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Smoking Disrupts Lipid Profil

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Abstract:
Cigarette smoking is an important and independent risk factor of atherosclerosis, coronary artery disease and peripheral vascular disorders. Apart from active smokers, passive-smokers are also prone to the development of smoking related disorders. Smoking adversely affects the concentration of the plasma lipids and lipoprotein levels. The lipid profile was measured from 100 selected smokers and nonsmokers and the study shows that as the intensity and duration of smoking increases a significant increase in the levels of very low density lipoprotein -cholesterol, low density lipoprotein -cholesterol, triglyceride and total cholesterol are noted in almost all groups of cigarette smokers as compared to nonsmokers. Simultaneously a significant reduction in the level of High density lipoprotein-cholesterol is observed in cigarette smokers as the intensity and duration is increased. These findings add another health enhancing benefit by the cessation of smoking.
Methods: Fifty adult smokers were selected along with thirty normal controls. Lipid profile was studied and estimations of cholesterol, total lipids, triglycerides, HDL, LDL, VLDL and chylomicrons were made.
Results: All these parameters except HDL level were significantly increased in smokers while the HDL level was significantly decreased, showing greater risk of the persons to atherosclerosis and coronary heart disease (CHD). Various ratios like LDL/HDL, VLDL/HDL, TG/HDL and TC/HDL were calculated to find out the risk of atherosclerosis and CHD so that early measures could be adopted to avoid the complicated disease process.

Key words: *Cigarette smoking, Triglyceride, low density lipoprotein cholesterol, very low density*

1. Introduction

Smoking is an escalating health problem, especially in developing countries such as India. Cigarette smoking is a known risk factor for peripheral, coronary and cerebral atherosclerotic vascular diseases. Cigarette smoking leads to the uptake of many hazardous compounds and their metabolites extracted from burning tobacco. These substances may be electrophilic and react with biological molecules, and give rise to oxidative stress through the formation of reactive species or the initiation of lipid peroxidation chain reactions in the membranes [1]. Plasma lipoprotein abnormalities are a major risk factor for the occurrence of atherosclerotic vascular disease [2]. The prevalence of smoking in India varies from about 15% to over 50% among men [3]. However, smoking is less common among women with prevalence of 4% or less [4]. Cigarette smoking has been found to alter the lipoprotein levels [5].
Previously published reports suggest their oxidatively modified low density lipoprotein (LDL) is taken up by macrophages to form foam cells in culture and aggravate the process of atherosclerosis [6]. Also, the effects of elevated lipid levels and changes in lipoprotein among cigarette smokers were demonstrated earlier [7], [8-10]. The effects of cigarette smoking on serum apolipoprotein A1 (Apo A1) and apolipoprotein B (Apo B) in smokers free from other risk factors of atherosclerotic vascular disease and dose response relationship were studied. The correlation of Apo A1 with high density lipoprotein cholesterol (HDL) and Apo B with LDL as coronary risk factors was also examined along with the effects of smoking on HDL/Apo A1 and LDL/Apo B. However, studies on the interaction between smoking and apolipoproteins are scarce. Hence, the objectives of the present study were: (i) to investigate into the lipid profile, including Apo-A1 and Apo-B among smokers, and (ii) to relate lipid profile alteration with smoking pack years.

2. Materials and Methods

2.1. Selection of Subjects

Middle aged healthy males smoking cigarettes over 10 pack years and hailing from Chennai, capital of Tamil Nadu State of Southern India attending Master health checkup program (MHCP) formed the subjects of the study. Another group of non-smoking age

matched male candidates attending MHCP were included as control subjects. The work was carried out after an approval from the Institutional ethical committee and informed consent from each participant according to Helsinki Declaration Guidelines.

