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A Study of Lipid Profile in Tobacco Chewers and Smokers

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Abstract

Aim: *To evaluate & compare the atherogenic ratio and to determine the extent of cardiovascular risk in tobacco chewers (T) & smokers (S).* ***Methods:*** *The present cross-sectional study was conducted at GMCH, Nagpur between November 2004 to June 2006. In all 100 subjects & 50 controls attending OPD of GMCH, Nagpur between age groups 20-60 years satisfying all the inclusion & exclusion criteria and willing to give consent were included in the study. Samples were withdrawn in fasting state & lipid profile was estimated using Accurex kits (Autozyme). Total cholesterol (TC) was estimated by CHOD-POD method, Triglycerides (TG) by GPO-PAP method, High density lipoprotein cholesterol (HDLc) by sodium phosphotungstate-Mg++ method. Very low density lipoprotein cholesterol & Low density lipoprotein cholesterol by using formula; VLDLc = TG/5 & LDLc = TC – HDLc – VLDLc respectively.* ***Results:*** *We observed significant increase in mean levels of TC, LDLc, LDLc/HDLc & TC/HDLc and significant decrease in mean HDLc in T & S as compared to controls.* ***Conclusions:*** *Tobacco chewing & smoking causes decrease in HDLc & increase in TC, LDLc, LDLc/HDLc & TC/HDLc indicating that they were independently associated with such an unfavorable lipid profile thereby greatly increasing the cardiovascular risk particularly for coronary artery disease. Cigarette smoking was found to be more atherogenic than tobacco chewing. However, bidi & cigarette smoking carry equal cardiovascular risk as far as alterations in lipid profile was concerned.*

Keywords*: CHOD-POD, GPO-PAP, Sodium phosphotungstate-Mg++ method*

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Introduction

In the 16th century Portuguese sailors and merchants were the first to introduce tobacco in Asia. Indian sailors took tobacco along with Yopa from Rio Guaviare in Colombia during mid 16th century and spread its use as a common practice all over India.[1] From national level studies such as from National Sample Survey Organization (1998) it was reported that 47% of men and 14% of women consumed some form of tobacco with 16.2% current smokers (29.4% men and 2.3% women) and 20.5% tobacco chewers (28.3% men and 18% women).[2] India is one among the world's top five tobacco producers and consumers.[3] Two major forms of tobacco use in India are smoking (bidis or cigarettes) and chewing. A 'bidi' is a local cigarette made of strong tobacco rolled in a special leaf (temburni) which is grown abundantly all over India. Tobacco is chewed either alone or mixed with slaked lime, betel leaf and areca nut.[4] The currently estimated 1.25 billion smokers (1 billion males and 250 million females) represent one-third of the world's population. The WHO attributed 4 million tobacco related deaths every year and is expected to raise 8.4 million deaths by 2020.[3] Cigarette smoking increases the risk of coronary heart disease, stroke, peripheral vascular disease (PVD) and aortic aneurysm.[5] On the other hand smokeless tobacco may increase the risk of cardiovascular disease and cancers of larynx, esophagus and of other sites as well as gingival and periodontal diseases.[6] The American heart

association estimates that one-fourth of fatal heart attacks are caused by cigarette smoking (120,000/year). Tobacco smoke contains 43 carcinogenic substances, over 4,000 gases, particles and compounds such as tar, nicotine and carbon monoxide which pollute tobacco. Nicotine is a psychoactive compound and is only found in tobacco. This compound is the primary cause for addiction to tobacco products because it mildly stimulates the central nervous system.[7] Despite the fact that the epidemiological evidence linking cigarette smoking with cardiovascular diseases is overwhelming, the precise components of the cigarette smoke responsible for this relationship and the mechanism by which they exert their effects have not been elucidated. There is considerable evidence that cigarette smoking can result in both morphological and biochemical disturbances to the endothelium both in vivo and in cell culture system.[8] An association between smoking and plasma lipids and lipoproteins concentration has been shown in general studies.[5] There are very few studies regarding the effect of tobacco chewing on lipid profile. Hence, present study is conducted to determine and to compare the extent of effect of tobacco both smoked and chewed on lipid profile.

Materials and Methods

The present cross-sectional study was conducted in Government medical college and hospital (GMCH), Nagpur from November 2004 to June 2006. Here we included only those tobacco chewers who chewed either tobacco lime preparation, ghutka or khaini and only those smokers who smoked either bidi or cigarette according to availability, although various tobacco habits are prevalent in India. In all 100 subjects attending out patient department (OPD) or admitted in medicine/surgery wards of GMCH, Nagpur in the age group between 20-60 years, satisfying all the inclusion and exclusion criteria and willing to give consent were included in this study. Ethical clearance was obtained from institutional ethics committee. These criteria are as follows:

1] Inclusion criteria:

a] Tobacco chewers (T) (n = 50) These subjects were habitual tobacco chewers with no other mode of tobacco use, consuming approximately 7 grams of tobacco either through tobacco slake lime preparation, ghutka or khaini for more than 5-10 years. They were further subdivided into; those consuming > 2 pouches/ day, those consuming 1-2 pouches/ day, those consuming < 1 pouch/ day.

b] Smokers (S) (n = 50) These were males with following mode of tobacco use and were subdivided into...

