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ORIGINAL ARTICLE

Effect of smoking on differential white cell count and hemoglobin level in healthy smokers and controls: A comparative study

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ABSTRACT

Background: Cigarette smoking alters inflammation indicators, which has been linked to cardiovascular disease as well as inflammatory disorders. The toxicity of tobacco has an effect on the oxygen saturation of haemoglobin. Total and differential leukocyte count (DLC), as well as oxygen saturation of haemoglobin, were measured in healthy smokers and nonsmokers in order to determine whether or not they were smoking.
Methods: The participants in this cross-sectional study totaled 80 persons in good health. A questionnaire was utilised to gather information on smoking habits as well as anthropometric measurements such as height, weight, and body mass index (BMI). In order to count total and DLC cells in blood samples, the MS-9 automated haematological cell counter was employed. The fingertip pulse oximeter was used to test the oxygen saturation of the haemoglobin.
Results: Compared to non-smokers, smokers had higher TLC (P <0.001), lymphocyte (P< 0.002), granulocyte (P 0.01), and monocyte counts (P 0.03) and lower SpO2 (P 0.03).
Conclusion: The study concluded that smokers' TLC, DLC, and haemoglobin oxygen saturation should be evaluated during diagnosis, interpretation, and therapy. The elevated TLC and DLCs seen in this study may be linked to chronic inflammation and increased CVD risk in smokers. Quitting smoking is therefore beneficial to health.
Keywords: SpO2; Oxygen Saturation of Hemoglobin; Total and Differential Leukocyte Count; Smokers

INTRODUCTION

Smoking is a leading cause of mortality internationally. Numerous studies have shown that smoking is harmful to one's health and contributes to the development of pathological conditions and diseases such as COPD, cancer, pancreatitis, gastrointestinal problems, periodontal disease, metabolic syndrome, and several autoimmune diseases. Smokers are more prone to cardiovascular disease, peripheral vascular disease, and cerebrovascular illnesses such as strokes.

As many as 4000 compounds present in cigarette smoke might cause inflammation, affecting haematological parameters. Smokers inhale nicotine, free radicals, carbon monoxide, and other gaseous compounds. Further, inhaled nicotine mimics nicotinic acetylcholine, which binds to nicotinic acetylcholine receptors, inhibiting acetylcholine absorption, resulting in ACh buildup at neuromuscular junctions Smokers have greater white blood cell counts than non-smokers, according to research published in the last decade. Smokers had larger mean platelet volume and platelet distribution breadth than non-smokers in healthy males.

Leukocytes have emerged as substantial indicators of both cardiac and cerebrovascular illness in smokers. The above-mentioned components of tobacco smoke enhance leukocyte counts, with nicotine having a major impact. Nicotine appears to increase leukocyte count via stimulating hormone production. Smoke also irritates the respiratory mucosa, causing inflammation and producing cytokines, which raises the leukocyte count.

The respiratory system is clearly designed for oxygenation. When oxygen enters the alveoli (mainly via haemoglobin), some of it stays molten. 0.003ml/100 ml dissolved O2, with 1g haemoglobin transporting 1.34 ml. Oxygen saturation is the quantity of oxygen carried by haemoglobin in the circulation (SpO2). Smoking is a major source of disease and mortality, with prevalence rates ranging from 20–40% in industrialised nations to 2–10% in poor ones. Cigarette smoking harms the lungs and other organs in many ways. Our work may contribute to the literature on smoking's influence on arterial blood and haemoglobin saturation. To better diagnose, interpret data, and treat patients, this study studies the effect of cigarette smoking on total and differential leukocyte count (DLC) and oxygen saturation of haemoglobin.

MATERIALS AND METHODS

The study was conducted in the Physiology Department. The study included 80 healthy persons aged 20–50 years. Smokers were defined as those who smoked cigarettes/bidis everyday for at least a year. The study excluded ex-smokers or former smokers. The following WHO (1998) classification criteria were used: Smokers are characterised as those who smoke tobacco products regularly or sometimes, whereas non-smokers are people who don't smoke at all. Also, an ex-smoker is someone who used to smoke but no longer smokes. Adults having a history of acute or chronic sickness, bleeding or bleeding disorders, or drug addiction were excluded from the research. The research excluded pregnant women and those who had given birth within 3 months.

Anthropometric measurements were made, including height, weight, and BMI. A questionnaire collected data on smoking behaviours. Hemoglobin total, DLC, and O_2 saturation estimation: The antecubital vein was pricked and blood was collected into 3 ml EDTA vacutainers. The EDTA blood samples were counted using an MS-9 automated haematology cell counter for TLC and DLC (in percentage). Samples were processed within 3–5 hours following collection. The fingertip pulse oximeter measured haemoglobin oxygen saturation. Subject sat quietly for 5–15 minutes. A non-invasive sensor was put on the index finger (or another finger if the index finger was absent), and six readings were taken every 10 seconds for 10 seconds. We used the average of six measurements.

RESULTS

Table 1 compares baseline demographic data (age and BMI) between smokers and non-smokers in a total of 80 participants. A lack of substantial differences in baseline demographics between smokers and non-smokers enables unbiased comparison.

