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Original Research Article

Exploring the Association Between Echocardiographic Parameters and Routine Laboratory Parameters of Heart Failure Patients in Abakaliki, Ebonyi State, Nigeria

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Abstract
Echocardiography is a crucial tool in the diagnosis of heart failure. At the same time, routine laboratory parameter results reflect changes associated with heart failure. This study investigated the association between routine laboratory parameters and echocardiographic parameters in heart failure patients in Abakaliki, Nigeria. Two hundred and fifty patients diagnosed with heart failure who underwent concurrent clinical evaluation, laboratory testing, and transthoracic echocardiography were enrolled in this study. The data generated were analysed using Python 3.12 and associated libraries to determine Spearman's correlation, multivariate linear regression, and Receiver Operating Characteristic (ROC) analysis. Cystatin C correlated positively with LVEF ($\rho = 0.271$) and negatively with LVEDD ($\rho = -0.242$). MPV and INR proved to be associated with both left ventricular ejection fraction and left ventricular end-diastolic diameter. The results revealed a strong association between MCV and MCHC with ventricular size in patients with HFrEF, while in HFpEF, sodium, chloride, and monocyte count correlated with diastolic function. ROC analysis revealed that MPV (AUC = 0.69), INR (AUC = 0.62), and cystatin C (AUC = 0.60) were the most effective laboratory tests in distinguishing heart failure phenotypes. These results demonstrate that routine laboratory parameters can still provide valuable information for assessing heart failure when echocardiography is unavailable.

Keywords: *Heart failure, echocardiography, routine laboratory parameters, resource-limited settings*

1.0 Introduction
Echocardiography plays a crucial role in evaluating both systolic and diastolic function through key parameters like mitral inflow velocities, left ventricular end-diastolic diameter (LVEDD), tricuspid valve regurgitant velocity (TRV), left ventricular ejection fraction (LVEF) (1,2) in heart failure. Clinicians use LVEF calculated through Simpson's biplane or three-dimensional imaging to classify heart failure (HF) into three phenotypes (3,4). These metrics reflect underlying cardiac pathophysiology: LVEF for systolic function, LVEDD for preload and dilation, E/A ratio for diastolic compliance, and TRV for pulmonary pressure. Many of these parameters predict prognosis and guide management in both acute and chronic HF (5,6). Given this utility, echocardiography remains essential in heart failure (HF) diagnosis, treatment planning, and long-term follow-up.

Results of routine laboratory parameters in heart failure can provide valuable insights that reflect the heart profile, particularly when inflammation, poor blood flow, or organ damage is present. For example, decreased lymphocytes, increased neutrophils, signs such as anemia, and high red cell distribution width (RDW) often indicate ongoing inflammation, oxidative stress, or changes in the immune system's functioning. Kidney-related values, including estimated glomerular filtration rate (eGFR), blood urea nitrogen (BUN), and serum creatinine, can identify problems with blood flow and hormonal activity related to heart function. Impaired renal function predicts poor outcomes in cardiorenal syndrome (9,10). Liver enzymes (ALT, AST, GGT), low albumin, and high bilirubin may suggest hepatic congestion due to venous hypertension (11,12). These abnormalities often reflect the severity of HF and may exacerbate the disease through cytokine release and metabolic impairment. In clinical settings with limited imaging resources, laboratory data can support HF diagnosis and monitoring.

Evidence continues to grow regarding the relationship between echocardiographic measurements and circulating biomarkers in HF. Studies have shown that LVEF increases as soluble urokinase plasminogen activator receptor (suPAR) decreases and rises with higher BNP levels, which may reflect worsening systolic function independent of kidney or inflammatory status (13). The left atrial volume index (LAVI) exhibits a positive correlation with soluble ST2 (sST2), which is associated with myocardial strain and fibrosis (14). Tricuspid annular plane systolic excursion (TAPSE) also correlates with both NT-proBNP and renal dysfunction, even in patients who maintain a normal LVEF (15). These findings show how systemic changes reflect the overall cardiac health. Recognizing these patterns can assist doctors in making better informed decisions using simple laboratory data available.

In many low-resource settings, getting access to echocardiography can be difficult. However, routine Laboratory tests. (this should be used consistently) are often easier to get and more affordable, making them a practical tool for evaluating patients when advanced imaging is not an option. Recent machine learning studies have combined echocardiographic and laboratory data to predict HF progression better than traditional models (16). These studies utilize many variables, including RDW, NT-proBNP, LVEF, TAPSE,suPAR, and LAVI, to construct personalized disease profiles. Combining laboratory and echocardiographic results may improve diagnostic accuracy while reducing costs. This approach could support clinical decisions in healthcare systems with limited resources, especially in sub-Saharan Africa, where the HF burden remains high.

