

## Original Article

# Dyslipidemia and Electrolyte Metabolism in Essential Hypertension - A Study Among North Indian Population

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## Abstract

**Objective:** To evaluate the lipid profile and Electrolyte levels (Sodium and Potassium in Serum and Urine) in Essential Hypertensives and in healthy controls in North Indian Population. **Materials and Methods:** A total of 166 age and sex matched essential hypertensives and healthy controls were included in our study from outpatient department (OPD) of Medicine in King George Medical University, Lucknow, India. First group consisting of 91 subjects were Known E. hypertensive patients (B.P  $\leq$  139/89mm of Hg). Another group consisting of 75 subjects were healthy controls (B.P  $\leq$  120/80mm of Hg) with no history of hypertension. Fasting venous blood sample was collected from all the subjects in plain vacutainers and the sample was centrifuged for the estimation of lipid profile and Serum Sodium (Na<sup>+</sup>) & Potassium (K<sup>+</sup>). Lipid profile was measured with an automated analyzer (Biochem) and Electrolytes was measured using ion-selective electrolyte auto-analyzer in the Clinical lab of biochemistry in KGMU. **Results:** Most of the control subjects had normal lipid profile levels. In patients with E. hypertension there was a highly significant increase in serum total Cholesterol ( $216.1 \pm 2.77 - 150.6 \pm 1.183$ ,  $p < 0.0001$ ), LDL Cholesterol ( $81.22 \pm 0.483 - 67.94 \pm 1.081$ ,  $p < 0.0001$ ) and Triglycerides ( $172.9 \pm 1.094 - 86.07 \pm 0.963$ ,  $p < 0.001$ ). HDL Cholesterol ( $43.76 \pm 0.49 - 49.25 \pm 1.204$ ,  $p < 0.0001$ ) is also significant reduced as compared to controls. No significant difference was found in serum Na<sup>+</sup> and K<sup>+</sup> level. The Urinary Na<sup>+</sup> ( $87.78 \pm 1.307 - 125.1 \pm 1.55$ ,  $p < 0.0001$ ) levels were significantly lower in E. hypertensive patients when compared to controls while differences in Urinary K<sup>+</sup> levels were not significant. **Conclusion:** We conclude that dyslipidemia is associated with essential hypertension and this may due to the genetic predisposition, sedentary lifestyles and fatty food consumption. Saturated fat and cholesterol in the food increase the blood cholesterol and saturated fat is the main culprit.

**Key Words-** Essential Hypertension, Cardiovascular disease, Cholesterol

## Introduction

Hypertension is an important medical and public health problem both in developed and developing countries and a major risk factor for cardiovascular diseases, stroke and chronic renal disease. It has been deemed the "biggest single risk factor for deaths worldwide" causing around 7 million deaths each year. With the current rates of this disease, India is projected to have the largest number of people with hypertension in the world and has a potential to become the 'Hypertension capital of world'.<sup>[1]</sup>

Basically hypertension is classified into Primary or Essential hypertension and Secondary hypertension. Essential hypertension is defined as a "rise of blood pressure of unknown cause" and Secondary hypertension as the increase in blood pressure caused by diseases of kidney, endocrines or some other organs. Essential hypertension has been appropriately called as a Silent Killer because it is usually asymptomatic and untreated. Abnormalities in Serum lipid levels (dyslipidemia) are recognized as a major risk factor for cardiovascular disease and essential hypertension.<sup>[2]</sup> Lipid profile is the earliest marker for coronary heart disease and includes total cholesterol, HDL-Cholesterol, LDL-Cholesterol, Triglycerides and VLDL. The objectives of this study was to compare the blood lipid levels in Essential hypertensives and normoten-

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sive's by making the association of hypertension with lipid profile in hypertensive cases.

