Study of the Relationship of Lipid Profile, BMI and Blood Pressure among Non-diabetic Hypertensive Patients - A Hospital Based Study

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ABSTRACT

Introduction: Several research have demonstrated the connection between lipid levels, diabetes mellitus (DM), and hypertension, but none have focused specifically on the association between lipid levels and essential hypertension in Nepal's non-diabetic population. **Aims:** To study the relationship between lipid profile, body mass index (BMI), and blood pressure (BP) in the non-diabetic hypertensive population. **Methods:** A hospital-based cross-sectional study was conducted among 150 participants. Anthropometric measurements including height, weight and BP were taken. Blood was collected in serum vial for lipid profile [total cholesterol (TC), triglyceride (TG) and low and high density lipoprotein cholesterol (LDLC and HDLC)] after an overnight fasting. **Results:** 44.7% were female, and 55.3% were male with the age ranging between 28-80 years. Among 48.67% of the overweight participants, 56.8% were male. 25.33% were obese, among which 52.6% were male. 56% of participants had systolic blood pressure (SBP) ≥140 mmHg and 58% had diastolic blood pressure (DBP) ≥90 mmHg and showed no significance among the gender. Borderline TG level was observed in 76.2% of participants with SBP ≥140 mmHg and in 74.7% with DBP ≥90 mmHg. HDLC vs gender showed significance. SBP and DBP had a significant relationship with LDLC and TC. The association of BMI with BP and lipid profile was not significant. **Conclusion**: Dyslipidemia is prevalent among non-diabetic hypertensive individuals. The majority of participants were overweight, however blood pressure and lipid profile did not correlate with BMI presumably because other anthropometric characteristics have better predictive values than BMI.

Keywords: Body mass index, blood pressure, hypertension, lipid profile, obesity

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INTRODUCTION

The prevalence of hypertension has increased from 21.5% to 24.5% in Nepal according to the last two periodic surveys conducted in Nepal, WHO STEP wise approach to Surveillance (STEPS) survey 2007 and 2019.[1] Most low-income and middle-income South Asian nations, including Nepal, have seen the increase in the prevalence of hypertension[2] which is the leading cause of cardiovasvular disease and premature death worldwide.[3] In a study conducted in 2016 among the indigenous population of Nepal, the overall prevalence of hypertension was 23.8%.[4] Several previous studies have shown the association between hypertension and dyslipidemia.[5,6] Obesity is one of the major contributors to the prevalence

of cardiovascular risks, hypertension and diabetes.[7] Central obesity and insulin resistance coexists in the pathogenesis of both hypertension and dyslipidaemia.[8] Although Insulin is a major hormone regulating lipid metabolism and type 2 DM is often associated with dyslipidemia.[9] Dyslipidemia in hypertension is associated with high TC level and isolated elevation of the LDLC or TG.[10]

Numerous research have demonstrated the link between lipid levels, DM and hypertension, but only a few studies have concentrated on the interaction between lipid levels and essential hypertension in Nepal's non-diabetic population. Obesity is regarded as a risk factor for dyslipidemia and essential hypertension.[11] We aimed to determine the pattern of dyslipidemia and the relationship between lipid profile, BMI and BP in the non-diabetic hypertensive population.

