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Influence of Cigarette Smoking on Liver Function and Lipids Levels Tests: A comparative Study Directed among Smokers and Non-smokers in Babylon Governorate

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Abstract

Cigarette smoking currently is considered as one of the greatest problems in public health worldwide and is risk factor for peripheral vascular disorders and heart disease. The monitoring of liver function and total cholesterol are very important to give an estimation of the future cardiovascular diseases among smokers. The objective of this study is to evaluate the effect of cigarette smoking on the serum levels of liver enzymes: aspartate aminotransferase (GOT), alanine transaminase (GPT) and cholesterol activities among Iraqi smokers in Babylon City, compared to apparently healthy individuals (non-smokers) as a control group (all were males). A case- control study was carried out on forty Iraqi male smokers who smoke at least 10 cigarettes per day for at least15 years. The group includes smokers with age range between 15-55 years. Non-smokers, (control, n= 20) group were collected with the same range of age for statistical comparison. The whole blood samples were drawn by venipuncture from each individual; levels of cholesterol and liver functions test were estimated in the blood serum of smokers and non-smokers by diagnostic kit (Randox corporation-UK) using automatic analyzer.The findings of this study showed that there was a significant higher level of total cholesterol, ALT, and AST in the smokers group compared to non-smokers (P<0.05). As well as, the results showed a significant positive correlation between the smokers' age and serum GOT, GPT and cholesterol activity (Increasing) especially in age between 45 to 55 years old, as compared to control. In addition, total cholesterol and liver function enzymes were significantly positive correlated with the duration of smoking (P<0.05).Cigarette smoking leads to oxidative stress by free radical generation by the mechanism of lipid peroxidation that is affected by the heaviness of smoking. Smoking exerts negative influence on liver functions test that should be carefully interpreted and preventive strategies needed to avoid the future cardiovascular diseases.

Key words: liver enzymes, aspartate aminotransferase (GOT), alanine transaminase (GPT), Cholesterol (TG)

Introduction

Cigarette smoking presently considered one of the greatest problems in public health worldwide and it is one of the most preventable reasons of death. The World Health Organization (WHO) has revealed that current tobacco use kills 6 million people annually - an average of one person every six seconds and represents one in 10 deaths among adults worldwide, and this number is required to increase the number of deaths to 10 million people Annually by 2020 (1).

The risk of death in smokers is estimated by the amount of cigarettes smoked each day, the duration of smoking, the degree of inhalation, and the age of initiation (2). It have been discovered that tobacco smoke comprises more than 4000 different chemicals, 400 of which are demonstrated cancer-causing; It also contains various oxidants, such as oxygen free radicals and volatile aldehydes, most likely the significant reasons for harm to biomolecules (3), as well as other chemical substances which have highly cytotoxic potentials that are considered gases, such as, carbon monoxide (CO), hydrogen cyanide (HCN), and nitrogen oxides. Different types of cigarette smoking contain formaldehyde, acrolein, benzene and some N-nitrosamines, which are volatile chemicals present in the liquid smoke portion of the vaporized smoke. Others, such as nicotine, phenols, polycyclic hydrocarbons (PAHs), and some tobacco express nitrosamines (TSNAs), are still present in solid particles of sub-micron size suspended in cigarette smoke (4).

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Additionally, cigarette smoke contains massive amounts of free radicals that are able to initiate or enhance oxidative stress. Cigarette smokers are at a higher risk of developing cardiovascular disease, respiratory disorders, cancers, peptic ulcers, gastroesophageal reflux disease, fascination, loss of bone matrix, and hepatotoxicity, compared to non-smokers (5). Cigarette smoking causes a variety of harmful effects on organs that do not have direct contact with the smoke itself, for example, the liver. The liver is an important organ with many functions; Such as the responsibility of processing drugs, alcohol and various toxins to excrete them from the body (6).

Some studies have demonstrated increase activity of AST, ALT and ALP in smoker compared to nonsmoker in population.

