The effect of cigarette smoking on some blood parameters, blood pressure and renal function test.

تأثير تدخين السكائر على بعض معايير الدم وضغط الدم واختبار وظيفة الكلية

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Abstract

This study investigated the effect of cigarette smoking on some hematological test profiles which comprised of complete blood count (CBC) and blood preasure, heart rate and renal function test including blood urea nitrogen (BUN), serum creatinine (Scr). The study included 60 males from students and the staff at Basrah University's: (30) smokers and (30) nonsmokers aged 20-49 years old. The results Showed that white blood cell counts WBC was significantly higher ($p<0.05$) in 20-49 years of age smokers group compared with nonsmokers group , red blood cell RBC counts , hemoglobin (Hb) and hematocrit (Hct) were significantly higher ($p<0.05$) in 30-49 years of age smokers group compared with the same ages of nonsmokers, mean corpuscular volume (MCV) was significantly increased ($p<0.05$) in 20-49 years of age smokers compared with control group, while mean corpuscular hemoglobin concentration (MCHC) was significantly lower in 30-49 years of age smokers compared with the same ages of nonsmokers , but no changes significant in mean corpuscular hemoglobin (MCH) and platelet (Plt) count were observed in all age groups ($p<0.05$). The results Showed that Lymphocytes (Lym) was significantly higher ($p<0.05$) in 20-49 years of age smokers compared with the same ages of nonsmokers, eosinophils (Eosi) was significantly higher ($p<0.05$) in 40-49 years of smokers compared with the same ages of nonsmokers . The neutrophil (Neut), monocyte (Mono) and basophil (Baso) showed a non-significant changes in all age groups (($p<0.05$)). Whereas systolic and diastolic blood pressures and heart rate showed a significant increases in the 30-49 years of age smokers compared with the same ages of nonsmokers, serum creatinine (Scr) and blood urea nitrogen (BUN) were significantly higher ($p<0.05$) in 20-49 years of age smokers compared with the same ages of nonsmokers. The study aims to identify the impact of cigarette smoking on some hematological test profiles which comprised of complete blood count , blood pressure, heart rate and renal function test.

Conclusion: The cigarette smoking causes significant higher increases in WBC, RBC, Hb,Hct,,MCV , Lym and Eosi. count Significantly lower in MCHC ,while no significant in MCH, Plt,, Neut, Mono and Baso. Also there was a significant increases in systolic and diastolic blood pressure, heart rate, serum creatinine and blood urea nitrogen.

Keywords: cigarette smoking, complete blood count, blood pressure, renal function.

الخلاصة

هدفت الدراسة الحالية إلى معرفة تأثير التدخين على اختبار الدم التي تتألف من تعداد الدم الكامل وضغط الدم ومعدل ضربات القلب واختبار وظيفة الكلى بما في ذلك الكرياتينين و اليوريا ، شملت الدراسة 60 رجلا من الطلاب والموظفين في جامعة البصرة: (30) مدخن و (30) غير مدخن الذين تتراوح أعمارهم بين 20-49 سنة. كان هناك ارتفاع معنوي في عدد كريات الدم البيضاء في مجموعة المدخنين بين 20- 40 سنة مقارنة مع غير المدخنين لنفس العمر وارتفاع معنوي في عدد كريات الدم الحمراء وتركيز الهيموكلوبين والهيماتوكريت في مجموعة العمر 30-49 سنة مقارنة مع غير المدخنين لنفس العمر ، اما معدل حجم كريات الدم سجل ارتفاع معنوي في مجموعة المدخنين بين 20- 40 سنة مقارنة مع غير المدخنين لنفس العمر ، بينما هناك انخفاض معنوي في معدل تركيز هيموكلوبين الخلية في مجموعة المدخنين بين 30- 49 سنة مقارنة مع غير المدخنين ، و لا يوجد فرق معنوي في كل من معدل هيموكلوبين الخلية والصفائح الدموية وبينت النتائج وجود ارتفاع معنوي في الخلايا اللمفاوية في مجموعة المدخنين بين 20- 40 سنة عند مقارنتها مع غير المدخنين وكذلك وجود ارتفاع

معنوي في الخلايا الحمضية في مجموعة المدخنين فوق 40 سنة مقارنة مع غير المدخنين لنفس العمر بينما لايوجد فرق معنوي في كل من الخلايا العدلة والخلايا وحيدة النواة وخلايا القاعدية ، اما ضغط الدم الانبساطي والانقباضي ومعدل ضربات القلب سجّل ارتفاع معنوي في المدخنين فوق 30 سنة مقارنة مع غير المدخنين لنفس العمر ,وجود ارتفاع معنوي في كل من الكرياتينين واليوريا في مجموعة المدخنين بين 20- 40 سنة مقارنة مع غير المدخنين لنفس العمر.

