Original Article

Evaluation The Effect Of Smoking On Some Biomarkers In Human In Baghdad City

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Abstract

Introduction: Cholesterol is a fatty molecule required by the body for the formation of cell membranes. It is produced by the liver and can also be Meat and eggs are examples of animal goods. A cigarette is a considerable risk factor for vascular blood system and respiratory disorders, as well as it is extremely addictive. When compared to non-tobacco users, tobacco users have a considerable rise in total cholesterol levels. AS well as smoking cigarettes is one of the most common causes of death worldwide. The effects of smoking on haematological markers are acute and chronic and impact biological parameters (kidney and liver) functions. The study involved fifty smokers in Baghdad city who smoked between 15 and 25 cigarettes daily. The group consists of 50 smokers between the ages of 18 and 55, as well as 50 non-smokers in about the same age range for combined analysis.

Aim: The study's goal is to explain the impact of cigarette smoking on total cholesterol levels compared to a non-smoker and also the study looked at the impact of cigarette smoking on several factors that are thought to be indicators of serious health problems in the blood parameters human body

Materials and Methods: A cross-control study was carried out in the outpatient Clinic in Baghdad, Iraq. This outpatient facility is the city's major general center, where a considerably large population is attending. The study was carried out from October 2021 to April 2022. Participants in this study included adult smokers and non-smokers of both genders, serum specimens were pooled from blood specimens from each individual. Each serum specimen was investigated for cholesterol content following, liver functions, and kidney functions [1] and This study included adult smokers and non-smokers, both males and females, who had their whole blood cell count examined by a CELL-DYN 3700 completely automatic haematological analyzer.

Results: Results revealed that there was a large difference in the average total cholesterol, Non-smokers and smokers have different values. In exclusive cigarette users, daily cigarette smoking total cholesterol levels (271.466 mg/dl). None smoker individual's sera were showing normal cholesterol values. As well as Hemoglobin (Hb, 16.445 g/dl), packed cell volume (PCV, 59.489 %), and platelets (PLT, 219.127 $x10^{12}$/L) all showed substantial increases in the study compared to non-smoking. About serum biochemical measures, similar results were seen in kidney function (urea, 56.240 mg/dl; creatinine, 2.361 mg/dl) as well as liver function (alanine aminotransferase (ALT) 140.890 U/l; aspartate transaminase (AST) 145.291U/l; alkaline phosphatase (ALP) 210.2000U/l).

Conclusion: Cholesterol levels are upregulation in current smokers of a cigarette compared to non-smokers, which is similar to findings in other populations. Furthermore, current cigarette smokers have higher levels of bad total cholesterol, which is linked to adverse cardiovascular consequences than non-smokers. Smoking influences blood and biochemical markers, according to our findings. The bulk of the parameters showed that smokers had greater values than non-smokers. Haemoglobin, packed cell volume (PCV), and platelets were the most common (PLT). renal functions, a liver enzyme. The direct source of the observed variances in our study is still unknown, with the hope that future research will shed light on it.

Keywords: blood human, cholesterol, urea, creatinine, liver enzyme, parameters blood, and cigarette smoking.

INTRODUCTION:

Smoking cigarettes is one of the most important risk factors for vascular blood disease. as well as Atherosclerosis caused by smoking. Furthermore, smoking is a significant modifiable risk factor for cardiovascular illness, including cardiovascular sickness, stable ischemia, acute coronary syndromes, sudden cardiac arrest, stroke, and heart problems. [1, 2]. Cholesterol must be carried in the plasma in interaction with different lipoprotein particles because it is a hydrophobic molecule (no charged parts) lipid component (undissolved in water) [3]. Tobacco cigarettes cause a wider range of cardiac developing problems [5]. Furthermore, the silibinin (*Silybum marianum*) inhibits cardio myogenesis via Ang II-mediated signaling pathway and that is important in reducing cardiac problems [6]. Silybin treatment dramatically reduced the total cholesterol [7]. Cigarette smoking can cause changes in lipoprotein structure that are independent of serum cholesterol and unhealthy fats [8]. Total cholesterol levels that are too high, as well as smoking activities, are risk factors for

