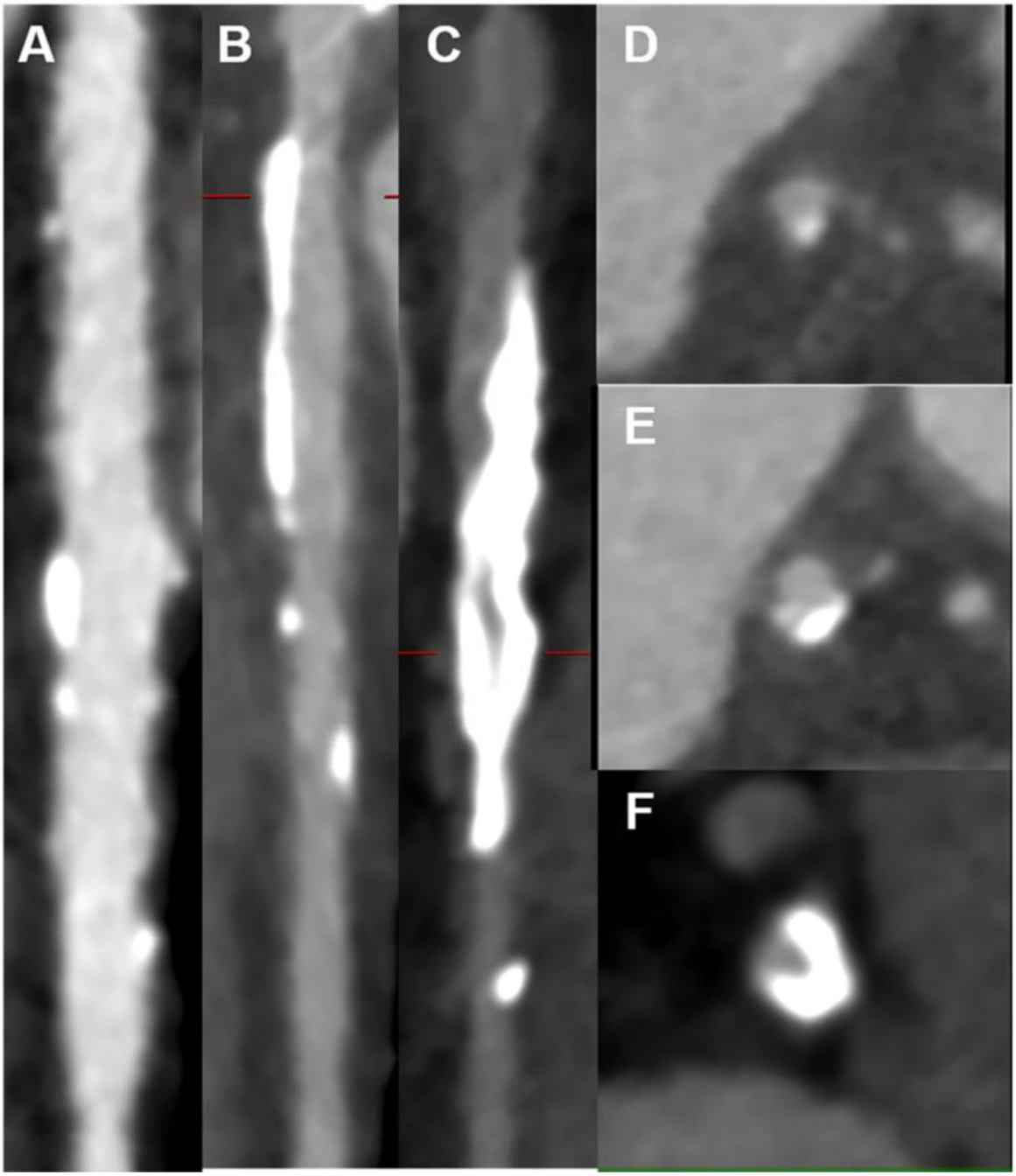

Figure 1. Examples of total plaque score.

A-C: Straight view of curved multiplanner reconstruction images, D-E: Cross-sectional images.

Table 1.

Descriptive statistics for the 347 participants from the Multicenter AIDS Cohort Study with coronary computed tomographic angiography and coronary artery calcium > 0.

	All participants (n=347)
Age (years)	58 ± 6
Body mass index (kg/m^2) (Mean ± SD)	26 ± 4
Race (%)	
White, non-Hispanic	246 (71%)
African American	74 (21%)
Hispanic/other	27 (8%)
Systolic blood pressure (mmHg)	129 ± 15
Diabetes (%)	35 (10%)
Smoking (%)	
Never	78 (23%)
Current	80 (23%)
Former	185 (54%)
Cumulative Pack-years smoking	17 ± 21
Glucose (mg/dl)	100 ± 27
Total cholesterol (mg/dl)	183 ± 39
LDL cholesterol (mg/dl)	106 ± 34
HDL cholesterol (mg/dl)	49 ± 15
Triglycerides (mg/dl)	147±102
Statin medication use (%)	155 (45%)
Diabetes medication use (%)	32 (9%)
Framingham risk score	9.8 ± 6.4

Numbers are count (%) for dichotomous variables and mean ± SD for continuous variables. SD- standard deviation

Table 2.

