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THE EFFECT OF CIGARETTE SMOKING ON HAEMATOLOGICAL PARAMETERS IN HEALTHY COLLEGE STUDENTS IN THE CAPITAL, BAGHDAD

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Abstract

It was shown that cigarette smoking (CS) was a major environmental factor which effect on hematological parameters in healthy person (no clinical signs). Thus the current study aimed to assess the levels of some hematological parameters in 40 individuals who smoking cigarette and compared them to 20 non-smokers group. there was no statistically significant difference in age, gender, body mass index (BMI), waist/Hip ratio, Waist/Height ratio and blood pressure between the smokers and non- smokers. The results of the study revealed a statistically significant increase in total White blood cells, Red blood cells, lemphocyte count, neutrophil count and hematocrit, and there are a highly significant increase in mean corpuscular hemoglobin (MCH) and hemoglobin in smokers, But there are no significant differences in MCV, MID and PLT, when we compares the haematological parameters between smokers and non-smokers.

1- Introduction:

Cigarette smoke is a complex mixture of chemicals. Some smoke components, such as carbon monoxide (CO), hydrogen cyanide (HCN), and nitrogen oxides, are gases. Others, such as formaldehyde, acrolein, benzene, and certain N-nitrosamines, are volatile chemicals contained in the liquidvapor portion of the smoke aerosol.(1) Still others, such as nicotine, phenol, polyaromatic hydrocarbons (PAHs), and certain tobacco-specific nitrosamines (TSNAs), are contained in the submicron-sized solid particles that are suspended in cigarette smoke. In view of this chemical complexity, cigarette smoke has multiple,

highly diverse effects on human health. (2) It is not unexpected that multiple chemicals in cigarette smoke can contribute to any single adverse health effect. The major health effects of cigarette smoke include: Cancer, noncancerous lung diseases, atherosclerotic diseases of the heart and blood vessels, and toxicity to the human reproductive system. According to the data of the World Health Organization, approximately 5 million people die globally each year from the diseases caused by smoking, and if this trend continues, it is expected that by 2030, that number would be 10 million. (3) Numerous studies indicated that smoking had adverse effects on human health and represented a predisposing factor for development of various pathological conditions and diseases, such as the chronic obstructive pulmonary disease, cancer, pancreatitis, gastrointestinal disorders, periodontal disease, metabolic syndrome, and some autoimmune diseases. (4) Cigarette smoking is associated with an increased risk of cardiovascular diseases, including coronary artery disease, peripheral vascular disease, ischaemic heart disease, atherosclerosis, myocardial infarction and stroke. The aim of the present study is to evaluate the effects of CS on some hematological parameters in male smokers. (5)

2- Methods:

Subject

Present study was carried out to investigate the relationship effect of cigarette smoking on haematological parameters in a group of clinically healthy volunteers. A total of 60 subjects were enrolled in the study, 40 smokers and 20 non-smokers in the age range 19-29 years. (6) The subjects were recruited from Dijlah University. The smokers were regularly consuming 10-20 cigarettes per day for at least 3 years. Each subject gave an information. Data on smoking habits and the amount of tobacco consumed were collected by a self-administered questionnaire to be filled in by the participants. (7) Subjects included in this study were free of evidence of active liver and kidney disease, chronic pancreatitis, gastrointestinal disease, inflammatory bowel, history of ischaemic heart disease or and diastolic blood pressure, endocrine disorders, infection, and hormonal therapy. (8)

Anthropometric and haematological measurements

In all the subjects blood pressure and anthropometric data (height, weight and waist circumference) were measured. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest. BMI was calculated as body weight (kg) divided by body height (m) squared. Blood pressure of each subject was measured with a mercury Sphygmomanometer and a standard stethoscope. (9) The subjects underwent the following tests: Blood pressure examination to rule out hypertension, estimation of red blood cell count, total leukocyte count, differential leukocyte count, platelet count, packed cell volume and

hemoglobin. Complete blood cell count was analyzed by Genex fully automatic haematological analyzer. (10)

Statistical analysis

Statistical analysis was performed using SPSS version 20.0 (SPSS Inc.). Before statistical analysis, normal distribution and homogeneity of the variances were tested using Kolmogorov-Smirnov test respectively. Groups were compared using Student's unpaired t test for parameters with normal distribution or Mann–Whitney test for parameters with non-normal distribution. Data are expressed as mean ± standard deviation. $P < 0, 05$ was considered significant. (11)

3- Results:

Table 1 shows that there was no statistically significant difference in age, gender, body mass index (BMI), waist/Hip ratio, Waist/Height ratio and blood pressure between the smokers and non- smokers.

Table (1): Differences of gender, age, body mass index, waist-to-hip ratio and waist-to-height ratio in smoker (1) and non-smoker (2) groups (mean±SD).

