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ORIGINAL ARTICLE

Correlation between Smoking and Dyslipidemia in Elderly Males: An Analytical Cross-Sectional Study

NARINDAR KUMAR[1], SHAFAQ NAZIA SHAIKH[2], ATIF IQBAL[3], FAIMA RANI MEMON[4], TAZEEM HUSSAIN[5], SAIMA RAFIQUE[6]
[1]*Associate Professor Medicine, Bhitai Dental and Medical College Mirpurkhas Pakistan*
[2]*Assistant Professor Medicine, Liaquat University of Medical and Health Sciences Jamshoro Pakistan*
[3]*Practitioner Family Medicine, Ministry of Health United Arab Emirates*
[4]*Lecturer Department of Physiology, BMC/Liaquat University of Medical and Health Sciences Jamshoro Pakistan*
[5]*Acting Consultant Medicine / Supervisor Family Medicine Academy & Family Medicine Department. King Saud Medical City Riyadh, Kingdom of Saudi Arabia*
[6]*Senior Registrar Medicine, Bhitai Dental and Medical College Mirpurkhas Pakistan*
Correspondence to: Narindar Kumar, Email: narindersitani@gmail.com

ABSTRACT

Aim: To investigate the association between smoking and dyslipidemia in elderly males while accounting for age, BMI, and alcohol use.

Study design: Analytical cross-sectional study

Place and Duration: This study was conducted at Bhitai Dental and Medical College Mirpurkhas from march 2021 to march 2022

Methodology: The research comprised 2,160 participants who were divided into 3 different groups: current smokers, non-smokers, & past smokers. The research excluded those who smoked just sometimes (one cigarette per day). This study separated smokers into four categories: light (26.7 pack-years), medium (426.7-90.5 pack-years), heavy (440.5-75.5 pack-years), and extremely heavy (455.5-165.5 pack-years).On the same day, a survey, physical examination, and laboratory study of blood samples were used to evaluate the study population. The person was assessed while wearing light clothes and no shoes, and anthropometric measures comprised height and body weight. Weight was divided by height squared (kg/m2) to get the BMI. Using established protocols in the clinical laboratory, an automated biochemistry analyzer measured the blood levels of total cholesterol, triglycerides, high-density lipid cholesterol, low- density lipid cholesterol, Apo protein B, and Lipoprotein.

Result: When compared to present smokers, the levels of triglycerides and high-density lipid cholesterol among non-smokers and former smokers were statistically significantly different (P=0.001). Other variables, such as Age, body mass index, use of liquor, systolic and diastolic blood pressure, did not significantly differ between non-, past, and present smokers. In comparison to light smokers, moderate, heavy, and very heavy smokers more commonly displayed abnormal levels of TGs, Apo-B, and TC, while very heavy smokers less frequently displayed abnormal levels of low-density lipid cholesterol. It was unexpected to find that among very heavy smokers, aberrant Apo-B levels were seen more frequently than low-density lipid cholesterol levels. The prevalence of abnormal HDL-C and lipoprotein (a) was higher among light smokers. After accounting for alcohol, BMI, and age, smoking remained a significant independent threat fact for dyslipidemia (P = 0.032).

Conclusion: The study revealed that smoking caused TGs and apoB levels to rise in elderly males, and HDL-C levels to decrease, making it an independent risk factor for dyslipidemia.

