Afr. J. Med. Med. Sci. (2024) **53,** 39-48

Arrhythmia Burden in Heart Failure Patients in a Referral Centre in Southern Nigeria

AO Ajala*[1], S Dodiyi-Manuel[1], B Oyan[2] and MR Akpa[1]
[1]*Department of Internal Medicine, University of Port Harcourt Teaching Hospital, Rivers State, Nigeria.*
[2]*Department of Internal Medicine, Rivers State University Teaching Hospital, Rivers State, Nigeria.*

Abstract
Background: Heart failure is the ultimate effect of all cardiovascular disorders with a rapid increase in its prevalence. The risk of sudden death is high in heart failure and has been attributed to progressive pump failure and arrhythmias especially ventricular arrhythmia.
Objectives: This study aims to determine the arrhythmia burden in heart failure patients
Methodology: Ninety adults with heart failure were assessed. A 24-hr Holter Electrocardiographic monitoring was done using a 12 - channel Edan® Holter recorder, trans-thoracic echocardiography and rest ECG was performed on all subjects. Continuous variables were compared by the student's t- test while categorical parameters were compared with the chi-square test or two tailed Fisher's exact test as appropriate. Pearson's correlation coefficient was used to assess the relationship between echocardiographic parameters against the number of arrhythmia/hours on Holter ECG.
Results: The mean age of the subjects was 49.8±14.4years with a female preponderance of 1.4:1. Arrhythmias such as isolated ventricular ectopic (100%), isolated atrial ectopics (91.1%) non-sustained ventricular tachycardia (16.7%) and atrial fibrillation (20%) were noted on Holter ECG. There was a significant positive correlation between the left ventricular internal wall diameters in diastole (LVIDd) and the number of VE/hour (p = <0.001) with a negative correlation between the ejection fraction and the number of VE/hour (p = 0.023).
Conclusion: The frequency of arrhythmias is high in heart failure patients. Left ventricular systolic dysfunction and chamber size were major determinants of arrhythmias and could be used in identifying high-risk patients in a bid to initiate preventive measures if necessary.

Keywords: *arrhythmias, acute, cardiovascular, chronic, heart failure, ventricular.*

Correspondence: Dr Aisha O. Ajala, Department of Internal Medicine, University of Port Harcourt Teaching Hospital, Rivers State, Nigeria. E-mail: ajalabunmi@gmail.com, +2348173400065

Abstrait
Contexte: L'insuffisance cardiaque est l'effet ultime de tous les troubles cardiovasculaires avec une augmentation rapide de sa prévalence. Le risque de mort subite est élevé dans l'insuffisance cardiaque et a été attribué à une défaillance progressive de la pompe et à des arythmies, en particulier une arythmie ventriculaire.
Objectifs: Cette étude vise à déterminer le fardeau de l'arythmie chez les patients insuffisants cardiaques
Méthodologie: Quatre-vingt-dix adultes souffrant d'insuffisance cardiaque ont été évalués. Une surveillance électrocardiographique Holter de 24 heures a été effectuée à l'aide d'un enregistreur Edan® Holter à 12 canaux, une échocardiographie transthoracique et un ECG de repos ont été effectués sur tous les sujets. Les variables continues ont été comparées par le test t de Student, tandis que les paramètres catégoriels ont été comparés au test du chi carré ou au test exact de Fisher à deux queues, selon le cas. Le coefficient de corrélation de Pearson a été utilisé pour évaluer la relation entre les paramètres échocardiographiques et le nombre d'arythmies/heures sur Holter ECG.
Résultats: L'âge moyen des sujets était de 49.8 ± 14.4 ans avec une prépondérance féminine de 1.4:1. Des arythmies telles que des ectopiques ventriculaires isolées (100%), des ectopiques auriculaires isolées (91.1%), une tachycardie ventriculaire non soutenue (16.7%) et une fibrillation auriculaire (20%) ont été notées sur l'ECG Holter. Il y avait une corrélation positive significative entre les diamètres de la paroi interne du ventricule gauche en diastole (LVIDd) et le nombre de VE/heure (p=<0,001) avec une corrélation négative entre la fraction d'éjection et le nombre de VE/heure (p = 0.023).
Conclusion: La fréquence des arythmies est élevée chez les insuffisants cardiaques. La dysfonction systolique ventriculaire gauche et la taille de la chambre étaient des déterminants majeurs des arythmies et pourraient être utilisées pour identifier les patients à haut risque dans le but d'initier des mesures préventives si nécessaire.

Background

Heart failure has become a growing public health issue of concern with an estimated prevalence of 37.7 million globally and associated increased morbidity and mortality and confers a substantial burden on the health-care system.[1] In the past few decades, heart failure has emerged as a major public health problem in developed countries. The prevalence of HF is approximately 1–2% of the adult population in developed countries, rising to e"10% among people >70 years of age. [2] The prevalence continues to rise because of increasing cardiovascular risks burden and an ageing population.

Heart failure ranks among the major causes of death of cardiovascular origin in Africa. Hospital death from congestive heart failure (CHF) in Africa ranges from 9% to 12.5%.[3] The major causes of HF in sub-Saharan Africa and Nigeria are largely hypertension, cardiomyopathy, and rheumatic valvular heart disease.[4] Although HF management has benefited from major advances in recent years, case-fatality among people with heart failure remains high worldwide.

