# Association of Serum Low Density Lipoprotein Cholesterol and High Density Lipoprotein Cholesterol with Hypertension in Adult Female

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### Abstract

**Background:** Hypertension is a chronic and debilitating disease. Its complications are increasing all over the world, leading to life threatening medical problems like cardiovascular diseases, stroke and end stage renal diseases. An association between hyperlipidemia and hypertension has been identified.

**Objectives:** The study was carried out to observe the association of serum low density lipoprotein cholesterol and high density lipoprotein cholesterol level with hypertension in adult female subjects.

Method: 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 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 serum fasting serum low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C) 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.

**Result:** The value of fasting serum LDL level was significantly higher in subjects with hypertension than those of control. Fasting serum HDL level was significantly lower in subjects with hypertension in comparison to control. In subjects with hypertension fasting serum LDL-C showed positive correlation with systolic and diastolic blood pressure and serum HDL-C showed negative correlation with systolic and diastolic blood pressure.

**Conclusion:** Present study revealed that systolic and diastolic blood pressure has positive relationship with low density lipoprotein cholesterol (LDL-C) and negative relationship with high density lipoprotein cholesterol (HDL-C) levels.

**Key words:** Fasting serum low density lipoprotein, fasting serum high density lipoprotein, systolic blood pressure, diastolic blood pressure.

# Introduction

Hypertension (HTN) remains a major modifiable risk factor for cardiovascular diseases. High blood pressure increases the risk of cardiovascular diseases for millions of people worldwide and there is evidence that the problem is only getting worse<sup>1</sup>. Hypertension is a major public health problem in many parts of 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. It has been identified as the most common, most potent and most universal contributor to cardiovascular mortality, which accounts for 20-50% of all death.2 It is the first sign of a chronic progressive process that may end in serious and potentially fatal complications such as stroke or renal failure and it is a major risk factor of coronary artery disease.3 Again, hypertension is the principal risk factors for mortality and second leading cause of disability of the life around the world. It is the most common chronic disease in the elderly with prevalence of about 60% in the America and Caribbean<sup>4</sup>. 90% causes are idiopathic, but a number of factors increase the blood pressure including obesity, insulin resistance, hyperlipidemia, high alcohol intake, high salt intake,

sedentary life style, stress<sup>5</sup>. Among the other causes, hyperlipidemia one of the important factor in developing hypertension<sup>6</sup>.

Hyperlipidemia is a medical condition characterized by an elevation of any or all lipid profile or lipoproteins in the blood. It can significantly increase the risk of developing cardiovascular disease including disease of blood vessels supplying the heart (coronary heart disease), brain (cerebrovascular disease), and limbs (peripheral vascular disease). These conditions can in turn lead to chest pain, heart attack, stroke and other problems<sup>6,7</sup>.

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

There are five major type of lipoprotein. Among them low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C) are most important

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cholesterols that carry lipid to and from cells Low density lipoprotein cholesterol (LDL-C) is called "bad" cholesterol, because it contributes to build up fatty plaque in arteries and raise the risk for heart attack, stroke and peripheral artery diseases. Again high density lipoprotein cholesterol (HDL-C) is called "good "cholesterol because it acts as a scavenger, carries lipid away from the arteries and back to the liver. There it's broken down and passed from the body. So increased level of HDL-C protects against heart attack and stroke9.

A number of studies have found the relationship between hyperlipidemia and hypertension. They found that patients with hyperlipidemia have more chance for development of hypertension. In developed countries at least one third of all cardiovascular vascular disease (CVD) is due to elevated blood pressure and hyperlipidemia. They suggested that arterial hypertension is frequently observed in combination with elevated low density lipoprotein cholesterol (LDL-C) and low level of high density lipoprotein cholesterol (HDL-C)<sup>10-14</sup>.

A study was done in Western Europe and they found that at least one-third of the population with hyperlipidemia have hypertension and patient with hypertension also show evidence of hyperlipidemia. Thus hypertension and hyperlipidemia are common intertwined condition that shares a significant overlapping risk factors and complications<sup>15</sup>.

