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Impact of Tobacco Use on Lipid Metabolism in Adult Females: A Study from Erbil-Kurdistan Region of Iraq

Mohammed Merza[1, *4], Huda Jabbar Jawhar[2], Azheen Sedeeq Ahmed[1], Roshna Hiwa Hady[1], Rundk Hwaiz[4]

[1]Department of Clinical Analysis, College of Pharmacy, Hawler Medical University, Erbil,

[2]Department of Pharmacy, College of Pharmacy, Knowledge University, Erbil 44001, Iraq

[3]Department of Pharmacy, College of Pharmacy, Knowledge University, Erbil, Iraq

[4]Nutrition and Dietetics Department, Tishk International University, Erbil, Iraq.

* Corresponding Author:

MSc, PhD, Department of Clinical Analysis, College of Pharmacy, Hawler Medical University, Erbil, Kurdistan, 100M street Erbil.

Email ID: mohammed.merza@hmu.edu.krd

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ABSTRACT

Background: Limited studies have explored the relationship between smoking and lipid profiles in adult female smokers.

Materials and Methods: This study aimed to assess and compare the serum lipid profiles of adult female smokers with those of non-smoking females (control group). Serum lipid levels were measured in 200 female participants.

Results: Among the participants, 110 were smokers and 70 were non-smokers (controls), aged between 25 and 50 years. Only smokers with a history of smoking for more than five years were included. The findings showed that the mean serum levels of total cholesterol (365.2 ± 42.2 mg/dl), triacylglycerol (206.4 ± 40.6 mg/dl), very low-density lipoprotein (42.2 ± 14.2 mg/dl), and low-density lipoprotein (146.2 ± 8.81 mg/dl) were significantly higher in female smokers compared to non-smokers, whose mean levels were total cholesterol (182.0 ± 28.2 mg/dl), very low-density lipoprotein (18.8 ± 8.2 mg/dl), triacylglycerol (96 ± 6.4 mg/dl), and low-density lipoprotein (84.54 ± 8.2 mg/dl). Conversely, the mean serum level of high-density lipoprotein cholesterol was lower in chronic female smokers (36.6 ± 2.6 mg/dl) compared to non-smokers (65.3 ± 8.0 mg/dl).

Conclusion: The findings suggest that cigarette smoking in females leads to dyslipidemia, which increases the risk of cardiovascular disease.

Keywords: *Cholesterol, LDL, HDL, Smoking*

1. INTRODUCTION

Changes in lipid profiles are a significant risk factor for cardiovascular diseases, especially with the rise in sedentary lifestyles and smoking [1]. Smoking can disrupt the normal plasma lipoprotein composition. Smoking is a well-established risk factor for myocardial infarction, as it affects various biological processes that accelerate atherogenesis. Research has confirmed its association with active smoking [2]. There is a strong correlation between smoking and cardiovascular disease, with studies indicating that smokers tend to have higher levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), while high-density lipoprotein cholesterol (HDL-C) levels are reduced [3]. Furthermore, other studies have shown that total cholesterol and triglyceride levels decline after smoking cessation [4].

The precise mechanism by which smoking increases cardiovascular risk remains unclear. However, it is believed that the presence of approximately 5,000 harmful and carcinogenic chemicals in tobacco plays a significant role in smoking-related diseases [5]. Some of the most common health conditions linked to smoking include cardiovascular disease, chronic

obstructive pulmonary disease (COPD), and lung cancer [6]. Nicotine, the primary active component in cigarette smoke, is an alkaloid derived from tobacco leaves that stimulates the body's physiological responses [7]. Cigarette smoking influences lipid metabolism, increasing the risk of coronary heart disease (CHD) and atherosclerosis [5].

Several mechanisms contribute to smoking-induced lipid alterations, particularly due to nicotine. Nicotine stimulates the secretion of hepatic free fatty acids and triglycerides into the bloodstream, along with very-low-density lipoprotein cholesterol (VLDL-C), by enhancing catecholamine secretion. This, in turn, activates the sympathetic adrenal system, leading to increased lipolysis [8]. Additionally, smoking has been associated with elevated homocysteine levels, which contribute to oxidative modifications in LDL-C and a reduction in HDL-C. Studies suggest that homocysteine inhibits Apolipoprotein A1 (Apo A-I), a crucial protein component of HDL-C in plasma, further impacting lipid balance [8].

Nicotine elevates the levels of harmful lipids such as total cholesterol, LDL-C, and triglycerides, while simultaneously reducing beneficial lipids like HDL-C [9]. Moreover, nicotine induces oxidative stress by generating free radicals, which damage membrane lipids, leading to the formation of malondialdehyde (MDA). This oxidative damage can result in peroxidative injury to body tissues [10]. Lipoprotein oxidation is thought to occur in the bloodstream, generating reactive species such as superoxide radicals, hydrogen peroxide, and lipid peroxides, all of which contribute to LDL oxidation [10]. However, there is ongoing debate regarding which components of the lipid profile are most affected by cigarette smoking and whether these alterations occur directly or indirectly.

In this study, we aim to investigate and analyze the effects of smoking on the lipid profile of females in Erbil, Kurdistan Region of Iraq.

