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Plasma Levels of Total Antioxidant Capacity in Relation to Cardiovascular Risk Factor in Cigarette Smokers

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ABSTRACT: Accumulating evidence suggests that smoking is associated with stress oxidative and cardiovascular risk factors. To analyze whether lipid profile markers as cardiovascular risk factors such as total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL) and high density lipoprotein (HDL) are associated with Total antioxidant capacity in smokers. Twenty one adult smoker men, 42 ± 4.5 year-olds were recruit fin this study. Fasting blood samples were collected of all participants with regard to measure all variables. Pair-wise correlations between total antioxidant capacity and cardiovascular risk factors were assessed by Pearson's partial correlation coefficients. Level of significance was set at 95%. Analysis data showed that total antioxidant capacity was negatively correlated with TG (p = 0.000) and positively correlated with HDL (p = 0.000). The association between total antioxidant capacity and TC was borderline significant (p = 0.000). No significant correlation was observed between total antioxidant capacity and LDL in studied subjects (p = 0.000). These results support important role of cigarette smoking on both stress oxidative and cardiovascular status.

Keywords: Stress oxidative, smoking, cardiovascular risk factor

INTRODUCTION

Today, smoking (tobacco products) is one of the major threats to human health. Over the past two decades, smoking has been reported to be a major cause of mortality in developed or developing countries [1]. Clinical studies showed that cigarette smoke contains 4000 poisonous substances [2].

Several factors are involved in cardiovascular disease deaths in both developed and developing countries. Among them, the role of cigarettes and other tobacco products has always been stressed by healthcare academics. According to a study in 1990, nearly 20% of cardiovascular deaths were smoking-related [3]. Research evidence suggests that cigarette smoking damages vascular endothelium which has an important role in the pathogenesis of cardiovascular diseases. However, the mechanisms by which smoking can alter endothelial homeostasis remains unknown [4].

On the other hand, some studies have suggested that quitting smoking delays the risk of cardiovascular disease for 5 to 10 years [5]. However, the immediate effects of quitting smoking on biomarkers associated with cardiovascular risk factors have not yet been fully studied. Researchers have noted that smoking increases cardiovascular risk factors, such as TG and LDL, and reduces HDL [6]. Literature has stressed the higher levels of dyslipidemia among smokers compared to non-smokers [5]. The researchers noted that smoking considerably decreases HDL and increases triglycerides compared to non-smokers [7,8]. These studies indicated a direct relationship between cigarette smoking and levels cardiovascular risk factors.

However, clinical studies showed that more than 1014 free radicals and 4700 complex mixtures of chemical compositions exist in a pack of cigarettes [9]. Extensive studies on the increased cardiovascular disease in smokers often have indicated decreased antioxidants levels and increased oxidized lipid and lipoprotein levels due to smoking [10, 11]. Literature has noted that the level of lipid peroxidation was significantly higher in smokers compared to non-smokers, which leads to atherosclerosis due to the destruction of lipids [12]. Although the literature has noted both reduced total antioxidant capacity and increased cardiovascular risk factors, their relationship in smokers is not well-studied. Hence, the present study aimed at identifying the relationship between total antioxidant capacity and lipid profiles indicators and is entitled cardiovascular risk factors in a group of male smokers.

METHOD AND MATHODS

A. Study Subjects and inclusion

Subjects were twenty one sedentary adult smokers (men) aged 42 ± 4.5 year and body weight 94 ± 4.7 kg that selected in this study by accidentally. All smokers were non athletes and non-alcoholic. After the nature of the study was explained in detail, informed consent was obtained from all participants.

Inclusion criteria to study were smoking history of at least 10 cigarettes a day for 5 years. Participants were included if they had not been involved in regular physical activity or diet in the previous 6 months. Exclusion criteria for the study group were: diagnosed type 2 diabetes, coronary artery disease, cerebrovascular disease, and peripheral artery disease, using medicine or hormone preparations that affect the carbohydrate and lipid metabolism.

B. Anthropometrical measurements

Anthropometric measurements were performed in all study participants before breakfast, with the subject wearing light clothing without shoes. All anthropometric measurements were made by the same trained general physician. Height was measured without shoes on standing while the shoulders were tangent with the wall. Body weight was measured in duplicate in the morning following a 12-h fast. Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared (m2). The abdominal circumference was measured to the nearest 0.1 cm, using a non-extendable flexible tape applied above the iliac crest and parallel to the ground; with the subject standing erect with abdomen relaxed, arms along the body, and feet together. Hip circumference was measured at the maximum circumference between the iliac crest and the crotch while the participant was standing and was recorded to the nearest 0.1 cm.

C. Laboratory

In each subjects, a venous blood sample was collected from all the subjects who came after a 12-h overnight fast between the hours of 8 to 9 am. Blood samples used to measure clinical mentioned markers. Subjects were asked to avoid doing any heavy physical activity for 48 hours before blood sampling. Total cholesterol, HDL and LDL cholesterol and triglyceride were measured using the colorimetric enzymatic method with COBAS MIRA from Roche (Lörrach, Germany). To measure total antioxidant capacity in plasma, we used FRAP method (the sensitivity of method was 0.1 Units/ml).

D. Statistical analyses

Data were expressed as individual values or the mean ± SD. Statistical analysis was performed with the SPSS software version 15.0. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. The bivariate associations between total antioxidant capacity and cardiovascular risk factors were examined with the Pearson rank correlation analysis in studied subjects. P value of <0.05 was accepted as significant.

RESULTS

In present study, we aimed to determine the association of plasma total antioxidant capacity with cardiovascular risk factors such as TG, TC, LDL and HDL-cholesterol. Analysis was performed by Pearson correlation method. Subject characteristics of anthropometrical, lipid profile and total antioxidant capacity are summarized in Table 1. All values are given as mean and standard deviation.

