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C-reactive Protein is an Independent Predictor of Left Ventricular Mass in Offspring of Hypertensive Subjects in Nigeria

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Authors' contributions

This work was carried out in collaboration among all authors. Author OMA designed, performed the project work, analyzed and interpreted the data. Author OMA was the main contributor in writing the manuscript. Author AAA designed, analyzed and interpreted the data, also a major contributor in writing the manuscript. Authors FTA, OAA, YAA, OGO and ABO designed, analyzed and interpreted the data, also contributor in writing the manuscript. Author JOA Analyzed and interpreted the data. All authors read and approved the final manuscript.

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ABSTRACT

Offspring of hypertensive parents have been shown to be at increased risk of developing systemic hypertension and adverse cardiovascular events later in life. The pathological antecedents of this are thought to be alterations in the structure and function of left ventricle. However, it is currently unclear if these abnormalities are due to genetic factors or a result of higher biomarker levels such as highly sensitive C reactive protein (hsCRP). An improved understanding of the associations of hsCRP with left ventricular structure may offer additional insight. Therefore, this study aims at determining the correlation of left ventricular mass with hsCRP among offspring of hypertensive parents compared with controls.

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Methodology: A cross sectional Hospital based study, with 100 subjects and 100 controls. A questionnaire was administered to obtain relevant history, physical examination, blood tests, ECG, Echocardiography were done for the two groups. The results were analysed using SPSS 20.
Results: The left ventricular mass and mass index was significantly elevated in the subjects compared with the control group. The median hsCRP was significantly higher in the subjects [1.85 (0.28-10.20) vs. 1.34 (0.17-8.49) mg/L: $P < 0.010$]. It progressively increases significantly as the number of parent with hypertension increases [1.34 (0.17-8.49), 2.00(0.28-9.66) and 2.54(0.91-10.20) mg/L $P < 0.001$] from zero, to single and both parent respectively. There was a significant correlation between hsCRP levels, blood pressure, left ventricular mass and left ventricular mass index (R = 0.165, 0.316, 0.274: $P = 0.021$, 0.004, 0.014) respectively.
Conclusion: The study shows that offspring of hypertensive parents had higher echocardiographic left ventricular mass, left ventricular mass index and hsCRP levels compared with controls and this hsCRP increases as the number of parents with hypertension increases. Blood pressure and left ventricular mass index increase with increasing Plasma hsCRP: This may suggest possible role of hsCRP in the development of hypertension and cardiac remodeling.

Keywords: C-reactive protein; hypertension; left ventricular mass; offsprings.

1. INTRODUCTION

Hypertension (HTN) is a chronic medical condition in which the blood pressure (BP) is persistently elevated [1]. It is an important risk factor that is associated with increased cardiovascular (CV) morbidity and mortality. Hypertension constitutes a significant public health burden in both developed and developing countries. It is the most important modifiable risk factor for coronary heart disease (CHD), stroke, congestive heart failure (CHF), end-stage renal disease (ESRD), and peripheral vascular disease (PVD), hence the need to promote a healthy lifestyle and preventive strategies to decrease the prevalence of hypertension in the general population [2] .

Genetics and environmental factors interplay in the aetiopathogenesis of hypertension. Furthermore, it is well established that HTN clusters in families, and that a positive family history represents a major risk factor for future HTN in normotensive offsprings. Adoption, twin, and family studies document a significant heritable component to BP levels and HTN [3,4]. The specific mechanisms are not fully understood. However, some studies have shown that circulating concentrations of several biomarkers that represent distinct biological pathways are correlated with blood pressure levels cross-sectionally [5,6]. For instance, a report from the Framingham Heart Study demonstrated that circulating concentrations of C-reactive protein (CRP) and plasminogen activator inhibitor-1 (PAI-1) predicts incident hypertension in non-hypertensive individuals during short-term follow-up(5). Circulating levels of highly sensitive C reactive protein (hsCRP) are clinically used to predict occurrence of cardiovascular events through several mechanisms [7–12]. Finally, parental HTN has been shown to be associated with higher hsCRP concentration in normotensive offsprings with 15% increase in hsCRP levels per parent with HTN [13,14]. HsCRP may thus be useful in identifying offsprings with higher risk for developing HTN, due to the pro-artherogenic metabolic status of higher inflammatory biomarker level in them which makes them susceptible to more rapid progression and attendant complications of cardiovascular disease. However, it is not clear if higher serum levels of hsCRP are associated with higher levels of echocardiographic abnormalities that have been shown to be higher among offsprings of hypertensive subjects in Nigeria. However, to the best of the investigator's knowledge, none of the previous studies on offspring of hypertensive subjects have assayed for hsCRP. Thus, there is no local data on the possible association between hsCRP and other cardiovascular risk factors among offsprings of hypertensive subjects in Nigeria.

