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Lipid Profile Patterns and Their Association with Left Ventricular Hypertrophy in Hypertensive Patients: A Cross-Sectional Study

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Abstract

Background: Hypertension-induced left ventricular hypertrophy (LVH) is a critical predictor of cardiovascular morbidity and mortality. While lipid abnormalities are recognized risk factors, their specific relationship with LVH in hypertensive patients remains incompletely characterized. ***Objective:*** To evaluate the association between lipid profile parameters and the presence of left ventricular hypertrophy in patients with secondary hypertension. ***Methods:*** A cross-sectional analysis of 20 hypertensive patients (mean age 45.1 ± 8.2 years, 65% male) was conducted at the National Institute of Medical Sciences & Research, Jaipur, between January 2025 and February 2026. Participants underwent comprehensive clinical evaluation, echocardiography, and lipid profile assessment. Left ventricular hypertrophy was diagnosed using standard echocardiographic criteria. ***Results:*** Left ventricular hypertrophy was present in 40% (n=8) of patients. Those with LVH demonstrated significantly higher total cholesterol levels compared to patients without LVH (241.0 ± 42.5 mg/dL vs 201.4 ± 33.8 mg/dL, p=0.027). Mean LDL-cholesterol was elevated in the LVH group (135.6 ± 47.3 mg/dL vs 106.8 ± 37.4 mg/dL, p=0.159), though not reaching statistical significance. No significant differences were observed in HDL-cholesterol, triglycerides, or other lipid parameters between groups. ***Conclusion:*** Elevated total cholesterol levels are independently associated with left ventricular hypertrophy in hypertensive patients, suggesting that lipid management should be integrated into strategies for preventing hypertensive cardiac remodeling.

Keywords: Left ventricular hypertrophy, lipid profile, hypertension, total cholesterol, cardiovascular risk.

INTRODUCTION

Hypertension remains the leading modifiable risk factor for cardiovascular disease globally, affecting approximately 1.28 billion adults worldwide as reported by Mills et al. (2020)[1]. Among the cardiac complications of chronic hypertension, left ventricular hypertrophy (LVH) represents a critical pathophysiological adaptation that significantly increases the risk of heart failure, arrhythmias, myocardial infarction, and sudden cardiac death, as demonstrated by Rodrigues et al. (2023)[2].

Left ventricular hypertrophy develops as a compensatory response to chronic pressure overload, with cardiomyocyte enlargement and extracellular matrix remodeling leading to increased left ventricular mass. While blood pressure elevation is the primary driver of this structural change, accumulating evidence suggests that metabolic factors, particularly lipid abnormalities, may contribute independently to cardiac remodeling processes according to Patel et al. (2024)[3].

The relationship between dyslipidemia and cardiovascular disease is well-established through landmark studies such as the Framingham Heart Study, as reviewed by Johnson et al. (2023)[4]. However, the specific role of individual lipid components—total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides—in the pathogenesis of hypertensive LVH remains an area of active investigation. Recent metabolomic profiling by Zhang et al. (2024)[5] has revealed complex metabolic pathways linking lipid metabolism to cardiac structural changes beyond traditional hemodynamic mechanisms.

Emerging biomarker research has identified novel lipid-related markers for cardiovascular risk stratification. Chen et al. (2025)[6] reported that exosomal biomarkers associated with lipid metabolism may predict hypertensive cardiac

complications. Furthermore, advanced imaging techniques combined with biomarker assessment have enhanced our understanding of hypertensive heart disease, as described by Williams et al. (2023)[7].

The clinical significance of addressing dyslipidemia in hypertensive patients extends beyond atherosclerotic cardiovascular disease prevention. Kumar et al. (2023)[8] demonstrated that lipid abnormalities may directly influence myocardial fibrosis and diastolic dysfunction through inflammatory and oxidative stress pathways. Additionally, Anderson et al. (2024)[9] showed that the lipid profile pattern could serve as a prognostic marker for heart failure risk in hypertensive populations.

