Research Article

**Dyslipidemia and electrolyte metabolism in essential hypertensive North Indians**

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**ABSTRACT**

**Background:** In the present study we are going to evaluate lipid profile and Electrolytes levels (Sodium, Potassium in Serum & Urine) in Essential Hypertensive and in healthy controls in North Indian Population.

**Methods:** A total of 210 age and sex matched E. hypertensive & healthy controls were included in our study from outpatient department (OPD) of Medicine in King George Medical University, Lucknow, India. First group consist of 110 subjects were known E. hypertensive patients (B.P ≤ 139/89mm of Hg). Another group is control group consist of 100 subjects who were healthy controls (B.P ≤ 120/80mm of Hg) with no history of hypertension. Fasting venous blood sample was collected from all the subjects in plane vacationer and the sample was centrifuged for the estimation of lipid profile & electrolyte i.e. Sodium (Na⁺) & Potassium (K⁺). Lipid profile was measured with an automated analyzer (Biochem) & Electrolytes was measured using ion-selective electrolyte auto-analyzer in the Clinical lab of biochemistry in KGMU.

**Results:** After analyzing results almost control subjects had normal lipid profile level. In patients of E. hypertension there was a highly significant increase in serum Total Cholesterol (p<0.0001), LDL-Cholesterol (p<0.0001) & Triglycerides (p<0.001). HDL-Cholesterol (p<0.03) is also significant as compare to controls. Not significant difference was found in serum sodium & potassium level. The Urinary Na⁺ levels were significantly lower in E. hypertensive patients when compared to controls while Urinary K⁺ levels were not significant.

**Conclusion:** So we conclude that dyslipidaemia is associated with essential hypertension this may due to the genetic predisposition, secondary lifestyles, fatty food consumption, saturated fat, cholesterol in the food increase the blood cholesterol and saturated fat is the main culprit. Essential hypertensive is linked with increased Na⁺, K⁺ - ATPase activity and increased renal tubular sodium reabsorption.

**Keywords:** Essential hypertension, Cardiovascular, Cholesterol

**INTRODUCTION**

Hypertension is an increasingly important medical and public health issue and a major risk factor for cardiovascular diseases, stroke and renal disease. It has been deemed the “biggest single risk factor for deaths worldwide” causing around 7 million deaths each year. It is a major health problem in developed as well as in developing countries, and one of the most important public health challenge in worldwide because of its high frequency and concomitant risks of cardiovascular and kidney disease. With the current rate of hypertension, India will have the largest number of people with hypertension in the world, with the potential of becoming the ‘Hypertension capital of world!’(1) The purpose of this study was to compare the blood lipid levels in Essential hypertensive with normotensive by making the association of hypertension with lipid profile in hypertensive cases.
Hypertension is classified into two groups Primary or essential hypertension and Secondary hypertension. Primary or Essential hypertension is defined as a “rise of blood pressure of unknown cause.” Secondary hypertension is the increase in blood pressure causes by diseases of kidney, endocrines or some other organs. Essential hypertension has been appropriately called Silent Killer because it is usually asymptomatic and untreated. Dyslipidaemia and hypertension are major risk factors for coronary artery diseases and arteriosclerosis. Moreover when they occur together, risk factors are very high. Abnormalities in Serum lipid levels are recognized as a major risk factor for cardiovascular disease and essential hypertension. (2) Lipid profile is the earliest marker, even today it is the first line marker done in almost all clinical laboratories. It is the group of 5 tests, often ordered together determine the risk of coronary artery disease. The test that make up lipid profile are tests that have been shown to be good indicators of whether sometime is likely to have a heart attack or stroke caused by blockage of blood vessels (Hardening of the arteries). The lipid profile includes total cholesterol, HDL-Cholesterol, LDL-Cholesterol, Triglycerides and VLDL.

Objective

The purpose of this study was to compare the blood lipid levels in Essential hypertensive with normotensive by making the association of hypertension with lipid profile in hypertensive cases.