### Materials and Methods

This case-control study was carried out in the Department of Physiology and Biochemistry, King George Medical University, Lucknow. The total number of subjects in our study were 166, and were divided into two groups. The case group consists of 91 subjects who were known essential hypertensives and whose B.P was in the range of 120/80 to 139/89 mm of Hg. The control group consists of 75 subjects who were healthy and normotensive (B.P < 120/80mmHg) with no past / present and family history of hypertension. Subjects with cardio vascular disease, (CVD), renal disease, stroke, endocrine and thyroid disorders were not included for the study. Subjects were selected after filling the informed consent form. The study was cleared by Institutional Ethics Committee. Records of E. Hypertensive patients diagnosed by the attending physician and medical history and life style information were maintained on a pre-tested performa. Blood pressure readings were obtained using a mercury sphygmomanometer. Three readings each of systolic blood pressure (SBP) and diastolic pressure (DBP) were taken after 2 minutes interval from the seated subjects and were averaged. Hypertension was diagnosed based on the JNC-7 Criteria.<sup>[7]</sup> Anthropometric measurements (height, weight, hip and waist circumferences) were taken for each participant. Fasting venous blood sample was collected from the subjects in a plain vacutainer and was centrifuged at 3000 rpm for 10 minutes for the estimation of lipid profile. Serum levels of TG, TC and HDL-C were measured in an automated analyser (EEBA CHEM-7) using commercial kit and LDL-C and VLDL was calculated using the standard formula (Friedwald et. al. 1972).

### Results

The clinical and biochemical characteristics of 91 Essential Hypertensive cases and 75 controls are presented in table 1 and Fig 1. The E. Hypertensives are in the age group of 18-45 years and the mean age is  $33.42 \pm 0.88$  years whereas among the controls the age group is 18-40 years and mean age is  $33.99 \pm 1.06$  years. The blood pressure is calculated separately as systolic blood pressure and diastolic blood pressure. The mean SBP is  $125.7 \pm 1.18$  among essential hypertensives and  $113.80 \pm 0.94$  mm Hg among healthy controls. The mean DBP is  $86.96 \pm 0.56$  among essential hypertension was  $76.71 \pm 0.71$  mmHg among healthy controls. The mean value of total Cholesterol in E. Hypertensive cases were  $216.1 \pm 2.77$  mg/dl and

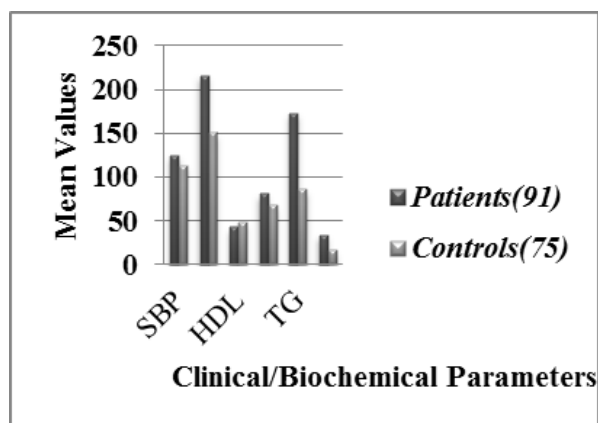
of essential hypertensive patients were higher than controls ( $P < 0.001$ ). The mean HDL of E. Hypertensive cases are  $43.76 \pm 0.49$  mg/dl and that of controls is  $49.25 \pm 1.20$  mg/dl. The increase in mean value of HDL among E. Hypertensive than in the controls was significant ( $P < 0.05$ ). The mean value of LDL in E. Hypertensive cases were  $81.22 \pm 0.48$  mg/dl and in the controls were  $67.94 \pm 1.08$  mg/dl. The mean values of LDL in cases were higher than controls ( $P < 0.001$ ). The mean value of Triglycerides in E. Hypertensive subjects were  $172.9 \pm 1.09$  mg/dl and in controls were  $86.07 \pm 0.96$  mg/dl. The mean value of TG in cases is significantly higher than the controls ( $P < 0.001$ ). The mean value of VLDL in E. Hypertensive subjects were  $34.57 \pm 2.08$  mg/dl and in the controls were  $17.21 \pm 1.66$  mg/dl. The mean values of VLDL in cases were higher than controls ( $P < 0.001$ ).

