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Echocardiographic Corelation of Pulmonary Artery Pressure and Right Heart Parameters in HIV Positive Haart Naïve Individuals.

COOKEY SN, BRIGGS FK, AMACHREE E.

Cookey S N
Consultant Cardiologist, Department of Internal Medicine,
Rivers State University Teaching Hospital
No 8-10 Harley Street, Old GRA
Port Harcourt, Rivers State
Briggs FK
Senior Registrar Cardiology, Department of Internal Medicine,
Rivers State University Teaching Hospital
No 8-10 Harley Street, Old GRA
Port Harcourt, Rivers State
Amachree E
Consultant Family Physician, Department of Family Medicine.
Rivers State University Teaching Hospital
No 8-10 Harley Street, Old GRA
Port Harcourt, Rivers State.
Corresponding Author: Stella N. Cookey

ABSTRACT

BACKGROUND

Echocardiography is a non-invasive assessment tool for the assessment of pulmonary artery pressures. The prevalence of Pulmonary hypertension is higher in HIV patients than in the general population. The pathogenesis of HIV associated PHT is still uncertain however it has been associated chronic chest infection, thromboembolism and right sided heart disease. Few African studies have evaluated the prevalence of pulmonary hypertension in this group of patients.This study seeks to contribute to knowledge with regards to this subject area and to understand the impact of right heart disease on the prevalence of pulmonary hypertension in HIV and AIDs.

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: *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 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 age of 31.82 ±8.72years and male to female ratio of 1: 1.7. The frequency of pulmonary artery regurgitation(PR) was more than tricuspid regurgitation(TR): Seventy of the study showed evidence of pulmonary artery regurgitation of the pulmonary artery with fifty-five (55) having tricuspid valve regurgitation. There was significant difference between prevalence of TR and PR in HIV positive patients relative to controls. The estimated mean ±SD of pulmonary pressure was significantly higher in HIV group in comparison with the normal population with Estimated PASP of 19.13 ± 10.3mmHg relative to controls of 13.60 ± 5.3mmHg, for PADP: 17.92±8.1mmHg and 14.41 ± 6.0mmHg.

Conclusion: The prevalence of pulmonary hypertension was 7.5% in the study population. TR was associated with dilated right atrial and right ventricle. The estimated PASP showed positive corelation(1 tailed) with the

tricuspid E/A(.68,.05), while the estimated PADP (-.80,.05),showed negative correlation with right ventricular ejection fraction.

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I. INTRODUCTION

The incidence of HIV-associated pulmonary hypertension is 1 in 200[1]. This is higher than the in the general population. Primary pulmonary hypertension has been reported in HIV infected patients without evidence of thromboembolic disease, intravenous drug use, right sided endocarditis or pulmonary infection. A case report of a young female presenting with exertional dyspnoea found to have pulmonary hypertension was related to HIV. In this report the patient had a CD4 count of 195. Most African studies have not evaluated the prevalence of pulmonary hypertension in this group of patients.

Idiopathic HIV associated pulmonary hypertension is not the only cause of pulmonary hypertension in HIV disease. Pulmonary hypertension is caused by chronic chest infection, thromboembolism and right sided heart disease.Pellicelli and colleagues[2-4] reported statistically significant difference between HIV positive patients with AIDS and those without AIDS with regard to degree of pulmonary hypertension, with AIDS patients having a higher pulmonary artery systolic pressure. Saidi and Bricker[5] also reported the association of pulmonary hypertension with HIV.

The pathogenesis of HIV associated PHT is still uncertain[6-9]. A hypothesis of direct effect of HIV on pulmonary vascular endothelium or smooth muscle has not been validated as HIV was not demonstrated in pulmonary vascular endothelium. However it has become clear that HIV may indirectly stimulate the vascular endothelium and smooth muscle cell growth, along with vasoconstriction via some mediators and cytokines. In patients with PHT, the increase in serum interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor alpha (TNF-α) concentrations, supports this hypothesis. Genetic predisposition is believed to play a role since only a small percentage of HIV infected patients present with the disease. Morse, et al[4]. documented a significant increase frequency of HLA-DR6 and of HLA-DR52 in the HIV patients with PHT compared with non-HIV matched subjects and HIV matched subjects who do not have PHT. The largest collective review to date showed that progressive shortness of breath is the most common presentation, along with pedal oedema, non-productive cough, fatigue, syncope or near-syncope and chest pain. Physical findings include the common signs of pulmonary hypertension, i.e. jugular venous distension, a loud P2, right-sided S3 gallop, tricuspid and pulmonary regurgitation murmur and peripheral oedema.

