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Clinical and procedural characteristics of persons living with HIV presenting with acute coronary syndrome

Caitlin A. Moran^{a,c}, Geoffrey Southmayd^b, Chandan M. Devireddy^b, Arshed A. Quyyumi^b, Ighovwerha Ofotokun^{a,c}, Henry A. Liberman^b, Wissam Jaber^b, Anandi N. Sheth^{a,c}

^aDivision of Infectious Diseases, Department of Medicine, Emory University School of Medicine, Atlanta, Georgia, USA.

^bDivision of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, Georgia, USA.

^cDepartment of Medicine, Grady Healthcare System, Atlanta, Georgia, USA.

Abstract

Objectives: Persons living with HIV (PLWH) are at greater risk for acute coronary syndrome (ACS). Practice patterns of ACS management by HIV serostatus are unknown. We examined the presentation and management of ACS in PLWH.

Design: Retrospective case–control study.

Methods: We included 86 PLWH and 263 sex-matched and race-matched HIV-negative controls hospitalized with ACS between 2004 and 2013. We performed multivariable conditional logistic regression to determine the associations between HIV serostatus and ACS type and management.

Results: Both groups were predominantly of black race and male sex. PLWH were significantly younger (53 vs. 60 years) and more likely to smoke (48 vs. 31%). Among PLWH, 30% had CD4⁺ cell count less than 200 cells/μl and 58% had undetectable HIV RNA. PLWH had more single-vessel disease and a higher median Gensini score among those with single-vessel disease (32 vs. 4.25) than controls. HIV serostatus was positively associated with ST-elevation myocardial infarction (STEMI) [adjusted odds ratio (aOR) (95% confidence interval (CI)): 5.05 (1.82–14.02)], and any revascularization procedure after ACS [aOR (95% CI): 2.90 (1.01–8.39)] and negatively associated with non-STEMI [aOR (95% CI): 0.33 (0.14–0.79)] presentation. PLWH who underwent stent placement had a higher likelihood of bare metal stent placement compared with controls [70 vs. 15%, aOR (95% CI): 5.94 (1.33–26.55)]. Among PLWH, ACS characteristics were not significantly associated with CD4⁺ cell count, HIV RNA, or antiretroviral therapy.

Correspondence to Caitlin A. Moran, Division of Infectious Diseases, Department of Medicine, Emory University School of Medicine, 49 Jesse Hill Jr Drive SE, Atlanta, GA 30303, USA. Tel: +1 404 778 1620; fax: +1 404 616 0592; caitlin.moran@emory.edu.

Authors' contribution: G.S., C.M.D., A.A.Q., and A.N.S. conceived of the study design. G.S., C.M.D., A.A.Q., H.A.L., and J.W. were involved in data collection. C.A.M. led the analysis and interpretation of study data with significant input from G.S. and A.N.S. The article was drafted by C.A.M. and critically revised for intellectual content by G.S., C.M.D., A.A.Q., H.A.L., J.W., I.O., and A.N.S.

Conflicts of interest

There are no conflicts of interest.

Conclusion: PLWH hospitalized with ACS were more likely to have severe single-vessel disease, present with STEMI rather than non-STEMI, and undergo revascularization, and less likely to have a drug-eluting stent placed than matched HIV-negative controls, suggesting that coronary plaque morphology and/or distribution is different with HIV infection and warrants further investigation.

Keywords

acute coronary syndrome; coronary revascularization; Gensini score; HIV

Introduction

With effective antiretroviral therapy (ART), persons living with HIV (PLWH) can expect to attain a near-normal life expectancy [1,2]. However, they are at increased risk for age-related comorbidities, including cardiovascular disease (CVD) [3–5]. Indeed, among PLWH, acute coronary syndrome (ACS) events tend to occur at younger ages and with greater morbidity than in the general population [6–8]. This increased risk is thought to be multifactorial, resulting from a greater prevalence of metabolic and behavioral risk factors including smoking [4,9], the effects of the HIV virus on inflammation and immune activation despite viral suppression with ART [10], T-cell depletion and senescence [11,12], and ART, particularly the protease inhibitors [13] and the nucleoside reverse transcriptase inhibitor abacavir [14]. Despite this risk, there are relatively few data on coronary artery disease (CAD) severity among PLWH. The Gensini score attempts to quantify the functional significance of a patient's coronary atherosclerotic burden by incorporating a point system for the degree of vessel luminal narrowing that accounts for the specific anatomic location of the stenosis [15]. It is strongly correlated with coronary plaque burden, plaque area, and prognosis [16], and correlates with the duration of other traditional cardiovascular risk factors including smoking [17] and diabetes [18]. However, to our knowledge, only one prior study examined Gensini scores among PLWH presenting with ACS [19].

