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Human Immunodeficiency Virus, Cardiac abnormalities on Echocardiogram and the use of Highly Active Antiretroviral Therapy in Nigerian Patients

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ABSTRACT
Background: An increasing relationship between HIV/AIDS, highly active anti - retroviral therapy (HAART) and cardiovascular diseases (CVD) have been noted over time. The occurrence of acquired immunodeficiency syndrome (AIDS) - associated heart disease found in post - mortem studies is notably higher than those diagnosed clinically implying that many HIV/AIDS patients may have cardiac abnormalities that are not diagnosed during their lifetime. This misdiagnosis persists even in the presence of dire consequences such as overt heart failure or even death.
Aim: This study set to determine what cardiac abnormalities are present in Nigerian HIV positive patients and what differences exist in the manifestations of these cardiac abnormalities between HIV positive patients who have been on HAART and those who are non-treated with HAART.
Methods: This was a cross-sectional analytical study with a comparison group in which two groups consisting of 76 HIV positive treatment naïve and 76 HIV positive HAART treated patients who met the inclusion criteria were sampled. The study protocol was reviewed and approved by the Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City. All patients had an echocardiography done and data obtained was entered into and analyzed using the IBM-SPSS version 22.0. A p-value ≤ 0.05 was considered significant for all statistical comparisons done.
Results: Total prevalence of ECHO abnormalities was 91.4% in HIV Positive patients. Echocardiographic cardiac abnormalities were more prevalent in HAART treated patients [94.7%] than treatment naïve patients [ECHO = 88.2%]. The cardiac abnormalities found include increased LVMI, left ventricular diastolic dysfunction, increased left ventricular mass, pericardial effusion and abnormalities in left ventricular geometry. Pericardial effusion was more prevalent in treatment naïve patients with treatment naïve patients also noted to have the worst form of left ventricular geometry with over half having abnormal left ventricular geometric patterns compared to about 1/3rd in HAART treated patients.
Conclusion: Overall, HAART treated patients had cardiac abnormalities on echocardiogram than treatment naïve patients.

KEYWORDS: Echocardiography: Cardiac Abnormalities; HIV/AIDS; HAART Treated.

INTRODUCTION

The pathogenesis of human immunodeficiency virus (HIV) related cardiac disorders is multifactorial. It has been shown to result from the viral infection itself, opportunistic infections, nutritional deficiencies, autoimmunity or cardiac toxicity resulting from drugs and long-standing immune suppression.[1] Before the advent of highly active antiretroviral therapy (HAART), the cardiac manifestation noted in patients with HIV were: cardiomyopathy, pericarditis, pulmonary hypertension, heart failure, conduction system abnormalities and neoplastic infiltration.[2] Treatment of HIV - infected patients has resulted in increased survival and a longer lifespan. However this has resulted in the identification of the problems of the late stage of HIV infection, of which HIV - related cardiovascular diseases is an example.[3]

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It has also been observed that the occurrence of acquired immunodeficiency syndrome (AIDS) - associated heart disease found in post - mortem studies is notably higher than those diagnosed clinically.[4] This implies that many AIDS patients may have cardiac abnormalities that are not recognized during the course of their illness.[5] Early recognition of cardiac involvement in HIV/AIDS has become important for better prognosis.

Cardiovascular complications represent an increasingly important health concern in HIV infected patients;[6] and is also gaining recognition in the developing world. An increasing relationship between HIV/AIDS, highly active anti - retroviral therapy (HAART) and cardiovascular diseases(CVD) have been noted over time.[7] The prevalence of cardiac involvement in AIDS patients has been documented to range between 28% and 73%.[8] Cardiac abnormalities in HIV positive patients if left unidentified, could lead to overt cardiac failure and other cardiac complications with increased morbidity and mortality.[9]

Cardiovascular diseases in HIV/AIDS are becoming increasingly recognized, especially following the advent of HAART necessitating this study which aims to compare the cardiac abnormalities in HIV patients on HAART and treatment naive patient using echocardiographic investigative modalities with a bid to answer the two key research questions; (1) what cardiac abnormalities are present in Nigerian HIV positive patient and (2) what differences exist in the manifestations of these cardiac abnormalities between HIV positive patients who have been on HAART and those who are non-treated with HAART.

