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Evaluation of Serum Lipid Profile, Homocysteine and C- Reactive Protein (CRP)In Smokers and Non-Smokers

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KEYWORDS

Smoking, Total Cholesterol, Triacylglycerol, VLDL-C, LDL-C, HDL-C, Homocysteine and C-reactive Protein (CRP).

ABSTRACT:

Background: In comparison to non-smokers, cigarette smoking is regarded as a high risk factor that might result in tissue damage and complaints, including atherosclerosis, cerebral vascular disease, and cardiovascular disease. Smoking intensity and duration change the levels of triglycerides (TG), VLDL-C, LDL-C, and total cholesterol (TC) in the blood. These are elevated, lowering HDL-C levels while raising serum homocysteine and CRP levels.Aim and Objectives:The aim of the study is to investigate and compare the level of serum lipid profile, Homocysteine and CRP level, in smokers and non-smokers.Materials and Methods:The study was to examine the levels of CRP, homocysteine, and serum lipid profile in smokers and non-smokers. For this study, a total of 100 subjects were enrolled and split into two groups: Group I was the Control group (n = 50, non-smokers) and Group II was the Case group (n = 50, smokers). The study was carried out at the KarpagaVinayaga Institute of Medical Science and Research Centre, Madhuranthagam, Chengalpattu, in the departments of clinical biochemistry and general medicine.Results:According to the current study, smokers' serum total cholesterol was substantially higher (175.9 ± 51.04) than nonsmokers' (155.2 ± 39.40). While the LDL level was considerably elevated at 107.9 ± 41.44 in smokers and 91.16 ± 33.48 in nonsmokers, the VLDL level was 29.50 ± 10.14 in smokers and 19.68 ± 7.84 in nonsmokers. Smokers' serum homocysteine mean and SD were 13.87±4.29, while nonsmokers' were 11.39± 3.14. After measuring the CRP level, the mean value rose to 7.823 ± 3.36 for smokers and 5.919 ± 2.25 for nonsmokers. In comparison to the control group, all measures are

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statistically significant.Conclusion:In order to change the serum lipid profile and raise the levels of homocysteine and CRP, smoking intensity and duration increase.

1.INTRODUCTION:

The habit of cigarette smoking is considered as high risk factor may leading to atherosclerosis, cerebral vascular disease, and cardiovascular disease as compared to nonsmokers. Smoking lowers the concentrations of antiatherogenic HDL cholesterol while raising serum levels of total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol. (1) Additionally, smoking raises serum homocysteine levels, which results in hyperhomocysteinemia. In relation to coronary artery disease (CAD), it has been considered an independent risk factor.Elevated blood homocysteine levels can harm platelet aggregation, causing inflammation and plaque to form. These factors can ultimately impede blood flow to the heart. The CRP level is consequently raised by this high homocysteine level. The liver's ability to produce positive acute phase proteins like CRP is directly impacted by smoking. This could lead to chronic inflammatory reactions, which have been linked to disease and destruction of tissue. (2) A higher incidence of certain chronic illnesses, such as heart disease, lung disease, colon cancer, etc., has been associated with elevated CRP. (3,4)Nicotine-containing cigarettes stimulate the release of adrenal hormones, such as catecholamines, which leads to increased lipolysis. This, in turn, raises plasma free fatty acids (FFA) and activates adenyl cyclase in adipose tissues, which in turn causes the bloodstream to secrete more hepatic FFAs and hepatic triglycerides, as well as VLDL cholesterol. (5,6,7) Additionally, smoking tends to reduce levels of B-complex vitamins, including folate, vitamin B6, and vitamin B12 [8,9,10], all of which have an impact on the metabolism of homocysteine.The B-complex vitamins perform as coenzymes for the enzymes that regulate the metabolism of homocysteine [11–16]. elevated plasma homocysteine levels, which lower HDL cholesterol and oxidatively modify LDL cholesterol (17–19). According to several investigations, homocysteine reduced the synthesis of HDL cholesterol and hindered the expression of the Apo A-I protein (20, 21). Thus, the purpose of this study was to determine the relationship between smoking and the levels of CRP, lipid profile, and serum homocysteine in our local community.

