UKR Journal of Medicine and Medical Research (UKRJMMR)
Homepage: https://ukrpublisher.com/ukrjmmr/
Email: submit.ukrpublisher@gmail.com
Volume 2, Issue 2, March-April 2026
ISSN: 3107-5061 (Online)

Right-Heart Echocardiographic Determinants of Estimated Pulmonary Artery Pressure in HAART-Naïve Adults Living With HIV: A Case–Control Study in Port Harcourt, Nigeria

Cookey S. N[1*]; Briggs F. K[2]; Amachree E.[3]

[1]Consultant Cardiologist, Department of Internal Medicine, Rivers State University/ Rivers State University Teaching Hospital, Port Harcourt, Rivers State, Nigeria.

[2]Consultant Cardiologist (Cardiology), Department of Internal Medicine, Rivers State University/Rivers State University Teaching Hospital, Port Harcourt, Rivers State, Nigeria.

[3]Senior Registrar, Department of Internal Medicine, Rivers State University/Rivers State University Teaching Hospital, Port Harcourt, Rivers State, Nigeria.

***Corresponding Author**: Cookey S. N.

DOI: *https://doi.org/10.5281/zenodo.18873676*

Article History

Original Research Article

Received: 15-02-2026

Accepted: 03-03-2026

Published: 05-03-2026

Citation: Cookey S. N; Briggs F. K; Amachree E. (2026). Right-Heart Echocardiographic Determinants of Estimated Pulmonary Artery Pressure in HAART-Naïve Adults Living With HIV: A Case–Control Study in Port Harcourt, Nigeria. UKR Journal of Medicine and Medical Research (UKRJMMR), Volume 2(2), 01-10.

Abstract

Background:

Echocardiography is a practical, non-invasive tool for estimating pulmonary artery pressures and assessing right-heart structure and function. Pulmonary hypertension (PH) occurs more frequently among people living with HIV than in the general population, yet data from African populations remain limited. The development of HIV-associated PH is likely multifactorial, involving recurrent respiratory infections, thromboembolic disease, chronic inflammation, and intrinsic right-heart pathology. In view of these considerations, this study aimed to determine the prevalence of echocardiographically defined pulmonary hypertension among HAART-naïve adults living with HIV and to evaluate the relationship between estimated pulmonary artery pressures and right-heart structural and functional parameters

Methods

This prospective, descriptive cross-sectional case–control study was performed at the University of Port Harcourt Teaching Hospital from July 2011 to July 2014. The trial comprised two hundred HAART-naïve HIV-positive persons and one hundred HIV-negative controls, all matched for age and sex, and devoid of hypertension, diabetes, or known cardiac illness. Everyone who took part got an echocardiogram. The modified Bernoulli equation was used to figure out the pulmonary artery systolic pressure (PASP). We used SPSS to look at the data, and we set the significance level at $p<0.05$.

Results:

Among 200 HAART-naïve HIV participants (mean age 33.13 ± 8.4 years), pulmonary regurgitation was more frequent than tricuspid regurgitation (35% vs 27.5%), both occurring more often than in controls. Estimated PASP and PADP were higher in the HIV group (19.31 ± 10.3 vs 13.60 ± 5.3 mmHg; 17.92 ± 8.1 vs 14.41 ± 6.0 mmHg). Pulmonary hypertension was present in 7.5%. Tricuspid regurgitation was associated with right atrial and ventricular dilatation. PASP correlated positively with tricuspid E/A ($r = 0.68$), while PADP correlated inversely with right ventricular ejection fraction ($r = -0.80$).

Conclusion:

HAART-naïve adults with HIV demonstrated higher pulmonary artery pressures and a 7.5% prevalence of pulmonary hypertension. Right-heart remodeling and the inverse PADP–RVEF relationship suggest early right-sided cardiac involvement in untreated HIV.

Keywords: *HIV, HAART-naïve, pulmonary hypertension, echocardiography, right ventricle.*

Introduction

Pulmonary hypertension in HIV infection has been reported at rates substantially above those observed in the general population, with a rate of 1 in 200.[1] It presents in different disease conditions, even in the absence of thromboembolic disease, intravenous drug exposure, pulmonary infections, or right-sided endocarditis. While idiopathic (HIV-associated) pulmonary arterial hypertension is recognized, secondary contributors including chronic chest infections and thromboembolism are particularly relevant in settings where opportunistic infections such as tuberculosis are common. Pellicelli et al demonstrated a significant disparity in pulmonary hypertension severity between HIV-infected individuals with AIDS and those without advanced disease, noting that patients with AIDS exhibited markedly higher pulmonary artery systolic pressures.[2-4] Similarly, Saidi and Bricker also highlighted a clear relationship between HIV infection and the development of pulmonary hypertension, strengthening the role of HIV as an important contributing factor in pulmonary vascular disease.[5]

