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Epidemiology of Coronary Atherosclerosis among People Living with HIV in Uganda: A Cross-Sectional Study

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Abstract

Background—HIV is an established risk factor for coronary atherosclerotic disease (CAD) in the United States and Europe, but data in the African region are lacking.

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Objective—To determine whether well-controlled HIV infection in Uganda is associated with increased presence or severity of CAD.

Design—Cross-sectional cohort study.

Setting—Southwestern Uganda.

Participants—People over 40 years of age, living with HIV, and on antiretroviral therapy (ART) for 3+ years, and population-based, age- and sex-similar people without HIV (PWoH).

Measurements—Participants underwent cardiovascular disease (CVD) risk profiling and computed tomography (CT) for detection of CAD, defined as presence of calcified or non-calcified plaque.

Results—586 participants met inclusion criteria and had evaluable images. Of these, 287 (49.0%) were PWH and nearly all (272/287, 97%) were virologically suppressed. Prevalence of CAD was low overall (54/586, 7.7%) and similar between PWH (26/287, 9.1%) and PWoH (19/299, 6.4%; absolute prevalence difference 2.7%, 95%CI −1.6, 7.0%). Findings were similar in multivariable regression models adjusted for CVD risk factors (adjusted prevalence ratio for PWH vs. PWoH 1.57, 95%CI 0.90, 2.74, P=0.11).

Limitations—Our findings may not generalize to symptomatic populations or those with known CAD. We cannot exclude residual confounding between HIV and CAD, and we may have inadequate power to detect increased prevalence of CAD.

Conclusion—We found no difference in the prevalence of CAD or non-calcified plaque between PWH and PWoH in Uganda in the ART era and extremely low prevalence compared to other risk-similar population-based cohorts in the Global North. Our results suggest that CAD may not be a major cause of morbidity in PWH in Uganda.

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Introduction

In the United States and Europe, people living with HIV (PWH) have an increased prevalence of coronary atherosclerotic disease (CAD),(2,3) a higher incidence of myocardial infarction,(4–7) and greater burden of cardiovascular (CV) mortality compared to age similar people without HIV (PWoH).(8–10) Although the mechanisms are not fully characterized, the increased risk of CAD among PWH appears to be partially due to increased HIV-associated subclinical immune activation, and is mitigated by antiretroviral therapy (ART)-mediated viral suppression.(11) Nonetheless, the increased risk of CV events persists despite suppressive ART; and, as such, HIV is now considered to be an independent CV risk equivalent.(12)

The contribution of HIV to CAD risk, and the potential mitigating role for lipid-lowering and anti-inflammatory therapies to prevent CAD events was recently corroborated by the REPRIEVE trial, which demonstrated an approximate 30% reduced risk of major adverse cardiovascular events (MACE) with the use of statins in PWH at low to moderate risk of CVD events.(1,13) Subsequent to this trial, HIV treatment guidelines in the United States

and United Kingdom now recommend statins in people living with HIV over 40 years of age, and at low to intermediate risk of MACE.(14,15)

Notwithstanding the compelling evidence linking HIV infection with CAD risk in the United States and Europe, similar data from low- and middle-income countries are sparse. People residing in sub-Saharan African region were included in the REPRIEVE trial, with over 1,000 participants recruited from sites in Botswana, South Africa and Uganda. However, there were only 12 captured MACE events during follow-up at those sites in both study arms.(1) The overall event rate in the region (2.7/1000 person-years [py]) was substantially lower than that observed in high-income countries in both the placebo (11.1/1000py) and pitavastatin arms (7.3/1000py). It is unknown whether this discrepancy in MACE risk was due to a lower absolute risk of events in the African region, difficulty in identifying MACE in the region due to limited diagnostic capacity, or a combination thereof.

Consequently, it remains unknown whether the increased risk of CAD among PWH in the United States and Europe extends to other global regions, where traditional CAD risk factors, behavioral and dietary pattens, and genetics all meaningfully differ. To help respond to this gap in knowledge, we designed the Epidemiology of Coronary Artery Disease among People with HIV in Uganda Study. We recruited approximately 600 PWH and PWoH in Uganda and conducted computed tomography (CT) angiograms (CCTA) of the coronary arteries and calcium artery calcium (CAC) scoring to estimate the prevalence and severity of CAD in both groups. We hypothesized that PWH would have an increased prevalence of CAD, both with and without adjustment for traditional CAD risk factors.

Methods

Study design

The Epidemiology of Coronary Artery Disease among People with HIV in Rural sub-Saharan Africa Study was a cross-sectional study of PWH on ART and an age- and sex-matched, population-based control group of PWoH in southwestern Uganda.

Participants and inclusion criteria

Participants were enrolled from two sites in Mbarara and Kabwohe, Uganda. We first selected PWH, who were eligible if they were greater than 40 years old, in clinical care at either the Mbarara Regional Referral Hospital (MRRH) Immune Suppression Syndrome Clinic or the Kabwohe HIV Clinic, and who were on antiretroviral therapy (ART) for a minimum of 3 years. We then enrolled a comparator group of PWoH, who were eligible if they lived within 20 kilometers of the HIV clinics and were age- and sex-similar to PWH. We used census data from the clinic catchment areas to identify demographically similar (by sex and age quartile of PWH) community-based comparators.(16) The selection of community-based comparators was done to reduce selection bias associated with health center-based recruitment of controls. Individuals were excluded if they had contraindications to CT, including reduced kidney function, as determined by the Chronic Kidney Disease Epidemiology (CKD-EPI) glomerular filtration rate of <60 mL/min/1.73m^2 or pregnancy,

which was confirmed through use of β-HCG testing among all women under the age of 60 years. We conducted confirmatory HIV testing in PWoH prior to study procedures.