2. MATERIALS AND METHODS

The study was a cross sectional study, carried out at the Cardiology unit of the Department of Internal Medicine, LAUTECH Teaching Hospital, Ogbomosho, Oyo state, Nigeria. One hundred (100) Subjects who were offsprings of hypertensive subjects with essential HTN who presented at the cardiology clinic of LAUTECH Teaching Hospital, Ogbomoso, Oyo State. Healthy subjects, sex and age-matched were

also recruited from amongst offsprings of normotensive patients who were treated for other medical conditions other than hypertension and from GOPD after having confirmed to be normotensives and do not have any significant medical history that may suggest otherwise, to serve as controls. Only those who satisfied the selection criteria among consecutive subjects were involved in the study. The subjects comprised consenting normotensive individuals who were 18 years and older, but less than 45 years with a positive family history of HTN, asymptomatic for hyperglycaemia and have normal venous glucose levels. They were normotensive as at the time of the examination and were not on antihypertensive medications and not previously diagnosed with HTN. We also excluded those with acute or chronic illnesses, or on medications.

Patients with essential HTN were randomly selected by balloting at each cardiology clinic over the study period (five subjects per clinic for convenience and considering the evaluations and investigations to be done); those selected were asked to bring their offsprings (first order offspring who fall into the age category to be studied) and spouse during the next clinic visit. Subjects were consecutively recruited through consenting parents.

Informed consent was obtained from all the parents including recruited offsprings, after which a preformed composite interviewer-administered questionnaire was used to obtain information on socio-demographics and relevant history. Physical examination was done, after which ECG, ECHO and laboratory profile (Urea, creatinine, fasting plasma glucose, uric acid, fasting lipid profile and hsCRP) were carried out on consenting participants.

Clinical characteristics were descriptively compared between normotensive offsprings of parent with and without hypertension. Numerical values were reported as means± standard deviations, while categorical variables were expressed as proportions and percentages. Student's *t*-test was used to ascertain significance for continuous variables while chi square test was used for categorical data. Non-parametric test was used to analyze data that were not normally distributed. Differences between more than two means for continuous variables were analyzed using one-way ANOVA. A multivariable linear regression analysis was performed in successive steps to estimate the association between hsCRP (independent variable) and other CV risk factors (dependent variables). Highly selective CRP concentrations among offspring with 1 and 2 parents with HTN were compared with levels in offspring of non-hypertensive parents adjusting for relevant covariates (model 1: age and sex; model 2: age, sex, BMI, systolic and diastolic BP, HDL cholesterol ratio and triglycerides) that have been previously related to biomarker levels in cohort study [15]. A p value <0.05 or 95% CI (CI95%) defined statistical significance. Data was analyzed using the statistical package SPSS for Windows version 20.0 (IBM Company, Chicago, IL).

All the cost of the study was borne by the investigator.

3. RESULTS

3.1 Characteristics of Participants

A total of 100 NOHP and 100 NONP were analysed. Out of the 100 normotensive offsprings of hypertensive parents, 79 had single parent with hypertension. Forty-three (43) had hypertension in the mother, while thirty-six (36) had paternal hypertension. Twenty-one (21) had both parent hypertensive.

