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Effect of Smoking on Some Biochemical and Hematological Indicators in Smokers

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Annotation: Objective: The aim of our study is to investigate the effect of smoking on hematological levels, lipid profile, renal and liver function tests, and cardiac enzymes in current smokers.

Materials and Methods: This investigation was conducted in various locations within Diyala Province, Iraq, from January to April 2024. A total of 100 blood samples were collected from smokers and 50 samples from non-smokers, which served as the control group. HbA1C was measured using a Biorex machine (Denmark), and hemoglobin (Hb) and platelets (PLTs) were measured using a Hematology Analyzer (CBC machine) (India). Serum concentrations of fasting blood sugar (FBS), blood urea, serum creatinine, lipid profile, ALT, AST, ALP, and high-sensitivity cardiac troponin (hs-cTn) markers were determined using a Roche Cobas e411 (Switzerland). The SPSS v. 20.0 program was used to analyze our data at a significance level of $P \leq 0.05$.

Results: The outcomes of the current study showed that most smokers were within the age groups 31-40 (35%) and 21-30 years (30%), with fewer in the age groups 51-60 (12%) and >60 years (9%), showing significant differences among age groups ($p<0.05$). Additionally, we

found a significant increase ($p<0.05$) in the levels of hemoglobin, platelets, cholesterol, triglycerides, ALT, AST, ALP, hs-cTn, and creatinine in smokers compared to non-smokers. In contrast, the study revealed no significant differences ($p>0.05$) in the levels of HbA1c and urea between smokers and non-smokers.

Conclusions: This investigation suggests that chronic cigarette smoking increases blood levels of hemoglobin, platelets, serum creatinine and urea, lipid profile, and cardiac and liver enzymes. These elevated levels may be associated with a higher risk of atherosclerosis, polycythemia vera, chronic obstructive pulmonary disease, and/or cardiovascular diseases.

Recommendations: The current study recommends conducting further research on the effects of smoking on biochemical variables in smokers and those who quit smoking, compared to non-smokers.

Keywords: Smoking, enzymes, lipids, biochemical and hematological markers.

Introduction

One of the biggest causes for arterial blood disorders and smoking-related atherosclerosis is smoking cigarettes. Moreover, smoking is a major changeable contributory factor for a number of cardiovascular illnesses, such as stable ischemia, acute coronary syndromes, heart difficulties, stroke, and sudden cardiac death (Guo et al., 2023).

It is thought that smoking alters blood lipid levels. Although a definitive correlation between smoking and blood lipids cannot be established, certain research has indicated that smoking cigarettes might potentially modify serum lipid levels in the blood by absorbing nicotine, which modifies the blood lipid process (Herath et al., 2022). According to certain research, nicotine lowers HDL cholesterol (HDL-C), raises triglycerides, total cholesterol, and LDL cholesterol (LDL-C). According to other research, smoking raises triglycerides and lowers total, LDL, and HDL cholesterol (Hussein et al., 2024).It is well known that smoking increases the risk of tumors in the liver. Jang et al.'s research from 2023 also showed that smoking increases the chance of cirrhosis and may slow the progression of chronic liver illnesses. Studies have shown that smoking deteriorates kidneys function in individuals with glomerulonephritis and hypertensive renal sclerosis, two kidney disorders (Chen et al., 2023).

A class of enzymes found in cardiac muscle fibers that control the contraction of muscles is known as cardiac troponins (cTn). Cardiovascular troponin T (cTnT) and troponin I (cTnI) are commonly used as diagnostic tools that are highly accurate and reliable for myocardial infarction, or MI. Even when circulating cTn levels are within the typical range, they are substantially linked to the risk of cardiovascular events in the chronic situation and are thought to be a sign of subclinical chronic myocardial damage (Jakubiak, 2024). According to a prior study,

smokers had lesser amounts of troponin than non-smokers do, and those levels rise within the standard range after quitting (Zaman et al., 2023).

Study Objective

Our study aim to study effect of smoking on levels of hematological, lipid profile, renal and liver function test, and cardiac enzyme in current smokers.

Patients and Methods

1. Samples Collection

The conducted investigation was applied in different places within Diyala province / Iraq from January to April 2024. 100 blood samples were collected from smokers and 50 samples from non smokers that considered them as control group. All participants were males and within age group 21-70 years.