Though the mechanisms responsible for HIV-associated PH remain incompletely defined, there exists many postulations.[6-9] Direct viral invasion of pulmonary vascular endothelium has not been consistently demonstrated; however, indirect pathways involving inflammatory mediators and cytokines (including IL-1, IL-6, and TNF-α) may drive endothelial dysfunction, vasoconstriction, and smooth muscle proliferation. Genetic susceptibility may also contribute a significant role, given that only a minority of HIV-infected individuals develop clinically significant PH. Morse and colleagues[9] reported a significantly higher prevalence of HLA-DR6 and HLA-DR52 among individuals living with HIV who developed pulmonary hypertension compared with both non-HIV controls and HIV-positive patients without pulmonary hypertension thus strengthening the genetic predisposition.

A large pooled review further identified progressive exertional dyspnoea as the predominant presenting complaint. Additional symptoms commonly observed included pedal oedema, dry cough, fatigue, syncope or presyncope, and chest discomfort. On clinical examination, patients typically demonstrate classical features of pulmonary hypertension such as elevated jugular venous pressure, an accentuated pulmonary component of the second heart sound (loud P2), a right-sided third heart sound, murmurs of tricuspid and pulmonary regurgitation, and varying degrees of peripheral oedema.[4]

Echocardiography is widely used to identify right-heart changes suggestive of elevated pulmonary pressures, including right atrial and right ventricular enlargement, pulmonary artery dilatation, interventricular septal shift, and Doppler evidence of regurgitant jets. Using Doppler-based estimates, principally TR velocity for PASP and PR velocity for PADP, echocardiography enables feasible screening and correlation analyses in resource-constrained environments. This study therefore assessed estimated pulmonary artery pressures and right-heart parameters in HAART-naïve adults with HIV in Port Harcourt, Nigeria. Morse and colleagues reported a significantly higher prevalence of HLA-DR6 and HLA-DR52 among individuals living with HIV who developed pulmonary hypertension compared with both non-HIV controls and HIV-positive patients without pulmonary hypertension.[1]

There are several echocardiographic methods that can be used to measure the pressure in the pulmonary arteries. In the absence of restriction in the right ventricular outflow tract and pulmonary artery stenosis, the right ventricular systolic pressure roughly approximates the pulmonary artery systolic pressure. Doppler echocardiography estimates systolic pulmonary artery pressure by measuring the peak velocity of tricuspid regurgitation. It may also use shunt-related velocities from a ventricular septal defect or patent ductus arteriosus, as well as indices derived from right ventricular isovolumetric time intervals. You can figure out the pulmonary artery diastolic pressure by looking at the speed of the pulmonary regurgitant jet, and you can figure out the mean pulmonary artery pressure by looking at the pulmonary Doppler acceleration time. Up to 70% of people with pulmonary systolic hypertension also have tricuspid and pulmonary regurgitation. This makes these Doppler-derived measures very valuable. A tricuspid regurgitation peak velocity beyond 2.9 m/s is typically considered indicative of increased pulmonary artery systolic pressure.[10-13]

Ryan and his coworkers first came up with the eccentricity index, which gives a number to how much the interventricular septum moves when there is too much pressure or volume in the right ventricle in pulmonary hypertension. An improper systolic eccentricity index indicates elevated pulmonary artery systolic pressure, while an abnormal diastolic eccentricity index correlates with increased right ventricular diastolic pressure and has been associated with adverse clinical outcomes and the progression of right ventricular failure.

Management of secondary pulmonary hypertension in individuals with HIV primarily focuses on addressing the underlying pathology alongside targeted pulmonary vasodilator therapy. Therapeutic options include endothelin receptor antagonists, calcium channel blockers, and phosphodiesterase inhibitors, among others. However, a definitive, standardized treatment strategy for HIV-associated pulmonary hypertension has yet to be established. Many patients, therefore, receive supportive

care directed at right heart failure, including oxygen therapy and diuretic use.

Although oral anticoagulation has demonstrated survival benefit in idiopathic pulmonary arterial hypertension, similar advantages have not been consistently observed in HIV-associated disease. The contribution of antiretroviral therapy remains uncertain; while some studies suggest potential reductions in right-sided pressures, others report minimal clinical improvement and, in certain cases, progression of symptoms despite treatment.[15-20]

Methods

Study Design and Setting

A prospective, descriptive cross-sectional case–control study was carried out at the University of Port Harcourt Teaching Hospital, a major referral center serving Rivers State, Bayelsa State and surrounding regions.

