

Erythrocyte Sedimentation Rate (ESR) in Male Adult Smokers

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Cigarette smoking is a common problem in Bangladesh and also a major public health problem associated with morbidity and mortality. The prevalence of cigarette smoking has peaked among the adults. Inflammation is currently considered to play a central role in carcinogenesis and chronic inflammation has been hypothesized to play a role in the pathogenesis of several cancers. Erythrocyte Sedimentation Rate (ESR) is a certain biomarker of inflammation. This study was done to find out the cross-sectional association of changes in ESR in relation to cigarette smoking and also with different intensity of cigarette smoking. The study population consisted of 105 male adult non-smokers and smokers, aged 20~40 years, of different socio-economic classes, 30 of them were apparently healthy non-smokers, taken as control group for the study, whereas the remaining 75 apparently healthy smokers, who were smoking one or more cigarette per day, regularly for at least last one year, considered as the experimental group of this study. Smokers were again divided according to the number of cigarettes they consume per day into three categories. Smoking habit, clinical history and examination performed by pre-tested questionnaire. Unpaired “t” test was performed to find significant statistical difference between non-smokers and different smokers groups. The ESR was significantly ($p < 0.01$) differ between smokers and non-smokers, but it did not significantly differ with intensity of smoking. The finding in this study suggests that cigarette smoking does changes the ESR which is a strong biomarker of chronic inflammation.

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Key words: Cigarette Smoking, ESR, Adult Male

Introduction

Tobacco is the second major cause of death in the world. It is currently responsible for the death of one in ten adult worldwide (about 5 million deaths each year). If current smoking patterns continue, it will cause some 10 million deaths each year

by 2020. Half the people that smoke today – that is about 650 million people – will eventually be killed by tobacco.¹ The Expert Committee observed that tobacco related diseases are on the rise in developing countries.²

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In 2003, the smoking prevalence in Bangladesh, among adults was 54.8% in males and 16.6% in females. The prevalence of smoking in age group 18-29 yrs, 30-39 yrs, 40-49 yrs is 36.3%, 64.2%, and 70.8%, respectively.³ [Fig. 1]

Researchers have demonstrated that certain biomarkers of cardiovascular disease risk factors (eg, hemoglobin, hematocrit, RBC count, WBC count, mean corpuscular volume (MCV), Mean corpuscular hemoglobin (MCH), lipoproteins concentrations, heart rate, respiratory symptoms) known to be affected by smoking are relatively stable over time when amount of smoking is maintained at a constant rate but are altered in response to changes in smoking behavior. This suggests that some of these measures are likely to be sensitive to changes in tobacco toxin exposure.⁴

Other investigators have also found that at all ages erythrocyte counts of men are higher in non-smokers than in smokers. Smokers have larger Erythrocytes than non-smokers in both sexes and at all ages.⁵ Inflammation is currently considered to play a central role in carcinogenesis, a hypothesis that has been addressed by assessing white blood cell count as a predictor of risk for cancer.⁶ Chronic inflammation has been hypothesized to play a role in the pathogenesis of several cancers.⁷ Some other researchers observed that cigarette smoking can change the ESR associated with hypersensitivity pneumonitis.⁸ Investigator demonstrated that Erythrocyte Sedimentation Rate as a main marker of inflammation, was a significant predictor of heart failure, independent of established risk factors for heart failure, and interim myocardial infarction after three decades of follow-up in a population-based sample of middle-aged men.⁹

Howell RW found that mean ESR about 10% higher in cigarette smokers than in non-smokers.¹⁰

In this study, we hypothesized that cigarette smoking can alter the ESR and which might be associated with intensity of smoking.

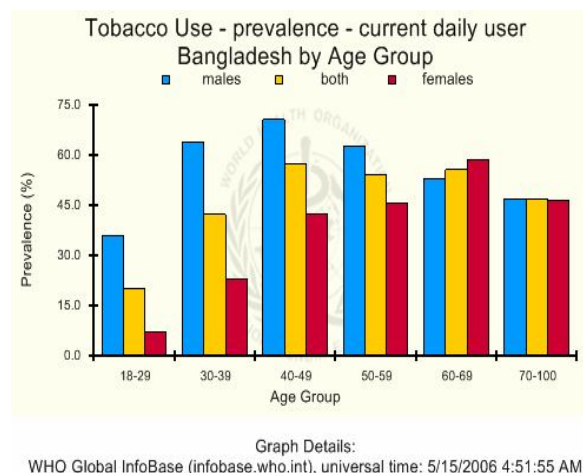


Figure 1. Tobacco use, prevalence in Bangladesh by age group. Reported from WHO Global InfoBase, 2006

Methods

This is a cross sectional comparative study, which was carried out in the department of Physiology of the Dhaka Medical College from July 2005 to June 2006. A total of 105 apparently healthy subjects age ranged from 20 to 40 years were selected, of whom 30 were non smokers (control – Group A) and 75 were smokers (experimental – Group B) who smoked for at least last one year. The smokers were again sub-grouped into 3 categories accordingly to the number of cigarette smoked per day (Group B₁: Consisted of 25 smokers consuming 1~9 cigarettes / day; Group B₂: Consisted of 25 smokers consuming 10~19 cigarettes/day; Group B₃: Consisted of 25 smokers consuming \geq 20 cigarettes/day). They were selected from

different areas of Dhaka city and different socio-economic classes of subjects were included in the study.

Subjects suffering from any acute or chronic respiratory illness, hypertension, diabetes mellitus, angina, endocrine, hepatic, allergic disorders, any infectious or debilitating illness etc. and subjects with history of recent hospitalization and surgery were also excluded. Persons taking the drugs such as antibiotic, steroids, thiazide diuretics, aspirin etc. or taking radiotherapy and subjects who drink alcohol were also excluded. Passive smokers were not included for control group.