The primary causes of death in heart failure patients are progressive pump failure or sudden cardiac death secondary to ventricular arrhythmias. The morbidity also remains poor, with a high rate of re-hospitalization (up to 50% a year), placing a significant burden on national healthcare systems. Poor prognostic indices include black race, structural heart disease, high New York Heart Association (NYHA) class (III and IV), electrolyte abnormalities, systolic/diastolic dysfunction, and arrhythmias among many others.[5] Significant arrhythmias are independent risk factors for the progression of HF and sudden cardiac death.[6]

Supraventricular and ventricular arrhythmias are common in HF patients and can exacerbate the HF symptoms by decreasing the effective cardiac output, as well as complicating the management of these patients.[7] Ventricular arrhythmias are found in up to 45% of patients with severe HF and are also responsible for an increased risk of re-hospitalization in one-third of HF patients.[8] The management strategy depends on the type of arrhythmia, the underlying structural heart disease and the severity of heart failure; with their control requiring pharmacological, electrical, or catheter-based intervention.[7]

HF hospitalizations are increasing, and many of these may be related to supraventricular arrhythmias (SVAs) such as atrial fibrillation (AF) which contributes to an increased risk of stroke and thromboembolism.[9] Sudden cardiac death (SCD) is also a major cause of mortality among HF patients and is commonly related to cardiac arrythmias particularly ventricular arrhythmias (VAs). Bradyarrhythmia are also common in HF, and these includes sinus node dysfunction, conduction disease and tachy-brady syndrome.[10]

Pathogenesis of arrhythmia in heart failure

There are multiple factors responsible for the frequency of arrhythmias in patients with heart failure; underlying structural disease, mechanical factors, neuro-hormonal factors, electrolyte abnormalities, ischemia and drugs are the common operating mechanisms.

Underlying structural disease - Extensive myocardial damage, fibrosis and focal areas of post-infarct scar or the loss of cell-to-cell coupling in patients with dilated cardiomyopathy provides the proper substrate for re-entry, the mechanism thought to be responsible for most ventricular arrhythmias due to anisotropy. Chamber Hypertrophy and Stretch in patients with HF also contribute to numerous compensatory mechanisms which are active to improve cardiac output, but these mechanisms can contribute to arrhythmogenic milieu.[8]

Mechanical factors - From a mechanistic standpoint, the structural changes that occur in patients with advanced stages of HF, including fibrosis, regional ventricular hypertrophy, and changes in electrophysiologic properties (electromechanical feedback) of the myocardial tissue promotes the development and maintenance arrhythmias in heart failure.[8]

Neurohormonal Activation: - The increased plasma levels of adrenaline and noradrenaline plays an important role in the pathophysiology of HF contribute to the development of arrhythmias. Cao and colleagues describe an association between the density of nerve sympathetic fibers (which are increased in patients with HF) and the risk of VAs.[11]

Electrolytes Abnormalities: - Hyperkalemia is common in patients with HF and is often linked to the use of ACE inhibitors, angiotensin receptor blockers and aldosterone antagonists. Hyperkalemia slows the ascent phase of action potential leading to atrioventricular block. Diuretic-induced hypokalemia and hypomagnesemia and from increased activity of the renin-angiotensin system results is a more automaticity in Purkinje fibers and a rapid increase repolarization, with consequent onset of VT/VF. [12]

Pharmacologic Agents: - The drugs that interfere with neuro-hormonal system can often contribute to the pro-arrhythmic milieu. Diuretic therapy, ACE inhibitors and aldosterone antagonists often cause electrolyte imbalances that contribute significantly to arrhythmogenesis and can increase the proarrhythmic effect of other drugs. The use of inotropic agents in patients with acute HF and hypoperfusion may further increase the risk of arrhythmias. [13]

The aim of this study was to identify the burden of arrhythmia in patients admitted for acute heart failure and its association with structural and functional cardiac abnormalities.

Methodology

Study site

The study was carried out at the medical wards and medical out-patient department of the University of Port Harcourt Teaching Hospital, Rivers State, Nigeria. The hospital is a tertiary hospital located in Port Harcourt and serves as a referral medical centre for neighboring states.

Participants

The study population consisted of ninety adults (90) admitted into the medical wards with a confirmed diagnosis of acute HF recruited via a consecutive sampling method. Patient recruitment was done within one week of admission into the hospital. Acute heart failure was defined as a rapid onset of new or worsening signs and symptoms of pre-existing HF. Heart failure was diagnosed using the Framingham Criteria [14] and confirmed by a trans-thoracic echocardiography after meeting the eligibility criteria as follows: Adults aged 18 years and above and who have given informed consent. All HF patients irrespective of their ejection fraction were recruited into the study. Patients who refused to give informed consent were excluded from the study. Ninety healthy subjects with no previous or present history of HF were recruited as controls.

