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Evaluation of Role of Serum Lipoprotein and Lipid Profile in Essential Hypertension Patients in a Tertiary Care Hospital

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Abstract

Introduction: Hypertension is a leading cause of mortality and morbidity worldwide. It doubles the risk of cardiovascular diseases. Elevated serum lipoprotein (a) are associated with an increasing risk of cardiovascular disease in hypertensive patients.

Aims and Objectives: To determine levels of serum lipoprotein (a) and lipid profile in hypertensive patients and to correlate the serum lipoprotein (a) levels with lipid parameters.

Material and Methods: Lipid profile and serum lipoprotein (a) were estimated in 68 hypertensive patients and compared with 63 age and sex matched healthy controls. Serum lipoprotein (a) was estimated by immunoturbidometric assay and lipid profile by standard biochemical methods.

Result: Serum lipoprotein (a) was found to be significantly increased ($p<0.001$) in hypertensive patients (36.52 ± 9.34 mg/dl) as compared to healthy individuals (17.96 ± 8.42 mg/dl). Total cholesterol, LDL, triglycerides were significantly increased ($p<0.001$) and HDL was significantly decreased ($p<0.001$) in cases compared to controls. The correlation study between serum lipoprotein (a) and lipid profile in hypertensive cases showed, that only LDLc had a significant positive correlation with lipoprotein (a).

Conclusion: Elevated lipoprotein (a) in hypertensive patients can be a independent risk factor for development of cardiovascular disease.

Keywords: Essential hypertension; Lipoprotein(a); Atherosclerosis; Arterial hypertension; Lipid profile; Dyslipidemia

Introduction

Hypertension or high blood pressure has emerged as a leading cause of the global burden of disease in both developed as well as developing countries. Data from the National Health And Nutrition Examination Survey (NHANES) have indicated that 50 million or more Americans have high blood pressure warranting some form of treatment [1,2]. Worldwide prevalence estimates for hypertension may be as much as 1 billion individuals, and approximately 7.1 million deaths per year may be attributable to hypertension [3].

Hypertension is classified as either primary/essential hypertension or secondary hypertension. Primary/Essential hypertension is the form of hypertension that by definition has no underlying cause [4]. It is the most common cause of hypertension affecting 90-95% of total hypertensive patients. It is mostly of familial origin. Its prevalence increases with age. Secondary hypertension is caused by an identifiable underlying change. It is much less common than primary hypertension consisting only 5% of hypertensive individuals. It has many different causes including endocrine diseases, kidney diseases, tumours, also due to side effects of certain medications.

Essential hypertension remains a major modifiable risk factor for cardiovascular disease (CVD) despite important advances in our understanding of its patho-physiology and the availability of effective treatment strategies. High blood pressure (BP) increases the risk of CVD for millions of people worldwide. Several prospective studies have identified the major risk factors for hypertension like obesity, smoking and alcohol consumption, dyslipidemia apart from dietary patterns [5].

Lp(a) has been of interest in hypertensive patients, since epidemiological studies indicated it to be an independent risk factor for cardiovascular diseases. Lipoprotein (a) is a complex lipoprotein molecule that contains apolipoprotein (a), which shares homology with plasminogen [6,7]. It acts as a competitive inhibitor of tissue type plasminogen activator and there by helps in modulating the fibrinolytic system consistent with an atherogenic role [8,9]. Lp(a) levels are known to exhibit significant inter-individual variation and strictly under genetic control [10,11].

Elevated serum Lp(a) levels are associated with an increasing risk of cardiovascular disease and renal failure in hypertensive patients [12]. As Lp (a) levels are genetically determined, screening for Lp (a) levels in asymptomatic individuals has been suggested to identify the subjects at risk [13].

Elevated Lp (a) could be an independent risk factor for atherosclerosis, and could contribute towards increasing the incidence of cardiovascular disease in people with essential arterial hypertension. There are very limited case- control studies determining association between Lp (a) excess and essential hypertension. Therefore the aim of our present study is to measure the serum concentrations of lipoprotein (a) in a group of hypertensive patients and to find its association with blood pressure and lipid profile.

