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HIV Infection Is Associated with Greater Left Ventricular Mass in the Multicenter AIDS Cohort Study

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Abstract

HIV infection has been associated with diastolic heart failure and atrial fibrillation. The purpose of this study is to determine whether HIV infection is associated with differences in left ventricular mass (LVM), left ventricular end-diastolic volume (LVEDV), and left atrial volume (LAV) indexed to body surface area (left ventricular mass index, left ventricular end-diastolic volume index [LVEDVI], and left atrial volume index [LAVI], respectively). Cross-sectional study of 721 men [425 HIV-infected (HIV+), 296 HIV-uninfected (HIV−) enrolled in the cardiovascular substudy of the Multicenter AIDS Cohort Study (MACS). Participants underwent cardiac computed tomography imaging. A blinded reader measured LVM, LVEDV, and LAV. We used multivariable linear regression models to evaluate whether LVEDVI, left ventricular mass index (LVMI), and LAVI differed by HIV serostatus, adjusting for demographics and cardiovascular disease risk factors. LVMI was significantly greater in HIV+ compared with HIV− men, with adjusted difference of 2.65 g/m^2 (95% confidence interval 0.53–4.77, $p < .001$). Left ventricular end-diastolic index and LAVI did not differ significantly between the two groups. HIV-related factors (nadir CD4 count, clinical AIDS diagnosis, cumulative antiretroviral therapy use, and cumulative protease inhibitor use) were not significantly associated with LVMI, LVEDVI, or LAVI. LVM was significantly higher in HIV+ than HIV− men, which may contribute to the observed increased risk for diastolic heart failure associated with HIV infection. Although HIV infection has been associated with an increased risk for atrial fibrillation, we did not find any difference in LAV by HIV serostatus.

Keywords: AIDS, HIV, cardiomyopathy, heart failure, diastolic, tomography, X-ray computed tomography, atrial fibrillation

Introduction

Since the advent of highly active antiretroviral therapy (HAART) in 1996, life expectancy of persons living with HIV (PLWH) has increased dramatically. As per CDC estimates, in 2015, roughly half of PLWH were older than 50 years.[1] With increased life expectancy, PLWH are prone to chronic health conditions, particularly cardiac diseases. PLWH have increased risk of cardiovascular events,[2] heart failure,[3,4] and atrial fibrillation,[5] however, the underlying mechanisms are not fully understood. In the general population, alterations in cardiac structure underlie many cardiac syndromes. In particular, left ventricular hypertrophy is a predictor of cardiovascular events (sudden cardiac death, ventricular arrhythmias) and heart failure[6–8] and increased left atrial volume (LAV) is associated with atrial fibrillation[9] and heart failure.[10]

Some studies have shown alterations in cardiac structure in PLWH, particularly with regard to increased left ventricular mass (LVM).[11] More evidence is needed, however, to understand the impact of long-term HIV infection on cardiac structure. In this study, we compare body surface area (BSA)-indexed LVM, left ventricular end-diastolic volume (LVEDV), and LAV using contrast-enhanced cardiac computed tomography (CT) in HIV-infected (HIV+) and uninfected (HIV−) men enrolled in the Multicenter AIDS Cohort Study (MACS).

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Materials and Methods

Study population

The study population consisted of HIV+ and HIV− men enrolled in the MACS who underwent CT coronary angiography as part of an MACS Cardiovascular Ancillary Study. Detailed description of MACS and the subset of participants who had cardiac CT scans has been reported previously.[12,13] Briefly, MACS is an ongoing cohort study of the natural and treated histories of HIV-1 infection in homosexual and bisexual men, conducted in four U.S. communities in Baltimore/Washington, DC, Chicago, Pittsburgh, and Los Angeles. The study had initial enrollment in 1984–1985, with additional enrollments in 1987–1991 and 2001–2003. The cohort has recruited both HIV+ and HIV− men who attend semiannual research visits during which they undergo standardized interviews and physical examinations, and blood and urine samples are collected.

Inclusion criteria for this MACS Cardiovascular Ancillary Study are as follows: being an active MACS participant (with oversampling of HIV-infected men), age 40 to 70 years, weight less than 300 pounds, and no history of cardiac surgery or percutaneous coronary intervention, as these procedures would interfere with the measurement of coronary atherosclerosis. Exclusion criteria for CT coronary angiography included the following: atrial fibrillation, chronic kidney disease (estimated glomerular filtration rate <60 mL/min/m^2 within 30 days of the CT scan), or a history of intravenous contrast allergy. The study was approved by the institutional review boards of all participating sites, and all participants signed informed consent. All eligible participants who were enrolled completed cardiac CT scanning between January 2010 and June 2013.

