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Research Article

ESTIMATION OF LIPID PROFILE AND ASSESSMENT OF CARDIOVASCULAR RISK IN SMOKERS BY USING NEW ATHEROGENIC INDICES

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ABSTRACT

Objective: Smoking habit leads to elevated levels of lipid profile thus increasing the cardiovascular disease risk in coronary heart disease. The aim of study is undertaken to evaluate plasma lipid profile, of the male smoker with non-smoker's healthy matched control and assessing the cardiovascular risk by using new atherogenic indices.

Methods: Fasting blood samples were collected form both cases and controls and estimation of lipid profile by using by using autoanalyzer. A detailed physical and anthropometric parameters information was collected form each participant subjects.

Results: Plasma total cholesterol (TC) (221.52±8.34), triglyceride (TG) (274.94±28.70) low density lipoprotein cholesterol (LDL-c) (129.22±7.76), very LDL-c (VLDL-c) (54.98±5.74) and non-high-density lipoprotein cholesterol NonHDL-c (HDL-c) (183.26±7.58) in smokers subjects, which were significantly ($p<0.0001$) higher compared with non-smokers, while HDL-c significantly (38.25±1.34; $p<0.001$) decreased in smokers as compared to non-smokers. Further, atherogenic ratios like, Castelli's risk index (CRI-I)=TC/HDL-c, CRI-II=LDL-c/HDL-c, atherogenic coefficient = (TC–HDL-c)/HDL-c TG/HDL-c ratio, and atherogenic index of plasma =log (TG/HDL-c) were calculated for individual subjects by using lipid profile. All these lipid ratio are significantly ($p<0.0001$) in smoker group.

Conclusion: Our conclusion, these ratio's could be used for identifying individual at higher risk of cardiovascular disease in the clinical practices especially, when the absolute values of lipid profile seem normal or higher and not markedly deranged or in centers with insufficient resources.

Keywords: Cigarette smoking, Lipid profile, Cardiovascular risk, Atherogenic indices.

INTRODUCTION

Cigarette smoking is generally associated with increased risk for causes of cancer, cardiovascular disorders and others associated disorder. According to the survey of Tobacco Control Policy Evaluation Project India (TCP India Project) in September 12, 2013 revealed the results of a study, approximately 275 million tobacco users in India and it is estimated that there will be 1.5 million tobacco-related deaths annually by 2020 (www.itcproject.org). Smoking is considered as a major cardiovascular risk factor [1]. There is a dose response relationship between the number of cigarettes smoked and cardiovascular morbidity and mortality [2]. During the 1990s, research clarified the pathophysiology of the atherosclerotic effects of cigarette smoking and demonstrating that smoking increases the risk for thrombosis [3-5]. A number of research groups are explained; smoking increases the concentration of serum total cholesterol (TC), triglycerides (TGs), low density lipoprotein cholesterol (LDL-c), very LDL-c (VLDL-c) and decreases the levels of anti-atherogenic high-density lipoprotein cholesterol (HDL-c) [6-10]. Plasma lipoprotein abnormalities are said to be the underlying major risk factors and may even be essential for the common occurrence of atherosclerotic vascular diseases [11]. The aim of this study, estimation of plasma levels of lipid profile and evaluating the role and contribution of the new atherogenic indices in determines the risk cardio vascular risk in smokers.

METHODS

The present study was carried out at Dr. Ramesh Cardiac and Multispecialty Hospital, Vijayawada, Andhra Pradesh, India. Study subjects were selected, who visit to the hospital for their general health checkup. The study protocol was approved by the Institutional Ethical Committee, and the study was conducted during the period from 2012 to 2014. A total number of 31 male smokers and 46 male non-smokers were participated in our study. Selection of subjects based upon implementing the certain inclusion criteria such as, no history of diabetes, no history of hypertension, no history of alcohol abuse, and no exiting of other metabolic disorders. Likewise, certain exclusion criteria are history of diabetes, renal disorders, hypertension, liver diseases and use of lipid lower drugs. Persons with five cigarettes smoking or more per day continuously for a year were selected as smokers considered as cases and person had no habit of smoking considered as control subjects. An informed written consent was obtained from all the study subjects who participated in our study. Proper history of each subject was recorded anthropometric parameters like, name, age, address, height, weight and body mass index (BMI). A detailed physical examination of the subjects of both groups was done.

