Original Article

Comparative study of lipid profile in non-smokers, chronic smokers, and chronic smokers with acute myocardial infarction in men

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Abstract:
Introduction: Smoking is a major risk factor in the genesis of coronary atherosclerosis and development of coronary heart disease. Smoking may alter normal plasma lipoprotein levels. The present study was undertaken to compare the lipid profile between non-smokers (Group A) and chronic smokers (Group B) and also between chronic smokers (Group B) and chronic smokers with acute myocardial infarction (AMI) (Group C).

Methods: Thirty six apparently healthy non-smokers, 36 apparently healthy chronic smokers and 36 chronic smokers with AMI were selected for the study. Fasting venous blood samples were collected; triglycerides (TG), total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) were measured. Low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) were calculated by Friedwald's formula.

Results: The lipid profile was compared between Group A and Group B and also between Group B and Group C. There was a significant rise in TG, TC, LDL-C, VLDL-C and significant decrease in HDL-C in Group B compared to Group A. There was a significant rise in TG, TC, LDL-C, VLDL-C and significant decrease in HDL-C in Group C compared to Group B.

Conclusion: Smoking increases the risk of atherosclerosis and smoking modulates the ischemic heart disease risk through gene-environment interaction. Further studies are required to ascertain the gene environment interaction.

Bibliographic Information of this article:

Keywords: Non-smokers; Chronic smokers; Acute myocardial infarction; Lipid profile

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1. Introduction
Smoking is a serious public health problem and the most important avoidable cause of death in the world. Smoking has been a strongly implicated risk factor and also leading cause of death for chronic obstructive pulmonary disease (COPD), lung cancer and atherosclerosis (1). It has been suggested that regularly smoking more than 10 per day, constitutes a major risk for coronary heart disease (CHD) (2).

Numerous reports have demonstrated the increased risk of coronary problems in smokers. Smoking is thought to have an influence on the prevalence of myocardial infarction by means of several mechanisms, including atherosclerotic injury, increased platelet aggregation, increase in the levels of adhesion molecules, and fibrinogen leading to vasoconstriction (1). Cigarette smoke is a complex mixture of chemicals containing more than 4000 different constituents. Some of the compounds identified include pyridine alkaloids such as nicotine, ammonia,
acrolein, phenols, acetaldehyde N-nitrosamine, polycyclic aromatic hydrocarbons such as benzopyrene, combustion gases such as carbon monoxide, nitrogen oxides, hydrogen cyanide, and trace metals, α-emitter radioactive elements such as polonium, radium and thorium (1). In smokers, nicotine causes a significant increase in the levels of serum cholesterol, triglycerides and LDL-C, but HDL-C is lower in smokers than non-smokers (2). Many studies have been compared the level of lipid profile among smokers and non-smokers, and non-smokers and smokers with myocardial infarction. None of the studies that we have reviewed have compared chronic smokers without myocardial infarction and chronic smokers with myocardial infarction. Therefore, the focus of this study was to find any other component that could play a role in lipid profile among chronic smokers and subsequent complications. In this context, the present study was undertaken to compare the extent of lipid profile in non-smokers (Group A), apparently healthy chronic smokers (Group B), and chronic smokers with acute myocardial infarction (AMI) (Group C).

2. Material and Methods

The present study was conducted at Hanagal Shri Kumareshwara (HSK) Hospital Bagalkot, Karnataka, India from May 2009 to June 2010. In the present study, 36 controls who were apparently healthy non-smokers were included after appropriate matching for age and sex. Thirty six apparently healthy chronic smokers and 36 chronic smokers with diagnosed AMI patients were selected after appropriate matching for age, sex and smoking habit. All of them were male subjects. Both chronic smokers and chronic smokers with AMI, who smoked 13 or more cigarettes per day for more than 12 years, were selected from HSK Hospital. Those excluded from the study were persons abusing alcohol, ex-smokers, patients with diabetes mellitus, hypertension, renal diseases, hepatic impairment, endocrine disorders, obese individuals and patients on drugs like β-blockers, lipid lowering drugs and thiazide diuretics. Ethical clearance was obtained from the S.Nijalingappa Medical College Ethical Committee. Informed consent was obtained from all subjects.

Each subject was interviewed and demographic details and smoking history was obtained. The demographic details included age, sex, body weight and body mass index (BMI). Smoking habits included smoking period and number of cigarettes smoked daily. Fasting venous blood samples were collected, and serum was separated and analyzed for the following parameters; Triglycerides (TG) was measured by GOP-PAP method (3, 4), optical density was measured at 546 nm. Total cholesterol (TC) and High Density Lipoprotein cholesterol (HDL-C) were measured by CHOD-PAP method (3, 4, 5, 6, and 7) optical density was measured at 500 nm. Low-Density Lipoprotein cholesterol (LDL-C) and Very Low Density Lipoprotein cholesterol (VLDL-C) were calculated by Friedwald’s formula (8). Statistical analysis was done by ‘z’ test using SPSS version 15.0. The p< 0.05 was considered statistically significant. All the results were expressed as mean±SD.

