Relationship between cigarette smoking and novel risk factors for cardiovascular disease

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Abstract

Background & Objectives: The aim of the present study was to assess the association between cigarette smoking and the alteration of plasma concentration of cardiovascular markers.

Methods: Twenty male cigarette smokers and 20 healthy age matched male non-smokers were included in the study. Plasma levels of fasting cholesterol, triglycerides, High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), Very Low Density Lipoprotein (VLDL), C-reactive protein and homocysteine were estimated.

Results: Both serum total homocysteine and C-reactive protein levels were significantly increased in cigarette smokers than in non-smokers (p<0.001). Fasting serum levels of total cholesterol, triglycerides, LDL, VLDL and total cholesterol / HDL ratio were observed to be significantly higher in smokers than non-smokers (p<0.001). However, fasting HDL concentration was significantly decreased in smokers than non-smokers (p<0.001). In conclusion, our study identifies a strong, positive relationship between cigarette smoking and elevated levels of all three novel risk factors for cardiovascular disease.

Interpretation & Conclusion: These findings suggest that inflammation and hyperhomocysteinemia may be important mechanisms by which smoking promotes atherosclerotic disease.

Key words: Smoking, Homocysteine, Lipid profile, C-reactive protein.

Introduction

Smoking remains the single most common cause of preventable deaths. Each year, over 430,000 people die as a result of a smoking related disease (1,2). Cigarette smokers have a higher risk of coronary artery disease than non-smokers. Several possible explanations have been offered for this association, including altered blood coagulation (3), impaired integrity of the arterial wall (4), changes in blood lipid and lipoproteins concentrations (5), elevated plasma levels of homocysteine (6,7,8) and elevated plasma C-Reactive protein levels (9). Cigarette smoking is known to be associated with a raised plasma homocysteine level (10-13). Cigarette smoking also leads to increase in the concentration of serum lipids (14-17). Smoking has been associated with increased CRP levels, possibly caused by the tissue damaging effects of tobacco smoke (18,19).

In contrast to the information on cigarette smoking, few studies have examined the relationship between cigarette smoking and novel risk factors for cardiovascular disease in a general population (20,21). To understand further the health risks associated with cigar smoking, we examined the association between cigarette smoking and the alteration of plasma concentration of cardiovascular markers.
Material and Methods

Twenty non smoking healthy male subjects aged between 27-48 years (mean ± SD: 35.35 ± 6.72) and twenty male cigarette smokers aged between 26-58 years (mean ± SD: 36.25 ± 7.48) were included in the study. All the subjects were non-alcoholic and no history of cardiovascular disease, diabetes, other systemic and metabolic diseases and also they were from similar socioeconomic status.

Ten ml of fasting blood was collected by venipuncture into evacuated tubes that were free of anticoagulant to clot, samples were centrifuged. Serum was used to analyze total cholesterol, HDL cholesterol, and triglyceride concentrations were quantified enzymatically with an autoanalyzer (Maxmat PL analyzer, France). LDL - cholesterol were calculated by using the Friedewald equation (22). VLDL-cholesterol was calculated from measured triglycerides divided by 5. serum concentration of total homocysteine was determined by the fluorescence polarization immunoassay (Abbott Axsym, USA) and serum C-Reactive Protein (CRP) concentration was determined by means of nephelometry method (Dade-Behring, BN-100).

The results are expressed as Mean ± SD for all the parameters and the statistical significance of differences among groups was examined by using t-test. The logistic regression analysis was carried out to find the significance of each risk factor after adjusting for other factors.

Results

As shown in Table 1, both serum total homocysteine and C-reactive protein levels were significantly increased in cigarette smokers than in non-smokers (p<0.001). Effect of cigarette smoking on serum lipids and lipoproteins are reported in Table 2. Fasting serum levels of total cholesterol, triglycerides, LDL, VLDL and total cholesterol/HDL ratio were observed to be significantly higher in smokers (p<0.001) than non-smokers. However, fasting HDL concentration was significantly decreased in smokers than non-smokers (p<0.001). The logistic regression analysis also showed significant changes in all the parameters in smokers when compared to non-smokers.

Table 1. Effect of Cigarette Smoking on Homocysteine and C-Reactive Protein (values are mean ± SD).

<table>
<thead>
<tr>
<th>Subject</th>
<th>n</th>
<th>Age (Years)</th>
<th>Homocysteine (µmol/L)</th>
<th>C-Reactive Protein (mg %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smokers</td>
<td>20</td>
<td>35.35 ± 6.72</td>
<td>9.75 ± 2.39</td>
<td>5.14 ± 0.85</td>
</tr>
<tr>
<td>Smokers</td>
<td>20</td>
<td>36.25 ± 7.48</td>
<td>27.35 ± 10.06</td>
<td>10.81 ± 1.68</td>
</tr>
<tr>
<td>p-value</td>
<td></td>
<td>NS*</td>
<td>&lt; 0.001</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

* Not Significant

Table 2. Effect of Cigarette Smoking on Lipid Profile.

<table>
<thead>
<tr>
<th>Subject</th>
<th>n</th>
<th>Total Cholesterol (mg%)</th>
<th>Triglycerides (mg%)</th>
<th>HDL (mg%)</th>
<th>LDL (mg%)</th>
<th>VLDL (mg%)</th>
<th>Total Cholesterol/HDL Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smokers</td>
<td>20</td>
<td>163.80 ± 13.94</td>
<td>120.30 ± 12.89</td>
<td>50.25 ± 5.81</td>
<td>89.28 ± 9.80</td>
<td>24.07 ± 2.60</td>
<td>3.30 ± 0.24</td>
</tr>
<tr>
<td>Smokers</td>
<td>20</td>
<td>241.80 ± 35.12</td>
<td>148.75 ± 26.15</td>
<td>40.30 ± 2.64</td>
<td>171.41 ± 38.50</td>
<td>29.75 ± 5.23</td>
<td>6.08 ± 1.03</td>
</tr>
<tr>
<td>p-value</td>
<td></td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt; 0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Discussion

Few studies have demonstrated that elevated levels of C-reactive protein, lipids and homocysteine are positively associated with risk for coronary heart disease and stroke. Increased levels of inflammatory mediators, such as C-reactive protein and fibrinogen, have been implicated in the pathogenesis of atherosclerosis (23). Serum homocysteine level has also been related to increased risk for cardiovascular disease (24, 25). In addition, Tracy and colleagues (26) reported that pack-years of smoking but not current cigarette smoking was positively associated with log C-reactive protein levels among 400 health elderly participants in the cardiovascular health study.

Our study reports that all the three novel risk factors for cardiovascular disease, serum lipids, C-reactive protein and homocysteine were elevated in cigarette smokers when compared with non-smokers. This results provide further insight into the role that cigarette smoking may play in the development of atherosclerosis.

The effects of smoking on human health are serious and in many cases, deadly. There are approximately 4000 chemicals in cigarettes, hundreds of which are toxic. The ingredients in cigarettes affect everything from the internal functioning of organs to the efficiency of the body’s immune system. Quitting smoking has immediate as well as long-term benefits, reducing risks for diseases caused by smoking and improving health in general.

In conclusion, our study identifies a strong, positive relationship between cigarette smoking and elevated levels of all three novel risk factors for cardiovascular disease. These findings suggest that inflammation and hyperhomocysteinemia may be important mechanisms by which smoking promotes atherosclerotic disease.
References


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