2.MATERIALS AND METHODS

The study was designed to compare values of serum lipid profile, homocysteine, and CRP in smokers vs. non-smokers. A total number of 100 subjects were included for this study and divided into two groups: Group I as Control (n=50, non-smokers) and Group II as Cases (n=50, smokers). The study was conducted in the Department of General Medicine and Clinical Biochemistry at KarpagaVinayaga Institute of Medical Science and Research Centre, Madhuranthagam, Chengalpattu.

These groups were evaluated for the following tests:

1. Serum Lipid Profiles (Total Cholesterol Triacylglycerol, VLDL-C, LDL-C, HDL-C)

2. Serum homocysteine

3. Serum CRP levels

2.1 Inclusion Criteria:

1. Patients with a smoking history of a duration of 1 year and above and of 18-60 years of age.

2. Only male subjects are included in this study(female subjects not available).

3. Nonsmokers (healthy volunteers, Age 18-60 years).

2.2 Exclusion Criteria:

1. Patients with pre-respiratory diseases (asthma, lung diseases, bronchitis).

2. Known diabetic and hypertension patients.

3.Sample collection

The venous blood samples were collected after 12 hours of an overnight fast into plain tubes. Serum was obtained by centrifugation, and samples were immediately separated into aliquots and stored at -20°C until analyzed. Total cholesterol, triglyceride, VLDL-cholesterol, LDL-cholesterol, HDL-cholesterol,

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homocysteine, and CRP levels were analyzed on a fully autoanalyzer of Vitros 5600.

3.1 Statistical Analysis:

GraphPad Prism version 9.00, statistical software, was used to analyze the data obtained. An unpaired t-test was used to test the significance of the means.

4.Results:

Throughout a six-month period, 100 serum samples were analyzed Out of these, 50 were chosen as Cases and 50 as Controls.

We are tested and compared the Lipid Profile, Homocysteine and CRP in both groups.

Table 1: A comparison of Serum Lipid profile between Smokers and Non-smokers.

Parameters	**Group – I Non Smokers (mean ± SD)**	**Group – II Smokers (mean ± SD)**	**t- test**
Total Cholesterol(mg/dL)	155.2 ± 39.40	175.9 ± 51.04	P<0. 0288
Triacylglycerol (mg/dL)	98.32 ± 39.55	148.2 ± 51.00	P<0.0001
VLDL- C (mg/dL)	19.68 ± 7.84	29.50 ± 10.14	P<0.0001
LDL- C (mg/dL)	91.16 ± 33.48	107.9 ± 41.44	p<0.0317
HDL -C (mg/dL)	43.10 ± 8.89	35.82 ± 7.79	P<0.0001
T.Chol/HDL-C Ratio	3.67 ± 0.94	4.96 ±1.27	P<0.0001
Non- HDL-C (mg/dL)	112.6 ± 38.14	140.1 ± 47.40	P<0.0023

The current study, smokers' serum total cholesterol was substantially higher (175.9 ± 51.04) than nonsmokers' (155.2 ± 39.40). The mean TGL value was found to be 148.2 ± 51.00 in smokers and 98.32 ± 39.55 in nonsmokers. While the LDL level was considerably elevated at 107.9 ± 41.44 in smokers and 91.16 ± 33.48 in nonsmokers, the VLDL level was 29.50 ± 10.14 in smokers and 19.68 ± 7.84 in nonsmokers. Smokers' mean HDL-C levels were determined to be 35.82 ± 7.79, while nonsmokers' mean values were 43.10 ± 8.89 points. According to our study, smokers had higher levels of the total cholesterol/HDL-C ratio and non-HDL-C (mg/dL), which is statistically significant when compared to nonsmokers (Table 1).

