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LIPID PROFILE STATUS AND CRP LEVELS IN MALE SMOKERS AND NON SMOKERS OF SOUTH WEST REGION OF PUNJAB.

Medical Biochemistry

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ABSTRACT

Introduction: Lipids are involved in almost every aspect of biological life. Serving as hormones or hormone precursors, aiding digestion, supplying energy, storing function, and metabolic fuels; operating as functional and structural chemicals in biomembranes; and producing insulation to facilitate nerve conduction or prevent heat loss are just a few of these functions. CRP is a member of the pentraxin family, so named after the Greek word penta (five) (berries). CRP is a short pentraxin protein with 206 amino acid residues.CRP is separated into four non-glycosylated ellipsoidal divisions, one of which is consist of multiple -pleated sheets that are non-covalently connected and arranged symmetrically in a cyclic sequence together around central pore, resulting in a pentameric, discoidal, and flattened structure.

Objectives

1. To study the lipid profile status in male smokers and non smokers.
2. Examine CRP levels in male smokers and nonsmokers.
3. To compare the lipid levels status of male smokers versus non - smokers.
4. To compare CRP levels in male smokers versus non - smokers.

Material & Methods: For Lipid profile and CRP testing, all blood samples were taken in plain vacutainer tubes were sent to Biochemistry laboratory of the AIMSR. The study was carried out from October 2021 to March 2022; a total 100 samples were received in Biochemistry laboratory all the samples were collected from the patient attending IPD and OPD Department of Adesh Institute of Medical Science and Research, Bathinda. All the parameters were done on fully automated Biosystems Analyzer A15. **Results:** Overall the CRP levels in smokers (cases) is 11.74±11.57 and the CRP level in non-smokers is 6.32±3.44, On comparison of CRP with the Lipid parameters (TG, TC, HDL, LDL, VLDL) the CRP gives the positive correlation with all the Lipid parameters except the HDL which gives negative correlation with CRP. **Conclusion:** CRP rates were increased in smokers (64%) than in non-smokers (36%), indicating that people who smoke were more highly susceptible. (This, in turn, is affected by the quantity and duration of smoking) More cigarettes consumed per day for a longer period of time resulted in an increase in CRP. Smokers had higher lipid levels, such as TC, TG, LDL, and VLDL, while their HDL remained lower.

KEYWORDS

Lipid profile, total cholesterol, triglyceride, high density lipoprotein, low density lipoprotein, very low density lipoprotein, CRP

INTRODUCTION

Lipids are involved in almost every aspect of biological life. Serving as hormones or hormone precursors, supplying energy, storing function, and metabolic fuels; operating as functional and structural chemicals in biomembranes; and producing insulation to facilitate nerve conduction or prevent heat loss are just a few of these functions. For every week of smoking, the average smoker loses more than a day of their life. More than one in every three frequent smokers dies as a result of their habit. [1] Since 1950, the harmful effects of smoking on one's health have been documented. [2] Nicotine and carbon monoxide have been linked to the onset of clinical illnesses, aberrant blood cholesterol levels, and the accumulation of atherogenic lipoproteins. Smoking has been linked to higher lipid profile levels in adults, according to research. [3] Smoking cigarette is considered as the risk factor for coronary heart disease (CHD) as compared to non- smokers. The possible reason for this association is may be due to 13 altered blood coagulability, decreased fibrinolysis, impaired integrity of the arterial wall, and changes in the blood lipid and lipoprotein concentrations. [4] One of the poisons found in cigarette smoke is nicotine. It has been discovered that it has an effect on a person's catecholamine and cortisol secretion. Catecholamine and cortisol levels that are too high can affect a person's glucose and lipid metabolism. Dyslipidemia can result from alterations in lipid metabolism, which can be a risk factor for atherosclerosis and ischemic heart disease, resulting in higher morbidity and mortality in smokers. [5] As the female smokers are uncommon in India, this study exclusively includes adult male smokers. In this work, we attempted to investigate and compare lipid profile changes in smokers and non-smokers of various ages. [6] Tillet and Francis identified C-reactive protein (CRP) in the sera of individuals with acute Pneumococcus infection in 1930. Pneumococcus capsular (C)-polysaccharide was being used to name it. Polysaccharides bind to CRP on bacteria, such as Phosphocholine (PCh), and activates C1q, triggering the classical complement cascade of innate immunity. [7] The liver produces C - reactive protein (CRP) in reaction to inflammation. One of the most used hematological tests for measuring non-specific inflammation is the CRP test in human blood. CRP

Levels are rather steady in the absence of an acute period of inflammation. A high baseline Inflammatory status, as assessed by CRP, has been linked to an increased risk of various chronic diseases, including heart disease, lung cancer, and colorectal cancer. [8] Several studies have found a clear link between higher levels of CRP, as well as other inflammatory markers, and cigarette smoking, with the majority exhibiting a dose-response relationship between CRP levels and smoking intensity and/or duration. [9] Smoking cessation has been shown to induce immediate reduction in the Levels of several inflammation markers. With specific reference to CRP, various cross-sectional studies showed that ex-smokers have reduced CRP. [10]

MATERIALS AND METHODS

Material Used

For Lipid Profile and CRP testing, all blood samples taken in plain vacutainer tubes were sent to the Biochemistry laboratory of the Biochemistry Department AIMSR. The investigation lasted six months (October 2021 to March 2022) after receiving clearance from the AIMSR Research Committee and the Ethics Committee of Biomedical and Health Research, Adesh University, Bathinda.