METHODS

This was a cross-sectional analytical study with a comparison group in which a total of one hundred and fifty-two consenting HIV positive patients were sampled. The study consisted of two groups of 76 HIV positive treatment naïve and 76 HIV positive HAART treated patients who met the inclusion criteria. The study protocol was reviewed and approved by the Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City, Edo State, Nigeria. Additionally, a written signed informed consent was obtained from every patient who participated in this study.

Patients included in this study were individuals who were HIV – positive patients confirmed by a polymerase chain reaction test, eighteen (18) years or older, and attended the adult President's Emergency Plan for AIDS Relief (PEPFAR) clinic of the University of Benin Teaching Hospital diagnosed for a minimum duration of one year without highly active antiretroviral therapy (HAART) treatment or patients on HAART for at least six completed (6) months.

Treatment with Highly active antiretroviral therapy was defined as a combination of at least three (3) classes of anti-retroviral medications, namely protease inhibitor (PIs), non - nucleoside reverse transcriptase inhibitors (NNRTIs) and nucleoside reverse transcriptase inhibitors (NRTIs).

The exclusion criteria for the study included patients less than 18years of age, patients who have documented congenital heart disease, patients with established cardiovascular diseases (e.g. hypertension, coronary artery disease), pregnant women and women on oral contraceptives plus patients with a history of diabetes mellitus, kidney disease or stroke.

Data from this study was obtained over a twelve (12) month period. Patients were sampled consecutively on arrival at the PEPFAR clinic and assigned to each group based on their HAART treatment status as stated in the inclusion criteria. All patients had their bio-data obtained and a physical/cardiovascular examination done. In addition, all patients had echocardiography done. Echocardiography was done using Philips HD7 XE with a 3.5 – 5MHz transducer. Each patient was placed in the left lateral position and various views (parasternal, apical, suprasternal and subcostal) were obtained. Measurements/recordings were performed with complete M-mode, 2 - dimensional, pulse wave, continuous - wave and colour doppler using standard guidelines of the Joint European Association of Echocardiography and American Society of Echocardiography.

Spectral Doppler Imaging was used in assessing flow across mitral valve, aortic valve, pulmonary valve and tricuspid valve. Mitral valve E/A velocity, Isovolumetric relaxation time (IVRT) and deceleration time were measured.

Data obtained was entered into and analyzed using the International Business Machines Statistical Product and Service Solutions (IBM-SPSS) version 22.0. Categorical data were expressed as frequencies and percentages while continuous data were presented as means (standard deviation). Where the data was skewed, continuous data was expressed as median (inter-quartile range) and compared using the Mann Whitney U test. Frequencies were compared using the Pearson's Chi-Square test; Fishers exact Chi square test was used to

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compare frequencies as applicable (specifically stated when used). Continuous data was compared between two groups using the independent t-test (e.g. for comparisons of left ventricular ejection fraction between treatment naive vs. HAART treated patients). A p-value ≤ 0.05 was considered significant for all statistical comparisons. Tables and charts were drawn using Microsoft Word and Excel 2017.

RESULTS

Most of the patients in the HAART treated group were diagnosed at a younger age but have been diagnosed for a longer period compared to treatment-naive HIV-positive patients in the study. Furthermore, there was a statistically significant difference noted between the age at diagnosis ($p < 0.001$) and duration since diagnosis ($p < 0.001$) in the treatment-naive vs. HAART treated HIV patients. Also, HAART treated patients had been on HAART for an average 6.24 (4.01) years.

The findings of this study show that an average 5.93years have exceeded since patients in the treatment naïve group patients were diagnosed. However, a longer duration since diagnosis was found in HAART treated patients (11.29years) but with a shorter treatment duration (6.24years) showing a time-lag between diagnosis and commencement of HAART of approximately 5years. The CD4 count of treatment naive HIV patients was 112.75 (106.75) cells which was lower than the 543.70 (985.58) cells recorded in HIV positive HAART treated patients. Also, the viral load of HIV positive HAART treated patients was lower than that of treatment naive patients. There was a statistically significant difference between the observed values of viral load ($p < 0.001$) and CD4 counts ($p < 0.001$) of both HIV positive patients' groups.

Assessment of patients' blood pressures showed that patients in the study were normotensive, with jugular venous pressures within normal limits and tachycardia noted in about 36% of patients studied (more prevalent in HAART treated patients). In addition, the slight numerical differences observed between both groups with respect to their blood pressure when compared found that the jugular venous pressure was not statistically significant but systolic and diastolic blood pressures were numerically higher in HAART treated patients and also statistically significant ($p<0.001$ for both comparisons).

