Study of Lipid Profile among Healthy Smokers and Non Smokers

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Abstract

Introduction: India is one of the largest consumers of tobacco worldwide. Addiction of tobacco smoking is related with many health hazards. Nicotine of tobacco can be attributed to changes in lipid profile and its atherogenic complications.

Objectives: This study was done to compare changes & evaluate role of estimation of lipid profile among healthy smokers and non smokers.

Materials and methods: Case control study was done taking 35 healthy male smokers as cases and 35 age matched healthy male non smokers as controls. Serum lipid profile was measured. Unpaired student’s t-test was used for comparison. Statistical analysis was done using SPSS 17.0.

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Results: The mean serum total cholesterol (TC), serum triglycerides (TG), serum LDL & serum VLDL were significantly higher while serum HDL cholesterol were significantly lower in cases when compared with controls (p<0.05).

Conclusions: Significantly higher serum level of TC, TG, LDL & VLDL with lower level of HDL suggests that smoking is associated with dyslipidemia. Dyslipidemia is an independent risk factor for vascular diseases so it can be used as a biomarker to identify smokers at risk.

Keywords: High density lipoprotein, low density lipoprotein, smoking, total cholesterol, triglycerides.

INTRODUCTION

India is one of largest producer and exporter of tobacco in the world. Tons of tobacco is grown every year in India. Approximately half of it is released for local consumption [1]. Tobacco is consumed in many ways such as chewing, smoking, etc [2]. Smoking of tobacco is done in various forms like cigar, cigarette, beedi, hukka, pipe, etc. Beedi and cigarette smoking is highly prevalent in rural as well as urban India [3].

Tobacco smoke is a complex, dynamic and reactive mixture containing an estimated 5,000 chemicals. Many of them can harm our body in various aspects [4]. Tobacco smoking is one of the well known modifiable risk factor for atherosclerosis, coronary heart diseases, lung & oral cancers, chronic obstructive pulmonary diseases, etc [5]. In India, tobacco kills 8–10 lakh people each year and majority of these deaths occur in young age. An estimate says that an average of five-and-a-half minutes of life is lost for each cigarette smoked [6].

Nicotine is one of the toxins present in tobacco smoke [7]. It is found to have effect on person’s catecholamine & cortisol secretion [8,9]. Elevated catecholamine and cortisol can alter carbohydrate and lipid metabolism in such person [10,11]. Alteration in lipid metabolism may lead to dyslipidemic changes which may become a predisposing factor for atherosclerosis and ischemic heart disease leading to increased morbidity and mortality in smokers [12].

This study was done to compare changes & evaluate role of estimation of serum lipid profile among smokers and non smokers.

MATERIALS & METHOD:

A cross sectional study was conducted taking healthy male smokers as cases and age matched healthy male non smokers as controls. The study cases & controls were
selected from staff, volunteers and patients attending the hospital OPD of Bapuji Hospital and Chigateri Hospital, Davangere. Each participant gave an informed consent and this study was approved by the institutional ethical and research committee to conduct the research study. A proforma was used to record relevant information and study subject’s data.

**INCLUSION CRITERIA FOR SELECTION OF STUDY SUBJECTS:**

**Cases-** It included 35 healthy male smokers in age group of 20-60 years, who have smoked more than 10 cigarettes or 15 beedis / day for 5 years or more.

**Controls-** It included 35 age matched healthy men who have never smoked.

**Exclusion Criteria:**

The subjects with history of hypertension, diabetes mellitus, alcoholism, liver, cardiac or renal diseases or any other major illness were excluded from the study. Subjects who are on medications which can affect serum lipid profile level were also excluded from study.

**Sample collection**

3 ml of venous blood was collected in plain vial after 12 hours overnight fasting. Serum was separated by centrifugation and used for the estimation of serum lipid profile estimation.

**Sample analysis**

Serum lipid profile was determined by using analytical kit from ERBA Diagnostics Mannheim GmbH in semi-autoanalylyzer (CHEM-5 plus V2, Erba Mannheim). Determination of serum triglycerides (TG) was done by Enzymatic glycerol phosphate oxidase – Phenol aminoantipyrine method (GPO-PAP) [13]; serum total cholesterol (TC) by Enzymatic cholesterol oxidase – Phenol aminoantipyrine method (CHOD-PAP) [13] & serum high density lipoprotein (HDL) by Phosphotungstic acid and CHOD-PAP method [13]. Serum low density lipoprotein (LDL) and very low density lipoprotein (VLDL) concentration was calculated by Friedwald’s formula [14].

