Original Article

Structural Echocardiographic Abnormalities Seen in HIV/AIDS Patients are Independent of CD4 Count

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Abstract

Introduction: The human immunodeficiency virus (HIV) infection remains one of the most daunting public health challenges today. Cardiac involvement in HIV/acquired immune deficiency syndrome (AIDS) is frequent and has been recognized on autopsy since the emergence of the pandemic. The objective of the study was to assess the pattern of structural echocardiographic (echo) findings in HIV/AIDS patients and compare this to the echo findings in apparently healthy HIV-negative controls. **Materials and Methods:** One hundred and fifty HIV-positive patients were recruited consecutively from the HIV patients attending the University of Ilorin Teaching Hospital, Ilorin, North Central, Nigeria. One hundred and fifty age- and sex-matched controls were also recruited from the surrounding community. All the individuals had clinical examination, electrocardiography (ECG) and echocardiography (echo) done. **Results:** ECG abnormalities were seen in 55.3% of the HIV-positive patients compared with 2.7% of controls ($P < 0.001$). The overall prevalence of echo abnormalities among the patients was 54%, against 15.3% ($P < 0.001$) of the controls. All the structural dimensions of the cardiac chambers were significantly greater than the cardiac chamber dimensions in the controls except for left atrial dimension (LAD). When the patients were considered in two groups of those with CD4 count less than 200 cells/mm^3 than those with CD4 count more than 200 cells/mm^3, the structural chamber dimensions were similar between both groups. **Conclusions:** Echo is an important tool for detecting cardiac abnormalities in HIV/AIDS patients. There is a high prevalence of echo abnormalities among HIV patients seen in our centre. The HIV infection was associated with increased structural dimensions of cardiac chambers compared with HIV-negative controls. This however did not seem to be related to disease severity as the chamber dimensions were similar between those with CD4 count below and above 200 cells/mm^3.

Keywords: *Echocardiography, CD4 count, human immunodeficiency virus/acquired immune deficiency syndrome*

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Introduction

The human immunodeficiency virus (HIV) infection is one of the greatest health challenges facing the developing world today. Cardiac involvement in HIV/acquired immune deficiency syndrome (AIDS) has been recognized at autopsy since the beginning of the pandemic.[1] Recent advances in the knowledge of HIV replication and transmission and the emergence of effective antiretroviral therapies are leading to longer survival times for HIV-infected individuals. As a result, organ-related manifestations of late-stage HIV infection, including HIV-related heart diseases have become more prevalent.[2]

Cardiac involvement in HIV-seropositive patients is relatively common and is associated with increased morbidity and mortality.[3,4] It could occur very early in the evolution of the disease and echocardiography (echo) has been shown to be a useful noninvasive tool in detecting cardiac abnormalities in all stages of the disease.[5,6] The purpose of this study was to describe the pattern of electrocardiographic and structural echocardiographic findings among patients attending the

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HIV clinics of the University of Ilorin Teaching Hospital (UITH), Ilorin, Nigeria, and compare the findings with those of an apparently healthy HIV-negative cohort.

Materials and Methods

Study population and recruitment

This consisted of adult HIV/AIDS patients aged 18 years and above who were consecutively recruited from the Medical Outpatient Department and other HIV clinics of UITH. All individuals who had preexisting systemic hypertension, other cardiovascular disease, diabetes mellitus, sickle cell disease, or who were presently pregnant were excluded from the study. The individuals were newly presenting patients at the clinic who were newly diagnosed with HIV for 3 months or less but not yet commenced on highly active anti-retroviral therapy (HAART).

Healthy controls were also recruited comprising individuals of similar age and sex as the HIV-positive individuals. The controls were recruited from apparently healthy HIV-negative blood donors at the UITH blood bank, students, and other members of the hospital community at large willing to participate in the study. HIV screening was carried out on controls after appropriate pretest counseling to determine their HIV status and confirm that they were negative. All controls who had preexisting systemic hypertension, other cardiovascular disease, diabetes mellitus, sickle cell disease, or who were presently pregnant were excluded from the study.

Sample size determination

The required sample size was obtained using Fisher's statistical formula for estimating minimum sample size in health studies.[7]

$$n = \frac{Z^2 pq}{d^2}$$

where n is the desired sample size; Z, standard deviation, usually set at 1.96 which corresponds to 95% confidence level; p, proportion in the target population estimated to have a particular characteristics. The regional prevalence rate of HIV infection was used 5.7% was used.[8] Therefore, $P = 0.057$.