Bidi smokers (n = 25)	Cigarette smokers (n = 25)
Those smoking > 20 bidis/ day	Those smoking > 20 cigarettes/ day
Those smoking 11 -19 bidis/ day	Those smoking 11-19 cigarettes/day
Those smoking < 10 bidis/ day	Those smoking < 10 cigarettes/ day

2] Exclusion Criteria[9, 10]

Subjects having any of the following were not taken into study; subjects with multiple tobacco habits, females, alcoholics, liver diseases, chronic renal failure, nephrotic syndrome, hypothyroidism, diabetes mellitus, drugs (β blockers, glucocorticoids, thiazide diuretics, lipid lowering drugs).

After satisfying the inclusion or exclusion criteria the study subjects were examined according to the predesigned proforma and the findings were recorded along with the consent from each subjects. We selected 50 healthy normal male volunteers between 20-60 years of age as controls. The selected study groups i.e; Controls (n = 50), tobacco chewers (n = 50) and smokers (n = 50) were asked to fast overnight for at least 12 hours.[11] The next day in the morning about 10 ml of blood sample was collected in plain bulb through disposable syringe and needle under proper aseptic precautions from the veins of the forearm according to feasibility in the sitting position. The sample was centrifuged in centrifuge machine and the serum was collected in serum collectors.[11] The serum tubes were preserved at 2°- 8° C in the refrigerator until analysis.[11] The stored serum were analyzed within 1-2 days for lipid profile using ACCUREX KITS (Autozyme) at Clinical Biochemistry laboratory, GMCH, Nagpur. The samples were processed on semiautoanalyser - ERBA CHEM - 5 at

Super Speciality Hospital and Post Graguate Research Institute, Nagpur. TG was measured by GPO-PAP method.[12] TC was estimated by CHOD-POD method.[12] High Density Lipoprotein Cholesterol (HDLc) was estimated by sodium-phosphotungstate-Mg++ method.[12] VLDLc & LDLc were calculated using formula; VLDLc = TG/5 & LDLc = TC – HDLc - VLDLc respectively.[12]

Results

Table 1 shows that when mean TG levels in C was compared with mean TG levels in T & S it was found to be statistically nonsignificant with p>0.05. Similarly, when mean TG levels in T and S were compared it was found to be nonsignificant with p>0.05. Further, when mean TG levels in B was compared with mean TG levels in Ci it was found to be again nonsignificant with p>0.05. Only 05 controls, 05 T, 03 B and 07 Ci were having serum TG levels of >150.

Table- 1: TG, TC, HDLc, LDLc (mg%) among study groups

Study groups	TG mg%	p-value	TC mg%	p value	HDLc mg%	p value	LDLc mg%	p value
Controls (C) (n = 50)	121.42 ± 24.16	C vs T p > 0.05	179.06 ± 19.43	C vs T p < 0.01	38.9 ± 3.54	C vs T p < 0.01	115.57 ±18.07	C vs T p < 0.01
Tobacco chewers (T) (n = 50)	124.84 ± 26.28	C vs S p > 0.05	198.04 ± 21.59	C vs S p < 0.01	34.24 ± 4.41	C vs S p < 0.01	138.81 ±21.24	C vs S p < 0.01
Smokers (S = B + Ci) (n = 50)	129.5 ± 18.29	T vs S p > 0.05	215.36 ± 14.23	T vs S p < 0.01	32.28 ± 3.79	T vs S p < 0.02	157.06 ±14.36	T vs S p < 0.01
Bidis (B) (n=25)	126.44 ± 18.06	B vs Ci p > 0.05	207.28 ± 11.30	B vs Ci p < 0.01	32.16 ± 3.70	B vs Ci p>0.05	149.07 ±11.96	B vs Ci p < 0.01
Cigarettes (Ci) (n=25)	132.56 ± 18.36	Ci vs B p > 0.05	223.44 ± 12.25	Ci vs B p < 0.01	32.4 ± 3.92	Ci vs B p>0.05	165.05 ±12.04	Ci vs B p < 0.01

Table 2: VLDLc, LDLc/HDLc, TC/HDLc among study groups

Study groups	VLDLc mg%	p value	LDLc /HDLc	p value	TC/ HDLc	p valve
Controls (C) (n = 50)	24.38 ± 4.79	C vs T p > 0.05	3.03 ± 0.70	C vs T p < 0.01	4.67 ± 0.88	C vs T P < 0.01
Tobacco chewers (T) (n = 50)	24.93 ± 5.16	C vs S p > 0.05	4.20 ± 1.19	C vs S p < 0.01	4.95 ± 1.43	C vs S P < 0.01
Smokers (S = B + Ci) (n = 50)	25.9 ± 3.66	T vs S p > 0.05	4.94 ± 0.79	T vs S p < 0.01	6.77 ± 0.94	T vs S p < 0.01
Bidis (B) (n=25)	25.28 ± 3.61	B vs Ci p > 0.05	4.74 ± 0.76	B vs Ci p > 0.05	6.54 ± 0.91	B vs Ci p > 0.05
Cigarettes (Ci) (n=25)	26.51 ± 3.67	Ci vs B p > 0.05	5.16 ± 0.78	Ci vs B p > 0.05	6.99 ± 0.94	Ci vs B p > 0.05