Introduction:

Although tobacco has dangerous effect on human health, it still highly consumed throughout the world. Smoking is one of the most common addictions of modern times. Its etiological agent for various chronic diseases, including a variety of infections, cancers, heart diseases and respiratory illnesses [1]. Cigarette smoke (CS) contains over 4000 compounds, including at least 200 toxicant, 80 known or suspected carcinogens. Moreover, cigarette smoking generates many toxic and carcinogenic compounds harmful to the health, such as nicotine, nitrogen oxides, carbon monoxide, hydrogen cyanide and free radicals [2]. Nicotine is commonly consumed via smoking cigarettes, cigars or pipes [3]. Carbon monoxide in tobacco smoker exerts a negative effect on the heart by reducing the blood's ability to carry oxygen .Although cigarette smoking is a strong risk factor for cardiovascular disease, its relationship with hypertension remains unclear [4].

Cigarette smoking contributes to the development of many chronic diseases associated with age [5]. One major risk factor for morbidity and mortality among adults was smoking cigarette. Smoking responsible for 90% of lung cancer cases among males, the major risk factors that cannot be changed which associated with heart disease was age, smokers who are still relatively young can reverse some lung damage by quitting smoking, but in middle-aged and older smokers some damage seems to be irreversible [6]. The main way for the arrival of cigarette smoke into the smoker's blood by gaseous exchange that occurs in the lungs, so the lungs damaged larger than the rest of the members of the body.

Materials and Methods:

The sample was selected at random (60) from male volunteers from the students and the staff at Basrah University's: (30) smoker and (30) nonsmokers were stratified by age into three groups of 20-29, 30-39, 40-49 years equal number in each group (10),: The sample of this study were normotensive and free of other cardiovascular risk factors and were not taking any medications. Each individual included in this study was asked about cigarette smoking habit, number of cigarettes consumed per day, and duration of smoking. Blood pressure (systolic and diastolic) was measured from the brachial artery, using a standard mercury sphygmomanometer in the right arm in a sitting position, after a steady state at 3-5 minutes.

Three milliliter of blood was collected from the vein and injected in EDTA test tube gentle mixing was done immediately to ensure complete anticoagulation of the blood. Serum was obtained by Centrifugation 1 ml of blood in EDTA test tube for hematological tests (WBC, RBC, Hb, Hct, plt, Mcv, Mch , Mchc, lumph, mono, Baso, Eso) by using Sysmax device , 2 ml of blood for renal functions tests (Bun, Scr) by using kit of Fabricante Biolabo

Statistical analysis:

The researcher used the statistical program (spss) in data processing and extract results, the statistical methods used (ANOVA test, arithmetic mean test, and standard deviation).

Results:

(table 1) showed a significant increase (p<0.05) in WBC,RBC count, hematocrit valve and MCV in smokers compared with non- smoker of the same age group. The MCHC was significantly decreased (p<0.05) in smoker of age groups 30-40 compared with non- smoker of the same age group. A non-significant changes were observed in MCH valves and platelet count in all age groups.

(Table 1) The effect of smoking on hematological parameters of smoker and nonsmoker groups (value are mean ±SD).

