



Original Research Article

Effects of cigarette smoking on erythrocyte sedimentation rate, platelet count, total and differential leucocyte counts in adult male smokers

Safia Sultana¹, Nabila Afsar², Mohammed Jawad¹, Mohammed Abdul Hannan

Hazari³ 

¹Graduate Student, ²Department of Pathology, ³Department of Physiology; Deccan College of Medical Sciences, DMRL 'X' Road, Kanchanbagh, Hyderabad-500058, Telangana, India.

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Abstract

Smoking is one of the leading causes of death worldwide. Smokers have higher risk for coronary heart disease, atherosclerosis, acute myocardial infarction, hypertension, clotting disorders, inflammation, respiratory diseases, cancers, etc. A cigarette smoker is exposed to a number of harmful substances. In this study we hypothesized that smoking causes inflammatory reactions and induces hyperthrombic state in the body which may be reflected in erythrocyte sedimentation rate (ESR), total leucocyte count (TLC), differential leucocyte count (DLC) and platelet count values. The purpose of the study was to study the effects of cigarette smoking on erythrocyte sedimentation rate, total leucocyte count and platelet count in adult male smokers and to compare the results with non-smokers and to establish a relationship between the duration and quantity of smoking with the change in ESR, TLC, DLC and platelet count. A cross sectional study was conducted in the department of Pathology on 86 healthy male subjects (smokers=43 and non-smokers=43). ESR was estimated using Westergrens method. TLC, DLC and platelet counts were estimated using HORIBA Pentra ES60 auto-analyzer. TLC and basophil counts were significantly higher in smokers than in non-smokers ($p < 0.05$). The mean value of ESR was higher among smokers than non-smokers but it was statistically insignificant. Platelets counts showed no significant difference between smokers and non-smokers. No correlation was observed in various blood parameters and smoking (in pack years). We conclude that smoking initiates an inflammatory response as evidenced from raised TLC, monocyte and basophil counts.

Introduction

Smoking is amongst the leading causes of death worldwide. In India, 24.3% of the adult men are tobacco smokers [1]. 1 to 1.5 million people die every year in India due to consequences of smoking. Cigarette smoke contains a number of

harmful substances like carbon monoxide, tar, nicotine, etc. which exert effects on many systems of the body. Smokers are at increased risk for coronary artery disease (CAD), atherosclerosis, acute myocardial infarction, hypertension (HTN),

Corresponding author

Ms. Safia Sultana

Graduate Student,

Deccan College of Medical Sciences,
DMRL 'X' Road, Kanchanbagh, Hyderabad-500058,
Telangana, India.

Phone: +91-9966196410

Email: sultanasafia2013@gmail.com



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clotting disorders, inflammation, respiratory diseases, cancers, etc.

Previous studies have found that smoking causes oxidative stress by releasing free radicals like hydrogen peroxide (H₂O₂) and nitric oxide (NO). These free radicals cause endothelial damage leading to increased whole blood viscosity, rouleaux formation and inflammatory reactions. This can be reflected by change in erythrocyte sedimentation rate (ESR) [2,3,4,5]. Previous studies have reported that smoking can effect total leucocyte counts (TLC). Smoking has an irritant effect on respiratory system with resultant inflammation, releasing inflammatory cytokines which can influence growth, differentiation and activation of leucocytes [6,7,8]. Leucocyte counts are considered as an independent risk factor for various cardiovascular diseases [9]. Studies on effects of smoking on differential leucocyte counts (DLC) have shown conflicting results.

In smokers, hyperthrombic state could be due to increased platelet activity which may be reflected in platelet count. Increased platelet activity may trigger clotting cascade that can cause occlusive vascular disease. Previous studies on platelet count have reported inconclusive results. Some studies reported platelet count increases due to smoking [10] whereas others have reported no significant change [5]. In view of these published literature, we hypothesized that smoking causes inflammatory reactions and induces prothrombic / procoagulant state in the body which may be reflected in ESR, TLC, DLC and platelet count.

Materials and methods

A cross-sectional study was conducted in the department of Pathology of our medical college between May and June 2016. A total of 86 male subjects (smokers: 43; non-smokers: 43) in the age group of 18-40 years were included in the study. Ethics approval was obtained from Institutional Review Board (IRB number: 2016/11/003) before the start of this study.

Selection of subjects

Inclusion criteria: Apparently healthy smokers (n=43) in the age group 18-40 years who smoke one or more cigarette per day and healthy non-smokers (n=43) of the age group 18-40 years were included in the study.

