

Original Research Article

Correlation between echocardiographic left ventricular mass and atherogenic index of plasma in adult hypertensive subjects in Olabisi Onabanjo university teaching hospital, Sagamu

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ABSTRACT

Background: Hypertension and dyslipidaemia are two major modifiable cardiovascular risk factors with their co-existence having more than an additive effect on endothelial function causing atherosclerosis. The purpose of this study was to determine the prevalence of dyslipidaemia in hypertensive subjects and to determine its relationship left ventricular hypertrophy.

Methods: The study was a cross-sectional comparative one involving 120 hypertensive participants with LVH (subjects) and 60 age and sex-matched hypertensive participants without LVH (controls). Detailed history, physical examination, fasting lipid profile test, and echocardiogram were carried out on all participants.

Results: The overall prevalence rate of dyslipidaemia in the study was 61.1%. The prevalence of dyslipidaemia in subjects (60.8%) was slightly lower than in controls (61.7%), though the difference was not statistically significant ($p=0.914$). The most common isolated lipid abnormality in the study was elevated serum LDL-C (55% in subjects, 46.7% in controls), though the difference did not achieve statistical significance ($p=0.370$). The mean atherogenic index of plasma (AIP) was significantly higher in the subjects (0.34 ± 0.23) than in the controls (0.22 ± 0.28) ($p=0.001$). There was a positive correlation between echocardiographic left ventricular mass and AIP ($r=0.298$, $p=0.001$).

Conclusion: There is a high prevalence of dyslipidaemia among hypertensive adults. There is also a positive correlation between echocardiographic left ventricular mass and AIP among adult hypertensive subjects.

Keywords: Hypertension, Echocardiogram, Left ventricular mass, Dyslipidaemia, AIP, Atherosclerosis

INTRODUCTION

Non-communicable diseases (NCDs) kill 41 million people each year, equivalent to 71% of all deaths globally. Cardiovascular diseases account for most non-communicable disease deaths, or 17.9 million people annually especially in developing countries.¹ Dyslipidaemia and hypertension are two major modifiable risk factors for cardiovascular disease.² The

co-existence of the two risk factors has more than an additive adverse impact on the vascular endothelium, which results in enhanced atherosclerosis.³ Nigeria has estimated proportional mortality attributable to cardiovascular diseases of 11%.⁴

Hypertension damages the endothelium through altered shear and oxidative stress, resulting in an increased endothelial synthesis of collagen and fibronectin, reduced

nitric oxide-dependent vascular relaxation and increased permeability to lipoproteins, promoting atherosclerosis.⁵ Hypertension is also associated with an up-regulation of lipid-oxidizing enzymes. Low-Density Lipoprotein (LDL), especially oxidized LDL, is a major cause of endothelial dysfunction.⁵ Left ventricular hypertrophy (LVH) is an adaptive response and hallmark of increased volume and/or pressure overload in exercise training (physiological), hypertension, cardiomyopathy or valvular heart disease (pathological) and serves to normalize ventricular wall stress.⁶ LVH is a complex and multifactorial condition whose pathogenesis may include many different genetic and signalling pathways, which involve the initiation of a foetal-like gene program.⁷ Although it involves a process of adaptive remodelling, which is usually a compensatory mechanism in response to increased hemodynamic load, it is ultimately characterized by structural changes, mainly in the form of myocardial fibrosis that leads to diastolic dysfunction and diminished contractility.⁸ Epidemiologic studies have shown that LVH is a strong independent predictor of cardiovascular events and all-cause mortality.⁹

