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STUDY OF SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS IN A TERTIARY CARE HOSPITAL, BAREILLY

Deependra Yadav[1], Pankaj Parmar[2], Sayan Bhattacharjee[3], Mukesh Dube[4], Ajit Sawhney[5], W.P. Singh[6]

[1]*PG Resident, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[2]*PG Resident, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[3]*Senior Resident, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[4]*Associate Professor, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[5]*Professor, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[6]*Professor & Head, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*

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Corresponding Author:
Dr. W. P. Singh,
Professor and Head, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.
Email: profdrwpsingh@gmail.com

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ABSTRACT

Background: Hypertension frequently coexists with dyslipidemia, significantly increasing cardiovascular risk. Regional data regarding the prevalence and pattern of lipid abnormalities among hypertensive patients in Bareilly are limited. The objective is to evaluate the prevalence, pattern, and clinical associations of serum lipid abnormalities among hypertensive patients attending a tertiary care hospital in Bareilly.

Materials and Methods: This observational cross-sectional study included 112 adult hypertensive patients attending the Department of General Medicine over one year (July 2024 - June 2025). Demographic details, clinical history, lifestyle factors, and anthropometric measurements were recorded. Fasting lipid profiles were analyzed using standardized laboratory methods. Statistical analysis included chi-square tests, t-tests, and correlation analysis, with $p < 0.05$ considered significant.

Results: Dyslipidemia was present in 83 patients (74.11%). Significant associations were observed between dyslipidemia and comorbid conditions such as CKD, diabetes, NAFLD, and obesity ($p = 0.02$). Lifestyle factors including smoking ($p = 0.0017$), alcohol consumption ($p = 0.0039$), and sedentary behavior ($p = 0.011$) were strongly linked to lipid abnormalities. Dietary habits such as high salt intake ($p = 0.010$), low fruit and vegetable consumption ($p = 0.00075$), and high-fat diet ($p = 0.0023$) showed significant associations. Mixed dyslipidemia, particularly elevated total cholesterol, LDL, and triglycerides, was the most common pattern. Hypertension severity demonstrated significant correlation with lipid parameters, especially triglycerides ($p < 0.001$).

Conclusion: A high prevalence of dyslipidemia exists among hypertensive patients in this tertiary care setting, with strong associations to metabolic comorbidities, lifestyle factors, and hypertension severity. Integrated dual-risk-factor management incorporating routine lipid screening and aggressive lifestyle modification is essential to reduce cardiovascular morbidity and mortality.

Keywords: Hypertension; Dyslipidemia; Serum Lipid Profile; Mixed Dyslipidemia; Cardiometabolic Risk; Lifestyle Factors; Tertiary Care Hospital.

INTRODUCTION

Hypertension is one of the most prevalent non-communicable diseases globally and a major cause of preventable morbidity and mortality. It is defined as a sustained systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg.[1] Because it frequently remains asymptomatic in its early stages, hypertension is often described as a "silent killer," as significant target organ damage may occur before clinical symptoms become

evident.[2] Persistent elevation of blood pressure is strongly associated with cardiovascular disease, stroke, chronic kidney disease, and peripheral vascular disease, making it a critical global public health concern.

According to the World Health Organization, approximately 1.28 billion adults aged 30–79 years worldwide are affected by hypertension, with nearly two-thirds living in low- and middle-income countries.[3] In India, the prevalence of hypertension has increased steadily over the past few decades due to rapid urbanization, sedentary lifestyles, dietary transitions, obesity, and rising life expectancy.[4] National surveys have shown increasing trends in both urban and rural populations, with a concerning shift toward earlier onset among younger individuals.[4]

Hypertension often coexists with metabolic abnormalities, among which dyslipidemia is one of the most clinically significant.[5] Dyslipidemia refers to abnormal serum lipid levels, including elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and/or reduced high-density lipoprotein cholesterol (HDL-C). The coexistence of hypertension and dyslipidemia significantly increases cardiovascular risk due to their synergistic adverse effects on vascular endothelium.[6]

