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Original Article

Effects of Tobacco Chewing on Serum Lipid Profile in South Indian Population

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Abstract

Introduction: Nicotine which is an active ingredient in tobacco stimulates adrenal medulla to release catecholamine. Catecholamines activate the adenyl cyclase of adipose tissue which causes lipolysis of stored triglyceride (TG) and the release of free fatty acids into plasma.

Materials and Methods: A total of 40 healthy adult male participants were recruited in that 20 were non-chewers and 20 were chewers of tobacco. The chewers were again divided into the users of <10 years and users of more than 10 years. 5 ml of blood samples were collected and the serum was separated. The total cholesterol, TG, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) were estimated by enzymatic and precipitation methods.

Results: A significant increase in the total cholesterol, LDL levels were observed in the long-term users of tobacco when compared with non-users and short-term users. However, HDL levels were similar in all the 3 groups. TGs were higher in the control group when compared with tobacco chewers.

Conclusion: Increased levels of total cholesterol and LDL could be considered as risk factors in the developing coronary heart disease. Tobacco chewing can be considered as one of the preventable risk factors of coronary heart disease.

Key words: Coronary artery disease, Lipid profile, Tobacco chewing

INTRODUCTION

Tobacco was introduced by Portuguese merchants in the 16th century and now India is one among the world's top five tobacco producers and consumers.[1] The WHO attributed 4 million tobacco-related deaths every year and is expected to raise 8.4 million deaths by 2020.[2] Various forms of smokeless tobacco products are available which include pan (piper betel leaf filled with sliced areca nut, lime, catechu, and other spices chewed with or without tobacco), pan-masala or gutkha (a chewable tobacco containing areca nut), and mishri (a powdered tobacco rubbed on the gums as toothpaste).[3]

Tobacco is pathogenetically a cholesterol-dependent risk factor and it acts synergistically with other risk factors for the causation of coronary heart disease as the cause of coronary heart disease is a multifactorial. Thus, a strong synergistic interaction exists between hyper cholesterolemia and tobacco consumption in the genesis of coronary heart disease.[4]

Nicotine is the active ingredient in tobacco.[5] Nicotine stimulates adrenal medulla to release catecholamine.[6] Catecholamines are the only hormones which effectively stimulates lipolysis in humans.[7] Tobacco smoking and its effects on lipid profile have been proved by several studies.[8,9] There are very few studies regarding the effect of tobacco chewing on lipid profile. Hence, this study is conducted to determine the effect of tobacco chewing on lipid profile.

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MATERIALS AND METHODS

This study was conducted on 40 male subjects in the age group ranging from 20 to 50 years. Twenty subjects were non-chewers of tobacco and 20 were chewers of tobacco. The chewers or tobacco users were divided into

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2 groups again. They were tobacco chewers of <10 years (9 subjects) and tobacco chewers of more than 10 years (11 subjects). Thus, there were total 3 groups - non-users, users <10 years, and users more than 10 years. Care was taken to see the average age of controls and chewers were same. All the research participants were explained about the procedures and recruited after obtaining informed consent. Subjects with multiple tobacco habits, alcoholics, liver diseases, chronic renal failure, nephrotic syndrome, hypothyroidism, diabetes mellitus, drugs (β blockers, glucocorticoids, thiazide diuretics, and lipid lowering drugs), and also with other chronic illness were excluded from the study.

About 5 ml blood samples were collected after an overnight fasting and serum was separated from the blood. The serum lipid profile was studied and the lipid levels were calculated by Freidewald's formula. Estimation of total cholesterol, triglycerides (TGs), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) was done by standard methods.

RESULTS

The mean and standard deviations of total cholesterol levels of the non-users, users of <10 years and >10 years were 148.6 ± 32.63, 144.1 ± 23.84 and 169.6 ± 25.95, respectively. It is found that there is no significant difference in cholesterol values between non-users and users of <10 years. However, long-term users (<10 years) show increased cholesterol levels compared to short-term users. This may be due to the long-term effects of sustained blood nicotine values. These data are shown diagrammatically in Table 1 and Figure 1.

Serum TG levels in three groups were shown in Table 2. Remarkably non-users have higher levels than the other two groups. In fact, there is statistically significant difference between non-users and users of <10 years. This may be an incidental finding as this is a cross-sectional study.

There seems to be no difference in the HDL cholesterols (HDL-C) of three groups, but the LDL cholesterol (LDL-C) levels seem to be increasing with chewing tobacco. There is gradual increase in the blood levels of LDL-C with the users of over 10 years showing maximum blood levels (Table 3). There is a statistically significant difference between non-users and users as well as between users of <10 years duration and the users of >10 years duration.