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Materials and Methods

The study was conducted in the Department of Biochemistry, S.C.B. Medical College and Hospital, Cuttack from February 2013 to August 2014. 68 patients of age group 35-74 years, attending OPD and indoor in the Department of Medicine, S.C.B. Medical College and Hospital, Cuttack, were included in the study. Patients with Hypertension were selected and diagnosed based on their history, physical examination, biochemical investigations and according to the JNC 7 (Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure) Criteria for the diagnosis of hypertension. 63 age and sex matched healthy adults with normal serum lipid profile, having no symptoms and signs suggestive of hypertension and also with no family history of the disease were selected as controls.

Three ml of blood was collected after overnight fasting of eight hours from all enrolled patients and healthy controls for the assessment of Lipoprotein (a) levels and other biochemical parameters like fasting plasma glucose, serum urea, creatinine, uric acid, lipid profile. Demographic characteristics (name, age, sex), history of risk factors (smoking, family history, medications, alcohol intake etc.), systolic and diastolic blood pressures, body mass index were recorded in detail (Table 1).

The inclusion criteria consists of Adults aged 35 years and above diagnosed with hypertension according to JNC 7 (Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure). Age and sex matched healthy adults with systolic blood pressure <120 mmHg and diastolic blood pressure <80 mmHg were taken as controls.

Patient with Diabetes Mellitus, renal disease, endocrinopathy, pregnancy Induced Hypertension, Immunosupression and history of illicit drug use were excluded from the study. In the control group, subjects with family history of hypertension, renal diseases and chronic metabolic diseases were excluded. All the case records were collected in a specified proforma. Written, informed consent was obtained from all subjects. The instruments used were automated Clinical Analyzer Biolis24i Premium (Tokyo Boeki Machinery Ltd.) and Ecolyte (ISE) (Table 2).

Table 2 Depicts the mean age to be 56.11 ± 6.84 yrs in healthy individuals in the control group (n=63); 55.72 ± 6.58 yrs in stage 1 hypertensives (n=39); and 56.9 ± 7.25 in stage 2 hypertensives (n=29). The mean body mass index was 21.67 ± 1.77 kg/m² in control group; 25.06 ± 1.95 kg/m² in stage 1 hypertensives; 25.91 ± 2.11 kg/m² in stage 2 hypertensives. In both stage 1 and 2 hypertensives the BMI values were found to be statistically significant ($p<0.001$) when compared to control.

Discussion

The present study was carried out in the Department of Biochemistry S.C.B. Medical College and Hospital, Cuttack from February 2013 to August 2014 in collaboration with the Department of Medicine, S.C.B. Medical College and Hospital, Cuttack. The study has been approved by Institutional Ethical Committee.

All statistical analyses was performed using SPSS version 16.0 software and Microsoft Office Excel 2007. Data are expressed as mean ± SD. Student's t-test and Anova test were used to compare mean values and calculate significance. Data were considered statistically significant if p values were <0.05. Pearsons coefficient of correlation was used to assess linear correlation between serum lipoprotein-a and other variables.

In this study the mean age of stage 1 hypertensives was 55.72 ± 6.58 years, stage 2 hypertensives was 56.9 ± 7.25 years and of healthy individuals was 56.11 ± 6.84 years (Table 2). The age distribution was in consistent with that of Catalano et al. [14].