Parameter	Group 1 n=40	Group 2 n=20	P value	Significance
Gender (male)	(40)	(20)	-	-
Age (year) (min-max)	(19– 29)	(19-29)	-	Not significant
BMI(kg/m^2)	26.3±1.67	26.75±2.43	>0.05	Not significant
WHR	0.926±0.04	0.934±0.03	>0.05	Not significant
WHtR	0.561±0.04	0.55±0.05	>0.05	Not significant
Systolic B.P. (mm Hg)	124.23±5.03	126.78±9.34	>0.05	Not significant
Diastolic B.P. (mmHg)	76.92±4.70	79.85±5.85	>0.05	Not significant

Significant difference from group1 using t-test for two independent means
(HS : $P < 0.01$, S : $P< 0.05$, NS = $P> 0.05$).

Table 2 compares the haematological parameters between smokers and non-smokers which shows a statistically significant increase in total White blood cells, Red blood cells, lemphocyte count, neutrophil count and hematocrit, and there are a highly significant increase in mean corpuscular hemoglobin (MCH) and hemoglobin in smokers, But there are no significant differences in MCV, MID and PLT.

Table (2): The differences of various CBC parameters in smoker and non-smoker groups (Mean±SD).

Parameter	Group 1 Smoker students Mean±SD	Group 2 Non-Smoker students Mean±SD	P - value	Significance
WBC(10^{*9}/L)	6.75±1.25	6±1.11	0.02	S
RBC(10^{*12}/L)	5.45±0.56	5.77±0.46	0.02	S
LYM(10^{*9}/L)	2.07±0.56	1.81±0.32	0.02	S
NEUT(10^{*9}/L)	4.34±1.06	3.84±1	0.06	S
HGB g/dL	14.65±1.46	13.16±2.02	< 0.01	HS
HCT %	50.8±4.05	48.68±3.63	0.03	S
MCV fL	127.8±173.5	88.04±4.96	0.11	NS
MCH pg	27.32±1.92	22.68±4.31	< 0.01	HS
MID(10^{*9}/L)	0.32±0.07	0.34±0.09	0.2	NS
PLT (10^{*9}/L)	197.85±48.03	176.82±45.86	0.07	NS

Significant difference from group1 using t-test for two independent means
(HS : P < 0.01, S : P< 0.05, NS = P> 0.05)

4- Discussion:

Our data revealed that cigarette smoking had a significant negative impact on haematological markers (e.g., haemoglobin - Hb, hematocrit - HCT, white blood cells count - WBC, red blood cells count - RBC, MVC, MCH). The overall erythrocyte count was nearly identical among smokers and nonsmokers. In comparison to female smokers, men smokers had statistically significant higher erythrocyte values. (12) In our study, smokers had considerably higher haemoglobin levels than non-smokers, regardless of gender, whereas there was no significant difference in hematocrit levels between the two groups of patients. (13-14) Male smokers, on the other hand, had significantly higher hematocrit values than female smokers. Prior research has linked the large increase in Hb among smokers to previous investigations (13-15). In a study by Lakshmi et al. (16), smokers had significantly greater haematocrit and Hb levels, and their RBC count grew significantly as their smoking intensity increased. In their investigation, Whitehead et al. discovered that those who smoked more than 10 cigarettes per day had considerably higher haemoglobin concentration and hematocrit (17). Carbon monoxide exposure is thought to cause an increase in haemoglobin concentration, and some scientists believe that a rise in haemoglobin levels in smokers' blood could represent a compensatory mechanism. Carbon monoxide bonds to Hb to generate carboxy haemoglobin, an inactive form of haemoglobin that carries no oxygen. Carboxyhemoglobin also shifts the

