PREDICTORS OF ACUTE HEART FAILURE OUTCOMES AT UNIVERSITY COLLEGE HOSPITAL, IBADAN, NIGERIA

O. Adebayo[1,2], A. Adebiyi[2,3], O.S Ogah[1,2,3], M.A. Adeoye[1,2,3], A. Aje[2], O.O Oladapo[1,2,3]

1. Institute of Cardiovascular Diseases, College of Medicine, University of Ibadan, Ibadan
2. Cardiology Unit, Department of Medicine, University College Hospital, Ibadan
3. Department of Medicine, Faculty of Clinical Sciences, University of Ibadan, Ibadan

Correspondence:
Dr. O. Adebayo
Institute of Cardiovascular Diseases,
College of Medicine,
University of Ibadan,
Ibadan
Email: doctorladi@yahoo.com

ABSTRACT

Background: **Studies of acute heart failure (AHF) outcomes suggest that there are different predictors of mortality depending on region, ethnicity, and gender.**

Objective: **The purpose of this study was to identify predictors of six months' post discharge outcome among AHF patients in a Nigerian tertiary hospital.**

Methods and Materials: **This study was a prospective observational study conducted at the cardiology unit of the Department of Medicine at the University College Hospital Ibadan. One hundred and sixty AHF participants ≥ 18 years old were recruited.**

Results: **The mean age of the cohort was 58.0±15.1 years and most were males (59.4%). The independent predictors for death outcome after six months of discharge for AHF and the adjusted hazard ratio) (95% CI) were male gender 2.77 (1.17 -6.56); p = *0.020*; systolic blood pressure (mmHg) 0.98 (0.96 - 0.99); p = *0.011*; and the presence of hepatomegaly 2.58 (1.02 - 6.51); p = *0.045*. Independent predictors for readmission or rehospitalization within 6 months after discharge were presence of right abdominal pain adjusted HR (95% CI): 2.07(1.14 - 3.76), p=*0.017*; SBP 0.98(0.96 - 0.99), p= *0.005*. Independent predictors for composite endpoint were male gender: adjusted HR: 2.08 (1.16 - 3.72), p= *0.014* and pericardial effusion and tamponade: 5.31(1.79 - 15.74), p=*0.003*.**

Conclusion: **The study provided an insight into the factors contributing to outcomes six-month after admission in a tertiary centre in South-Western Nigeria, and it highlighted the predictive role of systolic blood pressure.**

INTRODUCTION

Acute heart failure (AHF) is a clinical syndrome characterised by the sudden or gradual onset of heart failure symptoms and/or signs severe enough to necessitate hospitalization or an emergency department visit.[1] Interestingly, the understanding of its complexity and heterogeneity has evolved over the last few decades. Despite the increased understanding of clinical intervention and therapeutics on the condition, survival remains poor in terms of intrahospital mortality, readmission, or post-discharge mortality.[2-4]

Few literatures available suggest multiple independent factors are associated with AHF outcomes such as mortality, readmission, or all-cause events (mortality combined with readmission). These factors include sociodemographic, past medical history, clinical profile and comorbidities, laboratory parameters, electro-cardiographic and echocardiographic features among others.[5-7] In IN-HF Outcome Registry, predictors found for AHF outcome included age, low systolic blood pressure, anaemia, and renal dysfunction, signs of cerebral hypo-perfusion, low serum sodium, chronic obstructive pulmonary disease, and pulmonary oedema.[8] There is a direct relationship between renal function and AHF outcomes, whether in the short or long term.[5, 6]

Although most AHF studies were conducted in non-black African populations, the first multinational study on the subject in Africa identified only few factors as predictors.[7] In one study in Northern Nigeria, the identified risk factors included low left ventricular ejection fraction (VEF) (≤40%), renal impairment, cardiac rhythm abnormalities such as atrial fibrillation (AF) at six months, prolonged corrected QT interval (QTC) and complete left bundle branch block (LBBB), anaemia and advanced age.[9, 10] While lower body mass index, low literacy, low serum sodium level, the presence of atrial fibrillation, renal dysfunction, and valvular dysfunction were predictors of readmission

with a six-month follow-up in the Abeokuta HF registry study.[11]

Overall, there are diverse-predictors of outcomes in AHF depending on the location of study.

This study explored a broad range of the variables, notably clinical variables and socio-demographic to identify predictors of 180-day outcomes among AHF patients presenting at the University College Hospital (UCH), Ibadan, Nigeria

METHODS AND MATERIALS
Study Design
The study was a prospective observational study.[3]

Study Location
The study was carried out at the Cardiology Unit of the Department of Medicine, University College Hospital (UCH) in Ibadan. The hospital is a 1000-bed Federal tertiary health care facility that serves the people of Oyo State and other Nigerian states.

Study Population
Acute heart failure patients were consecutively recruited if they fulfil the inclusion criteria. The recruiting between 07/06/2018 and 07/01/2019.

Participants had to be at least 18 years old and have either de novo or acute decompensated chronic HF, regardless of the primary cause. Participants who declined enrolment and did not meet the inclusion criteria, pregnant women, and HIV-1 and HIV-2 patients were all excluded from the study.

