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RESEARCH ARTICLE

Effect of cigarette smoking on erythrocyte sedimentation rate and total leukocyte count

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ABSTRACT

Background: Cigarette smoking is one of the major lifestyle factors which influence the health of human beings. Erythrocyte sedimentation rate (ESR) is a certain biomarker of inflammation. It was a significant predictor of heart failure**. Aims and Objectives**: This study was conducted to find the effect of cigarette smoking on ESR and total leukocyte count (TLC). **Materials and Methods**: A total of 120 healthy male smokers and 120 healthy male non-smokers among hospital employees and people from surrounding areas of Navodaya Medical College, Raichur (India), were studied. The TLC was done using Beckman Coulter Automatic Analyzer; ESR estimation was done by Westergren's method. **Results**: The mean ESR for smokers is 11.74 mm/h and for non-smokers 7.38 mm/h. The mean TLC for smokers is 8050 per mm^3 and for non-smokers 6858 per mm^3. The difference between mean ESR and TLC of smokers and non-smokers was statistically significant ($P < 0.0001$). **Conclusion**: We concluded that, in smokers, ESR and TLC increased significantly. Regular monitoring of these two parameters in smokers is advised.

KEY WORDS: Cigarette Smoking; Erythrocyte Sedimentation Rate; Total Leukocyte Count

INTRODUCTION

Cigarette smoking is one of the major lifestyle factors influencing the health of human beings. Cigarette smoking is a serious health problem to smokers and to those exposed to it. Lung cancer is the major danger for smokers, but diseases of the blood vessels and the heart account for one-third of all excess death in smokers.[1] It is a powerful risk factor for atherosclerosis and coronary heart disease. There is a direct relationship between number of cigarette smoked and cardiovascular morbidity and mortality.[2]

A cigarette smoke contains over 4000 chemicals.[3] A cigarette smoker is exposed to a number of harmful substances including nicotine, free radicals, carbon monoxide, and other gaseous products. All these substances potentially affect atherogenesis and thrombosis.[4] Nicotine is now speculated to be responsible for the development of dependence, while carbon monoxide and other combustion substances are responsible for smoking-related cardiovascular disorders.[5]

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Erythrocyte sedimentation rate (ESR) is a certain biomarker of inflammation. It was a significant predictor of heart failure.[6] Cigarette smoke induces endothelial damage by producing free radicals such as nitric oxide and hydrogen peroxide. These free radicals cause oxidative stress which promotes a systemic acute phase reaction. This reaction increases inflammatory cytokines, C-reactive protein, fibrinogen, blood cell count, whole blood viscosity, and roulaux formation and eventually leads to rise in ESR values.[2]

Total leukocyte count (TLC) has been demonstrated in several studies to be a strong independent predictor of future coronary heart disease. Recent studies suggest that an increase in TLC is instrumental in pathogenesis of myocardial ischemia.[7]

Several studies on hematology and smoking had been conducted on Caucasian and other ethnic groups, but very less studies done in the past on Indian population. Thus, the present study was undertaken to find the effect of cigarette smoking on two hematological parameters (ESR and TLC) and to apply this information for better investigation and management.

MATERIALS AND METHODS

Subjects

A total of 120 healthy male smokers and 120 healthy male non-smokers following an informed consent were studied. Both smokers and controls (non-smokers) were hospital employees and people from surrounding areas of Navodaya Medical College, Raichur (India). Both smokers and controls were aged 30–60 years. This cross-sectional study carried out during February–September 2017 at the Department of Physiology, Navodaya Medical College, Raichur (India), with prior permission of ethical and research committee of Navodaya Medical College, Raichur.

Male smokers with the frequency of 20 or more cigarette per day with more than 20-year duration of smoking were selected for the study. Male smokers with blood pressure 100–140 mm Hg systolic and 60–90 mm Hg diastolic were selected for the study. Male smokers had no history of diabetes mellitus, and their random blood sugar was 100–140 mg/dl.

Controls (non-smokers) had no history of diabetes mellitus, and their random blood sugar was 100–140 mg/dl. They had blood pressure 100–140 mm Hg systolic and 60–90 mm Hg diastolic.

