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Research Article

**LIPID ABNORMALITIES IN NON-DIABETIC, NON-OBESE
AND PATIENTS OF HYPERTENSION**Dr Abdur Rahman Abshar¹¹MBBS, Jinnah Medical College Peshawar., Email: abshar403@gmail.com

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Abstract:

Background: Dyslipidemia and hypertension are among the main contributors to the pathogenesis of coronary artery disease, and their coexistence does not increase the risk, but rather increases it.

OBJECTIVE: The aim of this study was to determine the frequency of dyslipidemia in non-obese, non-diabetic hypertensive patients and to compare it with the same frequency in non-obese, non-diabetic, normotensive patients.

Methods: This case-control study was conducted in the Department of Medicine, Khyber Teaching Hospital Peshawar for six months duration from January 2021 to June 2021. 120 adults of both sexes were included in the study; 40 non-obese, non-diabetic, hypertensive patients and 80 non-obese, non-diabetic, normotensive individuals. Demographic data and eating habits of each subject were recorded using a questionnaire. Fasting lipid profile and blood glucose of all subjects were evaluated. All data were compared between the two groups.

Results: The hypertensive group showed higher total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG), and lower high-density lipoprotein cholesterol (HDL-C) values compared to the hypertensive group. Normo-tensive group. In males only, TC, LDL-C, HDL-C abnormalities and TG were more common in the hypertensive group (odds ratio = 2.96, 2.67, 4.28 and 4.57), respectively. HDL-C abnormality in women was similar in both groups (odds ratio = 1.47).

Conclusion: Hypertensive individuals are more likely to have various lipid abnormalities such as total cholesterol, LDL-C, HDL-C (men), and triglycerides compared to the normotensive population.

Keywords: Non-obese, Non-diabetic, Hypertensive, Dyslipidemia.

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INTRODUCTION:

Hypertension is an important health problem worldwide due to its high prevalence and associated increased risk of cardiovascular disease. Advances in diagnosis and treatment have played an important role in the recent dramatic decline in deaths from coronary heart disease and stroke in the Western world [1-2]. In normotensive individuals aged 55 years, the lifetime risk of developing hypertension is 90%. The higher the blood pressure, the higher the chance of heart attack, heart failure, stroke, and kidney disease [3]. Hypertension is the strongest and most important modifiable risk factor that triples the risk of stroke. There are many contributing factors that can play a fundamental role in the occurrence of hypertension, such as age, gender, occupation, alcohol intake, salt intake, the amount of blood pumped by the heart, the condition of the blood vessels, and various hormones levels [4-5]. The role of obesity and diabetes mellitus among these factors is universally known. Dyslipidemia often coexists with essential hypertension [6]. The proportion of patients with cardiovascular disease is much higher in patients with concomitant hypertension and dyslipidemia than in patients with isolated hypertension or dyslipidemia. Total and non-HDL (high-density lipoprotein) cholesterol levels increase significantly with increased systolic and diastolic blood pressure in both sexes. Hypertension has been reported to affect 17.7% of the non-obese and non-diabetic adult population in Punjab, Pakistan. Low-density lipoprotein cholesterol (LDL-C) is not a direct cause of hypertension, but acts indirectly by accelerating atherosclerosis [7-8]. HDL cholesterol is believed to mobilize cholesterol from existing and developing atheromas and transport it to the liver for excretion in the bile. Metabolic syndrome is another clinical scenario in which dyslipidemia and hypertension can occur hand in hand with obesity and glucose intolerance. The main culprit is visceral or upper body obesity. Hypertension is rarely found as an isolated condition; rather, it is found in a constellation of other risk factors. In 1991, the Tromso study showed that total and non-HDL cholesterol levels increased significantly with increased systolic and diastolic blood pressure in both sexes [9-10]. Baral *et al.* reported that dyslipidemia is closely related to hypertension. In addition, there is hypertension, possibly resulting in convection of LDL and other atherogenic particles into the intima of the arteries. A study was conducted in Southern Punjab to see the association of age and gender with lipid abnormalities in non-obese, non-diabetic hypertensive subjects [11-12]. We conducted this study in central Punjab to see the pattern of lipid abnormalities in non-obese, non-diabetic

