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ORIGINAL ARTICLE

Cigarette Smoking and its Association with Dyslipidemia among Middle-aged Population in Rajshahi District

Farhana Yasmin,[1] Parvez Hassan,[2] Md. Jawadul Haque,[3]

ABSTRACT

Background & objective: Dyslipidemia is one of the major risk factors for ischemic heart disease. Many factors contribute to the development of dyslipidemia. Smoking is one of them. Smoking and dyslipidemia are claimed to interact with each other and synergistically increase the risk of heart disease. The present study is, therefore, intended to find the association between smoking and dyslipidemia in the context of the Bangladeshi population.

Methods: The present cross-sectional study was conducted in the Department of Biochemistry, Rajshahi Medical College (RMC), Rajshahi in collaboration with the Institute of Biological Science, Rajshahi University, Rajshahi over a period of five years between 2017 and 2022. A total of 230 adult (ranging from 30 to 60 years) male subjects who attended at Biochemistry Lab of RMC to have their blood tested for lipid profile were consecutively included in the study. However, individuals with the habit of chewing tobacco, ex-smokers, or those having any diseases influencing lipid profile, taking drugs (like β-blockers, thiazide, statins, fibric acid derivatives, and nicotinic acid) that may influence lipid profile, or obese persons under dietary restrictions were excluded from the study. The subjects who exhibited either serum TC: HDL ratio > 4.5 or serum Tg: HDL ratio > 3.5 or both conditions were considered as cases and the subjects who did not have either of the conditions were considered as controls in the present study.

Result: In the present study, over one-third (34.8%) of subjects had a smoking habit with the mean duration of smoking being 13.6 ± 5.0 years. Dyslipidaemia (as defined by serum TC: HDL > 4.5) was found in 33% of subjects, while the same condition, defined by serum Tg: HDL > 3.5, was found in 42.6% of the subjects. While either of the two conditions was present in 13% of subjects, both conditions were concurrently observed in 31.3% of subjects. Of the total 230 subjects, 102(44.3%) were dyslipidaemic. Smoking habit was observed to be significantly higher in the case group (54.9%) than that in the control group (18.8%). The risk of having dyslipidaemia in smokers was > 5-fold (95% CI = 2.9 – 9.5) higher than that in non-smokers ($p < 0.001$). However, as the duration of smoking was correlated with different lipid profiles, it was not found to be significantly correlated with any of the lipid profiles.

Conclusion: The study concluded that smokers are more likely to have dyslipidaemia than non-smokers. The risk of having dyslipidaemia in smokers is much higher than that in non-smokers. While serum total cholesterol, low-density lipoprotein (LDL) and serum triglycerides are increased, serum high-density lipoprotein (HDL) is reduced.

Keywords: Cigarette smoking, dyslipidemia middle-aged male subjects etc.

Authors' information:

[1] ***Dr. Farhana Yasmin,*** *Assistant Professor & Head, Department of Community Medicine, Rajshahi Medical College, Rajshahi , Bangladesh.*

[2] ***Professor Dr. Parvez Hassan,*** *Institute of Biological Sciences, Rajshahi University, Rajshahi, Bangladesh.*

[3] ***Professor Dr. Md. Jawadul Haque,*** *Professor of Community Medicine, Vice Principal, Islami Bank Medical College, Rajshahi, Bangladesh.*

Correspondence: ***Dr. Farhana Yasmin,*** *Mobile: 01753713857, E-mail: f.yasmin5115@gmail.com*

INTRODUCTION:

Coronary artery diseases and stroke are the leading causes of disability and mortality worldwide. Atherosclerosis and coronary artery disease (CAD) are predisposed by a large number of risk factors; some are modifiable and some are non-modifiable. While the modifiable ones include hypertension, dyslipidemia, smoking, diabetes mellitus, sedentary lifestyle, non-modifiable ones are biological factors like age and sex. Cigarette smoking is an escalating health concern, especially in developing countries. Constituents of cigarette smoke that contribute to CVDs are nicotine, carbon monoxide, and oxidant gases.[1] Dyslipidemia is the presence of abnormal levels of lipids in the blood, characterized by an elevation of the level of serum total cholesterol (TC), low-density lipoprotein (LDL), or triglycerides (TG), and/or a decrease in high-density lipoprotein cholesterol (HDL).[2-3] An evidenced interaction exists between tobacco consumption in any form and hypercholesterolemia & they together synergistically act to induce the development of coronary heart diseases and or stroke. Therefore, it is crucial to screen for dyslipidemia at a young age to prevent and manage its effects.

Although smoking is an independent risk factor for coronary heart disease (CAD), the exact mechanism by which it increases the risk of coronary heart disease is still a myth. Whatever the mechanisms be, there is a significant increase in levels of total cholesterol, triglycerides, low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and reduced levels of high-density lipoprotein (HDL) among smokers. Heavy smokers showed significant dyslipidemia, an increase in red blood cell count, total leukocyte count, and neutrophil count as well.[4] The Framingham offspring study measured HDL in fasting blood samples of 4107 men and women. Cigarette smoking was found to be associated with an average difference in HDL cholesterol of about 4 mg/dl in men and 6 mg/dl in women. Furthermore, when heavy alcohol drinkers were adjusted by regression analysis, a significant inverse association between the number of cigarettes smoked and HDL cholesterol was demonstrated in both men and women.[5]

Smoking is rampantly prevailing in Bangladesh. Keeping pace with this habit, the incidence of coronary artery disease is also going up. Cigarette smoking is a well-known major risk factor for atherosclerotic change and CVD.[6-8] The results of previous studies suggest that cigarette smoking can produce oxygen-derived free radicals that damage blood lipids. These damaged lipids may promote atherogenesis & lead to atherosclerosis.[9] However, the results vary in different studies, and the effect of smoking on lipid levels is not well-defined.[10,11] A recent Korean study showed that over 40% of adults (aged over 30 years) are reported to have dyslipidemia, and the degree is much higher in men.[12] Screening for dyslipidemia and its risk factors at an early age is, therefore, of immense importance to prevent and control its consequences like ischemic heart diseases. However, there is a paucity of data on the association between smoking and dyslipidemia in the male population of Bangladesh. Hence, the present study was undertaken to find the relationship between smoking and lipid profile in young and early middle-aged male subjects with the hypothesis in mind that smokers are more likely to be dyslipidaemic than their non-smoker counterparts.

METHODS:

This cross-sectional analytical study was conducted in the Department of Biochemistry, Rajshahi Medical College (RMC), Rajshahi in collaboration with the Institute of Biological Science, Rajshahi University, Rajshahi over a period of 5 years between 2017 and 2022. Adult male subjects who came to the Biochemistry Lab of RMC to have their blood tested for lipid profile were the study population; of those who were suffering from dyslipidaemia were considered as cases, and who had lipid profiles within normal physiological range were considered as control. The study included a total of 230 adult male individuals (both healthy and diseased) from the Department of Biochemistry who attended there

to have their lipid profiles checked; of them, 102 were found to be dyslipidaemic (case) and the rest had normal lipid profiles (control). The subjects who exhibited either serum TC: HDL ratio > 4.5 or serum Tg: HDL ratio > 3.5 or both conditions were considered as cases and the subjects who did not have either of the conditions were considered as controls. Smokers in this study were meant to present smokers who have been smoking for at least one year. However, individuals with the habit of chewing tobacco, ex-smokers, or those having any diseases influencing lipid profile, taking drugs (like β-blockers, thiazide, statins, fibric acid derivatives, and nicotinic acid) that may influence lipid profile or obese persons under dietary restrictions were excluded from the study.