2.2. Master Health Checkup Program (MHCP)

State Government of Tamil Nadu has introduced MHCP in Government Medical College hospital and district headquarters, hospitals wherein any interested or referred persons can ask for health check-up. Subjects are instructed to come in fasting for 12 hours. During a checkup, qualified physician takes the complete health history, including health complaints, past, family and social histories, and physician does general and physical examination of body systems. Then, individuals are subjected to laboratory evaluation such as complete blood count, blood sugar, lipid profile, renal and liver function, and urine analysis, including chest X-ray, ultra sonogram of the abdomen and electrocardiogram for a nominal fee. If the individual is found to have any illness, he/she will be referred to respective specialty for management. Counseling and guidance will be provided to those who require life style modification.

2.3. Inclusion and Exclusion Criteria

Since smoking is extremely rare among women in this area due to cultural reasons, women were not included. Individuals if found to have any associated co-morbid illness or taking regular medication, including vitamin/mineral supplements/herbal/native medicines were excluded. Clinical history was elicited to rule out any acute injury or infectious episode and/or anti-microbial therapy over the last six weeks. Patients with the family history of lipid disorders were not included.

2.4. Data Collection

The socio-demographic and clinical data were collected. Dietary history and physical activity were elicited as per Indian Council of Medical Research.

2.5. Categorization of Smokers

Smoking history was elicited in detail and smoking pack year was then calculated by using formula, {(Number of cigarettes smoked per day × Number of years smoked)/20}. In our study, smokers were classified into mild, moderate and heavy based on the number of pack years as 10 to 14, 15 to 19, and 20 and above, respectively.

2.6. Laboratory Aspects

Blood was drawn from the subjects after 12 hours fasting with staple food for two days. Enzymatic method was used to estimate total cholesterol (TC) and triglycerides (TGL) using commercial kits. HDL cholesterol was determined by precipitation of phosphotungstic acid MgCl [11] and LDL cholesterol was then calculated. By kinetic nephelometry,

The data were tabulated and analyzed. Differences between mean values were evaluated by student 't' test. The statistical significance was assessed by using chi-square test [12].

Smoking is one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease 1, 2. Relationship of CHD and smoking was first developed by White et al. 3 and later Doll et al.4.

Incidence of developing CHD is directly related to the number of cigarette smoked5. Sudden death is 2-4 times more in heavy smokers than in nonsmokers Stanler6. It has been suggested that cigarette smoking when it is consumed more than 10/day on regular constitute a major risk factor for CHD7 Some studies of smoking and serum lipids, however, have shown that plasma HDL cholesterol level tend to be lower in smokers than in non-smokers8. Smoking which is recognized as a major risk factor for the development of ischemic heart disease may lead to alter the normal plasma lipoprotein pattern. Earlier Friedman5 has showed that increased cholesterol levels and CHD are observed in smokers. Cook et al.9 has also suggested that apart from other risk factors hypercholesterolemia and cigarette smoking are the major ones for CHD Tobacco smoke contains many constituents, nicotine is one of the main constituents. Nicotine causes an increase in triglyceride, cholesterol and VLDL levels and decrease in HDL levels, Augestin 10, later with Cluette Brown11 also studied that long term consumption of oral nicotine increased LDL cholesterol and decreased HDL cholesterol. It has been described that nicotine increases the circulatory pool of atherogenic LDL via accelerated transfer of lipids from HDL and impaired clearance of LDL from plasma compartment; therefore it increases the deposition of LDL cholesterol in the arterial wall Honjack 12. Serum Lipid Concentrations According to Smoke Exposure

	Patient Group		
Variable	**Nonsmokers (n=75)**	**Passive Smokers (n=28)**	**P**
Total cholesterol, mg/dL	237±5	229±13	NS
LDL-C, mg/dL	171±5	166±13	NS
HDL-C, mg/dL	43.6±1.2	38.7±1.2	.005
Triglyceride, mg/dL	112±7	123±10	NS
Total cholesterol/HDL-C	5.7±0.2	6.0±0.3	NS
LDL-C/HDL-C	4.1±0.2	4.4±0.3	NS

C indicates cholesterol. Values are mean±SEM.