Sample Size

A Kish-based sample size approach was described in the protocol, with adjustment for the study population size; the final recruited sample comprised 200 HIV cases and 100 controls.[21]

Ethical Approval and Consent

The Hospital 's ethic committee provided the ethical approval. All participants provided informed consent, and standard research governance procedures were maintained throughout.

Participants

Cases (HIV Group)

Two hundred adults (≥18 years) with newly diagnosed HIV infection who were antiretroviral-naïve were randomly selected using a table of random numbers. Selection was performed without prior knowledge of CD4 count to limit allocation bias.

Inclusion criteria (cases):

Confirmed HIV infection
HAART-naïve
Age ≥18 years
Consent provided

Exclusion criteria (cases):

Hypertension
Diabetes mellitus
Significant alcohol intake (operationalized in the protocol as ≥30 g/day)
Cigarette smoking history
Poor echocardiographic acoustic window

Controls

One hundred HIV-negative adults with no history of cardiac disease and without hypertension or diabetes were recruited and matched to cases by age and sex.

Clinical and Laboratory Assessment

Demographic characteristics, symptom history, physical examination, and blood pressure were recorded. Laboratory tests included packed cell volume (PCV), fasting blood glucose, and CD4 count. HIV diagnosis was confirmed using a double-ELISA rapid testing algorithm as practiced at the study site. CD4 count was assayed using the Apogee A50 micro flow cytometer.[22-23]

Echocardiographic Assessment

After explaining the technique to both patients and control subjects, all participants had transthoracic echocardiography done with the Aloka ProSound SSD-4000 machine. The individuals were placed in a steep left lateral decubitus position with the left arm raised above the head to improve acoustic windows for the tests.

Comprehensive imaging encompassed M-mode, two-dimensional, and Doppler techniques (pulsed, continuous-wave, and colour flow). The American Society of Echocardiography suggested that planimetric chamber area measures be used more than linear internal diameters to measure chamber size.

A standardized acquisition protocol was implemented for all variables. Two representative cardiac cycles were analyzed for each parameter, and the averaged values were recorded. Echocardiographic evaluation was performed prior to knowledge of CD4 counts to minimize observer bias. Random studies were independently reviewed by a supervising cardiologist for quality assurance. The cost of investigations was covered through an institutional waiver and project funding obtained from the University of Port Harcourt Teaching Hospital.

Imaging Protocol

1. Right Ventricular End-Diastolic Area (RVEDA):

   RVEDA was obtained from the apical four-chamber view by tracing the right ventricular endocardial border and the tricuspid annular plane at end-diastole. To account for body size variability, the measured area was indexed to patient height.[24-25]

2. Right Ventricular Systolic Performance:

   Systolic function was estimated using the right ventricular fractional area change. End-systolic (RVESA) and end-diastolic areas were determined as described above, and fractional area change was

calculated as: Fractional area change (%) = 100 × (RVEDA − RVESA) / RVEDA.[24-25]

3. Right Ventricular Diastolic Filling:

   Tricuspid inflow velocities were recorded by placing the Doppler sample volume at the leaflet tips. Peak E and A wave velocities, their ratio, and E-wave deceleration time were measured in subjects with sinus rhythm.

4. Right Atrial Area:

   The right atrium was assessed by planimetry in the apical four-chamber view at end-systole and subsequently indexed to height.

5. Pericardial Effusion:

   Effusion was defined as a clear diastolic separation of pericardial layers, particularly visible posterior to the heart in parasternal long- and short-axis views.

6. Regurgitant Jet Velocities:

   Continuous-wave Doppler was used to determine the peak instantaneous velocities of tricuspid and pulmonary regurgitation.

7. Estimation of Pulmonary Artery Pressures:[26-27]

   Pulmonary pressures were derived using the modified Bernoulli equation.

   Pulmonary artery systolic pressure (PASP) was calculated as:

   PASP = $4(VTR)^2$ + RAP, where *VTR* represents the peak tricuspid regurgitant velocity and *RAP* denotes right atrial pressure.

   In the absence of intracardiac shunts or pulmonary outflow obstruction, right ventricular systolic pressure was considered equivalent to pulmonary artery systolic pressure.

   Pulmonary artery diastolic pressure (PADP) was estimated using:

   PADP = $4(VPR)^2$ + RAP, where *VPR* is the peak pulmonary regurgitant velocity.

   Pulmonary hypertension was defined by **PASP greater than 30 mmHg** and **pulmonary artery diastolic pressure exceeding 19 mmHg**.

