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Lipid Profile in Drug Naive Hypertensive Patients visiting Cardiology OPD in a Central Hospital

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ABSTRACT

Background: Hypertension and dyslipidemia are two major risk factors for cardiovascular disease and often coexist and share common pathophysiological mechanisms. Their coexistence has a multiplicative adverse effect on cardiovascular risk. Understanding the lipid profile in drug-naive hypertensive patients is important for effective management and prevention of cardiovascular complications. The objective of the study was to assess the lipid profile in drug-naive hypertensive patients visiting the Cardiology Outpatient Department (OPD) of a central hospital.

Methods: This was a prospective observational study conducted at the Cardiology OPD of Bir Hospital, National Academy of Medical Sciences, Kathmandu, Nepal. A total of 113 consecutive drug-naive hypertensive patients were enrolled. Demographic data including age, gender, and ethnicity were recorded. Blood pressure measurements were recorded and fasting lipid profiles (total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides) were collected in the subsequent visit. Data were analyzed using SPSS version 23.

Results: The study included 53 males (46.9%) and 60 females (53.1%). The mean age distribution showed 38.1% young adults, 46.9% middle-aged, 13.3% elderly, and 1.8% very elderly patients. Mean systolic blood pressure was 157.62±11.755 mmHg and diastolic blood pressure was 96.73±10.556 mmHg. The mean values for lipids were- total cholesterol 184.77±41.51 mg/dL, HDL cholesterol 47.32±22.35 mg/dL, LDL cholesterol 111.84±38.49 mg/dL and triglycerides 165.20±78.60 mg/dL. A large proportion of patients had abnormal lipid levels, with elevated triglycerides and borderline HDL being the common pattern.

Conclusion: A large proportion of drug-naive hypertensive patients demonstrated dyslipidemia, specifically elevated triglycerides and borderline HDL cholesterol. The coexistence of hypertension and dyslipidemia increases cardiovascular risk many-fold and requires comprehensive management strategies. Early detection and aggressive treatment of both conditions are essential to prevent future cardiovascular events.

Keywords: hypertension; dyslipidemia; lipid profile; cardiovascular disease.

INTRODUCTION

Hypertension is the leading cause of cardiovascular disease and premature death worldwide affecting more than one billion people globally.[1] The prevalence of hypertension continues to rise due to population aging and increased exposure to unhealthy lifestyle measures, including physical inactivity and unhealthy diets.[2] In parallel, dyslipidemia has emerged as another major contributor to the growing burden of cardiovascular disease, particularly in low to middle income countries (LMIC).[3] The coexistence of hypertension and dyslipidemia,

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termed "lipitension" by some, is a stronger foe of mankind, with prevalence ranging from 15% to 31% across various populations.[4] These two conditions share common pathophysiological mechanisms that incorporates endothelial dysfunction, oxidative stress, and inflammation. Importantly, their coexistence produces multiplicative effect on cardiovascular risk. The Multiple Risk Factor Intervention Trial (MRFIT), which followed 356,222 men for 12 years, demonstrated that even mild-to-moderate elevations in both blood pressure and cholesterol levels had more than additive adverse impact on coronary heart disease risk.[5] Hypertension is frequently associated with alterations in lipid metabolism, including elevated triglyceride levels, reduced HDL cholesterol, and increased LDL cholesterol.[6] These metabolic abnormalities appear to develop early in life in individuals with genetic predisposition to hypertension. Borderline hypertensive individuals, when compared with normotensive subjects, demonstrate higher cholesterol levels, elevated triglycerides, lower HDL cholesterol, and increased insulin levels.[7]

The pathophysiological interplay between hypertension and dyslipidemia is multifaceted. The renin-angiotensin-aldosterone system (RAAS) promotes atherogenesis, with angiotensin II stimulating the AT1 receptor and thus increasing lipid uptake in cells and causing vasoconstriction and free radicals production that foster both hypertension and atherosclerosis. Conversely, dyslipidemia causes endothelial damage and loss of vasomotor activity that manifests as elevated blood pressure.[8] Understanding the lipid profile in hypertensive patients is crucial for comprehensive cardiovascular risk assessment and management. Both parameters are incorporated into cardiovascular disease (CVD) risk calculators for estimating 10-year risk of cardiovascular events. Despite the well-established relationship between hypertension and dyslipidemia, limited data exist from Nepal regarding the lipid patterns in drug-naive hypertensive patients. This study aimed to investigate the lipid profile of drug-naive hypertensive patients presenting to the Cardiology OPD of a central tertiary care hospital in Kathmandu, Nepal.

