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Research Article

Study of serum lipids and lipoprotein (a) levels in essential hypertension patients

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ABSTRACT

Objectives: To study of serum lipids and lipoprotein (a) levels in essential hypertension patients.

Methods and Collection of Data: The Study was conducted atDHQ Hospital Hafizabadfrom January 2017 to June 2017. 30 patients of essential hypertension in the age group of 22-75 years wereincluded as cases.30 healthy subjects of comparable age and sex were chosen ascontrols. Cases with history of drug treatment with β blockers and diuretics, diabetesmellitus, obesity, family history of hyperlipidemia, renal and liver failure, endocrinedisorders, taking systemic drugs especially lipid lowering agents, smoking andalcohol users were excluded from the study.

Blood and urine samples were collected after obtaining proper consent from allcases and controls. Serum lipids and Lp(a) levels were measured by enzymaticmethods and turbidimetric immunoassay respectively. Serum fasting blood sugar,blood urea, serum creatinine, urinary sugar and proteins were also measuredsimultaneously using routine laboratory methods.

Result: Serum TC, TG, LDL-C, VLDL-C and HDL-C levels were not found to bestatistically significant between the two groups. Patients with essential hypertensionshowed significantly higher serum levels of Lp(a) relative to controls.

Conclusion: Dyslipidemia and Lp(a) may be a factor contributing to an increased risk ofcoronary heart disease in patients with essential hypertension. In addition we canconclude that Lp(a) may be aindependent risk factor for atherosclerosis inhypertensive patients.

Key Words: Essential hypertension, lipid profile, lipoprotein(a).

INTRODUCTION

Hypertension is an important worldwide public-health challenge because of itshigh frequency and concomitant risks of cardiovascular and kidney disease.[1]It iscommon, asymptomatic, readily detectable, usually easily treatable, and often leads tolethal complications if left untreated.[2] It has been identified as the leading risk factorfor mortality and is ranked third as a cause of disability-adjusted life-years. A recentreport on the global burden of hypertension indicates that nearly 1 billion adults (morethan a quarter of the world's population) had hypertension in 2000 and this ispredicted to increase to 1.56 billion by 2025.[1] Pooling of epidemiological studiesshows that hypertension is present in 25% urban and 10% rural subjects in India. Itwas reported that of a total of 9.4 million deaths in India in 1990, cardiovasculardiseases caused 2.3 million deaths (25%). A total of 1.2 million deaths were due tocoronary heart disease and 0.5 million due to stroke. It has been predicted that by2020, there would be a 111% increase in cardiovascular deaths in India. Hypertensionis directly

responsible for 57% of all stroke deaths and 24% of all coronary heartdisease deaths in India.[3]

For countries which is in the second stage of development, infectious diseaseburdens are reduced and diseases related to hypertension, such as hemorrhagic strokeand hypertensive heart disease, becomes more common. For example, in the sevencountries study, low CHD rates were observed in Japan and the Mediterraneancountries, and high CHD rates in Finland and the US These differences were in largepart explained by differences in diet, serum cholesterol and blood pressure.[4]

Population studies suggest the blood pressure (BP) is a continuous variable,with no absolute dividing line between normal and abnormal values. It is aheterogenous disorder in which patients can be stratified by pathophysiologiccharacteristics that have a direct bearing on the efficacy of specifically targetedantihypertensive medications, on the detection of potentially curable forms ofhypertension and on the risk of cardiovascular complications.[5]

High blood pressure is a trait as oppose to a specific disorder and represents aquantitative rather than a qualitative deviation from normal. Any definition ofhypertension is therefore arbitrary.[6]

Hypertension is one of the most common complex genetic disorders, withgenetic heritability averaging 30%. Like other multifactorial human traits, it is causedby the interplay of several different 'risk' genes and multiple environmental factors.

The threshold model of multifactorial inheritance predicts that those with an inheritedgenetic liability above a certain threshold will develop hypertension, especially whenexposed to aggravating environmental stimuli.[7]

The pathogenesis of essential hypertension is not clearly understood. Differentinvestigators have proposed the kidney, the peripheral resistance vessels and thesympathetic nervous system as the seat of primary abnormality.[6]It is associated withfuctional and morphological alterations of the endothelium. Due to its positionbetween blood stream and smooth muscle cells, the endothelium is thought to be bothtarget and mediator of arterial hypertension. The endothelial dysfunction couldfacilitate the maintenance of elevated peripheral resistance, which would favor theoccurrence of complications, such as atherosclerosis. The increased vascularresistance in essential hypertension is related to the imbalance of action of vasodilator(NO and PGI2) and vasoconstrictors (ET 1 and TXA2).[8]

