Additive Effect of Tobacco Chewing on Lipid Profile Among Smokers

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ABSTRACT

Background: Tobacco use in form of smoking or smokeless is a major preventable cause of disease and premature death, currently leading to over 5 million deaths each year worldwide. This study was conducted to find out the additive effect of tobacco chewing on lipid profile among smokers. Method: Serum lipid profile was studied in 30 control (non smoker and non tobacco chewer); 30 smokers and 30 smokers with tobacco chewer; Standard methods were adopted to measure lipid profile. Data were analyzed statistically. Result: mean serum HDL-cholesterol was lower both in smokers (p<0.01) and smokers with tobacco chewers (p<0.001) than controls. Smokers had higher values of total cholesterol, LDL- cholesterol and triglycerides. Smokers with tobacco chewers had highly significant (p<0.001) raised triglycerides, LDL-cholesterol and VLDL cholesterol as compared to smokers. Conclusion: Though different mode of consumption, tobacco chewing had an addictive effect on lipid profile among smokers.

Introduction :-

Tobacco is a herbal product, which man has been using over last more than 3000yrs, but smoking in the form of cigarette is twentieth century phenomenon with major medical and economic consequences. India is the second largest consumer of Tobacco products and third largest producer of tobacco in the world (WHO framework convention on Tobacco control, Geneva, 2009). Tobacco use in the form of smoking (cigarette,bidi,cigarpipe) or smokeless (gutkha,khaini,panmasala,gul, mawa) is a major preventable cause of disease and premature death. Nearly 8-9lakh people die every year in India due to disease related to tobacco use. (Gajalakshmi V, et al 2003). Majority of cardiovascular disease, cancers and chronic lung disease are directly attributed to tobacco consumption.

Globally, cigarette smoking is a dominant form of tobacco use. In Indian context, tobacco use implies a varied range of chewing and smoking forms of tobacco available at different price points, reflecting the varying socio economic and demographic patterns of consumption.(John R.M., et al 2010).

The Global Adult Tobacco Survey India (GATS), revealed that more than 35% of adult use tobacco in some or another form. Among them 21% adult use only smokeless tobacco 9% only smoke and 5% smoke as well as use smokeless tobacco (WHO Global Youth Tobacco Survey, India. 2006).

When tobacco is burned, the resultant smoke contain in addition to nicotine, carbonmonoxide and 4000 other compound that acts primarily on brain and leads to addiction. Depending on “How tobacco is consumed”. Nicotine can reach peak levels in the bloodstream and brain rapidly. Nicotine stimulation of adrenergic drive raise both blood pressure and myocardial oxygen demand (Shaper A. G., et al 1981). Abnormal lipid metabolism with significant fall in “Protective” High density lipoprotein. Smoking has been identified as a major CHD risk factor (Slone D., et al 2003). Majority of cardiovascular disease, cancers and chronic lung disease are directly attributed to tobacco consumption.

Material and Methods:

The Present study was conducted in JLN, medical college and Hospital, Ajmer, a tertiary care center. After obtaining approval from Research Review Board of the institutional ethical committee, 90 subjects were included in the study on voluntary participation basis, of age group 18 - 60years, irrespective of gender. The study protocol was explained to the subjects and after obtaining informed written consent, the study was started. All subjects were screened for inclusion and exclusion criteria. In the present study, the subjects with H/o cigarette smoking or tobacco chewing (Guttaka) along with smoking were included.

Subjects not included in the study were, Alcoholics, Diabetic, Hypertensive, Renal and Hepatic impairment diseases, Endocrinometabolic disorders, those who were on lipid lowering drugs; β blockers; Thiazide diuretics, oral contraceptive pills, and Those who were not willing for study. The blood sample were collected during the morning hours after taking consent. Serum total cholesterol was estimated by enzymatic CHOD-PAP, end point method (Allain CC; 1974); HDL-cholesterol was estimated by Phosphotungstic acid, end point method (Finley PR; 1978); Triglyceride estimation was done by GPO-PAP, end point assay (Fossati P; 1978); LDL-cholesterol and VLDL- cholesterol calculated from Friedewalds formula;

Statistical Analysis of Data:

The data obtained for smokers and smokers with tobacco chewers were presented as Mean ± standard Deviation. Mean and standard deviation of the observations for all the parameters were calculated and comparison was done by applying student ‘t’test (unpaired t test).

Observations and Results:-

Table 1 Lipid Profile in various groups

<table>
<thead>
<tr>
<th>Lipid Profile</th>
<th>Control N= 30</th>
<th>Smokers N= 30</th>
<th>P value</th>
<th>Smokers with tobacco chewers N= 30</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>165.87±18.86</td>
<td>174.1±35.56</td>
<td>&lt;0.05</td>
<td>200.16±36.98</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LDL-Cholesterol</td>
<td>82.69±11.26</td>
<td>104.6±28.93</td>
<td>&lt;0.05</td>
<td>109.05±34.22</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>91.0±20.65</td>
<td>146.81±41.21</td>
<td>&lt;0.01</td>
<td>152.51±29.87</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Increased lipid profile with fall in protective high density lipoprotein, increased plasma cholesterol and also raising atherogenic LDL-cholesterol.

It can be concluded that cigarette smoking as well as tobacco chewing adversely affect the lipid profile. These are significant risk factor for coronary disease and other vascular lesion. Tobacco chewing has an additive effect on lipid profile.

References:-
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All values in mg%