METHODS

This was a prospective observational study conducted at the Cardiology Outpatient Department of Bir Hospital, National Academy of Medical Sciences (NAMS), Kathmandu, Nepal. The study was conducted over a six-month period from May 2025 to October 2025. Sample size was calculated based on the prevalence of abnormal lipids (35%) in South Asian populations[9] keeping the confidence interval at 95% and margin of error at 10%. All drug-naïve comers above 18 years of age presenting to the Cardiology Department with established- or incidental finding of hypertension were enrolled in the study. Patients already on lipid lowering drugs and with significant comorbidities were excluded from the study. A total of 113 patients were included in the study. Hypertension was defined as office systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, according to European Society of Cardiology (ESC)/European Society of Hypertension (ESH) guidelines.[10] Dyslipidemia was defined as abnormal levels of lipids in the blood characterized by total cholesterol >200 mg/dL, HDL cholesterol <40 mg/dL in males or <50 mg/dL in females, LDL cholesterol >100 mg/dL and triglycerides >150 mg/dL in isolation or in combination[11]. Patient information was collected through structured interviews and clinical examinations. Demographic details including age, gender, and ethnicity were recorded. Blood pressure was measured using standardized protocols in the OPD setting. Blood samples for lipid profile analysis were obtained after a minimum of nine hours of fasting. Serum total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were measured using standard laboratory methods. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula: LDL-C = Total Cholesterol - HDL-C -(Triglycerides/5).

Data were analyzed using IBM Statistical Package for the Social Sciences (SPSS) version 20.

Continuous variables were presented as mean ± standard deviation (SD). Categorical variables were presented as frequencies and percentages. Statistical significance was set at p-value<0.05. The study protocol was approved by the Institutional Research Board of NAMS, Bir Hospital. Written informed consent was obtained from all participants prior to enrollment. The study purpose and procedures were clearly explained to all participants. Confidentiality of collected information was maintained throughout the study. Participants were informed of their right to withdraw from the study at any time without affecting their medical care.

RESULTS

A total of 113 drug-naive hypertensive patients were enrolled in the study. The study population comprised 53 males (46.9%) and 60 females (53.1%), showing a slightly higher female representation. The ethnic distribution showed predominance of Brahmin (29.2%), Baishya (27.4%), and Kshetry (23.9%) communities, reflecting the demographic composition of the patients visiting to Bir Hospital. The majority of patients were middle-aged (46.9%) followed by young adults (38.1%), indicating that hypertension affects individuals predominantly during their productive years. The demographic distribution is summarized in Table 1.

Table 1. Demographic profiles of patients.

Variables	Frequency (%)
Mean age (years) ± SD	
Total population	49.96 ±12.74
Male	49.08 ±13.40
Female	50.73 ± 12.20
Age Groups (years)	22 - 82
Adults (18 – 44)	43 (38.1%)
Middle-aged (45 – 64)	53 (46.9%)
Elderly (65 – 79)	15 (13.3%)
Very elderly (≥80)	2 (1.8%)
Gender	
Male	53 (46.9%)
Female	60 (53.1%)
Ethnicity	
Brahmin	33 (29.2%)
Kshetry	27 (23.9%)
Baishya	31 (27.4%)
Shudra	6 (5.3%)
Madheshi	9 (8.0%)
Others	7 (6.2%)

The mean systolic blood pressure was 157.62±11.755 mmHg, and the mean diastolic blood pressure was 96.73±10.556 mmHg. These values indicate that the majority of patients presented with stage 1 or stage 2 hypertension (Table 2).

