Left Ventricular Hypertrophy in African Black Patients with Chronic Renal Failure at First Evaluation

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Objective: Chronic kidney disease (CKD) is a major cause of cardiovascular morbidity and mortality all over the world. The combined effect of volume and pressure overload seen in patients with CKD is the primary cause of left ventricular hypertrophy (LVH). Though it accounts for a significant proportion of patients dying in hospitals in Nigeria, information on CKD in African Blacks is lacking. This study evaluates the prevalence of LVH and factors affecting it in pre-dialysis patients by using echocardiography.

Design, Setting and Patients: One hundred consecutive patients with CKD who were attending the medical outpatient and renal clinics of University of Nigeria Teaching Hospital, Enugu, who satisfied the inclusion criteria were screened for the study. Eighty-eight patients completed the study. Forty-five age- and sex-matched subjects were selected as controls. Clinical and laboratory parameters and echocardiographic indices were measured.

Results: Left ventricular hypertrophy (LVH), defined in absolute terms as left ventricular mass index >134 g/m^2 in men and >110 g/m^2 in women was present in 95.5% of patients and 6.7% of controls. The most prevalent type of LVH was eccentric hypertrophy, which was found in 54.6%, while concentric was seen in 40.9%. Hypertension was present in 85.2% of the patients. The predominant causes of CKD were chronic glomerulonephritis (43.2%), hypertension (25%), and diabetes mellitus (14.8%). All the patients studied had advanced CKD, either stage 4 or 5 of the Kidney Disease Outcome Quality Initiative classification of CKD. Stepwise method of multiple linear regressions identified mean arterial pressure (32%), hemoglobin concentration (22%), male sex (17%), and creatinine clearance (24%) as predictors of LVH in CKD.

Conclusion: This study showed a strong association between CKD and LVH in patients in developing countries at the time of first evaluation by a nephrologist. It demonstrated a high prevalence of LVH in patients at first evaluation. The patients were often anemic and had severe hypertension even at first presentation. Early detection and treatment of causes of CKD should be pursued aggressively at the primary prevention level, as has been advocated by the International Society of Nephrology to reduce the effects of CKD and its attendant complications in the society. (*Ethn Dis*. 2006;16:859–864)

Key Words: Anemia, Cardiovascular Risk, CKD, Hypertension, Left Ventricular Hypertrophy, Mean Arterial Pressure

Introduction

Chronic kidney disease (CKD) is a major cause of cardiovascular morbidity and mortality all over the world.[1] In Nigeria and most parts of Africa, chronic renal failure accounts for a significant proportion of deaths in hospitals.[2] Even in advanced countries, CKD remains a major health problem,[3,4] causing cardiovascular diseases such as congestive cardiac failure, myocardial infarction, hypertension, stroke, and sudden cardiac death in patients with chronic renal failure (CRF). The hemodynamic and metabolic changes associated with CRF affect the cardiovascular system (especially the heart) adversely; more often than not, various cardiac layers and coronary vessels are affected.[5] Many patients on dialysis die of unknown cardiac causes that are thought to be related to high prevalence of left ventricular hypertrophy (LVH).[6] Co-founding factors of LVH seen in these patients include hemodynamic factors such as hypertension, fluid overload, and anemia and non-hemodynamic factors such as metabolic and endocrine abnormalities, autonomic dysfunction, ischemic heart disease, and cardiomyopathy.[5] Other putative risk factors may include iron and aluminum overload, hyperparathyroidism, and some unknown uremic toxins.