However, some studies of smoking and serum fat have shown that the level of plasma cholesterol tends to be lower in smokers than in non-smokers (7). Smoking, which is a major risk factor for developing ischemic heart disease, may alter normal plasma lipoprotein style. Earlier (8) it had been shown that high cholesterol levels were observed in smokers. It has also been suggested that, apart from other risk factors, hypercholesterolemia. Tobacco smoke contains many ingredients, and nicotine is one of the main ingredients. Nicotine causes an increase in cholesterol levels, and some have studied that long-term consumption of nicotine by mouth increases cholesterol (2, 4).

Thus, the current study was constructed to assess the relationship between cigarette smoking and the biochemical state of the liver by exploring the effect of cigarette smoking on liver function that was conducted to assess the activity of AST and ALT among male smokers in Hilla City. As well as knowing whether there is a relationship between the duration of smoking and the number of cigarettes smoked per day.

Materials and methods

Study population

In this study, samples were randomly collected based on several simple variables: age, smoking period, and number of cigarettes consumed daily. The study included (40) male individuals who smoked and (20) non-smokers. Male smokers and non-smokers as a control group select apparently strong volunteers in Hilla City.

The study groups

The first group: for the age group (15-25 years) included (10) smokers people and (5) non-smokers people as controls.

The second group: for the age group (25-35 years) included (10) smokers people and (5) non-smokers people as controls.

The third group: for the age group (35-45 years) included (10) smokers people and (5) non-smokers people as controls.

The fourth group: for the age group (45-55 years) included (10) smokers people and (5) non-smokers people as controls.

Blood samples collection

About five milliliters (5 mL) of venous blood were collected from each fasting patient. Samples were collected under sterile conditions and placed in sterile normal containers (gel tube), after which the coagulants were centrifuged for 15 minutes at 6000 rpm to obtain blood serum, and then the resulting serum was kept at -20 ° C until the time of analysis.

Estimation of serum aspartate aminotransferase

AST enzyme determines its activity by diagnostic kit make by randox corporation (UK).

Principle of method:

AST enzyme determined by regulated oxaloacetate hydrozoan Concentration that forms with 2-4 dinitrophenyl hydrozoans that give brown color. It may measure by spectrophotometer at 546nm wavelength it will be proportion intensity of color with amount of enzyme in blood serum (9).

Estimation of serum alanin aminotransferase

ALT enzyme is determining their activity in blood serum by diagnostic kit that make by Randox Corporation (UK).

Principle of method:

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ALT enzyme determined by regulated Pyruvate hydrazine Concentration that form with 2-4dinitrophenyl hydrozoan that give brown color. It may measure by spectrophotometer at 546nm wavelength it will be proportion intensity of color with amount of enzyme in blood serum (9).

Enzymatic method described by (10).
1- Mix. Let stand for 5 min at 37 ºC or 10 min at room temperature.
2- Let stand reagent and specimens at room temperature.
3- Record absorbance at 500nm against reagent blank.
4- Measure absorbance of samples and record the result.
The color is stable for 1 hour.

Statistical analysis

The statistically significant differences between the experimental means were dictated by the less significant distinction. The SPSS 14.0 statistical tool was used to analyze the information obtained. Results were considered statistically significant at p <0.05 with different Duncan scale tests.

Results

The current study consisted of 60 males divided into 20 healthy non-smokers as a control group and 40 dynamic smokers whose ages ranged from 15 to 55 years as an experimental group. Individual are divided into four groups (15-25), (25-35), (35-45) and (45-55), were taken a crack at this study to study the impact of smoking on some liver enzymes and serum total cholesterol.

Effect of smoking on enzyme activity of GOT and GPT and Cholesterol levels

The results demonstrated statistically significant differences (P value>0.05) were seen in serum GOT and GPT levels, were significantly high (P value<0.05) in non- smoker group when contrasted with heavy smoker nonsmokers controls group. The results of the current study are given in 2 tables and 4 charts, according to age's group and smoking duration.