Recent data also suggest that there are disparities in ACS management for PLWH. Extensive evidence demonstrates that outcomes after ACS are better with drug-eluting stents (DES) than with bare metal stents (BMS) [20–22], including for PLWH [23]. Nonetheless, in one recent sampling of nationwide data in the United States, PLWH who underwent percutaneous coronary intervention (PCI) following ST-elevation myocardial infarction (MI) (STEMI) had a lower rate of DES placement compared with the general population, even when adjusting for income, hospital type, and primary payer source [24]. National US data also suggest that PLWH hospitalized with MI are less likely to undergo any type of revascularization procedure than HIV-negative controls [25,26]. However, these studies may be confounded by practice and population differences among hospitals located in areas with varying HIV prevalence, and do not include measures of functional CAD severity. The local practice differences where the same physicians are caring for HIV-positive and HIV-negative patients presenting with ACS, and whether CAD severity accounts for any practice differences are unknown. Given the high CAD risk in an aging HIV population, it is increasingly important to define differences in ACS in this population. Here, we present data from PLWH and matched HIV-negative controls who were hospitalized with ACS at two

urban academic medical centers in the United States to determine any differences in presentation or management of ACS, or CAD severity by HIV serostatus.

Methods

Study design and patient population

We retrospectively reviewed the medical records of patients admitted to either Emory University Hospital (EUH) or Emory University Hospital Midtown (EUHM), two academic medical centers in the Emory Healthcare network in Atlanta, GA, between October 2006 and October 2012 with a diagnosis of ACS who underwent left heart catheterization (LHC) and had a concurrent diagnosis of HIV. These cases were then matched 1 : 3 on race and sex with an existing cohort of patients without HIV infection who presented to the same hospitals from 2004 to 2013 with a diagnosis of ACS. EUH is a 733-bed tertiary care academic hospital located approximately 7 miles from downtown Atlanta, and EUHM is a 511-bed, community-based, urban-located teaching hospital located just one mile from downtown Atlanta. Combined, EUH and EUHM perform an average of 2105 LHC annually for ACS (945/year at EUH and 1160/year at EUHM). Both hospitals have large catchment areas that include the eight-county metro Atlanta area as well as referrals from throughout the Southeastern United States.

Outcomes

The primary outcome was ACS type. ACS was defined and classified using the 2013 American College of Cardiology/American Heart Association guidelines [27]. STEMI was defined as ST-segment elevation of greater than 0.1 mV in at least two contiguous precordial or adjacent limb leads, a new left bundle branch block, or true posterior MI. Non-STEMI (NSTEMI) was defined as a troponin I elevation in the setting of chest pain or angina equivalence without ST-segment elevation. Unstable angina was defined as chest pain or angina equivalence of new or increasing frequency in the absence of cardiac biomarker elevation or ST-segment elevation.

The secondary outcome was ACS management. Management decisions including surgical, percutaneous, or medical therapies were made according to usual clinical practice and performed with standard technique.

Covariates

HIV infection was determined by third-generation ELISA testing and confirmed by Western Blot. HIV RNA (viral load) and CD4⁺ T-cell count values were obtained from the most recent data available at the time of admission, including up to 30 days after discharge. CD4⁺ T-cell count was determined using standard flow cytometric assays. HIV RNA was determined by real-time PCR with a lower limit of detection of 50 copies/ml through most of the study that decreased to 20 copies/ml in 2012. HIV RNA values below the limit of detection were recorded as undetectable. ART and other medication history was obtained from chart review. Additional comorbidity data including hypertension, dyslipidemia, diabetes mellitus, congestive heart failure and prior stroke was obtained from detailed chart abstraction in addition to International Classification of Diseases, 9th edition (ICD-9)

Clinical Modification diagnosis codes. Because diagnosis of most comorbidities requires more than one measurement or fasting measurements [28,29], diagnostic criteria could not be used to determine comorbidity prevalence at the time of ACS presentation. Demographic and behavioral characteristics were obtained from chart review.

Quantification of CAD severity was determined by the Gensini score [15]. Coronary vessel involvement was defined as the number of vessels including the left anterior descending (LAD), left circumflex and right coronary arteries with more than 50% stenosis. When the ramus branch was present and stenotic, it was considered a fourth vessel.