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These factors cause volume and/or pressure overload, and the combined effect of these overloads is thought to be the primary cause of LVH.[7,8] Cardiac hypertrophy occurs in patterns specific to inciting mechanical stress; therefore, volume overload results in eccentric hypertrophy while pressure overload would give concentric hypertrophy.[9]

In end-stage renal disease (ESRD) patients, LVH is closely related to systolic or pulse pressure.[9] London described systolic blood pressure (SBP) and pulse pressure as simplified markers of pressure load that result from interaction between cardiac factors, ie, stroke volume, ejection velocity, and the opposition to left ventricular ejection.[8]

The prevalence of LVH increases as kidney function worsens and may be as high as 70%–80% before initiation of dialysis.[5,8,10] Left ventricular hypertrophy (LVH) is documented as the most frequent cardiac alteration in ESRD and is an independent risk factor for survival in these patients.[6,8] Though LVH has been reported in essential hypertension and CKD, a close association of LVH with blood pressure level has not been uniformly documented in patients with CKD.[11]

Chronic renal failure (CRF) patients make up to 2%–8% of all hospital admissions in Nigeria,[2] and though cardiovascular complications are prevalent in them, information on LVH in African Black CKD patients is lacking. This study evaluates the prevalence of LVH and factors affecting it in pre-dialysis CRF patients by using echocardiography.

Method

The study was done at the University of Nigeria Teaching Hospital,

Enugu, Nigeria, a 750-bed tertiary health institution, between January 2002 and December 2003. The hospital serves approximately a quarter to a third of the Nigerian population, currently estimated to be 129 million.[12]

Ethical clearance was obtained from the ethics committee of the University of Nigeria Teaching Hospital, and the study group was drawn from patients attending the medical outpatient and renal clinics of the hospital. The first 100 patients with CKD who satisfied the inclusion criteria were recruited after informed consent was obtained. Patients ≥15 years of age with established CKD—ie, patients who had symptoms and signs of renal disease for ≥3 months, laboratory (proteinuria >1 g/dL) or imaging (kidney size <9 cm long) signs, and glomerular filtration rate (GFR) <60 mL/min/1.73m^2—and who had not received any form of renal replacement therapy and were presenting for the first time were included in the study. Patients who were on antihypertensives or any medication that could affect blood pressure were excluded. Some of the patients who had previously been diagnosed with hypertension but had stopped medication years before presenting to the unit with CKD were included in the study. Several investigators in the study area have documented similar observations, ie, patients are diagnosed and they default, only to present years later with advanced CKD.[2,13] Patients with preexisting primary cardiac disease, liver disease, arteriovenous fistula, or acute renal failure determined from past medical history, physical examination, electrocardiographic, radiologic, and laboratory results as well as from medical records from referring doctor were excluded. The control subjects were randomly selected from a population of normal subjects attending the hospital for medical examination for employment and pre-marriage evaluation and hospital staff in the ratio of two patients to one control subject. The control subjects had normal hemoglobin concentration level and blood pressure and no evidence of renal insufficiency, cardiac, liver, or bone disease. They were matched for age and sex with the patients. None of the patients or control subjects abused drugs or alcohol. The study was explained in full to them, and their consent was obtained.

Anthropometric data comprising height (Ht) and weight (Wt) were obtained from each patient, and body surface area (BSA) calculated from data using the formula BSA=(.0001) (71.84) ($Wt^{.425}$) ($Ht^{.725}$), where weight was in kilograms and height was in centimeters.[14] Systolic (SBP) and diastolic blood pressures (DBP) were measured with Accoson's mercury sphygmomanometer. The blood pressure of patients and control subjects were taken in a sitting position from the right arm after 15 minutes of rest. The mean of three values 10 minutes apart was taken. Hypertension was defined as mean arterial pressure ≥105 mm Hg and/or SBP ≥140 mm Hg and/or DBP using fifth Korotkoff phase ≥90 mm Hg.[15] Hypertension was classified according to JNC VI:

- mild: SBP 140–159 mm Hg and/or DBP 90–99 mm Hg
- moderate: SBP 160–179 mm Hg and/or DBP 100–109 mm Hg
- severe: SBP ≥180 mm Hg and/or DBP ≥110 mm Hg

Hemoglobin level, serum albumin, calcium, phosphate and alkaline phosphatase levels were measured and documented. Hemoglobin <12 g/dL for female subjects and <13 g/dL for male subjects were considered anemia. Serum calcium levels were corrected in relation to the albumin with the formula .01 mmol/L was added to the serum calcium concentration for every 4 g/L that albumin was <40 g/L; for albumin levels >40 g/L, .01 mmol/L was subtracted.[16]