Liver enzyme levels and the GPT GOT in the blood of smokers and non-smokers serum according to age groups

The Table indicates (1) and graph (1,2) to the emergence of a significant difference in liver enzyme levels GOT and the GPT in the serum blood of smokers compared to the totals of control were significantly (P<0.05), and by age levels of the four groups of the enzyme GOT (93.14 , 107.01 , 116.5 and 134.94) unit / liter, respectively and enzyme GPT (95.12 , 111.2 , 132.13 and 156.3) unit / liter thereby defeating groups of control for GOT (89.41 , 95.51 , 99.99 and 99.5) unit / liter and GPT (85.7 , 99.7 , 104.3 and 104.9) unit / liter respectively for all ages group, the results of statistical analysis also showed a significant increase for enzymes age group (45-55) and get over the rest of the groups of smokers and non-smokers.

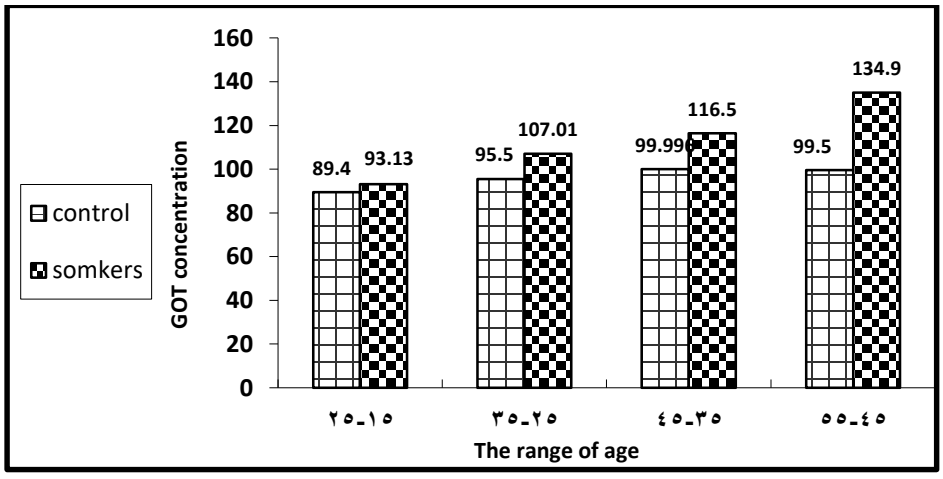

Figure (1): GOT level with the range of age

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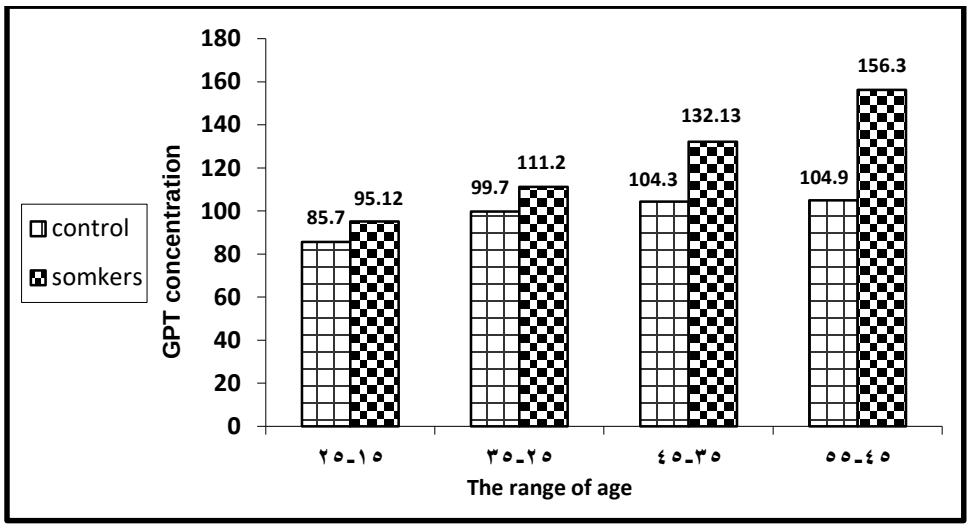

Figure (2): GPT level with the range of age

Levels of liver enzymes GOT and GPT according to smoking duration

When smokers divided into two groups according to the smoking duration (5-10) (15 – 25) years, we note from the Table (2) and graphs (3) the emergence of a simple significant rise in the levels of enzymes GOT to increase the smoking duration, as it stood at the effectiveness of the enzyme (100.10) (125.7) unit/liter for a smoking duration (5-10) and (10-15) years compared with the control group respectively, while the liver GPT enzyme levels (103.16) (788.01) unit/liter for a smoking duration (5-10) and (10-15) years) compared with the control group (96.11) (96.11) unit/liter for GOT and (98.7) (98.7) unit/liter for GPT respectively that note in Table (2) and graphs (4), and recorded the highest levels of the Smoking (10-15) a year and beating up on liver enzyme levels GOT.

