



COMARATIVE STUDY OF LIPID PROFILE ON HEALTHY SMOKERS AND NON SMOKERS

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ABSTRACT

Smoking is considered to cause heart disease, cancer, stroke and also have close relationship with gastric ulcer, periodontal disease, sudden infant death syndrome, and metabolic syndrome. Our aims was to:- (1) Study the effect of smoking on lipid profile of healthy smokers (2) To compare the lipid profile of both smokers and non-smokers (3) To study the effect of severity and duration of smoking on lipid profile. The present study "Comparative study of lipid profile on healthy smoker and non-smokers" was carried out at Department of Physiology, RIMS, RAIPUR, CG, on 100 healthy smokers and non-smokers. 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/bidis 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 inverse relationship with cigarettes/b idis smoked per day. Increase in duration of smoking adversely affects 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. The results shows that smoking influences the lipid profile adversely hence causing dyslipidaemia in smokers. Smoking results in increase in oxidized LDL -cholesterol level which plays a key role in the development of atherosclerosis, and also raising the cardiovascular disease risk. 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.

KEYWORDS : Substance Abuse; College Students; Prevalence

INTRODUCTION

Smoking has significant detrimental effect on various systems of body especially on cardiovascular system. Smoking in different forms is a major risk factor for atherosclerosis and coronary heart disease. Smoking cigarette/bidi leads to increase in concentration of serum total cholesterol, triglyceride, low density lipoprotein and very low density lipoprotein and fall in levels of anti-atherogenic high density lipoprotein as reported by various workers. There is a dose response relationship between number of cigarettes/bidis smoked and cardiovascular morbidity and mortality. It leads to altered physiological factors which include altered coagulation state, damaged vascular wall and alteration in lipid and lipoprotein content. Thus lipid profile is a simple investigation which helps estimate future cardiovascular morbidity and mortality among smokers. The present study was conducted to demonstrate the effect of smoking on lipid profile and thus on cardiovascular system. The aim of study is to make aware smokers, the hazards of smoking and discourage tobacco usage in any form. To study the effect of smoking on lipid profile of healthy smokers. To compare the lipid profile of both smokers and non-smokers. To study the effect of severity and duration of smoking on lipid profile.

MATERIALS AND METHOD:-

The present study was undertaken in the department of Physiology, RIMS, RAIPUR, CG from January 2013 to June 2014. The study was conducted on 50 healthy male smokers and 50 healthy non-smokers selected from staff, volunteers and patients attending the hospital OPD. The smokers were defined as those who had smoked at least once every week for last one year. The non-smokers were defined as those who had never smoked. The smokers and non-smokers were comparable in age, sex and socioeconomic status. The inclusion and exclusion criteria were as follows: Inclusion Criteria: (1) Age: 25-45 years; (2) Control: Who never smoked; (3) Subjects: Who smoked at least once every week for a year or more and are non-symptomatic. Exclusion Criteria: (1) Diabetes and endocrine Disorder: Both itself can lead to dyslipidemia or oxidation of LDL. Diabetes is a known risk factor for coronary heart disease as it

increases vascular reactivity and microvascular disease. (2) Hypertension: It is a known risk factor for coronary heart disease as it promotes atherosclerosis by increasing vascular permeability or endothelial injury that promotes oxidized LDL. (3) Renal Disorder: It leads to altered lipid profile by itself. (4) Coronary Artery Disease; (5) History of Drug intake: β -blockers, Lipid lowering drugs, Steroids; (6) History of Alcohol Intake/Drug abuse A detailed history was taken. Subjects were explained in detail about the study and written informed consent was taken. Prior approval of Institute's Ethical Committee was taken. Blood Sample was collected after overnight fasting under all aseptic precautions and sample was centrifuged at 2000rpm for one minute. Lipid profile estimation which includes serum cholesterol, serum triglyceride, High density lipoprotein, low density lipoprotein and very low density lipoprotein was done on MIURA autoanalyzer. It works on spectrophotometric/colorimetric principle. The statistical analysis was done by computer programs using Microsoft Excel and SIGMASTAT 2.01. All data were expressed in terms of mean \pm SD and differences were considered significant if P-value was 0.05 or low.

RESULT

The present study comprises of comparison of lipid profile of 50 healthy male smokers with 50 healthy male non-smokers. The study was carried out in the period extending from January 2013 to June 2014. All of them belonged to middle or lower socioeconomic status and had similar physical activity. Data was collected in pre-designed validated Proforma and results were tabulated in All differences are statistically significant except difference in HDL value between Mild smokers and non-smokers.