A surrogate for hyperparathyroidism was taken as serum alkaline phosphatase >120 U/L.[17] Raised alkaline phosphatase has been shown to correlate with radiologic changes of hyperparathyroidism, and the isoenzyme pattern in CKD patients has been shown to be predominantly of osseous origin.[18] Creatinine clearance was calculated by using the Cockroft and Gault equation,[19] which correlates well with measured creatinine clearance in Nigerians.[20]

$$\text{Creatinine clearance} = \frac{(140 - \text{age}) \times \text{weight (kg)}}{.81 \times \text{serum creatinine } (\mu\text{mol/L})}$$

For females multiply by a factor of .85.

Degree of renal impairment was classified using the Kidney Disease Quality Outcome Initiative (K/DQOI) classification[21]:

Stage 0: GFR ≥90 mL/min with no renal damage
Stage 1: GFR ≥90 mL/min with renal damage
Stage 2: GFR 60 – 89.9 mL/min
Stage 3: GFR 30 – 59.9 mL/min
Stage 4: GFR 15 – 29.9 mL/min
Stage 5: GFR <15 mL/min

Echocardiography (M-mode, two-dimensional and Doppler) was done with the Siemens sonoline CD echocardiographic machine. The machine is equipped with 3.5 MHz transducer, a video recorder, and print-out processor. It has capabilities for M-mode, two-dimensional, pulsed-wave, and continuous-wave Doppler echocardiography. All patients underwent two-dimensionally guided M-mode echocardiogram and Doppler recorded in the left decubitus position. Left ventricular M-mode echograms of the parasternal long axis view were frozen on the display monitor and measured according to the Penn convention (p), taking the peak of the R wave as end diastole.[22]

Measurements were taken at or just below the tip of the mitral valve leaflets in areas of recording that showed the largest left ventricular internal dimen-

sion. Two cardiologists/echocardiographers did and read the echocardiograms to reduce intra-observer bias. Left ventricular mass was calculated by using an anatomically validated formula.[22]

$$\text{Left ventricular mass (p)} = 1.04\{(\text{IVSTp} + \text{LVEDDp} + \text{LVPWTp})^3 - (\text{LVEDDp})^3\} - 13.6 \text{ g}.$$

IVSTp — Interventricular septal thickness by Penn convention.

LVEDDp — Left ventricular end-diastolic dimension by Penn convention.

LVPWTp — Left ventricular posterior wall thickness by Penn convention.

Left ventricular mass index (LVMI) was calculated by dividing the left ventricular mass by body surface area. Left ventricular hypertrophy was defined in absolute terms as LVMI >134 g/m^2 in men and >110 g/m^2 in women.[17] Further characterization of LVH into eccentric or concentric hypertrophy was based on measurements of relative wall thickness.[7] Eccentric hypertrophy was present if the relative wall thickness (RWT) was <.45 in presence of LVH while LVH with RWT>.45 was termed concentric hypertrophy. Patients who had RWT>.45 but no LVH were considered to have concentric remodeling.[10]

Statistical Analysis

The Statistical Package for Social Sciences (SSPS Inc, Chicago, Ill) version 11.5 statistical software was used for data analysis. For continuous variables, mean values and standard deviations were calculated, and the means were compared by using analysis of variance or two sample *t* test. Categorical variables were compared by using the nonparametric tests chi-square and Kruskal Wallis. The cross-tabulation was used to analyze the relationship between the stages of CKD and LVH and other variables. All tests were two-tailed, and $P<.05$ was taken to be statistically significant.

Pearson's correlation was used to assess the relationship between LVMI and the variables (age, sex, body mass index, SBP, DBP, mean arterial pressure, pulse pressure, hemoglobin, creatinine, creatinine clearance, calcium and calcium phosphorous product) affecting it. Significant variables were further analyzed by using the stepwise method of multiple linear regression analysis to isolate possible determinants of LVMI based on their level of importance.