A significantly higher level of BMI was found in stage 1 and 2 hypertensives when compared to healthy controls (Table 2). This finding was in accordance to that of Charles U Osuji et al. [15] Gowda et al. [16] and Gupta et al. [17]. It is due to the fact that increased BMI is associated with an increase in plasma volume and cardiac output. Thus obesity is a risk factor for hypertension. Table 3 shows mean systolic

	Control Group			Study Group (n=68)					
Age Group	**(n=63)**			**Stage 1 hypertensives**			**Stage 2 hypertensives**		
(In Yrs.)	M	F	Total	M	F	Total	M	F	Total
35 – 42	2	1	3	1	1	2	-	1	1
43 – 50	3	4	7	3	2	5	3	2	5
51 – 58	17	10	27	16	5	21	4	4	8
59 – 66	12	10	22	7	2	9	5	7	12
67 – 74	2	2	4	1	1	2	1	2	3
TOTAL	36	27	**63**	28	11	**39**	13	16	**29**

Table 1: Age and sex distribution in control and cases. It shows the age, sex distribution of hypertensive cases and healthy individuals. The control group consisted of 63 healthy individuals consisting of 36 males and 27 females. The study group consisted of 68 hypertensive patients, out of which 39 were stage 1 hypertensive cases and 29 were stage 2 hypertensives. The stage 1 hypertensives consisted of 28 males and 11 females; the stage 2 hypertensives consisted of 13 males and 16 females. Majority of stage 1 mhypertensives were in the age group of 51-58 yrs and majority of stage 2 hypertensives were in the age group of 59-66 yrs.

			Study Group (n=68)	
Sl No.	**Parameter**	**Control Group**		
		(n=63)	**Stage 1 hypertensive (n=39)**	**Stage 2 hypertensive (n=29)**
		Mean ± SD	Mean ± SD	Mean ± SD
1	Age (in yrs.)	56.11 ± 6.84	55.72 ± 6.58	56.9 ± 7.25
2	BMI (Kg/m²)	21.67 ± 1.77	25.06 ± 1.95*	25.91 ± 2.11*

*Statistically significant ($p<0.001$) as compared to control group

Table 2: Characteristics of control group and study group.

and diastolic blood pressure (SBP and DBP) in control and cases. Mean SBP and DBP in stage 1 and stage 2 hypertensives was significantly raised when compared to SBP and DBP of control. Table 3 shows the systolic and diastolic blood pressure in control and study

group. The mean SBP was 126. 38 ± 4.87 mmHg in control group; 151.08 ± 6.03 mmHg in stage 1 hypertensives; 169.86 ± 9.23 mmHg in stage 2 hypertensives. The mean diastolic blood pressure was 80.38 ± 3.17 mmHg in control group; 92.26 ± 3.82 mmHg in stage 1 hypertensives; 103.93 ± 5.46 mmHg in stage 2 hypertensives.

The systolic and diastolic blood pressures were found to be significantly higher ($p<0.001$) in both stage 1 and 2 hypertensives as compared to control group.

This finding was in accordance with the studies of, Bhavani BA, et al. [5], Charles U. Osuji, et al. [15], Kashem et al. [18] and Pooja et al. [19]. Blood pressure has a continuous and consistent relationship with the risk of cardiovascular events; the higher the BP, the higher the chance of CVD. The presence of each additional risk factor multiplies the risk for hypertension (Tables 4-6).

Elevated SBP confers significantly higher risk of coronary heart disease mortality than elevated DBP or combined systolic/diastolic hypertension [20,21] especially with advancing age. As age increases, mean blood pressure levels tend to rise and the prevalence of hypertension increases. After age 60, however, mean diastolic pressures tend to plateau or fall, whereas systolic pressures continue to increase [22]. Because the majority of coronary heart disease events and cardiovascular morbidity occur in older individuals, the result is that there is also a greater attributable risk conferred by SBP elevation than by DBP elevation. The rise in systolic blood pressure with ageing is mainly caused by an increase in vascular stiffness of the great arteries in combination with atherosclerotic changes in the vessel wall.

Of the lipid parameters in hypertensive cases and control, a significantly higher level of total cholesterol, triglycerides and LDL was found in stage 1 and 2 hypertensives when compared to controls. While serum HDL was significantly reduced.