3. Materials and Methods

Fifty non obese smokers who smoked more than ten cigarettes per day regularly were selected and thirty non-smokers non-obese persons were included in the study as controls. Diabetics, hypertensives and those with a history of angina were not included in the study Fasting blood sample was taken, serum was separated and was analyzed for Total lipids, cholesterol, triglycerides, HDL, LDL, VLDL and chylomicrons. Only males were included in the study, as smoke females are almost nonexistent in our area.

4. Results

The results of total lipids in controls and smokers are shown in Table-1 showing mean value, standard deviation (SD) and standard error of the mean.

PARAMETERS	**NONSMOKERS**	**CIGARETTE SMOKERS**		
	GROUP – I	**GROUP –II A**	**GROUP –II B**	**GROUP –II C**
	(n=25)	**(n=25)**	**(n=25)**	**(n=25)**
Total Cholesterol (mg %)	155.28 ± 24.09 P<0.001	196.38 ±18.54 P <0.001	202.78 ± 0.56 P<0.001	202. ± 20.567 P<0.0018
Triglycerides (Mg %)	121.20 ± 32.70	167.78 ± 25.41	P<0.001	171.57± 32.42
HDL-Cholesterol (mg %)	6.90 ± 6.71	438.63 ± 3.06	P<0.001	35.46 ± 3.50
LDL-Cholesterol (mg%) ol	84.08 ± 24.42	124.01 ± 18.12	P<0.001	133.50 ± 21.76

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	Controls-	**Smokers**
Mean	724	969
S.D.	39.34	40.1
S.E.M	7.	5.67

Table 1: TOTAL LIPIDS (mg/dl)
Control vs. Smokers: P < .05

- Serum triglyceride values are shown in Table-2.

	Controls	**Smokers**
Mean	151	204
S.D.	28.86	29.2
S.E.M	5.25	4.13

Table 2: TRIGLYCERIDES (mg/dl)
Control vs. Smokers: P < .05

- Mean serum cholesterol level are shown in Table-3

	Controls	Smokers
Mean	176	234
S.D.	16.10	33.35
S.E.M	2.94	4.72

Table 3: CHOLESTEROL (mg/dl)
Control vs. Smokers: P < .05

- HDL, LDL, VLDL and chylomicrons are useful and important components of lipid transport and utilization in the body. These parameters were also analyzed. The results of serum level of chylomicrons are shown in the following table.

	Controls	Smokers
Mean	49	103
S.D.	14.30	26.64
S.E.M	2.61	3.77

Table 4: CHYLOMICRONS (mg/dl)
Control vs. Smokers: P < .05

- HDL levels are shown in Table-5

	Controls	Smokers
Mean	39.4	32.04
S.D.	5 4.	4
S.E.M	0.91	0.62

Table 5: H.D.L (mg/dl)
Control vs. Smokers: P < .05

- Serum LDL and VLDL values are shown in the tables 6 &7.

	Controls	Smokers
Mean	165	206
S.D.	32.6	31
S.E.M	5.95	4.38

Table 6: L.D.L (mg/dl)
Control vs. Smokers: P < .05

	Controls	Smokers
Mean	97	113
S.D.	15.68	29
S.E.M	2.86	4.10

Table 7: V.L.D.L (mg/dl)
Control vs. Smokers: P < .05

- LDL/HDL ratios and VLDL/HDL ratios were also calculated as shown in Tab. 8 and 9.

	Controls	Smokers
Mean	5.23	10.37
S.D.	0.97	1.48
S.E.M	0.19	0.21

Table 8: LDL/HDL Ratio

	Controls	Smokers
Mean	2.48	3.53
S.D.	0.46	0.504
S.E.M	0.083	0.071

Table 9: VLDL/HDL Ratio

- Discussion

5. Conclusion

The total serum cholesterol, LDL, VLDL and Triglyceride values were higher in smokers as compared to Non-smokers. These values increased with increase in number of Cigarette, smoked. Serum levels of HDL are lower in smokers than the same in non-smokers. Serum HDL levels decrease with increase in number of Cigarette/ bidis smoked. Association of HDL had an inverse relationship with cigarettes/bidis smoked per day. Increase in duration of smoking adversely affects the lipid profile. Bidi smoking has more adverse effects HDL than cigarette smoking, although statistically significant results were not obtained.