Dyslipidaemia includes abnormalities in serum LDL-Cholesterol, High-Density Lipoprotein Cholesterol (HDL-Cholesterol), and triglycerides in various combinations. The AIP is a critical index that can be used as a stand-alone index for cardiac risk estimation.¹⁰ It is defined as the base 10 logarithms of the ratio of plasma triglyceride (TG) to high-density lipoprotein cholesterol (HDL-C), which has been shown to be a significant predictor of atherosclerosis and cardiovascular risk.¹⁰ It can act as an adjunct over individual lipid profile and be calculated easily from the standard lipid profile. AIP values increase with increased cardiovascular risk. It can be classified according to the values obtained: -0.3 to 0.1 for low risk, 0.1 to 0.24 for medium, and more than 0.24 for high risk of cardiovascular diseases.¹⁰ Several Nigerian studies have reported a huge burden of dyslipidaemia among apparently healthy adults, elderly, hypertensive subjects, diabetic subjects, with a low HDL-C and elevated LDL-C being the common pattern observed.¹¹⁻¹⁶

Reports from observational studies of a causal relationship between dyslipidaemia and LV remodelling have been inconsistent.¹⁷⁻¹⁹ Some studies documented that low HDL-C may unfavourably modify LV structure in the setting of elevated LVM or the presence of LVH, whereas others have not.¹⁷⁻²² Some other reports have implicated excess triglycerides and elevated total cholesterol in increased LVM.^{21,23} Recent emphasis has been placed on the clinical implications of non-traditional lipid profiles as a powerful and independent predictor of cardiovascular disease (CVD) outcomes.²⁴⁻²⁶ Suggestions to replace the traditional lipid risk factors with non-traditional lipid profile (lipid ratios, or single index) have been driven by the notion that the non-traditional lipid profile parameters are a better representation of the underlying vascular atherogenesis.³¹ Previous studies

showed that the TC/HDL-C ratio could represent a simple atherogenic particle burden tool informing on lipoprotein particle concentration and size not available in cholesterol-based measurements.^{27,28} Furthermore, TG/HDL-C has been documented to assist in identifying insulin resistance and the concentrations of small dense LDL particles as a novel, inexpensive, and readily available biomarker for quantifying atherogenic and cardio metabolic risk.^{24,29,30} A recent study by Wang et al reported an independent positive association on non-traditional lipid profile (TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, and non-HDL-C) with concentric LVH, in a general population of rural China.³¹

This study intends to determine the prevalence of dyslipidaemia in hypertensive subjects with LVH, compare the prevalence of dyslipidaemia in hypertensive individuals with and without LVH, and determine the relationship between left ventricular mass and the AIP among hypertensive subjects in a tertiary hospital in Sagamu, South- West Nigeria.

METHODS

The study was carried out between January and June 2018 at the Olabisi Onabanjo University Teaching Hospital (OOUTH), Sagamu, Ogun State, South-West Nigeria.

Study design

This was a cross-sectional comparative hospital-based study. One hundred and twenty hypertensive patients with LVH were consecutively recruited in the Cardiology clinic irrespective of the duration of hypertension, blood pressure control and whether on medications (anti-hypertensives) or not. Sixty age- and sex-matched hypertensive patients without LVH were also recruited from the same center.

Subjects were adults aged 18 years and above who were hypertensive with LVH on echocardiogram and gave informed consent. Controls were adults aged 18 years and above, who were hypertensive without LVH on echocardiogram who gave informed consent. They were matched by age and sex.

Individuals who were obese, pregnant, had kidney disease, heart failure, liver disease, malignancy, on medications that could significantly alter lipid profile like steroids (including hormonal contraceptives), diuretics (>25 mg hydrochlorothiazide), non-cardio selective beta-blockers, statins, and non-statin hypolipemic drugs- fibrates, niacin were excluded from the study.

Ethical consideration

Approval for the study was obtained from the Health Research and Ethics Committee of OOUTH. Written

informed consent was obtained from each participant. All relevant standards of the revised declaration of Helsinki were followed.

Sampling design

The sample size was calculated using the Cochran formula $N = Z^2pq/d^2$. With a prevalence of 7.6%, this resulted in a sample size of 107.9, which was approximated to 120 for the study group to accommodate for 10% attrition.¹⁵ Sixty age- and sex-matched hypertensive participants without LVH (by echocardiogram) were recruited for the study.

