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Association between Serum Total Cholesterol and Cigarette Smoking among Adult Male population of Mymensingh District, Bangladesh

*Naznin R[1]

Abstract

Cigarette smoking is one of the most widespread potentially harmful social habits practiced worldwide. A cross-sectional, comparative study was conducted in the Department of Physiology, Mymensingh Medical College, Bangladesh, and the Mymensingh locality, between July 2014 and June 2015, to determine the effect of smoking on serum total cholesterol levels among male smokers. A total of 150 adult males participated in this study recruited from outpatient department of Mymensingh Medical College Hopspital hailing from different parts of Mymensingh district. Among them, apparently healthy 50 non-smokers were considered as control in group-I, while the other 100 cigarette smokers (smoking ≥20 cigarettes per day) were divided equally into two sub-groups, as group-IIA (5-10 years duration of smoking) and group-IIB (having history of >10 years of smoking). We adopted a purposive, non-randomized sampling method. A semi-structured questionnaire including sociodemographic data and habits of smoking was completed by all study participants. Serum total cholesterol level was measured by enzymatic hydrolysis and oxidation method. There was no significant difference regarding the mean age and BMI of the participants in different groups ($p<0.05$). However, the mean systolic and diastolic blood pressure were increased gradually with duration of smoking in comparison to control (group-I), which was statistically significant ($p<0.001$); however, all the values were within physiological limits. The mean serum total cholesterol levels 152.28±1.22 mg/dL, 190.8±1.71 mg/dL, and 190.28±1.71 mg/dL in group-I, group-IIA, and group-IIB, respectively. Serum total cholesterol levels were found increased in both group-IIA and group-IIB compared to control (group-I). The differences were statistically significant ($p<0.05$). Serum total cholesterol level also found a bit higher in group-IIB compared to group-IIA. However, the difference was not statistically significant ($p>0.05$). Our study shows that cigarette smoking has a strong effects that is reflected through increase serum total cholesterol levels with the duration of smoking.

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Introduction

Smoking continues to be the second major cause of death in the world.[1] According to the World Health Organization (WHO), smoking is responsible for more than 8 million deaths annually, and one in every five cardiovascular patients dies because of smoking.[2] Cigarette smoking is a common problem in Bangladesh and also a major public health problem associated with morbidity and mortality.[3] Recent studies indicate smoking is responsible for nearly 25% of all fatalities among Bangladeshi men aged 25 to 69 years, with each smoker losing an average of seven years of life.[4] An individual was identified as a smoker if he/she smokes cigarette or bidi or a combination of these products, at least once a week. If he/she did not meet this criterion, he/she was classified as a non-smoker.[5] A cigarette contains around 6000 chemicals, which increases to 7000 chemicals when burnt. These ingredients in the cigarettes have detrimental effects on almost all organs of the body of the smoker.[6] Currently, reactive oxygen species (ROS) play a significant role in the development of atherosclerosis. The source of ROS varies between the gas or tar phase of cigarette smoke, monocytes, macrophages, neutrophils, and endogenous sources from xanthine oxidase, endothelial nitric oxide synthase (eNOS), and the mitochondrial electron transport chain.[7] Plasma lipoprotein metabolism is strongly influenced by cigarette smoking.[8] Smoking has severe adverse effect on blood lipoprotein level and associated with a greater risk for developing atherosclerosis, ischemic

1. *Dr. Rubiat Naznin, Associate Professor, Department of Physiology, Community Based Medical College, Bangladesh, Mymensingh.

Address of Correspondence:
Email: rubiatnaznin@gmail.com

heart disease, hypertension, stroke and peripheral vascular disease.[9]

The human serum contains a variety of lipid sub classes that can exert diverse impacts on the individual's physiological state. The maintenance of optimal levels of each subclass is crucial for the promotion and preservation of overall health and well-being.[10] Smoking-induced changes in cholesterol metabolism involve alterations in lipid transport enzymes, oxidative modifications of lipoproteins, and changes in serum total cholesterol levels. These effects can impact HDL metabolism, contribute to changes in lipid profile, and potentially increase the risk of cardiovascular disease.[11] Increased serum cholesterol in smokers increases the risk by 9%. Furthermore, the dose-response effect of smoking on serum cholesterol concentration suggests a gradient of increased absolute risk of coronary artery disease between light and heavy smokers.[12]

Under the circumstances, we proposed this study to comprehensively elucidate the dissimilarities in the impact of smoking on serum cholesterol levels, since smoking is a well-known risk factor for cardiovascular disease (CVD). The present study aimed to determine the association between smoking and serum cholesterol levels in a male population of Mymensingh district, Bangladesh.

Methods

This cross-sectional, comparative study was conducted in the Department of Physiology, Mymensingh Medical College, Bangladesh, between July 2014 and June 2015. A total of 150 adult males participated in this study recruited from outpatient department of Mymensingh Medical College Hospital hailing from different parts of Mymensingh district. Among them, apparently healthy 50 non-smokers were considered as control in group-I, while the other 100 cigarette smokers (smoking ≥20 cigarettes per day) were divided into two sub-groups, as group-IIA (5-10 years duration of smoking) and group-IIB (having history of >10 years of smoking), i.e., 50 in each group. We adopted a purposive, non-randomized sampling method. A semi-structured questionnaire including sociodemographic data and habits of smoking was completed by all study participants. Their body weight was measured in kilogram and height in meter. Fasting serum total cholesterol level was done by enzymetric hydrolysis and oxidation method. The participants were asked to fast for at least 8 hours and to avoid smoking and heavy physical activity for more than 2 hours before the examination. 5 ml of venous blood were drawn from the antecubital vein in the early morning between 8.00 am to 9.00 am.

