Original Research Article

Correlation between echocardiographic left ventricular mass and atherogenic index of plasma in hypertensive adult patients

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Abstract

Aim: to determine the prevalence of dyslipidaemia in hypertensive subjects and to determine its relationship with left ventricular hypertrophy. **Methods:** The cross-sectional comparative study was conducted at Arc Hospital Bhagalpur, India and Jawaharlal Nehru Medical College Hospital, Bhagalpur from December 2018 to July 2019, involving 100 hypertensive participants with LVH (subjects) and 50 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 mean age of the subjects was 54.21±7.84 years which did not differ significantly that of controls at 53.33±7.52 years (p=0.387).The mean AIP in the hypertensive participants with LVH (0.33±0.21) was higher than in their hypertensive counterparts without LVH, and the difference was statistically significant. **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

Left ventricular hypertrophy (LVH), a potent marker of end-organ damage, is an independent predictor and potentially modifiable risk factor for coronary heart disease (CHD), heart failure (HF), and other major cardiovascular (CV) morbidity and mortality[1,2]. In light of left ventricular mass index (LVMI) along with relative wall thickness (RWT), four distinct patterns of left ventricular (LV) geometry are further proposed to explain the pathophysiologic basis for cardiac remodeling, with concentric LVH conferring a stronger prognostic value of morbid CV events[3,4]. Although greater left ventricular mass (LVM) might initially be considered beneficial by a concomitant LV wall stress reduction and avoidance of hemodynamic compromise, in long-term, the progression of LV geometric abnormalities, particularly LVH, can prove to be maladaptive and portend a poor prognosis[5-7] Identification and intervention of the CV risk factors underlying a specific phenotype of LV geometry before development of manifest disease are of paramount importance, given that knowledge of these geometric adaptations has been a fundamental precursor of CV disorders. Reports from observational studies on the possibility of dyslipidemia-induced LV remodeling have been inconsistent[8-16]. Several lines of evidence have demonstrated that low high-density lipoprotein cholesterol (HDL-C) may unfavorably modify LV structure in the setting of elevated LVM or presence of LVH[8-10] whereas others have not[11-13].Historically, there were conflicting data as to whether greater total cholesterol (TC) has been implicated in increased LVM[15,16]. It was also plausible that alterations in LV morphology would be related at least in part to the excess of triglyceride (TG) which favors a high LVM-to-volume ratio and LV end-diastolic volume.[12] Conversely, lipid derangements, represented as cholesterol remnants and TG, were insufficient to cause an overt increase in LVM and LV wall thickness[16]. Recent emphasis has been placed in the clinical implications of nontraditional lipid profiles as powerful and independent predictors of cardiovascular disease (CVD) outcomes.[17-21] For instance, previous studies revealed that 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[22,23]. Notably, TC/HDL-C ratio offered significant incremental prognostic information over low-density lipoprotein cholesterol (LDL-C) and non-HDL-C for predicting CVD events[18-24].Moreover, TG/HDLC ratio has received growing interest in identifying insulin resistance and concentrations of sd-LDL particles as a novel, inexpensive, and readily available biomarker for quantifying atherogenic and cardio metabolic risk.[17,19,25,26] Further, assessment of lipid metabolism and atherosclerotic status using a simple tool, such as LDL-C/ HDL-C ratio, not only could help closely reflecting the interactions between lipid fractions but also could better predict plasma atherogenicity than isolated lipid

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values[27,28]. It has recently been proposed that non-HDL-C that includes all of the atherogenic lipoproteins, such as TG-rich lipoproteins, intermediate-density lipoprotein cholesterol, LDL-C, and lipoprotein(a) could better predict cardiovascular outcomes in patients on LDLC-lowering therapy[20,24]. To date, data are sparse regarding the relative performance of nontraditional lipid profiles for the purpose of reclassification of LV geometry risk. Di Bonito P et al. advocated that a high TG/HDL-C ratio, independent of blood pressure and visceral adiposity, was a key determinant of worrisome concentric LVH[29] Interestingly, an epidemiological investigation from the Framingham Heart Study did not support an independent correlation of non-HDL-C and LV remodelling In this regard, it is likely that the adverse impacts of non-traditional lipid profiles on CVD incidence may partly be mediated by it adverse effects on LV structural remodelling. However, no prior work has examined this premise comprehensively. We hypothesized that evaluating the influence of non-traditional lipid profiles on the probability of subclinical LV geometry change is helpful to understand the pathogenesis of CVD related to dyslipidemia. 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.

Material and Methods

The cross-sectional analytical study was conducted at Arc Hospital Bhagalpur, India and Jawahar Lal Nehru Medical College Hospital, Bhagalpur from December 2018 to July 2019.

Methodology

One hundred 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-hypertensive) or not. 50 age and sex-matched hypertensive patients without LVH were included in this study. Individuals who were obese, pregnant, had kidney disease, heart failure, liver disease, malignancy, on medications that could that 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.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 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 Weight (kg)/Height (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[30].