Imaging and laboratory

The CT scanning methods and analytic approach were previously described.[14] Briefly, noncontrast CT and coronary CT angiography were performed using a 64-slice multidetector scanner at three centers and a 320-slice scanner at the fourth center.

Men were pretreated with beta-blockers or calcium channel blockers as needed to slow the heart rate and with sublingual nitroglycerin unless contraindicated. Quantitative data analyses were performed using automated methods on a workstation and software (AW 4.6; GE Medical Systems, Waukesha, WI) that used a Hounsfield unit-based endocardial border detection technique. Images were reconstructed with a 1.25 mm slice thickness. The mid-diastolic phases were chosen for measurements of left atrium (LA) and left ventricle (LV) volume and LV mass. The LA appendage and pulmonary veins were not included in the LA volume measurement. LVM was also simultaneously calculated automatically. The user acquiring the measurements was blind to participant characteristics and HIV serostatus. Use of contrast CT for measurement of chamber size and LVM has been validated in other studies.[15–17]

Clinical data from the MACS semiannual research visit most proximal to the CT scan were used. Laboratory assessment included serum creatinine levels within 30 days of CT angiography, serum glucose levels, total and high-density lipoprotein (HDL) cholesterol and triglyceride levels, and low-density lipoprotein (LDL) levels calculated using the Friedewald equation or measured directly if nonfasting or if triglycerides >400 mg/dL. HIV-related characteristics included plasma HIV RNA levels and CD4 T cell counts (cells/μL; current and nadir), years on antiretroviral therapy, and history of clinical AIDS. Ethnicity and smoking status were self-reported. Hypertension was defined as blood pressure >140/90 mmHg or the use of antihypertensive agents with a self-reported history of hypertension. Diabetes was defined as fasting plasma glucose ≥126 mg/dL or use of medications prescribed for diabetes with a self-reported history of diabetes.

Statistical analyses

Demographic and clinical characteristics were determined at the time of baseline cardiac CT visit and compared by HIV serostatus. Wilcoxon rank-sum test and chi-squared test were used for continuous and categorical variables, respectively. Chamber size measurements were indexed to BSA to generate left ventricular mass index (LVMI), left ventricular end-diastolic index (LVEDI), and left atrial volume index (LAVI), which were used for statistical analyses. We conducted two analyses to assess the relationships between HIV infection and LVMI, left ventricular end-diastolic volume index (LVEDVI), and LAVI.

In the first analysis, we used linear regression to evaluate whether the levels of the three indices obtained from the baseline cardiac CT scan differed by HIV serostatus. We constructed separate models for each outcome. We first reported results from a univariable model that included HIV serostatus as the exposure. Then, in a minimally adjusted model, we accounted for the differences in demographic characteristics between HIV+ and HIV− men by including age, race (African American, Hispanic/other vs. white), MACS center, and enrollment cohort (pre/post 2001). Finally, we accounted for potential confounding due to cardiovascular disease (CVD) risk factors using a model that additionally adjusted for systolic and diastolic blood pressure (BP), use of antihypertensive medication, fasting glucose, use of diabetic medication, total and HDL cholesterol levels, use of lipid-lowering medication, and tobacco smoking (never, former, and current).

We also conducted analysis only among HIV+ men to investigate the relationships between the baseline levels of the three outcomes and HIV-related factors (detectable viral load, defined as HIV viral load >50 copies/mL, the most recent CD4 cell count, nadir CD4 cell count, prior clinically defined AIDS diagnosis, and the duration of HAART use and protease inhibitor [PI] use). We added each of the HIV-related factors to a separate model that simultaneously accounted for demographic and CVD risk factors as described previously.

In the multivariable regression analyses, data were missing for CVD risk factors (blood pressure: $N=2$; antihypertensive medication: $N=6$; fasting glucose: $N=9$; diabetic medication: $N=15$; cholesterol levels: $N=2$; cholesterol-lowering medication: $N=17$; tobacco smoking: $N=6$). To account for missing values in these covariates, we conducted multiple imputations using the Markov-chain Monte Carlo method by assuming multivariable normality.[18] Ten multiply imputed data sets were obtained. Model coefficients were estimated in each imputed data set and then pooled using the PROC MIANALYZE procedure in SAS. All statistical analyses were conducted using SAS, version 9.4 (SAS Institute, North Carolina). p Values below .05 were considered statistically significant.

