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Correlation of Blood Pressure with Fasting Blood Glucose Levels and Lipid Profile in a Selected Hypertensive and Normotensive

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ABSTRACT

Introduction

Hypertension increases the risk of diabetes mellitus (DM) and dyslipidemia. Objective of the present study was to investigate the association of blood pressure with fasting blood glucose and lipid profile in selected hypertensive and normotensive subjects. Study included, control: normotensive healthy individuals (n=41); case: known primary hypertensive patients (n=39)). Blood pressure (BP) was measured and recorded as SBP and DBP. Venous blood (5 mL) were collected from each subject for the analysis of total cholesterol (TC), triglycerides (TG) and high density lipoprotein (HDL) and fasting blood glucose (FBG). FBG levels were measured using Glucose Oxidase method. Total cholesterol and triglyceride levels were measured by enzymatic colorimetric method. HDL levels measured using precipitation method where LDL levels calculated from Freidewald equation. The median SBP and DBP values were significantly different (p=0.000) among hypertensives and normotensives. A positive weak correlation (r=0.364, p=0.023) observed between DBP and FBG in hypertensives. No significant correlation observed between SBP and FBG either in hypertensives or in normotensives. Hypertensives had higher median FBG level than normotensives. The median TC (p=0.042) and HDL (p=0.004) values were significantly different among hypertensives and normotensives. The median TG (p=0.725) and LDL (p=0.113) values were not significantly different among hypertensives and normotensives. However, there was no significant correlation between SBP as well as DBP with TC, TG, HDL, LDL either in hypertensives or in normotensives. Hypertensives had significantly higher SBP and DBP levels compared to normotensives. The FBG level of hypertensives increases gradually as DBP level increases. There were no significant correlations between SBP level and FBG in both hypertensives and normotensives. There were no significant correlations between SBP and/or DBP levels and lipid profile categories (TC, TG, HDL, LDL) in hypertensives as well as in normotensives.

1. Introduction

Hypertension can be defined as the elevation of blood pressure (BP) above optimal value. Optimal blood pressure range is systolic blood pressure (SBP) lower than 120 mmHg and diastolic blood pressure (DBP) less than 80 mm Hg (normotension). If SBP level is higher than 130 mmHg and/or a DBP higher than 80 mmHg, it is diagnosed as stage 1 hypertension [1]. Hypertension is usually asymptomatic and often detected while opportunistic screening. But its progression is strongly associated with functional and structural cardiac and vascular abnormalities that lead to premature morbidity and death [2]. According to WHO, hypertension is one of the major cause for the premature death in the world. Prevalence of hypertension in Sri Lanka was 23.7% in adults. Further, there was no significant

difference in the prevalence of hypertension between men (23.4%) and women (23.8%) [3].

Increasing age, physical inactivity, over weight/obesity, excess salt consumption, alcohol intake, stress and many other conditions increase the risk of hypertension. Most patients are asymptomatic and individuals with optimal BP and no identifiable signs and symptoms may exhibit occasional elevation of BP in the absence of hypertension [4].

Hypertension and diabetes are components of metabolic syndrome which resulted from unhealthy diet, sedentary life style and high psychological stress. Hypertensive individuals often develop diabetics and hypertension in diabetes patients leads to develop cardiovascular disease thus increase the morbidity and mortality [5]. According to the available data, elevated blood pressure increases the risk of diabetes by insulin resistance. Insulin involves blood pressure regulation by several mechanisms such as inducing vasorelaxation by stimulating nitric oxide production in endothelium and sodium metabolism in kidney. Insulin resistance which enhances sodium reabsorption causes the elevation of blood pressure level [6].

Hypertension and diabetes mellitus (DM) commonly coexist and patients with both diseases are vulnerable to cardiovascular diseases. According to published data, increase in SBP of 20 mmHg is associated with a 58% higher risk of developing diabetes and raised DBP of 10 mmHg increase the risk of diabetes by 52% [7]. Many previous studies revealed high fasting blood sugar (FBS) levels increase the risk of hypertension by increasing advanced glycation end products, oxidative stress, inflammation, vascular dysfunction [8].