Study procedures

Participants completed questionnaires on demographics, occupation, household assets,(17) medical and family history, smoking history,[16] and physical activity levels.(18,19) Study nurses measured height and weight and collected blood for creatinine, lipids and hemoglobin A1c testing.

Computed tomography imaging

Scans were performed using a 128-slice single-source CT scanner (SOMATOM AS; Siemens Healthineers) utilizing electrocardiogram (ECG)-based synchronization by a qualified technologist, with monitoring of all exams by a local subspecialty radiologist. Coronary CT angiography was initially performed at Nsambya Hospital, and later a site with identical equipment (Nakasero) was added to improve access. To ensure image quality, a board-certified radiologist (B.G.) visited Nsambya Hospital to implement and perform quality control of scan protocols. Full details of the CT scan methods are described in the study CT scan study operating procedure, which is included in a supplementary appendix. In summary, prospectively ECG-gated non-contrast axial acquisitions from the lung apices to the diaphragm were performed in all patients for non-contrast, calcium scoring. Retrospectively ECG-gated helical acquisitions of the heart were performed for contrast-enhanced CCTA. CT tube current was automatically optimized (CAREDose 4D, Siemens Healthineers). Breast displacement was performed as applicable.(20) Scan acquisitions were performed during a single breath-hold at end-inspiration, with the z-axis scan ranging from the level of the apices to the diaphragm (non-contrast scans) and carina to the diaphragm (contrast scans). Bolus-triggering (with manual override by an expert technologist as necessary) was used for contrast timing. Pre-recorded exam instructions were given in the native language of participants.

CCTA scans were conducted with heart rate control, including use of an oral beta-blocker to target a heart rate under 70 beats per minute. Unless contraindicated, sublingual nitroglycerin was administered during the exam to facilitate coronary artery vasodilatation and provide improved visualization.(21) Systolic, diastolic, and multiphase/functional datasets were reconstructed. Anonymized images were archived in a local picture archiving and communication system (PACS) and anonymized datasets were electronically shared with the US-based central core lab for quality control review and research interpretation. Imaging datasets received at the study core lab were reviewed for completeness and adherence to the CT acquisition protocol. Radiation exposure was monitored, and an established radiation safety plan was utilized to ensure that radiation doses did not exceed pre-specified thresholds,(22) with stepwise interventions triggered as necessary.

Computed tomography interpretation

A radiologist (B.G.) with 20 years' experience and cardiovascular radiology subspecialty fellowship training, along with three subspecialty research cardiovascular imaging fellows (V.T., A.K.T., and M.K.R.) analyzed CCTA results. Radiologists were blinded to participant

HIV serostatus and clinical characteristics. Images were interpreted at the participant and vessel (i.e., left main, left anterior descending, left circumflex, right coronary artery, and, where applicable, ramus intermedius) levels. Lesions >1.5mm in lumen diameter were graded by study radiologists according to the clinically reported stenosis per the CAD-RADS scoring system.(23) CAC were calculated using the Agatston method from non-contrast ECG-gated cardiac CT scans using dedicated software (Aquarius, TeraRecon, V. 4.7.0.22–111, Andover).(24) Plaque was characterized using all available data for each participant, including calcium scans and CCTA. If data were absent for either scan or for certain coronary segments, available data were reviewed to the extent possible. For each subject with any plaque identified on CCTA, total segment involvement and segment stenosis scores were also calculated. (25)

Statistical analysis

Our primary outcome of interest was the presence of CAD, which was defined in participants with either (a) any plaque (calcified or non-calcified, independent of stenosis) on CCTA in at least one of the major coronary arteries (i.e., left main, left anterior descending, left circumflex, right coronary artery or their major branches or (b) an Agatston CAC score > 0. Our primary exposure of interest was HIV serostatus. For secondary outcomes we compared the following outcomes by HIV serostatus: (a) the proportion with coronary atherosclerosis sub-categories as non-calcified, mixed or calcified plaque; (b) the CAD-RADS score, (c) the Agatston CAC score; and (d) the segment involvement and segment stenosis scores, which quantify the number of affected coronary artery sub-segments out of 18 total segments in the major coronary arteries.

We first summarized cohort sociodemographic and clinical characteristics overall and by HIV serostatus. We then estimated the prevalence of each of our primary and secondary outcomes by HIV serostatus, and by ASCVD risk score category (<2.5%, 2.5–5%, and >5%, selected to divide the cohort into roughly evenly sized groups). Finally, we fit univariable and multivariable Poisson regression models with robust standard errors to estimate the comparative prevalence of coronary artery atherosclerosis by HIV serostatus with and without multivariable adjustment. The exponentiated regression coefficients from the Poisson regression model can be interpreted as prevalence ratios, and the robust estimates of variance can be used to correct the standard errors that would otherwise be obtained given the progressive increase in variance of the Poisson distribution.(26,27) We fitted two multivariable models. In the first model, we included HIV serostatus and CVD risk factors, including: age, sex, smoking status (never, current, former), body mass index (<18.5, 18–25, 25–30 and >30 kg/m^2), treatment for hypertension (yes, no), average of two systolic blood pressure readings, hemoglobin A1c percentage, high density lipoprotein (HDL) and non-HDL cholesterol. In the second model we included HIV serostatus and the pooled cohort equation ASCVD risk score,(28) categorized as <2.5%, 2.5–5%, and >5%.

