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Research Article

LIPID PROFILE, PLASMA FIBRINOGEN, AND PLATELET COUNT AS MARKERS OF CARDIO VASCULAR DISEASE IN SMOKERS DUE TO FREE RADICAL GENERATION

N Kannan[1], R Aravind Kumar[1*], Ramaprabha P[1], Satish Kumar M[1], Ivvala Anand Shaker[2]
[1]Department of Physiology, Melmaruvathur Adhiparasakthi Institute of Medical Sciences, Melmaruvathur, Tamilnadu, India
[2]Department of Biochemistry, Melmaruvathur Adhiparasakthi Institute of Medical Sciences, Melmaruvathur, Tamilnadu, India
*Corresponding Author Email: arvindr84@gmail.com
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ABSTRACT
Cigarette smoking & tobacco chewing are risk factors not only for oral and lung tumours but also for the development of systemic disorders like atherosclerosis, coronary artery disease and peripheral vascular disease. This study was undertaken to evaluate the lipid profile, plasma fibrinogen and platelet count in male smokers, compared with healthy non smokers in rural area of south India, Out of 100 male healthy volunteers, 50 members were healthy smokers and 50 healthy non smokers, subjects were divided in both groups in age around 30 to 45yrs, with no past history of diabetes mellitus, hypertension, hepatic disorders and were neither on anti hypertensive, lipid lowering drugs were included in the study. Lipid profile, plasma fibrinogen and platelet count were analyzed by standard methods. Our results showed mean platelet count for smokers is 2, 86,345per mm^3 and for non-smokers 2, 04,484.6per mm^3. The mean plasma fibrinogen concentration for smokers is 3.48gm/dl and for non smokers is 3.12gm/dl. The platelet count and plasma fibrinogen concentration shows a higher value for smokers when compared to non- smokers. This is statistically significant. The mean total cholesterol level for smokers (186±30.10) mg/dl and non smokers (166.3±24.26) mg/dl and the mean triglyceride level for smokers (175±59.43) mg/dl and non smokers (132.09±+33.80) mg/dl are also statistically significant. The mean HDL level for smokers (40.4±4.13) mg/dl and for non smokers (44.68±4.13) mg/dl, the mean LDL level for smokers (105.8±28.16) mg/dl and non smokers (89.68±16.50) mg/dl and the mean VLDL level for smokers (28.4± 8.16) mg/dl and non smokers (14.3.±3.2) mg/dl indicate that the Lipid profile also is statistically significant between the two groups. We concluded that there is an elevated lipid profile; plasma fibrinogen and platelet count in smokers when compared to non smokers, which shows that smokers have high risk of prevalence of cardiovascular and vessel wall diseases.
Keywords: Plasma fibrinogen, Platelet count, Lipid profile, Cardio vascular disease, Free radicals.

INTRODUCTION

Smokers are easily prone for higher levels of chronic inflammation, due to generation of free radicals. It damages various tissues, there by affecting the function of the internal organs in the body. Smokers have high levels of oxidative stress which promotes atherosclerosis and cardiovascular diseases[1]. Cell repair and damaged cell recycling process are normally done by our body antioxidants. Antioxidant level is markedly decreased in smokers which lead to improper cell damage and repair[2]. Cigarette contains a very high level of carcinogenic agents like benzoic α Pyrennes and major part of cigarette provides tar, carbon monoxide, hydrogen cyanide, free radicals, metals and radioactive compounds[3]. Smoking reduces the pulmonary function by narrowing airways and also increases the risk of stroke and myocardial infarction by blocking the blood flow to the brain and heart respectively[4]. Bhatt *et al*. found that cigarette smoking alters the coagulation state, promotes vessel wall damaged, by altering the lipid content of circulating blood[5]. Burning *et al.* suggested that HDL levels were too low in smokers, which indicate smokers have high risk to develop cardiovascular disease[6]. Factors responsible for coagulation are produced in the liver. Increased inflammatory responses are associated with alteration in fibrinogen content. Impairment of bleeding occurs if the platelet count and plasma fibrinogen levels are too low[7]. The present study was carried out to assess the effect of cigarette smoking on plasma fibrinogen, platelet count and lipid profile in chronic smokers.