$q = 1\text{-}p$

d is the degree of accuracy desired usually set at 0.05. Therefore, the sample size for this study was

$$n = \frac{(1.96)^2 (0.057) (0.943)}{(0.05)^2}$$

$n = 82$

The minimum required sample size of patients was 82. However, to improve the power of the study, 150 HIV-positive patients and 150 controls were recruited consecutively during clinic visits.

A structured questionnaire was used to obtain relevant information on demographic characteristics, presence of cardiovascular risk factors, or other ongoing treatment for any disease from all the participants. Venous blood was drawn from all the HIV-positive participants for CD4 count, fasting blood sugar (FBS), total cholesterol (T-CHOL), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), and triglycerides. The viral load of the patients was not done because of nonavailability of the facility at UITH.

ECG was done on all the patients and controls using a Schiller Cardiovit AT-10 ECG machine operating at a speed of 25 mm/s and sensitivity of 10 mm/mV. This was performed in accordance with the American Heart Association specifications.[9] ECG abnormalities were defined as follows: left atrial enlargement (LAE) was diagnosed by the presence of P mitrale-a notched P wave of greater than 120 ms duration in Lead II (with the duration of the notch exceeding 40 ms), duration of the terminal deflection of the P wave in V1 greater than 40 ms; right atrial enlargement (RAE)-presence of a tall peaked P wave in Lead II, duration of the initial deflection of the P wave in V1 greater than 40 ms; right ventricular hypertrophy (RVH) was established when the ratio of R:S wave amplitude in V1 was more than 1, when R wave amplitude in V1 was greater than 7 mm; left ventricular hypertrophy was diagnosed the sum of the R wave in V5 or V6 and the S wave amplitude in V1 exceeded 3.5 mV, or when The R wave amplitude in lead V5 exceeds 26 mm, or when the sum of the S wave amplitude in V3 and R wave in a VL exceeded 2 mV in females and 2.8 mV in males.[10,11]

A single cardiologist who was blinded to the HIV status of the study participants conducted an echocardiographic study on all the study individuals. An Aloka SSD-4000 (2004) echocardiography machine was used for this assessment using established standard technique and parameters by the American Society of Echocardiography (ASE) recommendation.[12] 2-D-guided M-mode images of the right ventricle (RV), left ventricle (LV), and the left atrium were displayed and the right ventricular (RVIDd) left ventricular internal dimension in diastole (LVIDd) and in systole (LVIDs), interventricular septum thickness in diastole (IVSd) and in systole (IVSs), posterior wall thickness in diastole (PWd) and systole (PWs), aortic root dimension (AO) and left atrial dimension (LAD) were measured using the leading edge-to-leading edge technique. The presence of pericardial disease, pericardial

effusion (PE), or evidence of myocardial disease was also noted. Dilated cardiomyopathy (DCM) was diagnosed when there was presence of left ventricular dilation with thinned out walls and systolic dysfunction. Ethical clearance for the study was obtained from the ethics and research committee of the UITH. An informed and written consent was obtained from each participant.

Null hypotheses

[1] Cardiac abnormalities as detected by echo will occur with similar frequency among patients with HIV/AIDS and apparently healthy individuals.

[2] The occurrence of structural abnormalities in HIV/AIDS patients, if at all, will be unrelated to disease progression as measured immunologically by the CD4 count.

Statistical analysis

Quantitative and qualitative data obtained were analyzed using SPSS 16 computer software package. The frequencies and mean ± standard deviation were generated. Quantitative and qualitative demographic characteristics were summarized and the data tabulated. The student's independent *t* test was used to compare the means of continuous variables between two groups whereas $\chi 2$ was used to compare proportions.

Results

Demographic and physical characteristics of study patients and controls

As shown in [Table 1], 150 HIV-positive individuals comprising 64 males (42.7%) and 86 females (57.3%) were studied. The sex distribution among the 150 controls was the same. The mean age of the patients was 37.3 ± 8.9 years, which was similar to that of the controls, 40.1 ± 16.9 years ($P = 0.074$). The ages of patients ranged between 18 and 65 years whereas those of the controls ranged between 18 and 70 years.

