

## EFFECT OF DURATION OF SMOKELESS TOBACCO (GUTKA) CHEWING ON LIPID PROFILE

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**Abstracts: Background:** Tobacco use in the form of smoking /smokeless is a major preventable cause of disease and premature death, currently leading to over five million deaths each year worldwide, which is expected to rise to over 8 million deaths yearly by 2030. **Objectives:** To assess the effect of duration of smokeless tobacco chewing on lipid profile. **Method:** A total of 100 subjects were included, of which 50 were tobacco chewers and 50 were non tobacco chewers (control group) and were sub grouped according to duration of consumption of tobacco. **Method:** Fasting serum lipid profile was done by standard Enzymatic and Precipitate method. **Results:** Levels of serum Triglyceride and VLDL were significantly high ( $P < 0.05$ ) and C- HDL was significantly decreased ( $P < 0.05$ ;  $P < 0.01$ ;  $P < 0.001$ ) in study group with respect to increased tobacco chewing duration. **Conclusion:** Tobacco chewing (Gutka) can be considered as one of the preventive risk factor of Ischemic heart disease.

**Key Words:** Smokeless tobacco, Lipid Profile, Duration.

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### Introduction:

India is the second largest consumer of Tobacco products and third largest producer of tobacco in the world<sup>1</sup> (WHO framework convention on Tobacco control, Geneva, 2009). Nearly 8-9 lakh people die every year in India due to disease related to tobacco use (Gajalakshmi V, *et al* 2003)<sup>2</sup>. Majority of cardiovascular disease, cancers and chronic lung disease are directly attributed to tobacco consumption.

Tobacco use in the form of smoking /smokeless is a major preventable cause of disease and premature death, currently leading to over five million deaths each year worldwide, which is expected to rise to over 8 million deaths yearly by 2030<sup>3</sup>. Nearly 8-9 lakh people die every year in India due to disease related to tobacco use<sup>4</sup>. 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).

In Indian context, Tobacco is consumed in a variety of, both smoking and smokeless form e.g. – bidi, gutkha, khaini, panmasala, hookah, cigarette,

cigars, chillum, chutta, gul, mawamisri etc. The Global adult Tobacco Survey India (GATS India)<sup>5</sup> revealed that more than one-third (35%) of adult use tobacco in some form or the other. Among them 21% adult use only smokeless tobacco 9% only smoke and 5% smoke as well as use smokeless tobacco. The prevalence of overall tobacco use among males is 48% and that among females is 20%. In India, khaini or tobacco lime mixtures (12%) is the most commonly uses smokeless tobacco product, followed by gutkha, a mixture of tobacco, lime and areca nut mixture (8%), betel quid with tobacco (6%) and applying tobacco as dentifrice (5%). The prevalence of smokeless tobacco product is higher in rural than urban areas, however, gutkha is almost equally prevalent in both urban and rural areas<sup>6</sup>.

The mean age at initiation of daily tobacco use for tobacco users is 17.8 years. 60% of these daily tobacco users, use tobacco within 30 minutes of waking up in the morning, 52% of adults are exposed to second hand smoke at home<sup>7</sup>. Smokeless tobacco products such as snuff and chewing tobacco also contain high level of nicotine, which reaches through the skin and mucosal lining of the mouth and nose or by inhalation into the lungs. Depending on “How the tobacco is consumed”, nicotine can reach peak levels in the bloodstream and brain rapidly. Tobacco

contains nicotine which is absorbed on chewing. Gaede et al (1941)<sup>8</sup> found 0.6gm of tobacco contained 15 mg of nicotine, of which 33% was absorbed in ½ an Hour after chewing; 50% at 2Hours; 60% at 4Hours and 90% at 8Hours.

Nicotine stimulation of adrenergic drive raises both blood pressure and myocardial oxygen demand, lipid metabolism with fall in “protective” high density lipoprotein. Active tobacco consumption alters the total serum cholesterol concentration and lipoprotein composition, which directly increases the risk of coronary heart disease<sup>9</sup>.

According to the AHA(American Heart Association), nicotine causes short-term increases in blood pressure, heart rate, and blood flow through the heart. Over time, these can contribute to fatty build-up in the arteries, known as Atheroma which leads to coronary heart disease. Taking into account the above (hazard/ loss/disease) the present study was conducted to find out, the effect of duration (years) of tobacco chewing on lipid profile.