We designed the study to provide at least 80% power to detect a crude difference in the unadjusted prevalence of CAD among PWH versus PWoH as low as 12% (assuming a prevalence of 35% in the sub-group of PWoH), consistent with the smaller adjusted relative difference reported in prior studies in the United States.(29,30) To do so, we planned

enrollment of 600 eligible participants, to allow for evaluation of 570 images (n=285 per HIV serostatus group), to accommodate a 5% rate of uninterpretable images.

Ethical considerations

All participants gave written informed consent. Study protocols were approved by the ethics review committees at Mbarara University Science of Technology, Mass General Brigham, and the Uganda National Council for Science and Technology.

Role of the funding source

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Results

A total of 630 individuals were approached for participation, out of whom 627 consented for participation, and 586 were eligible and completed CT imaging with interpretable scans (Figure 1). Of these, 586 (100%) had interpretable CAC scans and 560 (95.6%) had interpretable CCTA scans. All arteries were evaluable for the majority of both the CAC scans (579/581, 99.7%) and the CCTA scans (478/565, 84.6%, Supplemental Table 1).

Demographic and clinical characteristics of participants in the primary analysis are summarized in Table 1. All participants were Black Africans. The cohort was nearly equally divided by sex and HIV serostatus, with a mean age of 57 years (standard deviation [SD] 6 years). All PWH were on active ART. Approximately half (148/287, 52%) had a current CD4 count over 500 cells/uL and nearly all (272/287, 97%) had an HIV-1 RNA viral load <40 copies/mL. PWH and PWoH were generally similar in terms of demographic and CV risk factor characteristics, with the exception of systolic blood pressure and non-HDL cholesterol, which were modestly higher in PWH (126.4 vs 122.9 mmHg, P=0.005) and (116.6 mg/dL vs. 110.0 mg/dL, P=0.03), respectively. The overall prevalence of angina was 10% overall, with no difference by HIV status.

The prevalence of any CAD was low overall in the cohort, and similar between groups (9.1 % [26/287] in PWH vs 6.3% [19/299] in PWoH, resulting in an absolute prevalence difference of 2.7% (95%CI −1.6, 7.0%, *P*=0.22, Table 2 and Figure 2). Although samples were small, of those with any CAD, the proportion with calcified, non-calcified or partially calcified plaque was similar between groups, with a nominally higher prevalence of calcified-only plaque among PWH (12/26 [46.2%] vs 7/19 [36.8%], *P*=0.60). We also found no difference in the median segment involvement score (median 1, interquartile range 1–2 in both groups, *P*=0.33) or CAD-RADS score, with the vast majority in both groups having a score of 0 (93.5 vs 95.1%, P=0.24), indicating no CAD. Finally, the proportion of participants with a calcium score equal to 0 was similar between groups (285/299 [95.3%]

in PWoH versus 269/287 [93.7%] in PWH), but there was a higher median Agatston calcium score among those with any CAD (27.2 vs 3.6, *P*=0.02) in PWoH.

Living with HIV was not associated with a statistically significant increased prevalence of CAD in multivariable regression models adjusted for traditional CV risk factors or the pooled cohort equation ASCVD risk score (aPR 1.60, 95%CI 0.93, 2.74, P=0.091, Table 3). There was an increased prevalence of CAD with increasing age (adjusted prevalence ratio [aPR], 1.54 for each 10-year increase in age, 95%CI 1.10, 2.16, P=0.012) and in men versus women (aPR 2.52, 95%CI 1.13, 5.63, P=0.024, Table 3). In the model with the summary ASCVD risk score in place of individual CV risk factors, those with a ≥5% 10-year ASCVD risk had an aPR of 5.34 (95%CI 2.13, 13.37, P<0.001) compared with those with a <2.5% 10-year risk.

Discussion

In a cohort of older adult PWH on ART in public-sector clinical care in Uganda we found an extremely low prevalence of CAD. Among both PWH and PWoH, the prevalence of any CAD was less than 10%, the prevalence of a CAC score > 0 was 5% and the prevalence of a CAD-RADS score > 1 was 3%. Moreover, and in contrast to numerous reports from high-income regions, PWH did not have a meaningful difference (<3% absolute difference) in the prevalence of CAD compared to demographically similar PWoH from their same communities. These data, which represent some of the first in the region that use CT angiography to directly measure end-organ CAD, suggest a unique clinical phenotype for PWH in Uganda. Further, these results challenge perceptions that CAD is a major cause of morbidity in this population.