   RAP was estimated from IVC size and inspiratory collapsibility:

Table 1. RAP estimation by IVC assessment

IVC Size and Respiratory Variation	Estimated RAP (mmHg)
Small IVC (< 1.5 cm) with collapse	0 – 5
Normal IVC (1.5 – 2.5 cm) with > 50% inspiratory collapse	5 – 10
Normal IVC (1.5 – 2.5 cm) with < 50% inspiratory collapse	10 – 15
Dilated IVC (> 2.5 cm) with < 50% inspiratory collapse	15 – 20
Dilated IVC with hepatic vein dilatation and no respiratory variation	> 20

Operational Definitions

Depressed RV ejection fraction: **<40%** [28]

RV diastolic dysfunction: defined relative to internally generated reference mean tricuspid E/A (1.27±0.4)

Pulmonary hypertension: **PASP >30 mmHg and PADP >19 mmHg** (per the protocol's cited threshold)

Statistical Analysis: SPSS was used for analysis. Continuous variables were summarized as mean ± SD and categorical variables as proportions. Group comparisons used t-tests for means and chi-square for categorical variables. Correlation analyses employed Pearson correlation coefficients. Statistical significance was set at $p<0.05$.

Results: Participant Characteristics: The HIV group (n=200) comprised 76 males (38%) and 124 females (62%) with mean age 33.13±8.4 years (range 18–56). Controls (n=100) comprised 36 males (36%) and 64 females (64%) with mean age 31.82±8.72 years. BMI was significantly lower in the HIV group, while mean blood pressures were similar between groups. Pulse rate was higher in HIV participants.

Clinical Profile of HIV Participants: Fifteen (7.5%) cases were inpatients and 185 (92.5%) were outpatients from the retroviral clinic. Cardiovascular symptoms were reported in 56 (28%), and 12 of these were in heart failure. Frequently documented symptoms/signs included fever, weight loss, lethargy, cough, dyspnea, pedal swelling, displaced apex beat, S3, loud P2, and functional murmurs.

Echocardiographic Right-Heart Abnormalities: PR occurred more frequently than TR in the HIV group (PR 35% vs TR 27.5%). Both regurgitant lesions were significantly more prevalent in HIV cases than controls. Pulmonary hypertension was identified in **15 (7.5%)** of HIV cases and was significantly more common than in controls.

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 (years)	33.13 ± 8.4	32.83 ± 8.72	0.11	0.91
Age Range (years)	18 – 53	19 – 50	—	—
BMI (kg/m²)	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/µL)	**234.50 ± 154.36**	—	—	—

* $p < 0.05$ indicates statistical significance.

Table 3. Mean Variables of HIV Cohort

Variables	Mean ± SD
Age (years)	34.81 ± 8.64
BMI (kg/m²)	21.98 ± 4.18
Heart rate (beats/min)	98.33 ± 15.82
SBP (mmHg)	127.75 ± 25.98
DBP (mmHg)	70.00 ± 5.35
PCV (%)	31.67 ± 5.22
CD4 count (cells/µL)	234.50 ± 154.36
RAA	12.93 ± 3.82
RVEDA	17.52 ± 10.97
RVESA	9.74 ± 3.95
Ejection fraction (%)	46.66 ± 13.38
Tricuspid E/A	1.27 ± 0.40
PADP (mmHg)	17.92 ± 8.1
PASP (mmHg)	19.31 ± 10.3
IVC diameter (mm)	22.92 ± 7.56

Table 4: Comparison of Cardiac Abnormalities and Echocardiographic Parameters between HIV Subjects and Controls

Variable	HIV Positive	Controls	t-test / Statistic	p-value
Cardiac Abnormalities (N, %)				
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*
Combined 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*
Echocardiographic Parameters (Mean ± SD)				
RAA/M (cm²/m)	7.85 ± 2.29	8.44 ± 1.5	-1.401	0.162

Variable	HIV Positive	Controls	t-test / Statistic	p-value
RVEDA/M (cm²/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
RV E/A ratio	1.27 ± 0.4	1.37 ± 0.5	-1.164	0.246
RV deceleration time (msec)	168.57 ± 64.1	168.57 ± 64.1	-1.895	0.358
RV cardiac output (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*

Abbreviations: RAA/M — right atrial area indexed to height; RVEDA/M — right ventricular end-diastolic area indexed to height; RVEF — right ventricular ejection fraction; RV E/A — tricuspid inflow ratio; RV deceleration time — right ventricular deceleration time; RVCO — right ventricular cardiac output; PASP — pulmonary artery systolic pressure; PADP — pulmonary artery diastolic pressure.