Enrolment and data collection
Informed consent was obtained from all the participants. Each participant was interviewed in complete privacy. Interview and assessment sessions lasted approximately 40 minutes per patient. To facilitate follow-up, phone numbers and addresses of participants and their next-of-kin were collected. The interviewer read questionnaire questions to the participants and recorded their responses. Basic demographic information as well as clinical history were gathered. Cigarette smoking was assessed and recorded as ever, never, and current smoking and alcohol use were similarly assessed. Cardiovascular risks factors were assessed in the participants. This has also been described in elsewhere.[3] The causes and precipitating factors of AHF episodes were also documented. There were four interviews during the assessment process: during recruitment, one month, three months, and six months after discharge. All recruitment interviews were conducted in person, and all follow-up sessions were conducted in person as well. When the patient was unable or refused to meet the follow-up schedule, a phone interview was conducted. The purpose of the interview was to determine readmission, the presence of an event, or complications. For death outside of the study facility and the investigator does not have access to the records, a verbal autopsy was performed. The interviews with the participants were conducted by an interviewer who was fluent in the patients' language as well as English if the patient could adequately converse in any of these. For speakers of other Nigerian dialects who are unable to communicate in English, a clinical staff member who is fluent in that dialect was used as a translator.

Diagnosis of acute AHF
Acute heart failure was diagnosed using the European Society of Cardiology (ESC)criteria and were classified as de novo AHF or acute decompensated chronic HF.[12]

Definition of AHF terms:
(a) De novo AHF (New-onset AHF) is meant to be AHF in patients with no prior history of HF
(b) Acute decompensated chronic HF (CHF) is worsening HF features in a known HF patient, i.e., acutely decompensated CHF patients.

Procedures
Laboratory
Fasting blood glucose and fasting lipid samples were collected after 8-10hours overnight fasting.

Serum creatinine level was used to determine the eGFR with the Cockcroft–Gault formula[13].

Electrocardiography
A standard 12-lead electrocardiography studies were done for the participants using a commercially available CONTEC® Workstation Model, CONTEC EC8000G, ECG machine (Made in China) at a speed of 25mm/s and 1mV/cm calibration while they were supine and at rest.[3] Various parameters such as PR intervals, QRS duration and axis, rate, rhythm, conduction abnormalities, types of arrhythmias, and QT intervals were assessed.[3]

Echocardiography
Transthoracic echocardiography were done for the participants based on the American Society of Echocardiography guidelines.[14] The participants had the echocardiographic procedure within 72 hours of admission in the partial left lateral decubitus position using a Toshiba Xario (Toshiba Medical Systems Corp) with a 3.5 MHz transducer.[3]

Follow up Details

Participants were followed-up for 6 months for major adverse cardiovascular events (MACEs) such as cardiac death, hospitalization for HF, and cardiovascular events. They were assessed at one, three and six months in the clinic. Medication intake were also recorded as types and either yes or no and those lost to follow up were documented. The composite all-cause mortality for HF during 6 months of follow up after discharge from hospital were documented. Deaths were further classified into the cardiovascular cause and non-cardiovascular cause. The ECG, echocardiographic, haematologic and biochemistry studies conducted for the patients at baseline were not repeated at follow up.

Ethical Considerations

Ethics approval was obtained from the institution's ethics review committee (approval number -(UI/EC/18/0004) and the study was conducted according to international and local ethics principles.

The study's questionnaires were kept secure, and the data generated was also kept safe from third parties.

Data Management and Statistical Analysis

Data was entered into a secured electronic database. International Business Machines (IBM) Corporation Statistical Product and Service Solutions (SPSS) for Windows version 23.0 was used for the statistical analysis (Armonk, NY: IBM Corp).

Sociodemographic, lifestyle, co-morbidities, risk factors, clinical symptoms at admission, vital signs at admission, anthropometry, laboratory results, ECG and echocardiographic results, precipitants of AHF, medication on admission and post-discharge status, and outcome made up the explanatory variables. Readmissions for HF, readmissions for non-HF cause, intrahospital deaths, and intrahospital cardiovascular complications were the study's primary outcome variables, while all-cause mortality, cardiac deaths, and cardiovascular events were its secondary outcomes. The normality test was performed on the data. Means, standard deviations, and other summary statistics for normally distributed continuous variables were compared with one-way analysis of variance (ANOVA).

The Mann-Whitney U test was used to compare the non-normally distributed data, which were summarised as median and interquartile range. Quantitative (categorical) variables were compared using the Pearson chi-square test and were expressed as frequencies and percentages. To identify the independent predictors of mortality, readmission, and all-cause events (composite events), Cox regression models were fitted. The models were also built with the time from admission to the first event in mind. For patients without the relevant event, the times were censored at the earlier of the relevant period of interest or the last date the patient was known to be alive. To find explanatory factors connected to the outcome variable that were statistically significant, Cox analysis was used. In order to have a strong mix of predictors to take into account, factors with a p-value of 0.1 or above were excluded from the multivariate model. Previously identified factors in the literature were also included. To identify potential risk factors for mortality six months after discharge, readmission six months after discharge, and all-cause events, multivariate Cox proportional hazards models were fitted. To determine every target outcome, all statistically significant univariate analysis variables were progressively fitted into the multivariate logistics regression model. Kaplan Meir survival analysis was used to analyse survival at six months. Two-sided p-values of 0.05 or less were regarded as significant for all tests.