3. Results

The demographic characteristics of all the subjects are shown in Table 1. All the subjects were male. There was no significant difference in mean age, body weight and BMI among Group A, Group B and Group C. The Group B and Group C were hypertensive, both groups smoked 13 or more cigarettes per day for 12 or more years.

Table 2 Shows the lipid profile parameters in non-smokers (Group A) and chronic smokers (Group B). The mean TG in Group A is 120.25 ± 17.6 mg/dL and that of Group B is 168.12±15.5 mg/dL. The TC of Group A is 160.05 ± 15.3 mg/dL and Group B is 192.37 ± 20.98 mg/dL. The mean HDL-C in Group A and Group B is 37.52± 4.22 mg/dL and 30.51± 3.75 mg/dL, respectively. Group A have a LDL-C mean of 98.48± 7.56 mg/dL and Group B 128.24± 14.3 mg/dL. The mean VLDL-C in Group A is 24.05± 3.52 mg/dL and Group B is 33.38± 2.86 mg/dL. The lipid parameters i.e. TG (p<0.000), TC (p<0.001), LDL-C (p<0.001) and VLDL-C (p<0.000), were significantly higher in Group B as compared to Group A. The HDL-C level (p<0.001) was significantly lower in Group B than in Group A.

Table 3 shows lipid profile in chronic smokers (Group B) and chronic smokers with AMI (Group C). The Group B have a mean TG of 168.12± 15.5 mg/dL and Group C, 198.16± 10.2 mg/dL. The mean TC in Group B and Group C was 192.37± 20.98 mg/dL and 221.13± 19.10 mg/dL, respectively. The mean HDL-C in Group B was 30.51± 3.75 mg/dL and in Group C, 29.23 ± 3.05 mg/dL. The mean LDL-C in Group B was 128.24± 14.3 mg/dL and in Group C, 152.27± 14.01 mg/dL. The mean VLDL-C in Group B and Group C was 33.38±2.86 mg/dL and 39.63±2.04 mg/dL, respectively. The TG, TC, LDL-C and VLDL-C in Group C were significantly higher compared to Group B (p<0.001 for all parameters). But there was no significant difference in HDL-C in Group B and Group C (p>0.05).
Table 1. Demographic characters of non-smokers, chronic smokers and chronic smokers with acute myocardial infarction (AMI) *

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Non-smokers (Group A)</th>
<th>Chronic smokers (Group B)</th>
<th>Chronic smokers with AMI (Group C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (Years)</td>
<td>42.3±9.7</td>
<td>43.1±10.3</td>
<td>45.6±10.4</td>
</tr>
<tr>
<td>BMI (Kg/m²)</td>
<td>25.53±3.08</td>
<td>25.82±0.96</td>
<td>26.92±0.85</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>120.62±3.40</td>
<td>138.21±5.73</td>
<td>144.67±10.56</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>80.05±2.02</td>
<td>90.76±4.10</td>
<td>97.73±5.7</td>
</tr>
<tr>
<td>Smoking status</td>
<td>_</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>No. of cigarettes smoked daily</td>
<td>13±2</td>
<td>14±2 or more</td>
<td></td>
</tr>
<tr>
<td>Smoking period (Years)</td>
<td>_</td>
<td>12.2±2.1</td>
<td>12.90±2.50</td>
</tr>
</tbody>
</table>

**BMI:** Body mass index; **SBP:** Systolic blood pressure; **DBP:** Diastolic blood pressure.
* Values are presented as mean±SD.

Table 2. Lipid profile of non-smokers and chronic smokers *

<table>
<thead>
<tr>
<th>Parameter</th>
<th>mg/dL</th>
<th>Non-smokers (Group A)</th>
<th>Chronic smokers (Group B)</th>
<th>’z’ Value</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>120.25±17.6</td>
<td>168.12±15.5</td>
<td>12.27</td>
<td>p&lt;0.000</td>
<td></td>
</tr>
<tr>
<td>TC</td>
<td>160.05±15.3</td>
<td>192.37±20.98</td>
<td>7.46</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>HDL-C</td>
<td>37.52±4.22</td>
<td>30.51±3.75</td>
<td>7.53</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>LDL-C</td>
<td>98.48±7.56</td>
<td>128.24±14.3</td>
<td>11.06</td>
<td>p&lt;0.000</td>
<td></td>
</tr>
<tr>
<td>VLDL-C</td>
<td>24.05±3.52</td>
<td>33.38±2.86</td>
<td>12.6</td>
<td>p&lt;0.000</td>
<td></td>
</tr>
</tbody>
</table>

**TC:** Total cholesterol; **TG:** Triglycerides; **HDL-C:** High density lipoprotein cholesterol; **LDL-C:** Low density lipoprotein cholesterol; **VLDL-C:** Very low density lipoprotein cholesterol.
* Values are presented as mean±SD.

