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# CORRELATION OF DYSLIPIDEMIA/ HYPERLIPIDEMIA AND BLOOD PRESSURE IN CARDIOVASCULAR DISEASE PATIENTS

*Original Research*

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## ABSTRACT

**Background:** Hypertension and dyslipidemia are among the leading contributors to cardiovascular disease (CVD) morbidity and mortality. These conditions frequently coexist, amplifying the risk of atherosclerosis and cardiac events. Dyslipidemia, characterized by elevated total cholesterol, low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), and triglycerides, alongside low high-density lipoprotein (HDL), is further exacerbated by obesity. Despite the well-established individual risks, their interrelationship—especially in hypertensive CVD patients with obesity—requires deeper investigation to enable earlier intervention and more precise management.

**Objective:** To determine the relationship between lipid profile parameters and blood pressure in cardiovascular disease patients and evaluate how CVD status, obesity, gender, and age influence lipid abnormalities among hypertensive individuals.

**Methods:** This cross-sectional observational study was conducted over six months at CMA Research Lab Lahore, DHQ Hospital Sahiwal, Social Security Hospital Lahore, and Punjab Institute of Cardiology Lahore. A total of 103 patients aged 30–70 years were enrolled via non-probability convenience sampling. Inclusion criteria comprised patients with confirmed dyslipidemia and hypertension (≥140/90 mmHg), while those with secondary hypertension, stage 4+ chronic kidney disease, or recent myocardial infarction were excluded. Blood pressure was measured using a digital sphygmomanometer, and lipid profiles were analyzed using automated chemistry analyzers. Statistical analysis involved SPSS, with Pearson's correlation, independent t-tests, and chi-square tests; p-values <0.05 were considered significant.

**Results:** Among 103 patients, 65 (63.1%) were males and 38 (36.9%) females, with most aged 40–49 years (29.1%). Hypertensive patients had significantly elevated total cholesterol (232.34 ± 34.18 mg/dL), LDL (143.94 ± 30.80 mg/dL), VLDL (53.16 ± 16.91 mg/dL), and triglycerides (273.16 ± 90.76 mg/dL) compared to non-hypertensives ($p < 0.05$). In hypertensive CVD patients, total cholesterol (223.79 ± 41.03 mg/dL) and VLDL (53.79 ± 16.03 mg/dL) were significantly higher than in non-CVD hypertensives. Obese hypertensive individuals had higher total cholesterol (227.91 ± 36.50 mg/dL), LDL (137.21 ± 34.88 mg/dL), VLDL (54.07 ± 16.27 mg/dL), and cholesterol/HDL ratio (5.74 ± 1.04) than non-obese counterparts ($p < 0.05$).

**Conclusion:** There is a clear association between hypertension and dyslipidemia, further intensified by obesity and cardiovascular disease status. These findings advocate for early lipid screening, lifestyle changes, and targeted therapeutic strategies to prevent cardiovascular complications in hypertensive patients.

**Keywords:** Atherosclerosis; Blood Pressure; Cardiovascular Diseases; Dyslipidemias; Hypertension; Obesity; Risk Factors.


## INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of global mortality, accounting for approximately 17.9 million deaths annually, and continues to represent a significant public health challenge (1). Encompassing a spectrum of conditions such as coronary artery disease, heart failure, and cerebrovascular disease, CVD imposes a growing burden on healthcare systems worldwide (2). Among the major modifiable contributors to this burden are hypertension and hyperlipidemia, both of which have been well-established as critical and interrelated risk factors. The interaction between these conditions not only exacerbates vascular injury but also accelerates the progression of atherosclerosis, highlighting the need for integrated research and clinical approaches (3). Hyperlipidemia, particularly elevated low-density lipoprotein cholesterol (LDL-C), plays a pivotal role in atherogenesis. LDL-C promotes the formation of atherosclerotic plaques, narrowing arterial lumens and impairing blood flow, thereby elevating the risk of ischemic cardiovascular events. This condition has become increasingly prevalent, driven largely by sedentary lifestyles, obesity, and diets rich in saturated and trans fats (4). In parallel, hypertension, defined as persistent systolic blood pressure ≥140 mmHg or diastolic pressure ≥90 mmHg, contributes to endothelial dysfunction, vascular remodeling, and increased cardiac workload, ultimately predisposing individuals to heart failure and other complications (5,6).

