ISSN: 0975-1556

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2021; 13(4); 230-235

Original Research Article

Variation in Total and Differential Leukocyte Count, As Well As Oxygen Saturation of Haemoglobin, Between Healthy Smokers and Non smokers

Sunil Kumar[1], Bharat Kumar[2], Richa Kumari[3], Malti Kumari[4]

[1]Tutor,Department of Physiology, Jan Nayak Karpoori Thakur Medical College and Hospital, Madhepura, Bihar, India

[2]Tutor,Department of Physiology, Jan Nayak Karpoori Thakur Medical College and Hospital, Madhepura, Bihar, India

[3]Senior Resident, Department of Physiology, AIIMS, Rishikesh, Uttarakhand, India.

[4]Professor and HOD, Department of Physiology, Jan Nayak Karpoori Thakur Medical College and Hospital, Madhepura, Bihar, India

Received: 05-06-2021 / Revised: 17-06-2021 / Accepted: 24-07-2021
Corresponding author: Dr. Bharat Kumar
Conflict of interest: Nil

Abstract

Aim: To evaluate the total and differential leukocyte count and oxygen saturation of hemoglobin changes in healthy smokers and non-smokers in Bihar region. **Methods:** The present Prospective study was conducted in the Department of Physiology, Jan Nayak Karpoori Thakur Medical College and Hospital, Madhepura, Bihar, India from February 2019 to feb 2020. A total of 140 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 1 year were considered as smokers. **Results:** The mean values of TLC ($P < 0.001$), lymphocyte count ($P < 0.001$), monocyte count ($P = 0.03$), and granulocyte count ($P = 0.01$) were significantly higher in smokers as compared to non-smokers, while the mean values of SpO_2 ($P = 0.03$) were significantly lower in smokers as compared to non-smokers. A significant difference was observed ($P < 0.001$) in TLC between non-smokers and smokers (103/mm3). Smoking builds an inflammatory environment in the human body which, in turn, triggers immune response in general, subsequently raising the leukocyte count. **Conclusion:** We concluded 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.

Keywords: Leukocyte Count, Haemoglobin, Smokers and Non smokers.

Introduction

Cigarette smoking is one of the leading causes of death globally. Numerous studies indicated that smoking had adverse effects on human health and represented a predisposing factor for the 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. Smokers have elevated risk of all varieties cardiovascular diseases (CVD), peripheral vascular disease, and cerebrovascular diseases, for example, stroke. In India, 337 million people above 10yrs of age consume tobacco. The World Health Organization predicts that tobacco deaths in India may exceed 1.5 million annually by 2020[1]. Cigarette smoking is an important and independent risk factor for atherosclerosis, coronary artery disease, peripheral vascular disorders, etc and several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile[2,3]. However, inspite of all that information, there is still much controversy about which component in the lipid profile are mainly altered in response to cigarette smoking, and whether those lipid profile components influence other parts directly or indirectly and vice versa. Differing results were obtained by various investigators, for example, Siekmeier et al.[4] concludes that HDL-C levels are same for smokers and non-smokers whereas Ito et al.[5] obtained low levels of HDL-C in cigarette smokers. Although some studies[6,7] have reported that leukocyte count increases with the number of cigarettes smoked daily and decreases after cessation of smoking, data on smoking characteristics, such as duration of smoking, intensity of smoking, smoked pack-year and their association with leukocytes count is scanty.

Material and methods

The present Prospective study was conducted in the Department of Physiology, Jan Nayak Karpoori Thakur Medical College and Hospital, Madhepura, Bihar, India from February 2019 to February 2020. A total of 140 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 1 year were considered as smokers. Ex-smokers or past smokers were excluded from the study. Classification criteria as suggested by WHO (1998) were used as below: 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.[8] 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 and those who had delivered within 3 months were also excluded from the study.

Methods

Anthropometric parameters which include height, weight, and body mass index (BMI) were 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 ml ethylene diamine tetra acetic acid (EDTA) vacutainers (Akuret, eastern medkit limited). The EDTA blood samples were processed using MS-9 automated hematology cell counter for total leukocyte count (TLC) (in thousands) and DLC (in percentage). Samples were processed on the same day approximately within 3–5 hours of collection. Oxygen saturation of hemoglobin was done using fingertip pulse oximeter Nidek Medical 5300. Subject was made to sit quietly for 5–15 min. A non-invasive sensor was placed on the index finger (occasionally on another finger if a reading was not obtained promptly or if the index finger was missing), waited for 10–15 s after the first reading on the screen and then noted six consecutive readings at an interval of 10 s.

Average of six measurements was taken as final value.

Statistical Analysis

Simple correlation analysis was performed to evaluate the association between the above said parameters. Alpha levels were set at $P < 0.05$ and data are expressed as mean ± standard deviation. All statistical analyses were completed with SPSS version 21.0 (SPSS Inc., Chicago, IL, USA).

Results

Table 1 shows that in a total of 140 subjects (100 non- smokers and 40 smoker's 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 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 ($P = 0.03$), and granulocyte count ($P = 0.01$) were significantly higher in smokers as compared to non-smokers, while the mean values of SpO2 ($P = 0.03$) were significantly lower in smokers as compared to non-smokers. A significant difference was observed ($P < 0.001$) in TLC between non-smokers and smokers ($103/mm^3$). Smoking builds an inflammatory environment in the human body which, in turn, triggers immune response in general, subsequently raising the leukocyte count.

