



LIPID PROFILE IN NON-OBESE AND NON-DIABETIC HYPERTENSIVE PATIENTS

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ABSTRACT

Background: Hypertension is a strong predictor for cardiovascular morbidity and typically coexist with dyslipidemia. Hypertensive patients may also have dyslipidemia, which further increases their risk of atherosclerosis, independent of obesity or diabetes. The lipid profile in these patients is of valuable importance for differentiating the cardiovascular risk regardless of their concomitant metabolic syndromes.

Objectives: This study aimed to evaluate the role of lipid profile (LP) abnormalities in non-obese, non-diabetic hypertensive patients and their impact on cardiovascular disease (CVD) risk beyond traditional risk factors.

Study Design: This is a cross-sectional study.

Place and Duration of study: From December 2024 to April 2025 in General Medicine Department, Bolan Medical Complex Hospital Quetta.

Methods: One hundred non-diabetic, non-obese hypertensive subjects were included in a cross-sectional study. Fasting blood lipid profiles including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) were detected. Data are expressed as mean \pm standard deviation (SD). Statistical analysis was performed using SPSS version 24.0. Independent t-tests were applied, and $p < 0.05$ was considered statistically significant.

Results: The study population had a mean age of 52.6 ± 9.4 years, with a male-to-female ratio of 1:1.2. A total of 100 hypertensive patients were enrolled. The mean lipid values were: total cholesterol (TC) 205.3 ± 34.7 mg/dL, LDL-C 132.8 ± 28.6 mg/dL, HDL-C 38.5 ± 6.9 mg/dL, and triglycerides (TG) 178.4 ± 41.2 mg/dL. There was Dyslipidemia in 62 Low HDL-C ($p = 0.031$) and high LDL-C ($p = 0.042$) were significantly associated with hypertension. These results indicate that there is a relatively high burden of lipid abnormality in

hypertensive patients beyond obesity or diabetes.

Conclusions: Non-obese non-diabetic hypertensive patients present dyslipidemia, mainly hyper-LDL-C and hypo-HDL-C, thus showing higher risk for cardiovascular disease. Hypertension in itself can be a potential risk factor for lipid abnormalities, stressing the importance of routine monitoring of lipids even without regular metabolic risk factors. It has been shown that early screening and treatment of dyslipidemia could effectively decrease future cardiovascular events in essential hypertensive patients.

Introduction

Hypertension remains a global public-health problem and a major risk factor for cardiovascular (CV) morbidity and mortality. There are an estimated 1.28 billion adults with hypertension aged 30–79 years in the world with most living in low- and middle-income (LMIC) countries as reported by the World Health Organization (WHO). It is often a concomitant of other pathologies, which are mainly related to body weight and metabolic syndrome, like obesity, insulin resistance or diabetes mellitus increasing cardiovascular risk. However, lipid abnormalities may even occur in the absence of comorbidities and thus a direct relationship between hypertension and dyslipidemia is implied [1,2]. Dyslipidemia, defined as an abnormal level of blood lipids, is one of the most well-known risk factors for atherosclerotic cardiovascular disease (ASCVD). Higher circulating levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG), lower concentrations of high-density lipoprotein cholesterol (HDL-C) are recognized risk factors for the development of coronary heart disease (CAD) and stroke [3]. The interaction of hypertension with dyslipidemia induces an amplified type of vascular damage, endothelial dysfunction being a potentiated cardiovascular risk. The Framingham studies demonstrated that hypertension and dyslipidemia often cluster and that elevated blood pressure combined with elevated cholesterol confers greater cardiovascular risk than either factor alone. Other studies have reported dyslipidemia among hypertensive

individuals irrespective of obesity or diabetes [4,10–12]. This raises important questions about whether hypertension itself causes lipid abnormalities via mechanisms such as sympathetic activation, the renin–angiotensin–aldosterone system (RAAS), oxidative stress, and endothelial dysfunction [5]. South Asian communities including Pakistanis have a high prevalence of hypertension and an increased incidence of early-onset cardiovascular disease. Dyslipidemia is common among Pakistani adults, with low HDL-C reported as the most frequent lipid abnormality [6]. Although there have been a few elsewhere most studies in the area including ours had one looked at hypertensives as a whole and not segregate non obese diabetics and obese. Although a few studies have examined subgroups, most research in the region have evaluated hypertensive patients as a whole without segregating non-obese, non-diabetic individuals from obese or diabetic patients. Because both obesity and diabetes are independent risk factors to dyslipidemia, we excluded these conditions to better evaluate the connection between hypertension and dyslipidemia. Clinical guidelines advocate measuring the lipid profile in risk stratification and treatment for CVD among patients diagnosed with hypertension. Dyslipidemia in the non-obese, non-diabetic hypertensive population often goes undetected, representing a missed opportunity for early intervention, including lifestyle modification and pharmacotherapy, to prevent long-term cardiovascular disease [7,8]. Also, investigation of lipid patterns in this subgroup