Hypertension and dyslipidemia are two of the main risk factors for vascular diseases and are often associated. The co-existence of the two risk factors has more than an additive adverse impact on the vascular

SL. No.	Parameter (mmHg)	Control group (n=63)	Study group (n=68)	
			Stage 1 hypertensive (n=39)	Stage 2 hypertensive (n=29)
		Mean ± SD	Mean ± SD	Mean ± SD
1.	SBP	126.38 ± 4.87	151.08 ± 6.03*	169.86 ± 9.23*
2.	DBP	80.38 ± 3.17	92.26 ± 3.82*	103.93 ± 5.46*

*Statistically significant ($p<0.001$) as compared to control group.SBP=Systolic blood pressure_DBP=Diastolic blood pressure

Table 3: Systolic and Diastolic Blood Pressure in Control and Cases.

Sl. No.	Parameter (mg/dl)	Control group (n=63)	Study group (n=68)	
			Stage 1 hypertensive (n=39)	Stage 2 hypertensive (n=29)
		Mean ± SD	Mean ± SD	Mean ± SD
1.	FPG	93.06 ± 13.28	94.33 ± 13.39	98.38 ± 12.87
2.	Serum Urea	34.57 ± 9.86	35.03 ± 8.63	41.9 ± 9.20
3.	Serum Creatinine	1.06 ± 0.25	1.05 ± 0.24	1.16 ± 0.24
4.	Serum Uric acid	5.2 ± 1.0	6.13 ± 1.08*	6.12 ± 1.08*

+statistically significant ($p<0.01$) as compared to control *statistically significant ($p<0.001$) as compared to control FPG=Fasting plasma glucose

Table 4: Biochemical parameters in control group and study group.

Sl. No.	Parameter (mg/dl)	Control group (n=63)	Study group (n=68)	
			Stage 1 hypertensive (n=39)	Stage 2 hypertensive (n=29)
		Mean ± SD	Mean ± SD	Mean ± SD
1.	Total Cholesterol	160.48 ± 23.73	223.13 ± 37.15*	228.03 ± 30.84*
2.	Triglycerides	136.11 ± 33.97	156.72 ± 38.16**	167.07 ± 24.31*
3.	HDLc	44.83 ± 6.70	40.49 ± 6.34**	36.45 ± 5.53*
4.	LDLc	88.42 ± 18.07	151.30 ± 36.52*	158.17 ± 30.98*
5.	VLDLc	27.22 ± 6.79	31.34 ± 7.63**	33.41 ± 4.86*

Statistically significant *$p< 0.001$, **$p< 0.01$ as compared to control

Table 5: Lipid profile of control group and study group. It shows the lipid profile parameters. The mean total cholesterol level was 160.48 ± 23.73 mg/dl in healthy controls ; 223.13 ± 37.15 mg/dl in type 1 hypertensives and 228.03 ± 30.84 mg/dl in stage 2 hypertensives. The mean serum triglycerides level was 136.11 ± 33.97 mg/dl in healthy controls; 156.72 ± 38.16 mg/dl in type 1 hypertensives and 167.07 ± 24.31 mg/dl in type 2 hypertensives.

The serum HDLc levels in controls, type 1 and 2 hypertensives were 44.83 ± 6.70 , 40.49 ± 6.34 and 36.45 ± 5.53 mg/dl respectively. The serum LDLc levels in controls, type 1 and 2 hypertensives were 88.42 ± 18.07, 151.30 ± 36.52 and 158.17 ± 30.98 mg/dl respectively. The serum VLDLc levels in controls, type 1 and 2 hypertensives were 27.22 ± 6.79, 31.34 ± 7.63 and 33.41 ± 4.86 mg/dl respectively. Serum total cholesterol, triglycerides, LDLc, VLDLc were found to be significantly raised and HDLc was significantly lower when compared with control.

endothelium, which results in enhanced atherosclerosis, leading to CVD.

The exact mechanism by which a low HDL-C increases CVD risk has however not been fully elucidated, though experimental studies suggest a direct role for HDL-C in promoting reverse cholesterol transport from foam cells in the atherosclerotic plaque depots in blood vessels to the liver for excretion. HDL-C also exhibits potent anti-inflammatory and antioxidant effects that inhibit the atherogenic process [23,24]. It has additionally been shown that a low HDL-C level correlates with the presence of other atherogenic risk factor.

