

Evaluation of lipoprotein (a) [Lp(a)] and lipid abnormalities in patients with newly detected hypertension and its association with severity of hypertension

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ABSTRACT

Introduction: Hypertension remains the major preventable cause of cardiovascular disease (CVD). Lipoprotein (a) is seen to be associated with established essential hypertension and contributes to atherogenesis or to thrombogenesis or both. **Aim:** Correlation between lipoprotein (a) [Lp(a)] and lipid abnormalities in patients with newly detected hypertension and its association with severity of hypertension. **Methods:** It was a cross-sectional observational study carried out at PGIMER, DR. RML Hospital, New Delhi, India. Estimation of serum Lp (a) and lipid parameters along with routine laboratory investigations were carried out in 100 newly diagnosed cases with hypertension and compared with age and sex matched 50 healthy normotensive controls. **Result:** Amongst 100 cases the mean systolic and diastolic blood pressure was 160.68 ± 19.75 mmHg and 84.44 ± 4.32 mmHg respectively. The mean serum Lp (a) in cases was 34.03 ± 7.55 mg/dl as compared to 24.13 ± 4.41 mg/dl in controls ($p < 0.0001$). 62% of cases as compared to 12% of controls had elevated serum Lp (a) levels. Apart from that, the levels of Lp (a) and lipid parameters increased significantly with higher stage of disease ($p < 0.0001$). Approximately 8% of cases had left ventricular hypertrophy as compared to 1% of control. Similarly, 18% of cases had Non-alcoholic fatty liver disease as compared to 4% of controls. 5% of cases had retinopathy as compared to nil in controls. 4% of cases had microalbuminuria as compared to nil in controls. **Conclusion:** It was observed that newly detected hypertension is associated with major derangements of Lp (a) and lipid parameters. We also concluded that end organ involvement is significantly higher in newly detected hypertensives as compared to normotensive subjects and it was attributed to be due to lipid abnormalities observed in the group.

Keywords: Grading of hypertension, lipid profile, lipoprotein (a), newly detected hypertension

Introduction

The total number of people estimated to be suffering with hypertension is estimated to be 1.13 billion in 2015.^[1] The

prevalence of hypertension is 11.3% in India.^[2] The overall prevalence of hypertension in adults is around 30-45%^[3] with a global age standardized prevalence of 24% and 20% in men and women respectively.^[1] Elevated blood pressure (BP) was the leading global contributor to premature deaths in 2015, accounting for almost 10 million deaths and over 200 million disability-adjusted life years. Systolic blood pressure (SBP) >140 mmHg accounts for most of the mortality and disability burden (70%) and the largest number of SBP-related deaths per

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year are due to ischaemic heart disease (4.9 million), haemorrhagic stroke (2.0 million), and ischaemic stroke (1.5 million).^[4] It has been estimated that 10 mmHg reduction in SBP or a 5 mmHg reduction in diastolic blood pressure (DBP) is associated with significant reductions in all major CV events by 20%, all-cause mortality by 10-15%, stroke by 35%, coronary events by 20%, and heart failure by 40%.^[5,6] Hypertension rarely occurs in isolation, and often clusters with other CV risk factors such as dyslipidaemia and glucose intolerance^[7,8] which are itself proatherogenic and together comprise metabolic syndrome. UKPDS trial proved that the reduction of macrovascular events in type 2 diabetics are primarily by reduction of hypertension rather than glycaemic reduction.^[9] Similarity HOPE-3^[10] trial proved that dyslipidaemia alone or together leads to increased CVD mortality in patients with hypertension.

Dyslipidaemia is more common in hypertensives than normotensives and lipid levels increase as BP increases.^[11] In hypertensive patients, a changes in the lipid profile has been observed, with increased levels of total cholesterol (TC), triglycerides (TG) and low density lipoprotein cholesterol (LDL-C) and low blood concentrations of high-density lipoproteins cholesterol (HDL-C) as compared to non-hypertensive.^[12] Apart from that high plasma concentration of lipoprotein (a) [Lp(a)] has been found to be higher in these individuals as compared to healthy population and is found to be an independent risk factor for cardiovascular diseases in these patients.^[13] The role of Lp(a) as an independent risk factor of vascular disease has been investigated for more than 20 years, but recently the European Atherosclerosis Society (EAS) has issued a new consensus statement endorsing routine measurement of Lp(a) among patients with moderate to high risk of cardiovascular disease.^[14] With the results of HOPE-3^[10] trial it is now proven beyond doubt that mortality reduction in hypertensive individuals after BP control is achieved maximally after suppression of lipid parameters even when they are minimally raised or within normal limits. It was seen that rosuvastatin to these hypertensive subjects with normal lipid parameters reduced subsequent CVD substantially. However the same is not well studied in treatment naïve newly detected hypertensive.

