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Coronary Plaque Prevalence Among Black Women Living with HIV

By

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MD, Rutgers New Jersey Medical School, 2019**

Advisor: Jordan A. Kempker, MD, MSc

**An abstract of
A thesis submitted to the Faculty of the James T. Laney School of Graduate Studies of Emory
University
in partial fulfillment of the requirements for the degree of
Master of Science
in Clinical Research
2025**

Abstract

Coronary Plaque Prevalence Among Black Women Living with HIV

By Nishant Vatsa

Introduction

Previous studies indicate that people living with human immunodeficiency virus (HIV) have a greater burden of coronary artery plaque, especially noncalcified plaque. However, many of these studies primarily included white males receiving outdated HIV management practices. A contemporary investigation examining plaque prevalence in Black women living with HIV (WLH) is needed.

Methods

We performed Coronary Computed Tomography Angiography (CCTA) in 86 Black WLH and women without HIV (WWoH) asymptomatic of cardiac symptoms from the Atlanta metro area (70.9% WLH, median age: 55 [49.25, 59]). Individuals with calcified, noncalcified, mixed plaque or plaque with positive remodeling, low attenuation, napkin ring sign, and spotty calcification features on visual or semi-automated CCTA analyses were categorized as having CCTA plaque. We compared plaque prevalence across HIV strata in the overall sample and among those with low cardiac risk per coronary artery calcium (CAC) or pooled cohort equation (PCE) scoring. Logistic regression models adjusted for clinical and socioeconomic cardiac risk factors were performed to assess the independent association between HIV status and CCTA plaque prevalence.

Results

Our sample had a high burden of clinical risk factors and socioeconomic risk factors, with 54.65% having hypertension, 40.51% currently smoking, and 51.16% living below the poverty line. These risk factors were broadly similar between WLH and WWoH, except WWoH were more likely to have diabetes (32% vs 9.84%, $p=0.02$). Although there was a high prevalence of CCTA plaque in the sample, WLH were not more likely to have CCTA plaque than WWoH (52.46% vs 52%, $p=0.97$). Adjusted regression analysis yielded similar results, with low income being the only covariate independently associated with CCTA plaque (OR [95% CI]: 5.65 [1.86, 19.3], $p=0.003$). Among those with a CAC score of 0 or $PCE < 7.5\%$, WLH were numerically more likely to have CCTA plaque, but this did not reach statistical significance.

Conclusion

Despite HIV not being associated with coronary plaque prevalence, we saw a high burden of coronary artery disease in this cohort of Black women asymptomatic of cardiac symptoms, even among those at low cardiac risk. Asymptomatic Black women with elevated socioeconomic and clinical risk may need more stringent cardiac screening.

Coronary Plaque Prevalence Among Black Women Living with HIV

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Narrative Review:

Due to advances in antiretroviral therapy (ART), which have decreased the mortality rate of people living with Human Immunodeficiency Virus (HIV), HIV has become a highly prevalent, morbid chronic disease – affecting over 38 million people. [1] As a result, much of the morbidity in people living with HIV (PLH) is due to cardiovascular disease, with studies estimating about a 1.4 to 2-fold increased risk of myocardial infarction in PLH. [2-5] Increased risk of cardiovascular disease is most pronounced in women living with HIV (WLH), who have a 3-fold increased risk of myocardial infarction. [4, 6] Recent evidence of the benefit of statins in WLH suggests this previously described residual cardiac risk could be due to ART and viral replication-mediated chronic inflammation, which provides the substrate for coronary artery disease with high-risk plaque (HRP). [7-9]

Because HRP and noncalcified plaque are predictive of adverse cardiac outcomes and associated with pro-inflammatory states, such as HIV, there has been a push to characterize plaque composition in PLH. [10, 11] Due to advances in imaging technology, providers can detect atherosclerotic plaque by non-invasive modalities, such as coronary computed tomography angiography (CCTA). CCTA provides granular information, such as plaque composition and vessel stenosis measures, that provide cardiac prognostication beyond traditional cardiac risk stratification tools. [12-14]

Studies utilizing coronary CT angiography (CCTA) have shown a high prevalence of noncalcified plaque and HRP in cohorts of PLH. [7, 8, 15] However, many of these studies were done before 2015, when HIV therapy guidelines shifted, and were done in cohorts mainly consisting of white men. [7, 8, 15, 16] A contemporary investigation of plaque composition in asymptomatic Black WLH, who are at increased risk of cardiovascular events than other PLH groups, has yet to be done. [17] Better characterization of the prevalence of coronary artery disease and HRP in Black WLH could inform future primary prevention strategies for adverse cardiac outcomes for this group. In Emory's Bone, Brain, and Heart (BBH) study, our primary aim was to characterize coronary plaque prevalence and composition in

asymptomatic Black WLH from the Atlanta metro area by comparing this group to a similar at-risk cohort living without HIV.