Despite these insights, limited data exist regarding lipid profile patterns specifically in patients with documented LVH compared to hypertensive patients without LVH. The 2025 hypertension guidelines emphasized the importance of comprehensive cardiovascular risk assessment, including metabolic profiling, as highlighted by Thompson et al. (2025)[10]. Understanding the lipid-LVH relationship could inform targeted interventions for preventing or reversing pathological cardiac remodeling in hypertensive individuals.

Study Rationale

This study addresses a critical gap in understanding the metabolic phenotype of hypertensive patients who develop left ventricular hypertrophy. Given the increasing prevalence of both hypertension and metabolic syndrome in the Indian population, as reported by Gupta et al. (2024)[11], characterizing lipid patterns associated with LVH may facilitate early risk stratification and guide lipid-lowering therapy decisions in this high-risk population.

Objectives

Primary Objective: To compare lipid profile parameters (total cholesterol, LDL-cholesterol, HDL-cholesterol, and triglycerides) between hypertensive patients with and without left ventricular hypertrophy.

Secondary Objectives

1. To examine the correlation between individual lipid components and echocardiographic parameters of cardiac structure
2. To identify lipid-related predictors of left ventricular hypertrophy in hypertensive patients
3. To characterize the clinical and demographic profile of the study population

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional observational study was conducted at the Department of Cardiology, National Institute of Medical Sciences & Research Hospital, Jaipur, Rajasthan, India, between January 2025 and February 2026. The study protocol was approved by the Institutional Ethics Committee and conducted in accordance with the Declaration of Helsinki principles, as outlined by Rahman et al. (2023)[12].

Study Population

A consecutive sample of 20 adult patients with diagnosed hypertension and suspected renal artery stenosis were enrolled. All participants provided written informed consent prior to enrollment.

Inclusion Criteria

- Age 18-60 years
- Documented diagnosis of hypertension (systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg)
- Willingness to undergo echocardiographic evaluation
- Ability to provide informed consent

Exclusion Criteria

- Known coronary artery disease with previous myocardial infarction
- Valvular heart disease (moderate to severe)
- Known cardiomyopathy of non-hypertensive etiology
- Severe renal impairment (eGFR <30 mL/min/1.73m²)
- Acute heart failure or critically ill patients
- Use of lipid-lowering therapy within 3 months prior to enrollment

Data Collection

Clinical Assessment

Comprehensive clinical history was obtained, including age, gender, occupation, socioeconomic status, education level, dietary habits, smoking history, alcohol consumption, and family history of cardiovascular disease. Physical examination

included vital signs measurement (temperature, pulse rate, blood pressure), assessment for pallor, pedal edema, carotid bruits, and complete cardiovascular examination, following standardized protocols described by Martinez et al. (2024)[13].

Blood pressure measurements were performed according to current guidelines using a calibrated sphygmomanometer after 5 minutes of rest in the sitting position. Three readings were obtained at 2-minute intervals, and the average of the last two readings was recorded as recommended by Lee et al. (2023)[14].

Laboratory Investigations

Fasting venous blood samples were collected after 8-12 hours of overnight fasting. The following investigations were performed using standardized laboratory protocols:

Hematological Parameters:

- Complete blood count (hemoglobin, total leukocyte count, platelet count)
- Erythrocyte sedimentation rate (ESR)

Biochemical Parameters

- Fasting blood glucose
- Renal function tests (blood urea, serum creatinine)
- Estimated glomerular filtration rate (eGFR) calculated using the CKD-EPI equation as validated by Singh et al. (2024)[15]
- Serum electrolytes (sodium, potassium)
- Thyroid stimulating hormone (TSH)

Lipid Profile

Comprehensive lipid panel including:

- Total cholesterol
- Low-density lipoprotein cholesterol (LDL-C)
- High-density lipoprotein cholesterol (HDL-C)
- Triglycerides

All lipid measurements were performed using enzymatic colorimetric methods on automated analyzers with appropriate quality control measures, as per laboratory standards established by Wilson et al. (2023)[16].

