

Hypertensive Heart Failure in Nigerian Africans: Insights from the Abeokuta Heart Failure Registry

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Data from the Abeokuta Heart Failure Registry were used to determine the clinical characteristics, mode of treatment, and short- and medium-term outcomes of patients with hypertensive heart failure. A total of 320 patients were consecutively studied, comprising 184 men (57.5%) and 136 women (42.5%) aged 58.4±12.4 and 60.6±14.5 years, respectively. Most patients (80%) presented with New York Heart Association functional class III or IV and around one third (35%) had preserved systolic function. Median hospital stay was 9 days (interquartile range 5–21) while intra-hospital mortality was 3.4%. The 30-day, 90-day, and 180-day mortality rates were 0.9%

(95% confidence interval, –0.2 to 3.5), 3.5% (95% confidence interval, –1.7 to 7.3), and 11.7% (95% confidence interval, –7.8 to 17.5), respectively. In a multiple logistic regression analysis, only serum creatinine was an independent predictor of mortality at 180 days (adjusted odds ratio, 1.76; 95% confidence interval, –1.17 to 2.64). Hypertension is the most common etiological risk factor for heart failure in Nigeria. Most patients present in the fourth decade of life with severe heart failure and secondary valvular dysfunction and significant in-hospital mortality. *J Clin Hypertens (Greenwich)*. 2015;1–10. © 2015 Wiley Periodicals, Inc.

High blood pressure (BP) is the leading risk factor for cardiovascular (CV) diseases (CVDs) and CV-related morbidity and mortality globally. It is responsible for about 7.5 million deaths every year worldwide.^{1–3} More than 80% of these deaths occur in young and middle-aged men and women in developing/low-income countries.^{2,4} It has been projected that hypertension will increase by 89% in countries in sub-Saharan Africa compared with a rate of 24% in advanced countries.²

In a recent report on the national, regional, and global trends in systolic BP (SBP) since 1980, Danaei and colleagues¹ showed that while SBP fell in many developed countries, it actually rose in many developing countries including countries in East and West Africa.

In Nigeria, for example, the pooled prevalence of high BP in the country increased from 8.6% during the period from 1970–1979 to 22.5% during 2000–2011. Awareness, treatment, and control of hypertension are generally low with an attendant high burden of hypertension-related complications. Hypertension is the most common risk factor for heart and kidney disease in Nigeria.^{5–10}

Recently, hypertension and other noncommunicable diseases have been included as a point of emphasis for

global initiative. It has been added to the agenda of the World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC), especially the new global standardized treatment initiative for hypertension.¹¹

The aim of this study is to describe the sociodemographic characteristics, clinical and echocardiographic characteristics, and clinical outcome of individuals with hypertensive heart failure (HHF) in Abeokuta, Nigeria.

MATERIAL AND METHODS

This was a hospital-based prospective and observational study conducted in the cardiology unit at the Department of Medicine, Federal Medical Centre Abeokuta, Nigeria, as part of the Abeokuta Heart Failure Registry. The baseline characteristics and clinical profile of patients have been published elsewhere.¹⁰ The center is a tertiary institution located in the capital of Ogun State in the southwestern region of Nigeria, which is one of the 36 states that make up the Federal Republic of Nigeria.

The center receives referrals from primary and secondary health facilities within and outside the state. The city has a population of about a million people.¹²

During a 2-year period (January 2009–December 2010), a total of 452 patients who were admitted for heart failure (HF) (constituting 9% of total medical admissions for the period) at the center were recruited into the registry.¹⁰ A total of 355 cases (78.5%) were caused by hypertensive heart disease. A total of 320 (90.1%) of these were included in the current subanalysis. Thirty-five patients were excluded because of insufficient BP data.

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A standardized diagnosis of HF was made using Framingham criteria¹³ as well as according to the guidelines of the European Society of Cardiology on the diagnosis and treatment of acute HF.¹⁴ As such, both de novo presentations of acute HF as well as recurrent presentations of typically decompensated HF (ie, acute-on-chronic HF) were recruited.

All patients gave consent before being enrolled into the study. Ethical clearance was obtained from the institution's ethics review board, and the study conformed to international ethics standards.¹⁵

Clinical Evaluation

A standard case report form was used in data collection. Patients' baseline clinical and demographic variables such as age, sex, contact addresses, telephone number and their relations, marital status, occupation, educational background, history of cardiovascular risk factors such as hypertension, family history, and cigarette smoking were collected.

Signs and symptoms, clinical diagnoses, comorbidities, medications, results of investigation, data on discharge, and short- and medium-term outcomes were also obtained.

BP was recorded according to standard guidelines¹⁶ with the use of a mercury sphygmomanometer. Systolic and diastolic BPs were measured at Korotkoff sounds phases I and V, respectively.

BP was measured in the right arm with the patient in the sitting position and cuff size based on the size of the patient's arm. An average of three readings were taken after 5 minutes of rest.

A diagnosis of hypertensive HF (HHF) was made if the following criteria were met: clinical and echocardiographic-confirmed diagnosis of HF in a known hypertensive or persistent BP $\geq 140/90$ mm Hg in those who were not previously known hypertensive. Patients with de novo HHF were those presenting for the first time with HF while patients with decompensated HHF were those who had a previous history of HF as a result of hypertensive heart disease.^{17,18}

Patients with clinically overt renal failure or on dialysis therapy were excluded from the study. Patients with HF primarily caused by ischemic heart disease and right HF by chronic obstructive pulmonary disease were also excluded. Criteria for the diagnosis or exclusion of other causes of HF in our environment has been previously reported.¹⁰

Body mass index (BMI) was calculated using a standard formula. A BMI of 25 kg/m^2 to 29.9 kg/m^2 and $\geq 30 \text{ kg/m}^2$ defined overweight and obesity, respectively. Anemia was defined as a hematocrit level of $<10 \text{ g/dL}$. Glomerular filtration rate (GFR) was estimated using the four variables of the Modification of Diet in Renal Disease (MDRD) formula.¹⁹ Renal dysfunction was defined as an estimated GFR of $<60 \text{ mL/min/1.73 m}^2$.