Procedure

A structured questionnaire was administered by the investigators to collect demographic information and disease related variables from the subjects. A detailed physical examination was conducted to determine weight, height, abdominal circumference, and blood pressure. A 24-hr Holter Electrocardiographic monitoring was done using a 12 - channel Edan® Holter recorder and in compliance with the American College of Cardiology/American Heart Association guidelines for Ambulatory ECG (AECG).[15] The skin over the electrode area was shaved for patients who were very hairy after consent was sort and thoroughly cleansed with an alcohol swab, the subjects were encouraged to undertake their usual daily activities but to avoid bathing or bodily contact of water with the electrodes, the Holter ECG was disconnected after 24 hours and thereafter results were transferred to a computer and analyzed using the software of the Holter machine. Parameters such as Trend analysis (of heart rate and ST-segment deviations), underlying rhythm, arrhythmias (including ectopic beats, tachyarrhythmia, bradyarrhythmia) [15] were assessed.

Echocardiography was performed using a Mindray® ultrasound machine model DC- N6 equipped with 2.5MHz transducer. Two- dimensional (2D) M-mode, pulse-wave, continuous wave, and color Doppler echocardiography assessment was done with the subject in left lateral decubitus position and targeted echocardiographic estimations were taken according to the recommendations of the American Society of Echocardiography.[16] LV Systolic function was calculated using the biplane method. LV mass was calculated using the formula of Devereux and Reichek.[17] LV geometry was defined according to standard criteria.[17] Left atrial dimension and area were measured using standard methods and a value of $>34ml/m^2$ considered abnormal in accordance with the ASE.[16] Trans-mitral flow velocities were obtained with the Doppler sample volume placed just beyond the tip of mitral valve leaflets, and standard measurements were obtained.[18] Tissue Doppler imaging was applied to identify true pseudo-normal filling pattern.[18]

Statistical analysis

Data was analyzed using Statistical package for social sciences package 22 (SPSS- 22). Results were presented as means, standard deviation, ranges, and medians as appropriate for continuous variables while categorical variables were expressed as proportions or percentages. Graphs and tables were used to illustrate results where appropriate. Continuous variables were compared by the student's t- test, while proportions or categorical parameters were compared with the chi-square test or two tailed Fisher's exact test as appropriate. Pearson's correlation coefficient was used to assess the linear relationship between echocardiographic parameters such as LVIDd, LAVI and EF against the number of arrhythmia/hours on Holter ECG (SVE and VE). A p value of less than 0.05 was considered statistically significant.

Ethical Consideration

Ethical approval was obtained from the Ethics and Research Committee of the University of Port Harcourt Teaching Hospital (UPTH) prior to commencement of the study.

Results

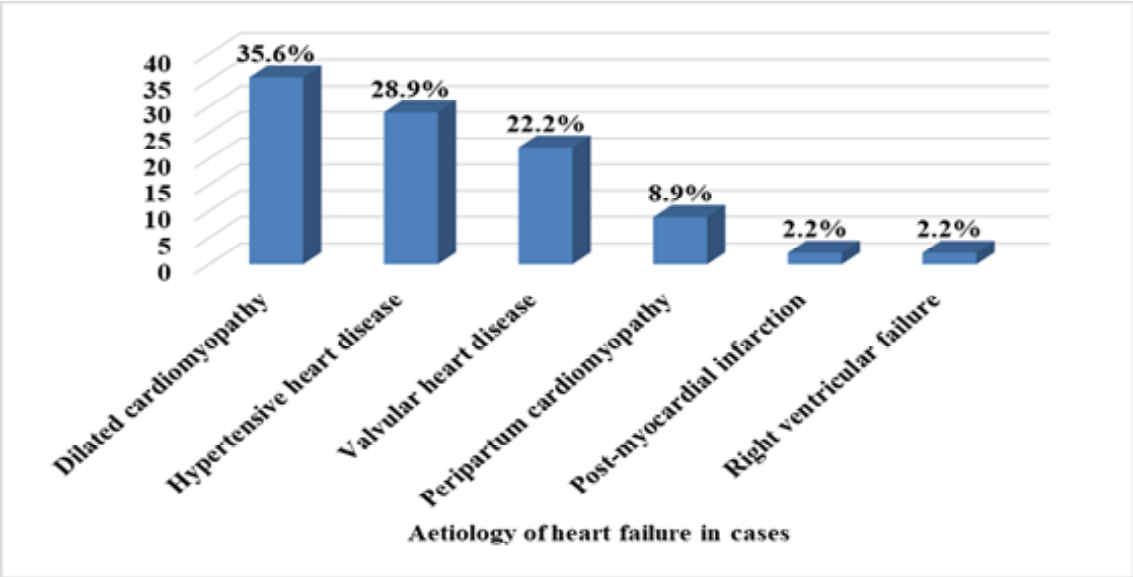
A total of 180 subjects were included in this study of which 90 constituted the cases with heart failure and 90 healthy age and sex matched controls. The mean age of the study cohorts was 49.8± 14.4 years (range 27 - 72 years). Most of the cohorts were between 40 - 59 years (Table 1). There was a female preponderance among the study cohorts with a female to male ratio of 2:1. The mean systolic and

diastolic blood pressure (SBP & DBP) among the cases was significantly lower than the healthy controls, 112.9±15.6mmHg versus 129.1 ±8.03mmHg for SBP (p=0.001) and 70.9 ± 6.59mmHg versus 82.6 ± 4.6mmHg for DBP (p = 0.001). The etiology of HF in the cases were dilated cardiomyopathy seen in 35.6%, HHD in 28.9%, valvular heart disease (rheumatic and degenerative) in 22.2%, peripartum cardiomyopathy in 8.9% and post-M.I HF in 2.2% of cases. About 85% of the cases were presenting with an acute decompensation of a CHF while the remaining 14 (15.6%) had de-novo heart failure (figure 1).