Results

Participant characteristics

Among the 759 participants who underwent contrast-enhanced CT scanning, 721 had scans suitable for LVMI, LVEDVI, and LAVI; of those, 425 were HIV+ and 296 HIV−. HIV+ men were younger (mean 51 years vs. 54 years, $p < .001$) and more likely to be non-white (68% vs. 49% $p < .001$) (Table 1). HIV+ men had lower HDL (45.3 mg/dL vs. 51.1 mg/dL) and LDL cholesterol (106 mg/dL vs. 116 mg/dL) and higher triglyceride levels (128.5 mg/dL vs. 107.5 mg/dL). Both groups had similar prevalence of hypertension and diabetes. HIV+ men were less likely to drink alcohol and had a lower body mass index. Median cumulative tobacco use

Table 1. Baseline Characteristics by HIV Serostatus

Characteristics	*HIV-infected* (N = *425*)	*HIV-uninfected* (N = *296*)	p
Age, median (IQR), years	51 (47–57)	54 (50–61)	<.001
Race, *n* (%)			
White, non-Hispanic	210 (49)	201 (68)	<.001
Black, non-Hispanic	148 (35)	72 (24)	
Hispanic/other	67 (16)	23 (8)	
Center, *n* (%)			
Baltimore	107 (25)	94 (32)	.094
Chicago	121 (29)	67 (23)	
Pittsburgh	72 (17)	58 (20)	
LA	125 (29)	77 (26)	
Cohort, *n* (%)			
Pre-2001	194 (46)	190 (64)	<.001
2001+	231 (54)	106 (36)	
Hypertension, *n* (%)	178 (44)	116 (42)	.54
Systolic blood pressure, median (IQR), mmHg	125 (115–136)	128 (118–137)	.10
Diastolic blood pressure, median (IQR), mmHg	77 (72–84)	78 (71–83)	.70
Hypertension medication use, *n* (%)	132 (31)	86 (30)	.65
Diabetes, *n* (%)	45 (11)	20 (7)	.086
Glucose level, median (IQR), mg/dL	97 (90–107)	96.5 (88.5–103)	.032
Diabetes medication use, *n* (%)	35 (8)	16 (6)	.15
Total cholesterol, median (IQR), mg/dL	187 (163–214)	195 (168–220)	.016
LDL cholesterol, median (IQR), mg/dL	106 (84–134)	116 (94–139)	.001
HDL cholesterol, median (IQR), mg/dL	45 (39–55)	51 (42–61)	<.001
Triglyceride, median (IQR), mg/dL	129 (89–197)	108 (74–147)	<.001
Lipid-lowering medication, *n* (%)	137 (33)	89 (31)	.57
BMI, median (IQR), kg/m^2	25.6 (23.1–28.3)	26.5 (24.0–29.7)	.002
Tobacco use, *n* (%)			
Never	109 (26)	67 (23)	.011
Former	187 (44)	161 (55)	
Current	127 (30)	64 (22)	
Cumulative tobacco use, median (IQR), pack-years	5.9 (0–22.2)	4.5 (0–23.1)	.59
Alcohol use, *n* (%)			
None	95 (23)	39 (13)	<.001
1 to 3 drinks/week	227 (54)	140 (48)	
4 to 13 drinks/week	79 (19)	83 (28)	
More than 13 drinks/week	22 (5)	30 (10)	
HIV clinical characteristics			
Undetectable HIV RNA copies, *n* (%)	342 (81)		
HIV RNA level, median (IQR), copies/mL	993 (143–26,350)		
Current CD4+ T cell count, median (IQR), cells/mL	599 (437–766)		
Nadir CD4+ T cell count, median (IQR), cells/mL	299 (187–432)		
Current HAART use, *n* (%)	378 (89)		
Cumulative HAART exposure, median (IQR), years	9.2 (6.2–12.2)		
Current PI use, *n* (%)	199 (47)		
Cumulative PI exposure, median (IQR), years	4.8 (0.3–9.2)		
Current NNRTI use, *n* (%)	202 (48)		
Cumulative NNRTI exposure, median (IQR), years	3.9 (0.6–7.9)		
Current NRTI use, *n* (%)	375 (89)		
Cumulative NRTI exposure, median (IQR), years	11.4 (7.6–14.2)		
History of AIDS, *n* (%)	47 (11)		

BMI, body mass index; HAART, highly active antiretroviral therapy; HDL, high-density lipoprotein; IQR, interquartile range; LA, Los Angeles; LDL, low-density lipoprotein; NNRTI, non-nucleoside reverse transcriptase inhibitors; NRTI, nucleoside analog reverse-transcriptase inhibitors; PI, protease inhibitor.

