Smoking and its association with serum lipid levels

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Abstract

Objective: To compare the effect of smoking over lipid profile. Method: Total 100 subjects were included in this study, 50 were smoker and 50 non-smoker. All the factors other then smoking those can lead to dyslipidemia were ruled out. All patient’s fasting blood sample was collected and lipid profile was estimated. Data was analysed and ‘t’ value was calculated. Results: Serum levels of total cholesterol (TC), triglycerides (TG), low density cholesterol (LDL), very low density cholesterol (VLDL) were found to be significantly high in smokers in comparison to non-smokers but high density cholesterol (HDL) did not any significant difference between two groups. Conclusion: Smoking affects lipid profile significantly, number and duration of smoking having correlation with serum lipoprotein levels but type of smoking (cigarette or biddi) doesn’t change the outcome.

Key word- Smoking, Total cholesterol, LDL, VLDL, Triglyceride, HDL

Introduction

Smoking is one of the major cause of mortality and morbidity throughout the world. And as per the reports of World Health Organisation, India is home for 12% of the world’s smokers and approximately 900,000 people die every year in India due to smoking as of 2009 [1].

Smokers have a higher risk of coronary artery disease than non-smokers, various explanation have been offered for its association, including altered blood coagulation [2] impaired integrity of arterial wall [3] and changes in blood lipid and lipoprotein leading to increase in concentration of total cholesterol, LDL-cholesterol, VLDL-cholesterol, triglyceride, and fall in level of anti-atherogenic HDL-cholesterol, as per the reports of various workers [4-8].

Dyslipidemia, as a risk factor for cardiovascular diseases, is manifested by elevation or attenuation of plasma concentration of lipoproteins [9]. It is presence of abnormal level of lipids in blood characterised by elevation of concentration of total cholesterol, LDL (low density lipoprotein), TG (triglyceride) and decrease in concentration of HDL (high density lipoprotein) [10].

A comprehensive meta-analysis by Craig et al. examined published data from 1966 to 1987 and estimated the risk caused by smoking on CVD with particular emphasis on lipid and lipoprotein involvement [11]. Various mechanisms leading to lipid alteration by smoking are:

(a) Nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamine’s resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further results in increased secretion of hepatic FFAs and hepatic TG along with VLDL-C in blood stream [8,12]. (b) Fall in oestrogen levels occur due to smoking which further leads to decreased HDL-cholesterol[13] (c) presence of hyperinsulinemia in smokers leads to increased cholesterol, LDL-C, VLDL-C and TG due to decreased activity of lipoprotein lipase [14-15].
(d) Consumption of diet rich in fat and cholesterol as well as diet low in fibre and cereals content by smokers as compared to non-smokers [8,16].

Thus a strong synergistic interaction exist between hypercholesterolemia and smoking in genesis of various vascular complications, clearing the relationship might be important for increasing the proportion of longevity in population.

Material and Method

The aims of our study are:
1. To study alteration in lipid profile in healthy smokers and compare the same with lipid profile of non-smokers
2. To find out dose response correlation between the numbers of cigarette/biddi smoked to the degree of alteration in lipid profile
3. To study lipid profile alteration with duration of smoking.

Study Design: Comparative cross sectional study.

Study Population: subjects taken from the healthy attendants of OPD patient visiting our hospital. Study population taken from age 17-60 years. It was divided into control and study group. Subjects selected for the study were 50 healthy smokers and 50 healthy non-smokers. Our population was included male subjects because in our society male smoke frequently and openly as compared to female, and female if do smoke are reluctant in admitting it.

Sample Size: 100 subjects were taken.

Selection Criteria (on the basis of history, general examination, and systemic examination).

Observation and Results

The study included 100 male subjects who were classified into smokers and non-smokers. The BMI for all the subject was determined and the difference for BMI and age was statistically insignificant (p value= 1.000)

Duration of smoking is around 17 yrs (16.98) and amount is around 15/day (14.8).

Table-1: Showing distribution of number of smokers and non-smokers in various age group.

