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Research Article

Trace Elements as Risk Indicator for Coronary Artery Disease among Cigarette Smokers

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ABSTRACT
Cigarette smoking has been accepted as a risk factor for coronary artery disease. The present study investigate the influence of cigarette smoking on Copper (Cu), Zinc (Zn), Chromium (Cr), Nickel (Ni) and cholesterol (TC) and triglycerides (TG). Thirty three healthy men (21 Smokers and 12 non-smokers) from Thanjavur Transport Corporation, Thanjavur, volunteered to participate in this study. A questionnaire was used to assess smoking habits, blood pressure and anthropometric measurements. The random blood samples were collected. Two statistical methods were used. The Pearson correlation co-efficient indicates the strong positive relationship of Cu with smokers, Ni and Zn. Likewise Zn reciprocates its positive relation with smokers and Ni. TG is significantly correlated with smokers and Cu. As per PCA analysis, Factor 1 shows strong influence due to smoking, Zn, Cu, and TG. The factors in 2 and 3 one finds the role of Cr and Ni, and TC and TG respectively. These findings suggest that cigarette smoking may cause an imbalance among trace elements which may initiate the deterioration process associated with cardiovascular disease; thereby it becomes a risk factor to coronary artery disease.

Keywords :- Cigarette Smoking, Trace elements, Occupational risk.

INTRODUCTION

Cigarette smokers have generally higher order of risk in becoming a victim to coronary artery disease (CAD) than non-smokers. Several possible explanations have been offered for this association, Plasma lipids are well-known factors in leading to the development of cardiovascular disease. Cigarette smoke is the known source of oxidants, responsible for the pathogenesis of lungs and vascular system. A number of researchers have already shown that classical and extrinsic factors such as smoking, high cholesterol levels and high blood pressure have a significant role in the pathogenesis of cardiovascular disease. Recently, many other factors have been repeatedly pointed out to influence this disease. Tobacco smoke contains many oxidants and free radicals that can cause damage to lipids, proteins, DNA, carbohydrates and other bio-molecules. In-vivo antioxidant nutrients which include vitamin C, Se, Zn and Cu play a crucial role in defending against oxidant damage [1]. Each puff of a tobacco contains 104 oxidants in the tar phase and 105 in the gas phase. It has been demonstrated that one of the prominent risk factors for increased lipid peroxidation is smoking [2]. In some studies, it has been shown that tobacco smoking can alter trace elements metabolism [3]. Since trace elements are required in small quantities as an essential component of antioxidative enzymes (cytoplasmic Cu-Zn- superoxide dismutase contains copper and zinc metals as cofactors), tobacco smoking can affect its activities, thereby indirectly damaging trace elemental metabolism [4]. The search for its etiology has led to some theories that dietary intake of minerals and in particular, trace elements may have a role in the progress of atherosclerosis [5]. The present study was conducted to obtain data on the effects of cigarette smoking to trace elements, total cholesterol and triglycerides in leading to the risk of CAD.

MATERIALS AND METHODS

Participants: The study was conducted on thirty three healthy drivers and conductors (21 Smokers and 12 non-smokers) from Thanjavur Transport Corporation (TTC), Tamil Nadu, who volunteered to participate in this study. The protocol was approved by the ethical committee of SASTRA University. Subjects were clearly informed, in advance about the purpose and a written consent was obtained from them.