Collection of a blood sample and estimation of lipid profile

Fasting blood samples were collected in the morning between 7 a.m. and 8 a.m. by venepuncture of antecubital vein with all aseptic precautions, using a dry disposable syringe under sterile conditions in a sterile plain vial. Serum was separated by centrifugation at 3000 rpm for 15 minutes. Fresh serum was used for estimation of TC, TG and HDL-c. The tests were carried out in an automated clinical auto analyzer. Further, LDL-c, VLDL-c, and non-HDL-c were calculated by using Friedewald's formula [12]. Plasma lipid abnormality was based on the expert panel of the National Cholesterol Education Programme cut of values [13]. Further, atherogenic ratios like, Castelli's risk index (CRI-I)=TC/HDL-c, CRI-II=LDL-c/HDL-c, atherogenic coefficient (AC)=(TC–HDL-c)/

HDL-c [14-16], TG/HDL-c ratio [17] and atherogenic index of plasma (AIP)=log (TG/HDL-c) [18] were calculated for individual subjects by using lipid profile.

Statistical analysis

The collected data were analyzed by using Graph pad prism version 5. The differences in groups were determined by performing by unpaired t-test, data were expressed either as mean ± standard error mean (SEM). The statistical significance was set at the p value of $p<0.05$; $p<0.001$; and $p<0.0001$ and $p>0.05$; non-significant.

RESULTS AND DISCUSSION

Table 1 shows the mean ± SEM values of age in years, weight in kilograms and BMI of non-smokers and smokers groups. BMI of smokers (26.36±0.65) group showed significantly (<0.009) higher compared with non-smoker (24.25±0.47) group. Obesity and smoking are important causes of morbidity and mortality worldwide [19]. According to the Framingham study, obese individuals, who smoke have a 14 years reduction in life expectancy at the age of 40 years [20]. A large prospective study showed that smoking coupled with obesity contributes substantially to all-cause mortality, with a 3.5 - 5-fold greater risk for severely obese smokers than for normal weight non-smokers [21]. Some other research studies clear that, smoking and obesity are the main causes of preventable morbidity and mortality in developed countries [22]. Our study also supports the previous studies and hence BMI is very important to predict cardio vascular risk and relationship between smoking and obesity.

Table 2 shows the mean ± SEM values of lipid profiles (mg/dl) of non-smokers and smokers groups. In our study, we were observed the elevated levels (Fig. 1) of TC (221.52±8.34), TGs (274.94±28.70,) LDL-c (129.22±7.76), VLDL-c (54.98±5.74) and non-HDL-c (183.26±7.58) in smokers subjects, which were significantly ($p<0.0001$) higher compared with non-smokers, whereas HDL-c significantly (38.25±1.34; $p<0.001$) decreased in smokers as compared to non-smokers. Similar findings were found in other studies done by Alharbi [23], Venkatesan *et al*. [24], Rastogi *et al*., [25], Sinha *et al*., [26] and Krupski [27] in smokers. Smoking causes an immediate constriction of both proximal and distal coronary arteries as well as an increase in coronary vessel tone and hence resistance [28] that leads to induces the release of catecholamines (epinephrine and norepinephrine) [29,30], resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA), which further result in increased secretion of hepatic FFAs and hepatic TGs along with VLDL-c in the blood stream [31] and also associated with an increased baseline heart rate and contractility [32]. These changes contribute to the atherosclerotic potential of cigarette smoke. So, smoking seriously affects lipid metabolism and LDL modification. Some other researchers are explained, smokers have higher levels of serum malondialdehyde [3], it may modify LDL cholesterol to promote uptake by macrophages and decrease cholesterol transport from cell membranes to plasma. Malondialdehyde is an important marker of oxidation, and evidence indicates that smoking may promote lipid peroxidation, which is hypothesized to be one key element in the causal pathway of atherogenesis [33]. Bloomer explained that, cigarette

Table 1: The comparison mean±SEM values of age, weight and BMI of non-smokers with smokers by t-test