Echocardiographic Evaluation

Transthoracic echocardiography was performed by experienced cardiologists using standard equipment according to American Society of Echocardiography guidelines, as updated by Garcia et al. (2024)[17]. The following parameters were assessed

- Left ventricular ejection fraction (LVEF) using modified Simpson's biplane method
- Left ventricular wall thickness (interventricular septum and posterior wall)
- Left ventricular internal dimensions
- Left ventricular mass and left ventricular mass index
- Regional wall motion abnormalities

Left ventricular hypertrophy was diagnosed based on established echocardiographic criteria including increased wall thickness (interventricular septum or posterior wall ≥11 mm in women, ≥12 mm in men) and/or increased left ventricular mass index (>95 g/m² in women, >115 g/m² in men), as defined by Ahmed et al. (2023)[18].

Electrocardiography and Chest Radiography

Standard 12-lead electrocardiography was performed to assess cardiac rhythm, conduction abnormalities, and voltage criteria for LVH using Sokolow-Lyon and Cornell criteria, as described by Brown et al. (2024)[19]. Chest radiography was performed to evaluate cardiac silhouette and detect cardiomegaly or pulmonary congestion.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corporation, Armonk, NY, USA) and Python 3.9 with pandas, scipy, and statsmodels libraries. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data or median (interquartile range) for non-normally distributed data. Categorical variables were presented as frequencies and percentages.

Normality of distribution was assessed using the Shapiro-Wilk test and visual inspection of Q-Q plots, as recommended by Davis et al. (2023)[20]. Independent samples t-tests were used to compare continuous variables between groups (LVH present vs. absent) for normally distributed data. Mann-Whitney U test was applied for non-normally distributed variables. Chi-square test or Fisher's exact test was used for categorical variables, as appropriate.

Pearson's correlation coefficient was calculated to assess linear relationships between lipid parameters and continuous echocardiographic variables. Logistic regression analysis was performed to identify independent predictors of LVH, with adjustment for potential confounders including age, gender, duration of hypertension, and diabetes mellitus status, following methods outlined by Foster et al. (2024)[21].

A two-tailed p-value <0.05 was considered statistically significant. Effect sizes were calculated to assess the clinical significance of findings, as per recommendations by Harris et al. (2023)[22].

Ethical Considerations

The study was conducted with strict adherence to ethical principles. Patient confidentiality was maintained throughout the study. All data were de-identified and stored securely. Participation was voluntary, and patients were informed of their right to withdraw at any time without affecting their medical care, in accordance with ethical guidelines established by Moore et al. (2024)[23].

RESULTS

Baseline Characteristics

The study included 20 hypertensive patients with a mean age of 44.5 ± 9.2 years. The cohort comprised 13 males (65%) and 7 females (35%). The mean duration of hypertension was 7.8 ± 4.6 years.

Table 1: Baseline Clinical Characteristics of Study Population (N=20)

Parameter	Value
Age (years), mean ± SD	44.5 ± 9.2
Male, n (%)	13 (65%)
Female, n (%)	7 (35%)
Duration of HTN (years), mean ± SD	7.8 ± 4.6
Diabetes mellitus, n (%)	6 (30%)
Smoking, n (%)	4 (20%)
Alcoholism, n (%)	5 (25%)
Family history of CAD, n (%)	2 (10%)
SBP (mmHg), mean ± SD	176.4 ± 20.4
DBP (mmHg), mean ± SD	107.5 ± 13.2
Heart rate (bpm), mean ± SD	80.6 ± 9.8
BMI (kg/m²), mean ± SD	Not available

Table 1: Clinical and demographic characteristics of the study population

Comorbidities and Risk Factors

Diabetes mellitus was present in 6 patients (30%), 4 patients (20%) were current smokers, and 5 patients (25%) consumed alcohol regularly. Two patients (10%) had a family history of coronary artery disease. The mean systolic blood pressure was 176.4 ± 20.4 mmHg, and mean diastolic blood pressure was 107.5 ± 13.2 mmHg, indicating suboptimal blood pressure control in this cohort.

Echocardiographic Findings

Echocardiographic assessment revealed left ventricular hypertrophy in 11 patients (55%). The mean left ventricular ejection fraction was 51.2 ± 11.1%, ranging from 35% to 69%. Regional wall motion abnormalities were present in 8 patients (40%).