RESULTS

The age range of participants was 18 to 90years, with about half being older than 60 years. The mean ± standard deviation age was 58.0±15.1years and most were males (59.4%) (Table 1). About four out of five participants were married. Hypertension (77.5%), kidney disease (34.4%), and diabetes mellitus (18.1%) were the most prevalent comorbidities. Alcohol consumption and smoking, were the common behavioural cardiovascular risk factors. Reduced exercise tolerance, fatigue & tiredness, difficulty recovering from exercise, and dyspnoea were present in 88.1%, 83.8%, 83.8%, and 83.1% respectively (Table 2).

The commonest physical finding at baseline evaluation were tachypnoea, ascites, laterally displaced apical impulse and the presence of third heart sound with the following frequencies 98.8%, 90.0% and 73.8%,
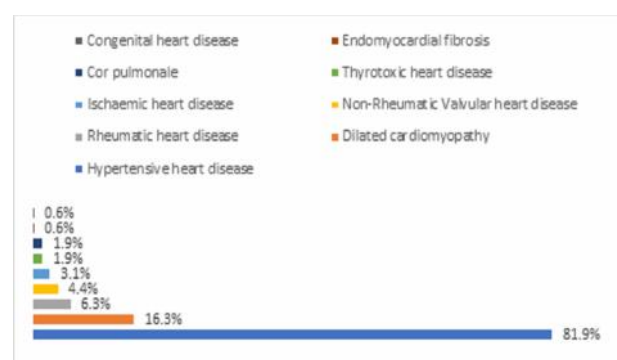
Figure 1: Diagnosis of heart failure

**Table 1:** Sociodemographic characteristics of the 160 participants

Variables	All (n=160)
Age (mean ± SD) years	58.0±15.1
Gender	
Male n (%)	95(59.4)
Female n (%)	65(40.6)
Aged ≥60 years n (%)	76(47.5)
Marital Status n (%)	
Single	6(3.8)
Married	138(86.3)
Divorced	1(0.6)
Separated	5(3.1)
Widowed	10(6.3)
Educational level n (%)	
None	15(9.4)
Primary	32(20.0)
Secondary	65(40.6)
Tertiary	45(28.1)
Postgraduate	3(1.9)
Employment status n (%)	
Unemployed	73(45.6)
Employed	87(54.4)
Estimated monthly income n (%)	
≤₦100.000	146(93.1)
>₦100 000	14(6.9)
Comorbidities n (%)	
Hypertension	124(77.5)
Kidney disease	55(34.4)
Diabetes Mellitus	29(18.1)
Obesity	16(10.0)
COPD	8(5.0)
Asthma	3(1.9)
Arthritis	3(5.1)
Family history of hypertension n (%)	16(10.0)
Family history of heart failure n (%)	1(0.6)
History of atrial fibrillation n (%)	17(10.7)
History of stroke n (%)	2(1.3)
History of thyroid disease n (%)	1(0.6)
Smoking n (%)	
Never	139(86.9)
Previous	18(11.3)
Current	3(1.9)
Alcohol intake n (%)	
Never	108(67.5)
Previous	41(25.6)
Current	11(6.9)

[**] SD= *standard deviation*

respectively (Table 2). About 80% were in NYHA class III/IV. Majority were either overweight (63.1%) or obese (10.0%) (Table 2). About half of the AHF was precipitated by infection (51.3%). Other common precipitants were arrhythmias (25.0%) and poor medication adherence (18.1%). Physical stress and emotional excesses (0.6%) and fluid overload (0.1%) were the least common precipitants. (Table 2).

The biophysical profile and laboratory findings are presented in Table II. The packed cell volume (PCV) mean± SD was 40.1±6.3%. Only 156 (97.5%) of the AHF patients in this study were on loop diuretics at the time of recruitment/admission. Similarly, spironolactone (93.1%) and ACE inhibitor (83.8%) were prescribed in high proportions (Table 3).

The electrocardiographic results of the study participants were also displayed in Table 4. Approximately two out of every five people had
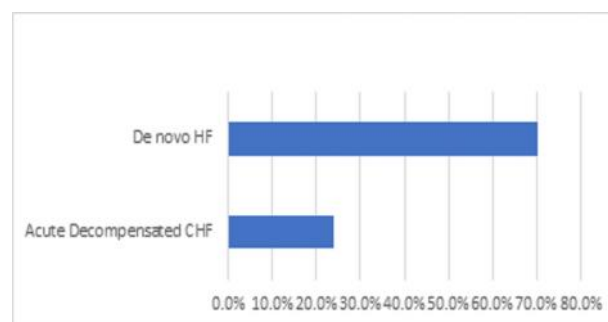
Figure 2: Type of heart failure

arrhythmias. The prevalence of left ventricular systolic dysfunction is high (75.0%). (Table 4). Mitral incompetence (84.4%) and tricuspid incompetence (78.1%) were the most prevalent valvular abnormalities (Table 4).

