Effect of Smoking on Erythrocyte Sedimentation Rate (ESR) and Lipid Profile in Adults

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ABSTRACT

Objective: The study was designed to find out changes in ESR and serum lipid profile in adult smokers and non-smokers and risk of development of atherosclerosis.

Study Design: It is a comparative and non-interventional study.

Study Period: 1st October to 31st October, 2005

Setting: Study was carried out in Department of Pathology, PGMI, Lahore.

Subjects and Methods: A total of 100 males of adult age (18-65 years) were selected, out of which 50 were smokers and 50 were non-smokers. ESR was done by modified Westergren method and lipid profile was done by chemistry analyzer (Microlab 300 MERCK).

Results: A significant increase in ESR, serum total cholesterol (TC), low density lipoprotein cholesterol (LDL-c) and mean serum triglyceride (TG) levels were observed in smokers as compared with non-smokers. While levels of serum high density lipoprotein cholesterol (HDL-c) were significantly lower in smokers as compared with non-smokers.

Conclusion: Smoking produces adverse effect on lipid profile and ESR thus enhancing the risk of atherosclerosis.

Key Words: Serum total cholesterol, ESR, smoking

INTRODUCTION

Cigarette smoke induces endothelial damage by producing free radicals such as nitric oxide and hydrogen peroxide. This oxidative stress promotes a systemic acute phase reaction thus increasing inflammatory cytokines, C-reactive protein, fibrinogen, blood cell count, whole blood viscosity and rouleaux formation. Eventually this leads to rise in ESR values. Cigarette smoking is a powerful risk factor for atherosclerosis and coronary heart disease. There is direct relationship between number of cigarettes smoked and cardiovascular morbidity and mortality. Rise in serum TC, LDL-c, TG and fall in antiatherogenic cholesterol (HDL-c) have been reported by many authors.

Nicotine stimulates catecholamines resulting in lipolysis and increased concentration of plasma free fatty acids (FFAs) which further results in increased secretion of hepatic FFAs and triglycerides along with very low density lipoprotein cholesterol (VLDL-c) in blood. Fall in estrogen due to smoking leads to decreased HDL-c, while hyperinsulinemia in smokers leads to increased cholesterol, LDL-c, VLDL-c and TG due to decreased activity of lipoprotein lipase.

AIMS AND OBJECTIVES

This study was carried out to see changes in ESR and serum lipid profile in smokers and non-smokers of middle age group and their effect on coronary artery disease.

MATERIAL AND METHODS

A total of 100 males in adult age (18-65) years were selected, out of which 50 who have been smoking at least five cigarettes daily for the last five years were kept in group I, while other 50 weight matched non-smokers were kept as control group II. Patients with renal failure, liver disease, endocrine disorders, diabetes, hypertension, alcoholism, obesity and those on lipid lowering agents or thiazide diuretics were excluded from the study. A detailed physical examination was carried out in both groups. After an overnight fast of 12 hours, 3 cc blood was drawn, out of which 2 cc was delivered to a tube containing dried ethyle diamine tetraacetic acid (EDTA) in the concentration of 1.5±0.25 mg/dl for estimation of erythrocyte sedimentation rate (ESR) by modified Westergren method and 1 cc was kept in syringe for lipid profile (TC, TG, LDL-c HDL-c) by chemistry analyzer named Microlab 3000 MERCK.

RESULTS

Most of the smokers had been smoking at least five cigarettes daily for the last five years. The results are given in Tables 1 and 2.

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Table 1: Lipid profile in non-smokers and smokers

<table>
<thead>
<tr>
<th>Lipid Profile values/mg/dl</th>
<th>Non-smokers (n=50)</th>
<th>Smokers (n=50)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean TC</td>
<td>175.0±19.0</td>
<td>193.0±24.0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Mean TG</td>
<td>152.0±30.0</td>
<td>196.0±64.0</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Mean LDL-c</td>
<td>96.0±17.0</td>
<td>116.0±20.0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Mean HDL-c</td>
<td>45.0±5.0</td>
<td>42.0±6.0</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

(P value <0.05 significant, P<0.001 Highly significant)

Table 2: Erythrocyte sedimentation rate (ESR) values in mm/first hour in smokers and non-smokers

<table>
<thead>
<tr>
<th>Mean ESR value mm/First hour</th>
<th>Non-smokers (N=50)</th>
<th>Smokers (N=50)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.0±1.0</td>
<td>9.0±2.0</td>
<td></td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

(P value <0.001 Highly significant)

DISCUSSION

The mean total serum cholesterol in non-smokers was 175.0±19.0 mg/dl while it was significant (P<0.05) in smokers i.e. 193.0±24.0 mg/dl. These observations are similar to the findings of Muscat and Harris.10 The mean serum triglyceride level in non-smokers and smokers were 152.0±30.0 mg/dl and 196.0±64.0 mg/dl respectively (P<0.001). These findings are consistent with Rustogi and Shrivastva.8 The mean LDL-c in non-smokers and smokers was 96.0±17.0 mg/dl and 116.0±20.0 mg/dl respectively (P<0.05) showing significant rise in smokers similar to Rustogi et al.8 The mean HDL-c in non-smokers was 45.0±5.0 mg/dl and in smokers 42.0±6.0 mg/dl (P<0.001). This finding is consistent with the study of Rosenson13 who reported that there is a fall in HDL-c level in smokers. The erythrocyte sedimentation rate values given in Table 2 show a highly significant rise of ESR (P<0.001) in smokers as compared to non-smokers indicating a strong association of markers of systemic inflammation with smoking. These findings are in agreement with Bennudez et al².

CONCLUSION

A significant increase in ESR, total cholesterol, TG, LDL-c was observed in smokers, while lowering of HDL-c was seen in same group, showing greater risk of development of atherosclerosis in smokers as compared to non-smokers.

REFERENCES


