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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 Cardiology and Medicine Unit-II of Services Hospital Lahore for six-months duration from March 2020 to August 2020.*

Methods: *A total of 102 (30 non-smokers and 72 smokers) healthy men with similar diets Habits and socio-economic conditions were selected for the study.*

Results and Conclusion: *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. 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 triglycerides6. Therefore, the proposed study was designed to evaluate the effect of chronic cigarette smoking on the lipid profile.

METHODOLOGY:

The study was conducted at the Cardiology and Medicine Unit-II of Services Hospital Lahore for six-months duration from March 2020 to August 2020. In total, 102 apparently healthy men aged 25-35 were selected. They are 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 a day (n = 36).

Group B2 = heavy smokers who consumed> 20 cigarettes per day (n = 36)

People who took a multivitamin supplement or suffered from any acute or chronic medical condition 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 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, thus mobilizing cholesterol away from the atherosclerotic lesion. Serum LDL-C levels were significantly elevated in smokers compared to non-smokers, and the difference was statistically highly significant. Similar

results were reported by Craig et al. (1989) 9 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 FFA mobilization from adipose tissue. Free fatty acids stimulate the 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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