Data collection

Detailed history including socio-demographic data, anthropometric indices, personal and family history of hypertension, diabetes and other diseases, medication history was obtained. The Blood Pressure (BP) of all participants was measured after five minutes' rest each to eliminate anxiety. There was no consumption of stimulants (coffee, cigarette smoking) before BP measurement. The blood pressure was taken with the participant seated comfortably upright with the feet on the floor and arm at the level of the heart, free of any constrictive clothing or lying supine using the standard mercury sphygmomanometer (Accoson[®]) and appropriate-sized cuff. The BP of participants was taken in both arms, with five minutes between readings and the average was used.

The body weight of all subjects was determined in kilograms while the height of the participants was measured in meters using a stadiometer.

The Body mass index (BMI) of all participants was calculated as $\text{Weight}(\text{kg})/\text{Height}(\text{m}^2)$.

Plasma lipid profile estimation

Laboratory assessment included the collection of 3ml of fasting venous blood taken from the ante-cubital vein via venipuncture into plain specimen bottles after routine aseptic preparation to determine lipid profile parameters. The Third Report of the Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults, which is one of the most current and most frequently referenced diagnostic criteria for dyslipidemia, was used.³² It defines dyslipidemia as follows.

Total Cholesterol >200 mg/dl (>5.17 mmol/l)
 LDL- Cholesterol >130 mg/dl (>3.36 mmol/l)
 HDL- Cholesterol <40 mg/dl (<1.03 mmol/l) (for males)
 <50 mg/dl (<1.29 mmol/l) (for females)
 Triglyceride >150 mg/dl (>1.7 mmol/l)
 The AIP was defined as:¹⁰

$\text{Log}_{10}(\text{Triglyceride}/\text{HDL-Cholesterol})$ (both parameters in mmol/L).

Cardiovascular risk assessment was defined as 0.3 to 0.1-low risk, 0.1 to 0.24 intermediate risk, >0.24 high risk.¹⁰

Echocardiogram

Echocardiographic studies were done using the commercially available echo-machine (ALOKA SSD 900) with a 3.5 MHz linear array transducer probe.

Left ventricular hypertrophy was defined as LVM greater than 51 g/m in both men and women.^{27,33} Relative wall thickness was calculated as twice the posterior wall thickness/LV Internal dimension in diastole. A relative wall thickness of 0.44 or greater was considered abnormal.³⁴

Statistical analysis

The data were analyzed using the Statistical Product and Service Solutions (SPSS) version 20 software (SPSS, Inc, Chicago Illinois, USA). Continuous variables were expressed as mean \pm SD (standard deviation) and categorical variables as percentages. Differences in categorical variables among the two groups were assessed by Chi-square analysis while unpaired t-test was used for comparison of continuous variables. A comparison of normally distributed continuous variables among three groups was performed by analysis of variance (ANOVA). Correlation between two normally distributed variables was done with Pearson's correlation coefficient while Spearman's rank correlation was used for non-normally distributed variables. Spearman's correlation analysis was used to assess variables with a significant correlation with left ventricular mass. A p-value above 0.05 was generally considered to be statistically insignificant.

RESULTS

Demographic and clinical parameters in the study

One hundred and twenty hypertensive participants with LVH (subjects) and 60 age and sex-matched hypertensive participants without LVH (controls) were studied. The mean age of the subjects was 53.72 ± 8.67 years which did not differ significantly that of controls at 52.63 ± 8.04 years ($p=0.419$) as shown in Table 1.

There was no statistically significant difference in the body mass index, systolic blood pressure, diastolic blood pressure between the subjects and controls as depicted in Table 1.

The mean AIP in the hypertensive participants with LVH (0.34 ± 0.23) was higher than in their hypertensive counterparts without LVH, and the difference was statistically significant, as shown in Table 1.