Clinical Examination: Volunteers were subjected to medical examination including anthropometric by well-trained staff. A questionnaire was given to assess the smoking habits. The random blood samples were collected during April 2006 from all volunteers. Serum concentrations of total cholesterol (TC) and triglyceride (TG) determinations were performed by a fully automated A15 Biosystem analyzer. Serum concentrations of trace elements were measured by AAS coupled with graphite furnace (Perkin Elmer, USA). Worksite BP was measured with a digital sphygmomanometer after the subject had rested 5 minutes in a reclining position to eliminate observer's bias. The above examinations were conducted by

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Table 1:- Pearson Correlation Coefficients

TC	1						
TG	-0.111	1					
Cu	0.143	0.421*	1				
Zn	-0.166	-0.328	-0.856**	1			
Cr	0.017	-0.13	-0.312	0.08	1		
Ni	0.215	-0.019	0.396*	-0.478*	-0.433*	1	
Smoke	0.182	0.379*	0.89**	-0.919**	-0.078	.327	1
	TC	TG	Cu	Zn	Cr	Ni	Smoke

** Correlation is significant at the 0.05 level (two-tailed)*
***Correlation is significant at the 0.01 level (two-tailed)*

physicians from Vaidhyanatha Arokiyasala, hospital, SASTRA UNIVERSITY. To investigate the influence of cigarette smoking on serum Cu, Zn, Cr, Ni and TC, TG Pearson correlation coefficient and Factor analysis (PCA) model were used. We used SPSS 12.0 Software for all the statistical analysis.

RESULTS

The demographic variables like, age mean of workers was observed to be 45 yrs. ranging from 36 to 56 yrs. The mean body weight and body mass index (BMI) were similar for both smoker and non-smoker groups. Mean systolic and diastolic blood pressure values were not significantly higher for smokers than those of non-smokers.

Table 1 presents Pearson correlation coefficients of smokers and non-smokers variables are correlated with at least one variable. Moreover, all correlations are significant at 0.01 or 0.05 levels of significance in a two tailed test. Cu was found to correlate significantly with smoking, Ni and negatively with Zn. Zn was found to negatively correlate significantly with smoking and Ni. Cr was found to negatively correlate significantly with Ni. TG was found to correlate significantly with smoking and Cu.

Principle factor analysis was used to identify the initial set of uncorrelated factors. The number of components to be retained was based on Scree plot analysis and eigenvalue criteria (1.0). Varimax (orthogonal) rotation was used to obtain a set of independent interpretable factors. The resulting factor pattern was interpreted using factor loadings of ≥ 0.4. Factor analysis was used to delineate the role of trace elements among the smoking habits. A total of 7 variables were analyzed. The data were measured using Kaiser-Meyer-Olkin (KMO) of Sampling Adequacy (0.679) and Bartlett's test of sphericity ($p < 0.0001$). Each of the variables significantly correlated between five and nine of the others, showing that they were all interrelated. The factor patterns were extracted from a correlation matrix after principal-component analysis and orthogonal rotation (Table.2). With three uncorrelated factors, in aggregate, was amounted to 81.5 % of the total variance in the data set was significantly found. Factor 1 had strong influence with smoking ($p < 0.0001$), Zn ($p < 0.0001$) and Cu ($p < 0.0001$), in addition to a strong correlations with TG ($p = 0.0014$). This factor occupied 43.8 % of the total variance. Factor 2 had strong contributions from Cr ($p < 0.0001$) significant correlation with Ni ($p < 0.0001$). It spread to 20.5 % of the total variance. Factor 3 pointed out the strong relation of TC ($p < 0.0001$) and TG ($p = 0.0002$). This factor accounted for 17.0 % of the total variance.