The pathophysiological link between hypertension and dyslipidemia is multifactorial. Both conditions share common mechanisms such as endothelial dysfunction, insulin resistance, oxidative stress, chronic inflammation, and activation of the renin–angiotensin–aldosterone system.[7] Dyslipidemia reduces nitric oxide availability and promotes lipid deposition in the vascular intima, accelerating atherosclerosis. Conversely, chronic hypertension enhances vascular permeability and oxidative stress, thereby aggravating lipid abnormalities and promoting plaque formation.[8] This bidirectional interaction contributes to arterial stiffness, vascular remodeling, and progressive target organ damage.

Epidemiological studies have consistently demonstrated a higher prevalence of lipid abnormalities among hypertensive individuals compared to normotensive controls.[5] Atherogenic dyslipidemia—characterized by elevated LDL-C and triglycerides along with reduced HDL-C—is commonly observed in hypertensive patients and is strongly linked to coronary artery disease and stroke.[6] In the Indian population, genetic predisposition to insulin resistance, central obesity, and adverse lipid patterns further amplifies cardiovascular risk, even at relatively lower body mass indices.[9] Lifestyle factors such as diets rich in saturated fats and refined carbohydrates, physical inactivity, tobacco use, and alcohol consumption also contribute significantly to altered lipid metabolism.[4,9]

The coexistence of hypertension and dyslipidemia markedly worsens prognosis. Dyslipidemia accelerates atherosclerosis, increases plaque instability, and elevates thrombotic risk.[8] Even moderate abnormalities in lipid levels can significantly increase cardiovascular morbidity and mortality among hypertensive patients.[10]. herefore, comprehensive cardiovascular risk assessment should include routine evaluation of serum lipid profiles in hypertensive individuals.

Tertiary care hospitals play a crucial role in the integrated evaluation and management of hypertensive patients, particularly those with comorbid conditions. However, region-specific data regarding the prevalence and pattern of dyslipidemia among hypertensive patients in Bareilly remain limited. Generating such local evidence is essential for early identification of high-risk individuals, appropriate risk stratification, and implementation of targeted preventive strategies.

In this context, the present study aims to evaluate the serum lipid profile among hypertensive patients attending a tertiary care hospital in Bareilly. Understanding the prevalence, pattern, and demographic associations of lipid abnormalities in this population will contribute to improved integrated cardiovascular risk management.

MATERIALS AND METHODS

This observational cross-sectional study was conducted in the Department of General Medicine at a tertiary care teaching hospital in Bareilly over a period of one year (July 2024 - June 2025). The study population comprised adult patients attending the General Medicine Outpatient Department (OPD) as well as those admitted to the Inpatient Department (IPD) with a confirmed diagnosis of hypertension. Both newly diagnosed and previously known hypertensive patients receiving treatment were considered eligible for participation. Consecutive sampling was adopted during the study period to minimize selection bias and ensure adequate representation of the hypertensive population.

Hypertension was defined as a sustained systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, or current use of antihypertensive medication. Patients aged 18 years and above of either sex who were willing to provide written informed consent were included in the study. Exclusion criteria comprised individuals without a formal diagnosis of hypertension, patients below 18 years of age, pregnant or lactating women, patients with a prior history of major cardiovascular events such as myocardial infarction or stroke, and those with significant systemic illnesses known to affect lipid metabolism including thyroid disorders and chronic liver disease. Patients taking medications known to alter lipid levels (unless prescribed as part of hypertension management) and those unwilling or unable to provide informed consent were also excluded.

The sample size was calculated using the standard formula for estimating prevalence in cross-sectional studies:

$n = (Z^2 \times P \times Q) / d^2$

where Z represents the standard normal variate at 95% confidence level (1.96; squared ≈3.84), P is the expected prevalence (assumed 25%), Q = 100 – P, and d is the allowable error (8%). This method of prevalence-based sample size estimation is widely accepted in epidemiological research [11]. Based on feasibility and pilot observations, approximately 14 eligible cases were identified per month. Over an estimated eight-month recruitment period, a total sample size of 112 hypertensive patients was finalized. A pilot study was conducted prior to full-scale data collection to assess feasibility, refine data collection tools, and validate recruitment rates.

