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Assessment of the effect of smoking on haematological profile and SpO_2 of hemoglobin: A comparative research

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Abstract
Aim: To evaluate the changes associated with the extent of adverse effects of tobacco smoking in total and differential leukocyte count (DLC) and oxygen saturation of hemoglobin in healthy smokers and non-smokers.
Materials and Methods: This analytical observational study was carried out in the Department of Physiology, India for the period of 1 year. A total of 200 clinically healthy volunteers of Bihar, in the age group of 20–60 years participated in the present study. Individuals with a history of smoking cigarettes/bidis daily for at least 6 months were considered as smokers. Another 100 non-smokers of the same age group were included separately in this study as a control group. TLC, DLC and other parameters were analyzed using standard methods.
Results: A total of 320 subjects (160 non- smokers and 160 smokers cases), in which baseline demographic parameters (age and BMI) are compared between smokers and non-smokers. No significant difference between the baseline demographic parameters between the smokers and non-smokers ensures optimum comparison avoiding bias. The difference between TLC, lymphocyte count, monocyte count, granulocyte count, and oxygen saturation of hemoglobin among smokers and non-smoker subjects. The mean values of TLC (P<0.001), lymphocyte count (P<0.001), monocyte count (P=0.01), and granulocyte count (P=0.01) were significantly higher in smokers as compared to non-smokers, while the mean values of SpO_2 (P = 0.02) were significantly lower in smokers as compared to non-smokers.
Conclusion: The study has shown that altered values of TLC and DLC and oxygen saturation of hemoglobin in smokers should be considered during diagnosis, interpretation of result, and treatment of patients. A high TLC and DLCs exhibited in this research may be responsible for chronic inflammation and subsequent high risk of CVD in smokers. Therefore, quitting smoking should be encouraged for better health.

Keywords: SpO_2, oxygen saturation of hemoglobin, total and differential leukocyte count, smokers

Introduction
The greatest serious threat to public health is the prevalence of smoking. Organ systems such as respiratory, reticuloendothelial, and cardiovascular have been shown to be negatively affected by it [1]. An estimated 2.4 billion individuals throughout the globe have used tobacco at some point in their lives, according to the World Health Organization [2]. Tobacco use is expected to kill 8.3 million people in 2030 and one billion people worldwide by the end of the twenty-first century, according to the World Health Organization [2].
The time it takes to smoke a cigarette is nearly equivalent to the time it takes to lose a cigarette [3]. One's life expectancy is shortened by an average of eight years at age 15, and by an average of four years at age 25 if one starts smoking. Cigarette smoking's extra mortality is mostly caused by heart disease, cancer, and a variety of respiratory illnesses and conditions [4].
Comparing smokers to nonsmokers, the average risk of lung cancer is 16 times higher, the risk of COPD is 12 times higher, and the risk of a myocardial infarction is two times higher.
Tobacco use has been linked to a variety of health issues, including haematological parameters, peripheral vascular disease, and stroke. However, it wasn't until 1964 that a US Surgeon General's study warned of a possible link between smoking and emphysema that the connection was first discovered [6].
Tobacco smoking is the leading cause of ischemic heart disease and mortality among those between the ages of 30 and 40 with no other known risk factors for coronary artery disease, according to a study published in the journal Circulation [7].
Hemoglobin cut-off values for smokers should be lowered because chronic smoking seems to push the dissociation curve northward, obscuring the influence of smoking on the diagnosis of anaemia in smokers, according to this study [8].

It is clear that the respiratory system's primary function is to provide oxygen to the bloodstream. A tiny amount of molten oxygen remains in the alveoli after haemoglobin transports it there.

In the blood, there is 0.003 ml of O2 per 100 ml, with a haemoglobin molecule containing around 1.34 ml of O2 per gram. Oxygen saturation is a term used to describe how much oxygen is in the circulation and being delivered by haemoglobin (SpO2).