METHODS

After receiving approval from the Institutional Review Board, People's Dental College and Hospital, a hospital-based cross-sectional study was conducted from September 2021 to March 2022, in the department of Internal Medicine at People's General Hospital, Balaju, Kathmandu. A total of 150 patients between 20-80 years of age, diagnosed with essential hypertension were chosen. Major comorbidities like DM, cardiac, renal, and hepatic problems were excluded from the study. Pregnant ladies and those on thiazides, steroids, oral contraceptive pills (OCPs), lipid-lowering drugs for dyslipidemia were not included. Anthropometric measurements were collected after receiving informed consent and were taken by trained nurses or doctors with allowing only light clothes. Hypertension was defined according to JNC 7 as cut off point of 140mmHg and above for SBP and / or 90mmHg and above for DBP, or reported about the use of antihypertensive medication during the study.[12] A digital portable scale was used to measure body weight as kilogram (kg). Height was measured in centimeter with a stadiometer. BMI kg/m^2 was calculated and classified according to their BMI into three groups: normal weight BMI: 18.5–24.9 kg/m^2, overweight BMI: 25.0–29.0 kg/m^2 and obese BMI ≥ 30 kg/m^2 as per World Health Organisation (WHO). 3.5ml of venous blood was collected in serum vial, prior which the patients were advised 12 hours overnight fasting. Serum was separated within 30 min of blood collection and sent to the lab for estimation of TC, TG, LDLC, HDLC. Using commercial kits by Biosystems (BA-400 biosystems S.A. Spain) analyzer, serum samples were evaluated. TC, HDLC, LDLC and TG were determined directly using enzymatic spectrophotometric techniques. For serum lipid reference level, National Cholesterol Education Programme (NCEP) Adult Treatment Panel III (ATP III) guideline was referred. The desirable level of TC is <200mg/dl, borderline high is between 200-239 mg/dl, high TC is considered when the level is >240mg/dl. LDLC is defined as optimum level when it is <100mg/dl, near optimal 100-129mg/dl, borderline high 130-159mg/dl, high 160-189mg/dl, very high >190mg/dl. TG level is considered normal when it is < 150mg/dl, borderline high 150-199mg/dl, high 200-499mg/dl, very high when it is >500mg/dl. Desirable HDLC is when it is >40mg/dl, and low when it is <40mg/dl.[13] Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 24 software. Simple descriptive statistics were used to present the demographic characteristics of the study participants. Chi-square test was conducted to compare proportions of categorical variables. Mann-Whitney U test and Kruksal-Wallis test was used to test for medians on non-normally distributed numerical variables. All tests were two tailed and p value of <0.05 was considered to be statistically significant.

RESULTS

Among 150 participants 67(44.7%) were female, and 83 (55.3%) were male. The mean age was 58.21± 11.57, range (28-80 years). Mean SBP was 139±17 and mean DBP was 87± 9 with 84(56%) of participants having systolic BP ≥140 mmHg and 87(58%) had diastolic BP ≥ 90 mmHg. 9(6%) cases were newly diagnosed hypertensive cases. Approximately 74(48.67%) of the participants were overweight, with 42(56.8%) being male, and 20(52.6%) being male among the 38(25.33%) obese individuals. Mean TG was 209±79, TC 203±48, LDLC 121±34 and HDLC 38±6. Among the total participants, 83 males had mean HDLC levels below the normal range, which was significant with a p value of 0.001. Characteristics of the study participants are given in Table I.

Study variables	Female (N=67)	Male (N=83)	Total (N=150)	P-value
SBP (mmHg)	140±17	139±17	139±17	0.766
DBP (mmHg)	87±11	87±8	87±9	0.704
Wt (Kgs)	65±11	72±11	69±11	0.000
Ht (m)	1.54±.07	1.62±0.7	1.59±0.8	0.000
BMI (kg/m2)	27.90±5.05	27.30±3.65	27.57±4.32	0.683
TG (mg/dl)	208±81	209±78	209±79	0.582
TC (mg/dl)	202±38	204±54	203±48	0.837
LDLC (mg/dl)	120±32	121±36	121±34	0.965
HDLC (mg/dl)	40±6	37±6	38±6	0.001

*A p-value of <0.5 shows that there is a significant association between two variables

Table I: Characteristics of the study participants

Table II shows the characteristic of lipid profile among genders. TG levels were borderline in 76.1% of females and 79.5% of males. 34.3% of females and 27.7% of males have borderline TC. The optimal high range of LDLC was found in 35.8% of females and 36.1% of males. 56.7% of females had HDLC in the desirable range, while 75.9% of males had low HDLC.

Lipid profile	NCEP ATP III Classification	Female(N=67)	Male(n=83)
TG	Normal	16 (23.9%)	16 (19.3%)
	Borderline	51 (76.1%)	66 (79.5%)
	High	0 (0%)	1 (1.2%)

TC	Desirable	31 (46.3%)	41 (49.4%)
	Borderline	23 (34.3%)	23 (27.7%)
	High	13 (19.4%)	19 (22.9%)
LDLC	Optimal	18 (26.9%)	24 (28.9%)
	Optimal high	24 (35.8%)	30 (36.1%)
	Borderline	17 (25.4%)	16 (19.3%)
	High	5 (7.5%)	7 (8.4%)
	Very High	3 (4.5%)	6(7.2%)
HDLC	Desirable	38 (56.7%)	20 (24.1%)
	low	29 (43.3%)	63 (75.9%)

*National Cholesterol Education Programme (NCEP) Adult Treatment Panel III (ATP III)

