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HIV and the Heart, an Echocardiographic Study.

Cookey SN.
Consultant Cardiologist, Department of Internal Medicine,
Rivers State University Teaching Hospital.
No 8-12 Harley Street, old GRA, Port Harcourt

ABSTRACT
BACKGROUND
Various screening modalities have been used to assess cardiac involvement in patients infected with HIV.There is varying prevalence depending on the screening modalitiy used. Echocardiography is a valuable tool used in assessing cardiac diseases and has been used in a large number of studies to evaluate cardiac abnormalities in HIV patients. This study is aimed at assessing the incidence of cardiac abnormalities among HIV positive individuals using echocardiography.
METHODS: *Study cases were randomly selected amongst adult patients aged 18 years and above, who fufilled the inclusion criteria, presenting at the University of Port Harcourt Teaching Hospital with a diagnosis of HIV disease. Controls were subjects from the hospital population who had no history of cardiac disease, non hypertensives, non diabetics and tested negative for the human immunodeficiency virus. The controls were carefully matched with the cases for sex and age. The study period was from July 2011 to July 2014.*
Ethical clearance was obtained from the the Ethical Committee, University of Port Harcourt Teaching Hospital. Subjects were evaluated for cardiac abnormalities using the Aloka 4000ssd ultrasound machine and appropriate cardiac probe after clinical examination and laboratory investigations, including packed cell voume, and CD4 count. Data was analysed using the SPSS 11 statitical software package.
RESULTS:*The study subjects were 200 HIV positive patients:76 (38%) males and 124 (62%) females with a male to female ratio of 1:1.6. They were aged between 18 yrs and 56 years, with a mean age ± SD of 33.13 ±8.4years.*
The controls were made up of 100 individuals who met the inclusion criteria: 64 females (64%) and 36males(36%) with age range between 19 and 54 years with a mean ± SD age of 31.82 ±8.72years and male to female ratio of 1: 1.7. The mean ages for the female cases and controls were similar with no statistical difference; as were the mean ages of the male cases and the male controls.
The mean blood pressure also compared favourably. However, there were significant differences in the packed cell volume (PCV), pulse rate and the body mass index (BMI) of controls, as compared to HIV patients.
The echocardiographic abnormalities were in order of frequency pericardial effusion, left ventricular (LV) diastolic dysfunction, depressed LV ejection fraction, depressed LV and Right Ventricular (RV) ejection fraction, and regional wall motion abnormality.
CONCLUSION
Cardiac abnormalities are more common in HIV infected individuals when compared to normal individuals. This prospective descriptive, cross sectional study has shown a high prevalence of cardiac abnormality; 63% as shown by echocardiography. Strikingly most of the subjects studied had no cardiac symptoms but already had on going cardiac pathologies, suggesting the need for managing physicians to be on the lookout, to allow for prompt diagnosis and management.

KEYWORDS: HIV, HEART, ECHOCARDIOGRAPHY.

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I. INTRODUCTION

Human immuno deficiency virus (HIV) belongs to the family of human retroviruses (Retroviridae) and the subfamily lentiviruses. There are two main types of human immunodeficiency viruses; HIV-1 and HIV-2. The most common cause of HIV disease throughout the world is HIV-1**[1]**, which comprises several subtypes with different geographic distributions. HIV-2 was first identified in 1986 in West Africa[1].

There has been a significant reduction in incidence of HIV and AIDs globally since 1997; 50% was the estimate put forward United Nations programme on HIV/AIDS. (UNAIDS**)**[2] in 2021. There is also a greater access to antiretroviral medications. This implies an increase in non-opportunistic complication of HIV, and

increase in complications arising from highly active antiretroviral therapy (HAART). Cardiac involvement in AIDS was first reported in 1983 in a post-mortem study of a 24-year-old female with Kaposi sarcoma.[3] The effect of HIV on the heart is dependent on the stage of the disease, the opportunistic co-infections and also on the drug therapy. [1,4]

HIV is well known to penetrate cells that bear CD4 receptors, notably the cardiac insterstitial cells and cardiac myocytes because they do not bear CD4 receptors were thought to evade invasion by the HIV. However studies[5,6] have isolated viral sequences in cardiac myocytes obtained from endocardial biopsies. The effect on the heart is belived to be both direct and indirect by stimulating the release of cytokines that are cardiotoxic and by opportunistic co-infections [7].

Cardiovascular disease in HIV Patients has also been associated with antiretroviral medications. Soon after the introduction of protease inhibitors and nonnucleoside reverse-transcriptase inhibitors for the management of human immunodeficiency virus (HIV) infection, clinicians observed unexpected cardiovascular events among patients receiving these new, combination, "highly active" antiretroviral regimens. Angina, myocardial infarction, and stroke were seen in patients who were relatively young [8].

In the african study by Danbauchi et al [9] the patients on antiretroviral were said to gain weight and tended to obesity.

Cardiac abnormalities in HIV are important non opportunistic complication of HIV infection however it can also be as a result of opportunistic infections.

The effect of HAART on HIV cardiac abnormalities is still being evaluated though the effect of opportunistic co infection on the heart are believed to be amelorated by HAART but the direct effect of the virus continues despite therapy with antiretrovirals. The effect of HAART [10] is however reversible once therapy is stopped.

Echocardiography remains a very valuable diagnostic tool in cardiology is because it allows real time visualization of cardiac anatomy and echocardiographic studies have shown frequent involvement of the heart in advanced stages of HIV infection. The range of cardiac abnormalities include: pericardial effusion, dilated cardiomyopathy (frequently with myocarditis), infective endocarditis, and malignancy (myocardial Kaposi's sarcoma and B-cell immunoblastic lymphoma).[6] There has however been a discrepancy on the frequency of the cardiac abnormalities. Largely, pericardial effusion has been reported to be the most common cardiac abnormality seen by echocardiography in African studies and other studies. This has been corroborated by some authors. [10, 11]

The clinical expression of cardiac involvement is variable and is affected by the stage of the HIV infection, antiretroviral therapy and the use of drugs to prevent opportunistic infections and neoplasms. As a primary consequence of HIV infection, the most clinically significant finding is a dilated cardiomyopathy,usually associated with congestive cardiac failure referred to as *HIV cardiomyopathy is the most studied* the cardiac complications of HIV.{12-19}

The annual incidence of DCM was 15.9 cases per 1000 patients from a study [20], that evaluated asymyomatic patients, followed up for a period of 5 years, other studies revealed 5.1% of patients having DCM in their study population.

Many factors have been implicated as the cause of dilated cardiomyopathy in HIV positive individuals. The myocytes do not have CD4 receptors and are therefore not believed to be penetrated by the HIV. On the other hand the dendritic cells in the insterstium of the heart bear CD4 receptors and believed to be the channel through which the HIV penetrates the heart.

However, the large-scale prospective study published by Barbaro et al [11], a study which recruited 952 HIV- infected patients, followed up by echocardiography to determine the incidence of DCM, all patients with the diagnosis of DCM by echocardiogarphy underwent endomyocardial biopsy for histopathological, virological and immuno-histological study. A histopathological diagnosis of myocarditis was established in 83% of the patients with DCM. Hybridization *in situ* detected the HIV nucleic acid sequence in the myocytes of 58 patients, and 36 of these had active myocarditis. The authors concluded that DCM can be caused by the direct action of HIV on the myocardium or by an autoimmune process, possibly associated with other cardiotropic viruses,this was corroborated by the study of Rodriguez et al.[21]

Pathologic features of HIV-associated cardiomyopathy are similar to those observed in sero-negative patients with dilated cardiomyopathy. [22-24]There is ventricular dilation and apical rounding. There is fibrosis and myocyte hypertrophy which results in increase in the heart weight. On cut surface, there is eccentric hypertrophy of the ventricle, that is, a mass increase with chamber volume enlargement histologically showing an increase in interstitial and endocardial fibrillar collagen is a constant feature in this cardiomyopathy.

Myocarditis and viral myocardial infection: Myocardial infection with HIV has been described by Babaro et al[24] as the best-studied causes of dilated cardiomyopathy in HIV disease. HIV-1 virons appear to infect myocardial cells in a patchy distribution with no direct association between the presence of the virus and myocyte dysfunction.