Parameter	Non-smokers (n=46)	Smokers (n=31)	t value	p value
Age (in years)	45.32±2.45	42.61±2.45	0.7519	0.4545^{ns}
Wight (Kg)	70.98±1.53	75.62±2.00	1.868	0.0657^{ns}
BMI (kg/m^2)	24.25±0.47	26.36±0.65	2.676	<0.0091

Where $p<0.001$; $p<0.0001$ considered significant and >0.05. ns: Non-significant, SEM: Standard error of mean, BMI: Body mass index

Table 2: The comparison mean±SEM values of lipid profiles of non-smokers with smokers by t-test

Parameter (mg/dl)	Non-smokers (n=46)	Smokers (n=31)	t value	p value
TC	150.0±2.33	221.52±8.34	9.658	<0.0001
TG	106.67±3.84	274.94±28.70	7.026	<0.0001
LDL-c	85.77±2.14	129.22±7.76	6.320	<0.0001
VLDL-c	21.33±0.76	54.98±5.74	7.028	<0.0001
HDL-c	42.91±0.64	38.25±1.34	3.438	<0.001
Non-HDL-c	107.11±2.28	183.26±7.58	11.191	<0.0001

Where $p<0.001$; $p<0.0001$ considered significant and >0.05. NS: Non-significant, SEM: Standard error of mean, TC: Total cholesterol, TG: Triglycerides, LDL-c: Low density lipoprotein cholesterol, VLDL-c: Very low density lipoprotein cholesterol, HDL-c: High density lipoprotein cholesterol

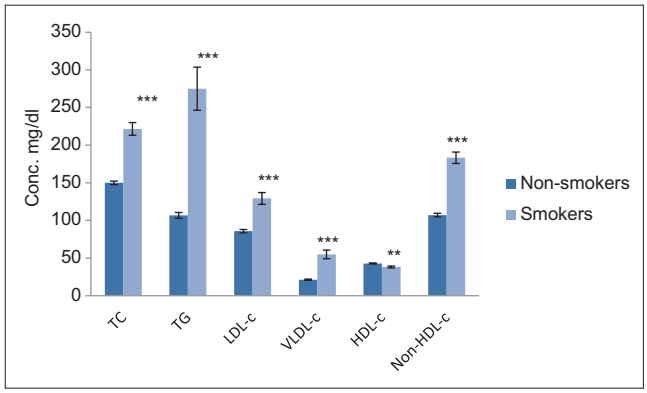

Fig. 1: Comparison of lipid profiles in non-smokers and smokers

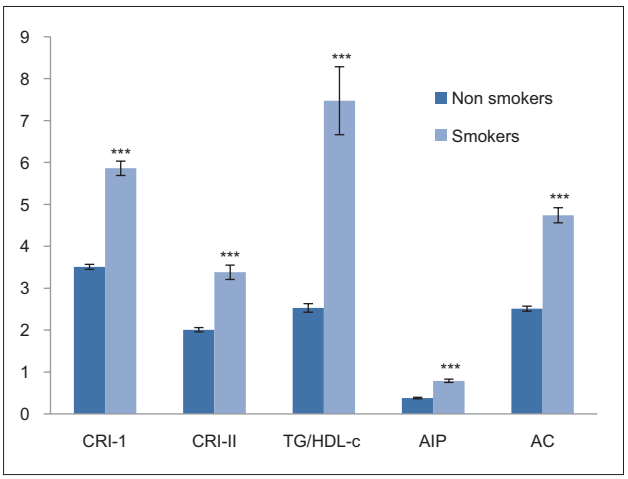

Fig. 2: The comparison of atherogenic indices of non-smokers and smokers

Table 3: The comparison mean±SEM values of atherogenic indices of non-smokers with smokers by t-test

Parameter (mg/dl)	Non-smokers (n=46)	Smokers (n=31)	t value	p value
CRI-1	3.51±0.06	5.86±0.17	14.162	<0.0001
CRI-II	2.01±0.05	3.38±0.17	8.914	<0.0001
TG/HDL-c	2.53±0.10	7.47±0.81	7.284	<0.0001
AIP	0.38±0.02	0.79±0.04	8.460	<0.0001
AC	2.51±0.06	4.74±0.18	13.321	<0.0001

