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Effects of Cigarette Smoking on Serum Cholesterol and Triglyceride Levels; Implication for Cardiovascular Morbidity

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Abstract: ***This study was designed to compare serum Cholesterol and triglyceride levels between cigarette smokers and non-smokers and to evaluate the effect of smoking intensity on these parameters. This was a cross-sectional comparative study between 40 non-cigarette smokers and 60 cigarette smokers between ages 25 and 55 years residing in Ekpoma, Nigeria. Volunteers were apparently healthy non-obese male subjects who gave informed consents. Questionnaires were used to collect data on age and smoking intensity. Blood samples were collected for analysis of serum lipids using standard procedures. Data were the analyzed using analysis of variance (ANOVA) and paired t-test on SPSS version 20.0 and P values of less than 0.05 was considered significant. The results showed significantly lower mean total cholesterol and triglyceride levels in non-smokers as compared to cigarette smokers. There was a dose dependent significant increase (p<0.05) in mean serum total cholesterol and triglyceride levels between light and heavy cigarette smokers. The findings of this study showed that cigarette smoking has an adverse effect on lipid profile thereby predisposing smokers to increase risk of cardiovascular diseases such as atherosclerosis.***

Keywords: Cigarette smoking, Cardiovascular diseases, Cholesterol, Triglyceride

1. Introduction

Smoking is a major risk factor for cardiovascular morbidity and mortality, and is considered to be the leading preventable cause of death in the world[1, 2]. Epidemiologic studies strongly support the assertion that cigarette smoking (CS) in both men and women increases the incidence of myocardial infarction (MI) and fatal coronary artery disease (CAD)[3, 4, 5, 6]. Even low-tar cigarettes and smokeless tobacco have been shown to increase the risk of cardiovascular events in comparison to nonsmokers[7, 8]. Furthermore, passive smoking (environmental tobacco exposure) with a smoke exposure about one hundredth that of active CS is associated with approximately a 30% increase in risk of CAD, compared with an 80% increase in active smokers[9, 10].

Despite all the publicity concerning the documented adverse effects of smoking, the prevalence still remains high as studies showed that cigarette smoking is on the increase and even among the young. In fact, smoking is reported to account for 25% of middle-aged cardiovascular deaths[11] and has been recognized as the second most important risk factor in the burden of disability-adjusted life years as well as the primary risk factor for premature mortality, associated with about 1.6 million deaths annually[12] and other diseases in the body such as compromising renal function[13]. The European Society of Cardiology reported recently that smoking causes 28% of cardiovascular deaths in men aged 35 to 69 years and 13% in women of the same age[14].

Smoking ranks among the top causes of cardiovascular disease, including coronary heart disease, ischemic stroke, peripheral artery disease and abdominal aortic aneurysm[14]. Smoking, either active or passive, can cause cardiovascular disease via a series of interdependent processes, such as enhanced oxidative stress, haemodynamic and autonomic alterations, endothelial dysfunction, thrombosis, inflammation, hyperlipidaemia, or other effects[15]. Smokers have significantly higher serum cholesterol, triglyceride, and low-density lipoprotein (LDL) levels, but high-density lipoprotein is lower in smokers than in nonsmokers[16].

2. Materials and Methods

Study design

The study was a comparative cross-sectional study among apparently healthy non-cigarette smokers and apparently healthy cigarette smokers around Ekpoma, Esan West Local Government Area of Edo State, Nigeria.

Study sample

The study was carried out among adult males between the ages of 25 and 59 years. Non-smokers group consisted of 40 male subjects who had never smoked as at the time of the study. Cigarette smoker group consisted of 60 subjects who have being smoking cigarettes for at least six month before the study. The cigarette smokers group was then classified into heavy smokers (those smoking more than seven sticks daily) and light smokers (those smoking less than seven sticks and irregular daily).While the non-smoker group was sampled randomly, the smokers group was via purposive convenience sampling method.

Exclusion criteria

Those who reported using alcohol were excluded as well as subjects on any medication(s) and having any disease conditions like hypertension, diabetes, renal, hepatic

diseases and other chronic diseases. In addition, ex-smokers were excluded from this study.