#### **Material and Methods:**

The Present study was conducted in JLN, medical college and Hospital, Ajmer, a tertiary care centre. After obtaining approval from Research Review Board of the institutional ethical committee, a total of 100 subjects were included in prospective study, on voluntary participation basis, of age group between 18 - 60years, irrespective of gender. Out of 100 subjects, 50 were tobacco chewers (Gutka) and 50 were non-tobacco chewers (control group), the study group was further subdivided into 4 groups, according to the duration of consumption of tobacco (10years interval). 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 habit of tobacco chewing (Gutka) 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,  $\beta$  blockers, Thiazide diuretics, oral contraceptive pills, and those who were not willing to participate in study. Every time the quantity consumed is considered to be more of less

equal. The subjects were considered to have been eating an average Indian diet.

5ml of venous blood sample was collected from antecubital vein in the sitting position. After overnight fast (12-14hrs), under aseptic condition sample from all the study participants were collected. All samples were centrifuged and analyzed for serum lipid profile. The serum Total cholesterol (TC), Triglyceride (TG), and High density lipoprotein (HDL-C) were determined enzymatically, while Low density lipoprotein (LDL-C) was calculated using the Friedewalds formula. Elevated TC was defined as having Total cholesterol levels of (>200mg/dl), Low HDL-C was defined as having HDL-C levels of (<40 mg/dl), elevated LDL-C was defined as having LDL-C levels of (>130mg/dl), elevated TG was defined as having Triglyceride levels of (>150mg/dl), as reference.

#### **Statistical Analysis of Data:**

The data obtained for tobacco chewers and control group 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). ANOVA test was used for comparison of intergroup of study variables. P value less than 0.05 was considered as significant. All data were analysed by software SPSS version 2015.

#### **Result:**

This was a hospital based randomized case-control study, comprised of 100 subjects attending OPD at the tertiary care centre, over a period of 2 years. The results are summarized in Table 1. Which shows that serum TC and VLDL-C were significantly high (<0.05) in tobacco chewers (study group) as compared to non-tobacco chewers (control group). It was also found that TC and VLDL-C were highly significantly in duration of 21-30 years and more as compared to lesser period of duration, while HDL-C was significantly decreased ( $P < 0.01$ ) in study group with tobacco chewing duration 11-20 years and very significantly decreased ( $P < 0.001$ ) in study group with tobacco chewing duration of 21-30 years and more as compared to control group. With an increase in duration of tobacco chewing there was increase in TC and LDL-C level, which become statistically highly significant with the

duration of 31-40 years ( $p < 0.001$ ). The mean LDL-C showed an increase with greater duration.

In the present study the mean HDL-C with duration of tobacco chewing in group 0-10 years was 40.6mg/dl. In group 11-20 years was 38.1mg/dl. In group 21-30 years was 37.9mg/dl. The mean HDL-C with duration of tobacco chewing in group 31-40 years was 31.67mg/dl. Statistical

highly significant decrease in HDL-C was observed in 31-40 years duration when compared to controls ( $p < 0.001$ ). Our findings are in agreement with the study of Axelsen A et al<sup>10</sup> who also observed a statistically significant fall in HDL with increased duration of smoking.

**Table:1 Study results showing impact on lipid profile in relation with tobacco chewing duration.**

Sr.no	Parameters	Control group (nontobacco chewers)	Study group (tobacco chewing duration in years)				ANOVA Fvalue (P-value)
			N=50	0-10yrs (n=20)	11-20yrs (n=13)	21-30yrs (n=10)	
1	TC	165.87±18.86	159.8±66.6	168.6±21.10	191.6±20.97	212.5±24.9	0.626 (0.5990)
2	TG	91.04±20.65	128.0± 56.9	152.2±38.16	160.8± 37.3	178.3± 38.31	2.35 (0.075)
3	LDL-C	82.69±11.26	82.8± 17.37	104.6± 16.5	114.9±26.59	134.0± 37.7	0.939 (0.423)
4	VLDL-C	20.97±4.38	28.1±1.72	33.20±6.77	33.95±7.96	38.0±2.08	2.223 (0.008)
5	HDL-C	48.01±10.07	40.69±8.65	38.1±6.97	37.9±3.78	31.67±7.51	5.691 (0.001)

#### Discussion:

The present study comprised of 100 subjects, with age group between 16-60 years. They were further subdivided into groups. Subjects were randomly selected in all the 5 groups, keeping in mind a matching age and sex distribution. The data assembled was subjected to suitable statistical analysis.