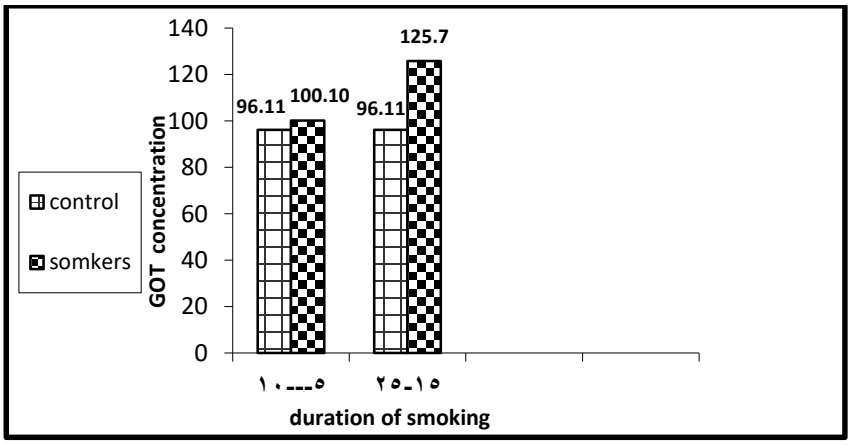

Figure (3): GOT level with smoking duration

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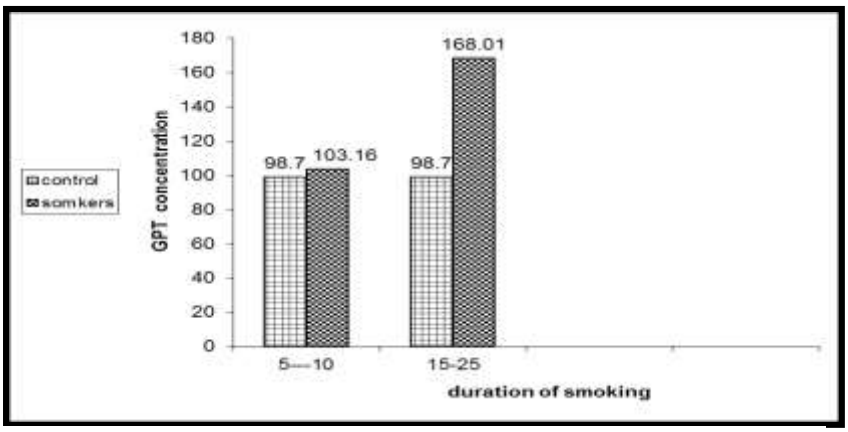

Figure (4): GPT duration of smoking

Levels of cholesterol concentration in the blood serum of smokers and non-smokers

The Table indicates (1) and graphs (5) to the emergence of statistically significant differences in the concentration of cholesterol levels in the blood serum of smokers compared to control at significant (P<0.05), and by age levels of the four groups (138.85, 183.34, 299.75 and 432.11) mg / deciliter than compare with control (156.77, 161.1, 181.86 and 203.51) mg / deciliter respectively , as well as note overcome age group (45-55) cholesterol levels in the rest of the age groups for smokers as well as the control group.