<table>
<thead>
<tr>
<th>Age-group</th>
<th>Smokers(n1)</th>
<th>Non-smokers(n2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-25 yrs</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>26-35 yrs</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>36-45 yrs</td>
<td>17</td>
<td>15</td>
</tr>
<tr>
<td>46-55 yrs</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>&gt;55 yrs</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>50</td>
</tr>
</tbody>
</table>

*Most of the smokers were of age group 36-45yrs.
Table-2: Showing lipid profile in smokers and non-smokers.

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Smoker (mean ± 2 SD)</th>
<th>Non-smoker (mean ± 2 SD)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>135.98 ± 60.12</td>
<td>101.98 ± 62.74</td>
<td>&lt;0.0001**</td>
</tr>
<tr>
<td>LDL</td>
<td>122.14 ± 62</td>
<td>100.06 ± 58.55</td>
<td>0.0004*</td>
</tr>
<tr>
<td>HDL</td>
<td>38.94 ± 33.70</td>
<td>39.66 ± 18.58</td>
<td>0.7919</td>
</tr>
<tr>
<td>VLDL</td>
<td>25.64 ± 15.79</td>
<td>21.18 ± 12.33</td>
<td>0.0022**</td>
</tr>
<tr>
<td>TC</td>
<td>184.98 ± 62.60</td>
<td>159.88 ± 65.78</td>
<td>0.0002**</td>
</tr>
</tbody>
</table>

**highly significant *significant # 't' test for 2 samples.

Above table shows significant increase in value of TG, LDL-C, VLDL-C, and TC in smokers as compared to non-smokers, while decrease in HDL-C values in smokers is statistically insignificant.

Table-3: Showing BMI of smokers and non-smokers.

<table>
<thead>
<tr>
<th></th>
<th>Smokers (mean±2SD)</th>
<th>Non-smokers(mean±2SD)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>23.85±4.86</td>
<td>23.85±5.34</td>
<td>1</td>
</tr>
</tbody>
</table>

Table-4: Correlation of duration of smoking with lipid value as per Pearson’s correlation score(r)

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Duration(r)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>0.391</td>
<td>0.005</td>
</tr>
<tr>
<td>LDL</td>
<td>0.478</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL</td>
<td>-0.432</td>
<td>0.002</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.303</td>
<td>0.032</td>
</tr>
<tr>
<td>TC</td>
<td>0.428</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Pearson’s correlation coefficient shows less correlation between duration of smoking and rise in lipid values i.e. TC TG LDL VLDL and fall in level of HDL.

Table-5: Correlation of number of biddi/cigarette with lipid value as per Pearson’s correlation score(r)

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Number (r)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>0.345</td>
<td>0.014</td>
</tr>
<tr>
<td>LDL</td>
<td>0.273</td>
<td>0.055</td>
</tr>
<tr>
<td>HDL</td>
<td>-0.281</td>
<td>0.048</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.275</td>
<td>0.053</td>
</tr>
<tr>
<td>TC</td>
<td>0.248</td>
<td>0.083</td>
</tr>
</tbody>
</table>

Pearson’s correlation coefficient shows very less correlation with number of cigarette/biddi smoked per day to alteration in lipid value.

Hence with above data we can say that duration and numbers of cigarette/biddi smoking having little correlation with alteration of lipid values but values are more significant for duration of smoking when we compare with number of cigarette/biddi smoked per day.
Discussion

Our study revealed that smoking causes significant increase in concentration of triglyceride, TC, LDL-C and VLDL-C in smokers as compared to non-smokers. While the alteration in levels of HDL-C was not statistically significant (p value = 0.7919) among the two groups. Mean serum total cholesterol was 184.98 ± 62.60 in smokers as compared to that in non-smokers where the value was 159.88 ± 65.78, hence it is significantly raised (p=0.0002) which is similar to the findings observed in other studies.[11,17,19,21,22,23].

