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Consequence of smoking on hematological parameters in apparently healthy medical students

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ABSTRACT

Background: One of the greatest causes of death in the globe is tobacco cigarette smoking. Smoking affects hematological markers both acutely and chronically. The current study's objective was to evaluate the severity of the negative effects of cigarette smoking on biochemical traits in healthy smokers.
Materials and Methods: This study included 280 participants, only 56 smokers and 100 non-smokers. They were smoking for three years, the smokers regularly smoked 10 to 20 cigarettes per day. The fully automatic hematological analyzer CELL-DYN 3700 examined the total number of blood cells.
Results: White blood cells (p0,001), hemoglobin (p=0,042), mean corpuscular volume (p=0,001), and mean corpuscular hemoglobin concentration (p0,001) were all considerably greater in the smokers. No other measured metrics varied considerably from one another. Red blood cells, white blood cells, hemoglobin, hematocrit, and mean corpuscular hemoglobin were all significantly higher (p0,001) in male smokers compared to female smokers after smoking cigarettes.
Conclusion: Our study concluded that smoking cigarettes continuously has severe negative effects on hematological variables, including hemoglobin, white blood cell count, mean corpuscular volume, mean corpuscular hemoglobin concentration, red blood cell count, and hematocrit. These modifications may be linked to an increased risk of polycythemia vera, atherosclerosis, chronic obstructive pulmonary disease, and/or cardiovascular diseases.

1. Introduction

The World Health Organization estimates that 5 million people worldwide pass away each year from diseases brought on by smoking, and that number will rise to 10 million by 2015 if the current trend continues. Numerous studies revealed that smoking had negative effects on human health and was a risk factor for the onset of a number of pathological conditions and diseases, including cancer, chronic obstructive pulmonary disease, pancreatitis, gastro-intestinal disorders, periodontal disease, metabolic syndrome, and some autoimmune diseases.[1]

In addition to chronic heart disease, atherosclerosis, myocardial infarction, ischemic heart disease, and stroke, smoking cigarettes is linked to an elevated risk of these cardiovascular disorders.[2] The precise causes of these illnesses in smokers are unknown are not known, but it is presumed that these effects are caused by abnormalities in the blood rheology, infection and inflammation, oxidative stress, and alterations of antithrombotic and fibrinolysis system. Tobacco smoke contains over 4000 compounds that have a more or a less adverse effect on human health, among which free radicals, the nicotine and the carbon monoxide are considered the most responsible for pharmacological

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effects.[3]

Although the exact causes of these diseases in smokers are unknown, it is assumed that infections and inflammation, oxidative stress, and changes to the antithrombotic and fibrinolysis systems are responsible. Around 2.4 billion individuals worldwide smoke, chew, snuff, or dip tobacco. Around 6.4 million fatalities were attributed to tobacco use in 2015, and it is predicted that number will climb to 8.3 million in 2030 and one billion in the twenty-first century.[4] The percentage of smokers is expected to be high as the population grows. Smoking addiction is growing among people, most of them are in the youthful group. Along with increasing awareness, the other harms induced by smoking (such as higher WBC) must be understood in order to better understand and treat the health issues brought on by smoking in order to address this crucial situation.

The nicotine decreases vascular function, causes clot formation in the coronary arteries, and worsens endothelial dysfunction. Increased levels of carboxy-hemoglobin may result in hypoxia, and they are also to blame for subendothelial edema because they change vascular permeability and lipid accumulation.[5] Free radicals and peroxides from the tobacco smoke are clearly linked with physiological phenomenon such as synthesis of prostaglandins and thromboxane, and they play a role in the development of many diseases, including atherosclerosis, cancer, and inflammatory processes. Numerous research on the effects of smoking on changes in the hemostatic and fibrinolytics system, antioxidant status, and hematology parameters were conducted, but the results were mixed.[6] The present study was undertaken to measure how smoking cigarettes affected some hematological markers in smokers compared to age-matched non-smoking controls.