DISCUSSION

The mean serum triglycerides levels in non-smokers and smokers were 115.9 ± 47.67 mg/dl and 173.44 ± 46.87 mg/dl respectively. These findings are similar to those observed by Wynder et al and Rustogi et al. The mean values of s. triglycerides were significantly higher in those subjects smoking 11-20 cigarettes/bidis per day as compared to those smoking 1-10 cigarettes/bidis per day and even

higher in case of those who smoked >20 cigarettes/bidis per day. These findings are similar to those of Rustogi et al. Recent studies have suggested that triglyceride levels are the most important factor leading to CHD. Triglyceride as a risk factor has been suggested by various research workers. The reduced Lipoprotein lipase activity in smokers as observed by Freeman et al. My explain impaired triglyceride metabolism and higher triglyceride levels. The mean serum total cholesterol in non-smokers was 166.08 ± 28.71 mg/dl while it was significantly higher in smokers, i.e., 194.06 ± 37.61 mg/dl. The total cholesterol values in subjects smoking 1-10 cigarettes/bidis per day was 185.61 mg/dl and those smoking 11-20 cigarettes/bidis per day was 192.12 and > 20 cigarettes/bidis per day was 205.5 mg/dl. These findings are in accordance with those of Muscat JE et al., NSNeeki et al. Increased cholesterol levels and CHD are observed in cigarette smokers. Higher level of cholesterol are associated with CHD. Cigarette smoking substantially increases the risk of coronary heart disease and ischaemic stroke 21-24. According to Zamir et al. nicotine causes increase in cholesterol levels. These findings are in accordance with the work of Rustogi and Shrivastva and Austine. It was observed that HDL-cholesterol level was decreased in smokers when compared with non-smokers. The mean HDL-C in non-smokers was 53.58 ± 9.87 and 47.32 ± 6.90 in smokers respectively (p value <0.002). This finding is similar to that of Rosenson²¹ who reported that there is fall in HDL-C level by 3-5 mg/dl in smokers. The fall in oestrogen level that occurs due to smoking further results in decreased HDL cholesterol. These findings are in conformity with the work of Zamir et al. and scrot. Direct relationship of smoking towards CHD has been mentioned by MRFIT, who described that increase in HDL level by 1 mg/dl was associated with decrease in the risk of CHD by 3%. Further, the subjects smoking 1-10 cigarettes/bidis per day had significantly higher HDL-C (48.61 mg/dl) as compared to those who smoked 11-20 cigarettes/bidis per day, 46.43 mg/dl and 46.75 mg/dl in those who smoked > 20 cigarettes/bidis per day. Similar findings have been reported by Brischetto et al. On the other hand, LDL & VLDL levels were also significantly increased in smokers than non-smokers and are in agreement with results of Kesaneimi and Grundy. The mean LDL-C and VLDL-C values in non-smokers were 78.36 ± 21.74 mg/dl and 23.18 ± 9.75 mg/dl respectively. But these values were significantly higher in subjects with severe smoking than that of mild and moderate smokers. These observations are also similar to those of Rustogi et al. It has been described that nicotine contained in cigarette increased the circulatory pool of atherogenic LDL through accelerated transfer of lipids from HDL and impaired clearance of LDL from plasma compartment, and hence LDL cholesterol in the arterial wall increased. The results shows that smoking influences the lipid profile negatively hence causing dyslipidaemia in smokers. Smoking results in increase in oxidized LDL-cholesterol level which plays a key role in the development of atherosclerosis, and also raising the cardiovascular disease risk¹⁸. Therefore, it could be probably deduced that smoking is very dangerous to health and should be discouraged. The values of serum triglycerides, cholesterol and VLDL were higher in those subjects smoking cigarettes/bidis for more than 15 years as compared to those who smoked for 10-15 years, 5-10 years and 1-5 years. A rising trend of mean values was seen with increase in duration of smoking but the differences were not statistically significant. It clearly shows that duration of smoking directly affects lipoprotein value in smokers suggesting increase in cardiovascular risk. The mean values of HDL were lower in smokers who had history of smoking for more than 15 years as compared to 1 to 5 years of smoking. The differences were not statistically significant. A larger sample size could probably give more accurate results. Difference in mean values for cigarette and bidi smokers were not statistically significant which is similar to the observation of Rustogi et al. but the mean value of HDL was lower in bidi smokers than in cigarette smoker. This could be due to higher nicotine, hydrogen cyanide and carbon monoxide contents of bidi. Also, bidi smoke delivers three times amount of nicotine and carbon monoxide and five times the amount of tar compared to cigarette smoke. Probably a larger sample size would help to establish statistically significant effect of bidi smoking on lipid

profile.

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/ bidis 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 inverse relationship with cigarettes/bidis smoked per day. Increase in duration of smoking adversely affects lipid profile. Bidi smoking has more adverse effects on 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. The results show that smoking influences the lipid profile adversely hence causing dyslipidaemia in smokers. Smoking results in 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.

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