It has been postulated that dendritic reservoir cells may play a role by activating multifunctional cytokines that contribute to progressive and late tissue damage, such as tumour necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), interleukin-6 (IL-6), and interleukin-10 (IL-10) Co infection with other viruses (usually coxsackievirus B3 and cytomegalovirus) may also play an important pathogenetic role.

Autoimmunity as a factor is said to be contributory in the cardiomyopathy found in this group of individuals. This has been further supported by the presence of cardiac-specific autoantibodies (anti-alpha-myosin autoantibodies) **[25]** supporting the theory that cardiac autoimmunity plays a role in the pathogenesis of HIV related heart disease and suggesting and also the possibility of using these antibodies as markers of left ventricular dysfunction in HIV-positive patients with previously normal echocardiographic findings.

In addition, it's been found that monthly intravenous immunoglobulin in HIV infected paediatric patients minimizes left ventricular dysfunction [25], increases left ventricular wall thickness, and reduces peak left ventricular wall stress, suggesting that both impaired myocardial growth and left ventricular dysfunction may be immunologically mediated.

Myocardial cytokine expression: **[26-28]** Cytokines play a role in development of HIV-related cardiomyopathy. It has also been postulated that these cytokines may be produced by opportunistic co-infection with the HIV virus such as: Coxsackie A and B, Toxoplasma Gondi, Epstein Barr virus and Adenovirus. These alongside the HIV cause a viral myocarditis

Nutritional deficiencies: **[29-31]** In HIV infection Nutritional deficiencies are quite common, resulting from malabsorption and diaorrhea, causing loss of trace elements for example, Selenium and vit B_{12} deficiency. These trace elements have been directly or indirectly associated with cardiomyopathy. Nutitional deficiencies usually occur late in the disease.

Drug cardiotoxicity; Studies of transgenic mice suggest that zidovudine **[7]**is associated with diffuse destruction of cardiac mitochondrial ultrastructure and inhibition of mitochondrial DNA replication. This mitochondrial dysfunction may result in lactic acidosis, which could also contribute to myocardial cell dysfunction.

Cardiac abnormalities:

Depressed Ejection Fraction:

Depressed ejection fraction without frank dilatation of the cardiac chambers has been described by some authors **[13,14]**. It is not yet certain if this is a prelude to frank DCM. Danbauchi et al also noted depressed ejection fraction in their study from Zaria, Nigeria **[9]**. Another study by a team in Athens Apostolos et al [32] which employed Tissue doppler echocardiography of the Mitral annulus. The systolic Sm Doppler wave was measured and compared between normal subjects and HIV positive patients the Doppler tissue imaging in that study was significantly lower in that group.

Diastolic Dysfunction: Clinical and echocardiographic findings suggest that diastolic dysfunction is relatively common in long-term survivors of HIV infection. Left ventricular diastolic dysfunction preceded systolic dysfunction in some cases. One large multicentre echocardiographic study found that asymptomatic HIV-infected patients had 34.6 percent lower E/A ratio (Doppler-derived parameter of diastolic dysfunction: peak early [E] over peak atrial [A] velocity) and 19.7 percent longer Isovolumetric relaxation time than healthy adults.**[30]**

This has also been corroborated by other studies.

Pericardial Effusion: this is the most frequent finding in most Nigerian study. HIV/AIDS most of which are said to be assymptomatic and the prevalence prior to HAART estimated at 11%**[1,9,33.34]**

In the 5-year Prospective Evaluation of Cardiac Involvement in AIDS (PRECIA) study , 16 of 231**1** patients developed pericardial effusions. The incidence of pericardial effusion among those with AIDS was 11 percent per year. The prevalence of effusion in AIDS patients rose over time, reaching a mean in asymptomatic patients of about 22 percent after 25 months of follow-up. Pericardial effusion **[35-37],**in HIV disease is related to opportunistic infections or malignancy, but most often a clear aetiology is not found. Predisposing factors include TB, congestive heart failure, mycobacterium infection, cryptococcal infection, pulmonary infection, lymphoma, and Kaposi Sarcoma, staphylococcus and salmonella specie.

Endocarditis:

The prevalence of infective endocarditis in HIV-infected patients is said to be similar to that of patients in other risk groups, such as intravenous drug addicts ranging from 6.3 to 34%, independent of antiretroviral therapy.**[1]**

Infective endocarditis may be due to either pyogenic or opportunistic pathogens. Infective endocarditis occurs more frequently in HIV-infected intravenous drug addicts. These patients have frequent bacteraemia, higher in intravenous drug addicts who abuse multiple drugs in addition to alcohol.

Coronary Artery Disease: Echocardiography especially stress echo has been used to evaluate coronary artery disease**3**. Regional wall motion abnormalities can be assessed using the segmental approach described by the American Society of Echocardiography **[3]**most of the studies on coronary artery disease in patients with AIDS

have implicated the protease inhibitor.
In a large-scale observational study reported in the lancet which recruited 33000 HIV positive individuals two important specific medications has been implicated and said to cause heart attack. They were Glaxo Smith Kline Ziage(abacavir) and Bristol Myers-\Squibb Vidox (didanosine)[38]reported high prevalence of asymptomatic ischaemic heart disease a study that evaluated cardiac diseases as seen by electrocardiography in 10% of the patients enrolled for the smart study. The study enrolled a total of 5472 patients from 300 HIV treatment centres in 33 countries. However, most of these patients were on antiretroviral therapy some were diabetic and hypertensive.
However, in some studies HIV has been shown to accelerate artherosclerosis. [5]
Endothelial dysfunction and injury have been described in HIV infection [5]. This is evident by circulating markers of endothelial activation, such as soluble adhesion molecules and procoagulant proteins that are elaborated in HIV infection. The means by which HIV penetrates endothelium may be via CD4 or galactosyl-ceramide receptors. Other possible mechanisms of entry include chemokine receptors. Coronary endothelium strongly expresses CXCR4, CCR3and CCCR2A co-receptors.
Opportunistic agents, such as cytomegalovirus, frequently co infect HIV-infected patients and may contribute to the development of endothelial damage. Moreover, a retrospective analysis of post-mortem reports revealed a strong correlation between Kaposi's sarcoma, the most frequent AIDS-related neoplasm, and the presence of atheroma. On the basis of this observation and previous experimental data, the authors hypothesize that human herpes virus-8 HHV-8 (a virus that is found in all forms of Kaposi's sarcoma) may trigger or accelerate the development of atheroma in the presence of hyperlipideamia [39].

Malignancy: Common malignancies affecting the heart in HIV patients are Kaposi sarcoma and malignant lymphoma. Retrospective autopsy studies in the pre HAART period estimated the prevalence of cardiac Kaposi in AIDS to be from 12-28%[40,41]. The lesions are typically less than 1 cm in size and may be pericardial or, less frequently, myocardial, and are only rarely associated with obstruction, dysfunction, morbidity, or mortality. Malignant lymphoma involving the heart in AIDS is infrequent, mostly limited to case reports before the commencement of HAART .
Other cardiac tumors are leiomyosarcomas; these are rare and largely non cardiac and often affecting the smooth muscles of the arterial wall and is associated with Epstein Barr virus.
Danbauchi et al recorded an intra-pericardial tumour, though there was no histological confirmation.[9]
Isolated Right Heart Disease: Isolated right ventricular hypertrophy[42] with or without right ventricular dilation is rare in HIV-infected individuals and is generally related to pulmonary disease that increases pulmonary vascular resistance. Possible causes include multiple bronchopulmonary infections, pulmonary arteritis from the immunological effects of HIV disease, or micro vascular pulmonary emboli caused by thrombus or contaminants in injected drugs.
Cardiovascular Alterations In Chronic Anaemias; Chronic anaemia has a series of well described effects on the heart viz; cardiac dilation and hypertrophy are known to be associated with chronic anaemia. HIV is usually associated with chronic anaemia. The reduced oxygen carrying capacity due to anaemia increases demand on the heart with an increase in cardiac output, this increase is achieved in chronic anaemia by increased in stroke volume, with resultant hyper dynamic circulation, heart murmur and cardiomegaly. This is also the issue in other disease conditions associated with chronic anaemia like sickle cell. [43,44]

II. METHOD

This was a prospective, descriptive, cross-sectional study.

Study Site:

This study was a hospital based study conducted at the University of Port Harcourt Teaching Hospital.