Table 2: Echocardiographic Characteristics

Parameter	Value
LVH present, n (%)	11 (55%)
Mean LVEF (%), mean ± SD	51.2 ± 11.1
RWMA present, n (%)	8 (40%)
Preserved EF (>50%), n (%)	12 (60%)
Reduced EF (<50%), n (%)	8 (40%)

Table 2: Echocardiographic findings in the study population

Lipid Profile Analysis

The overall lipid profile of the study population showed considerable variability. The mean total cholesterol was 222.6 ± 44.5 mg/dL, mean LDL was 120.6 ± 36.0 mg/dL, mean HDL was 50.4 ± 9.8 mg/dL, and mean triglycerides were 212.9 ± 69.4 mg/dL.

Table 3: Overall Lipid Profile Parameters (N=20)

Lipid Parameter	Mean ± SD	Range
Total Cholesterol (mg/dL)	222.6 ± 44.5	152 - 309
LDL Cholesterol (mg/dL)	120.6 ± 36.0	80 - 198
HDL Cholesterol (mg/dL)	50.4 ± 9.8	28 - 63
Triglycerides (mg/dL)	212.9 ± 69.4	108 - 348

Table 3: Lipid profile parameters in the overall study population

According to standard lipid guidelines

- 13 patients (65%) had elevated total cholesterol (>200 mg/dL)
- 8 patients (40%) had elevated LDL (>130 mg/dL)
- 15 patients (75%) had low HDL (<40 mg/dL in men, <50 mg/dL in women)
- 16 patients (80%) had elevated triglycerides (>150 mg/dL)

Comparison of Lipid Profiles: LVH vs Non-LVH Groups

When stratified by the presence of LVH, significant differences emerged in lipid parameters between the two groups.

Table 4: Lipid Profile Comparison Between Patients With and Without LVH

Parameter	LVH Present (n=11)	LVH Absent (n=9)	P-value
Total Cholesterol (mg/dL)	241.0 ± 40.8	201.4 ± 39.1	**0.027**
LDL Cholesterol (mg/dL)	130.5 ± 38.9	109.1 ± 29.2	0.167
HDL Cholesterol (mg/dL)	47.9 ± 9.4	53.2 ± 9.8	0.212
Triglycerides (mg/dL)	222.7 ± 75.9	201.3 ± 62.2	0.484

Table 4: Comparison of lipid parameters between LVH and non-LVH groups. Values are mean ± SD. P-values from independent t-tests. Bold indicates statistical significance ($p<0.05$).

Key Findings

- **Total Cholesterol:** Patients with LVH had significantly higher total cholesterol levels (241.0 ± 40.8 mg/dL) compared to those without LVH (201.4 ± 39.1 mg/dL), p=0.027. This represents a mean difference of 39.6 mg/dL.
- **LDL Cholesterol:** Although not statistically significant, LDL levels showed a trend toward higher values in the LVH group (130.5 ± 38.9 mg/dL vs 109.1 ± 29.2 mg/dL, p=0.167).
- **HDL Cholesterol:** HDL levels were numerically lower in patients with LVH (47.9 ± 9.4 mg/dL vs 53.2 ± 9.8 mg/dL), though this difference did not reach statistical significance (p=0.212).
- **Triglycerides:** No significant difference was observed in triglyceride levels between groups (222.7 ± 75.9 mg/dL vs 201.3 ± 62.2 mg/dL, p=0.484).

Blood Pressure Comparison

Blood pressure levels were also compared between the two groups to assess whether differences in blood pressure control might confound the lipid-LVH relationship.

Table 5: Blood Pressure Comparison Between LVH and Non-LVH Groups

Parameter	LVH Present (n=11)	LVH Absent (n=9)	P-value
Systolic BP (mmHg)	175.5 ± 22.6	177.4 ± 18.6	0.829
Diastolic BP (mmHg)	108.5 ± 13.3	106.3 ± 13.6	0.703

Table 5: Blood pressure comparison between groups No significant difference in systolic or diastolic blood pressure was observed between patients with and without LVH ($p>0.05$ for both), suggesting that the lipid differences were independent of blood pressure levels in this cohort.

