

Cardiovascular Topics

Clinical and echocardiographic findings in a cross-sectional study of HIV-infected adults in Enugu, Nigeria

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Abstract

Background: Human immunodeficiency virus (HIV) infection and highly active antiretroviral therapy (HAART) are implicated in cardiovascular diseases. The objective of this study was to evaluate the clinical and echocardiographic findings in HIV-infected adults.

Methods: One hundred HIV subjects on HAART, 100 HAART-naïve patients and 100 controls were recruited in this cross-sectional study.

Results: Mean CD4 cell count was significantly higher in the HAART-exposed (408.43 ± 221.62) than the HAART-naïve groups (250.06 ± 154.26) ($p < 0.001$). Weight loss (49%), skin lesions (14%), body weakness (24%), oral thrush (10%) and lymphadenopathy (10%) were more prevalent in HAART-naïve patients ($p < 0.05$). Dimensions of aortic root (2.71 cm), left atrium (3.27 cm) and left ventricular mass index (79.95) were significantly higher in HIV-positive subjects on HAART ($p < 0.05$).

Conclusion: Clinical features of HIV and the CD4 nadir were more prevalent in the HIV-positive, HAART-naïve subjects. Dimensions of the aortic root, left atrium and left ventricle were relatively larger in the HAART-exposed patients while wall thickness and ejection fraction were higher in the HAART-naïve subjects.

Keywords: human immunodeficiency virus, antiretroviral therapy, CD4 cell, cardiovascular, echocardiography, dimension

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Despite the decreasing national prevalence of human immunodeficiency virus (HIV) infection in Nigeria (1.4%)¹ and some other developing countries, the challenge of managing the

burden of HIV infection still remains high due to the estimated 1.9 million¹ and 36.9 million² people in Nigeria and the world, respectively, living with HIV infection. These figures will likely increase with time, because of improved longevity arising from the availability of more potent antiretroviral and other antimicrobial agents used in treating affected individuals.^{3,4} This challenge is compounded by cardiovascular diseases, which occur in this group of people due to the effect of both the HI virus and the antiretroviral medications employed in its treatment.

The effects of HIV on the cardiovascular system are many and related to immunosuppression, with the occurrence of myocarditis, pericarditis, opportunistic infections and tumours.⁵ Antiretroviral therapy used in treating HIV has been identified to cause metabolic disorders. Highly active antiretroviral therapy (HAART), especially protease inhibitors (PI), have been found to induce disorders of lipid metabolism such as diabetes and dyslipidaemia, which have been implicated in the increased incidence of cardiovascular disease in this patient population.^{6,7}

A few echocardiographic studies have evaluated the effect of antiretroviral therapy on cardiac function in children.^{8,9} While some other similar studies in adults compared findings in HIV-positive patients as a group, with controls,¹⁰⁻¹⁶ studies evaluating findings in HIV-positive HAART-naïve, HIV-positive HAART-exposed and control subjects are few and therefore underscore the need for more studies in cohorts of HIV-positive subjects taking antiretroviral therapy. This study evaluated the clinical and echocardiographic findings in two groups of HIV-infected adults and non-infected controls in Enugu, south-east Nigeria.

Methods

We carried out a cross-sectional study between November 2010 and November 2011 at the University of Nigeria Teaching Hospital (UNTH), Enugu, Nigeria, and adhered to ethical standards of the Helsinki Declaration¹⁷ (1964, amended 2008) of the World Medical Association. Approval for the study was given by the ethics committee of the UNTH, Enugu, and written consent was given by the subjects. Information obtained was made anonymous.

The inclusion criteria were: adult Nigerians who were aged 18 years and above, in addition to confirmed HIV-positive serology. Enzyme-linked immunoassay (ELISA) was the method of HIV screening while confirmation was by Western blot electrophoresis. Flow cytometry was used to quantify CD4 T-lymphocytes.

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Fisher's formula was used to calculate the sample size:¹⁸

$$n = \frac{z^2 pq}{d^2},$$

where n = minimum sample size; z = 95% confidence level, i.e. 1.96; d = level of precision (0.075);¹⁹ p = maximum prevalence reported in a study of a similar population²⁰ (13.6%); and $q = 1-p$.

