Original Article

Association of Serum Triglycerides and Total Cholesterol levels with Hypertension in Adult Female

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Abstract:

Hyperlipidemia is one of the most widespread medical disorder in female and its complications are increasing all over the world, leading to life threatening medical problems like cardiovascular diseases, stroke and peripheral vascular diseases. An association between hyperlipidemia and hypertension has been identified. The study was carried out to observe the association of serum triglycerides and total cholesterol level with hypertension in adult female subjects. This cross sectional study was conducted in the Department of Physiology, Dhaka Medical College, Dhaka, during the period of January 2011 to December 2011. A total number of sixty female subjects were selected with age ranging from 30 to 50 years. Among them 30 female subjects with hypertension were included from the out-patient department of Medicine, Dhaka Medical College Hospital, Dhaka as study group (B) and 30 apparently healthy females were taken as control group (A) for comparison. Estimation of fasting serum triglycerides (TG) and total cholesterol (TC) levels were done by enzymatic method in the Department of Physiology, Dhaka Medical College Dhaka in both groups. Systolic blood pressure and diastolic blood pressure of all participants were recorded by standard method. Data were analyzed by Unpaired Student's- test and Pearson's correlation co-efficient (r) test as applicable. The values of fasting serum TG level were significantly higher in subjects with hypertension than those of control. Fasting serum TC level was significantly higher in subjects with hypertension in comparison to controls also. In subjects with hypertension fasting serum TG and fasting serum TC levels were higher and showed positive correlation with systolic and diastolic blood pressure. Present study revealed that systolic and diastolic blood pressure has positive relationship with fasting serum triglyceride (TG) and fasting serum cholesterol (TC) levels.

Key words: *Fasting serum triglyceride, fasting serum total cholesterol, systolic blood pressure, diastolic blood pressure.*

Introduction:

Hyperlipidemia is the most important risk factor for premature cerebrovascular disease, stroke and peripheral vascular disease[1]. Hyperlipidemia stands for abnormal elevation of various lipids and lipoprotein in the blood thus leading to atherosclerosis of arteries, especially in the aorta, coronaries, carotids and cerebral arteries.

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Hypercholesterolemia and hypertriglyceridemia both constitute major risk factor for cardiovascular diseases[2].

Hyperlipidemia accounts for an estimated 54% of all strokes and 50% of all ischemic heart diseases[3]. A study in the USA found approximately one in every six adults has hyperlipidemia or high cholesterol in their blood. People with high cholesterol have twice the risk of heart disease than people with lower level of cholesterol but they are unaware of this condition because there are no symptoms[4].

The prevalence of hypertension is increasing throughout the world. It is known as "the silent killer", it may exit for prolonged periods without symptoms and may manifest only after causing serious complications[5]. Hypertension is a recognized modifiable risk factor of cardiovascular disease, stroke and end stage renal disease[6].

A number of studies have shown the relationship between hyperlipidemia and hypertension. They found that patients with hyperlipidemia have more chance for development of hypertension.[7-12.] In developed countries at least one third of all cardiovascular disease (CVD) is due to elevated blood pressure and hyperlipidemia. They suggested that arterial hypertension is frequently observed in combination with elevated triglyceride and cholesterol[13.]

It has been shown that raised triglyceride (TG) level is a key feature of the lipid disturbance that is more strongly associated with hypertension. Higher TG has a link for development of central obesity and insulin resistance and both

are important factors for development of hypertension[14]. The risk associated with triglycerides is more in women than men. Studies comparing the risk from high triglycerides in men and women found the risk of coronary heart diseases approximately six times higher for men and more than fifteen times higher for women. They also suggested that high TG level have several metabolic effects on lipids. It decreases high density lipoprotein cholesterol, increases small dense low density lipoprotein, increases serum viscosity, hypercoagulability and endothelial dysfunction. All these factors are associated with development of hypertension[15,16]. Again, some investigators suggested that the prevalence and severity of atherosclerosis and ischemic heart diseases are related to hyperlipidemia more specifically hypercholesterolemia which is a major risk factor for the development of hypertension. Hypercholesterolemia causes endothelial injury, resulting in adhesion of platelets and monocyte, release of growth factors which lead to smooth muscle cell migration and proliferation resulting in formation of atherosclerotic plaques and development of hypertension.[12,17]