Table 5. Correlation of Right-Heart Parameters with PASP and PADP in HIV Cohort

Parameter	CD4	RAA	RVEDA	RVESA	RVEF	Tricuspid E/A	PADP	PASP	IVC
PASP (r)	0.57	0.16	0.07	0.06	0.34	0.68*	1.00	—	0.24
p-value	0.17	0.38	0.45	0.45	0.26	0.05	—	—	0.30
PADP (r)	0.28	0.10	0.48	0.54	−0.80*	−0.14	—	−0.27	0.28
p-value	0.30	0.43	0.21	0.17	0.05	0.39	—	0.33	0.30
BMI (r)	0.44*	−0.29	−0.25	−0.44	0.05	−0.58*	−0.67*	−0.58*	−0.14
p-value	0.05	0.17	0.23	0.09	0.44	0.02	0.01	0.02	0.32
HR (r)	1.00**	−0.03	0.73	0.01*	−0.99*	0.68	1.00**	1.00**	−0.51
p-value	0.00	0.49	0.24	0.02	0.05	0.26	0.00	0.00	0.33
SBP (r)	−0.17	0.43	0.99	0.57	−0.79	−0.70	−0.60	−0.30	0.28
p-value	0.36	0.24	0.05	0.31	0.21	0.06	0.10	0.35	0.30
DBP (r)	−0.07	−0.37	−0.35	0.42	−0.14	0.62	0.60	0.19	0.27
p-value	0.44	0.27	0.39	0.36	0.46	0.09	0.10	0.40	0.30
PCV (r)	0.21	0.77**	0.15	0.15	−0.11	−0.42	−0.28	−0.21	−0.10
p-value	0.24	0.00	0.35	0.35	0.39	0.10	0.23	—	—

* $p < 0.05$ indicates statistical significance.

Table 6. Correlation of Right-Heart Parameters with PASP and PADP in Patients with Pulmonary Hypertension

Variable	RAA	RVEDA	RVESA	RVEF	Tricuspid E/A	PASP	PADP	IVC
PCV (r)	**0.82***	0.15	0.35	−0.12	**−0.73***	−0.60	−0.17	0.08
p value	0.01	0.39	0.25	0.41	0.03	0.14	0.39	0.44
CD4 (r)	−0.30	−0.58	−0.38	−0.28	**−0.83**	−0.54	0.57	−0.14
p value	0.28	0.16	0.26	0.33	**0.01**	0.17	0.16	0.39
PASP (r)	0.156	0.072	0.063	0.337	**0.684***	1	−0.277	0.242
p value	0.384	0.446	0.453	0.257	**0.045**	—	0.326	0.301
PADP (r)	0.099	0.478	0.540	−0.798	−0.137	−0.277	1	0.278
p value	0.426	0.208	0.174	0.053	0.385	0.326	—	0.297

Discussion

The index study demonstrates that HAART-naïve adults living with HIV in Port Harcourt exhibit higher echocardiographically estimated pulmonary artery pressures compared with age and sex matched HIV-negative controls. Although the mean pulmonary pressures in the overall cohort remained below diagnostic thresholds at the group level, the finding that 7.5% of participants fulfilled criteria for pulmonary hypertension is clinically significant. This prevalence is particularly relevant in a population with a substantial burden of respiratory infections and other contributors to pulmonary vascular disease, where secondary mechanisms may coexist with HIV-associated vascular dysfunction.

In addition to the observed prevalence of pulmonary hypertension, estimated pulmonary artery systolic and diastolic pressures were significantly elevated in the HIV cohort relative to controls. These differences reached statistical significance and are consistent with prior reports demonstrating higher pulmonary pressures among individuals living with HIV. Pellicelli and colleagues documented similar elevations in pulmonary artery systolic pressure in HIV-infected patients, particularly those with more advanced disease,[16,17] and Saidi and Bricker[5] likewise described an increased frequency of pulmonary hypertension in this population. Although idiopathic pulmonary arterial hypertension remains rare, HIV-related pulmonary vascular involvement is thought to arise through a multifactorial process that includes chronic pulmonary infections, thromboembolic phenomena, inflammatory endothelial activation, and cytokine-mediated smooth muscle proliferation. In sub-Saharan African settings, the contribution of recurrent respiratory infections may be especially important.