Statistical analysis

Baseline characteristics of cases and controls were compared by chi-square tests or Fisher's exact tests for categorical variables, and Student's *t* tests or Wilcoxon rank-sum tests for continuous variables, as appropriate. To determine the association of the primary and secondary outcomes with HIV status, conditional logistic regression was performed matching on race and sex. Two separate models were developed, the first to determine the associations between HIV serostatus and ACS type and management when adjusting for age and traditional cardiovascular risk factors, and the second to adjust for CAD severity in addition to cardiovascular risk factors. The first model adjusted for age, smoking status, BMI, hypertension, dyslipidemia, diabetes mellitus, and year of intervention, while conditioning on race and sex. The second model included all covariates from the first model as well as the Gensini score to control for CAD severity. Finally, a separate analysis was conducted among PLWH. Given changes in HIV care over time, two-way ANOVA and Chi-square tests were used to determine if differences in ACS presentation or CAD severity occurred by year among PLWH. Similar to the entire cohort, two models were developed to account for age, traditional cardiovascular risk factors, and CAD severity. A logistic regression model controlling for age, sex, race, smoking status, and year of intervention and a second model that included Gensini score were developed to determine the association between the HIV-specific variables CD4⁺ cell count, HIV viral load, and ART (specifically HIV protease inhibitors and abacavir, which have been epidemiologically associated with CVD risk and/or metabolic complications [14,30]) with ACS type and management. Analyses were conducted using SAS v9.4 (SAS Institute, Cary, North Carolina, USA). Statistical significance was determined by a *P* value of less than 0.05. Missing data were handled with a complete case analysis, in which participants with missing covariate data were excluded from models. All data were collected and maintained with approval from the Emory University Institutional Review Board.

Sensitivity analyses

To account for disparate years in which PLWH and HIV-negative controls were enrolled in the study, we performed a sensitivity analysis restricting years from 2006 to 2012 for both groups using fully adjusted models as described above for ACS outcomes and management. Chi-squared tests were performed to assess for differences associated with stent type by year for each group. Finally, to account for up to 20% missingness in some data, we performed a sensitivity analysis with multiple imputation (25 datasets) for our full model for the main outcome measures.

Results

Baseline characteristics

Eighty-six PLWH hospitalized with ACS were matched for sex and race with 263 HIV-negative patients also hospitalized with ACS. PLWH were significantly younger (mean age 53 vs. 60 years), with lower SBP, lower BMI, lower LDL cholesterol, less frequent statin use, lower Framingham risk scores, higher rates of current smoking and higher baseline creatinine levels (Table 1). Median (interquartile range) year of hospitalization was 2010 (2008–2011) for PLWH and 2006 (2005–2011) for controls. Cases and controls had similar prevalence of hypertension, dyslipidemia, diabetes mellitus, congestive heart failure, and prior stroke. Among PLWH, the mean time since HIV diagnosis was just over 10 years. The mean (SD) CD4⁺ cell count was 389 (304) cells/ μ l. Thirty percentage had a CD4⁺ cell count less than 200 cells/ μ l; 58% were virologically suppressed. Seventy-six percent of PLWH were on ART with 54% on protease inhibitors and 21% on abacavir.

CAD severity at baseline is described in Table 2. PLWH were more likely to have single vessel CAD, while controls were more likely to have three-vessel CAD. However, median Gensini score was significantly higher among PLWH overall (44 vs. 28, $P = 0.01$), and for those with single-vessel disease (32 vs. 4.25, $P < 0.0001$). Among PLWH, the most commonly affected vessel was the right coronary artery [38 (44%)], followed by the LAD artery [31 (36%)]. Six (7%) PLWH had left main disease.

Acute coronary syndrome types and interventions

ACS type and interventions are presented in Table 3. Twenty-five (29%) PLWH presented with a STEMI, compared with 31 (12%) controls (unadjusted $P = 0.0002$). In fully adjusted conditional logistic regression models matched on sex and race and adjusted for year, traditional cardiovascular risk factors (age, smoking, hypertension, BMI, dyslipidemia and diabetes mellitus), and Gensini score, PLWH with ACS were more likely to present with STEMI compared with controls [adjusted odds ratio (aOR) 5.05 [95% confidence interval (CI): 1.82–14.02]], and less likely to present with NSTEMI [aOR (95% CI): 0.33 (0.14–0.79)].

Among all patients, in fully adjusted models PLWH had greater odds of any revascularization procedure [aOR (95% CI): 2.90 (1.01–8.39)] than controls. Among those undergoing revascularization, there was no statistically significant difference in coronary artery bypass graft (CABG) for PLWH compared with controls [aOR (95% CI): 3.55 (0.70–18.03)] after controlling for the above covariates. There were too few events to model the odds of CABG vs. PCI among those presenting with STEMI, although four (17%) of PLWH and no controls presenting with STEMI underwent CABG. Among those PLWH who underwent CABG, most had multivessel disease, although three had LAD disease only, and one each had isolated left main, left circumflex, and first obtuse marginal disease. There was no significant difference in the mean (SD) number of vessels bypassed for PLWH compared with controls [2.67 (0.62) vs. 2.93 (0.27), $P = 0.15$], although the median Gensini score for PLWH undergoing CABG was higher compared with controls (67 vs. 30.5, $P = 0.02$).