Table 2. Association of HIV Serostatus with LVMI: Unadjusted Model (Model 1), Minimally Adjusted Model (Model 2), Fully Adjusted Model (Model 3)

	Model 1		*Model 2*		*Model 3*	
Variables	*Mean difference in LV mass (95% CI)*	p	*Mean difference in LV mass (95% CI)*	p	*Mean difference in LV mass (95% CI)*	p
HIV infection	2.93 (0.78 to 5.07)	.01	2.66 (0.48 to 4.84)	.02	2.65 (0.53 to 4.77)	.01
Age, per 10 years			−1.34 (−3.23 to 0.56)	.17	−2.99 (−4.87 to −1.11)	.00
African American (ref = white)			4.02 (1.24 to 6.79)	.01	2.97 (0.20 to 5.74)	.04
Hispanic/other (ref = white)			−1.36 (−5.14 to 2.42)	.48	−0.39 (−4.05 to 3.26)	.83
2001+ cohort (ref = pre-2001)			−0.66 (−3.45 to 2.14)	.64	−1.61 (−4.31 to 1.09)	.24
Systolic blood pressure, per 10 mmHg					3.41 (2.45 to 4.38)	<.001
Diastolic blood pressure, per 10 mmHg					−0.82 (−2.23 to 0.60)	.26
On antihypertensive medication					1.03 (−1.28 to 3.34)	.38
Fasting glucose, per 10 mg/dL					0.14 (−0.33 to 0.62)	.55
On diabetes medication					2.07 (−2.30 to 6.44)	.35
Total cholesterol, per 10 mg/dL					−0.28 (−0.56 to −0.00)	.05
HDL cholesterol, per 10 mg/dL					0.02 (−0.68 to 0.73)	.95
On lipid-lowering medication					1.03 (−1.30 to 3.36)	.38
Former smoker (ref = never)					0.36 (−2.13 to 2.85)	.78
Current smoker (ref = never)					3.16 (0.25 to 6.06)	.03

Also includes adjustment for MACS clinic site.
CI, confidence interval; LV, left ventricle; LVMI, left ventricular mass index; MACS, Multicenter Aids Cohort Study.

between the two groups was similar; however, HIV+ men were more likely to be current smokers. Among the HIV+ men, average current CD4+ count was 599 cells/μL, 81% had undetectable viral load, 91% were currently on HAART, and 13% had a history of AIDS (Table 1).

Cardiac CT findings

There were 721 participants with scans suitable for measurement and included in the analysis. To address whether LVMI, LVEDVI, and LAVI differed by serotype, the three models described above were constructed for each variable of interest as shown in Tables 2–4, respectively. For each variable of interest (LVMI, LVEDI, LAVI), model 1 reports unadjusted values by serotype, model 2 adjusts for demographic variables (minimally adjusted model), and model 3 is a fully adjusted model, adjusting for both demographic variables and CVD risk factors.

In all three models, HIV+ men had greater LVMI than HIV− men, with a mean difference of 2.90 g/m^2 (54.48 vs.

Table 3. Association of HIV Serostatus with LVEDVI: Unadjusted Model (Model 1), Minimally Adjusted Model (Model 2), Fully Adjusted Model (Model 3)

	Model 1		*Model 2*		*Model 3*	
Variables	*Mean difference in LV volume (95% CI)*	p	*Mean difference in LV volume (95% CI)*	p	*Mean difference in LV volume (95% CI)*	p
HIV infection	1.85 (−0.41 to 4.10)	.11	0.25 (−1.99 to 2.49)	.83	1.13 (−1.14 to 3.39)	.33
Age, per 10 years			−3.41 (−5.35 to −1.47)	<.001	−2.85 (−4.86 to −0.84)	.01
African American (ref = white)			0.15 (−2.70 to 3.00)	.92	0.21 (−2.75 to 3.17)	.89
Hispanic/other (ref = white)			3.13 (−0.75 to 7.01)	.11	3.63 (−0.28 to 7.54)	.07
2001+ cohort (ref = pre-2001)			2.24 (−0.63 to 5.11)	.13	2.20 (−0.68 to 5.09)	.13
Systolic blood pressure, per 10 mmHg					0.48 (−0.56 to 1.52)	.365
Diastolic blood pressure, per 10 mmHg					−1.87 (−3.39 to −0.36)	.02
On antihypertensive medication					−1.59 (−4.06 to 0.88)	.21
Fasting glucose, per 10 mg/dL					−0.39 (−0.89 to 0.12)	.14
On diabetes medication					0.02 (−4.66 to 4.69)	.00
Total cholesterol, per 10 mg/dL					−0.07 (−0.36 to 0.23)	.66
HDL cholesterol, per 10 mg/dL					0.98 (0.23 to 1.74)	.01
On lipid-lowering medication					−2.96 (−5.43 to −0.48)	.02
Former smoker (ref = never)					−1.58 (−4.24 to 1.08)	.24
Current smoker (ref = never)					−2.42 (−5.53 to 0.68)	.13