cardiovascular disease [9]. Cigarette smoking is now widely regarded as the leading preventable cause of death in the United States. Smokers develop diabetes, higher norepinephrine levels, which cause an increase in breathing, and an excess of cholesterol in the bloodstream, that all increase the chance of heart disease. The association between upregulate cholesterol levels and heart disease has been well-established and cannot be refuted. Furthermore, because cigarette smoking has been linked to the promotion of lipoprotein, this association is strengthened in smokers [10]. The frequency of atherosclerotic cardiovascular disease is opposite associated with high-density lipoproteins (HDL) cholesterol such as high blood sugar, metabolic disorders, and diabetes mellitus [11]. Nicotine from cigarette smoking causes a clot to form in the coronary arteries, reduces vascular activity, and worsens endothelial dysfunction. Increased carboxy-haemoglobin levels can cause hypoxia, and they're also linked to sub-endothelialoedema since they affect vascular permeability and lipid buildup [12]. The kidneys are important in maintaining the internal environment's consistency. The blood that passes through the kidneys is first filtered (glomerular filtration) to ensure that all constituents, except blood cells and plasma proteins, are filtered out and sent to the microtubular systems. Useful compounds are easily reabsorbed in the kidneys, whereas undesired substances escape filtration and are expelled in urine [13]. To measure renal function, most clinicians utilize the concentration of creatinine, also known as urea. These tests are sufficient for determining whether a patient has renal disease. The effects of smoking on renal function were first observed in patients with kidney disorders, such as kidney failure [14]. In addition, Liver function tests are helpful tools in clinical practice for assessing suspected liver disorders and monitoring therapy responses. As a result, the goal of this study is to see how smoking cigarettes affects cholesterol, blood parameters, blood creatinine, urea, and liver enzymes (Alanine aminotransferase (ALT), Aspartate aminotransferase (AST) activities, and alkaline phosphatase (ALP)).

MATERIALS AND METHODS:

Two groups were among the participants in this study, participants in this study included adult smokers and non-smokers of both genders, the subject with cholesterol, Hemoglobin (Hb), packed cell volume (PCV), and platelets (PLT), as well as, urea, creatinine, and liver enzymes (alanine aminotransferase (ALT); aspartate transaminase (AST), and alkaline phosphatase (ALP).

The first group was non-smokers, as a control group, these were used and the second group was smokers, these were the people that smoked 11 to 20 cigarettes each day. Smokers and non-smokers who were patients, attendants, volunteers, or workers at the Outpatient Clinic. The study's participants were chosen from among the workers. Those who claimed that they had hypertension, diabetes, and renal or hepatic disease are all conditions that can lead to death.

The participants were chosen from various age groups using convenience sampling with a purposive approach. In total, 30 individuals were approached in a row as being eligible for the study's inclusion criteria. The participants who were present at the outpatient clinic were interviewed. After explaining the study's objectives to everyone that took involved, a written informed consent form was signed by them. A self-administered questionnaire was used to collect demographic information from the participants.

Their smoking reports, as well as their medical history, were collected in case they had chronic illnesses or were taking lipid-lowering drugs. Furthermore, all participants were required to conduct their blood tests while fasting for at least 14 hours before taking the sample and every test depend on the procedure of the company that made it.