A 12-lead electrocardiographic (ECG) tracing was obtained with Schiller electrocardiography (Schiller AG, Switzerland) The ECG paper speed was adjusted to

25 mm/s. All ECGs were recorded in the supine position. For each recording, the styli control was set at 10 mm/mV (except in very high voltages when it was set at 5 mm/mV). ECG reports were analyzed by one of the authors blinded to the clinical history of the patients.

ECG left ventricular hypertrophy by voltage was based on Sokolow-Lyon (SV1+RV5 or V6)²⁰ and/or Cornell (SV3+RaVL with 0.6 mV added in women).²¹

Repolarization abnormalities in the lateral leads (V5–V6, I, and aVL) indicated typical strain when there is a down-sloping convex ST-segment depression ($\geq 0.1 \text{ mV}$) with an inverted asymmetrical T-wave opposite to the QRS axis.^{22,23}

Voltages in individual leads were calculated as the mean of three complexes wherever possible.

M mode, two-dimensional, and Doppler echocardiography were performed using an ALOKA 4000 SSD machine (ALOKA Co. Ltd, Tokyo, Japan). Two dimensionally guided M-mode measurements were made according to standard guidelines. The left atrial dimensions and area were measured according to standard guidelines.^{24,25}

Measurements of left ventricular (LV) dimensions were made from M-mode readings according to the recommendations of the American Society of Echocardiography (ASE).²⁶

Measurements were averaged over three cardiac cycles. Where optimal M-mode imaging could not be obtained, two-dimensional linear measurements were taken according to ASE guidelines. LV fractional shortening fraction was calculated accordingly. Where there was regional wall motion abnormality, the Simpson's method of disc was used for LV function assessment.

LV mass (LVM) was calculated according to the equation²⁷: $\text{LVM} = 0.8[1.05 (\text{IVSTd} + \text{LVIDd} + \text{PWTd})^3 - (\text{LVIDd})^3] + 0.6 \text{ g}$. This formula had good interobserver reproducibility ($\rho=0.93$) in one study.²⁸

LVM index (LVMI) was calculated by dividing the value of LVM by height to its allometric growth rate of 2.7 ($\text{LVMHt}^{2.7}$).^{29,30} The partition value of $51 \text{ g/ht}^{2.7}$ was used since this was the only criterion that demonstrated the optimal threshold value for LV hypertrophy in black patients irrespective of sex in two previous studies.^{31,32}

Relative wall thickness (RWT) was derived from $2 \times$ posterior wall thickness/LV internal diameter. Increased RWT was considered to be present when RWT exceeded 0.43. This represented the 97.5th percentile in normal patients.³³ LV geometric was defined accordingly.³⁴

Doppler flow mapping was used in the assessment of presence of valvular regurgitation. LV diastolic function was evaluated by studying the filling dynamics of the left ventricle. This was quantified by measuring the transmitral E wave velocity (peak early mitral inflow velocity) and the A wave velocity (peak atrial inflow velocity), the E/A ratio, and the deceleration time: time interval of peak E wave velocity to its extrapolation to the baseline. The E/A ratio was not evaluated in individuals with atrial fibrillation or marked tachycardia.

Follow-Up

The cohort was prospectively followed for 180 days. The patients were contacted through clinic visit, telephone calls, or home visits at 30 days, 90 days, and 180 days. Information obtained during follow-up included patient well-being, medication use, history of rehospitalization, and death (from next of kin). In addition to patient or relation telephone interviews, necessary referring physicians were contacted for additional information.

Outcomes Measures

Outcomes measures included: (1) length of hospital stay, (2) intra-hospital outcome, (3) rehospitalization at 180 days, and (4) survival within 180 days.

Quality Control

One of the authors (OSO) performed all echocardiography studies. In our laboratory, the intra-observer concordance correlation coefficient and measurement error have been reported.³⁵

Data Analysis and Statistics

All data were entered into EpiData data management software (EpiData, Odense, Denmark). Analysis was performed with SPSS statistical package (SPSS Inc, IBM, Armonk, NY). Categorical variables are expressed as percentages. Continuous variables are presented as mean and standard deviation or median and interquartile ranges where applicable. Comparison of continuous variables was performed with Student *t* test while categorical variables used chi-square statistics. Non-parametric tests were used where necessary.

Survival function estimates were assessed by the Kaplan-Meier method and the difference was tested using log-rank test. The follow-up was censored at 180 days post-admission. Patients who died or who were readmitted within the period of 180 days constituted the event of interest for purpose of survival analysis while those still alive or lost to follow-up were treated as censored.

Predictors of survival were determined using univariate Cox proportional regression analyses. Thereafter, multiple logistic regression analyses were performed to identify independent predictors of survivals ($P < .1$ used for selection of variables). Results were expressed as odds ratio (OR) and 95% confidence interval (CI).

A two-tailed *P* value of <0.05 was considered statistically significant.

RESULTS

A total of 320 patients were analyzed. There were 184 (57.5%) men and 136 (42.5%) women aged 58.4 ± 12.4 years and 60.6 ± 14.5 years, respectively. The mean age for all patients was 59.3 ± 13.4 years. Figure S1 shows a histogram of the age distribution of all patients and in men and women.

Table I shows the sociodemographic characteristics of the patients. More than 90% were of the Yoruba

tribe (the dominant tribe in the South Western region of Nigeria). The majority of patients were married (72.8%), had at least a primary education (63.5%), were employed (95%), and resided in the urban area (73.4%).

Men significantly attained higher levels of education than their female counterparts and were more often in the skilled and professional occupation category. In terms of CVD risk factors, women had higher body mass indices than men. On the other hand, cigarette smoking and alcohol consumption were significantly higher in men. Only about 15% had acute or chronic HHF. A total of 290 patients (90.6%) were known hypertensives 70 (92.4%) men and 120 (88.2%) women. There was a history of diabetes mellitus (DM) in 12.2% of the patients.