Table shows that when mean TC levels in C was compared with mean TC levels in T & S it was found to be statistically highly significant with $p<0.01$. When mean TC levels in T was compared with mean TC levels in S, it was found to be highly significant with $p<0.01$. When mean TC levels in B was compared with mean TC levels in Ci it was found to be highly significant with $p<0.01$. Only 07 C and 22 T, while majority of B (19) and Ci (25) were having serum TC levels of ≥200. When mean HDLc levels in C was compared with mean HDLc levels in T & S it was found to be statistically highly significant with $p<0.01$. When mean HDLc levels in T and S were compared it was found to be significant with $p<0.02$. However, when mean HDLc levels in B was compared with mean HDLc levels in Ci it was found to be nonsignificant with $p>0.05$. None of the C and 24 T, while majority of B (21) and Ci (18) were having serum HDLc levels of <35. When mean LDLc levels in C was compared with mean LDLc levels in T & S it was found to be statistically highly significant with $p<0.01$. Similarly when mean LDLc levels in T and S were compared it was found to be highly significant with $p<0.01$. However, when mean LDLc levels in B was compared with mean LDLc levels in Ci it was found to be highly significant with $p<0.01$. None of the C and 09 T, 07 B, and majority of Ci (17) were having serum LDLc levels of ≥ 160. Table 2 shows that when mean VLDLc levels in C was compared with mean VLDLc levels in T & S it was found to be statistically nonsignificant with $p>0.05$. When mean VLDLc levels in T was compared with that in smokers it was found to be nonsignificant with $p>0.05$. When mean VLDLc levels in B was compared with mean VLDLc levels in Ci it was found to be nonsignificant with $p>0.05$. Only 05 C, 05 T, 03 B and 07 Ci were having serum VLDLc levels of ≥30. When mean atherogenic ratio LDLc/HDLc & TC/HDLc levels in C were compared with mean of those in T & S it was found to be statistically highly significant with $p<0.01$. When mean LDLc/HDLc & TC/HDLc levels in T were compared with mean of those in smokers it was found to be highly significant with $p<0.01$. When mean LDLc/HDLc & TC/HDLc levels in B were compared with those in Ci it was found to be nonsignificant with $p>0.05$. None of the C and 20 T, while majority of B (19) and Ci (21) were having LDLc/HDLc ratio levels of > 4.5. 42 C and all the T, B and Ci were having TC/HDLc ratio levels of ≥3.5.

Discussion

According to Brischetto C S et al[13] increased in blood carbon monoxide in the blood of cigarette smokers may damage the endothelium and accelerate the entry of cholesterol into the wall of coronary artery. The formation of carboxyhemoglobin in smokers creates relative anoxemia in the tissues including the myocardium. Smoking enhances platelet aggregation. Nicotine absorption from cigarette smoke may induce cardiac arrhythmias through its pharmacological actions. Smoking adversely affects the concentration of the plasma lipids and lipoproteins. Nicotine absorbed from cigarette smoke stimulates the secretion of epinephrine and norepinephrine as well as other hormones like cortisol and growth hormone. This enhanced hormonal secretion would occur 20 or more times a day in a habitual smoker. Catecholamines and other hormone activate the adenyl cyclase of adipose tissue which causes lipolysis of stored TG and the release of free fatty acids into plasma. Chain smokers have increased plasma free fatty acids levels 3 fold over the baseline value. The released free fatty acids [FFA] are immediately bound to plasma albumin and are then transported to various tissues of the body particularly to the liver. Hepatic TG and VLDLc synthesis is stimulated by increased influx of FFA. In smokers, due to the effect of smoking the plasma free fatty acids level increases which then could act to depress the plasma HDLc & increases plasma TG & VLDLc. According to Sinha A K et al[14] smokers are believed to consume diet rich in fats and cholesterol and poorer in fibre and cereals. There are differences in fat intake between smokers and nonsmokers due to psychological and attitudinal changes. Following food intake normally there is increased levels of HDLc & TC & decreased levels of LDLc. According to Neki N S et al[15] nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased concentration of plasma FFA which further results in increased secretion of hepatic FFA, TG along with VLDLc in the

blood stream. Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL.

Conclusions

Tobacco chewing & smoking causes decrease in HDLc & increase in TC, LDLc, LDLc/HDLc & TC/HDLc indicating that they were independently associated with such an unfavorable lipid profile thereby greatly increasing the cardiovascular risk particularly for coronary artery disease. Cigarette smoking was found to be more atherogenic than tobacco chewing. However, bidi & cigarette smoking carry equal cardiovascular risk as far as alterations in lipid profile was concerned.

Conflict of Interest: *None declared*
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