Table 1: Socio-demographic and clinical characteristics of all study participants.

Variables (Mean±SD)	Subject (%) (n=120)	Control (%) (n=60)	Test	Statistical test value	P value
Age (in years)	53.72±8.67	52.63±8.04	t	-0.809	0.419
Gender					
Male	76 (63.3)	37 (61.7)	X	0.003	0.957
Female	44 (36.7)	23 (38.3)			
BMI	26.93±2.79	26.47±2.89	t	-1.047	0.296
SBP	148.29±17.70	145.55±18.95	t	-0.957	0.340
DBP	94.69±14.79	94.35±13.19	t	-0.151	0.880
LDL-C	131.42±45.77	133.42±53.34	t	0.262	0.794
HDL-C	45.57±10.92	60.65±37.94	t	4.042	0.001*
Triglyceride	106.49±44.43	98.70±38.51	t	-1.157	0.249
Total cholesterol	195.31±41.30	207.45±55.26	t	1.655	0.100
AIP	0.34±0.23	0.22±0.28	t	-3.235	0.001*

BMI-body mass index, SBP- systolic blood pressure, DBP-diastric blood pressure, LDL-C-low density lipoprotein cholesterol, HDL-C-high density lipoprotein cholesterol, AIP- atherogenic index of plasma.

Table 2: Prevalence of dyslipidaemia among the study subjects and controls.

Lipid	Subjects LVH ⁺ (%)	Controls LVH ⁻ (%)	Chi square value	P value
LDL-C				
Normal	54 (45.0)	32 (53.3)	0.804	0.370
Elevated	66 (55.0)	28 (46.7)		
HDL-C				
Normal	69 (57.5)	45 (75.0)	4.548	0.033*
Decreased	51 (42.5)	15 (25.0)		
Triglyceride				
Normal	106 (88.3)	55 (91.7)	0.184	0.668
Increased	14 (11.7)	5 (8.3)		
Total cholesterol				
Normal	63 (52.5)	34 (56.7)	0.137	0.711
Increased	57 (47.5)	26 (43.3)		
Total dyslipidemia				
Normal	47 (39.2)	23 (38.3)	0.012	0.914
Dyslipidemia	73 (60.8)	37 (61.7)		
AIP				
Low risk of CV	21 (17.5)	17 (28.3)	4.970	0.083
Intermediate risk of CVD	13 (10.8)	10 (16.7)		
High risk of CVD	86 (71.7)	33 (55.0)		

HDL-C- high density lipoprotein cholesterol, LDL-C-Low Density Lipoprotein Cholesterol, LVH⁺ - with left ventricular hypertrophy, LVH⁻-without left ventricular hypertrophy, AIP-atherogenic index of plasma, CVD- cardiovascular disease.

Prevalence of dyslipidaemia in the study

The overall prevalence of dyslipidaemia in the study participants was 61.1% (110/180). The prevalence of dyslipidaemia in the subjects was 60.8% (73/120), which was slightly lower than that observed in the controls 61.7% (37/60). The difference, was however, not statistically significant (p=0.914) as shown in Table 2.

The most common isolated dyslipidaemia was elevated serum LDL-C levels, found in both subjects (55%) and controls (46.7%).

The prevalence of serum LDL-C was higher in the subjects 55% (66/120) than in controls 46.7% (28/60), though the difference was not statistically significant (p=0.370) as shown in Table 2.

Table 3: Prevalence of combined dyslipidaemia in the study participants.