METHODS

Blood Pressure measurements

Blood pressure readings were obtained using a mercury sphygmomanometer. Averaged three readings each of systolic blood pressure (SBP) & diastolic pressure (DBP) were taken after 2 minutes interval from seated subjects. Hypertension was diagnosed based on the JNC-7 Criteria.

This case-control study carried out from Dept. of Physiology & Biochemistry, King George Medical University, Lucknow. A Total number of subjects in our study were 210, which were divided into two groups. First group was case group consists of 110 subjects with known essential hypertensive patients. (BP > 120/80 & < 139/89mm of Hg). Another group is control group consists of 100 subjects who were healthy and normotensive (B.P < 120/80mmHg) with no past or present and family history of hypertension. Subjects with CVD, Renal disease, stroke, endocrine and thyroid disorder were excluded from the study. Subjects were selected after filling informed consent form. The study was cleared by Institutional Ethics Committee. Records of E. Hypertensive patients diagnosed by the attending physician for medical history & life style information were maintained on a pre-diagnosed Performa. Anthropometric measurements (height, weight, hip & waist circumferences) were taken for each participant. Fasting venous blood sample was collected from the subjects in plain vacationer and sample was centrifuged at 3000 rpm for 10 minutes for the estimation of lipid profile. Serum levels of TG, TC and HDL-C were measured on an automated analyzer (EEBA CHEM-7) using commercial kit & LDL-C & VLDL was calculated using the standard formula (Friedwald et. al. 1972).

RESULTS

The present study is undertaken to evaluate the significance of Serum lipids, Electrolyte & Urinary Electrolyte levels in essential hypertension. 110 essential hypertensive cases were considered for this study. 100 age and sex (approximately) matched healthy individuals were chosen as controls.

The values of age and BMI in controls and cases are presented in Table 1. The mean age of controls was 33.97 ± 8.92 with the male female ratio being 61/39. The mean age of cases was 33.96 ± 8.44 with the male female ratio being 64/46. The mean age in hypertensive cases compared to controls was not statistically significant (P = 0.902). The mean BMI value among cases as compared to controls was statistically not significant (P = 0.439). The BMI distribution in control and cases is graphically depicted in Figure 1.

<table>
<thead>
<tr>
<th>Table 1: Age, BMI &amp; BP in Cases &amp; Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
</tr>
<tr>
<td>-----</td>
</tr>
<tr>
<td>Cases</td>
</tr>
<tr>
<td>Controls</td>
</tr>
</tbody>
</table>

Figure 1: Age, BMI and BP distribution in Cases & Controls

The mean value distribution of serum lipid parameters of the study groups are projected in Table 2. The mean cholesterol was 152.9 ± 11.71 in control group whereas in hypertensive cases it was 196.89 ± 28.05. When compared to the controls, rise in the mean serum total cholesterol level, in the cases, was statistically significant (P = 0.0001). The mean serum triglycerides levels were higher in E Hypertensive cases as compared to controls. This difference is statistically significant (P = 0.0001).

The serum HDL-C level was statistically significant in cases compared to controls and it shows less statistical significant difference when it compares to other cholesterol with P value = 0.03.
The mean serum LDL-C levels in the E. Hypertensive cases were higher and they were also show statistically significant difference between cases and controls with P value = 0.0001.

Rise in serum VLDL-C levels in cases as compared to controls was statistically significant (P value = 0.0001).

