A STUDY ON EFFECTS OF CIGARETTE SMOKING ON BLOOD CHOLESTEROL IN YOUNG POPULATION OF AHMEDABAD

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Abstracts: **Backgrounds and objectives**: According to the Indian Council of Medical Research (ICMR), India records about 800,000 tobacco deaths every year or 2,200 deaths a day. Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease.**Material and method**: Our study was performed on 100 normal asymptomatic healthy males (50 controls, 50 cigarette smokers) with age-group between 18-32 years. The effects of smoking on blood cholesterol were studied. **Results and Interpretation**: They had elevated total cholesterol compared to age and sex matched controls. Cigarette leads to increase in the concentration of serum total cholesterol, triglycerides, LDL-cholesterol, VLDL-cholesterol and fall in the levels of anti-atherogenic HDL cholesterol, as reported by various workers. **Conclusion**: We observed a dose response relationship between the number of cigarettes smoked and cardiovascular morbidity and mortality.
Key Words: Cigarette smoking, atherosclerosis, cholesterol, lipoprotein.

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Introduction:

Tobacco consumption in different forms is a common addiction in the socio-economically handicapped population in many developing countries. Although tobacco abuse in any form is a widely recognized health hazard and a major cause of mortality, people continue to consume it on a regular basis.[22] People sufferes many consequences of smoking such as cardiovascular disease, pulmonary disease and oxidative stress.

According to the world health organization (WHO) approximately one third of world population older than 15years, are consuming tobacco.[11,17] Cigarette smoking is one of the most extensively used potentially hazardous social habits throughout the world but more extensively prevalent in South East Asia . Tobacco consumption is now increasing rapidly throughout the developing world and is one of the biggest threats to current and future world health.[22] The highest prevalence of it is observed in young adult male during their reproductive period between 20 to 39 years.[23]

Today tobacco consumption has been established as a number one preventable cause of death and disease in the countries worldwide. About 30-40% of the all the death from cancer are associated with tobacco consumption. Recent data suggest that tobacco consumption is not only associated with lung cancer but also associated with increased incidence of cancer in larynx, oral, esophagus, cervix, bladder, pancreas and even leukemia [13].

Cigarette smoking is considered a major risk factor for coronary heart disease1 .It has also been shown that a decrease of high density lipoprotein (HDL) cholesterol is a risk factor for the same [2].In normal individuals; HDL cholesterol should be about 20 per cent of the total cholesterol[3]. Craig et aI have analysed data from 54 studies on blood lipid changes in smokers. These studies are mostly on Western subjects. We have studied serum lipids in Indian smokers to investigate any relationship between smoking and serum cholesterol and the HDL fraction.

Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further result in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL- C in the blood stream; (b) Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL – cholesterol; (c) Presence of hyperinsulinaemia in smokers leads to increased

cholesterol, LDL-C, VLDL-C, and TG due to decreased activity of lipoprotein lipase; (d)

Consumption of a diet rich in fat and cholesterol as well as a diet low in fibre and cereal content by smokers as compared to non-smokers.[3,4]
Cigarette smoke contains a large number of substances including nicotine, carbon monoxide and recognizes carcinogens and mutagens such as radioactive polonium, benzopyrene, dimethylnitrosamine. Various mutagens and carcinogens or other toxic components in cigarette smoke disrupt the testicular microcirculation and cause DNA or chromosomal damage in germinal cells. [1]

Although increased total cholesterol raises the risk of cardiovascular disease, a lower level of serum cholesterol is associated with an increased risk of non-cardiovascular causes of mortality, including cancer and respiratory disease. Thus, the present study was aimed to affirm the deleterious effects of smoking on lipid profile and to study association between lipid profile and smoking.

Material and Methods:
The study was conducted on 100 subjects (50 control and 50 smokers) after obtaining permission from Institutional Ethics Committee. The subjects, enrolled for the study were informed about the study and procedural details and an informed consent was obtained. In order to exclude conditions that might influence the results, the recruitment of subject was done on the basis of following criteria. They were all vegetarians and belonged to the age group of 18 to 32 years. They were also indulging in equivalent physical exercise and did not consume alcohol.

