Determinants of HIV-related cardiac disease among adults in north central Nigeria

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ABSTRACT

Objective The aim of the present study was to evaluate the determinants of HIV-related cardiac disease (HRCD) among adults in north central Nigeria. This was a hospital-based cross-sectional study recruiting patients who were HIV positive attending the HIV clinic at Jos University teaching Hospital, Nigeria.

Methods A total of 200 adults who were HIV positive and aged ≥18 years were consecutively recruited. All patients were administered a questionnaire and underwent clinical examination, laboratory investigation for haemoglobin estimation, CD4 cell count, viral load, serum lipid profile, hepatitis B surface antigen, anti-hepatitis C virus antibody, electrocardiogram and two-dimensional echocardiography Doppler studies. The outcome measure was echocardiography-defined cardiac disease, such as systolic dysfunction, diastolic dysfunction, isolated left ventricular dilatation, right ventricular dysfunction or pulmonary hypertension.

Results The mean age of the study population was 38±9 years. The majority (71%) were women and were on average younger than the men (36±8 years vs 47±9 years, p<0.0002). Highly active anti-retroviral therapy (HAART) use was seen in 84.4% of subjects. The median CD4 cell count for the study population was 358 cells/μL; the count was 459 (95% CI 321 to 550) cells/μL for subjects without HRCD and 193 (95% CI 126 to 357) cells/μL for subjects with HRCD (p<0.001). HAART-naive subjects with HRCD had a mean CD4 cell count of 121 cells/μL vs 200 cells/μL for those on HAART (p<0.01). CD4 cell count (OR = 0.25, 95% CI 0.15 to 0.45) and duration of diagnosis (OR=3.88, 95% CI 1.20 to 13.71) were the significant determinants of HRCD on multivariate analysis.

Conclusions Duration of HIV diagnosis and degree of immunosuppression were the significant determinants of HRCD. There is therefore a need to reduce cardiovascular morbidity in patients infected with HIV through early diagnosis/sustained use of HAART, early screening for HRCD and prompt intervention.

INTRODUCTION

Data from the US Centers for Disease Control and Prevention (CDC) predict that by 2015, 50% of the people living with HIV (PLHIV) will be older than 50 years,[1] reflecting the impact of highly active anti-retroviral therapy (HAART). This good news comes with increased challenges of non-communicable diseases among this population, with cardiovascular diseases being the prominent sequelae in affected individuals. HIV-related cardiac disease (HRCD) presents in different ways, with the most frequent findings being left ventricular dysfunction, isolated left ventricular dilatation, right ventricular dilatation, pulmonary hypertension and cardiomyopathy.[2–4] Sub-Saharan Africa, which is home to 70% of the global population of those infected by HIV, will definitely bear the brunt of these emerging epidemics. In a review article by Magula *et al*,[5] geography, access to and use of HAART, degree of immunosuppression, susceptibility to cardiac infections and spectrum of opportunistic infections were noted to be some of the factors that influence the frequency and pattern of cardiac dysfunction in patients infected with HIV.[5] Evidence also indicates that some anti-retroviral drugs are associated with an increased risk of coronary artery disease.[6]

In Nigeria, data on HRCD is scarce; the few available studies were performed before anti-retroviral therapy became widely accessible. We evaluated determinants of HRCD among adults receiving care and treatment in a tertiary hospital in Nigeria.

MATERIALS AND METHODS

The present work was part of a larger study of HRCD, a cross-sectional study involving 200 patients who were HIV positive aged 18 years and above, 84.4% of whom were on HAART. Approval for the study was given by the ethical committee of Jos University Teaching Hospital (JUTH) and the participants gave informed consent. The following were excluded from the study: those with obvious respiratory diseases, heart disease prior to diagnosis of HIV infection, patients with severe anaemia, diabetes mellitus and those with a history of significant alcohol consumption (defined as greater than 21 IU/week in men and greater than 14 IU/week in women). Subjects with connective tissue disease, sickle cell anaemia and those with peripartum cardiomyopathy were also excluded. Those eligible were then interviewed about the symptoms of heart disease using the New York Heart Association (NYHA) dyspnoea questionnaire. This was followed by a physical examination and relevant investigations, which included haemoglobin estimation, CD4 cell count, HIV viral load, serum lipid profile, hepatitis B surface antigen (HBsAg) and anti-hepatitis C virus antibody (anti-HCV) testing.