After obtaining informed written consent, demographic details including age and sex were recorded using a structured and pretested proforma. A detailed clinical history was obtained, including duration of hypertension, treatment status, and relevant lifestyle factors such as dietary habits, physical activity, tobacco use, and alcohol consumption. Physical examination was performed for all participants.

Blood pressure measurement was carried out using a standardized sphygmomanometer following recommended clinical guidelines to ensure accuracy and reproducibility.[12] Measurements were taken in the sitting position after at least five minutes of rest, and two readings were recorded at an interval of five minutes. The average of the two readings was considered for analysis to reduce observer variability. Anthropometric measurements including weight and height were obtained using calibrated instruments, and body mass index (BMI) was calculated using the standard formula.

Venous blood samples were collected under aseptic precautions after an overnight fast of 8–12 hours for estimation of serum lipid profile. The lipid parameters assessed included total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides. All biochemical analyses were performed using automated analyzers available in the hospital's central laboratory, adhering to standard operating procedures and internal quality control measures to ensure accuracy and reliability.

To ensure validity and reliability of data, standardized instruments were used for blood pressure and anthropometric measurements. Duplicate blood pressure recordings minimized observer bias. Laboratory investigations were conducted in a quality-controlled environment, and data collection was performed using a uniform structured proforma to maintain consistency.

Data were entered into a secure database and analyzed using appropriate statistical software. Descriptive statistics were used to summarize demographic characteristics, blood pressure values, and lipid profile parameters. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The prevalence of dyslipidemia was calculated as the proportion of hypertensive patients exhibiting abnormal lipid parameters according to standard reference ranges.

Correlation analysis was performed to assess the relationship between severity of hypertension and variations in lipid parameters using Pearson or Spearman correlation coefficients, depending on the distribution of data. Comparative analyses across demographic groups were conducted using independent t-tests or one-way analysis of variance (ANOVA) for continuous variables and chi-square tests for categorical variables. Multivariate regression analysis was undertaken to evaluate the independent association of demographic and clinical variables with serum lipid levels. Statistical analysis was conducted in accordance with established principles of medical statistics,[13] and a p-value <0.05 was considered statistically significant.

The study was conducted in strict accordance with ethical principles for biomedical research involving human participants as outlined in the Declaration of Helsinki.[14] Approval was obtained from the Institutional Ethics Committee prior to commencement of the study. Written informed consent was secured from all participants, and confidentiality of patient information was maintained throughout the study.

RESULTS

Among 112 hypertensive patients, 83 (74.11%) had dyslipidemia, indicating a high burden of lipid abnormalities. As demonstrated in [Table 1], dyslipidemia was significantly associated with comorbid conditions ($\chi^2 = 13.12$, $p = 0.02$), with highest prevalence in CKD (100%), diabetes (88.24%), NAFLD (88.89%), and obesity (83.33%).

Table 1: Other Medical Conditions versus Dyslipidemia

Other Medical Condition	No Dyslipidemia	Yes Dyslipidemia	Total
CKD	0 (0%)	8 (100%)	8
Diabetes	2 (11.76%)	15 (88.24%)	17
NAFLD	1 (11.11%)	8 (88.89%)	9
None	20 (33.33%)	40 (66.67%)	60
Obesity	1 (16.67%)	5 (83.33%)	6
Thyroid Disorder	5 (41.67%)	7 (58.33%)	12
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 13.12$, p-value: 0.02			

Lifestyle factors (combined findings from [Table 2-4] showed strong associations. Current smokers (89.47%) and alcohol consumers (occasional 90.62%, regular 87.50%) had markedly higher dyslipidemia prevalence compared to non-smokers and non-drinkers ($p < 0.01$). Sedentary individuals also demonstrated higher prevalence (82.05%) compared to active participants (61.54%) ($p = 0.011$).