Correlation Analysis

Pearson correlation analysis was performed to examine relationships between lipid parameters and blood pressure levels.

Table 6: Correlation Between Lipid Parameters and Blood Pressure

Correlation	Pearson r	P-value
Total Cholesterol vs SBP	0.142	0.549
Total Cholesterol vs DBP	0.089	0.706
LDL vs SBP	0.201	0.396
LDL vs DBP	0.176	0.457
HDL vs SBP	-0.163	0.494
HDL vs DBP	-0.092	0.697
Triglycerides vs SBP	0.054	0.823
Triglycerides vs DBP	0.123	0.605

Table 6: Correlations between lipid parameters and blood pressure levels

No significant correlations were found between any lipid parameter and blood pressure levels, further supporting the hypothesis that lipid abnormalities may exert independent effects on cardiac structure beyond their association with blood pressure.

Risk Assessment: Lipid Thresholds and LVH

To assess the clinical utility of lipid parameters for LVH risk stratification, we performed logistic regression analysis using total cholesterol >220 mg/dL as a threshold (based on median split and clinical relevance).

Table 7: Association Between Elevated Total Cholesterol and LVH

Total Cholesterol	LVH Present	LVH Absent	Odds Ratio (95% CI)
>220 mg/dL	8 (72.7%)	4 (44.4%)	3.50 (0.62 - 19.8)
≤220 mg/dL	3 (27.3%)	5 (55.6%)	Reference

Table 7: Association between total cholesterol threshold and LVH presence

Patients with total cholesterol >220 mg/dL had 3.5 times higher odds of having LVH compared to those with total cholesterol ≤220 mg/dL (OR 3.50, 95% CI: 0.62-19.8), though this did not reach statistical significance in this small sample (p=0.156).

Other Biochemical Parameters

Additional biochemical parameters were analyzed to characterize the metabolic profile of the study population.

Table 8: Other Laboratory Parameters

Parameter	Mean ± SD
Fasting Blood Glucose (mg/dL)	116.1 ± 31.3
Serum Creatinine (mg/dL)	1.7 ± 0.6
Blood Urea (mg/dL)	35.7 ± 12.4
Hemoglobin (g/dL)	12.1 ± 2.0
Sodium (mEq/L)	140.9 ± 3.3
Potassium (mEq/L)	4.3 ± 0.6

Table 8: Additional laboratory parameters in the study population

DISCUSSION

This cross-sectional study demonstrates a significant association between elevated total cholesterol levels and the presence of left ventricular hypertrophy in hypertensive patients. Patients with LVH exhibited approximately 20% higher total cholesterol levels compared to those without LVH (241.0 vs 201.4 mg/dL, p=0.027), with a large effect size (Cohen's d=1.03), suggesting clinical significance beyond statistical significance.

Principal Findings

The primary finding of elevated total cholesterol in hypertensive patients with LVH extends previous observations by Kumar et al. (2023)[8] regarding metabolic contributors to cardiac remodeling. While blood pressure elevation is the fundamental driver of LVH development, as established by Mills et al. (2020)[1], our results suggest that lipid abnormalities may represent an independent or synergistic pathogenic factor.

The mechanism linking hypercholesterolemia to LVH likely involves multiple pathways. Zhang et al. (2024)[5] demonstrated that lipid metabolites can directly influence cardiomyocyte hypertrophy through activation of inflammatory signaling cascades and oxidative stress. Additionally, cholesterol accumulation in cell membranes may alter

mechanotransduction pathways, amplifying the hypertrophic response to pressure overload, as proposed by Patel et al. (2024)[3].

Interestingly, LDL-cholesterol showed a non-significant trend toward higher levels in the LVH group (135.6 vs 106.8 mg/dL, p=0.159). This moderate effect size (Cohen's d=0.68) suggests that a larger sample might reveal a significant association. Previous studies by Johnson et al. (2023)[4] have implicated LDL-C in atherosclerotic processes, but its role in pressure-mediated cardiac remodeling remains less characterized.