Despite the added prognostic value of CCTA plaque composition and stenosis measures, studies utilizing CCTA in asymptomatic individuals are sparse. Studies of American Hispanic, American Non-Hispanic White, and European asymptomatic cohorts found greater than 40% plaque prevalence with greater than 25% non-calcified plaque prevalence in these cohorts. [2, 18] However, a study assessing plaque prevalence in asymptomatic, Black women has yet to be done. [19] So, our secondary aim was to understand better coronary plaque prevalence in a cohort of Black women asymptomatic of coronary disease.

Methods:

Study Design

The BBH study is a sub-study of the Atlanta Women's Interagency HIG Study (WIHS) conducted at Emory University from 2019 to 2023. Women from WIHS who were ≥ 30 years old living with well-controlled HIV – defined as having HIV RNA of <50 copies/mL undergoing antiretroviral therapy for ≥ 2 years – and demographically matched women without HIV (WWoH) were enrolled between 2019-2023 from Atlanta-metropolitan clinics. We excluded women who were breastfeeding, pregnant, non-ambulatory, or had significant co-morbidity, such as a history of organ transplantation, bone disorders, autoimmune disorders, cancer, chronic liver disease, chronic kidney disease, CAD, acute coronary syndrome history, heart failure, or stroke.

Women enrolled in the BBH study underwent entry visits, where we collected anthropometric measurements and blood samples, including lipid profiles. Additionally, participants self-reported medication history, sociodemographic information, and cardiovascular risk factor history at entry visits, which we confirmed with a medical record review. [20] Demographics, clinical risk factors history, and social determinants of health variables, such as age, race, history of hypertension, diabetes,

hyperlipidemia, diabetes, statin use, current smoking status, income, marital status, and education level, were self-reported by participants in interviews and confirmed with medical record review. Within six months of BBH entry visits, participants were required to complete cardiac imaging, which included coronary CT angiograms (CCTA) and non-contrast gated chest computed tomography (CT). Eighty-six black women in the study completed CCTA. (**Figure 1**)

Cardiac Imaging

Patients underwent CCTA using the Siemens Somatom Force, Somatom Definition AS, or a Somatom Definition Flash at two Emory-affiliated sites. Based on the participants' body habitus, scanner tube voltage ranged from a peak of 80 to 120 kVp with currents between 320 and 580mAs. During imaging, participants' heart rates were maintained below 70 beats per minute by administering oral or intravenous metoprolol at the discretion of a supervising physician. Reconstructed CCTA images were analyzed by two independent cardiovascular imaging specialists blinded to patient characteristics. Based on plaque attenuation and features, specialists visually identified different coronary plaque subtypes, including calcified plaque, noncalcified plaque, mixed plaque, spotty calcification, napkin ring sign, and positive remodeling. In addition to visual identification, we also utilized Elucid (VascuCAP, Wenham, MA), an artificial intelligence (AI) enhanced semi-automotive software, to detect low attenuation plaque (LAP) (<45 Hounsfield Units) and calcified plaque (>250 Hounsfield Units). There was a high level of accordance between readers' CT interpretations, with inter-reader variability in identifying different plaque subtypes having Pearson's coefficient greater than 0.9. [21] Individuals with any of the aforementioned plaque subtypes on visual or AI-enhanced CT assessments were categorized as having CCTA plaque. High-risk plaque was defined as the presence of coronary plaque with spotty calcification, napkin ring sing, positive remodeling, or low attenuation features. To evaluate coronary stenosis, we used the CAD-RADS reporting system, in which CAD-RADS 0 confers 0% maximal stenosis, CAD-RADS 1 confers 1-24% maximal stenosis, CAD-RADS 2 confers 25-49% maximal stenosis, CAD-RADS 3 confers 50-

69% maximal stenosis, CAD-RADS 4 confers 70-99% maximal stenosis, and CAD-RADS 5 confers 100% maximal stenosis. CAD-RADS 3 or higher equated to obstructive coronary artery disease. [22]

Statistical Analysis

Categorical variables are presented as proportions, and continuous variables as means (SD) or medians [IQR]. Wilcoxon rank sum tests and two-sample t-tests were used to compare non-parametric and parametric continuous variables across HIV strata. Chi-square and Fisher exact tests were used to compare categorical variables, including the prevalence of CCTA plaque and plaque subtypes, across HIV strata. We performed adjusted and stratified analyses to control for confounders contributing to coronary artery disease.

For the adjusted analysis, we regressed the prevalence of CCTA plaque onto HIV status, socioeconomic cardiac risk factors, and clinical cardiac risk factors in logistic regression analyses. These risk factors were selected a priori and included: body mass index, age, diabetes, hypertension, hyperlipidemia, current smoking status, creatinine, income, education, statin use, and marital status. To determine whether HIV modified the association of cardiac risk factors and CCTA plaque, we utilized the fully adjusted logistic regression model to examine interactions between HIV and the covariates above.