RESULTS

Table 1 describes the characteristics of the study and control populations. The predominant causes of CKD were chronic glomerulonephritis (43.2%), hypertension (25%) and diabetes mellitus (14.8%). All the patients in this study had either stage 4 (17.9%) or stage 5 (82.1%) CKD, a reflection of how late these patients present to healthcare facilities in the study area.

Prevalence of anemia was high (98.86%) in the patients; only one male patient did not have anemia.

The prevalence of LVH in this study was high, 95.5% in patients and 6.7% in controls. Apparently healthy and normal individuals who engage in intensive physical activity and athletes may have physiologic LVH.[7] Many Nigerians are subsistence farmers who engage in manual mode of farming. The most prevalent type of hypertrophy was eccentric (54.6%), followed by concentric (40.9%) and concentric–remodeling (1.1%). Hyperparathyroidism, defined in this study as alkaline phosphatase >120 IU/L, was found in three patients, and all of them had LVH. Additionally, LVH was not influenced by the primary kidney disease (R^2 .001, F .128, P=.721). Since only four patients did not have LVH in this study, no further analysis of patients by presence or absence of LVH was done.

Table 2 shows the Pearson correlation values for LVMI and factors that affect it in the study population (patients and control subjects). Creatinine clearance correlated best with LVMI. Significant variables were further analyzed with the stepwise method of multiple linear regression analysis to isolate variables based on their importance. Only mean arterial pressure, hemoglobin, male sex, and creatinine clearance were isolated as predictors, and mean arterial pressure was the best predictor when both patients and control subjects were included (Table 3). All the other variables were excluded. When only control subjects were analyzed, male sex and pulse pressure were isolated; mean arterial pressure was the only predictor identified when only patients were analyzed (Table 3). Approximately 52% of the variation in LVMI could be explained by these variables.

Prevalence of Hypertension

The mean arterial, SBP, and DBPs of the patients were high being 130.21 mm Hg, 172.73 mm Hg and 109.22 mm Hg respectively (Table 1). The prevalence of hypertension was very high being 85.2% and most had severe hypertension, ie, SBP ≥180 mm Hg and/or DBP ≥110 mm Hg (63.6%).

DISCUSSION

This study was undertaken to determine the prevalence of LVH and factors associated with it in adult Nigerians with CKD at first presentation before any form of renal replacement therapy (RRT) began. The prevalence of LVH was high (95.5%) in our patients, much higher than has been documented for pre-dialysis patients. Other studies have shown 40%–80% prevalence before initiation of RRT.[5,8,10] Our patients' evaluation was at first presentation to the hospital. In most developing countries, patients