Subjects underwent two-dimensional transthoracic echocardiography with an Aloka-SSD4000 using a 3.5 MHz transducer from which M-mode images were derived, as well as Doppler flow examination. Standard views in the supine and left lateral positions were obtained, and the cardiac measurements taken using the American Society of echocardiography guidelines.[7 8]

Table 1 Echocardiography parameters used for patient classification

Issue	Parameters and values
Left ventricular (LV) systolic dysfunction	Fractional shortening <28%; ejection fraction <50%; without LV dilatation (LVIDD <55 mm)
Isolated left ventricular dilatation	LVIDD ≥55 mm with normal fractional shortening (≥28%)
Dilated cardiomyopathy (DCM)	Ejection fraction <50%; fractional shortening <28%; LVIDD >55 mm, with global hypokinaesia
Right ventricular dysfunction	RVIDD ≥30 mm or RVIDD>LVIDD
Pulmonary hypertension	PASP ≥30 mm Hg and/or peak tricuspid regurgitation (VTR) >2.5 m/s: mild 30–40 mm Hg, moderate 41–50 mm Hg, severe >50 mm Hg
Diastolic dysfunction	Grade 1: E/A <1.0, deceleration time (DCT) >220 ms, IVRT >92 ms (<30 years), >100 ms (30–50 years), >105 ms (>50 years); grade 2: E/A 1–2, DCT 150–200 ms; grade 3: E/A >2.0, DCT <150 ms

E/A ratio, ratio of the early (E) to late (A) ventricular filling velocities; IVRT, isovolumetric relaxation time; L/RVIDD, left/right ventricular internal dimension – diastole; PASP, pulmonary arterial systolic pressure; VTR, velocity of tricuspid regurgitation.

Transvalvular Doppler determination of presence and severity of valvular regurgitation were performed. Using continuous wave Doppler, sampling of peak regurgitation jet velocity across tricuspid valve was used to estimate the right ventricular to right atrial systolic pressure gradient, according to the modified Bernoulli equation. The right atrial pressure (RAP) was added to the Doppler derived peak tricuspid regurgitation velocity (VTR) as stated in the Bernoulli equation, to estimate the pulmonary arterial systolic pressure. The percentage collapse of the inferior vena cava (IVC) was used to estimate the RAP.[9 10]

Cardiac disease was defined based on echocardiography findings[8] as shown in table 1; subjects were either classed as having cardiac disease (if they had pulmonary hypertension or any other form of cardiac disease) or without cardiac disease if their echocardiography findings were normal. Subjects were classified based on the CDC immunological classification of HIV disease into those with a CD4 cell count ≥500 cells/μL, a CD4 cell count 200–499 cells/μL and those with CD4 cell count <200 cells/μL.

Statistical analysis was performed using Epi info V.3.5.3 (CDC, Atlanta, Georgia, USA). Descriptive statistics and ORs were calculated, and continuous variables were expressed as means and SD. Differences between group means were tested by two-tailed Student t test, and χ^2 statistics were calculated to test differences between proportions. Analysis of variance was used to compare means where more than two groups were involved. Multiple logistic regression models were used to determine the factors independently associated with cardiac disease. A p value<0.05 was considered significant.

RESULTS

Subjects were divided into two groups: those with cardiac disease (HIV-positive subjects with pulmonary hypertension (HRPH) and those with any other form of cardiac disease) and those who were HIV positive without cardiac disease.

A total of 71% of the study population were women. The mean age of the study population was 37±9 years, and women were younger than the men (36±8 years vs 47±10 years), χ^2 3.8, p<0.0002.

In all, 84 out of 200 patients (39.5%) had cardiac disease. The mean of the echocardiography parameters are highlighted in table 2. The commonest forms of cardiac disease among the study subjects noted were left ventricular systolic dysfunction (21 patients, 10.5%), diastolic dysfunction (20 patients, 10.0%) and pericardial disease (17 patients, 8.5%). Significantly more men (52%), compared to women (35%) had cardiac disease on univariate analysis (χ^2=8.6, p=0.01). The mean body mass index (BMI) of the study population was 24.5±4.7 and BMI had no statistically significant association with cardiac disease, p=0.41 (table 3). More subjects had normal BMI, while overweight was more frequent than underweight and obesity. A total of 53.3% of underweight subjects had HRCD, higher than any other BMI class, while 39.1% of subjects with normal BMI had HRCD. Subjects with cardiac disease (other HRCD or HRPH), 59.7% had normal BMI, 25.8% overweight, 11.3% obese and 3.2% underweight.

