Statistical OF LIPID PROFILE IN ACTIVE SMOKERS

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ABSTRACT

BACKGROUND: Smoking alters the lipid profile adversely and dyslipidemia is a well-established risk factor for the development of coronary artery disease, hence the objective of the present study was to assess the impact of active tobacco smoking on lipids profile. MATERIAL AND METHODS: In this cross sectional study a total of 302 participants (252 smokers and 50 non smokers) were enrolled. Smokers were classified into mild to moderate (Group I) and severe (Group II) based on the number of pack years as 2 – 10 and more than 10 respectively. Standard methods were adopted to check the lipid levels. Data analysis was performed with the SPSS 15.0 statistical software. RESULTS: The observed values for fasting serum lipid profile indicates abnormalities of lipid profile worsen with increasing smoking pack year and atherogenic index as indicated total cholesterol/HDL, LDL cholesterol/HDL cholesterol was significantly elevated in both the groups. CONCLUSION: Measurements of lipid profile in smokers are good predictive tools in the assessment of risk for cardiovascular diseases.

Keywords: lipid profile, smoking, cardiovascular disease

INTRODUCTION: Cigarette smoking is the most common type of tobacco use and is now acknowledged to be one of the leading causes of preventable morbidity and mortality and is one of the largest single preventable causes of ill health in the world. By 2030, if current trends continue, smoking will kill more than 9 million people annually.

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In India 337 million people above 10 years of age consume tobacco and Indian consumers not paid much attention to the tobacco smoking related diseases.1 Cigarette smoking is an independent cardio vascular risk factor, i.e. it increases the incidence of myocardial infarction and sudden coronary death.2 The adverse effects of smoking on cardiovascular disease (CVD) risk are mediated through multiple interrelated mechanisms, including increased oxidative stress, endothelial injury and dysfunction, altered blood coagulation, and derangements of lipid composition and metabolism.3 We examined the last of these explanations by analyzing the effects of current smoking on lipids and lipoproteins. Keeping in view the paucity of data on smoking...
related issues in India and the expected adverse effects of smoking on coronary function profile, the objective of the present study was to assess the impact of active tobacco smoking on lipids profile function tests to compare the effect of smokers and non-smokers in male population.

MATERIALS AND METHODS:
This was an analytical observational cross-sectional study comprising of 252 healthy regular habitual adult male smokers defined by persons smoking cigarettes over 2 pack years and 50 adult healthy non-smokers selected through convenient non probability sampling. Participants with any severe illness (such as diabetes mellitus, malignancy, severe infection, respiratory disease, renal disease, liver disease), using drugs with potential metabolic effects (namely \( \beta \) – blockers, \( \beta \) – agonists, theophylline, diuretics, glucocorticoids and phenytoin), ex-smokers, family history of lipid disorders, alcoholic, lack of approval by physician and persons showing disinterest were excluded from the study. All participants were interviewed for medical and nutritional history. Present and past history of each case was recorded in detail regarding their general information, nutritional and personal habits, medication and history suggestive of any systemic illness. Age was defined as the age at the time of interview (though no documentary proof had been entertained) and smoking history was obtained from the patient which was then used to calculate smoking pack per year by using formula, \( \text{Number of cigarettes smoked per day } \times \text{Number of years smoked}/20 \).4 In our study, smokers were classified into mild to moderate (Group I) and severe (Group II) based on the number of pack years as 2 – 10 and more than 10 respectively. A sample of blood was drawn after overnight fasting of 12 hours with an aseptic technique. Serum was separated from the blood sample and were subjected for following analytical procedures: Serum cholesterol by Cholesterol Oxidase p-aminophenazone (CHOD-PAP, CV\%:3),5 serum triglycerides by Glycerol phosphate oxidase p-aminophenazone (GPO-PAP, CV\%: 3)6 and high-density lipoprotein (HDL) cholesterol by precipitation method (CV\%: 4).7 Low-density lipoprotein (LDL) cholesterol was calculated with Friedwald’s formula.8 Adult Treatment Panel III (ATP III) criteria9 were used to classify plasma lipid levels. Total cholesterol, triglyceride and LDL levels exceeding 200, 150, and 100 mg/dl respectively, and HDL levels below 45 mg/dl were considered as abnormal. Biochemical tests were analyzed on a Bayer express plus auto analyzer. Quality was controlled using standard solutions. This study was approved by Institutional Human Research Ethical Committee. Written informed consent was obtained from all participants. Data analyses were performed with the SPSS 15.0 statistical software. The results for continuous variables are mean + SD and are well within the normal curve (i.e. normality is maintained). The two tailed (unpaired) student’s test for independent samples, analysis of variance (ANOVA) was used, in assessment of the significance of difference between group means. For all analyses, the nominal level of statistical significance was <0.05.