2. Materials and Methods

The present study was carried out to investigate the relationship effect of cigarette smoking on hematological parameters in a group of medical student's volunteers apparently normal from Inaya Medical Colleges. A total of 280 male and female medical students were registered in the study, only 156 students were accepted for further investigations. The age of the accepted participants ranged between 20-45 years old, all registered students under 20 years are excluded and all participants over 45 years were excluded too. 56 smokers from both genders and 100 non-smokers. The smokers from both genders were regularly consuming 15-30 cigarettes per day for at least 3 years.

All students' samples were analyzed in King Salman Hospital (Hematology and Blood banking Department).

2.1. Ethical approval

Students were enrolled gave an informed consent and the study protocol was approved by the Ethical Review Committee in Inaya Medical Colleges.

The study was conducted in compliance with the ethical standards from Research unit in the Colleges. Data on smoking habits and the amount of tobacco consumed were collected by a self-administered questionnaire to be filled in by each participant.

Students included were free of evidence of active liver and kidney disease, chronic pancreatitis, gastrointestinal disease, inflammatory bowel, history of ischemic heart disease or and diastolic blood pressure, endocrine disorders, infection, and hormonal therapy.

2.2. Anthropometric and biochemical measurements

In all the volunteer student's anthropometric data (weight and height), blood pressure, and arterial blood pressure were measured. BMI was calculated as body weight (kg) divided by body height (m) squared. Blood pressure of each student was measured with a mercury Sphygmomanometer and a standard stethoscope. Blood samples were drawn after an overnight fast. Each test procedure was carried out between 8-10 a.m.

The subjects underwent the following tests: Blood pressure examination to rule out hypertension, estimation of the serum lipid profile, evaluation of the fasting and post prandial blood glucose levels to rule out diabetes, red blood cell count, total leukocyte count, differential leukocyte count, platelet count, packed cell volume, hemoglobin, serum urea and creatinine levels. Complete blood cell count was analyzed by CELL-DYN 3700 fully automatic hematological analyzer in King Salman Hospital.

2.3. Questionnaire tool

The questionnaire aimed to collect demographic information and medical history of the students. Students who accepted to participate in the study, were asked to attend to the Clinical Laboratory Sciences Department (Hematology and Phlebotomy labs). The participants were asked to provide a written consent. (Appendix 1).

2.4. Statistical analysis

The Kolmogorov-Smirnov test was used to determine whether the variances in each student's sample were homogeneous and distributed normally. SPSS version 20.0 was used for the statistical analysis (SPSS Inc.). Groups were compared using the Mann-Whitney test for parameters with a non-normal distribution or the student's unpaired t test for parameters with a normal distribution prior to statistical analysis. Correlations employing the Spearman test for variables with a non-normal distribution and the Pearson R test for variables with a normal distribution, respectively. Data is presented as a mean ± standard deviation or medians (interquartile range). $P < 0,05$ was considered significant.

3. Results

156 students' initial characteristics are shown in (Table 1). The ages ranged from 20,00 to 0,477 years, on average. All of the individuals had normal blood pressure, with a mean systolic of 122,33 1,093 and a mean diastolic of 79.85 ± 0.498.

The results appeared in Table 2 compares the hematological parameters between smokers and non-smokers which shows a statistically significant increase in hematocrit, mean corpuscular hemoglobin, total leucocyte count, and hemoglobin in smokers. The rest of the parameters were not statistically significant. Data are presented as means ± SD or medians (interquartile range). *Significance of difference in Mann-Whitney test for data following non-normal distributed and t-test for normal distributed date. WBC-White blood cells, RBC-Red blood cells, Hemoglobin, HTC-Hematocrit, MCV-Mean Corpuscular Volume, MCH- Mean Corpuscular Hemoglobin, MCHC-Mean Corpuscular Hemoglobin Concentration, RDW-Red Blood Cell Distribution Width.

Following a previous analysis of the effects of smoking on all research participants' hematological parameters, the 56 smokers were separated into groups based on gender. As Table 3 shown, there was no statistically significant difference. Data are reported as means SD or medians (interquartile range), and the Mann-Whitney test for data returns NS, or non-significance of difference. Following non-normal distributed and t-test for normal distributed date. BMI-body mass index.