DISCUSSION

The risk of developing CHD increases with length and intensity of exposure to cigarette smoke. In this study, relationships of trace elements among smokers related to coronary artery disease were studied using factor analysis. Factor 1 shows significant correlation with smoking, Cu, Zn, and TG. During smoking, the chemicals in the cigarette, particularly nicotine and carbon monoxide, damage the cardiovascular system. Nicotine causes both immediate and long term increase in blood pressure, heart rate, cardiac output and coronary blood flow. Carbon monoxide binds to the haemoglobin, which is what normally carries oxygen from the lungs via the bloodstream, and therefore, reduces the amount of oxygen reaching body tissues. Smoking also makes blood vessels and blood cells sticky, allowing cholesterol and other dangerous fatty materials to develop atherosclerosis. In this study, Cu negatively correlated but significantly with smoking, Zn and Ni. Low zinc levels have been related to excess release of steroids. Due to the release of leucocyte endogenous mediators which redistributes in the body the Zn from serum and may cause a drop in serum Zn. Due to these decreased levels of Zn, the cholesterol content may increase in the blood. The injured heart muscle shows decreased Zn concentration, which may be related to the loss of LDH from infracted heart tissue. The increase in Cu may also be due to injury and subsequent necrosis of myocardial cells. The increased levels of Cu may be due to a rise in the copper-binding capacity of ceruloplasmin. Sam and Amal, (2004) have suggested that the smokers have significantly lowered level of retinol, alpha-tocopherol, Se, and Zn and increased concentrations of Cu [6] it can be inferred that the smoking decreases the concentrations of trace elements. Lekakis and Kalofoutis, (1980) have reported that measurement of Zn in the serum may have diagnostic value for acute myocardial infraction. Large amount of Zn is needed by the myocardium for the synthesis of enzymes depending upon this metal ion, because these enzymes function in repair of the myocardial damage [7]. Cr deficiency has been shown to increase aortic lesions. Factor 2 correlated with Cr and

Table 2:– Rotated Factor pattern

		Component		
S.No	Variable	1	2	3
1	Smoke	.961		
2	Zn	-.939		
3	Cu	.910		
4	Cr		.916	
5	Ni		-.717	
6	Cho			.806
7	TG	.533		-.606

Loadings ≥ 0.40 in bold type

Ni. This factor principally relates to atheromatous plaque formation and, to a lesser extent, hypernickelemia for acute myocardial infraction. The role of Ni in CHD has been implicated. Low concentration of exogenous Ni chloride has been shown to induce coronary vasoconstriction in rat heart. Francesco, et al (2004) has concluded that the serum levels of Ni are more elevated in traffic policemen than those administrative workers. So, the presence of Ni as fuel additive in lead free fuels and as catalyser in catalytic exhausts, could be one of the causes for the increased serum levels of Ni [8] Similarly, this study population working in transport corporation that contained same fuel additive and air pollution may cause serum levels of trace elements. The heavier petroleum products, such residual fuel oil fly ash (ROFA) contains a relatively high content of toxic trace metals (e.g., soluble nickel and vanadium sulfate salts) and compared to coal fly ash is highly toxic to pulmonary alveolar macrophages, in part due to the soluble toxic metals. Kromhout, et al, (1985) have inferred that the serum Zn was inversely related to resting heart rate; serum Cu was positively related to cigarette smoking and inversely to HDL [9]. Serum Cu was positively associated with fasting serum TG [10] the similar trend was found to be significantly correlated with Cu and TG. Factor 3 strongly correlated between TC and TG. This factor is primarily related to hyperlipidemia with relationship to hypertriglyceridemia. A decrease in the levels of Zn and HDL-c strongly suggests the role of Zn in cholesterol and lipid metabolism. Limal, et al, (2006) evaluated the greater alterations in the lipid profile in HDL-c, LDL-c, and TG levels, with trace elements [11] Issa Nourmohammadi, et al, (2001) has compared the status of serum Zn, Cu, Cr and Ni as well as levels of TG, TC, LDL and HDL-C in patients with and without atherosclerosis [12].

Conclusion

Cigarette smoking and its influence on CAD is a well known fact. However, the influence of cigarette smoking in enriching the trace elements so as to expedite the occurrence of CAD has not been attempted. Such influence varies according to race, age, climate, behavior, etc. this work is on effort to unravel the cigarette smoking's effect on this region among Dravidian race. Smoking is directly propositional to Cu while Zn, Cr, keeps anti-pathic relation. In conclusion, the effect of cigarette smoking can change or damage the abnormal levels of trace elements that may play a role in the development of coronary artery diseases. We need to undertake further elaborate surveys and observe the effects of cigarette smoking that alter the serum trace element levels on CHD risk factors in a massive scale.

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