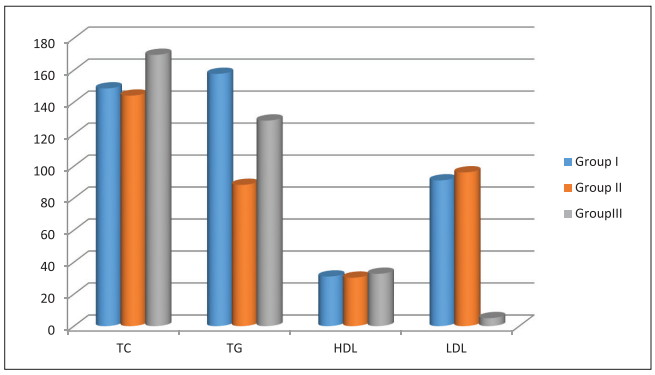

Figure 1: The parameters (triglyceride, high-density lipoprotein and low-density lipoprotein) in three groups

Table 1: Serum cholesterol levels in all three groups

Mean, SD and SE	Non users	Users <10 years	Users >10 years
Number	20	9	11
Mean	148.6	144.1	169.6
SD	32.63	23.84	25.95
SE of mean	7.30	7.95	7.83

t=2.27, *P*<0.02 (between users of <10 years and >10 years). SD: Standard deviation, SE: Standard error

Table 2: Serum triglyceride levels in all three groups

Mean, SD and SE	Non users	Users <10 years	Users >10 years
Number	20	9	11
Mean	157.7	88.3	128.5
SD	90.29	61.44	61.78
SE of mean	20.19	20.48	18.63

t=2.09, *P*<0.05 (between non-users and users of <10 years). SD: Standard deviation, SE: Standard error

Table 3: HDL-cholesterol levels

Mean, SD and SE	Non users	Users <10 years	Users >10 years
Number	20	9	11
Mean	31.0	30.3	32.7
SD	2.71	3.12	5.18
SE of mean	0.61	1.04	1.56

Not significant. SD: Standard deviation, SE: Standard error, HDL: High-density lipoprotein

Table 4: Serum LDL-cholesterol levels

Mean, SD and SE	Non users	Users <10 years	Users >10 years
Number	20	9	11
Mean	91.1	96.1	111.2
SD	28.07	12.07	13.68
SE of mean	6.28	4.02	4.12

t=2.22, *P*<0.05 (between non-users and users of<10 years). *t*=2.59, *P*<0.02 (between users of <10 years and users of >10 years. SD: Standard deviation, SE: Standard error, HDL: High-density lipoprotein

This proves that there is gradual increase of LDL-C levels in tobacco chewers. These data are shown diagrammatically in Figure 1 and Table 4.

DISCUSSION

Nicotine which is an active ingredient in tobacco stimulates adrenal medulla to release catecholamine. Catecholamines activate the adenyl cyclase of adipose tissue which causes lipolysis of stored TG and the release of free fatty acids (FFAs) into plasma. The released FFAs are immediately bound to plasma albumin and are then transported to various tissues of the body particularly to the liver. Hepatic TG and very LDL-C (VLDL-C) synthesis is stimulated by increased influx of FFA. The increased levels of plasma FFAs could act to depress the plasma HDL-C and increases plasma TG and VLDL-C.[1]

In this study, total serum cholesterol was higher in long-term users when compared to non-users and users of <10 years. There was no significant difference between the non-users and the users of <10 years. This could be explained as due to long-term effects of sustained blood nicotine levels. This was also recorded by other Indian workers. Khurana *et al.* and Rao and Subash observed a rise in the levels of total cholesterol, TG, LDL, and VLDL with a decrease in the HDL level in smokers and tobacco chewers, which was in concurrence with the results of this study in relation to total cholesterol and LDL levels, whereas TGs were higher in non-users than users and the levels of HDL were similar in all the groups.[4,10] Latha *et al.* administered nicotine to rats and found that the concentration of TGs increased in both serum and tissues.[11] Our study results are consistent with the above two studies with reference to total serum cholesterol.

The serum TG levels in this study, however, show a different picture. It is higher in the non-users compared to users of more than 10 years. This is perhaps an incidental finding because this is a cross-sectional study and the average age of non-users is slightly higher than users. However, the long-term users (<10 years) have higher levels of TGs compared to the short-term users (users of <10 years).

The HDL levels in the study and control groups did not show any difference. However, there was a notable gradual increase in the blood levels of LDL-C. The non-users have the lowest levels and the long-term users have the highest levels. This can be explained as one of the chronic effects of sustained blood nicotine.

CONCLUSION

With the limitations of this study, we could conclude that there is a definite impact of chewing tobacco on the serum lipid profile. Tobacco chewing causing increased total cholesterol and LDL levels in the blood serum which is harmful and may be responsible for the greater risk of developing atherosclerosis in the tobacco users than in the non-tobacco users.

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