Echocardiography has proven a very useful non invasive tool for assessing pulmonary hypertension. In pulmonary hypertension [1] echocardiography usually demonstrates enlargement of the right atrium and ventricle, dilated pulmonary artery, normal or small left ventricular dimensions and a thickened interventricular septum. The septal/posterior left ventricular wall ratio may be abnormally increased, as in Hypertrophic cardiomyopathy, but other echocardiographic signs characteristic of that condition is not observed. Spectral Doppler has been recommended for use in patients with pulmonary hypertension.

There are various methods of estimating pulmonary artery pressure. In the absence of right ventricular outflow tract obstruction or pulmonary artery stenosis the right ventricular pressure equals the pulmonary artery pressure [10,11]. The systolic pulmonary artery pressure can be estimated using Doppler velocity of tricuspid regurgitation, ventricular septal defect or the patent ductus arteriosus and the right ventricular isovolumetric time. Pulmonary artery diastolic pressure can be estimated by pulmonary regurgitant jet. The mean pressure gradient can be estimated from the pulmonary doppler activation time. In 70% of patients with pulmonary systolic hypertension there is usually an accompanying tricuspid regurgitation and pulmonary regurgitation. Pulmonary artery systolic pressure has been defined as tricuspid peak velocity of > 2.9m/s.[12-13]

The eccentricity index was originally described by Ryan et al [14] as a method of quantifying the septal shift observed in right ventricular pressure and volume overload states in pulmonary hypertension. An abnormal systolic eccentricity index suggests high pulmonary arterial systolic pressures. An abnormal diastolic eccentricity index, the parameter associated with adverse outcomes in patients, implies an elevated right ventricular diastolic pressure and right ventricular failure.

Concerning the treatment of secondary pulmonary hypertension in HIV patients; [15-20] treatment is usually that of the underlying cause and treatment of the pulmonary hypertension with vasodialtor therapy; edothelin receptor antagonist, calcium channel antagonist and phosphodiesterase inhibitors amongst others. To date, there is no certain therapeutic approach to the patients with HIV associated PHT. Most patients receive supportive treatment for right-sided heart failure and pulmonary hypertension, such as oxygen supplement and diuretics. Oral anticoagulation has been shown to significantly improve survival in patients with non-HIV related primary PHT, but results in HIV associated PHT has not been noted.

The role of antiretroviral agents in HIV associated PHT have been disappointing. Some data showed to decrease right heart pressure with antiretroviral medications but some study showed no clinical benefit, but rapid deterioration of clinical course in some patients.

II. METHOD

STUDY DESIGN:
This was a prospective, descriptive, cross sectional study.
STUDY SITE:
This study was conducted at the University of Port Harcourt Teaching Hospital. This hospital serves as a major referral centre for Rivers state, Bayelsa state and its subregions. It is situated in in Choba. Port Harcourt is a cosmopolitan city with residents from all over the country.
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: The minimum number of patient required for this study was calculated from the method of kish:[21]

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

$$n = 0.5 * 0.5 * 1.96^2 \Big/ 0.05^2 = 384$$

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

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.
PATIENTS: The study sample was made up of 200 HIV positive patients, who were antiretroviral naive. They were 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 [22]. 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.[23]
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.[23]

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

Protocol:

1. 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.
2. 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.
3. The ventricular diastolic inflow were measured by positioning the sample volume at the tip of the 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.
4. Right atrial area was measured by planimetry in the apical four-chamber view at end-systole, corrected for height.
5. 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.
6. Maximal instantaneous velocity of the tricuspid regurgitation and pulmonary regurgitation signal were measured using continuous wave Doppler.
7. Pulmonary artery pressures were assessed by the modified Bernoulli equation: the peak velocities of TR and PR were measured and the pressures estimated using the formulae below:

Pulmonary Artery Systolic Pressure(PASP) $=4(VTR)^2 + RAP$

VTR = peak velocity of tricuspid regurgitant jet.

RAP = right atrial pressure.

Pulmonary artery pressure is equal to the right ventricular pressure provided there is no shunt or stenosis.

Pulmonary artery diastolic pressure was estimated $= 4(VPR)^2 + RAP$.

Where VPR = peak pulmonary regurgitant velocity.

RAP =right atrial pressure.