Table 1: Demographic and physical characteristics of patients and controls

Variable	Patients (mean ± SD) *N* = 150	Controls (mean ± SD) *N* = 150	*P*
Age (years)	37.3 ± 8.9	40.1 ± 16.9	0.074
Sex			
Male	64 (42.7%)	64 (42.7%)	1.000
Female	86 (57.3%)	86 (57.3%)	1.000
BMI (kg/m^2)	21.3 ± 5.0	25.1 ± 4.7	0.001*
Weight (kg)	67.7 ± 15	73.1 ± 15.2	0.002*
SBP (mmHg)	112.2 ± 12.7	115.3 ± 9.5	0.017*
DBP (mmHg)	73.4 ± 9	74.1 ± 7.7	0.518

BMI = body mass index, DBP = diastolic blood pressure, SBP = systolic blood pressure. *$P < 0.05$ is statistically significant. Values are mean ± SD except when indicated.

The mean weight of the controls, 73.1 ± 15.2 kg was significantly higher than that of the patients 67.7 ± 15 kg ($P = 0.002$) and the mean BMI of the controls 25.1 ± 4.7 kg/m2 was also significantly higher than that of the patients 21.3 ± 5 kg/m2 ($P < 0.001$). The systolic blood pressure (BP) of the patients 112.2 ± 12.7 mmHg was significantly lower than that of the controls 115.3 ± 9.5 mmHg ($P = 0.017$). Among the patients, 8.7% gave history of alcohol consumption whereas 10.2% of controls took alcohol. The median CD4 count among the HIV patients was 137 [inter quartile range (IQR) = 46-246], the median FBS was 4.7 mmol/L (IQR = 4.1-5.1 mmol/L), median total cholesterol was 3.2 mmol/L (IQR = 2.3-3.8), median HDL-C 1.1 mmol/L (IQR = 0.73-3.9 mmol/L), median LDL-C was 1.9 mmol/L (IQR = 1.75-2.5 mmol/L), median triglyceride was 1.31 mmol/L (IQR = 0.92-1.71 mmol/L).

Electrocardiographic findings in patients and controls

[Table 2] shows the ECG parameters of patients and controls. ECG abnormalities occurred more frequently among the patients (55.3%) than the controls (2.7%) ($P < 0.001$). The mean heart rate of the patients was higher than that of the controls (92.9 ± 21.9 vs. 73.2 ± 9.9, respectively, $P < 0.001$). The prevalence of LAE was 20% among the patients, which was significantly higher than 2% found among the controls ($P < 0.001$). The prevalence of LVH was also higher among

Table 2: Electrocardiographic findings in patients and controls

Variable	Patients (%) *N* = 150	Controls (%) *N* = 150	*P*
Abnormal ECG	55.3	2.7	<0.001*
Heart rate (b/min) (mean ± SD)	92.9 ± 21.9	73.2 ± 9.9	<0.001*
QTc (s) (mean ± SD)	0.43 ± 0.03	0.39 ± 0.03	<0.001*
QTc prolongation	34.7	5.3	<0.001*
LAE	20	2	<0.001*
RAE	7.3	1.3	0.011*
LVH	17.3	4	<0.001*
RVH	8.7	2.0	0.01*
PVC	8	1.3	0.002*
First-degree AV block	3.3	1.3	<0.201

AV = atrioventricular, LAE = left atrial enlargement, LVH = left ventricular hypertrophy, PVC = premature ventricular contraction, RAE = right atrial enlargement, RVH = right ventricular hypertrophy. *$P < 0.05$ is statistically significant. Values are percentage unless otherwise stated.

patients 17.3% than among controls 4% ($P < 0.001$). The prevalence of RAE among the patients was 7.3%, and this was higher than 1.3% found among controls ($P = 0.011$). The prevalence of RVH among patients was 8.7%; this was also significantly higher than 2% found among controls. Premature ventricular contractions were seen in 8% of the patients whereas 0.7% prevalence was found among controls ($P = 0.002$). Five patients (3.3%) had first-degree atrioventricular block while two of the controls (1.3%) had it ($P < 0.201$). Two of the patients (1.3%) had generalized low ECG voltages, in one of them pericardial effusion was demonstrated on echo whereas the other was obese.