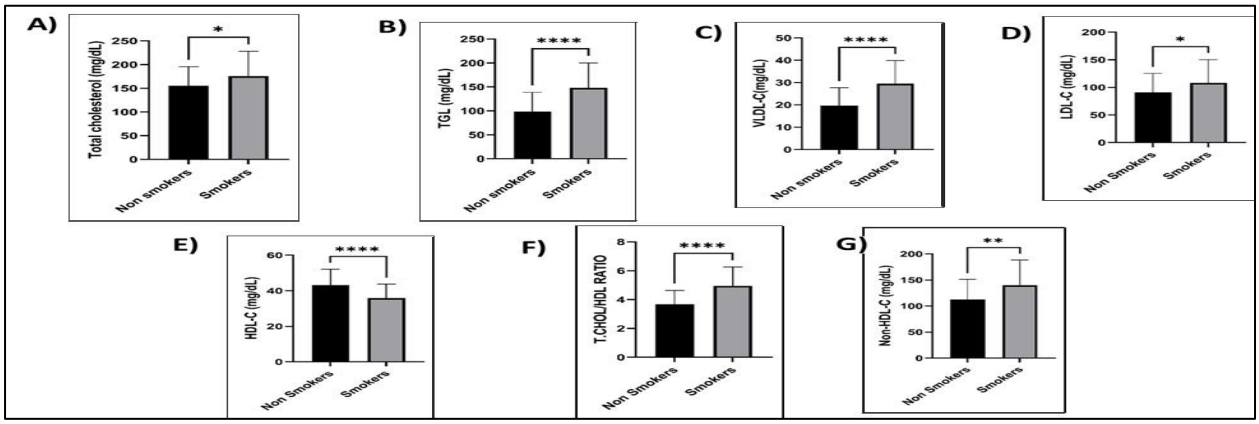

Figure: 1. Comparison of lipid profile parameters between non-smokers and smokers. Bar graphs show mean ± SD values for (A) total cholesterol, (B) triglycerides (TGL), (C) very low-density lipoprotein

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cholesterol (VLDL-C), (D) low-density lipoprotein cholesterol (LDL-C), (E) high-density lipoprotein cholesterol (HDL-C), (F) total cholesterol to HDL-C ratio (T.CHOL/HDL ratio), and (G) non–HDL cholesterol in non-smokers and smokers**.**Statistical significance is indicated as *p < 0.05, **p < 0.01, and ****p < 0.0001.

Table 1: A comparison of Serum homocysteine level between Smokers and Non-smokers.

Parameters	**Group – I Non Smokers (mean ± SD)**	**Group – II Smokers (mean ± SD)**	**t- test**
Homocysteine (µmol/L)	11.39 ± 3.14	13.87 ± 4.29	P<0.0017

The mean and SD of Homocysteine in smokers was found to be 13.87±4.29 and 11.39± 3.14 in nonsmokers. Which is statistically significant as compared to nonsmokers (table 2).

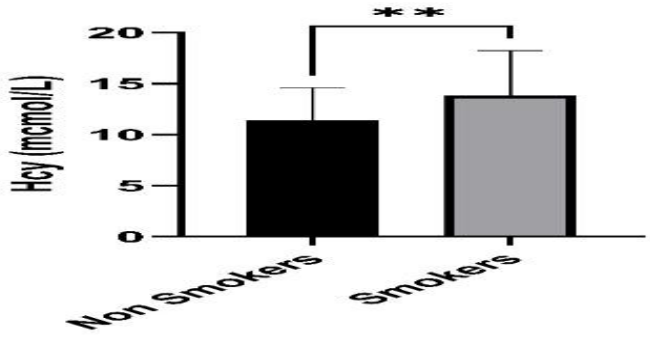

Figure: 2. Graphical comparison of Homocysteine parameters between non-smokers and smokers.Bar graphs show mean ± SD values forHomocysteine. Statistical significance is indicated as *p < 0.05.

Table 1: A comparison of Serum CRPlevel between Smokers and Non-smokers.

Parameters	**Group – I Non Smokers (mean ± SD)**	**Group – II Smokers (mean ± SD)**	**t- test**
CRP (mg/L)	6.243± 2.093	7.931± 3.344	P<0.0032

The mean level of CRP was found to be7.931 ± 3.344in smokers and 6.243 ± 2.093in nonsmokers, which is statistically significant (Table 3).

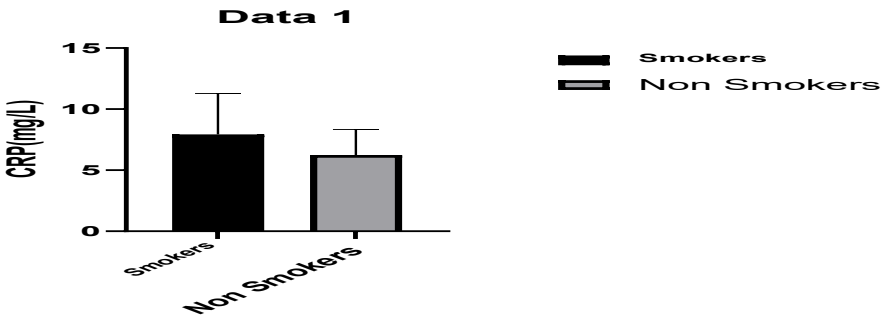

Figure: 3. Graphical comparison of CRP parameters between non-smokers and smokers.Bar graphs show mean ± SD values forCRP. Statistical significance is indicated as *p < 0.05.

