The Effect of Cigarette Smoking on Blood and Biochemical Parameters: A Comparative Study Among Male Smokers and Non-Smokers In Baghdad City

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Abstract

   The impact of smoking on human health is remarkable and can lead to death. This research was performed to test the effects of cigarette smoking on some parameters that are considered as signs of critical problems in human body. The study was carried out on fifty Iraqi male smokers in Baghdad city, who smoked at least 10 cigarettes per day for at least 15 years. The group includes 25 male smokers with an age range of 20 -55 years and 25 male non-smokers who were collected with the same range of age for statistical comparison. The results of the study revealed significant increases in blood parameters, including hemoglobin (Hb, 16.0917 (g/dl)   , packed cell volume (PCV, 49.2%), red blood cells (RBC, 5.4763 $X10^{12}$/L), white blood cell (WBC, 12.5565 $X10^9$/L), and platelet (PLT, 430.000 $x10^{12}$/L). Similar effects were observed in relation to the serum biochemical parameters o of kidney function (urea, 53.2400 mg/ dl; creatinine, 1.5480 mg/ dl) as well as liver function (alanine aminotransferase, ALT, 104.9200 U/l; aspartate transaminase, AST, 122.3040 U/l; alkaline phosphatase, ALP, 337.4000U/l); total serum bilirubin, TSB, 0.6780 mg/ dl). However, significantly decreased levels of total protein (60.6800 mg/ dl) and uric acid (4.2400 mg/ dl) were recorded in cigarette smokers when compared with non-smokers group.

Keywords: Smoking, Liver Enzymes, Kidney Enzyme, Total Protein And Blood Parameters.

تأثير تدخين السجائر على معايير الدم والكيمياء الحيوية: دراسة مقارنة ما بين المدخنين وغير المدخنين من الذكور في مدينة بغداد

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الخلاصه

   تأثير التدخين على صحة الإنسان ملحوظ ويمكن أن يؤدي إلى الوفاة. تم إجراء البحث لتقييم تأثير تدخين السجائر على بعض العوامل التي تعتبر علامة على مشكلة النقطة الحرجة في الجسم. أجريت الدراسة على خمسين مدخنا عراقيا في مدينة بغداد يدخنون ما لا يقل عن 10 سجائر يوميا لمدة 15 سنة على الأقل. تضم المجموعة المدخنين 25 ذكور تتراوح أعمارهم بين 20–55 سنة. تم جمع غير الذكور 25 مجموعة من الذكور مع نفس النطاق من العمر للمقارنة الإحصائية. كشفت نتائج الدراسة عن زيادة ملحوظة في قياسات

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الدم الهيموغلوبينdl / g 16.0917   وحجم الخلايا المعبأة ٪49.2 PCV وخلايا الدم الحمراء 5.4763

L / 10X^{12} وخلايا الدم البيضاء L / X10^9 12.5565 والصفائح الدموية 10^{12} × 430.000 / لتر

بالإضافة إلى المعلمات الكيميائية الحيوية واختبار وظائف الكلى في الدم: اليوريا 53.2400 ملغم / دل

والكرياتينين 1.5480 ملغم / دل. واختبارات وظائف الكبد في المصل: alanine aminotransferase

ا / U 104.9200 ALT، AST 122.3040 U Aspartate transaminase. الفوسفاتيز القلوي

ALP 337.4000U / لتر ومصل البيليروبين الكلي TSB 0.6780 ملغ / دل.نخفض بشكل ملحوظ في

البروتين الكلي 60.6800 ملغم / دل وحامض اليوريك 4.2400 ملغم / دل في مدخني السجائر مقارنة مع

مجموعة غير المدخنين.