Contrary to our primary hypothesis, PWH in Uganda did not have a substantially elevated prevalence of CAD compared to PWoH. Our results suggest that absolute CAD risk is predominantly driven local population a *priori* risk. For example, a recent sub-analysis of asymptomatic PWH in REPREIVE found a prevalence of CAD of 49%, compared to 40% in a Swedish population of PWoH.(31) Importantly, we observed a similar prevalence of CAD between groups for both calcified plaque and non-calcified plaque, which mitigates concern that PWH in this region may harbor higher-risk, non-classified plaque, as has been reported among PWH in the United States.(32–34) By contrast, in our study the prevalence of calcified-only plaque among those with any CAD was nominally higher among PWH than PWoH (12/26, [46%] vs 7/19 [37%]). Our findings are consistent with those of a smaller mixed HIV cohort from urban Uganda, which was enriched for people with at least one CVD risk factor, and also found a low prevalence of CAD and a null association between HIV and CAD prevalence.(35,36) These data contrast with assumptions made in modeling exercises that suggest a large and growing burden of CAD attributable to HIV in the region, and may support region-specific guidelines for CAD prevention measures among PWH.(37,38) Moreover, in our opinion these findings should prompt additional study of the priority disease states for PWH on suppressive ART, to help target prevention and treatment strategies that most impact health.(39)

Overall, we found extremely low rates of CAD in the general population in Uganda. For example, the approximately 8% prevalence of any CAD and 4% prevalence of non-calcified plaque we detected in this cohort are considerably lower than those observed in population-based cohorts with similar age distributions in the United States (49% for any CAD and 27% for non-calcified plaque),(40) Sweden (42% for any CAD and 8% for non-calcified plaque), (41) and South Korea (21% for any CAD and 4% for non-calcified plaque), respectively. (42) Similarly, the 5% prevalence of a CAC score > 0 in our cohort is substantially lower than estimates reported among minorities in the United States in the Multi-Ethnic Study of Atherosclerosis, which reported a prevalence of coronary artery calcification of 52% among African American men and 38% among African American women (Figure 4).(43) In our prior study in Kampala, Uganda and Cleveland, USA, Ugandans had 13-times lower odds of CAC >0 compared to people in Cleveland after adjustment for cardiovascular disease risk factors.(35) Although we did not measure MACE rates, in most populations, there are strong correlations between the presence of CAD and future risk of events.(44,45) If such relationships hold true in this region, other conditions, such as stroke and non-ischemic heart failure, or perhaps non-cardiovascular conditions, may be more likely to dominate morbidity in the region.(46,47) Regardless, these data reinforce the importance of regional data collection to more precisely identify health priorities and reduce reliance on externally generated data to advise health policy.

Our study should be interpreted with limitations in mind. We measured prevalent CAD in a population-based cohort. Our findings cannot be generalized to symptomatic populations or those with known CAD. Moreover, this was a cross-sectional study and did not measure MACE, which will be needed in future studies to confirm the risk of myocardial infarction (Ml) events in this population. That said, the overall low rates of CAD we observed are consistent with our prior studies from the region(35,36) and are potentially affirmed by the significantly reduced rates of MACE reported in the REPRIEVE trial in the African region.(1) As with all observational studies, we cannot eliminate the risk of confounding between our primary exposure and outcome. We took steps to mitigate this risk, including enrollment of controls directly from the community in the same communities as those with HIV, and adjustment for well-established CVD risk factors in our models. These risk factors generally correlated with CAD prevalence as predicted and did not affect our estimates on the relationship between HIV and CAD. Moreover, the low overall prevalence and small absolute difference between groups further reduces concern for significant confounding. Finally, our study was powered to detect a >12% difference in the absolute difference in CAD prevalence between PWH and PWoH; a difference derived from studies in the United States. We found a much smaller absolute difference (<3%), and our study was not adequately powered to determine whether PWH might have an increased prevalence of that magnitude.

In conclusion, we found low rates of CAD among PWH in Uganda, and a similar prevalence as demographically similar people in the same communities. Our findings reduce suspicion that HIV is associated with significant CAD risk in the region and should prompt future work to better define health priorities for this population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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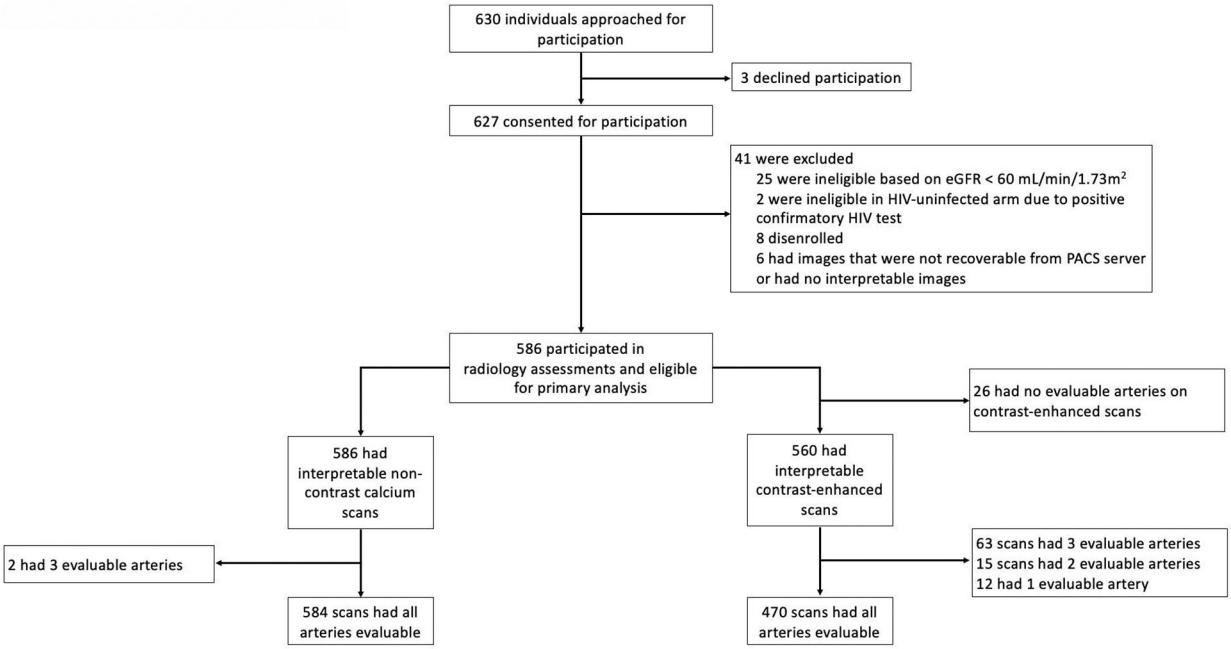