The most common comorbidity was osteoarthritis (67 [20.9%]), which was significantly more common in women than in men (29 [15.8%] vs 38 [27.9%]; $P = .006$). Chronic obstructive pulmonary disease (COPD), asthma, and stroke were present in 8 (2.5%), 7 (2.2%), and 1 (0.3%) patient, respectively. One patient each had stroke, migraine, inguino-scrotal hernia, and psychosis. Dyspnea on exertion was the most common symptom and was present in 94.4% of patients, while basal crepitation/crackles were seen in 84.4% of patients. More than 80% of patients were in New York Heart Association (NYHA) functional class III or IV.

Figure 1 shows the identified precipitating factors for decompensation.

The mean heart rate on admission was 96.2 beats per minute while mean systolic and diastolic BPs were 143.5 mm Hg and 90.5 mm Hg, respectively. The mean packed cell volume, total white cell count, serum sodium, serum potassium, and blood glucose levels were similar between men and women. Total cholesterol was higher in women but not statistically significant. Blood urea and creatinine levels were higher in men (49.9 mg/dL vs 38.2 mg/dL and 1.8 vs 1.3 mg/dL, respectively). In addition, 2.7% of those who had HIV screening tested positive (Table I).

QRS duration and QT intervals were higher in men, but this was not statistically significant, and 12.8% of the cohort had atrial fibrillation.

Almost all echocardiographic variables were statistically higher in men compared with women (Table II).

In terms of drug administration, 278 (86.9%) patients were taking loop diuretics, 25 (7.8) were taking thiazide diuretics, 319 (99.1%) were taking angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and 98 (30.6%) were placed on long-acting calcium channel blockers (CCBs). A total of 81.3%, 73.1%, 15.3%, and 2.7% patients were taking spironolactone, digoxin, hydralazine/isosorbide, and β -blockers, respectively (Figure 2).

Outcome

The median length of hospital stay was 9 days, and this was the same in both men and women. There were 11

TABLE I. Baseline Sociodemographic and Clinical Profile of 320 Hypertensive Patients

Variable	All (N=320)	Men (n=184)	Women (n=136)
Demographic characteristics			
Age, y	59.3±13.4	58.4±12.4	60.6±14.5
Married, No. (%) ^a	233 (72.8)	149 (81.0)	84 (61.8)
No education, No. (%) ^b	85 (37.3)	36 (27.1)	49 (51.6)
Unemployed, No. (%)	16 (5.0)	6 (3.3)	10 (7.4)
Rural dweller, No. (%)	85 (26.6)	48 (26.1)	37 (27.2)
CV risk factors and comorbidities			
Never smoked, No. (%) ^a	260 (81.3)	131 (71.2)	129 (94.9)
Ever consumed alcohol ^a	156 (48.7)	118 (64.2)	38 (28.0)
Current alcohol consumption, No. (%)	42 (13.1)	34 (18.5)	8 (5.9)
Diabetes mellitus, No. (%)	39 (12.2)	22 (12.0)	17 (12.5)
Bronchial asthma, No. (%)	7 (2.2)	3 (1.6)	4 (2.9)
COPD, No. (%)	8 (2.5)	6 (3.3)	2 (1.5)
Arthritis, No. (%) ^b	67 (20.9)	29 (15.8)	38 (27.9)
Family history of heart disease, No. (%)	20 (6.3)	13 (7.1)	7 (5.1)
Clinical and laboratory features			
NYHA class, No. (%)			
II	57 (17.8)	36 (19.6)	21 (15.4)
III	201 (62.8)	109 (59.2)	92 (67.6)
IV	62 (19.4)	39 (21.2)	23 (16.9)
Symptoms and signs			
Nocturnal cough, No. (%)	292 (91.3)	166 (90.2)	126 (92.6)
Dyspnea on exertion, No. (%)	302 (94.4)	172 (93.5)	130 (95.6)
Leg edema, No. (%)	242 (75.6)	140 (76.1)	102 (75.0)
Paroxysmal nocturnal dyspnea, No. (%)	262 (81.9)	157 (82.1)	111 (81.6)
Raised JVP, No. (%)	210 (65.6)	128 (69.6)	82 (60.3)
Basal crepitation, No. (%)	270 (84.4)	160 (87.0)	110 (80.9)
Hepatomegaly, No. (%)	210 (62.8)	116 (63.0)	85 (62.5)
Third heart sound, No. (%)	207 (64.7)	115 (62.5)	92 (67.6)
Temperature, °C	36.4±0.82	36.4±0.77	36.3±0.88
Respiratory rate, breaths per min	28.7±8.9	29.2±10.3	28.0±6.6
Pulse, beats per min	96.2±18.9	97.4±19.1	94.7±18.7
Systolic BP, mm Hg	143.5±32.1	143.8±32.9	143.0±19.4
Diastolic BP, mm Hg	90.5±20.6	91.1±21.5	89.6±19.4
Body mass index, kg/m ²	24.2±5.3	24.0±5.0	24.5±5.7
Overweight, No. (%)	43 (23.3)	41 (30.3)	84 (26.1)
Obese, No. (%)	19 (10.1)	19 (13.8)	38 (11.6)
De novo HF, No. (%)	274 (85.6)	115 (84.2)	119 (87.5)
Length of admission, d	10.4±5.2	10.1±4.8	10.8±5.5
Laboratory findings			
Packed cell volume, %	37.4±6.9	37.3±6.7	37.5±3.3
White cell count	6.9±3.6	7.1±3.8	6.9±3.3
Serum sodium, mmol	135.8±6.9	135.3±7.1	136.4±6.7
Serum potassium, mmol/L	3.66±0.79	3.71±0.81	3.60±0.78
Total cholesterol, mg/dL	166.2±74.1	162.9±76.7	174.7±68.6
Blood glucose, mg/dL	113.3±51.2	113.4±47.6	113.3±56.2
Urine protein, No. (%) (164 tested)	58 (35.4)	26 (25)	32 (42.1)
Blood urea, mg/dL ^c	44.5± 42.8 ^c	49.9±49.7	38.2±32.0
Blood creatinine, mg/dL ^c	32.8 (27.5) ^d	37.0 (29.7)	28.0 (22.0)
Blood creatinine, mg/dL ^c	1.5±2.0 ^c	1.8±2.4	1.3±1.5
Blood creatinine, mg/dL ^c	0.9 (0.8) ^d	1.0 (0.8)	0.8 (0.5)
Estimated glomerular filtration rate ^c	94.6±51.7 ^c	95.2±52.6	93.8±50.7
Estimated glomerular filtration rate ^c	(90.2) ^e	(89.1)	(91.2)
HIV positive, No. (%) (150 tested)	4 (2.7)	3 (3.4)	1 (1.5)

Abbreviations: BP, blood pressure; COPD, chronic obstructive pulmonary disease; CV, cardiovascular; HF, heart failure + data are skewed; JVP, jugular venous pressure; NYHA, New York Heart Association. ^aP<.001. ^bP<.05. ^cMean (standard deviation). ^dMedian (interquartile range). ^eMedian value.