Table 1. Characteristics of patients and control subjects

Characteristics	Controls	Patients	F	*P* value
Number	45	88		
Mean age (years)	42.84 ± 14.16	42.00 ± 15.23	.10	.757
Age range (years)	18 – 70	16 – 76		
Sex ratio	1.5:1	1.67:1		.781
Mean Body mass index (kg/m^2)	26.59 ± 5.05	24.50 ± 4.69	5.59	.02
Mean SBP (mm Hg)	118.78 ± 9.42	172.73 ± 32.37	119.43	<.001
Mean DBP (mm Hg)	73.56 ± 7.04	109.22 ± 21.51	116.91	<.001
Mean MAP (mm Hg)	86.59 ± 12.78	130.21 ± 24.08	128.72	<.001
Mean Pulse pressure (mm Hg)	45.18 ± 9.70	63.06 ± 19.02	35.34	<.001
Mean Hemoglobin (g/dL)	13.08 ± .85	7.79 ± 2.13	256.10	<.001
Mean Calcium x phosphorous product	3.42 ± .63	5.72 ± 2.77	30.17	<.001
Mean serum alkaline phosphatase (IU/L)	50.91 ± 13.99	51.71 ± 23.82	.15	.702
Mean serum albumin (g/L)	46.00 ± 6.93	30.97 ± 6.65	121.93	<.001
Mean serum creatinine (µmol/L)	102.72 ± 22.56	956.77 ± 420.77	137.01	<.001
Creatinine clearance (mL/min)				
Mean	101.65 ± 9.27	9.68 ± 5.49	5150.89	<.001
Range	90.30 – 126	3 – 34		
LVMI (g/m^2)	82.88 ± 22.19	216.19 ± 80.00	91.26	<.001
LVEDD (mm)	47.06 ± 5.60	54.64 ± 6.99	31.97	<.001
RWT (mm)	.35 ± .09	.47 ± .17	15.68	<.001
IVS (mm)	8.92 ± 1.60	13.72 ± 3.62	55.48	<.001
Ejection fraction (%)	60.82 ± 8.66	56.32 ± 12.91	3.58	.063
Prevalence of hypertension (%)	0	85.2		<.001
Severity of hypertension (%)				
Mild		3.4		
Moderate		18.2		
Severe		63.6		
Prevalence of LVH (%)	6.7	95.5		]<.001
Eccentric	0	54.6		
Concentric	6.7	40.9		
C-remodel	0	1.1		
No hypertrophy	93.3	3.4		
Primary cause of kidney disease [number (%)]				
Chronic glomerulonephritis		38 (43.2)		
Hypertensive nephrosclerosis		22 (25.0)		
Diabetic nephropathy		13 (14.8)		
Unknown		6 (6.8)		
HIV-associated nephropathy		5 (5.7)		
Adult polycystic kidney disease		2 (2.3)		
Chronic pyelonephritis		1 (1.1)		
Obstructive uropathy		1 (1.1)		

Abbreviations: SBP=systolic blood pressure; DBP=diastolic blood pressure; MAP=mean arterial pressure; LVMI=left ventricular mass index; IVS=intra-ventricular septum; LVEDD=left ventricular end diastolic dimension; RWT=relative wall thickness; LVH=left ventricular hypertrophy.

present late to health facilities for several reasons, ranging from prohibitive cost of services to use of alternative treatment like spiritual healing and traditional/native healers.[13,23] Most patients in our study had hypertension (85.2%), and hypertension is one of the factors shown to best predict LVH.[24]

Of the variables tested, mean arterial pressure, hemoglobin concentration, male sex and creatinine clearance predicted the occurrence of LVH. While mean arterial pressure and male sex correlated positively, hemoglobin concentration and creatinine clearance correlated inversely with LVMI. They accounted for 32%, 17%, 22%, and 24%, respectively, of the variation in LVMI. Both pressure overload and volume overload are the main stimuli for LVH. Interplay between the two is complex and influences the type of LVH that develops.[9] The findings in our study suggest that the primary stimulus may be volume overload, which produces more eccentric LVH. Factors contributing to volume overload include anemia and sodium and water retention, which are often marked in CKD patients in the study area.[2] Unlike other studies in which LVH was more closely related to SBP or pulse pressure,[9,24] in our study, LVH was more related to mean arterial pressure. However, studies have shown that peripheral resistance and mean arterial pressure as well as DBP are frequently increased in early renal disease and CRF.[7] But as anemia worsens with

Table 2. Correlations values of left ventricular mass index left ventricular mass index and various parameters in study population (patients and control)

Parameters	Pearson Correlation	
	r	*P* value
Age	.03	.761
Sex	.23	.012
Body mass index	.16	.112
Systolic blood pressure	.60	<.001
Diastolic blood pressure	.61	<.001
Mean arterial blood pressure	.62	<.001
Pulse pressure	.38	<.001
Hemoglobin	.56	<.001
Creatinine	.57	<.001
Creatinine clearance	.64	<.001
Calcium	.06	.51
Calcium-phosphorous product	.17	.07

progression of CKD, blood viscosity is decreased; this, in addition to the creation of fistulas, causes peripheral resistance to normalize or decrease in uncomplicated ESRD.[7,25] Our patients did not have fistulas, but they had significant anemia as well as significantly high mean arterial pressure; therefore, peripheral resistance may have played a significant role in LVH seen in our patients. The mean hemoglobin concentration of patients was 7.79 g/dL, and only one of the patients studied had normal hemoglobin. LVH and hemoglobin were significantly correlated. Anemia has been consistently associated with LVH in CKD population; anemia was a significant independent factor in development of LVH in this study.