Data were processed and analyzed using the statistical software SPSS (Statistical Package for Social Sciences), version 25.0. Both descriptive and inferential statistics were used to analyze the data. The prevalence of smoking and other confounding variables was compared between cases and controls using Chi-squared (χ^2) Tests. The level of significance was set at 5% and a p-value < 0.05 was considered statistically significant.

RESULTS:

Age distribution shows that over half (53.5%) of the individuals were 30 – 40 years old, 32.6% were 20 – 30 years and 13.9% were 40 – 50 years old with the mean age of the subjects being 32.2 ± 5.9 years. In terms of occupation, 17% were service-holders, 23% were farmers, 18.3% were businessmen and 41.7% were involved in diverse odd jobs. Level of education depicts that 37% were SSC-level educated followed by 24.8% primary level, 18.7% HSC level, 16.5% graduate, and higher level educated. Almost two-thirds (66%) of the study subjects were urban residents. Nearly half (46.5%) had a monthly income of Taka 10000 – 20000, 23.9% between Taka 20000 – 30000 and 15.6% Taka < 10000 (Table I). Over two-thirds (68.3%) of the study subjects were accustomed to a sedentary lifestyle and the rest (31.7%) to an active lifestyle. Nutritional status measured in terms of body mass index (BMI) shows that approximately 44% were of normal nutritional status, 42.6% were overweight, and 13% were obese (Table II). Over one-third (34.8%) of the subjects were smokers with the mean duration of smoking being 13.6 ± 5.0 years. The average number of sticks smoked per day was 8 ± 3 days (Table III).

In terms of comorbidities, 17.4% of subjects had diabetes mellitus and 22.6% had hypertension with the mean duration of diabetes and hypertension being 6.4 ± 5.2 and 5.1 ± 3.7 years respectively. The fasting blood sugar > 6.4 mmol/L was found in 61(26.5%) cases (Table IV). Analyses of serum lipids show that about one-quarter (25.2%) had hypercholesterolemia (> 200 mg/dl). While raised serum LDL (≥ 130 mg/dl) was found in 21.7% of cases, the prevalence of low HDL (< 40 mg/dl) was observed in 22.6% of cases. The prevalence of elevated serum Tg (≥ 150 mg/dl) was much higher (62.6%) (Table V). Dyslipidaemia as defined by serum TC: HDL > 4.5 was found in 33% of subjects, while the same condition defined by serum Tg: HDL > 3.5 was found in 42.6% of the subjects. While either of the two conditions was present in 13% of subjects, both conditions were concurrently observed in 31.3% of subjects. Of the total 230 subjects, 102(44.3%) were dyslipidaemic (Table VI).

As smoking habit was compared between case and control groups, it was observed to be significantly higher in the case group (54.9%) than in the control group (18.8%) with the risk of having dyslipidaemia in smokers being > 5-fold (95% CI = 2.9 – 9.5) higher than that in non-smokers (p < 0.001) (Table VII). The association of smoking with different lipid profiles demonstrates that hypercholesterolemia was significantly higher among smokers with the risk of having hypercholesterolemia in smokers being 4.7(95% CI=2.7–8.9) times higher than that in non-smokers (p<0.001). Likewise, hypertriglyceridemia was staggeringly higher among smokers compared to that among non-smokers with the risk of having the condition in the former group being 4.3(95% CI=2.2–8.4) times more likely

than in the non- smokers (p<0.001). Elevated serum LDL (130mg/dl) & low HDL (<40mg/dl) also demonstrated their significant presence in the former group with risks of developing the conditions in the former group being 3.1(95% CI=1.6–5.9) and 2.1(95% CI=1.1-3.8) times higher respectively than those in the latter group (Table VIII).