Ethical Consideration:

Clearance for this study was obtained from the Ethical Committee of the University of Port Harcourt Teaching Hospital. At all stages the researcher adhered to the guidelines of the ethical committee and standard research protocol. All case and control subjects gave informed consent.

Study Population:

Patients: The study sample was made up of 200 HIV positive patients, who were antiretroviral naïve,randomly selected without fore knowledge of their CD4+ count. The random numbers were generated using the table of random numbers.

Inclusion Criteria For Patients

Newly diagnosed HIV positive, antiretroviral naïve individuals, irrespective of
CD4 count and who have consented to be a part of the study.

Exclusion Criteria For Patients
1. Hypertensives.
2. Diabetics.
3. Patients with no significant history of alcohol ingestion. (Patients that consume less than 30g/day)
4. History of cigarette smoking.
5. Poor Echocardiography window.

Controls:
Normal values for echocardiographic variables were generated by analysing echocardiograms of 100 healthy adults with age range 18-80yrs of age, with no history of cardiovascular disease or any other medical condition. They were of comparable ages and sex with the HIV positive patients.
Clinical Evaluation:
Baseline demographics,clinical history and detailed physical examination of all subjects were carried out including their age, ,gender, height, weight and baseline blood pressure.Packed cell volume and fasting blood sugar was assessed.
Hiv Confirmation: Double Elisa using a rapid screening kit was used to confirm a diagnosis of HIV infection. This is the method used in University of Port Harcourt Teaching Hospital. This was also used in the Enugu study by Ikechebelu et al. The World Health Organization (WHO) endorses alternative algorithm for use in recourses-limited setting where a double Elisa confirms HIV positivity [45].

Cd4 Count:CD4 count was assayed using the Apogee A50 micro flow cytometer.

Echocardiography
All subjects had Echocardiography done, using the Aloka prosonic SSD 4000 after due explanation to the patients and controls alike. Subjects were asked to lie in a steep lateral decubitus position with the patients left arm extending over their heads.
Standard M-Mode, 2D, Doppler(pulsed, continious and colour wave) was performed on all subjects.The area of the chambers was used as opposed to the internal diameter following the guidelines of the American Society of Echocardiography.[3]
A predefined imaging protocol was used. For each variable, two representative beats were analysed and the mean results calculated. Echocardiography was carried out before checking the result for the CD4 count to eliminate bias. Supervisor cross checked random samples of echocardiography findings, for quality control. The bills of the investigations were not borne by the patients.A waiver and project grant was obtained from the authorities of the University of Port Harcourt Teaching Hospital.

Echocardiography protocol.
1. Left ventricular end-diastolic area (LVEDA) was measured in the apical four-chamber view, by tracing the endocardial edges of the left ventricle and the plane of the mitral valve at end-diastole. The calculated area was be divided by height to account for differences in body size.

2. Left Ventricular Function was estimated by the left ventricular percent change in area. Areas of the left ventricle at end-systole (LVESA) and end-diastole was determined and the percent change in area calculated as: 100 × (LVEDA - LVESA)/LVEDA, following the American Society of Echocardiography guidelines.

3. Right ventricular end-diastolic area (RVEDA) was measured in the apical four-chamber view by tracing the endocardial edges of the right ventricle and the plane of the tricuspid valve at end-diastole. The calculated area was divided by height to account for differences in body size.

4. Right ventricular systolic function was estimated by the right ventricular percent change in area. Areas of the right ventricle at end-systole (RVESA) and end-diastole were determined using the method described above, and the percent change in area calculated as:100 × (RVEDA - RVESA)/RVEDA.
5. The ventricular diastolic inflow were measured by positioning the sample volume at the tip of the mitral /tricuspid leaflets, with measurement of the E and A wave (in sinus rhythm) Velocities, and their ratios were calculated. The deceleration time of the E wave was also measured.
6. Right atrial area was measured by planimetry in the apical four-chamber view at end-systole, corrected for height.
7. Left atrial area was measured by planimetry in the apical four-chamber view at end-systole, corrected for height.

8. Pericardial effusion, defined as a distinct diastolic separation of the pericardial layers posterior to the heart on the parasternal long-axis and short-axis views.
9. Maximal instantaneous velocity of the tricuspid regurgitation and pulmonary regurgitation signal were measured using continuous wave Doppler.
10. Using continuous wave Doppler echocardiography, cardiac output was calculated as the product of aortic/pulmonary stroke volume time's heart rate.
13. Regional wall motion abnormality was noted.

Definition Of Cardiac Abnormalities:
1. Depressed LV ejection fraction was defined by the guidelines of the American college of cardiology as an ejection fraction ‹55%.[22]
2. Depressed RV ejection fraction was defined by the publication of Pfisterer et al as an ejection fraction ‹40%.[46]
2. Diastolic dysfunction was defined by LV E/A ratio of 1-1.8' for those in sinus rhythm and E- wave deceleration time, with normal DT: 60 – 240ms. While RV E/A was defined by the normal generated from this study with a mean of 1.27± 0.4.
3. Dilated cardiomyopathy was defined as depressed ejection fraction below 40% with accompanying cardiac chamber and global hypokinesia [47].
4. Heart failure was defined using the Framingham's criteria [47] described below.
5. LV depressed Ejection fraction; depressed LV ejection fraction with normal RV ejection fraction but normal LV chamber dimension.
6. RV depressed ejection fraction: depressed RV ejection fraction with normal LV ejection fraction but normal RV chamber dimension.
7. Depressed LV and RV ejection fraction: both LV and RV depressed ejection fraction but both chambers are normal in dimension.
8. Pericardial effusion, defined as a distinct diastolic separation of the pericardial layers posterior to the heart on the parasternal long-axis and short-axis views.it was further classified into mild moderate and severe where mild is less than 1cm in diameter, moderate 1-2 cm and severe is above 2cm [47]
9. Normal cardiac output [4] : 4 – 8l/min [2] [47]
10. Abnormal regional wall motion abnormality was described using the 16- segment model approach as described by the echocardiography guidelines of the American College of Cardiology and American Heart Association[22]**.** The wall segments are identified according to internal anatomical landmarks of the left ventricle in the standard short axis at the mitral, papillary and apical levels. These 3 levels are further subdivided to produce 16 segments. The basal level is at the level of the mitral annulus, this has six segment the anterior, antero-septal, inferio-septal, inferior, inferio-lateral and antero-lateral. The mid is at the level of tips of the papillary muscles this also has six segments similar to that of the basal level. The apical is at the apex of the ventricle and is divided into four equal segments namely: anterior, septal, inferior and lateral.
11. LVM/BSA was estimated using the Penn cube method:
$LVM = 1.04 (LVID + IVST + PWT)^3 - LVID^3 - 136$
Where 1.04 = specific gravity of the myocardium(g/ml)
LVID = Left ventricular internal dimension (in diastole)
PWT = Posterior wall thickness(cm) (in diastole)
IVST = Interventricular Septal thickness (in diastole)
which was later indexed for BSA.
Sample Size Estimation: The minimum number of patient required for this study was calculated from the method of kish:[48]

$$NF = \frac{n}{1 + (n)/N} \quad \text{And} \quad n = pgz^2/d^2$$

$$n = \frac{0.5 * 0.5 * 1.96^2}{0.05^2} = 384$$

$$NF = \frac{384}{1 + (384)/380} = \frac{384}{2.01} = 190$$

NF= 190

NF= final sample size

n= the desired sample size
z= the standard normal deviation usually set at 1.96 which corresponds to the 95% confidence level .
p= proportion of likely patient with cardiovascular disease estimated at 50%
q= 1.0 - p =50%
d= degree of accuracy desired 0.05
N = estimation of population size i.e new patients with HIV disease managed in UPTH port Harcourt annually = 500.

Data Analysis

Aanalysis was performed using the SPSS 11 software package.Continuous variables were expressed as means (standard deviation) while categorical variables were expressed as percentages. Differences of the means between two groups were compared with students ' t' test. Proportions or the categorical parameters was be analysed with the chi-square. A p -value of < 0.05 was considered statistically significant. Correlation was carried out between the CD4 count and echocardiography parameters, using Pearson correlation coefficient.