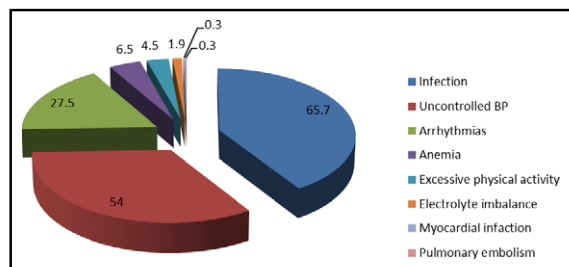


FIGURE 1. Pie chart showing the precipitating factors identified in the 320 hypertensive patients. BP indicates blood pressure.

intra-hospital deaths (5 men and 6 women), with an in-hospital mortality rate of 3.6% (3.4% and 3.8% for men and women, respectively).

The rehospitalization rates at 30 days, 90 days, and 180 days were 4.2% (95% CI, -2.3 to 7.6), 5.6% (95% CI, -3.3 to 9.5), and 7.3% (95% CI, -4.5 to 11.7), respectively (Figure 3a).

Similarly, the 30-, 90-, and 180-day mortality rates were 0.9% (95% CI, -0.2 to 3.5), 3.5% (95% CI, -1.7 to 7.3), and 11.7% (95% CI, -7.8 to 17.5), respectively (Figure 3b).

Compared with those who survived at 180 days, patients who died were more in NYHA class III or IV (86.7% vs 38.7%, $P < .001$). They also had significantly lower SBP (130.7 ± 20.9 mm Hg vs 144.1 ± 32.4 mm Hg, $P = .030$), lower diastolic BP (80.0 ± 13.7 mm Hg vs 89.3 ± 18.9 mm Hg, $P = .044$), pulse pressure (42.7 ± 13.3 mm Hg vs 53.5 ± 12.1 mm Hg, $P = .009$), lower packed cell volume (31.7 ± 8.8 vs 37.7 ± 6.6 , $P = .011$), higher serum creatinine (2.2 ± 2.6 vs 1.2 ± 1.0 , $P = .005$), and higher total white cell count (9.4 ± 5.2 vs 7.0 ± 3.5 , $P = .019$).

Patients who died also had significantly larger left atrial size, lower mitral A wave, and higher E/A ratio.

After multiple logistic regression analysis, only serum creatinine was an independent predictor of mortality at 180 days (adjusted OR, 1.76; 95% CI -1.17 to 2.64).

The only significant difference in rehospitalization status of the cohort was serum potassium level. This was significantly higher in the rehospitalized group (4.3 ± 0.9 vs 3.6 ± 0.7 , $P = .011$).

DISCUSSION

There have been limited investigations on the characteristics and outcomes of HHF in Nigeria and this is the first comprehensive study of this disorder in the country.

Our findings indicate that HHF is a problem in middle-aged Nigerians. Only 46% were older than 60 years, with the majority of patients presenting with severe symptoms and worse NYHA functional class (82% with NYHA III or IV functional class). We also noted that severe LV remodeling patterns and frequent functional valvular dysfunction were common in predominantly de novo HF (moderate/severe aortic

regurgitation, mitral regurgitation, and tricuspid regurgitation were present in 3.6%, 31.6%, and 21.1% of patients, respectively). Comorbid conditions were common. While the use of disease-modifying drugs such as angiotensin-converting enzyme inhibitors/angiotensin receptor blockers and spironolactone was high, the rate of β -blocker and hydralazine/isosorbide use was very low. Finally, an intra-hospital mortality rate of 3.4% (95% CI, 1.5%–6.6%) was recorded.

Table III depicts the comparison of our data with previous studies in other parts of Africa and high-income countries. While our patients were slightly older than those in a Cameroonian study (59.3 vs 54.9 years),³⁶ they were younger than hypertensive patients in studies from South Africa (61 years),³⁷ Europe (69.8 years),¹⁸ or North America (74.8 years).³⁸

The male preponderance in our cohort is similar to the report from Europe.^{18,39} Higher frequency in women has been reported elsewhere.^{37,38} The rate of comorbidities (diabetes mellitus, CAD, COPD, stroke, atrial fibrillation) was lower in our cohort compared with similar data in Europe and America (Table III). This was associated with higher rates of CVD risk factors in Europe and America such as cigarette smoking, obesity, and dyslipidemia. The frequency of anemia was similar. The mean BP was similar with the report from South Africa³⁷ but lower than the mean BP in the Acute Decompensated Heart Failure Syndromes (ATTEND) registry.³⁸ Our patients had more severe LV systolic dysfunction and larger LA size (surrogate of LV diastolic dysfunction) compared with previous reports.^{18,37,38}

The use of disease-modifying drugs such as ACE inhibitors in our study was comparable to that in previous reports; however, β -blocker utilization was low in our report.

The intra-hospital outcome was better in our study than in the report from a Polish investigation but worse than that of the EuroHF survey and the ATTEND registry.

Several factors could explain the severity of the disease in our cohort. Late presentation is one reason. Most often patients are unaware that they have high BP. In a recent review, it was reported that only 14.2% to 30% of Nigerians are aware that they are hypertensive, 18% to 21% are taking treatment for hypertension, and only 9% are controlled.⁵ Other factors may include ignorance, lack of health education, weak health-care system, and poor social support.