Table 2: Smoking Status versus Dyslipidemia

Smoking Status	No Dyslipidemia	Yes Dyslipidemia	Total
Current	4 (10.53%)	34 (89.47%)	38
Former	4 (16.0%)	21 (84.0%)	25
Never	21 (42.86%)	28 (57.14%)	49
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 12.78$, p-value: 0.0017			

Table 3: Alcohol Consumption versus Dyslipidemia

Alcohol Consumption	No Dyslipidemia	Yes Dyslipidemia	Total
Never	25 (34.72%)	47 (65.28%)	72
Occasionally	3 (9.38%)	29 (90.62%)	32
Regularly	1 (12.5%)	7 (87.5%)	8
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 11.04$, p-value: 0.0039			

Table 4: Physical Activity versus Dyslipidemia

Physical Activity	No Dyslipidemia	Yes Dyslipidemia	Total
Active	10 (38.46%)	16 (61.54%)	26
Moderate	12 (25.53%)	35 (74.47%)	47
Sedentary	7 (17.95%)	32 (82.05%)	39
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 8.94$, p-value: 0.011			

Dietary factors were significantly related to lipid abnormalities. High salt intake [Table 5] was associated with higher dyslipidemia prevalence (85.71%, $p = 0.010$). Low fruit and vegetable intake [Table 6] showed the highest prevalence (86%), while high intake was protective (44.44%) ($p = 0.00075$). Similarly, high-fat food consumption [Table 7] was strongly associated with dyslipidemia (88.57%, $p = 0.0023$).

Table 5: Salt Intake versus Dyslipidemia

Salt Intake	No Dyslipidemia	Yes Dyslipidemia	Total
High	3 (14.29%)	18 (85.71%)	21
Low	15 (39.47%)	23 (60.53%)	38
Moderate	11 (20.75%)	42 (79.25%)	53
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 9.18$, p-value: 0.010			

Table 6: Fruit/Vegetable Consumption versus Dyslipidemia

Fruit/Vegetable Consumption	No Dyslipidemia	Yes Dyslipidemia	Total
High	10 (55.56%)	8 (44.44%)	18
Low	7 (14.00%)	43 (86.00%)	50
Moderate	12 (27.27%)	32 (72.73%)	44
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 14.38$, p-value: 0.00075			

Table 7: High-Fat Food Consumption versus Dyslipidemia

High-Fat Food Consumption	No Dyslipidemia	Yes Dyslipidemia	Total
High	4 (11.43%)	31 (88.57%)	35
Low	12 (50.00%)	12 (50.00%)	24
Moderate	13 (24.53%)	40 (75.47%)	53
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 12.16$, p-value: 0.0023			

A statistically significant association was observed between hypertension stage and type of dyslipidemia in [Table 8] ($\chi^2 \approx 6.42$, $p = 0.040$), with mixed LDL/TG abnormalities predominating. Clinical and biochemical comparisons in [Table 9] revealed significantly higher BMI, blood pressure, total cholesterol, LDL, and triglycerides and lower HDL among dyslipidemic patients (all $p < 0.05$).

Table 8: Hypertension Stage versus Type of Dyslipidemia

Hypertension Stage	Normal (n=29)	High LDL/TG/Mixed (n=83)	Total
Prehypertension	10 (18.18%)	45 (81.82%)	55
Stage 1 Hypertension	19 (34.55%)	36 (65.45%)	55
Stage 2 Hypertension	0 (0%)	2 (100%)	2
Total	29 (25.89%)	83 (74.11%)	112
Chi-square value: $\chi^2 \approx 6.42$, p-value ≈ 0.040			