2. Methods

Each person gave five milliliters of blood, which was then placed in an EDTA tube and a gel tube (to contain serum). HbA1C was measured using a Biorex machine (Denmark) and Hb and PLTs by a Hematology Analyzer (CBC machine) (India) using blood taken in an EDTA tube. Blood was placed in a gel tube and spun for six minutes at 3000 rpm to extract serum. By using Roshe Cobas e411 (Switzerland), serum concentrations of FBS, B. urea, S. creatinine, lipid profile, ALT, AST, ALP, and hs-cTn markers were determined. This test was carried out in accordance with the manufacturer's procedure protocol, which was contained in the kit packaging.

Statistical Analysis

All indicators were mentioned as Mean± SD. Independent t test based to detection differences between smokers and non smokers groups. Variations among age groups were calculated by Pearson-Chi-square test. Standard significant level was $P \leq 0.05$. SPSS v. 20.0 program was used to measure our data.

Results

1. Age of Participants

Outcomes of current study showed the most smokers were lie within age groups 31-40 (35%) and 21-30 years (30%) and a little of them lie at 51-60 (12%) and >60 years (9%) with significant differences among age groups ($p<0.05$) (table 1).

2. Relation of Biochemical Indicators with Smoking

Data of conducted study showed there is significant increase ($p<0.05$) in levels of hemoglobin, platelets, cholesterol, triglycerides, ALT, AST, ALP, hs-cTn and creatinine in smokers (16.33±2.91, 398.92±70.83, 287.78±56.37, 162.93±30.23, 132.98±19.23, 145.32±14.32, 210.87±56.84, 18.27±3.91 and 2.04±0.34) than non smokers peoples (13.23±1.09, 190.67±30.91, 165.72±27.23, 119.22±20.87, 52.89±11.76, 56.74±9.08, 73.24±12.31, 11.21±2.90 and 0.91±0.25). In contrast, current study revealed no significant differences ($p>0.05$) in levels of HbA1c and urea between smokers versus non smokers (table 2).

Table 1; Distribution of Smokers According to Age Groups

Age groups (years)	N	%	P value
21-30	35	35%	p<0.01**
31-40	30	30%	
41-50	14	14%	
51-60	12	12%	

>60	9	9%	
Total	100	100%	

Table 2; Comparative Mean Levels of Biochemical Indicators Between Smokers and No Smokers Persons

Biochemical parameters		Groups	Number	Mean	St. deviation	P value
Hematological Indicators	Hemoglobin (g/dl)	Smokers	100	16.33	2.91	P<0.05*
		No smokers	50	13.23	1.09	
	Platelets (× 10^9/L)	Smokers	100	398.92	70.83	P<0.05*
		No smokers	50	190.67	30.91	
Lipids	Cholesterol (mg/dl)	Smokers	100	287.78	56.37	P<0.05*
		No smokers	50	165.72	27.23	
	Triglycerides (mg/dl)	Smokers	100	162.93	30.23	P<0.05*
		No smokers	50	119.22	20.87	
Liver function tests	ALT (mg/dl)	Smokers	100	132.98	19.23	P<0.05*
		No smokers	50	52.89	11.76	
	AST (mg/dl)	Smokers	100	145.32	14.32	P<0.05*
		No smokers	50	56.74	9.08	
	ALP (mg/dl)	Smokers	100	210.87	56.84	P<0.05*
		No smokers	50	73.24	12.31	
Sugar	HbA1c mmol/mol	Smokers	100	6.8	1.87	**P>0.05**
		No smokers	50	6.32	1.25	
Cardiac enzyme	hs-cTn (ng/l)	Smokers	100	18.27	3.91	P<0.05*
		No smokers	50	11.21	2.9	
Renal function tests	Urea (mg/dl)	Smokers	100	55.78	12.39	**P>0.05**
		No smokers	50	37.89	3.82	
	Creatinine (mg/dl)	Smokers	100	2.04	0.34	P<0.05*
		No smokers	50	0.91	0.25	

Discussion

According to recent data, the majority of smokers were between the ages of 21 and 40. The results obtained were consistent with those of Colyer-Patel et al. (2023). Early stopping will save most of the years that smoking would otherwise cost you. One can significantly extend their lifespan even if they stop smoking at 65 years of age or more (Le et al., 2024).