left side of the Hb dissociation curve, reducing Hb's ability to supply oxygen to the tissue. Smokers maintain a higher haemoglobin level than non-smokers to compensate for the diminished oxygen delivering capacity (18). Increased erythropoiesis and haematocrit values in male smokers can be explained by tissue hypoxia produced by increased carboxy haemoglobin production, which leads to enhanced erythropoietin secretion and consequently increased erythropoiesis. Tobacco smoke causes an increase in capillary permeability, which reduces plasma volume and mimics the condition of polycythemia, which is defined by an increased proportion of erythrocytes in the blood volume, as well as higher hematocrit levels (14, 19). The three primary red blood cell indices, MCV, MCH, are used to determine the average size and haemoglobin content of red blood cells. Smokers had significantly higher levels of mean corpuscular volume (MCV) and mean corpuscular haemoglobin (MCH). Other studies have also found that smokers have higher MCV and MCH readings than non-smokers (20, 21). The levels of these parameters surpassed the reference interval in the study of Kung et al. (21) and were typical of findings for illnesses such as kidney dysfunction, hyperuricaemia, hypertension, and hypercholesterolemia. These findings contrast those of Pankaj et alstudy. .'s(22) There were no significant differences in MCV and MCH between smokers and non-smokers, according to the researchers(23). In the two groups, there was no difference in platelet count or MCV. In smokers, Asif et al. discovered a rise in MCV and a decrease in MCH levels (24). MCV measures the size of a red blood cell, and the presence of red blood cells that are smaller or larger than normal indicates anaemia. High MCV levels in our study indicate that subjects may have megaloblastic, haemolytic, pernicious, or macrocytic anaemia, which is caused by iron and folic acid deficiencies (24). MCH signifies the amount of haemoglobin in a certain volume of 'packed' red corpuscles or cells. In addition, male smokers' leukocyte counts were statistically considerably higher. The increased total leukocyte count seen in smokers is consistent with previous research (25, 26). Although the specific method of how smoking increases the amount of leukocytes is unknown, smoking-induced leukocytosis is caused by a number of elements that can be explained in a variety of ways. According to some experts, nicotine-induced release of catecholamine and steroid hormones from the centre of the adrenal gland can result in an increase in the number of leukocytes. Increases in the levels of some endogenous hormones, such as epinephrine and cortisol, are known to result in an increase in the number of leukocytes (27, 28). Furthermore, the irritating action of tobacco smoke on the respiratory tree, as well as the resultant inflammation, can lead to an increase in leukocyte numbers. It has been proven that inflammatory stimulation of the respiratory tract causes an increase in inflammatory markers in the bloodstream, particularly cytokines, which influence the quantity of leukocytes. Reduced vasomotor function, reduced capillary perfusion, leukocyte and thrombocyte adhesion, activation of the coagulation cascade and increased thrombosis, increased vascular permeability, and an increase in the rate of proliferation of blood and lymphatic vessels are the best-known microcirculation responses to inflammation. The activation of numerous cells that ordinarily circulate in the blood (leukocytes, thrombocytes), exist in the walls of blood arteries (endothelial cells, pericytes), or exist in the perivascular

space (fat cells, macrophages) is the answer to inflammation (29). Inflammatory processes are marked by leukocyte adhesion to vascular endothelium. After being bound to the endothelium and being inactive, leukocytes might move in the intercellular space between those same cells. Sequential activation of distinct families of adhesion molecules, which are located on the cell surface and promote cell contact, fixing of cells on the wall of blood arteries, and movement, regulates the entire process of leukocyte adherence on endothelium cells. acrophages) acrophages) acrophages) (29). Selectins are lecithin-like adhesion glycoproteins that mediate leukocyte movement, whereas integrin (CD11/CD18, VLA-4) on leukocytes and immunoglobulin-like adhesion molecules on endothelium cells (ICAM-1, VCAM-1) on endothelium cells mediate firm adhesion and subsequent trans-endothelium migration (30). It's possible that the increased number of leukocytes in healthy smokers' peripheral blood is linked to the phenomenon of cell movement from other lymphoid organs into the bloodstream, or that smoking reduces these cells' ability to adhere to endothelium cells in blood vessels, resulting in a general increase in the number of blood cells (31). The results of smoking-related differential blood tests are inconsistent. In smokers, Aula and Qadir found a considerable increase in leukocytes, neutrophils, eosinophils, basophils, lymphocytes, and monocytes compared to non-smokers (32). Kastelein et al., on the other hand, found no significant difference in neutrophil levels between middle-aged smokers and non-smokers (33). Our investigation found the same results, with granulocytes, which comprise neutrophils, not significantly increasing. Unlike Kasteleinaet alwork, .'s which found statistically significantly higher levels of basophils, lymphocytes, and monocytes in smokers, our investigation could not substantiate the influence of smoking status on the aforementioned white blood line characteristics in healthy participants. The atherogenic effect of cigarette smoking is thought to be mediated in part by leukocytes. The quantity of leukocytes is perhaps the most useful and straightforward biomarker of endothelial deterioration. Because enhanced leukocyte aggregation predisposes microcirculatory blockage and vascular damage, the presence of a persistently elevated number of leukocytes in smokers contributes to the pathogenesis of smoking-related disorders, including ischemic vascular disease. The amount of leukocytes is an independent predictor of atherosclerosis and cardiovascular illnesses, according to several studies (34, 35). Our findings show that smokers, particularly male smokers, have a higher risk of atherosclerosis and cardiovascular disease than non-smokers.

5-CONCLUSION

We can conclude from this study that continuous cigarette smoking raises erythrocyte count, haemoglobin concentration, hematocrit, leukocyte count, mean corpuscular volume, and mean corpuscular haemoglobin

concentration, and that these changes may be linked to an increased risk of atherosclerosis, polycythemia vera, chronic obstructive pulmonary disease, and/or cardiovascular diseases.

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