Systemic blood pressure rises with age and the incidence of cardiovasculardisease is closely related to average blood pressure at all ages, even when the bloodpressure readings are within the so called 'normal range'. The adverse effects ofhypertension principally involve the blood vessels, central nervous system, the retina,the heart and kidneys.[6]In patients with essential hypertension there are often ofcoronary risk factors. Indeed, in these patients, a modification of the lipid profile hasbeen observed, with increased levels of total cholesterol, triglycerides and LDL-C andlow blood concentrations of HDL-C. High plasma concentration of lipoprotein (a) [Lp(a)] has been found to be an risk factor for cardiovascular diseases in thesepatients.[9-10]

Lipoprotein (a) is a complex lipoprotein macromolecule that containsapolipoprotein (a) [Apo(a)], which shares homology with plasminogen. It acts as acompetitive inhibitor of tissue type plasminogen activator and thereby helps inmodulating the fibrinolytic system consistent with an atherogenic role.10

There are very limited studies determining association between Lp(a) excessand essential hypertension. So we undertook this study to evaluate lipid profile andLp(a) level in essential hypertensive patients.

METHODOLOGY

The study was carried out on 30 normotensive controls and 30 essentialhypertensive patients who attended the outpatient and inpatient

departments ofDHQ Hospital Hafizabadfrom January 2017 to June 2017.

The diagnosis of hypertension was established in accordance with therecommendations of world health organizations, international society of hypertension.

Inclusion Criteria: Cases of essential hypertension in the age group of 22-75 years.

Exclusion Criteria: patients with the following conditions are excluded from thestudy. Cases of essential hypertension who were on drug treatment with β blockersand diuretics, diabetes mellitus, obesity, family history of hyperlipidemia, renal andliver failure endocrine disorders, taking systemic drugs especially lipid loweringagents, smoking and alcohol users.

The institutional ethical committee approved the study protocol. History and personalphysical data was obtained from both cases and controls.

Materials

Informed consent was taken from patients and control subjects. 5ml of venoussample was collected from the subjects after an overnight fast of 10-12hrs. A urine sample wascollected and analyzed immediately. The blood samples were analyzed for, bloodglucose, serum total cholesterol and serum triglycerides, HDL cholesterol, blood urea,serum creatinine and serum Lipoprotein (a).

Urine sample wasanalyzed for protein and sugar. All the estimations were done immediately aftercollection of specimen and separation of serum.

Collected data was analyzed by using SPSS version 16. Mean and SD was calculated for numerical data and frequencies were calculated for categorical data.

RESULTS

The present study is undertaken to evaluate the significance of serum lipid and Lipoprotein (a) levels in essential hypertension. 30 essential hypertensive cases were considered for the study. 30 age and sex matched healthy individuals were chosen as controls.

Thirty hypertensives are further categorized according to their blood pressure as mild hypertensive (six patients), moderate hypertensive (fourteen patients), and severe hypertensive (ten patients). The values are presented in Table 1.

The mean age of controls were 56.87±10.26 with the male female ratio being 11:19. The mean age of cases were 56.63±11.24 with the male female ratio being 11:19. Age, gender and BMI distribution was showed in table 2, 3 and 4)

The mean value distribution of serum lipid parameters of the study groups are projected in Table 5. The mean total cholesterol was 183.07±39.36 in control group whereas in hypertensive cases it was 195.10±30.36. When compared to the controls, rise in the mean serum total cholesterol level, in the cases, was not statistically significant (p value=0.190).

The mean serum triglyceride levels were higher among hypertensive cases as compared to controls but this difference was also not statistically significant (p value=0.059).

The serum HDL-C level was lowered in cases compared to controls. This difference was statistically not significant with p value =0.45.

The mean serum LDL-C levels in the hypertensive cases were higher and were statistically not significant (P=0.323). Rise in the serum VLDL-C levels in cases, as compared to the controls was statistically not significant (p value=0.056).Total cholesterol/HDL-C ratio was higher among hypertensive cases as compared to controls. The difference was statistically significant in both the groups (p value=0.021*).

The Levels of LDL/HDL ratio was statistically similar between two groups with P=0.233.

The mean values of lipoprotein(a) level in serum of the study groups are also projected in Table 5. When compared to the controls, rise in the serum Lipoprotein (a), in the cases, was highly significant (p value <0.001).