Table 9: Clinical and Biochemical Variables versus Dyslipidemia

Variable	No Dyslipidemia (n=29)	Yes Dyslipidemia (n=83)	p-value (updated)
Height (cm)	161.20 ± 6.10	164.90 ± 7.20	0.02
Weight (kg)	63.10 ± 8.90	70.40 ± 10.80	0.004
Waist Circumference (cm)	84.20 ± 5.10	89.30 ± 7.10	0.001
BMI (kg/m²)	23.90 ± 3.90	26.70 ± 4.30	0.003
SBP (mmHg)	146 ± 8.10	150.80 ± 9.10	0.015
DBP (mmHg)	89 ± 6.90	93.80 ± 7.40	0.01
Total Cholesterol (mg/dL)	193.50 ± 21.10	218.00 ± 29.40	<0.001
LDL (mg/dL)	128.20 ± 17.20	150.10 ± 21.80	<0.001
HDL (mg/dL)	47.10 ± 4.20	41.30 ± 4.60	<0.001
Triglycerides (mg/dL)	141.00 ± 34.20	184.70 ± 36.90	<0.001

Pattern analysis in [Table 10] confirmed mixed dyslipidemia—particularly the combination of high total cholesterol, high LDL, and high triglycerides (26.79%)—as the most common pattern ($p < 0.001$).

Correlation analysis in [Table 11] demonstrated significant relationships between hypertension severity and lipid parameters, especially triglycerides ($\rho = -0.34$, $p < 0.001$).

Table 10: Patterns of Lipid Abnormalities among Hypertensive Patients

Pattern	Count (n)	Percentage (%)
Normal	29	25.89%
High Cholesterol, High LDL, High TG	30	26.79%
High LDL, High TG	18	16.07%
High TG	5	4.46%
High Cholesterol, High TG	5	4.46%
High Cholesterol, High LDL	7	6.25%
High Cholesterol, High LDL, Low HDL, High TG	6	5.36%
High Cholesterol, High LDL, Low HDL	4	3.57%
High LDL, Low HDL, High TG	4	3.57%
High Cholesterol	2	1.79%
Low HDL, High TG	1	0.89%
High LDL, Low HDL	1	0.89%
High Cholesterol, Low HDL, High TG	0	0.00%
High LDL	0	0.00%
Total	112	100%
Chi-square value: $\chi^2 \approx 46.3$, p-value: < 0.001		

Table 11: Correlation between Hypertension Severity and Lipid Profiles

Variable	Spearman's rho	p-value
Total Cholesterol (mg/dL)	−0.32	0.001
LDL (mg/dL)	−0.36	<0.001
HDL (mg/dL)	0.28	0.003
Triglycerides (mg/dL)	−0.34	<0.001

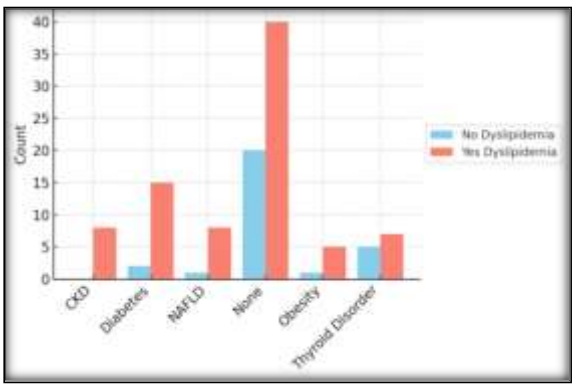

Figure 1: Other Medical Conditions versus Dyslipidemia

The overall trends are visually represented in [Figure 1] (comorbid conditions and dyslipidemia), [Figure 2] (physical activity and dyslipidemia), and [Figure 3] (patterns of lipid abnormalities), collectively reinforcing the strong interrelationship between hypertension severity, metabolic comorbidities, lifestyle behaviors, and adverse lipid profiles.