It has been shown that high level of LDL-C impairs endothelial cell function. This endothelial injury increases permeability and accumulation of LDL-C in intima of blood

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vessels, then LDL-C is oxidized by free radicals and oxidized LDL-C is ingested by macrophage and transformed into foam cells. These lipid laden foam cells release cytokines interlukin-1 and monocyte chemotactic protein which creates a stimulus for a recruitment of additional mononuclear cells. These mononuclear cells increase secretion of growth factors; these cause smooth muscle cells proliferation. As a result, narrowing of blood vessels occurs and atherosclerosis develops<sup>16</sup>. A study found that low level of HDL-C contributed to structural and functional alteration and which are associated with increased blood pressure Again, low HDL-C are associated with endothelial dysfunction and peripheral vasoconstriction. All these are important factors for development of hypertension<sup>17,18</sup>. In addition higher level of high density lipoprotein (HDL-C) protect against development of atherosclerosis by transport of cholesterol from the peripheral tissues back to liver for subsequent catabolism and excretion. So HDL-C helps in transport of cholesterol from the arterial wall thus retards progression of atherosclerosis<sup>19</sup>. Furthermore, HDL-C stimulate nitric oxide (NO) production and inhibit adhesion of monocytes to endothelium<sup>20</sup>. So, low level HDL-C 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<sup>21</sup>.

Some studies have been done on 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 LDL-C and HDL-C level with hypertension in adult female. Better understanding of these metabolic changes with 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

# Method

This cross sectional study was conducted in the department of Physiology, Dhaka Medical College, Dhaka, during the period of January 2011 to December 2011. Thirty females of 30-50 years of age range with hyperlipidemia 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 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. Ethical permission was taken from 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. Ethical permission was taken from ethical committee of Dhaka Medical College. 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. Detailed personal history, medical history, drugs history, family history and all the clinical examinations were done. Then with all aseptic precautions 5ml 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 an eppendrof tube and was preserved in refrigerator in Department of Physiology of Dhaka Medical College, Dhaka. Fasting triglycerides, total cholesterol and high density lipoprotein level were estimated by enzymatic method in the laboratory of Department of Physiology of Dhaka Medical College and low density lipoprotein was calculated using the Fried wald equation. The blood pressure of the subjects was measured after five 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<sup>22</sup>. Statistical analyses 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.

### Result

In this study the mean ( $\pm$ SD) of low density lipoprotein was 77.53 $\pm$  16.72 mg/dl in group A and 134.20 $\pm$ 27.48 mg/dl in group B. The level of serum low density lipoprotein was significantly higher in group B than that of controls and the result was statistically significant (p<0.001). The mean ( $\pm$ SD) of serum high density lipoprotein was 58.27 $\pm$  10.09 mg/dl in group A and 42.60 $\pm$ 7.84 mg/dl in group B. The level of high density lipoprotein was significantly lower in group B than that of controls and the result was statistically significant (p<0.001)( Table-I).

In this study the mean ( $\pm$ SD) of systolic blood pressure was 113.17 $\pm$  13.38 mm/Hg in group A and 137.50 $\pm$ 8.73 mm/Hg in group B. The level of systolic blood pressure was significantly higher in group B than that of controls and the result was statistically significant (p<0.001). The mean ( $\pm$ SD) of diastolic blood pressure was 73.67 $\pm$  10.24 mm/Hg in group A and 89.17 $\pm$ 7.31 mm/Hg in group B. The level of diastolic blood pressure was significantly higher in group B than that of controls and the result was statistically significant (p<0.001)(Table-II).

Serum fasting LDL-C level showed positive correlation (r=0.911) with systolic blood pressure in study group and result was statistically significant. Serum LDL-C level also showed positive correlation (r=0.952) with diastolic blood pressure in study group and result was also statistically significant (Table- III and Figure-1 & 2)

Again, fasting serum HDL-C level showed negative correlation (r=-0.752) with systolic blood pressure and also showed negative correlation (r=-0.734) with diastolic blood

pressure in study group and result was statistically significant (Table-IV and Figure 3 & 4).

**Table-I:** Fasting serum LDL-C and Fasting serum HDL-C in both groups

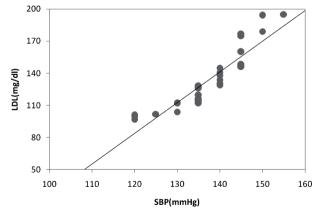
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Groups	n	LDL-C (mg/dl) (Mean±SD)	HDL-C (mg/dl) (Mean±SD)
A	30	77.53±.16.72	58.8.27±.10.09
В	30	$134.20 \pm .27.48$	$42.60.\pm7.84$
Statistica	al analysis		
Groups		LDL-C	HDL-C
		(p value)	(p value)
A vs B		0.001	0.001

**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

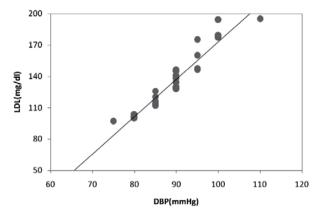
**Table-III:** Correlation of fasting serum LDL-C level with SBP and DBP in study group (B)