Lipid	LDL-C (%)		Chi-square value	P value
	Normal	Dyslipidemia		
HDL-C				
Normal	71 (82.6)	43 (45.7)	24.648	0.001
Dyslipidemia	15 (17.4)	51 (54.3)		
Triglyceride				
Normal	84 (97.7)	77 (81.9)	10.204	0.001
Dyslipidemia	2 (2.3)	17 (18.1)		
Total Cholesterol				
Normal	84 (97.7)	13 (13.8)	123.705	0.001
Dyslipidemia	2 (2.3)	81 (86.2)		
HDL-C (%)				
Triglyceride				
Normal	107 (93.9)	54 (81.8)	5.207	0.022
Dyslipidemia	7 (6.1)	12 (18.2)		
Total Cholesterol				
Normal	72 (63.2)	25 (37.9)	9.756	0.002
Dyslipidemia	42 (36.8)	41 (62.1)		
Triglyceride (%)				
Total Cholesterol				
Normal	96 (59.6)	1 (5.3)	18.084	0.001
Dyslipidemia	65 (40.4)	18 (94.7)		

HDL-C- high density lipoprotein cholesterol, LDL-C- low density lipoprotein cholesterol.

Table 4: Correlation between echocardiographic left ventricular mass and lipid profile parameters among hypertensive subjects with LVH.

Variables	Mean±SD	r	P value
LVM (Absolute)	202.90 ± 28.97		
LDL-C	131.42 ± 45.77	-0.166	0.071
HDL-C	45.57 ± 10.93	0.063	0.492
TG	106.49 ± 44.43	0.250	0.006*
TC	195.31 ± 41.30	-0.144	0.117
AIP	0.34 ± 0.24	0.274	0.002*

LVM- left ventricular mass, LDL-C-low density lipoprotein cholesterol, HDL-C-high density lipoprotein cholesterol, TG- triglycerides, TC-total cholesterol, AIP- atherogenic index of plasma, r- correlation co-efficient.

Table 5: Linear Regression analysis of lipid profile parameters as predictor of LVH in hypertensive subjects.

Lipid profile	B	OR	CI at 95%	P value
LDL-C	-0.104	-0.165	-0.458 -0.249	0.560
HDL-C	2.283	0.861	1.358-3.208	0.001*
TG	-0.525	-0.805	-0.836--0.215	0.001*
TC	-0.174	-0.248	-0.553-0.205	0.356
AIP	212.998	1.740	143.245- 282.751	0.001*

LDL-C-low density lipoprotein cholesterol, HDL-C-high density lipoprotein cholesterol, TG- triglyceride, TC- total cholesterol, AIP- atherogenic index of plasma, OR-Odds ratio, CI- confidence interval.

DISCUSSION

To the best of knowledge, this study represents the first report on the relationship between echocardiographic left ventricular mass and AIP in hypertensive subjects in South-West Nigeria.

The mean age of study participants was similar to that observed in similar studies.^{14,35}

The prevalence of dyslipidaemia in the study participants was 61.1%, which is comparable with similar studies carried out in Bida, North-Central Nigeria (64%), Oshogbo, South-West Nigeria (58.9%), Owerri, South-East Nigeria (60.5%).^{14,35,36}

The prevalence of dyslipidaemia in hypertensive participants without LVH (61.7%) was slightly higher than in hypertensive participants with LVH (60.8%), though the difference was not statistically significant.

The most common isolated lipid abnormality in the present study was elevated serum LDL-C (55% in subjects, 46.7% in controls) while the least common was elevated serum triglycerides (11.7% in the subjects, 8.3% in controls). This was at variance with some previous Nigerian studies in which low serum HDL-C was reported as the most common lipid abnormality in hypertensive subjects.^{14,15,35}

Combined dyslipidaemia was also common in the study, with elevated serum triglyceride and total cholesterol being the most frequent abnormality, while elevated LDL-C and triglycerides were the least common. This is in keeping with results from a similar study conducted in Abuja, North-Central Nigeria.¹⁵ However, this finding was at variance with another study carried out in North-Central Nigeria in which elevated serum TG and low HDL were the most frequent combined dyslipidaemia among hypertensive subjects.³⁵ This disparity in the prevalence of combined dyslipidaemia in this study may be due to regional dietary differences, as well as concomitant use of medications. Most of the participants in the present study were already on anti-lipidemic drugs which could have altered the lipid profile. Participants in the prior studies were newly diagnosed, dry-naïve hypertensive individuals.