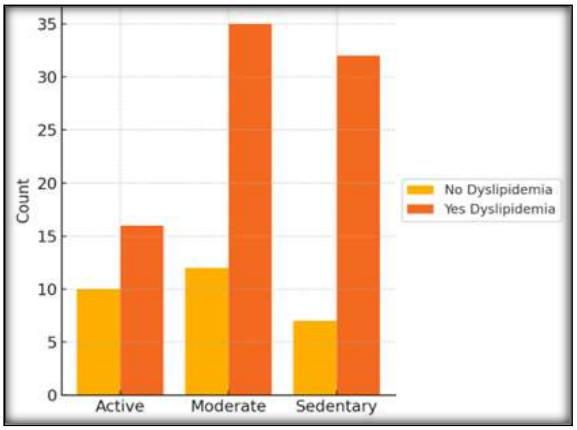

Figure 2: Physical Activity versus Dyslipidemia

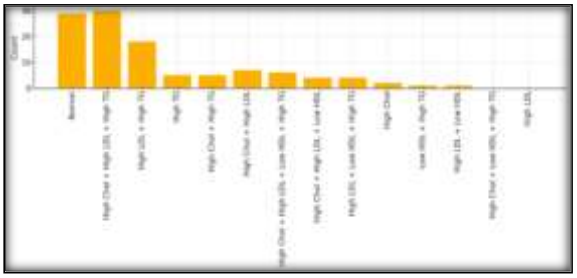

Figure 3: Patterns of Lipid Abnormalities among Hypertensive Patients

DISCUSSION

The present study demonstrated a high prevalence of dyslipidemia (74.11%) among hypertensive patients, underscoring the substantial coexistence of lipid abnormalities with elevated blood pressure. This finding aligns with prior Indian and international data showing that hypertension frequently clusters with atherogenic lipid profiles, thereby markedly increasing cardiovascular risk.[15,16] The coexistence of these two conditions accelerates endothelial dysfunction, oxidative stress, and vascular remodeling, contributing to premature atherosclerosis.

A significant association was observed between comorbid medical conditions and dyslipidemia, with the highest prevalence among patients with chronic kidney disease, diabetes mellitus, NAFLD, and obesity. This clustering reflects the broader cardiometabolic syndrome phenotype, where insulin resistance and chronic low-grade inflammation play central roles.[17] Diabetic and CKD patients are particularly predisposed to elevated triglycerides and small dense LDL particles, which have high atherogenic potential.[18] These findings emphasize the need for comprehensive lipid evaluation in hypertensive patients with metabolic comorbidities.

Lifestyle factors showed strong and statistically significant relationships with dyslipidemia. Current smokers and alcohol consumers had markedly higher prevalence of lipid abnormalities compared to non-users. Cigarette smoking promotes oxidative modification of LDL cholesterol and impairs HDL-mediated reverse cholesterol transport, thereby enhancing atherogenesis.[19] Similarly, excessive alcohol intake is associated with hypertriglyceridemia and mixed dyslipidemia due to altered hepatic lipid metabolism.[20] Physical inactivity further contributed to adverse lipid patterns, while active individuals demonstrated comparatively lower prevalence, supporting evidence that regular exercise improves HDL levels and reduces triglycerides.[21] These results reinforce the critical role of behavioral modification in cardiovascular risk reduction.

Dietary habits were also significantly associated with lipid abnormalities. High salt intake correlated with increased dyslipidemia prevalence. Although sodium primarily affects blood pressure, emerging evidence suggests it may indirectly worsen metabolic parameters through insulin resistance and endothelial dysfunction.[15] Low fruit and vegetable intake showed a strong association with dyslipidemia, whereas higher intake demonstrated a protective effect. Diets rich in fiber and antioxidants improve lipid metabolism and reduce cardiovascular risk.[21] Similarly, high-fat food consumption was strongly associated with dyslipidemia, consistent with established evidence that saturated fats elevate LDL cholesterol and triglycerides.[16]

A statistically significant association was found between hypertension stage and type of dyslipidemia, with mixed LDL and triglyceride abnormalities predominating. Correlation analysis further demonstrated significant relationships between hypertension severity and lipid parameters, particularly triglycerides and LDL cholesterol. These findings support the concept that worsening blood pressure parallels progressive metabolic deterioration, amplifying overall cardiovascular risk.[22]