Table 2. Mean blood pressure of patients.

Parameter	Systolic BP (mmHg)	Diastolic BP (mmHg)
Mean	157.62±11.75	96.73±10.55
Minimum	140	70
Maximum	200	130

Systolic blood pressure showed variation across age groups, with the elderly group displaying the highest mean value (163.20±16.12 mmHg), while young adults had the lowest (154.74±12.58 mmHg). Inter-group variation was, however, not statistically significant (0.095). Conversely, DBP showed an inverse age relationship, with young adults exhibiting the highest values (98.33±10.50 mmHg) and elderly patients the lowest (90.00±12.53 mmHg) The variation in DBP across age-groups was, however, statistically significant (p-value=0.039) (Table 3).

Table 3. Blood pressure as per age-groups.

Age groups	Systolic BP	p- value	Diastolic BP	p- value
Young adults	154.74±12.58	0.095	98.33±10.50	0.039
Middle-aged	158.47±9.04		97.60±9.33	
Elderly	163.20±16.12		90±12.53	
Very elderly	155±7.07		90±14.14	

Regarding ethnicity, the Shudra group exhibited the highest mean systolic (160 ± 20 mmHg) and diastolic (103.33±10.32 mmHg) blood pressure values, while the 'Others' showed the lowest values for both measurements. Inter-ethnic blood pressure variations did not achieve statistical significance (Table 4).

Gender analysis revealed significantly higher SBP in females (160.92±12.18 mmHg) compared to males (153.89 ± 10.12 mmHg; p=0.001). No significant gender difference was observed for DBP (p-value=0.207) (Table 5).

Table 4: Blood pressure as per ethnicity.				
Ethnicity	**SBP**	**p- value**	**DBP**	**p- value**
Brahmin	155.55±8.66	0.466	96.39±11.18	0.494
Kshetry	157.78±11.46		96.89±11.01	
Baishya	159.58±11.84		97.55±8.85	
Shudra	160±20		103.33±10.32	
Madheshi	161±17.30		93.33±14.14	
Others	151.71±8.28		92.86±7.55	

Table 5. Blood pressure as per gender.				
Gender	**SBP**	**p- value**	**DBP**	**p- value**
Male	153.89±10.12	0.001	95.40±10.45	0.207
Female	160.92±12.18		97.92±10.59	

Mean total cholesterol (TC) was 184.77 ± 41.51 mg/dL, HDL cholesterol (HDL-C) was 47.32 ± 22.35 mg/dL, LDL cholesterol (LDL-C) was 111.84 ± 38.49 mg/dL, and triglyceride (TG) was 165.20 ± 78.60 mg/dL (Table 6).

Middle-aged patients exhibited the highest values for TC (193.97 ± 37.05 mg/dL), HDL-C (51.04 ± 28.68 mg/dL) and LDL-C (116.10 ± 36.36 mg/dL). TG (181.90 ± 87.05 mg/dL) was highest in elderly. These age-related variations did not achieve statistical significance (Table-7).

No significant inter-ethnic variations were observed for lipid parameters (Table 8).

Males exhibited significantly lower HDL-C (42.94±13.11 mg/dL) compared to females (51.17±27.64 mg/dL; p-value=0.050). Males also demonstrated higher TG levels (177.73±94.69 mg/dL) compared to females (154.13±59.63 mg/dL) but the difference was not statistically significant (p-value=0.111) (Table 9).

Table 6. Lipid profile statistics.				
Parameter	**Total Cholesterol**	**HDL Cholesterol**	**LDL Cholesterol**	**Triglycerides**
Mean±SD (mg/dL)	184.77±41.51	47.32±22.35	111.84±38.49	165.20±78.60
Range (mg/dl)	260 (88.0-348)	196 (27-223)	219 (23-242)	547 (58-605)