Heart failure causes changes not only in the heart but also in the kidneys, liver, blood, and immune system, which laboratory parameters can detect early and cost-effectively. In contrast, echocardiography, although vital, is not always readily available, especially in low- and middle-income health facilities. Clinicians may use laboratory values as indirect indicators of cardiac status if specific echocardiographic findings consistently correlate with them.

This study aims to identify these relationships, which will support low-cost diagnostic approaches in settings with limited access to imaging. By identifying accessible laboratory-based indicators of cardiac dysfunction, the study may support early diagnosis, risk stratification, and context-appropriate prediction models for resource-limited healthcare settings.

2.0 Materials and Methods

2.1 Study Design

We carried out a cross-sectional analytical study with patients from the Cardiology Unit and Medical Outpatient Department at Alex Ekwueme Federal University Teaching Hospital Abakaliki (AE-FUTHA), a tertiary referral center in Ebonyi State, Nigeria. The AE-FUTHA Research and Ethics Committee approved the study (Ref: AEFUTHA/REC/VOL3/2020/119). We followed all relevant ethical guidelines and obtained written informed consent from each participant after explaining the study's purpose and procedures.

2.2 Study Population

We enrolled 250 subjects aged 18 years or older who had a confirmed diagnosis of heart failure, as defined by the 2021 European Society of Cardiology (ESC) or 2022 American Heart Association (AHA)/American College of Cardiology (ACC) guidelines. Eligible participants had a recent transthoracic echocardiogram (TTE) completed within one week of blood sample collection and provided informed consent. We excluded individuals with incomplete laboratory or echocardiographic data, current infections, hematologic cancers, chronic liver disease unrelated to heart failure, end-stage renal disease requiring dialysis, known congenital heart defects, or pregnancy.

2.3 Data and Blood Sample Collection

Clinical and demographic data, including Charlson Comorbidity Index (CCI) score, body mass index (BMI), age, sex, blood pressure, pulse rate, and etiology of heart failure, were collected using structured questionnaires and patients records. Transthoracic echocardiography was performed by experienced cardiologists using a Philips HD11XE machine, according to the American Society of Echocardiography (ASE) recommendations. Measurements averaged across three cardiac cycles included left ventricular ejection fraction (LVEF, by Simpson's biplane method), tricuspid annular plane

systolic excursion (TAPSE), left atrial volume index (LAVI), mitral E/A ratio, tricuspid regurgitation velocity (TRV), left ventricular end-diastolic diameter (LVEDD), and right ventricular systolic pressure (RVSP). We applied standardization protocols and conducted periodic training to reduce inter-observer variation. We collected blood samples within 48 hours of echocardiography into anticoagulant tubes and processed them at the AE-FUTHA Central Laboratory using standard operating procedures. The tests included Haematological parameters, renal and liver function markers, lipid profile, enzyme levels, and coagulation parameters. The data generated were entered into Microsoft Excel with double-entry validation, and incomplete records were excluded from analysis.

2.4 Assay Procedure

We analyzed hematologic parameters using the Sysmex XN-500 automated hematology analyzer. Coagulation tests were run on the Sysmex CA-1500 analyzer. Biochemistry profiles—including renal, hepatic, and lipid markers were assessed using the Selectra 4 automated chemistry analyzer. Standard laboratory protocols were followed for all procedures.

2.5 Data Analysis

The data generated were analyzed using Python version 3.12, following a stepwise plan that included descriptive summaries, correlation testing, regression modeling, and diagnostic evaluation. Data management was performed using Pandas and NumPy, while plots and charts were created with Matplotlib and Seaborn. We used SciPy. Stats for descriptive and inferential statistics, stats models for regression analysis, and scikit-learn for ROC curve analysis.

Demographic, clinical, echocardiographic, and laboratory variables were summarized using medians and interquartile ranges. We assessed distribution using histograms, quantile–quantile plots, and the Shapiro-Wilk test. Log transformations were applied where appropriate. We calculated Spearman correlation coefficients to examine pairwise relationships between laboratory and echocardiographic parameters.

We developed multivariate linear regression models to identify independent laboratory predictors of key echocardiographic findings. Variables with a p-value below 0.1 in the bivariate step were included in the models. Results were reported as adjusted coefficients with 95% confidence intervals. Subgroup analyses were conducted based on heart failure phenotype (HFrEF, HFrEF), with interaction terms included to assess potential effect modification. These were visualized using interaction plots.

To assess the diagnostic accuracy of laboratory markers in identifying HF phenotypes, we constructed ROC curves. We reported the area under the curve (AUC), sensitivity, specificity, and optimal cut-off values based on Youden's index.