Notably, the presence of hyperlipidemia can influence renal function by impairing glomerular filtration, leading to sodium and fluid retention, which in turn contributes to elevated blood pressure levels (7). Conversely, hypertension has been shown to adversely alter lipid metabolism, leading to increased LDL-C and decreased high-density lipoprotein cholesterol (HDL-C), thus intensifying the cardiovascular risk profile (8). While the individual pathophysiology of both hypertension and hyperlipidemia is well-documented, their combined impact—especially in patients with established CVD—remains underexplored. Emerging data suggests that over 70% of patients with cardiovascular disease also present with coexisting hypertension and hyperlipidemia, creating a detrimental feedback loop that accelerates vascular inflammation, endothelial damage, and atherosclerotic plaque development (9). Despite this high prevalence, most studies have historically focused on these risk factors in isolation, overlooking the synergistic effects they exert when co-present. Furthermore, critical determinants such as obesity and gender differences in this context have been insufficiently studied, leaving significant gaps in understanding the nuanced interplay between lipid disorders and blood pressure regulation.

The growing body of evidence has also started to explore the genetic and epigenetic mechanisms underpinning these conditions. Variations in genes such as those encoding LDL receptors may influence susceptibility to both hypertension and hyperlipidemia (10). Epigenetic changes, including DNA methylation and histone modifications, have also been implicated in the pathogenesis of these diseases, suggesting a complex regulatory network that may inform future individualized therapies (11). Advances in pharmacological interventions, including the use of PCSK9 inhibitors, show promise not only in lowering LDL-C but also in exerting modest antihypertensive effects, underscoring the potential for dual-targeted strategies (12). Despite progress in lifestyle interventions and pharmacotherapy, the concurrent evaluation of blood pressure and lipid profile in patients with cardiovascular disease—particularly with respect to the modifying effects of obesity and gender—remains an understudied yet clinically relevant area. Addressing this knowledge gap is essential for improving risk stratification and tailoring therapeutic strategies for high-risk individuals. Therefore, the objective of this study is to investigate the association between blood pressure and lipid profiles in patients with established cardiovascular disease, with specific focus on hypertensive versus normotensive and obese versus non-obese subgroups. This approach aims to contribute to more personalized and effective preventive cardiology practices.

## METHODS

This retrospective observational study was conducted to explore the correlation between blood pressure and lipid profile parameters in patients diagnosed with cardiovascular disease (CVD). The study was carried out across multiple clinical sites, including CMA Research Lab, Lahore; DHQ Hospital, Sahiwal; Social Security Hospital, Lahore; and the Punjab Institute of Cardiology, Lahore, over a period of four months. A total of 103 patients were included in the study using non-probability convenience sampling. Ethical approval was obtained from the respective institutional review boards of the participating centers prior to data collection, and written informed consent was secured from all participants prior to their inclusion in the study, in accordance with the Declaration of Helsinki. Participants were

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eligible for inclusion if they were between 30 and 70 years of age, had a confirmed diagnosis of cardiovascular disease, presented with an abnormal lipid profile, and had elevated blood pressure readings defined as systolic ≥140 mmHg or diastolic ≥90 mmHg. Patients were excluded if they had secondary hypertension, chronic kidney disease at stage 4 or higher, hepatic dysfunction, were pregnant, had experienced a recent myocardial infarction, or had been on lipid-lowering or antihypertensive medications for more than six months prior to study entry. These criteria were established to minimize confounding variables that could independently affect lipid and blood pressure parameters.

Data collection involved the review of medical records to retrieve clinical and biochemical information. Blood pressure was measured using a standard digital sphygmomanometer following established protocols to ensure accuracy and consistency. Lipid profiles—including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides, and very-low-density lipoprotein (VLDL)—were assessed using automated and semi-automated chemistry analyzers (Beckman Coulter AU480 and Microlab 300). All blood samples were drawn within six hours of myocardial infarction symptom onset in eligible patients and analyzed at the time of hospital admission to reflect acute-phase levels. A structured data collection proforma was employed to capture demographics (age, gender, obesity status), medical history, comorbidities (hypertension and diabetes), and cardiovascular disease status. The primary dependent variables were systolic and diastolic blood pressure, while independent variables included lipid profile components. Additional variables such as obesity and gender were also analyzed to determine their association with hypertension and dyslipidemia. Statistical analysis was conducted using SPSS software. Continuous variables were summarized as means and standard deviations, while categorical variables were presented as frequencies and percentages. Pearson's correlation coefficient was used to determine the relationship between lipid parameters and blood pressure levels. Independent t-tests were applied to compare means between hypertensive versus non-hypertensive patients, obese versus non-obese patients, and CVD versus non-CVD subgroups. The chi-square test was used to assess associations between categorical variables such as gender and obesity in relation to hypertension and dyslipidemia. A p-value of less than 0.05 was considered statistically significant. Microsoft Excel was utilized to generate graphs, tables, and visual representations of the data.