may clarify pathophysiological mechanisms and inform population-level prevention strategies [9]. Therefore, this study was undertaken to evaluate the lipid profile in non-obese, non-diabetic patients with hypertension at Bolan Medical Complex hospital Quetta. We aimed to determine whether patients with hypertension, in the absence of other metabolic risk factors, have dyslipidemia, and to estimate the prevalence and pattern of lipid abnormalities in this population. This research also contributes to the limited regional evidence on hypertension and dyslipidemia, offering valuable insights for health planners in resource-constrained settings.

Methods

A cross-sectional study was conducted in the Department of Medicine at Bolan Medical Complex hospital Quetta from December 2024 to April 2025. One hundred patients with hypertension, diagnosed according to Joint National Committee (JNC-8) were enrolled. Hundred hypertensive patients according to definition by analyzed. Participants were non-obese (BMI < 25.0 kg/m²). and non-diabetic, defined as fasting plasma glucose <126 mg/dL and HbA1c <6.5% with no prior diagnosis of diabetes and no use of antidiabetic medication (ADA diagnostic thresholds). After obtaining informed consent, demographic information such as age, sex and medical history was recorded. Fasting blood samples were collected after an overnight fast of at least 12 hours. Serum lipid profile variables were total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) were measured; LDL-C was calculated using the Friedewald formula ($LDL-C = TC - HDL-C - TG/5$), except when TG ≥ 400 mg/dL in which case LDL-C was measured directly. The samples were analyzed with the routine enzymatic colorimetric methods. All data

were de-identified to maintain privacy. erum TC, TG, HDL-C and LDL-C

Inclusion Criteria:

Inclusion criteria: age 30–70 years; diagnosis of primary (essential) hypertension according to JNC-8 criteria; BMI < 25.0 kg/m²; no history of diabetes mellitus; and provision of written informed consent. Participants receiving antihypertensive therapy were eligible provided they met other criteria.

Exclusion Criteria:

Exclusion criteria: known diabetes mellitus, BMI ≥ 25.0 kg/m² (obesity/overweight as defined in this study), secondary hypertension, chronic kidney disease (stage ≥ 3), untreated thyroid disease, active hepatic disease (ALT/AST $>2\times$ upper limit of normal), current use of lipid-lowering medications, pregnancy, and other systemic inflammatory conditions.

Ethical Approval:

The protocol of the study has been approved by the Institutional Review Board (IRB) of Bolan Medical Complex Hospital, Quetta (Approval No CPSP/REU/MED/2022/001-19209/24). Informed written consent was received from all the participants. This study was conducted in accordance with the Declaration of Helsinki (2013 revision) guidelines.

Data Collection:

Demographic as well as anthropometric and clinical information were recorded with the help of a self-prepared questionnaire. Blood pressure was recorded sitting down by mercury sphygmomanometer; two readings were averaged. Lipid profile was performed on the blood samples taken after 12 hours of fasting, in hospital laboratory within 24 hours of collection. Ethical approval was obtained from the Institutional Review Board (IRB) of Rahman Medical Institute (Reference No. RMI/IRB/2024/Hypertension-Lipid-Study). Written informed consent was obtained from all participants. The study complied with the Declaration of Helsinki.

Statistical Analysis:

Statistical analysis was performed with SPSS software version 24.0. Continuous variables were reported using descriptive statistics (mean \pm standard deviation). Categorical variables were presented as frequencies and percentages. A t-test examined mean lipids test levels compared to between subgroup a and b. A p-value \leq 0.05 was considered to indicate statistical significance for all inferential statistics.