A total of 203 patients underwent PCI with stent placement. Types of stents placed during each year of the study by HIV status are presented in Fig. 1. Among the 46 PLWH who underwent PCI, 13 (28%) had a DES placed, while 33 (70%) had a BMS placed (Table 3). In contrast, among the 157 control patients who underwent PCI, 125 (83%) had a DES placed, while 23 (15%) had a BMS placed. In the fully adjusted model, PLWH had greater odds of BMS vs. DES placement [aOR (95% CI): 5.94 (1.33–26.55)]. When using multiple imputation to account for missing data in the fully adjusted model, differences in ACS type or stent type remained significant; however, differences in total revascularization were no longer significant.

HIV-specific analysis

The associations of ACS type and any revascularization procedure with HIV-specific variables are presented in Table 4. Among PLWH, the proportion of STEMI vs. other ACS types, and CABG vs. PCI did not change by year ($P = 0.40$ and 0.99 , respectively). There was no difference in severity of CAD as measured by number of vessels involved ($P = 0.64$) or median Gensini score ($P = 0.80$) by year. Among PLWH, neither $CD4^+$ cell count nor HIV viral load were significantly associated with either STEMI or revascularization after adjusting for age, race, sex, smoking status, year, and Gensini score. Adjusting for the same covariates, there was no significant association between STEMI and use of any ART [aOR (95% CI): 0.72 (0.17–3.16)]. Among those PLWH who were taking ART, there was no association with protease inhibitor-containing regimens and STEMI [aOR (95% CI): 2.87 (0.64–12.98)], there were greater odds of presenting with STEMI vs. NSTEMI/unstable angina [aOR (95% CI): 5.08 (1.02–25.26)] for abacavir-containing regimens. There were also no significant associations between any revascularization and use of any ART [aOR (95% CI): 1.18 (0.22–6.22)], protease inhibitor-containing ART [aOR (95% CI): 2.29 (0.48–11.03)], and abacavir-containing ART [aOR (95% CI): 1.42 (0.24–8.48)].

Discussion

In this retrospective study of PLWH and race-matched and sex-matched HIV-negative controls who presented with ACS to two urban academic medical centers within the same healthcare system in a high HIV burden area of the southeastern United States, we comprehensively describe ACS type, severity, disease characteristics, and management and compare these by HIV serostatus adjusting for known CVD risk factors and coronary plaque burden. We found that PLWH were younger, were more likely to present with STEMI and less likely to present with NSTEMI, have more frequent single-vessel disease and less frequent triple-vessel disease, than race-matched and sex-matched HIV-negative patients with ACS. Even with more single-vessel disease, PLWH had higher Gensini scores compared with controls, indicating greater and more proximal occlusive disease at presentation.

In contrast to recently published studies comparing nationwide trends in ACS management by HIV status during the same time period that reported a lower rate of revascularization among PLWH [7,26], we observed a greater rate of coronary revascularization procedures among PLWH compared with HIV-negative controls in our fully adjusted model, although

this difference did not persist when accounting for missing data with multiple imputations. However, similar to national trends [24,26], among those who underwent PCI with stent placement, PLWH in our cohort were more likely to be treated with BMS and less likely to be treated with DES than HIV-negative controls. Reasons for this discrepancy are unclear and are likely multifactorial. Since this analysis included a single healthcare system, it is unlikely that this difference in stent type was due to variation in practice patterns by region or hospital type as has likely confounded analyses from national datasets. Concerns for increased stent thrombosis risk with medication nonaccess or non-adherence may have directly influenced stent choice in PLWH; however, data on comorbid substance use disorders, income or payer sources of patients were lacking, as historic payer source data is not maintained in the electronic medical record. Substance use disorders are more prevalent among PLWH in general and can be a barrier to medication adherence [31]. Socioeconomic or insurance status may have influenced the decision to shorten the necessary duration of dual antiplatelet therapy (DAPT) by favoring BMS among PLWH. Outcomes following ACS are improved with DES rather than BMS regardless of HIV serostatus [23,32], and risk of stent thrombosis with newer generation DES is lower, even with shorter duration DAPT [33]; however, guidelines still recommend 12 months of DAPT following DES for ACS [34], which may still affect stent choice among practitioners. Although current management of ACS is moving toward DES for all persons who undergo PCI, these data point to a need for further research to determine if DES are used as extensively among PLWH.