hypertensive patients compared to non-obese, non-diabetic, normotensive subjects. Non-obese, non-diabetic hypertensive patients are as important as obese and diabetic patients because insulin resistance is also found in non-obese individuals and can result in dyslipidemia, its presence has been reported in Southeast Asian and non-obese population. individuals can inherit this problem. The main aim of this study was to determine the frequency of lipid abnormalities in obese and non-diabetic patients with essential hypertension.

METHODS:

This case-control study was conducted in the Department of Medicine, Khyber Teaching Hospital Peshawar for six months duration from January 2021 to June 2021. 120 people aged 35-65 years of both sexes were included in the study; 40 were non-obese, non-diabetic with essential hypertension (group A, cases), and 80 were non-obese, non-diabetic, and normotensive individuals (group B, controls). Patients were selected by non-probability intentional sampling. Patients with myocardial infarction (MI), congestive heart failure (CCF), renal failure, cerebrovascular accident (CVA), glucose intolerance, any medical condition requiring long-term treatment, and those taking medication were excluded from the study. medicines. Forty patients from the emergency and outpatient departments were included in the study group. 80 control subjects (healthcare workers and patient assistants) were enrolled according to the above-mentioned criteria. Informed consent was obtained from all study participants and their data was recorded. Selected individuals were summoned with a 10-hour fast in the morning. They sat for half an hour in a quiet environment without smoking. Blood samples were taken for biochemical analysis. A medical history such as smoking, drugs, family history of dyslipidemia was taken, and a specific physical examination was performed, which included measurement of blood pressure in a sitting position. All this information was recorded on a form. Non-obesity status was defined on the basis of a BMI between 18.5 and 24.9. Non-diabetic status was defined based on fasting blood glucose (FG) <100 mg/dl (5.6 mmol/l) and values between 100 mg/dl (5.6 mmol/l) and 126 mg/dl (7 mmol/l) labeled as glucose intolerance (IGT). If no cause was found on clinical examination and preliminary laboratory data, and any of the following were present, essential systemic hypertension was diagnosed: diastolic blood pressure >90 mmHg at at least two visits or mean systolic >140 mmHg of multiple blood pressure readings at two or more visits. The diagnosis of dyslipidemia was made if any of the following conditions were present; total cholesterol > 200

mg/dl, triglycerides > 150 mg/dl, LDL > 100 mg/dl, and HDL <40 mg/dl (male) and 50 mg/dl (female).

All collected information was entered and analyzed using SPSS version 21.0. Quantitative variables such as age, duration of hypertension, blood pressure, weight, BMI, blood glucose level and lipid levels were presented by calculating mean + SD (standard deviation). Qualitative variables such as gender, smoking, eating habits were presented as frequency and percentage. Odds ratios for various lipid abnormalities were calculated in exposed subjects (Study Group) and unexposed subjects (Control Group).

RESULTS:

The mean age was 46.43 ± 7.58 years for group A (study group) and 46.96 ± 7.76 years for group B (control group). Group B had 40 (50%) and the lowest number of cases was in the 56-65 age group, that is, 7 (17.5%) in group A and 12 (15%) in group B. In the 46-55 age group, 12 (30%) people were in group A and 28 (35%) people were in group B. Gender distribution was similar in both groups; 23/40 (57.5%) men in group A and 47/80 (58.7%) men in group B, Table 1. Both groups were similar in terms of smoking, glycemic status, height, weight and BMI (Table 1).