Hypertensive heart disease was the most frequent risk factor in AHF (131 (84.2%)). (Figure 2). Only 26 people (16.3%) and 10 people (6.3%), respectively, were found to have dilated cardiomyopathy and rheumatic heart disease (Figure 2). Majority of participants had de novo AHF (75.6%) compared to 24.4% for ADCHF, p=0.350. De novo AHF and ADCHF had mean SD
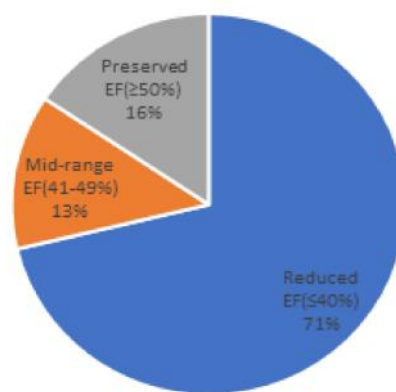

Figure 3: Type of heart failure based on EF

Table 2: Heart failure symptoms and frequencies of previous AHF admissions among the 160 AHF participants

Variables	All(n=160)
Symptoms at presentation/recruitment n (%)	
Reduced exercise tolerance	141(88.1)
Fatigue & tiredness	134(83.8)
Difficulty to recover after exercise	134(83.8)
Dyspnoea	133(83.1)
Paroxysmal nocturnal dyspnea (PND)	127(79.4)
Leg swelling	126(78.8)
Cough	118(73.8)
Orthopnoea	117(73.1)
Easy Satiety	76(47.5)
Right abdominal pain	57(35.6)
Palpitation	50(31.3)
Abdominal swelling	50(31.3)
Chest pain	43(26.9)
Loss of appetite	30(18.8)
Nausea	24(15.0)
Vomiting	23(14.4)
Headache	21(13.1)
Confusion	9(5.6)
Wheezing	8(5.0)
Bloated feeling	5(3.1)
Tachypnoea	158 (98.8)
Physical signs and New York Heart Association (NYHA) functional class of the participants	
Laterally displaced apical impulse (Displaced Apex)	128 (80.0)
Third heart sound (gallop rhythm)	118 (73.8)
Pulmonary crepitations	118 (73.1)
Peripheral oedema (ankle, sacral, scrotal)	117 (73.1)
Elevated jugular venous pressure (JVP)	109 (68.1)
Hepatomegaly	97 (60.6)
Ascites	96 (90.0)
Narrow pulse pressure	84 (52.5)
Tachycardia	73 (45.6)
Systolic murmur	59 (36.9)
Irregular pulse	52 (32.5)
Cold extremities	46 (28.8)
Pallor	30 (18.8)
Reduced breath sound and dullness to percussion at lung bases	26 (16.3)
Fever	18 (11.3)
Oliguria	12 (7.5)
Jaundice	10 (6.3)
Central cyanosis	10 (6.3)
Cheyne–Stokes respiration	9 (5.6)
Diastolic murmur	7 (4.4)
Finger clubbing	5 (3.1)
New York Heart Association Class n (%)	
I	1(0.6)
II	25(15.6)
III	103(64.4)
IV	31(19.4)

Table 3: Biophysical profile, laboratory results and use of medications of the 160 participants

Variables	All(n=160)
Biophysical profile	
Weight median (IQR) Kg	69.8 (62.78-76.82)
Height median (IQR) metre	1.63 (1.36- 1.90)
BMI median (IQR) Kg/m²	26.5(24.66-28.44)
BMI Category n (%)	
<18.5kg/m²	5(3.1)
18.5-24.9kg/m²	38(23.8)
25-29.9kg/m²	101(63.1)
≥30Kg/m²	16(10.0)
Pulse median (IQR) cycles/min	90.0(70.0-110.0)
Respiratory rate median (IQR) beats/min	28.0(20.0-36.0)
SBP median (IQR) mmHg	110.0(80.0-140.0)
DBP median (IQR) mmHg	80.0(51.5-108.5)
Heart rate mean± SD beats/min	97.9± 16.3
Tachycardia n (%)	52(32.5)
Pulse Pressure median (IQR) mmHg	38.5(22.5-54.5)
Body surface area (mean ± SD) m²	1.74± 0.12
Laboratory results	
PCV mean± SD %	40.1±6.3
WBC median (IQR) /10mm³	7.5(4.2-10.8)
Platelet median (IQR) /mm³	220.5(121.7-319.3)
Sodium (median ± IQR)	137.0(130.0-144.0)
Potassium (median ± IQR)	3.8(3.8-4.0)
Urea median (IQR) mg/dl	45.4(8.6-82.2)
Creatinine median (IQR) mg/dl	1.30(0.5-2.1)
eGFR median (IQR) mL/min/1.73 m²	53.1(19.9-86.3)
Total cholesterol mean± SD mg/dl	153.6 ± 30.6
LDL mean ± SD mg/dl	94.4 ±24.9
HDL mean ± SD mg/dl	40.4± 15.1
Triglyceride mean ±SD mg/dl	99.0±28.7
Use of medications at presentation	
Loop diuretics	156 (97.50)
Mineralocorticoid receptor inhibitors	149 (93.1)
ACEi/ARB	138 (86.3)
Anticoagulant agents	126 (78.8)
Beta-blocker	90 (56.3)
Digoxin	34 (21.3)
CCBs (Amlodipine)	21 (31.1)
Amiodarone	10 (6.3)
Aspirin	7 (4.4)
Anti-glycaemic agent	5 (3.1)
Thiazide	3 (1.9)
Centrally acting	1 (0.6)