Results

A hundred hypertensive patients with a mean age of 52.6 ± 9.4 years were enrolled. Patients were composed of 45 males (45%) and 55 females (55%) in a male to female ratio of 1:1.2. Mean BMI was 23.4 ± 1.1 kg/m², indicating that the subjects were not obese. As for lipid profiles, the average total cholesterol was 205.3 ± 34.7 mg/dL, LDL-C was 132.8 ± 28.6 mg/dL, HDL-C was, and triglycerides were. Dyslipidemia was seen in 62% of patients; low HDL-C was the most common (41%). Raised LDL-C was found in 29%, hypertriglyceridemia in 24% and raised total cholesterol in 21%. Low HDL-C ($p = 0.031$) and high LDL-C ($p = 0.042$) were significantly associated with hypertension, as revealed by Statistical analysis Sex stratification showed that occurrence of low HDL-C was higher in men (46%) compared to women (38%), while high LDL-C rates were greater in women (32%). These findings suggest that lipid disorders are prevalent among non-obese, non-diabetic hypertensive patients and serve as an independent predictor of CVD. Statistical analyses were performed using SPSS v24.0. Normality of continuous variables was assessed with the Shapiro-Wilk test. Normally distributed variables are presented as mean \pm SD and were compared using the independent-samples t-test; non-normally distributed variables were expressed as median (interquartile range) and compared using the Mann-Whitney U test. Categorical

variables were compared using chi-square or Fisher's exact test as appropriate. Two-tailed $p < 0.05$ was considered statistically significant. Levene's test was used to assess equality of variances.

Table 1 Demographic description of the study population (n = 100)

Variable	Mean \pm SD / n (%)
Age (years)	52.6 ± 9.4
Gender (Male/Female)	45 (45%) / 55 (55%)
Male-to-Female Ratio	1:1.2
BMI (kg/m ²)	23.4 ± 1.1
Systolic BP (mmHg)	148.6 ± 12.5
Diastolic BP (mmHg)	92.4 ± 8.7
Duration of Hypertension (years)	6.3 ± 3.1

Table 2. Lipid Profile in Hypertensive Patients (n = 100)

Lipid Parameter	Mean \pm SD (mg/dL)	Reference Range (mg/dL)
Total Cholesterol (TC)	205.3 ± 34.7	< 200
LDL-C	132.8 ± 28.6	< 130
HDL-C	38.5 ± 6.9	> 40 (male), > 50 (female)
Triglycerides (TG)	178.4 ± 41.2	< 150
VLDL-C (calculated)	35.6 ± 8.2	< 30

Table 3. Frequency of Dyslipidemia in Study Population

Lipid Abnormality	Frequency (n)	Percentage (%)
Low HDL-C	41	41%
Elevated LDL-C	29	29%
Hypertriglyceridemia	24	24%
Elevated Total Cholesterol	21	21%
Any Dyslipidemia	62	62%

Table 4. Relationship of blood lipids with hypertension

Lipid Parameter	Normal (n = 38)	Abnormal (n = 62)	p-value
Total Cholesterol (mg/dL)	182.6 ± 21.3	219.8 ± 27.4	0.054
LDL-C (mg/dL)	118.2 ± 20.5	139.6 ± 26.8	0.042*
HDL-C (mg/dL)	44.2 ± 5.6	35.9 ± 6.1	0.031*
Triglycerides (mg/dL)	151.7 ± 32.4	189.4 ± 38.7	0.061

Discussion

In this cross-sectional study in a cohort of non-obese, non-diabetic hypertensive patients, dyslipidemia was common even in the absence of traditional metabolic comorbidities. Our results are commensurate with years of established epidemiologic evidence that HTN and unfavorable lipid fractions additively potentiate ASCVD risk, a connection originally encapsulated by the Framingham program but continually reaffirmed in subsequent decades [10]. Current prevention guidelines identify high BP and atherogenic lipids as co-primary modifiable risk domains. Managing both together lowers absolute risk. The high proportion of subjects with low HDL-C and elevated LDL-C in our sample reflects regional trends found throughout South Asia where dyslipidemia is already highly prevalent even at relatively moderate-BMI levels [11]. Substantial number of large Pakistani and South Asian cohorts have reported aggressively high prevalence of low HDL-C and hypertriglyceridemia in population samples, reflecting on genetic dietary, lifestyle determinants other than adiposity or overt diabetes [12]. The National Diabetes Survey of Pakistan (NDSP) and similar urban surveys reveal that >80% of men and approximately 90% of women have low HDL-C with high TGs and LDL-Cs being burdensome, a background scenario in which