A sample size of 100 HIV-positive, HAART-naïve patients was recruited consecutively. One hundred age- and gender-matched HIV-positive patients on HAART for at least three months and 100 controls with HIV-negative serology were recruited for comparison. Antiretroviral therapy was commenced according to Nigerian guidelines for HIV and AIDS treatment and care.²¹ The controls were recruited from subjects being screened for marriage, blood donation and insurance purposes.

We excluded patients in end-stage AIDS disease, classified as category C by the Centre for Disease Control, 1993.²² Also excluded were those less than 18 years of age and subjects with a history or laboratory evidence of arterial hypertension, coronary artery or ischaemic heart disease, congestive heart failure, cardiomyopathy, peripheral or cerebrovascular disease and diabetes mellitus. In addition, pregnant women or those in puerperium, those with a significant history of tobacco and/or alcohol use, as well as those who used drugs known to affect the cardiovascular system were excluded.

Clinical evaluation was carried out on every subject. Their anthropometric parameters such as height (m) and weight (kg) were measured, while body mass index (BMI) (kg/m²) and body surface area (m²) were calculated.

A resting 12-lead surface electrocardiogram (ECG) was done on all recruited subjects in the supine position, at a speed of 25 mm/s, using a two-channel automated Techmel ECG machine (USA), ECG-1101 model. Analysis of the ECG tracings from each participant was done in the standard fashion, and long-lead II tracing was used as the rhythm strip. Parameters analysed were heart rate, rhythm, P wave (duration, shape), height (paroxysmal atrial complexes), PR interval, QRS wave (duration, shape, height, axis), paroxysmal ventricular complexes, QT interval, QTc, Q wave, T wave (shape), ST-segment (shape), and R and S waves for ventricular hypertrophy.

Resting two-dimensional echocardiography was carried out on all subjects using the SonoScape SS1-5000 machine and transducer of frequency 3.5 MHz. M-mode, two-dimensional, pulsed-wave, continuous-wave, tissue Doppler imaging and colour Doppler assessments were carried out on each subject in the left lateral decubitus position. Measurements were taken (in cm) using the American Society of Echocardiography guidelines (leading-edge methodology).²³

Table 1. Comparison of some demographic parameters across the three groups using one-way ANOVA

Parameters	HIV+ on HAART	HIV+ HAART-naïve	Controls	F-value	p-value
Age	35.85 ± 8.94	34.43 ± 9.49	35.76 ± 9.74	0.716	0.490
Weight	65.77 ± 13.92*	62.40 ± 12.45	68.69 ± 8.67*	7.007	0.001
BMI	24.14 ± 4.55*	22.47 ± 3.65	24.18 ± 3.32*	6.301	0.002
HR	82.92 ± 14.08*	84.28 ± 16.79*	68.77 ± 8.02	40.232	< 0.001

*Duncan's *post hoc* multiple comparisons test indicating means for groups in homogenous subsets (means not significantly different). BMI: body mass index; HR: heart rate.

Table 2. Demographic characteristics of the study participants

Parameters	HIV+ on HAART	HIV+ HAART-naïve	Control	F-value	p-value
Gender					
Male	51	48	52	0.347	0.841
Female	49	52	48		
Age group, years					
< 26	11	19	16	6.058	0.913
26–30	22	19	17		
31–35	21	24	25		
36–40	17	16	15		
41–45	13	7	8		
46–50	9	9	11		
> 50	7	6	8		

Statistical analysis

Data were analysed using EPI INFO version 6 software. Association between categorical variables was done using the chi-squared test. The Student's *t*-test was used to compare means of normally distributed continuous variables while the Mann-Whitney *U*-test was used to compare median of skewed data. The means ± standard deviations of parameters across the three groups were compared using one-way ANOVA, and the Duncan *post hoc* multiple comparisons test was done to indicate means for groups in homogenous subsets (means not significantly different). A *p*-value < 0.05 was taken as statistically significant.