3.0 Results

A total of 250 patients with heart failure were included in the analysis. The median age was 64 years (IQR: 59–74), and the median body mass index (BMI) was 21.2 kg/m² (IQR: 19.0–23.5), reflecting a mostly underweight group. The median Charlson Comorbidity Index (CCI) score was 2.0, with systolic and diastolic blood pressures at 130 mmHg (IQR: 118–147) and 80 mmHg (IQR: 65–86), respectively. Pulse and respiratory rates remained within normal limits (Table 1). Echocardiographic findings showed that the median left ventricular ejection fraction (LVEF) was 49.0% (IQR: 40.0–60.0), placing many patients in the mid-range category (HFrEF). The median left ventricular end-diastolic diameter (LVEDD) was 53.0 mm (IQR: 43.5–60.0). Additional parameters such as mitral valve E-wave slope (EMS), A-wave slope (AMS), and E/A ratio were also recorded (Table 2).

Laboratory profiles revealed mild anemia, with a median hemoglobin level of 114 g/L (IQR: 95.0–124.5). Red and white cell counts were 3.80 $\times 10^{12}$/L and 6.13 $\times 10^{9}$/L, respectively. Platelet indices included a mean platelet volume (MPV) of 12.0 fL and RDW-SD of 47.9 fL (Table 3.1). Coagulation markers showed a median international normalized ratio (INR) of 1.18 and a prothrombin time ratio of 1.19. D-dimer was moderately elevated at 1.60 mg/L FEU, while fibrinogen was 2.93 g/L (Table 3.2).

Liver function tests indicated stable values, with ALT at 20.5 U/L, albumin at 35.6 g/L, and total bilirubin at 16.95 µmol/L (Table 3.3). Renal function was mildly impaired in some cases, with a median creatinine of 72.0 µmol/L, cystatin C of 1.33 mg/L, and estimated glomerular filtration rate (eGFR) of 74.76 mL/min/1.73m² (Table 3.5). Lipid measurements were generally low: total cholesterol was 3.51 mmol/L and LDL-C was 1.62 mmol/L (Table 3.4). Enzyme levels such as CK, CK-MB, LDH, and HBDH fell within expected values (Table 3.6).

Analysis of associations between laboratory and echocardiographic measures showed several statistically significant findings (Table 4). Cystatin C was positively related to LVEF ($\rho = 0.271$, $p = 0.011$) and negatively to LVEDD ($\rho = -0.242$, $p = 0.024$). MPV showed an inverse relationship with LVEF ($\rho = -0.285$, $p = 0.007$) and a positive one with LVEDD ($\rho = 0.243$, $p = 0.023$). INR and prothrombin time ratio had inverse relationships with LVEF but positive links with LVEDD. Monocyte count and ratio were inversely related to mitral EMS and E/A ratio. Hemoglobin and hematocrit levels were negatively correlated with AMS. Electrolytes, including chloride ($\rho = 0.260$, $p = 0.015$) and sodium ($\rho = 0.240$, $p = 0.025$), showed positive relationships with mitral EMS.

Multivariable regression identified several predictors of cardiac function (Table 5). LVEF was associated with prothrombin time ratio (β = 3.51, p = 0.036), MPV (β = –0.27, p = 0.008), age (β = 0.25, p = 0.027), respiratory rate (β = $–7.6 \times 10^{-15}$, p = 0.036), and INR (β = –3.68, p = 0.029). MPV was also a significant predictor of LVEDD (β = 0.21, p = 0.044). Pulse rate predicted mitral AMS (β = 0.38, p = 0.001), and monocyte count predicted EA ratio (β = –0.24, p = 0.042).

In patients with HFpEF, LVEDD was negatively correlated with markers of inflammation and red cell parameters, including WBC (ρ = –0.348, p = 0.022), monocyte ratio (ρ = –0.322, p = 0.035), and neutrophil ratio (ρ = –0.453, p = 0.002). Chloride (ρ = 0.515, p < 0.001) and sodium (ρ = 0.303, p = 0.048) were positively linked to mitral EMS. Albumin (ρ = –0.479, p = 0.001) and HDL-C (ρ = –0.359, p = 0.018) had inverse relationships with tricuspid return velocity (Table 6).

Among HFrEF patients, red cell indices were strongly associated with ventricular size. MCV (ρ = 0.431), MCHC (ρ = 0.412), and hematocrit (ρ = 0.380) all had positive links with LVEDD. INR and prothrombin time ratio were negatively associated with LVEF (ρ = –0.440 and –0.443, respectively, both p = 0.003). Additional associations were found between albumin, globulin, and echocardiographic variables (Table 7).

Regression models stratified by phenotype showed significant interaction terms in HFrEF (Table 8). LVEF was negatively associated with mean hemoglobin concentration (β = –0.216, p = 0.046), INR (β = –0.241, p = 0.024), and prothrombin time ratio (β = –0.247, p = 0.021). Positive associations were noted between LVEF and both chloride (β = 0.243, p = 0.021) and sodium (β = 0.207, p = 0.050). For LVEDD, hemoglobin, MCV, MCH, MCHC, PDW, and basophil count all showed positive associations.

Diagnostic evaluation using ROC analysis (Table 9) showed that MPV had the highest area under the curve (AUC = 0.69), followed by INR and creatine kinase (both AUC = 0.62), and cystatin C (AUC = 0.60). INR achieved 59% sensitivity and 67% specificity at its optimal threshold. Other parameters, such as GGT, albumin, and LDH, showed moderate performance.