Keywords: smoking, dyslipidemia, elderly, males

INTRODUCTION

Dyslipidemia is the medical term for the condition where the blood has abnormally high levels of lipids. Triglycerides, LDL-C, and total cholesterol concentrations all rise in this syndrome, whereas high-density lipoprotein cholesterol levels fall. There are a variety of factors that might affect dyslipidemia, such as your age, whether or not you smoke, your body mass index (BMI), how much alcohol you drink, and your lifestyle. (1) Smoking causes dyslipidemia and endothelial damage, which speed up the development of atherosclerosis, cardiovascular disease, and stroke. These are the potential processes. Smoking's negative effects on health and the resulting medical costs are quite concerning. (2, 3)In China, cigarette smoking has already had a considerable negative impact on public health, with more than 1 million people dying each year from smoking-related diseases. (4)It has not been shown that smoking affects the levels of different blood lipids. According to several research, smokers had greater degrees of triglycerides, TC, and low–density lipid cholesterol and low levels of high–density lipid cholesterol than nonsmokers (1). According to other studies, smoking can increase TG levels while lowering TC, LDL cholesterol, and HDL cholesterol. (5, 6) These studies cannot conclusively demonstrate how smoking affects TC, LDL-C, Apo protein B, and lipoprotein, even while they consistently demonstrate that smoking raises TG levels and lowers HDL-C. The majority of research has targeted adolescents, young adults, and adults through cross-sectional or longitudinal studies involving chosen groups. To the best of our knowledge, there have not been many studies that just looked at elderly men. One research focused on the connection between smoking and dyslipidemia in those above 50 years of age. (5)Another research examined the connection between smoking and dyslipidemia in those over 65 years of age , however it was unclear how smoking history and dyslipidemia related to one another (6) Most research have also not taken into account the effects of alcohol use. The current research aims to investigate the association between smoking and dyslipidemia in elderly men while accounting for age, BMI, and alcohol use. The decision to concentrate on elderly men in this study was made owing to the dearth of prior published studies in this age group as well as the fact that male smoking is far more prevalent than female smoking. Future moves of prevention, education, and therapeutic involvement for elderly male smokers will have a stronger foundation if the link between smoking and blood lipids is understood.

METHODOLOGY

A sample of 2,160 persons over 65 years of age was used for this analytical cross-sectional study. Those individuals who were smoking occasionally or those taking medications of dyslipidemia were excluded from the study. All members provided informed permission and the Institutional Review Board of the hospital authorized this research.

On the same day, a Questionnaire, physical examination and laboratory analysis of blood samples were used to evaluate the study population. The person was assessed while wearing light clothes and no shoes, and anthropometric measures comprised height and body weight. Weight was divided by height squared (kg/m2) to get the BMI. Using venipuncture, fasting blood samples were taken. Using established protocols in the clinical laboratory, an automated biochemistry analyzer measured the blood levels of total cholesterol, triglycerides, high-density lipid cholesterol, low-density lipid cholesterol, Apo protein B, and Lipoprotein (a). The reference ranges for triglycerides at the laboratory were 0.34-1.70

mmol/L, 2.80-5.70 mmol/L, and 0.60-1.60 mmol/L for HDL-C, 1.07-3.10 mmol/L for LDL-C, 0.60-1.10 g/L for apoB, and 0-300 mg/L for lipoprotein (a). For LDL-C, the laboratory's reference ranges were 1.07-3.10 mmol. If the readings for of total cholesterol, triglycerides, low- density lipid cholesterol, Apo protein B, and Lipoprotein (a) were greater than the mention range and the readings for high-density lipid cholesterol were lower than the reference range, dyslipidemia wasdiagnosed. The lab took part in a nationwide quality control programme.

Two criteria were used to evaluate the subjects' alcohol consumption: average frequency (days/week) and average quantity (ml). The following were the alcohol content calculation techniques. White wine had an average alcohol content of 56 percent, wine 12.5%, and beer 4%. The average daily alcohol consumption for each individual was then determined (ethanol equivalent [g/day]).

Participants self-reported whether they were: 1) nonsmokers,never smoked; 2) former - smokers, those who stop smoking of last 6 months; or 3) current smokers, smoke at least one cigarette per day. Applicants were questioned about their current smoking behaviors as well as their smoking history. The research excluded those who smoked just sometimes (one cigarette per day). To determine a person's pack-years, a degree of smoking experience that takes into consideration both the quantity smoked and the length of time they have been a smoker, the number of packs smoked on a daily basis was multiplied by the number of years they have been a smoker. Light smokers had an average of 26.7 pack-years of experience, medium smokers had 426.7 pack-years of experience, heavy smokers had 440.5 pack-years of experience, and very heavy smokers had 455.5 pack-years of experience. (7)

Statistical Analysis: Analysis of variance was used to compare factors between nonsmokers, former smokers, and current smokers (ANOVA). Using the w2 -test, the Odds Ratio and 95% Confidence Interval were obtained. Statistics were judged significant at a P value of< 0.05. Means and SD are used to display data. The influence of the factors on blood lipids was evaluated using multiple linear regression analysis. To ascertain if smoking was an independent risk factor and to manage possible impacts, logistic regression analysis was utilized. Version 20.0 of SPSS for Windows was used for all of these analyses.