Difference in age, body mass index (BMI), waist circumference and blood pressure between the smokers who are male and female. Leukocyte counts differed between the sexes in the group of smokers according to an examination of gender disparities. Male individuals' leukocyte values were statistically greater than those of female respondents. There isn't a statistically significant difference between other white blood cell metrics. The percentage of red blood cells, hemoglobin, hematocrit, mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH) values were statistically greater in the male smoker population as appeared (Table 4).

Data are shown as means, standard deviations, or medians (interquartile range). *The difference between the Mann-Whitney test for data with a non-normal distribution and the t-test for data with a normal distribution. The abbreviations RBC, HB, MCV, HTC, MCH, MCHC, WBC, and RDW stand for white blood cells, red blood cells, mean corpuscular volume, mean corpuscular hemoglobin, and red blood cell distribution width.

4. Discussion

Our data demonstrated that smoking had serious negative impact on hematological parameters (e.g., hemoglobin - Hb, hematocrit - HCT, white blood cells count - WBC, red blood cells count - RBC, MVC, MCH). The overall erythrocyte count was nearly the same in smokers and non-smokers. Male smokers were shown to have statistically significant higher erythrocyte values than female smokers. In our study, there was no significant difference in the levels of hematocrit between the two groups of participants, however the hemoglobin values were substantially higher in smokers than in non-smokers regardless of sex. However, compared to female smokers, male smokers had significantly higher hematocrit values. In one of the Previous studies is related to the smokers' large increase in Hb (13-15). In a study conducted by Lakshmi et al.[7] smokers had significantly higher hemoglobin and hematocrit levels, and as smoking intensity increased, smokers' RBC counts also increased dramatically. In their study, White head et al.[8] found that smokers had considerably higher hemoglobin concentration and hematocrit levels more than 10 cigarettes per day.

Some researchers hypothesized that an increase in hemoglobin levels in smokers' blood might represent a compensatory mechanism. The rise in hemoglobin concentration is thought to be mediated by exposure to carbon monoxide. To create carboxy hemoglobin, which is an inactive version of hemoglobin with no ability to deliver oxygen, carbon monoxide binds to hemoglobin (Hb). Additionally, carboxyhemoglobin pushes the left side of the Hb dissociation curve, which reduces Hb's capacity to carry oxygen to the tissue. Smokers continue to maintain a higher hemoglobin level than non-smokers in order to make up for the reduced oxygen-delivering capacity.[9] The fact that tissue hypoxia brought on by an increase in the production of carboxy hemoglobin results in an increase in the release of erythropoietin, which in turn increases erythropoiesis, can be used to explain why male smokers have higher levels of erythrocytes and hematocrit.

Additionally, carbon monoxide from tobacco smoke increases capillary permeability, which reduces plasma volume and ultimately resembles polycythemia, which is marked by a greater proportion of erythrocytes in blood volume and is also demonstrated by higher hematocrit readings.[10,11] MCV, MCH and MCHC are three main red blood cell indices that help in measuring the average size and hemoglobin composition of the red blood cells. Our study established significantly larger values of mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) among smokers, while differences in values of MCHC and RDW were not significant between smokers and non-smokers. Larger values of MCV and MCH in smokers, in relation to non-smokers were confirmed by other studies as well.[12,13] The values of these parameters in the study by Kung et al.[14] were above those of the reference interval and were indicative of several disorders, such as kidney dysfunction, hyperuricemia, hypertension, or hypercholesterolemia. These findings contrast with those

Table 1: Baseline characteristics of participants. Data expressed as Mean ± SD.

Variable	Mean ± SD	Range
Age (years)	20.00 ± 0.477	(20-45)
BMI (kg/m2)	26.35 ± 0.389	(23.9-27.9)
Waist circumference (cm)	0.86 ± 0.05	(0.79-0.92)
Systolic blood pressure (mmHg)	126.33 ± 1.093	(75-145)
Diastolic blood pressure (mmHg)	79.85 ± 0.498	(50-110)

Table 2: Hematological comparison parameters between smokers and non-smokers students.