Hypertension accelerates the progression of atherosclerosis primarily in small arteries and arterioles. In hypertension, the blood flows through the vessels under high force which accelerates the formation of plaques by injuring the endothelial cells and enhancing deposition of low density lipoproteins (LDL). That recruits migration and adherence of monocyte to endothelium and also enhances the atherosclerotic response. Hypertension also destabilizes the plaques. However, hypertension cannot induce atherosclerosis in large arteries in the absence of high plasma cholesterol level. Therefore, hypertension in patients with dyslipidemia induce atherosclerosis in large arteries and progress to ischemic conditions such as myocardial infarction. Development of atherosclerosis and cardiovascular diseases can be significantly reduced by antihypertensive drugs [9].

It is believed that untreated hypertension associated with alteration of lipid metabolism which leads to derangement of serum lipids and lipoprotein levels. However, there is no specific pattern of dyslipidemia in hypertension. Studies indicated that lipid levels tend to increase as BP increases. Hence, dyslipidemia is an independent risk factor for primary hypertension [10]. Reported literature have shown significant correlation between serum total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL), triglycerides (TG), and blood pressure in hypertensive. Many researchers reported increased serum level of TC, TG, LDL and decreased HDL in hypertensive subjects. Elevation of lipids in the blood (dyslipidemia) and hypertension increase the risk for cardiovascular disease (CVD) which accounts for majority of deaths in the world [3,11,12]

Hence, the present study aims to determine the association of blood pressure with fasting blood glucose (FBG) levels and lipid profile in selected hypertensive and normotensive subjects.

2. Material and Methods (Arial-9.5)

2.1 Study design

This is an unmatched case-control study. Cases were individuals with diagnosed primary hypertension and the controls were normotensive subjects selected from general population in few selected districts of Sri Lanka.

2.2 Study population

A case control study was conducted including patients with primary hypertension (case group) and apparently healthy normotensive individuals (control group) between 30-60 years of age. The participants recruited through a self-administered questionnaire. All participants were screened for the exclusion criteria. We excluded subjects with previously diagnosed history of diabetes mellitus, heart diseases, dyslipidemias and secondary hypertension.

Investigators had discussions with study participants prior to the study in a separate room and ensured that all the participants have received a full disclosure of the nature of the study, the risks, benefits, and alternatives with an extended opportunity to ask questions. An information sheet printed in English, Sinhala and Tamil were distributed among the participants. A written informed consent was obtained by each participant and the participation in the study was entirely voluntary.

2.3 Study setting

The study was conducted in the Faculty of Allied Health Sciences, University of Sri

Jayewardenepura, Sri Lanka. Ethical approval was granted by the Ethics Review Committee of University of Sri Jayewardenepura, Sri Lanka.

2.4 Sample size calculation

Sample size was calculated according to the two-sample t-test calculation criteria. Minitab version 16 software calculator was used for the calculation and calculated minimum sample size was 40 in each category of study subjects.

2.5 Sample collection

Blood samples (12 hours fasting) for the cases were taken from a private laboratory after performing their analysis with in the stipulated time period of sample stability for each test of fasting blood glucose (FBG) and lipid profile and no additional bleeding was done for the cases. However, blood samples for controls were taken from apparently healthy normotensive subjects. A sample of five milliliter (5 mL) of 12 hours fasting whole blood sample was collected by venipuncture by a qualified phlebotomist from each control subject. Blood pressure of each subject was measured after 10 minutes from venipuncture using a standard mercury sphygmomanometer according to the guidelines of the American Heart Association and recorded separately for SBP and DBP [13] Height and weight of each subject was measured and body mass index (BMI) of all study participants was calculated (weight/height^2). Blood samples were analyzed for FBG and lipid profile using HUMALYZER PRIMUS semi-automated analyzer.

