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Original Article

Effect of Smoking on Low-Density Lipoproteins Level in Human

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Abstract

Smoking destroys the vascular system, increases plaque deposits in atherosclerosis, and increases inflammation. The present study was performed to determine the effect of smoking on the levels of low-density lipoprotein and very low-density lipoprotein in smokers. A cross-control study was carried out in the outpatient clinic in Baghdad, Iraq. The study was carried out 60 by individuals from February 2021 to July 2021. Participants in this study included adult smokers and non-smokers of both genders, and the levels of LDL and VLDL were determined using an Automatic Chemistry Analyze. The results revealed that the total number of smokers was 60 individuals from both genders; there was a significant difference in mean low-density lipoprotein and very low-density lipoprotein levels. There is a significant difference in LDL Values between non-smokers and smokers (129.853±7.880; $P<0.05$). Non-smokers showed lower LDL values (104.460±7.950; $P<0.05$). Regarding VLDL values, results revealed that smokers were showing higher values than a non-smoker (49.641±4.02), (28.986±1.676) respectively, $P<0.05$). LDL and VLDL levels are more prevalent in current cigarette smokers than in non-smokers. Heavy smokers have higher LDL and VLDL values for a cigarette than non-smokers, which is consistent with observations in other populations.

Keywords: Lipid profile, LDL, VLDL, Cigarette smoking

1. Introduction

Smoking is an essential factor in the development of cardiovascular disease in more than 30% of the world's population (1). Smoking may significantly affect changes in serum lipids in ordinary people (2). Compared to non-smokers, smokers have significantly higher levels of low-density lipoprotein (LDL-C) cholesterol. (3). However, after controlling for smoking, the risk of heart disease and serum lipids varied slightly (4). LDL-C is a lipoprotein that transfer lipids through the blood and deposits in the walls of the arteries. It is also required to regulate cellular activity. Because cholesterol is highly insoluble, it must be bound to hydrophilic proteins in the bloodstream (5). The outer layer of LDL particles comprises cholesterol glucosides, while the inner core comprises cholesterol fatty acid esters. The envelope consists of one copy of the hydrophobic apo-B protein, which mediates the attachment of Low - density lipoproteins to specific receptor molecules (6). Lipoproteins, including the apolipoprotein B protein, are arteriosclerosis that disease finds when damage occurs within arteries. In people with low plasma LDL-C levels, the remaining cardiovascular damage may be induced by triglyceride-rich lipoproteins such as very-low-density lipoproteins (VLDL) (7). According to the studies, their oxidatively modified low-density lipoprotein (LDL) is taken up by macrophages in

culture, causing foam cells to develop and aggravating the atherosclerotic process (8). Furthermore, the consequences of raised cholesterol levels and changes in LDL and VLDL among cigarette smokers have already been documented (9). Smoking cigarettes has also been linked to minor but highly significant increases in low-density lipoprotein cholesterol levels (10). Smoking cigarettes induces a rise in LDL cholesterol and an elevation in VLDL cholesterol, resulting in a fat buildup in the artery wall. Because of this, smokers have a higher chance of atherosclerosis than non-smokers (11). Given that there is no updated local data to contradict this hypothesis in Iraq, the effect of smoking on the level of low-density lipoproteins in humans was evaluated in this study.

2. Materials and Methods

2.1. Study Population

Sixty adults of both genders within the age group 20-50 years old were evaluated in this study; the study was extended from February 2021 to July 2021.

The study group included smokers who smoked 10 to 25 cigarettes daily, and the control group included non-smokers. Patients with other diseases such as diabetes, liver disease, and pancreatic disorders were excluded from the study to avoid the effect on lipid factors.

Interviews were conducted with those present at the Medical City Outpatient Clinic. Written consent was obtained from each individual to participate in this study. The participants' demographic information was taken via a personal questionnaire.

2.2. Blood Sampling and Biochemical Assays

After an overnight fast of roughly fourteen hours, five ml of blood samples were taken, and the serum was pooled by centrifuge at 3000 rpm (800-1Centrifugal Machine, China) for 15 minutes. The lipoprotein profiles in the serum were investigated, and the levels of LDL and VLDL were determined using an Automatic Chemistry Analyzer and the standard homogeneous enzymatic approach (Linear), with the Linear Company providing the calibration and internal controls (EMC LAB, Germany).

2.3. Statistical Analysis

Excel and Statistics (SPSS version 18) were used for data analysis. Continuous data were expressed using the mean and standard deviation. The Student's t-test was used to examine the differences in LDL and VLDL concentration measured by the direct method and other formulae between smokers and non-smokers. (P<0.05) was measured statistically significant.

3. Results

The study's findings demonstrated that there was a substantial variation in LDL and VLDL levels between cigarette non-smokers and smokers, with cigarette smokers having much higher LDL (129.853±7.880; P<0.05) than non-smokers (104.460±7.950; P<0.05) (Figure 1).

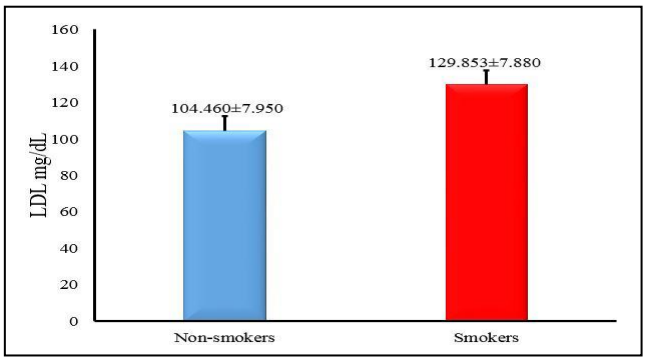

Figure 1. LDL levels in serum of cigarette non-smokers and smokers

As well as, the level of VLDL was showed up-regulated in cigarette smokers (49.640±4.029; $P<0.05$) compared to non-cigarette smokers (28.986±1.676; P<0.05) as in figure 2.