Table 3: The structural echocardiographic characteristics of patients and controls

Variable	Patients (mean ± SD) $N = 150$	Controls (mean ± SD) $N = 150$	*P*
Structural Echo abnormality	54%	15.3%	<0.001*
Pericardial effusion	4%	0	<0.001*
DCM	2.7%	0	0.008*
RVIDd (cm)	1.53 ± 0.48	1.36 ± 0.40	0.01*
LVIDd (cm)	4.69 ± 0.65	4.51 ± 0.50	0.006*
IVSDd (cm)	1.16 ± 0.34	1.03 ± 0.24	<0.001*
PWd (cm)	0.91 ± 0.18	0.81 ± 0.23	<0.001*
LVM (g)	192.46 ± 71.87	173.42 ± 47.42	0.023*
LVMI	118.13 ± 38.38	96.51 ± 18.77	<0.001*
LAD (cm)	3.07 ± 0.66	3.12 ± 0.52	0.542

DCM = dilated cardiomyopathy, IVSd = interventricular septal thickness in diastole, LAD = left atrial dimension, LVIDd = left ventricular dimension in diastole, LVM = left ventricular mass, LVMI = left ventricular mass index, PWd = posterior wall thickness in diastole, RVIDd = right ventricular dimension in diastole. *$P < 0.05$ is significant. Values are mean ± SD unless otherwise stated.

The prevalence of QTc prolongation among patients was 34.7%, which was significantly higher than 5.3% recorded among controls ($P < 0.001$). The mean corrected QT interval (QTc) calculated using the Bazett's formula[13] was significantly higher in the patients (0.43 ± 0.03 s) than in the controls (0.39 ± 0.03 s) ($P < 0.001$). Among the patients however, the mean QTc was similar in males (0.43 ± 0.036 s) and females (0.44 ± 0.028 s) ($P = 0.238$).

Structural echocardiographic findings in patients and controls

As shown in [Table 3], the overall prevalence of all echocardiographic abnormalities were 54% among the HIV-positive cohorts and 15.3% ($P < 0.001$) in the controls. The prevalence of PE was 4% among the patients whereas none was observed among the controls

Table 4: Relationship between CD4 count and echocardiographic abnormalities in HIV patients with CD4 count <200/mm3 and >200/mm3

ECHO abnormality	CD4 count <200/ mm^3 mean ± SD ($N = 72$)	CD4 count >200/ mm^3 mean ± SD ($N = 78$)	*P*
DCM (%)	4.2	1.3	0.273
Pericardial effusion (%)	5.6	2.6	0.35
RVIDd	1.6 ± 0.5	1.47 ± 0.5	0.1
IVSDd	1.17 ± 0.3	1.16 ± 0.4	0.848
LVIDd	4.74 ± 0.6	4.64 ± 0.7	0.382
PWd	0.82 ± 0.2	0.80 ± 0.2	0.468
LVMI	122.06 ± 36.9	113 .88 ± 39.7	0.193
LAD	3.14 ± 0.6	3.0 ± 0.7	0.205

DCM = dilated cardiomyopathy, IVSDd = interventricular septal thickness in diastole, LAD = left atrial dimension, LV = left ventricular, LVIDd = left ventricular internal dimension in diastole, PWd = posterior wall thickness in diastole, RVIDd = right ventricular internal dimension in diastole.