2.6 Statistical analysis

Statistical analysis was performed by using IBM SPSS version 25.0. The distribution of SBP levels and DBP levels in control and case groups deviate from normal distribution, therefore, independent sample Mann-Whitney U non parametric test was used to compare the median of SBP and DBP across the cases and controls groups. According to results of shapiro-wilk test, distribution of levels of FBG, TC, TG, HDL and LDL were not normal. Therefore, we compared the medians of all those variables for cases and controls using independent sample Mann-Whitney U non parametric test. Pearson's correlation 2-tailed test was done to investigate the association between SBP and DBP separately with FBG, TC, TG, HDL, LDL in both groups. The value of "r" was calculated in control and case group respectively.

3 Results and Discussion

3.1 Demographic characteristics of study population

The study was conduct using 80 individuals between 30-60 years of age. Out of 80 individuals 39 were hypertensive subjects (case group) (48.8%) and 41 were normotensive apparently healthy subjects (control group) (51.3%). Mean age of the cases (52.0±6.3 years) was significantly higher (p=0.000) compared to controls (45.1±8.5 years). Although, the case group showed a slightly elevated BMI level (25.2 kg/m^2) compared to that of controls (24.4 kg/m^2), no statistical significance was observed in BMI among two study groups (p=0.281). When consider the control group, out of 41 normotensive individuals there were 56.1% males and 43.9% females. Among cases there were 59% females and 41% male subjects.

3.2 Distribution of blood pressure in study population

Distribution of SBP level is significantly different (p=0.000) across the control and case group. Cases showed higher median SBP level (140 mmHg) compared to control group (114 mmHg). Similarly, distribution of DBP level is significantly different (p=0.000) across the control and case group. Cases showed higher median DBP level (90 mmHg) compared to control group (76 mmHg).

3.3 Association of blood pressure with FBG in hypertensive and normotensive subjects

The median FBG level of those with hypertension (case group) (100 mg/dl) was significantly higher (p=0.000) compared to control group (82 mg/dl). There is no significant correlation between SBP level and FBG level in both control and case groups. Although, DBP level in control group did not show significant association with FBG, cases showed a significant positive weak correlation (p=0.023) between DBP level and FBG levels.

Table1: Correlation between SBP and DBP level with FBG level in the control and case group

	FBG(mg/dl)			
	Control		**Case**	
	Coefficient (r)	p value	Coefficient (r)	p value
DBP (mmHg)	- 0.003	0.985	0.364	0.023*
SBP (mmHg)	0.000	0.999	-0.103	0.531

* Correlation is significant at the 0.05 level (2-tailed)

3.4 Association of blood pressure with Total cholesterol (TC), Triglycerides(TG), HDL cholesterol, LDL cholesterol in study subjects

Normotensive control subjects showed a significantly higher (p=0.042) median TC level (197.4 mg/dl) compared to that of case group (182 mg/dl). Further, control subjects showed higher median TG level (122 mmHg) compared to cases group (110 mmHg) and normotensive control subjects showed a significantly higher median (p=0.004) HDL level (50 mmHg) compared to cases group (46 mmHg). Present study could not observe statistically significant difference (p=0.113) across the control and case groups for LDL parameter, although, controls showed higher median LDL level (125 mmHg) compared to case group (114.4 mmHg).

Surprisingly, no statistically significant correlation was observed between SBP level and TC, TG, HDL or LDL level in the control group as well as case group. Similarly, there is no significant correlation observed between DBP level and TC, TG, HDL or LDL level either the control or the case group. According to Choudhury *et al.,* (2014) hypertensive patients had higher TC, TG and LDL, lower HDL than normotensives and dyslipidemia in hypertension is a major risk factor for CVD [14].

The present study was conducted to study the association of blood pressure with fasting blood glucose level and lipid profile in selected hypertensive subjects (n=39) compared to normotensive subjects (n=41). According to results of statistical analysis, hypertensive subjects had higher median value of FBG than normotensives subjects. We obtained a significant positive weak correlation between DBP level and FBG level in the case group and did not obtain a significant correlation between DBP level and FBG level in the control group. Similarly, we did not obtain a significant correlation between SBP level and FBG level in hypertensive cases as well as in normotensive controls.