**Table1.** Comparison of Clinical/Biochemical parameters of hypertensive and non-hypertensive subjects.

Parameters	Patients	Controls	P value
Number (N)	91	75	
Age (years)	$33.42 \pm 0.8$	$33.99 \pm 1.06$	0.6780
BMI (Kg/m <sup>2</sup> )	$25.36 \pm 0.76$	$23.93 \pm 0.78$	0.1958
Serum Sodium (mmol/l)	$137.9 \pm 0.81$	$137.5 \pm 0.9$	0.7779
Serum Potassium (mmol/l)	$3.45 \pm 0.52$	$3.99 \pm 0.48$	0.9904
HDL cholesterol (mg/dl)	$43.76 \pm 0.49$	$49.25 \pm 1.20$	0.0001*
Triglyceride (mg/dl)	$172.9 \pm 1.09$	$86.07 \pm 0.96$	0.0001*
LDL cholesterol (mg/dl)	$81.22 \pm 0.48$	$67.94 \pm 1.08$	0.0001*
Systolic BP (mm Hg)	$125.7 \pm 1.18$	$113.8 \pm 0.94$	0.0001*
Diastolic BP (mm Hg)	$86.98 \pm 0.56$	$76.51 \pm 0.71$	0.0001*
<b>Number (N)</b>	<b>n = 40</b>	<b>n = 45</b>	
Urinary Sodium (mmol/l)	$87.78 \pm 1.3$	$125.1 \pm 1.15$	0.0001*
Urinary Potassium (mmol/l)	$3.110 \pm 0.09$	$3.253 \pm 0.10$	0.3231

\*Significant difference between groups.

**Fig 1.** Lipid profile measurement in Essential Hypertensives and Healthy Controls



## Discussion

Hypertension already affects one billion people worldwide and the prevalence of hypertension between three and six decades in India, has increased by about 30 times among urban residents and by about 10 times among the rural residents. It is the major risk factor for myocardial infarction, stroke and renal diseases and is responsible for most deaths worldwide.<sup>[3]</sup> Whatever the precise mechanism of the underlying pathophysiology, it is generally thought to be a combination of genetic and environmental factors. Changes in the environment of modern society have allowed the expression of genetic susceptibility in populations with physical inactivity and weight gain.<sup>[4]</sup> In our study, Serum TC, TG and LDL-C concentrations are significantly higher in E. Hypertensive patients than among normotensive controls. This observation may be due to common risk factor for E. Hypertension the young population. The exact pathogenetic mechanisms underlying the CVD risk mediated by dyslipidemia are not fully elucidated, but high levels of serum cholesterol are known to increase the risk of developing macrovascular complications such as coronary artery disease (CHD) and stroke.<sup>[5]</sup> A study conducted on hypertensive persons in Nigeria found a significantly higher lipid profile except HDL-Cholesterol and the findings were similar to the observations of our study.<sup>[6]</sup> Serum HDL-C level in hypertensive patients was found to be lower than the findings of Shahadat et al. (1999) Castilli et al. (1977), Wilson et al. (1980), Person et al. (1979) and Miller et al. (1977) but serum LDL Cholesterol level corroborated with the all above studies and the Framingham offspring study and the co-operative phenotyping study in USA, who demonstrated a positive correlation between the levels of LDL- Cholesterol and Coronary risk.<sup>[7]</sup> Epidemiological studies shows that the TC levels were very high

in CHD patients.<sup>[8]</sup> It is thus generally recognised and recommended that treatment of hypertension should, in addition to lowering blood pressure, target correction of dyslipidemia if present, to reduce overall CVD risk and increase the cost effectiveness of therapy. The exact mechanism by which a low HDL-C increases CVD risk has not been fully elucidated, though experimental studies suggest a direct role for HDL-C in promoting cholesterol efflux (reverse cholesterol transport) from foam cells in the atherosclerotic plaque depots in blood vessels to the liver for the excretion.<sup>[9]</sup> Furthermore, blood sampling for measuring the lipid profile of hypertensive is an essential part of management.

## Conclusion

We conclude that the increase in systolic blood pressure was more significant than the diastolic blood pressure with increasing age groups. Based on the results obtained from the present study, we concluded that serum cholesterol, triglycerides and LDL Cholesterol levels are positively correlated with high blood pressure, whereas HDL Cholesterol has less significant changes with hypertension. These observations taken together with the data demonstrating the importance of dyslipidemia in patients' risk stratification imply that patients who have high blood pressure and impaired lipid profile are at high risk and should be the target of aggressive primary preventive strategies to reduce the burden of hypertension and subsequent CVD. This study demonstrated that dyslipidemia is associated with hypertension and this may be due to the genetic predisposition, increased consumption of dietary animal fat, sedentary life styles, fatty food consumption, cholesterol in the food increasing the blood cholesterol and consumption of saturated fats, Smoking and increased alcohol intake. We must encourage a change in the life style of hypertensive: A healthy diet and more physical activity will result in healthier community.

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**Conflict of interest:** The authors claim to have no conflict of interests in the context of this work.