D	C D	(20)
Parameters	Group B	(n=30)
	r	p
Systolic blood pressure (SBP)	+0.911	0.001
Diastolic blood pressure (DBP)	+0.952	0.001



n = number of subjects in study group; r = 0.911; p = <0.001

**Figure-1:** Correlation of serum LDL-C with SBP in study group (n=30)

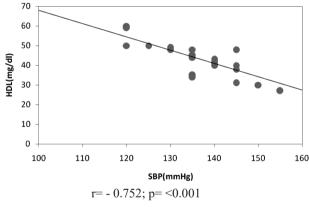


n = number of subjects in study group, r= 0.952; p= <0.001

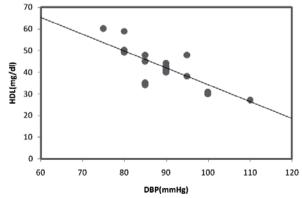
**Figure-2:** Correlation of serum LDL-C with DBP in study group (n=30)

**Table IV:** Correlation of serum HDL-C level with SBP and DBP in study group

Parameters	Group B r	(n=30)
Systolic blood pressure (SBP)	-0.752	0.001
Diastolic blood pressure (DBP)	-0.734	0.001



**Figuer-3:** Correlation of serum HDL-C with SBP in study group (n=30)



n = number of subjects in study group; r= -0.734; p=<0.001 **Figure-4:** Correlation of serum HDL-C with DBP in study group (n=30)

### Discussion

In the present study, the values of fasting low density lipoprotein (LDL-C) and high density lipoprotein (HDL-C) in control were almost within normal range and also similar to reported by the several investigators from abroad<sup>8-12</sup>.

In this study, LDL-C in study group was higher than those of controls and result was statistically significant. Similar types of observation were found by other workers<sup>8-12</sup>. The level of HDL-C in study subjects were lower than those of control and result was statistically significant. Similar types of findings were reported by different researchers of different countries<sup>8,10</sup>. On the contrary, similar observations were made by other researcher but they did not find any significant difference in LDL-C level between the groups. It may be due to different life style, nutritional and environmental variations in their study population<sup>13</sup>.

Again, correlation analysis showed positive correlation of serum LDL-C level with systolic and diastolic blood pressure. The results were statistically significant. Similar observations were also reported by some investigators. But serum HDL-C level showed negative correlation with systolic and diastolic blood pressure. The results were statistically significant. Similar observations were also reported by some investigators 16,23.

Hyperlipidemia are well established and overlapping risk factors for developing hypertension in adult female. Elevated LDL-C and low HDL-C level are key factors in developing atherosclerosis and hypertension in study subject. The exact possible mechanisms 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.

Increased LDL-C may impair endothelial function which may consequently disrupt production of nitric oxide and regulation of blood pressure. Increased LDL-C may predispose individuals to development of hypertension by reducing baroreflex sensitivity. Increased LDL-C decreases dispensability of large elastic arteries. This may reduce the wind Kassel reflex which in turn increase blood pressure. Again, LDL--C causes injury of renal microvasculature and increased blood pressure<sup>24</sup>.

Moreover, elevated level of LDL-C and low HDL-C are key reason for the development of insulin resistance and hyperinsulinemia which are associated with development of hypertension in study subjects. They suggested that hyperinsulinemia associated with increased sympathetic activity which leads to increase in circulating catecholamine. Increase in circulating catecholamine elevates the 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 increase blood pressure. Again, angiotensin II acts directly on kidney and increase water, salt retention by the kidney and increase blood volume, cardiac output and blood pressure<sup>25</sup>. In addition, hyperinsulinemia directly increase renal tubular

reabsorption of sodium and also stimulates the synthesis of sodium retaining hormone, aldosterone and ultimately hypertension develops<sup>26,27</sup>. Similarly, hyperinsulinemia with insulin resistance increase intracellular Ca++ accumulation, increases secretion of growth factors and vascular smooth muscle cell proliferation, as a result narrowing of blood vessels and hypertension develops<sup>28,29</sup>. Again, some researcher suggested that decreased level of HDL-C is one of the important factor for development of oxidative stress. Oxidative stress plays a key role in pathogenesis of atherosclerosis and also causes endothelial dysfunction. Endothelial dysfunction associated with increase adhesion molecules, decrease endothelial-dependent vasodilation and nitric oxide (NO) production. All these are important factors for development of hypertension in study subjects<sup>30</sup>. A study found that low level of HDL-C contributed to structural and functional alteration and also has antithrombotic. anti-inflammatory and antioxidant effects<sup>31</sup>.

In the present study, level of low density lipoprotein (LDL-C) are higher and level of high density lipoprotein are lower in study group than that of control. Furthermore, in the present study, fasting serum LDL-C level showed positive correlation with systolic and diastolic blood pressure and HDL-C level showed negative correlation with systolic and diastolic blood pressure in study group. These finding supports the relationship between hyperlipidemia and elevated blood pressure in study group.