A study was done in Western Europe and they found that at least one-third of the population with hyperlipidemia has hypertension and people with hypertension also show evidence of hyperlipidemia. Thus hypertension and hyperlipidemia are common intertwined condition that shares a significant overlapping risk factors and complications[18]. A number of studies have shown the relation between elevated triglyceride (TG) and total cholesterol (TC) level and hypertension. They reported that lowering hyperlipidemia through diet and by using lipid lowering agents have been associated with reduction in blood pressure. They also suggested that several neuro-endocrine mechanisms are involved in these relations. The most investigated mechanisms are excess sodium retention, renin angiotensin aldosterone system, oxidative stress, endothelial dysfunction and increased production of endothelin-1[19][20]. Some studies have been done in this regard in abroad, but no published data has yet been available on this aspect in our country. Therefore, the present study has been designed to observe the association of serum TG and TC level with hypertension in adult females. Better understanding of these metabolic changes of hyperlipidemia will help in the detection of women at risk for future cardiovascular diseases and renal diseases. Treatment and preventive measure can reduce the risk of developing cardiovascular diseases and thus reduce burden on our health budget.

Methods

This cross sectional study was conducted in the Department of Physiology, Dhaka Medical College during the period of January 2011 to December 2011. Thirty females of 30-50 years of age range with hypertension were included in this study as study (B) group. They were selected from out-patient Department of Medicine of Dhaka Medical College Hospital, Dhaka. Age matched thirty apparently healthy females were studied as control (A) group for comparison. Subjects having history of heart, liver, kidney diseases, endocrine disorders and women taking hormone replacement therapy, steroid, alcohol user and smoker were excluded from the study. After selection of the subjects, the objectives, nature, purpose and benefit of the study were explained to the subjects in details. Permission was taken from the ethical committee of Dhaka Medical College. After selection of subjects, the objectives, nature, purpose and benefit of the study were explained to the subjects in details. Written informed consents were taken from the participants. They were encouraged for voluntary participation. They were also allowed to withdraw from the study whenever they feel like. Detailed medical history, menstrual history and family history of the subjects were taken and recorded in a pre-designed data collection form. The subjects were advised to attend the laboratory in the Department of Physiology of Dhaka Medical College, Dhaka between 8AM to 10AM in fasting state. Detailed history and complete physical examination were done. Then with all aseptic precautions 5 ml of venous blood was drawn from antecubital vein by disposable plastic syringe. Blood was allowed to clot and then centrifuged at rate of 3000 rpm and supernatant clear serum was separated. Serum was taken in a eppendrof tube and was preserved in refrigerator in the Department of Physiology of Dhaka Medical College, Dhaka. Fasting serum TG and TC level was estimated by enzymatic method in the laboratory. The blood pressure of the subjects was measured after 5 minutes rest with a sphygmomanometer using an appropriate cuff size in the right arm. Hypertension was defined according to the Joint National Committee (JNC) VII criteria.[13] Statistical analysis was done by Unpaired Student's t- test. Correlation was analyzed by Pearson's correlation co-efficient (r) test. P value <0.05 was taken as of significance.

Results

In this study the mean (±SD) of TG was 99.93±.32.37 mg/dl in group A and 238.77±.85.05 mg/dl in group B. The level of TG was significantly higher in group B than that of controls and the result was statistically significant (p<0.001). The mean (±SD) of TC was 130.20±.41.33 mg/dl in group A and 226.±35.24 mg/dl in group B. The level of TC was significantly higher in group B than that of controls and the result was statistically significant (p<0.001) (Table-I). Mean values of systolic blood pressure and diastolic blood pressure were higher in study group than those of controls and results were statistically significant (Table-II).

Serum fasting TG level showed positive correlation (r= 0.861) with systolic blood pressure in study group and result was statistically significant. Again, serum TG level showed positive correlation (r=0.936) with diastolic blood pressure in study group and result was also statistically significant (Table-III and Figure-1,2).

Again, fasting serum TC level showed positive correlation (r=0.926) with systolic blood pressure and also showed positive correlation (0.913) with diastolic blood pressure in study group and result was statistically significant. (Table IV and Figure 3,4).