Alcohol use was seen in 23% of the study subjects, and had no significant association with cardiac disease (p=0.88). Men were more likely to have a history of significant alcohol use (17.2%) than women (7.7%) p<0.01. In addition, more of the men who consumed alcohol compared to women had HRCD: 43% vs 12.2%, p<0.01.

The median duration since diagnosis of HIV infection was 46 months (IQR 18–64 months). There was noticeable statistical significance between the duration of HIV diagnosis and

Table 2 Echocardiography characteristics of the study population

Parameter	Study population, n=200, mean (SD)	Cardiac disease, n=85, mean (SD)	No cardiac disease, n=115, mean (SD)	p Value
E/A ratio	1.4 (0.4)	1.5 (0.6)	1.4 (0.3)	0.06
Ejection fraction, %	62.2 (14.9)	56.3 (18.0)	66.7 (10.0)	<0.01
Fractional shortening, %	35.1 (9.6)	31.8 (11.0)	37.5 (7.6)	<0.01
LVIDD, mm	45.5 (9.8)	47.6 (9.5)	43.9 (5.7)	<0.01
LVIDS, mm	31.1 (8.1)	34.3 (9.3)	28.7 (6.0)	<0.01
LVPWD, mm	10.9 (3.4)	11.2 (4.2)	10.7 (2.6)	0.38
LVPWS, mm	13.7 (3.0)	13.6 (3.0)	13.8 (2.5)	0.66
RVSP, mm	14.4 (6.0)	15.0 (8.1)	13.6 (3.8)	0.19

E/A ratio, ratio of the early (E) to late (A) ventricular filling velocities; LVIDD, left ventricular internal dimension – diastole; LVIDS, left ventricular internal dimension – systole; LVPWD, left ventricular posterior wall dimensions, LVPWS, left ventricular posterior wall thickness at systole; RVSP, right ventricular systolic pressure

Table 3 Clinical and laboratory parameters of subjects with and without cardiac disease

Parameters	Cardiac disease, n=85	No cardiac disease, n=115	χ^2	p Value
Duration of HIV ≥40 months	49 (34)	41 (36)	1.57	0.04
HAART use, n (%)	67 (78.8)	101 (87.8)	2.95	0.09
Duration on HAART in months, mean (SD)	39 (23)	42 (24.0)	0.91	0.36
History of past TB treatment, n (%)	21 (25)	32 (28.0)	0.20	0.66
WHO staging of HIV/AIDS, n (%):			4.26	0.04
1	26 (31.0)	46 (38.6)		
2	20 (28.8)	35 (30.7)		
3	30 (35.7)	31 (27.2)		
4	8 (9.5)	4 (3.5)		
NYHA dyspnoea class, n (%):			5.07	0.02
I	13 (15.3)	17 (15.0)		
II	5 (5.9)	11 (9.2)		
III	21 (24.7)	16 (14.2)		
IV	7 (8.2	0 (0.0)		
BMI, mean (SD)	23.9 (5.0)	24.4 (4.5)	0.83	0.41
SBP, mean (SD)	118 (15.9)	119 (19.0)	0.57	0.57
DBP, mean (SD)	77 (11.7)	78 (11.0)	0.07	0.94
Hb concentration, mean (SD)	11.9 (2.0)	12.3 (1.8)	1.57	0.12
CD4 cell count, median	193	459	6.83	<0.001
Log $_{10}$ viral load, mean (SD)	3.16 (1.1)	3.22 (1.1)	0.3	0.7

Heart disease=presence of HIV-related cardiac disease (HRCD) or HRPH.
BMI, body mass index; DBP, diastolic blood pressure; HAART, highly active anti-retroviral therapy; Hb, haemoglobin concentration; NYHA, New York Heart Association; SBP, systolic blood pressure; TB, tuberculosis.

presence of cardiac disease (χ^2=1.57, p=0.04). In all, 84.4% were on anti-retroviral therapy at the time of the study, with the average duration of treatment of 41±24 months. HAART use was not associated with less risk of HRCD, 78.5% of those on it had HRCD, while 25.7% of patients on HAART still had CD4 cell count less than 200 μL. Zidovudin use did not predispose patients to heart disease, only 1.8% of patients were on protease inhibitors, and 80.6% of subjects had been on HAART for less than 60 months.