Similar to present findings, Kumar *et al* (2010) reported that hypertensive cases had higher blood glucose levels compared to normotensive controls [15]. A study conducted by Rantala *et al.,* (1999) reported that drug-treated hypertensive subjects tend to have poor control in glucose and insulin metabolism [16]. A study done by Bamrara and Mittal (2014) have shown significant higher FBG levels in hypertensives than normotensives which further strengthen present study findings [17]. Slmasi *et al.,* (2004) reported higher prevalence of glucose abnormalities among hypertensive subjects [18] which gives positive insights to the present study. Similarly, Sheikh *et al.,* (2012) also found higher FBG levels in hypertensives. Authors further commented that it may be due to the presence of 55% diabetic cases in hypertensive group [19].

Kuwabara *et al.,* (2019) reported significant association of FBG with hypertension [20] while Manicardi *et al.,* (1986) reported that obese subjects with essential hypertension showed more insulin resistance compared to normotensives [21]

One way of explaining our findings is that raised level of glucose could be a result of endothelial dysfunction and subsequent insulin resistance. High blood pressure damages the blood vessels and alter the structure and function of blood vessels. These vascular alterations are responsible for the incidence of diabetic [22]. Further, hypertension increase vascular and systemic inflammation responses and oxidative stress which promote the vascular dysfunction. Inflammation linked with both insulin resistance and blood pressure elevation. As a result of insulin resistance, plasma levels of glucose will be elevated (6). Thus, impaired glucose tolerance, develops among hypertensives increase the risk for developing type 2 DM [23].

Hypertension and DM are component of metabolic syndrome and commonly coexist. Conen *et al.,* (2007) stated pre-hypertension and hypertension strongly predict the future development of type 2 DM. It was believed that endothelial dysfunction, inflammation and insulin resistance could be factors that associate with blood pressure and new-onset of diabetes [24].

Even though we got a positive weak association between FBG and DBP, we did not get the trend of increasing FBG with SBP. The reason for not obtaining an association may be because the case and control groups are not age and sex matched as blood pressure is known to be affected by age and gender.

A study done by Midha *et al.,* (2015) also showed similar findings to the present study. According to their findings, there was no statistically significant association between SBP and FBG. However, they have found a significant association between DBP and FBG. However, this study was done among young hypertensive adults [25].

According to study conduct by Yaogai *et al.,* (2018) a positive association of BP with FBG was recorded, which also different among genders. Authors also revealed that females had lower risk for developing hypertension than males and only females with high SBP were associated with high FBG levels [26].

Many previous studies revealed chronic hyperglycemia induce hypertension through several mechanisms. Higher FBG induced several pathological changes such as increasing advanced glycation end products, oxidative stress, inflammation and vascular dysfunction. Hence, higher FBG level increase the risk for future hypertension [8]. In the present study we investigated FBG and lipid profile pattern in primary hypertensive subjects and therefore, investigators excluded the hyperglycemic subjects to reduce the effect on results.

Even though we excluded the hyperglycemia subjects during recruitment of participants, one normotensive subject in the control group and 13 hypertensive subjects in the case group had higher levels of FBG than the normal reference range. This undiagnosed hyperglycemia is mainly due to lack of awareness among people and also poor access to health care facilities specially due to the COVID 19 pandemic situation.

According to results of statistical analysis, SBP and DBP had no significant associations with TC, TG, LDL and HDL level in the control group as well as case group. The distribution of TG and LDL were same across case and control group while the distribution of TC and HDL were not same across case and control group. Normotensive control group had higher median TC level (197.4 mg/dl) and higher median HDL level (50 mg/dl) than hypertensive cases.