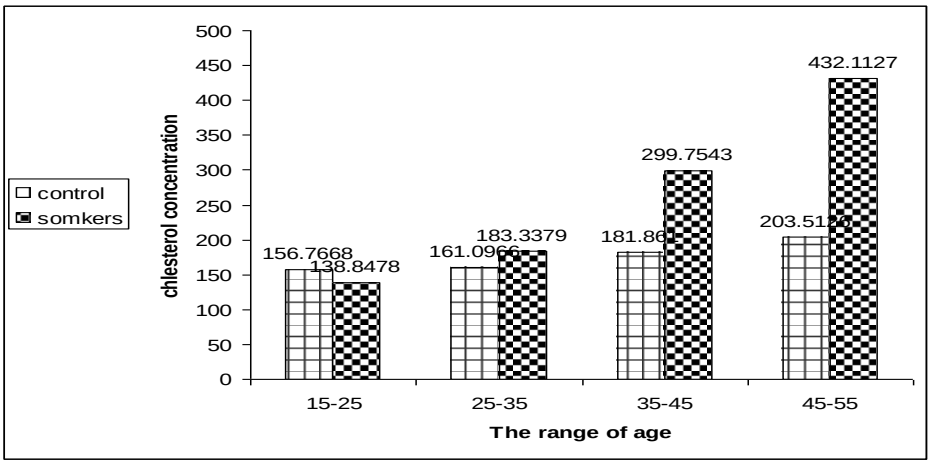

Figure (5): level of cholesterol with the range of age

Levels of cholesterol concentration according to smoking duration

when smokers divided into two groups according to the smoking (5-10) and (10-15) years, we note from the table (2) and graph (6) the emergence of a significant concentration of high cholesterol levels increase the smoking duration, reaching Hits (161.09) and (365.9) mg / deciliter for a duration smoking years compared with a group control (175.8) and (175.8) mg/deciliter respectively. It is the results of statistical analysis note for a significant increase in the level of (15-25) for a smoking duration (10-15) and beating by a wide margin over a smoking duration (5-10) and compared to the control group, respectively.

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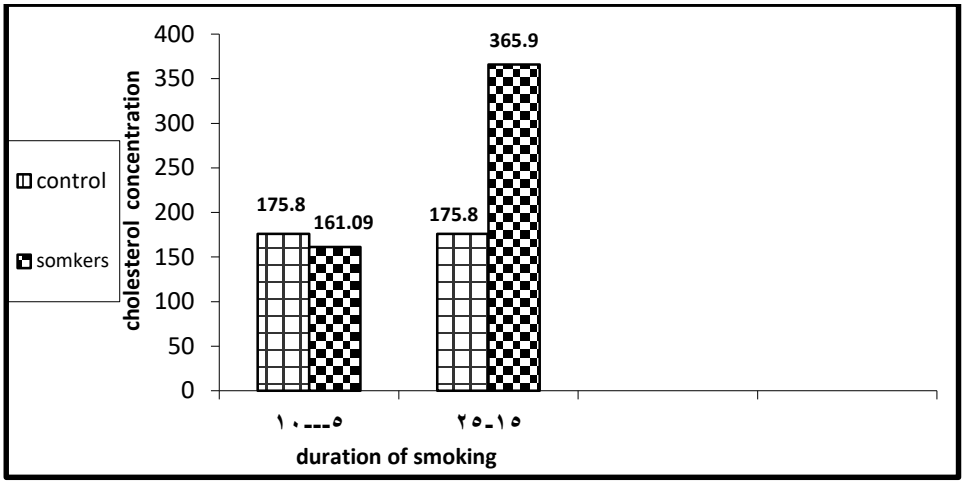

Figure (6): cholesterol with duration of smoking

Table (1): level of GOT, GPT and Cholesterol in blood serum smokers and non-smokers according to age

parameters	According to age							
	15-25		25-35		35-45		45-55	
	Control	Smoker	Control	Smoker	Control	Smoker	Control	Smoker
GOT unit/litter Mean	89.41	93.14	95.51	107.01	99.99	116.5	99.5	134.94
GPT unit/litter Mean	85.7	95.12	99.7	111.2	104.3	132.13	104.9	156.3
CHOLESTEROL mg/dislater Mean	156.77	138.85	161.1	183.34	181.86	299.75	203.51	432.11

Table (2) level of GOT, GPT and Cholesterol in blood serum smokers and non-smokers according to smoking duration

parameters	According to smoking duration			
	5-10		15-20	
	Control	Smoker	Control	Smoker
GOT unit/litter Mean	96.11	100.10	96.11	125.7
GPT unit/litter Mean	98.7	103.16	98.7	168.01
CHOLESTEROL mg/dislater Mean	175.8	161.09	175.8	365.9

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Correlation between levels Cholesterol, GOT and GPT in blood serum

The Table indicates (3) and to the emergence of a significant correlation in the concentration of GOT levels in the blood serum with GPT, this Pearson Correlation is 0.881 at significant (P<0.05) for control. But do not found correlation between cholesterol and liver enzymes for control.