Smoking also increases TG the value of which is significantly raised in smokers i.e. 135.98 ± 60.12 as compared to non-smokers where the value is 101.98 ± 62.74 which is statistically very significant (p < 0.0001) [17,19,21]. The triglyceride / high-density lipoprotein abnormalities have recently been suggested to be related to insulin resistance. In fact, it has been proposed that insulin resistance is a potential key link between cigarette Smoking and cardiovascular disease [25]. Similarly values are also raised for LDL that is 122.14 ± 62.00 in smokers as compared to 100.06 ± 58.55 in non-smokers and VLDL which is 25.64 ± 15.78 in smokers as compared to 21.18 ± 12.33 in non-smokers, which is supported by studies done by Craig et al., Anila Jaleel et al., NS Neki, Naisargi Joshi et al., Cheryl S Brischetto et al., D. J. Freeman et al. and Sinha A K et al [11, 17, 19, 21, 22, 23, 26] But Gepher AD et al., Dirican M et al and Nesje LA et al haven’t seen significant rise in LDL values in smokers and non-smokers [20, 27, 28] while Zhang Yan-Ling et al. in their study over residents of age group 90yrs or more have experienced decrease in value of LDL, TC, TG, and VLDL in smokers as compared to non-smokers [18].

One study showed slightly lower mean triglyceride levels in smokers as compared to that of non-smokers, but this study was conducted in young population of 20-25 years of age group those who used to smoke for 5-7 cigarette per day and duration of smoking was only 5 year. [24]. Cigarette smoking also increases oxidative modification of LDL. Circulating products of lipid peroxidation and autoantibody titers to oxidized LDL are significantly increased in smokers [29]. In 1988, Yakode et al [30] reported that exposure to cigarette smoking caused a modification of LDL, which was actively taken up by the macrophages to form foam-cells in culture. Frei et al [31] observed that exposure of human plasma to the gas phase of cigarette smoke caused oxidative modification of plasma LDL. Smoking associated with vasomotor dysfunction, inflammation, and modification of lipids which are integral components for the initiation and progression of atherosclerosis. These components precede the apparent structural and clinicopathologic manifestations of atherosclerosis [32, 33].

Although several studies [34, 35] provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile, there is still inconclusive evidence regarding the alteration of a particular lipoprotein, particularly to high density lipoprotein (HDL) levels. Some authors have concluded that HDL levels were same for smokers and non-smokers [36], while others have found conflicting results wherein significant variations (low levels of HDL in cigarette smokers) were obtained [37, 38]. Our study doesn’t show significant alteration in the values of HDL-C in smokers as well as non-smokers (p=0.7919) while different studies revealed that absenters had shown increase in HDL, total HDL and large HDL particles compared with those continuing smoking which is seen to be raised in non-smokers. While other studies shown a decrement in values in smokers [17, 18, 19, 20, 21, 22, 23]. In another study conducted by Majos O. D. et al. [39] reported that there is significant decrease in HDL-C, but there is no change in total cholesterol and triglycerides. Another report shows lower but no significant HDL levels in smokers [40].

But a study conducted by Siekmeier et al [41] reported the HDL levels are same for smokers and non-smokers.

Here we also proved that alteration of lipid values shows correlation with duration and amount of cigarette/biddi smoked but values are more significant for duration of smoking as compared to number of cigarette/biddi smoked per day and studies by Craig et al. and Naisargi Joshi et al also showed correlation with the number of cigarette/biddi smoked [11, 21]. While studies conducted by Suleyman H et al. And Khurana M et al. Showed that change in serum lipids tends to be high with the increase in duration and intensity both [42, 43].

The study by Gepher AD et al showed effects stronger in women, while our study mainly focused on males. [20].
This study was conducted primarily to study the effect of smoking over lipid profile and as this study has shown significant alteration in lipid profile with smoking and dyslipidemia is associated with increased cardiovascular disorders so result of this study can be used to create awareness about ill effects of smoking and that will be helpful in decreasing cardiovascular morbidity and mortality [44].

There are some limitation of this study also as results cannot be generalized because of small sample size and same geographical distribution. In this study we did not include Apolipoprotein A1 and Apolipoprotein B in lipid profile.

Reporting of smoking habits was by subjects them self which sometimes may not be accurate. Further studies should be multicentric with large sample size and should cover wide geographical area.

**Conclusion**

Serum levels of TC, LDL, VLDL, TG were significantly raised in smokers as compared to non-smokers, while the value of HDL remained statistically unchanged in both the groups. Serum lipoprotein levels also showed correlation with number and duration of biddi/cigarette smoked, but results were more significant for duration than number.

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**References**


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