Effect of cigarette smoking on blood parameters in young smokers

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Received: Mar 15, 2023/ Revised: Apr 9, 2023/ Accepted: Apr 11, 2023

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Abstract

This study set out to evaluate the detrimental impact of cigarette smoking on the metabolic characteristics of smokers, looking at how smoking altered physiological factors differently for both smokers and nonsmokers. The results showed a significant difference in hemoglobin content, a gauge of blood oxygenation, between smokers and non-smokers. The findings of the current investigation enable us to reach the following judgment: Leukocytes, average erythrocyte volume, and average hemoglobin content in erythrocytes are all increased by smoking cigarettes on a regular basis. These changes could raise the risk of cardiovascular disease, polycythemia, pulmonary disease, and/or atherosclerosis. According to the number of years of smoking, the groups of smokers in the analysis were divided, with smoking periods of 10 years or longer being the longest.

Keywords: Smoking, Blood Parameters, HB, RBC, WBC, HCT, PLT, MCH, MCHC, MCV

Introduction

Smoking has detrimental effects on human health and increases the chance of developing a variety of pathological disorders and diseases, including obstructive lung disease. As a result, it is difficult to handle the issue both internationally and locally. The following diseases are chronic (Buist and McBurnie, 2008), cancer (Vineis et al., 2004), pancreatitis (Barreto, 2016), gastroenteritis (Li et al., 2014), periodontitis (Gautam et al., 2011), metabolic (Slagter et al., 2013), and a few autoimmune (Harel-Meir et al., 2007).

Moreover, smoking increases the risk of stroke, peripheral vascular disease, atherosclerosis, ischemic heart disease (Lu and Creager, 2004), myocardial infarction, (Messner and Bernhard, 2014) and coronary artery disease (Law et al., 1997) and stroke (Shah and Cole, 2010). Smokers may experience these issues for a variety of reasons, such as changes to the coagulation system and fibrinolysis, anomalies in blood biology, infections and inflammation, oxidative stress, and other unknown causes. Smoke from tobacco contains more than 4,000 different substances, including free radicals, nicotine, and carbon monoxide. Human health is adversely affected by pyridine, tar, lead, polonium, sulfuric hydrogen, ammonia compounds, sulfuric acid, and a variety of other substances (Messner and Bernhard, 2014; Shah and Cole, 2010).

The effects of nicotine on blood vessel health, endothelial dysfunction, and the formation of blood clots in the coronary vessels are all deteriorated. Excessive levels of carboxyhemoglobin can cause hypoxia, which changes the permeability of the blood vessels and causes lipid buildup and subendothelial edema (Gossett et al., 2009). Tobacco smoke contains free radicals and peroxides, which have a clear connection to physiological processes including the production of prostaglandins and thromboxanes as well as their part in the pathogenesis of a number of diseases like atherosclerosis, cancer, and inflammatory processes.

Materials and Method

Blood sample collection

In a group of clinically healthy volunteers, the current research was performed to look into the relationship between cigarette
smoking and blood parameters. The research included 100 participants in the 20–40 age range, including 30 nonsmokers and 70 smokers. For at least three years the smokers had been 10–20 cigarettes a day smokers. To learn more about the individuals’ smoking habits and the amount of tobacco they ingested, a self-administered questionnaire was used. The study subjects showed no symptoms of chronic pancreatitis, liver disease, or renal disease.

The following tests were performed on the volunteers: estimation of packed cell volume, total white blood cell count, platelet count, and hemoglobin in the blood. A completely automatic CBC was used to examine the whole blood cell count.

The following mathematical relationship was used to compute the length of time and amount of smoking between (three years to twenty years) and compare it to the blood analysis (Complete Blood Count).

Analytical statistics

Utilizing SPSS software, statistical analysis was carried out.

Results and Discussion

![Fig. 1 Compares the levels of WBC, RBC, HB, and HCT in Smokers and non-smokers](image)

When compared to non-smokers, Fig. 1 demonstrates a considerable rise in Hb concentration, RBC HCT, and WBC in smokers. These findings corroborated the study’s findings (Shah et al., 2012; Nadia et al., 2015; Jen et al., 2013), which showed a considerable rise in hemoglobin levels in the group of smokers. These findings are also in line with the findings of (Lakshmhi et al., 2014) study, which found that smokers had greater hemoglobin concentrations than non-smokers and that the number of red blood cells rises markedly with increasing smoking intensity.