There are significant correlation in the concentration of GOT levels in the blood serum with concentration of cholesterol in blood serum, this Pearson Correlation is 0.845 at significant (P<0.05) for smoker, but do not found correlation GPT with other parameters at significant (P<0.05) for smoker as indicates in Table (4), and note in this Table also there are correlation between concentration of GOT and GPT levels in blood serum, this Pearson Correlation is 0.845 at significant (P<0.05) for smokers.

Table (3): Correlation between levels Cholesterol, GOT and GPT in blood serum for control

		cholesterol	GOT	GPT
cholesterol	Pearson Correlation	1	0.240	0.355
	Sig.		.309	0.125
GOT	Pearson Correlation	0.240	1	o.881**
	Sig.	0.309		0.000
GPT	Pearson Correlation	0.355	0.881**	1
	Sig.	0.125	0.000	

Table (4): Correlation between levels Cholesterol, GOT and GPT in blood serum for smoker

		cholesterol	GOT	GPT
cholesterol	Pearson Correlation	1	0.845**	0.182
	Sig.		0.000	0.261
GOT	Pearson Correlation	0.845**	1	0.291
	Sig.	0.000		0.068
GPT	Pearson Correlation	0.182	0.291	1
	Sig.	0.261	0.098	

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Discussion

Smoking on the effectiveness of the liver enzymes GOT effect and GPT

This is a case study aimed at studying the effect of smoking on liver and cholesterol enzyme activities. Sixty Hilla City males (40 smokers and 20 non-smokers) were registered to study the effect of smoking on liver enzymes and cholesterol. After evaluation of enzymes and cholesterol activities by auto analyzer, the statistical analysis was done by using SPSS computer program and the results showed that all liver enzyme levels, it was significantly higher in the smokers' group compared to the non-smokers group Table (1), increase in activity of GOT and GPT, in smokers indicate tissue damage due to loss of functional integrity of cell membrane.

Results of this study revealed that increases in GOT and GPT is proportional with duration of smoking per years, also The serum GOT and GPT activities are a significant positive correlation with age. The serum GOT and GPT activities are a significant positive correlation with duration of smoking.

It noted the current data to the existence of a moral high $p<0.05$ in the effectiveness of the enzymes (GOT and GPT) in smokers than controls, and noted previous studies that the moral high effectiveness of these enzymes in the blood serum or tissue due to excess nitric oxide effort. Noted the scientific literature that cigarette smoke components suffer metabolic activity in with enzymes cytochrome E p450 of reactive electrophiles that causes excessive nitric oxide leading to toxic the cells and mutations and cancer (11), as the Cigarette smoke contains great numbers of toxic substances that cause toxic cells of liver, such as nicotine (7), that the oxidative effort caused by cigarette smoke leads to stimulate NADPH oxidase which reduces defense anti-oxidants and increases the oxidation of lipids (12), and cigarette smoke have a significant impact on liver function, because it contains free radicals, leading to the events of oxidant effort and increases the oxidation of fat (13).

Nicotine is the significant part of tobacco smoke plays an important role in the development of numerous diseases (14). It makes oxidative damage to kidney, lung, liver and heart. It is a likely oxidant, which is capable of producing free radical and receptive oxygen species (15).

The nicotine induce free radical to react with bio membrane causing oxidative destruction of poly unsaturated fatty acid and forming cytotoxic aldehydes by lipid per-oxidation, lipid per-oxidation implicated in pathogenesis of number of disease (14).

The results indicated a significant increase in the effect of GOT and GPT in the blood serum of cigarette smokers compared to the control group. This may be caused by nitric stress and is a condition that occurs when the production of highly reactive chemicals containing nitrogen, such as nitrous oxide, exceeds the human body's ability to neutralize and eliminate them. The nitric stress can lead to reactions that alter protein structure and thus interfere with normal body functions (16). Cigarette smoke contains countless synthetic substances with the potential to poison the liver, including nicotine (13).