Figure 1.
Study flow diagram

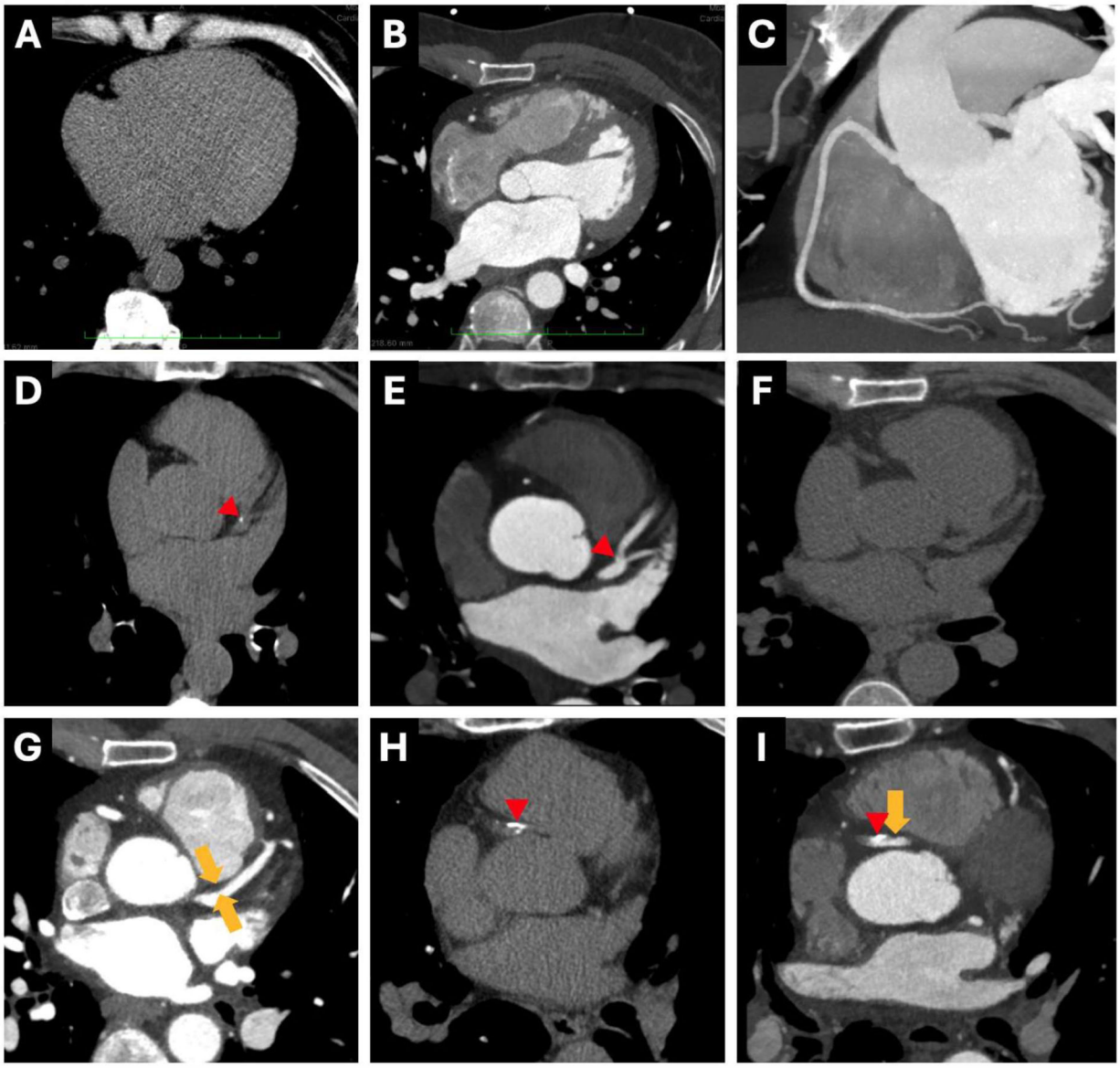

Figure 2.

Representative images of individuals with the spectrum of coronary artery disease identified in this study. Axial non-enhanced coronary artery calcium scoring (CAC) scan image shows no calcified plaque (panel A); corresponding axial (panel B) and double-oblique right coronary artery maximum intensity projection coronary contrast-enhanced computed tomography angiography (CTA) (panel C) confirm no noncalcified plaque in the same subject. A second subject's matched axial CAC and CTA scans reveal punctate calcified plaque at the level of the proximal LAD (red arrowheads, panels D and E). A third subject illustrates the incremental yield of CTA images; no calcified plaque is identified at CAC (panel F), while corresponding CTA reveals noncalcified plaque along the luminal wall (yellow arrows, panel G). A fourth subject's right coronary artery CAC scan detects a calcified plaque (red arrowhead, panel H) while the corresponding CTA dataset reveals an

additional proximal lipid-rich noncalcified component (yellow arrow, panel I) adjacent to the calcified plaque (red arrowhead, panel I).