RESULTS:
A total of 252 smokers and 50 controls were included in the study. Smokers were grouped based on pack years to group I and group II. Both the groups were comparable in age. There was no difference noticed among smokers and non-smokers with reference to diet, physical activity and any other lifestyle. The observed values for fasting serum lipid profile are given in table 1 and data clearly indicates abnormalities of lipid profile worsen with increasing smoking pack year. The given data provided a basic concept about the general influence of cigarette smoking in healthy male subjects. Although the mean values of total cholesterol and LDL- cholesterol were comparable between smokers and non-smokers, these markers were strongly correlated with the level of smoking (table1). Triglycerides and HDL-C were statistically significant between control and severe smokers only (Table 1).
Table 1: Lipid profile in Smokers and controls

<table>
<thead>
<tr>
<th>Lipid profile**</th>
<th>Control (n=50)</th>
<th>Group I (n=142)</th>
<th>Group II (n=110)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol (mg/dL)</td>
<td>163.37 ± 21.76</td>
<td>181.64 ± 25.05*</td>
<td>212.81 ± 28.40†</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>100.57 ± 24.39</td>
<td>113.21 ± 40.27</td>
<td>155.63 ± 39.29*</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>43.00 ± 2.65</td>
<td>41.21 ± 2.36</td>
<td>39.44 ± 2.61*</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>100.60 ± 20.81</td>
<td>117.71 ± 21.18*</td>
<td>142.13 ± 24.53†</td>
</tr>
</tbody>
</table>

*P<0.05, †P<0.01

** Values of lipid profile are depicted as mean ± S.D

Table 2: Atherogenic index as indicated by various risk factors

<table>
<thead>
<tr>
<th>Atherogenic index **</th>
<th>Control (n=50)</th>
<th>Group I (n=142)</th>
<th>Group II (n=110)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC/HDL-C</td>
<td>3.80 ± 0.49</td>
<td>4.42 ± 0.67*</td>
<td>5.42 ± 0.83†</td>
</tr>
<tr>
<td>LDL-C/HDL-C</td>
<td>2.34 ± 0.49</td>
<td>2.87 ± 0.55*</td>
<td>3.63 ± 0.71†</td>
</tr>
</tbody>
</table>

*P<0.05, †P<0.01

** Values are depicted as mean ± S.D

TC: Total Cholesterol, HDL-C: High density Lipoprotein cholesterol, LDL-C: Low density Lipoprotein cholesterol

Atherogenic index as indicated by various risk ratios are shown in table 2. The risk ratio calculated as total cholesterol/HDL, LDL cholesterol/HDL cholesterol was significantly elevated in both the groups

DISCUSSION:

In the study, the two groups of subjects (smokers and non-smokers) were of Comparable age, BMI and diet. They were non-diabetic, non-alcoholic, normotensive subjects. Dyslipidemia is a well-established risk factor for the development of coronary artery disease. Our study demonstrated that smoking alters the lipid profile adversely causing dyslipidemia in smokers and the changes become more marked with the number of cigarettes smoked. Smoking causes an increase in oxidized LDL-cholesterol level which plays the key role for atherosclerotic process. It has been reported that the stimulation of the sympathetic adrenal system caused by nicotine lead to an increase in catecholamine secretion resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further result in increased secretion of hepatic triglycerides and VLDL-C in the blood stream, triglycerides along with VLDL-C in the blood stream These changes contribute to the atherosclerotic potential of cigarette smoke. The most prominent feature in our assessment lies in the level of the antiatherogenic HDL-C, which showed a highly significant decrease (p<0.01) in smokers as compared to non smokers, 39.44±2.61 mg/dL versus 43.00 ± 2.65 mg/dL respectively. In
agreement with our finding, a fall in HDL-C level by 3-5 mg/dL in smokers has previously been reported by Rosenson.13

CONCLUSION:

Thus, measurements of lipid profile in smokers are good predictive tools in the assessment of cardiovascular diseases. The present study provides opportunity to explain the physiological consequences of the cigarette smoking activity. As the current report concerns solely to the study of lipid profile in normal healthy controls and smoking male subjects, the results of this study can be correlated with other biochemical, physiological and clinical aspects. This approach is hence, helpful for future studies in understanding the underlying mechanism causing series of changes influenced by smoking activity.

REFERENCES:

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