Table 7. Lipid profiles as per age-groups.								
Age Group	**Total cholesterol**	**p-value**	**HDL cholesterol**	**p-value**	**LDL cholesterol**	**p-value**	**Triglyceride**	**p-value**
Young adults	176.837±38.03	0.173	42.94±15.49	0.376	110.12±37.12	0.65	153.62±71.70	0.134
Middle-aged	193.97±37.05		51.04±28.68		116.10±36.36		181.90±87.05	
Elderly	175.14±61.05		46.86±9.82		102.57±51.25		136.12±56.81	
Very elderly	183.50±6.36		46±12.72		105.20±1.69		189.50±47.37	

Table 8. Lipid profiles as per ethnicity.								
Ethnicity	**Total cholesterol**	**p-value**	**HDL cholesterol**	**p-value**	**LDL cholesterol**	**p-value**	**Triglyceride**	**p-value**
Brahmin	183.63±47.41	0.85	48.63±22.14	0.62	109.98±42.	0.815	173.20±76.14	0.735
Kshetry	184.00±34.89		52.81±36.35		107,54±31.08		171.44±60.64	
Baishya	181.71±39.46		42.51±9.69		113.71±36.67		155.02±101.07	
Shudra	202.10±34.04		48.20±10.21		128.40±17.13		130.85±62.39	
Madheshi	180.11±40.26		42.77±4.73		106.11±48.99		183.77±72.92	
Others	197.74±58.29		46.28±9.82		122.02±54.99		154.00±64.24	

Table 9. lipid Profiles as per gender.								
Gender	**Total Cholesterol**	**p- value**	**HDL Cholesterol**	**p- value**	**LDL Cholesterol**	**p- value**	**Triglyceride**	**p- value**
Male	183.82±44.80	0.821	42.94±13.11	0.05	112.26±40.49	0.913	177.73±94.69	0.111
Female	185.60±38.73		51.17±27.64		111.46±36.96		154.13±59.63	

The lipid profile pattern observed in this study is consistent with the metabolic abnormalities commonly associated with hypertension and reflects the "lipitension" phenotype described in the literature.

DISCUSSION

This prospective observational study evaluated the lipid profiles of 113 drug-naive hypertensive patients who presented to the Cardiology OPD of a central tertiary care hospital in Nepal. The findings reveal important insights into the relationship between hypertension and dyslipidemia in this population. The study population showed a slight female predominance (53.1% vs. 46.9%). It differs from some international studies that report higher hypertension prevalence in males[12]. This finding may reflect healthcare-seeking behavior patterns in Nepal or may be the true reflection of demographic differences in hypertension prevalence by gender in this setting. The mean blood pressure values (157.62/96.73 mmHg) indicate that most patients presented with stage 1 or stage 2 hypertension. The relatively narrow standard deviations suggest homogeneity in the severity of hypertension at presentation.

The finding of significantly higher SBP in females (p-value=0.001) coincides with evidence that systolic blood pressure rises more steeply in aging women compared to men, potentially related to hormonal changes during menopause.[13] Estrogen deficiency has multifaceted effects leading to endothelial dysfunction, reduced nitric oxide bioavailability, increased sympathetic nervous system activity, and upregulation of the renin-angiotensin-aldosterone system.[14] Given that the mean age of female participants (50.73±12.20) approximated the typical menopausal age in South Asian populations, the observed higher SBP likely reflects the loss of estrogen-mediated cardiovascular benefits.[15] Approximately 75% of postmenopausal women in Western populations have hypertension, revealing the global occurrence of this phenomenon.[16]

The majority of patients were middle-aged (46.9%) and young adults (38.1%), highlighting that hypertension and its associated metabolic abnormalities affect individuals during their most productive years with significant socioeconomic implications. Studies confirm that SBP continues to rise after age 50 in response to age-induced arterial stiffening and DBP plateaus around age 50 and subsequently decreases.[17] The statistically significant variation in DBP across age groups (p-value=0.039) underscores the clinical importance of isolated systolic hypertension as the predominant phenotype in elderly populations.[18]

The ethnic distribution predominantly included Brahmin, Baishya, and Kshetry communities reflecting the demographic composition of the patients visiting to cardiology OPD. However, the variation of SBP and DBP across ethnicity were not statistically significant. This could be because of sharing similar lifestyles across ethnic groups. Ethnic categories in Nepal are different from others, so direct comparison could not be made with similar studies from other corners of the world.

