A Study of Various Biochemical Parameters of Lipid Metabolism in Renal Disorders

Manishi Singh*, S.P. Singh and B.K. Agarwal

Department of Biochemistry, MCB Medical College, Jhansi, India.

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Dyslipidemia contributes to the rate of progression of atherosclerosis and chronic kidney disease. Also chronic kidney disease leads to the development of secondary abnormalities in lipid metabolism that contribute to increased cardiovascular morbidity and mortality. Present study was conducted to assess cardiac risk among the patient of CKD. The study comprised into two groups 30 morbid and 42 healthy control, all were in age group of 20 to 50 years having nephrotic syndrome with ESRD undergoing haemodialysis attending nephrology clinic, male, female ratio 3:2, blood urea, S. creatinine, lipid profile, apolipoprotein A and B were estimated it was found that patient of NS had significantly decreased level of HDL-Chol, Apo A1, Apo A1/Apo B, HDL-Chol./T.Chol. as compared to control same time significantly increased level of T-Chol., TG, LDL-Chol., VLDL-Chol, APO-B, LDL-Chol/HDL-Chol., Apo B/Apo A1.

Key words: Dyslipidemia, Glomerulosclerosis, Nephrotic syndrome (NS), ESRD.
MATERIAL AND METHODS

The study was carried out at the department of Biochemistry at MLB Medical College, Jhansi with the collaboration of associated hospital. Blood was obtained from 30 with NS and 42 healthy control group all were age group of 20 to 50 years, mean age 40 years and weight 55 kg and male female ratio 3:2.

Before collecting the blood sample from patients and control 12 hours overnight fasting and collected 5 ml heparinised blood from each patient in a sterilized vial and kept for half an hour proper coagulation and than serum was aspirated by centrifugation at 3000 rpm for 10 minutes and lipid profile measured by fully automated clinical chemistry analyzer –

1. Plasma total chol. By CE-CO PAP enzymatic end point method
3. VLDL and LDL were estimated by simple calculation by using friedewald’s equation
4. Estimation of S. TG by GPO-PAP end point method
5. Estimation of blood glucose by GOD-POD end point method. Value have been expressed as mean±SD the result were analyzed using student ‘t’ Test p<0.05 was considered as significant.
6. APO lipoprotein A1 and B estimation by orion diagnostic as immunochemical assay sample were studied by colorimetrically for blood urea, creatinine, blood sugar described in practical clinical biochemical by Varley.

RESULTS

In our study high TG (P<0.001, 91.7%), T.chol. (IP<0.005, 28.2%), LDL Chol. (P<0.05, 33.25%), VLDL Chol. (P<0.00192, 93%), APOB (P<0.001, 43.28%) significantly increase and HDL Chol. was significantly low (P<0.005, 17%) as compared to controls, hence this group has shown increased risk of atherosclerosis. It is well established that increased rates of synthesis and secretion of VLDL cholesterol by the liver play a major role(22-25) (Table 1A & B).

Table 1.A

<table>
<thead>
<tr>
<th></th>
<th>Group V (n=30)</th>
<th>Control (n=42)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tch. mg% mean±SD</td>
<td>218.57±50.2</td>
<td>183.56±47.32</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TG mg% mean±SD</td>
<td>207.74±48.21</td>
<td>100.9±32.0</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>HDLch mg% Mean±SD</td>
<td>32.77±8.31</td>
<td>37.37±11.52</td>
<td>NS</td>
</tr>
<tr>
<td>LDLch mg% Mean±SD</td>
<td>144.24±34.12</td>
<td>125.59±31.76</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VLDLch mg% Mean±SD</td>
<td>41.54±12.27</td>
<td>19.98±4.31</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>APO A1 mg% Mean±SD</td>
<td>102.98±29.71</td>
<td>98.12±31.10</td>
<td>NS</td>
</tr>
<tr>
<td>APO B mg% Mean±SD</td>
<td>96.53±28.54</td>
<td>63.41±18.10</td>
<td>&lt;0.005</td>
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Table 1.B

<table>
<thead>
<tr>
<th></th>
<th>Group V (n=30)</th>
<th>Control (n=42)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>APO A1/B Mean±SD</td>
<td>1.06±0.32</td>
<td>1.55±0.39</td>
<td>&lt;0.05</td>
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<tr>
<td>LDL-ch/HDL-ch</td>
<td>4.5±1.14</td>
<td>3.48±0.99</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>HDL -ch/Tchol Mean±SD</td>
<td>0.146±0.04</td>
<td>0.202±0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TG/Tchol Mean±SD</td>
<td>0.949±0.19</td>
<td>0.546±0.10</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Tchol/HDL -ch Mean±SD</td>
<td>6.81±1.31</td>
<td>4.945±0.98</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Apo B/A1 Mean±SD</td>
<td>0.946±0.30</td>
<td>0.642±0.27</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Calculated HDL Mean±SD</td>
<td>35.01±9.20</td>
<td>38.81±12.32</td>
<td>NS</td>
</tr>
</tbody>
</table>

DISCUSSION

The study group compared of 30 patients with 42 control group significantly increased level of TG, LDL Chol., VLDL Chol., APO B, and T.Chol., APOB/A1, (P<0.005), it may because of increased synthesis and decreased catabolism of lipoproteins may accounts for severe hyperlipidemia which frequently occurs in patients with NS, NS is defined is proteinuria, hypoalbuminemia edema, and hypercholesterolemia, high lipid levels are not only marker of disease, but also contribute to the process of glomerulosclerosis26 hyperlipidemia is integral part of the NS. It causes increased risk of atherosclerosis27-30.

Recent experiments have suggested that hyperlipidemia may also play a role in the progression of renal disease.

CONCLUSIONS

There was general trend towards increase in TG level along with increase in total cholesterol and LDL & VLDL accompanied by decrease in HDL level., APO A1, APO A1/ APO B ratio an atherogenic index was evaluated in all patient dialyzed and undialyzed in both groups. This is the scope of dyslipidemia in nephrotic is an addition to the effect of basic disease. Dislipidemia is one of the possible factors contributing to atherosclerosis may be worsened by haemodialysis.

REFERENCES

228-239.


