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 Association of CigaRETTE SMOKING AND RAISED BLOOD CHOLESTEROL EVELS IN YOUNG POPULATION ATTENDING OUTPATIENT DEPARTMENT OF A TERTIARY CARE HOSPITAL

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 ABSTRACT
 BACKGROUND AND OBJECTIVES: According to the WHO, India is home to 12% of the world's smokers. More than Inilion die each year due to tobacco in India. Cigarette smoking is one of the most extensively used potentially

hazardous social habits throughout the world.

MATERIAL AND METHOD: The study was conducted on 150 normal asymptomatic healthy males. Out of which 75 were controls and 75 were cigarette smokers with age-group between 17-35 years. The association of cigarette smoking and raised blood cholesterol levels was studied.

RESULTS: The study showed, cigarette had raised concentration of serum total cholesterol, triglycerides, LDL-cholesterol, VLDL-cholesterol and had reduced levels of HDL cholesterol.

CONCLUSION: We observed a dose response relationship between the number of cigarettes smoked and cardiovascular morbidity and mortality.

KEYWORDS : Cigarette smoking, cholesterol, atherosclerosis.

INTRODUCTION:

Tobacco smoking is the practice of smoking tobacco and inhaling tobacco smoke consisting of particle and gaseous phases. Tobacco consumption in different forms is a common addiction in the socioeconomically handicapped population in many developing countries.¹ According to a 2002 WHO estimate, 30% of adult males in India smoke. Among adult females, the figure is much lower at between 3–5%. Tobacco smoking harms nearly every organ of the body, causes many diseases and reduces the health of smokers in general.²³

Cigarette smoking is more extensively prevalent in South East Asia. In 2010, an estimated 120 million Indian adults smoked, making India second only to China in number of smokers. In 2010, smoking caused about 1 million deaths, or 10% of all deaths in India, with about 70% of these deaths occurring at the ages of 30–69 years.⁴ Cigarette smoking contributes to Cardiovascular Disease in a number of ways and leading to atherosclerosis or plaque (fatty streaks) deposited within the inner layers of the arteries, it is slow and complex, often starting in childhood and progressing with age. Smoking also has a direct effect on platelets, leading to increased activation and stickiness. This in turn causes an increased risk of thrombosis or development of blood clots.⁵

According to the Centers for Disease Control and Prevention (CDC), one-third of all deaths from cardiovascular disease are caused by smoking. A significant increase of serum Cholesterol, Triglyceride and Low-density lipoprotein with significant decrease in serum High density lipoprotein level (HDL) has been documented in smokers. The most well-documented impact that smoking has on cholesterol is how it lowers levels of HDL. HDL has protective effects against heart disease, it's particularly beneficial for women, who typically have higher levels of the cholesterol than men.⁶ Thus, the study was aimed to determine the deleterious effects of smoking on lipid profile and to study association between lipid profile and smoking.

MATERIAL AND METHODS:

A cross-sectional descriptive study was conducted on 150 subjects (75 control and 75 smokers) attending the outpatient clinic of the Medicine department of LAMC, Raigarh after obtaining permission from Institutional Ethics Committee. The study was carried out from April–May 2018.

Smokers and non-smokers who attended the outpatient clinic as a patient, attendant, volunteer and staff were recruited in the study. 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, belonged to the age group of 17 to 35 years, indulged in equivalent physical exercise and did not consume alcohol. Patient suffering from chronic diseases or taking medications for lipid lowering were excluded.

Patients were categorised into mild smokers (1-10 cigarettes per day), moderate smokers (11-20 Cigarettes per day) and sever smokers (> 20 cigarettes per day).

A self-administered questionnaire was used to obtain the demographic data of the participants. 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). In addition, all participants were asked to perform their blood test in fasting condition for at least 12 hours before collecting the blood sample. All blood samples were tested at the hospital laboratory. 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 managanous chloride and estimated as cholesterol in the supernatant.

STATISTICAL ANALYSIS- The recorded observations were put into Epi info software. Data was expressed as mean value \pm standard deviation at 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 (smoker and Non-smoker).

RESULTS: Table 1- Anthropometric and Lipid Profile in Smokers and Non-Smokers

Parameters	Smokers (n=75)	Non- Smokers (n=75)	p- value
Age (years)	41.52±7.20	34.20±12.68	<0.05*
Weight (kg)	62.08±10.47	60.20±10.70	NS

Height (cm)	165.40±6.22	164.96±8.80	NS
Cholesterol (mg/dl)	202±38.32	164±34.11	<0.05*
LDL (mg/dl)	128.11±46.71	138.20±86.11	<0.05*
Triglycerides	192±78.40	88.10±38.38	NS
HDL	35.44±8.90	48.76±16.20	<0.05*

*P<0.05 is significant, NS- not significant.

As per Table 1 it was clearly seen that Lipid profile of smokers was significantly higher in terms of cholesterol and LDL, while HDL is significantly higher in Non-Smokers. (p<0.05).

Table 2- Comparison of Lipid Profile parameters with Severity of Smoking

Parameters	Mild (n=20)	Moderate (n=27)	Severe (n=28)
Cholesterol	182.22±12.10*	198.10±22.20*	220.42±34.46*
LDL	130.11±44.67*	134.32±48.98*	142.48±48.99*
Triglycerides	188.10±54.56*	190.34±52.50*	200.10±58.98*
HDL	36.34±4.50*	32.42±3.32*	28.98±2.10*

*P<0.05 is significant

As per table 2 mean cholesterol, LDL, Triglycerides levels were increased with the severity of smoking which was found to be significant. While level of HDLA decreases with severity which was also statistically significant. (p<0.05)

DISCUSSION:

Historically, most of the smoked tobacco in India has been in the form of cigarettes with tobacco wrapped inside a Tendu leaf. Tobacco smoking, raised blood pressure, elevated blood cholesterol, insufficient physical activity, overweight and obesity, poor nutrition, drinking at harmful levels and diabetes are major preventable risk factors for CVD. Toxic products from cigarette smoke, nicotine and carbon monoxide, circulate in the bloodstream, interfering with the efficient working of the endothelium, eliciting blood fat abnormalities and impairing glucose regulation. Each effect is implicated in the development of atherosclerotic lesions in the arterial walls. These collections narrow the arteries, gradually impairing blood flow and making the arteries harder, less elastic and more liable to rupture and the process leading to atherosclerosis. In the present study, the results showed that the serum level of total cholesterol, triglyceride, LDL-C, were significantly higher in moderate and severe smoker as compared to non-smokers, thereby revealing a direct dose response relationship. Previous studies have reported the same findings that smokers have a higher risk lipid profile than non-smokers7-9. On the contrary, a study by Dirican et al. reported that there was no significant difference in lipid profile among smokers and non-smokers.¹⁰. 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.^{11,12}

CONCLUSION- It was concluded from the present study that the dose response relationship between cigarettes smoked and increase in the atherogenic parameters and decline in anti-atherogenic parameters.

CONFLICT OF INTEREST-None declared SOURCE OF FUNDING-None

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