Pulmonary hypertension was defined by an elevated pulmonary artery systolic pressure >30mmHg and pulmonary artery diastolic pressure >19mmHg[24-25].

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Where VPR = peak pulmonary regurgitant velocity.

RAP =right atrial pressure.

TABLE 1

The Right Atrial Pressure(RAP):

The RAP was estimated from the evaluation of the inferior vena cava (IVC) during respiration from the subcostal view.[87,88,89.] the estimation was as follows

IVC	CHANGE WITH RESPIRATION	ESTIMATED RAP IN mmHg
Small(<1.5cm)	Collapse	0 – 5
Normal(1.5-2.5cm)	Decrease by >50%	5 – 10
Normal(1.5-2.5cm	Decrease by <50%	10 – 15
Dilated(>2.5)	Decrease by <50%	15 – 20
Dilated with dilated hepatic veins	No change	>20

Definition of Cardiac Abnormalities:

1. Depressed RV ejection fraction was defined by the publication of Pfisterer et al as an ejection fraction ‹40%.[26]

2. Diastolic dysfunction (RV E/A) was defined by the normal generated from this study with a mean of 1.27± 0.4.

3.Pulmonary hypertension was defined by an elevated pulmonary artery systolic pressure >30mmHg and pulmonary artery diastolic pressure >19mmHg[26].

DATA ANALYSIS

Analysis was performed using the SPSS 23 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 Paired ' 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 PASP, PADP : RAA and RVEDA, using Pearson correlation coefficient

III. RESULTS

CARDIOVASCULAR FINDINGS:

Fifteen (7.5%) of the HIV cases were on admission, while 185(92.5%) were from the HIV retroviral clinic. Fifty-six of the two hundred patients (28%) presented with cardiovascular symptoms. Twelve of these fifty-six patients were in cardiac failure. Common cardiac symptoms and clinical findings were fever 98(49%), weight loss120(60%), diarrhoea 24(12%), rashes 56(28%) , lethargy128(64%)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.

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) and 27.5%, 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.

TABLE 2
Comparison of demographic data of cases and controls

CHARACTERISTICS	CASES N=200	CONTROLS **N=100**	t-test	p-value
Gender				
Male	76	36		
Female	124	64		
Mean Age ± SD	33.13 ± 8.4	32.83 ± 8.72	0.11	0.91
Age Range	18-53	19-50		
BMI(kg/m^2)	21.09 ± 4.0	25.06 ± 6.2	-6.40	<0.001*
Systolic BP(mmHg)	113.09 ± 16.1	114.9 ±22.3	-1.69	0.094
Diastolic BP(mmHg)	71.87 ± 11.3	72.72 ± 15.11	0.52	0.60
Pulse Rate(beats/min)	90.24 ± 18.3	67.92 ± 15.71	7.04	<0.001*
CD4 Count(cells/l)				

***P values ‹ 0.05 are significant**

The Table below, shows the different forms of right heart abnormalities seen on echocardiography in the study cases. Isolated RV depression was 20(10%) without chamber dilatation, while combined LV and RV diastolic dysfunction without chamber dilatation was seen in 11(9%), by pulmonary hypertension in15(7.5%) of the study population. However pulmonary regurgitation and tricuspid regurgitation were present in 70 and 55 respectively accounting for 35% and 27.5%.

TABLE 3 Comparison of Cardiac Abnormalities between HIV and Controls

CARDIAC ABNORMALITIES	HIV POSITIVE N(%)	CONTROLS N(%)	P-Value
Depressed RV Ejection Fraction (Normal Chamber)	20(10)	6(6)	0.25
Depressed RV and LV Ejection Fraction (Normal Chamber)	11(9)	3(3)	0.06
Pulmonary Hypertension	15(7.5)		0.01*
Isolated RV Diastolic dysfunction	12(6)	3(3)	0.02*
RV and LV diastolic dysfunction	36(18)	6(6)	0.26
Tricuspid Regurgitation	55(27.5)	5(5)	0.00*
Pulmonary Regurgitation	70(35)	18(18)	0.00*

TABLE 4 Comparison of Cardiac Parameters between HIV and Controls.