III. Results

A total of 200 HIV positive, antiretroviral naive patients and 100 HIV negative controls who met the inclusion criteria for this study were recruited**.**

Base Line Demographic Parameters

The study subjects were made of 76(38%) males and 124(62%) femaleswith a male to female ratio of 1:1.6. The age range was 18 - 56 years, with a mean of 33.13 ± 8.4years. The controls were made up of 64 females (64%) and 36(36%) males with age range between 19 to 54 years and a mean of 31.82 ±8.72years.There was no statistically significant difference between the ages of the cases as compared to the controls.(See table 1 on page 44)

The mean ages for the female cases and controls; 30.95 ± 8.04 ,31.25 .± 10.26 years respestively were similar; as were the mean ages of the male cases and the male controls; 32.13.± 9.9 and 32 .± 9.1 years repectively.The second and third decades made up the largest no of subjects in both control and HIV cases.

TABLE 1

Comparison of demographic data of cases and controls

CHARACTERISTICS	**CASES**	**CONTROLS**	**P-Value**
Gender	**N=200**	N=100	X^2 =0.00 Df = 1 P = 1.00
Male	76	36	
Female	124	64	
Mean Age ± SD	33.13 ± 8.4	32.83 ± 8.72	**T = 0.11** **P = 0.91**
Age Range	18-53	19-50	

Significant P = ≤ 0.05

TABLE 4

AGE DISTRIBUTION OF CASES /CONTROLS

AGE	**MALES**		**FEMALES**	
	HIV POPULATION N(%)	**CONTROLS N(%)**	**HIV POPULATION N(%)**	**CONTROLS N(%)**
<20yrs			**3(1.5)**	**2(2)**
20-29yrs	**13(6.5)**	**7(7)**	**53(26.5)**	**24(24)**
30-39yrs	**33(16.50**	**15(15)**	**52(26.5)**	**26(26)**
40-49yrs	**23(11.5)**	**10(10)**	**10(50**	**7(7)**
50-59yrs	**7(3.5)**	**4(4)**	**6(3)**	**5(5)**
Total years	**76(38)**	**36(36)**	**124(62)**	**64(64)**

Fifteen (7.5%) of the HIV cases were on admission, while 185(92.5%) were from the HIV retroviral clinic. Common cardiac symptoms and clinical findings were fever 98(49%), weight loss120(60%), diarrhoea 24(12%), rashes 56(28%) , lethargy128(64%).

The packed cell volume ranged from 11.7 to 53% with a mean of 30.8+ 7.8% The BMI ranged from14.09 - 33.8kg/m^2 with a mean of 21.2 + 3.5.kg/m^2 . The CD4 count ranged from 26 – 986cells/l with a mean of 246.51 + 176.1cells/l.(see table 5 on page 47)

CARDIOVASCULAR FINDINGS:
Fifty six of the two hundred patients(28%) presented with cardiovascular symptoms. Twelve of these fifty six patients were in cardiac failure. The symptoms were cough 92(46%) dyspnoea 43 (20%) Paroxysmal nocturnal dyspnoea (5%) pedal swelling (28%) displaced apex(22%) third heart sound was heard in10 (5%) while loud P2 was heard in13 (6.5%) and heart murmurs were heard in(11%). The murmurs were essentially functional murmurs of mitral and tricuspid regurgitation.

Comparison of Cardiovascular findings of Controls and HIV cases.
The pulse rate ranged from 55 to 137 beats/min with a mean of 90.83.± 16.6 beats/min While the pulse rate for the controls ranged from 65- 95beats/min with a mean of 69.62 .± 11.5.beats/min. This was significantly different.
The systolic blood pressure of the cases ranged from 70-140 mmHg with a mean 114.02.± 12.9mmHg. Controls had a range of 100-140 mmHg with a mean of 117.95 ± 11.9 .mmHg. There was no significant difference.
The diastolic blood pressure of the cases ranged from 70 - 90mmHg with a mean 72.44.± 9.5 mmHg. Controls had a range of 50- 90 mmHg with a mean of 74.63 ± 9.38 mmHg. There was no significant difference.
However the mean BMI for HIV patients and controls were 21.22.± 3.50 kg/m^2(ranging from14.09 to 33.8kg/m^2) and 25.71.± 4.7 kg/m^2(ranging from15.41 to 36.33kg/m^2) respectively with a t-test and p-value of -6.40 and .0001.the BMI was significantly lower in the HIV group.(see Table 2)

TABLE 2:COMPARISON OF SOME CLINICAL AND LABORATORY PARAMETERS OF CASES AND CONTROLS

PARAMETER	CASES N = 200	CONTROLS N = 100	t-test	p-value
BMI(kg/m^2)	21.09 ± 4.0	25.06 ± 6.2	-6.40	<0.001*
DIASTOLIC BP(mmHg)	71.87 ± 11.3	72.72 ± 15.11	0.52	0.60
SYSTOLIC BP (mmHg)	113 .09 ± 16.1	114.9 ± 22.3	-1.69	0.094
PULSE RATE (beats/min)	90.24 ± 18.3	67.82 ± 15.71	7.04	< 0.001*
CD4 COUNT(cells/l)	246 ± 176			

***P values ‹ 0.05 are significant**

The mean ±SD pulse rate for the male HIV patients was 85.31.± 18.8 beats/min, while that of the females was 92.17 ± 19.9beats/min this was significantly different.
The systolic blood pressure of the HIV males ranged from 70-140 mmHg with a mean ± SD 114.45 ± 20.58mmHg. HIV females had systolic blood pressure range of 100-140 mmHg with a mean .± SD of 111.13.± 17.27 mmHg. There was no significant difference.
The diastolic blood pressure of the males ranged from 70 - 90 mmHg with a mean.±SD of 71.6. ±14.0 mmHg. Females had diastolic blood pressure ranging from 50 - 90mmHg with a mean. ± SD of 71.31. ± 12.04 mmHg. There was no significant difference.
Comparing the BMI of the male HIV patients to that of the female HIV patients; males had a mean.± SD BMI of 20.71 ± 4.2kg/m^2 and females 21.05 ± 4.4 kg/m^2. There was no significant difference.
However, the PCV of the male HIV patients was significantly higher than that of the female HIV patients with mean±SD: PCV of 33.14 ± 8.66% for males and females 28.67 ± 7.4%..
The mean ±SD CD4 T lymphocyte count for the HIV patients was 246.51 ± 176.2cells /l with a mean ± SD of 251.34 ± 188.6cells /l for males and 237.91 ± 171.0 cells /l for females. This was not significantly different.

TABLE 3:
CLINICAL AND BIOCHEMICAL PARAMETERS AMONG HIV CASES ACCORDING TO SEX.

PARAMETERS	MALES N=76 Mean± SD	FEMALES N=124 Mean± SD	t-test	P-Value
PCV(%)	33.14 ± 8.66	28.67 ± 7.40	-3.86	0.000*
DIASTOLIC BP(mmHg)	71.6 ± 14.0	71.31± 12.04	0.791	0.430
SYSTOLIC BP (mmHg)	114 .45 ± 20.58	111.13 ± 17.27	1.886	0.064
PULSE RATE (Beats/min)	85.31 ± 18.8	92.17 ± 19.9	. 2.21	0 .029*
CD4 COUNT (Cells/l)	251 .34 ± 188.6	237.91 ± 171.0	-.050	0.618
BMI Kg/m^2	**20.71 ± 4.2**	**21.05 ± 4.41**	**0.313**	**0.744**

P values ‹ 0.05 are significant

TABLE 4: THE SPECTRUM OF ECHOCARDIOGRAPHIC CARDIAC ABNORMALITIES IN HIV POSITIVE PATIENTS .