Table 3. Lipid profile of chronic smokers and chronic smokers with acute myocardial infarction (AMI) *

<table>
<thead>
<tr>
<th>Parameter</th>
<th>mg/dL</th>
<th>Chronic smokers (Group B)</th>
<th>Chronic smokers with AMI (Group C)</th>
<th>’z’ Value</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>168.12±15.5</td>
<td>198.16±10.2</td>
<td>9.88</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>TC</td>
<td>192.37±20.98</td>
<td>221.13±19.10</td>
<td>6.90</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>HDL-C</td>
<td>30.51±3.75</td>
<td>29.23±3.05</td>
<td>-</td>
<td>p&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>LDL-C</td>
<td>128.24±14.3</td>
<td>152.27±14.01</td>
<td>7.28</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>VLDL-C</td>
<td>33.38±2.86</td>
<td>39.63±2.04</td>
<td>10.69</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

**TC:** Total cholesterol; **TG:** Triglycerides; **HDL-C:** High density lipoprotein cholesterol; **LDL-C:** Low density lipoprotein cholesterol; **VLDL-C:** Very low density lipoprotein cholesterol.
* Values are presented as mean±SD.

4. Discussions
In the present study there is a significant elevation of TG (p<0.000), TC (p<0.001), LDL-C (p<0.000) and VLDL-C (p<0.000) in smokers compared to non smokers, and there is a significant decrease in HDL-C (P<0.001) in chronic smokers than non-smokers. Cigarette smoking is the most important risk factor for myocardial infarction.
Smoking can trigger myocardial infarction in individuals with minimal atherosclerosis or even with normal coronary arteries, promoting temporary coronary vessel occlusion, as a result of thrombus formation, coronary artery spasm or both (9). Hyperlipidemia is a well-known risk factor for development of atherosclerosis; evidence suggests that oxidatively modified LDL contributes to the pathogenesis of atherosclerosis. Nicotine increases the circulatory pool of atherogenic LDL via accelerated transfer of lipids from HDL and impaired clearance of LDL from plasma compartment. Therefore, it increases the deposition of LDL-C in the arterial wall (2). Increased oxidative stress and conversion of LDL to oxidized LDL could lead to atherosclerotic lesions. Circulating products of lipid peroxidation and autoantibody titer to oxidized LDL are significantly increased in smokers. It has been reported that exposure to cigarette smoke extract (CSE) causes modification of LDL and actively taken up by the macrophages to form foam cells in culture. CSE exposure may also decrease the plasma activity of paraoxonase, an enzyme that protects against LDL-oxidation (9). Various other mechanisms leading to lipid alteration by smoking are:

1. Nicotine stimulates sympathetic adrenal system leading to increased lipolysis and increased concentration of plasma free fatty acids (FFA), which further result in increased secretion of hepatic FFA and hepatic triglycerides along with VLDL-C in the blood stream.
2. Fall of estrogen levels occurs due to smoking, which further leads to decreased HDL – cholesterol.
3. Presence of hyperlipidemia in smokers leads to increased TG, TC, LDL-C and VLDL-C due to decreased activity of lipoprotein lipase.
4. Consumption of a diet rich in fat and cholesterol as well as a diet low in fiber and cereal content by smokers as compared to non-smokers (10)

The present study is consistent with the previous studies. In this study, we have also compared chronic smokers and chronic smokers with AMI. The lipid parameters TG (p<0.001), TC (p<0.001), LDL-C (p<0.001) and VLDL-C (p<0.001) were significantly higher in chronic smokers with AMI than chronic smokers, even when both the groups were matched for age, body weight, BMI and smoking habits. Although cigarette smoking is a well established risk factor for vascular diseases, the genetic mechanisms that link cigarette smoking to an increased incidence of stroke are not well understood. C alleles were associated with higher risk of carotid plaque formation and were found to act synergistically with other inflammatory single nucleotide polymorphisms (SNPs) to increase carotid intima media thickness (IMT) specifically among the smokers. Cigarette smoke has been shown to increase the expression of proinflammatory cytokines including IL-6. Furthermore, several studies have demonstrated that IL-6 polymorphism may mediate carotid artery intima-media wall thickness. Genetic variation in IL-6 may modify stroke risk, and this increased risk may be due to synergistic effect between pro-inflammatory genotypes and smoking. The inflammatory gene SNPs are associated with early onset ischemic stroke among African-American women (IL-6) and cigarette smoking may modulate stroke risk through a gene-environment interaction (IL6, CD14) (11). The limitations of present study were:

1. Small sample size
2. Diet history was not taken in detail
3. Extent of smoking i.e. amount of smoke exposed to the lungs was not documented, which is very difficult to measure
4. All the participants were male

5. Conclusion

In conclusion, the present study suggests that cigarette smoking is one of the most important exogenous factors that causes significant rise in TG, TC, LDL-C, VLDL-C and significant decrease in HDL-C, thereby increasing the risk of atherosclerosis; and smoking may modulate the ischemic heart disease risk through a gene-environment interaction.

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