Procedure

Interview was carried out after explaining the aims of the study and obtaining consent to all of the participants. Thereafter, using interviewer administered questionnaire, information on demographic profile, smoking and medical history were obtained from all participants. Thereafter 5mls of whole blood sample was then collected from all that met the inclusion criteria after a fast for at least 12hrs via venipuncture following standard laboratory procedures. The blood samples were then taken to the laboratory and centrifuged at 3000rpm for 10 minutes to obtain the serum for the analysis of serum cholesterol and Triglyceride.

Serum cholesterol was estimated using the method described by Allain et al[17] with kit assay system (Radox United Kingdom). Serum triglyceride was estimated following the method described by Abell et al[18] using kit (Radox United Kingdom). Both serum cholesterol and triglyceride were then obtained from the calculation using the concentration of the standard.

Data analysis

The obtained data were then analyzed using the Statistical Package for Social Sciences (SPSS,version 20) and results were described using mean and standard deviation. Where applicable, the student "t –test" and ANOVA were used to compare the differences of serum cholesterol and triglyceride between groups at significant level of p-value of <0.05 with a confidence level of 95%.

3. Results

A total of 100 subjects took part in this study and they consisted of 40 non-smoker herein represent the control and 60 cigarette smokers herein represent the cigarette smoker group (test). Figure 1 compared the different in total cholesterol level (mg/dl) between smokers and non-smokers. It was observed that mean total cholesterol increased significantly ($p<0.05$) in the cigarette smoker group (215.76±38.97mg/dl) compared to the non-smoker group (173.71±23.10mg/dl). Similarly, mean serum triglyceride (figure 2) was observed to significantly increase ($p<0.05$) in the cigarette smoker group (162.97±7.95mg/dl) compared to the non-smoker group (126.58±7.71mg/dl).

Table 1 showed the effect of smoking intensity on serum mean total cholesterol and triglyceride levels in cigarette smokers and non-smokers. Mean serum total cholesterol and triglyceride levels were observed to increase in smokers in a dose dependent manner. Specifically, mean serum total cholesterol significantly increase ($p<0.05$) in heavy smoker (n= 32; 240.95±31.29mg/dl) than light smoker (n=28; 186.96±24.15mg/dl) and these were significantly higher ($p<0.05$) compared to the non-smoker group (n=40; 173.71±23.10mg/dl).Similarly, mean serum triglyceride was significantly higher ($p<0.05$) in the heavy smokers(165.28±7.70mg/dl) compared to light smokers (160.32±7.38mg/dl) and these were significantly increased compared to the non-smoker group (126.58±7.71mg/dl).

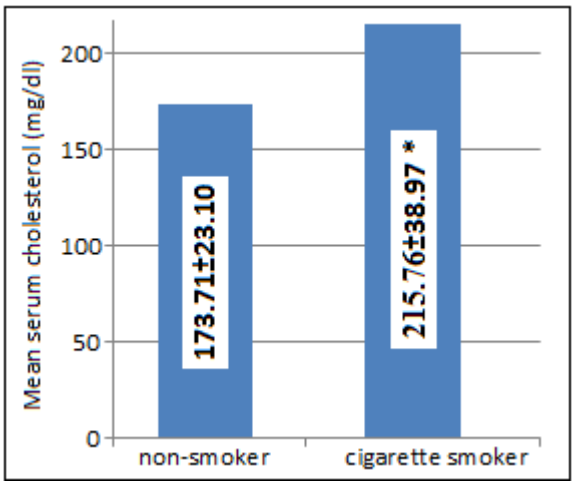

Figure 1: Mean serum total cholesterol level comparism between smokers and non-smokers*(values are mean±Standard deviation and unit is in mg/dl; * indicates statistical significant at p<0.05)*