Similar to present findings, a study conducted by Rekha and Prasad (2016) also did not show significance difference in LDL and HDL levels between hypertensive and normal healthy individuals during their study in Bihar state in 2016. However, they have found significant high levels of total cholesterol and triglyceride in hypertensive patients compared to normotensives [27]. Similarly, Akintunde A (2010) did not find statistically significant difference of serum lipid levels between hypertensives and normotensives even though they have found higher levels of serum lipids in hypertensives [28].

However, contradictory findings to the present study have been recorded by many studies. Those have stated that hypertensives are more prone to develop lipid abnormalities and most commonly they showed elevated levels of serum TC, TG and LDL and decreased level of serum HDL than normotensives. However, the pattern of dyslipidemia changes according to population sub groups across worldwide [12].

A study conduct by Osuji *et al.,* (2012) reported that newly diagnosed hypertensive patients had significantly higher SBP, DBP, FBS, TC, TG, and LDL levels compared to normotensive subjects. According to their findings, elevated TC and LDL was the most common lipid abnormality and also they have reported lipid abnormalities in hypertensive often appeared as group of lipid abnormalities rather than single lipid abnormality [10].

Choudhury *et al.,* (2014) reported that TC, TG, LDL levels were higher and HDL levels were lower in hypertensive subjects compared to normotensive subjects [14] while Gebrie *et al.,* (2018) found higher TC, TG and LDL levels are associated with uncontrolled hypertensives [29]. Ghooshchi *et al.,* (2014) reported only TG levels in hypertensives were significantly different from normotensives [30].

Evidence suggests that hypertension associated with alteration of lipid metabolism and this may be due to genetic locus for hypertension linked to the LDL receptor [15]. Even though the exact mechanism for occurrence of dyslipidemia in hypertension is not known, the evidence presented is consistent with serum lipid levels that gradually increase as BP increases.

Despite the comparable results of serum lipid levels with other studies, present study found lower serum lipid level pattern in hypertensives than normotensive study group which can be supported by the controlled dietary habits of hypertensives but dietary habits were not studied in the present study. The reason for not obtaining an association between BP and lipid profile may be because the control and case groups are not age and sex-matched. Most of the individuals were above 50 years old in hypertensive case group.

Wang *et al.,* (2011) reported association between sex hormones and lipid metabolism thus, results in different serum lipid profiles in men and women at different ages [31]. Also Pingsen *et al.,* (2018) reported lipid profiles significantly different according to age and sex among patients with ischemic stroke. Their results demonstrated dyslipidemia is more prevalent in non-elderly (40-69 years) than elderly (>80 years) and females have higher TC, TG, LDL and low HDL than males [32].

Another reason for not obtaining an association between BP and lipid profile may be because we recruited hypertensive individuals who were taking antihypertensive drugs. Subjects with lipid lowering drugs: statins, were also excluded as it affects lipid metabolism. Different antihypertensive drugs have different mechanisms to control blood pressure. According to Ferrari *et al.,* (1991) many antihypertensive medications such as calcium channel blockers, angiotensin

converting-enzyme inhibitors have no or detrimental effect on lipoprotein or carbohydrate profiles while thiazide-type diuretics can increase serum LDL and TG levels [33].

A study conducted by Kumar *et al.,* (2010) reported that β-blockers used in hypertension are associated with lowering the level of HDL, while ACE-inhibitors show small beneficial effect on TC and LDL levels [15]. Ning *et al.,* (2018) reported that development of atherosclerosis and cardiovascular diseases can be significantly reduced by antihypertensive drugs [9]. These study findings further support the present study results.

Katulanda *et al.,* (2014) demonstrated that urban adult population had a higher prevalence for hypertension than rural adult population in Sri Lanka. According to their findings nearly one-third of adult population was hypertensive and obese, increasing age, male gender, moor ethnicity, diabetics and physical inactivity have significant influence on hypertension [3].

4 Conclusion

Hypertensive patients had significantly higher SBP and DBP levels compared to normotensives, even they are on antihypertensive medications. The FBG level of hypertensives increases gradually as DBP level increases. There was no significant correlation between SBP level and FBG in hypertensives as well as in normotensives.
There were no significant correlations between SBP and/or DBP levels and lipid profile categories (TC, TG, HDL, LDL) in hypertensives as well as in normotensives.