Total prevalence of echocardiographic abnormalities was 91.4% in HIV Positive patients. Also, echocardiographic cardiac abnormalities were more prevalent in HAART treated patients [94.7%] than treatment naïve patients [ECHO = 88.2%].

There was statistically significant difference observed for the higher deceleration time ($p=0.001$) and TR ($p<0.001$) between both groups. In addition, LVMI showed a statistically significant difference between treatment naive and HAART treated patients ($p = 0.044$).

Grade 1 diastolic dysfunction was present in 10 patients in total (5 in each group) while Grade III diastolic dysfunction was present in 14.5% of HAART treated HIV-positive patients. These findings were found to be statistically significant when compared ($p = 0.003$). Over 10% of HAART treated patients had valvular thickening with 69.7% of treatment naive patients and over 40% of HAART treated patients having pericardial effusion. There was a statistically significant difference in the prevalence of valvular thickening ($p=0.028$) and pericardial effusion ($p=0.001$) between both groups with pericardial effusion showing significant odd ratio comparison.

The most common left ventricular geometric abnormality was eccentric hypertrophy present in over 1/3rd of all patients. Concentric hypertrophy was also more common than eccentric hypertrophy with these findings noted not to be statistically significant ($p = 0.162$).

DISCUSSION

Existing evidence shows that ninety seven percent of HIV patients are usually diagnosed between the ages of 22 – 24 years. [10] Late diagnosis of HIV could hold deleterious effects for both the patient and the general population as about 25% of newly diagnosed patients progress to AIDS within the first one year[10] contravening the natural history of HIV which follows a long course.

Earlier studies reviewed have shown that older age and male gender are contributory to late entry to care. [11] The prevalence of Late presentation has risen over the years despite the increased campaigns on awareness, prevention and the availability of voluntary counselling, testing and treatment facilities.[12] Late presentation can be elucidated from the results of this study showing that an average 5.93years have exceeded since patients in the treatment naïve group patients were diagnosed. However, a longer duration since diagnosis was found in HAART treated patients (11.29years) but with a lower treatment duration (6.24years) showing a time-lag between diagnosis and commencement of HAART of approximately 5years.

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Patients who commence treatment late have not benefited from patient education services available in treatment centres targeted at enhancing prevention strategies and combating the spread of the virus.[13] This education would have further reduced transmission risk to other individuals in the time lag before presentation in both groups. Furthermore, transmission risk to intimate partners would have been reduced if treatment using HAART had been commenced earlier.[13] This study showed that late presentation is a problem and the time-lag observed is alarming and could worsen transmission especially to intimate partners if no intervention plans are put in place.

The mean systolic and diastolic blood pressures were significantly higher in HAART treated compared to treatment naïve patients although earlier studies have suggested that an elevation in blood pressure is consequent upon receiving HAART therapy possibly from alterations in immune modulating mechanisms that assist in restoration of patients' immunity and concomitant reduction in viral load upon commencement of HAART.[14]

Diastolic dysfunction is also noted to be present in HIV patients in this study. Twenty-one patients had either grade I or III diastolic dysfunction. Diastolic dysfunction is known to be associated with antiretroviral use[15] and thus explains our study's finding of 14.5% of HAART treated patients with severe forms of diastolic dysfunction. Furthermore, the presence of an increased LVMI and hypertension are known to increase predisposition to diastolic dysfunction in HIV positive patients just as direct invasion of the myocardium by the HIV cells, can account for these findings[16] although these mechanisms have not been shown to be direct causative factors. Specifically, the use of NRTI has been found to be strongly associated with cardiomyopathy and mitochondrial damage.[17] However, it is still a debate as to whether HIV positive patients with diastolic dysfunction will require treatment or not.[18]

Previous research has also shown that the use of Lopinavir / Ritonavir significantly increases the incidence of new onset hypertension.[19] Apart from the effects of protease inhibitors, lipodystrophy[20] dyslipidaemia[21,22] and microalbuminuria[21] in addition to chronic inflammatory mechanisms[23] in HIV patients are suggested pathophysiologic pathways for the onset of hypertension secondary to HIV infection.