Table 1: Hypertension according to severity

Blood pressure	Number of patients	%
Mild	6	20.0
Moderate	14	46.7
Severe	10	33.3
Total	30	100.0

Table 2: Age distribution in controls and cases

Age in years	Control		Cases	
	No	%	No	%
36-40	3	10.0	3	10.0
41-50	6	20.0	7	23.3
51-60	10	33.3	10	33.3
61-70	9	30.0	8	26.7
>70	2	6.7	2	6.7
Total	30	100.0	30	100.0
Mean ± SD	56.87±10.26		56.63±11.24	

Table 3: Gender distribution among controls and cases

Gender	Control		Cases	
	No	%	No	%
Male	11	36.7	11	36.7
Female	19	63.3	19	63.3
Total	30	100.0	30	100.0

Table 4: BMI distribution of controls and cases

BMI (kg/m^2)	Control		Ca	ses
	No	%	No	%
18-25	26	86.6	24	80
25.1-30.0	4	13.4	6	20
>30.0	0	0.0	0	0.0
Total	30	100.0	30	100.0

Table 5: Comparison of levels of Lipid parameters and lipoprotein in two groups of patients

Variables	Controls	Cases	Significance	Effect size
Total cholesterol (mg/dl)	183.07±39.36	195.10±30.36	t=1.326; P=0.190	0.34(S)
Triglycerides (mg/dl)	138.13±48.09	167.67±68.84	t=1.926; P=0.059+	0.50(M)
HDL (mg/dl)	41.40±7.18	39.80±9.19	t=0.752; P=0.455	0.19(N)
LDL (mg/dl)	114.37±32.38	122.03±27	t=0.996; P=0.323	0.25(S)
VLDL(mg/dl)	27.30±9.58	33.27±13.72	t=1.954; P=0.056+	0.50(M)
TChol/HDL	4.41±0.81	5.09±1.34	t=2.371; P=0.021*	0.60(M)
LDL/HDL	2.73±0.72	3.21±1.15	t=1.915; P=0.060+	0.50(M)
Lp(a) mg/dl	24.27±5.38	31.95±9.55	t=3.841; P<0.001**	0.98(L)

DISCUSSION

This study attempted to know the serum Lp(a) and lipid profile levels in uncomplicated essential hypertensive patients. This study has shown that the prevalence of hypertension is highest in age group 51-60 years. This is consistent with earlier study in Ibadan, Nigeria. Several studies in both developed and developing countries have consistently shown a positive relationship between age and blood pressure.[11]

Hypertension and hyperlipidemia occur together more often than it is expected by chance. There is some evidence that hyperlipidemia itself may predispose to hypertension and that lipid-lowering interventions may have a beneficial effect on blood pressure, or at least on vascular reactivity. Hypertension and hyperlipidemia have a more than additive effect on cardiovascular risk and it is important to consider them both along with other risk factors before embarking on drug therapy.[12]

It was found that there was a higher TC, TG and LDL-C in the hypertensive population, but the difference between the two groups was not statistically significant in our study.

A study carried out on 3182 Indian hypertensive patients detected high TC and low HDL-C but no disturbance in LDL-C.[12] This study is in line with our study concerning LDL-C.

A review of seven studies including over 41,000 subjects showed that significant correlations existed between blood pressure and high TC, LDL-C and TG.[12]

In our study there was no statistically significant difference in HDL-C level between thetwo groups of population. This finding is in good agreement with the study done by Idemudiaet al.[11]

In our study though TC was not significantly raised but its ratio with HDL-C was raised significantly in cases. This finding is in accordance with theHalperinet al[13]

CHD and stroke are the main causes of morbidity and mortality in patients with arterial hypertension and in many studies, Lp(a) has been found to be an important risk factor for cardiovascular disease. The mechanisms by means of which Lp(a) increases the risk of thrombosis is not completely clear.[9]

Lp(a) excess may promote premature atherosclerosis by the following mechanisms: inhibition of clot lysis by Lp(a) leading to a thrombogenic state , increased binding to proteoglycans or to the very low density lipoprotein (VLDL) receptor. Lp(a) also promotes increased uptake by macrophages, direct chemoattraction of monocytes with induction of monocyte chemotactic activity in endothelial cells and promotion of smooth muscle cell proliferation by blocking the plasmin-dependent activation of transforming growth factor-b (TGF-b). Oxidized Lp(a) may also contribute directly to accumulation of lipids in macrophages.[14]

In the present study, it was found that the hypertensive patients had statistically significant higher plasma concentrations of Lp (a) than in the controls. In a similar study, Catalano et al reported significantly elevated levels of plasma Lp (a) in 123 Caucasian essential arterial hypertensive patients. Studies in Indian population have shown that Lp (a) levels are significantly higher among pulmonary arterial hypertension. Mohan et al showed that Lp (a) was an independent risk factor for CAD in diabetic patients. In the present study, majority of hypertensive patients had levels >30 mg/dl which in general, is taken as high-risk level for atherogenesis[10].

CONCLUSION

To conclude, we can say that hypertensive patients are more prone to atherosclerosis due to deranged Lp(a) and lipid parameter. In addition we can conclude that Lp(a) may be aindependent risk factor for atherosclerosis in hypertensive patients. Obviously, further studies are needed to establish the usefulness of Lp(a) in assessing the risk of cardiovascular diseases in hypertensive patients.

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