Table 1. Baseline Demographic and Clinical Characteristics of Heart Failure Subjects.

Variable	Median (IQR)
Age (years)	64.0 (59.0–74.0)
Body Temperature (°C)	36.4 (36.2–36.5)
Pulse (beats/min)	81.0 (70.0–93.5)
Respiration (breaths/min)	19.0 (18.0–19.0)
Systolic Blood Pressure (mmHg)	130.0 (118.0–147.0)
Diastolic Blood Pressure (mmHg)	80.0 (65.0–86.0)
Mean Arterial Pressure (mmHg)	95.3 (86.7–105.3)
Weight (kg)	50.0 (45.0–56.5)
Height (m)	1.55 (1.50–1.60)
Body Mass Index (kg/m²)	21.21 (19.03–23.46)
Charlson Comorbidity Index Score[1]	2.0 (1.0–2.0)

[1]Charlson Comorbidity Index is used to predict 10-year survival in patients with multiple comorbidities.

Table 2. Echocardiographic Parameters of Heart Failure Subjects.

Parameter	Median (IQR)
Left Ventricular Ejection Fraction (%)	49.0 (40.0–60.0)
LV End-Diastolic Diameter (mm)	53.0 (43.5–60.0)
Mitral Valve E-wave Slope (EMS) (m/s)	0.98 (0.75–1.14)
Mitral Valve A-wave Slope (AMS) (m/s)	0.80 (0.54–0.96)
E/A Ratio	1.24 (0.78–1.84)
Tricuspid Regurgitant Jet Velocity (m/s)	3.30 (2.90–3.70)

LV = Left ventricle; E/A ratio = early (E) to late (A) diastolic filling velocities.

Table 3. Laboratory Parameters of Heart Failure Subjects

Table 3.1: Hematological Parameters

Parameter	Median (IQR)
Hemoglobin (g/L)	114.0 (95.0–124.5)
Hematocrit (L/L)	0.348 (0.300–0.382)
RBC Count (×10¹²/L)	3.80 (3.30–4.11)
WBC Count (×10⁹/L)	6.13 (4.82–7.63)
Lymphocytes (×10⁹/L)	0.92 (0.675–1.25)
Monocytes (×10⁹/L)	0.36 (0.31–0.465)
Neutrophils (×10⁹/L)	4.63 (3.21–5.80)
Eosinophils (×10⁹/L)	0.05 (0.025–0.13)
Basophils (×10⁹/L)	0.02 (0.02–0.03)
Platelet Count (×10⁹/L)	142.0 (110.0–184.5)
Mean Platelet Volume (fL)	12.0 (10.85–13.25)
RDW-CV (%)	14.3 (13.55–15.15)
RDW-SD (fL)	47.9 (45.55–50.40)
MCV (fL)	93.2 (89.85–97.1)
MCH (pg)	30.3 (29.0–31.75)
MCHC (g/L)	326.0 (317.0–333.0)

RBC = Red blood cells; WBC = White blood cells; RDW = Red cell distribution width;

MCV = Mean corpuscular volume; MCH = Mean corpuscular hemoglobin; MCHC = MCH concentration.

Table 3.2: Coagulation Parameters

Parameter	Median (IQR)
D-dimer (mg/L FEU)	1.60 (0.97–2.57)
INR	1.18 (1.13–1.31)
aPTT (sec)	33.95 (30.55–36.80)
Thrombin Time (sec)	17.15 (16.0–17.9)
Prothrombin Activity (%)	70.4 (62.3–80.05)
Prothrombin Time Ratio	1.19 (1.12–1.31)
Fibrinogen (g/L)	2.93 (2.35–3.32)

INR = International Normalized Ratio; aPTT = Activated partial thromboplastin time.

Table 3.3: Liver Function Parameters

Parameter	Median (IQR)
Total Bilirubin (µmol/L)	16.95 (11.6–23.95)
ALT (U/L)	20.5 (14.5–39.5)
Albumin (g/L)	35.6 (32.95–37.5)
Globulin (g/L)	27.8 (24.25–30.65)
Total Protein (g/L)	63.1 (58.55–66.45)
Albumin/Globulin Ratio	1.30 (1.10–1.50)

ALT = Alanine transaminase.

Table 3.4: Lipid Profile

Parameter	Median (IQR)
Total Cholesterol (mmol/L)	3.51 (2.94–4.08)
LDL-C (mmol/L)	1.62 (1.27–2.11)
HDL-C (mmol/L)	1.05 (0.945–1.31)
Triglycerides (mmol/L)	0.89 (0.66–1.13)

LDL-C = Low-density lipoprotein cholesterol; HDL-C = High-density lipoprotein cholesterol.