It defines dyslipidemia as follows:

- Total Cholesterol >200 mg/dl (>5.17mmol/l)
- LDL- Cholesterol >130 mg/dl (>3.36mmol/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:

Log_{10} (Triglyceride /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.[31]

Echocardiogram

Echocardiographic studies were done using the commercially available echo-machine (GE Vivid) 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[32] 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[33]

Statistical analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2010) and then exported to data editor page of SPSS version 19 (SPSS Inc., Chicago, Illinois, USA). Descriptive statistics included computation of percentages and means. Test applied for the analysis were chi-square test and t-test. The confidence interval and p-value were set at 95% and 5% for both the test.

Results

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

Variables (Mean±SD)	Subject (%) (n=100)	Control (%) (n=50)	Test	Statistical test value	P value
Age (in years)	54.21±7.84	53.33±7.52	t	-0.754	0.387
Gender					
Male	66(66)	31 (62)	X	0.002	0.897

Female	34(34)	19 (38)			
BMI	27.21±2.79	26.98±2.75	t	-1.047	0.276
SBP	149.12±18.45	146.17±18.87	t	-0.897	0.331
DBP	94.87±13.87	94.48±13.07	t	-0.157	0.871
LDL-C	132.04±46.32	133.74±52.47	t	0.254	0.709
HDL-C	46.04±11.12	59.87±36.24	t	3.658	0.001*
Triglyceride	106.87±45.32	99.80±37.50	t	-1.159	0.270
Total cholesterol	194.73±40.10	208.85±51.07	t	1.604	0.110
AIP	0.33±0.21	0.21±0.31	t	-3.287	0.001*

BMI-body mass index, SBP- systolic blood pressure, DBP-diastolic 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	47(47.0)	28 (56)	0.789	0.357
Elevated	53(53.0)	22 (44)		
HDL-C				
Normal	60 (60)	40(80)	4.488	0.030*
Decreased	40(40)	10(20)		
Triglyceride				
Normal	90(90)	46 (92)	0.178	0.662
Increased	10(10)	4 (8)		
Total cholesterol				
Normal	51 (51)	28 (56)	0.130	0.733
Increased	49 (49)	22 (44)		
Total dyslipidemia				
Normal	38 (38)	18 (36)	0.010	0.888
Dyslipidemia	62(62)	32 (64)		
AIP				
Low risk of CV	24 (24)	14(28)		
Intermediate risk of CVD	6(6)	9 (18)	4.989	0.090
High risk of CVD	70 (70)	27 (54)		

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

Table 3: Prevalence of combined dyslipidaemia in the study participants

Lipid	**LDL-C (%)** **Normal**	**Dyslipidemia**	**Chi-square value**	**P value**
HDL-C				
Normal	59 (78.67)	36 (48)	23.957	0.001
Dyslipidemia	16 (21.33)	39(52)		
Triglyceride				
Normal	73 (97.33)	63 (84)		
Dyslipidemia	2(2.67)	12(16)	10.187	0.001
Total Cholesterol				
Normal	73 (97.33)	10(13.33)	124.304	0.001
Dyslipidemia	2 (2.67)	65 (86.67)		
HDL-C (%)				
Triglyceride				

Normal	92 (92)	39(78)	5.211	0.020
Dyslipidemia	8 (8)	11(22)		
Total Cholesterol				
Normal	65 (65)	20 (40)		
Dyslipidemia	35 (35)	30 (60)	9.678	0.002
Triglyceride (%)				
Total Cholesterol				
Normal	83 (61.02)	1 (7.14)		
Dyslipidemia	53 (38.98)	13 (92.85)	18.104	0.001

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)	204.10 ± 29.63		
LDL-C	132.04±46.32	-0.187	0.068
HDL-C	46.04±11.12	0.057	0.472
TG	106.87±45.32	0.265	0.005*
TC	194.73±40.10	-0.134	0.121
AIP	0.33±0.21	0.281	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	-0.110	-0.187	-0.474-0.252	0.572
HDL-C	2.306	0.854	1.367-3.214	0.001*
TG	-0.647	-0.785	-0.820--0.209	0.001*
TC	-0.187	-0.267	-0.575-0.242	0.361
AIP	214.987	1.689	139.175- 282.874	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 Bihar region, India. The mean age of study participants was similar to that observed in similar studies[14,34].The prevalence of dyslipidaemia in the study participants was 62.67% (94/150). 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%)[34-36]

The prevalence of dyslipidaemia in hypertensive participants without LVH (62%) was slightly higher than in hypertensive participants with LVH (60%), though the difference was not statistically significant. The most common isolated lipid abnormality in the present study was elevated serum LDL-C (53% (53/100) in subjects, 44(22/50)% in controls) while the least common was elevated serum triglycerides (10% in the subjects, 8% in controls). This was at variance with some previous studies in which low serum HDL-C was reported as the most common lipid abnormality in hypertensive subjects[34-37]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.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 subjectsThis 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, drug-naive 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 unfavourably

modify the left ventricular structure in the setting of elevated left ventricular mass or presence of LVH 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[38,39]

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 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[11-16]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[40,41]. 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[22].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[8-11]. 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[21]. 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 overtime[41,42]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 Bihar region, India.

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