Table II: Lipid profile vs gender

The relationship of BMI with SBP and DBP (Table III) was not significant, with p values of 0.676 and 0.657, respectively. In terms of the relationship between high blood pressure and BMI, females followed the same pattern as males. Borderline TG levels were found in 76.2% (64) of participants with SBP ≥140 mmHg and 74.7% (65) of participants with DBP≥90 mmHg. Using the Kruskal-Wallis test, it was determined that SBP and DBP had a significant relationship with LDLC (p values of 0.025 and 0.018, respectively) and TC (p-values of 0.032 and 0.01 respectively). None of the tests that could be used revealed any significance in the relationship between BMI and lipid levels.

Variables	Classification	SBP	P-value	DBP	P-value
TG	Desirable	19 (22.6%)	0.324	22 (25.3%)	0.643
	Borderline	64 (76.2%)		65 (74.7%)	
	High	1 (1.2%)		0 (0%)	
TC	Desirable	34 (40.5%)	0.032	36 (41.4%)	0.01
	Borderline	33 (39.3%)		34 (39.1%)	
	High	17 (20.2%)		17 (19.5%)	
LDLC	Optimal	17 (20.2%)	0.025	18 (20.7%)	0.018
	Optimal high	30 (35.7%)		33 (37.9%)	
	Borderline	22 (26.2%)		20 (23%)	
	High	10 (11.9%)		9 (10.3%)	
	Very High	5 (6%)		7 (8%)	
HDLC	Desirable	37 (44%)	0.645	38 (43.7%)	0.340
	low	47 (56%)		49 (56.3%)	
BMI	Normal	21 (25%)	0.676	23 (26.4%)	0.657
	Overweight	42 (50%)		41 (47.1%)	
	Obese	21 (25%)		23 (26.4%)	

*A p-value of <0.5 shows that there is a significant association between two variables.

Table III: Relationship of Systolic and Diastolic blood pressure with lipid profile and BMI

DISCUSSION

Numerous studies have demonstrated the relationship between DM and hypertension and insulin is now generally acknowledged to play a role in the pathophysiology of obesity-related hypertension.[14] Therefore the main purpose of this study was to examine the association between serum lipid profiles, essential hypertension, and BMI in the non-diabetic participants. According to the findings of this study, approximately half of the total number of hypertensive cases had uncontrolled blood pressure despite being on treatment, (56%) of participants had systolic BP ≥140 mmHg and (58%) had diastolic BP ≥90 mmHg. Similar results were seen among those with treated hypertension, 58.0% had uncontrolled BP: 52.8% in Bangladesh, 70.6% in Pakistan, and 56.5% in Sri Lanka.[15] This result could be due to poor adherence to antihypertensive medications. A study conducted among the Nepalese population suggested that since control of hypertension depends on patient's adherence to therapy and unfortunately, a specific validated tool in the Nepalese version to assess the adherence of Nepalese patients to anti-hypertensive therapy is lacking.[16] There were no significant differences in SPB and DBP among genders in our study. According to recent NHANES (National Health and Nutrition Examination Survey) data, the prevalence of hypertension is 42.5 percent among obese people, compared to 27.8 percent for overweight people.[14] This is in contrast to our survey where we discovered that 25.33% of the participants were obese and 48.67% were overweight, with a predominance of men in both groups. A study conducted at Dharan also suggests a higher percentage of obesity in males, and urbanization and less physical activity being the culprit for it.[17] Our study cannot find a significant relationship between BMI and hypertension

in both genders possibly due to the small sample size. Many previous studies[18,19] indicated that visceral obesity measured by waist circumference (WC) or waist-hip ratio (WHR) was more closely associated with blood pressure and/or the presence of hypertension than overall obesity measured by BMI. A study among large Chinese population showed that BMI is closely associated with hypertension among females and not in males. Similar to their study,[20] the role of insulin resistance and abdominal fat content was not evaluated in our study as well.

In this study the serum levels of TC, LDLC in majority of participants was found to be in higher range than their respective cut-off values and TG was present in borderline range. Additionally HLDC was significantly low in a group of hypertensive males. This is comparable to the studies in the other parts of the world.[21,22] TC and LDLC were significantly related with SBP and DBP in our study whereas TG and HDLC has no significant relation with blood pressure. According to Saha M. et al[23] study, which similarly revealed no significant link between HDLC and hypertension, but contrary to our study they found the significant relationship of TG with blood pressure. Likewise, there were no significant differences between male and female hypertensive patients' serum levels of TG, TC, and LDLC in their study which is a similar finding to ours.