Table 2: Comparison of Clinical/Biochemical parameters between hypertensive and non-hypertensive subjects.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (110)</th>
<th>Controls (100)</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.12 ± 8.40</td>
<td>33.97 ± 8.92</td>
<td>0.122</td>
<td>0.902</td>
</tr>
<tr>
<td>BMI</td>
<td>25.10 ± 7.46</td>
<td>24.28 ± 7.46</td>
<td>0.77</td>
<td>0.439</td>
</tr>
<tr>
<td>SBP</td>
<td>125.39 ± 10.96</td>
<td>114.1 ± 7.77</td>
<td>8.41</td>
<td>0.0001</td>
</tr>
<tr>
<td>DBP</td>
<td>87.09 ± 5.20</td>
<td>75.97 ± 5.90</td>
<td>14.12</td>
<td>0.0001</td>
</tr>
<tr>
<td>HDL</td>
<td>46.26 ± 6.37</td>
<td>48.93 ± 10.92</td>
<td>2.10</td>
<td>0.03</td>
</tr>
<tr>
<td>T-Cholesterol</td>
<td>196.89 ± 28.05</td>
<td>152.9 ± 11.71</td>
<td>14.46</td>
<td>0.0001</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>159.55 ± 38.33</td>
<td>93.03 ± 12.15</td>
<td>16.54</td>
<td>0.0001</td>
</tr>
<tr>
<td>LDL</td>
<td>119.02 ± 28.12</td>
<td>84.74 ± 16.43</td>
<td>10.52</td>
<td>0.0001</td>
</tr>
<tr>
<td>VLDL</td>
<td>31.91 ± 7.66</td>
<td>18.61 ± 2.43</td>
<td>16.54</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Figure 2: Distribution of lipid profile in cases & controls.

Serum & Urinary Electrolytes

The mean Serum Sodium and Potassium level in the cases compared to controls was statistically not significant. Mean Urinary potassium level was also not statistically significant in the cases when it compared to controls, but Urinary Sodium level was highly significant in the cases compared to controls.

The distributions of Serum and Urinary electrolytes in the both groups are presented in Fig. 3 & 4. The values of serum and urinary electrolytes in the cases and controls are indicated in Table 3.

Table 3: Distribution of Serum & Urinary Electrolytes in cases and controls.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases (110)</th>
<th>Controls (100)</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Sodium</td>
<td>138.12 ± 7.64</td>
<td>138.3 ± 8.54</td>
<td>0.19</td>
<td>0.848</td>
</tr>
<tr>
<td>S. Potassium</td>
<td>3.83 ± 0.69</td>
<td>3.95 ± 0.48</td>
<td>1.36</td>
<td>0.173</td>
</tr>
<tr>
<td>U. Sodium</td>
<td>77.57 ± 9.84</td>
<td>84.45 ± 8.43</td>
<td>1.44</td>
<td>0.001</td>
</tr>
<tr>
<td>U. Potassium</td>
<td>31.55 ± 5.75</td>
<td>33.71 ± 7.64</td>
<td>3.29</td>
<td>0.153</td>
</tr>
</tbody>
</table>

DISCUSSION

E. Hypertension affects approximately 75% of the adult population 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 death worldwide.\(^3\) 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.  

In our study, Serum TC, TG and LDL-C concentrations are significantly higher in E. Hypertensive patients than in normotensive controls. This observation may be due to common risk factor for E. Hypertension in young population. The exact pathogenic 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) & stroke.  

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. 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. The Framingham offspring study and also with the co-operative phenotyping study in USA, who demonstrated a positive correlation between the levels of LDL- Cholesterol & Coronary risk. 

Epidemiological Studies shows that the TC levels were very high in CHD patients. 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 costeffectiveness of therapy. The exact mechanism by which a low HDL-C increases CVD risk has not been fully elucidated, through experimental studies suggest a direct role for HDL-C in promoting cholesterol efflux (this is called reverse cholesterol transport) from foam cells in the atherosclerotic plaque deposits in blood vessels to the liver for the excretion. Furthermore, blood sampling for measuring the lipid profile of hypertensive is an essential part of managing them. We must encourage a change in the life style of hypertensive: a healthy diet and more physical activity will result in healthier community.

CONCLUSION

Based on the results obtained from the present study, we concluded that serum cholesterol; triglyceride & LDL Cholesterol levels are positively correlated with essential hypertensive patients whereas HDL Cholesterol has less significant changes with essential 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 HT and subsequent CV disease. From the above study that dyslipidemia is associated with hypertension this may due to the genetic predisposition, increased consumption of dietary animal fat, secondary life styles, fatty food consumption, cholesterol in the food increase the blood cholesterol and saturated fat is the main culprit, Smoking and increased alcohol intake.

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES


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