RESULTS

A total of 3,411 individuals were assessed, and 2,160 persons eligible to participate in the research. A total of 613 female respondents, 112 people who sometimes smoked and 526 applicants who were taking medicine for dyslipidemia were eliminated from the study. Table 1 displays the baseline parameters of the research population by smoking status. When compared to present smokers, the levels of triglycerides and high-density lipid cholesterol among non-smokers and former smokers were statistically significantly different (P = 0.001). Non-smokers, former-smokers and current smokers did not vary significantly on any other variable, including age, BMI, alcohol consumption, or systolic and diastolic blood pressure. Table 2 shows the Odds Ratio of dyslipidemia according to smoking experience level. In comparison to light smokers, medium, heavy, and very heavy smokers more commonly displayed abnormal levels of TGs, apoB, and TC, while very heavy smokers less frequently displayed abnormal levels of low-density lipid cholesterol. It was unexpected to find that among very heavy smokers, aberrant apoB levels were more commonly seen than low-density lipid cholesterol levels. The prevalence of abnormal HDL-C and lipoprotein (a) levels was higher among light smokers. The multiple linear regression's standardized coefficients for dyslipidemia are shown in Table 3. Smoking, alcohol use, BMI, and age were shown to have varying degrees of effect on dyslipidemia, with smoking having the most impact. The results of Table 4 demonstrate that smoking is an independent risk factor for dyslipidemia (P = 0.032) after adjusting for alcohol intake, BMI, and age.

Table 1: baseline parameters of the research population by smoking status

	Current smokers	Past smokers	Non-smokers
TGs	2.97	1.32	1.30
TC	4.87	4.84	4.82
HDL-C	0.95	1.26	1.24
LDL-C	2.74	2.69	2.80
ApoB	1.03	0.96	1.01
LP	282.4	317..86	373.34

Table 2: Odds Ratio of dyslipidemia according to smoking experience level.

	Light	Medium	Heavy	Very heavy
TGs	1	1.286	2.571	5.571
TC	1	1.333	1.250	0.667
HDL-C	1	0.857	0.857	0.75
LDL-C	1	4	3	0.66
ApoB	1	3	5	24
LP	1	0.5	0.222	0.18

Table 3: multiple linear regression's standardized coefficients for dyslipidemia

	Standard coefficients
Age	-0.031
BMI	-0.032
Smoking	0.219
Alcohol intake	0.137

Table 4: smoking status after adjusting for alcohol intake, BMI, and age

	B	p-value
Age	0.015	0.090
BMI	-0.048	0.120
Smoking	0.84	0.032
Alcohol intake	0.083	0.150

DISCUSSIONS

Although few research have been conducted in elderly smokers, several studies have shown that smoking may increase levels of triglycerides, lower levels of high-density lipid cholesterol have an uncertain effect on total cholesterol, low-density lipid cholesterol, Apo protein B, and lipoprotein (a)(8). However, the current extensive research of the elderly male smoker population suggests that smoking might result in dyslipidemia. In comparison to nonsmokers, smokers had higher levels of total cholesterol (3%), triglycerides (9.1%), and low-density lipid cholesterol (1.7%), as well as lower levels of high-density lipid cholesterol (5.7%), according to Craig et al (1) meta-analysis of 54 published studies. According to some research, smoking raises TG levels while lowering total cholesterol, low density lipid cholesterol, and high-density lipid cholesterol levels. (6) According to prior research, smoking has a dose-dependent influence on blood lipid levels, while alcohol intake and BMI also affect serum lipid levels. (9) However, the confounding effects of age, BMI, alcohol use, and smoking history have often not been taken into account in this research, nor have they been accounted for. It is also unknown if the findings from earlier research can be generalized to elderly male smokers since elderly male smokers have not been investigated separately. Current smokers were more possibly to have aberrant triglycerides and high-density lipid cholesterol values, according to the current research. This is in line with the majority of previous research, showing that this impact is widespread across the populations that have been examined and may be the primary mechanism through which smoking promotes Atherosclerosis, Cardiovascular disease and Stroke.The levels of total cholesterol, low-density lipid cholesterol, Apo-protein B and Lipoprotein (a) among non-smokers, former smokers, and current smokers did not show any significant differences. In the meta-analysis led by Craig et al., it was shown that smokers had high levels of total cholesterol and low-density lipid cholesterol. (1)linked to nonsmokers, but Kuzuya et al. (6) found that this was not the case. In line with the findings of the present investigation, Yasue et al.(8)hypothesized that there were no appreciable changes in total cholesterol and low-density lipid cholesterol levels between none, former, and current smokers, and Current smokers