2. MATERIALS AND METHODS

This study was conducted in Erbil city, Kurdistan Region of Iraq, involving a total of 200 female participants. Among them, 100 were lifelong smokers who had been smoking for more than seven years, aged between 20 and 60 years, and classified as **Group 1**. The control group (**Group 2**) consisted of 100 non-smoking females, closely matched in age and weight with the participants in Group A. The control group comprised healthy individuals with no history of smoking, ensuring comparability with the smokers.

Blood samples were collected after 8–10 hours of fasting. Each sample was transferred to plastic centrifuge tubes and transported to the Rizgari Teaching Hospital laboratory for analysis. The following exclusion criteria were applied to both groups:

1. Former smokers
2. Hepatic dysfunction
3. Endocrine disorders
4. Obesity
5. Renal disease
6. Alcohol consumption
7. Family history of heart disease or previous cardiovascular conditions
8. Hypertension
9. Diabetes mellitus
10. Use of lipid-lowering or other relevant medications

The collected blood serum was centrifuged, aliquoted, and stored at -20°C until further analysis. Lipid profile parameters, including total cholesterol (TC), triglycerides (TG), LDL cholesterol (LDL-C), and HDL cholesterol (HDL-C), were measured using the Cobas 400 Integra colorimetric method. The reference ranges for lipid profile levels were:

- Total cholesterol: Normal (<200 mg/dl), borderline high (200–250 mg/dl), high (>250 mg/dl)
- Triglycerides (TG): Normal (<150 mg/dl), based on Wahlefeld's method
- LDL-C: Normal (<150 mg/dl), measured through cholesterol ester hydrolysis
- HDL-C: Normal range for females (34–52 mg/dl)

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 20. Data were expressed as mean ± standard deviation (SD). A t-test was used for statistical evaluations, and a p-value ≤ 0.05 was considered statistically significant. The difference in mean total cholesterol, TG, HDL, and LDL levels between the two groups was analyzed using Student's t-test.

Results

As presented in Table 1, female smokers exhibited significantly higher levels of total cholesterol (TC), triglycerides (TG), very-low-density lipoprotein cholesterol (VLDL-C), and low-density lipoprotein cholesterol (LDL-C) compared to the control group. Conversely, the HDL-C levels in Group A were notably lower than those observed in Group B.

Table 1: Parameters for the lipid profiles

Lipid profile mg/dl	**Smokers Group A Mean ± SD**	**Non-Smokers Group B Mean ± SD**	***P*-value**
TC	365.2 ± 42.2	182.0 ± 28.2	<0.05
TG	206 ± 40.6	96 ± 6.4	<0.05
VLDL-Cholesterol	42.2 ± 14.2	18.8 ± 8.2	<0.05
LDL-Cholesterol	146.2 ± 8.8	84.54 ± 8.2	<0.05
HDL-Cholesterol	36.6 ± 2.6	65.3 ± 8.0	<0.05

3. DISCUSSION

The rate of CHD in female smokers were higher compared to non-female smokers in Erbil city. This might due to cigarette will leads to increase the levels of lipoprotein. According to this rate, we evaluated the levels of lipoprotein among female smokers with non-smokers in different age. In general, in cigarette smokers the risk of CHD is higher than in non-smokers and can be interpreted by different interactions among degradation of the integrity of arterial walls (AW), blood lipid, lipoprotein derangements and blood coagulation. The result data of lipid profiles such TC, TG, VLDL-C, LDL-C levels in smokers of group compared were higher than to non-smokers in group B with *p* values as shown in Table 1. It may because of nicotines, the nicotine leads to secrete high levels of hepatic free fatty acids (HFFA) and TC along with VLDL-C in the blood stream by increasing the secretion of catecholamines. This cateholamines then stimulating sympathetic adrenal system followed by in increased lipolysis. Or the spike in lipids in female smokers may be because of consuming tobacco induces the absorption of nicotine into the bloodstream. This absorption may induces lipolysis. Then free fatty acids will pass through into the bloodstream via activation of adenyl cyclase in the body tissue and then nicotine will induces catecholamine secretion [11]. It is important to mention that elevated free fatty acids in the liver contribute to decreased hepatic TG and VLDL synthesis and it may raising the blood production of TG and VLDL-C [12]. Our data has also shown a significant reduction in HDLC levels (p <0.05) in female smokers compared to non-female smokers (Table 1). In general, HDL biosynthesis involves in synthesis of A-I (apoA-1) followed by the acquisition of liver cholesterol and phospholipids, forming nascent HDL particles. Smoking cigarettes can decrease the apoA-I levels. Lack of ApoA-I results in defective synthesis of HDLs. Smoking cessation regained attention at apoA-I. This data is in line with many studies that have reported high homocysteine plasma levels in smokers [13,14], and stated that in plasma homocysteine negatively associated with HDL-C and Apo A-I. Increased levels of homocysteine may result in decreased levels of HDL-C via multiple mechanisms as described by Huang F., et al. 2016 [13]. Genetic study to evaluate the influence of blood lipid levels on the risk CHD are more recommended.

4. CONCLUSION

Smoking-induced can increased release of catecholamine may also justify further reduction of HDL-C in lifelong smokers, resulting in decreased VLDL-C and reduced concentrations of HDL-C. However, our data clearly showed a very strong relationship between smoking and lipid profile increase. The risk of an increase in serum cholesterol in female smokers with an increase in LDLC is associated with coronary heart disease.

CONFLICT OF INTEREST:

The authors state no conflict of interest.

AUTHOR CONTRIBUTIONS

All authors contributed equally in this study.

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