ECHO PARAMETER	***HIV***	***CONTROLS***	***T-TEST***	***P- VALUE***
RAA/M (cm^2/m)	7.85 ± 2.29	8.44 ± 1.5	-1.401	0.162
RVEDA/M(cm^2/m)	8.54 ± 4.9	8.51 ± 1.9	0.048	0.962
RVEF (%)	51.99 ± 13.3	53.57 ± 12.5	-0.618	0.520
RVE/A	1.27 ± 0 .4	1.37 ± 0.5	-1.164	0.246
RV DECT (msec)	168.57±64.1	168.57 ± 64.1	-1.895	0.358
RVCO (l/min)	8.72 ± 16.7	5.90 ± 1.8	0.997	0.320
PASP(mmHg)	19.31 ± 10.3	13.60 ± 5.3	4.624	0.001*
PADP (mmHg)	17.92 ± 8.1	14.41 ± 6.0	2.90	0.005*

RVDT right ventricular deceleration time; ; RvEF: Right ventricular ejection fraction; RVCO: Right ventricular cardiac oputput; PASP: pulmonary artery systolic pressure, PADP; pulmonary artery diastolic pressure;RAA/m: right atrial area indexed for height, RVEDA/m : right ventricular end diastolic area indexed for height RVESA/m: right ventricular end systolic area indexed for height. RVE/A: :ratio of tricuspid inflow measurement of E-velocity to A velocity.

Table 5: *Mean Variables Amongst HIV Cases with obvious PR and TR*

	Minimum	*Maximum*	*Mean ± Std. Deviation*
AGE	*23*	*53*	*34.81 ± 8.64*
BMI	*16.14*	*31.65*	*21.98 ± 4.18*
HR	*81*	*112*	*98.33 ± 15.82*
SBP	*110*	*190*	*127.75 ± 25.98*
DBP	*60*	*80*	*70.00 ± 5.35*

PCV	*20*	*38*	*31.67 ± 5.22*
CD4	*28*	*476*	*234.50 ± 154.36*
RAA	*7.76*	*23.84*	*12.93 ± 3.82*
RVEDA	*8.62*	*47.00*	*17.52 ± 10.97*
RVESA	*4.20*	*16.35*	*9.74 ± 3.95*
EF	*24.2*	*69.0*	*46.66 ± 13.38*
T E/A	*.560*	*2.15*	*1.27 ± .40*
PADP	*7.31*	*30.76*	17.92 ± 8.1
PASP	*6.12*	*38.31*	19.31 ± 10.3
IVC	*14*	*36*	*22.92 ± 7.56*

TABLE 6:CORELATION OF RIGHT HEART PARAMETERS AND PASP AND PADP

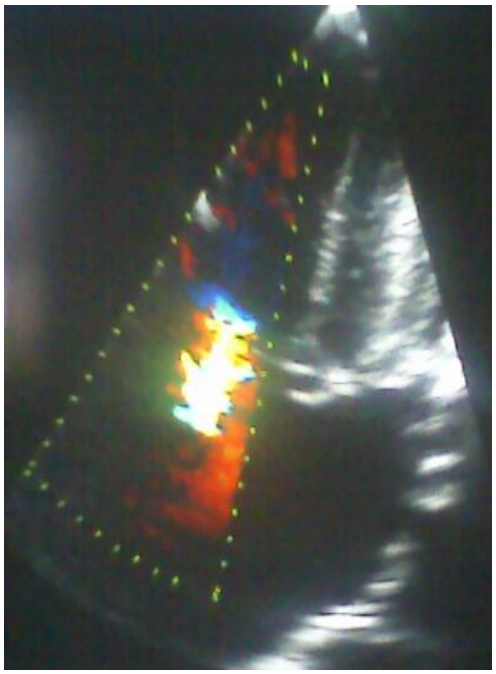
		CD4	RAA	RVEDA	RVESA	RVEF	T E/A	PADP	PASP	IVC
PASP	Pearson Correlation	.57	.16	.07	.06	.34	.68*	1	-.27	.24
	Sig. (1-tailed)	.17	.38	.45	.45	.26	.05	.33	1	.30
PADP	Pearson Correlation	.28	.10	.48	.54	-.80*	-.14	-27	-.27	.28
	Sig. (1-tailed)	.30	.43	.21	.17	.05	.39	1	.33	.30
BMI	Pearson Correlation	.44*	-.29	-.25	-.44	.05	-.58*	-.67*	-.58*	-.14
	Sig. (1-tailed)	.05	.17	.23	.09	.44	.02	.01	.02	.32
HR	Pearson Correlation	1.00**	-.03	.73	.01*	-.99*	.68	1.00**	1.00**	-.51
	Sig. (1-tailed)	.00.	.49	.24	.02	.05	.26	.00	.00	.33
SBP	Pearson Correlation	-.17	.43	.99	.57	-.79	-.70	-.60	-.30	.28
	Sig. (1-tailed)	.36	.24	.05	.31	.21	.06	.10	.35	.30
DBP	Pearson Correlation	-.07	-.37	-.35	.42	-.14	.62	.60	.19	.27
	Sig. (1-tailed)	.44	.27	.39	.36	.46	.09	.10	.40	.30
PCV	Pearson Correlation	.21	.77**	.15	.15	-.11	-.42	-.28	-.21	-.10
	Sig. (1-tailed)	.24	.00	.35	.35	.39	.1	.23	.29	.38