Cigarette smoke contains a variety of dangerous substances, the most common of which are tobacco, carbon monoxide, and oxidant chemicals, which are all linked to the development of cardiovascular disorders. Smoking causes lipid profile alterations, hypertension, resistant to insulin, endothelial problems, modifications to circulation dynamics, and hypercoagulability. All

of these contribute to a better knowledge of the processes of atherothrombosis in tobacco users (Ishida et al., 2024). Smoking increases levels of triglycerides and cholesterol and is associated with a decrease in lipoprotein lipase activity (Parmar et al., 2023). Cigarette smoking was shown to be associated with elevated cholesterol levels in the current investigation, which was concurrent with the findings of Ali et al. (2022).

In our research, smokers had significantly greater levels of hemoglobin than nonsmokers, irrespective of gender. Prior studies have demonstrated that smoking significantly raises Hb levels (Vivek et al., 2023; Nurjanah et al., 2023). It is believed that being exposed to carbon monoxide raises hemoglobin pleased, and some research indicates that an increase in hemoglobin levels in smokers' blood may be a compensatory strategy. Hemoglobin that does not carry oxygen is called carboxy hemoglobin, which is created when carbon monoxide attaches to hemoglobin. Moreover, carboxyhemoglobin causes the Hb dissociation curve to move to the left, which lowers Hb's ability to carry oxygen into the tissue. To make up for their decreased ability to supply oxygen, smokers have greater hemoglobin levels than non-smokers (Wijaya and Nataprawira, 2024). Smoking causes oxidative damage, which increases platelet activation and aggregation as well as nicotine-induced endothelial damage. It has been proposed that smoking cigarettes interferes with activation of platelets, accumulation, and coagulation, which causes thrombosis (Ramotowski et al., 2023). The present research demonstrates the impact of cigarette smoking on the renal system as determined by blood creatinine and urea levels. It demonstrates that there is a substantial correlation between smoking and kidney damage, which may result in problems related to renal function in the future. Smokers had greater amounts of blood creatinine and urea ($p < 0.05$) than non-smokers. These findings align with the findings of previous studies (Ali et al., 2022; Lang and Schiffl, 2024).

The results show that smokers have significantly higher levels of the liver enzymes ALT, AST, and ALP activity than non-smokers do, and that the proportion rises with the length of smoking. These results align with the elevation of liver enzyme levels (ALT, AST, and ALP) observed with contact to smoke or high amounts of cellular reactive radicals, as documented in (Kalyanikutty, 2024). According to (Barapatre et al., 2024), there was a multivariable influence on serum levels of ALT and AST after controlling for gender, age, BMI, daily current smoking, and lifetime smoking. However, the daily quantity of smoking had a substantial impact on these levels.

According to Grossmann et al. (2022), smokers had lower levels of hs-cTnI than non-smokers, which is in contrast to the current study's findings, which indicate that smokers had higher levels of hs-cTnI than non-smokers. Previous research has demonstrated a correlation between lower quantities of hs-cTnI with cigarette smoking, both past and present. There has been a dosage response connection between pack-years of smoking and hs-cTnI in ex-smokers. Increasing quantities of hs-cTnI were correlated with time after smoking cessation, suggesting a continuum of hs-cTnI from current tobacco users to never-smokers (Skranes et al., 2022). The results of the research conducted by the authors imply that tobacco use may affect cardiac troponin's production or breakdown. This might have an impact on how cardiac troponin is used to diagnose and predict heart disease. It is still unclear if smoking and cardiac troponin have an inverse relationship in an emergency situation, thus further research on this topic is crucial (Skranes, 2022).

Conclusions

This investigation suggests that chronic cigarette smoking increases blood levels of hemoglobin, platelets, serum creatinine and urea, lipid profile, and cardiac and liver enzymes. These elevated levels may be associated with a higher risk of atherosclerosis, polycythaemia vera, chronic obstructive pulmonary disease, and/or cardiovascular diseases.

Recommendations

The current study recommends conducting further studies on the effect of smoking on biochemical variables in smokers and smoking quitting, and comparing them to non-smokers.

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