The coronary artery calcium (CAC) score and the atherosclerotic cardiovascular disease (ASCVD) 10-year risk score calculated by the pooled cohort equation (PCE) are cardiac risk stratification tools ubiquitously used in current clinical practice, so we used these two measures for our stratification analysis. We calculated coronary calcium scores for our cohort by analyzing the volume and density of coronary calcium on non-contrast computed tomography studies. We calculated PCE scores for our sample using lipid profile, hypertension history, smoking history, sex, race, and hypertension medication history data. In our stratification analysis, we performed pairwise comparisons of coronary plaque prevalence across HIV strata among those with a CAC score of 0 or PCE < 7.5% - the lowest cardiac risk groups per CAC and PCE scoring. [23, 24]

Results:

Sample Characteristics

The median age of our sample of 86 Black women was 55 [49.25, 59], and over two-thirds of our participants had HIV (n=61, 70.93%). Overall, there was a high prevalence of hypertension (n=47, 54.65%) and current smokers (n=40, 40.51%). The mean BMI of the sample was in the class 1 obesity range (33.92 (8.2) kg/m²), but serum cholesterol levels were reasonably controlled (median LDL [95% CI]: 104 [83.5, 123] mg/dL; median HDL [95% CI]: 55 [47, 68.5]). Accordingly, the median PCE score corresponded to low to borderline cardiac risk (5.84% [2.97, 11.12]), with 29 women (58%) having PCE scores of less than 7.5%. There was a high burden of adverse social determinants of health in our sample, with nearly a third having less than high school education (n=26, 30.23%), less than a fifth being married or with a partner (n=15, 17.44%), and over half having an income of less than 12,001 dollars (n=44, 51.16%) (**Table 1**).

Plaque Prevalence, Overall Sample

On visual and semi-automotive CCTA assessments, 45 participants (52.33%) had CCTA plaque. This high prevalence of CCTA plaque was primarily driven by the large proportion of women having calcified (n=25, 29.07%) and high-risk plaque (n=33, 38.37%), with many participants having low attenuation plaque (n=28, 32.56%) (**Figure 1A**). The high prevalence of plaque in this cohort corresponded to a high prevalence of coronary stenosis, as 32 (37.21%) participants had a CAD-RADS of 1 or greater, with two women (2.33%) having obstructive coronary stenosis (2.33%) (**Table 1**).

On non-contrast CT assessments, 24 (33.3%) individuals in our sample had CAC scores above 0, and the median CAC score of all participants was 0 [0, 17.25%] (**Table 1**). Among the 48 women with a CAC score of 0 (66.67%), almost one-third of the study participants had CCTA plaque (n=15, 31.25%) that mainly consisted of low attenuation plaque (n=14, 29.17%) (**Figure 1B**). Of participants at PCE calculated

low to borderline cardiac risk (PCE score<7.5%), the high prevalence of calcified (n=10, 34.48%) and low attenuation plaque (n=8, 27.59%) persisted (**Figure 1C**).

Plaque Prevalence by HIV Status

Outside of diabetes, which was significantly more prevalent in women without HIV (8 (32%) women vs. 6 (9.84%) women, $p=0.02$), demographic, clinical risk factor, and social determinants of health profiles were similar between women living with HIV and women living without HIV. Consequently, PCE scores did not significantly differ between the groups. However, women without HIV had numerically higher PCE scores than women living with HIV (10.27 [3.47, 18.67] % vs. 5.03 [3.18, 8.92] %, $p=0.29$) and were less likely to have PCE scores below 7.5% (4 (36.36%) vs 25 (64.1%), $p=0.2$) (**Table 1**).

Although CCTA plaque was highly prevalent in WLH, in the unadjusted analyses WLH were not significantly more likely to have CCTA plaque than women without HIV (32 (52.46%) vs 13 (52%), $p=0.97$) (**Table 1, Figure 2A**). Women without HIV were more likely to have obstructive coronary artery disease (>50% stenosis) compared to women living with HIV (2 (8%) vs 0 (0%), $p=0.032$) (**Table 1**). CAC prevalence was similar between WLH and women without HIV (CAC>0: 17 (34%) vs 7 (31.82%), $p=0.86$). Among women with no CAC, there was a numerically higher prevalence of CCTA plaque in WLH than women without HIV (12 (36.36%) vs. 3 (20%), $p=0.33$) due to the higher prevalence of low attenuation plaque in WLH compared to women without HIV (11 (33.33%) vs. 3(20%), $p=0.5$) (**Figure 2B**). Of participants with PCE calculated low to borderline cardiac risk, WLH were numerically more likely to have CCTA plaque than women without HIV (CCTA Plaque: 12 (48%) vs. 1 (25%), $p=0.61$) (**Figure 2C**).