Table I. Distribution of patients by their demographic characteristics (n = 230)

Demographic characteristics	Frequency	Percentage
Age distribution		
20 – 30	75	32.6
30 – 40	123	53.5
40 – 50	32	13.9
Occupation		
Service	39	17.0
Farmers	53	23.0
Business	42	18.3
Others	96	41.7
Education		
Illiterate	7	3.0
Primary	57	24.8
Secondary	85	37.0
Higher secondary	43	18.7
Graduate plus	38	16.5
Residence		
Urban	151	65.7
Rural	79	34.3
Income (Taka)		
< 10000	36	15.6
10000 - 20000	107	46.5
20000 - 30000	55	23.9
30000 - 40000	16	7.0
≥ 40000	16	7.0

***Mean age** = 32.2 ± 5.9 years; **range** = (20 – 49) yrs.

Table II. Subjects stratified by physical activities and nutritional status (n = 230)

Physical activities & Nutrition	Frequency	Percentage
Physical activities		
Active	73	31.7
Sedentary	157	68.3
BMI (kg/m^2)		
18.5 – 25 (normal)	101	43.9
25 – 30 (overweight)	98	42.6
30 – 40 (obese)	30	13.0
≥ 40 (morbidly obese)	01	0.4

Table III. Distribution of study subjects by smoking profile

Smoking profile	Frequency	Percentage	Mean ± SD
Smoking habit (n = 230)			
Present	80	34.8	---
Absent	150	65.2	---
Duration of smoking (years)	---	---	13.6 ± 5.0
Number of sticks smoked per day	---	---	8 ± 3

Table IV. Distribution of study subjects by presence of co-morbidities

Comorbidities	Frequency	Percentage	Mean ± SD
Diabetes mellitus (n = 230)	40	17.4	---
Duration of diabetes (years)	---	---	6.4 ± 5.2
FBS > 6.4 mmol/L	61	26.5	---
Hypertension (n = 230)	52	22.6	---
Duration of hypertension (years)	---	---	5.1 ± 3.7

Table V. Distribution of study subjects by lipid profile (n = 230)

Lipid profile	Frequency	Percentage	Mean ± SD
Serum total cholesterol (mg/dl)			
≤ 200	172	74.8	199.2 ± 41.2
> 200	58	25.2	
Serum LDL (mg/dl)			
< 130	180	78.3	114.6 ± 36.6
≥ 130	50	21.7	
Serum HDL (mg/dl)			
< 40	52	22.6	48.1 ± 10.1
≥ 40	178	77.4	
Serum Tg (mg/dl)			
< 150	86	37.4	182.7 ± 67.2
≥ 150	144	62.6	

Table VI. Distribution of study subjects by disorders of lipid profile (n = 230)

Disorders of lipid profile	Frequency	Percentage
Serum TC:HDL > 4.5		
Yes	76	33.0
No	154	67.0
Serum Tg:HDL > 3.5		
Yes	98	42.6
No	132	57.4
Either TC:HDL > 4.5 or Tg:HDL > 3.5		
None	128	55.7
Either of the two conditions	30	13.0
Both	72	31.3
Dyslipidaemia		
Yes	102	44.3
No	128	55.7

Table VII : Association between smoking habit and dyslipidaemia

Smoking	Outcome		Odds Ratio (95% CI of OR)	p-value
	Case (n =120)	Control (n =128)		
Yes	56(54.9)	24(18.8)	5.2(2.9 – 9.5)	< 0.001
No	46(45.1)	104(81.2)		

*Data were analyzed using the **Chi-squared** (χ^2) **Test**.
Figures in the parentheses denote the corresponding **percentage.**

Table VIII. Association of smoking with different lipid profiles

Lipid profile	Smoking status		Odds Ratio (95% CI of OR)	p-value
	Smoke (n =80)	Non-Smoke (n =150)		
Serum TC (mg/dl)				
> 200	36(45.0)	22(14.7)	4.7(2.7 – 8.9)	< 0.001
≤ 200	44(55.0)	128(85.3)		
Serum LDL (mg/dl)				
≥ 130	28(35.0)	22(14.7)	3.1(1.6 – 5.9)	< 0.001
< 130	52(65.0)	128(85.3)		
Serum HDL (mg/dl)				
< 40	25(31.3)	27(18.0)	2.1(1.1 – 3.8)	0.022
≥ 40	55(68.7)	123(82.0)		
Serum Tg (mg/dl)				
≥ 150	66(82.5)	78(52.0)	4.3(2.2 – 8.4)	< 0.001
< 150	14(17.5)	72(48.0)		