Results

There was a significant difference in the mean weight, height, BMI and heart rate among the study groups ($p < 0.05$) (Table 1). The mean age for HIV-positive, HAART-naïve patients was 34.43 ± 9.49 years with a range of 18–59, while that for HIV-positive patients on HAART was 35.85 ± 8.94 years with a range of 23–59, and that for the controls was 35.76 ± 9.74 years with a range of 18–57 years. The highest number of subjects (70) was from the age bracket 31–35 years, while the least number (21) was from the age group above 50 years (Table 2, Fig. 1). There were 51 HIV-positive patients on HAART, 48 HIV-positive, HAART-naïve patients and 52 controls ($p > 0.841$).

The median time from diagnosis of HIV for HIV-positive, HAART-naïve patients was one year with minimum and maximum durations of one and 21 years, respectively, while that for HIV-positive patients on HAART was three years with minimum and maximum durations of one and 13 years,

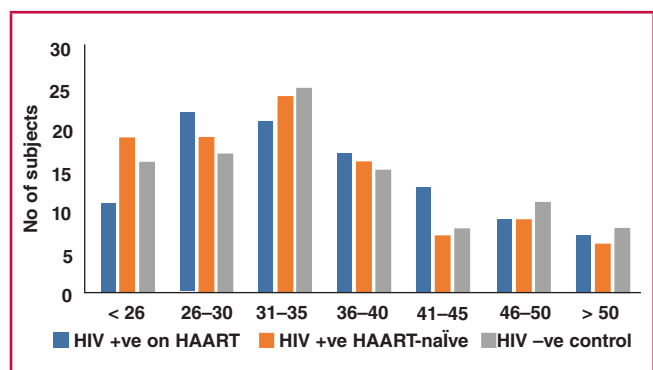


Fig. 1. Age distribution of the study groups.

Table 3. Proportions of HAART received by the treated group

First-line drugs	Second-line drugs
ZDV + 3TC + EFV OR If d4T or AZT used in first-line therapy	TDF + 3TC or FTC + ATV/r or LPVr OR AZT + 3TC + ATV/r or LPVr.
ZDV + 3TC + NVP	If TDF used in first-line therapy

ZDV, Zidovudine; 3TC, Lamivudine; EFV, Efavirenz; NVP, Nevirapine; TDF, Tenofovir; FTC, Emtricitabine; ATV/r, Atazanavir/ritonavir; LPV/r, Lopinavir/ritonavir; d4T, Stavudine; AZT, Zidovudine.

respectively. This was statistically significant ($U = 2\ 144$, $p < 0.001$).

The types of HAART received by the treated group were the non-nucleoside reverse-transcriptase inhibitors (NNRTIs), namely Nevirapine (NVP) 200 mg BD, Efavirenz (EFV) 600 mg OD; nucleoside reverse-transcriptase inhibitors (NRTIs), namely Zidovudine (ZDV) 250 mg BD, Lamivudine (3TC) 150 mg BD, Stavudine (d4T) 30 mg BD, Tenofovir (TDF) 300 mg OD; and the protease inhibitors, namely Ritonavir (RTV) 100 mg BD, Lopinavir-ritonavir (LPV/r) 400/100 mg BD. They were given in proportion according to the Nigerian guidelines²¹ (Table 3). Other medications received were mainly Co-trimoxazole 960 mg, Artemether/Lumefantrine 80 mg/480 mg, Clotrimazole cream, Fluconazole 200 mg, Diphenoxylate 5 mg, Metronidazole 400 mg, Mebendazole 400 mg, Fesolate 200 mg, paracetamol and multivitamins.

For the patients on HAART, the mean duration of HAART medication was 4.0 ± 2.4 years with a minimum and maximum duration of one and 10 years, respectively. Seven per cent of these patients were on a PI-containing HAART regimen while 93% were on a non-PI regimen. Those on a PI regimen received it for less than six months.