No.	hematological parameters	age/yrs	smokers	Non-smokers	L.S.D (p<0.05)
1	WBC (Cells/mm3)	20-29	**8.631^c** ±1.90	6.550 ±1.706	1.972
		30-39	**8.786^b** ±2.363	7.201 ±1.862	
		40-49	**10.061^a** ±2.356	7.618 ±1.537	
2	RBC(Million) Cells/mm3)	20-29	5.175 ±0.226	5.619 ±0.277	1.009
		30-39	**6.491b** ±1.639	5.278 ±0.263	
		40-49	**7.459^a** ±1.325	5.111 ±0.297	
3	Hb (g/dl)	20-29	15.350 0.607	15.530 ±0.653	2.342
		30-39	**17.530^b** ±3.997	14.620 ±0.687	
		40-49	**20.913^a** 3.533	15.248 ±1.848	
4	HCT%	20-29	47.04 ±1.093	47.570 ±1.911	6.402
		30-39	**52.960b** ±10.950	45.000 ±2.168	
		40-49	**59.847a** ±8.333	45.319 ±6.988	
5	MCV (fl)	20-29	**91.040^c** ±3.807	84.770 ±4.067	5.099
		30-39	**114.640^a** ±8.116	84.140 ±3.104	
		40-49	**101.542^b** 3.788	87.290 ±6.018	
6	MCH (pg)	20-29	25.900 ±0.917	27.690 ±1.401	**N.S**
		30-39	27.260 ±2.456	26.910 ±2.100	
		40-49	27.830 ±3.356	29.380 ±2.023	
7.	MCHC (g/dl)	20-29	31.988 ±0.676	32.670 ±0.940	1.561
		30-39	**29.214^b** ±1.136	32.380 ±1.270	
		40-49	**29.048^a** ±2.819	33.513 ±1.592	
8.	PLT 103/mL	20-29	248.813 ±43.590	250. 772 ±25.869	**N.S**
		30-39	260.500 ±54.324	268.115 ±86.724	
		40-49	245.251 ±66.050	255.651 ±49.330	

* (a,b signify p<0.05).

(Table2) showed a significant increase (p<0.05) in Lymph, eosi count in smokers compared with non- smoker of the same age group. A non-significant changes were observed in Neut, Mono and baso count in all age groups.

(Table2):The effect of smoking on differential Leucocyte count in smoker and non-smokers groups. (value are mean ±SD).

NO.	%Differential Leucocyte	age/yrs	smoker	Non-smoker	L.S.D
1.	NEUT %	20-29	56.540 ±12.237	54.250 ±13.295	N.S
		30-39	55.590 ±9.926	55.440 ±8.421	
		40-49	46.387 ±6.347	58.475a ±3.039	
2.	LYMPH %	20-29	**43.710b** ±9.188	34.610 ±11.769	**9.368**
		30-39	**42.880c** ±10.180	35.860 ±7.443	
		40-49	**43.787a** ±7.702	33.275 ±5.606	
3.	MONO %	20-29	6.810 ±2.717	7.950 ±2.199	N.S
		30-39	6.940 ±2.188	6.400 ±2.957	
		40-49	6.251 ±2.914	7.502 ±1.954	
4.	Eosi %	20-29	1.990 ±1.074	1.900 ±1.360	1.224
		30-39	2.230 ±1.380	2.170 ±1.208	
		40-49	**3.126[a]** ±1.691	2.125 ±0.670	
5.	BASO %	20-29	0.500 ±0.377	0.380 ±0.239	N.S
		30-39	0.360 ±0.164	0.330 ±0.163	
		40-49	0.442 ±0.127	0.425 ±0.275	

*(a,b signifiy p<0.05)

The current study, as shown in the (table 3) indicates a significant increases of systolic and diastolic- blood pressure ($p<0.05$) in smokers compared to non-smokers groups

(Table 3) : The effect of smoking on blood pressure of smokers and nonsmokers groups (value are mean ±SD).

NO.	Blood p.	age	smoker	Non-smoker	L.S.D
1.	SYS mmHg	20-29	119.00 15.289	120.800 6.358	11.06
		30-39	133.500[b] 19.927	119.500 7.852	
		40-49	140.600[a] 11.502	122.000 20.215	
2.	DIA mmHg	20-29	76.200 15.375	75.80 8.715	9.75
		30-39	86.250[b] 12.012	79.250 7.888	
		40-49	89.666[a] 8.213	78.600 10.945	

The (table 4) showed a significant increase in heart rate ($p<0.05$) in smokers compared to non-smokers groups.

(Table 4): Heart rate in smokers and nonsmokers male (value are mean ±SD).

No.	HEART r.	Age/yre	smoker	Non-smoker	L.S.D
1.		20-29	82.800 10.643	75.600 11.777	10.554
		30-39	86.000[b] 12.821	71.750 18.191	
		40-49	88.200[a] 14.788	80.500 5.972	

*(a,b signify $p<0.05$).