## RESULTS

The study included a sample of 103 patients, with a predominance of middle-aged males. Analysis of age and gender distribution revealed that male representation was highest in middle age, whereas the proportion of females increased in older age groups, potentially reflecting longer female life expectancy. Comparative evaluation of lipid profiles between hypertensive and non-hypertensive patients revealed statistically significant differences in multiple lipid parameters. Hypertensive individuals exhibited elevated mean total cholesterol levels (232.34 ± 34.18 mg/dL) compared to non-hypertensives (185.06 ± 34.28 mg/dL, $p < 0.001$). Similarly, triglyceride levels were higher in hypertensive patients (273.16 ± 90.76 mg/dL) versus controls (222.60 ± 59.54 mg/dL, $p = 0.004$). Mean LDL was notably raised among hypertensives (143.94 ± 30.80 mg/dL) compared to non-hypertensives (99.23 ± 38.58 mg/dL, $p < 0.001$), as was VLDL (53.16 ± 16.91 mg/dL vs. 44.49 ± 11.91 mg/dL, $p = 0.008$). The cholesterol to HDL ratio was also significantly elevated in hypertensives (5.94 ± 0.99 vs. 4.64 ± 0.89, $p = 0.001$). Although HDL levels were slightly lower in hypertensive patients (39.35 ± 8.72 mg/dL) compared to non-hypertensive ones (42.78 ± 12.98 mg/dL), the difference was not statistically significant ($p = 0.114$).

Among hypertensive individuals, further comparison between those with and without diagnosed cardiovascular disease demonstrated that CVD patients had higher total cholesterol (223.79 ± 41.03 mg/dL vs. 206.96 ± 38.89 mg/dL, $p = 0.037$) and VLDL levels (53.79 ± 16.03 mg/dL vs. 45.79 ± 14.71 mg/dL, $p = 0.01$). Other lipid parameters, including triglycerides, LDL, HDL, and cholesterol to HDL ratio, were higher in the CVD group but did not reach statistical significance. Assessment of obese versus non-obese hypertensive patients showed that obesity was associated with significantly worse lipid profiles. Obese hypertensive patients had significantly higher total cholesterol levels (227.91 ± 36.50 mg/dL) than their non-obese counterparts (201.85 ± 41.63 mg/dL, $p = 0.001$). LDL levels were also elevated in the obese group (137.21 ± 34.88 mg/dL vs. 118.27 ± 42.97 mg/dL, $p = 0.015$), as were VLDL values (54.07 ± 16.27 mg/dL vs. 45.43 ± 14.16 mg/dL, $p = 0.006$). The cholesterol to HDL ratio was notably higher in obese hypertensives (5.74 ± 1.04) compared to non-obese individuals (5.22 ± 1.20, $p = 0.021$). Triglycerides showed a borderline significant elevation (270.47 ± 81.39 mg/dL vs. 238.02 ± 86.11 mg/dL, $p = 0.053$), while HDL levels did not differ significantly ($p = 0.768$).

Pearson correlation analysis was performed to assess the relationship between blood pressure measurements (systolic and diastolic) and lipid profile parameters among hypertensive patients. The results indicated weak and statistically non-significant correlations across most parameters. Systolic blood pressure showed a slight negative correlation with triglycerides ($r = -0.307$, $p = 0.099$) and HDL ($r = -$

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0.080, p = 0.674), while its association with LDL (r = 0.211, p = 0.264) and VLDL (r = 0.109, p = 0.565) was weakly positive. Similarly, diastolic blood pressure demonstrated a modest positive correlation with LDL (r = 0.300, p = 0.107) and triglycerides (r = 0.166, p = 0.382), though these did not reach statistical significance. Overall, no lipid parameter exhibited a statistically significant linear correlation with either systolic or diastolic blood pressure. These findings suggest that while lipid abnormalities are prevalent among hypertensive patients, the direct linear relationship between individual lipid components and blood pressure may be limited or influenced by additional confounding factors such as medication history, genetic predisposition, or duration of disease.