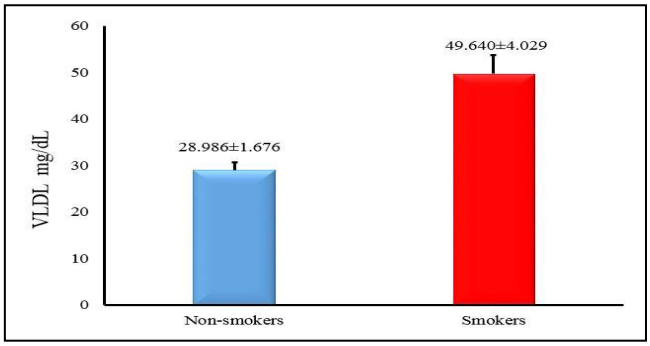

Figure 2. VLDL levels in serum of cigarette non-smokers and smokers

4. Discussion

Smoking has been described as one of the main factors of cardiovascular diseases and is associated with changes in the regular pattern of plasma lipoproteins (12). It is found in cigarettes containing nicotine, which significantly affects lipid levels in the blood (3).The predominant cholesterol carriers in circulation are low-density lipoprotein (LDL) particles, which have the physiological role of transporting cholesterol to cells. Inhibition in the function of LDL receptors leads to hypercholesterolemia. The liver produces very low-density lipoprotein (VLDL) (3). Jain and Ducatman (13), through their research on the relationship between serum lipids and continuous smoking, found that people who smoke have higher levels of LDL and VLDL; this was consistent with the results of our research. Also, the present study's findings were consistent with the results of Gossett, Johnson (14), who found that LDL and VLDL levels increased. As a result, LDL and VLDL levels are higher in current smokers than in non-smokers. Heavy smokers have higher LDL and VLDL values for a cigarette than non-smokers, which is consistent with observations in other studies (10, 11, 13). One factor that increases LDL and VLDL in the blood serum of smokers is increased secretion of catecholamines and, as a result, increased free fatty acids, which may increase the concentration of VLDL and LDL (9). Smoking also decreased lecithin-cholesterol acyl transferase, which is responsible for esterifying free cholesterol and increasing HDL, and may also lower cholesterol ester transfer protein levels (15). According to the findings of studies, community education must be oriented that smoking is a risk factor for community health through induction and acceleration of hyperlipidemia so that atherosclerosis and its complications are the risky output of smoking, particularly in heavy smokers.

Authors' Contribution

Study concept and design: E. H. A.
Acquisition of data: E. H. A.
Analysis and interpretation of data: K. H. A. A.
Drafting of the manuscript: K. H. A. A.
Critical revision of the manuscript for important intellectual content: E. H. A. and A. H. A.
Statistical analysis: E. H. A.
Administrative, technical, and material support:

Ethics

This study comprised experiment protocols permitted by the Ethical Committee of the Kufa University of Iraq.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

1. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52.
2. Śliwińska-Mossoń M, Mihułka E, Milnerowicz H. Assessment of lipid profile in non-smoking and smoking young health persons. Prz Lek. 2014;71(11):585-7.
3. Rao Ch S, Subash YE. The effect of chronic tobacco smoking and chewing on the lipid profile. J Clin Diagn Res. 2013;7(1):31-4.
4. Selya AS, Hesse ND. Time to first cigarette and serum cholesterol levels. Soc Sci Med. 2017;174:213-9.
5. Hevonoja T, Pentikäinen MO, Hyvönen MT, Kovanen PT, Ala-Korpela M. Structure of low density lipoprotein (LDL) particles: Basis for understanding molecular changes in modified LDL. Biochim Biophys Acta Mol Cell Biol Lipids. 2000;1488(3):189-210.
6. Heidemann BE, Koopal C, Bots ML, Asselbergs FW, Westerink J, Visseren FLJ. The relation between VLDL-cholesterol and risk of cardiovascular events in patients with manifest cardiovascular disease. Int J Cardiol. 2021;322:251-7.
7. Meenakshisundaram R, Rajendiran C, Thirumalaikolundusubramanian P. Lipid and lipoprotein profiles among middle aged male smokers: a study from southern India. Tob Induc Dis. 2010;8:11.

8. Venkatesan A, Hemalatha A, Bobby Z, Selvaraj N, Sathiyapriya V. Effect of smoking on lipid profile and lipid peroxidation in normal subjects. Indian journal of physiology and pharmacology. 2006;50(3):273-8.
9. Chelland Campbell S, Moffatt RJ, Stamford BA. Smoking and smoking cessation -- the relationship between cardiovascular disease and lipoprotein metabolism: a review. Atherosclerosis. 2008;201(2):225-35.
10. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. Am Heart J. 2011;161(1):145-51.
11. Hallit S, Zoghbi M, Hallit R, Youssef L, Costantine R, Kheir N, et al. Effect of exclusive cigarette smoking and in combination with waterpipe smoking on lipoproteins. J Epidemiol Glob Health. 2017;7(4):269-75.
12. Krupski WC. The peripheral vascular consequences of smoking. Ann Vasc Surg. 1991;5(3):291-304.
13. Jain RB, Ducatman A. Associations between smoking and lipid/lipoprotein concentrations among US adults aged >/=20 years. J Circ Biomark. 2018;7:1849454418779310.
14. Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking intensity and lipoprotein abnormalities in active smokers. J Clin Lipidol. 2009;3(6):372-8.
15. Freeman DJ, Griffin BA, Murray E, Lindsay GM, Gaffney D, Packard CJ, et al. 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.