Table 3.5: Renal Function Parameters

Parameter	Median (IQR)
Urea (mmol/L)	7.78 (6.14–10.28)
Creatinine (µmol/L)	72.0 (59.2–105.7)
eGFR (mL/min/1.73m²)	74.76 (46.57–96.06)
Cystatin C (mg/L)	1.33 (1.11–1.86)
Uric Acid (µmol/L)	413.0 (339.0–528.5)
Sodium (mmol/L)	139.65 (136.3–141.35)
Potassium (mmol/L)	3.89 (3.54–4.20)
Chloride (mmol/L)	102.95 (100.05–106.1)
Calcium (mmol/L)	2.25 (2.14–2.33)

eGFR = Estimated glomerular filtration rate.

Table 4. Significant Correlations Between Routine Laboratory and Echocardiographic Parameters (p < 0.05)

Laboratory Variable	Echocardiographic Parameter	Spearman's ρ	p-value
GFR	LV End-Diastolic Diameter	0.222	0.038
Cystatin C	LVEF	0.271	0.011
Cystatin C	LV End-Diastolic Diameter	–0.242	0.024
Monocyte Ratio	Mitral Valve EMS	–0.246	0.022
Monocyte Count	LV End-Diastolic Diameter	–0.247	0.021
Monocyte Count	Mitral Valve EMS	–0.257	0.016
Monocyte Count	E/A Ratio	–0.219	0.041
Hematocrit	Mitral Valve AMS	–0.235	0.028
Mean Platelet Volume	LVEF	–0.285	0.007
Mean Platelet Volume	LV End-Diastolic Diameter	0.243	0.023
Hemoglobin	Mitral Valve AMS	–0.233	0.030
Neutrophil Ratio	LV End-Diastolic Diameter	–0.222	0.038
INR	LVEF	–0.296	0.005
INR	LV End-Diastolic Diameter	0.214	0.047
Prothrombin Time Ratio	LVEF	–0.291	0.006
Chloride	Mitral Valve EMS	0.260	0.015
Sodium	Mitral Valve EMS	0.240	0.025
CK-MB/CK Ratio	LV End-Diastolic Diameter	–0.300	0.005
Creatine Kinase	LV End-Diastolic Diameter	0.337	0.001
Creatine Kinase	Mitral Valve EMS	0.231	0.032
White Globulin Ratio	LV End-Diastolic Diameter	0.251	0.019

Values represent Spearman's correlation coefficient (ρ).
Abbreviations: LVEF = Left Ventricular Ejection Fraction; EMS = E-wave Slope; AMS = A-wave Slope; LV = Left Ventricle; CK = Creatine Kinase.

Table 5. Multivariate Linear Regression: Laboratory Predictors of Echocardiographic Parameters

Echocardiographic Parameter	Variable	Coef.	SE	t-value	p-value	95% CI
LVEF	Prothrombin Time Ratio	3.51	1.65	2.13	0.036	0.23 to 6.80
	Mean Platelet Volume	–0.27	0.10	–2.75	0.008	–0.47 to –0.08
	Age	0.25	0.11	2.25	0.027	0.03 to 0.48
	Respiration Rate	–7.6e-15	3.6e-15	–2.14	0.036	–1.47e-14 to –5.1e-16
	INR	–3.68	1.65	–2.23	0.029	–6.96 to –0.40
LVEDD	Mean Platelet Volume	0.21	0.10	2.05	0.044	0.01 to 0.41
Mitral Valve AMS	Pulse Rate	0.38	0.11	3.43	0.001	0.16 to 0.61
E/A Ratio	Monocyte Count	–0.24	0.12	–2.07	0.042	–0.47 to –0.01

Abbreviations: LVEF = Left Ventricular Ejection Fraction; LVEDD = Left Ventricular End-Diastolic Diameter; AMS = A-wave Slope.
Coefficients (Coef.), standard errors (SE), and 95% confidence intervals (CI) are shown.

Table 6. HFpEF Group: Spearman Correlations Between Laboratory and Echocardiographic Parameters

Laboratory Variable	Echocardiographic Parameter	Spearman's ρ	p-value
White Blood Cell Count	LV End-Diastolic Diameter	–0.348	0.022
Monocyte Ratio	Mitral Valve EMS	–0.322	0.035
MCV	Tricuspid Valve Return Velocity	–0.390	0.010
MCH	Tricuspid Valve Return Velocity	–0.368	0.015
MCHC	LV End-Diastolic Diameter	–0.347	0.023
Eosinophil Ratio	LV End-Diastolic Diameter	0.390	0.010
Eosinophil Count	LV End-Diastolic Diameter	0.309	0.044
Chloride	Mitral Valve EMS	0.515	<0.001
Sodium	Mitral Valve EMS	0.303	0.048
CK-MB/CK Ratio	LV End-Diastolic Diameter	–0.331	0.030
HBDH/LDH Ratio	LV End-Diastolic Diameter	–0.315	0.040
Neutrophil Ratio	LV End-Diastolic Diameter	–0.453	0.002
Neutrophil Count	LV End-Diastolic Diameter	–0.404	0.007
Direct Bilirubin	Mitral Valve EMS	–0.319	0.037
Total Bilirubin	Mitral Valve EMS	–0.313	0.041
Total Protein	Tricuspid Valve Return Velocity	–0.320	0.036
Total Cholesterol	Tricuspid Valve Return Velocity	–0.441	0.003
LDL-C	Tricuspid Valve Return Velocity	–0.329	0.031
Calcium	Mitral Valve AMS	–0.335	0.028
Calcium	Tricuspid Valve Return Velocity	–0.337	0.027
Albumin	Tricuspid Valve Return Velocity	–0.479	0.001
HDL-C	Tricuspid Valve Return Velocity	–0.359	0.018