In the present study, 70.6% of hypertensive patients had Lp(a) levels ≥ 30 mg/dL when compared to 9.5% in controls, which, in general, is considered as a high-risk level for atherogenesis (Table 7). Bhavani et al. [5] found that in Indians 40.5% of hypertensive patients had Lp(a) >30 mg/dl when compared to 10% in controls. Catalano et al. [14] in their study found that in Caucasians only 13% of hypertensive patients had Lp(a) >30 mg/dL when compared to 8% in controls.

Elevated serum Lp (a) values could play an important role in essential hypertension pathogenesis and can be considered as an individual risk factor in hypertensive patients. Lp (a) could be an independent risk factor for atherosclerosis and can contribute towards increasing the risk for cardiovascular disease in persons with essential hypertension.

The correlation study between serum lipoprotein (a) concentrations and BMI in stage 1 and 2 hypertensives (Table 8) showed a positive correlation (r=0.024) in stage 1 hypertensives but it was not statistically significant (p=0.88). Similarly a positive correlation (r=0.035) was obtained between Lp(a) and BMI in stage 2 hypertensives but was not statistically significant (p=0.86).

The correlation study between serum lipoprotein (a) and systolic blood pressure (SBP) showed a significant positive correlation (r=0.390, p<0.05) in stage 1 hypertensives and in Stage 2 hypertensives (r=0.492, p<0.01) (Tables 9 and 10).

The correlation study between serum lipoprotein (a) and diastolic blood pressure (DBP) showed a significant positive correlation (r=0.444, p<0.01) in stage 1 hypertensives and in stage 2 hypertensives (r=0.415, p<0.05) (Tables 11 and 12).

The correlation between serum Lp(a) and lipid profile in stage 1 and 2 hypertensives showed that Total cholesterol, triglycerides, VLDLc were positively correlated while HDLc was negatively correlated with Lp(a) in both stage 1 and 2 hypertensives but were not found to be statistically significant (Table 13).

However, a significant positive correlation (r=0.321, p<0.05) was found in stage 1 hypertensives and in stage 2 hypertensives (r=0.379, p<0.05) between Lp(a) and LDLc (Table 13).

CATEGORY		MEAN ± SD(mg/dl)	CI (MEAN ± 2SE)(mg/dl)
control group(n=63)		17.96 ± 8.42	15.8 – 20.13
Study Group (N=68)	STAGE 1 HYPERTENSIVES (n=39)	32.61 ± 9.04*	29.86 – 35.36
	STAGE 2 HYPERTENSIVES (n=29)	36.52 ± 9.34*	33.31 – 39.74

*Statistically significant (p<0.001) as compared to control group.

CI=Confidence Interval

Table 6: Serum lipoprotein (a) levels in control group and study group. It shows the mean serum lipoprotein (a) concentration in the control group to be 17.96 ± 8.42 mg/dl with a 95% confidence interval of mean to be 15.8-20.13. Patients with stage 1 hypertension had a mean serum lipoprotein (a) concentration of 32.61 ± 9.04 mg/dl and 95% confidence interval of mean of 29.86-35.36. In patients with stage 2 hypertension the mean serum lipoprotein (a) level was found to be 36.52 ± 9.34 mg/dl and 95% confidence interval of mean was 33.31-39.74. The serum lipoprotein (a) concentrations were significantly higher in both stage 1 and 2 hypertension cases as compared to the healthy control group.

Lp(a) mg/dl	Controls (n=63)	Cases (n=68)
< 30.0	57 (90.5%)	20 (29.4%)
> 30.0	6 (9.5%)	48 (70.6%)

Table 7: Distribution of control and cases according to serum lp(a) levels. Table 7 shows the distribution of hypertensive cases and control according to serum Lp(a) levels. Out of the 63 normotensive controls, 57 (90.5%) have serum Lp(a) <30 mg/dl and only 6 (9.5%) have >30 mg/dl of serum Lp(a). Out of 68 hypertensive cases 20 (29.4%) have Lp(a) <30 mg/dl while 48 (70.6%) have Lp(a) > 30 mg/dl.

