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RESEARCH ARTICLE

Study of serum lipid profile in the smokers

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ABSTRACT

Background: Smoking of cigarettes has been found to alter lipoprotein levels. Smoking raises the blood total cholesterol (TC) content, triglycerides (TG), low-density lipoprotein (LDL), and very LDL (VLDL) and reduces the level of good cholesterol, i.e., high-density lipoprotein (HDL). **Aim and Objective:** The aim of this study was to find out the association between lipid profile and smoking among the patients. **Materials and Methods:** A hospital-based cross-sectional study at Outpatient Department, Gandhi Medical College, Hyderabad, for 6 months from January 2019 to July 2019. The study was performed among 100 people who were being treated with various illnesses at the hospital. There were 50 non-smokers and 50 smokers in this study. Based on smoking frequency, the smokers were further split in three classes. Serum lipid profile was evaluated in all the patients. **Results:** The mean TC was found to be 160.29 ± 27.89 in the non-smokers compared to 192.96 ± 32.37 in the smokers. The mean TG in non-smokers was found to be comparatively lesser at 105.61 ± 25.82 than smoker group with 165.29 ± 28.95. The mean HDL was found to be less in smokers at 44.62 ± 9.98 compared to non-smokers, where the mean was 48.91 ± 8.70. The mean LDL was comparatively lower in non-smokers with a mean of 81.20 ± 15.93 when compared to smokers with a mean of 104.09 ± 19.54. The VLDL was lower in the non-smokers with a mean of 20.96 ± 6.52 when compared to smokers 29.23 ± 9.20. **Conclusions:** A strong correlation between smoking and increased serum lipids is clearly established in this research. The risk of increasing serum cholesterol in heavy smokers by raising LDL cholesterol and decreasing the amount of HDL cholesterol is significant because this effect is correlated with a coronary heart disease.

KEY WORDS: Cigarette Smoking; Triglycerides; Low-density Lipoprotein; Very High-density Lipoprotein; High-density Lipoprotein; Cholesterol

INTRODUCTION

Smoking is now growing rapidly across the developing world and is one of the greatest threats to current and future health and accounts for an estimated global mortality of 1.17 million citizens.[1] Smoking is the world's second most common cause of death. Almost 20% of all deaths from heart disease can be attributed to smoking.[2] It is a major risk factor for atherosclerosis, peripheral vascular disease, stroke, and cancer.[3-5] This is also closely linked to the stomach ulcer, periodontal, and metabolic diseases. Smoking cigarettes lead to a significant number of dangerous compounds and metabolites derived from tobacco consumption. The substances can be electrophilic and lead to oxidation by the initiation and formation of lipid peroxidation chains in membranes with biomolecules. Smoking of cigarettes has been found to alter lipoprotein levels. Smoking raises the blood total cholesterol (TC) content, triglycerides (TG), low-density lipoprotein (LDL), and very LDL (VLDL) and reduces the level of good cholesterol, i.e. high-density lipoprotein (HDL).[6-10] The main risk factor for the common occurrence

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of atherosclerosis is said to be lipoprotein abnormalities in plasma.[11,12] During a number of studies, LDL and VLDL were shown to be atherographic and HDL was a protective factor. HDL is able to move cholesterol from peripheral to the liver.

The purpose of this research was to determine the effect of TC, HDL, and LDL rates in the smokers

MATERIALS AND METHODS

Place of Study

This study was conducted at the Outpatient Department, Gandhi Medical College.

Type of Study

This was a hospital-based cross-sectional study.

Sample Collection

The sample size was 50 non-smokers and 50 smokers.

Sampling Methods

This was a consecutive sampling method.

Inclusion Criteria

Fifty non-smokers and 50 smokers were included in the study.

Exclusion Criteria

Obese, people on diet restriction, people taking drugs known to alter lipid profile (beta-blockers, thiazides, statins, etc.), alcoholics, diabetes mellitus, hypothyroidism, renal failure, hypertension and coronary artery disease (CAD),and history of dyslipidemia were excluded from the study.

Statistical Analysis

Data were presented in the form of statistical tables and charts. SPSS software version 20 was used for statistical analysis.

Ethical Approval

Approval was taken from the Institutional Ethics Committee before commencement of the study.