Non-contrast and contrast CTA findings

	All participants (n=347)
Non-contrast CT findings (median (IQR))	
Agatston score	82 (26, 205)
Density-volume score	195 (73, 476)
Number of plaques (all types), per participant	4 (2, 6)
Contrast CT findings (mean ± SD)	
Number of total plaques	4.3 ± 2.4
Number of non-calcified plaques	1.5 ± 1.8
Number of partially calcified plaques	1.2 ± 1.4
Number of calcified plaques	1.7 ± 1.3
Total plaque score	6.2 ± 4.3
Non-calcified plaque score	2.1 ± 2.6
Partially calcified plaque score	1.9 ± 2.6
Calcified plaque score	2.2 ± 2.7

CTA- computed tomographic angiography, CAC-coronary artery calcium

Table 3.

Univariate analysis to predict the number of total plaques and partially calcified plaques, as measured using contrast coronary computed tomographic scans.

Model 1. Number of total plaques per unit of Log-transformed score					
	# plaques (95% CI)	**p-value**		**# plaques (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	0.89 (0.78, 1.01)	<0.001	*Log (Agatston score) per standard deviation*	0.60 (0.52 to 0.68)	<0.001
Model adjusted r^2	0.40		*Model adjusted* r^2	0.36	
Model 2. Number of partially calcified plaques per unit of Log-transformed score					
	# plaques (95% CI)	**p-value**		**# plaques (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	0.36 (0.28, 0.44)	<0.001	*Log (Agatston score) per standard deviation*	0.24 (0.19, 0.29)	<0.001
Model adjusted r^2	0.20		*Model adjusted* r^2	0.18	
Model 3. Total plaque score					
	Plaque score (95% CI)	**p-value**		**Plaque score (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	1.67 (1.47, 1.87)	<0.001	*Log (Agatston score) per standard deviation*	1.13 (0.97, 1.27)	<0.001
Model adjusted r^2	0.44		*Model adjusted* r^2	0.40	
Model 4. Partially calcified plaque score					
	Plaque score (95% CI)	**p-value**		**Plaque score (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	0.76 (0.60, 0.89)	<0.001	*Log (Agatston score) per standard deviation*	0.49 (0.39, 0.60)	<0.001
Model adjusted r^2	0.24		*Model adjusted* r^2	0.21	

Models were estimated using linear regression without additional covariates.

Table 4.

Adjusted Associations between log-transformed density volume score or the log transformed Agatston score with the number of total plaques and partially calcified plaques or the total plaque and partially calcified plaque scores.

Model 1. Number of total plaques per unit of Log-transformed score					
	# plaques (95% CI)	**p-value**		**# plaques (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	0.86 (0.75, 0.98)	<0.001	*Log (Agatston score) per standard deviation*	0.58 (0.49 to 0.67)	<0.001
HIV infection	0.34 (−0.09, 077)	0.12	*HIV infection*	0.34 (−0.10 to 0.77)	0.13
Model adjusted r^2	0.42		*Model adjusted* r^2	0.39	
Model 2. Number of partially calcified plaques per unit of Log-transformed score					
	# plaques (95% CI)	**p-value**		**# plaques (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	0.37 (0.29, 0.45)	<0.001	*Log (Agatston score) per standard deviation*	0.24 (0.19, 0.29)	<0.001
HIV infection	0.20 (−0.08, 0.49)	0.17	*HIV infection*	0.22 (−0.06, 0.51)	0.12
Model adjusted r^2	0.24		*Model adjusted* r^2	0.18	
Model 3. Total plaque score					
	Plaque score (95% CI)	**p-value**		**Plaque score (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	1.64 (1.44, 1.85)	<0.001	*Log (Agatston score) per standard deviation*	1.09 (0.95, 1.24)	<0.001
HIV infection	0.76 (0.0\3, 1.44)	0.04	*HIV infection*	0.75 (−0.01, 1.50)	0.05
Model adjusted r^2	0.47		*Model adjusted* r^2	0.44	
Model 4. Partially calcified plaque score					
	Plaque score (95% CI)	**p-value**		**Plaque score (95% CI)**	**p-value**
Log (Density-Volume score) per standard deviation	0.76 (0.61, 0.91)	<0.001	*Log (Agatston score) per standard deviation*	0.49 (0.39, 0.60)	<0.001
HIV infection	0.43 (−0.09, 0.96)	0.10	*HIV infection*	0.43 (−0.14, 0.95)	0.12
Model adjusted r^2	0.25		*Model adjusted* r^2	0.22	

Models were estimated using linear regression, adjusted for (in addition to HIV): Age, systolic blood pressure, diabetes, total cholesterol, HDL cholesterol, glucose, cumulative pack-years smoking (current, former, never), race/ethnicity, body mass index, and statin medication use.

Table 5.

Correlation matrix between different plaque scores

N=347	*Log (Density-Volume Score)*	*Log (Agatston Score)*	*Total Plaque Score*	*Partially calcified Plaque Score*
Log (Density-Volume Score)	1.00	0.96	0.67	0.49
Log (Agatston Score)	0.96	1.00	0.63	0.46
Total Plaque Score	0.67	0.63	1.00	0.64
Partially calcified Plaque Score	0.49	0.46	0.64	1.00

All correlations are statistically significant at $p < 0.001$