Inclusion And Exclusion Criteria

Inclusion Criteria:

1. Patients with smoking history of duration of 1 year and above and of 18-50 years of age.
2. Only Male subjects are included in this study.

Exclusion Criteria:

1. Patient with pre respiratory diseases (Asthma, lungs diseases, Bronchitis).
2. Known diabetic and hypertension patients.
3. Female subjects are excluded from the study.

Methodologies And Experimental Strategy:

Serum Preparation

Blood samples were obtained in Becton Dickinson's commercially available red capped tubes vacutainers (BD). After that, blood samples were left undisturbed at room temperature for 15-30 minutes to coagulate. For 5 minutes, the tubes were centrifuged at 3000 rpm. After

centrifugation, the sample solution (serum) was transferred to a fresh polypropylene tube with a Pasteur pipette.[11]

Specimen Storage And Handling During Testing.

During testing, the temperature of the specimens was kept between 20 and 25 degrees Celsius. Specimens were kept at 4-8°C for a maximum of 8 days. If the samples required to be preserved for a longer amount of time, they were put in a deep freezer at -20°C.[12]

Procedure:

All the samples were collected from the patient attending Surgery IPD and OPD Department of Adesh Institute of Medical Science and Research, Bathinda. All the parameters were done on fully automated Biosystems Analyzer A15.

RESULTS

During a six-month period, 100 serum samples were tested for Lipid Profile and CRP. Out of these, 50 were chosen as Cases and 50 as Controls.

Table 1 Distribution Of Controls According To Age.

AGE GROUP	NO OF PATIENTS	%
20-30	13	26
31-40	13	26
41-50	24	48
TOTAL	50	100

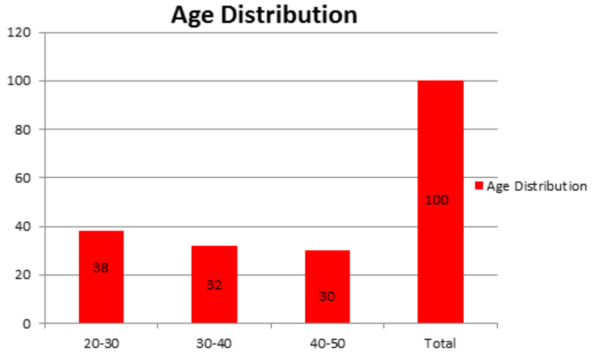

Figure 1 Age Distribution

Table 2 OPD/IPD Wise Distribution Controls.

OPD	39	78%
IPD	11	22%
TOTAL	50	100%

Table 3 OPD/IPD Wise Distribution Cases.

OPD	36	72%
IPD	14	28%
TOTAL	50	100%

Table 4: Showing Comparison Of Vital Parameters

PARAMETERS	MEAN±SD		t-test
	CASES	CONTROL	
TC mg/dl	206.76±74.50	166.28±45.78	P<0.0028
TG mg/dl	291.74±191.68	175.84±63.34	P<0.0001
HDL mg/dl	43.64±2.81	47.46±8.40	P<0.0029
LDL mg/dl	102.98±64.99	81.58±42.04	P<0.0483
VLDL mg/dl	58.14±38.33	34.94±12.72	P<0.0001

Table 5: Showing Comparison Of Vital Parameters

PARAMETERS	MEAN±SD		t-test
	CASES	CONTROL	
CRP	11.74±11.57	6.32±3.44	P<0.0028

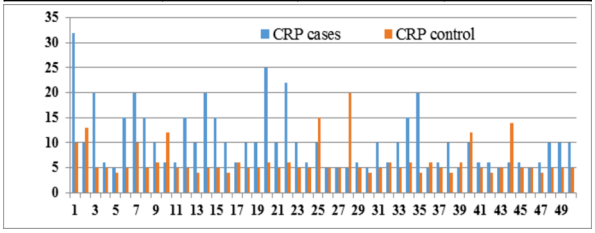

Figure 3: Comparison Between CRP Cases And Control

DISCUSSION

The goal of the study was to look at changes in lipid profiles and CRP levels in male smokers and non-smokers who had been smoking for more than a year. The following are some of the study's various findings:

Age Distribution

Study	Age (years)
Jamal et al [13]	43.48 ± 9.53
Elfadil et al [14]	22.9 ± 3.9
Helmersson et al. [15]	77.3 ± 6.1
Wannamethee et al. [16]	60.06 ± 9.5
Lowe et al. [17]	44.0±10.7
Present study	38.7±11.3

Total Cholesterol (TC)