Table 1: Baseline characteristics of the study population

Variable	Cases n=90 (%)	Controls n=90 (%)	Total n=180 (%)	Chi-square (p- value)
SEX				
Female	52 (57.8)	60 (66.7)	112(62.2)	1.51 (0.2187)
Male	38 (42.2)	30 (33.3)	68 (37.8)	
AGE GROUP (years)				
20 - 29	8 (8.9)	9 (10.0)	17 (9.4)	
30 - 39	17 (18.9)	19 (21.1)	36 (20.0)	
40 - 49	18 (20.0)	19 (21.1)	37 (20.6)	0.64 (0.9856)
50 - 59	26 (28.9)	26 (28.9)	52 (28.9)	
60 - 69	13 (14.4)	11 (12.2)	24 (13.3)	
≥ 70	8 (8.9)	6 (6.7)	14 (7.8)	
EDUCATIONAL STATUS				
None	0 (0.0)	1 (1.0)	1 (0.5)	
Primary	18 (20.0)	16 (18.0)	34 (19.0)	2.68 (0.8470)
Secondary	42 (47.0)	47 (52.0)	89 (49.5)	
Tertiary	30 (33.0)	26 (29.0)	56 (31.0)	
MARITAL STATUS				
Single	17 (18.9)	23 (25.6)	40 (22.2)	
Married	57 (63.3)	52 (57.8)	109 (60.6)	3.82 (0.3498)
Separated	1 (1.1)	1 (1.1)	2 (1.1)	
Divorced	4 (4.4)	5 (5.6)	9 (5.0)	
Widowed	11 (12.2)	9 (10.0)	20 (11.1)	
BMI CATEGORY				
Underweight	4 (4.4)	2 (2.2)	6 (3.3)	4.32 (0.319)
Normal	59 (65.6)	56 (62.2)	115 (63.9)	
Overweight	16 (17.8)	23 (25.6)	39 (21.7)	
Obese	11 (12.2)	9 (10.0)	20 (11.1)	
BLOOD PRESSURE	**Mean±SD**	**Mean±SD**	**Student-t test.**	
SBP (mmhg)	112.9±15.6	129.1 ±8.03	0.001*	
DBP (mmhg)	70.9 ± 6.59	82.6 ± 4.6	0.001*	

**Shows statistically significant (p < 0.05) SBP = systolic blood pressure ; DBP = diastolic blood pressure*



Holter electrocardiographic findings among study subjects.

As shown in table 2, the total heart rate in 24 hours was higher among the cases 140511 ± 139847 than the controls 113433 ± 19315 although this difference was not statistically significant (p= 0.189). However, there was a statistically significant difference in the maximum and minimum heart rates between the cases and healthy controls (p value = 0.0001 and 0.039 respectively). The total supraventricular ectopic and ventricular ectopic were also significantly higher among the cases than the control group (p = 0.0001). The standard deviation of normal-to-normal R-R intervals (SDNN) was also significantly lower in the cases than in the controls (p = 0.0042). The burden of isolated ventricular and atrial ectopics were greater than 10% in 22.2% and 17.8% of the cases. Table 3 shows the prevalence of rhythm abnormalities among the cases when rest ECG was compared with Holter ECG. The use of Holter ECG at detection of arrhythmias was statistically significant when compared to rest ECG.

Table 2. 24-hour Holter electrocardiographic findings among study subjects

Holter findings	Cases n =90(%)	Controls n =90(%)	p value
Total QRS Beats	140511±139847	113433±19315	0.1892
Max HR	168.5±53.2	135.8±34.5	0.0001*
Min HR	54.8±22.4	64.2±20.2	0.0391*
Average HR	84.7±17.0	82.1±18.1	0.5505
Total SVE	2017.6±690.9	97.4±42.8	0.0001*
SVE/hour	82.6±62.1	6.8±9.7	0.0049
SVE Bigeminy	70.4±33.9	2.77±6.1	0.0043*
SVT	22.8±15.2	0.3±0.8	0.0144*
Total VEVE/hour	3089.1±941.3137.1±22.9	140.6±88.66.3±5.7	0.0001*0.0001*
VE Bigeminy	111.1±48	1.15±2.9	0.2015
VTD (secs)	5.7±3.0	0.1±0.01	0.0001*
SDNN	83.5±63.1	117.6±44.4	0.0042*
TRI Index	132.3±109.6	262.2±104.4	0.0001*

**Shows statistically significant (p < 0.05) HR- heart rate; SVE =supraventricular ectopics, SVT – supraventricular tachycardia, VE = ventricular ectopics, VTD – ventricular tachycardia duration, SDNN – standard deviation of normal-to-normal R-R intervals*

Table 3: Comparison of frequency of arrhythmias in the cases.