Specially studies on evaluation of Lp(a) and lipid abnormalities in newly detected hypertension are very scarce in India hence the present study was planned. The objectives of this research was to detect the prevalence of Lp(a) and lipid abnormalities in subjects with newly detected hypertension and their association with grades of hypertension.

Materials and Methods

It was a cross sectional observational study conducted at the department of Medicine, PGIMER, Dr RML Hospital, New Delhi, India over a span of two years.

100 consecutively newly diagnosed treatment naïve patients with hypertension as per European Society of Hypertension (ESH)

and the European Society of Cardiology (ESC-2018)^[15] [Table 1] were recruited as cases.

All hypertensive patients were graded according to the severity^[15] thus:

- Grade 1 = 140–159/90–99 mmHg,
- Grade 2 = 160–179/100–109 mmHg, and
- Grade 3 = >180/>110 mmHg.

50 non-hypertensive, age and sex matched, healthy volunteers were recruited as controls. The ethical clearance was taken from the institutional ethical review board committee (Number: 01-40/18/2013/IEC/Thesis/PGIMER-RMLH/10234). The cases and controls were recruited from the general outpatient clinics of the hospital. All cases with history of chronic alcoholism or smoking, renal disease (serum creatinine >1.5 mg/L), liver related disorder, diabetes mellitus, pregnancy, hypothyroidism/hyperthyroidism, history of CAD in the past, hypertriglyceridemia (>400 mg/dL) and hereditary familial hypercholesterolemia/dyslipoproteinemia were excluded. Patients taking phenytoin, carbamazepine, metformin, pentoxifylline, methotrexate, vitamin D supplements, and lipid lowering agents were also excluded. A detailed history was taken including past or current co-morbidities. Height was measured by a stadiometer, weight was recorded using a spring based weighing scale. BMI was calculated using the formula of BMI = Weight (in Kg.)/Height² (in m).

Blood pressure was measured by using standard BP measurement protocol after the patient had rested for 10 minutes. Two measurements were taken by aneroid sphygmomanometer, with at least a 5-minute interval between successive measurements. The reading at the first appearance of the Korotkoff sound (phase I) was taken as the systolic blood pressure and that at its disappearance (phase V) was taken as the diastolic blood pressure.^[15] Hypertension was defined as values >140 mmHg SBP and/or >90 mmHg DBP on two or more different occasions.^[15]

After overnight fasting, 10 ml of venous sample was drawn for haematological and laboratory investigation including kidney function test (KFT), liver function test (LFT), complete lipid profile, Lp(a), and thyroid function test. Biochemical tests were performed by standard operating protocols (SOP) in the laboratories of department of Biochemistry and Pathology. Total

Table 1: Classification of blood pressure as per ESH/ESC-2018

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	>80
Normal	120-129	and/or	80-84
High-normal	130-139	and/or	85-89
Grade 1 hypertension	140-159	and/or	90-99
Grade 2 hypertension	160-179	and/or	100-109
Grade 3 hypertension	>180	and/or	>110

cholesterol, HDL-cholesterol, and triglycerides were measured by enzymatic method. Friedewald formula was used in the calculation of LDL Cholesterol as shown in the following:

LDL Cholesterol = total serum cholesterol - (HDL + total TG/5) mg/dL and VLDL = 1/5 triglyceride.