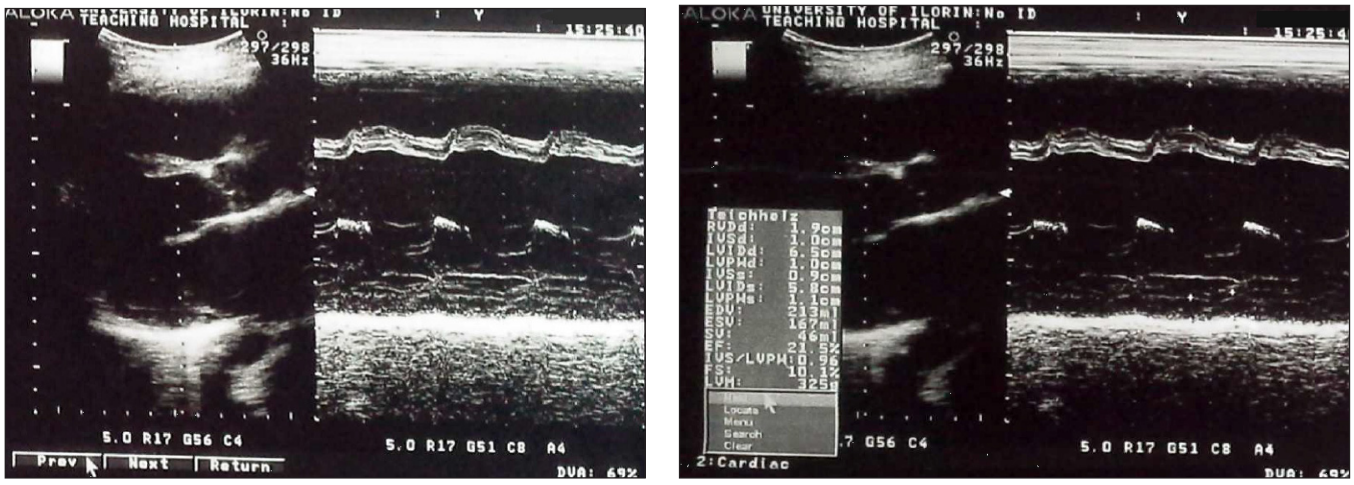

Figure 1: a and b: Echocardiographic image showing the parasternal long-axis view of a patient with dilated cardiomyopathy showing a dilated left ventricle with poor systolic function.

($P < 0.001$). Structural changes in keeping with DCM with dilated left ventricle and LVEF less than 50% occurred in 2.7% of the patients whereas none was found in the controls ($P = 0.008$) [Figure 1a] and [Figure 1b]. The mean LVIDd, RVIDd, and IVSd were higher among patients than controls ($P = 0.006$, $P = 0.010$, $P < 0.001$, respectively) whereas PWd was significantly higher in patients than controls ($P < 0.001$). The in diastole LAD was however similar in both groups ($P = 0.54$). The left ventricular mass (LVM) and the left ventricular mass index (LVMI) were higher in the patients than in the controls ($P = 0.023$ and $P < 0.001$, respectively).

Relationship between the CD4 count and electrocardiographic and structural echocardiographic parameters in the patients

When the patients were grouped according to their CD4 counts into those who had CD4 count less than 200/mm3 and those who had CD4 count more than 200/mm3, significantly more echocardiographic abnormalities were found in the earlier group (87.5 vs. 69.2%, respectively, $P = 0.007$). The structural dimensions of the cardiac chambers were however similar in both groups [Table 4].

Discussion

The subject of cardiovascular involvement in HIV infection is of great interest in view of the current dynamics and realities of care of HIV/AIDS patients. Advances in the development of effective antiretroviral therapies have caused the disease to evolve from an inevitably fatal condition to a chronic manageable illness. The patients are also beginning to live longer, and are now expected to live long enough to develop cardiac illness and other effects of long-term disease.

The contribution of HIV/AIDS to hospital admission and mortality statistics has increased gradually over the years. In the UITH, HIV/AIDS-related admissions increased from 199 to 219 in the 5-year period between 2004 and 2008 accounting for 1.6% of all hospital admissions and 0.9% of all hospital deaths during the stated period (Source - Department of Medical Health Records UITH). Chijioke and Kolo,[14] in reviewing the mortality pattern in adult medical wards in UITH over a 10-year period from 1996 to 2006 found that HIV infection accounted for 6.7% of all medical admissions. Sani *et al*.[15] in Kano, North western Nigeria, also reported an upward trend in HIV/AIDS-related admissions.

In this study, the mean weight and BMI of the controls was expectedly higher than that of the patients. Weight loss is a cardinal feature of the HIV infection and about 20% of the patients were underweight using a body mass index (BMI) cutoff of 18 kg/m2. The impact of weight loss on blood pressure is much studied and appears to be evident among our study patients. Though the etiology is largely unknown, several explanations have been postulated. Tuck *et al*.[16] observed that a decline in plasma renin activity correlated with weight reduction in obese patients placed on a weight-reducing diet. Obese individuals are also known to have higher levels of inhibition of the natriuretic peptides; hence, greater tendency to fluid retention and vasoconstriction.[17] Weight reduction is thus associated with a lesser degree of this inhibition, and consequently a lower blood pressure. This tendency to have a lower blood pressure in HIV patients may however not lower their overall cardiovascular risk. The HIV potentiates other risk factors that add to a patient's predisposition to cardiovascular disease. For example, the virus causes inflammatory reaction in the coronary vessels, which is believed to promote endothelial dysfunction and atherosclerosis[18] as well as early carotid artery atherosclerosis. Indeed HIV-1 genomic sequences have been demonstrated in the coronary vessels of HIV-infected patients who died of acute myocardial infarction.[19]