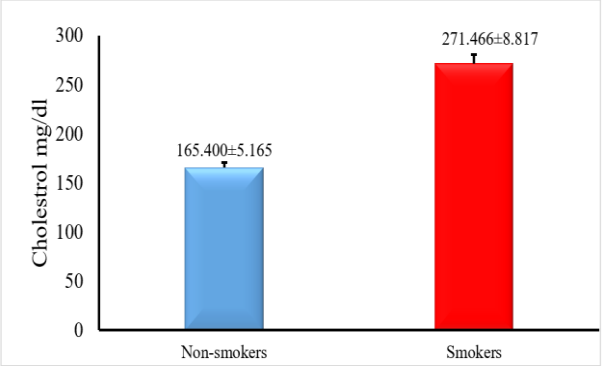
After a 14-hour fast, blood was drawn into basic tubes and centrifuged at 3000 rpm (China) for 15 minutes to collect the serum. All samples were analyzed to determine Cholesterol by using an Automatic Chemistry Analyzer and the usual homogeneous enzymatic technique (Linear) and the Linear Company with Spectrophotometer provided the calibration and internal controls (EMCLAB, Germany). Also, these samples were analyzed to determine kidney tests and liver enzymes.
The blood collected for Hemoglobin (Hb), packed cell volume (PCV), and platelets (PLT) by a 5ml syringe was used to collect 4 ml of venous blood from subjects in a reclining position between 10:00 am and 12:00 noon (to avoid diurnal fluctuations in cell count). With the needle removed, the samples were rapidly and carefully poured into a dipotassium ethylene diamine tetra-acetic acid (K2 EDTA) vial. The blood was gently inverted 5 times to mix it with the anticoagulant. To get the hemoglobin (Hb), packed cell volume (PCV), and platelets (PLT), a complete blood count and division were performed using an automated hematology analyzer [Cobac Swelab].

STATISTICAL ANALYSIS:

Excel and Statistics (SPSS version 18) were used for statistical analysis. The mean and standard deviation were used to express continuous data. Paired The difference between smokers and non-smokers for Cholesterol, Hb, PCV, PLT, urea, creatinine, ALT, AST, and ALP concentration assessed by the direct method and other formulas were compared using the Student's t-test. $P < 0.05$ was used as the level of significance.

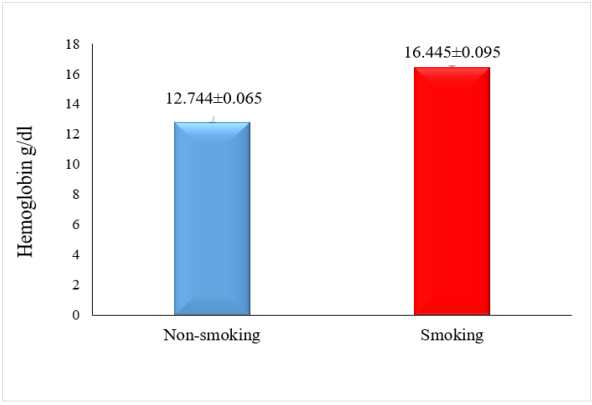
RESULTS:

The findings of the study revealed that there was a large difference between non-smokers and people who smoke in mean the total cholesterol values and showed total cholesterol levels compared to smokers a greatly increased (271.466±8.817; $p < 0.05$) than non-smokers (165.400±5.165; $p < 0.05$) (control) [Table/Fig-1].