3.2 Anthropometric, Clinical and Bio-chemical Parameters of Participants

The mean weight of the subjects was 62.84 ± 11.69 kg and was not statistically different from the control 60.02±9.57 kg (*P*=0.063). Likewise, the mean height in the subjects and controls were similar: 1.66±0.08 m vs 1.64±0.08 m respectively (*P*=0.079) as shown in Table 1. However, the waist circumference (80.37±15.52 vs 75.99±8.84) cm, hip circumference (94.55±9.15 vs 90.39±9.93) cm and neck circumference (33.77±4.60 vs 32.31±4.43) cm were significantly higher in the subjects compared to the control group as shown in Table 1 with p values 0.015, 0.002 and 0.023 respectively. The mean blood pressure was 111.82 ± 13.58 vs 110.02 ± 9.96 mmHg for systolic and 70.06 ± 9.52 vs 70.44 ± 8.97 mmHg for diastolic BP, there was no statistically significant difference between the two groups (*P*=0.287 and 0.772 respectively) as show in Table 1. The mean LDL (3.87 ± 0.61 vs 3.73 ± 0.59) mmol/L, triglycerides (1.64 ± 0.27 vs 1.59 ± 0.26) mmol/L and total cholesterol (5.35 ± 1.11 vs 5.27 ± 1.01) mmol/L were higher in the subjects than control group though not

statistically significant (*P* = 0.101, 0.184 and 0.595 respectively). The mean HDL (1.23 ± 0.33 vs 1.34 ± 0.34) was however, significantly lower among the subjects compared with controls (p=0.021) the atherogenic index of plasma was also significantly higher among the subjects than controls {0.51(-9.94 to 10.41) vs 0.41(-19.25 to 9.13): *P*=0.039} (Table 1).

3.3 Echocardiographic Indices among Study Participants

The mean LVIDd in the subject group (42.75 ± 5.09 mm) was significantly higher than that of the control group (40.92 ± 4.89 cmm) *P* = 0.010. Higher values were also seen in the LVIDs (28.80 ± 3.53 vs 28.63 ± 3.00), IVSTd (10.73 ± 2.21 vs 10.54 ± 2.47) and PWTd (9.39 ± 4.01 Vs 9.12 ± 1.99) parameters of the subject group when compared to the control group, though they were not statistically significant (*P* = 0.714, 0.567, 0.547 respectively) as shown in Table 2. The mean LVM and LVMI were significantly higher among subjects compared with controls (171.37 ± 48.83 g vs 149.68 ± 47.03 g) and (105.20 ± 28.67 g/m^2 vs 92.45±16.87 g/m^2) respectively with *P* = 0.002, 0.002 as shown in below. Twenty-eight (28%) of the subject group had echocardiographic LVH compared with thirteen (13) in the control group (*P* = 0.009).

The mean LVEF of the subjects was 59.58 ± 6.24% while that of the control group was 61.14 ± 5.81% (*P* = 0.069).

3.4 HsCRP Levels between Subjects and Control Group

The distribution of the hsCRP was skewed, so median (range) was used as a measure of central tendency to represent the distribution. The hsCRP was significantly higher in subjects [1.85 (0.28-10.20) mg/L] than the controls [1.34 (0.17-8.49) mg/L] with *P* = 0.010. Using the American Heart Association CRP risk stratification, 37% of the subjects compared with 21% of the controls were in the high risk category (> 3 mg/L) for hsCRP levels (*P* = 0.036) as shown in Table 3.

Table 1. Socio-demographic characteristics and labouratory parameters of subjects and controls