Hematological parameters	Smokers (n=56)	Nonsmokers (n=100)	P
WBC (x10e9/L)	7.03 (6.00-8.12)	6.00 (4.89-7.11)	0.001*
Lymphocyte (%)	33.20 (24.58-39.35)	29.00 (19.15-35.40)	0.097
Monocyte (%)	4.55 (1.43-6.10)	4.50 (0.00-6.00)	0.643
Granulocyte (%)	56.25 (50.08-64.83)	55.90 (0.00-65.90)	0.675
RBC (x10e12/L)	4.88 (4.56-5.24)	4.88 (4.53-5.22)	0.959
HCT (%)	41.65 ± 1.03	40.63 ± 0.55	0.336
MCV (fL)	88.50 (81.90-92.25)	84.00 (80.00- 88.00)	0.001
Hb (g/dL)	147.00 (132.60-157.00)	139.00 (127.15- 151.30)	0.042*
MCH (pg)	29.82 (28.49-30.92)	28.70 (27.05-29.75)	<0.001*
MCHC (g/L)	333.90 (324.00-344.15)	333.90 (324.00-346.75)	0.526
RDW (%CV)	14.10 (13.28-14.90)	14.00 (13.50-15.20)	0.598

Table 3: Baseline characteristics of the smoker subjects -gender differences.

Variable	Male smokers (n=26)	Female-smokers (n=30)	P
Age (years)	48.60 ± 1.31	49.65 ± 1.45	NS
BMI (kg/m2)	26.30 (24.70-28.30)	26.19 (23.90-27.90)	NS
Waist circumference (cm)	0.89 ± 0.05	0.80 ± 0.12	NS
Systolic blood pressure (mmHg)	136.06 ± 1.378	130.43 ± 1.682	NS
Diastolic blood pressure (mmHg)	88.11 ± 0.512	83.40 ± 1.436	NS

Table 4: Differences hematological parameters between male and female smokers.

Hematological parameters	Male-smokers (n=26)	Female-smokers (n=30)	p
WBC (x10e9/L)	7.67 ± 0.40	6.67 ± 0.287	0.040*
Lymphocyte (%)	29.80 (22.65- 36.65)	33.90 (24.00- 40.78)	0.158
Granulocyte (%)	57.50 (30.00- 67.65)	55.10 (36.15- 62.63)	0.561
RBC (x10e12/L)	5.12 ± 0.07	4.66 ± 0.06	<0.001*
HB (g/dL)	155.78 ± 2.03	134.65 ± 2.44	<0.001*
HCT (%)	43.84 ± 1.97	39.76 ± 0.76	0.047*
MCV (fL)	88.92 ± 1.34	85.38 ± 1.17	0.052*
MCH (pg)	30.58 (29.75- 31.20)	29.43 (27.62- 30.32)	0.001*
MCHC (g/L)	336.00 (329.00- 344.50)	335.75 (323.75- 345.20)	0.554
RDW (%CV)	14.10 (13.29- 14.95)	14.10 (13.29- 14.95) 0.722	0.722

of Pankaj et al.,[15] which found no appreciable differences in MCV and MCH between smokers and non-smokers. They found significantly low value of MCHC (p <0,009) among smokers. In contrast our finding, in study made by Salamzadeh, when compared to the non-smoking group, the smoker group's levels of MCH and MCHC (p 0.05) were considerably lower. The MCV in two groups did not differ from the platelets count.

According to Asif et al.,[16] smokers had higher MCV levels and lower MCH and MCHC levels. Elevated levels of MCV in our study indicate that people may have megaloblastic, haemoytic, pernicious, or macrocytic anemia, which is typically brought on by iron and folic acid deficits. Red blood cells smaller or larger than normal size indicate anemia.

In contrast to MCHC, MCH is the average weight of hemoglobin found inside a single red blood cell indicates how much hemoglobin is present in a given volume of densely packed red blood cells or corpuscles. In comparison to non-smokers, smokers of both sexes had a significantly higher number of leukocytes, according to our study.