The (table 5) showed a significant increase ($p<0.05$) in smokers compared to non-smokers groups.

(Table 5): Renal function test in smokers and nonsmokers males (valve are mean ± SD).

NO.	Renal Function	Age/year	Smoker	Non-smoker	L.S.D
1.	Scr	20-29	2.08[c] ±1.037	1.142 ±0.361	1.186
		30-39	2.32[b] ±0.969	0.902 ±0.158	
		40-49	2.96[a] ±1.213	1.025 ±0.419	
2.	BUN	20-29	34.3[d] ±9.978	28.5 ±19.213	5.901
		30-39	38[c] ±5.958	28.25 ±9.912	
		40-49	41.3[a, b] ±7.885	30.2 ±17.23	

*(a,b.c signify a $p<0.05$).

Discussion:

The current study showed significant differences in hematological parameters of smokers and non-smokers where the WBC, RBC, Hb, HCT and MCV were significantly high, whereas MCHC was significantly low in smokers as compared to non-smokers. We did not find any significant difference in, MCH, and PLT level We found that the smokers had significantly higher in WBC count ($p<0.05$) compared to non-smokers this result agree with that of [7,6,8] .

The relation of high WBC count with smoking could be due to the presence of a subclinical inflammatory reaction [9]. The leukocytosis may simply be a marker of smoking-induced tissue damage, this high count can promote cardiovascular diseases through multiple pathologic mechanisms that mediate inflammation, plug the microvasculature, induce hypercoagulability and promote infarct expansion [10]. Some researchers
 found that the blood leukocyte count was increased by as much as 30% in smokers, because the biologic mechanisms for a persistent effect of smoking on white blood cell count [11].

The current study showed the smokers had significant increase in RBC count ($p<0.05$) compared to non-smokers this rustle agree with [12] and Contrary to [13] . The increase of RBC count is termed as polycythemia and very high of RBC mass slows blood velocity and increase the risk of intravascular clotting, coronary vascular resistance decreased coronary blood flow, and a predisposition to thrombosis [14] . The mechanism by which polycythemia causes thrombosis is still under investigation, but smoking cigarettes creates a unique condition of combined polycythemia to chronic hypoxia, leading to elevated red cell production because of an elevated carboxyhemoglobin level with concomitant plasma volume reduction. [15]. Some possible mechanisms are reported by which cigarette smoking could cause such changes in the red blood cells. Carbon monoxide, one of the chemicals identified in tobacco smoke, may induce hypoxia, usually the body responds to hypoxia by increasing the number of erythrocytes [16].Excessive tobacco can be result in polycythemia because this situation created a real oxy-carbon poisoning, so there was an extra oxygen demand and production of red blood cells increases to cope [17].

We found that the smokers had significantly higher hemoglobin concentrations ($p<0.05$) compared to non-smokers this rustle agreement with [18]. Elevated levels of hemoglobin are correlated with increased numbers or sizes of RBCs. and are consistent with other studies [19,20]. Both high and low hacmoglohin levels increase mortality and morbidity, smoking cessation lowered mean hemoglobin 1.6 g/dl compared with nonsmokers [6]. Hemoglobin concentrations were significantly associated with increasing age in cigarette smoking men . The study were demonstrated other studies support by [21,22]. Who found that increasing age was significantly associated with higher hemoglobin concentrations and smoking was significantly associated with higher hemoglobin, smoking cessation becomes more difficult when habit was developed early in life because of the nicotine tolerance that is built up through years of smoking, [6]. Hematocrit values were also significantly higher in smokers than those of non-smokers ($p<0.05$) and consistent with previous studies [23]. Higher levels of hematocrit may cause polycythemia vera (PV), a myeloproliferative disorder in which the RBCs are produced excessively by bone marrow and also related to an increased risk of development of atherosclerosis and cardiovascular disease [18].

An increased hematocrit may reduce the coronary blood flow and increase adhesion of platelets to the aortic subendothelium, which may contribute to the early formation of atherosclerosis and thromboembolic disease [16].

MCV, MCH and MCHC are three main red blood cell indices that help to measure the average size and hemoglobin composition of the red blood cells . We found an increase in MCV and decrease in MCHC levels in smokers than those of non-smokers ($p<0.05$) and this result is consistent with previous studies [18].

