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IMPACT OF CHRONIC CIGARETTE SMOKING ON LIPID PROFILE

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Abstract:
Aim: *This study aims to observe the effects of chronic cigarette smoking on the lipid profile, including triglycerides, total cholesterol, HDL-C, and LDL-C.*
Place and Duration: *In the Department or Medicine Unit II of Nishtar Hospital Multan for six-months duration from October 2019 to March 2020.*
Methods: *A total of 102 (30 non-smokers and 72 smokers) healthy men with similar diets. Socio-economic habits and conditions were selected for the study. Total cholesterol, triglycerides and LDL-C were significantly increased in smokers (group B) compared to non-smokers (group A), while HDL-C levels in smokers were significantly reduced compared to non-smokers.*
Results and Conclusion: *Total cholesterol, triglycerides, and LDL-C were significantly increased in heavy smokers (Group B2) compared to moderate smokers (Group B1), and HDL-C in heavy smokers was significantly reduced compared to moderate smokers.*
Key words: *triglycerides, HDL-C, LDL-C, smokers*

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INTRODUCTION:

Smoking is a major risk factor for cardiovascular disease, cancer, chronic obstructive pulmonary disease, and cerebrovascular disease. Smoking cigarettes causes a variety of physiological responses. Some of them appear to be involved in accelerating atherogenesis or making thrombosis more likely. These responses include lowering plasma HDL cholesterol followed by increasing LDL cholesterol and triglycerides. Therefore, the proposed study was designed to evaluate the effect of chronic cigarette smoking on the lipid profile. Cigarette smoking is one of the most important modifiable risk factors for atherosclerosis and increasing morbidity and mortality of Chronic Heart Diseases (CHD). Although the precise mechanism of tobacco smoke role in the atherosclerotic process remains not fully understood, several chemicals among thousands that exist within tobacco smoke produce harmful and toxic effects on health. Many changes which could promote atherosclerosis in chronic tobacco smokers have been reported by many research workers, whereby these changes include; alternation of lipid profile, increased oxidative LDL-C, decreased nitric oxide (NO) availability, endothelial dysfunction, increased insulin resistance, alternation in fibrinolysis, platelet dysfunction, high blood viscosity, on-going inflammation with increasing inflammatory markers, and more recently free radicals-mediated oxidative stress appear to play an important role in mediation of athero-thrombotic disease in chronic smokers. Cigarette smoking increases plasma catecholamine which induces lipolysis and release of free fatty acid, which will be taken up by the liver. Atherosclerosis has been described as a lipid driven inflammatory disorder of the arterial wall. Low Density Lipoprotein-Cholesterol (LDL-C) and Very Low-Density Lipoprotein-Cholesterol (VLDL-C) are atherogenic and the High-Density Lipoprotein-Cholesterol (HDL-C) is a protective factor against coronary atherosclerosis. Previous research workers have reported that tobacco smoking is associated with increased levels of total cholesterol, triglyceride, LDL–C, VLDL and decreased level of HDL-C. However, other studies were reported with conflicting results. It seemed that cigarette smoking could promote atherosclerosis, in part, by its effect on lipid profile. It is also found that the risk of development of CVD is directly related to the number of cigarettes smoked. The non-HDL-C is a progressing parameter which includes all potentially atherogenic apo B -containing lipoprotein partials including LDL-C, IDL-C and VLDL.

METHODOLOGY:

The study was conducted at the Department or Medicine Unit II of Nishtar Hospital Multan for six-months duration from October 2019 to March 2020. In total, 102 apparently healthy men ranging in age from 25 to 35 were selected. They were divided into different groups: Group A = non-smokers (n = 30) Group B = non-stop smokers for> 5 years (n = 72) Group B1 = moderate smoker, consuming <20 cigarettes per day (n = 36). Group B2 = heavy smokers who consumed> 20 cigarettes per day (n = 36). People who took multivitamin supplements or suffered from any acute or chronic illness were excluded from the study. A fasting blood sample (5 ml) was collected from all selected patients. Total cholesterol, triglycerides and serum HDL-C were determined by the enzymatic-colorimetric method, and LDL-C was calculated according to the Fried-wald formula.

RESULTS:

A comparison of total cholesterol, triglycerides, HDL-c, and LDL-c in nonsmokers (Group A), smokers (Group B), moderate smokers (Group B1), and heavy smokers (Group B2) is given in Tables 1 and 2.

Table 1: Comparison of Lipid Profile in Non-Smokers (Group A) and Smokers (Group B)

Parameters (mg/dl)	Non-smokers (Group A) (n=30)	Smokers (Group B) (n=72)	Level of Significance (A vs B)
Total cholesterol	173.27±1.70	190.82±1.75	HS
Triglycerides	115.17±2.99	161.82±1.95	HS
HDL-C	41.23±0.95	33.25±0.28	HS
LDL-C	109.00±2.13	125.21±1.80	HS

Table 2: Comparison of S-Cholesterol, Triglycerides, HDL-C and LDL-C in Non-Smokers (Group A), Moderate Smokers (Group B1) and Heavy Smokers (Group B2)

Parameters (mg/dl)	Non-smokers (Group A) (n=30)	Moderate Smokers (Group B1) (n=36)	Heavy smokers (Group B2) (n = 36)	Level of Significance		
				A Vs B1	A Vs B2	B1 Vs B2
Total cholesterol	173.27 ± 1.70	184.36 ± 2.17	197.28 ± 2.32	HS	HS	HS
Triglycerides	115.17 ± 2.99	151.42 ± 2.21	172.22 ± 2.11	HS	HS	HS
HDL-C	41.23 ± 0.95	34.44 ± 0.4	32.06 ± 0.26	HS	HS	S
LDL-C	109.00 ± 2.13	119.63 ± 2.37	130.80 ± 2.40	HS	HS	HS

DISCUSSION:

Smoking cigarettes adversely affects the lipid profile. Smoking is said to work in two ways. First, by altering the serum lipid concentration, and second, by chemically modifying the lipids. Total cholesterol in smokers was found to be higher than in non-smokers, and the difference was statistically highly significant. Dose-related increases have also been observed among smokers. Similar results were presented by Craig et al. (1989) and Hallfrish et al. (1994). It was found that the serum triglyceride concentration was increased in smokers compared to non-smokers, and the difference was statistically highly significant. These findings are consistent with those of Willett et al. (1983), Craig et al. (1989). Facchini et al. (1992) and Marangon et al. (1998). HDL-C has been found to be significantly lower in smokers than in non-smokers. The same results were presented by Mjos (1988), Craig et al. (1989), Facchini et al. (1992) and Hallfrisch et al. (1994). HDL-c provides protection against atherosclerosis by competing with LDL-c, thereby mobilizing cholesterol away from the atherosclerotic lesion. Serum LDL-C concentration was significantly increased in smokers compared to non-smokers, and the difference was statistically highly significant. Similar results were presented by Craig et al. (1989) Hughes et al. (1993) and Sniderman et al. (1997). The most common class of atherogenic lipoproteins in human plasma are low-density lipoproteins (LDL), which transport cholesterol from the liver to the tissues. We know that nicotine stimulates the release of adrenaline, which in turn increases the concentration of free fatty acids (FFA) in the plasma through increased lipolysis and the mobilization of FFA from adipose tissue. Free fatty acids stimulate hepatic secretion of VLDL, triglycerides, and also cholesterol. Inhibition of lipoprotein lipase activity by smoking is believed to increase triglyceride and LDL-C levels.

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