Leukocyte count values were also statistically considerably higher in male smokers. Smokers increased total leukocyte count is consistent with past findings.[17,18] Smoking-induced leukocytosis has a lot of components and can be explained in a number of ways, despite the fact that the precise process by which it increases the number of leukocytes is not yet fully understood. According to some experts, nicotine-induced release of catecholamine and steroid hormones from the center of the adrenal gland can lead to an increase in the number of leukocytes. It is understood that a rise in the concentration of several endogenic hormones, such as cortisol and adrenaline, causes an increase in the number of leukocytes.[19,20]

Leukocyte production can also be aided by the irritating effects of cigarette smoke on the respiratory system and the subsequent inflammation. It has been proven that inflammatory stimulation of the respiratory tract causes an increase in circulating inflammatory markers, particularly cytokines, which have an impact on the leukocyte count. A decrease in vasomotor activity, a reduction in capillary perfusion, leukocyte and thrombocyte adhesion, activation of the coagulation cascade and an increase in thrombosis, increased vascular permeability, and an increase in the rate of blood and lymphatic vessel proliferation are some of the best-characterized responses of the microcirculation to inflammation. Activating various cells that typically circulate in the blood (leukocytes, thrombocytes), are found in the blood vessel walls (endothelial cells, pericytes), or are found in the perivascular space (fat cells, macrophages) is the solution to inflammation.[14]

A defining characteristic of inflammatory events is leukocyte adhesion to vascular endothelium. Leukocytes are bound to the endothelium and remain dormant until migrating in the gap between those very cells. Sequential activation of distinct families of adhesion molecules, which are found on the cell surface and promote cell contact, fixing of the cells on the wall of blood arteries, and their movement, controls the entire process of leukocyte adherence on endothelium cells. Leukocyte movement is mediated by lecithin-like adhesion glycoproteins known as selectins, whereas firm adhesion and subsequent trans-endothelium leukocytosis migration are mediated by interactions between integrin (CD11/CD18, VLA-4) on leukocytes and immunoglobulin-like adhesion molecules on endothelium cells (ICAM-1, VCAM-1).[21]

It's possible that smoking reduces the capacity of these cells to adhere to endothelial cells or that a rise in leukocytes in the peripheral blood of healthy smokers is related to the phenomena of cell movement from other lymphoid organs in the blood. Increase in the amount of blood cells worldwide due to the expansion of blood arteries.[22] There are conflicting reports on how smoking affects blood tests. Smokers' leukocyte, neutrophil, eosinophil, basophil, lymphocyte, and monocyte counts were significantly higher than those of the control group of non-smokers, according to research by Aula and Qadir.[23]

On the other hand, middle-aged smokers and non-smokers showed no discernible change in neutrophil levels, according to Kastelein et al.[24] Our study also showed the same outcomes, showing that the levels of granulocytes, which comprise neutrophils, were not significantly elevated. However, unlike the study by Kasteleina et al., which found that basophils, lymphocytes, and monocyte values were statistically substantially higher in smokers, our investigation did not support the relationship between smoking status and the aforementioned white blood cell parameters in healthy participants. Leukocytes are thought to play a role in the atherogenic effects of cigarette smoking.

Perhaps the most practical and straightforward biomarker of endothelial impairment is the leukocyte count. Given that an increase in leukocyte aggregation predisposes microcirculatory blockage and vascular damage, the presence of a persistently elevated number of leukocytes in smokers contributes to the development of smoking-related disorders, particularly ischemic vascular disease. The prevalence of leukocytes is an independent predictor of atherosclerosis and cardiovascular illnesses, according to several studies.[25,26]

In the present study finding of a high leukocyte number among smokers, particularly in male students, indicates that they are at an increased risk for cardiovascular disease and atherosclerosis compared to non-smokers.

5. Conclusion

According to the results of the current study, smoking cigarettes continuously increases erythrocyte count, hemoglobin concentration, hematocrit, leukocyte count, mean corpuscular volume, and mean corpuscular hemoglobin concentration. 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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8. Conflict of Interest

The authors declare that there are no potential conflicts of interest for the authorship and publication of the article.

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