The mean total cholesterol was 184.77±41.51 mg/dL, which falls within the desirable range (<200 mg/dL). However, the maximum value of 348.0 mg/dL indicats that a substantial proportion of patients had elevated cholesterol levels. The wide range (260.0 mg/dL) suggests considerable heterogeneity in cholesterol levels among the study population. The variation in total cholesterol was not statistically significant across gender, age and ethnicity. With regard to gender, the finding is similar to other international studies[19], however, the variations seen in other studies with age and ethnicity were not apparent in our study. This may be the result of relatively small sample size in our study.

The mean HDL-C of 47.32 mg/dL is borderline low, particularly concerning for females (normal >50 mg/dL for males, the value is >40mg/dl). Low HDL-C has emerged as the most frequent lipid abnormality among hypertensive individuals in multiple studies[20]. The variation was statistically significant across gender but not with age-groups and ethnicity. The mean LDL cholesterol was 111.84±38.49 mg/dL. This value falls within the near-optimal to borderline high range (100-129 mg/dL). However, the maximum value of 242.0 mg/dL indicates that some patients

had very high LDL cholesterol levels placing them at substantially increased cardiovascular risk. The variation of this bad cholesterol was statistically not significant across age-groups, gender and ethnicity. This finding is similar to the findings of the south African study by A. A. Akintunde, which revealed that the hypertensives tend to have higher baseline lipid levels but without significant differences across age and gender[21].

The mean triglyceride level (165.20±78.60 mg/dL) in our study exceeds the normal range (<150 mg/dL) and indicates mild hypertriglyceridemia. This finding is particularly important because elevated triglycerides are common in South Asian populations and are strongly associated with cardiovascular disease risk[22]. The maximum level was 605.0 mg/dL, which indicates that some patients had moderately to very high range of triglycerides. The combination of elevated triglycerides and low HDL cholesterol observed in this study represents an atherogenic dyslipidemia pattern. This pattern has been proven to be strongly associated with cardiovascular disease risk. Recent research from the Systolic Blood Pressure Intervention Trial (SPRINT) demonstrated that atherogenic dyslipidemia was associated with increased cardiovascular risk among adults with hypertension that is independent of whether blood pressure was controlled to intensive (<120 mmHg) or standard (<140 mmHg) targets[23].

Strengths and Limitations

Strengths

1. The study provides important baseline data on lipid profiles in drug-naive hypertensive patients from Nepal.
2. The study included a diverse ethnic representation reflecting the demographic composition of the study setting.
3. All patients were drug-naïve so that potential confounding effects of antihypertensive or lipid-lowering medications were eliminated.

Limitations

1. The cross-sectional nature of the study limits causal inference regarding the relationship between hypertension and dyslipidemia.
2. The relatively small sample size (n=113) may limit the generalizability of findings and power of the study for subgroup analyses by ethnicity or age group.
3. Other cardiovascular risk factors such as smoking, physical activity, dietary patterns, or family history were not looked into. These may influence both blood pressure and lipid profiles in the study population.
4. The study was conducted at a single tertiary care center that may introduce selection bias.
5. Follow-up data were not collected, preventing assessment of the natural history of lipid abnormalities in these patients or the impact of treatment.

CONCLUSIONS

Drug-naive hypertensive patients in Nepal show a high prevalence of atherogenic dyslipidemia. It is characterized by elevated triglycerides and low HDL cholesterol. This "lipitension" phenotype-the coexistence of hypertension and dyslipidemia—arises from shared mechanisms including endothelial dysfunction, RAAS activation, and insulin resistance, producing a multiplicative increase in cardiovascular risk. Routine lipid screening in newly diagnosed hypertensive patients is crucial. Comprehensive management addressing both blood pressure and lipid abnormalities through lifestyle modifications and pharmacotherapy can prevent future cardiovascular events. Public health strategies in Nepal and South Asia must prioritize primary prevention and accessible screening programs to combat this growing epidemic.

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