MATERIALS AND METHODS

The present study was done in 100 male volunteers divided into two groups. 50 male volunteers were smokers and 50 male volunteers were non smokers and all were aged between 30 to 45yrs. Smokers were selected under the criteria that those who were smoking more than ten cigarettes a day for a period of more than five years. Totally 100 members who were participated in our study underwent detailed medical history, which includes, blood pressure, heart rate, BMI and routine blood investigations with questionnaire of smoking frequencies and intervals between each cigarettes. All the subjects were explained in detail about the duration and purpose of the study. Since the study was only investigative oral informed consent was obtained. Local institutional Ethical committee approval was obtained.

Inclusion criteria

Healthy Subjects Assessed By A General Physician And Certified That They Are Free From Any Symptoms And Disease.

Exclusion criteria

Patients on lipid lowering drugs, chewing tobacco, ex-smokers, obese, alcoholic, hypertension, diabetes mellitus and dyslipidemia were excluded from the study.

Collection of blood sample

Overnight 12hr fasting blood samples were collected. Nine millilitres of venous blood were withdrawn from the antecubital vein under sterile precaution. 5ml of blood sample for estimating lipid profile was collected in plain test tube, 2ml of blood was taken in EDTA bulb for estimating platelet count and another 2ml of blood was mixed in a tube containing sodium citrate for estimation of plasma fibrinogen concentration. Platelet count was analysed by Beckman coulter automatic analyzer. Plasma fibrinogen was analyzed by turbid metric immunoassay. Serum was separated by

centrifugation at 4000rpm for seven minutes and lipid profile assay was done by using fried dewfalls formula.

LDL and VLDL were calculated[8] as LDL (mg %) =
Total cholesterol – (HDL + TG/5), VLDL (mg %) = TG/5.

Statistical Analysis

Statistical analysis was done using SPSS Ver16.0 (Statistical package for social sciences). The mean variables were stated by mean ± SD where frequencies and percentages were applied for qualitative variables. Independent sample "t –test" to observe group mean difference was done.

Table 1: Showing Plasma Fibrinogen and Platelet Count In Smokers and Non Smokers

Variables	Smokers	Non smokers	P value
Plasma fibrinogen(gm/l)	3.48 gm/l	3.12 gm/l	<0.0001*
Platelet count (/mm^3)	286345/mm^3	204484.6/mm^3	<0.0001*

Both Plasma Fibrinogen and Platelet Count in Smokers were high, when compared to Non Smokers. * Significant

Table 2: Average Values of Lipid Profile in Smokers and Non Smokers

Variables	TC	TG	HDL	LDL	VLDL
Smokers mean ±SD mg/dl	186±30.10	175±59.43	40.4±4.13	105.8±28.16	28.4±8.16
Non - smokers mean ±SD mg/dl	166.3±24.26	132.09±33.80	44.68±4.13	89±16.50	14.3±3.2
P value	<0.005*	<0.01**	<0.01**	<0.005*	<0.005*

There was a significant increase in mean level of TG, TC, LDL and VLDL in smokers and significant decrease in HDL was observed in smokers when compared to non smokers. * Significant, ** Highly Significant

RESULTS

The present study was conducted on 100 healthy male volunteers. 50 members were smokers and 50 non smokers between age group of 30-45 yrs depending upon the duration and intensity of smoking. Lipid profile, platelet count, plasma fibrinogen were analyzed

Table 1: Shows the mean value of plasma fibrinogen, platelet count in smokers and non smokers. The mean plasma fibrinogen concentration for smokers is 3.48gm/l and for non smokers 3.12gm/l. The increase in serum plasma fibrinogen level observed in smokers when compared to non smokers was statistically significant p<0.0001. The mean platelet count for smokers is 2, 86, 345 cells/mm^3 and for non smokers 2, 04, 484.6 cells/mm^3. The difference between mean platelet count for smokers and non smokers was statistically significant p< 0.0001.