Despite these differences in pulmonary pressures, several indexed right-heart structural parameters did not differ significantly between groups. The mean right atrial area indexed for height was 7.85 ± 2.2 cm²/m in the HIV group compared with 8.44 ± 1.5 cm²/m in controls, and the mean right ventricular end-diastolic area indexed for height was 8.54 ± 4.9 cm²/m versus 8.51 ± 1.9 cm²/m, respectively, with no statistically significant differences. Similarly, right ventricular ejection fraction, tricuspid E/A ratio, right ventricular deceleration time, and right ventricular cardiac output did not demonstrate significant intergroup differences. Nevertheless, qualitative patterns within the HIV cohort were noteworthy. Pulmonary regurgitation was more frequent than tricuspid regurgitation among HIV participants, and when tricuspid regurgitation was present, it was associated with enlargement of the right atrium and right ventricle, consistent with chronic pressure or volume loading. The higher frequency of pulmonary regurgitation may reflect pulmonary arterial or vascular involvement that is detectable through Doppler interrogation even when tricuspid regurgitation signals are absent or technically suboptimal.

Correlation analyses further clarified the relationship between pulmonary pressures and right ventricular function. Estimated PASP demonstrated a positive correlation with the tricuspid inflow E/A ratio ($r = 0.68$, $p = 0.05$; one-tailed), suggesting that rising systolic pulmonary pressures may influence right ventricular filling dynamics. This relationship implies an interaction between afterload and diastolic function, whereby altered compliance or loading conditions modify inflow patterns. In contrast, estimated PADP showed a strong inverse correlation with right ventricular ejection fraction ($r = -0.80$, $p = 0.05$; one-tailed). This negative association provides a physiologically coherent explanation: sustained elevation of pulmonary diastolic pressure increases right ventricular afterload, promotes chamber dilatation, and ultimately impairs systolic performance. The resultant reduction in ejection fraction reflects progressive ventricular compromise under chronic pressure stress. Similar interactions between pulmonary pressures and ventricular dysfunction have been described in heart failure populations, where pulmonary hypertension frequently complicates ventricular impairment.

In the context of HIV infection, inflammatory activation and myocardial remodeling may further compound hemodynamic stress. Elevated circulating cytokines, endothelial dysfunction, and diffuse myocardial fibrosis have been reported in HIV-infected individuals, potentially reducing myocardial compliance and contractility. Such structural and functional alterations may amplify the adverse impact of pulmonary vascular loading, thereby accelerating right ventricular dysfunction beyond what would be expected from pressure overload alone. Observational studies examining myocardial steatosis and fibrosis in HIV-infected adults lend support to this broader inflammatory–fibrotic paradigm.[14,29-33]

The correlation analysis in patients with pulmonary hypertension demonstrates meaningful interactions between right-heart structural parameters, hematologic status, immune function, and pulmonary artery pressures. A particularly notable finding was the strong positive correlation between packed cell volume and right atrial area ($r = 0.82$, $p = 0.01$), suggesting that increased hematocrit may contribute to right atrial enlargement in this subgroup. This relationship may reflect chronic hypoxic adaptation and increased blood viscosity, both of which are recognized contributors to pulmonary vascular resistance and right-heart loading. Similar associations have been described in pulmonary hypertension populations where secondary

erythrocytosis contributes to increased right-sided pressures and chamber remodeling.[14,34]

Packed cell volume also demonstrated a significant negative correlation with tricuspid E/A ratio ($r = -0.73$, $p = 0.03$), indicating potential impairment in right ventricular diastolic filling with increasing hematocrit levels. This finding aligns with hemodynamic models in which elevated viscosity and pulmonary vascular resistance reduce ventricular compliance and alter filling dynamics.[35-36]

CD4 count showed a strong negative correlation with tricuspid E/A ratio ($r = -0.83$, $p = 0.01$), suggesting worsening right ventricular diastolic function with progressive immunosuppression. This observation supports the hypothesis that advanced HIV disease contributes to myocardial involvement through inflammatory and fibrotic mechanisms rather than solely through pressure overload. Previous studies have reported diastolic dysfunction in HIV populations independent of overt pulmonary hypertension, reinforcing the role of chronic immune activation and myocardial fibrosis.[30,37]

The relationship between PASP and tricuspid E/A ratio ($r = 0.684$, $p = 0.045$) highlights the interaction between elevated pulmonary systolic pressure and right ventricular filling characteristics. As pulmonary pressure increases, alterations in ventricular compliance and preload conditions may shift diastolic filling patterns. Comparable findings have been reported in pulmonary arterial hypertension cohorts, where diastolic abnormalities correlate with rising pulmonary pressures and predict functional decline.[35,38]