Smoking is a leading cause of disease and death in affluent nations, where it affects 20–40% of women and 30–40% of men, compared to 2–10% of women and 40–60% of men in underdeveloped countries. As a result, smoking cigarettes harms the lungs as well as other organ systems [9]. The present study thus investigates the effect of tobacco smoking on total and differential leukocyte count (DLC) and oxygen saturation of hemoglobin for better diagnosis, interpretation of results, and treatment.

Materials and Methods

This analytical observational study was carried out in the Department of Physiology, after taking the approval of the protocol review committee and institutional ethics committee. After taking informed consent detailed history was taken from the Participant.

Methodology

A total of 320 clinically healthy volunteers in the age group of 20-60 years participated in the present study. Individuals with a history of smoking cigarettes/bidis daily for at least 6 months were considered as smokers. Ex-smokers or past smokers were excluded from the study. Smokers are defined as someone who, at the time of the study, smokes any tobacco product either daily or occasionally, while a non-smoker is someone who, at the time of the study, does not smoke at all. Moreover, an ex-smoker is someone who was formerly a daily or occasional smoker but currently does not smoke at all.

Unhealthy adults with any history of acute or chronic illness, bleeding and bleeding disorders, drug addiction, and if they had donated blood within the previous 6 months were not included in the study. Pregnant women were also excluded from the study.

Anthropometric parameters which include height, weight, and body mass index (BMI) was taken. Information of the smoking habits was obtained by a questionnaire.

Estimation of total, DLC, and oxygen saturation of hemoglobin: After taking antiseptic precautions, blood samples were taken from the antecubital vein and collected into 3-5 ml ethylenediaminetetraacetic acid (EDTA) vacutainers. The EDTA blood samples were processed using automated hematology cell counter for total leukocyte count (TLC) (in thousands) and DLC (in percentage). Oxygen saturation of hemoglobin was done using fingertip pulse oximeter.

Statistical analysis

The data were analyzed using statistical software, SPSS (ver. 20.0) (IBM Inc, Armonk, New York, USA). Descriptive statistics and bivariate and regression analysis were carried out to find association and correlation and considered significant at $P<0.05$. The internal consistency, i.e., Cronbach's alpha value was 0.87 that was suggestive of high reliability.

Results

Table 1 shows that in a total of 320 subjects (160 non-smokers and 160 smokers cases), in which baseline demographic parameters (age and BMI) are compared between smokers and non-smokers. No significant difference between the baseline demographic parameters between the smokers and non-smokers ensures optimum comparison avoiding bias.

Table 1: Comparison of baseline demographic parameters of smokers and non-smokers subjects

Smoking status	N=320	Range	Minimum	Maximum	Mean	Standard deviation
Non-smoker						
Age	100	33	18	61	32.67	9.457
BMI	100	21.37	17.04	37.08	23.1897	3.16224
Smoker						
Age	100	30	20	57	33.24	7.276
BMI	100	12.78	19.11	30.87	25.1528	2.85245
p-value	>0.05					

Test applied: student t-test, BMI: Body mass index

Table 2 shows the difference between TLC, lymphocyte count, monocyte count, granulocyte count, and oxygen saturation of hemoglobin among smokers and non-smoker subjects. The mean values of TLC ($P<0.001$), lymphocyte count ($P<0.001$), monocyte count ($P=0.01$), and granulocyte count ($P=0.01$) were significantly higher in smokers as compared to non-smokers, while the mean values of SpO^2 ($P = 0.02$) were significantly lower in smokers as compared to non-smokers.

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

Parameter	Non-smokers (N=160)	Smokers (N=160)	*P*-value
TLC	6.8787	7.3577	<0.001
DLC			
Lymphocyte count	0.3619	0.3723	<0.001
Monocyte count	0.061	0.0537	0.01
Granulocyte count	0.5878	0.50	0.01
SpO_2	0.9767	0.9830	0.02

Test applied: student t-test, TLC: Total leukocyte count, DLC: Differential leukocyte count