Our observation that PLWH hospitalized with ACS were more likely to have STEMI than their HIV-negative counterparts even after adjusting for age is consistent with the literature. Data from both the United States and France have shown higher rates of STEMI in PLWH who are hospitalized with ACS than in matched HIV-negative controls [25,35], likely related to coronary plaque composition. Two studies evaluating coronary plaque composition among men by computed tomography angiography found that PLWH had a higher prevalence of noncalcified coronary plaques compared with age-matched HIV-negative controls despite similar rates of overall plaque burden [36,37]. Noncalcified plaques are at greater risk for rupture than calcified or mixed plaques, and the higher burden of noncalcified plaques among PLWH likely puts them at greater risk for plaque rupture and STEMI. This hypothesis is supported by the fact that the association between HIV status and STEMI remained statistically significant even when Gensini score was added to traditional cardiovascular risk factors in our multivariable models.

Significantly, we also found that PLWH were more likely to have single-vessel CAD and less likely to have triple-vessel disease compared with the HIV-negative controls, consistent with previous studies that have reported a predominance of single-vessel disease among PLWH [38], likely due to younger age at ACS presentation [39]. However, PLWH with single-vessel disease in our study had higher Gensini scores than controls with single-vessel disease, indicating a greater degree of vessel stenosis. Because the Gensini score accounts for the degree of vessel narrowing and the anatomic location of stenosis, the higher Gensini score among PLWH despite fewer vessels involved suggests that those vessels may have had more severely occlusive proximal disease, more diffuse disease or higher risk lesions. This finding contrasts with an Australian study of 73 PLWH presenting with ACS and matched on age, sex, and smoking status with 87 HIV-negative controls, that found no difference in

the number of coronary vessels involved, but that the Gensini score was actually lower among PLWH [19]. Importantly, PLWH in the Australian study had less advanced HIV than in our study (mean CD4⁺ cell count 510 cells/μl and 89% with an undetectable HIV viral load) [19], suggesting that ongoing HIV viremia and immunosuppression may contribute to atherosclerosis.

In our study among PLWH, current CD4⁺ cell count, HIV viral load, and ART use were not significantly associated with ACS type or intervention in fully adjusted models; however, sample size limited our ability to draw further conclusions. Among those taking ART, there was no association between protease inhibitor use and STEMI presentation rather than NSTEMI/unstable angina; however, there was a statistically significant association between abacavir use and STEMI, as has been seen in prior observational studies [14,40]. Data are mixed regarding the cardiovascular effects of individual antiretroviral drugs or classes. The CVD risk associated with protease inhibitors is thought to be mediated by dyslipidemia and insulin resistance [30]; however, this risk is less clear with the newer protease inhibitors atazanavir and darunavir [41,42]. In fact, ritonavir-boosted atazanavir is associated with a slower progression of atherosclerosis than the lipid-neutral integrase strand transfer inhibitor (INSTI) raltegravir [43]. In our study, the two most commonly used protease inhibitors were ritonavir-boosted lopinavir ($n = 17$) and ritonavir-boosted atazanavir ($n = 10$). Therefore, it is possible that the lack of effect of protease inhibitors seen in our study may be due in part to the heterogeneity of protease inhibitors used. The association between abacavir use and MI is somewhat controversial, with some studies showing a positive association [14,40], and others showing no significant association [44]. Some experimental ex-vivo data suggest that abacavir may impact endothelial inflammation and/or platelet activation in a reversible manner [45,46]. While we did observe an association between abacavir and STEMI rather than NSTEMI/unstable angina presentation in fully adjusted models, the numbers were small (only seven PLWH on abacavir presented with a STEMI), and these findings would need to be validated in larger studies. Other HIV-specific variables including CD4⁺ cell count and HIV viral load were not significantly associated with ACS type or management in our study, although both low CD4⁺ nadir and high HIV viral load are known to be associated with CVD risk [6,47]. It is likely that the cumulative effects of these variables could not be elucidated in our cross-sectional study.

Our study has several limitations. First, its retrospective observational design limits our ability to measure additional confounders or control for unmeasured confounders while reliance on detailed chart abstraction and ICD-9 codes for comorbidity data can result in misclassification bias if either group was more engaged in care prior to their ACS event. Since the ACS event was many patients' first encounter with our healthcare system, detailed comorbidity data prior to presentation could not be obtained for some patients. In analyzing data from 2004 to 2013, we were unable to account for the most modern PCI practices; however, our study demonstrates that a discrepancy in stent choice existed at the time that would be important to address in a follow-up study. In this cross-sectional analysis, we were unable to assess mortality, revascularization rate, recurrent ACS, or other major adverse cardiac events, which would additionally inform the management of PLWH who are hospitalized with ACS. Among PLWH, we were unable to assess the duration of ART exposure, CD4⁺ nadir, and peak or duration of HIV viremia, all of which have been shown