Cigarette smoking is a significant risk factor for cardiovascular disease. Smokers are more likely to develop coronary artery disease than non-smokers. Some potential explanations for this relationship have been presented, including modified blood clotting, impaired arterial wall integrity, and changes in blood lipids (17). Several studies have indicated an association between cigarette smoking and altered blood lipids.

The effect of smoking on the cholesterol

Table (1) shows the TG cholesterol levels in the smokers and control groups. The total cholesterol level was significantly increased ($P <0.05$). A significant increase ($P <0.05$) was observed in the level of blood cholesterol in smokers compared to non-smokers.

It has been observed that blood cholesterol levels are elevated in all groups of cigarette smokers, but the risk is greater in heavy smokers. When investigating the results regarding smoking duration, it was generally noted that there was a significant increase in blood cholesterol level in relation to the increase in the duration and intensity of cigarette smoking.

Cigarette smokers are more likely to develop CHD than non-smokers. Some possible clarifications have been provided for this relationship that led to altered blood clotting, disturbances in the integrity of arterial walls, and changes in blood lipids and lipoprotein

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concentration. Smoking leads to the development of coronary artery disease and atherosclerosis.

This may be due to the nicotine in tobacco smoke, which increases the oxygen requirements of the heart muscle by increasing the use of free fatty acids, and smoking with an unknown mechanism reduces the anti-hormone HDL-C factor, and remains an important and independent predictor of coronary artery disease.

Cigarette smoking may lead to atherosclerosis, to some extent, through its effect on the lipid profile. Changes in plasma lipid concentration including cholesterol are complications often observed in patients that contribute to the development of vascular disease. Cholesterol has been identified as a primary factor in the development of atherosclerosis.

Smokers have high oxidative stress and usually have a lower intake of antioxidant vitamins and a lower plasma level, which may make LDL cholesterol more vulnerable to lipid peroxidation. The oxidation of LDL cholesterol is thought to play an important role in early atherosclerosis by precipitation of cholesterol in macrophages, which leads to the formation of lipid streaks.

The oxidation of harmful LDL cholesterol is supported in smokers by increasing the levels of lipid peroxides reported in the plasma of smokers. The sensitivity of low-density lipoprotein to oxidative stress decreased in smokers after quitting (18).

We found that the level of cholesterol increased significantly in a smoker compared to non-smokers. The various components that stimulate lipid change through smoking are (a) an increase in nicotine in the adrenal sympathetic system primarily to increase the excretion of catechol amines which leads to increased lipolysis and an increase in plasma-free fatty acid concentration leading to increased excretion of fatty acids free from liver and fat Triglycerides in the liver. With VLDL cholesterol in the circulatory system (b) the presence of hyperinsulinemia in smokers broadens the lipoprotein movement leading to increased lipid levels in smokers (13).

The elevation in blood lipid levels in smokers may be through tissue lipolysis induced by the catecholamine and adenyl cyclic axons as suggested in the graph (1).

Relationship between liver enzyme and lipid

There is an incredible relationship between liver and fat because all parameters of the lipid profile are synthesized and metabolized in the liver (19). The current study revealed that serum TG was mainly higher in heavy smokers as opposed to non-smokers along these lines which revealed an immediate reaction relationship. The lipid alteration in cigarette smokers may be due to nicotine stimulating the secretion of catechol amines resulting in an increased lipolysis rate and an increase in plasma free fatty acid (FFA) concentration which also leads to an increased release of hepatic FFA and hepatic triglycerides. With VLDL into circulation (16). From the results of this work one can confirm that total cholesterol levels are influenced by the dose response of tobacco exposure.