Exclusion criteria: Male subjects suffering from coagulation disorders, hypertension, diabetes, infections, acute or chronic respiratory illness or debilitating illness have been excluded from the study. Male subjects taking non-steroidal anti-inflammatory drugs (NSAIDs) or any other anti-platelet aggregation drugs were excluded from this

study. Non-smokers who are subjected to passive smoking were also excluded from the study.

Methodology

The subjects were divided into 2 groups i.e. smokers (n=43) and non-smokers (n=43). Informed consent was obtained from each subject. The particulars of the subjects and required history were taken from all the subjects. Under aseptic precautions, 4ml venous blood sample was collected from the median cubital vein using a 5ml disposable syringe. 2ml of blood was mixed in K3 EDTA containing vacutainer for estimation of red blood cell (RBC) count, hemoglobin (Hb) concentration, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RCDW), total leucocyte count (TLC), differential leucocyte count (DLC) i.e. counts of neutrophils, eosiniphils, basophils, lymphocytes and monocytes; platelet count, plateletcrit, mean platelet volume (MPV), platelet distribution width (PDW) using HORIBA Pentra ES60 auto-analyzer. Additional parameters documented were atypical lymphocytes and large immature cells. 2ml of blood was mixed in 3.8% trisodium citrate (in the ratio of 1:4) containing vacutainer for estimation of erythrocyte sedimentation rate by Westergren's method. The samples were evaluated within 1 hour of sample collection.

Manual confirmation of platelet count was done in few samples by making blood smear and staining with Leishman's stain and examination under microscope. Controls were used prior to testing on auto-analyzer for quality control.

Statistical analysis

Data collected was analyzed using independent sample 't' test and partial correlation available in SPSS 17.0 statistical software (SPSS Inc., Chicago, USA).

Results

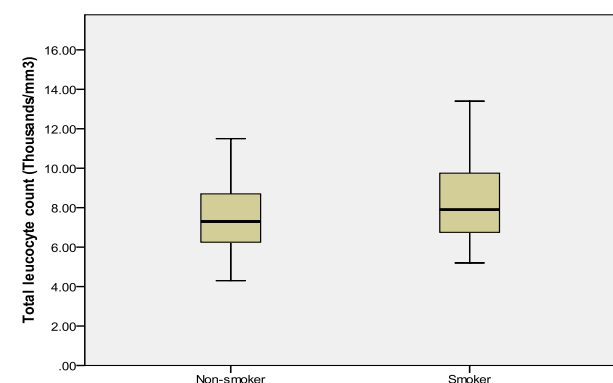


Figure 1. Differences in TLC between smokers and non-smokers

Table 1: Differences in hematological parameters between smokers and non-smokers

	Non-smokers (n=43)	Smokers (n=43)	p value
Age (years)	21.49±4.54	26.02±6.48	0.000*
No. of cigarettes (per day)	-	8.35±7.65	
Duration of smoking (years)	-	8.09±6.22	
ESR (mm/1 st hour)	6.47±4.70	10.51±12.58	0.053
TLC (thousands/mm ³)	7.41±1.63	8.44±2.28	0.019*
Platelet count (lakhs/mm ³)	2.67±0.64	2.65±0.57	0.909
RBCs (millions/mm ³)	5.36±0.39	5.24±0.44	0.173
Hemoglobin (gm/dl)	15.11±0.98	14.77±2.05	0.320
Hematocrit (%)	45.82±2.86	44.74±3.73	0.136
MCV (fl)	85.65±5.04	85.72±6.24	0.955
MCH (pg)	28.27±2.05	28.72±2.57	0.363
MCHC (%)	32.99±0.68	33.50±0.91	0.004*
RCDW (%)	12.28±1.02	12.37±1.53	0.772
Neutrophils (thousands/mm ³)	4.10±0.98	4.54±1.67	0.135
Lymphocytes (thousands/mm ³)	2.52±0.65	2.72±0.70	0.171
Monocytes (thousands/mm ³)	0.53±0.18	0.62±0.20	0.021*
Eosinophils (thousands/mm ³)	0.23±0.16	0.27±0.17	0.292
Basophils (thousands/mm ³)	0.16±0.12	0.28±0.31	0.023*
Atypical lymphocytes (%)	0.06±0.03	0.07±0.04	0.439
Large immature cells (%)	0.06±0.03	0.08±0.07	0.070
MPV (fl)	7.99±0.75	7.87±0.80	0.461
Plateletcrit (%)	6.77±42.51	0.21±0.04	0.317
PDW (%)	13.18±2.45	12.70±2.07	0.329

Data is presented as Mean±SD. *Statistically significant

Table 1 shows the mean values of hematological parameters. ESR was higher in smokers than non-smokers but the difference was not statistically significant. Total leucocyte count (TLC) was significantly higher ($p=0.019$) in smokers than in non-smokers (**Figure 1**). Counts of monocytes ($p=0.021$) and basophils ($p=0.023$) were significantly higher among smokers. RBC count, hematocrit and hemoglobin concentration was not significantly different between the two groups. Among RBC indices, only MCHC was significantly different between smokers and non-smokers. Platelet counts, plateletcrit and platelet indices showed no significant difference between smokers and non-smokers.