CARDIAC ABNOMALITIES	HIV POSITIVE N(%)	CONTROLS (N%)	p-value
DCM	7(3.5)	0(0)	0.058
PERICARDIAL EFFUSION	89(44.5)	5(5)	< 0.001*
RWMA	10(5)	0(0)	0.023*
DEPRESSED LV EF	25(12.5)	3(3)	0.007
DEPRESSED RV EF	20(10)	6(6)	0.246
DEPRESSED LV EF/ DEPRESSED RV EF	11(9)	3(3)	0.055
ISOLATED LV DIASTOLIC DYSFUNCTION	55(27.5)	21(21)	0.222
CARDIAC ABNOMALITIES	HIV	CONTROLS	p-value
ISOLATED RV DIASTOLIC DYSFUNCTION	12(6)	3(3)	0.017*
RV AND LV DIASTOLIC DYSFUNCTION	36(18)	6(6)	0.261
TUMOUR	1(0.5)	0(0)	0.478
AORTIC ROOT DILATATION	2(1)	0(0)	
DESCENDING AORTA DILATATION	1(.5)	0(0)	0.316
MR	7 (3.5)	1(1)	0.103
TR	55(27.5)	5(5)	0.001*
PR	70(35)	18(18)	0.002*
AR	15(7.5)	1(1)	0.018*
ENDOCARDITIS	1(.5)	0(0)	0.478
MVP	1(0.5)	0	0.478

DCM: dilated cardiomyopathy; RWMA: regional wall motion abnormality;EF: ejection fraction; MR: mitral regurgitation; TR:tricuspid regurgitation; PR:pulmonary regurgitation, AR: aortic regurgitation. MVP: mitral valve prolapse. Significant = $p<0.05$.

Classification Of Pericardial Effusion

Eighty-nine (89)HIV positive patients had pericardial effusion, 62(69.6%) of the pericardial effusions were mild and mostly asymptomatic. 15(16.9%) had moderate effusion (16.9%) while massive effusion was found in 12(13.5%) one had clinical evidence of tamponade while 4 showed only echocardiographic evidence of tamponing with right ventricular diastolic collapse. Some of the effusions were fibrinous.

Distribution Of Cd4 Count: With regards to the CD4 lymphocyte distribution amongst the study population 50% of the patients had AIDS (CD4 lymphocyte count ≤ 200), while 50% had CD4 counts above 200cells/l: however, of these only 8% had CD4 count above 500.

Echocardiography Parameters:

Table5. The mean ± SD RAA /M was 7.85 ± 2.2 cm^2/m in the HIV group, with 8.44 ± 1.5cm^2/m in the controls with no significant difference. The mean ± SD LVEDA/M was 16.49 ± 3.6 cm^2/m in the HIV positive group but in the controls was 17.17 ± 3.4 cm^2/m. The mean ± SD of RVEDA/M was 8.54 ± 4.9 cm^2/m in the HIV group but 8.51 ± 1.9 cm^2/m in the controls, both the RVEDA/M and the LVEDA/M showed no significant difference. Similarly the mean RV E/A compared without difference at 51.99 ± 13.33 and 53.57 ± 12.50 in the

HIV positive group and controls respectively; the LVCO at 7.01 ± 9.5 l/min,5.70 ± 1.6 l/min for HIV positive patients and controls respectively; and the RVCO at 8.72 ± 16.7 l/min and 5.90 ± 1.8 l/min for HIV and controls respectively; the LVDT at 166.34 ± 52.8ms and178 ± 35ms for HIV and controls respectively; the RVDT 168.57± 64.1ms and 168.57 ± 64.1ms for HIV positive patients and controls respectively; the LVM/BSA at 118.58 ± 40.8g/m^2 and 105 ± 28.8g/m^2 respectively.

The PASP and PDAP are significantly higher in the HIV group than in the controls with a mean of 19.31± 10.3mmHg and 13.60 ± 5.3mmHg respectively, for PASP and 17.92mmHg ± 8.1mmHg and 14.41 ± 6.0mmHg for the PADP.

The HIV group had significantly lower LV ejection fraction , lower trans mitral E/A ratio, significantly higher Pulmonary artery systolic and pulmonary artery diastolic pressures, when compared with the normal controls. Also, the mean ± SD of LAA/M in the HIV positive group 7.95 ± 2.3cm 2/m was significantly smaller than controls at 8.65 ± 1.5cm^2/m. However the RV Ejection fraction, RV E/A ratio , LV DECT, RVDECT, the cardiac output at the pulmonary and the aortic did not show any statistically significant difference when compared using the P-value. Nevertheless, cardiac output was relatively higher in the HIV cases while mitral and tricuspid E deceleration time for both ventricles were lower. The cardiac chambers were relatively smaller in the HIV group as compared to the controls and they had higher LVM/BSA.

In comparing the mean values of echocardiographic abnormalities between sexes; The mean RAA for males was 14.22 ± 4.1cm^2 and for the females was 12.25 ± 3.7cm^2 The mean LVEDA for males was 29.33 ± 5.30 cm^2 and for females 23.88 ± 5.32cm^2. The mean LVEDA/M for males was 16.87 ± 3.9cm^2/m and 14.65 ± 3.2cm^2/m for females. The mean RVEDA for males was 15.3 ± 4.1cm^2 and for females 12.71 ± 4.5cm^2. the mean LVM/BSA for males was 136 ± 44.0g/m^2 and females;107 ± 34.6g/m^2. There were significant differences in their RAA, RAA/M, LVEDA, LVEDA/M^2, RVEDA, RVEDA/M, LVM/BSA.

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TABLE 5
Comparison of Echocardiographic Abnormalities Between HIV and Controls

ECHO PARAMETER	HIV	CONTROLS	T-TEST	P- VALUE
LAA/M (cm^2/m)	7.95 ± 2.3	8.65 ± 1.5	2.052	0.041[8]
RAA/M (cm^2/m)	7.85 ± 2.29	8.44 ± 1.5	-1.401	0.162
LVEDA/M (cm^2/m)	16.49 ± 3.6	17.17 ± 3.4	-1.011	0.313
RVEDA/M (cm^2/m)	8.54 ± 4.9	8.51 ± 1.9	0.048	0.962
LVEF(%)	58.49 ± 12.3	69.60 ± 7.4	6.80	< 0.001*
RVEF (%)	51.99 ± 13.3	53.57 ± 12.5	-0.618	0.520
LVE/A	1.34 ± 0 .4	1.72 ± 0.5	- 4.505	< 0.001*

***P values ≤ 0.05 are significant**

TABLE 6. ECHOCARDIOGRAPHIC PARAMETERS OF HIV +VE PATIENTS AND CONTROLS CONTD

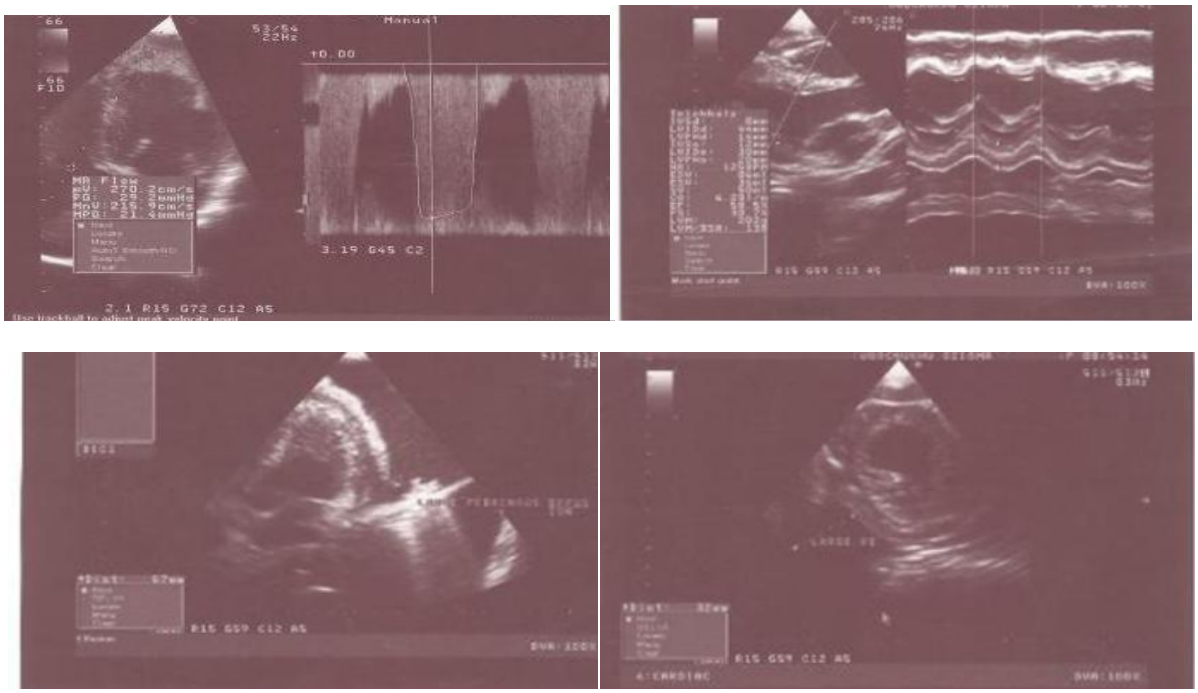
ECHO PARAMETER	HIV	CONTROLS	T-TEST	P- VALUE
RVE/A	1.27 ± 0 .4	1.37 ± 0.5	-1.164	0.246
LVCO (l/min)	7.01 ± 9.5	5.70 ± 1.6	0.830	0.408
LV DECT (msec)	166.34±52.8	178.14 ± 38.5	-1.225	0.221
RV DECT (msec)	168.57±64.1	168.57 ± 64.1	-1.895	0.358
LVM/BSA (g/m^2)	118.58±40.8	105.00 ± 28.8	1.807	.0730
RVCO (l/min)	8.72 ± 16.7	5.90 ± 1.8	0.997	0.320