Rhythmabnormalities	Holter ECG n = 90	Rest ECG n =90	Chi-square(p-value)
Atrial fibrillation	18 (20.0)	8 (8.9)	4.49 (0.033)*
Bradycardia	10 (11.1)	3 (3.3)	4.06 (0.044)*
Atrial ectopic	82 (91.1)	15 (16.7)	128.5 (0.0001)*
Atrial bi/trigeminy	24 (26.7)	2 (2.2)	21.75 (0.0001)*
Isolated VES	90 (100.0)	17 (18.8)	122.8 (0.0001)*
Couplets	39 (43.3)	5 (5.6)	34.77 (0.0001)*
Ventricular bigeminy	27 (30.0)	2 (2.2)	25.69 (0.0001)*
NSVT	15 (16.7)	0 (0.0)	16.36 (0.0001)*

**Statistically significant; VES = ventricular ectopics ; NSVT = non-sustained ventricular tachycardia*

Echocardiographic parameters among study subjects

There was a significant difference between the mean left ventricular mass (LVM) and left ventricular mass index between the cases and controls (p = 0.0001). The left atrial volume index (LAVI) was significantly higher among the cases when compared with the controls (p = 0.0001). The mean left ventricular systolic function assessed with the ejection fraction (EF) and fractional shortening (FS) was significantly lower among the cases. (Table 4) Fifty –three of the cases had eccentric hypertrophy while 37 had concentric hypertrophy. Majority of the controls had either normal geometry or concentric remodeling (46.7% and 35.6% respectively) See Table 5. The majority of the cases 35 (38.9%) had grade 3 diastolic dysfunction, followed by grade 2 and 4 diastolic dysfunction in 27.8% and 24.4% respectively while the diastolic function was normal in 64 (72.7%) of the controls. There was a positive correlation between the mean LAVI and the number of SVE/hour, however this was not statistically significant (r = 0.14,

Table 4: Mean echocardiographic and ECG parameters among study subjects

Echo Findings	Cases (n = 90) Mean ±SD	Controls (n = 90) Mean ±SD	p value
AVD (cm)	1.8 ±0.2	1.9 ±0.2	0.4093
LAD (cm)	4.9 ±0.4	3.3 ±0.4	0.0001*
AODd (cm)	2.8 ±0.4	2.4 ±0.3	0.0002*
LAVI (ml/m²)	49.4±24.6	23.0±4.7	0.0001*
E (ms)	108.8 ±21.7	71.7 ±16.1	0.0001*
A (ms)	49.8 ±19.4	42.3 ±10.8	0.0598
E/A	2.5 ±1.0	1.9 ±0.7	0.0079*
E/e'	18.1±5.6	7.1±1.9	0.0001*
EDV (ml)	242.4 ±132.1	108 ±29.5	0.0001*
ESV (ml)	176.3 ±112.2	29.0 ±12.5	0.0001*
SV (ml)	66.8 ±33.5	82.6 ±20.6	0.0128*
LVEF (%)	36.8 ±15.4	71.1 ±9.2	0.0001*
LVFS	18.5 ±8.4	0.4 ±0.1	0.0001*
IVSD (cm)	1.09 ±0.4	0.9 ±0.2	0.0498*
LVIDd (cm)	6.4 ±1.3	4.7 ±0.5	0.0001*
LVPWd (cm)	1.2 ±0.3	0.9 ±0.1	0.0001*
LVM (g)	377.2 ±144.8	161.5 ±29.5a	0.0001*
LVMI (g/m²)	207.4 ±82.2	94.6 ±19.5[a]	0.0001*
RWT	0.4 ±0.1	0.4 ±0.1[b]	0.6278
TAPSE (cm)	1.6 ±0.8	2.1 ±0.3[a]	0.0001*

**Statistically significant. LAD = left atrial diameter; AOD = aortic root diameter; IVSd=interventricular septal thickness in diastole; LVIDd= left ventricular internal diameter in diastole; LVPWd= left ventricular posterior wall thickness in diastole; LVM= left ventricular mass; LVMI= left ventricular mass indexed to body surface area; RWT = relative wall thickness; TAPSE = tricuspid annular plane systolic excursion; EDV = end-diastolic volume; ESV = end-systolic volume; SV= stroke volume; LVEF = left ventricular ejection fraction; LVFS = left ventricular fractional shortening; E = early diastolic filling; A = atrial contraction; E/A = ratio of early (E) to late (A) diastolic filling velocities in mitral inflow.*

P = 0.053). The mean EF had a significant negative correlation with the mean VE/hour (r = -0.03, p = 0.023) while the mean LVIDd also had a significant positive correlation with the mean VE/hour (r = 0.03, p = 0.001).

Table 5: Distribution of LV Geometry among study subjects

LV Geometry	Case n = 90(%)	Control n =90(%)	Chi-square (p- value)
Concentric hypertrophy	37 (41.1)	4 (8.9)	15.11 (0.0005)*
Eccentric hypertrophy	53 (58.9)	4 (8.9)	35.5 (0.0001)*
Concentric remodeling	0 (0.0)	16 (35.6)	52.6 (0.0001)*
Normal geometry	0 (0.0)	21 (46.7)	71.3 (0.0001)*