The Non-smoker control volunteers were also 50 in number, and were age and sex matched, and had comparable diet and physical activity and no addiction. Those excluded from the study were persons abusing alcohol, ex-smokers, suffering from coronary artery disease, heart failure, cardiac valve disease, respiratory disease, diabetes mellitus, hypertension, renal disease, hepatic impairment, endocrine disorders, obese, on drugs like β-blockers, lipid lowering drugs, thiazide diuretics, involved in competitive sports, hard-working labours and persons with sedentary life style. They were categorised into mild smokers (1-10 cigarettes per day) moderate smokers (11-20

Cigarettes per day) and sever smokers (> 20 cigarettes per day).

Clinical assessment

All participants provided information on age, family history, personal habits (alcohol intake, tobacco consumption, type and level of physical exercise, drug ingestion, known pathological conditions). A detailed physical examination was conducted to exclude cardiac or pulmonary diseases.
Anthropometric variables like height and weight were obtained

Blood sample were collected after overnight fasting. 4cc of blood was collected in plain bulb and the fresh, clear serum with no Hemolysis was separated for following tests:

1. Total serum cholesterol
2. Serum triglyceride
3. Serum HDL
4. Serum LDL and Serum VLDL were derived by formula.

Blood was taken at the same time in all cases and the investigations were done within 2 hours of collecting blood. Total cholesterol as estimated by the method of Schoenheimer and Sperry modified by Venugopala Rao and Ramakrishnan while
HDL cholesterol was analysed by the technique of polyanion precipitation with Heparin and managa•nous chloride and estimated as cholesterol in the supernatant.

Statistical Analysis:

Data was expressed as mean value ± standard deviation and corresponding 95% confidence intervals (CIs) and comparisons between the three groups were performed using one-way analysis of variance (ANOVA), and unpaired t test was used for comparisons between two groups.

Result:

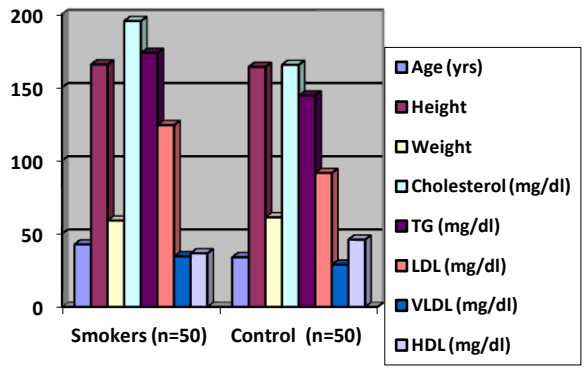
TABLE I: Showing fasting serum lipid profile in smokers and non-smokers.

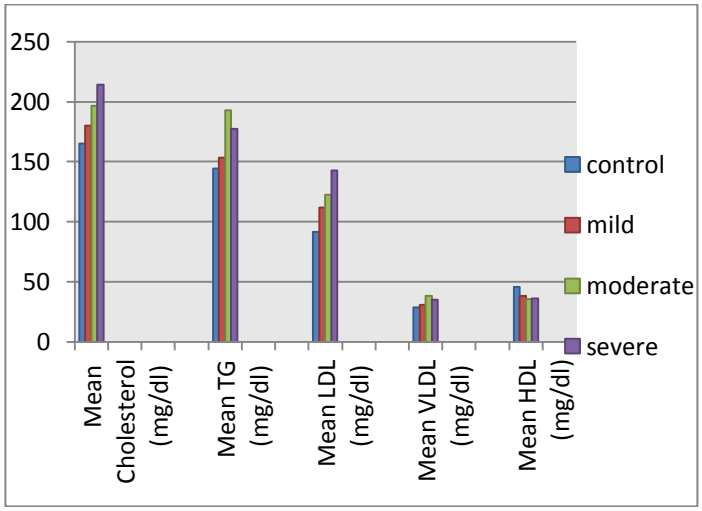
Parameters	*Smokers (n=50)*	*Control (n=50)*	P Value
Age (yrs)	42.68±6.99	33.9±11.67	<0.05
Height	165.6±5.94	163.96±8.82	<0.05
Weight	59±11.17	61.2±11.71	>0.05
Cholesterol (mg/dl)	196.25 ±50.32	162.54±42.19	<0.05
TG (mg/dl)	183.46 ±80.42	140.35 ±90.13	>0.05
LDL (mg/dl)	124.17 ±50.79	90.15±40.38	<0.05
VLDL (mg/dl)	34.55 ±15.69	28.9 ±18.97	>0.05
HDL (mg/dl)	35.04 ±10.19	46.06±15.29	<0.05

TABLE II: Showing the comparison of lipid profile with severity of cigarette smoking.