** Kg- *kilogramme* IQR-*Interquartile range* SBP-*Systolic Blood Pressure* DBP-*Diastolic Blood Pressure* BMI- *Body Mass Index* PCV-*Packed cell volume* WBC-*White blood cells* IQR-*Interquartile range* SD-*Standard deviation* eGFR- *estimated glomerular filtration rate* LDL-*low-density lipoproteins cholesterol* HDL-*high-density lipoprotein cholesterol* LMWH= *Low Molecular Weight Heparin* ACEi= *Angiotensin-converting enzyme inhibitor* ARB=*Angiotensin II Receptor Blockers* CCBs-Calcium channel blockers Anticoagulant agent (LMWH, Warfarin, Traditional heparin) Anti-glycaemic agent (Insulin and oral hypoglycaemic agent)

of 38.6 12.9% and 39.1 16.6, respectively, with a p-value of 0.830. 51 (67.1%), 10 (13.2%), and 15 (19.2%) fall into the categories of reduced, mid-range, and preserved ejection AHF, respectively. De novo HF and ADCHF had 59 (36.1%) and 17 (10.6%) more people over the age of 60, respectively (p=0.929) (Figure 3).

The survivors that were followed up during 6 months' post-discharge were 150(93.8%) with 16(10.0%) of the total study population lost to follow up. A total of 41(25.6%) of the total study population died during the study period.

Most death were after 8 days of stay for both gender while more males died on admission compared to females HR (95% confidence interval (CI)), *p*-value; 1.51(0.86,2.62),*0.151*. More of the death at post-discharge 6 months' follow-up were within the first 3 months of discharge (70.7%).

Table 4: Electrocardiographic profile of the participants

Variables	All (n=160)
Electrocardiographic results	
Heart rate mean ±SD/min	97.9±16.3
Sinus rhythm n (%)	98(61.3)
Normal ECG axis n (%)	110(68)
Atrial fibrillation n (%)	23(14.4)
Atrial flutter n (%)	4(2.5)
Premature Ventricular Contractions (PVCs) n (%)	28(17.5)
Premature Supraventricular Contractions n (%)	2(1.3)
Atrial chamber enlargement n (%)	
Left	24(15.0)
Right	15(9.4)
Bi-atrial	6(3.8)
None	115(71.9)
Ventricular Chamber hypertrophy	
Left Ventricular hypertrophy (LVH) n (%)	48(30.0)
Left Ventricular hypertrophy (LVH) with strain n (%)	37(23.1)
Right Ventricular hypertrophy (LVH) n (%)	13(8.1)
Right Ventricular hypertrophy (LVH) with strain n (%)	8(5.0)
Conduction abnormalities n (%)	22(13.8)
First degree AV block n (%)	9(5.6)
Second degree AV block	3(1.9)
Complete heart block	0
Right Bundle Branch Block (RBBB) n (%)	3(1.9)
Left Bundle Branch Block (LBBB) n (%)	6(3.8)
Intermediate interventricular block n (%)	1(0.6)
Presence of infarction	2(1.3)
QT interval mean± SD ms	369.7± 71.5
QT$_c$ interval mean± SD ms	415.6± 65.3
QRS mean± SD ms	112.7± 39.7
Echocardiographic results	
Aortic valve opening (mean ± SD) cm	2.01± 0.38
Aortic root diameter (mean ± SD) cm	2.92±0.57
Left atrial diameter (mean ± SD) cm	4.47±7.28
IVST(d) mean ± SD cm	1.06± 0.25
IVST(d) mean ± SD cm	1.16±0.28
LVPWT(d) mean ± SD cm	1.07± 0.27
LVPWT(s) mean ± SD cm	1.32± 0.33
LVID(d) mean ± SD cm	5.87± 1.07
LVID(s) mean ± SD cm	4.68± 1.16
Pulsed wave Doppler study of trans-mitral inflow	
Mitral E wave velocity (mean ± SD) m/s	0.95±0.33
Mitral A wave velocity (mean ± SD) m/s	0.57± 0.47
E/A wave ratio median (IQR)	1.67(0.80,2.54)
DT of Mitral E mean ± SD ms	202.8 ±112.9
IVRT (mean ± SD) (ms)	122.7±31.1
LV filling pattern n (%)	
Normal	102(63.8)
Impaired relaxation	23(14.4)
Pseudo normalization	5(3.1)
Restrictive pattern	
Reversible	4(2.5)
Fixed	26(16.3)
LV geometry n (%)	
Normal geometry	19(11.9)
Concentric remodeling	10(6.3)
Concentric geometry	109(68.1)
Eccentric geometry	22(13.8)
EDV (mean ± SD) ml	187.1±71.4
ESV (mean ± SD) ml	116.0± 51.6
Fractional shortening (mean ± SD)	19.8± 8.7
Ejection fraction (EF) (mean ± SD)	38.7± 13.8
EF Category n (%)	
Reduced EF	114(71.3)
Mid-range EF	21(13.1)
Preserved EF	25(15.6)
Relative wall thickness (RWT) mean ± SD cm	0.37±0.10)
TAPSE mean ± SD cm	1.63± 0.46
LV mass (mean ± SD) g	258.0± 84.5
LV mass indexed to BSA (mean ± SD) g	149.1±50.8
LV mass indexed to height$^{2.7}$ (mean ± SD) g	70.0±25.7
Valvular abnormalities n (%)	
Mitral incompetence	135(84.4)
Tricuspid incompetence	125(78.1)
Aortic incompetence	28(17.5)
Pulmonary incompetence	12(7.5)
Aortic stenosis	4(2.5)
Mitral stenosis	1(0.6)
Tricuspid stenosis	0
Pulmonary stenosis	0
Presence of wall motion abnormality n (%)	48(30.0)
Pericardium pericardial effusion n (%)	84(52.5)
Size of pericardial effusion n (%)	
Nil	84(52.5)
Negligible	59(36.9)
Less than 1cm	8(5.0)
Between 1-2cm	4(2.5)
More than 2cm	5(3.1)
Spontaneous echo contrast n (%)	9(5.6)
Intra-cardiac clots n (%)	2(1.3)