**Statistically significant.*

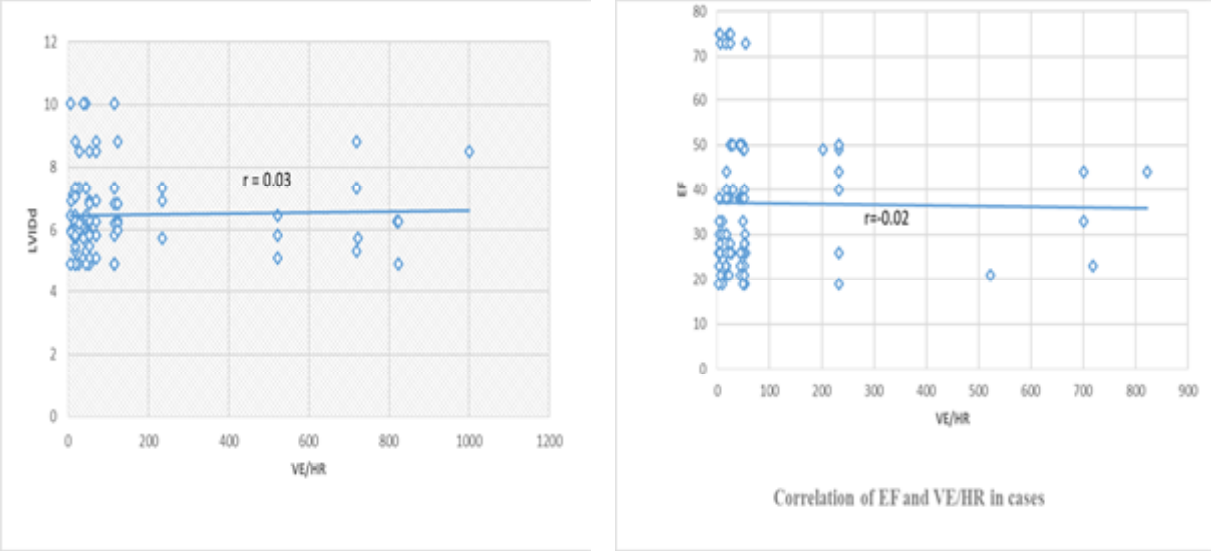


Fig 2: Scatter diagram showing correlation between mean LVIDd/EF against number of Ventricular ectopics/hour (VE/HR)

Discussion

The mean age of study subjects was found to be about 50yrs, African heart failure patients in the THESUS-HF study had a mean age of 52 years [19]. Caucasians' HF patients have however been found to have a higher mean age according to data from the EHFS, ADHERE and OPTIMIZE HF studies [20-22]. The younger mean age of onset of HF in blacks when compared to Caucasians counterparts have been attributed to early onset of hypertension in blacks as this accounts for the major etiology of HF in these population.

Acute decompensation of chronic heart failure (CHF) tends to be more common than de-novo HF and it accounted for 84% of HF amongst the cohort. Ogah et al, [23] in a study done in a tertiary Hospital in the South- Western part of Nigeria to determine the profile of AHF patients found 91.8% to have acute decompensation of CHF. Acute decompensation of chronic heart failure accounts for 80% of AHF admissions which may be due to easily identifiable precipitants like chest infections, arrhythmias, electrolytes imbalance and poor adherence. However, no clear explanation for decompensation might be seen in some of the patients. Precipitants for decompensation noted in our study cohorts were chest infections, arrhythmias, and poor adherence to anti-failure therapy.

Hypertensive heart disease tends to be the predominant cause of heart failure in Africans according to the THESUS-HF study [19]. However, dilated cardiomyopathy was found to be slightly more predominant cause of HF in our cohorts at 35.6% followed closely by HHDx. The commonest cause of HF in Nigeria remains HHDx with DCM or rheumatic heart disease accounting for the second commonest. Data from Abeokuta heart failure registry identifies hypertensive heart disease as the commonest cause of HF with a high prevalence of 75.6% among acute HF patients [23].

A few studies have also found DCM as a prevalent cause of HF, Lasisi et al found a higher

prevalence of DCM (53.3%) among CHF patients when assessing the prevalence of ventricular arrhythmias and heart rate variability in chronic HF patients [24]. Alcoholic DCM due to the high consumption of alcohol in the South-South region of Nigeria of which Rivers state is included could explain the high prevalence of DCM in our cohorts [25]. The small sample size of this study may also be the reason for this finding.

The prevalent arrhythmias in the study cohorts includes ventricular ectopic , atrial ectopic , ventricular bigeminy or couplets, Non-sustained ventricular tachycardia and atrial fibrillation. Cardiac arrhythmias are more commonly found in HF when compared with normal subjects. These arrythmias are common in patient with structural heart disease. Increased risk of ventricular arrhythmias is found in patients with structural heart disease especially in patients with heart failure with reduced EF [6] [24] [26] Resting ECG remains essential in the management of HF patients, however Holter monitoring serves as a valuable tool in risk stratification and prognostication of HF patients.