**Table 1: Comparison of lipid levels between hypertensive patients and healthy controls (Mean ± S.D).**

| Lipid Profile Parameter | Hypertensive Patients (Mean ± SD) | Non-Hypertensive Patients (Mean ± SD) | Significance (p-value) |
|---|---|---|---|
| Total Cholesterol (mg/dL) | 232.34 ± 34.18 | 185.06 ± 34.28 | < 0.001 |
| Triglyceride (mg/dL) | 273.16 ± 90.76 | 222.60 ± 59.54 | 0.004 |
| HDL (mg/dL) | 39.35 ± 8.72 | 42.78 ± 12.98 | 0.114 |
| LDL (mg/dL) | 143.94 ± 30.80 | 99.23 ± 38.58 | < 0.001 |
| VLDL (mg/dL) | 53.16 ± 16.91 | 44.49 ± 11.91 | 0.008 |
| Cholesterol/HDL Ratio | 5.94 ± 0.99 | 4.64 ± 0.89 | 0.001 |

**Table 2: Comparison of lipid levels between hypertensive CVD patients and hypertensive non-CVD patients (Mean ± S.D).**

| Lipid Profile Parameter | CVD Patients (Mean ± SD) | Non-CVD Patients (Mean ± SD) | Significance (p-value) |
|---|---|---|---|
| Total Cholesterol (mg/dL) | 223.79 ± 41.03 | 206.96 ± 38.89 | 0.037 |
| Triglycerides (mg/dL) | 269.12 ± 80.28 | 239.70 ± 88.01 | 0.079 |
| HDL (mg/dL) | 40.65 ± 10.42 | 40.36 ± 10.56 | 0.89 |
| LDL (mg/dL) | 133.35 ± 39.22 | 123.04 ± 39.88 | 0.191 |
| VLDL (mg/dL) | 53.79 ± 16.03 | 45.79 ± 14.71 | 0.01 |
| Cholesterol/HDL Ratio | 5.64 ± 1.08 | 5.34 ± 1.21 | 0.185 |

**Table 3: Comparison of lipid levels between hypertensive obese patients and hypertensive non obese patients (Mean ± S.D).**

| Lipid Parameter | Obese (Mean ± SD) | Non-Obese (Mean ± SD) | p-value |
|---|---|---|---|
| Total Cholesterol (mg/dL) | 227.91 ± 36.50 | 201.85 ± 41.63 | 0.001 (Significant) |
| Triglycerides (mg/dL) | 270.47 ± 81.39 | 238.02 ± 86.11 | 0.053 (Borderline) |
| HDL (mg/dL) | 40.24 ± 8.39 | 40.86 ± 12.60 | 0.768 (Not Significant) |
| LDL (mg/dL) | 137.21 ± 34.88 | 118.27 ± 42.97 | 0.015 (Significant) |
| VLDL (mg/dL) | 54.07 ± 16.27 | 45.43 ± 14.16 | 0.006 (Significant) |
| Cholesterol/HDL Ratio | 5.74 ± 1.04 | 5.22 ± 1.20 | 0.021 (Significant) |

**Table 4: Pearson correlation analysis between blood pressure (systolic and diastolic) and lipid profile parameters**

| Lipid Parameter | r (Systolic BP) | p-value (Systolic BP) | r (Diastolic BP) | p-value (Diastolic BP) |
|---|---|---|---|---|
| Total Cholesterol | -0.011 | 0.955 | 0.032 | 0.868 |
| Triglycerides | -0.307 | 0.099 | 0.166 | 0.382 |
| HDL | -0.080 | 0.674 | 0.097 | 0.611 |
| LDL | 0.211 | 0.264 | 0.300 | 0.107 |
| VLDL | 0.109 | 0.565 | 0.031 | 0.870 |