According to previous studies primary dyslipidemia related to obesity is characterized by increased TG, decreased HDLC levels, and abnormal LDLC composition[24], however we fail to relate BMI with lipid levels, additionally dyslipidemia was equally present in non-obese hypertensives in our study. The presence of dyslipidemia in non-obese population could be due to different socioeconomic conditions, dietary habits, sedentary life, urbanization and insulin resistance.[25] We did not evaluate these conditions, which is also one of the limitation to our study. Previously only BMI was used as an indicator of obesity. But several studies have shown that even normal weight subject, those with a BMI <25kg/m^2 may have the presence of metabolic risk factors. Upper body fat, which was linked to dyslipidemia in normal weight people, WC, WHR, but not BMI, may be relevant for screening and managing dyslipidemia in these individuals.[20]

LIMITATIONS

The small sample size and the fact that our study was done in a single centre, our findings do not adequately represent the total Nepalese population. Because our study was cross-sectional, we did not analyze diverse socioeconomic conditions, dietary habits, sedentary life or urbanization. As a result, we were unable to reach a causal conclusion. We failed to assess participants' insulin resistance in our case, despite the fact that obesity and dyslipidemia are strongly related to it. Anthropometric parameters including WHR, WC and Body Mass (BM) were to be evaluated overBMI since body fat distribution plays a significant influence in dyslipidemia among normal weight obese individuals.[20]

CONCLUSION

The results of the current study highlight the prevalence of obesity, hypertension, and dyslipidemia in Nepalese urban residents. In our study, we discovered that hypertensive individuals had high mean levels of TG, LDLC and TC. HDLC was typically low and was much lower in hypertensive men than in women. However, TG and HDLC did not substantially correlate with SBP, only LDLC and TC did. The majority of participants were overweight, however, blood pressure and lipid levels did not correlate with BMI, presumably because other anthropometric characteristics had better predictive values than BMI and could be taken into account.

REFERENCES

1. Dhungana RR, Pandey AR, Shrestha N. Trends in the Prevalence, Awareness, Treatment, and Control of Hypertension in Nepal between 2000 and 2025: A Systematic Review and Meta-Analysis. Int J Hypertens. 2021; 2021: 1-11. DOI: 10.1155/2021/6610649.

2. Das Gupta R, Bin Zaman S, Wagle K, Crispen R, Hashan MR, Al Kibria GM. Factors associated with hypertension among adults in Nepal as per the Joint National Committee 7 and 2017 American College of Cardiology/American Heart Association hypertension guidelines: A cross-sectional analysis of the demographic and health survey 20. BMJ Open. 2019; 9(8): 1–8. DOI: 10.1136/bmjopen-2019-030206.

3. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nat Rev Nephrol. 2020; 16(4): 223–37. DOI: 10.1038/s41581-019-0244-2.

4. Denekew TW, Gautam Y, Bhandari D, Gautam GP, Sherchand JB, Pokhrel AK, et al. Prevalence and determinants of hypertension in underrepresented indigenous populations of Nepal. PLOS Glob Public Health. 2022: 1-18. DOI:10.1371/journal.pgph.0000133.

5. Srivastava A, Binawara BK. Comparative Study on Lipid Profile of Hypertensive Patients and Non-hypertensive. PBJ. 2016; 4: 62–66. DOI: 10.20510/UKJPB/4/I4/112412.

6. Ghooshchi G, Masoomian M, Yazdi MS, et al. Evaluation of the Lipid Profile of Hypertensive Patients Compared to Non-Hypertensive Individuals Article history : Patient Saf Qual Improv. 2014; 2(3): 120-2. DOI: 10.22038/PSJ.2014.2989.

7. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract. 2010; 87(1): 4–14. DOI: 10.1016/j.diabres.2009.10.007.

8. Osuji CU, Omejua EG, Onwubuya EI, et al. Serum lipid profile of newly diagnosed hypertensive patients in Nnewi, South-East nigeria. Int J Hypertens. 2012; 2012: 1-7. DOI: 10.1155/2012/710486.

9. Bhambhani G, Bhambhani R, Thakor N. Lipid profile of patients with diabetes mellitus: a cross sectional study. Int J Res Med Sci. 2015; 3: 3292–3295. DOI:10.18203/2320-6012.ijrms20151179.