References

1. Toto, Robert D. Defining Hypertension: Role of New Trials and Guidelines. Clinical Journal of the American Society of Nephrology. 2018:13(10):1578-1580.
2. Lovibond K, Jowett S, Barton P, Caulfield M, Heneghan C, et al. B. Cost-effectiveness of options for the diagnosis of high blood pressure in primary care: A modelling study. The lancet. 2011;378(9798):1219–30. DOI:10.1016/S0140-6736(11)61184-7
3. Katulanda P, Ranasinghe P, Jayawardena R, Constantine GR, Rezvi SMH, et al. The prevalence, predictors and associations of hypertension in Sri Lanka: A crosssectional population based national survey. Clinical Experimental Hypertension. 2014;36(7):484-91. DOI: 10.3109/10641963.2013.863321
4. Gaware RJ, Shinde NG, Chandrawanshi MJ, Wattamwar PB, Belle SD. High Rates Of Hypertension In India. 2018:17(7), 523-537.
5. Petrie JR, Guzik TJ, Touyz RM. Diabetes, Hypertension, and Cardiovascular Disease.Clinical Insights and Vascular Mechanisms. Can J Cardiol. 2018:34(5):575-584.
6. Zhou MS, Wang A, and Yu H. Link between insulin resistance and hypertension: What is the evidence from evolutionary biology?. Diabetology & Metabolic Syndrome. 2014;6(1):12. DOI:10.1186/1758-5996-6-12
7. Emdin CA, Anderson SG, Woodward M, Rahimi K. Usual blood pressure and risk of new-onset diabetes evidence from 4.1 Million adults and a meta-analysis of prospective studies. Journal of the American College of Cardiology. 2015;66(14):1552–62.
8. Kuwabara M, Hisatome I. The Relationship between Fasting Blood Glucose and Hypertension. American Journal of Hypertension. 2019;32(12):1143–5. DOI:10.1093/ajh/hpz147
9. Ning B, Chen Y, Waqar AB, Yan H, Shiomi M, et al. Hypertension Enhances Advanced Atherosclerosis and Induces cardiac death in watanabe heritable hyperlipidemic rabbits. The American Journal of Pathology. 2018;188(12):2936–47. DOI:10.1016/j.ajpath-2018-08-007
10. Osuji CU, Omejua EG, Onwubuya EI, Ahaneku GI. Serum lipid profile of newly diagnosed hypertensive patients in Nnewi, South-East nigeria. International Journal of Hypertension. 2012;2012. DOI:10.1155/2012/710486
11. Zhang Y, Vittinghoff E, Pletcher MJ, Allen NB, Al Hazzouri AZ, et al. Y. Association of blood pressure and cholesterol levels during young adulthood with later cardiovascular events. Journal of American College of Cardiology. 2019;74(3):330-41. DOI: 10.1016/j.jacc.2019.03.529
12. Bonaa KH , Thelle DS. Association between blood pressure and serum lipids in a population: The Tromsø study. Circulation. 1991;83(4)1305–14.
13. Paul KW, Robert MC, Giuseppe M, Reinhold K, Joshua DB, Bryan W. Harmonization of the American college of Cardiology/American Heart Association and European Society of Cardiology/ European Society of Hypertension and Blood Pressure/ Hypertension Guidelines: Comparisons, Reflections and Recommendations. European Heart journal,2022:35(43):3302-3311
14. Choudhury KN, Mainuddin AKM, Wahiduzzaman M, Ohamme SM, Islam S. Serum lipid profile and its association with hypertension in Bangladesh. Vascular Health and Risk Management. 2014;10:327–332