After collection, data was scrutinized, coded and then analyzed by using Statistical Package for Social Sciences (SPSS) version 12.0 for windows. Data was expressed as mean±SD (standard deviation). Comparison among the groups was done using unpaired Student's t-test. A p-value <0.05 was considered as statistically significant. The study was approved by the Institutional Review Board of Mymensingh Medical College, Mymensingh, Bangladesh.

Results

Table-I shows age distribution of the study participants. Among non-smokers (group-I), group-IIA and group-IIB smokers, the maximum study participants were found in the age group of 35–45 years (44%, 50% and 46%, respectively). Table-II shows physical parameters of the groups. There was no significant difference regarding the mean age and BMI of the study participants in different groups

(p<0.05). However, the mean systolic and diastolic blood pressure were found increased gradually with duration of smoking in comparison to control group, which was statistically significant (p<0.001); all the values were within physiological limits though. Table-III shows that the mean serum total cholesterol levels were 152.28±1.22 mg/dL, 190.8±1.71 mg/dL, and 190.28±1.71 mg/dL in group-I, group-IIA, and group-IIB, respectively. Serum total cholesterol levels were found increased in both group-IIA and group-IIB compared to control (group-I). The differences were statistically significant (p<0.001). Serum total cholesterol level also found a bit higher in group IIB compared to group IIA. However, the difference was not statistically significant (p>0.05).

Table-I: Age distribution of study participants (N=150)

Age group (in years)	Group I (n=50)	Group IIA (n-50)	Group IIB (n=50)
25–35	13 (26%)	11 (22%)	12 (24%)
35–45	22 (44%)	25 (50%)	23 (46%)
45–55	15 (30%)	14 (28%)	15 (30%)

Table-II: Comparison of study groups regarding physical parameters (N=150)

Variables	Group I (n=50) Mean±SD	Group IIA (n-50) Mean±SD	Group IIB (n=50) Mean±SD	p-value
Age	45.14±0.93	45.2±0.90	45.8±0.9	>0.05^{NS}
BMI	23.39± 0.44	23.47±0.34	23±0.38	>0.05^{NS}
Systolic BP	115.80±0.97	127±0.98	132±1.18	<0.001^{S}
Diastolic BP	75.40±1.11	84.50±0.94	89.20±1.15	<0.001^{S}

Unpaired Student's t-test was applied; S=significant, NS=not significant.

Table-III: Comparison of mean serum cholesterol levels (mg/dL) (N=150)

Group I (n=50) Mean±SD	Group IIA (n=50) Mean±SD	Group IIB (n=50) Mean±SD	p-value
152.28±1.22	190.8±1.71	–	<0.001^{S}
152.28±1.22	–	190.28±1.71	<0.001^{S}
–	190.8±1.71	190.28±1.71	>0.05^{NS}

Unpaired Student's t-test was applied; S=significant, NS=not significant.

Discussion

In the present study, the mean serum total cholesterol levels were found 152.28±1.22 mg/dL, 190.8±1.71 mg/dL, and 190.28±1.71 mg/dL in group-I, group-IIA, and group-IIB, respectively. Serum total cholesterol levels were found increased in both group-IIA and group-IIB compared to control (group-I). The differences were statistically significant (p<0.05). Serum total cholesterol level also found a bit higher in group-IIB compared to group-IIA. However, the difference was not statistically significant (p>0.05). Similar results were observed by Anandha Lakshmi *et al.*[9], Yasmin *et al.*[11], Moosazadeh *et al.*[13], Anwari *et al.*[14], and Sousa *et al.*[15], as they reported that serum total cholesterol got significantly increased, along with a reduction in high-density lipoprotein (HDL) level in smokers and those changes were more evident in longer duration of smoking. Meenakshisundaram *et al.*[16] also observed the same, as the mean values of total cholesterol among mild, moderate, heavy smokers, and control subjects were 198±30.6 mg/dL, 224±27.2 mg/dL, 240±24.3 mg/dL, and 160±20.4 mg/dL, respectively. Momayyezi *et al.*[17] reported that smokers have nearly 2-fold higher chances of dyslipidemia compared to non-smokers.

Nicotine, the primary ingredient of tobacco, is a sympathetic stimulant that acts on the central and peripheral nervous systems to stimulate the release of catecholamine and other neurotransmitters. It leads to cardiovascular consequences, such as elevating the heart rate, blood pressure, and cardiac output. Furthermore, it leads to a rise in low-density lipoprotein and decrease in high-density lipoprotein by mobilizing free fatty acids, thereby intensifying vasoconstriction and accelerating the progression of blood epithelial cell injury and atherosclerosis. Moreover, cigarette smoke contains numerous free radicals and reactive oxygen species (ROS) which interfere with lipid homeostasis, accelerating lipid peroxidation and altering serum lipid levels.[18-20]

Conclusion

Increased serum cholesterol levels among smokers compared to non smokers is an indicator for development of atherosclerosis. These differences became more pronounced with greater smoking intensity. Our findings highlight the harmful cardio-metabolic effects of smoking and underscore the need for urgent public health awareness, including early intervention, lifestyle modification with proper health education and routine lipid monitoring for smokers. Moreover, we need coordinate national strategies for prevention, smoking cessation programs and control of cigarette smoking to establish tobacco free environment.

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