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5.Discussion:

Examining changes in lipid profiles, homocysteine, and CRP levels in male smokers and non-smokers was the study's goal. The results of this study show that smokers' plasma had lower levels of serum HDL-C and higher levels of TC, TGL, VLDL-C, LDL-C, Hcy, and CRP when compared to controls. The majority of research demonstrates that smokers had a worse lipid profile than control groups. According to a recent study report, the mean ± SD of the blood total cholesterol was 206.76±74.50 for Bilal Shakoor et al. and 194.1±18.3 for Chandrashekar V et al. According to the current study, smokers' serum total cholesterol was substantially higher (175.9 ± 51.04) than nonsmokers' (155.2 ± 39.40).Additionally, smoking raised serum TGL levels, which is more significant than control. The previous study indicated that the mean TGL value was 148.2 ± 51.00 for smokers and 98.32 ± 39.55 for nonsmokers. Srinivas S.P. et al. and Mamatha B. Patil et al. According to Singh D.P. et al. and Salman Shafi Siddiqui et al., the LDL level was also significantly elevated, measuring 107.9 ± 41.44 in smokers and 91.16 ± 33.48 in nonsmokers, while the VLDL level was 29.50 ± 10.14 in smokers and 19.68 ± 7.84 in nonsmokers. Smokers' mean HDL-C levels were determined to be 35.82 ± 7.79, while nonsmokers' mean values were 43.10 ± 8.89 points.According to our study, smokers had higher levels of the total cholesterol/HDL-C ratio and non-HDL-C (mg/dL), which is statistically significant when compared to nonsmokers. According to the current study, smokers' lipid profiles differ significantly from those of nonsmokers. Smokers had low levels of HDL-C and high levels of TGL, VLDL-C, LDL-C, and total cholesterol. changed lipid profile, with the most obvious alterations being a rise in triglyceride levels and a fall in HDL cholesterol levels. Plaque deposits on artery walls, which narrow the channel and limit blood flow, have been connected to high lipid profile levels that surpass a particular threshold.

Numerous oxidants are found in cigarette smoke, and new research has identified oxygen-derived free radicals that can raise endothelial angiotensin II production while lowering nitric oxide activity, potentially leading to endothelial dysfunction in smokers.Raising Homocysteine levels is one of smoking's most harmful effects, as it can lead to atherosclerosis and endothelial damage. (1,6-8) The current investigation demonstrates statistically significant elevated serum homocysteine levels in smokers (mean and SD 13.87±4.29 and 11.39± 3.14 in nonsmokers) as compared to the control group. Higher levels of homocysteine have been identified as an independent risk factor for vascular disease among smokers, and Jahan S. et al. and Deepa Singh et al. observed a similar impact. The mean CRP level rose to 7.93 ± 3.34in smokers and 6.24 ± 2.09in nonsmokers, according to the measurement. The similar effect of CRP level (mean ± SD 6.99 ± 2.4 in smokers and 4.6 ± 1.5) was found in KM Rajul, Ashish jain. Et al., The current study revealed smoking causes increased the serum lipid profile TC, TGL, VLDL-C and LDL-C, decreased level of HDL-C, smoking also causes hyperhomocysteinemia and CRP level when compared to nonsmokers.

6.Conclusion:

According to the study's findings, smoking raises blood levels of serum total cholesterol, TGL, VLDL-C, LDL-C, Homocysteine and CRP while lowering HDL-C. Thus, it can be said based on the present study that smoking affects and deranges the lipid profile, hyperhomocysteinemia and elevated level of CRP. These finding thus suggest the mechanism through which smoking promote cardiovascular disease and as well as atherosclerosis. Counseling should be given to smokers to help them quit, maintain healthy eating habits, and engage in regular exercise.

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Conflict of interest:

The authors declare that there is no conflict of interest.

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