Several reasons may account for the lower rates of stroke in our study compared with data from high-income countries. It is possible that most of our stroke patients die and therefore do not live long enough to develop HF. Better facilities available for care of stroke patients may have accounted for the higher number of patients with history of stroke in advanced countries. In a 10-year review of stroke, Ogun and colleagues⁴⁰ documented that the case fatality at 24 hours, 7 days, 30

TABLE II. 12-Lead ECG and Echocardiographic Abnormalities in 320 Hypertensive Patients

Variable	All (N=320)	Men (n=184)	Women (n=136)
Ventricular rate, beats per min	96.8±20.4	95.8±17.1	98.4±25.2
QRS duration, ms	109.8±24.7	113.1±23.9	104.4±25.5
QT interval, ms	364.8±44.3	368.5±36.6	358.8±54.6
Corrected QT, ms	454.8±35.4	457.5±35.5	450.3±35.5
Atrial fibrillation/flutter	41 (12.8)	23 (12.5)	18 (13.2)
ECG LVH ^a	309 (96.5)	173 (94.3)	136 (100)
ECG LVH with strain	138 (43.0)	87 (47.2)	49 (36.4)
Aortic root diameter, cm	3.10±0.50	3.24±0.50	2.90±0.39
Left atrial diameter, cm	4.68±0.85	4.77±0.86	4.55±0.81
Left atrial area ^b	26.6±8.1	27.7±8.3	24.8±7.36
IVSTd, cm ^b	1.35±0.37	1.40±0.41	1.28±0.29
IVSTs, cm ^b	1.60±0.85	1.67±0.55	1.48±0.34
LVIDd, cm ^b	5.52±1.49	5.84±1.47	5.08±1.40
LVIDs, cm ^b	4.57±1.49	4.86±1.43	4.15±1.31
PWTd, cm ^b	1.19±0.36	1.22±0.30	1.15±0.35
PWTs, cm ^b	1.65±0.37	1.66±0.38	1.65±0.37
Fractional shortening, %	17.9±8.7	17.4±8.7	18.7±8.6
Ejection fraction, %	42.9±16.5	41.8±16.6	44.5±16.3
LV mass, g ^b	338.4±133.6	376.4±140.8	283.9±100.4
Indexed LV mass, g/ht ^{2.7b}	90.4±37.7	96.7±41.8	81.1±28.0
Relative wall thickness	0.45±0.15	0.44±0.15	0.47±0.15
LV geometry (n=263)			
Normal geometry, %	4.2	4.5	3.7
Concentric remodeling, %	7.2	3.9	12.0
Concentric hypertrophy, %	42.6	43.2	41.7
Eccentric hypertrophy, %	46.0	48.4	42.6
Mitral E wave	0.82±0.29	0.80±0.28	0.85±0.32
Mitral A wave	0.53±0.25	0.50±0.23	0.57±0.28
E/A ratio	2.05±1.45	2.11±1.52	1.95±1.35
Deceleration time, ms	144.3±57.6	141.5±55.1	148.6±61.4
IVRT, ms	116.4±34.8	118.5±36.5	113.0±31.9
LV filling pattern (n=262)			
Impaired relaxation, %	26.7	26.4	27.2
Pseudonormalized filling, %	45.5	45.3	45.6
Restrictive filling, %	27.9	28.3	27.2
Mitral regurgitation, % ^c	79.1	78	80.3
Tricuspid regurgitation, % ^c	60.8	58.2	63.9
Aortic regurgitation, % ^c	47.7	43	46.7
Systolic HF, No. (%)	182 (65.5)	114 (69.9)	68 (59.1)
Diastolic HF, No. (%)	96 (34.5)	49 (30.1)	47 (40.9)

Abbreviations: A, left ventricular late filling velocity; E, left ventricular early filling velocity; ECG, electrocardiographic; EF, ejection fraction; FS, fractional shortening; HF, heart failure; IVRT, isovolumic relaxation time; IVSTd, interventricular septal wall thickness in diastole; LV, left ventricular; LVIDd, left ventricular internal diameter in diastole; LVIDs, left ventricular internal diameter in systole; PWTd, left ventricular posterior wall thickness in diastole; PWTs, left ventricular posterior wall thickness in systole. ^aLV hypertrophy (LVH) diagnosed by Sokolow-Lyon and/or Cornell Criteria. ^bP<.05. ^cModerate/severe atrial regurgitation, mitral regurgitation, and tricuspid regurgitation were present in 3.6%, 31.6%, and 21.1% of patients, respectively.

days, and 6 months were 9%, 28%, 40%, and 46%, respectively [154]. In 2007, mortality from stroke in the country was 126/100,000 population.⁴¹

An important feature of our study is the assessment of LV geometry and filling pattern in HHF patients, which has been scarcely studied in Africa. Severe forms of LV geometry (concentric or eccentric hypertrophy) were frequent, which is indicative of severe myocardial remodeling in our HF patients.

Presence of high frequency of pseudo-normalized and restrictive LV filling pattern indicates severe degrees of

LV diastolic dysfunction. This may also explain the high frequency of valvular abnormalities in our cohort.

Some of the potential reasons for the high prevalence of severe diastolic dysfunction in our study population may include severity of hypertension and left ventricular hypertrophy. Race as well as salt sensitivity may also be responsible.

The most common reasons for the low utilization of β -blockers in our cohort could include severity of disease and edema at presentation, low BP on admission, poverty (since patients pay out of pocket), and

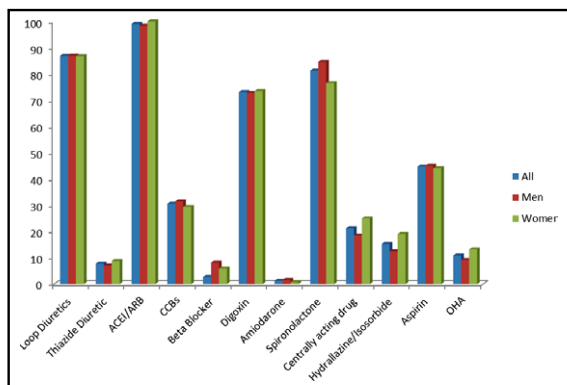


FIGURE 2. Bar chart showing the pattern of drug prescription in the 320 hypertensive patients. ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCBs, calcium channel blockers; DHA, docosahexaenoic acid.