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LDL (\text{mg/dl}) = \text{total cholesterol (mg/dl)} - \text{HDL (mg/dl)} - \frac{\text{Triglycerides (mg/dl)}}{5}
\]

\[
VLDL (\text{mg/dl}) = \frac{\text{Triglycerides (mg/dl)}}{5}
\]
Statistical analysis

Values were presented as mean ± SD and the statistical analysis was done using SPSS 17.0 software. Student’s unpaired t-test was used for comparison of parameters between two groups. The p-value of less than 0.05 was considered as statistically significant.

RESULTS:

Table 1 shows that the mean levels of serum TG, TC, LDL & VLDL were significantly higher while mean level of serum HDL were significantly lower in cases when compared with controls. (p<0.001)

<table>
<thead>
<tr>
<th></th>
<th>Controls (Mean ± S.D.)</th>
<th>Cases (Mean ± S.D.)</th>
<th>p value of unpaired student’s t test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Serum triglycerides (mg/dL)</strong></td>
<td>116.14 ± 39.34</td>
<td>164.26 ± 41.56</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td><strong>Serum total cholesterol (mg/dL)</strong></td>
<td>161.56 ± 27.67</td>
<td>184.45 ± 35.52</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td><strong>Serum HDL (mg/dL)</strong></td>
<td>47.38 ± 7.64</td>
<td>39.26 ± 7.16</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td><strong>Serum LDL (mg/dL)</strong></td>
<td>88.31 ± 21.57</td>
<td>112.45 ± 25.52</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td><strong>Serum VLDL (mg/dL)</strong></td>
<td>23.22± 7.86</td>
<td>32.91 ± 8.35</td>
<td>&lt; 0.001*</td>
</tr>
</tbody>
</table>

* Highly significant

DISCUSSION:

Present study shows comparison of serum lipid profile among smokers and non-smokers. Tobacco smoke contains many toxic compounds and free radicals which can alter body metabolic pathways significantly [4]. Inhalation of such smoke regularly for long time can aggravate the situation and lead to serious health hazards [15].

In our study, we found significantly elevated levels of serum triglycerides (TG) & serum VLDL among smokers than non-smokers. This finding is in accordance with studies done by Sharma P et al [16], Mouhamed DH et al [17] and Fariduddin JM et al [18]. Nicotine is one of the major content of tobacco smoke. Nicotine is found to cause stimulation of catecholamine and cortisol secretion [8,9]. These hormones
activate adenyl cyclase in adipose tissue leading to increased lipolysis of stored TG and release of free fatty acids (FFA) in plasma [19,20]. Repeated spikes of their secretion occur in heavy smokers leading to sustained high level of FFA in plasma. These FFA are taken up by hepatocytes and there is increased hepatic TG synthesis. Endogenously synthesized TGs are secreted in the form of VLDL particles [21]. Thus, serum levels of TG and VLDL are elevated in smokers.

As serum TG & VLDL increases, probability of their encounter with HDL through cholesterol ester transfer protein (CETP) increases. CETP is responsible for exchange of TG with cholesterol ester (CE) between VLDL & HDL particles. Such TG rich and CE poor HDL particles are less stable so they get disintegrated and serum HDL level falls [22]. Thus, increased TG & VLDL is associated with decreased HDL level in smokers.

LDL particles are derived from VLDL particles. Thus, increased VLDL leads to elevated LDL in plasma. As serum HDL level decreases, reverse cholesterol transport also decreases. Due to these reasons, cholesterol deposition in peripheral tissue increases. Dyslipidemia in the form of elevated TG, LDL & VLDL along with decreased HDL is associated with occurrence of atherosclerotic lesions in vascular endothelium [23]. Therefore, dyslipidemia in smokers predisposes them to increased risk of occurrence of cardiovascular diseases [24].

Smoking is also an independent and modifiable risk factor for development of pulmonary diseases, cancers, cerebrovascular diseases, peripheral vascular diseases, etc. Risk of occurrence of these complications is directly proportional to amount of smoking [25]. Therefore, smokers should be counseled regarding health hazard to them as well as to people around them who becomes victims due to passive smoking [26]. They should be encouraged to quit smoking and adopt healthy lifestyle to reduce the risk of developing health related problems [27].

**CONCLUSION:**

Smoking is one of the major causes of preventable morbidity and mortality in India. Dyslipidemic changes are seen in smokers in the form of increased serum TC, TG, LDL & VLDL and decreased serum HDL. Such changes are associated with occurrence of atherosclerosis and cardiovascular diseases. Smokers should be screened for serum lipid profile to assess the risk of atherosclerosis & related disease. They should be counseled about health hazards of smoking. They should be encouraged to quit smoking and adapt healthy lifestyle to improve the life.
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REFERENCES


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