to be significantly associated with CVD risk [5,14,47]. Overall small numbers of events limited our ability to perform additional subgroup analyses. Among PLWH we were unable to include those on the most modern ART regimens including second-generation INSTIs, and after guidelines changed to recommend starting ART in all PLWH regardless of CD4⁺ cell count [48]. Therefore, it may be difficult to generalize these results to PLWH who have been newly diagnosed and treated only with more current ART. On the contrary, data beyond 2013 are not available, but our data remain relevant as individuals currently aging with HIV often have already experienced years of therapy with older regimens under previous HIV treatment guidelines, which may influence their ACS presentation even in the present day. In addition, our HIV population had more advanced disease (30% with stage 3 disease), which may limit generalizability to much of the United States, although is typical for the Southeastern United States [49]. Finally, in limiting our analysis to a single healthcare system, our results may not be generalizable to the rest of the population. However, unlike other studies that have used national databases, our study is more likely to be unaffected by regional and institutional variations in practice patterns and can therefore better assess differences by HIV serostatus.

Conclusion

In our study, PLWH hospitalized with ACS were more likely to present with STEMI, have more severe single-vessel disease, and have higher Gensini scores than their HIV-negative counterparts, suggesting differences in coronary plaque morphology. An observed greater likelihood of BMS vs. DES use in PLWH in the years studied points to the need to ensure that PLWH receive optimal care for non-HIV comorbidities. There were no associations between CD4⁺ cell count or HIV viral load and ACS management. These findings suggest further investigation of the mechanisms and optimal management of ACS in PLWH is urgently needed to address health conditions in the aging population of PLWH.

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Fig. 1. Comparison of stent type between persons living with HIV and HIV-negative controls who presented with acute coronary syndrome between 2004 and 2013 and underwent percutaneous coronary intervention with stent placement.

BMS, bare metal stent; DES, drug-eluting stent; PCI, percutaneous coronary intervention.

Table 1.

Baseline characteristics of persons living with HIV and HIV-negative controls presenting with acute coronary syndrome between 2004 and 2013.

Characteristic, n (%) except where specified ^a	HIV-positive, n = 86	Controls, n = 263	P value*
Age (years) mean (SD)	52.5 (9.4)	59.9 (11.8)	<0.0001
Sex (male)	72 (84)	201 (76)	0.15
Race (African-American)	53 (62)	172 (65)	0.53
Hypertension	72 (84)	198 (75)	0.10
SBP (mmHg), mean (SD)	128 (24)	137 (23)	0.003
DBP (mmHg), mean (SD)	76 (16)	77 (14)	0.57
Antihypertensive use	54 (70)	150 (75)	0.33
Dyslipidemia	51 (59)	175 (67)	0.22
Total cholesterol (mg/dl) mean (SD)	161 (46)	169 (50)	0.25
HDL cholesterol (mg/dl) mean (SD)	38 (16)	39 (12)	0.41
LDL cholesterol (mg/dl) mean (SD)	89 (40)	104 (47)	0.01
Triglycerides (mg/dl) median (IQR)	138 (83–188)	113 (75–170)	0.08
Statin use	27 (36)	112 (56)	0.002
Creatinine, mg/dl, median (IQR)	1.2 (1.0–1.8)	1.1 (0.9–1.3)	0.002
Diabetes mellitus	22 (26)	88 (33)	0.17
Prior CVA	7 (8)	25 (10)	0.70
Congestive heart failure	15 (18)	52 (20)	0.65
BMI (kg/m ²) mean (SD)	25.6 (5.4)	29.6 (5.6)	<0.0001
Smoking status			0.01
Current	39 (48)	81 (31)	
Former	19 (23)	100 (38)	
Never	24 (29)	82 (31)	
Framingham risk score, median (IQR)	12 (9–14.5)	14 (11–16)	0.001
Framingham 10-year risk (%), median (IQR)	9 (3.5–12)	10 (5–20)	0.04
HIV-specific variables			
Duration of HIV infection (years) mean (SD)	10.3 (6.7)	–	–
On ART	65 (76)	–	–
NRTI	63 (74)	–	–

Characteristic, <i>n</i> (%) except where specified ^a	HIV-positive, <i>n</i> = 86	Controls, <i>n</i> = 263	<i>P</i> value [*]
Abacavir	18 (21)	—	—
Tenofovir disoproxil fumarate	38 (45)	—	—
Zidovudine	14 (16)	—	—
Protease inhibitor	46 (54)	—	—
NNRTI	22 (26)	—	—
INSTI	13 (15)	—	—
Entry inhibitor (T20)	2 (2)	—	—
CD4 ⁺ cell count (range 0–1526 cells/ μ l)			
<200 cells/ μ l	23 (30)	—	—
200–499 cells/ μ l	32 (42)	—	—
500 cells/ μ l	22 (29)	—	—
HIV viral load (range <50–2610000 copies/ml)			
Undetectable (<50 copies/ml)	38 (58)	—	—
50–1000 copies/ml	10 (15)	—	—
>1000 copies/ml	18 (27)	—	—

ART, antiretroviral therapy; BP, blood pressure; CVA, cerebrovascular accident; IQR, interquartile range; NNRTI, nonnucleoside reverse; NRTI, nucleos(t)ide reverse transcriptase inhibitor; PLWH, persons living with HIV.