have higher apoB levels than non- and ex-smokers. Five possible reasons are offered. 1) Ethnicity; 2) Age and Gender; 3) food, lifestyle and Public health awareness; 4) Age, BMI, and Alcohol intake; and 5) other factors.According to Craig et al.(1), Light, moderate, and heavy smokers had a dose-response influence on TC (1.8, 4.3, & 4.5%), TGs (10.7, 11.5, & 18.0%), LDL-C (-1.1, 1.4, & 11.0%), and HDL-C (-4.6, 6.3, & -8.9%)(1). There was an association between dyslipidemia and smoking exposure in the current study, which was divided into 4 categories: light, medium, moderate, and extremely heavy. However, there were no definite pattern. The most prevalent abnormalities were abnormal TG and apoB levels, abnormal high-density lipid cholesterol and lipoprotein (a) levels, abnormal total cholesterol and LDL-C levels, and the least frequent abnormalities were abnormal triglycerides and Apo protein B levels in extremely heavy smokers. It is interesting that very heavy smokers were more likely to have increased apoB levels than normal LDL-C. This shows that even while very heavy smokers had the highest risk of vascular disease, physicians frequently observed that their LDL-C levels were normal in these patients. It might be due to an increase in apoB atherogenic particles. One molecule of apoB is present in every VLDL, low-density lipoprotein and LDL particle, making plasma apoB equal to the total number of atherogenic particles, of which low density lipoprotein particles account for 490 percent. (10)Recent large-scale prospective epidemiological studies have shown that Apo protein B predicts vascular disease risk more accurately than LDL-C. (11). Cross-sectional studies have shown that hypertriglyceridemia does not enhance the risk of coronary disease if the apoB level is normal. However, hypertriglyceridemia that is associated with a high apoB level does increase the risk of coronary disease. (12) In the prospective epidemiologic Quebec cardiovascular study, it was found that the risk of coronary disease was three times higher in hypertriglyceridemia with increase Apo protein B level than it was in hypertriglyceridemia with a normal apoB level. This was found to be the case when comparing the two types of hypertriglyceridemia. There is a wide range of sizes and densities that may be found in LDL particles, with some being much bigger and denser than others. (13)Tiny, dense low density lipid particles enhance the risk for Vascular disease, according to multiple cross-sectional investigations(14), and other prospective epidemiologic studies have observed a link between the occurrence of small, dense low density lipid particles and poor clinical outcomes. (15)

Additionally, prospective double-blinded studies have examined how different lipid-lowering medications affect the rates at which coronary disease advances as measured by angiography. (16)All of them demonstrated that treatment improved results, and among other factors, the advantage was connected to a reduction in the amount of micro, dense LDL particles. The atherogenic particle count is reliably estimated by measuring apoB, which enables correct diagnosis and evaluation of the therapeutic response. Low density lipid cholesterol is frequently used to determine the likelihood of vascular disease and the effectiveness of treatment, while apoB is now employed in clinical practice very seldom. Our results provide more evidence for the necessity for a reevaluation in that area.

CONCLUSION

According to our study, age, BMI, smoking, alcohol, and BMI had the most and smallest impacts on blood lipid levels. In conclusion, smoking was shown to be a distinct risk factor for dyslipidemia, promoting the blood levels of TGs and apoB to be elevated and HDL-C to be decreased in elderly males.

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