Table 1: baseline Characteristics of study population:

Parameter	Group-A (n = 40)	Group-B (n = 80)	P value
Age (years) Mean+ SD	46.43±7.58	46.96±7.76	0.63
Age Groups (years) - n (%) 35-45	21 (52.5)	40 (50)	1.00
46-55	12 (30)	28 (35)	0.73
56-65	07 (17.5)	12 (15)	0.92
Sex- n (%)Male	23 (57.3)	47 (58.7)	0.896
Female	17 (42.5)	33 (41.3)	
Smoking- n (%)	15 (37.5)	19 (23.8)	0.12
Fasting blood glucose Mean+ SD	88.83±3.87	89.80±3.95	0.202
Weight (kg) -Mean+ SD	66.00±9.86	67.47±9.14	0.419
Height (m)- Mean+ SD	1.68±0.10	1.69±0.10	0.642
Body mass index (BMI)- Mean+ SD	23.38±1.20	23.66±0.98	0.164

Mean systolic and diastolic blood pressures in group A were 158.07 ± 9.20 and 102.55 ± 4.8 mmHg. The relevant values for group B were 121.04 ± 6.86 and 79.60 ± 6.38 mmHg. Most of the hypertensive patients in the study group, namely 37 (92.5%) had a duration of hypertension between 1 and 5 years.

The dietary habits of the study group and controls in terms of daily consumption of meat, chicken/fish, fruit/vegetable, egg/dairy products, pulses and rice are shown in Table 2 and are comparable in both groups.

Table 2: Dietary Habits of Two Groups.

Variable	Group-A (n = 40)	Group-B (n = 80)	P value
Consumption of Red Meat -n (%)	09 (22.5)	20 (25.0)	0.763
Consumption of Chicken/ Fish - n (%)	30 (75.0)	59 (73.8)	0.883
Consumption of Vegetables/ Fruits- n (%)	39 (97.5)	76 (95.0)	0.518
Eggs/Dairy Products- n (%)	23 (57.5)	41 (51.3)	0.518
Use of Pulses - n (%)	23 (57.5)	49 (61.3)	0.692
Use of rice - n (%)	13 (32.5)	38 (47.5)	0.117

Table 3 shows the mean values of the different lipids in the 2 groups. The hypertensive group (A) had significantly higher total cholesterol, LDL-C and triglyceride levels compared to the normotensive group (B).

Table 3: Lipid profile (in mg/dL) of Two Groups.

Variable	Group-A (n = 40)	Group-B (n = 80)	P value
Total Cholesterol (Mean \pm SD)	208.5 \pm 43.7	178.4 \pm 23.1	<0.001
LDL-C (Mean \pm SD)	121.7 \pm 29.6	101.5 \pm 15.3	<0.001
HDL-C (Mean \pm SD)	43.2 \pm 7.8	45.8 \pm 6.0	0.044
Triglyceride (Mean \pm SD)	220.8 \pm 99.8	155.5 \pm 56.3	0.002

HDL-C level was lower in group A (43.2 \pm 7.8 mg/dl) than group B (45.8 \pm 6.0 mg/dl.), P = 0.044. Table 4 shows a comparison of the different lipid abnormalities in the two groups. Our hypertensive group was more likely in women to have all abnormalities except HDL-C abnormality; [Odds ratio (OR) = 2.96 (95% CI: 1.2-7.4) for CT, OR = 2.67 (95% CI: 1.12-6.41) for LDL-C, OR = 4.28 (95% CI: 1.35-13.89) HDL-C abnormality in men, OR = 1.47 (95% CI: 0.36 to 6.01), OR for HDL-C abnormality in women = 4.57 (95% CI 1.89 to 11.22) TG abnormality].

Table 4: Comparison of Two Groups on the basis of Different Lipid abnormalities (values in mg/dL).