Also includes adjustment for MACS clinic site.
LVEDV, left ventricular end-diastolic volume index.

Table 4. Association of HIV Serostatus with LAVI: Unadjusted Model (Model 1), Minimally Adjusted Model (Model 2), Fully Adjusted Model (Model 3)

	Model 1		*Model 2*		*Model 3*	
Variables	*Mean difference in LA volume (95% CI)*	p	*Mean difference in LA volume (95% CI)*	p	*Mean difference in LA volume (95% CI)*	p
HIV infection	−0.06 (−1.74 to 1.61)	.94	0.68 (−0.98 to 2.34)	.42	1.03 (−0.66 to 2.72)	.23
Age, per 10 years			3.10 (1.66 to 4.54)	<.001	3.30 (1.80 to 4.80)	<.001
African American (ref = white)			−1.47 (−3.58 to 0.64)	.17	−2.16 (−4.37 to 0.05)	.06
Hispanic/other (ref = white)			2.56 (−0.31 to 5.44)	.08	2.45 (−0.47 to 5.37)	.10
2001+ cohort (ref = pre-2001)			2.87 (0.74 to 5.00)	.01	2.91 (0.76 to 5.07)	.01
Systolic blood pressure, per 10 mmHg					0.37 (−0.41 to 1.15)	.35
Diastolic blood pressure, per 10 mmHg					0.01 (−1.12 to 1.15)	.98
On antihypertensive medication					0.66 (−1.20 to 2.52)	.49
Fasting glucose, per 10 mg/dL					0.09 (−0.29 to 0.47)	.64
On diabetes medication					1.26 (−2.28 to 4.80)	.48
Total cholesterol, per 10 mg/dL					−0.08 (−0.30 to 0.15)	.50
HDL, per 10 mg/dL					0.49 (−0.07 to 1.05)	.09
On lipid-lowering medication					−2.98 (−4.83 to −1.14)	.00
Former smoker (ref = never)					−1.69 (−3.68 to 0.30)	.10
Current smoker (ref = never)					−1.83 (−4.15 to 0.50)	.12

Also includes adjustment for MACS clinic site.
LAVI, left atrial volume index.

51.58, $p < .001$) in the fully adjusted model. In model 3, our study also demonstrated well-known associations. In particular, higher systolic BP, African American race, and smoking were positively associated with LVMI, and increasing age was inversely associated with LVMI.

The adjusted difference in mean LVEDV index between the two groups in the fully adjusted model was 0.97 mL/m^2 and not statistically significant (59.47 mL/m^2 vs. 58.50 mL/m^2 in HIV+ vs. HIV−, respectively, $p = .42$) (Table 3). Similarly, there was no difference in mean LAVI (mL/m^2) between HIV+ and HIV− men (46.09 mL/m^2 vs. 45.39 mL/m^2, respectively, $p = .43$) (Table 4).

Association of HIV clinical factors with chamber size

Among HIV+ men, there were no associations between cardiac chamber size and clinical HIV factors measured. Specifically, detectable viral load (defined as HIV viral load >50 copies/mL), the most recent CD4 cell count, nadir CD4 cell count, prior clinically defined AIDS diagnosis, and the duration of HAART use and PI use were not related to LVMI, LVEDVI, and LAVI (Table 5).