LVDT: Left ventricular deceleration time; RVDT right ventricular deceleration time; LVEF: left ventricular ejection fraction; RvEF: Right ventricular ejection fraction; LVCO: left ventricular cardiac output RVCO: Right ventricular cardiac output; pressure, PADP; pulmonary artery diastolic pressure;LAA/m:left atrial area indexed for height RAA/m: right atrial area indexed for height, LVEDA/M: left ventricular end diastolic area indexed for height RVEDA/m : right ventricular end diastolic area indexed for height LVESA/m: left ventricular end systolic area indexed for height RVESA/m: right ventricular end systolic area indexed for height. LVM/BSA: left ventricular mass indexed for body surface area. LVE/A: ratio of Mitral inflow measurement of E-velocity to A velocity, RVE/A: :ratio of tricuspid inflow measurement of E-velocity to A velocity.* significant=p value≤ 0.05.

TABLE 7: SEX DISTRIBUTION OF ECHOCARDIOGRAPHIC PARAMETERS

ECHO PARAMETER	MALES	FEMALES	t-test	p-value
LAA(cm^2)	15.81 ± 15.9	122.94 ± 3.1	-1.621	0.107
LAA/M^2(cm^2/m)	7.98 ± 2.1	7.95 ± 2.0	-0.081	0.937
RAA(cm^2)	14.22 ±4.1	12.25 ± 3.7	-2.921	0.004*
RAA/M^2(cm^2/m)	8.35 ± 2.38	7.55 ± 2.2	-2.004	0.047*
LVEDA(cm^2)	29.33 ± 5.3	23.88 ± 5.32	-5.358	0.000*
LVEDA/M^2(cm^2/m)	16.87 ± 3.9	14.65 ± 3.2	-5.358	0.000*
RVEDA(cm^2)	15.31 ± 4.1	12.71 ± 4.5	-3.025	0.003*
RVEDA/M^2(cm^2/m)	9.78 ± 7.0	7.77 ± 2.54	-2.404	0.018*
LVM/BSA(g/m^2)	136.21±44.0	107 ±34.6	-4.253	0.000*
LVEF(%)	56.98 ± 13.6	59.43 ± 11.33	1.141	0.256
RVEF(%)	49.85 ± 12.2	53.32 ± 13.8	1.493	0.138
LVE/A	1.36 ± 0.4	1.34 ± 0.4	-0.236	0.814
RVE/A	1.22 ± 0.4	1.30 ± 0.5	1.010	0.314
LVCO(l/min)	8.124± 14.9	6.22 ± 2.9	-1.096	0.275
RVCO (l/min)	6.29 ±2.9	7.48 ± 3.73	-0.993	0.322

LVDT: Left ventricular deceleration time; RVDT right ventricular deceleration time; LVEF: left ventricular ejection fraction; RvEF: Right ventricular ejection fraction; LVCO: left ventricular cardiac output RVCO: Right ventricular cardiac oputput; PASP: pulmonary artery systolic pressure, PADP; pulmonary artery diastolic pressure;LAA/m:left atrial area indexed for height RAA/m: right atrial area indexed for height,LVEDA/M: left ventricular end diastolic area indexed for height RVEDA/m : right ventricular end diastolic area indexed for height LVESA/m: left ventricular end systolic area indexed for height RVESA/m: right ventricular end systolic area indexed for height.LVM/BSA: left ventricular mass indexed for body surface area. LVE/A:ratio of Mitral inflow measurement of E-velocity to A velocity, RVE/A: :ratio of tricuspid inflow measurement of E-velocity to A velocity



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IV. DISCUSSION

The effect of HIV on the heart is a subject that has attracted a lot of investigators and various studies have been carried and still going on to evaluate the incidence, pattern, pathophysiology of cardiovascular involvement in HIV/AIDS. This study was a prospective analytical cross-sectional study, aimed at evaluating the pattern of heart disease as seen by echocardiography; by assessing the echocardiographic pattern in HIV positive patients (antiretroviral naive) attending HIV clinic in the University of Port Harcourt Teaching Hospital. The incidence of cardiac abnormalities before commencement of HAART.

Most of the work done locally and in Africa have emphasised the impact of HIV on the left ventricle and pericardium, few studies have given attention to the right ventricle and fewer have assessed the impact on the heart as a whole organ. The two parts of the heart though serving different functions are largely dependent on one another[1].

This study evaluated the heart in totality and thereby contributes to the limited information on this subject in this environment.

Demographic Characteristics Of The Study Population

Two hundred HIV positive patients, irrespective of CD4 T lymphocyte count and 100 controls were recruited in this study matched at a ratio of 2:1.

The study subjects were made of 76 males(38%)and 124 females(62%) with a male to female ratio of 1:1.6. They were aged between 18 yrs and 56 years, with a mean age of 33.13 ± 8.4years. The controls were made up of 64 females ((64%) and 36(36%) males with age range between 19 and 54yrs with a mean age of 31.82 .±8.72years, the HIV study population showed more women affected than male this corroborates the UNAIDS**[94]** finding that of the 33.3 million adults living with HIV more than half are women..

The subjects and Controls were adequately matched for sex and age. Their systolic and diastolic blood pressures showed no significant difference. However, there were significant differences in their BMI and pulse rates. This can be explained by the disease process, HIV /AIDS is associated with cachexia and anaemia.

There was no significant variation in the sex distribution in BMI, but the heart rate was significantly higher in the HIV females when compared to the males. Anaemia was more marked in the female HIV patients this may explain the difference in heart rate. In addition, males are known to have a higher normal PCV when compared to the females while the females on the other hand are more susceptible to anaemia because of the periodic loss of blood from menstruation despite the disease state**.**

HIV is CD4 T lymphocyte depletory and the concentration of CD4 T lymphocyte in the blood has been used to classify the disease condition[1]. The mean CD4 count of the patients in this study did not show significant sex variation. The range was from 26-936cells/l. AIDS is also defined as CD4 count of 200cells /l and below. One hundred (100) patients fell into this category accounting for 50 % of the study cases. Eight four (42%) patients had CD4 count between the range of 201 to 500 cells/l. While only 16(8%) had CD4 count of

above 500. This explains the high prevalence of cardiac abnormalities seen in this study, as HIV /AIDS cardiovascular disease is said to be a late

Echocardiography Findings:

Pericardial Effusion: Pericardial effusion was found in 44.5% of the HIV group and was the commonest cardiac abnormality. This was corroborated by the low voltages seen on ECG. This is agreeable with a large number of studies. The Danbauchi et al[9] study from Zaria had pericardial effusion as the commonest finding. However in contrast the frequency of occurrence was lower than in the Danbauchi study, which reported 60%. This can be explained by the population type studied, the Danbauchi et al study recruited only patients with advanced disease but in this study the number of patients with CD4 count lower than 200 was 50% of the entire study population and majority of the pericardial effusion was found in this group. However, the study by Morozov et al **[48]** showed pericardial effusion in only 8%. The difference may be accounted for by varying opportunistic co-infections in different regions.

The pericardial effusion found in this study was largely asymptomatic with mild effusion accounting for over two thirds (69%) of the pericardial effusion, moderate pericardial effusion (16.9%) and severe pericardial effusion (13.5%).