Between the HIV-positive patients on HAART and HIV-positive, HAART-naïve groups, there was a significant difference in the number of subjects with weight loss, skin lesions, body weakness, oral thrush and peripheral lymphadenopathy ($p < 0.05$) (Table 4). Forty-nine per cent of HIV-positive, HAART-naïve patients had weight loss compared to 12% in the group of HIV-positive patients on HAART. Similarly, 14% of HIV-positive, HAART-naïve patients had dermatological lesions, compared to only 2% of the group of HIV-positive patients on HAART. Twenty-four per cent of HIV-positive, HAART-naïve patients had generalised body weakness compared to 5% of

Table 4. Clinical features in the study population

Parameters	HIV+ on HAART (n)	HIV+ HAART-naïve (n) ²	χ^2	p-value
Weight loss	12	49	32.292	< 0.001
Skin lesion	2	14	9.783	0.002
Pruritus	1	0	1.005	0.316
Hepatomegally	1	0	1.005	0.316
Palpitation	5	8	0.740	0.390
Breathlessness	3	3	0.000	1.000
Weakness	5	35	14.559	< 0.001
Fever	4	8	1.418	0.234
Diarrhoea	3	7	1.684	0.194
Cough	2	6	2.083	0.149
Oral thrush	1	10	7.792	0.005
Peripheral lymph node enlargement	1	10	7.792	0.005

Table 5. ECG abnormalities in the study groups and controls

ECG abnormalities	HIV+ on HAART	HIV+ HAART-naïve	Controls	χ^2	p-value
LAD	15	10	8	2.656	0.265
T-wave inversion leads VI–VIII	44	22	14	24.682	< 0.001
Low-voltage complex	1	0	0	2.007	0.367
1st-degree heart block	3	1	2	1.020	0.600
T-wave inversion leads II, III aVF (inferior leads)	2	1	6	4.811	0.090
VEB	0	1	0	2.007	0.367
T-wave inversion leads I, avL, V5–V6	0	2	2	2.027	0.363
LBBB	1	0	0	2.007	0.367
RBBB	1	0	2	2.020	0.364
LVH	0	8	0	16.438	< 0.001
Tachycardia	0	1	2	2.020	0.364
ST-segment elevation	0	2	0	4.027	0.134
Bradycardia	0	0	12	25.000	< 0.001
Mean QTc, mean \pm SD	0.42 \pm 0.04	0.41 \pm 0.04	0.39 \pm 0.03		
Prolonged QTc, n (%)	17 (18.2)	12 (16.4)	4 (10.5)	8.784	0.012
Total, n (%)	93 (100)	73 (100)	38 (100)		

For QTc, $F = 15.779$; $p < 0.001$. Duncan's *post hoc* multiple comparisons test showed all significantly different.
LAD: left-axis deviation; VEB: ventricular ectopic beat; LBBB: left bundle branch block; RBBB: right bundle branch block; LVH: left ventricular hypertrophy.

patients in the HIV-positive group on HAART. Similarly, 10% of HIV-positive, HAART-naïve patients had oral thrush and peripheral lymphadenopathy, respectively, compared to 1% in the HIV-positive group on HAART.

The mean CD4 cell count for the HIV-positive patients on HAART was 408.43 ± 221.62 cells/mm³, while that of the HIV-positive, HAART-naïve group was 250.06 ± 154.26 cells/mm³. There was a significant difference between the means of the CD4 cell counts between HIV-positive patients on HAART and HIV-positive, HAART-naïve patients ($t = 5.865$, $p < 0.001$). There was also a significant difference in the number of subjects with CD4 cell counts < 200 cells/mm³ and those with CD4 cell counts \geq 200 cells/mm³ in both HIV-positive patients on HAART and HIV-positive, HAART-naïve groups ($\chi^2 = 16.095$, $p < 0.001$).

There were more ECG abnormalities in HIV patients on HAART compared to HAART-naïve patients and the controls (Table 5). T-wave inversion in leads VI–VIII occurred in 44% of the HIV-positive patients on HAART, 22% of HIV-positive, HAART-naïve patients and 8% of the controls ($\chi^2 = 24.682$, $p < 0.001$). Left ventricular hypertrophy (LVH) was found in only the HIV-positive, HAART-naïve patients ($p < 0.001$).

Comparing some echocardiographic parameters measured across the groups using one-way ANOVA, there was a significant difference in the mean aorta, left atrium, end-diastolic diameter, interventricular septum and ejection fraction, respectively, among the study groups ($p < 0.05$) (Table 6).