Tobacco chewing is associated with changes in blood lipid, resulting in atherogenic risk factor primarily low HDL-C. Nicotine increase causes lipolysis and increases free fatty acids content. Increased fatty acid turnover leads to overproduction of VLDL-C; Total triglycerides; LDL-C and Lowered HDL-C. Cross-sectional studies have shown that smokeless tobacco use seems to have an adverse effect on lipid profile. In a study of 2840 adult men who were smokeless tobacco users had 2.5 times the risk of hypercholesterolemia compared with normal subjects. In another study,

involving 90% of smokeless tobacco users had lower HDL-C and higher triglycerides levels than control group.

In the present study, the mean TC and LDL-C with duration of tobacco chewing in group 0-10 years was 159mg/dl and 82.8mg/dl respectively. The mean TC and LDL-C with duration of tobacco chewing in group 11-20 years was 168.6 and 104.6mg/dl respectively. The mean TC and LDL-C with duration of tobacco chewing in group 21-30 years was 191 and 114mg/dl respectively. The mean TC and LDL-C with duration of tobacco chewing in group 31-40 years was 212.5 and 134mg/dl respectively. The mean TC and LDL-C showed a statistically significant increase with duration ( $p < 0.01$ ).

In the present study it was found that Triglyceride and VLDL-C were progressively increased according to tobacco chewing duration in years. Triglycerides and VLDL-C were significantly increased ( $P < 0.05$ ) in

study group for duration 21-30 years in tobacco chewers as compared to non tobacco chewers.

IngrarHijenmann et al<sup>11</sup> in their study investigated the correlation of serum cholesterol, cigarette smoking and body weight and it was found to be more pronounced. Increasing daily exposure to cigarette smoke was paralleled by increasing cholesterol levels. This study had comparable result to our studies. Smoking causes stimulation of adrenal medulla leading to higher concentration of free fatty acids and nicotine causes higher concentration of catecholamine in plasma leading to pro-atherogenic state and alteration of lipid metabolism and increased profile with fall in protective high density lipoprotein, increased plasma cholesterol and raising atherogenic LDL cholesterol.

Wiley et al<sup>12</sup> found a statistically significant association between the TG levels of smokers and non smokers. In their study mean TG levels among current smokers was 100 mg/dl and among non smokers was 68.4 mg/dl ( $p < 0.05$ ). These observations were similar to the present study.

Brischetto and Connert al<sup>13</sup> have shown that plasma levels of lipid lipo-proteins were related to the duration and amount of cigarette smoked per day. Nicotine from cigarette smoking causes stimulation of adrenal medulla to higher concentration of catecholamine in plasma leading to both decreased HDL-C and attenuated the anti atherogenic properties of HDL constituents.

Similar observations were made by Brischetto and Conner et al, showed that plasma levels of lipid profile and lipoproteins were related to duration of cigarette smoking per day. They found, TC was higher with increasing duration of smoking. On statistical comparison significant association was observed between TC and LDL-C with duration. Though there was an increase in TC and LDL-C with increased duration of smoking for 1-10 years, 11-20 years, 21-30 years also but it was not statistically significant.

In the present study the mean TG and VLDL-C with duration of tobacco chewing in group 0-10 years was 128 and 28.1 mg/dl respectively. The mean TG and VLDL-C with duration of tobacco chewing in group 11-20 years was 152.2 and 33.2 mg/dl respectively. The mean TG and LDL-C with duration of tobacco chewing in group 21-30 years was

160.8 mg/dl and 33.9 mg/dl respectively. The mean TG and VLDL-C with duration of tobacco chewing in group 31-40 years was 178 and 38 mg/dl respectively. On statistical analysis significant increase in TG and VLDL was observed in 31-40 years duration as compared to other groups. Also TG and VLDL show an increase with increase duration.

Our study is in accordance with the above mentioned studies, demonstrate that Total cholesterol, LDL-C and Triglyceride levels were significantly higher in tobacco chewer group in compared to control group, while HDL-C level were significantly lower.

#### **Conclusion:**

From this study, we concluded that an increase prevalence of Triglyceride, VLDL-C and decreased level of HDL-C is found in tobacco chewers. Smokeless tobacco (Gutka) produces adverse effect on lipid profile and the changes become more significant with the increased tobacco chewing duration in years

With the limitations of the study, we conclude that there is a definite impact of tobacco chewing on the lipid profile. Tobacco chewing causes increased Total cholesterol and LDL-C in blood serum which is harmful and may be responsible for, the great risk of developing atherosclerosis in tobacco users than in nontobacco users.

This is a small scale study, however to confirm these findings, more detailed and large scale study is required. It appears that there is increased need of general awareness about the risk factor of chewing tobacco.

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