15. Kumar NL, Deepthi J, Rao YN, Deedi MK. Study of lipid profile, serum magnesium and blood glucose in hypertension. Biol Med. 2010;2(1):6–16.
16. Rantala AO, Kauma H, Lilja M, Savolainen MJ, Reunanen A, et al. Prevalence of the metabolic syndrome in drug treated hypertensives and control subjects. Journal of International Medicine. 1996;245:163-74.
17. Pooja Bamrara, Yashoda Mittal. Fasting blood glucose level in patients suffering from hypertension. Asian Journal of Biomedical and Pharmaceutical Sciences. 2014;04(29):19-22.
18. Slmasi AM, Alimo A, Dancy M. Prevalence of unrecognized abnormal glucose tolerance in patients attending a hospital hypertension clinic. Americn Journal of Hypertension. 2004;17(6):483-488. DOI:10.1016/j.amjhyper.2004.01.011
19. Sheikh MK, Akhtar J, Mustafa SG, Mangario, Kashif F, et al. Fasting blood glucose and serum magnesium in patients with hypertension. World Appliied Science Journal. 2012;17(10):1261-64.
20. Kuwabara M, Chintaluru Y, Kanbay M, Niwa K, Hisatome I, et al. Fasting blood glucose is predictive of hypertension in a general Japanese population. Journal of Hypertension. 2019;37(1):167–74. DOI: 10.1097/HJH.0000000000001895
21. Manicardi V, Camellini L, Bellodi G, Coscelli C, Ferrannini E. Evidence for an association of high blood pressure and hyperinsulinemia in obese man. The Journal of Clinical Endocrinology & Metabolism. 1986; 62(6):1302-4. DOI: 10.1210/jcem-62-6-1302
22. Hsueh WA, Anderson PW. Hypertension, the endothelial cell, and the vascular complications of diabetes mellitus. Hypertension. 1992:20:253-263.
23. Akintunde AA. Epidemiology of conventional cardiovascular risk factors among hypertensive subjects with normal and impaired fasting glucose. South African Medical Journal. 2010; 100(9):594-7.
24. Conen D, Ridker PM, Mora S, Buring JE, Glynn RJ. Blood pressure and risk of developing type 2 diabetes mellitus: The Women's Health Study. 2007:28:2937-2943.
25. Midha T, Krishna V, Shukla R, Katiyar P, Kaur S, et al. Correlation between hypertension and hyperglycemia among young adults in india. World Journal of Clinical Cases. 2015;3(2):171-179. DOI: 10.12998/wjcc.v3.i2.171
26. Lv Y, Yao Y, Ye J, Guo X, Dou J, et al. Association of blood pressure with fasting blood glucose levels in Northeast China: a cross-sectional study. Scientific Reports. 2018; 8(1):1-7.
27. Rekha K, Prasad RR. Effect of hypertension on lipid profile of individuals of Bihar State. International Journal of Scientific Study. 2016; 4(5):196-8. DOI: 10.17354/ijss/2016/459
28. Akintunde AA. Epidemiology of conventional cardiovascular risk factors among hypertensive subjects with normal and impaired fasting glucose. South African Medical Journal. 2010; 100(9):594-7.
29. 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 Medicine. 2018; 6:1-11. DOI: 10.1177/2050312118756663
30. Ghooshchi G, Masoomian M, Yazdi MS, Ramezan M.P. Evaluation of lipid profile of hypertensive patients compared to non-hypertensive individuals. Patient Safety & Quality Improvement Journal.2014; 2(3):120-122.
31. Wang X, Magkos F, Mittendorfer B. Sex hormones and lipid metabolism. Journal of Clinical Endocrinal Metabolism. 2011; 96(4):885-893. Doi: 10.1210/jc.2010-2061
32. Pingsen Z. Sadong L, Zhixiong Z, Jing L. Age and sex related difference of lipid profile in patients with ischemic stroke in china. Hypertension. 2018; 97(23):pe10930. DOI 10.1097/MD.0000000000010930
33. Ferrari P, Rosman J, Weidmann P. Antihypertensive agents,serum lipoproteins and glucose metabolism.The American Journal of Cardiology. 1991:67(10):B26-35.

