ORIGINAL ARTICLE

Assessment of LIPID PROFILE in smokers versus non-smokers

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ABSTRACT

BACKGROUND & OBJECTIVES: Smoking is one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease. 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. **PURPOSE:** This study assesses the changes of lipid profiles in smokers and non-smokers and to find out whether smoking is risk factor for atherosclerosis or not. Early intervention in smokers may decrease the chances of atherosclerosis. **MATERIAL & METHODS:** 40 adult non-obese smokers devoid of any major disease were selected along with 40 normal healthy age and weight matched, non-obese non-smokers who served as controls. Subjects in both groups were in the age range of 20 -50 years, having no history of alcohol abuse or diseases like diabetes mellitus, hypertension, hepatic impairment, renal disease obesity, and were neither on drugs like β-blockers, lipid lowering drugs, or thiazide diuretics. An Informed consent was taken from all the subjects and study was carried out in accordance with the world medical association declaration of Helsinki. Fasting venous sample was collected, under aseptic precaution in department of biochemistry at Sumandeep Vidyapeeth, Waghodiya, Vadodara. Lipid profile was studied and estimations of cholesterol, total lipids, triglycerides, HDL, LDL, VLDL and chylomicrons were made. Statistical analysis was done using unpaired 't' test using Graph pad software. **RESULT:** All these parameters except HDL level were significantly increased in smokers while HDL level was significantly decreased, showing greater risk of these 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 complicated disease process. **CONCLUSION:** Individually smoking is major risk factor for alteration of normal lipid profile leading to development of athroscerosis, coronary heart disease and other related morbidity leads to mortality.

key words: Smokers, Atherosclerosis, Lipid Profile, HDL (High Density Lipoprotein)

INTRODUCTION

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 non smokers 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 have 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 increase in triglyceride, cholesterol and VLDL levels and decrease in HDL levels, Augustine 10, later on 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

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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.Cigarette/*bidi* leads to increase in the concentration of serum total cholesterol, triglycerides, LDL-cholesterol, VLDL-cholesterol and fall in the levels of antiatherogenic HDLcholesterol, as reported by various workers5-9 Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further

result in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL- C in the blood stream9,10; (b) Fall in estrogen levels occurs due to smoking which further leads to decreased HDL – cholesterol11.

MATERIAL AND METHODS

40 non obese smokers who smoked more than ten cigarettes per day regularly were selected and 40 non-smokers non obese persons were included in the study as controls.

- 40 healthy, non-obese male smokers in the age group of 20 -50 years who smoked more than 10 cigarettes per day regularly for more than 3 years were recruited for the study after obtaining informed consent (group I).
- 40 healthy non- obese, non-smokers, age and weight matched selected as controls (group II).
- Those excluded from the study were persons abusing alcohol, ex-smokers, diabetes mellitus, hypertension, renal disease, hepatic impairment, endocrine disorders and obesity and on drugs like β-blockers, lipid lowering drugs, and thiazide diuretics.

Fasting blood sample was taken, serum was separated and was analysed for Total lipids, cholesterol, triglycerides, HDL, LDL, VLDL and chylomicrons. An Informed consent was taken from all the subjects and study was carried out in accordance with the world medical association declaration of Helsinki. Statistical analysis was done using unpaired 't' test using Graph pad software. Study was significant if p value is less than or 0.05, and highly significant if p value is <0.005.

RESULTS AND OBSERVATIONS

Table1: Mean Of Various Parameters Of Lipid Profile

LIPID PROFILE	SMOKERS (MEAN± S.D)	CONTROLS (MEAN ± S.D)	P value
TOTAL LIPIDS *(mg/dl)*	978 ± 38.87	698 ± 41.23	< 0.05
TRIGLYCERIDES (mg/dl)	211 ± 26.54	147 ± 24.34	< 0.05
CHOLESTEROL (mg/dl)	240 ± 29.32	171 ± 14.38	< 0.05
CHYLOMICRONS *(mg/dl)*	110 ± 21.67	52 ± 13.24	< 0.05
H.D.L *(mg/dl)*	35.1 ±5.3	42.13 ± 6	< 0.05
L.D.L *(mg/dl)*	210 ± 28	161 ± 29.12	< 0.05
V.L.D.L *(mg/dl)*	117 ± 25	93 ± 18	< 0.05
LDL/HDL Ratio	11.21 ± 1.28	4.13 ± 0.68	< 0.05
VLDL/HDL Ratio	3.14 ± 0.500	2.19 ± 0.41	< 0.05

DISCUSSION

It has 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 ischemic 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 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 on 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. 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 risk of developing CHD also increases proportionately. In addition to these TG/HDL & TC/HDL ratios are useful as quick summary of disease risk in smokers. These can be easily counted and risk of disease evaluated. TC/HDL ratio is of very high significance as values higher than 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.

CONCLUSION

Individually smoking is major risk factor for alteration of normal lipid profile leading to development of athroscerosis, coronary heart disease and other related morbidity leads to mortality.

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