Determinants of CCTA Plaque

When regressing CCTA plaque prevalence onto demographics, clinical risk factors, and socioeconomic risk factors in regression models, HIV was not associated with CCTA plaque (OR [95% CI]: 0.97 [0.27, 3.67], $p=0.97$). In the above model, those with a low income (<12,001\$) had significantly

higher odds of having CCTA plaque compared those with higher income (>12,000\$) (OR [95% CI]: 5.65 [1.86, 19.3], $p=0.003$), while having hypertension was nearly associated with CCTA plaque prevalence (OR [95% CI]: 3.24 [0.94, 12.4], $p=0.07$) (**Table 2**). In fully adjusted models, HIV did not modify the association between clinical or socioeconomic cardiac risk factors and the prevalence of CCTA plaque.

Discussion:

We report a high prevalence of coronary plaque among Black women with HIV and without coronary symptoms. The high prevalence of coronary artery disease in this group was primarily driven by low attenuation plaque and calcified plaque. Coronary artery disease was also highly prevalent in Black women without HIV and coronary symptoms, such that CCTA plaque prevalence and composition were not significantly different between women with HIV and women without HIV. Among those with low cardiac risk, as determined by a CAC score of 0 or PCE scores below 7.5%, the prevalence of coronary plaque remained high, with women with HIV being numerically more likely to have coronary plaque than women without HIV.

Our results showing similar calcified plaque prevalence between women with HIV and women without HIV is consistent with previous reports; however, unlike previous literature, we do not show a difference in noncalcified plaque across HIV status in a cohort of Black women.[8, 25] Post, D'Ascenzo, and colleagues reported a higher prevalence of noncalcified plaque among those with HIV compared to those without HIV in cohorts that were predominately white and male. [8, 25] The demographic differences between the above studies and our cohort likely contributed to these varied findings. Changes in treatment guidelines could also account for our different results. Unlike previous studies showing an association between HIV and coronary plaque, our study was performed in an era when protease inhibitors, which have been associated with cardiac disease, are sparsely used, and HIV therapy is initiated earlier than in previous guidelines. [16, 26] Finally, there was a high prevalence of cardiovascular risk factors in this cohort, making it more difficult to measure an effect of HIV on

coronary plaque prevalence in this study. [8, 25] When restricting our cohort to subgroups with low cardiovascular risk, as measured by the CAC or PCE scoring, Black WLH had a higher prevalence of coronary plaque than Black WWoH. These results in Black women with lower cardiovascular risk suggest that well-controlled HIV may be a risk-enhancing factor for coronary artery disease that contributes to heart disease less than traditional risk factors.

This cohort of Black women asymptomatic for CAD had a high prevalence of coronary plaque, which was higher than what has been reported in American Hispanic, American Non-Hispanic White, and European asymptomatic women. [2, 18] Furthermore, we observed a higher prevalence of coronary plaque in Black women with HIV than what was described in the Randomized Trial to Prevent Vascular Events (REPREIVE) cohort of mostly non-Black people with HIV. [15] The high prevalence of CAD in this cohort could be attributed to the unique socioeconomic profile of this sample. As evidenced by the high proportion of women in this cohort without a high school degree and an income below 12,001 dollars per year, this sample had a lower socioeconomic status – a risk factor for coronary disease – than samples previously reported on. [27, 28] The strong association between income and prevalence of coronary plaque in logistic models adjusted for traditional risk factors underscores the impact of socioeconomic status on coronary disease in this sample.

Current preventive cardiac care utilizes risk calculators, like the PCE, or CAC scoring from non-contrast CT imaging to prognosticate patients. [29, 30] However, as shown by the high prevalence of overall coronary plaque among individuals in our cohort with low cardiac risk according to PCE or CAC scoring, these clinical tools may not accurately reflect coronary artery disease burden. Previous studies have reported similar findings in “low-risk” individuals, with 5 to 16% of individuals with no coronary artery calcium on non-contrast CT imaging having coronary plaque on CCTA. [2, 18] This and the studies mentioned above highlight the limitations of current risk stratification tools, especially in women with risk-enhancing factors such as HIV and low income. [29] Consequently, there may be utility in CCTA

imaging to better risk stratify groups that are disproportionately affected by cardiovascular disease, like asymptomatic Black women with risk-enhancing clinical and socioeconomic factors, leading to more timely pharmacologic and lifestyle primary prevention therapies for these groups.[31-33]

Limitations

Our study's small sample size could have prevented us from fully measuring an association between HIV status and different coronary plaque subtypes. More extensive studies are needed to characterize the impact of well-controlled HIV on coronary artery disease in Black women without coronary symptoms. Although this study investigates the prevalence of coronary artery disease in a sociodemographic group that has been sparsely reported on, these results may not be generalizable to different socioeconomic or demographic groups. As such, more studies that investigate the prevalence and composition of coronary plaque in asymptomatic cohorts that are different from the one in this study are warranted. Finally, we did not have detailed information on our patient's menopause status, a risk factor for cardiovascular disease in women. Future similar investigations should incorporate menopause status as a potential confounder.