Abbreviations: MCV = Mean Corpuscular Volume; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration; LDL-C = Low-Density Lipoprotein Cholesterol; HDL-C = High-Density Lipoprotein Cholesterol; EMS = E-wave Slope; AMS = A-wave Slope.
Only significant values ($p < 0.05$) are shown.

Table 7. HFrEF Group: Spearman Correlations Between Laboratory and Echocardiographic Parameters ($p < 0.05$)

Laboratory Variable	Echocardiographic Parameter	Spearman's ρ	p-value
Red Blood Cell Count	Mitral Valve AMS	–0.309	0.041
Red Blood Cell Count	E/A Ratio	0.365	0.015
Hematocrit	E/A Ratio	0.380	0.011
Lymphocyte Count	LV End-Diastolic Diameter	0.333	0.027
MCH	LV End-Diastolic Diameter	0.431	0.004
MCH	Mitral Valve EMS	0.317	0.036
MCHC	LV End-Diastolic Diameter	0.412	0.005
Neutrophil Ratio	LV End-Diastolic Diameter	–0.376	0.012
INR	LVEF	–0.440	0.003
INR	LV End-Diastolic Diameter	0.334	0.027
Prothrombin Time Ratio	LVEF	–0.443	0.003
Prothrombin Time Ratio	LV End-Diastolic Diameter	0.339	0.024
Fibrinogen	LVEF	0.332	0.027
Chloride	Mitral Valve EMS	0.339	0.024
Chloride	LVEF	0.339	0.024
Sodium	Tricuspid Valve Return Velocity	–0.351	0.020
HBDH/LDH Ratio	Mitral Valve EMS	0.502	<0.001
Albumin	Mitral Valve EMS	0.316	0.037
Albumin	Mitral Valve AMS	0.301	0.047
White Globulin Ratio	LV End-Diastolic Diameter	0.392	0.008
White Globulin Ratio	Mitral Valve EMS	0.386	0.010
GGT	Mitral Valve AMS	–0.325	0.031

ALT (GPT)	LV End-Diastolic Diameter	0.320	0.034
Indirect Bilirubin	LV End-Diastolic Diameter	0.482	0.001
Direct Bilirubin	LV End-Diastolic Diameter	0.446	0.002
Total Bilirubin	LV End-Diastolic Diameter	0.482	0.001
Globulin	LV End-Diastolic Diameter	–0.365	0.015
Globulin	Mitral Valve EMS	–0.311	0.040

Abbreviations: LVEF = Left Ventricular Ejection Fraction; LV = Left Ventricle; EMS = E-wave Slope; AMS = A-wave Slope; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration; GGT = Gamma-Glutamyl Transferase; ALT = Alanine Transaminase. Only significant values are reported.

Table 8. Significant Laboratory Predictors of Echocardiographic Parameters in HFrEF (Interaction Terms)

Echocardiographic Parameter	Laboratory Variable	Interaction Term	Coefficient	95% CI	p-value
LVEF	Mean Hemoglobin Volume (MCH)	MCH:HF_phenotypes	–0.212	–0.421 to –0.002	0.048
LVEF	Mean Hemoglobin Concentration	MCHC:HF_phenotypes	–0.216	–0.429 to –0.004	0.046
LVEF	INR	INR:HF_phenotypes	–0.241	–0.450 to –0.032	0.024
LVEF	Prothrombin Time Ratio	PTR:HF_phenotypes	–0.247	–0.455 to –0.039	0.021
LVEF	Chloride	Chloride:HF_phenotypes	0.243	0.037 to 0.448	0.021
LVEF	Sodium	Sodium:HF_phenotypes	0.207	0.000 to 0.415	0.050
LVEDD	Mean Corpuscular Volume	MCV:HF_phenotypes	0.352	0.026 to 0.677	0.034
LVEDD	Hematocrit	Hematocrit:HF_phenotypes	0.383	0.049 to 0.717	0.025
LVEDD	Mean Hemoglobin Volume (MCH)	MCH:HF_phenotypes	0.526	0.209 to 0.842	0.001
LVEDD	Mean Hemoglobin Concentration	MCHC:HF_phenotypes	0.601	0.282 to 0.920	<0.001
LVEDD	Basophil Count	Basophil:HF_phenotypes	0.336	0.002 to 0.670	0.049
LVEDD	Hemoglobin	Hemoglobin:HF_phenotypes	0.428	0.097 to 0.760	0.012
LVEDD	Platelet Distribution Width	PDW:HF_phenotypes	0.424	0.036 to 0.812	0.032