Data from this study revealed that the level of serum HDL-C in hypertensive participants with LVH was significantly lower when compared with their counterparts with normal LV geometry. Also, a linear regression analysis further revealed that serum HDL-C level is an important predictor of left ventricular hypertrophy in hypertensive subjects. These findings further corroborate reports from other studies that low HDL-C may unfavorably modify the left ventricular structure in the setting of elevated left ventricular mass or presence of LVH.^{17,18,20} It has been reported that HDL-C exerts a protective effect by its anti-inflammatory properties, improving endothelial function and weakening LDL oxidation, and a reverse cholesterol transport from the tissues (like myocardium, blood vessels) to the liver for excretion, thereby preventing adverse cardiovascular remodelling, including left ventricular hypertrophy.^{17,37,38}

In the present study, serum triglyceride levels showed a positive correlation with LVM in hypertensive subjects with LVH, although total or LDL-cholesterol was not associated with these echocardiographic indices at all. This result was in agreement with some studies which implicated excess triglycerides in high LV mass-to-volume ratio and LV-end-diastolic volume.^{17,21,41} However, some other studies documented a divergent view, concluding that lipid abnormalities, represented as cholesterol remnants and triglycerides, were insufficient

to cause an overt increase in LVM and LV wall thickness.^{19,23}

Furthermore, this present study revealed a positive correlation between the AIP (TG/HDL-C) and left ventricular hypertrophy in hypertensive subjects. Hypertensive subjects with LVH had a higher AIP (and hence a higher risk of cardiovascular disease) when compared with hypertensive counterparts without LVH. Also, a linear regression analysis revealed that serum levels of triglycerides, HDL-C and AIP serve as predictors of LVH. These findings were in agreement with other studies which reported an association between high TG/HDL-C ratio and concentric LVH.^{31,39} This has further strengthened the submissions of studies which reported the usefulness of non-traditional lipid profile parameters like TC/HDL-C, TG/HDL-C, LDL-C/HDL-C, and non-HDL-C as a veritable screening tool for various populations, identifying those with a higher risk of subsequent cardiovascular complications and heart failure.^{24,26,27} TG/HDL-C is a strong correlate of insulin resistance or hyperinsulinemia, which is an important contributor to left ventricular hypertrophy and diastolic dysfunction in hypertensive subjects.¹⁷⁻¹⁹ In a very large population of Italian outpatient overweight children, Di Bonito P et al concluded that the TG/HDL-C ratio discriminated better than non-HDL-C with prevalent concentric LVH.^{39,40} Furthermore, in another prospective longitudinal cohort study, the LDL+HDL-C ratio contributed to the origin of LVH over 20 years, suggesting the changes occur over time.⁴¹

A limitation of the study was the cross-sectional design and thus cannot be reliably used to predict some of the causal relationships. Prospective studies with large sample sizes are therefore suggested. The duration and severity of systemic hypertension were also not put into consideration. Besides, the study participants were already on antihypertensive medications, some of which are known to alter lipid profile parameters. For instance, thiazide diuretics are known to increase serum triglyceride, Very Low-Density Lipoprotein (VLDL), total cholesterol, LDL-Cholesterol with no significant effect on HDL-Cholesterol.⁴² The study was hospital-based and so it is difficult to generalize findings to reflect the whole country. Finally, we did not measure or calculate other molar ratios of lipids, which may also have confounding effects.

CONCLUSION

In conclusion, the prevalence of dyslipidaemia in this study population was high in both hypertensive participants with and without LVH. The most common isolated dyslipidaemia in the study participants was elevated LDL-C, with hypertensive subjects with LVH having significantly lower HDL-C levels. There is a positive correlation between echocardiographic LV mass and the AIP among hypertensive subjects in OOUTH, Sagamu.

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