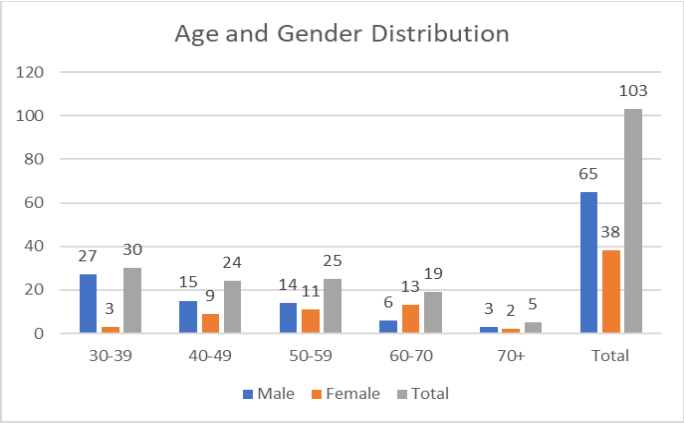


*Figure 2 Age and Gender Distribution*

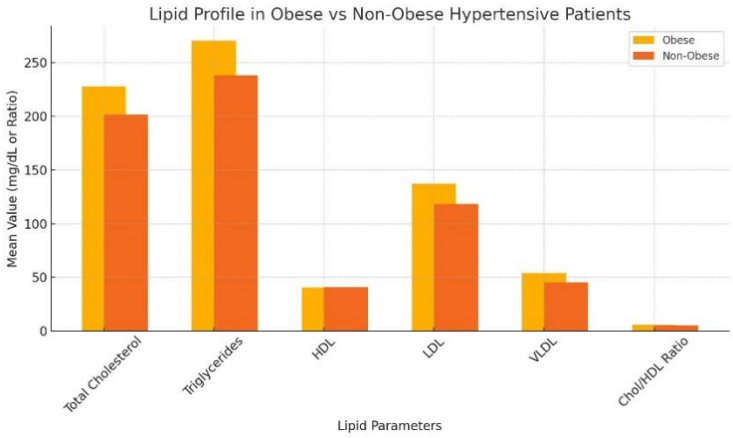


*Figure 3 Lipid Profile in Obese vs Non-Obese Hypertensive Patients*

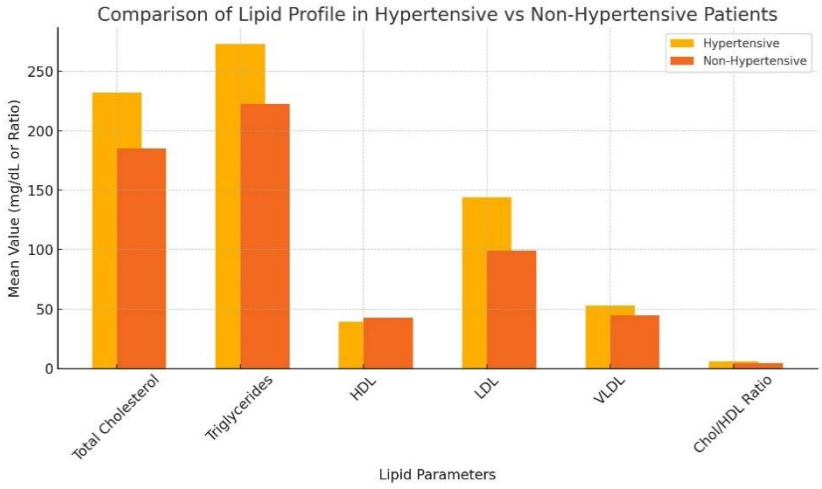


*Figure 1Comparison of Lipid Profile in Hypertensive vs Non-Hypertensive Patients*

## DISCUSSION

The findings of this study provide compelling evidence that hypertension is closely associated with dyslipidemia, particularly in patients with established cardiovascular disease and obesity. Significant elevations in total cholesterol, LDL, and VLDL levels among hypertensive patients underscore the atherogenic potential of combined lipid and blood pressure abnormalities. These results align with prior investigations that have linked visceral obesity with dyslipidemia through mechanisms involving increased free fatty acid flux and hepatic triglyceride production (13). The presence of small, dense LDL particles, though not directly assessed in this study, likely contributed to the observed elevation in LDL, which has been previously implicated in heightened atherosclerotic risk. Despite the consistent increase in atherogenic lipid components, HDL levels in this study did not significantly differ between hypertensive and non-hypertensive individuals. This contrasts with previous studies that have highlighted reductions in HDL as part of metabolic syndrome profiles (14,15). The lack of significant variation may reflect functional rather than quantitative deficiencies in HDL, as supported by emerging evidence that HDL cholesterol efflux capacity, rather than absolute plasma concentration, is more predictive of cardiovascular outcomes. The notably higher cholesterol/HDL ratio observed in hypertensive patients further reinforces the presence of dysfunctional lipid transport, indicating a shift toward a more atherogenic profile even when HDL levels appear within normal range (16,17).