Stepwise multiple regressions of control subjects showed that male sex and pulse pressure were predictors of LVH, which reflects what has been described in other studies.[8,9,26] Like in other studies,[10,27] eccentric hypertrophy (54.6%) was more prevalent than the concentric type (40.9%). Levin et al[10] noted 57.8% for eccentric and 24% for concentric in pre-dialysis patients. London noted that anemia is present in most patients initiating dialysis and that it could explain the high prevalence of LVH in the patients.[7]

Further analysis on the basis of presence or absence of LVH was not done because of the small number of patients who did not have LVH. All the three patients with serum alkaline phosphatase >120 IU/L (a surrogate of hyperthyroidism) had LVH (r=.500, P=.667). Because few patients in this present study had values >120 IU/L, not much can be said about the impact of hyperparathyroidism on LVH. Previous studies have not demonstrated a consistent relationship between parathyroid hormone and LVH.[28] Some showed a trend toward high absolute parathyroid hormone levels in patients with LVH, but the trend did not reach statistical significance.[10] Other factors, such as age, serum calcium, and calcium-phosphorous product, shown to be predictors of LVH in other studies,[11,17] did not appear important in this study.

This study has some limitations. Because few patients had early stages of CKD in this study, the influence of stage of CKD on LVH could not be assessed. A larger sample size would yield a clearer result. The absolute LVMI cutoff for Nigerians is not known. The only available study showed average LVMI for normal male population of 122.1 g/m^2, but this study considered only male subjects and the number was too small (25 subjects) for conclusive inference.[29] Figures from studies done elsewhere were used.[17] The use of serum alkaline phosphatase >120 IU/L as surrogate for hyperparathyroidism could give erroneous results, as hepatitis B virus infection, which also causes elevated alkaline phosphatase, is prevalent in the study area. Congestive heart failure, pancreatitis, malignancy, and vitamin D deficiency also influence alkaline phosphatase. Unfortunately, facilities for parathyroid hormone assay are not readily available.

In conclusion, this study has shown a strong association between CKD and LVH in patients in a developing country at the time of first evaluation by

Table 3. Stepwise multiple linear regressions of factors that correlate with left ventricular mass index in study population (patients and control), control subjects only, and patients only

Model	Unstandardized Coefficients		Beta	*t*	Significance	95% Confidence Interval for B
	B Error	Standard				
Patients and control						
Constant	130.56	42.07		3.10	.002	47.20 – 213.92
Creatinine clearance	−.56	.29	−.24	−1.94	.055	−1.14 – .12
Mean arterial pressure	.97	.28	.32	3.53	.001	.43 – 1.51
Male sex	31.35	12.49	.17	−2.51	.014	6.60 – 56.11
Hemoglobin	−6.98	3.36	−.22	−2.08	.040	−13.63 – −.33
Control subjects only						
Constant	35.06	19.69		1.78	.087	−5.41 – 75.54
Male sex	22.14	7.83	.48	2.83	.009	6.05 – 38.24
Pulse pressure	.82	.39	.36	2.11	.044	.023 – 1.62
Patients only						
Constant	55.45	44.98		1.24	.220	−33.88 – 144.96
Mean arterial pressure	1.23	.34	.37	3.62	.001	.55 – 1.90

a nephrologist. The patients are often anemic and have severe hypertension even at first presentation.

Unfortunately, the cost of treatment of CKD/ESRD is prohibitive. Most developing countries like Nigeria do not have a well-articulated health insurance program or social security system to help these patients. However, strategies and measures to prevent the development and progression of LVH at an early stage may prove more effective than attempts to induce regression of established LVH. Early detection and treatment of causes of CKD should be pursued aggressively at the primary prevention level, as has been advocated by International Society of Nephrology, to reduce the impact of CKD and its attendant complications in the society.

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