Lipid abnormality	Group-A (n= 40)	Group-B (n = 80)	P value
Total cholesterol >200 - n(%)	17 (42.5)	16 (20.0)	0.009
LDL-C >100 - n(%)	27 (67.5)	35 (43.7)	0.014
HDL-C (Men) <40 - n(%)	14 (56.0)	11 (22.9)	0.004
HDL-C (Women) <50 - n(%)	08 (53.3)	14 (43.7)	0.539
Triglyceride >150 - n(%)	27 (67.5)	25 (31.3)	< 0.001

DISCUSSION:

Our single-center observational study demonstrated a possible link between hypertension and lipid abnormalities in obese and non-diabetic patients. We found that all lipid abnormalities, except HDL-C abnormality, were more common in the hypertensive group in women. While a study by Baral et al showed a direct association with dyslipidemia in hypertensive patients, Foucan et al reported contradictory findings with this study; OR = 1.39 for dyslipidemia. Our results are supported by Lee et al., who noted that the combination of dyslipidemia and hypertension is common and more frequent than incidentally detected; can be considered as a different syndrome [13-14]. Saha et al. concluded that total cholesterol, triglycerides, and LDL-C were significantly increased in hypertensive patients compared to control subjects, while HDL-C levels were consistent with our results. They also stated that there was no significant change in lipid profile in male and female hypertensive patients, but abnormal lipid profile in control subjects was more common in males than

females. Most of our hypertensive patients are in the 35-45 age group [15]. Kotokey et al. reported that hypertension is more common in the 50-59 age group in their study in Dibrugarh, The global prevalence of hypertension was 27.9%, and 54% of hypertensive patients had dyslipidemia [16-17]. While their study population was only urban, we had a mixed population; Another factor that can explain this age group difference is geographical diversity. The high frequency of dyslipidemia in the hypertensive population is consistent with our study [18-20].

Important limitations of our study are the relatively small sample size and historical dependence on data on subjects' eating habits, as they did not keep a diet diary.

CONCLUSION:

In conclusion, there seems to be a high probability of a possible association between hypertension and dyslipidemia in the non-obese and non-diabetic population. These data cannot determine whether this

relationship is a relationship or a cause-effect relationship in both directions. A large-scale cohort study could answer these questions.

REFERENCES:

1. Santos AS, Rodrigues AP, Rosa LP, Sarrafzadegan N, Silveira EA. Cardiometabolic risk factors and Framingham Risk Score in severely obese patients: Baseline data from DieTBra trial. *Nutrition, Metabolism and Cardiovascular Diseases*. 2020 Mar 9;30(3):474-82.
2. Yandrapalli S, Jolly G, Horblitt A, Sanaani A, Aronow WS. Cardiovascular benefits and safety of non-insulin medications used in the treatment of type 2 diabetes mellitus. *Postgraduate medicine*. 2017 Nov 17;129(8):811-21.
3. Diwan AG, Kuvalekar AA, Dharamsi S, Vora AM, Nikam VA, Ghadge AA. Correlation of serum adiponectin and leptin levels in obesity and type 2 diabetes mellitus. *Indian journal of endocrinology and metabolism*. 2018 Jan;22(1):93.
4. Zhang H, Lin S, Gao T, Zhong F, Cai J, Sun Y, Ma A. Association between sarcopenia and metabolic syndrome in middle-aged and older non-obese adults: a systematic review and meta-analysis. *Nutrients*. 2018 Mar;10(3):364.
5. Opoku S, Gan Y, Fu W, Chen D, Addo-Yobo E, Trofimovitch D, Yue W, Yan F, Wang Z, Lu Z. Prevalence and risk factors for dyslipidemia among adults in rural and urban China: findings from the China National Stroke Screening and prevention project (CNSSPP). *BMC Public Health*. 2019 Dec;19(1):1-5.
6. Kotsis V, Tsioufis K, Antza C, Seravalle G, Coca A, Sierra C, Lurbe E, Stabouli S, Jelakovic B, Redon J, Redon P. Obesity and cardiovascular risk: a call for action from the European Society of Hypertension Working Group of Obesity, Diabetes and the High-risk Patient and European Association for the Study of Obesity: part B: obesity-induced cardiovascular disease, early prevention strategies and future research directions. *Journal of hypertension*. 2018 Jul 1;36(7):1441-55.
7. Gebreyes YF, Goshu DY, Geletew TK, Argefa TG, Zemedu TG, Lemu KA, Waka FC, Mengesha AB, Degefu FS, Deghebo AD, Wubie HT. Prevalence of high bloodpressure, hyperglycemia, dyslipidemia, metabolic syndrome and their determinants in Ethiopia: Evidences from the National NCDs STEPS Survey, 2015. *PloS one*. 2018 May 9;13(5):e0194819.
8. Ferrara D, Montecucco F, Dallegri F, Carbone F. Impact of different ectopic fat depots on cardiovascular and metabolic diseases. *Journal of cellular physiology*. 2019 Dec;234(12):21630-41.
9. Hall JE, Mouton AJ, da Silva AA, Omoto AC, Wang Z, Li X, do Carmo JM. Obesity, kidney dysfunction, and inflammation: Interactions in hypertension. *Cardiovascular Research*. 2021 Jul 1;117(8):1859-76.
10. Kuwabara M, Kuwabara R, Niwa K, Hisatome I, Smits G, Roncal-Jimenez CA, MacLean PS, Yracheta JM, Ohno M, Lanaspa MA, Johnson RJ. Different risk for hypertension, diabetes, dyslipidemia, and hyperuricemia according to level of body mass index in Japanese and American subjects. *Nutrients*. 2018 Aug;10(8):1011.
11. Ye Q, Zou B, Yeo YH, Li J, Huang DQ, Wu Y, Yang H, Liu C, Kam LY, Tan XX, Chien N. Global prevalence, incidence, and outcomes of non-obese or lean non-alcoholic fatty liver disease: a systematic review and meta-analysis. *The lancet Gastroenterology & hepatology*. 2020 Aug 1;5(8):739-52.
12. Naqvi S, Naveed S, Ali Z, Ahmad SM, Khan RA, Raj H, Shariff S, Rupareliya C, Zahra F, Khan S. Correlation between glycated hemoglobin and triglyceride level in type 2 diabetes mellitus. *Cureus*. 2017 Jun;9(6).
13. Aloud AA, Chinnadurai V, Govindasamy C, Alsaif MA, Al-Numair KS. Galangin, a dietary flavonoid, ameliorates hyperglycaemia and lipid abnormalities in rats with streptozotocin-induced hyperglycaemia. *Pharmaceutical biology*. 2018 Jan 1;56(1):302-8.
14. Chu DT, Nguyet NT, Dinh TC, Lien NV, Nguyen KH, Ngoc VT, Tao Y, Le DH, Nga VB, Jurgowski A, Tran QH. An update on physical health and economic consequences of overweight and obesity. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*. 2018 Nov 1;12(6):1095-100.
15. Ahadi M, Molooghi K, Masoudifar N, Namdar AB, Vossoughinia H, Farzanehfard M. A review of non-alcoholic fatty liver disease in non-obese and lean individuals. *Journal of Gastroenterology and Hepatology*. 2021 Jun;36(6):1497-507.
16. Iqbal J, Al Qarni A, Hawwari A, Alghanem AF, Ahmed G. Metabolic syndrome, dyslipidemia and regulation of lipoprotein metabolism. *Current diabetes reviews*. 2018 Oct 1;14(5):427-33.
17. Gaiz A, Mosawy S, Colson N, Singh I. Thrombotic and cardiovascular risks in type two diabetes; Role of platelet hyperactivity.

- Biomedicine & pharmacotherapy. 2017 Oct 1;94:679-86.
18. Yanai H, Yoshida H. Beneficial effects of adiponectin on glucose and lipid metabolism and atherosclerotic progression: Mechanisms and perspectives. *International journal of molecular sciences*. 2019 Jan;20(5):1190.
 19. Prakash GT, Das AK, Habeebullah S, Bhat V, Shamanna SB. Maternal and neonatal outcome in mothers with gestational diabetes mellitus. *Indian journal of endocrinology and metabolism*. 2017 Nov;21(6):854.
 20. Koliaki C, Liatis S, Kokkinos A. Obesity and cardiovascular disease: revisiting an old relationship. *Metabolism*. 2019 Mar 1;92:98-107.