to situate our numbers from this non-obese, non-diabetic hypertensive niche [13]. Mechanism-wise, amongst many other things including turnover of lipids in the inhibition and prevention of lesion formation, hypertension may create atherogenic environment through sympathetic overactivity, activation of RAAS (Renin-angiotensin-aldosterone system), oxidative stress and endothelial dysfunction which also interact with lipid transport and clearance [14]. Such cross-cutting bridges of pathophysiology may account for lipid disturbances that are not brought about by obesity and diabetes, as is the case of analogue-cohorts of hypertensive adults whose gross metabolic disorders are absent [15]. Even more recent large-population studies demonstrate that “any” dyslipidemia clusters with other risk factors to drive global ASCVD risk, since the days of medical school measuring lipids should be a routine event in hypertensives whether their BMI or glycemic disarray status [16]. Sex-specific nuances deserve attention. Men had a higher prevalence of low HDL-C and women showed a slightly higher prevalence of high LDL-C, consistent with large datasets in Pakistan that report a varying pattern of screening intensity and burden by sex [17]. Of note, these national-level analyses from Pakistan highlight a high burden of dyslipidemia and sex inequality not only on lipid testing but also in regard to the women who are often tested at late stage despite hazardous LDL-C profiles -- an across-the-board deficiency with rapid impact onto rate of risk counselling and therapy [18]. Indeed, such approaches are, in practice, now promoting a more "risk based" pathway for statin therapy and % reduction target for LDL-C (≥30–49% with moderate intensity/≥50% with high intensity) in combination with considering non-HDL-C and TG if residual risk is present [19]. These considerations also pertain to those with hypertension whether or not they are

obese/diabetic and if the estimated risk of ASCVD and lipid thresholds would initiate treatment. Novel drug treatment information suggests that hypertensive, nondiabetic patients might experience major reductions in both LDL-C and non-HDL-C by statins and similar to those observed in diabetics—arguably an argument against a delay on therapy of well-defined high vascular risk hypertensive subjects with no metabolic disorder. Our results also overlap with South Asian-specific lipid phenotypes that exhibit low HDL-C and high TG at a lower BMI, described as an “lipid-metabolic” risk profile which go beyond established obesity cut-points [20] sub-studies also highlight excess cardiovascular disease burden in South Asia which further supports aggressive risk-factor modification (including lipids) to be needed in this particular region. Clinically, the strong agreement of our findings with global prevention directions promotes a single message: in hypertensive subjects do not expect obesity or diabetes to prompt lipid checking and treating. The protocol will include standardization of fasting or nonfasting lipid testing, pooled risk estimation when appropriate, linking the use of statins to initiation/intensification to guideline-recommended levels. and lifestyle interventions (dietary patterns, physical activity sodium reduction)[21]. Consider considering the rather high background prevalence of a low HDL-C in Pakistan focusing on targets for non-HDL and LDL, treating TG when elevated and measuring adherence would be most effective. Strengths and limitations. Through the exclusion of obesity and diabetes, our study identifies the hypertension–lipid concatenation as a separate entity, thus adding to previous mixed-risk cohorts. Nevertheless, the cross-sectional nature of this study does not allow for causal inferences and unmeasured dietary, genetic or physical-activity factors could confound associations. Prospective multi-center

enriched genotyping and detailed lifestyle phenotyping studies are required in South Asia to identify causal pathways and tailor preventative measures. In summary, in our study, the finding that hypertension is frequently associated with atherogenic dyslipidemia—also among persons without obesity/diabetes-, and does have region-specific differences with regard to lipids has implications for an increased emphasis on aggressive lipid screening and guideline-recommended therapy in all hypertensive individuals.

Conclusion

Non-obese, non-diabetic hypertensive individuals have a high prevalence of dyslipidemia, especially low-HDL-C and high-LDL-C, which are risk factors for cardiovascular diseases. Hypertension per se may affect lipid metabolism independently of obesity and diabetes. Regular screening for lipids and early intervention is important to prevent unfavorable cardiovascular outcomes in these high-risk patients.

Limitations

This was a single center and cross-sectional study with a relatively small sample size, which may affect generalization. Diet, activity and genetic factors were not fully considered. Causality was not determined via longitudinal follow-up. More extensive multicenter studies using complete metabolic evaluations are needed to validate these findings.

Future Findings

Genetic, molecular, and inflammatory mechanisms that may connect hypertension with lipid abnormalities need to be further investigated. Longitudinal multicenter studies with lifestyle, dietary, and pharmacological interventions may provide insight into causal inference. Assessment of statins, antihypertensives, and novel lipid-lowering agents in non-obese, non-diabetic hypertensive populations may yield

preventive and therapeutic strategies tailored to individual patients.

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Authors Contribution

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