Variables	Subjects(100) Mean ± SD	Control(100) Mean ± SD	P value
Sex			
Male	54 (54.0)	56 (56.0)	0.776
Female	46(46.0)	44 (44.0)	
Age (in years)	25.42± 5.80	24.27±5.40	0.148
Weight (Kg)	62.84 ± 11.69	60.02±9.57	0.063
Height (M)	1.66±0.08	1.64±0.08	0.079
BMI(kg/m^2)	22.79±8.79	22.31±7.92	0.685
Waist Circum (cm)	80.37±15.52	75.99±8.84	0.015*
Hip Circum (cm)	94.55±9.15	90.39±9.93	0.002*
Waist/hip Ratio	0.85±0.07	0.84±0.08	0.348
Neck Circum (cm)	33.77±4.60	32.31±4.43	0.023*
Diastolic BP (mmHg)	70.06±9.52	70.44±8.97	0.772
Systolic BP (mmHg)	111.82±13.58	110.02±9.96	0.287
Glucose (mmol/L)	4.11±1.00	4.18±0.99	0.619
Urea(mmol/L)	3.88±0.78	3.68±0.71	0.059
Creatinine(µmol/L)Median(range)	96.35(43.20-189.34)	92.90(44.20-169.00)	0.826
LDL(mmol/L)	3.87±0.61	3.73±0.59	0.101
HDL(mmol/L)	1.23±0.33	1.34±0.34	0.021*
Triglycerides(mmol/L)	1.64±0.27	1.59±0.26	0.184
TC (mmol/L)	5.35±1.11	5.27±1.01	0.595
AIP Median(range)	0.51(-9.94-10.41)	0.41(-19.25-9.13)	0.039*

Key: SD=Standard deviation. Subjects with parental history of hypertension, Control group with no parental history of hypertension. AIP=Atherogenic index of plasma, TC= Total Cholesterol, LDL=Low density lipoprotein, HDL=High density lipoprotein

Table 2. Echocardiographic findings of subjects and controls

Variables	Subjects Mean ± SD	Control Mean ± SD	P value
LVIDd (mm)	42.75±5.09	40.92±4.89	0.010*
LVIDs (mm)	28.80±3.53	28.63±3.00	0.714
IVSTd (mm)	10.73±2.21	10.54±2.47	0.567
PWTd (mm)	9.39±4.01	9.12±1.99	0.547
RWT	0.42±0.07	0.41±0.05	0.246
LAD (mm)	34.28±3.59	33.47±4.27	0.148
AOD (mm)	28.11±4.01	27.39±3.56	0.181
EF (%)	59.58±6.24	61.14±5.81	0.069
LVM (g)	171.37±48.83	149.68±47.03	0.002*
LVMI (g/m^2)	105.20±28.67	92.45±16.87	0.002*
ECHO LVH			
No	72	87	0.009*
Yes	28	13	

*Key: *: Statistically significant (P value < 0.05). LVIDd = left ventricular internal dimension in diastole, LVIDs = left ventricular internal dimension in systole, IVSTd=interventricular septal thickness in diastole, EF = Ejection fraction, LVM = left ventricular mass, LVMI = left ventricular mass index, LVEF= left ventricular ejection fraction, LAD= Left atrial diameter, AOD= Aortic root diameter, SD=Standard deviation. Subjects with parental history of hypertension, Control group with no parental history of hypertension*

Table 3. Showing the plasma hsCRP of subjects and controls

Variables	Subjects n=100(%)	Control n=100(%)	P value
hsCRP(mg/L)Median(Range)	1.85(0.28-10.20)	1.34(0.17-8.49)	0.010*
Category			
<1	10 (10)	16(16)	
1-3	53 (53)	63 (63)	0.036*
>3	37 (37)	21 (21)	

*Key: *= Statistically significant (P value < 0.05), hsCRP= highly sensitive C reactive protein, SD= standard deviation. The division and distribution into 3 groups (<1, 1-3 and >3) is based on the American Heart Association CRP risk stratification levels. Subjects with parental history of hypertension, Control group with no parental history of hypertension*