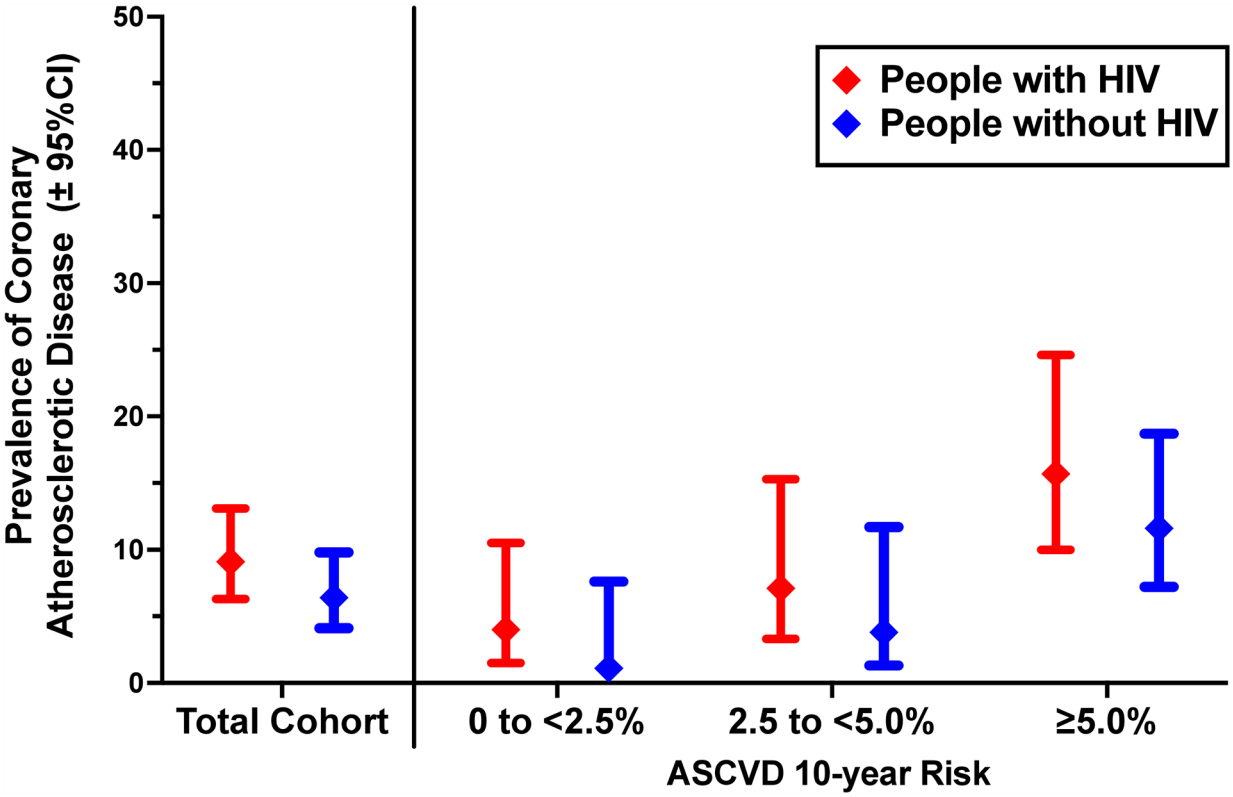

Figure 3.
Prevalence of any CAD overall and by ASCVD risk

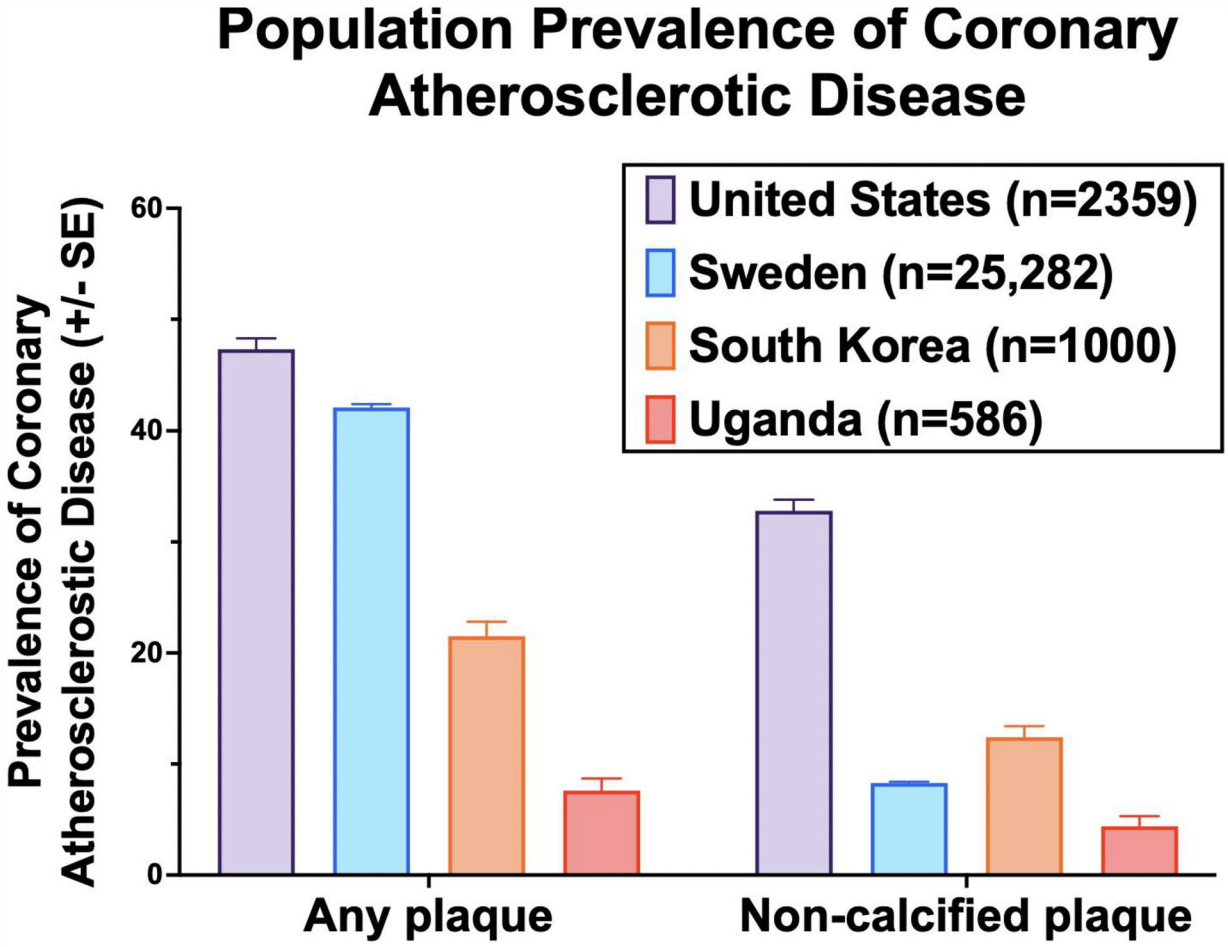

Figure 4.
Prevalence of coronary artery disease by computed tomography in global population-based studies from the United States,(42) Sweden,(41) South Korea,(42) and our study in Uganda.

Table 1.

Participant characteristics, stratified by HIV serostatus

	People without HIV (n=299)	People with HIV (n=287)	*P*-value
Sociodemographics and medical history			
Age, mean (SD), y	57.9 (6.9)	57.4 (6.4)	0.32
Women (n,%)	147 (49.2)	140 (48.8)	0.92
Race/ethnicity (n,%)			
Black African	299 (100)	287 (100)	
Occupation (n,%)			0.001
Non-manual workers	37 (12.4)	64 (22.3)	
Farm workers	230 (76.9)	181 (63.1)	
Manual workers/Other	32 (10.7)	40 (13.9)	
Unemployed	0 (0)	2 (0.7)	
Education (n,%)			0.56
Did not complete Primary School	156 (52.2)	140 (48.8)	
Completed Primary School	111 (37.1)	109 (38.0)	
Completed Secondary School	32 (10.7)	38 (13.2)	
Smoking status (n,%)			0.35
Current	29 (9.7)	19 (6.6)	
Former	94 (31.4)	99 (34.5)	
Never	176 (58.9)	169 (58.9)	
Cardiovascular disease characteristics			
ASCVD risk score, %			0.30
Median [IQR]	4.1 [2.1, 7.5]	3.4 [1.9, 7.3]	
0 to <2.5	92 (30.8)	100 (34.8)	
2.5 to <5	78 (26.1)	85 (29.6)	
5+	129 (43.1)	102 (35.5)	