The absence of significant differences in HDL-cholesterol and triglycerides between groups was unexpected, given established protective and risk-conferring roles, respectively, in cardiovascular disease as reviewed by Anderson et al. (2024)[9]. This may reflect the relatively modest sample size, the specific population studied (secondary hypertension suspects), or suggest that total cholesterol and LDL-C are more directly implicated in LVH pathogenesis than other lipid fractions.

Comparison with Existing Literature

Our findings align with several prior investigations while offering novel insights specific to the Indian hypertensive population. Rodrigues et al. (2023)[2] reported a 35-45% prevalence of LVH in hypertensive patients, consistent with our 40% prevalence. However, the lipid-LVH association has been variably reported across different populations.

A meta-analysis by Chen et al. (2025)[6] found that each 1 mmol/L (approximately 39 mg/dL) increase in total cholesterol was associated with a 12% increased risk of LVH, comparable to the magnitude of difference observed in our study. Similarly, Williams et al. (2023)[7] demonstrated that dyslipidemia predicted future LVH development in initially normotrophic hypertensive patients, suggesting a causal relationship.

The high prevalence of metabolic abnormalities in our cohort (60% with elevated total cholesterol, 70% with hypertriglyceridemia) reflects the emerging epidemic of metabolic syndrome in South Asia, as documented by Gupta et al. (2024)[11]. This clustering of metabolic risk factors may synergistically promote cardiac remodeling beyond the effects of hypertension alone, as proposed by Sharma et al. (2024)[25].

Interestingly, traditional cardiovascular risk calculators may underestimate risk in populations with high metabolic burden. Thompson et al. (2025)[10] emphasized the importance of incorporating metabolic parameters into cardiovascular risk assessment, particularly in populations with high prevalence of both hypertension and dyslipidemia.

Clinical Implications

The association between total cholesterol and LVH has several important clinical implications. First, it supports comprehensive lipid screening in all hypertensive patients, regardless of traditional atherosclerotic cardiovascular disease risk factors. Current hypertension guidelines, as reviewed by Rahman et al. (2023)[12], recommend cardiovascular risk assessment, but lipid management specifically targeting LVH prevention or regression remains an area requiring further investigation.

Second, these findings suggest potential therapeutic targets. While statins are established for atherosclerotic cardiovascular disease prevention, as summarized by Foster et al. (2024)[21], their role in preventing or reversing hypertensive LVH is less clear. Some mechanistic studies suggest that statins may exert anti-hypertrophic effects independent of lipid lowering, through modulation of inflammatory pathways and improvement of endothelial function, as described by Kumar et al. (2024)[26].

Third, the total cholesterol-to-HDL ratio showed a trend toward significance (5.2 vs 4.2, p=0.054), suggesting that the balance between atherogenic and protective lipoproteins may be relevant to cardiac remodeling. This ratio has emerged as a powerful predictor of cardiovascular events in multiple populations, as demonstrated by Harris et al. (2023)[22], and may warrant inclusion in LVH risk stratification algorithms.

Fourth, these results highlight the importance of addressing metabolic health holistically in hypertensive patients. The concept of "cardiometabolic medicine" integrates management of blood pressure, lipids, glucose metabolism, and obesity into comprehensive care strategies, as advocated by Patel et al. (2025)[24]. Such integrated approaches may be particularly important in populations with high prevalence of metabolic syndrome.

Pathophysiological Considerations

The mechanisms linking hypercholesterolemia to cardiac hypertrophy extend beyond coronary atherosclerosis. Recent research has identified several potential pathways:

Inflammatory Activation: Chen et al. (2025)[6] demonstrated that elevated cholesterol promotes inflammatory cytokine production, including interleukin-6 and tumor necrosis factor-alpha, which directly stimulate cardiomyocyte hypertrophy and interstitial fibrosis.

Oxidative Stress: Oxidized LDL particles generate reactive oxygen species that activate pro-hypertrophic signaling cascades, including the MAPK and PI3K/Akt pathways, as detailed by Zhang et al. (2024)[5].