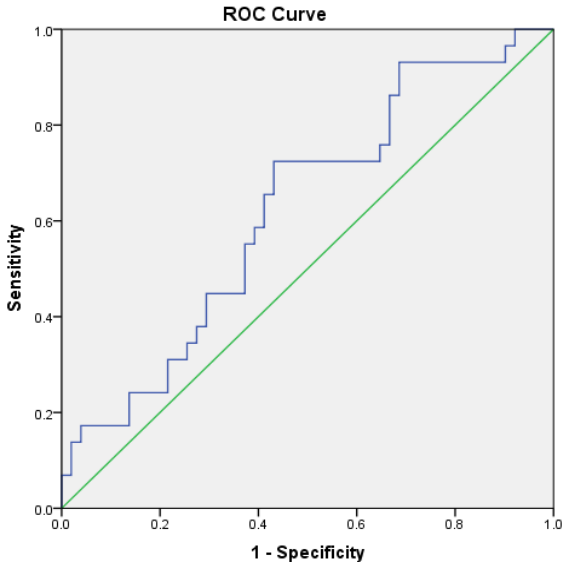

Fig. 1. ROC curve for hsCRP and left ventricular hypertrophy among NOHP

AUC = 0.623 (0.497-0.748); P=0.069

Key: ROC= Receiver Operator Characteristic Curve; AUC= Area under curve

Table 4. Linear regression between hsCRP, blood pressure parameters and echocardiographic LVH in the study subjects

Dependent variable	Univariate hsCRP model	Mean ± SE	B Estimate (95% CI)	*p*-value	Adjusted for age, sex, BMI, TC,HDL, Uric Acid	P-value
SBP	Low intermediate high	107.8 ± 3.1	-	< 0.001	-	
		113.3 ± 1.4	5.5 (-1.3 to 12.3)		7.2 (0.4 to 14.1)	<0.001
		122.4 ± 1.6	14.6 (7.5 to 21.7)		16.0(8.9 to 23.0)	
DBP	Low intermediate high	64.7 ± 3.6	-		-	
		67.9 ± 1.3	3.2 (-3.7 to 10.2)	0.110	4.7 (-2.3 to 1.6)	0.100
		71.5 ± 1.9	6.8 (-0.4 to 14.1)		7.4 (0.3 to 4.6)	
LVMI	Low intermediate high	92.6 ± 1.2	-		-	
		113.4 ± 2.8	20.8 (5.6 to 36.0)	<0.001	22.1(6.3 to 37.8)	<0.001
		136.9± 4.4	44.2 (28.6 to60.0)		45.6(29.4 to 61.8)	

*Key: R: Pearson correlation coefficient; *: Statistically significant (P value < 0.05). Key: Regression equation for SBP, DBP and LVMI= βo+ β1 (CRPm) + β2 (CRPh). βo= Estimate for low hsCRP category which is the reference point.β1= Estimate for intermediate hsCRP (CRPm)category.β2= Estimate for high hsCRP (CRPh)category, For individuals with: hsCRP <1, then CRPm and CRPh will be 0.hs CRP between 1-3, then CRPm=1, while CRPh=0hsCRP >3, then CRPm=0, while CRPh=1*

Table 5. Effect of number and type of parent with hypertension on parameters among the study subjects

Variables	MHH(43) Mean ± SD	PHH(36) Mean ± SD	BFHH (21) Mean ± SD	P value
LVMI	98.30±13.09	104.00±16.59	117.56±30.63	0.001* MHHvs PHH=0.465 MHHvs BHH<0.001 PHHvs BHH=0.018
LVM	156.23±20.73	170.89±24.39	194.22±49.18	<0.001* MHHvs PHH=0.093 MHHvs BHH<0.001 PHHvs BHH=0.015
hsCRP Median (range)	1.89(0.28-9.66)	2.50(0.51-9.40)	2.54(0.91-10.20)	0.127
Tp-e/QT	0.24±0.06	0.25±0.05	0.26±0.12	0.582

**: Statistically significant (P value < 0.05), PHH=paternal history of hypertension, MHH= Maternal history of hypertension, BPHH= Both parental history of hypertension, PHT=Post Hoc Test (Bonferroni), Z= Kruskal Wallis test*

3.5 Relationship of Hscrp to Echocardiographic Left

3.5.1 Ventricular parameters among subjects

The bivariate correlation between plasma hsCRP and Echo LV indices revealed a statistically significant correlation between plasma hsCRP and LVM (r= 0.472, $P < 0.001$) and LVMI (r =0.488, $P < 0.001$). Moreover, in a univariate linear regression model, the LVMI increases as the hsCRP risk stratification category increases from low (92.6 g/m^2), intermediate (113.4 g/m^2) to high (136.9 g/m^2) risk respectively ($P < 0.001$). In a multivariable analysis adjusting for age, sex, BMI, lipids and uric acid, there was still a significant relationship between the hsCRP and LVMI as shown in Table 4. HsCRP level was predictive for the development of left ventricular hypertrophy (Area under receiver operation curve = 0.623 (0.497-0.748); P=0.069) as shown in Fig. 1.