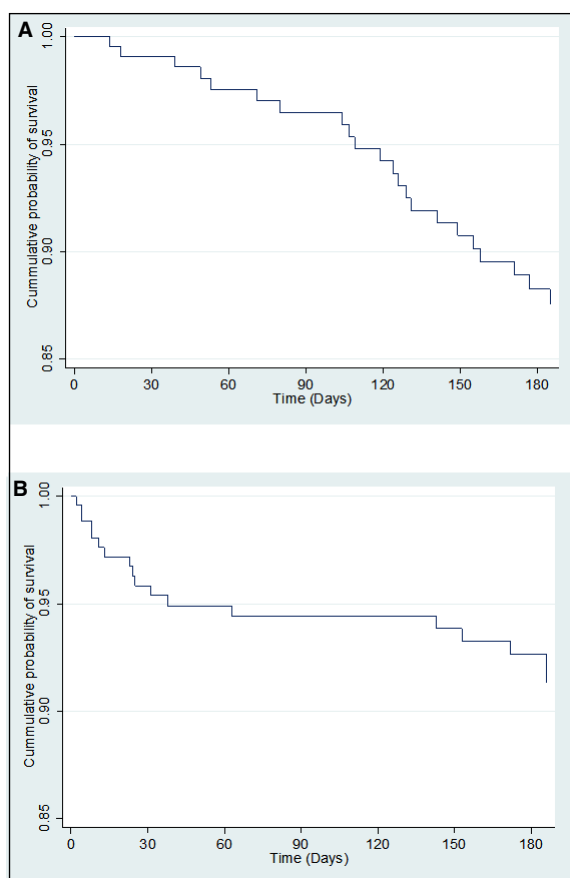


FIGURE 3. (a) Kaplan-Meier curve showing the rate of rehospitalization of the cohort after 180 days. (b) Kaplan-Meier curve showing the rate of mortality of the cohort after 180 days.

even ignorance on the side of care givers. The high rate of digitalis use could be related to the high burden of systolic HF as well as ignorance on the part of health professionals.

These findings provide an opportunity for intervention (education and training of health-care providers).

The finding that presence of renal impairment is an independent predictor of outcome in our cohort supports the known outcome of patients with chronic kidney disease (CKD) in Nigeria and in most African countries.^{42,43} In most cases, renal replacement is not an option for patients because of high cost or nonavailability.

Perspectives

This study has some important clinical and public health implications. In view of the rising burden of CVD risk factors, especially hypertension in Nigeria, our findings may indicate a possible increase in the burden of HF and other hypertensive target organ damage. This is against the background of the high burden of HIV/AIDS and other communicable diseases that place a lot of strain on the already weak health system of the country.

Therefore, there is a need for regular and systematic national and regional data necessary for policy. It is important to set up chronic disease clinics at the community level to cater for patients with diseases such as hypertension and diabetes mellitus. This will reduce the burden on general physicians as well as decrease the waiting time encountered by hypertensive patients in most general clinics. The government can also subsidize the cost of drugs as the burden of out-of-pocket payment of medications often leads to noncompliance.

Basic equipment for measuring BP should be made available at all levels of care in the country, especially at the primary health-care level. Capacity building in the form of training and retraining of health-care workers as well as education of the general population on the significance of the causes and control of hypertension is important.

At the individual level, the use of nurse-led primary health care for the diagnosis and follow-up of patients has been shown to be effective.⁴⁴ Integration of BP control into other control programs such as HIV/AIDS programs have been demonstrated to be useful and effective in countries such as Cambodia and South Africa.⁴⁵ The national policy on noncommunicable disease must be put into use especially at the primary health-care level. A national guideline on the management of hypertension has to be tailored to the needs and peculiarities of the Nigerian environment. The guideline should also be available and accessible to health-care providers at all levels of care. The process of procurement and distribution of antihypertensive medications has to be standardized in order to prevent “out of stock” syndrome. There must also be quality control in order to stop the flow of counterfeit medications in the country.

Reduction of salt intake is one of the most cost-effective approaches to BP control. In this regard,

TABLE III. Comparison of Our Findings With Similar Studies in Other Parts of the World

Characteristics	Present Study (N=320)	HOS ³³ (n=281)	EHFS II ¹¹ (n=200)	AHEAD ³¹ (n=179)	Venskutonyte et al ³⁴ (n=65)
Women, %	42.5	61	39.6	65.4	33.3
Mean age, y	59.3	61	69.8	74.8	65.5
De novo HF, %	85.6	NA	37.3	74.3	66.7
NYHA III and IV, %	82.2	29	NA	34.0	NA
History of hypertension, %	90.6	100	94.6	94.3	100
Diabetes mellitus, %	12.2	14	34.5	43.1	33.3
Previous MI or CAD, %	0.3	1.0	53.8	26.4	46.7
COPD, %	2.5	NA	18.0	17.8	26.7
History of stroke or TIA, %	0.3	12	16.0	26.4	20
Atrial fibrillation, %	12.8	9	37.7	19.0	46.7
Mean systolic BP, mm Hg	144	140	NA	198	NA
Mean diastolic BP, mm Hg	91	80	NA	100	NA
Heart rate, beats per min	96	NA	NA	93	NA
Body mass index, kg/m ²	24.2	NA	NA	28.0	33.9
Hospitalization for HF within past 12 months, %	82.2	NA	NA	45.1	46.6
Renal failure, %	14.4	27	18.7	NA	NA
Anemia, %	11.5	10	11.3	NA	NA
Infection, %	63.4	NA	15.6	NA	13.3
Noncompliance with therapy, %	74.1	NA	21.9	NA	66.7
ACE inhibitors, %	99.1	NA	NA	71.3	NA
β-Blockers, %	2.7	NA	NA	77.0	NA
Calcium antagonists, %	30.6	NA	NA	51.1	NA
Diuretics, %	86.9	NA	NA	88.5	NA
Spirolactone, %	81.3	NA	NA	36.2	NA
Digoxin, %	73.1	NA	NA	13.8	NA
LVEDD, mm	55	46	56	NA	50
Mean ejection fraction	42.7	53	44	55	50.5
LA, mm	47	NA	45	NA	42
Mitral regurgitation, %	79.1	7	77.6	NA	100
Tricuspid regurgitation, %	60.8	6	53.7	NA	93.3
LOS, median, d	9	NA	8	NA	13
Intra-hospital mortality, %	3.4	NA	1.5	2.2	6.6