[**] SD-*standard deviation,* QTc -*Corrected QT* IVST(d)- *Interventricular septal thickness(diastole)* IVST(s)- *Interventricular septal thickness(systole)* LVPWT(d)- *Left Ventricular posterior wall thickness(diastole)* LVPWT(s)- *Left Ventricular posterior wall thickness(systole)* LVID (d)- *Left Ventricular internal diameter-diastole* LVID (s)- *Left Ventricular internal diameter-systole* IQR-*Interquartile range* DT- *Deceleration time* IVRT- *Isovolumetric relaxation time* TAPSE-*Tricuspid annulus plane systolic excursion*

Table 5: Univariate analysis results of variables with significant differences between dead and survivors, between readmission after discharge and those not admitted and all composite endpoint 6 months after discharge and those no event

Dead and survivors				
Variables	**Dead**	**Survivors**	**Univariate analysis**	
			Crude HR (95% CI)	***P*-value**
Gender n (%)			2.40(1.03 to 5.56)	0.042**
Male	28(68.3)	56(54.4)		
Female	13(31.7)	47(45.6)		
SBP (mmHg)	107.2±22.1	119.7±22.9	0.94(0.96 to 0.99)	0.006**
Elevated JVP n (%)			1.96(1.16 to 3.32)	0.012**
Yes	37(90.2)	64(62.1)		
No	4(9.8)	39(37.9)		
Hepatomegaly n (%)			1.76(1.12 to 2.74)	0.013**
Yes	33(80.5)	58(56.3)		
No	8(19.5)	45(43.7)		
Ascites n (%)			1.56(1.04 to 2.33)	0.031**
Yes	33(80.5)	56(54.4)		
No	8(19.5)	47(45.6)		
Hyponatraemia n (%)			1.49(1.04 to 2.12)	0.029**
Yes	18(43.9)	26(25.2)		
No	23(56.1)	77(74.8)		
Pericardial effusion/tamponade n (%)			2.78(1.52 to 5.06)	0.001**
Yes	4(9.8)	0		
No	37(90.2))	103(100.0)		
Diabetes Mellitus n (%)			1.61(1.03 to 2.51)	0.037**
Yes	11(26.8)	8(7.8)		
No	30(73.2)	95(92.2)		
Readmission after discharge and those not admitted				
Variables	Readmitted	Not admitted	Univariate analysis	
			Crude HR (95% CI)	*P*-value
Right abdominal pain n (%)			1.35(1.02 to 1.80)	0.039**
Yes	24(63.2)	80(72.1)		
No	14(36.8)	31(27.9)		
SBP mean± SD mmHg	110.1±22.9	121.1±22.7	0.98(0.96 to 0.99)	0.004**
LAD mean± SD cm	4.7± 0.8	4.4± 0.7	1.46(1.01 to 2.10)	0.045**
LVPWT(d) cm	1.1± 0.2	1.0± 0.3	2.94(1.05 to 8.18)	0.040**
Pericardial effusion/Tamponade n (%)			2.17(1.07 to 4.41)	0.032**
Yes	1(4.3)	3(2.0)		
No	26(68.4)	87(78.4)		
Valvular heart disease n (%)			0.32(0.32 to 0.80)	0.015**
Yes	4(10.5)	1(0.9)		
No	34(89.5)	110(99.1)		
All composite endpoint 6 months after discharge and those no event				
Variables	Presence of event	Absence of event	Crude HR (95% CI)	*P*-value
Elderly n (%)			1.68(1.01 to 2.81)	0.047**
Yes	32(57.1)	44(42.5)		
No	24(42.9)	60(57.7)		
Gender n (%)			0.53(0.30 to 0.92)	0.025**
Male	43(76.8)	52(50.0)		
Female	13(23.2)	52(50.0)		
SBP mean ± SD mmHg	111.1±22.8	121.2±22.8	0.98(0.97 to 1.00)	0.007**
Narrow pulse pressure n (%)			1.32(1.02 to 1.72)	0.033**
Yes	33(58.9)	51(49.0)		
No	23(41.1)	53(51.0)		
LVPWT(d) mean± SD cm	1.1±0.3	1.0±0.3	2.88(1.81 to 7.04)	0.020**
Pericardial effusion/tamponade n (%)			2.55(1.53 to 4.24)	<0.0001**
Yes	3(5.4)	2(1.9)		
No	53(94.6)	102		
Uncontrolled hypertension n (%)			0.48(0.24 to 0.97)	0.042**
Yes	2(5.6)	15(14.4)		
No	54(94.4)	89(85.6)		