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Introduction

Smoking is one of the issues with most popular intemperance in the modern world. It has been involved as an etiological agent for various chronic diseases, including a variety of infections, cancers, heart diseases and respiratory illnesses [1, 2], Tobacco is an etiological agent for many chronic diseases, inclusive a variety of infections, cancers, heart diseases, and respiratory illnesses such as chronic obstructive pulmonary disease (COPD). These conditions are associated with impairment in the balance between cell growth and cell death and collectively cause high rates of morbidity and mortality in today's society [3]. Tobacco has both acute and chronic effects on hematological parameters and physiological alteration in human body [4, 5]. Smoking is a familiar cause of the increase in hemoglobin (Hb) concentration that is believed to be mediated by the exposure to carbonmonoxide. Carbonmonoxide binds to Hb to form carboxyhemoglobin, an inactive form of hemoglobin having no oxygen carrying capacity. Carboxyhemoglobin conveys the Hb detachment curve to the left side, resulting in a reduction in the ability of Hb to deliver oxygen to the tissues. To recompense the decreased oxygen delivering capacity, smokers preserve a higher hemoglobin level than non-smokers [6].

Smoking is recognized as a risk factor of liver cancer; Burns [7] demonstrated that smoking causes an increased risk of cirrhosis and may reversely affect the progress of chronic liver diseases. Researches revealed that smoking leads to deterioration in the renal function of patients with kidney diseases involving hypertensive nephrosclerosis and glomerulonephritis [8]. Therefore, the aim of the present study is to explore differences between smokers and non-smoker in different blood parameters and biochemical serum markers of the functions of the kidney and liver in human body.

Materials and methods

Design of the study

The experiment was conducted on healthy male subjects (50) in Baghdad city whose ages ranged from 20 to 55 years. The donors were divided into two groups; 25 smokers and 25 non-smokers (control). The blood samples were drawn by venipuncture (5 ml), placed in a non-heparinized tubes , and centrifuged at 3000 rpm for 15 minutes. Estimation of HB, PCV, WBC, RBC and platelets in blood samples was performed in EDTA-coated tubes. Assessments of blood cell count was performed using Mindray BC-30 automated hematological analyzer, which could perform 18 hematological parameters with high accuracy and precision. Each sample was left to clot and centrifuged for 15min in 3000rpm. Next, the levels of the serum parameters of the functions of the kidney (urea and creatinine) and the liver (ALT, AST, ALP, and TSB), as well as uric acid and total protein were estimated using Cobas-C311 High performance diagnostic reagent kits.

Statistical analysis

Statistical analysis was performed with the statistical package for social sciences (SPSS) 21.0 and Microsoft Excel 2013 To determine the significant differences between the values (Mean ± SEM) of the test and the control groups,   t-test (at $p \leq 0.05$) was applied

Results and Discussion

Blood parameters

This study showed a significantly elevated white blood cells count in smokers (12.5565×10^9/L) compared to non-smokers (6.1×10^9/L) as shown in Table-1.

The positive relationship between WBC and male smokers shown in the present study was also prviously demonstrated [9].

In addition, the values of hemoglobin, RBCs count, hematocrit (PCV) and red cell indices were compared between smokers and non-smokers. The smokers had significantly higher levels of HB (16.0917 g/dl), PCV (49.2917 %) and RBC (5.4763×10^{12}/L) as compared with nonsmoker (Table-1). Cigarette smoke has 4000 compounds, among which CO and tars are the major toxic substances. CO distributes rapidly through the alveolar capillaries, where it combines firmly to Hb (with binding ability of 200-250 times greater than that of O2), forming **c**arboxyhaemoglobin HbCO. Such a reaction results in tissue hypoxia, leading to increased values of RBCs, Hb and PCV [10].

In healthy individuals, smoking triggers a boost in Hb levels, likely mediated by exposure to carbon monoxide (CO) which binds to Hb to form HbCO. Mean Hb and HbCO levels increase gradually with the duration of chronic exposure to smoke. HbCO also correlates with the development of polycythemia [11].

The variations detected between the composition of peripheral blood leukocytes and erythrocytes of smokers and non-smokers are supposedly the reflections of the effects of the gaseous and solid phases of cigarette smoke toxic substance on the bone marrow. Another mechanism is possibly the adaptive immunologic reactions of the body to long-term active smoking [12].

It was concluded that readjustment to carbon monoxide inhaled in cigarette smoke is reflected by an increased red cell mass and hemoglobin [13].