4. DISCUSSION

4.1 Socio-demographic Characteristics of the Subjects/ Controls

The age of the study population ranges from 18-44 (mean age of 25.42± 5.8) years. This was similar to the study of Burke et al. [16] (18 -30 years), Beatty et al. [17] (18-32 years with a mean of 25.80 ± 3.9) and Adeoye et al. [18] in Ibadan (mean age of 25.00 ± 5.31). It was however slightly higher than the mean age used by Kolo et al. [19] (15-25 years) and Amadi et al. [20] (13-19 years). Although, Obasohan et al. [21] (31.05 ± 1.58) used older age group due to the inclusion of a third group of newly diagnosed hypertensive subjects with higher age group. The ages used in this study were young adults with lower likelihood to have developed essential hypertension or its complications.

4.2 Echocardiographic Indices in the Study Population

The study showed that LVDD, LVM and LVMI were significantly higher in the NOHP than in the controls. The observations agreed with findings from previous studies which also showed increased LVM in NOHP [19,20,22,23]. However, this result contrasts that of Adeoye et al. and Jalal et al. who did not find significant difference in the LVM between the two groups [18,24]. Several studies have shown the clinical impact of left ventricular hypertrophy on morbidity and mortality in cardiovascular diseases in adults, yet the origin of increased LVM and its clinical significance is still not clear in NOHP. Some investigators have suggested that increased LVM may be a forerunner of hypertension and future cardiovascular events in these individuals [25,26]. Before changes in hypertension become apparent, various changes have been proposed to occur at the cellular level that cause alterations in the myocardial architecture and collagen structure coupled with metabolic and neuro-endocrine changes which could explain the increased LVM in the offsprings of hypertensives [18,22]. Radice et al. had also suggested earlier that cardiac involvement may precede elevation of blood pressure in offsprings of hypertensive subjects [22].

4.3 Highly Selective C Reactive Protein among Subjects and Controls

This study showed a significantly higher hsCRP level among the NOHP when compared to the healthy matched control ($P = 0.001$). The higher plasma hsCRP level was similar to what was reported by Diaz et al. in a Spanish multicenter study (0.60 (0.15-8.4) vs 1.16(0.15-9.0): p=0.001) [13]. Idemudia *et al* had earlier shown similar higher trend albeit in hypertensive subjects in Nigeria with C- reactive protein values (0.18 ± 0.1 mg/dL vs 0.08 ± 0.04 mg/dL: $P < 0.0001$), thus establishing a positive association between elevated CRP levels and higher BP in adults [27]. Furthermore, in a recent prospective study by Sesso et al. elevated CRP levels were associated with increased risk of developing HTN (6). Ridker et al. observed that CRP level and parental history of cardiovascular disease increased the global cardiovascular risk prediction using the Reynolds risk score, thus NOPH with higher hsCRP in this study will be at higher risk for the development of HTN and other cardiovascular diseases later in life [28,29]. These observations of higher hsCRP among NOHP may also indicate that HTN could in some way be considered an inflammatory disorder like atherosclerosis. A chronic inflammatory state may induce endothelial dysfunction, impairing the ability of the endothelium to produce nitric oxide and prostacyclin, which can contribute to the development of HTN [30].

Plasma CRP levels have been used in the diagnosis and monitoring of inflammatory and acute infectious diseases in children and adults. Although conventional CRP assays are not able to detect blood levels <1 mg/l. The development

of high sensitivity CRP assays, such as that used throughout this study, has permitted detection of low-grade inflammation in healthy individuals. In this study, hsCRP was significantly elevated in a sample of young adults with at least one parent with HTN. The observation of elevated hsCRP values without consistent elevation of current BP levels does not exclude a possible role in hypertension and could indicate that hsCRP elevation is a phenomenon that takes place before actual HTN occurs. Using the American Heart Association CRP risk stratification levels, 37% of the NOHP had hsCRP levels > 3 mg/L compared to 21% of the control. The difference was statistically significant which is similar with Diaz *et al* observation (7.8% vs. 2.9%: *P* =0.029).