Twenty percent of the cohorts had AF. The presence of AF adversely affects mortality of HF patients. Atrial fibrillation can be paroxysmal, persistent, or permanent. Patients with paroxysmal AF are usually missed when a rest ECG is used [27] The overall prevalence of AF in HF according to Wang and colleagues in a prospective study of about 1500 patients recruited in the Framingham' study is between 13 – 41%. [28]

More than 90% of the HF patients had atrial and ventricular ectopics. A quarter of the patients in the Euro Heart failure Survey (EHFS II), a large survey involving 115 hospitals from 25 different countries were found to have SVES [21] Atrial Fibrillation (AF) however remains the most studied supraventricular arrhythmia in HF patients. Atrial fibrillation can precipitate or can be a consequence of HF, it can cause a further reduction in CO due to rapid ventricular rates, loss of atrial contraction, abnormal ventricular filling, and tachycardia- induced cardiomyopathy. The presence of AF in HF patients further increases the risk of thromboembolism. The number of VES/hours was associated with a lower mean EF, 137 VES/hr. was found in this study however Lasisi and colleagues found 226 VES/hr. The mean EF was 37 vs 30% respectively in both studies. Ejection fraction was also found to correlate better with ventricular arrhythmias than LVMI [24]. The length of NSVT rather than the rate is associated with the risk of major arrhythmic events.[29] The EHFS II found VT to be the precipitating arrhythmia of HF in 4.1% of patients while atrial arrhythmias, majorly AF, accounted for 29%. [21] About 17% of the cases had NSVT.

Heart failure patients can develop a range of bradyarrhythmia including various degrees of AV block, ventricular conduction delay and sinus node dysfunction. The prevalence of Brady-arrhythmias in this study cohorts was generally low. [30] Bradyarrhythmia were reported in 6% of the EHFS study subjects with up to 50% requiring a permanent pacemaker [21].

A significant correlation of arrhythmias with left atrial and ventricular structure as well as function was demonstrated in this study. Tsang et al in a multivariate analysis found LAVI to be independently associated with cardiovascular risk score, AF, transient ischemic attack, and history of smoking [31]. This study also showed that a larger LA volume was associated with a higher risk of AF in older patients. Lower EF values and chamber dilatation were shown to increase the likelihood of developing frequent premature ventricular complexes in this study. A multivariate analysis of premature ventricular complexes (PVCs) with causal predictors in the ARIC study found African descent, male sex and an increased LVMI to be related to PVCs prevalence [32]. This study found isolated ventricular and atrial ectopics burden to be greater than 10% in about 20% of the heart failure patients. A high PVC burden of greater than 10% had been shown to be an indication for comprehensive cardiac evaluation and an even higher burden of more than 20% results in arrhythmia-induced cardiomyopathy with further worsening of underlying structural heart disease [33].

Conclusion

Arrhythmias occur frequently in HF patients. The severity of LV systolic dysfunction and chamber dilatation are predictors for the development of arrhythmias. Sudden cardiac death due to arrhythmias remains a significant cause of mortality in heart failure patients especially in resource poor setting like ours. Routine use of Holter ECG to aid risk stratification is essential in heart failure patients, especially in those with impaired systolic function. Early risk stratification to identify high risk patients who will benefit from necessary preventive measures is vital in reducing morbidity & mortality.

Limitations of the study

The results of this study may be biased by the relatively small sample size.

Disclosure of conflict of interest

The authors declare no conflict of interest.

Acknowledgements

None

Statement of Ethical approval

Ethical approval was given by the University of Port Harcourt Teaching Hospital Ethical Committee and the Research Ethics group of the Centre for Medical Research and Training, College of Health Sciences, University of Port Harcourt. (UPTH/ADM/90/S. II/ VOL.XI/500).