Discussion

Since the advent of HAART, there has been a well-described evolution of the cardiac manifestations of HIV infection, otherwise termed HIV-associated cardiomyopathy (HIVAC).[19] In pre-HAART era, HIVAC was characterized as left-ventricular dilation with symptomatic systolic dysfunction.[20] In the current era of widespread HAART use, diastolic dysfunction is the hallmark of HIVAC.[21] The mechanism behind this shift in phenotype is not fully understood, although the antiretroviral medications themselves have been implicated.[20,22]

In the current study, HIV+ men had greater LVMI compared with HIV− men. These results add to a growing body of evidence demonstrating increased LVM in PLWH, and represent

Table 5. Association of HIV Clinical Factors with LVMI, LVEDVI, LAVI Among HIV-Infected Men: Fully Adjusted Model ($n = 425$)

Variables	*Mean difference in LVMI (95% CI)*	p	*Mean difference in LVEDVI (95% CI)*	p	*Mean difference in LAVI (95% CI)*	p
Detectable VL, VL >50 copies/mL	1.86 (−2.08 to 5.81)	.35	3.64 (−0.13 to 7.42)	.06	1.32 (−1.54 to 4.17)	.37
Most recent CD4+ T cell count, per 100 cells/mm^3	−0.33 (−0.89 to 0.23)	.24	−0.50 (−1.03 to 0.04)	.07	−0.25 (−0.66 to 0.15)	.22
Cumulative HAART use, per 1 year	0.18 (−0.18 to 0.53)	.33	0.06 (−0.28 to 0.40)	.72	−0.03 (−0.29 to 0.23)	.82
Nadir CD4+ T cell count, per 100 cells/mm^3	−0.20 (−0.74 to 0.35)	.47	−0.19 (−0.71 to 0.33)	.48	−0.22 (−0.61 to 0.18)	.28
Cumulative PI use, per 1 year	0.11 (−0.21 to 0.42)	.51	0.17 (−0.13 to 0.47)	.27	0.09 (−0.14 to 0.32)	.42
AIDS diagnosis	−2.23 (−6.99 to 2.52)	.36	1.56 (−3.01 to 6.12)	.50	0.08 (−3.36 to 3.52)	.96

VL, viral load.

the largest cohort to demonstrate this finding. Mansoor *et al.* demonstrated an association between increased echocardiographic LVM and HIV infection in women in the Women's Interagency Health Study.[11] Similarly, Hsue *et al.*, found greater LVM in HIV-infected persons compared with uninfected individuals using echocardiography and greater diastolic dysfunction in conjunction with increased LVM.[22] Lipshultz found this association to hold true for children.[23] These findings, in total, support the notion that HIVAC in the era of HAART is characterized by diastolic dysfunction, and that increased LVM may be underlying this observed clinical outcome.

Interestingly, none of the HIV clinical factors evaluated was associated with LVM. Neither degree of control of the virus (nadir and current CD4+ T cell count, detectable viral load, and history of clinical AIDS) nor antiretroviral use (cumulative HAART use and cumulative PI use) was associated with LVM (Table 5). Our inability to identify an HIV clinical factor related to LVM may due to the power of the study or to a relatively homogenous study population (with regard to control of HIV and HAART use). Alternatively, the lack of association between LVM and HIV clinical factors in our study may indicate that the effects of long-term HIV infection on LVM are unrelated to the level of HIV control achieved or the medications used to achieve control. The present study excluded subjects with chronic kidney disease (estimated glomerular filtration rate <60 mL/min/m^2 within 30 days of the CT scan). As chronic kidney disease (CKD) is known to correlate positively with LVM[24] as well as HIV infection,[25] examination of CKD as a possible covariate represents a potential avenue for future research. Future research areas could also include examining the effect of coinfections, opportunistic infections, and other individual HAART agents on LVM in PLWH.

The other cardiac parameters measured, LVEDV and LAV, did not differ between the HIV+ and HIV− men. HIV has been implicated as a risk factor for atrial fibrillation.[26] In this cohort, LAV was similar between HIV+ and HIV− men, suggesting that any increase in atrial fibrillation incidence in HIV populations is unrelated to increased chamber size and is perhaps due to the infiltrative properties of virus or a treatment side effect.

Our study has several limitations. There were differences in characteristics between HIV+ and HIV− men; however, hypertension, the strongest predictor of increased LVM, was similar between the two groups. Importantly, all measured cardiac risk factors were controlled for in the multivariable analyses. In addition, the cohort included men only, which limits generalizability to women. Of note, however, Mansoor also demonstrated an association between HIV and increased LVM in a study only including women enrolled in the Women's Interagency Health Study.[11]

The main result of the current investigation is the positive association between HIV infection and increased LVM. This finding has important implications in understanding the current manifestation of HIVAC and in the care of patients with HIV. Further research using the MACS CVD cohort will include change in cardiac structure over time and possibly clinical outcomes (such as heart failure, coronary events) in this population.

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Author Disclosure Statement

No competing financial interests exist.

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