Study of Cigarette Smoking on Haematological Parameters and Lipid Profile in Vidharbha Region, India

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Authors’ contributions

This work was carried out in collaboration among all authors. ‘All authors read and approved the final manuscript.

ABSTRACT

Introduction: Cigarette smoke increases the risk of cardiovascular disease, such as coronary heart disease and peripheral vascular disease. Atherosclerosis, myocardial infarction, and stroke are also examples of ischaemic heart disease. Cigarette smoke contains more than 4,000 substances that have a negative or minimal effect on human health, including free radicals, nicotine, and the most important carbon monoxide in the pharmacy. Tobacco smoke kills 6 million people a year, many from lung cancer, chronic obstructive pulmonary disease (COPD), and cardiovascular disease (CVD). When opposed to never-smokers, smokers lose 10–15 years of life on average, and they begin to develop tobacco-related disorders such as coronary disease.
### Aim: Study of Cigarette Smoking on Haematological parameters and Lipid Profile in Vidharbha Region

**Material and Method:** 25 subjects were smokers and 25 subjects were non-smokers. Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine and Dept. of Respiratory, Datta Meghe Medical College and Shalinitai Meghe Hospital and Research Center.

**Result:** Patients who were non-smokers and smokers were compared. Total cholesterol, triglycerides, LDL, and VLDL are statistically higher in smokers than non-smokers, but the same is true for HDL-cholesterol. Smokers had slightly lower HDL cholesterol than non-smokers.

**Conclusion:** The smokers in this sample had dyslipidaemia as well as a large rise in haemoglobin and haematocrit. The RBC count rises as the rate of smoking rises, as it does in heavy smokers, and the altered lipid profile worsens. This dyslipidaemia in smokers can expose the vascular endothelium to potentially atherogenic lipoproteins, placing smokers at higher risk of developing atherosclerotic plaques and heart disease. As a result, quitting smoking early can alter these processes, which may prevent any major health risks.

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**Keywords:** Smoking; CVD; COPD; HDL; Cigarette; Tobacco.

### 1. INTRODUCTION

According to WHO figures, about five million people die each year from smoking-related diseases, and if current trends continue, that number will rise to ten million by 2015. Numerous studies have shown that smoking has a detrimental effect on health and is a risk factor for a variety of diseases and ailments, including chronic obstructive pulmonary disease, cancer, pancreatitis, intestinal disorders, periodontal disease, metabolic syndrome, and certain autoimmune diseases. Cigarette smoke contains more than 4,000 substances that have a negative or minimal effect on human health, including free radicals, nicotine, and the most important carbon monoxide in the pharmacy.[1]

Cigarette smoke increases the risk of cardiovascular disease, such as coronary heart disease and peripheral vascular disease. Atherosclerosis, myocardial infarction, and stroke are also examples of ischaemic heart disease.[2]

Tobacco use is a leading cause of coronary heart disease, atherosclerosis, and peripheral vascular disease. A higher atherogenic lipid profile is linked to smoking. It raises serum total Cholesterol, triglycerides, LDL Cholesterol, and VLDL-Cholesterol levels while lowering HDL-Cholesterol levels. Atherosclerosis and coronary heart disease are both caused by smoke. The following are some of the pathways that trigger lipid changes as a result of smoking: (a) Nicotine increases the release of free hepatic acids and triglycerides, as well as VLDL-C, in the blood through can catecholamine secretion. Stimulating a sensitive adrenal system leading to increased lipolysis; (b) smokers' diet is low in fibre and cereal but high in fat and cholesterol, in contrast to non-smokers with a decrease in HDL cholesterol. Other studies have shown that Homocysteine inhibits the expression of Apo A-I proteins and lowers HDL cholesterol. [5,6]

#### 1.1 Aim

Study of Cigarette Smoking on Haematological parameters and Lipid Profile in Vidharbha Region

### 2. MATERIALS AND METHODS

The study conducted Department of Biochemistry this study included 50 healthy male subjects of age 25-40 years. Both smokers and Non-smoker subjects who were referred to Shalinitai Meghe hospital and Research center Consequently, 25 subjects were smokers and 25 subjects were non-smokers. Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine and Dept. of Respiratory, Datta Meghe Medical College and Shalinitai Meghe Hospital and Research Center,
Nagpur in collaboration with JNMC & ABVRH (Datta Meghe Institute of Medical Sciences Deemed To Be University), Sawangi, Wardha, Maharashtra.

- **Inclusion criteria:**
  1. Who drink alcohol
  2. Passive smokers
  3. Past Smokers
  4. Women were not included

- **Exclusion criteria:**
  1. DM
  2. Coronary artery disease
  3. Peripheral vascular disease
  4. Chronic Kidney Disease
  5. High blood pressures
  6. Non-steroidal anti-inflammatory drugs

### 2.1 Sample Collection

5ml of each patient’s blood sample was taken and separated in two tubes EDTA and plain tube. The sample was used to estimate the levels of Lipid profile and CBC.