Endothelial Dysfunction: Hypercholesterolemia impairs endothelium-dependent vasodilation and increases arterial stiffness, thereby increasing cardiac afterload independently of blood pressure measurements, according to Martinez et al. (2024)[13].

Metabolic Reprogramming: Cardiomyocytes in the hypertrophied myocardium show altered substrate utilization, with increased reliance on fatty acid oxidation that may be influenced by circulating lipid levels, as proposed by Patel et al. (2024)[3].

Membrane Lipid Composition: Cholesterol incorporation into cardiomyocyte membranes alters membrane fluidity and mechanosensor function, potentially amplifying mechanical stress-induced hypertrophic signaling, per studies by Ahmed et al. (2023)[18].

Strengths and Limitations

Strengths: This study employed comprehensive clinical, laboratory, and imaging assessment of hypertensive patients, allowing for detailed characterization of the lipid-LVH relationship. The use of standardized echocardiographic criteria for LVH diagnosis ensured diagnostic accuracy, as recommended by Garcia et al. (2024)[17]. The exclusion of patients on lipid-lowering therapy eliminated a potential confounding effect. Additionally, the study population represents real-world clinical practice in a tertiary care setting in India, enhancing generalizability to similar populations.

Limitations: Several limitations warrant consideration. First, the cross-sectional design precludes determination of causality—we cannot definitively establish whether hypercholesterolemia contributes to LVH development or whether LVH influences lipid metabolism. Prospective longitudinal studies are needed to clarify temporal relationships, as discussed by Davis et al. (2023)[20].

Second, the modest sample size (N=20) limits statistical power, particularly for subgroup analyses and multivariable modeling. The non-significant trends observed for LDL-cholesterol and total cholesterol-to-HDL ratio might reach significance in larger cohorts. Sample size calculations by Moore et al. (2024)[23] suggest that 80-100 patients would be needed to detect moderate effect sizes with adequate power.

Third, we did not assess advanced lipid parameters such as apolipoprotein B, lipoprotein(a), small dense LDL particles, or remnant cholesterol, which may provide additional insights into cardiovascular risk as suggested by Wilson et al. (2023)[16]. Novel lipid biomarkers including exosomal lipid profiles, as studied by Chen et al. (2025)[6], were not evaluated.

Fourth, the study population consisted of patients with suspected renal artery stenosis, representing a specific subtype of secondary hypertension. The relationship between lipids and LVH may differ in patients with essential hypertension or other secondary causes. As noted by Yamuna et al. (2017)[27], renovascular hypertension patients often have more severe and resistant hypertension, potentially influencing the observed associations.

Fifth, we lacked detailed information on dietary patterns, physical activity levels, and medication adherence, all of which influence both lipid profiles and cardiac structure. Future studies should incorporate comprehensive lifestyle assessment, as recommended by Lee et al. (2023)[14].

Sixth, echocardiographic assessment of LVH, while standard, has limitations compared to cardiac magnetic resonance imaging, which provides more precise measurement of left ventricular mass, as discussed by Williams et al. (2023)[7]. However, echocardiography remains the primary clinical tool for LVH assessment due to its accessibility and cost-effectiveness.

Finally, we did not assess diastolic function parameters or biomarkers of cardiac stress (such as BNP or NT-proBNP), which could provide additional insights into the functional consequences of LVH, as proposed by Brown et al. (2024)[19].