MCV indicates the size of a red blood cell and presence of red cells smaller or larger than normal size means the person has anemia, elevated levels of MCV indicates that subjects might suffer from megaloblastic , hemolytic, pernicious or macrocytic anemia usually caused by iron and folic acid deficiencies [18].

The increase in hemoglobin, hematocrit, MCV could be due to the inhaled carbon monoxide gas (CO), which is one of the inhaled components of cigarette smoke. CO present in cigarette smoke in more than 600 times the concentration considered safe in industrial plants. A smoker's blood typically contains 4 to 15 times as much CO as that of a nonsmoker. CO combines reversibly with oxygen-carrying sites on the hemoglobin molecule by about ranging from 210 to 240 times greater than that of oxygen, which results in decreased oxygen-carrying capacity of the blood, this decrease is compensated by an increase in hemoglobin and hematocrit [24] .

MCH is the average weight of hemoglobin that is present inside a single red blood cell whereas MCHC denotes the amount of hemoglobin in a specific volume of 'packed' red cell. We found significantly low value of MCHC ($p < 0.05$) in smokers indicating hypochromic anemia and might be due to paucity of folic acid or vitamin B12 or thyroid problems [18].

This study showed that Lymphocytes and eosinophils were significantly higher in the smokers than in smoker group ($p < 0.05$) This result was in agreement with [25]. Who reported that Lymphocytes strongly associated with number of cigarettes per day and packer per years, may be due to the glycoprotein from tobacco leaf may stimulate lymphocyte spread and differentiation [26]. And may be due to residual chronic inflammation of respiratory tract [25] . The lymphocytosis can be attributed to chronic tissue damage and inflammation produced by toxic smoke products. This result was in agreement with [27]. Also [13], reported that leukocytosis in smokers is mainly attributable to an increased lymphocyte count.

The increase in eosinophils in smoker groups is correlated with a study published in British Journal of Hematology The possible cause of increase in Eosinophil count may be due to smoking allergy in respiratory tract [28] .Or may be due to cannabis plants are contaminated with a number of fungal spore organisms, which cause secondary eosinophilic pneumonia [27]. They exhibit specialized function in certain disorders and are conspicuously active in their protection against foreign substances like smoke and cannabinoids [26]. The study demonstrated no significant differences in neutrophil, monocytes and basophil count. This result was in agreement with [13,25].

This study showed a significant increase in blood pressure in the smokers than in control (non smoker) group (($p < 0.05$)) This result was in agreement with [29,12].

Some epidemiologic studies reported a relation between smoking and blood pressure. [30]. Reported that each cigarette induces a similar and statistically significant acute increase in both blood pressure and heart rate which lasts for a long time and could be detected in 24-hour ambulatory blood pressure monitoring.

The cigarette smoking causes sympathetic activation, oxidative stress, and acute vasopressor effects that are associated with increased markers of inflammation that are linked with hypertension [31]. There are several potential biological mechanisms through which exposure to cigarette smoke may lead to high blood pressure, cigarette smoking also increases blood pressure through stiffening arteries, particularly with deleterious effects of chronic smoking that leads to the development of hypertension [32].

Smoking causes a significant increase in heart rate [33]. Smoking or intravenous nicotine administration increases arterial blood pressure and heart rate in humans [34]. This can be explained by an acute stimulation of the sympathetic nervous system causing heart rate increases [35]. The increase a of BP and HR derived from smoking has been attributed to the activation of the sympathetic nervous system with release of noradrenalin and adrenalin [36].

The results showed that there were an elevated values of serum creatinine in the smokers than in no smokers ($p<0.05$), this result was in agreement with [37,38]. These results can be explained as the cigarette smoking increases Renovascular resistance that lead to a significant fall in glomerular filtration rate (GFR), the decrease in GFR will lead to a decrease in distal tubular flow rate which leads to increase of urea reabsorption [39].

We found significant increase in BUN in the smokers than in no smokers ($p<0.05$) this result was in agreement with [40]. BUN is the breakdown product of protein in the blood. Studies have demonstrated that the cigarette smoke which contains various oxidants, have a greatly oxidative effect on human plasma proteins and tissue microsomal proteins [41]. Therefore, the increased BUN levels in smokers may be due to the increased proteolytic degradation of oxidized Proteins.

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