Discussion

The results of our study showed a significant increase in the total WBC, lymphocyte count, monocyte count, and the granulocyte count in smokers as compared to non-smokers. We have also exhibited that oxygen saturation of hemoglobin was found to be lower in smokers than in non-smokers. Pedersen *et al*. in the Copenhagen general population study found that smoking causes increased blood leukocytes, neutrophils, lymphocytes, and monocytes [10]. Asif *et al*. in their study also found that regular smokers exhibited significantly greater WBCs count compared to non-smokers ($P = 0.027$) [11]. They also found that the WBC count among male smokers was higher which also suggests that they may have greater risk of developing both atherosclerosis and CVDs than female smokers and non-smokers [11]. Airway epithelium acts as a physical barrier obstructing the entry of inhaled noxious particles into the submucosa. Leukocytosis has emerged as a potential marker of tissue damaged caused by cigarette smoke. Moreover, a rise in its count may account for an increased incidence of CVD through a plethora of postulated pathogenic mechanisms that mediate inflammation,

block microvasculature at various junctures, and induce hypercoagulability. Gitte and Taklikar also found in their study a sharp increase in total leukocyte count values of smokers with respect to the non-smokers [12]. Anitha and Manjunath also confirm this empirical positive association between smoking and total leukocyte count [13]. Our study also aimed at DLCs due to a probable association between cigarette smoking with TLC. Evidence suggests a strosng possibility of this association, however, its effect on the DLC is still a matter of debate. In our study, it was also demonstrated that there was a statistically significant increase in all leukocyte subtypes. Zei-Shung *et al*. in their study also found significantly higher TLCs along with its subtypes in smokers [14]. One of the possible mechanistic hypotheses of this increased TLC is the extracted glycoprotein from the tobacco leaf, which stimulates lymphocyte proliferation and differentiation by intermingling with a specific membrane component, commonly seen in antigenic response [15]. As for lymphocyte count, Shenwai and Aundhakar reveal that the lymphocyte count increases significantly from 32.4% in non-smokers to 38.3% in smokers, while neutrophil count showed a slight fall in smokers than non-smokers, however, the difference for neutrophil count is statistically non-significant. Furthermore, no significant change was observed in eosinophil, basophil, and monocyte counts [16]. It is quite evident that lymphocytosis is attributed to both chronic tissue damage and inflammation produced by toxic substances found in tobacco smoke. It has also been suggested that smoke causes stimulation of respiratory bronchial tract inflammatory markers, thus inducing their increase in the blood. Moreover, nicotine induces an increase in blood lymphocyte counts too [10]. Cigarette smoking encompasses a myriad of effects on the immune response of lymphocyte cells. Some of the noteworthy examples include immunoglobulin production, T4/T8 lymphocyte ratio change, enhanced NK activity, and low mutagen induced lymphocyte transformation [11]. In his research, Silverman *et al*. found that that smokers exhibit marked elevation in leukocytes especially T lymphocytes [17]. We are aware that saturation of arterial blood to oxygen is essential for all individuals. Ozdal *et al*. reported that non- smoker individuals had significantly higher oxygen saturation of hemoglobin than smoker individuals (P<0.05) which was similar as found in our study. The two main ingredients of cigarette smoke that potentially reduces oxygen supply to all tissues of the body are nicotine and carbon monoxide by combining themselves to transport proteins such as hemoglobin and myoglobin [9].

The strength of our study was that the authentic subject selection was done on the basis of inclusion and exclusion criteria. Meticulously statistical analysis was done and p value was obtained to prove statistically significance. Earlier detection of respiratory damage in asymptomatic smokers will prevent future complications. Reduction in smoking may prove useful in subjects undergoing treatment and can surely serve pivotal and an empirical cornerstone in people who are resistant to quitting. Limitations involve the limited sample size; the research should be carried out with larger sample sizes. Future direction in this kind of research is necessary to determine whether smoking cessation is advantageous and if yes to what extent smoking needs to be reduced for health benefits to occur.

Conclusion

This study has shown that the total and DLC were altered in smokers and thus should be considered during diagnosis, interpretation of result, and treatment of patients. Tobacco smoking has a negative impact on oxygen saturation of hemoglobin. Reduction in smoking can improve the changes which are sensitive to change in smoking intake. We advise regular monitoring of the above-mentioned hematological parameters in smokers to detect early changes and avoid future catastrophic outcomes.

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