Discussion

The mean ages of the three groups were 34.43 ± 9.49 years (HIV-positive, HAART-naïve), 35.85 ± 8.94 years (HIV-positive patients on HAART) and 35.76 ± 9.74 years (controls). Similar findings have been documented in a recent study done in a

Table 6. Comparison of echocardiographic parameters measured across the groups using one-way ANOVA

Parameters	HIV+ on HAART	HIV+ HAART-naïve	Controls	F-value	p-value
AO (cm)	2.71 ± 0.40*	2.41 ± 0.37	2.74 ± 0.42*	21.363	< 0.001
LA (cm)	3.27 ± 0.62	2.68 ± 0.51	3.11 ± 0.47	31.385	< 0.001
EDD (cm)	4.73 ± 0.70*	4.41 ± 0.55	4.75 ± 0.42*	11.240	< 0.001
ESD (cm)	3.01 ± 0.51	2.84 ± 0.57	2.92 ± 0.43	2.616	0.075
IVS (cm)	0.77 ± 0.17*	0.85 ± 0.17	0.78 ± 0.15*	6.098	0.003
PW (cm)	0.82 ± 0.16	0.87 ± 0.17	0.82 ± 0.13	2.878	0.058
EF (%)	68.95 ± 12.43*	72.81 ± 11.70	67.36 ± 9.04*	6.223	0.002
FS (%)	36.77 ± 9.81	36.51 ± 8.64	37.77 ± 6.53	0.623	0.537
LVM (g)	141.94 ± 49.75	138.61 ± 48.53	131.26 ± 31.55	1.540	0.216
LVMI (g/m ²)	79.95 ± 26.25	77.55 ± 25.91	72.37 ± 16.52	2.760	0.065

*Duncan's *post hoc* multiple comparisons test indicating means for groups in homogenous subsets (means not significantly different).
 AO: aorta; LA: left atrium; EDD: end-diastolic diameter of left ventricle; ESD: end-systolic diameter of left ventricle; IVS: interventricular septum; PW: posterior wall of left ventricle; EF: ejection fraction; FS: fractional shortening; LVM: left ventricular mass; LVMI: left ventricular mass index.

similar population²⁴ and in other related studies.^{12,25} A higher number of HIV-infected patients (70) were within the age group of 31–35 years and this represents the global age distribution in which most of the people infected with HIV/AIDS are within the sexually active age bracket of 15–35 years.²⁶

Clinical features of HIV^{21,27} and immunosuppression were more prevalent in the HIV-positive, HAART-naïve group compared to the group of HIV-positive patients on HAART in this study. It has been documented in other studies that HAART is effective in increasing CD4 cell count and decreasing the viral load, with an associated decrease in morbidity and mortality among HIV-infected individuals.^{28,29} For the same reason, opportunistic infection was seen less often in the HIV-positive patients on HAART.^{10,30-32}

The mean weight and BMI were higher in the HIV-positive patients on HAART than in the HAART-naïve group. There was no significant difference in the mean weight and BMI between the HIV-positive patients on HAART and the controls (Table 1).

Weight loss is a feature of HIV infection, and weight gain in the HIV patients on HAART could have been due to a HAART-induced decrease in viral burden,^{33,34} as well as the metabolic side effects of HAART. In addition, the use of antiretrovirals, such as NRTIs and PIs, has been found to cause an increase in weight and mean BMI of subjects on HAART.^{35,36} They are also associated with the metabolic abnormalities of diabetes, dyslipidaemia, altered body fat distribution, especially HIV lipodystrophy syndrome, as well as mitochondrial abnormalities.³⁷ Weight and BMI of HIV patients on HAART in this study therefore increased and became comparable to that of the controls. A similar finding was documented in a related study between a cohort of HIV/AIDS patients and normal controls,^{12,16} but there was no separation, as in our study, of the HIV patients into those on HAART and HAART-naïve groups. In our study, antimicrobials and multivitamins received by the group on HAART helped to control opportunistic infections and diarrhoeal diseases, and improved appetite, weight gain and over-all well-being of the subjects.