Lp(a) was measured by Immunoturbidimetry method (Normal serum level: <30 mg/dL).^[16] Serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), and triglyceride (TG) levels were classified on the basis of the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III).^[17]

Statistical analysis

Categorical variables were presented in number and percentage (%) and continuous variables were presented as mean \pm SD and median. Normality of data was tested by Kolmogorov-Smirnov test. If the normality was rejected then non-parametric test was used. Quantitative variables were compared using Independent t test (as the data sets were normally distributed) between the two groups and ANOVA for more than two groups. A *P* value of <0.05 was considered statistically significant. The data was entered in MS EXCEL spreadsheet and analysis was done using Statistical Package for Social Sciences (SPSS) version 21.0.

Results

A total of 100 cases and 50 controls were recruited in this study. Out of 100 cases, 60 (60%) were males and 40 (40%) were females while Out of 50 controls, 30 (60%) were males and 20 (40%) were females. The mean age amongst cases was 43.06 ± 10.68 years and was 45.24 ± 11.31 years in controls. Majority (85%) of the cases and controls were less than 50 years of age. The mean body mass index (BMI) was 26.58 ± 95 kg/m² in cases and 28.25 ± 2.36 kg/m² in controls, which was not statistically significant (*p* = 0.07). The mean systolic BP was 160.68 ± 19.75 mmHg in cases and 117.64 ± 8.45 mmHg in controls (*p* < 0.0001). Similarly, the mean diastolic BP among cases was 84.44 ± 4.32 mmHg and 78.78 ± 5.78 mmHg in controls (*p* < 0.0001). 58% of our cases had grade 1 hypertension, 18% cases had grade 2 hypertension and 24% cases had grade 3 hypertension.

The mean serum Lp(a) was 34.03 ± 7.55 mg/dl and 24.13 ± 4.41 mg/dl in cases and controls respectively (*p* < 0.001). Out of 100 cases, 62% had raised Lp(a) whereas only 12% controls had raised Lp(a) (*p* < 0.0001). Even amongst controls, high Lp(a) levels were observed in those with blood pressure 130-139 mmHg systolic and 80-89 mmHg diastolic (i.e. pre hypertension/high normal). The mean value of total cholesterol (TC), and low density lipoprotein and (LDL-C) were significantly higher and mean value of HDL was significantly lower in cases than in the controls (*p* < 0.001). There was no significant difference in levels of VLDL-C (*p* = 0.08685)

and TG (*p* = 0.0747) amongst cases and controls. [Table 2]. 8% of cases had left ventricular hypertrophy (LVH) as compared to 1% of control (*p* < 0.497). Similarly, 18% of cases had Non-alcoholic fatty liver disease (NAFLD) as compared to 4% of controls (*p* < 0.939). 5% of cases had retinopathy as compared to nil in controls (*p* < 0.170). 4% of cases had microalbuminuria (MAU) as compared to nil in controls (*p* < 0.302). No case and control were found to have abnormal nerve conduction velocity (NCV). [Table 3]. No correlation was found between levels of Lp (a) and lipids parameters with (*p* = 0.092) or sex (*p* = 0.254) of the cases or controls.

Mean value of serum Lp(a) were 31.11 ± 4.5 mg/dl, 33.22 ± 7.22 mg/dl, and 41.69 ± 8.82 mg/dl in grade1,2 and 3 of hypertension respectively (*p* < 0.001). Among of 100 cases, 51%, 55% and 91.67% cases had raised Lp (a) in grade1, 2 and 3 of hypertension respectively (*p* < 0.0001). The mean values of TC, and LDL-C increased proportionately with the increasing grade of hypertension (*p* < 0.001) while mean values of TG and VLDL-C also increased with the increasing grade of hypertension (*p* < 0.05). There was no significant changes in the levels of HDL-C, and with different grades of hypertension (*p* = 0.072) [Table 4].