All the subjects were explained about the aims and objectives of the study and the test procedure were briefed and written consent was taken before performing the test. A detailed history of smoking habit and health of each subject was obtained by using a pre-tested questioner and clinical examination sheet. Clinical examinations of these subjects were done on the first day of the visit. The subjects were advised overnight fasting and not to engage in unusual physical exercise and smoking before reporting on the next morning.

On the reporting day all the blood samples were collected at morning between 8.00 to 9.00 AM. With all aseptic (70% alcohol) precaution a venepuncture was done with a 3 ml disposable syringe in the antecubital fossa applying a tourniquet, which was released when the actual blood sampling began. 2 ml venous blood was drawn from each sample, in sitting position. After collection the sample of blood was mixed in a vial containing anticoagulant EDTA and transported to the Physiology laboratory of Dhaka Medical College in an air conditioned bus within one hour of collection. All the hematological analyses were done within 5 hours of sample collection.

With the collected samples total count of ESR was done by Westergren's method for each study subjects in the laboratory of Physiology of Dhaka Medical College and Hospital, Dhaka. After the collection of data these were checked, verified, edited for consistency to reduce error. All the results of laboratory investigations were analyzed by SPSS 12.0 programme and significance tests were done by unpaired student's "t" test.

Results

The mean (\pm SD) of ESR were 4.77 ± 3.66 and 9.69 ± 8.50 mm in 1st hour in group A (non-smokers) and group B (smokers), respectively. Again the means (\pm SD) of ESR were 11.1 ± 10.5 , 8.84 ± 5.79 , 9.12 ± 8.72 mm in 1st hour in B₁, B₂ and B₃ groups, respectively (Table I).

The difference of means (\pm SD) of ESR was significant ($p < .01$) between group A and B. The results were also significant between group A and smoker's group B₁ ($p < .01$), B₂ ($p < .01$), B₃ ($p < .05$). Again the differences of means of ESR among the smokers groups B₁ vs. B₂ ($p > .05$), B₂ vs. B₃ ($p > .05$), and B₁ vs. B₃ ($p < .05$) were not statistically significant (Table II, fig 2).

Table I: Mean and \pm SD of ESR (mm in 1st hour)

Groups	N	Minimum	Maximum	Mean	Std. Deviation
A	30	1	17	4.8	± 3.7
B	75	2.	40	9.7	± 8.5
B1	25	2	40	11.1	± 10.5
B2	25	2	22	8.8	± 5.8
B3	25	2	40	9.1	± 8.7

Table II: Statistical Analysis of ESR between different groups:

Groups	p	Significance	Remark
A vs. B	.0028	$P < .01$	Significant
A vs. B ₁	.0031	$P < .01$	Significant
A vs. B ₂	.0025	$P < .01$	Significant
A vs. B ₃	.016	$P < .05$	Significant
B ₂ vs. B ₁	.89	$P > .05$	Not
B ₁ vs. B ₃	.47	$P > .05$	Not
B ₁ vs. B ₂	.35	$P > .05$	Not

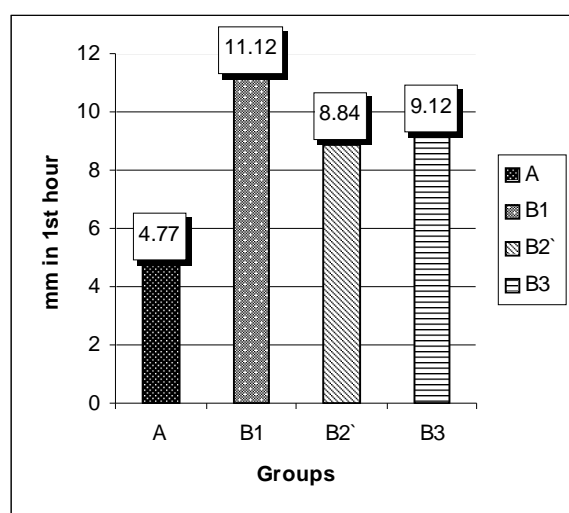


Figure 2. Mean (\pm SD) of ESR levels in non-smoker's and smoker's group

Discussion

The erythrocyte sedimentation rate values given in table I show a highly significant rise of ESR ($p < 0.01$) in smokers as compared to non-smokers indicating a strong association of markers of systemic inflammation with smoking. These findings are in agreement with Howell RW.¹⁰ But erythrocyte sedimentation rate was not significantly ($p > 0.05$) differ with intensity of cigarette smoking per day.

Cigarette smoke induces endothelial damage by producing free radicals such as nitric oxide and hydrogen peroxide. This oxidative stress promotes a systemic acute phase reaction thus increasing inflammatory cytokines, C-reactive protein, fibrinogen, blood cell count, whole blood viscosity and rouleaux formation. Eventually this leads to rise in ESR values.¹¹⁻¹³

So, the possible mechanism of increase ESR in the present study is chronic inflammatory response mediated by particulates of cigarette smoke.

In the present study we had some potential limitations which arise primarily due to lack of scope and resources, the tests could not done on the spot of blood collection, which would give better results. The ESR was done in traditional Westergren's method rather than using a auto analyzer. Blood cells were more or less hemolysied during transport. The sample included in the study only on basis of questionnaire and clinical examination.

Conclusion

From this study, it may be concluded that cigarette smoking increase erythrocyte sedimentation rate due to chronic inflammatory response mediate by particulates of cigarette smoke.

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Conflict of interest

None declared.

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