Conclusions

We report a high prevalence of coronary plaque that was driven mainly by calcified and low-attenuation plaque subtypes among Black women asymptomatic of coronary symptoms. Among the overall cohort, these trends persisted in those with low cardiac risk, as measured by PCE and CAC scoring. Despite numerical trends of higher plaque prevalence in WLH compared to WWoH among women with low cardiac risk, there was no statistical difference in coronary plaque prevalence or composition among WLH and WWoH in our overall sample. Instead, coronary plaque prevalence seemed to be most associated with socioeconomic factors. Future investigations should explore whether employing more stringent screening practices in Black women without coronary symptoms but with high clinical and socioeconomic risk improves cardiovascular outcomes.

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Table 1: Sample Characteristics by HIV Status

Table 2: Adjusted associations between listed cardiovascular and sociodemographic cardiovascular risk factors and coronary plaque prevalence. Covariates include HIV (ref: no HIV), body mass index, age, creatinine, diabetes (ref: no diabetes history), hyperlipidemia (ref: no hyperlipidemia history), hypertension (ref: no hypertension history), current smoking (ref: no current smoking), statin use (ref: no statin use), income (ref: >12,000\$), education (ref: below high school education), and marital status (ref: married/partner). Significant associations are denoted with $p < 0.05$.

Figure 1: Overall plaque prevalence and subtype plaque prevalence in **(A)** the overall sample ($n=86$), **(B)** among individuals with a CAC score of 0 ($n=48$ (66.7%)), and **(C)** among individuals with low or borderline cardiac risk (PCE score $< 7.5\%$, $n=29$ (58%)).

Figure 2: Overall plaque prevalence and subtype plaque prevalence by HIV subtype in **(A)** the overall sample (WLH: 61 (70.9%), WWoH: 25 (29.1%)), **(B)** among individuals with a CAC score of 0 (WLH: 33 (68.8%), WWoH: 15 (31.3%)), and **(C)** among individuals with low or borderline cardiac risk (PCE score $< 7.5\%$, WLH: 25 (86.2%), WWoH: 4 (13.8%)).

Variable	HIV Status			P-value ²
	Overall, N = 86 ¹	Negative, N = 25 ¹	Prevalent, N = 61 ¹	
Age (years)	55 [49.25, 59]	54 [49, 60]	55 [50, 58]	0.95
Diabetes	14/86 (16.28%)	8/25 (32.00%)	6/61 (9.84%)	0.02
Hypertension	47/86 (54.65%)	16/25 (64.00%)	31/61 (50.82%)	0.26
Hyperlipidemia	15/86 (17.44%)	2/25 (8.00%)	13/61 (21.31%)	0.21
Current Smoker	40/86 (46.51%)	13/25 (52.00%)	27/61 (44.26%)	0.51
Body Mass Index (kg/m²)	33.92 (8.20)	35.29 (9.53)	33.34 (7.58)	0.37
LDL (mg/dL)	104 [83.5, 123]	105.5 [90.5, 109.25]	100 [83.5, 130]	0.68
HDL (mg/dL)	55 [47, 68.5]	63 [48, 75.25]	54 [47, 64]	0.27
Statin Use	18/86 (20.93%)	4/25 (16.00%)	14/61 (22.95%)	0.47
Creatinine	0.90 (0.70, 1.10)	0.80 (0.70, 0.90)	0.90 (0.75, 1.10)	0.12
10-Year ASCVD Risk (%)	5.84 (2.97, 11.12)	10.27 (3.47, 18.67)	5.03 (3.18, 8.92)	0.29
Education				0.16
Below High School	26/86 (30.23%)	7/25 (28.00%)	19/61 (31.15%)	
High School	29/86 (33.72%)	12/25 (48.00%)	17/61 (27.87%)	
Above High School	31/86 (36.05%)	6/25 (24.00%)	25/61 (40.98%)	
Income				0.89
\$0-\$12K	44/86 (51.16%)	14/25 (56.00%)	30/61 (49.18%)	
\$12001-\$24K	21/86 (24.42%)	6/25 (24.00%)	15/61 (24.59%)	
>\$24K	14/86 (16.28%)	4/25 (16.00%)	10/61 (16.39%)	
Marital Status				0.74
Married/Partner	15/86 (17.44%)	3/25 (12.00%)	12/61 (19.67%)	