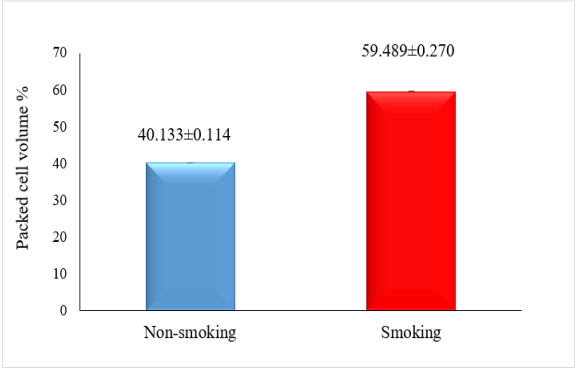


[Table/Fig-1]: Cholesterol levels in studied individuals

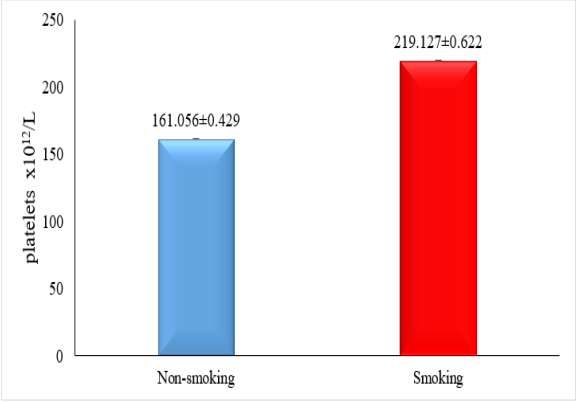
Hemoglobin (Hb), packed cell volume (PCV), and platelet (PLT) mean and standard Error for smoking16.445±0.095; $p < 0.05$, 59.489±0.270; $p < 0.05$, 219.127±0.622; $p < 0.05$, compared to non-smokers 12.744±0.065; $p < 0.05$ [Table/Fig-2], 40.133±0.114; $p < 0.05$ [Table/Fig-3], 161.056±0.429; $p < 0.05$ [Table/Fig-4], respectively.



[Table/Fig-2]: Hemoglobin concentrations in studied individuals

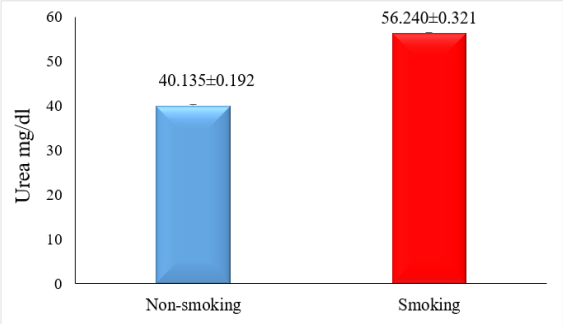


[Table/Fig-3]: Packed cell volume in studied individuals



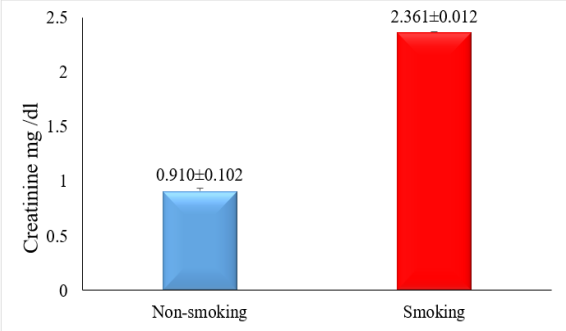
[Table/Fig-4]: Platelets in studied individuals

[Table/Fig-5] shows the urea, non-smoker, and smoker groups. The results demonstrated that smokers had significantly higher levels of urea 56.240±0.321 $p < 0.05$, than the non-smokers group (40.135±0.192).



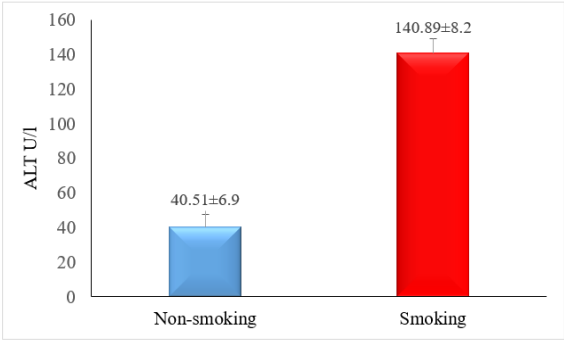
[Table/Fig-5]: Urea in studied individuals

[Table/Fig-6] shows the creatinine, non-smoker, and smoker groups. The results demonstrated that smokers had significantly higher levels of creatinine (2.361±0.012 $p < 0.05$, than the non-smokers group (0.910±0.102).



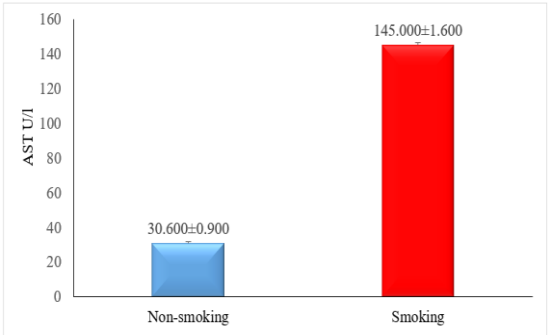
[Table/Fig-6]: Creatinine in studied individuals

[Table/Fig-7] indicates the effects of smoking on the parameters evaluated. There was a substantial increase in ALT 140.890±8.200; $p < 0.05$ compared to non-smokers 40.510±6.900; ; $p < 0.05$.