***Significant p-value Á0.05 SBP- Systolic Blood Pressure JVP- Jugular Venous Pressure*

Table 6: Independent predictors of surviving after 6 months after discharge, readmission after discharge and composite endpoint 6 months after discharge using multivariate analyses among the participants

Independent predictors of surviving after 6 months after discharge using multivariate analyses among the participants					
Variables	B	SE	Wald	adjusted HR (95.0% CI)	P-value
Gender(Male)	1.020	0.439	5.391	2.77(1.17 *to* 6.56)	0.020**
Systolic Blood Pressure (mmHg)	-0.026	0.010	6.497	0.98(0.96 *to* 0.99)	0.011**
Elevated jugular venous pressure (JVP)	0.962	0.544	3.133	2.62 (0.90 to 7.60)	0.077
Hepatomegaly	0.946	0.473	4.006	2.58(1.02 to 6.51)	0.045**
Ascites	0.475	0.427	1.238	1.61(0.70 *to* 3.71)	0.266
Hyponatraemia	0.356	0.385	.855	1.43(0.67 *to* 3.04)	0.355
Independent predictors for readmission after discharge using multivariate analysis					
Variables	B	SE	Wald	Adjusted HR (95.0% CI)	Sig.
Right abdominal pain	0.727	0.305	5.706	2.07(1.14 *to* 3.76)	0.017**
Systolic Blood Pressure (mmHg)	-0.023	0.008	7.989	0.98(0.96 *to* 0.99)	0.005**
Left atrial diameter (cm)	0.268	0.191	1.977	1.31((.90 *to* 1.90)	0.160
LVPWT(d)	0.834	0.565	2.177	2.30(0.76 *to* 6.98)	0.140
Pericardial effusion/tamponade	1.591	0.962	2.735	4.91(0.75 *to* 32.35)	0.098
VHD	0.089	0.683	0.017	1.09(0.29 *to* 4.17)	0.896
Independent predictors for composite endpoint 6 months after discharge by multivariate analysis					
Variables	B	SE	Wald	Adjusted HR (95.0% CI)	*p*-value
Elderly	0.377	0.266	2.016	1.46(0.87 *to* 2.46)	0.156
Gender (Male)	0.732	0.297	6.087	2.08(1.16 *to* 3.72)	0.014**
Systolic Blood Pressure (mmHg)	-0.011	0.007	2.578	1.00(0.98 *to* 1.00)	0.108
Narrow pulse pressure	0.274	0.285	0.924	1.32(0.75 *to* 2.30)	0.336
LVPWT(d)	0.891	0.466	3.648	2.44(0.98 *to* 6.08)	0.056
Pericardial effusion/tamponade	1.669	0.555	9.045	5.31(1.79 *to* 15.74)	0.003**
Uncontrolled hypertension	-1.007	0.737	1.866	0.37(0.09 *to* 1.55)	0.172

*****Significant p-value <0.05   SBP- Systolic blood pressure LAD- Left atrial diameter LVPWT(d)- Left Ventricular posterior wall thickness(diastole) VHD-Valvular heart disease*

The various categories of the variables assessed showed that the following were significantly different between the dead and survivors after six months of follow up, sociodemographic crude hard ratio (HR) (95% confidence interval (CI) gender: 2.40(1.03 to 5.56); *p*=0.042), physical signs (elevated jugular venous pressure: 1.96(1.16 to 3.32); *p*= 0.012), hepatomegaly: 1.76(1.12 to 2.74; *p*=0.013), ascites :1.56(1.04 to 2.12; *p*=0.029), and systolic blood pressure: 0.94(0.96 to 0.99); *p*-value=0.006) (Table 5). Others are laboratory results; hyponatremia:1.49(1.04 to 2.12); *p*=0.026), Echocardiographic finding: presence of pericardial effusion/tamponade: 2.78(1.52(1.52 *to* 5.06); *p*=0.001, presence of co-morbidities: diabetes mellitus: 1.61(1.03 *to* 2.51); *p*=0.037.

During univariate analysis the presence of right abdominal pain: crude HR 1.35(1.02 to 1.80), p-value- 0.039, systolic blood pressure 0.98(0.96 to 0.99), p=0.004, echocardiographic left atrial diameter 1.46(1.01 to 2.10),p=0.045, Ventricular Posterior Wall Thickness (diastole): 2.94(1.05 to 8.18), p=0.040, presence of pericardial effusion/tamponade during echocardiography: 2.17(1.07 to 4.41), p=0.032 and presence of valvular heart disease: 0.32(0.32 to 0.80), p=0.015 were different among survivors (Table 5).

Univariate analysis of composite endpoints(death and readmissions) were been elderly crude HR (95% CI): 1.68(1.01 to 2.81), *p*= 0.04&, male gender: 0.53(0.30 to 0.92), *p*=0.025, systolic blood pressure: 0.98(0.97 to 1.00), *p*=0.007, presence of narrow pulse pressure: 1.32(1.02 to 1.72), *p*=0.033, ventricular posterior wall thickness (diastole): 2.88(1.81 to 7.04), *p*=0.020, presence of pericardial effusion/tamponade: 2.55(1.53 to 4.24), *p*=<0.0001 and presence of uncontrolled hypertension: 0.48(0.24 to 0.97), *p*=0.042 Systolic Blood Pressure (mmHg) (Table 5).