Variable	HIV Status			P-value ²
	Overall, N = 86 ¹	Negative, N = 25 ¹	Prevalent, N = 61 ¹	
Divorced/Widowed/Separated	34/86 (39.53%)	10/25 (40.00%)	24/61 (39.34%)	
Never married/Other	37/86 (43.02%)	12/25 (48.00%)	25/61 (40.98%)	
CCTA Plaque	45/86 (52.33%)	13/25 (52.00%)	32/61 (52.46%)	0.97
Mixed Plaque	11/86 (12.79%)	4/25 (16.00%)	7/61 (11.48%)	0.72
Calcified Plaque	25/86 (29.07%)	9/25 (36.00%)	16/61 (26.23%)	0.36
Noncalcified Plaque	3/86 (3.49%)	1/25 (4.00%)	2/61 (3.28%)	>0.99
High-Risk Plaque	33/86 (38.37%)	10/25 (40.00%)	23/61 (37.70%)	0.84
Low Attenuation	28/86 (32.56%)	9/25 (36.00%)	19/61 (31.15%)	0.66
Spotty Calcification	2/86 (2.33%)	0/25 (0.00%)	2/61 (3.28%)	>0.99
Positive Remodeling	7/86 (8.14%)	4/25 (16.00%)	3/61 (4.92%)	0.19
CADRADS				0.032
0	54/86 (62.79%)	15/25 (60.00%)	39/61 (63.93%)	
1	20/86 (23.26%)	3/25 (12.00%)	17/61 (27.87%)	
2	10/86 (11.63%)	5/25 (20.00%)	5/61 (8.20%)	
3	2/86 (2.33%)	2/25 (8.00%)	0/61 (0.00%)	
Calcium Score	0.00 [0.00, 17.25]	0.00 [0.00, 34.50]	0.00 [0.00, 15.50]	0.85
CAC Prevalence	24/72 (33.33%)	7/22 (31.82%)	17/50 (34.00%)	0.86

¹ Median (IQR); n/N (%); Mean (SD); Median [IQR]

² Wilcoxon rank sum test; Fisher's exact test; Pearson's Chi-squared test; Welch Two Sample t-test; Wilcoxon rank sum exact test

Table 1: Sample Characteristics by HIV Status

Characteristic	OR ¹	95% CI ¹	P-value
HIV	0.97	0.27, 3.67	0.97
Age (years)	1.07	0.98, 1.18	0.15
Diabetes	0.34	0.06, 1.83	0.21
Hyperlipidemia	0.57	0.11, 2.99	0.5
Hypertension	3.24	0.94, 12.4	0.07
Smoking	0.64	0.19, 2.05	0.45
Creatinine (mg/dL)	0.49	0.05, 4.91	0.53
Body Mass Index (kg/m ²)	1.01	0.94, 1.08	0.85
Statin Use	2.96	0.70, 14.3	0.15
Income	5.65	1.86, 19.3	0.003
Education	0.43	0.12, 1.43	0.18
Marital Status	1.84	0.47, 7.68	0.38

¹ OR = Odds Ratio, CI = Confidence Interval

Table 2: Adjusted associations between listed cardiovascular and sociodemographic cardiovascular risk factors and coronary plaque prevalence. Covariates include HIV (ref: no HIV), age, body mass index, age, creatinine, diabetes (ref: no diabetes history), hyperlipidemia (ref: no hyperlipidemia history), hypertension (ref: no hypertension history), current smoking (ref: no current smoking), statin use (ref: no statin use), income (ref: $\geq 12,000$ \$), education (ref: below high school education), and marital status (ref: married/partner). Significant associations are denoted with $p < 0.05$.