Increased levels of GOT and GPT commonly occur with accumulation of triglycerides and fatty liver degeneration, and likewise there has been a wealth of data in the past decade to link these phenomena to health risks outside the liver, such as type 2 diabetes, metabolic syndrome, insulin resistance and cardiovascular disease, GOT serum activities may predict mortality from cardiovascular or cerebral vascular events (20). The Table indicates (3) and to the emergence of a significant correlation in the concentration of GOT levels in the blood serum with GPT, this Pearson Correlation is 0.881 at significant ($P<0.05$) for control. However, do not found correlation between cholesterol and liver enzymes for control.

There are significant correlation in the concentration of GOT levels in the blood serum with concentration of cholesterol in blood serum, this Pearson Correlation is 0.845 at significant ($P<0.05$) for smoker, but do not found correlation GPT with other parameters at significant ($P<0.05$) for smoker as indicates in Table (4), and note in this table also there are correlation between concentration of GOT and GPT levels in blood serum, this Pearson correlation is 0.845 at significant ($P<0.05$) for smokers.

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تأثير تدخين السجائر على وظائف الكبد ومستويات الدهون: دراسة مقارنة بين المدخنين وغير المدخنين في محافظة بابل

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الخلاصة

يعد تدخين السجائر في الوقت الحاضر أحد أكبر المشاكل التي تؤثر في الصحة العامة في جميع أنحاء العالم، ويعد عامل خطر لاضطرابات الأوعية الدموية الطرفية وأمراض القلب. تعد السيطرة على وظائف الكبد والكوليسترول الكلي في الجسم مهمة جدًا وهي تعطي مؤشر لأمراض القلب والأوعية الدموية في المستقبل بين المدخنين. الهدف من الدراسة الحالية هو تقييم تأثير تدخين السجائر على مستويات إنزيمات الكبد في مصل الدم: الأسبارتات أمينوترانسفيراز (GOT) ، ألانين ترانس أميناز (GPT) ونشاط مستويات الكوليسترول بين المدخنين العراقيين في محافظة بابل ، مقارنة بمجموعة السيطرة من الاصحاء. الأفراد (غير المدخنين) كمجموعة سيطرة (جميعهم من الذكور). تم إجراء الدراسة الحالية على أربعين مدخناً عراقياً يدخنون ما لا يقل عن 10 سجائر يومياً لمدة 15 سنة على الأقل. تضمنت الدراسة عدد من المجاميع للمدخنين التي تتراوح أعمارهم بين 15-55 سنة. تم اضافة مجموعة من الافراد غير المدخنين (السيطرة ، عدد = 20) من نفس الفئة العمرية للمقارنة الإحصائية. تم سحب عينات الدم عن طريق الوريد من كل فرد ولكل المجاميع. قدرت مستويات الكوليسترول واختبار وظائف الكبد في مصل الدم للمدخنين وغير المدخنين عن طريق عدة التشخيص (شركة راندوكس - المملكة المتحدة) باستخدام التحليل، أظهرت نتائج الدراسة الحالية وجود أعلى مستوى معنوي للكوليسترول الكلي وانزيمات الكبد (ALT و AST) في مجموعة المدخنين مقارنة بمجموعة غير المدخنين وبمستوى معنوي (P<0.05). كما أظهرت النتائج الحالية وجود علاقة ارتباط معنوية موجبة بين عمر المدخنين وانزيم الكبد GOT في الدم وانزيم الكبد GPT وفعالية الكوليسترول (العالي) في مجموعة الفئات العمرية بين 45 إلى 55 سنة مقارنة بمجموعة السيطرة. بالإضافة إلى أن الكوليسترول الكلي وأنزيمات وظائف الكبد كانت مرتبطة بشكل إيجابي مع مدة التدخين وبمستوى معنوي (P<0.05)، ان تدخين السجائر يؤدي إلى الإجهاد التأكسدي عن طريق توليد الجذور الحرة بآلية بيروكسيد الدهون التي تتأثر بكثرة التدخين. للتدخين تأثير سلبي على اختبار وظائف الكبد والذي يجب تجنبه بعناية باتباع الاجراءات الوقائية اللازمة لتجنب حدوث أمراض القلب والأوعية الدموية في المستقبل.

الكلمات المفتاحية: إنزيمات الكبد ، الأسبارتات أمينوترانسفيراز (GOT) ، ألانين ترانس أميناز (GPT) ، الكوليسترول (TG)