Serum lipoprotein (a) Vs Body Mass Index	r Value	p Value
Stage 1 hypertension	0.024	0.88
Stage 2 hypertension	0.035	0.86

Table 8: Correlation of serum lipoprotein (a) concentrations and bmi in stage 1 and stage 2 hypertension cases. Shows the correlation study between serum lipoprotein (a) concentrations and BMI in Kg/m² in stage 1 and stage 2 hypertension cases . A non-significant positive correlation was observed with a r value of 0.024 and p value of 0.88 in stage 1 hypertension and a non-significant positive correlation was observed with a r value of 0.035 and p value of 0.86 in cases of stage 2 hypertension.

Serum lipoprotein (a) Vs Systolic blood pressure (SBP)	r Value	p Value
Stage 1 hypertension	0.390	<0.05

Table 9: Correlation of serum lipoprotein (a) concentration and systolic blood pressure in stage 1 hypertension cases. It shows the correlation study between serum lipoprotein (a) concentration and systolic blood pressure (SBP) in mmHg in stage 1 hypertension cases. A significant positive correlation was observed with a r value of 0.390 and p value of <0.05 in stage 1 hypertension cases.

Serum lipoprotein (a) Vs Systolic blood pressure (SBP)	r Value	p Value
Stage 2 hypertension	0.492	<0.01

Table 10: Correlation of serum lipoprotein (a) concentration and systolic blood pressure in stage 2 hypertension cases. It shows the correlation study between serum lipoprotein (a) concentration and systolic blood pressure (SBP) in mmHg in stage 2 hypertension cases. A significant positive correlation was observed with a r value of 0.492 and p value of <0.01.

Serum lipoprotein (a) Vs Diastolic blood pressure (DBP)	r Value	p Value
Stage 1 hypertension	0.444	<0.01

Table 11: Correlation of serum lipoprotein (a) concentrations and diastolic blood pressure in stage 1 hypertension cases. It shows the correlation study between serum lipoprotein (a) concentration and diastolic blood pressure (DBP) in mmHg in stage 1 hypertension cases. A significant positive correlation was observed with a r value of 0.444 and p value of <0.01.

Serum lipoprotein (a) Vs Diastolic blood pressure (DBP)	r Value	p Value
Stage 2 hypertension	0.415	<0.05

Table 12: Correlation of serum lipoprotein (a) concentrations and diastolic blood pressure in stage 2 hypertension cases. It shows the correlation study between serum lipoprotein (a) concentration and diastolic blood pressure (DBP) in mmHg in stage 2 hypertension cases. A significant positive correlation was observed with a r value of 0.415 and p value of <0.05.

Parameter	Stage 1 hypertension		Stage 2 hypertension	
	r value	p value	r value	p value
Total cholesterol	0.315	NS	0.358	NS
Triglycerides	0.003	NS	0.142	NS
HDLc	-0.006	NS	-0.249	NS
LDLc	0.321	<0.05	0.379	<0.05
VLDLc	0.003	NS	0.142	NS

NS: Not Significant (p value >0.05)

Table 13: Correlation of serum lipoprotein (a) concentrations with lipid profile in stage 1 and stage 2 hypertension cases. It shows the correlation of serum lipoprotein (a) concentrations with lipid profile in stage 1 and 2 hypertension cases. Serum total cholesterol, triglyceride, VLDL cholesterol were positively correlated with lipoprotein (a) which were not statistically significant. Serum HDL cholesterol was negatively correlated with lipoprotein (a) and was not statistically significant.

Conclusion

The above findings suggest that in addition to conventional lipid profile parameters, estimation of Lp(a) can prove to be a valuable tool in risk assessment of population with hypertension and their progression to cardiovascular disease. Further, long term studies in a large group of population are needed to establish the role of Lp(a) in assessing the risk of cardiovascular disease in hypertensive patients.

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