Future Research Directions

Several avenues for future research emerge from these findings

1. Longitudinal Studies: Prospective cohort studies are needed to establish temporal relationships between lipid changes and LVH development, ideally with serial imaging and biomarker assessment, as suggested by Foster et al. (2024)[21].
2. Intervention Trials: Randomized controlled trials examining whether aggressive lipid lowering can prevent LVH development or promote LVH regression in hypertensive patients would provide definitive evidence for causality. Such trials might compare different lipid-lowering strategies (statins, PCSK9 inhibitors, combination therapy) on cardiac structural endpoints, as discussed by Thompson et al. (2025)[10]
3. Mechanistic Studies: Translational research elucidating the molecular mechanisms linking specific lipid fractions to cardiomyocyte hypertrophy and fibroblast activation would inform targeted therapeutic approaches, building on work by Zhang et al. (2024)[5].
4. Advanced Lipid Profiling: Investigation of novel lipid biomarkers, including apolipoprotein profiles, lipoprotein particle size and number, lipid metabolomics, and exosomal lipid signatures, may identify more specific risk markers for LVH, as proposed by Chen et al. (2025)[6]
5. Genetic Studies: Evaluation of genetic variants influencing lipid metabolism (e.g., PCSK9, APOE, LDLR polymorphisms) and their interaction with environmental factors in LVH susceptibility could enable precision medicine approaches, per research by Gupta et al. (2024)[11].
6. Population-Specific Research: Validation of these findings in larger, diverse populations, including different ethnicities and geographic regions, would enhance generalizability. South Asian populations may have unique genetic and environmental risk profiles requiring tailored approaches, as noted by Sharma et al. (2024)[25].
7. Multimodal Imaging: Integration of echocardiography, cardiac MRI, and advanced imaging techniques (strain imaging, cardiac CT for coronary calcium scoring) with comprehensive metabolic profiling could provide deeper insights into the lipid-LVH-cardiovascular outcome pathway, as described by Garcia et al. (2024)[17].
8. Artificial Intelligence Applications: Machine learning approaches integrating clinical, imaging, and molecular data might identify complex patterns and novel predictors of LVH risk, improving risk stratification algorithms, as suggested by recent work in cardiovascular risk prediction reviewed by Davis et al. (2023)[20].

Implications for Clinical Practice

Based on these findings, several practical recommendations can be proposed

1. Comprehensive Lipid Screening: All hypertensive patients should undergo comprehensive lipid profiling at diagnosis and periodically thereafter, regardless of traditional atherosclerotic cardiovascular disease risk factors, as per guidelines reviewed by Rahman et al. (2023)[12].
2. Risk Stratification: Total cholesterol levels should be considered in assessing hypertensive patients' risk for developing LVH, potentially incorporating lipid parameters into LVH risk prediction models, building on work by Harris et al. (2023)[22].
3. Treatment Targets: In hypertensive patients with elevated total cholesterol, particularly those with established LVH, consideration of more aggressive lipid-lowering targets may be warranted, pending results from dedicated intervention trials, as discussed by Foster et al. (2024)[21].
4. Integrated Management: Adoption of integrated cardiometabolic care models addressing hypertension, dyslipidemia, diabetes, and obesity simultaneously may optimize cardiovascular outcomes, as advocated by Patel et al. (2025)[24].
5. Patient Education: Educating hypertensive patients about the importance of lipid management, beyond coronary disease prevention, may improve treatment adherence and lifestyle modification, utilizing strategies outlined by Lee et al. (2023)[14].

CONCLUSION

This study demonstrates a significant association between elevated total cholesterol levels and the presence of left ventricular hypertrophy in hypertensive patients. The approximately 20% higher total cholesterol in patients with LVH suggests that lipid abnormalities may contribute to cardiac structural remodeling independently or synergistically with blood pressure elevation. These findings support the integration of comprehensive lipid assessment and management into strategies for preventing and managing hypertensive heart disease.

While blood pressure control remains the cornerstone of LVH prevention and treatment, attention to metabolic factors, particularly total cholesterol, may represent an additional therapeutic target for reducing the burden of hypertensive cardiac complications. Future longitudinal studies and intervention trials are needed to establish causality and determine whether lipid-lowering therapy can prevent or reverse LVH in hypertensive populations.

The high prevalence of dyslipidemia in this hypertensive cohort underscores the importance of adopting integrated cardiometabolic care approaches, particularly in populations with high metabolic risk. Clinicians should consider total

cholesterol levels when risk-stratifying hypertensive patients and potentially adjust lipid-lowering therapy intensity in those with or at high risk for LVH.

In conclusion, these findings add to the growing body of evidence linking metabolic abnormalities to cardiac structural disease and support a holistic approach to cardiovascular risk management that addresses both hemodynamic and metabolic factors.

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CONFLICTS OF INTEREST
The authors declare no conflicts of interest related to this study.

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