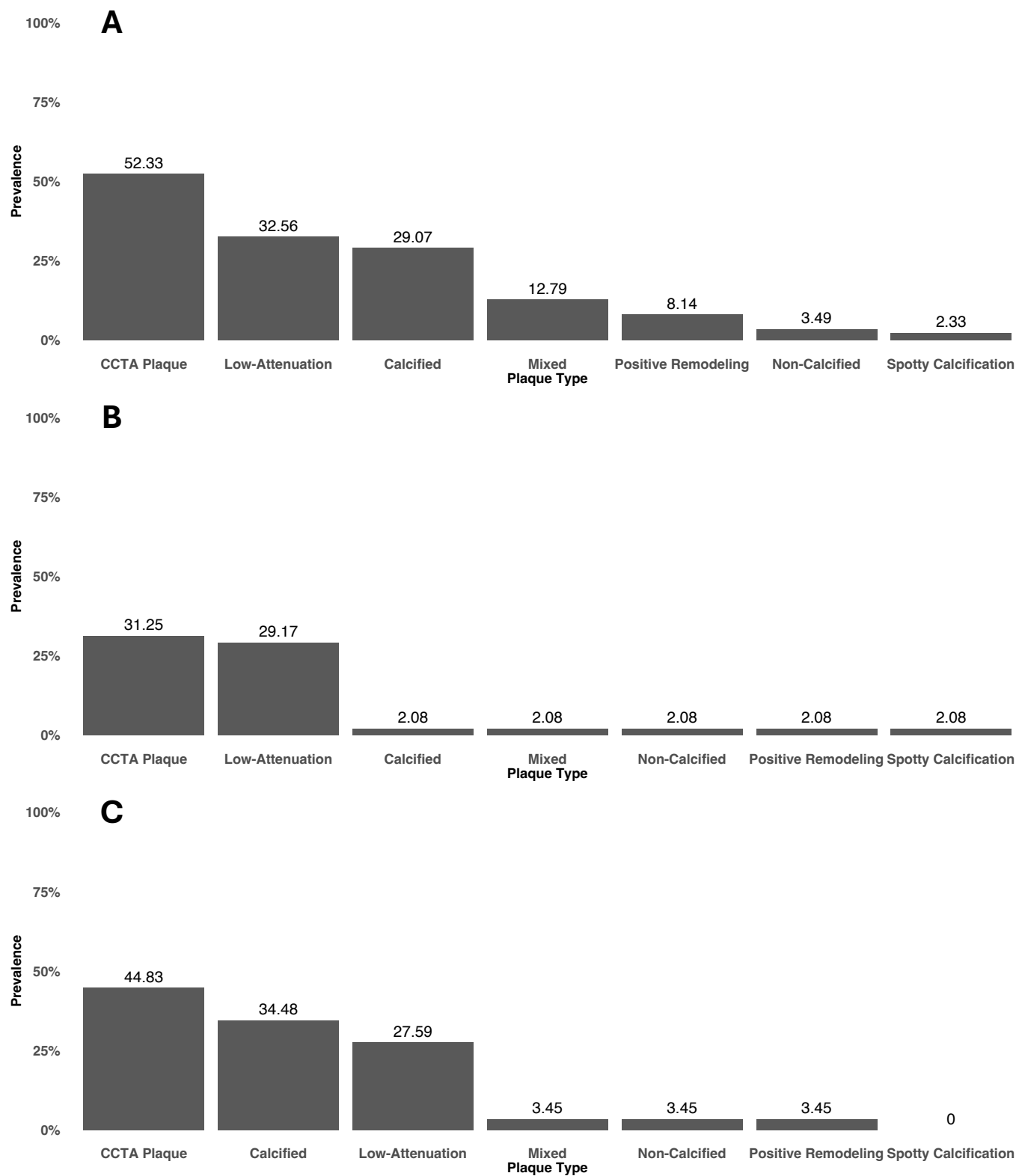


Figure 1: Overall plaque prevalence and subtype plaque prevalence in (A) the overall sample (n=86), (B) among Black women with a CAC score of 0 (n=48 (66.7%)), and (C) among individuals with low or borderline cardiac risk (PCE score <7.5%, n=29 (58%)).