Our study findings of a significantly lower viral load and higher CD4 count in HAART treated patients' shows the effectiveness of HAART in combating the progression of HIV/AIDS although not without side effects as shown. Notwithstanding, without intervention, the deleterious effects will far outweigh the side effects of HAART as shown in the low CD4 and high viral load values in treatment naïve patients in this study.

Treatment naïve patients were noted to have the worst form of left ventricular geometry with over half having abnormal left ventricular geometric patterns compared to about 1/3rd in HAART treated group. This finding is similar to earlier research showing the existence of worse forms of LV geometry in HIV positive patients due to the pathogenesis and natural progression of the infection.[24,25] Other mechanisms for increased LVM may be mediated by subclinical atherosclerosis and an inverse relationship existing between CD4 count and LVM with the former pathway progressing via carotid artery intima-media thickening in HIV patients.[18] Notably, the increased ventricular dimensions in HIV patients are better assessed on echocardiography rather than electrocardiography as done in this study due to the better sensitivity of echocardiography.[26]

Pericardial effusion was more prevalent in treatment naïve patients who also had a higher viral load. The CD4 cell count did not correlate with cardiac risk factors significantly in this study while viral load correlated positively with pulse rate. Meanwhile, CD4 count did not show any association with sinus arrhythmias or pericardial effusion in both HIV positive patient groups in this study.

HIV is related to the presence of opportunistic infections;[27] with HIV positive patients of African descent noted to have a high prevalence of pericardial effusion.[28] Thus, the presence of pericardial effusion has been related to increased mortality due to a reduced CD4 count and superimposed opportunistic infections with consequently weaker immunity.[27,28] Hence, increased CD4 count in the presence of pericardial effusion holds the possibility of a better outcome in HIV positive patients as our study shows that pericardial effusion was more prevalent in treatment naïve patients who also had a higher viral load with lower CD4 count.

CONCLUSION

Diastolic dysfunction, increased LVM and LVMI and the presence of pericardial effusion were the most significant findings on echocardiography in HIV patients in this study. In addition, over 90% of HAART treated patients had cardiac abnormalities on echocardiogram more than that recorded in treatment naïve patients who also had a lower CD4 count and a higher viral load.

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Conflict of Interest: All authors declare that there are no Conflict of Interest.

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Table 1: Clinical Profile of Patients

	HIV Positive (Treatment Naïve) N=76 N (%)	**HIV Positive (HAART treated) N=76 N (%)**	**t**	**p-value**
Age at Diagnosis (Years)	41.64 (7.70)	36.47 (8.22)	-4.33	**<0.001**
Duration Since Diagnosis	5.93 (9.21)	11.29 (5.61)	4.00	**<0.001**
Treatment Duration	NA	6.24 (4.01)	-	-
CD4 Count(cells/m3)	112.75 (106.75)	543.70 (985.58)	-3.79	**<0.001**
Viral Load(copies/ml)	328736.25 (680649.53)	14468.31 (67429.21)	4.01	**<0.001**

NA = Not applicable

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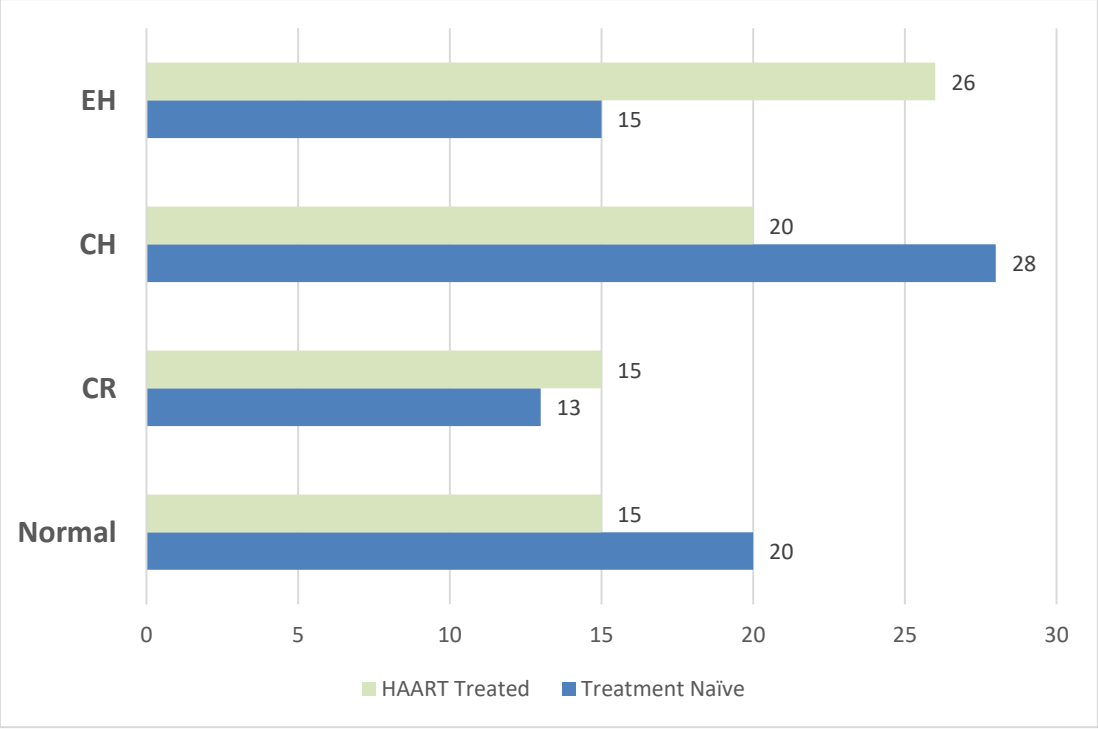
Table 2: Comparison of study's prevalence of Diastolic dysfunction, valvular thickening and pericardial effusion