References

1. Ziaeian B, Fonarow GC. Epidemiology and aetiology of heart failure. Nat Rev Cardiol [Internet]. 2016 3;13(6):368–78.
2. Mosterd A, Hoes AW. Clinical epidemiology of heart failure. Heart . 2007 ;93(9):1137–46.
3. Nwafor CE, Alikor CA. Pattern of cardiovascular disease admissions in the medical wards of university of Portharcourt Teaching Hospital. Niger Heal J. 2016;16 (2)
4. Ojji DB, Alfa J, Ajayi SO, Mamven M, Falase AO. Pattern of heart failure in Abuja , Nigeria: an echocardiographic study. Cardiovasc J Afr. 2009;20(6):349–52.
5. Levy WC, Mozaffarian D, Linker DT, Sutradhar SC, Anker SD, Cropp AB, et al. The Seattle Heart Failure Model. Circulation . 2006 Mar 21;113(11):1424–33.
6. Ajayi O, Abiodun O, Akintomide A, Adebayo R, Ogunyemi S, Balogun M, et al. Pattern of arrhythmias among Nigerians with congestive heart failure. Int J Gen Med [Internet]. 2015;8:125.
7. Masarone D, Limongelli G, Rubino M, Valente F, Vastarella R, Ammendola E, et al. Management of Arrhythmias in Heart Failure. J Cardiovasc Dev Dis 2017, Vol 4, Page 3 . 2017 ;4(1):3.
8. Santangeli P, Rame JE, Birati EY, Marchlinski FE. Management of Ventricular Arrhythmias in Patients With Advanced Heart Failure. J Am Coll Cardiol. 2017 11;69(14):1842–60.
9. Saltzman HE. Arrhythmias and Heart Failure. Cardiol Clin. 2014 Feb;32(1):125–33.
10. Masarone D, Limongelli G, Rubino M, Valente F, Vastarella R, Ammendola E, et al. Management of Arrhythmias in Heart Failure. J Cardiovasc Dev Dis . 2017 2019;4(1):3.
11. Lan SC, Czer L, Wolf PL, Denton TA, Shintaku IP, Chen P-S, et al. Relationship Between Regional Cardiac Hyperinnervation and Ventricular Arrhythmia. Circulation. 2000 ;101:1960–9.
12. Hoss S, Elizur Y, Luria D, Keren A, Lotan C, Gotsman I. Accepted Manuscript Serum Potassium Levels and Outcome in Patients with Chronic Heart Failure. Am J Cardiol. 2016;
13. Heist EK, Ruskin JN. Contemporary Reviews in Cardiovascular Medicine Drug-Induced Arrhythmia. Circulation.2010; 122:1426-1435.
14. Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, et al. 2009 Focused Update Incorporated Into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults. J Am Coll Cardiol . 2009;53(15):e1–90.
15. Crawford MH, Bernstein SJ, Deedwania PC, DiMarco JP, Ferrick KJ, Garson A, et al. ACC/ AHA Guidelines for Ambulatory Electrocardiography: Executive Summary and Recommendations. Circulation. 1999 ;100(8):886–93.
16. 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 Jan;28(1):1-39.e14.
17. Devereux RB. Detection of left ventricular hypertrophy by M-mode echocardiography. Anatomic validation, standardization, and comparison to other methods. Hypertension . 1987 ;9(2_pt_2).
18. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF, Dokainish H, Edvardsen T, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Hear J – Cardiovasc Imaging. 2016;17(12):1321–60.
19. Damasceno A, Mayosi BM, Sani M, Ogah OS, Mondo C, Ojji D, et al. The Causes, Treatment, and Outcome of Acute Heart Failure in 1006 Africans From 9 Countries. Arch Intern Med . 2012:;172(18):1386.
20. Yancy CW, Abraham WT, Albert NM, Clare R, Stough WG, Gheorghiade M, et al. Quality of Care of and Outcomes for African Americans Hospitalized With Heart Failure. J Am Coll Cardiol. 2008 Apr;51(17):1675–84.
21. Nieminen MS, Brutsaert D, Dickstein K, Drexler H, Follath F, Harjola V-P, et al. EuroHeart Failure Survey II (EHFS II): a survey on hospitalized acute heart failure patients: description of population. Eur Heart J . 2006;27(22):2725–36.
22. Abraham WT, Fonarow GC, Albert NM, Stough WG, Gheorghiade M, Greenberg BH, et al. Predictors of In-Hospital Mortality in Patients Hospitalized for Heart Failure. J Am Coll Cardiol [Internet]. 2008;52(5):347–56.
23. Ogah OS, Stewart S, Falase AO, Akinyemi JO, Adegbite GD, Alabi AA, et al. Contemporary Profile of Acute Heart Failure in Southern

Nigeria: Data From the Abeokuta Heart Failure Clinical Registry. ("Contemporary profile of acute heart failure in Southern Nigeria: Data ...") JACC Hear Fail. 2014;2(3):250–9.
24. Lasisi GT, Adebola AP, Ogah OS, Daniel FA. Prevalence of ventricular arrhythmias and heart rate variability pattern in chronic heart failure. Niger Postgrad Med J. 2012 Sep;19(3):157–62.
25. National bureau statistics. South-south leads Nigeria's huge alcohol consumption, NBS data shows. Alcohol Consum Niger. 2016; Available from: https://www.premiumtimesng.com/health/health-features/334887-south-south-leads-nigerias-huge-alcohol-consumption-nbs-data-shows.htm
26. Sarwar M, Muhammad S, Majeed I, Khan MA, Ahmed T, Shabbir F. Effect of Age and Gender on Arrhythmias in Patients With Chronic Heart Failure. Pak J Physiol. 2013;99(22):11–4.
27. Ehrlich JR, Nattel S, Hohnloser SH. Atrial fibrillation and congestive heart failure: Specific considerations at the intersection of two common and important cardiac disease sets. Journal of Cardiovascular Electrophysiology. 2002.
28. Wang TJ, Larson MG, Levy D, Vasan RS, Leip EP, Wolf PA, et al. Temporal Relations of Atrial Fibrillation and Congestive Heart Failure and Their Joint Influence on Mortality. Circulation. 2003;107(23):2920–5.
29. Cygankiewicz I, Zareba W, de Luna AB. Prognostic value of Holter monitoring in congestive heart failure. Cardiology Journal. 2008.
30. Adebayo R, Ikwu A, Balogun M, Akintomide A, Ajayi O, Adeyeye V, et al. Heart rate variability and arrhythmic patterns of 24-hour Holter electrocardiography among Nigerians with cardiovascular diseases. Vasc Health Risk Manag. 2015;11:353–9.
31. Tsang TSM, Barnes ME, Gersh BJ, Bailey KR, Seward JB. Left atrial volume as a morphophysiologic expression of left ventricular diastolic dysfunction and relation to cardiovascular risk burden. Am J Cardiol. 2002;90(12):1284–9.
32. Simpson RJ, Cascio WE, Schreiner PJ, Crow RS, Rautaharju PM, Heiss G. Prevalence of premature ventricular contractions in a population of African American and white men and women: The Atherosclerosis Risk in Communities (ARIC) study. Am Heart J . 2002;143(3):535–40.
33. Akdemir B, Yarmohammadi H, Alraies MC, Adkisson WO. Premature ventricular contractions: Reassure or refer? Cleve Clin J Med. 2016 ;83(7):524–30.

Received = 16/01/2023
Accepted = 06/04/2023