### 2.2 Biochemical Analysis

EDTA samples were used for the CBC count was estimated on 3 parts coulter counter.

The sample was used to estimate the levels of Lipid profile were estimated on AU480 Analyser.

### 3. RESULT

Table 1 show that comparison non-smokers and smokers patients Hb, RBC and Pcv are more than non-smokers to smokers’ patients is statistically significant.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Non-Smokers (n=25)</th>
<th>Smokers (n=25)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HB</td>
<td>13.4±1.7</td>
<td>15.11±2.1</td>
<td>P = 0.0027</td>
</tr>
<tr>
<td>RBC</td>
<td>4.62±0.82</td>
<td>5.01±0.34</td>
<td>P=0.0329</td>
</tr>
<tr>
<td>PCV</td>
<td>42.3±1.24</td>
<td>48.8±7.25</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>TC (mg/dl)</td>
<td>184.2±20.9</td>
<td>210.5±16.4</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Triglyceride(mg/dl)</td>
<td>110.8±23.6</td>
<td>174.6±22.6</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>HDL(mg/dl)</td>
<td>48.7±5.89</td>
<td>36.7±3.41</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>LDL(mg/dl)</td>
<td>98.4±21.3</td>
<td>194.3±13.7</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>VLDL(mg/dl)</td>
<td>21.4±6.11</td>
<td>34.8±9.87</td>
<td>P &lt; 0.001</td>
</tr>
</tbody>
</table>

Patients who were non-smokers and smokers were compared. Total cholesterol, triglycerides, LDL, and VLDL are statistically higher in smokers than non-smokers, but the same is true for HDL-cholesterol. Smokers had slightly lower HDL cholesterol than non-smokers.

### 4. DISCUSSION

The following procedure may explain the increase in lipid levels in smokers: Nicotine is injected into the body by smoking, Nicotine modulates catecholamine secretion and induces lipolysis and the release of free fats from the blood by opening adenyl cyclase in adipose tissue. Increased levels of free fatty acids in the liver lead to increased hepatic triglyceride and VLDL synthesis, which increases blood triglyceride levels and VLDL-C levels.

In our sample, smokers had higher levels of haematocrit and Hb, and their RBC count increased significantly as their smoking rate increased. In their study, Whitehead et al. found that people who smoked more than ten cigarettes a day had significantly lower haemoglobin and haematocrit content.

In a recent study, researchers discovered that smokers had significantly higher levels of total cholesterol, triglycerides, LDL-C, VLDL-C, and a significantly lower level of HDL-C than non-smokers. These findings were similar to those reported by Devaranavadgi BB et al.[7]

Smoking induces hyperinsulinemia and increases insulin tolerance. Since lipoprotein lipase activity is reduced in hyperinsulinemic environments, LDL, VLDL, and TGL levels rise, and hepatic lipase, which converts VLDL to LDL, is enabled.[8] Smoking reduces the activity of human serum paraoxygenase (PON1), compromising the anti-oxidant defense system.[9]

Ex-smokers’ lipid and lipoprotein concentrations were either the same as non-smokers’ or were moderate between smokers and non-smokers, adding to the informal relationship.[10]
Ex-smokers' HDL and Apo-A1 levels return to non-smokers' baseline levels after quitting smoking, according to a longitudinal report. Since quitting smoking, the prevalence of lipid profile changes and atherosclerotic symptoms among middle-aged smokers decreases due to a reversible phenomenon. HDL-C and Apo A-I are negatively associated with plasma Homocysteine. Increased Homocysteine levels can cause a reduction in HDL-C levels through a variety of mechanisms. The increase in catecholamine release caused by smoking, leading to an increase in VLDL-C and a decrease in HDL-C concentration, may also explain why HDL-C levels continue to fall in chronic smokers. As a result, smoking causes coronary artery disease and atherosclerosis by lowering the anti-atherogenic element HDL-C and actually increasing the atherogenic lipoproteins LDL-C, which severely weakens the vascular endothelium. [11-15]

5. CONCLUSION

The smokers in this sample had dyslipidaemia as well as a large rise in haemoglobin and haematocrit. The RBC count rises as the rate of smoking rises, as it does in heavy smokers, and the altered lipid profile worsens. This dyslipidaemia in smokers can expose the vascular endothelium to potentially atherogenic lipoproteins, placing smokers at higher risk of developing atherosclerotic plaques and heart disease. As a result, quitting smoking early can alter these processes, which may prevent any major health risks.

CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline patients consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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11. The insulin resistance syndrome and postprandial lipid intolerance in smokers Eliasson B, Mero N, Taskinen