^aMissing data: SBP 24; DBP 24; antihypertensive use 71; total cholesterol 27; HDL cholesterol 27; LDL cholesterol 34; triglycerides 28; BMI 49; statin use 71; CHF 2; smoking status 4; Framingham risk score 74; 10-year Framingham risk 74; HIV duration 32; ART 1; NRTI 1; PI 1; ABC 1; NNRTI 1; INSTI 1; entry inhibitor 1; CD4⁺ cell count 9; HIV viral load 20 transcriptase inhibitor; INSTI, integrase strand transfer inhibitor; T20, enfuvirtide.

^{*}*P*value comparing characteristics in HIV-positive versus control participants obtained by chi-square (categorical) or Student's *t* or Wilcoxon rank-sum (continuous) tests as appropriate.

Table 2.

Coronary artery disease severity at baseline for persons living with HIV and HIV-negative controls presenting with acute coronary syndrome between 2004 and 2013.

Outcome	PLWH, <i>n</i> = 86	Control, <i>n</i> = 263	<i>P</i> value*
Vessels involved, <i>n</i> (%) ^a			
0	2 (3)	17 (9)	0.09
1	27 (36)	8 (4)	<0.0001
2	17 (23)	67 (34)	0.08
3	28 (38)	104 (53)	0.03
Median (IQR) Gensini score	44 (31–76)	28 (13–76)	0.01
Median (IQR) Gensini score by vessels involved			
1 Vessel	32 (16–44)	4.25 (1.0–6.5)	<0.0001
3 Vessels	70 (45–106)	49 (27–130)	0.17

ACS, acute coronary syndrome; IQR, interquartile range; PLWH, persons living with HIV.

^aLeft main, left anterior descending, left circumflex, and right coronary arteries, and ramus branch, at least 50% stenotic. Vessel involvement is missing for 79 participants; Gensini score is missing for 80 participants

* *P*value obtained by chi-square (categorical) or Wilcoxon rank-sum (continuous) tests.

Acute coronary syndrome types and interventions for persons living with HIV and HIV-uninfected controls presenting with acute coronary syndrome between 2004 and 2013.

Table 3.

Outcome	PLWH, <i>n</i> = 86	Control, <i>n</i> = 263	<i>P</i> value	Crude OR (95% CI) ^a	Adjusted OR ^b (95% CI)	Adjusted OR ^c (95% CI)
ACS type, <i>n</i> (%)						
STEMI	25 (29)	31 (12)	0.0002	3.07 (1.68–5.60)	5.01 (2.17–11.57)	5.05 (1.82–14.02)
NSTEMI	50 (58)	187 (71)	0.03	0.62 (0.37–1.03)	0.37 (0.18–0.75)	0.33 (0.14–0.79)
UA	11 (13)	45 (17)	0.34	0.57 (0.26–1.22)	0.68 (0.24–1.93)	0.76 (0.23–2.52)
Intervention, <i>n</i> (%) ^d						
Overall						
Medical management	23 (27)	81 (31)	0.46	Ref	Ref	Ref
Any revascularization	63 (73)	181 (69)		1.14 (0.66–1.98)	1.51 (0.74–3.11)	2.90 (1.01–8.39)
PCI	46 (73)	161 (89)	0.002	Ref	Ref	Ref
CABG	17 (27)	20 (11)		2.91 (1.40–6.02)	1.28 (0.45–3.62)	3.55 (0.70–18.03)
Among STEMI						
Medical management	2 (8)	7 (23)	0.14	Ref	Ref	Ref
Any revascularization	23 (92)	24 (7)		3.30 (0.62–17.69)	3.98 (0.26–61.47)	–
PCI	19 (83)	24 (100)	0.05	Ref	Ref	Ref
CABG	4 (17)	0 (0)		–	–	–
Among NSTEMI/UA						
Medical management	21 (34)	74 (32)	0.72	Ref	Ref	Ref
Any revascularization	40 (66)	157 (68)		0.87 (0.44–1.48)	0.89 (0.40–2.01)	1.65 (0.51–5.30)
PCI	27 (68)	137 (87)	0.003	Ref	Ref	Ref
CABG	13 (33)	20 (13)		3.37 (1.49–7.66)	2.41 (0.67–8.64)	8.90 (1.39–56.99)
Stent type among patients who underwent PCI, <i>n</i> (%)						
BMS	33 (72)	23 (16)	<0.0001	13.72 (5.97–31.53)	10.04 (2.81–35.95)	5.94 (1.33–26.55)
DES	13 (28)	125 (84)		Ref	Ref	Ref

All ORs conditioned on race and sex. ACS, acute coronary syndrome; BMS, bare metal stent; CABG, coronary artery bypass graft; CI, confidence interval; DES, drug-eluting stent; NSTEMI, non-ST-segment myocardial infarction; OR, odds ratio; PCI, percutaneous coronary intervention; PLWH, persons living with HIV; STEMI, ST-segment elevation myocardial infarction; UA, unstable angina.