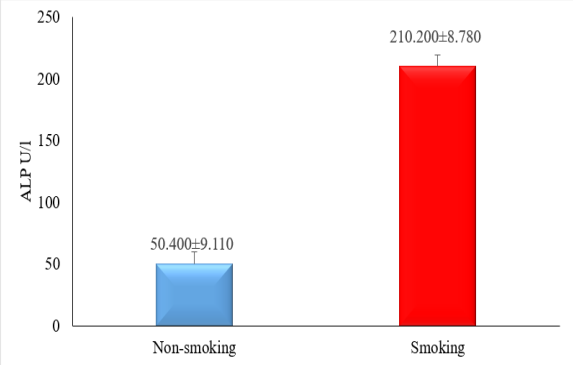
[Table/Fig-7]: ALT in studied individuals

[Table/Fig-8] indicates the effects of smoking on the parameters evaluated. There was a substantial increase in AST 145.000±1.600; $p < 0.05$ compared to non-smokers 30.600±0.900; $p < 0.05$.



[Table/Fig-8]: AST in studied individuals

[Table/Fig-9] indicates the effects of smoking on the parameters evaluated. There was a substantial increase in ALP 210.200±8.780; $p < 0.05$ compared to non-smokers 50.4010±9.110; ; $p < 0.05$.



[Table/Fig-9]: ALP in studied individuals

DISCUSSION:

Cigarette smoke is a mixture of numerous harmful compounds, the most often implicated in the etiology of cardiovascular disease being tobacco, carbon monoxide, and oxidant compounds are all found in cigarettes. Tobacco use leads to a problem with the endothelium, hypertension, insulin resistance, lipid profile changes, the dynamics of blood flow changes, and hypercoagulability. In tobacco smokers' atherothrombosis, all of this function together as a better understanding of the mechanisms [15,16]. Smoking raises cholesterol levels, linked to a reduction in lipoprotein lipase activity [17]. In this study, it was discovered that cigarette smoking was linked to higher cholesterol levels, which coincided with the discovery of Hajmouhamed et al., [18]. According to this research, there is an inverse relationship between smokers and non-smokers, and total cholesterol levels are consistent with the findings of our investigation. Regardless of gender, smokers had considerably higher hemoglobin levels than nonsmokers in our study. Previous research has shown that smoking causes a considerable increase in Hb [19, 20, 21]. Carbon monoxide exposure is thought to cause an elevation in hemoglobin content, and some studies suggest that a rise in hemoglobin levels in smokers' blood could represent a compensatory mechanism. Carbon monoxide binds to Hb to generate carboxy hemoglobin, a passive form of hemoglobin that carries no oxygen. Carboxyhemoglobin also shifts the left side of the Hb dissociation curve, reducing Hb's capacity to supply oxygen to the tissue. Smokers have a higher hemoglobin level than non-smokers to compensate for their reduced oxygen delivery capacity [22]. The effect of cigarette smoking on renal function, as measured by serum creatinine and urea, is shown in this study. It reveals that smokers had higher levels of serum creatinine and urea ($p < 0.05$) than non-smokers. These results are consistent with those of other investigations [13, 23]. The findings demonstrate a considerable increase in the activity of the liver enzymes ALT, AST, and ALP in smokers compared to non-smokers, with the proportion increasing with smoking duration. These findings are consistent with the levels of liver enzymes ALT, AST, and ALP, which rose in response to smoke exposure or the generation of high quantities of cellular reactive radicals, as reported in [24, 25]. The daily amount of smoking had a significant effect on serum levels of ALT and AST, although there was a multivariate effect after adjusting for gender, age, BMI, daily current smoking, and lifetime smoking, as reported in [25]. We may conclude from this study that persistent cigarette smoking raises hemoglobin concentrations, levels of serum creatinine and urea, and Liver enzymes which may be linked to an increased risk of atherosclerosis, polycythaemia vera, chronic obstructive pulmonary disease, and/or cardiovascular illnesses.

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