In all, 73% of the subjects had never been diagnosed as having tuberculosis. A total of 54 patients (27%) had been positive for tuberculosis, out of which 26.5% had completed treatment for tuberculosis and 0.5% were currently on anti-tuberculosis treatment. The incidence of pulmonary hypertension had no relationship to the diagnosis of tuberculosis; HRPH occurred in only two subjects (3.7%) of those who had tuberculosis or had been treated for it, while six (4.7%) of those negative had HRPH (OR 0.9, χ^2 0.02, p value 0.90). There were no intravenous drug users and no homosexuals among the study population.

ECG abnormalities were seen in 57.3% of study population with Left ventricular hypertrophy being the commonest ECG abnormality (29.0%). The various ECG findings are shown in table 4

The mean (SD) haemoglobin concentration was 12.2 (1.9) mg/dL in the overall population, 11.9 (1.9) mg/dL and 12.3 (1.8) mg/dL for subjects with cardiac dysfunction and those without cardiac disease respectively (p=0.11). In all, 37% and 7.5% of the study subjects were reactive to hepatitis B virus (HBsAg) and anti-HCV, respectively, but none had history or features of chronic liver disease. Of those reactive to HBsAg 22% had HRCD, while 7.8% of those reactive to anti-HCV had HRCD, but neither of these factors were statistically significant. Only 92% of subjects had HIV viral load results, with 47% showing detectable viral load.

Patients with cardiac disease had significantly lower median CD4 cell count (χ^2=1.57, p<0.001). The median CD4 cell count was 358 cells/μL; this was 395 cells/μL for HAART-naive subjects and 532 cells/μL for those on HAART (p<0.01). HAART-naive subjects with HRCD, had a median CD4 count of 131 cells/μL (IQR 74–422 cells/μL), while subjects with HRCD who were on HAART had median CD4 cell count of 259 cells/μL (IQR 160–498 cells/μL) (p<0.01). The median CD4 cell count for subjects on HAART without HRCD was 459 cells/μL (IQR 356–629 cells/μL) and 193 cells/μL (CI 126 to 357) (p<0.000). Dilated cardiomyopathy (DCM) tended to occur more at lower CD4 count, with an average of 180 cells/mm^3. Based on CD4 categorisation, 60% of subjects with HRCD had CD4 cell count less than 200 cells/μL, χ^2=24.3, p<0.001, (tables 5 and 6).

Table 4 ECG features of study population

ECG findings	Frequency, %
Arrhythmias	4
Bradycardia	3
First-degree heart block	3
Left atrial enlargement	1
Low voltage complex	1.5
LVH	29
LVH+bradycardia	4.5
LVH+PVC	0.5
Normal	42.5
Tachycardia	5
Right atrial enlargement	1.5
RBBB	0.5
RVH	0.5
RV strain	3

LVH, left ventricular hypertrophy; PVC, premature ventricular complex; RBBB, right bundle branch block; RV, right ventricle; RVH, right ventricular hypertrophy.

Table 5 Multiple regression analysis of determinants of HIV-related cardiac disease (HRCD)

Term	OR	95% CI	Coefficient	SE	Z statistics	p Value
Age	0.99	0.95 to 1.03	−0.02	0.02	−0.69	0.49
Sex	1.89	0.81 to 1.13	0.64	0.398	1.60	0.11
Duration of HIV diagnosis	3.88	1.20 to 13.71	1.36	0.64	2.11	0.04
HAART use	0.59	0.24 to 1.43	−0.05	0.45	−1.16	0.25
Alcohol use	0.96	0.43 to 2.16	−0.04	0.41	−0.09	0.93
BMI	1.06	0.98 to 1.14	0.06	0.04	1.41	0.16
CD4 cell count	0.25	0.15 to 0.45	−1.37	0.29	−4.81	<0.001
Log_{10} viral load	0.81	0.58 to 1.11	1.11	−0.22	−1.31	0.19
Constant	–	–	0.495	1.09	0.45	0.65

BMI, body mass index; HAART, highly active anti-retroviral therapy.

The mean (SD) of log_{10} of the viral load of the study population was 3.19 (1.1); for subjects with cardiac disease this was 3.16 (1.1) and for those without cardiac disease 3.22 (1.1) (p=0.7). Duration of HIV diagnosis (in months) and CD4 cell count showed significant association with HRCD on multivariate analysis.