The prevalence of ECG abnormalities was higher among patients (55.3%) than controls. The reported prevalence of ECG abnormalities in HIV patients is high. The finding in our study is similar to reports in other studies by Barbaro *et al*.[20] who observed an overall prevalence of 57.2% among their study population and that of Soliman *et al*.[21] reported a prevalence rate of 51.5% among his multiracial group of 4518 HIV patients. It is higher, however, than the prevalence rate of 44% Levy *et al*.[22] reported among their patients. As the gateway to cardiovascular evaluation in most clinical settings, a high prevalence of abnormalities detected on the ECG of HIV/AIDS patients underscores the importance of cardiac evaluation in these patients at every point of their care, rather than focus disproportionately only on opportunistic infections alone as obtains commonly in the African setting.[23] Specific important electrophysiologic findings that have been reported among HIV patients are rhythm abnormalities and sudden death.[24,25] Many of these are secondary to other cardiac comorbidities and the effect of HAART medications and other medications used to treat opportunistic infections such as pentamidine or macrolides as well as cardiac autonomic dysfunction.[26-28] The QTc is one of such parameters, and a higher prevalence of QTc prolongation was observed among HIV patients than healthy controls in our study. A similar trend has been reported in our environment by Sani *et al*.[29] among patients in North western Nigeria and Ogunmola *et al*.[30] in Ekiti State, South western Nigeria. The use of macrolides to treat opportunistic infections in HIV patients is common and this would have contributed to this observation in our patients.

A few of the HIV-positive patients in this study had PE. None was however symptomatic. The prevalence rate of 4% found in this study is lower than (11.4%) reported by Danbauchi *et al*.[31] and 22% reported by Heidenreich *et al*.[32] Patient selection may have accounted for these differences because a review of the study subject characteristics suggests that the earlier mentioned workers may have had more patients in the later stages of infection. Certain factors cause pericardial effusion in HIV/AIDS. These include pericarditis caused by the HIV itself or by other opportunistic pathogens such as *Mycobacterium tuberculosis*,[33] *Cryptococcus neoformans*,[34] *Staphylococcus aureus*,[35] *Herpes simplex*,[36] *Pseudomonas aeruginosa*, *Listeria monocytogenes*, and *Klebsiella pneumonia*.[35] Malignancies such as Kaposi sarcoma and lymphomas can also present with PE.

In our study, the HIV appears to cause an increase in the dimension of cardiac chambers. With the exception of LAD, all the structural dimensions of the right and left ventricles were significantly higher in the patients than in the healthy controls. This is similar to findings by Nzuobontane *et al*.[23] in a similar study comparing HIV/AIDS patients to healthy controls and Danbauchi *et al*.[31] in Zaria, North western Nigeria. It is striking to note that Uwanuruochi *et al*.[37] from Enugu, South eastern Nigeria actually observed that the LVIDd was actually significantly smaller among HIV patients than among healthy controls. The higher dimensions of the left ventricle in our patients probably explain the significantly higher LVMI in them. The LVMI has been shown in noninfected populations to be associated with a higher mortality.[38] Some authors have suggested the increase in the structural dimensions of the heart and LVMI in HIV/AIDS may be related to the effect subclinical atherosclerosis.[39] A similar increase in left ventricular mass has been observed in studies of patients with conditions that are significantly associated with inflammation such as systemic lupus erythematosus and rheumatoid arthritis.[40,41] Thus, the higher levels of inflammation that occurs in HIV patients may in theory contribute to increased LV mass. It will be important to study prospectively the effect of treatment on the increased LV mass.