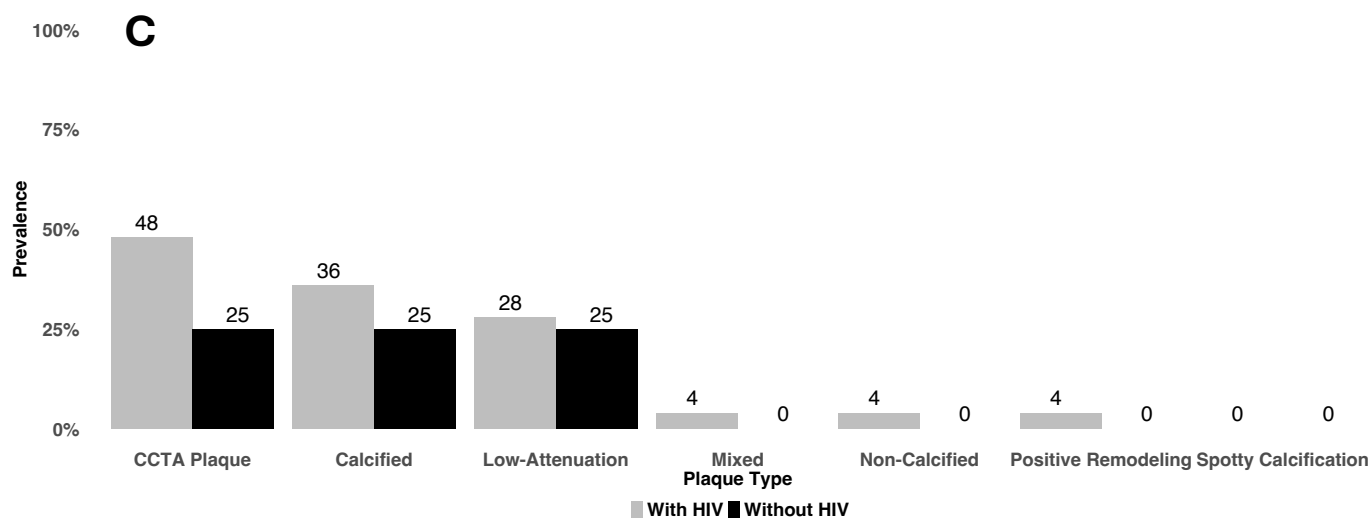
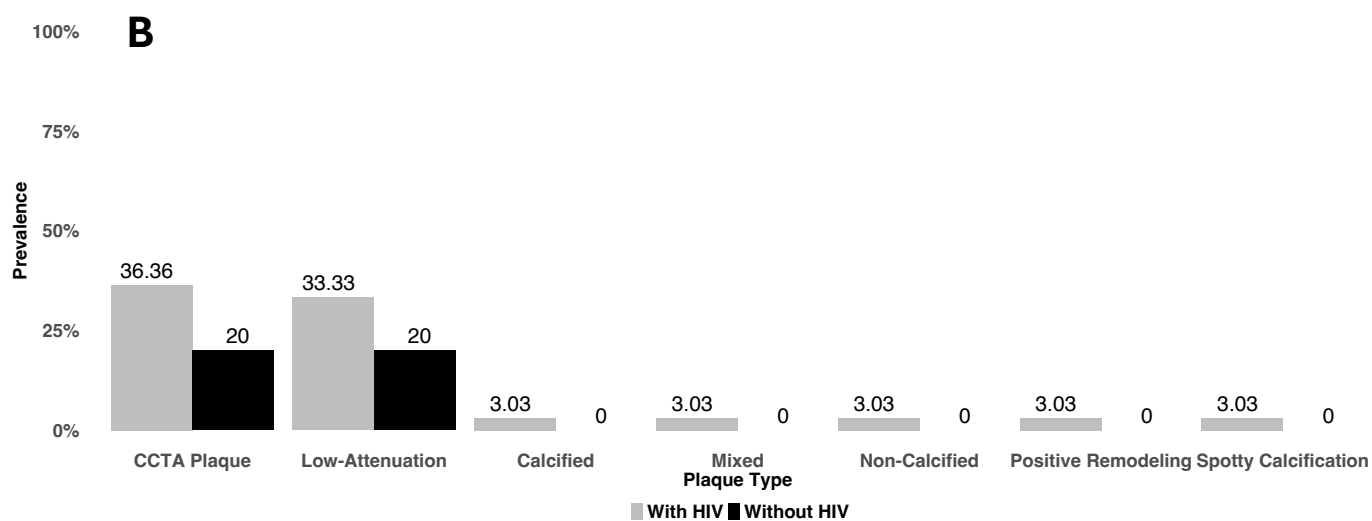
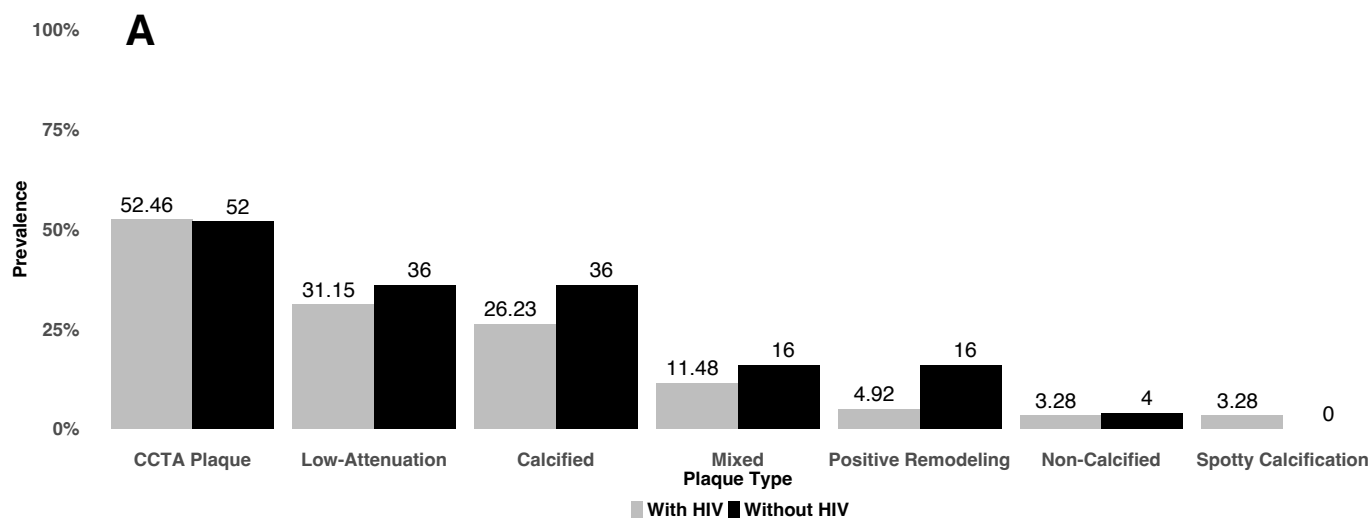


Figure 2: Overall plaque prevalence and subtype plaque prevalence by HIV status in (A) the overall sample (WLH: 61 (70.9%), WWoH: 25 (29.1%)), (B) among Black women with a CAC score of 0 (WLH: 33 (68.8%), WWoH: 15 (31.3%)), and (C) among individuals with low or borderline cardiac risk (PCE score <7.5%, WLH: 25 (86.2%), WWoH: 4 (13.8%)).

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