Discussion

In our study, 62% of cases with newly detected hypertension had higher lipoprotein (a) as compared to 12% in controls. This is in accordance with the findings of Lima *et al.*^[18] who stated that higher levels Lp(a) abnormality (62.5%) were seen irrespective of the size and degree of stenosis (mild/moderate and severe atheromatosis) in known hypertensives. Bhavani *et al.*^[19] also found that in Indians, 40.5% of hypertensive patients had Lp(a) >30 mg/dl when compared to 10% in controls. Cataloni *et al.*^[13] reported significantly elevated levels of plasma Lp(a) in 123 Caucasian essential arterial hypertensive patients (47 men and 76 women) and stated that 13% of hypertensive patients had Lp(a) >30 mg/dl when compared to 8% in controls. The prevalence of high had Lp(a) observed in the our study was quite high (62% in patients and 12% in controls) when compared to the Caucasian population. This can be explained by the fact that Indian population by their genetic structure are prone to dyslipidaemia, especially hypertriglyceridemia. The prevalence of metabolic syndrome in Indians is higher and manifests earlier as compared to Caucasians. This unique genetic tendency superimposed with a high fat, high carbohydrate and low protein diet, may be the causative factor behind the dyslipidaemia and high Lp(a) which is seen in even 12% of our controls (almost equal to the prevalence of high Lp(a) seen in Caucasian hypertensives).

Our study did not find any correlation of lipoprotein and lipid levels to the age or sex of the patients. This is in accordance with other studies including by Osuji *et al.*^[20] where no statistically significant relationship of sex with lipoprotein (a) and lipid levels

Table 2: Levels of Lipoprotein (a) and lipid parameters and percentage of participants with abnormal value in cases and controls

Lipoprotein(a) and lipid parameters (in mg/dl)	Case (n=100)	Control (n=50)	P*	(%) of cases with abnormal value	(%) of controls with abnormal value
Lp (a)	34.03±7.55	24.13±4.41	<0.001	62%	12%
TC	213.72±43.32	168.1±32.11	<0.001	60%	12%
HDL-C	34.38±5.98	40.3±6.89	<0.001	66%	26%
LDL-C	138.08±40.64	99.86±27.07	<0.001	30%	4%
VLDL-C	36.48±10.99	27.88±8.27	0.08685	32%	3%
TG	183.26±55.59	136.06±34.81	0.0747	62%	30%
LDL/HDL	4.21±1.65	2.5±0.59	<0.001	62%	4%
TC/HDL	6.39±2.07	4.21±0.68	<0.001	74%	43%

*Independent t-test. [Lipoprotein(a)- Lp (a)], Total cholesterol (TC), High density lipoprotein cholesterol (HDL-C), Low-density lipoprotein cholesterol (LDL-C), Very low-density lipoprotein cholesterol (VLDL-C), triglyceride (TG), Low-density lipoprotein cholesterol (LDL)/ High density lipoprotein (HDL), Total cholesterol (TC)/ High density lipoprotein (HDL)]

Table 3: Frequency of end organ damage in cases and controls

End organ damage	Frequency of end organ damage in cases (n=100)	Frequency of end organ damage in controls (n=50)	P*
Left ventricular hypertrophy (LVH)	8%	1%	0.497
Non-alcoholic fatty liver disease (NAFLD)	18%	4%	0.939
Retinopathy	5%	0	0.170
Micro albuminuria (MAU)	4%	0	0.302
Nerve conduction velocity (NCV)	0	0	0

*Independent t test

Table 4: Frequency of Lipoprotein (a) and lipid parameters abnormalities in cases with between different grades of hypertension and their comparison with controls

Lipoprotein(a) and lipid parameters (in mg/dl)	Grade 1 hypertension n=58	Grade 2 hypertension n=18	Grade 3 hypertension n=24	Control n=50	P*	F
Lp(a)	31.11±4.5	33.22±7.22	41.69±8.82	24.13±4.41	<0.001	12.243
(%) of cases with abnormal value	51%	55%	91.67%	12%		
LDL-C	121.9±29.69	134±27.48	180.25±43.82	99.86±27.07	<0.001	13.183
(%) of cases with abnormal value	10.34%	44.44%	66.67%	4%		
LDL/HDL	3.5±1.11	4.15±0.99	5.96±1.89	2.5±0.59	<0.001	14.813
(%) of cases with abnormal value	41.38%	100%	100%	4%		
HDL-C	36±6.84	32.67±4.06	31.75±3.36	40.3±6.89	0.072	2.784
(%) of cases with abnormal value	48.28%	88.89%	91.67%	26%		
TC/HDL	5.49±1.75	6.61±1.11	8.39±1.97	4.21±0.68	<0.001	12.277
(%) of cases with abnormal value	51.78%	100%	100%	43%		
TC	192.72±29.98	213.33±23.53	264.75±40.82	168.1±32.11	<0.001	21.594
(%) of cases with abnormal value	41.38%	66.66%	60%	12%		
TG	163.1±35.36	210.56±74.33	211.5±63.38	136.06±34.81	0.008	5.344
(%) of cases with abnormal value	51.72%	77.78%	75%	30%		
(VLDL-C)	32.93±7.26	42±14.69	40.92±12.92	27.88±8.27	0.023	4.084
(%) of cases with abnormal value	14.24%	55.56%	50%	3%		