Moreover, the value of PLT count (430.000×10^{12}/L) was significantly higher in smokers than non-smokers (160.6250 (10^9/L)). The present research is in agreement with the results of previous reports [14,15]. Mobarrez *et.al.* [16] clarified that acute smoking can cause endothelial damage which leads to an increase in platelet count. Moreover, the platelet production is controlled by hormonal metabolisms that might be potentially impaired via smoking, causing the production of increased numbers of platelets . Higher circulating thrombopoietin levels were reported in cigarette smokers, which is a humoral growth factor that is released in response to increasing platelet production [17].

Table 1-Comparison of blood parameters in cigarette smokers and non-smokers

Parameters	Control n =25			Treatment n =25			P-Value	Significant P ≤ 0.05
HB. (g/dl)	14.2000	±	0.2818	16.0917	±	0.2005	0.0000	S
WBC (10^9/L)	6.1458	±	0.1461	12.5565	±	0.1564	0.0000	S
RBC (10^{12}/L)	3.9200	±	0.0578	5.4763	±	0.0523	0.0000	S
PCV.%	44.0417	±	0.9640	49.2917	±	0.7134	0.0001	S
PLT(10^{12}/L)	160.6250	±	5.9172	430.000	±	15.6031	0.0000	S

Mean ± S.E   S.E= standard error, S= Significant
Biochemical parameters:

The results of the statically analysis revealed significant elevations in serum AST(122.3040 U/l) and ALP (337.4000U/l) levels, along with a non-significant change in ALT (104.9200 U/l) and TSB (0.6780 mg/ dl) levels in cigarette smokers as compared with the control (AST 80.3200 U/l , ALP 80.3200U/l)) (167.5200 U/l) , ALT 88.1200 U/l, TSB 0.6640 mg/ dl). as shown in Table-2**.**

The rise in the levels of ALT and AST is likely because of the deleterious effects of the chemical compounds of tobacco smoke on liver cells that lead them to higher secretion of these enzymes through inflammatory pathways, or due to the aggravation of pathogenic actions of other compounds on the liver [18]. These enzymes, in addition to other liver enzymes, can be used for the diagnosis of liver disorders such as hepatitis and cirrhosis [19].

Furthermore, the results demonstrated that smokers has significantly lower serum levels of total protein and uric acid (60.6800 mg/ dl and 4.2400 mg/ dl, respectively) in comparison with non-smokers (80.6400 mg/ dl and 5.3640mg/ dl, respectively), as exhibited in Table-2. High cigarettes consumption was correlated with low total protein levels because of the attachment between the three factors: tobacco, coffee consuming and alcohol use present high level of correlation and interaction is point between these factors [20]. Researches also indicated the reduction in antioxidant levels, including uric acid, in smokers [21] is caused by both chronic exposures to cigarette smoke, that is a major source of oxidative stress, and low intake of dietary antioxidants [22].

The result of the kidney function tests showed high significant in urea (53.2400 mg/ dl) and creatinine (1.5480 mg/ dl). as exhibited in Table-2.

Tobaccos was shown to cause a leakage in endothelial cells, while nicotine induces smooth muscle cell proliferation [23].

Table 3-Comparison of biochemical parameters in the serum of cigarette smokers and non-smokers

Parameters	Control n =25			Treatment n =25			P-Value	Significant P ≤ 0.05
AST(U/l)	80.3200	±	36.8892	122.3040	±	90.1571	0.0414	S
ALT(U/l)	88.1200	±	3.0569	104.9200	±	10.8417	0.1489	NS
TSB (mg/ dl)	0.6640	±	0.0532	0.6780	±	0.0802	0.8855	NS
ALP(U/l)	167.5200	±	16.7984	337.4000	±	49.9534	0.0036	S
Urea (mg/ dl)	29.7200	±	0.4454	53.2400	±	0.9350	0.0000	S
creatinine (mg/ dl)	1.0264	±	0.0574	1.5480	±	0.0874	0.0000	S
Uric acid(mg/ dl)	5.3640	±	0.1413	4.2400	±	0.0632	0.0000	S
Total protien(mg/ dl)	80.6400	±	1.1134	60.6800	±	1.6691	0.0000	S

Mean ± S.E S.E= standard error, S= Significant, NS = Non- Significant
WHY THERE ARE NO CONCLUSIONS?

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