Clinical and biochemical comparisons revealed that dyslipidemic patients had significantly higher BMI, waist circumference, systolic and diastolic blood pressure, total cholesterol, LDL, and triglycerides, along with lower HDL levels. This clustering of anthropometric and biochemical abnormalities represents a high-risk cardiometabolic phenotype.[17] Pattern analysis confirmed that mixed dyslipidemia—especially elevated total cholesterol, LDL, and triglycerides—was the most common presentation. Such atherogenic patterns are strongly linked with coronary artery disease and stroke.[16]

Overall, the findings highlight the strong interrelationship between hypertension severity, metabolic comorbidities, lifestyle behaviors, dietary practices, and dyslipidemia. The high prevalence of lipid abnormalities in this cohort underscores the necessity of routine lipid screening, integrated cardiovascular risk assessment, and aggressive lifestyle and pharmacological interventions to reduce long-term morbidity and mortality among hypertensive patients.

CONCLUSION

This hospital-based cross-sectional study conducted in a tertiary care center in Bareilly demonstrates a markedly high prevalence of dyslipidemia (74.11%) among hypertensive patients, highlighting the substantial coexistence of lipid abnormalities with elevated blood pressure. The predominance of mixed dyslipidemia—particularly elevated total cholesterol, LDL, and triglycerides—indicates the presence of a highly atherogenic lipid pattern in this population. Significant associations with metabolic comorbidities, adverse lifestyle behaviors, dietary practices, and increasing hypertension severity further support the concept of a clustered cardiometabolic risk phenotype rather than isolated hypertension. These findings clearly suggest that hypertension management should not be approached in isolation but as part of a broader cardiovascular risk reduction strategy.

A notable strength of this study lies in its comprehensive evaluation of hypertensive patients through integration of clinical, biochemical, lifestyle, dietary, and comorbidity-related variables within a single analytical framework. Standardized blood pressure measurement techniques, fasting lipid estimation under quality-controlled laboratory settings, and appropriate statistical analyses enhance the reliability and internal validity of the findings. Furthermore, the study provides valuable region-specific epidemiological data for Bareilly, where limited evidence was previously available regarding the coexistence and patterns of dyslipidemia among hypertensive individuals. The detailed assessment of lipid patterns and correlation with hypertension severity adds further clinical relevance to the observations.

However, several limitations must be acknowledged. The cross-sectional design restricts the ability to establish causal relationships or temporal sequencing between dyslipidemia and hypertension progression. As a single-center tertiary care study, the findings may not be fully generalizable to the broader community or primary care population, since tertiary hospitals often manage patients with more advanced or complicated disease profiles. Certain subgroups, such as stage 2 hypertension and specific comorbid conditions, had relatively small sample sizes, which may reduce statistical power and limit the robustness of subgroup analyses.

Lifestyle and dietary factors were assessed through self-reported data, introducing potential recall and social desirability bias. The dietary evaluation lacked detailed quantitative nutrient assessment, and psychosocial determinants such as stress levels, socioeconomic status, and health literacy were not explored. Additionally, LDL cholesterol was calculated rather than directly measured, and advanced lipid markers such as apolipoprotein B or lipoprotein(a) were not included. The absence of longitudinal follow-up data also prevents assessment of long-term cardiovascular outcomes associated with the identified lipid patterns.

Overall, the study reinforces the urgent need for integrated dual-risk-factor management in hypertensive patients. Routine lipid screening, careful selection of antihypertensive agents considering metabolic effects, and structured, culturally appropriate lifestyle interventions should form the cornerstone of management. A coordinated, multidisciplinary approach aimed at simultaneous control of blood pressure and lipid abnormalities is essential to reduce the growing burden of atherosclerotic cardiovascular disease in Bareilly and similar healthcare settings.

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