Method

Informed written consent was obtained from each subject. This study has got the permission from the ethical and research committee of Navodaya Medical College, Raichur (India). 5 ml of venous blood was withdrawn with minimum stasis into a clean disposable syringe 5 ml. The blood samples were stored in EDTA bulb. The TLC was done using a Beckman Coulter Automatic Analyzer; ESR estimation was done by Westergren's method.

Statistical Analysis

The data were entered in the computer and analyzed using NCSS statistical package. The differences in means of ESR and TLC were tested for statistical significance by independent sample *t*-test.

RESULTS

Table 1 shows the mean, maximum, minimum, standard deviation, and standard error of age, ESR, and TLC of smokers and non-smokers. The mean ESR for smokers is 11.74 mm/h and for non-smokers 7.38 mm/h. The difference between mean ESR of smokers and non-smokers was statistically significant ($P < 0.0001$). The mean TLC for smokers is 8050 per mm^3 and for non-smokers 6858 per mm^3. The difference between mean TLC smokers and non-smokers was statistically significant ($P < 0.0001$). The mean age for smokers is 44.93 years and for non-smokers is 43.84 years. The difference between the mean age of smokers and non-smokers was statistically non-significant ($P = 0.0646$).

DISCUSSION

In the present study, the mean ESR for smokers is 11.74 mm/h and for non-smokers is 7.38 mm/h. Thus, ESR values show a highly significant rise ($P < 0.01$) in smokers as compared to non-smokers. In the present study, the mean TLC for smokers is 8050 per mm^3 and for non-smokers 6858 per mm^3. Thus, TLC values of smokers showed a sharp increase with respect to the non-smokers.

This indicates a strong association of ESR as a marker of systemic inflammation with smoking. These findings are similar as Islam *et al*. Cigarette smoke can damage endothelium by producing free radicals such as nitric oxide and hydrogen peroxide. Systemic acute phase reaction promoted by this oxidative stress will increase inflammatory cytokines, C-reactive protein, fibrinogen, blood cell count, blood viscositym, and rouleaux formation. These things lead to rise in ESR values. Hence, the possible mechanism of increase ESR in the present study is chronic inflammatory response mediated by particulates of cigarette smoke.[6] The high TLC in male smokers in this

Table 1: Age, ESR, and TLC of smokers and non-smokers

Variable	*n*	Min.	Max.	Mean	S.D.	S.E.	*P* value
ESR smokers (mm/h)	120	02	43	11.74	10.778	1.524	0.006
ESR non-smokers (mm/h)	120	02	10	7.38	1.602	0.227	0.006
TLC of smokers (per mm^3)	120	4600	11600	8050	1879.562	265.810	0.0001
TLC of non-smokers (per mm^3)	120	4400	10900	6858	34862.03	176.128	0.0001
Age of smokers (years)	120	39	56	44.93	4.168	0.3805	0.0646
Age of non-smokers (years)	120	32	56	43.84	4.911	0.4483	0.0646

ESR: Erythrocyte sedimentation rate, TLC: Total leukocyte count, mm/h: Millimeter/hour, Min: Minimum, Max: Maximum, SD: Standard deviation, S E: Standard error, *n*: Sample size

study is similar to other published reports.[8,9] Increased TLC in smokers contributed by the irritant effect of cigarette smoke that causes the inflammation on respiratory tract. A published literature has reported that chronic bronchitis or leukocytes released from lymphoid organ to the periphery were two major causes for increased TLC in smokers.[10]

The present study results show a strong association of cigarette smoking and increased ESR and TLC values in smokers. The ESR was done by traditional Westergren's method rather than using an autoanalyzer. The sample included in this study only on basis of questionnaire and clinical examination. Additional research is clearly necessity to determine that what extent smoking should be reduced for health benefit.

CONCLUSION

Thus, we concluded that, in smokers, ESR and TLC increased significantly. Increase in ESR is due to chronic inflammatory response mediated by particulates of cigarette smoke.[6] Many blood cells present in the blood can make the smoker's blood viscous. Thus, blood does not flow efficiently and contributes to the formation of clot. This will increase the risk of complications such as stroke, deep vein thrombosis, pulmonary embolism, etc.[10] Regular monitoring of these ESR and TLC in smokers is advised so that changes can be detected at an earlier stage for the implementation of preventive measures such as cessation of smoking.

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