Framingham risk score, %			0.27
Median [IQR]	7.9 [4.7, 12.5]	6.6 [4.1, 11.7]	
0 to <2.5	15 (5.0)	24 (8.4)	
2.5 to <5	71 (23.7)	76 (26.5)	
5 to <10	107 (35.8)	98 (34.1)	
10+	106 (35.5)	89 (31.0)	
BMI, mean (SD)	24.7 (5.5)	23.7 (4.5)	
Self-reported hypertension (n,%)	75 (25.1)	45 (15.7)	0.005
Systolic blood pressure, mean (SD)	126.4 (19.8)	122.9 (19.0)	0.03
Diastolic blood pressure, mean (SD)	79.0 (11.5)	78.7 (12.3)	0.82
Self-reported diabetes (n,%)	13 (4.3)	16 (5.6)	0.49
Hemoglobin A1c (g/dL), mean (SD)	5.8 (1.0)	5.8 (1.3)	0.94
Proportion >6.5 (g/dL)	22 (7.4)	21 (7.3)	0.99
Total cholesterol (md/dL), mean (SD)	165.7 (36.7)	159.5 (40.9)	0.06
Non-HDL cholesterol (md/dL), mean (SD)	116.6 (37.1)	110.0 (38.4)	0.03

	People without HIV (n=299)	People with HIV (n=287)	*P*-value
HDL (mg/dL), mean (SD)	49.0 (14.5)	49.6 (21.9)	0.74
Any immediate family history of CAD (n, %)			0.01
Reported family history	3 (1.0)	1 (0.3)	
No family history	225 (75.3)	244 (85.0)	
Unknown	72 (23.7)	42 (14.6)	
HIV History			
Current CD4 count, cells/mm^3 (n,%)			
<350	N/A	53 (18.6)	
350–499	N/A	84 (29.5)	
≥500	N/A	148 (51.9)	
Nadir CD4 count, cells/mm^3, (n,%)			
<100	N/A	40 (13.9)	
100–250	N/A	87 (30.3)	
250–350	N/A	47 (16.4)	
350–500	N/A	56 (19.5)	
≥500	N/A	57 (19.9)	
Viral load			
HIV-1 RNA < 40 copies/mL (n,%)	N/A	272 (97.1)	

Table 2.