*ANOVA. F value is the value of ANOVA test. [Lipoprotein(a) {Lp(a)}, Low-density lipoprotein cholesterol (LDL-C), Low-density lipoprotein cholesterol (LDL)/ High density lipoprotein (HDL), High density lipoprotein cholesterol (HDL-C), Total cholesterol (TC)/ High density lipoprotein (HDL), Total cholesterol (TC), Triglyceride (TG), Very low-density lipoprotein cholesterol (VLDL-C)]

could be proved. The mean value of lipoprotein (a) in our study was 34.03 mg/dl in hypertensive patients which was significantly high than that in controls and is almost the same as that found by Tewari *et al.*^[21] Elevated serum Lp(a) could play an important role in essential hypertension pathogenesis and can be considered as an individual risk factor in hypertensive patients.^[13] That may explain the high CVD prevalence as well as mortality in Indians as compared to others. High Lp(a) creates a proinflammatory and prooxidative condition in blood vessels which may result into more endothelial injury and more seepage of LDL and Lp(a)

into arterial wall and further plaque formation.^[22] Eventually, less of elasticity as well dispensability may happen leading to hypertension. A significantly higher level of total cholesterol, triglycerides and LDL were found in cases as compared to controls. Similarly, HDL-C was significantly lesser in cases than in controls. Results of our study are supported by those done by Lima M. *et al.*^[18] where significantly higher levels of lipoprotein (a) and others lipid parameters were documented in hypertensive individuals on treatment as compared to normotensive people. Which can happen since many antihypertensive drugs specially

diuretics can have a negative impact on lipid parameters. However since our cases were treatment naïve so either it was because of disease per se or vice versa.

Hypertension and dyslipidaemia are two of the main risk factors for vascular diseases and are often associated with each other. The co-existence of these two risk factors has more than an additive adverse impact on the vascular 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 although 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. Diastolic dysfunction was associated with severity of HTN which was significantly raised in stage II HTN patients. In early stages of HTN, diastolic filling disorder can arise in view of delayed relaxation of left ventricle and subsequently diastolic failure occurs as a consequence of poor left ventricle compliance.^[25] In our study, statistical significant correlation of lipoprotein (a) and lipid parameters with the grade of hypertension was elucidated. This is in well accordance with other studies by in Dahlen *et al.*^[26] who evaluated the relationship of level of plasma Lp(a) and the severity of CHD, as assessed by coronary angiography and found that not only are high plasma concentrations of Lp(a) associated with the presence of CHD, but they are also directly related to its severity.

Limitations of the study

It was a hospital based study, so it may not truly represent the population. Our study did not collect data from all parts of the country and at best it could only be speculated whether observed relationship is similar all over the country. Secondly being a cross-sectional study by design it cannot associate causal relationships between the factors under study. India is a diverse country with different religious and dietary practices which was not taken into account.

Conclusion

This study has shown that lipoprotein (a) and lipid abnormalities are highly prevalent among newly diagnosed hypertensive and the severity increases with increasing grades of hypertension. Approximately 10%–15% of our newly detected hypertension had end organ damage. So efforts should be intensified to diagnose hypertension as early as possible in general population and if present then complete evaluation and management including lipid and Lp(a) should be the utmost task. Any abnormalities detected are to be taken into the therapeutic considerations in these high-risk individuals. Early diagnosis and treatment of the disease can decrease the morbidity and mortality, and can prevent further complications. However, long term randomized prospective trials are required to elicit the effect

of reduction of Lp(a) and lipid levels on the reduction of these complications.

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Conflicts of interest

There are no conflicts of interest.

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