Table I: Fasting serum triglyceride (TG) and Fasting serum total cholesterol (TC) in both groups

Groups	Number (n)	TG (mg/dl) (Mean±SD)	TC (mg/dl) (Mean±SD)
A (Control)	30	99.93±.32.37	130.20±.41.33
B (Study)	30	238.77±.85.05	226.±35.24
Groups		TG (p value)	TC (p value)
A vs B		0.001	0.001

Table II: Systolic blood pressure (SBP) and Diastolic blood pressure (DBP) in both groups

| Parameters	Group A (Control)	Group B (Study)
Systolic blood pressure (mmHg)	113.17±13.38	137.50±8.73.
Diastolic blood pressure (mmHg)	73.67±10.24	89.17±7.31
Groups	Systolic blood pressure (p value)	Diastolic blood pressure (p value)
A vs B	0.001	0.001

Table III: Correlation of fasting serum triglycerides level with SBP and DBP in study group (B)

Parameters	Group B (n=30)	
	r	p
Systolic blood pressure (SBP)	+ 0.861	0.001
Diastolic blood pressure (DBP)	+0.936	0.001
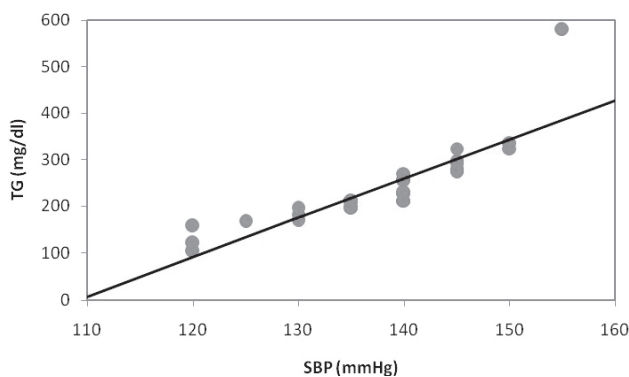
Figure 1: Correlation of serum TG with SBP in study group (n=30)

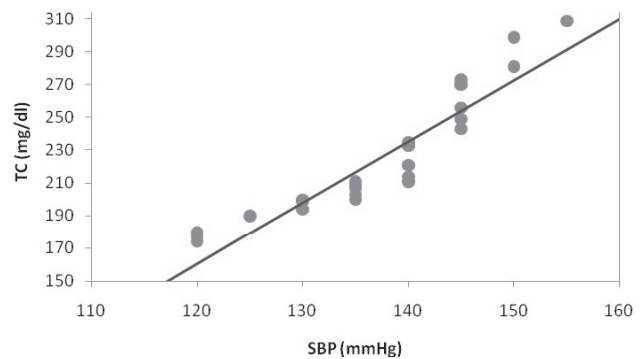(n = number of subjects in study group; r= 0.861; p= <0.001)
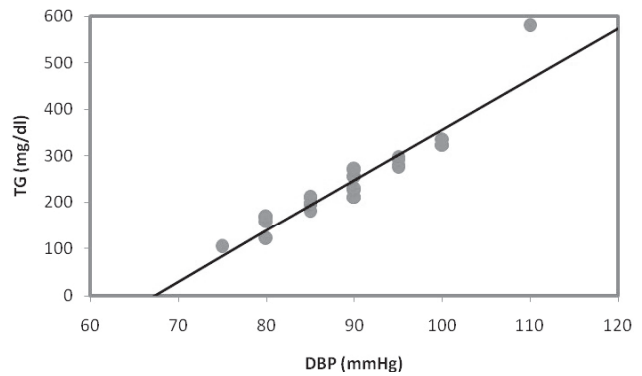
Figure 2: Correlation of Serum TG with DBP in study group (n=30)

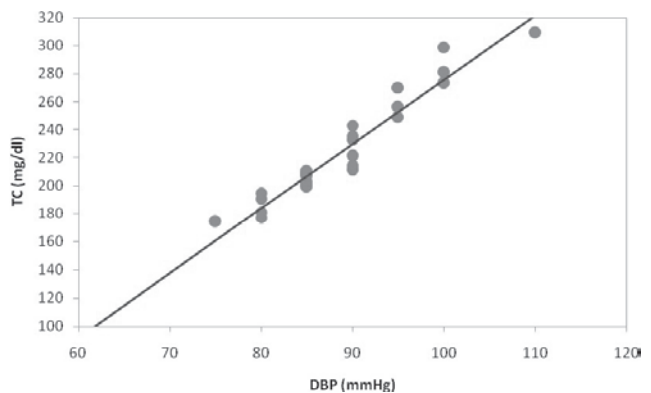(n = number of subjects in study group; r= 0.936; p= <0.0010

Table IV: Correlation of serum total cholesterol (TC) level with SBP and DBP in study group

Parameters	Group B (n=30)	
	r	p
Systolic blood pressure (SBP)	+0.926	0.001
Diastolic blood pressure (DBP)	+0.913	0.001

Figure 3: Correlation of serum TC with SBP in study group (n=30)

n = number of subjects in study group; r= 0.926; p= <0.001

Figure 4: Correlation of Serum TC with DBP in study group (n=30)

n = number of subjects in study group, r= 0.913; p= <0.001

Discussion

In the present study, the values of Triglyceride (TG) and Total Cholesterol (TC) in controls were almost within normal range and also similar to reported by the several investigators from abroad[2-5.]