10. Islam S, Choudhury KN, Mainuddin AKM, Wahiduzzaman M, et al. Serum lipid profile and its association with

hypertension in Bangladesh. Vasc Health Risk Manag. 2014; 10: 327–332. DOI: 10.2147/vhrm.s61019.

11. Nath JD, Biswas R. Association between Anthropometric Measurements and Lipid Profile in Newly Detected Hypertensive Patients at a Tertiary Hospital in Bangladesh. J Indian coll cardiol. 2020; 10: 6–9. DOI: 10.4103/JICC.JICC_54_19.

12. Chobanian AV., Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003; 42: 1206–52. DOI:10.1161/01.HYP.0000107251.49515.c2.

13. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation. 2002; 106(25): 3143-421. PMID: 12485966.

14. Landsberg L, Aronne LJ, Beilin LJ, et al. Obesity-related hypertension: Pathogenesis, cardiovascular risk, and treatment-a position paper of the the obesity society and the American society of hypertension. Obesity. 2013; 21: 8–24. DOI:10.1002/oby.20181.

15. Jafar TH, Gandhi M, Jehan I, Naheed A, de Silva HA, Shahab H, et al. Determinants of Uncontrolled Hypertension in Rural Communities in South Asia-Bangladesh, Pakistan, and Sri Lanka. Am J Hypertens. 2018; 31: 1205–14. DOI: 10.1093/ajh/hpy071.

16. Shakya R, Shrestha R, Shrestha S, Sapkota P, Gautam R, Rai L. Translation, Cultural Adaptation and Validation of the Hill Bone Compliance to High Blood Pressure Therapy Scale to Nepalese Language. Patient Prefer Adherence. 2022; 16: 957–70. DOI: 10.2147/PPA.S349760.

17. Vaidya AK, Pokharel PK, Nagesh S, Karki P, Kumar S, Majhi S. Association of obesity and physical activity in adult males of Dharan, Nepal. Kathmandu Univ Med J (KUMJ). 2006; (4)2: 192–7. PMID: 18603897.

18. Zhu SK, Wang ZM, Heshka S, Heo M, Faith MS, Heymsfield SB. Waist circumference and obesity-associated risk factors among whites in the third National Health and Nutrition Examination Survey: Clinical action thresholds. Am J Clin Nutr. 2002; 76(4): 743. DOI: 10.1093/ajcn/76.4.743.

19. Siani A, Cappuccio FP, Barba G, Trevisan M, Farinaro E, Iacone R, et al. The relationship of waist circumference to blood pressure: The Olivetti Heart Study. Am J Hypertens. 2002; 15: 780–86. DOI: 10.1016/s0895-7061(02)02976-x.

20. Zhou Z, Hu D, Chen J. Association between obesity indices and blood pressure or hypertension: which index is the best? Public Health Nutr. 2009; 12(8): 1061-71. DOI: 10.1017/S1368980008003601.

21. Gebrie A, Gnanasekaran N, Menon M, Sisay M, Zegeye A. Evaluation of lipid profiles and hematological parameters in hypertensive patients: Laboratory-based cross-sectional study. SAGE Open Med. 2018; 6: 1-11. DOI: 10.1177/2050312118756663.

22. Ajabnoor GMA, Bahijri S, Alamoudi AA, et al. The association between hypertension and other cardiovascular risk factors among non-diabetic Saudis adults-A cross sectional study. PLoS One. 2021; 16(2): 1-15. DOI: 10.1371/journal.pone.0246568.

23. Saha MS, Sana NK, Shaha RK. Serum Lipid Profile of Hypertensive Patients in the Northern Region of Bangladesh. J BioSci (Rajshri). 2006; 14: 93–8. DOI: 10.3329/jbs.v14i0.450.

24. Sharma SK, Ghimire A, Radhakrishnan J, Thapa L, Shrestha NR, Paudel N, et al. Prevalence of hypertension, obesity, diabetes, and metabolic syndrome in Nepal. Int J Hypertens. 2011:1-10. DOI:10.4061/2011/821971.

25. Jabbar A, Irfanullah A, Akhter J, Mirza YK. Dyslipidemia and its relation with body mass index versus waist hip ratio. J Pak Med Assoc. 1997; 47(12):308-10. PMID: 9510644.