Where $p<0.001$; $p<0.0001$ considered significant and >0.05. NS: Non-significant, SEM: Standard error of mean, CRI: Castelli's risk index, TG: Triglycerides, HDL-c: High density lipoprotein cholesterol, AIP: Atherogenic index of plasma, AC: Atherogenic coefficient

smokers displays decreased antioxidant capacity and increased oxidized lipids compared to non-smokers [34]. Previous studies also explained, oxidatively modified LDL contributes to the pathogenesis of atherosclerosis [35], also effect on lipid metabolism and also effect on morphologic changes in the endothelium of blood vessel [36]. This endothelial dysfunction in the early stages of atherosclerosis is now well recognized [37,38].

Table 3 shows the mean ± SEM values of atherogenic indices of non-smokers and smokers. Cases group showed significantly higher indices (Fig. 2) when compared to control group. All these indices are very useful in predicting the cardio vascular risk and conformed by number of other studies. Now, we are applying these indices for predicting the cardio vascular risk in smokers. The average ratio of TC to HDL-c (CRI-I) of healthy individuals is about 3.5 or lower [14,39] and in case of LDL-c/HDL-c ratio (CRI-II) is 3 or lower [40,41]. Another research study has showed that association of TC/HDL-c with coronary plaques formation [42]. In PROCAM study it was observed, subjects with LDL-c/HDL-c (CRI-II) >5 had 6 times higher rate of coronary events [43]. In our study observed, elevated levels of both CRI-I (5.86±0.17; p<0.0001) and CRI-II (3.38±0.17; p<0.0001), that greater risk in smokers cases.

The atherogenic link between high TGs and HDL-c is due to the higher plasma concentration of TGs, VLDL that generates small dense LDL-c during lipid exchange and lipolysis. These LDL-c particles accumulate in the circulation and form small, dense LDL-c particles, which undergo accelerated catabolism, thus closing the atherogenic circle [44,45]. da Luz *et al*., explained that ratio of TGs to HDL-c was found to be a powerful independent indicator of extensive coronary disease [17]. The ratio TG/HDL-c, initially proposed by Gaziano *et al*., is an atherogenic index that has proven to be a highly significant independent predictor of myocardial infarction, even stronger than TC/HDL-c and LDL-c/HDL-c [46]. Bampi *et al*., reported that TG/HDL-c ratio is possible to approximately determine the presence and extent of coronary artery disease (CAD) by non-invasive methods [47]. In smoker group observed higher values (7.47±0.81; p<0.0001) than the non-smokers.

AIP shown an inverse relationship that exist between TG and HDL-c and that the ratio of TG to HDL-c is a strong predictor of infarction and it was used by some practitioners as significant predictor of atherosclerosis [46]. Other researchers are suggested that, AIP is a highly sensitive marker of difference of lipoprotein in patients. AIP values of −0.3-0.1 are associated with low, 0.1-0.24 with medium and above 0.24 with high cardiovascular risk [48]. The API values of smoker group showed significantly higher (0.79±0.04; p<0.0001) than non-smoker.

AC is a measure of cholesterol in LDL-c, VLDL-c lipoprotein fractions with respect to good cholesterol or HDL-c. It reflects the atherogenic potential of the entire spectrum of lipoprotein fractions. The higher values, higher the risk of developing cardiovascular diseases and *vice versa* [49]. In smoker group observed significantly higher values (4.74±0.18; p<0.0001) of than non-smokers.

CONCLUSION

In this study, we observed the higher levels of BMI and elevated levels of lipid profiles in smoker group when compared to the non-smokers group. These results support the previous studies, smoking habit of individual alter lipid metabolism and elevated levels of lipid profiles. When compared of atherogenic indices, higher values are observed in smoker subjects. Based on earlier studies, these atherogenic indices are powerful indicator to predict the risk of CADs. As per our knowledge, this is the first report to predict cardio vascular risk in smoker by using these new atherogenic indices. So would conclude that these indices are lipid ratios could be used for identifying individual at higher risk of cardiovascular disease in the clinical practices especially, when the absolute values of lipid profile seem normal or higher and not markedly deranged or in centers with insufficient resources.

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