Table 2: Shows the mean value of lipid profile compared with smokers and non smokers. There was a significant increase in serum lipid profile level except HDL level with p value <0.0001 and decrease in HDL level in smokers when compared to non smokers with p value <0.005.

DISCUSSION

Cigarette smoking is a serious health hazard which is associated with many diseases. Nicotine released from cigarette and high VLDL levels promotes early onset of coronary artery disease by atherosclerotic plague formation in the blood vessels which leads to hypoxia, due to decreased blood supply to cardiac muscles resulting in myocardial infarction[9,12]. Nicotine increases the myocardial oxygen requirement by increasing free fatty acid level which in turn increases the risk of cardio vascular disease[10]. Long term use of tobacco and related products increases the risk of early onset of hypertension and atherosclerosis in adults[11]. In our study there was an increase in plasma fibrinogen and platelet count in smokers when compared to non smokers. In 2004, study conducted by Low AD *et al.* concludes that smokers are more at risk of early development of thrombus which leads to impaired circulation[13-15]. Gs Tell *et al*. suggested that there was increased platelet count in adolescent age group causing endothelial dysfunction and promotes vascular damage[16]. Lipid profile of our study revealed that there was decreased HDL level in smokers when compared to non smokers. Other parameters such as TC, TG, LDL and VLDL were increased in smokers when compared to non smokers. EJ Neufeld *et al*. (1997) studied the lipid level variations in 40 young volunteers (20 smokers & 20 non- smokers) and they found increase in mean level of TGL, TC, LDL and VLDL in smokers and significant decrease in HDL level in smokers when compared to non smokers. In our present study, total cholesterol level of smokers and non smokers were (186±30.10) mg/dl and (166.3±24.26) mg/dl respectively. There is a high risk of developing coronary artery disease, peripheral artery disease and stroke in smokers due to increased Mean Total cholesterol value[17,18]. The Mean Serum Triglyceride Levels were high in smokers (175±59.43) mg/dl when compared to non smokers (132.09±33.80) mg/dl in our study. Similar findings were observed by Y Wind *et al* and rusting *et al* in 1989[19]. The mean HDL in smokers and non smokers was around (40.4±4.13) mg/dl and (44.68±4.13) mg/dl with the p value at <0.01. HDL cholesterol level was increased in smokers. Rosen son *et al* in 1993 reported that there was a fall in HDL level by 3-8mg/dl in smokers. Decreased HDL level were observed as the duration of smoking increases[20]. Increased LDL level was found in smokers (105.8±28.16), when compared to non smokers (89±16.50). Increased LDL level indicates that smokers are at high risk in developing coronary artery disease. Stamper *et al*. in 1991 proved that increase in serum LDL is marker of early onset of developing coronary artery disease[21]. The mean VLDL levels were significantly high in smokers (28.4±8.16) when compared to non smokers (14.3±3.2). JOS.OD *et al*. in 1988 in his study showed that increased level of VLDL cholesterol in smokers[22] can be a marker for vascular diseases.

CONCULSION

The present study gave us a good opportunity to understand the health hazards of smoking. From our study we concluded that the level of plasma fibrinogen and platelet count was high in smokers. Both plasma fibrinogen and platelet s are an early stage marker of coronary and peripheral artery diseases. Considering the lipid profile parameters there was a decreased level of HDL and increased level of, TC, TGL,

VLDL, and LDL were seen. As suggested by many studies. Hence, we conclude that the combined effect of increased coagulation factors along with bad lipid profile can be used as markers to assess the liability of smokers to cardiovascular diseases. Also, this combined effect suggests that this may be due to increased free radical generation seen in smokers compared to non smokers.

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