Diastolic Dysfunction:

Another notable finding in this study, was diastolic dysfunction. Isolated LV diastolic dysfunction affected 27.5% of the study population. Danbauchi et al also noted LV diastolic dysfunction in 10% of the cases. This has also been corroborated by a large number of studies. **[13,29]** LV diastolic dysfunction has been described as the first abnormality of several cardiovascular diseases. In research carried out by Coudray et al**[19]**. LV diastolic function estimated by doppler Echocardiography in HIV patients showed an increase in the IVRT and a decrease in the E wave velocity when compared with control patients. Martinez et al**[49]**also noted diastolic dysfunction in their study. The African study by Longo Mbenza et al **[12]** noted diastolic dysfunction in 80% of its study population and attributed it to systemic amyloidosis and concentric LVH developed by the HIV patients in the course of the study.

This study enrolled 26% of patients in the 3rd and 26.5% in their 2nd decades of life. This accounts for 52.5% of the study population. Studies have corroborated a higher incidence of HIV in the second and third decades of life**[50]**. This large population of young people in this study may be contributory to the large percentage of diastolic dysfunction seen in this study as the E/A ratio simulates a restrictive pattern in the young. Endocardial fibrosis is a noted finding in hearts of patients infected with the HIV and can account for the diastolic dysfunction in this group. In addition myocarditis and myocardial fibrosis can account for impaired relaxation which is associated with diastolic dysfunction.**[51]**

Isolated RV diastolic dysfunction was found in only 6% of the study population and can be largely explained by the presence of pulmonary hypertension. Studies have corroborated right ventricular involvement in HIV. **[52-53]**

RV and LV diastolic dysfunction occurred in 18% of the study population this was also seen in some of the electrocardiography tracings. This variability may be explained by the patchy pattern in which the HIV affects the heart. HIV has been shown to affect the Heart in a patchy distribution probably more extensive in some than the other**[54-55]**. Explanation has not been put forward as to any sites of preference in the heart. Nevertheless, looking at the pattern of distribution it tends to affect the left more than the right.

Depressed Ejection Fraction:

Subjective but noteworthy is the gross appearance of the myocardium of the HIV patients on echocardiography testing. The myocardium appears 'slow' and hypoechoic. Depressed ejection fraction of the left ventricle without frank dilatation was seen in 12% of the study population, while another 8% had isolated depressed right ventricular and 9% showed both depressed LV and RV EF. This disparity still goes on to validate the patchy distribution of the effect of the virus on the myocardium. Yet the left ventricle appears more affected than the right ventricle. Depressed ejection fraction has been attributed to elaboration of inflamatory cytokines that depresses the myocardium notably nitric oxide **[23-24]**. Studies have shown that the myocardium of these patients stain more intensely for nitric oxides **[23-24]**. Autoimmunity has been also implicated and auto-antibodies have been seen in greater concentration **[24]**. This may go a long way to explain the globular nature of the hypokinesia seen in some of these patients implicating a systemic rather than a local elaboration of cytokines.

Cassilino et al**[56]** noted that right ventricular dysfunction may be an important feature in HIV infected patients. In this study there was a significant difference between the right ventricular diastolic volume and the right ventricular EF between the HIV patients and normal controls.

Coronary Artery Disease: Regional wall motion abnormalities corroborrated by the ECG findings point to coronary artery disease which has been shown to be common in HIV patients. This study noticed regional wall motion abnormalities in 10 patients accounting for 5% of the study population. The discrepancy may point to a possible co-existing risk factor. Most patients that showed regional wall motion abnormality had anterior medial hypokinesia, some had inferior and one patient showed a more extensive involvement with anterior and inferior hypokinesia. They were of a grade of 1 to 3.

Dilated Cardiomyopathy was found in 7 of the study population accounting for only 3.5% of the study population, higher prevalence has been reported but the majority of subjects recruited were asymptomatic.

Concerning aortic artery dilatation, two subjects had aortic sinus dilatation while one showed dilatation of the descending aorta. Most of the valvular pathologies were largely functional.

Echocardiographic Parameters:

The echocardiographic parameters showed statistically significant differences in the left atrial area when compared with controls, the left ventricular ejection fraction and the ratio of the left ventricular mitral inflow velocities: the early mitral velocity E and atrial contribution A(LV E/A). The values of these parameters were significantly lower than the HIV negative controls.

There were also significant differences in the pulmonary artery systolic and diastolic blood pressures. The pulmonary pressures were significantly higher in the HIV group. When the LAA was indexed for height, there was no significant difference.

The cardiac chambers, except the RVEDA, were however smaller in the HIV groups than those of the normal this can be accounted for by the endocardial fibrosis that has been noted in the histology of HIV patients.

V. CONCLUSION.

Cardiac abnormalities are more common in HIV infected individuals when compared to normal individuals. Echocardiographic assessment is a very valuable tool in detecting cardiac abnormalities early in people living with HIV.

Pericardial effusion and diastolic dysfunction are also common in HIV positive patients attending clinic in the University of Port Harcourt Teaching Hospital. HIV patients with CD4 count lower than 200 had a higher incidence of cardiac. Dilated cardiomyopathy in HIV/AIDS is associated with more advanced immunosuppression and lower CD4 lymphocyte count < 100cells/ l.

This prospective descriptive, cross sectional study has shown a high prevalence of cardiac abnormality; 63% as shown by echocardiography. Strikingly most of the subjects studied had no cardiac symptoms but already had on going cardiac pathologies, suggesting the need for managing physicians to be on the look out, to allow for prompt diagnosis and management.

REFERENCES

[1]. Anthony SF, Lane HC, Human Immunodeficiency Virus disease, AIDS and related disorders, Harrison principles of internal medicine, 17th edition. McGraw Hill Companies, 2008, 1076-1139.

[2]. Https://www.unaids.org/en/resources/fact-sheet. Accessed 2022/25/02

[3]. Nasidi A, Harry TO. The Epidemiology of HIV/AIDS in Nigeria. In Adeyi,s AIDS in Nigeria: A non on the threshold. Havard centre fo population and development Stues 2006:17-36.http://www.apin.havard .edu/chapter 2.pdf.

[4]. Jaffe HW, Francis DP, Mclane M et al. Transfus associated AIDS: serologic evidence of human T-cell leukemi virus infection of donors. Science1984;223:1309-1312.

[5]. Guillermo SG, HIV and cardiovascular manifestation of the disease, Circulation.2002 106: 1420-1425.

[6]. Barbaro G, Di Lorenzo G, Grisorio B. Incidence of Dilated Cardiomyopathy and Detection of HIV in Myocardial Cells of HIV/ Positive Patients.New England Journal of Medicine,1998:339;1093- 1099.

[7]. Barbaro G, Fisher S, Lipshultz S. Pathogenesis of HIV-associated cardiovascular complications. Lancet Infect Disease 2001; 1:115-124.

[8]. Stein, JH, Klein MA, Bellehumeur JL, McBride PE, Wiebe DA, Otvos JD, and Sosman JM. Use of human immunodeficiency virus-1 protease inhibitors is associated with atherogenic lipoprotein changes and endothelial dysfunction. Circulation 2001;104:257-262.

[9]. Danbauchi SS, Alhassan MA, Oyati AI et al, Cardiac manifestation of stage III and IV HIV/AIDS compared to subjects on ARV in Zaria, Nigeria. Nigerian Journal of Cardiology, 2006: 3; 5-10.

[10]. HIV Drugs, Abacavir Didanosine, Increase the risk of heart attack ,studsuggests,HTTP://www.Sciencedaily.com release /2008/ 102/ 080207155728 .html.

[11]. Babaro G, Di Lorrenzo G, Grisorio **,** et al: cardiac involvement in acquired Immune deficiency Syndrome. A multicentre clinical pathological study. AIDS Res 14: 1071,1998.

[12]. Longo-Mbenza B, Seghers KV, Phuati M et al. Heart Involvement And HIV Infection In African Patients : Determinants Of Survival.International Journal of Cardiology. 1998;64:63.

[13]. Rogstad K, Shah R, Tesfaladet G, Abdullah M et al. Cardiovascular Autonomic Neuropathy In HIV Infected Patients. *Sex Transm Inf* 1999;75:264 -267.

[14]. Nzuobontane D, Ngu BC, Kuaban C et al, Cardiovascular Autonomic Dysfunction In Africans Infected With Human Immunodeficiency Virus, R Soc Med 2002**;95**:445-447.

[15]. Lipshultz S E. Dilated cardiomyopathy in HIV-infected patients. N Engl J Med,1998; 339:1153-1155.