Multivariate analysis showed that the independent predictors for death outcome after 6 months of discharge for AHF and the adjusted hazard ratio (95% CI) are gender(male) 2.77(1.17 to 6.56); *p*=0.020, systolic Blood Pressure (mmHg) 0.98(0.96 to 0.99); p=0.011, the presence of hepatomegaly 2.58(1.02 to 6.51); p=0.045 (Table 6).

Independent predictors for readmission or rehospitalization within 6months after discharge were presence of right abdominal pain during admission adjusted HR (95% CI): 2.07(1.14 to 3.76), p=0.017, systolic blood pressure 0.98(0.96 to 0.99), p= 0.005 (Table 6).

Independent predictors from multivariate analysis are male gender: adjusted hazard ratio (HR): 2.081.16 to 3.72, p= 0.014 and pericardial effusion and tamponade: 5.31(1.79 to 15.74, p=0.003(Table 6).

DISCUSSION

Overall, this study showed the importance of systolic blood pressure(SBP) as an independent predictor of survival six months after admission and readmission after baseline hospital discharge. Although, it had no effect on the composite endpoints (mortality and readmission) six months after discharge. Furthermore, this study showed that variables such as hepatomegaly, right abdominal pain, and pericardial effusion/tamponade associated with congestion also play an important role in predicting the outcome of AHF after 180 days of discharge.

Low SBP, male gender, and hepatomegaly were independent predictors of AHF survival six months after discharge. The predictive role of SBP, a readily accessible vital sign at admission and on AHF post-discharge during clinic visits has been previously noted.[15,16] It has also been noted to play a great role in morbidity and mortality outcomes among black patients with AHF.[15] Elevated blood pressure at admission is associated with the likelihood of better response to treatment during admission.[15]

Systolic blood pressure was also an independent factor for readmission/rehospitalisation in this study population similar to previous studies.[15,16] SBP was also found to be statistically significant during univariate analysis in the Romanian Acute Heart Failure Registry (RO-AHF); however, this was not statistically significant after multivariate analysis.[17] Similar finding was documented in Abeokuta Heart failure registry and THESUS-HF registry.[18] The role of elevated SBP in readmission calls for better post-discharge care among patients with AHF, especially through the provision of HF home care and continuous optimization of therapy cannot be underemphasised.

Lower survival in males with AHF have been highlighted in previous studies.[19-21] The explanation for better survival among females after the onset of heart failure is probably because they have a better ventricular function and more frequently suffer from preserved HF compare to males.[15] Females in this study also had a higher mean EF, and a greater proportion had preserved EF compared to males.[3,22] Other explanations for better survival in females with HF found in this study include a lower burden of AF and ventricular arrhythmias.[3, 22] Notwithstanding that our study included a similar proportion of males and females.[3] Furthermore, females with non-ischaemic HF

are noted to have an attenuated sympathetic activation and parasympathetic withdrawal compared with men with its attendant prognostic implications.[22]

Evidence or markers of congestion, such as right abdominal pain, were independent predictors of survival, readmission/rehospitalization, and all-cause events. These are not unconnected with the key pathophysiology associated with AHF, which is congestion and the major reason for admission or readmission.[23, 24] Furthermore, systemic congestion in AHF predispose to multi-organ dysfunction and activation of neurohormonal system which is already activated above the physiological baseline level early during disease progression in patients with chronic HF.[16] This role of congestion suggest that adequate physical examination among AHF especially attempt to identify all HF features that maybe congestive in nature cannot be underestimated.[25]

Longer follow-up studies on prognosis in this setting are required to provide additional insights into patient prognosis. Given the limited contribution of ECG and echocardiography to prognosis determination in AHF, the importance of exploring the roles of biomarkers, particularly cardiac biomarkers, such as cystatin, natriuretic peptides, and troponin cannot be overemphasised.

The results of this study will aid in the efficient management of AHF, particularly in low-resource environments, such as Nigeria and black subjects. Additionally, knowledge of the clinical and laboratory predictors would help in the creation of prevention, outcome improvement, and adverse outcome reduction strategies for AHF in Nigeria. Finally, knowing the prognosis at six months would add to the knowledge about enhancing clinical outcomes for AHF among black patients and improving survival strategies.

This study has some limitations. First, because this study was conducted at a single tertiary centre, the results may not be applicable to primary and secondary institutions in the same region, and it may not have adequately captured the rural patient population, although the institution is a major tertiary institution in south-western Nigeria. This limitation may have reduced the generalisability of the results to some extent. Secondly, the study could be influenced by unmeasured confounding variables, such as socioeconomic status, patient preference, and even post-hospital care, despite the fact that efforts were made to investigate a broad range of sociodemo-graphic, bio-humoral or laboratory, electrocardio-graphic, echocardiographic, and therapeutic variables.

Finally, BNP testing was not included in the HF diagnosis and follow-up.

CONCLUSION
Overall, the study provided an understanding of the factors contributing to HF admissions in a population in South-Western, Nigeria. A better understanding of AHF will help with the development of the protocol for trials to improve the prognostic outcome of AHF. The study also highlighted the relevance of clinical profile, particularly systolic blood pressure, has shown in previous studies

Statements and Declarations
The authors have no financial or non-financial interests that were directly or indirectly related to the work submitted for publication to declare.

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