DISCUSSION

Sociodemographic parameters such as sex, age, marital status, occupation, alcohol use and educational level had no association with development of cardiac disease.

The mean age of the study population was 37.9±8.8 years, with the predominant age group being 30–39 years, while the mean age of subjects with cardiac disease was 38±9.8 years. This pattern is similar to the findings of other authors in Africa.[11–13] It is a worrisome trend, since this age bracket constitutes the work force and most productive segment of the population, with an adverse effect on the economy.[14]

However, in a report from the USA by Hsue *et al*,[15] subjects presenting with HRCD were older with a mean age of 47 ±8.2 years. This may be a reflection of better patient care, and easier and early access to HAART compared with what is obtainable in Africa. Men were older in the study (47±6.0 years), compared to women (36±7.4 years), similar to the findings of Hakim *et al*,[11] perhaps because of the better health-seeking behaviour of their patients. In our study, age did not show any significant correlation with cardiac disease, probably because the majority of the subjects were in same age group (21–50 years) with the older age group contributing minimally to the study population.

Table 6 CD4 cell count grouping and subject characteristics according to gender

CD4 cell count grouping	Normal HIV positive (%), n=115	Other HRCD (%), n=77	HRPH (%) n=8
<200 cells/μL:			
Women	29.7 (11)	64.9 (24)	5.4 (2)
Men	26.1 (6)	73.9 (17)	0.0
200–499 cells/μL:			
Women	67.2 (41)	29.5 (18)	3.3 (2)
Men	52.0 (13)	32.0 (8)	11.0 (4)
>500 cells/μL:			
Women	88.6 (39)	11.4 (5)	0.0
Men	62.5 (5)	37.5 (3)	0.0

HRCD, HIV-related cardiac disease; HRPH, HIV-positive subjects with pulmonary hypertension.

The mean BMI in the study was 24.2±4.7 kg/m^2. Normal BMI was seen in 57.5% of the subjects, with 26% overweight, 11% obese and only 6% underweight. BMI did not correlate with cardiac disease in the study population. However, underweight showed a trend toward development of cardiac disease. Although HIV/AIDS is known to be associated with weight loss, the fact that majority of the subjects (84.4%) were on HAART and asymptomatic for their HIV disease could have accounted for the findings. Okeahialam and Sani[16] documented that patients who were underweight and HIV positive had significant ECG findings, and in a 7-year population study in Congo, BMI <22 kg/m^2 was predictive of cardiac disease;[12] the latter study differs from the present work because it was a prospective study. An association has been established between an increase in leptin (a satiety hormone), wasting and cardiac myopathy in HIV/AIDS.[17] Torres and Purgilese[18] suggested that high serum interleukin 18 (IL-18) in subjects infected with HIV with a high CD4 cell count was a useful marker of metabolic disorders and fat redistribution, as well as a sensitive predictor of cardiovascular complications in patients treated.

This present study showed that there was no correlation between alcohol use and cardiac dysfunction. However, men with a history of more significant alcohol intake were more afflicted with cardiac disease, though age could have been a confounder since they were also older. Alcohol use has been reported to have positive as well as detrimental effects on cardiovascular disease. Contrarily, a report from Framingham study[19] showed that alcohol use was positively associated with left ventricular (LV) mass. The lack of relationship between alcohol use and HRCD in this study is not surprising since patients with significant alcohol use were excluded.

This study showed that the degree of immunosuppression as shown by the CD4 cell count was the most common determinant of cardiac disease (table 5), with 64.9% of women with HRCD and 73.9% of men with HRCD having a CD4 cell count of less than 200 cells/μL (table 6). This point is corroborated by the work of Sitbon *et al*[20] while studying HIV related pulmonary hypertension in the HAART era. They noted that in a high proportion of patients a CD4 cell count less than 200 cells/μL was associated with pulmonary hypertension. Barbaro and Di Lorenzo,[21] in a cross-sectional study, reported that ECG parameters correlated significantly with values of CD4 T cells, suggesting that the state of immunodeficiency could enhance a possible cardiopathogenic action of HIV. DCM was seen in 4% of the study population, a low fraction compared to higher percentages seen in most reports from Africa,[12 22 23] ranging from 9% to 57%. Subjects with DCM had very low CD4 cell count of 153 cells/μL when compared with other forms of cardiac