[16]. Fischer S and Lipshultz S; Cardiovascular abnormalities in HIV infected individuals Braunwald E. Heart disease: a textbook of cardiovascular medicine, 7th edn. W. B Saunders, Philadelphia; 2004, 1711-1729.

[17]. Currie PF, Jacob AJ, Foreman AR et al Heart Muscle Disease Related To HIV: Prognostic Implication, British Medical Journal.1994; 309:1605-1607.
[18]. Omotoso AB, Opadijo OG.Araoye MA,The Evolving Role Of HIV Infection In Dilated Cardiomyopathy In Ngerians. Trop cardiol.2000; 26:85-87.
[19]. Coudray N, De Zuttere D, Force G, et al. Left Ventricular Diastolic Function In Asymptomatic And Symptomatic Human Immunodeficiency Virus Carries: An Echocardiographic Study. Eur Heart J 1995; 16: 61-67.
[20]. Barbaro G, HIV and the cardiovascular system pre-HAART HIV AIDS Rev, 2004;3(1): 5-10.
[21]. 21. Rodriguez ER,Nasim S,Hasia J, et al cardiac myocytes and dendritic cells habour Human Immunodeficiency Virus in infected patients with and without cardiac dysfunctions:detection by multiplex, nested, polymerase chain reaction in individually microdissected cells from right ventricular endomyocardial biopsy tissues. JACC 1991:68; 1511 – 1520.
[22]. Barbaro .G. and. Klatt E.C, HIV and the cardiovascular system, *AIDS Rev* 2002; 4:93-103.
[23]. Barbaro .G, HIV infection and the cardiovascular system part II: the post-HAART era, HIV AIDS Rev3 2004 ;1: 14-20.
[24]. Babaro G, Di Lorenzo G, Maurizio S et al Intensity of Myocardial Expression of Inducible Nitric Oxide Synthase Influences the Clinical Course of Human Immunodeficiency Virus-Associated Cardiomyopathy, Circulation 1999;100: 933-939.
[25]. Currie PF, Godman JH, Caforio ALP et al Cardiac autoimmunity in HIV related Heart muscle disease. Heart, 1998; 79: 599-604.
[26]. Barbaro G, Fisher SD, Lipshultz SE. Pathogenesis of HIV-associated cardiovascular complications. Lancet Infectious Diseases,2001; 1:115-124.
[27]. Breuckmann F, Neumann T, Kondratieva J, et al. Dilated cardiomyopathy in two adult humanimmunodeficiency positive patients possibly related tohighly active antiretroviral therapy (HAART). European Journal ofMedical Research,1995;10: 395-399.
[28]. Chariot P, Perchet H, Monnet I. Dilated cardiomyopathy in HIV-infected patients. New England Journal of Medicine. 1999 ; 340:732-735.
[29]. Miller TL, Orav EJ, Colan SD, et al, Nutritional status and cardiac mass and function in children infected with the human immunodeficiency virus. Am J Clin Nutr 1997; 66:660-664.
[30]. Miller TL. Cardiac complications of nutritional disorders. In: Lipshultz SE, editor. Cardiology in AIDS. New York: Chapman & Hall, 1998: 307-316.
[31]. Hoffman M, Lipshultz SE, Miller TL. Malnutrition and cardiac abnormalities in the HIV-infected patients. In: Miller TL, Gorbach S, editors. Nutritional aspects of HIV infection. London: Arnold, 1999: 33-39.
[32]. Apostolos K, Manolis F, George L et al ,Assessment of cardiac function with Doppler tissue imaging in asymptomatic HIV patients , International Journal of STD, AIDS, 2008;19:227-231.
[33]. Nkuoa IL,Tsombou B,Bouamoue CH et al Non rheumatic pericarditis with effusion causes out comes and HIV linking. Trop Cardiol 1999;25:8-12
[34]. Cheitlin MD. AIDS and the cardiovascular system. In: Alexander RW, Schlant RC, Fuster V, eds - Hurst's the Heart, Arteries, and Veins. 9th edition. New York: 1998: 2145.
[35]. Heidenreich PA, Eisenberg MJ, Kee LL, et al. Pericardial effusion in AIDS: Incidence and survival. Circulation, 1995; 92: 3229-3234.
[36]. Zayas R, Anguita M, Torres F, et Al Incidence Of Specific Aetiologies And Role Of Methods Of Specific Etiologic Diagnosis Of Primary Acute Pericardial Effusion; AM J cardiol 1995;75:378-382.
[37]. Acheron LJ: Cardiac complications in acquired immunodeficiency syndrome (AIDS): A review. JACC, 1989; 13:1144 – 1146
[38]. Carter M, High prevalence of asympyomatic Heart Disease in HIV/AIDS,http/www.aidsmap.com,page 1429181.
[39]. Shannon RP, Simon MA, Mathier MA, et al Dilated Cardiomyopathy associated with Simian AIDS in non human primates. Circulation.2000:101;185- 193.
[40]. Sanna P, Bertoni F, Zucca E, et al. Cardiac involvement in HIVrelated non-Hodgkin's lymphoma: a case report and short review of the literature. Ann Hematol, 1998;77:75-78.
[41]. Duong M, Dubois C, Buisson M et al. Non-Hodgkin's lymphoma of the heart in patients infected with human immunodeficiency virus. Clin Cardiol 1997; 20(5):497-502.
[42]. Umamahesh C R, Atiar M R, Nasir H et al Reversible right ventricular dysfunction in patients with HIV infection. Southern medical journal,2006; 99: 274-278.
[43]. Adebayo RA .Balogun MA, Akinola MA et al. Cardiovascular Involvement In Sickle Cell Anaemia, Nigerian Journal of Medicine,2002;11: 145-150.
[44]. Adebiyi AA, Falase AO, AkinovaYA.Left ventricular systolic function of Nigerians with sickle cell anemia. , tropical cardiology,1999; 98:27-32 .
[45]. Ikechebelu JI, Kalu S, Muozi A. HIV counseling and testing for prevention of mother to child transmission: Impact of cost and Tesating and counseling Method. Tropical Journal of Medical Research; 2007; 11:11-13.
[46]. Pfisterer ME, Bahler A, Zaret BL, Range of normal values for left and right ventricular Ejection Fraction at rest and during exercise assessed by radionucleotide angiocardiography, Eur Heart J,1985;6:647-655.
[47]. Braunwald E, Diseases of the heart, Harrison principles of internal medicine, 15th edition. McGraw Hill Companies,1309 – 1355.
[48]. Morozov A, Feldman M, Antonova V. Study of electrocardiographic abnormalities in different evolutive phases of HIV/AIDS, The 3rd IAS Conference on HIV Pathogenesis and Treatment,July 24-27,2005.
[49]. Martinez- Garcia T, Sobino JM, Pujoi E, et al Ventricular Mass And Diastolic Function In Patients Infected By The Human Immunodeficiency Virus, Heart 2000;84:620-624.
[50]. Eghafora NO,Phillips MN, Uraih N, HIV- 1 Seroepidemiological profile in age groups and sexes in Benin city Nigeria. Trop Geog Med: 1993;45(60):308-309.
[51]. Lipshultz SE, Orav EJ, Sanders SP, et al. Immunoglobulins and left ventricular structure and function in pediatrics HIV infection. *Circulation.* 1995; 92: 2220–2225.
[52]. Morse JH, Barst RJ, Fotino M. Familial pulmonary hypertension: immunogenetic findings in four Caucasian kindreds. Am Rev Respir Dis 1992; 145:787-92
[53]. Carter M, High prevalence of asympyomatic Heart Disease in HIV/AIDS,http/www.aidsmap.com,page 1429181.
[54]. De Castro S, D'Amati G, Gallo P et al, Frequency Of Global Left Ventricular Dysfunction In Human Immunodeficiency Virus, Journal of America College of Cardiology,1994; 24:1018-1024.
[55]. Shannon RP, Simon MA, Mathier MA, et al Dilated Cardiomyopathy associated with Simian AIDS in non human primates. Circulation.2000:101;185-193.
[56]. Casalino E, Laissy JP, Servois Vet al Right ventricular dysfunction in HIV infected patients. Evaluation by magnetic resonance imaging (MRI). *Int Conf AIDS.* 1993; 9 :432 .

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