Also there was no significant linear relationship observed between various hematological parameters and cigarette smoking (in pack years). **Figure 2** shows the distribution of smokers into groups based on pack years.

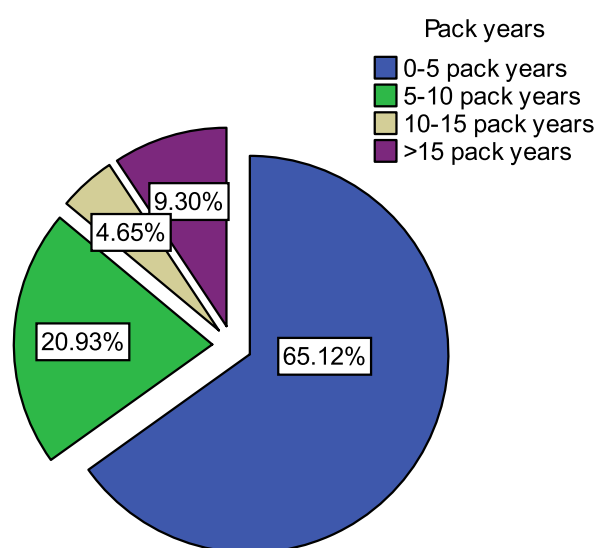


Figure 2: Distribution of smokers according to pack years

Discussion

The present study was done to document any change in total leucocyte count (TLC), differential leucocyte count (DLC), platelet count and erythrocyte sedimentation rate (ESR) among smokers and non-smokers and to evaluate any relationship between hematological values and duration/intensity of smoking among smokers. As the previous studies did not delve into the effect of chronicity of smoking and the intensity of smoking on the TLC, platelet count and ESR, this is the newer dimension in this study.

Total leucocyte count was significantly higher in smokers than non-smokers in this study which was consistent with the results of Oke et al [5], Shenwai et al [6], Asif et al [7], Ahmed [11], Nadia et al [12], Iqbal et al [13] and Fadiel et al [14]. It was conflicting with the study conducted by Khand et al [15]. Smoking has an irritant effect on the respiratory tree which can cause chronic inflammation. It causes increased release of inflammatory cytokines from the epithelial cells, these influence the activation and differentiation of leucocytes. This could be the reason of leucocytosis. Another mechanism proposed is that nicotine increases release of catecholamines which can increase the total leucocyte count [16]. Monocyte and basophil counts were significantly higher in smokers than non-smokers. High basophil counts indicate an ongoing inflammatory process in the body. This can increase the risk of atherosclerosis, cardiovascular disorders, chronic lung diseases and cause increased morbidity and mortality in smokers.

In this study, no significant correlation was obtained between smokers and non-smokers with respect to erythrocyte sedimentation rate. This was in contrast to the results of Nisa et al [2], Sharma et al [3], Islam et al [4] and Oke et al [5] who found that the erythrocyte sedimentation rate was increased in smokers when compared to non-smokers. This increase was independent of the number of cigarettes smoked per day [3,4].

There have been conflicting results in previous studies on the effect of smoking on platelet counts. In this study we found that platelet counts were similar amongst smokers and non-smokers. It is consistent with the results of Oke et al [5], Shenwai et al [6], Asif et al [7], Mohammed et al [17], Nadia et al [12] and Khand et al [15]. The results obtained were in contrast to some other studies. Ghahremanfard et al [10] and Ahmed [11] found that platelet counts were significantly increased in smokers when compared to non-smokers whereas Sandhya et al [18] concluded that the platelet counts of chronic smokers were significantly lower than that of non-smokers and

decreased significantly with increase in duration of smoking.

Limitations of the study

Sample size was relatively small for such type of case-control study. Since the hematological parameters are having wide range of normalcy, the differences in many parameters was not evident.

Conclusion

We found no statistically significant difference in erythrocyte sedimentation rate and platelet count of smokers and non-smokers. There was a significant increase in total leucocyte count, basophil count and monocyte count in smokers when compared to non-smokers which indicate inflammation in the body. As these changes are occurring in young healthy men with no other risk factors, smoking cessation can reduce the risk of developing cardiovascular and respiratory problems in future.

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Conflict of interest: None

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