The overall amount of cholesterol in your blood is known as total cholesterol. Low-density lipoprotein (LDL, or "bad") cholesterol and high-density lipoprotein (HDL, or "good") cholesterol make up your total cholesterol. Cholesterol is a waxy, fat-like natural compound in every cell in your body. In the present study the total cholesterol was analyzed to see the effects of smoking on a normal person. Studies by Sudeep Kumar et al [18] had mean± SD of Total Cholesterol as 284.48±16.25 whereas studies by Elfadil et al [14] and Teshome et al [19] analysed the values of Total Cholesterol as 185 ± 64.0 and 179.56 ± 28.8 respectively. In the current study, the mean± SD for Total Cholesterol came out to be 206.76+-74.50 mg/dl. It was in accordance with a study by Jamal et al [13] and Jain et al [20] who also had similar mean value of Total cholesterol although standard variation was somewhat variable. The value of high levels of Total Cholesterol in the present study is that as Smoking raises LDL, or "bad," cholesterol in the blood while lowering HDL, or "good," cholesterol. As a result, excessive cholesterol levels in the blood can cause plaque to build up in your arteries, narrowing them.

Triglyceride (TG)

In the present study the Triglyceride was analysed to see the effects of smoking on a normal person. Studies by Sudeep Kumar et al [18] had mean± SD of Triglyceride as 183.32±17.88 whereas studies by Elfadil et al [14] and Jamal et al [13] analyzed the values of Total Cholesterol as 141 ± 102 and 164.90 ± 122.51 respectively. In the current study, the mean± SD for Total Cholesterol came out to be 291.74±191.68 mg/dl. It was in accordance with a study by Teshome et al [19] and Jain et al [20] that also had similar mean value of Triglyceride although standard variation was somewhat variable. The value of high levels of Triglyceride in the present study is that as Cigarette smoke elevates LDL, or "bad" cholesterol, as well as triglycerides, a type of blood fat. These contribute to the formation of waxy plaque in your arteries. At the same time, it reduces HDL cholesterol, or "good" cholesterol, which helps to prevent plaque formation.

High Density Lipoprotein (HDL)

Study	High Density Lipoprotein (mg/dl)
Jamal et al [13]	48.27 ± 13.06
Elfadil et al [14]	34.0 ± 8.0
Sudeep Kumar et al [18]	25.16±3.09
Teshome et al [19]	45.76 ± 12.09
Jain et al [20]	41.76 ± 7.09
Present study	43.64±2.81

Low Density Lipoprotein (LDL)

Study	Low Density Lipoprotein (mg/dl)
Jamal et al [13]	131.82 ± 34.83
Elfadil et al [14]	109 ± 52.9
Sudeep Kumar et al [18]	223.12±18.09
Teshome et al [19]	89.92 ± 14.70
Jain et al [20]	100.68±44.69
Present study	102.98±64.99

Very Low Density Lipoprotein (VLDL)

The liver produces very-low-density lipoprotein (VLDL) cholesterol, which is then delivered into the bloodstream to provide a kind of fat to bodily tissues (triglycerides).Cholesterol comes in a variety of forms, each of which is made up of lipoproteins and lipids. Each form of lipoprotein has a different proportion of cholesterol, protein, and triglycerides. Triglycerides make up about half of a VLDL particle.

VLDL cholesterol levels beyond a certain threshold have been linked to the formation of plaque deposits on arterial walls, narrowing the route and restricting blood flow.

C - reactive protein (CRP)

The CRP was measured in order to determine the effects of smoking on a healthy person in this study. Sudeep Kumar et al [18] found that the mean SD of CRP was 3.99±0.21, but Teshome et al [19] and Elfadil et al [14] found CRP values of 4.02 ± 1.04 and 2.09± 1.85, respectively. CRP had a mean SD of 11.74±11.57 mg/dl in the current study. Despite having somewhat different standard variation, Lowe et al. [17] and Wannamethee et al.[16] found similar mean CRP values.

CONCLUSION

- CRP rates were increased in smokers (64%) than in non-smokers (36%), indicating that people who smoke were more highly susceptible. (This, in turn, is affected by the quantity and duration of smoking) More cigarettes consumed per day for a longer period of time resulted in an increase in CRP.
- Smokers had higher lipid levels, such as TC, TG, LDL, and VLDL, while their HDL remained lower.
- The age group with the greatest effect was 40-50 years, followed by 20-40 years, implying that CRP positivity was prevalent in the middle age group.
- CRP levels were elevated in 64 percent of patients with a smoking history and 36 percent of patients without a smoking history in this study. This study also discovered an increase in lipid profile, including TC, TG, LDL, and VLDL. High CRP and lipid levels are related with chronic inflammatory disorders as opposed to acute inflammatory diseases, according to this study, and serum CRP levels can be measured using the Nephelometry method. The patients' histories and test findings indicate a significant frequency of complications in smokers, with heart events, dyslipidemia, pulmonary illness, and depression being the most common clinical symptoms depending on the quantity and duration of smoking.

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