Although not reaching statistical significance, the strong inverse relationship between PADP and right ventricular ejection fraction ($r = -0.798$, $p = 0.053$) is physiologically important. Elevated pulmonary diastolic pressure increases right ventricular afterload, promoting ventricular dilatation and eventual systolic impairment. This trend is consistent with broader pulmonary hypertension literature demonstrating that sustained diastolic pressure elevation is a major determinant of right ventricular failure. Moderate positive relationships between PADP and right ventricular chamber dimensions suggest progressive structural remodeling with rising pulmonary diastolic pressure. Enlargement of right ventricular end-diastolic and end-systolic areas in the presence of elevated pulmonary pressure reflects chronic pressure overload and ventricular adaptation. The absence of strong correlations between inferior vena cava diameter and pulmonary pressures may reflect the multifactorial determinants of right atrial pressure in HIV patients, including intravascular volume status, autonomic dysfunction, and respiratory mechanics.[35,38]

Overall, these findings emphasize that pulmonary hypertension in HIV patients is not solely a vascular phenomenon but is closely linked to myocardial function, hematologic adaptation, and immune status. The interplay between CD4 depletion, right ventricular diastolic impairment, and pulmonary pressure elevation suggests a complex pathophysiologic continuum involving inflammatory myocardial injury and hemodynamic stress. This reinforces the importance of comprehensive right-heart echocardiographic assessment in HIV patients with suspected pulmonary hypertension, particularly in those with advanced immunosuppression.

Conclusion

Echocardiography remains a valuable non-invasive and practical modality for cardiovascular evaluation in individuals living with HIV, particularly in resource-limited settings. Early assessment prior to the initiation of antiretroviral therapy provides an opportunity to detect elevated pulmonary pressures, right-heart structural remodeling, and subclinical functional impairment that may otherwise remain unrecognized. Identification of these abnormalities enables improved clinical surveillance, more accurate risk stratification, and timely therapeutic intervention, thereby potentially mitigating progression to overt right ventricular dysfunction and advanced cardiopulmonary disease.

In this cohort of HAART-naïve adults in Port Harcourt, pulmonary hypertension was present in 7.5% of participants, with significantly higher estimated pulmonary artery systolic and diastolic pressures observed compared with matched controls. Tricuspid regurgitation was associated with enlargement of right-sided cardiac chambers, while pulmonary artery systolic pressure demonstrated a positive relationship with tricuspid inflow E/A ratio and pulmonary artery diastolic pressure showed an inverse association with right ventricular ejection fraction. Collectively, these findings highlight early right-heart involvement in untreated HIV infection and support the integration of focused right-heart echocardiographic assessment into baseline cardiovascular evaluation for this population.

References

1. Fauci AS, Lane HC. Human immunodeficiency virus disease, AIDS and related disorders. In: Harrison's Principles of Internal Medicine. 17th ed. New York: McGraw-Hill; 2008. p. 1076-1139.
2. Shavadia J, Das S, Yonga G. HIV-associated pulmonary hypertension: case report. East Afr Med J. 2008;85:564-567.

3. Pellicelli AM, Palmieri F, D'Ambrosio C, et al. Role of human immunodeficiency virus in primary pulmonary hypertension: case reports. Angiology. 1998;49:1005-1011.

4. Pellicelli A, Barbaro G, Palmieri F, et al. Primary pulmonary hypertension in HIV disease: a systematic review. Angiology. 2001;52:31-41.

5. Saidi A, Bricker JT. Pulmonary hypertension in patients infected with HIV. In: Cardiology in AIDS. New York: Chapman and Hall; 1998. p. 187-194.

6. Rich S, Panzer DR, Ayres SM, et al. Primary pulmonary hypertension: a national prospective study. Ann Intern Med. 1987;107:216-223.

7. Fuster V, Steele PM, Edwards WD, et al. Primary pulmonary hypertension: natural history and the importance of thrombosis. Circulation. 1984;70:580-587.

8. D'Alonzo GE, Barst RJ, Ayres SM, et al. Survival in patients with primary pulmonary hypertension: results from a national prospective registry. Ann Intern Med. 1991;115:343-349.

9. Morse JH, Barst RJ, Fotino M. Familial pulmonary hypertension: immunogenetic findings in four Caucasian kindreds. Am Rev Respir Dis. 1992;145:787-792.

10. Nasidi A, Harry TO. The epidemiology of HIV/AIDS in Nigeria. In: Adeyi O, ed. AIDS in Nigeria: A nation on the threshold. Cambridge: Harvard Center for Population and Development Studies; 2006. p. 17-36.

11. Jaffe HW, Francis DP, McLane MF, et al. Transfusion-associated AIDS: serologic evidence of human T-cell leukemia virus infection of donors. Science. 1984;223:1309-1312.

12. Rich S, Brundage BH. High-dose calcium channel-blocking therapy for primary pulmonary hypertension. Circulation. 1987;76:135-141.

13. Park MH. Advances in diagnosis and treatment in patients with pulmonary arterial hypertension. Catheter Cardiovasc Interv. 2008;71:205-213.