Abbreviations: LVEF = Left Ventricular Ejection Fraction; LVEDD = Left Ventricular End-Diastolic Diameter; MCH = Mean Corpuscular Hemoglobin; MCHC = Mean Corpuscular Hemoglobin Concentration; PDW = Platelet Distribution Width.
Results from regression models including interaction terms (laboratory variable × HF phenotype).

Table 9. Diagnostic Performance of Laboratory Parameters for Predicting HF Phenotypes *Based on Receiver Operating Characteristic (ROC) analysis.*

Laboratory Variable	Optimal Threshold	Sensitivity	Specificity	AUC
Mean Platelet Volume	0.1978	0.57	0.74	0.69
INR	–0.0528	0.59	0.67	0.62
Creatine Kinase	–0.5349	0.82	0.42	0.62
Cystatin C	0.4640	0.43	0.81	0.60
GGT	0.5195	0.43	0.79	0.60
Mean Hemoglobin Concentration	0.5372	0.45	0.79	0.60
GFR	0.4194	0.50	0.77	0.60
Uric Acid	0.6163	0.41	0.79	0.56
Total Bilirubin	0.0858	0.57	0.67	0.57
Indirect Bilirubin	0.4640	0.43	0.81	0.57
Albumin	0.5201	0.34	0.81	0.58
LDH	0.4781	0.36	0.84	0.58
Triglycerides	0.0729	0.64	0.56	0.63
LDL-C	–0.3335	0.75	0.49	0.56
ALT (GPT)	–0.1759	0.70	0.51	0.58
Hematocrit	–0.1774	0.77	0.51	0.58
Neutrophil Ratio	–1.018	0.98	0.26	0.57
Creatine Kinase Isoenzyme (CK-MB)	–0.3219	0.73	0.49	0.59
Alkaline Phosphatase	0.0909	0.52	0.70	0.56

Abbreviations: AUC = Area Under Curve; GGT = Gamma-Glutamyl Transferase; ALT = Alanine Transaminase; LDH = Lactate Dehydrogenase; GFR = Glomerular Filtration Rate; LDL-C = Low-Density Lipoprotein Cholesterol.

4.0 Discussion

We conducted this study to investigate the clinical, laboratory, and echocardiographic characteristics of heart failure (HF) patients in Abakaliki, Southeastern Nigeria. Our goal was to understand how standard laboratory tests relate to cardiac function and structure, especially when comparing HF with preserved ejection fraction (HFpEF) and reduced ejection fraction (HFrEF). This allowed us to study heart failure patterns in a setting where diagnostic resources are limited but the burden of HF is high.

Our patients were predominantly older adults, with a median age of 64 years, and were generally underweight (median BMI of 21.2 kg/m²). Most of them had a moderate Charlson Comorbidity Index score of 2. These values differ from those reported in more obese HFpEF groups in Western and urban Nigerian studies (17,18). The median LVEF of 49% and borderline E/A ratio (1.24), along with a left ventricular end-diastolic diameter (LVEDD) of 53 mm, suggest that many of our patients were in an intermediate phase between HFpEF and HFrEF. This aligns with views that HF is better described as a continuous disease process rather than being categorized into fixed stages (19,20).

We also noted that liver function was relatively well maintained, while early kidney injury was present, as indicated by increased cystatin C levels. Mild anemia was also common, with a median hemoglobin level of 114 g/L. Compared to advanced HF cases with severe weight loss and multi-organ failure, our findings point to an earlier stage of illness with a window for timely treatment (21,22).

Among hematologic markers, we found that red blood cell indices such as mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and mean corpuscular volume (MCV) were strongly linked to heart structure and function in patients with HFrEF. Lower MCH and MCHC were linked with reduced LVEF, while higher MCV, hemoglobin, hematocrit, and platelet distribution width (PDW) were linked with greater LVEDD. These findings are consistent with earlier studies that have demonstrated how red blood cell variations reflect remodeling, oxygen delivery issues, and impaired cardiac performance (23–25). The increased RDW-SD in our patients supports the idea that inflammation and nutritional deficiencies contribute to reduced red blood cell production (26,27).

Anemia in HF has many causes—such as kidney disease, poor nutrition, inflammation, and neurohormonal stress—and these are often seen together. The strong links between red cell indices and cardiac changes in HFrEF reflect this connection (28,29). On the other hand, HFpEF showed no such associations, which supports the theory that it follows a different disease path, driven more by inflammation and metabolic problems than volume overload (30). This suggests

that red blood cell markers may help distinguish between HFpEF and HFrEF in clinics where advanced testing is not available.