Venous blood samples for examination were obtained after full fasting for at least 12 h. On the day before sampling, the patients were advised to have a healthy, fat-free diet. The serum was separated within 2 h of collection to prevent artifactual changes in concentration of HDL. The sample was analyzed. The lipid and lipoprotein assay were done.

RESULTS

The majority of the smokers belonged to the age group of 31–40 years constituting 58%, 32% belonging to the age group of 21–30 years, and the least belonging to the age group of 41–50 years amounting to 4% [Table 1]. The mean TC was found to be 160.29 ± 27.89 in the non-smokers compared to 192.96 ± 32.37 in the smokers. The mean TGs in non-smokers were found to be comparatively lesser at 105.61 ± 25.82 than smoker group with 165.29 ± 28.95. The mean HDL was found to be less in smokers at 44.62 ± 9.98 compared to non-smokers, where the mean was 48.91 ± 8.70. The mean LDL was comparatively lower in non-smokers with a mean of 81.20 ± 15.93 when compared to smokers with a mean of 104.09 ± 19.54. The VLDL was lower in the non-smokers with a mean of 20.96 ± 6.52 when compared to smokers 29.23 ± 9.20 [Table 2].

DISCUSSION

Cigarette smoking likely occurs later in Indian life in comparison to Western society; the third decade of life has the highest number of smokers. In comparison to non-smokers, smokers are at greater risk of CAD. The different reasons given for the correlation include changes in blood coagulation, fibrinolysis decreased, arterial integrity compromised, and lipoprotein profile alterations. Some people are smoking addicted, but some people quit. There are others who use other substances such as cannabis. Beedi smoking is predominantly in India among the laborers. Filtered cigarettes are mostly used by elite people.

Since the study comprised of small group and the study population was entirely male, the mean TC was found to be lesser at 160.29 ± 27.89 in the non-smokers compared to 192.96 ± 32.37 in the smokers and *P* value obtained was

Table 1: Distribution of the study group according to the age

Age in years	Number of non-smokers	Number of smokers
21–30	23	20
31–40	26	29
41–50	1	2

Table 2: The mean lipid profile across various parameters compared in the smokers versus non–smokers

Lipid profile	Non-smokers (Mean±SD)	Smokers (Mean±SD)	*P* value
TC (mg/dL)	160.29±27.89	192.96±32.37	<0.001
TG (mg/dL)	105.61±25.82	165.29±28.95	<0.001
HDL (mg/dL)	48.91±8.70	44.62±9.98	=0.002
LDL (mg/dL)	81.20±15.93	104.09±19.54	<0.001
VLDL (mg/dL)	20.96±6.52	29.23±9.20	<0.001

VLDL: Very low-density lipoprotein, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglyceride, TC: Total cholesterol

<0.001. The mean TG in non-smokers was found to be comparatively lesser at 105.61 ± 25.82 than smoker group with 165.29 ± 28.95 and *P* value obtained was <0.001. The mean HDL was found to be less in smokers at 44.62 ± 9.98 in comparison to non-smokers, where the mean was 48.91 ± 8.70 and *P* value obtained was 0.002. The mean LDL was comparatively lower in non-smokers with mean of 81.20 ± 15.93 when compared to smokers with a mean of 104.09 ± 19.54 and *P* value obtained was <0.001. The VLDL was lower in the non-smokers with a mean of 20.96 ± 6.52 when compared to smokers 29.23 ± 9.20 and *P* value obtained was <0.001.

Our results are similar to the other studies done by Sinha *et al.*[13] (1994) showed a significant increase in serum TG levels. In their study on lipid profile variation in smokers by Tilwani *et al*.,[14] Rastogi *et al*.[15] also had come to the same conclusion.

The findings of our study suggest that there is a significant difference in the lipid profile of smokers when compared to non-smokers. The smokers have high cholesterol, VLDL, LDL, and TGs and lower HDL which mean reduced productivity when compared to smokers which is an alarming sign.

CONCLUSIONS

In all age groups with smoking history, the increase for TC, TG, and LDLs has been reported, while HDLs have a reverse relation. Increased levels of TC, TG, and LDL and decreased levels of HDL were found to be significant among those with ischemic heart disease.

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