P values <0.05 are significant

TABLE 7: CORELATION OF RIGHT HEART PARAMETERS AND PASP AND PADP in Patient with Pulmonary Hypertension.

		RAA	RVEDA	RVESA	EF	T E/A	PASP	PADP	IVC
PCV	Pearson Correlation	.82*	.15	.35	-.12	-.73*	-.60	-.17	.08
	Sig. (1-tailed)	.01	.39	.25	.41	.03	.14	.39	.44
CD4	Pearson Correlation	-.30	-.58	-.38	-.28	-.83**	-.54	.57	-.14
	Sig. (1-tailed)	.28	.16	.26	.33	.01	.17	.16	.39
PASP	Pearson Correlation	.156	.072	.063	.337	.684*	1	-.277	.242
	Sig. (1-tailed)	.384	.446	.453	.257	.045		.326	.301
PADP	Pearson Correlation	.099	.478	.540	-.798	-.137	-.277	1	.278
	Sig. (1-tailed)	.426	.208	.174	.053	.385	.326		.297

P values <0.05 are significant



IV. Discussion

As seen in the table below in the HIV group as compared to the controls. The mean RAA /M ± SD 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 RVEDA/M ± SD was 8.54 ± 4.9 cm^2/m in the HIV group but 8.51 ± 1.9 cm^2/m in the controls with no significant differences. 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 and the RVCO at 8.72 ± 16.7 l/min and 5.90 ± 1.8 l/min the RVDT 168.57± 64.1ms and 168.57 ± 64.1ms for HIV positive patients and controls respectively showed significant differences.

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 higher Pulmonary artery systolic and pulmonary artery diastolic pressures, when compared with the normal controls. However, the RV Ejection fraction, RV E/A ratio, RVDECT, the cardiac output at the pulmonary did not show any statistically significant difference when compared using the P-value. Nevertheless, RV cardiac output was relatively higher in the HIV cases while tricuspid E deceleration time for both ventricles were lower. The cardiac chambers were relatively smaller in the HIV group as compared to the controls.

Pulmonary hypertension is an important contribution of this study. Very few studies have evaluated pulmonary hypertension in HIV, most emphasis has been on HIV associated idiopathic pulmonary hypertension which is largely a diagnosis of exclusion. One of the problems associated with HIV is chronic chest infection which is a cause of secondary pulmonary hypertension. In Nigeria the commonest opportunistic infection of the HIV virus is tuberculosis. Pellicelli et al **[16,17]** Saidi and Bricker **[18]** noted elevated pulmonary artery systolic blood pressure when compared with normal controls. This study corroborated this as pulmonary artery systolic and diastolic were significantly higher than in the controls. There were no significant differences when compared amongst both sexes in the HIV group. The pulmonary trunks and chambers in the affected cases were dilated with marked PR and TR.

The estimated PASP showed positive corelation(1 tailed) with the tricuspid E/A(.68,.05), while the estimated PADP (-.80,.05),showed negative correlation with right ventricular ejection fraction. The negative corelation of the PADP with ejection fraction can be explained by the dilated cardiac chambers with consequent depressed ejection from impaired systolic function. Pulmonary hypertension has been known to complicate heart failure.[26] The corelation between PASP with diastolic function can exist with restrictive filling in heart failure but in addition, the role of endocardial fibrosis should also be considered as endocardial fibrosis has been described by other studies.[27-28].

V. Conclusion:

The prevalence of pulmonary hypertension was 7.5% in the study population. TR was associated with dilated right atrial and right ventricle. The estimated PASP showed positive corelation(1 tailed) with the tricuspid E/A(.68,.05) while the estimated PADP (-.80,.05) showed negative correlation with right ventricular ejection fraction. Heart failure and endocardial fibrosis should be considered as possible explanations for these findings.

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