A study by Whitehead and colleagues found that smokers who smoked more than 10 cigarettes per day had considerably higher hematocrit and hemoglobin concentrations. According to some scientists (Whitehead et al., 1995), carbon monoxide exposure is the likely source of elevated hemoglobin concentration, and smokers are more prone to have this issue. As a type of protection, their hemoglobin levels may have increased. Where carboxyhemoglobin, an inactive form of hemoglobin that cannot carry oxygen, is the product of the interaction between hemoglobin and carbon monoxide. Carboxyhemoglobin also modifies the left-sided hemoglobin dissociation curve, reducing hemoglobin's capacity to transport oxygen to the organs. Smokers nevertheless have higher hemoglobin levels than non-smokers, which offsets the loss of their ability to provide oxygen. Skin hypoxia may be the cause of the increased erythrocyte and hematocrit levels seen in male smokers. Increased carboxyhemoglobin production results in enhanced erythropoietin secretion and erythropoiesis (Aitchison and Russell, 1988).

Smoke from cigarettes also contains carbon monoxide, which reduces plasma volume and raises capillary permeability. This is similar to erythrocytosis, a disorder marked by an elevated proportion of erythrocytes in blood volume and evidenced by higher hematocrit levels (Verma and Patel, 2015).

Also, we discovered that smokers had a considerable increase in leukocytes compared to non-smokers. Leukocyte counts increased overall, which is consistent with earlier research. Although the precise mechanism by which smoking affects leukocyte count is still unknown, leukocytosis shows a number of traits (Linal et al., 2014; Higuchi et al., 2016).

According to several research, the release of nicotine from the adrenal core, which is prompted by catecholamine and steroid hormones, may be the reason for the rise in white blood cell count. (Kapoor and Jones, 2005). There is proof that certain hormones like cortisol and adrenaline, which are present in larger amounts, increase their synthesis. Uncolored blood cells, the amount of white blood cells can also be impacted by smoking’s effects on the respiratory system and the resulting inflammation (Deutsch et al., 2007). The amount of white blood cells can also be impacted by smoking’s effects on the respiratory system and the resulting inflammation. It is well established that inflammatory activation of the respiratory system raises blood levels of inflammatory markers, particularly cytokines, which in turn affects how white blood cells are produced. This result in a decline in White blood cell adhesion to vascular endothelium is an indication of inflammatory events. After adhering to the endothelium and entering dormancy, white blood cells might move among the same cells in the intercellular space (Granger and Senchenkova, 2010).

To control the entire leukocyte adherence to the endothelial cells process, various families of adhesion molecules that promote cell contact, cell fixation, and cell movement are successively activated in the arterial wall. Selectins are sticky glycoproteins that resemble lecithin and control how white blood cells travel (Langer and Chavakis, 2009).
Smoking may reduce the ability of these cells to adhere to endothelial cells, increasing the overall number of blood cells. Another possibility is that smoking causes these cells to move from other lymphoid organs into the peripheral blood, increasing the number of leukocytes in the peripheral blood of healthy smokers. (Parry et al., 1997).

Research reveals that the most practical and simple predictor of endothelial damage is the number of white blood cells, which may lead to atherosclerosis in smokers. Considering that smokers have more of these cells, it is possible that sclerosis and cerebral vascular disease, two related illnesses, are present as well. Many studies have shown that an increase in white blood cells destroys large blood arteries and obstructs small blood vessels. Arteries and cardiovascular problems are both impacted differently by leukemia (Madjid et al., 2004; Loimaa et al., 2006). Our research indicates that smoking raises the white blood cell count, particularly in men, which raises the risk of atherosclerosis and cardiovascular disease. (Khan et al., 2008).

In line with Asif et al., we also discovered that smoking was associated with decreased MCH and MCHC levels and higher MCV levels. The study subjects might have megaloblastic, hemolytic, pernicious, or refractory macrocytic anemias based on the high levels of MCV they had. When red blood cells are sized using the MCV, there are always red blood cells that are either smaller or larger than (Salamzadeh's, 2004). Anemia is implied by the phrase "normal." often due to iron and folic acid deficiencies. The average weight of hemoglobin retained within a single red blood cell is known as MCH, as opposed to MCHC, which describes the amount of hemoglobin in a certain volume of "packed" red blood cells (Asif et al., 2013).

Conclusion

Our research shows that smoking considerably impairs hematological parameters such as hemoglobin, hematocrit, and white blood cell count, red blood cell count, MCHC, and MCH.

Conflict of Interest

The author hereby declares no conflict of interest.

Ethical Considerations

The study was approved by the institutional ethical committee.

Consent for publication

The author declares that the work has consent for publication.

References


Granger, D. N., & Harris, N. R. (2012). Vascular Responses to Intestinal Inflammation in Inflammatory Bowel Disease. *Crohn's Disease and Ulcerative Colitis: From Epidemiology and Immunobiology to a Rational Diagnostic and Therapeutic Approach*, 133-139.


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