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

Smoking status	n	Range	Minimum	Maximum	Mean		Standard deviation
Non-smoker							
Age	**100**	**38.5**	**20**	**60**	**32.17**	**0.88**	**10.36**
BMI	**100**	**21.62**	**15.92**	**37.12**	**25.69**	**0.36**	**4.11**
Smoker							
Age	**40**	**30.5**	**22**	**57**	**33.87**	**1.69**	**9.67**
BMI	**40**	**12.36**	**19.62**	**30.45**	**26.97**	**0.46**	**3.66**

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

Parameter	Non-smokers (*n*=100)	Smokers (*n*=40)	*P*-value
TLC	**7.22**	**7.88**	**<0.001**
Lymphocyte count	**0.38**	**0.37**	**<0.001**
Monocyte count	**0.055**	**0.057**	**0.03**
Granulocyte count	**0.59**	**0.61**	**0.01**
SpO_2	**0.99**	**0.99**	**0.03**

Discussion

Tobacco smoking has been associated with a variety of major hematological ailments. 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[9]. Asif et al.

in their study also found that regular smokers exhibited significantly greater WBCs count compared to non-smokers (P = 0.027)[10]. 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[10]. 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[11]. Anitha and Manju Nath also confirm this empirical positive association between smoking and total leukocyte count[12]. Our study also aimed at DLCs due to a probable association between cigarette smoking with TLC. Evidence suggests a strong 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[13]. 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[14]. 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[15]. 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 mitogen- induced lymphocyte transformation[12]. In his research, Silverman et al. found that that smokers exhibit marked elevation in leukocytes especially “T” lymphocytes[16].

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[17].

Conclusion

We concluded 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.

Reference

1. Murray CJ, Lopez AD. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020. Cambridge, Massachussets: Harvard School of Public Health, 1996.
2. Guedes DP, Guedes JE, Barbosa DS, de Oliveira JA. Tobacco use and plasma lipid-lipoprotein profile in adolescents. Rev Assoc Med Bras. 2007; 53(1): 59-61.
3. Aggarwal A, Aggarwal S, Sarkar PG, Sharma V. Predisposing factors to premature coronary artery disease in young (age 45 years) smokers: a single center retrospective case control study from India. J Cardiovasc Thorac Res. 2014;6(1):15-9.
4. Siekmeier R, Wulfroth P, Wieland H, Gross W, Marz W. Low-density lipoprotein susceptibility of in vitro oxidation in healthy smokers and nonsmokers. Clin Chem. 1996; 42:524–30.
5. Ito T, Nishiwaki M, Ishikawa T, Nakamura H. CETP and LCAT activities are unrelated to smoking and moderate alcohol consumption in healthy normolipidemic men. Jpn Circ J. 1995; 59(8): 541-43.
6. Yeung MC, Buncio AD. Leukocyte count, smoking, and lung function. Am J Med. 1984; 76: 31-5.
7. Friedman GD, Siegelaub AB, Seltzer CC, Feldman R, Collen MF. Smoking habits and the leukocyte count. Arch Environ Health. 1973; 26:137- 40.
8. World Health Organization. Guidelines for Controlling and Monitoring the Tobacco Epidemic. Geneva: World Health Organization; 1998. p. 76-101.
9. Pedersen KM, Çolak Y, Ellervik C, Hasselbalch HC, Bojesen SE, Nordestgaard BG. Smoking and increased white and red blood cells. Arterioscler Thromb Vasc Biol 2019; 39:965-77.
10. Asif M, Karim S, Umar Z, Malik A, Ismail T, Chaudhary A, et al. Effect of cigarette smoking based on hematological parameters: Comparison between male smokers and nonsmokers. Turk J Biochem 2013; 38:75-80.
11. Gitte RN, Taklikar R. Effect of cigarette smoking on erythrocyte sedimentation rate and total leukocyte count. Natl J Physiol Pharm Pharmacol 2018; 8:1429-31.
12. Anitha K, Manjunath H. Does leucocyte count vary with beedi smoking? Natl J Physiol Pharm Pharmacol 2014; 4:69-71.
13. Zei-Shung H, Kuo-Liong C, Chi-Yu Y, Keh-Sung T, Chiu- Hwa W. Peripheral differential leukocyte counts in humans vary with hyperlipidemia, smoking, and body mass index. Lipids 2001; 36:237-45.
14. Aula FA, Qadir FA. Effects of cigarette smoking on some immunological and hematological parameters in male smokers in Erbil city. Jordan J Biol Sci 2013; 6:159-66.
15. Shenwai MR, Aundhakar NV. Effect of cigarette smoking on various hematological parameters in young male smokers. Indian J Basic Appl Med Res 2012; 2:386-92.
16. Silverman NA, Potvin C, Alexander JC Jr., Chretien PB. In vitro lymphocyte reactivity and T cell levels in chronic cigarette smokers. Clin Exp Immunol 1975; 22:285-92.
17. Ozdal M, Pancar Z, Çinar V, Bilgic M. Effect of smoking on oxygen saturation in healthy sedentary men and women. EC Pulmonol Respir Med 2017; 4:178-82.