It shows that serum anti-atherogenic HDL-C level is significantly low in chronic smokers irrespective of the number of cigarettes smoked. The serum level of total cholesterol, LDL-C and VLDL-C and TG are significantly increased with the severity of smoking. Naisargi Joshi et al. The results show that smoking influences the lipid profile adversely hence causing dyslipidaemia in smokers. Smoking results in an increase in oxidized LDL-cholesterol level, which plays a key role in the development of atherosclerosis, and also raising the cardiovascular disease risk.

Therefore, it could be probably deduced that smoking is very dangerous to health and should be discouraged. It was also concluded from the present study that tobacco smoking is associated with dyslipidaemia (Increase LDL-C and decrease HDL-C levels), which is atherogenic in nature. As tobacco smoking interacts with other risk factors, the tobacco smokers get additional benefit if these factors are diagnosed and managed adequately. These risk profiles may be helpful in developing preventive cardiovascular strategies for adolescents.

It has also been reported that incidence of coronary heart disease is directly related to number of cigarettes smoked10. Sudden death is 2-4 times more often in heavy smokers than in non smokers6 and smoking more than 10 cigarettes on regular basis constitutes a major risk factor for ischaemic heart disease. Those who continue to smoke have twice as many fatal and non fatal events as compared to those who do not smoke13. It has long been established that one of the constituents of tobacco i.e. nicotine has a considerable influence on increasing the lipid levels in blood14. The current study showed significantly higher levels of total lipids in smokers as compared to that of controls and the results are in accordance with study of Friedman10. Increased total lipids are considered to be an important contributory factor for development of atherosclerosis15 Increased cholesterol levels and CHD are observed in cigarette smokers2. In present study statistically significant increase ($P<0.05$) was observed in the serum cholesterol level in smokers as compared to that of control; these results are in agreement with those of Gorden15 . Higher levels of cholesterol are associated with CHD16 Similarly; higher levels of triglycerides were found in smokers as compared to controls. Recent studies have suggested that triglyceride levels are the most important factor leading to CHD17 although in fact triglyceride as a risk factor has been suggested by various research workers18. Chylomicron levels were slightly higher as compared to that of the normal range, but statistically significant levels were observed as compared to that of controls, as the serum chylomicron level is diet dependent and in this study 14 hours fasting samples were collected so much emphasis cannot be laid on this parameter due to the same reason considerable

Less work has been done with this parameter.HDL level showed statistically significant decrease in smokers as compared to controls. These results are in conformity with those of Scrot (1989) who observed low levels of HDL in smokers as the result of threat of development of atherosclerosis and CHD is increased. The direct relationship of smoking towards CHD has been mentioned by MRFIT20, who described that increase in HDL level by 1 mg/dl was associated with decrease in the risk of CHD by 3%. LDL & VLDL levels were also significantly increased in smokers and are in agreement with results of Kesaneimi and Grundy21. LDL/HDL and VLDL/HDL ratios were significantly higher in smokers as compared to that of controls as evidenced by Martin22 who suggested that with increase in these ratios risks of developing CHD also increases proportionately. In addition to these TG/HDL & TC/HDL ratios are useful as a quick summary of disease risk in smokers. These can be easily counted and risk of disease evaluated. TC/HDL ratio is o 21 very high significance as values higher than the accepted dangerous limit of >4.5 require intervention and indicate very high risk of CHD23, 24. TC/HDL ratio estimates the net effect of two way traffic of cholesterol in and out of tissues25. This ratio has been suggested to be the most important predictor of premature development of CHD26. Persons considered at higher risk of CHD can then be immediately identified and properly advised.

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