^aConditioned on race and sex.

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^b Adjusted for age, catheterization year, smoking, hypertension, BMI, dyslipidemia, diabetes mellitus.
^c Adjusted for age, catheterization year, smoking, hypertension, BMI, dyslipidemia, diabetes mellitus, and Gensini score.
^d One control patient was transferred to an outside facility for intervention (CABG vs. PCI) and not included in this analysis.

Table 4.

Association of HIV-specific variables with acute coronary syndrome type and revascularization for persons living with HIV who presented with acute coronary syndrome between 2004 and 2013.

Variable, n (%)	STEMI, n = 25	NSTEMI/UA, n = 61	P value	Crude OR (95% CI)	Adjusted OR ^a (95% CI)	Adjusted OR ^b (95% CI)
CD4 ⁺ cell count (cells/ μ l)			0.87			
<200	8 (33)	15 (28)		1.00 (ref)	1.00 (ref)	1.00 (ref)
200–499	9 (37)	23 (43)		0.73 (0.23–2.33)	0.46 (0.12–1.74)	0.20 (0.04–1.01)
500	7 (29)	15 (29)		0.88 (0.25–3.03)	0.76 (0.17–3.39)	0.36 (0.07–1.89)
Any ART	19 (76)	46 (75)	0.95	1.03 (0.35–3.06)	0.89 (0.24–3.32)	0.72 (0.17–3.16)
PI ^c	15 (79)	31 (69)	0.41	1.69 (0.48–6.03)	1.72 (0.42–7.05)	2.87 (0.64–12.98)
ABC ^c	7 (37)	11 (24)	0.31	1.80 (0.57–5.72)	2.40 (0.63–9.13)	5.08 (1.02–25.26)
HIV viral load (copies/ml)			0.43			
Undetectable	13 (57)	25 (58)		1.00 (ref)	1.00 (ref)	1.00 (ref)
50–1000	2 (9)	8 (19)		0.48 (0.09–4.84)	0.45 (0.07–2.75)	0.46 (0.08–2.78)
>1000	8 (35)	10 (23)		1.54 (0.49–4.84)	1.48 (0.38–5.76)	3.68 (0.68–19.90)
CABG or PCI, n = 63		No CABG or PCI, n = 23	P value	Crude OR (95% CI)	Adjusted OR ^a (95% CI)	Adjusted OR ^b (95% CI)
CD4 ⁺ cell count (cells/ μ l)			0.10			
<200	14 (25)	9 (40)		1.00 (ref)	1.00 (ref)	1.00 (ref)
200–499	27 (49)	5 (23)		3.47 (0.98–12.35)	5.25 (1.31–21.02)	1.68 (0.22–12.80)
500	14 (18)	8 (36)		1.13 (0.34–3.76)	1.43 (0.38–5.42)	0.25 (0.03–1.79)
Any ART	50 (79)	15 (65)	0.18	2.05 (0.72–5.88)	3.32 (0.99–11.17)	1.18 (0.22–6.22)
PI ^c	36 (57)	10 (45)	0.34	1.03 (0.28–3.83)	1.21 (0.31–4.79)	2.29 (0.48–11.03)
ABC ^c	12 (19)	6 (27)	0.41	0.42 (0.12–1.46)	0.59 (0.16–2.21)	1.42 (0.24–8.48)
HIV viral load (copies/ml)			0.58			
Undetectable	30 (61)	8 (47)		1.00 (ref)	1.00 (ref)	1.00 (ref)
50–1000	7 (14)	3 (18)		0.62 (0.13–2.97)	0.54 (0.11–2.75)	0.48 (0.08–3.07)
>1000	12 (24)	6 (35)		0.53 (0.15–1.87)	0.46 (0.11–1.93)	4.35 (0.28–67.36)

ABC, abacavir; ACS, acute coronary syndrome; ART, antiretroviral therapy; CABG, coronary artery bypass graft; CI, confidence interval; NSTEMI, non-STEMI; OR, odds ratio; PI, protease inhibitor; PLWH, persons living with HIV; STEMI, ST-segment elevation myocardial infarction; UA, unstable angina.

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^a Adjusted for age, sex, race, smoking status, and year.
^b Adjusted for age, sex, race, smoking status, year, and Gensini score.
^c Among those receiving ART.