disease a finding that affirms the assertion that cardiomyopathy is often seen in a state of severe immunosuppression, with low CD4 cell counts of less than 100 cells/μL being a marker of poor prognosis.[3 24] In a recent study of 20 775 individuals who were HIV positive and 215 158 who were HIV negative, HIV infection seemed only to be correlated with a higher risk of coronary heart disease when CD4 cell counts were less than 200 cells/μL, whereas patients infected with HIV with CD4 cell counts over 500 cells/μL had a similar risk to the general population.[24] In another study, over 81 000 individuals who were either HIV positive or HIV negative from the Virtual Cohort of the Veterans Aging Cohort Study were followed from 2003 to 2008. Results showed that, after accounting for other risk factors, HIV infection resulted in a twofold increased risk of clinically confirmed myocardial infarction (MI).[25]

Animal studies in simian monkeys with antigenic stimulation, using heat-killed mycobacterium to induce cardiac disease, noted significant correlation between CD4 T cells and evaluated ECG parameters.[26] It demonstrated the role of tumour necrosis factor α (TNFα) in DCM, showing that serum levels of TNF receptor 2 (TNFR2) and IL-18 correlated with left ventricular diastolic diameter. Their explanation was that the role of IL-18 may simply reflect connection between myocardial remodelling and overall systemic inflammatory response. However it has also been observed that lower levels of IL-18 at baseline, were associated with more severe decline in left ventricular ejection fraction (LVEF) at a later time. This suggests that the preinfection innate immune activation state may also play an important role in divergent postinfection myocardial effect. Inducible nitric oxide (INOS) has also been implicated in the impaired myocardial function through inflammation and HIV-1 envelope glycoprotein gp120, which is negatively ionotropic in vitro.[27]

The duration of HIV diagnosis showed some association with HRCD, patients who have had HIV infection for more than 48 months were more likely to develop HRCD compared to those who have been positive for less than 48 months (OR 3.88, 95% CI 1.20 to 13.28, p value 0.04). This observation has been likened to the effect of aging on the heart, with the HIV virus said to hasten the degeneration/aging of cardiac myocyte.[28] Its effect is related to length of time the patient has been infected, with the histological findings being similar to what is seen with chronic inflammation. There is reduced compliance, increased dimensions of the left ventricle and associated reduction of contractility, demonstrated by increased wall motion score, and ultimately reduced ejection fraction. In this study, this later effect of length of immunosuppression on cardiac dysfunction was seen on multiple regressions.

HAART did not have any significant association with cardiac disease in this study, similar to the opinion of Pugliese *et al*[29] that use of HAART does not seem to alter the long-term outcome in patients with established myocardial or pericardial disease. However, introduction of HAART before onset of cardiac disease has been reported to protect patients from developing these complications in the first place. Magula *et al*[5] noted from their review that access to HAART, degree of immunosuppression and opportunistic infections were determinant of cardiac disease. Several studies including the Strategies for Management of Antiretroviral Therapy (SMART) study and AIDS Clinical Trials Group (ACTG) 5142 suggest that using ART prevents cardiovascular disease associated outcomes (eg, myocardial infarction), at least in the short term.[30]

This study did not use a control population, which would have helped define the normal parameters in our population, the presence of primary pulmonary hypertension (PAH) could not be excluded for certainty among the patients, also we did not evaluate coronary artery disease and myocarditis important causes of cardiac dysfunction. Finally being a cross-sectional study, it is possible that the primary outcome were as a result of lifetime of exposure to risk factors rather than due to HIV/AIDS, a longitudinal study is therefore needed to measure all such factors.

CONCLUSIONS

Cardiotropism of HIV/AIDS has become an established aphorism, several comorbid factors fuel this association, but the degree of immunosuppression appears to stand out as the most obvious driver in the cardiac complications of HIV infection. It therefore necessary to sustain the awareness in early diagnosis of retroviral status of our population as well as access to HAART for those infected. These patients should also be availed of prompt cardiovascular evaluation so as to detect and intervene early if they are found to harbour markers of cardiovascular diseases.

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Contributors GI initiated the study and was directly involved in originating it; BO guided the study protocol and vetting of literature; SD undertook the echocardiography interpretation and verification; AO was involved in the statistics and organisation; MI did the proofreading and organising of the work for publication.

Competing interests None.

Ethics approval Ethics committee, Jos University Teaching Hospital.

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