In terms of liver-related and coagulation tests, we found that INR and PT ratio predicted lower LVEF values, while bilirubin levels were associated with chamber enlargement. This pattern reflects liver congestion and poor liver function, which are common in advanced HFrEF (31–33). Patients with HFrEF also had higher MPV and PDW levels, markers of platelet activity and vascular stress, which have been linked to inflammation and thrombotic risk (34,35).

What stood out in our study was that INR and PT ratio had opposite effects on LVEF, a finding not widely reported in African populations. This may reflect the complex ways in which liver dysfunction, medications, and congestion interact in HF (36,37). These findings support the notion that standard coagulation tests can aid in predicting cardiac function, particularly when used in combination with other tests.

We also studied inflammatory markers by looking at white blood cell counts and platelet indices. In our data, monocyte count and ratio were linked with higher EA ratios, a marker of diastolic function, while MPV was tied to lower LVEF. These findings are consistent with studies linking inflammation to diastolic dysfunction (38, 39). Surprisingly, we saw that HFpEF patients had negative associations between neutrophil counts, WBCs, and LVEDD. This could mean that the immune response is less active in early or mild HFpEF, or that local immune behavior in African patients differs from Western cohorts (40,41).

Unlike many studies that use derived ratios, such as NLR, we focused on absolute monocyte counts. This makes our results more practical for real-world settings, where laboratories may not have the capability to calculate complex ratios. Monocytes may serve as an early warning sign for diastolic dysfunction, especially in places with limited diagnostic options.

We also identified some new and unusual findings. In HFpEF, sodium and chloride were positively associated with mitral valve EMS, which measures early diastolic relaxation. This was especially true for chloride, which had a moderate to strong positive correlation. While this area is rarely studied, electrolyte balance may affect how well the heart muscle relaxes, possibly due to changes in volume status or vascular tone. This adds a new layer of insight into the factors that affect diastolic function.

We further found that HDL-C and albumin were linked with lower tricuspid return velocity in HFpEF. Low HDL-C and albumin levels have both been associated with poor outcomes and could reflect inflammation, poor nutrition, or both (42,43). Their link to proper heart measures suggests that these patients may have early right-sided pressure problems. These findings highlight the importance of measuring nutritional and metabolic markers, particularly in settings like ours, where patients may be both ill and undernourished.

Cystatin C was another useful marker in our study. We found that it was linked with both LVEF and LVEDD, confirming its role as a marker of early heart and kidney stress. This aligns with work from (44,45), which found that cystatin C reflects not only kidney function but also neurohormonal activity. Our work builds on this by tying cystatin C to structural heart changes, showing that it may help track both function and anatomy in HF.

When comparing HFpEF and HFrEF side by side, we observed distinct patterns. In HFrEF, structural changes in the heart were associated with red blood cell indices, liver markers, and coagulation tests. In HFpEF, however, only a few markers mainly those related to metabolism, inflammation, and diastolic function showed any association. These results align with current thinking that HFrEF results more from cardiac remodeling and systemic congestion, while HFpEF is associated more closely with comorbid conditions, vascular problems, and inflammation (46,47).

Regression analysis confirmed that MPV, INR, and cystatin C were strong indicators of cardiac dysfunction. MPV and INR had different but meaningful effects on LVEF and LVEDD, and they could provide a cost-effective method for classifying patients by HF type and severity. This is especially important in African settings, where high-cost markers such as NT-proBNP or GDF-15 are often unavailable (18,48).

Our approach differed from previous Nigerian research, which primarily focused on hemoglobin or reported HF as either present or absent (49,50). We used a larger panel of tests and linked them to both systolic and diastolic echo parameters. By doing so, we provided a more comprehensive picture of HF and developed a low-cost model that could be utilized in clinics across Nigeria and similar regions.

Limitations of the Study

One of the main strengths of this study lies in its use of accessible, low-cost laboratory parameters and detailed echocardiographic data to describe HF phenotypes in a real-world sub-Saharan setting. We used routine blood parameters

that are widely available in most Nigerian and African hospitals. This supports practical application in everyday clinical care.

However, the study has a few limitations. The cross-sectional design limits our ability to study outcomes over time or to identify cause–and–effect relationships between laboratory parameters and cardiac structure. We also used data from a single hospital in Southeastern Nigeria, so our findings may not be representative of all HF patients in the country or on the continent. Furthermore, we did not measure natriuretic peptides, cardiac MRI, or specific inflammatory cytokines, which could have provided more detailed mechanistic insights.

Conclusion

Routine blood tests such as red blood cell and platelet indices, coagulation, and metabolic parameters reveal meaningful correlations with cardiac function in Nigerian patients with heart failure. These patterns differ between HFpEF and HFrEF, supporting their use in early diagnosis and risk assessment, especially when resources are limited.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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