Table 1: Demographic parameters of smokers and non-smokers subjects

Descriptive statistics		
Smoking status	**Mean**	**Standard deviation**
Non-smoker		
Age	28.57	8.42
BMI	25.246	4.211
Smoker		
Age	33.51	7.365
BMI	26.266	3.143

Table 2 compares TLC, lymphocyte count, monocyte count, granulocyte count, and haemoglobin oxygen saturation between smokers and non-smokers. Smokers had considerably greater TLC (P 0.001) than non-smokers, although their SpO2 (P 0.03) was much lower.

Table 2: TLC, DLC, and oxygen saturation among smokers and non-smoker subjects

Parameter	Non-smokers	Smokers	*P*-value
TLC	7.22	8.116	<0.001
Lymphocyte count	0.425	0.4534	<0.002
Monocyte count	0.048	0.0623	0.03
Granulocyte count	0.621	0.56	0.01
SpO2	0.992	0.993	0.03

DISCUSSION

In addition to some serious haematological illnesses, tobacco smoking has been associated to several others. In our study, smokers had higher total white blood cell, lymphocyte, monocyte, and granulocyte counts than non-smokers, as well as higher total WBC and lymphocyte counts. Smokers' haemoglobin oxygen saturation was shown to be lower than that of non-smokers, which we discovered as well.

In the Copenhagen general population investigation, Pedersen et al. observed a rise in leukocytes, neutrophils, lymphocytes, and monocytes, among other things. Asif and colleagues discovered that frequent smokers had greater white blood cell counts than non-smokers in their study (P = 0.027). In comparison to female smokers and non-smokers, male smokers had a higher WBC count, which indicated a greater risk of atherosclerosis and cardiovascular disease (CVD). The epithelium prevents the entry of hazardous particles into the submucosa after they have been inhaled. The presence of leukocytosis has been identified as a probable indicator of tissue harm caused by cigarette smoke. Furthermore, a rise in its count may be associated with an increase in the risk of cardiovascular disease (CVD) through pathogenic pathways that promote inflammation, block microvasculature at various locations, and increase coagulability. Previous study observed that smokers' total leukocyte counts were significantly greater than those of non-smokers during their experiment. Manjunath establish that there is an empirically positive relationship between smoking and therapeutic lifestyle changes (TLC). Because of the potential relationship between TLC and smoking, we concentrated our investigation on DLCs. Although there is evidence to suggest this relationship, it is not clear what effect it will have on the DLC. According to our findings, there has been a statistically significant increase in all leukocyte subtypes. According to Zei-Shung et al., smokers have significantly higher TLCs and subtypes than nonsmokers. One possible mechanism for increased TLC is the interaction of a tobacco leaf glycoprotein with a specific membrane, which stimulates lymphocyte proliferation and differentiation antigenic response component found that lymphocyte count increased from 32.4 percent in non-smokers to 38.3 percent in smokers, whereas neutrophil count decreased somewhat in smokers compared to non-smokers, although the difference is not statistically significant. Eosinophil, basophil, and monocyte numbers did not alter significantly. Toxic chemicals present in tobacco smoke cause persistent tissue damage and inflammation, resulting in lymphocytosis. Smoke may also stimulate inflammatory markers in the pulmonary bronchial system, increasing their levels in the blood. Nicotine also increases blood lymphocyte numbers. Smoking has several consequences on lymphocyte immunological responses. Immunoglobulin synthesis, T4/T8 lymphocyte ratio alteration, increased NK activity, and decreased mitogen-induced lymphocyte transformation are notable. Silverman & colleagues. discovered that smokers had elevated leukocytes, particularly “T” lymphocytes.

We know that arterial blood oxygen saturation is vital for everyone. Ozdal et al. showed that non-smokers had greater oxygen saturation of haemoglobin than smokers (P <0.05), which matched our findings. Nicotine and carbon monoxide, which combine to transport proteins like haemoglobin and myoglobin, are the two primary components of cigarette smoke that may limit oxygen flow to all body tissues.

Our study's strength was that real subjects were chosen using inclusion and exclusion criteria. The p value was calculated meticulously to verify statistical significance. In asymptomatic smokers, finding respiratory damage early is critical. Smoking cessation may be beneficial in patients receiving therapy and can be an empirical cornerstone in those who are resistant to stopping. The study's sample size is too small; larger samples should be used. This type of research must continue to determine whether smoking cessation is beneficial and how much smoking must be reduced to achieve health benefits.

CONCLUSION

To summarise, smoking is one of the leading preventable causes of CVD mortality and morbidity. In our study, cigarette smoking increased WBC count and subtypes, indicating increased inflammatory responses. The total and DLC were found to be altered in smokers, and should be considered during diagnosis, interpretation, and treatment. Tobacco smoking reduces haemoglobin oxygen saturation. Smoking cessation can improve changes that are sensitive to smoking cessation. Monitor these haematological markers regularly in smokers to detect early alterations and avert future catastrophes.

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