		HIV Positive (Treatment Naïve) N=76 N (%)	HIV Positive (HAART treated) N=76 N (%)	χ^2	p-value
Diastolic Dysfunction	Normal	71 (93.4)	60 (80.0)	11.92	**0.003**
	Grade I	5 (6.6)	5 (6.5)		
	Grade III	0 (0.00)	11 (14.5)		
Valve Morphology	Normal	74 (97.4)	67 (88.2)	4.80	**0.028***
	Thickened	2 (2.6)	9 (11.8)		
Pericardial Effusion	Present	53 (69.7)	33 (43.4)	10.71	**0.001**
	Absent	23 (30.3)	43 (56.6)		

*Compared using Fishers exact Chi-square test.



CR *– concentric remodelling;* ***CH*** *– concentric hypertrophy;* ***EH*** *– Eccentric hypertrophy.*

Figure 1: Left Ventricular Geometry of Study Groups

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Table 3: Comparison of Echocardiographic characteristics between treatment naïve and HAART treated patients

	HIV Positive (Treatment Naïve) N=76 N (%)	HIV Positive (HAART treated) N=76 N (%)	t	p-value
Aortic Root	2.89 (0.33)	2.83 (0.45)	0.94	0.350
Left Atrial Size	3.24 (0.43)	3.37 (0.45)	-1.82	0.071
IVS Systole	1.52 (0.25)	1.44 (0.28)	- 1.86	0.065
IVS Diastole	1.21 (0.19)	1.15 (0.24)	1.71	0.090
LVID Systole	3.07 (0.48)	3.12 (0.59)	-0.57	0.567
LVID Diastole [median(IQR)]**	4.26 (0.68)	4.44 (0.87)	2553.50*	0.218
FS	32.96 (4.59)	33.82 (6.71)	- 0.92	0.358
LVPWD Systole	1.30 (0.27)	1.37 (0.27)	-1.60	0.112
LVPWD Diastole	0.98 (0.17)	1.00 (0.25)	-0.58	0.565
LVEF	61.57 (7.16)	62.16 (9.62)	-0.43	0.669
LVMI [median(IQR)]**	103.00 (21.00)	91.00 (34.00)	2342.50*	**0.044**
LVM [median(IQR)]**	168.00 (47.00)	101.00 (68.00)	2768.50*	0.660
RWT	0.44 (0.97)	0.44 (0.14)	0.00	1.000
Stroke Volume	54.12 (13.21)	62.35 (38.24)	-1.77	0.078
Mitral E/A [median(IQR)]**	1.30 (0.49)	1.40 (0.47)	244.50*	0.482
Deceleration Time	184.66 (18.69)	154.23 (74.89)	3.44	**0.001**
IVRT [median(IQR)]**	74.00 (14.00)	67.00 (27.00)	2521.00*	0.919
TR	1.51 (0.50)	2.02 (0.77)	- 4.84	**<0.001**

*U values in asterisk; **Median (Interquartile range) values compared using Mann-Whitney U test.

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