

Additive Effect of Tobacco Chewing on Lipid Profile Among Smokers



Medical Science

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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 world wide. 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, VLDL- 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 additive 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, cigar pipe) or smokeless (gutkha, khaini, panmasala, gul, mawa) is a major preventable cause of disease and premature death. Nearly 8-9 lakh 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 contains in addition to nicotine, carbon monoxide and 4000 other compounds that act primarily on the brain and lead to addiction. Depending on "How tobacco is consumed". Nicotine can reach peak levels in the bloodstream and brain rapidly. Nicotine stimulation of adrenergic drive raises 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* 1978). According to AHA (American Heart Association), nicotine causes short term increases in blood pressure and heart rate and blood flow through the heart. Over time, these can contribute to heart diseases. Furthermore, the carbon monoxide in cigarette smoke causes artery damage, resulting in fatty buildup in arteries. This leads to increased blood pressure, which over time leads to cardiovascular disease. Taking into account the above (hazards/loss/diseases) the present study was conducted to find out the additive effect of tobacco chewing on lipid profile among smokers.

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 - 60 years, 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 samples 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 Friedewald's formula;

Statistical Analysis of Data:

The data obtained for smokers and smokers with tobacco chewers were presented as Mean \pm 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					
Lipid Profile	Control N= 30	Smokers N= 30	P value	Smok- ers with tobacco chewers N= 30	P value
Total cho- lesterol	165.87±18.86	174.1±35.56	<0.05	200.16±36.98	<0.01
LDL-Chol- esterol	82.69±11.26	104.6±28.93	<0.05	109.05±34.22	<0.01
Triglycer- ide	91.04±20.65	146.81±41.21	<0.01	152.51±29.87	<0.001

VLDL-Cholesterol	20.97±4.38	32.89±6.35	<0.001	39.46±10.05	<0.001
HDL-Cholesterol	48.01±10.07	37.48±7.27	<0.001	35.80±5.52	<0.001

All values in mg%

The above table1 shows the lipid profile of control (Group A), Smoker (Group B) and Smoker with tobacco chewers (Group C). This table shows statistically significant ($P<0.05$) correlation of TC and LDL-C in the control Group A in relation to Group B, and Group C. The TG and VLDL-C was highly statistically significant ($P<0.001$) in Group B and C as compare to control Group A. The HDL-C was highly statistically significant ($P<0.001$) in Group B & C as compare to control Group A. The smokers group showed statistical significant ($p\leq 0.05$) rise in total cholesterol and LDL-cholesterol than control group, But serum TC and LDL-cholesterol in smokers with tobacco chewers group showed highly significant ($p\leq 0.01$) correlation. Serum triglyceride and VLDL-cholesterol levels were highly significant ($p\leq 0.01$) in smokers than control, but the values were highly significant ($p\leq 0.001$) in smokers with tobacco chewers than smokers. Serum HDL-cholesterol level was ($p\leq 0.05$) significantly lower in smokers than control, but there was highly significant ($p\leq 0.001$) reduction in smokers with tobacco chewers.

Discussion :-

Various biophysical parameters are determinant of the status of the health of an individual and the subjects of interest for clinical research and investigation. Tobacco consumption either smoking, chewing or both shows to have a strong evidence to modify, regulate and deteriorate many of these biophysical parameters. Findings showing the harmful effect of smoking or tobacco chewing by marked reduction in 'Protective' HDL-cholesterol and raised levels of triglycerides and VLDL-cholesterol, have been replicated in many studies.

Willet W. et al (1983) found a statistically significant difference between total cholesterol and LDL-cholesterol of smokers and nonsmokers. The results of the study are in concordance with present study.

M Khurana. et al (2000) also found high density lipoprotein lower in smokers ($p\leq 0.01$) as well as in tobacco chewers ($p\leq 0.001$) than the controls. Both smokers and tobacco chewers had higher values of total cholesterol, low density lipoproteins cholesterol, very low density lipoproteins cholesterol and triglyceride as compared to control group.

K Sirisali et al (1992) showed the smokers had significantly higher serum triglyceride and VLDL-cholesterol levels ($p\leq 0.001$), but significantly lower HDL-cholesterol levels ($p\leq 0.05$) than nonsmokers. Levels of lipid lipoprotein-cholesterol were related to the number of cigarette smoked per day.

Present study also showed statistically significant reduction in HDL-cholesterol levels in smoker and smoker with tobacco chewers. Triglycerides and VLDL-cholesterol levels were statistically highly significantly raised among smokers with tobacco chewers. These findings were quite consistent with most of the research works conducted by others.

Conclusion :-

From this study it is concluded that smoking with tobacco chewing cause stimulation of adrenal medulla leading to higher concentration of free fatty acid and nicotine causes higher concentration of catecholamine in plasma leading to atherogenic state and alteration of lipid metabolism and

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 a additive effect on lipid profile.

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