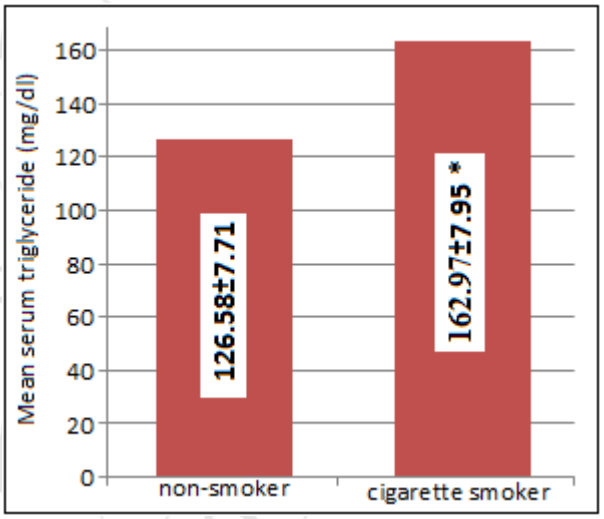

Figure 2: Mean serum triglyceride level comparism between smokers and non-smokers
*(values are mean±Standard deviation and unit is in mg/dl; * indicates statistical significant at p<0.05)*

Table 1: Effect of smoking intensity on serum mean total cholesterol and triglyceride levels in cigarette smokers and non-smokers

Mean serum parameters	Non-smoker (control; n=40)	Light smoker (n=28)	Heavy smoker (n=32)
Total cholesterol (mg/dl)	173.71±23.10[a]	186.96±24.15[b]	240.95±31.29[c]
Triglyceride (mg/dl)	126.58±7.71[a]	160.32±7.38[b]	165.28±7.70[c]

(Values are mean±Standard deviation; mean in a row having different superscripts are significantly different at p<0.05)

4. Discussion

Cigarette smoke contains more than 4000 chemical substances that have harmful effects on cardiovascular function[19] through which it is asserted to have a negative impact on lipid profile and in turn cardiovascular function. The present study investigates the effect of cigarette

smoking and its intensity on serum cholesterol and triglyceride levels.

The present study showed significantly higher mean value of serum total cholesterol and triglyceride in cigarette smokers compared to nonsmokers (figure 1 and 2). These findings contradict the findings by Mjos[20] that there is no change in total cholesterol and triglycerides levels in cigarette smokers as compared to non-smokers. However, in agreement with our results, significant increase in the total cholesterol, triglyceride and even low-density lipoproteins have been documented by Devaranavadgi et al[16, 21] in smokers compared to nonsmokers. In fact, it has long been reported that smoking exacerbates increased serum lipid levels[22].

Compared to non-smokers, we observed significantly higher serum cholesterol and triglyceride levels in all groups of cigarette smokers with increased risk in heavy smokers (table 1). This indicates that cigarette smoking has a dose dependent effect on serum lipid profile such as total cholesterol and triglyceride as shown in this study. In accordance to this finding, studies have reported dose dependent tendency lipoprotein cholesterol in smokers[16, 23]. These findings showed that cigarette smoking has significant effect on lipid metabolism and the regulation of lipid levels in the blood. Thus, cigarette smoke could promote atherosclerosis, in part, via its effects on the lipid profile as previously reported by Ambrose and Barua[15]. Thus, high cholesterol and triglyceride cause by smoking may be among the most important factors that may lead to coronary artery disease and this has previously been acknowledge by Zamir et al[24].

Based on our findings, lipid constituents may explain the way in which cigarette smoking affects plasma lipid profile and contribute to cardiovascular issues and this may possibly be via oxidative damage to protein. This assertion is based on the report by Craig et al[16] that cigarette smoking increases the oxidative modification of lipids specifically via low density lipoproteins. Two potential mechanisms have been suggested as ways smoking produce their deleterious effects on plasma lipoproteins[25]. According to McCall et al[25], the first was said to be by direct action of gas-phase cigarette smoke components interaction on the lung and the second via indirect action of gas-phase cigarette smoke activating macrophages and neutrophils in the lung to release enzymes and oxidants capable of damaging lipids and proteins.

In conclusion, the significantly raised serum cholesterol and triglyceride levels and the dose dependent relationship in these lipid profile between heavy and light cigarette smokers, emphasis the vascular endothelium damaging potentials of cigarette smoking and this no doubt predisposes to cardiovascular morbidity and mortality. In fact, the risk of significantly increase in serum cholesterol and triglyceride in cigarette smokers as compared to non-smoker indicate smokers to have greater risk of atherosclerotic plaques.

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