14. Ryan T, Petrovic O, Dillon JC, Feigenbaum H, et al. An echocardiographic index for separation of right ventricular volume and pressure overload. J Am Coll Cardiol. 1985;5:918-924.

15. Simonneau G, Robbins IM, Beghetti M, et al. Updated clinical classification of pulmonary hypertension. J Am Coll Cardiol. 2009;54:S43-S54.

16. Pellicelli AM, Barbaro G, Palmieri F. Primary pulmonary hypertension in HIV patients: a systematic review. Angiology. 2001;52:31-41.

17. D'Alonzo GE, Barst RJ, Ayres SM, et al. Survival in patients with primary pulmonary hypertension: results from a national registry. Ann Intern Med. 1991;115:343-349.

18. Hinderliter AL, Willis PW, Barst RJ, et al. Effects of long-term infusion of prostacyclin on echocardiographic measures of right ventricular structure and function. Circulation. 1997;95:1479-1486.

19. Nunes H, Humbert M, Sitbon O, et al. Prognostic factors for survival in HIV-associated pulmonary arterial hypertension. Am J Respir Crit Care Med. 2003;167:1433-1439.

20. Rich S, Brundage BH. High-dose calcium channel-blocking therapy for primary pulmonary hypertension. Circulation. 1987;76:135-141.

21. Kish L. Survey Sampling. New York: John Wiley & Sons; 1965. p. 17-21.

22. UNAIDS. UNAIDS report on the global AIDS epidemic. Geneva: UNAIDS; 2010.

23. Lubega S, Zirembuzi GW, Lwabi P. Heart disease among children with HIV/AIDS attending the paediatric infectious disease clinic at Mulago Hospital. Afr Health Sci. 2005;5:219-226.

24. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1-39.

25. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults. J Am Soc Echocardiogr. 2010;23(7):685-713..

26. Abbas AE, Fortuin FD, Schiller NB, Appleton CP, Moreno CA, Lester SJ. A simple method for noninvasive estimation of pulmonary vascular resistance using Doppler echocardiography. J Am Coll Cardiol. 2003;41(6):1021-1027.

27. Otto CM. Textbook of clinical echocardiography. 5th ed. Philadelphia: Elsevier; 2017.

28. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al.

Guidelines for the echocardiographic assessment of the right heart in adults. J Am Soc Echocardiogr. 2010;23(7):685-713.

29. Hsue PY, Tawakol A. Inflammation and fibrosis in HIV: getting to the heart of the matter. Circ Cardiovasc Imaging. 2016;9(3):e004427.

30. Thiara DK, Liu CY, Raman F, Mangat S, Purdy JB, Duarte HA, et al. Abnormal myocardial function is related to myocardial steatosis and diffuse myocardial fibrosis in HIV-infected adults. J Infect Dis. 2015;212(10):1544-1551.

31. Ntusi N, O'Dwyer E, Dorrell L, Wainwright E, Piechnik SK, Clutton G, et al. HIV-1-related cardiovascular disease is associated with chronic inflammation, frequent pericardial effusions, and myocardial fibrosis. J Am Coll Cardiol. 2016;68(5):455-463.

32. Enriquez-Sarano M, Rossi A, Seward JB, Bailey KR, Tajik AJ. Determinants of pulmonary hypertension in left ventricular dysfunction. *J Am Coll Cardiol*. 1997;29(1):153-159.

33. Marra AM, Benjamin N, Cittadini A, Bossone E, Grünig E. When pulmonary hypertension complicates heart failure. *Heart Fail Clin*. 2020;16(1):53-60.

34. Voelkel NF, Quaife RA, Leinwand LA, Barst RJ, McGoon MD, Meldrum DR, et al. Right ventricular function and failure: report of a National Heart, Lung, and Blood Institute working group. *Circulation*. 2006;114(17):1883-1891.

35. Haddad F, Doyle R, Murphy DJ, Hunt SA. Right ventricular function in cardiovascular disease, part II: pathophysiology, clinical importance, and management of right ventricular failure. *Circulation*. 2008;117(13):1717-1731.

36. Simonneau G, Montani D, Celermajer DS, Denton CP, Gatzoulis MA, Krowka M, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. *Eur Respir J*. 2019;53(1):1801913.

37. Hsue PY, Hunt PW, Ho JE, Farah HH, Schnell A, Hoh R, et al. Impact of HIV infection on diastolic function and left ventricular mass. *Circ Heart Fail*. 2010;3(1):132-139.

38. Barbaro G. Cardiovascular manifestations of HIV infection. *Circulation*. 2002;106(11):1420-1425.