The mean CD4 cell count in the HIV subjects was higher in the group on HAART (408.43 ± 221.62 cells/mm³) compared to the HAART-naïve group (250.06 ± 154.26 cells/mm³) (Fig. 2),

underscoring the benefits of HAART in this patient population.²⁸ The mean CD4 cell count for the HAART-naïve group was relatively high in this study because HIV patients with clinical features of end-stage AIDS, classified as category C by CDC 1993,²² were excluded.

Cardiac complications of HIV infection such as left ventricular dysfunction and dilated cardiomyopathy have been found to occur more often in patients with low CD4 counts.³⁸ In a study carried out in the pre-HAART era, global left ventricular hypokinesia was found to be associated with lower CD4 counts.¹¹ The dimensions of the aortic root, left atrium and left ventricle were within the normal range in the study groups. Although comparable to the controls, they were relatively higher in the HIV-positive patients on HAART than in the HIV-positive, HAART-naïve subjects (Table 6).

HAART has been found to induce myocardial toxicity and was believed to be the cause of right ventricular wall impairment and dilatation in a related study in children.³⁹ In a similar study by Ajala *et al.*,²⁴ in which the mean duration of HAART was not stated, no difference in chamber dimension was reported (Table 7). However, HIV disease has been found in some studies to be independently associated with increased left atrial volume in addition to increased left ventricular mass index,^{8,12} while other studies have shown no difference.¹³

Although the mean thickness of the interventricular septum was within normal limits in all the groups in our study, it was higher in the HIV-positive, HAART-naïve subjects but similar between the HIV-positive, HAART-exposed subjects and the controls (Table 6). Similarly, the posterior wall was higher in this group compared to the HAART-exposed or control groups. However, no difference in wall thickness was found in a recent study done in a similar population.²⁴ Disparate results have been reported, indicating possible HAART-induced increases in left ventricular wall thickness and mass in some studies,^{8,14}

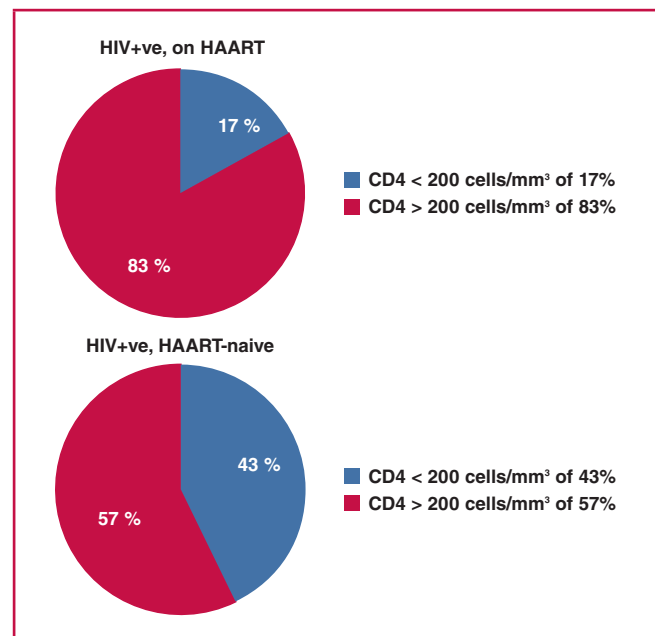


Fig. 2. Percentage of individuals and their mean CD4 counts in HIV-positive groups on HAART and HIV-positive, HAART-naïve groups.