The elevated triglyceride and VLDL levels observed in hypertensive patients are particularly notable in the context of residual cardiovascular risk. Even in the presence of LDL-lowering interventions, remnant cholesterol and triglyceride-rich lipoproteins have

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been recognized as contributors to ongoing atherogenesis. This study supports the view that a comprehensive lipid management strategy should address not only LDL-C but also VLDL and triglyceride levels, particularly in hypertensive populations where endothelial dysfunction and inflammation may amplify lipid-induced vascular injury (18,19). A comparison between hypertensive patients with and without diagnosed cardiovascular disease revealed that total cholesterol and VLDL were significantly higher in the CVD group, suggesting that these parameters may serve as better early indicators of cardiovascular complications than LDL alone (20). Although LDL levels were also elevated in CVD patients, the lack of statistical significance in this subset points to the possible role of qualitative differences in LDL particles—such as density and oxidation susceptibility—which were not evaluated in this study. These findings call attention to the need for more advanced lipid profiling techniques in clinical settings, especially among high-risk hypertensive patients.

The role of obesity in worsening lipid profiles was also clearly demonstrated, with obese hypertensive individuals showing significantly higher total cholesterol, LDL, and VLDL levels than their non-obese counterparts. This confirms the synergistic impact of adiposity and hypertension on lipid metabolism and cardiovascular risk. It also emphasizes the importance of incorporating weight management as a core component of therapeutic strategies aimed at controlling both blood pressure and dyslipidemia (21,22). This study's strength lies in its multi-center design and comprehensive assessment of lipid parameters in the context of blood pressure and comorbid cardiovascular disease. However, certain limitations must be acknowledged. The retrospective nature of the study may introduce information bias due to reliance on medical records. The use of non-probability sampling limits the generalizability of findings, and the absence of data on lipid particle size, insulin resistance, or inflammatory markers restricts a more nuanced interpretation of the metabolic interactions. Additionally, the study did not account for dietary patterns, medication adherence, or socioeconomic factors, all of which may significantly influence both blood pressure and lipid levels.

Future research should consider longitudinal designs to assess causality and progression, as well as incorporate advanced lipid testing and functional HDL assessment. Integrating genetic and epigenetic data could further elucidate the mechanisms linking hypertension and dyslipidemia. Larger sample sizes and inclusion of diverse ethnic and socioeconomic groups will enhance the applicability of findings to broader populations. In conclusion, the study reinforces the interconnected nature of hypertension and dyslipidemia, highlighting their collective contribution to cardiovascular risk. Early identification and management of these co-existing conditions, with particular attention to VLDL and triglycerides, may improve patient outcomes. A more holistic approach that integrates lifestyle interventions, pharmacotherapy, and possibly emerging biomarkers is essential to mitigate the burden of cardiovascular disease in hypertensive populations.

## CONCLUSION

This study concludes that hypertension is strongly associated with dyslipidemia, with hypertensive individuals—particularly those with cardiovascular disease or obesity—exhibiting more severe lipid abnormalities. These findings reinforce the importance of early lipid screening and integrated management in hypertensive patients to reduce cardiovascular risk. The study highlights obesity as a key modifiable factor that exacerbates lipid derangement, emphasizing the critical role of weight control and lifestyle interventions. Although HDL levels did not show significant variation, the overall lipid imbalance observed supports a comprehensive approach to lipid lowering in hypertensive populations. The interconnection between hypertension, dyslipidemia, cardiovascular disease, and obesity underscores the need for proactive, targeted prevention strategies and calls for future research involving larger cohorts to inform individualized care and long-term risk reduction.

**AUTHOR CONTRIBUTION**

| Author | Contribution |
|---|---|
| M. Saim Qasim* | Substantial Contribution to study design, analysis, acquisition of Data<br>Manuscript Writing<br>Has given Final Approval of the version to be published |
| Shahid Sultan* | Substantial Contribution to study design, acquisition and interpretation of Data<br>Critical Review and Manuscript Writing<br>Has given Final Approval of the version to be published |
| Tasra Bibi | Substantial Contribution to acquisition and interpretation of Data<br>Has given Final Approval of the version to be published |

| Author | Contribution |
|---|---|
| Muhammad Mushtaq | Contributed to Data Collection and Analysis<br>Has given Final Approval of the version to be published |
| Usama Abid | Contributed to Data Collection and Analysis<br>Has given Final Approval of the version to be published |
| Sheraz Ali | Substantial Contribution to study design and Data Analysis<br>Has given Final Approval of the version to be published |
| M. Tayyab | Contributed to study concept and Data collection<br>Has given Final Approval of the version to be published |
| Gulam Mustafsa | Writing - Review & Editing, Assistance with Data Curation |
| Ahram Hussain | Writing - Review & Editing, Assistance with Data Curation |
| Muhammad Faizan | Writing - Review & Editing, Assistance with Data Curation |

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