# Conclusion

From this study, it can be concluded that hypertension, present in study group, may be due to high level of serum LDL-C and low level of HDL-C.

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# Reference

- Oscar AC, Oparil. Essential hypertension: Definition and Etiology. Circulation 2000: 101(3): 329-335.
- AKI OA, Khairy AE, Abdel-Aal NM, Deghedi BS, Amer ZF. Knowledge, Attitude, Practice and Performance of Family Physician Concerning Holistic Management of Hypertension. J Egypt Public Assoc. 2006; 81(5-6): 337-53
- Kalavathy MC, Thankappan KR, Sasma PS, Vasan PS. Prevalence, awareness, treatment and control of HTN in an elderly community based sample in kerala, India. Nalt Med J India 2000; 13(1):9-15.
- WHO. Hypertension control. Technical Report Series No 862, Geneva: WHO 1996: 3-20.
- Kannel WB. Blood pressure as a cardiovascular risk factor.JAMA 1996; 275(20): 1571-6.
- Bassam Abdul Rasool Hassan. Overview on Hyperlipidemia.J Chromat Separation technique2013; 4(2): 12-6.
- 7. Lucy B.Adams.Hyperlipidemia.Am Fam Physician2000; 61(3):

- 109-15.
- Islam Kamal, Hadir M Abdelkader. Dyslipidemia: the hidden sector of hypertension.IOSR Journal of Pharmacy 2016; 6(5):69-4.
- 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): 554-8
- Francisco J. Felix-Redondo, Maria Grau, Daniel Fernandez-Berges. Chloesterol and cardiovascular disease in the eldery. Aging and disease 2013; 4(3):154-5.
- 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): 757-63.
- 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): 2420-7.
- Toshiaki O, Hirotaka T, YasuhiroN, Eitaro K.Dyslipidemia and the risk of developing hypertension in working age in male population. J Am Heart Assoc. 2016; 11(1):1-9.
- Borghi C, Veronesi M, Baccheli S, Esposti DD. Cosentono E. Serum cholesterol levels, blood pressure response to stress and incidence of hypertension in young subjects with blood pressure. J Hypertens. 2004;22(1):265-72.
- Derek Weyecker, Gregory A, Nichls, John E delsberg, Zebe M Khan, Satyin Kaura. Risk Factor clustering and cardiovascular risk in hypertensive patients. American Journal of hypertension 2007; 20 (6):599-8.
- Kumarv, Abbas, AK and Fauston, N. Rbbins and Cartan . Pathologic Basis of Disease. 7th ed. Elsivier Saunders Philadelphia.
- 17. Marco polo Dias Freitus, Antonio Ignacio de Loyola Filho, Maria Fernanda Lima Costa. Dyslipidemia and the risk of incident hypertension in apopulation of Community-dwelling Brazilian elderly: the Bambui cohort study of ageing. Cad. Saude publica, Rio de Janeiro 2011; 27(3):351-359.
- 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; 29:2561-7.
- 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: 270-7.
- M.Flesch, A. Sachinidis, Y.D. Ko, raft, H. Vetter. Plasma lipids and lipoproteins and Essential Hypertension. Clin Investig 1994; 72:044-5
- 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):495-3.
- Chobanian AV, Bakris GL, Black HR, Cushan WC, Green LALzzo
  JL.The seventh Report of the Joint National Committee on
  Prevention, Detection, JNC report. JAMA. 2003; 289:2560-72.
- Daniele V, Mario G, Dallit M, Elena D, Offer G, Claudi N, Liach O. Lerman. Hypertension hypercholesterolemia differentially affect the function and structure of Pig Carotid artery. Hypertens 2007; 50(1): 1063-68.
- 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): 521-25.

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- Daugherty A, Lult, Rateri DM, Classis LA. Augmentation of the renin-angiotensin system by hypercholesterol promotes vascular disease. Future Lipidol 2008; 3(6):625-35.
- 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; 26:2781-5.
- AK Al-Mahmood, Sf Afrin, N Hoqe. Dyslipidemia in insulin resistance: Cause or Effects. Bangadesh J Med Biochem 2014; 7(1):27-31
- Salvetti A, Broji G, Dileje V, Bernini GP. The inter-relationship between insulin resistance and hypertension 1993; 46(2):149-59.

- 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):1026-31.
- Alev E, Altinova, Fuson Toruner, Nuri Cakir. Decreased HDL is associated with oxidative stress in overweight subjects. Endocrine Journal 2007; 54(2):221-6.
- Fatiha tabet, Kery Anne Rye. High density lipoproteins, inflammation and oxidative stress. Clinical science 2009; 116 (2): 87-98.