Tests	(controls (n = 50)	Smokers Cigarettes per day		
		Mild (1-10) (n = 20)	Moderate (11-20) (n=14)	Severe More than 20 (n=16)
Mean Cholesterol	162.54 ± 42.19	179.85± 49.97*	196.42± 54.97*	214.06± 55.22*
Mean TG	140.35 ± 90.12	153.45± 54.00	192.77± 88.39*	177.33± 92.17*
Mean LDL	90.15 ± 40.38	111.58± 47.25*	122.38± 53.51*	142.69± 50.39*
Mean VLDL	28.9 ± 19.38	30.70± 10.83	38.33± 17.54*	35.23± 18.65*
Mean HDL	46.06± 15.29	38.17± 11.14*	35.46± 12.67*	36± 4.74*

*p value<0.05– significant





Discussion:

It is revealed that serum triglycerides, LDL, VLDL, and Cholesterol were significantly higher in smokers as compared to non-smokers thereby revealing a direct dose response relationship. It is also revealed that serum triglycerides, LDL, VLDL, and Cholesterol were significantly higher in moderate and severe smokers as compared to non-smokers, Details of present study showed (Table No-I, III) that the mean serum total cholesterol in non-smokers was 162.54±42.19mg/dl while it was significantly higher in smokers, i.e., 196.25 ±50.32 mg/dl. Cigarette smoking substantially increases the risk of coronary heart disease and ischaemic stroke. These observations are in tune with findings of other workers like NS Neki et al and Chattopadhyay et al [18]

The mean serum triglycerides levels in non smokers and smokers were 140.35±90.13 mg/dl and 183.46 ±80.42mg/dl respectively. The mean LDL value in nonsmokers and smokers were 90.15±40.38mg/dl and 124.17±50.79. the mean HDL-C in non-smokers and smokers was 46.06±15.29 mg/dl and 35.04 ±10.19 mg/dl respectively. An increase of cholesterol (as LDL) might be suggestive of an increase of VLDL from which LDL is formed by delipidation.

A greater increase of LDL and VLDL may cause a greater decrease of HDL as there is a reciprocal relation between the concentration of VLDL and HDL. The interesting finding is therefore a greater increase of total cholesterol and greater decrease of HDL cholesterol in Indian smokers compared to Westerners. As both an increase of total cholesterol and a decrease of HDL cholesterol are risk factors for coronary heart disease, Indian smokers appear more susceptible to heart disease as compared to their Western counterparts.[7,8]

The mechanism of increase of blood cholesterol is through an increase of free fatty acids of blood. In smokers, the nicotine ingested stimulates the secretion of catecholamines. These hormones increase
the FFA by lipolysis of adipose tissue fat. The FFA reaching the liver are esterified as Triacylglycerol and cholesteryl esters which are secreted into the blood stream as VLDL which gets converted to LDL in circulation. The greater the release of FFA, the greater the levels of LDL and cholesterol.[6,9,10,11]

The decrease of HDL may be due to 3 factors. Increase of VLDL will cause a decrease of HDL due to poorer availability of phospholipid remnants from VLDL for HDL formation. It may also be due to decreased Apo A content, as Apo A is needed for the formation of HDL, and finally, diminished lecithin cholesterol acyl transferase (LCAT) needed for the formation of HDL .[10,11]

A decrease of Apo A can cause a diminished activity of LCAT as Apo A is its activator. Again, an accumulation of Cadmium may cause a decrease of both Apo A and LCAT (which are proteins) by heavy metal precipitation.[16]

Conclusion:

We observed the dose response relationship between cigarettes smoked and increase in the atherogenic parameters and decline in anti atherogenic parameters.

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