In this study, TG in study group was higher than that of controls and result was statistically significant. Similar types of observations were found by other workers[2]-[8]. TC in study subjects was also higher than those of controls and the result was statistically significant. Similar types of findings were reported by different researchers of different countries [2, 8.] On the contrary, similar observations were made by other researcher but they did not find any significant difference in TG between the groups. It may be due to different life style, nutritional and environmental variations in their study population[12].

Again, correlation analysis showed positive correlation of serum TG level with systolic and diastolic blood pressure. In addition, serum TC level also showed positive correlation with systolic and diastolic blood pressure. The results were statistically significant. Similar observations were also reported by some investigators.[16]

The exact possible mechanism regarding these observed effects cannot be revealed directly from present study. However, several investigators of different countries proposed various suggestions on these aspects, which might be cause of our present findings It has been suggested that elevated levels of triglycerides and total cholesterol are key reasons for the development of obesity, insulin resistance, hyperinsulinemia and hyperglycemia which are associated with development of hypertension in study subjects. They suggested that hyperinsulinemia associated with increased sympathetic activity leads to increase in circulating catecholamine ultimately leading to increase plasma renin activity. This high level of plasma renin elevates tissue angiotensin II. This angiotensin II is a potent vasoconstrictor and increases the total peripheral resistance, ultimately increases blood pressure. Again, angiotensin II acts directly on kidney and increases water and salt retention by the kidney and increases blood volume, cardiac output and blood pressure.[19-20]. In addition, hyperinsulinemia directly increases renal tubular reabsorption of sodium and also stimulates the synthesis of sodium retaining hormone aldosterone and ultimately hypertension develops. Similarly, hyperinsulinemia with insulin resistance increases intracellular Ca^{++} accumulation, secretion of growth factors and vascular smooth muscle cell proliferation resulting in narrowing of blood vessels and thereby development of hypertension[21]. Again, some researcher suggested that hypertriglyceridemia is one of the important factors for development of oxidative stress. Oxidative stress plays a key role in pathogenesis of atherosclerosis and also causes endothelial dysfunction. Endothelial dysfunction is associated with increase adhesion molecules production, decrease endothelial-dependent vasodilation and nitric oxide (NO) production. All these are important factors for development of hypertension. Moreover, oxidative stress also stimulates the renin -angiotensin -aldosterone system (RAAS) which is an important factor for development of hypertension in study subjects[22,23]. Again, high blood cholesterol may also lead to high blood pressure through increased salt sensitivity. Cholesterol accumulation in kidney cells increases sodium retention. Sodium retention can increase blood volume leading to increase blood pressure. Sodium retention also decreases the production of nitric oxide by endothelial cells thereby decreases the elasticity of arteries and increases the blood pressure[24,25].

In the present study, levels of TG and TC are higher in study group than those of controls. Furthermore, in the present study, fasting serum TG and TC levels, showed positive correlation with systolic and diastolic blood pressure in study group. These findings support the relationship between serum TG and TC levels and elevated blood pressure in study group.

Conclusion

From this study, it can be concluded that hypertension present in the study group may be due to high level of serum triglyceride and total cholesterol.

Acknowledgement

References:

1. Islam Kamal, Hadir M Abdelkader. Dyslipidemia: the hidden sector of hypertension .IOSR Journal of Pharmacy.2016; 6(5):69-73.

2. GUO Zr, Hu Xr,Wu M,Zhou MH,Zhou Zy. A prospective study on the association between dyslipidemia and hypertension. National Institute of Health. 2009; 30 (6): 554-562.

3. Sritara P, Patoomanunt P, Wood.Ward M, Narksawnt K. Association between serum lipids and cases of mortality in a cohort of 3499 urban Thais. Angiology. 2008; 58(6): 757-763.

4. Sesso HD,Buring JE, Chown MJ, Ridker Pm, Gazino JM. A prospective study of plasma lipid and Hypertension in women. Arch Intern med.2008; 165(20): 2420-2427.

5. Toshiaki Otsuka, Hirotaka Takada, YasuhiroNishiyaama, Eitaro Kodani. Dyslipidemia and the risk of developing hypertension in working age in male population. J Am Heart Assoc.2016;10(1):1-9.