Table 7. Comparison of main findings with other similar studies

Studies	ECG abnormalities	Echocardiography: wall thickness	Echocardiography: mean chamber dimensions and LVEF
Index study	ECG abnormalities were higher in HAART-exposed (93%), and HAART-naïve (73%), compared to controls (38%). LVH was found in 11% of HAART-naïve cases but none in the HAART-exposed and controls	Mean thickness of IV septum was higher in the HAART-naïve but similar between HAART-exposed and controls. LVM and LVMI were higher in cases than controls	Aortic root, LA and LV were slightly higher in HAART-exposed than HAART-naïve and controls. LVEF was higher in cases (HAART-naïve, 72.8%, and HAART-exposed, 68.9%) compared to the controls (67.3%)
Ajala <i>et al.</i> ²⁴	ECG abnormalities were higher in cases (49%) than controls (42%)	IV septum was slightly higher in controls than cases but LVM and LVMI were higher in cases than controls	LA was slightly higher in cases than controls. Aortic root was slightly higher in controls than cases. The mean LVEF was higher in the cases (71.9%) compared to controls (68.9%)
Ogunmodede <i>et al.</i> ¹²	ECG abnormalities were more in HIV-positive patients (55.3%) than controls (2.7%). LVH was higher in cases (17.3%), than controls (4%)	IV septum, LVM and LVMI were higher in cases than controls	LV was slightly higher in cases than controls. LA was slightly higher in controls. LVEF not stated
Uwanuruochi <i>et al.</i> ¹⁶	Not stated	IV septum and LVMI were higher in cases than controls. PW slightly higher in controls than cases	LV was slightly higher in controls. LVEF was higher in cases (60.3%) than controls (57.9%)
Danbauchi <i>et al.</i> ²⁰	Not stated	IV septum, PW, LVM and LVMI were higher in cases than controls	Aortic root, LA and LV were higher in cases. The LVEF was the same in both the cases and controls (66%)
Reinsch <i>et al.</i> ¹⁴	Not stated	IV septum and PW were higher in 18 and 11% of cases, respectively	LV was higher in 10.0% of cases. LVEF was 57.5%

LVH, left ventricular hypertrophy; IV, interventricular; LA, left atrium; LV, left ventricle; LVM, left ventricular mass; LVMI, left ventricular mass index; LVEF, left ventricular ejection fraction; PW, posterior wall.

while others showed increased thickness of the interventricular septum and posterior wall,⁴⁰ and eccentric patterns of LVH, with an increase in left ventricular cavity size in HIV-infected persons who were not on HAART^{41,42} (Table 7).

Possible causes of a thicker interventricular septum and posterior wall in the HAART-naïve group were higher HIV viraemia and its toxic effect on the myocardial cells, causing local release of cytokines and other factors, leading to subclinical inflammation and myocarditis.⁴³ This is corroborated by the postulation of immune dysfunction, as measured by the CD4 nadir, which is an independent risk factor for increased left ventricular mass, LVH and dysfunction.⁴⁴⁻⁴⁶ The HAART-naïve group consisted of more immunosuppressed subjects with a CD4 nadir and HIV viraemia, compared to the HAART-exposed subjects (Fig. 2). ECG findings in this study showed a relatively high prevalence of LVH in the HIV-positive, HAART-naïve patients, compared to it not being seen in subjects on HAART (Table 5), as assessed by the voltage criteria of Sokolow and Lyon, and Araoye.^{47,48}

Heart rate and left ventricular ejection fraction (LVEF) were higher in the HIV-positive, HAART-naïve subjects compared to the controls and HIV-positive, HAART-exposed subjects in this study (Tables 1, 6). This could have been due to increased prevalence of opportunistic infections, fever, anaemia, diarrhoea and dehydration, which would drive increased sympathetic activity and cardiac contractility in many of the individuals in this study group.¹⁶ Hyperdynamic left ventricular performance with enhanced contractility was reported in this subgroup of patients by Lipshultz *et al.*⁴⁹ In a recent study done in a similar population, there was no statistically significant difference in LVEF in the two HIV-positive groups but LVEF was slightly higher in subjects who were on PI-based HAART.²⁴ However, duration of PI use and clinical characteristics of the study population were not stated.

A limitation of the study is the relatively short duration of the use of PI and HAART, which have been found to cause diabetes mellitus and dyslipidaemia, which in turn have been identified to cause cardiovascular disease among HIV patients.

Conclusion

In this study, clinical features of HIV infection, immunosuppression and reduced CD4 cell count were more prevalent in HIV-positive patients who were HAART-naïve than in HIV-positive patients on HAART. The dimensions of the aortic root, left atrium and left ventricle were within normal limits but relatively larger in the HIV-positive, HAART-exposed group, while the wall thickness and LVEF were higher in the HIV-positive, HAART-naïve subjects. A longitudinal study would help identify possible links in the use of antiretroviral therapy.

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