*Data were analyzed using the **Chi-squared** (χ^2) **Test**.
Figures in the parentheses denote the corresponding **percentage.**

DISCUSSION:

In the current study, smoking was found to be strongly linked to dyslipidaemia with the overall risk of having dyslipidaemia among smokers being > 5 times greater than among non-smokers. However, as the duration of smoking was correlated with different lipid profiles, it was not found to be significantly correlated with any of the lipid profiles indicating that duration of smoking does not have any significant influence on the status of lipid profile. A Chinese study, conducted on 2,160 older males, investigated the link between smoking and dyslipidemia. The study revealed that compared to non-smokers or those who had quit smoking, current smokers had considerably higher levels of TGs and lower levels of HDL-C. When the effect of smoking was adjusted for other factors, such as alcohol intake, body mass index (BMI), and age, smoking had the highest adverse impact on lipid profile and emerged to be an independent risk factor for dyslipidemia compared to other factors that affect blood lipids.[13] To determine the association of smoking with dyslipidemia in elderly males an analytical cross-sectional study was conducted at Bhitai Dental and Medical College Mirpur Khas Pakistan between March 2021-2022 divided the participants into 3 different groups – current smokers, non-smokers, & past smokers. The probable confounding factors like age, body mass index, alcohol usage, and systolic and diastolic blood pressures did not differ substantially between non-smokers, former smokers, and current smokers. The results demonstrated that non-smokers & past smokers had considerably lower levels of triglycerides and high-density lipoprotein cholesterol than current smokers (p = 0.001).[14]

Several studies conducted in Saudi Arabia observed smoking along with age, sex, higher BMI, greater waist circumference, low physical activity, and consumption of fast food, particularly margarine to be associated with dyslipidemia.[15-17] According to a meta-analysis of 54 published studies, smokers' TC, TG, and LDL-C levels increased by 3, 9.1, and 1.7%, respectively, while their HDL-C levels declined by 5.7%.[2] Very recently, a cross-sectional study conducted among Nigerian adults showed that the overall prevalence of reduced HDL-C, elevated LDL-C, hypertriglyceridaemia and hypercholesterolaemia were 72.5, 13.6, 21.4, and 7.5%, respectively. The prevalence of reduced HDL-C was significantly higher and elevated Tg was considerably higher among smokers than among non-smokers.[18]

CONCLUSION:

Summarizing the results of the present study, it can be concluded that smokers are significantly prone to being dyslipidaemic. The risk of having dyslipidaemia in smokers is several times higher than in non-smokers. While serum total cholesterol, low-density lipoprotein (LDL), and serum triglycerides are increased, serum high-density lipoprotein (HDL) is reduced.

REFERENCES:

1. Krishnaswami S, Richard J, Prasad NK, Alexander T, Thomas CS. Association between cigarette smoking nad coronary arterial disease in patients in India: How quantitative is it? An assessment by selective coronary arteriography. *Int J Cardiol* 1991;31(3):305-311.
2. Craig WY, Palomaki GE, Haddow JE. Cigarette Smoking and Serum lipid and lipoprotein concentrations: an analysis of published data. *BMJ* 1989;298:784-788.
3. Wang S, Xu L, Jonas JB, You QS, Wang YX, Yang H. Prevalence and associated factors of dyslipidemia in the adult chinese population. *PLoS One* 2011;6:e17326.
4. Lakshmi AS, Lakshmanan A, Kumar GP, Saravanan A. Effect of intensity of cigarette smoking on haematological & lipid parameters. *J Clin Diagn Res* 2014;8(7):BC11-3. doi: 10.7860/JCDR/2014/9545. 4612.
5. Garrison RJ, Kannel WB, Feinleib M, Castelli WP, McNamara PM, Padgett SJ. Cigarette smoking and HDL cholesterol: the Framingham offspring study. *Atherosclerosis* 1978;30(1):17-25. doi: 10.1016/0021-9150(78)90149-1 PMID: 209795.
6. McGill HC, Jr, McMahan CA, Malcom GT, Oalmann MC, Strong JP. Effects of serum lipoproteins and smoking on atherosclerosis in young men and women. The PDAY Research Group. Pathobiological Determinants of Atherosclerosis in Youth. *Arterioscler Thromb Vasc Biol* 1997;17:95–106.
7. Kannel WB. Some lessons in cardiovascular epidemiology from Framingham. *Am J Cardiol* 1976;37:269–282.
8. Hammond EC, Horn D. Landmark article March 15, 1958: Smoking and death rates: report on forty-four months of follow-up of 187,783 men. By E. Cuyler Hammond & Daniel Horn. *JAMA* 1984;251:2840–2853.
9. Pasupathi P, Rao YY, Farook J, Saravanan G, Bakthavathsalam G. Effect of cigarette smoking on lipids and oxidative stress biomarkers in patients with acute myocardial infarction. *Res J Med Med Sci* 2009; 4:151–159.
10. Freeman DJ, B A Griffin, E Murray, G M Lindsay, D Gaffney, C J Packard, J Shepherd. Smoking and plasma lipoproteins in man: effects on low density lipoprotein cholesterol levels and high-density lipoprotein subfraction distribution. *Eur J Clin Invest* 1993;23(10): 630-40. doi: 10.1111/j.1365-2362.1993.tb00724.x.\
11. Ludicke F, Magnette J, Baker G, Weitkunat R. A Japanese cross-sectional multicentre study of biomarkers associated with cardiovascular disease in smokers and non-smokers. *Biomarkers* 2015;20:411–421.
12. The Korean Society of Lipid and Atherosclerosis. Dyslipidemia Fact Sheets in Korea, 2018. Seoul (KR): The Korean Society of Lipid and Atherosclerosis; 2018.
13. Tan XJ, Jiao GP, Ren YJ, Gao XR, Ding Y, Wang XR, et. al. Relationship Between Smoking and Dyslipidemia in Western Chinese Elderly Males. *J Clin Lab Anal* 2008;22:159–163.
14. Kumar N, Shaikh SN, Iqbal A, Memon FR, Hussain T, Rafique S. Correlation between Smoking and Dyslipidemia in Elderly Males: An Analytical Cross-Sectional Study. *PJMHS* 2022;16(07):745-47. DOI:https://doi.org/10.53350/pjmhs22167745 [accessed Feb 22 2024]
15. Basulaiman M, El Bcheraoui C, Tuffaha M, Robinson M, Daoud F, Jaber S, et al. Hypercholesterolemia and its associated risk factors—Kingdom of Saudi Arabia, 2013. *Ann Epidemiol* 2014;24:801–808. Doi:10.1016/ j.annepidem.2014.08.001. 11.
16. Prevalence and Correlates of Dyslipidemia among Adults in Saudi Arabia: Results from a National Survey. *Open J Endocr Metab Dis* 2012;2:89. doi: 10.4236/ ojemd. 2012.24014. 12.
17. Al-Hassan Y, Fabella E, Estrella E, Aatif M. Prevalence and Determinants of Dyslipidemia: Data from a Saudi University Clinic. *Open Public Health J* 2018;11:416–424. Doi: 10.2174/1874944501811010416
18. Chori B, Danladi B, Nwakile P, Okoye I, Abdullahi U, Zawaya K. Prevalence, patterns and predictors of dyslipidaemia in Nigeria: a report from the REMAH study. *Cardiovasc J Afr* 2022;33:52–59. DOI: 10.5830/ CVJA-2021-037.