Prevalence and degree of coronary atherosclerotic disease and plaque type in Uganda

	People without HIV	People with HIV	Total Cohort	*P*-value
Any coronary atherosclerosis (n, %) – primary	19/299 (6.4)	26/287 (9.1)	45/586 (7.7)	0.22
Plaque composition §				0.60
Only calcified plaque (n, %)	7/19 (36.8)	12/26 (46.2)	19/45 (42.2)	
Only non-calcified plaque (n, %)	5/19 (26.3)	8/26 (30.8)	13/45 (28.9)	
Partially calcified plaque (n, %)	7/19 (36.8)	6/26 (23.1)	13/45 (28.9)	
Segment involvement score (median, IQR)§	1.0 [1.0, 2.0]	1.0 [1.0, 2.0]	1.0 [1.0, 2.0]	0.35
Segment stenosis score (median, IQR)§	1.0 [1.0, 3.5]	2.0 [1.0, 2.0]	2.0 [1.0, 2.0]	0.14
Agatston coronary calcium score (median, IQR)§	27.2 [0.6, 71.2]	3.6 [0, 14.0]	6.0 [0.0, 35.1]	0.02
Agatston coronary calcium category				0.08
Calcium score 0 (n, %)	285/299 (95.3)	269/287 (93.7)	554/586 (94.5)	
Calcium score >0 (n, %)	11/299 (3.7)	18/287 (6.3)	29/586 (4.9)	
Calcium score >100 (n, %)	3/299 (1.0)	0/287 (0.0)	3/586 (0.5)	
CAD-RADS Score (n, %)				0.24
0	271/285 (95.1)	244/266 (91.7)	515/551 (93.5)	
1	7/285 (2.5)	13/266 (4.9)	20/551 (3.6)	
2	7/285 (2.5)	9/266 (3.4)	16/551 (2.9)	

IQR: Interquartile Range; CAD-RADS: Coronary Artery Disease Reporting and Data System Score; where 0 = no stenosis, 1=1–24% (Minimal stenosis or plaque with no stenosis), 2 = 25–49% (Mild stenosis).

§Plaque composition, segment involvement score, segment stenosis score, and Agatston calcium scores are reported among those who with coronary atherosclerosis.

Table 3.

Correlates of coronary atherosclerosis in Uganda. Models were fit in two ways: adjusted for individual traditional cardiovascular risk factors (3A) and adjusted for atherosclerotic cardiovascular disease risk score (3B)

Table 3A.	**Univariable Model PR**	**95% CI**	***P*-value**	**Multivariable Model, aPR**	**95% CI**	***P*-value**	**ASCVD Model, APR**	**95%CI**	***P*-value**
Age, each 10 years	1.70	[1.27, 2.27]	<0.001	1.54	[1.10, 2.16]	0.012			
Sex									
Women	Reference			REF					
Men	2.64	[1.39, 5.01]	0.003	2.52	[1.13, 5.64]	0.024			
Smoking status (n,%)									
Never	Reference			REF					
Current	2.88	[1.34, 6.16]	0.007	1.88	[0.84, 4.19]	0.123			
Former	1.52	[0.82, 2.83]	0.188	0.97	[0.50, 1.87]	0.918			
BMI									
18–25	Reference			REF					
<18	1.61	[0.67, 3.87]	0.289	1.70	[0.71, 4.09]	0.234			
25–30	0.29	[0.10, 0.81]	0.018	0.35	[0.11, 1.12]	0.077			
>30	0.90	[0.39, 2.08]	0.803	1.21	[0.41, 3.55]	0.733			
On treatment for hypertension									
No	Reference								
Yes	1.41	[0.75, 2.65]	0.283	1.32	[0.65, 2.67]	0.437			
Systolic blood pressure, each 10mmHg	1.05	[0.92, 1.21]	0.465	0.99	[0.85, 1.16]	0.931			
Hemoglobin A1c, each %	1.15	[1.00, 1.32]	0.055	1.16	[1.00, 1.34]	0.055			
Non-HDL cholesterol, each 10 mg/dL	1.03	[0.95, 1.12]	0.499	1.06	[0.99, 1.15]	0.114			
HDL cholesterol, each 10mg/dL	0.94	[0.80, 1.10]	0.433	0.95	[0.82, 1.10]	0.501			
HIV serostatus									
People without HIV	Reference			REF					
People with HIV	1.43	[0.81, 2.52]	0.222	1.60	[0.93, 2.74]	0.091			
Table 3B.									
ASCVD risk score									
0 to <2.5	Reference						Reference		
2.5 to <5	2.12	[0.73, 6.20]	0.170				2.12	[0.73, 6.18]	0.169

5+	5.15	[2.04, 13.00]	<0.001	5.34	[2.13, 13.37]	<0.001
HIV serostatus						
People without HIV	Reference			Reference		
People with HIV	1.43	[0.81, 2.52]	0.222	1.57	[0.90, 2.74]	0.112

PR: prevalence ratio; aPR: adjusted prevalence ratio; ASCVD: Atherosclerotic cardiovascular disease; HDL: high density lipoprotein