The LAD appeared unaffected by the trend observed in the ventricular dimensions. This was similarly observed by Danbauchi *et al*.[42] in their comparison of late-stage HIV/AIDS patients and healthy controls. It will be expected that the LAD should increase in tandem with other cardiac chambers. The observation in this study may have been due to the fact that only M-mode measurements of LADs were planned at the time of development of the study protocol. The measurement of the LAD in this way shows significant clinical association with the development of adverse cardiovascular outcomes like atrial fibrillation.[43] However, it measures only the anteroposterior diameter of the left atrium. The enlarging left atrium is asymmetrical and in such instances, left atrial volume may provide more accurate measurement.[44]

Depending on patient selection, a wide range of frequencies of occurrence of DCM has been reported.[45] The association between HIV infection and the development of DCM has been variously investigated and evidence, although mostly indirect tend to support a direct etiological link. Omotoso *et al*.[46] in evaluating heart failure patients found that 40.5% of them had echocardiographically confirmed DCM. About 32.1% of these were HIV positive. This high rate is not comparable to this study because the primary study cohort comprised heart failure patients. This study's DCM prevalence is also lower than that reported in Twagirumukiza *et al*.'s Rwandan cohort.[47] However, the Rwandan cohort comprised a larger number of HIV patients who were, like in this study, also consecutively recruited.

Several explanations have been suggested for the link between HIV infection and DCM. One is the fact the HIV is cardiopathogenic and causes myocarditis, which may serve as a precursor to DCM.[48] Another explanation is the effect of opportunistic infections to which HIV patients are frequently prone. One autopsy series found evidence of cardiac toxoplasmosis in about 25% of HIV-infected patients.[49] The role of oxidant stress precipitated by deficiency of trace elements such as selenium, which are naturally occurring antioxidants has also been proposed. Chariot *et al*.[50] and Twagirumukiza *et al*.[47] demonstrated that selenium deficiency does occur in HIV-positive individuals with DCM. Correction of selenium deficiency led to improvement in symptoms in majority of them.[50]

The CD4 count has been proven to be a key factor predicting survival in HIV patients. It appears that the occurrence of echo abnormalities is also related to the level CD4 count as the group with CD4 count less than 200 cells/mm3 had a significantly higher overall prevalence of echo abnormalities. The fact that none of the structural dimensions reflected this pattern is because the significantly different abnormalities were mainly in the domain of Doppler evaluation.

The effect of the HIV infection that was seen on the structural dimensions of the hearts of the study patients appear not to be related to disease severity as measured

by the CD4 count. The evaluation of the relationship between CD4 count and structural dimensions of the heart has thrown up different results over the years. The observation in this study is similar to findings by Martinez-Garcia *et al*.[51] who observed that not only were the hearts of the HIV-positive patients in their study smaller in size than those of healthy controls, the LVMI was similar in both groups of patients on either side of the 200 cells/mm3 CD4 count divide. Also, Mansoor *et al*.[52] in a large study of 654 women found that the LVMI was not associated with the nadir CD4 less than 200 cells/mm3. Considering that the LVMI is influenced by cardiac chamber wall thickness and cavity dimension, the observation in this study that cardiac chamber structural dimensions are unrelated to CD4 count is in tandem with observations by these earlier workers on the evaluation of the relationship between structural cardiac dimensions and the CD4 count. The findings by Hsue *et al*.[39] are however different because they found a significant association between the CD4 count and LVMI. However, Hsue *et al*.'s cohort of HIV-infected patients included hypertensive individuals and the significance of the correlation between nadir CD4 count and LVMI in their study was actually borderline at a *P* value of 0.052. Interestingly however, Martinez-Garcia *et al*.[51] also found a significant relationship between the LVMI and HIV viral load with the LVMI smaller in HIV patients who had a viral load more than 10,000 copies/mL compared with those who had a viral load less than 10,000 copies/mL. This will suggest that factors, other than the CD4 count, such as the effect of direct viral invasion of cardiac myocytes and the subsequent immune-mediated fibrotic process it triggers also make impact on the left ventricular mass and other structural cardiac dimensions of HIV patients.

Conclusions

In conclusion, this study showed a high prevalence of cardiac involvement in HIV/AIDS patients managed in our center. On echo, their cardiac chambers were of significantly greater dimensions than those of healthy controls. The structural echo changes however appeared to be unrelated to disease severity as measured by the CD4 count. With growing evidence of cardiac involvement in HIV/AIDS, perhaps it is time to consider comprehensive cardiac evaluation, including echocardiography as part of the baseline evaluation of patients at diagnosis. This will make for early diagnosis and treatment of cardiovascular diseases and improve the overall care of the patients.

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Conflicts of interest

There are no conflicts of interest.

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