Moreover, CRP levels are clearly positively correlated with systolic and diastolic BP in these subjects. This is similar to what Idemudia observed though among adult hypertensive subjects in a study in Benin, Nigeria [27]. Cook et al. also observed a positive but weak correlation between CRP and SBP levels in a study where 699 healthy boys were recruited [31]. This study also shows that NOHP have a positive trend towards higher systolic and diastolic BP values as the hsCRP increases. Furthermore, in a linear regression multivariable model, the blood pressure increases significantly as the risk category of hsCRP increases from low (107.8 mmHg), intermediate (113.3 mmHg) and high (122.4 mmHg) risk group. The area under receiver operation curve of 0.608 observed in this study for the development of prehypertension suggest that hsCRP level may thus predict the development of hypertension. However, a prospective follow up study design will better establish this as was earlier shown by Sesso et al. [6].

4.4 Relationship between Plasma Hscrp Levels and Echocardiographic Indices of Left Ventricular Functions

This study revealed a significant positive correlation between LVM, LVMI and hsCRP level. Seyfeli et al. had earlier revealed significant associations of hsCRP with left ventricular mass in newly diagnosed hypertensives [32]. Masugata et al. [33] and Shi et al.[34] also observed that hsCRP was an independent predictor of LVM among subjects with cardiovascular risk and lupus nephritis respectively. Kaunang et al. however did not find any correlation between hsCRP and LVM among obese aldolescents in Indonesia [35]. Previous offspring studies that have looked at the echocardiographic indices in Nigeria did not assay for hsCRP levels thus hsCRP was not correlated with LVM. The finding of significant correlate with LVM and LVMI in this study may suggest the possible role os hsCRP in cardiac remodelling even before overt development of hypertension.

4.5 Effect of Number of Parent with Hypertension on Hscrp and Other Cardiovascular Risk Factors

This study showed that the hsCRP levels increase significantly as the number of parent with hypertension increases. This is similar to what Lieb et al. [14] reported that parental hypertension was associated with higher hsCRP concentration in offsprings (15% increase per parent with hypertension; *P*=0.004). This study revealed a stronger paternal than maternal influences on LVM and LVMI contrary to what had been documented by earlier studies [20,24]. Amadi et al. in LUTH found higher LVMI for offsprings of maternal hypertensives compared with paternal hypertensives [20]. Kuznetsova et al. studying adult offsprings of hypertensives, their parents and first degree relatives simultaneously found stronger maternal-offspring LVM correlation than paternal-offspring LVM correlation [36]. In this study, the cohort was the young adult age group only, parental LVM and those of first degree relatives were not determined and thus could not be correlated with the LVM of offspring. These differences in methodology and analysis probably can account for the observed difference.

5. CONCLUSION

This study shows that offspring of hypertensive parents have higher echocardiographic LVM, LVMI and hsCRP levels compared with controls. HsCRP was observed to rise as the number of parents with hypertension increases. Blood pressure and left ventricular mass index correlated significantly with increasing Plasma hsCRP which may suggest possible role of this biomarker in the cardiac remodeling and eventual development of LV hypertrophy. The study therefore recommends regular medical check for offsprings of parent with hypertension for early detection of cardiovascular and other metabolic abnormalities. Moreover, primary cardiovascular prevention should be advocated among offsprings of hypertensive subjects to retard progression of cardiovascular disease. A

large multicentre randomised study may be suggested in order to establish and validate above findings.

CONSENT

Informed consent was also obtained from all participants in the study.

ETHICAL APPROVAL

All experimental protocols were approved and performed in accordance with the guiding principles of LAUTECH Teaching Hospital Ethical Review Committee.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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