Abbreviations: ACE, angiotensin-converting enzyme; AHEAD, Acute Heart Failure Database; BP, blood pressure; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; EHFS II, European Heart Failure Survey II; HF, heart failure; HOS, Heart of Soweto study; LA, left atrium; LOS, length of hospital stay; LVEDD, left ventricular end-diastolic diameter; MI, myocardial infarction; NA, not available; NYHA, New York Heart Association; TIA, transient ischemic attack.

Nigeria can learn from South Africa regarding the recently passed law that will regulate the amount of salt in processed foods in the country.⁴⁶

The global risk management approach must remain the key message in Nigeria. This has been shown to be economically beneficial in many third-world countries.⁴⁷

Finally, community participation is an important approach in public health control programs in sub-Saharan Africa. The use of traditional and religious rulers has produced good results in many primary health-care programs and this can be applied to BP control.

Limitations

This study has some limitations. First, because the study was hospital-based, it may not have captured all cases of HF in the community, especially those with mild

disease. However, effort was made by the group to inform health-care providers (both in public and private health institutions) in the community about the registry. Second, our institution was the only one that had the services of a cardiologist and cardiac evaluation tools in the city. Third, we may not have captured the HF cases that presented to the outpatient department. Community-based data are superior in estimating the true burden of disease in the state.

Fourth, we were unable to assess biomarkers in this study because of cost constraints.

Fifth, LV systolic function evaluation did not include mid-wall function assessment, which has been repeatedly shown to be prognostically valuable and to respond to treatment in hypertensive heart disease.^{48,49} Finally, some of the newer parameters for assessing diastolic function were not used in this study.

CONCLUSIONS

HHF affects Nigerians in their productive age group with attendant high economic loss and disability-adjusted life-years. The disease is often severe as a result of late presentation. Efforts should be made at the community level to ensure primordial prevention, early detection, treatment, and control of hypertension in the country.