6. M.Tohidi, M.Hatami,F Hadaegh, F Azizi. Trigycerides and triglycerides to high density lipoprotein ratio are strong predictors of incident hypertension in middle Eastern women. Journal of Human hypertension.2012; 26:25-32.

7. Kearney PM, Whelton. M, Reynolds. K, Whelton. PK. Global burden of hypertension: analysis of world-wide data. Lencet.2005; 365:217-723.

8. B.Hozi, B.Paul Weber, F.Sandhofer J.R Patsch. Hypertriglyceridemia and hypertension. Journal of internal medicine. 1998; 243:79-81.

9. M.Flesch, A. Sachinidis, Y.D. Ko, raft, H. Vetter. Plasma lipids and lipoproteins and essential Hypertension.Clin Investig.1994; 72:944-749.

10. Patrick McBride. Triglycerides and risk for coronary Artery disease. Atherosclerosis.2008; 10(5)): 386-389.

11. David E. Laaksonen, Leo Niskanen, Kristiina Nyyssonen, Timo A.Lakka, Jari A.Laukkanen. Dyslipidemia as a predictor of hypertension in middle-aged men. European heart journal.2008; 29(20):2561-2568.

12. Jukka T.Salonen, Timo A.lakka, Hanna-Maaria Lakka,Veli-pekka valknen, Susan A.Everson. Hyperinsulinemia is associated with the incidence of hypertension and dyslipidemia in middle aged men.Diabetes.1998; 47: 270-277.

13. Marco polo Dias Freitus, Antonio Ignacio de Loyola Filho, Maria Fernanda Lima Costa. Dyslipidemia and the risk of incident hypertension in a population of Community-dwelling Brazilian elderly: the Bambui cohort study of ageing. Cad. Saude publica, Rio de Janeiro.2011; 27(3):351-359.

14. Chobanian AV, Bakris GL, Black HR, Cushan WC, Green LALzzo JL.The seventh Report of Joint National Committee on Prevention, Detection, JNC report.JAMA.2003;289(19):2560-2572.

15. Vijay Bhalavi, Pradeep Deshmukh, Manisha Atram, Bhushan Mahajan..Hypertension and Hyperlipidemia in the Adolescent of Central India. International journal of recent Trends in Science and Tecchnology.2014;10(3):495-497.

16. Daniele Versari, Mario Gossl, Dallit Mannheim, Elena Daghini, Offer Galili, Claudi Napoli et al. Hypertension hypercholesterolemia differentially affect the function and structure of Pig Carotid artery. Hypertens. 2007; 50: 1063-1068.

17. Jose Tunon, Jose Luis Martin-Ventura, Luis Miguel Blanco-Colio, Nieves Tarin, Jesus Egido. Common pathways of hypercholesterolemia and hypertension leading to atherombosis: the need for a global approach in the management of cardiovascular risk factors. Vascular Health and risk management.2007; 3(4): 521-526.

18. Branisalva ivanovic, Marijana Tadic. Hypercholesterolemia and Hypertension: Two sides of the same coin. American journal of cardiovascular drugs.2015; 15(6):403-514.

19. Yoshihide asato, Keisuke Katsuren,Tadashi Ohshiro, Kazuhide Kikawa,Tadao Shimabukuro, Takao Ohta. Relationship between lipid abnormalities and insulin resistance in Japanese school children. Arterioscler Thromb Vasc Biil. 2006; 26:2781-2786.

20. AK Al-Mahmood, Sf Afrin, N Hoqe. Dyslipidemia in insulin resistance: Cause or Effects. Bangadesh J Med Biochem.2014; 7(1):27-31

21. Salvetti A, Broji G, Dileje V, Bernini GP. The inter-relationship between insulin resistance and hypertension.1993; 46(2):149-159.

22. M.J.Veerkamp, J. de Graaf, A.F.H. Stalenhoef. Role of insulin resistance in familial combined Hypertension. Arterioscler Thromb Vasc Biol.2005; 25(1):1026-1031.

23. Surapon Tangvarasiittichi. Oxidative stress, insulin resistance, dyslipidemia and type 2 diabetes mellitus. World Journal of Diabetes 2015; 6(3):456-480.

24. Hyder A. AL-Aubaidy, Herbert Oxidative stress and Triglycerides as predictors in Prediabetes. Redox Report.2014; 19(2):87-91.

25. Michel R. Peluso. Hypercholesterolemia and hypertension. Am J Cardiovascular Drugs.2015; 15(6):403-417.