Table 1: Mean and standard deviation of anthropometric and metabolic characteristics of studied subjects.

Variable	Mean	Standard deviation
Age (years)	42	4.5
Weight (kg)	94	4.7
Height (cm)	175	3
Body mass index (kg/m^2)	30.8	1.67
Body Fat (%)	31.2	2.64
Abdominal Circumference (cm)	104	6.8
Hip circumference (cm)	102	4.7
Total Cholesterol (mg / dl)	176	40
Triglyceride (mg / dl)	168	55
Low density lipoprotein (mg / dl)	102	20
High density lipoprotein (mg / dl)	44.9	5.3
Total antioxidant capacity (mmol/L)	0.30	0.22

Plasma total antioxidant capacity were negatively correlated with TG (p = 0.018, r = 0.511, Fig 1). The plasma level of total antioxidant capacity was positively associated with HDL-cholesterol (p = 0.006, r = 0.58). A borderline signigcant negative association was observed between total antioxidant capacity and TC in studied subjects (p = 0.061, r = 0.415). There was no significant relationship between total antioxidant capacity and LDL-cholesterol in studied subjects (p = 0.289, r = 0.24).

Table 2: Relationship of plasma total antioxidant capacity with cardiovascular risk factors in smoker subjects.

		Total antioxidant capacity (mmol/L)	Total Cholesterol	Triglyceride	LDL-Cholesterol	HDL-cholesterol
Total antioxidant capacity (mmol/L)	Pearson Correlation	1	-.415	-.511*	-.243	.577**
	Sig. (2-tailed)		.061	.018	.289	.006
	N	21	21	21	21	21
Total Cholesterol	Pearson Correlation	-.415	1	.274	.682**	-.317
	Sig. (2-tailed)	.061		.230	.001	.162
	N	21	21	21	21	21
Triglyceride	Pearson Correlation	-.511*	.274	1	.069	.089
	Sig. (2-tailed)	.018	.230		.765	.703
	N	21	21	21	21	21
LDL-Cholesterol	Pearson Correlation	-.243	.682**	.069	1	-.398
	Sig. (2-tailed)	.289	.001	.765		.074
	N	21	21	21	21	21
HDL-cholesterol	Pearson Correlation	.577**	-.317	.089	-.398	1
	Sig. (2-tailed)	.006	.162	.703	.074	
	N	21	21	21	21	21

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

DISCUSSION

The findings of the present study support the direct relationship between total antioxidant capacity and HDL. These findings indicated that the increase or decrease in the total antioxidant capacity due to smoking is consistent with changes in HDL levels. It is reported that many cardiovascular incidents that commonly appear in middle age root in tobacco and cigarette smoking especially in adolescence and youth. These irreparable damages are not exclusive to smokers, and those non-smokers who are exposed to secondhand smoke are also affected, and the incidence of cardiovascular diseases and cancers in this population is much higher than those who are not exposed to cigarette smoke [13]. Long-term cigarettes smoking increased the incidence of common diseases such as atherosclerosis and COPD with detrimental effects on systemic inflammation [14]. Long-term cigarettes smoking increases inflammatory biomarkers such as CRP, fibrinogen, IL-6 and is accompanied by an increased number of WBCs [14].

The health effects of smoking are so serious that the increased levels of inflammatory mediators such as CRP is present even after 20 to 30 years of quitting [14]. The literature has also noted weight loss caused by smoking. Findings of some studies reported a significant relationship between regular smoking and decreased body mass index and atrophy in smokers [15, 16, 17].

In the present study, statistical findings revealed a direct relationship between total antioxidant capacity and HDL. Statistical findings also pointed out a significant inverse relationship between total antioxidant capacity and blood triglyceride levels as a cardiovascular risk factor. Furthermore, although the inverse relationship between total antioxidant capacity and TC was not significant, the inverse relationship was clinically considerable. The lack of relationship between total antioxidant capacity and LDL in the studied smokers may be attributed to the low number of samples. It is also possible that the blood LDL levels in smokers indirectly affect the total antioxidant capacity.

It was found that smokers have higher levels of cardiovascular risk factors such as triglycerides, total cholesterol, and LDL compared to non-smokers [18]. It is noted that the most important constituent factors of the metabolic syndrome in smokers is hypertriglyceridemia and low HDL [19]. However, some studies have reported the lack of relationship between cardiovascular risk factors such as cholesterol with smoking [20]. Some factors, such as reactive oxygen species (ROS), increased blood glucose, lipids, and lipoproteins play a key role in the endothelial dysfunction [21, 22], and the literature supports the potential impact of smoking on their impairment.

However, the results of another study suggested the acceleration in formation of blood lipid peroxides, and thus, the exacerbation of the adverse effects of these materials on the vessel wall in smokers [23]. The relationship between cigarette smoking and some metabolic and immune factors was found to be significant as research has showed a close significant relationship between cigarette smoking and blood homocysteine levels, folic acid, cholesterol, blood pressure, uric acid, and blood lipoproteins such as LDL and HDL [21]. Numerous studies indicated a close significant between cigarette smoking and high cholesterol levels [24, 25, 26]. However, some studies have reported an inverse relationship between smoking and blood cholesterol levels [27] or lack of relationship between smoking and hypertension [28, 29]. Some other studies have also reported increased levels of LDL and low levels of HDL in smokers compared to non-smokers [25, 26, 28]. Accumulation of nicotine products in plasma LDL and the structural proteins in the vessel wall and endothelial cells and the vascular damage caused by cigarette smoking increases the incidence or severity of vascular diseases [29, 30].

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