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References

- Danaei G, Finucane MM, Lin JK, et al. National, regional, and global trends in systolic blood pressure since 1980: systematic analysis of health examination surveys and epidemiological studies with 786 country-years and 5.4 million participants. *Lancet*. 2011;377:568–577.
- Kearney PM, Whelton M, Reynolds K, et al. Global burden of hypertension: analysis of worldwide data. *Lancet*. 2005;365:217–223.
- Mancia G, Fagard R, Narkiewicz K, et al. 2013 ESH/ESC Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *J Hypertens*. 2013;31:1281–1357.
- Modesti PA, Agostoni P, Agyemang C, et al. Cardiovascular risk assessment in low-resource settings: a consensus document of the European Society of Hypertension Working Group on Hypertension and Cardiovascular Risk in Low Resource Settings. *J Hypertens*. 2014;32:951–960.
- Ogah OS, Okpechi I, Chukwuonye II, et al. Blood pressure, prevalence of hypertension and hypertension related complications in Nigerian Africans: a review. *World J Cardiol*. 2012;4:327–340.
- Ogah OS. Hypertension in sub-Saharan African populations: the burden of hypertension in Nigeria. *Ethn Dis*. 2006;16:765.
- Ojji DB, Alfa J, Ajayi SO, et al. Pattern of heart failure in Abuja, Nigeria: an echocardiographic study. *Cardiovasc J Afr*. 2009;20:349–352.
- Ojji D, Stewart S, Ajayi S, et al. A predominance of hypertensive heart failure in the Abuja Heart Study cohort of urban Nigerians: a prospective clinical registry of 1515 de novo cases. *Eur J Heart Fail*. 2013;15:835–842.
- Damasceno A, Mayosi BM, Sani M, et al. The causes, treatment, and outcome of acute heart failure in 1006 Africans from 9 countries. *Arch Intern Med*. 2012;172:1386–1394.
- Ogah OS, Stewart S, Falase AO, et al. Contemporary profile of acute heart failure in Southern Nigeria: data from the Abeokuta heart failure clinical registry. *JACC Heart Fail*. 2014;2:250–259.
- The Global Standardized Hypertension Treatment Project. Centers for Diseases Control and Prevention 2014. <http://www.cdc.gov/global-health/ncd/hypertension-treatment.htm>. Accessed November 3, 2014.
- National Population Census. Abuja: National Population Commission; 2006.
- McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham study. *N Engl J Med*. 1971;285:1441–1446.
- Dickstein K, Cohen-Solal A, Filippatos G, et al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the diagnosis and treatment of acute and chronic heart failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). *Eur J Heart Fail*. 2008;10:933–989.
- Rickham PP. Human experimentation. Code of ethics of the world medical association. Declaration of Helsinki. *Br Med J*. 1964;2:177.
- Recommendations for routine blood pressure measurement by indirect cuff sphygmomanometry. American Society of Hypertension. *Am J Hypertens*. 1992;5:207–209.
- Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure (The JNC 7 Report). *JAMA*. 2003;289:2560–2572.
- Nieminen MS, Brutsaert D, Dickstein K, et al. EuroHeart Failure Survey II (EHFS II): a survey on hospitalized acute heart failure patients: description of population. *Eur Heart J*. 2006;27:2725–2736.
- Levey AS, Bosch JP, Lewis JB, et al. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med*. 1999;130:461–470.
- Sokolow M, Lyon TP. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am Heart J*. 1949;37:161–186.
- Casale PN, Devereux RB, Alonso DR, et al. Improved sex-specific criteria of left ventricular hypertrophy for clinical and computer interpretation of electrocardiograms: validation with autopsy findings. *Circulation*. 1987;75:565–572.
- Okin PM, Devereux RB, Nieminen MS, et al. Relationship of the electrocardiographic strain pattern to left ventricular structure and function in hypertensive patients: the LIFE study. Losartan Intervention For End point. *J Am Coll Cardiol*. 2001;38:514–520.
- Ogah OS, Adebisi AA, Oladapo OO, et al. Association between electrocardiographic left ventricular hypertrophy with strain pattern and left ventricular structure and function. *Cardiology*. 2006;106:14–21.
- Lester SJ, Ryan EW, Schiller NB, Foster E. Best method in clinical practice and in research studies to determine left atrial size. *Am J Cardiol*. 1999;84:829–832.
- Thomas L, Levett K, Boyd A, et al. Compensatory changes in atrial volumes with normal aging: is atrial enlargement inevitable? *J Am Coll Cardiol*. 2002;40:1630–1635.
- Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. *Circulation*. 1978;58:1072–1083.
- Reichek N. Two-dimensional echocardiography for determination of left ventricular mass. *Am J Card Imaging*. 1994;8:305–309.
- Palmieri V, Dahlof B, De Zeeuw D, et al. Reliability of echocardiographic assessment of left ventricular structure and function. The PRESERVE study. *J Am Coll Cardiol*. 1999;34:1625–1632.
- De Simone G, Daniels SR, Devereux RB, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. *J Am Coll Cardiol*. 1992;20:1251–1260.
- De Simone G, Devereux RB, Daniels SR, et al. Effect of growth on variability of left ventricular mass: assessment of allometric signals in adults and children and their capacity to predict cardiovascular risk. *J Am Coll Cardiol*. 1995;25:1056–1062.
- Nunez E, Arnett DK, Benjamin EJ, et al. Optimal threshold value for left ventricular hypertrophy in blacks: the Atherosclerosis Risk in Communities study. *Hypertension*. 2005;45:58–63.
- Adebisi AA, Ogah OS, Aje A, et al. Echocardiographic partition values and prevalence of left ventricular hypertrophy in hypertensive Nigerians. *BMC Med Imaging*. 2006;6:10.
- Roman MJ, Pickering TG, Schwartz JE, et al. Association of carotid atherosclerosis and left ventricular hypertrophy. *J Am Coll Cardiol*. 1995;25:83–90.
- Ganau A, Devereux RB, Roman MJ, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. *J Am Coll Cardiol*. 1992;19:1550–1558.
- Ogah OS, Adebajo AT, Otukoya AS, Jagusa TJ. Echocardiography in Nigeria: use, problems, reproducibility and potentials. *Cardiovasc Ultrasound*. 2006;4:13.
- Dzudie A, Kengne AP, Mbahe S, et al. Chronic heart failure, selected risk factors and co-morbidities among adults treated for hypertension in a cardiac referral hospital in Cameroon. *Eur J Heart Fail*. 2008;10:367–372.
- Stewart S, Wilkinson D, Hansen C, et al. Predominance of heart failure in the Heart of Soweto Study cohort: emerging challenges for urban African communities. *Circulation*. 2008;118:2360–2367.
- Sato N, Kajimoto K, Asai K, et al. Acute Decompensated Heart Failure Syndromes (ATTEND) registry. A prospective observational multicenter cohort study: rationale, design, and preliminary data. *Am Heart J*. 2010;159:949–955, e1.

39. Nieminen MS, Harjola VP, Hochadel M, et al. Gender related differences in patients presenting with acute heart failure. Results from EuroHeart Failure Survey II. *Eur J Heart Fail.* 2008; 10:140–148.
40. Ogun SA, Ojini FI, Ogungbo B, et al. Stroke in south west Nigeria: a 10-year review. *Stroke.* 2005;36:1120–1122.
41. Strong K, Mathers C, Bonita R. Preventing stroke: saving lives around the world. *Lancet Neurology.* 2007;6:182–187.
42. Seck SM, Diallo IM, Diagne SI. Epidemiological patterns of chronic kidney disease in black African elders: a retrospective study in West Africa. *Saudi J Kidney Dis Transpl.* 2013;24:1068–1072.
43. Ulasi II, Ijoma CK. The enormity of chronic kidney disease in Nigeria: the situation in a teaching hospital in South-East Nigeria. *J Trop Med.* 2010;2010:501957.
44. Sindhu S, Pholpet C, Puttakitpol S. Meeting the challenges of chronic illness: a nurse-led collaborative community care program in Thailand. *Collegian.* 2010;17:93–99.
45. Levitt NS, Steyn K, Dave J, Bradshaw D. Chronic noncommunicable diseases and HIV-AIDS on a collision course: relevance for health care delivery, particularly in low-resource settings—insights from South Africa. *Am J Clin Nutr.* 2011;94:1690S–1696S.
46. Foodstuffs, cosmetics and disinfectants Act, 1972 (Act 54 of 1972) Department of Health, Republic of South Africa, 2012. <http://www.doh.gov.za/docs/foodcontrol/comments/2012/fcr533.pdf>. Accessed April 6, 2013.
47. Gaziano TA, Young CR, Fitzmaurice G, et al. Laboratory-based versus non-laboratory-based method for assessment of cardiovascular disease risk: the NHANES I Follow-up Study cohort. *Lancet.* 2008;371:923–931.
48. Verdecchia P, Schillaci G, Reboldi G, et al. Prognostic value of midwall shortening fraction and its relation with left ventricular mass in systemic hypertension. *Am J Cardiol.* 2001;87:479–482, A7.
49. Wachtell K, Gerds E, Palmieri V, et al. In-treatment midwall and endocardial fractional shortening predict cardiovascular outcome in hypertensive patients with preserved baseline systolic ventricular function: the Losartan Intervention for Endpoint reduction study. *J Hypertens.* 2010;28:1541–1546.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Figure S1. Histogram showing the age distribution of the 320 hypertensive patients according to sex.