“EFFECT OF CIGARETTE SMOKING ON VARIOUS HEMATOLOGICAL PARAMETERS IN YOUNG MALE SMOKERS.”

DR. MRUNAL R. SHENWAI * 1, DR. MRS. N.V. AUNDHAKAR 2
1 Asst Prof. Dept. of Physiology, Smt. Kashibai Navale Medical College [SKNMC], Narhe – Ambegaon, Pune - 41.
2 Professor & Head, Dept. of Physiology, R.C.S.M. Govt. Medical College, Kolhapur.
Corresponding author: Dr Mrunal R. Shenwai; E-mail: drpmrunal@yahoo.com

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ABSTRACT:

Introduction: Coronary heart disease is one of the important causes of mortality in human beings. Cigarette smoking is one of the independent risk factors for coronary heart disease. Smoking increases mortality nearly five times between age group of 30-40 years who are likely to be free from other myocardial risk factors. The present work was undertaken to study the effect of cigarette smoking on various hematological parameters collectively in young & well educated population and compare the results with non-smokers.

Methods: The hematological parameters studied were Hemoglobin, Hematocrit, Blood Indices, R.B.C. count, Total leucocyte count and Differential leucocyte count. Total sample size was 70 which included a mixed population of male doctors & engineers [smokers (n=35) & non-smokers (n=35)] between the age group 26-40 yrs. To maintain accuracy all the parameters were studied using Sysmex K-4500 Autoanalyser. Differential counts were also done manually.

Observations: Our results showed a statistically significant increase in the total leucocyte count (P< 0.01 & Z >2) and lymphocyte count (P< 0.01 & Z >2). The change in rest of the parameters was statistically insignificant.

Conclusion: Increased Total leucocyte count and lymphocyte counts found in our subjects may be responsible for the chronic inflammatory state and high risk of coronary artery disease & neoplasia in smokers. Such young population which is otherwise free from the predisposing factors like obesity, hypertension, diabetes etc. can be encouraged to adopt healthier lifestyles and quit smoking so that future health related consequences can be avoided.

Key words: young smokers, Sysmex Autoanalyser, hematological parameters,

INTRODUCTION:

Heavy smoking is the commonest cause of ischemic heart disease and death in 30-40 yrs of age group who are likely to be free from other myocardial risk factors. Alterations in the hematological parameters may be responsible for the high risk of occlusive vascular disease in chronic smokers. Chronic smoking seems to cause an upward shift of hemoglobin dissociation curve, which may decrease the utility of hemoglobin levels in the detection of anaemia in smokers, suggesting that hemoglobin cutoff values should be adjusted for smokers to compensate for masking effect of smoking on detection of anemia. An association of total leucocyte count with cigarette smoking has been reported by several researchers but conflicting results have been obtained about the effect of smoking on differential leucocyte count. In most of the earlier studies blood parameters have been considered separately. In the present work an attempt has been made to study the effect of cigarette smoking on various hematological parameters collectively using automated counter for accuracy.
Our study focussed mainly on young, well educated (doctors & engineers) male population who are considered as role models for the society with an aim of creating awareness among them regarding their health and social responsibility. With this background in consideration the present work was planned.

MATERIAL & METHODS: Present study was conducted between two groups i.e. smokers and non-smokers. All were apparently healthy male subjects between the age group of 26-40 yrs. Total sample size was 70, out of which study group included smokers (n=35) and control group included non-smokers (n=35). The study group included male smokers who have been smoking filtered cigarettes minimum 5 (maximum 10) per day with duration \( \leq 10 \) yrs. Out of 35, 18 subjects were engineers belonging to a Pune based Software Company & 17 were doctors from BJMC & Sassoon General Hospital. The control group included 18 subjects (non-smokers) from the same software company & 17 doctors (non-smokers) from BJMC & Sassoon General Hospital. The socio-economic status, age, height, weight, daily activity and levels of stress were comparable between study group and the control group. Subjects suffering from coagulation disorders, diabetes, hypertension or any infection and those who are on any medication like aspirin or non-steroidal anti-inflammatory drugs (NSAIDs) were excluded from the study. All subjects were free from other habits like tobacco chewing and alcohol intake.

Written informed consent was taken from all the subjects before the procedure. Study protocol was approved by the ethical committee of the B.J. Medical College, Pune.

PROCEDURE: All the haematological parameters were studied using Sysmex K-4500 Autoanalyser. It is an 18-parameter, 3 part differential analyser which works on the principle of Aperture Impedance. Differential WBC count was also done manually by preparing a peripheral blood smear. All the blood samples were collected in the morning between 9.30-11.30 A.M. to avoid the effect of diurnal variation on blood counts. By a clean venepuncture of antecubital vein, 2 ml of blood was collected into a syringe with all aseptic precautions and immediately transferred to an EDTA (anticoagulant) bulb and automated counting was done using symsmex autoanalyser. A peripheral blood smear was done at the same time and differential WBC smear was also done manually using Leishmann’s stain.

STATISTICAL ANALYSIS OF THE DATA: The results are presented as mean ± S.D. All the results were statistically analysed by applying ‘Z’ test, as the sample size was more than 30. ‘Z’ value i.e. relative deviate and ‘P’ value were found out. Z'value of > 2 and ‘P’ value of < 0.001 has been taken as statistically significant.

RESULTS: Our study showed a statistically significant increase in the Total leucocyte count from a mean value of 7442/cu mm in non-smokers to about 8640/cu mm in smokers (P< 0.01 & Z >2) and an increase in the lymphocyte count amongst the differential leucocyte counts in smokers (P< 0.01 & Z >2) as compared to non-smokers. [Table I]. Table II shows changes in rest of the parameters which were statistically not significant.

DISCUSSION: Our results showed that Total leucocyte count is more in smokers as compared to non-smokers. Our findings corroborate with the findings of Tell G.S. & Grimm RH et al who reported increased TLC in smokers who have started smoking relatively recently. Marked leucocytosis has been reported by several other researchers even in subjects smoking less than 10 cigarettes per day. Moreover in our study casual blood samples were studied i.e. the subjects were not asked to smoke cigarettes or abstain from smoking prior to tests.
### TABLE I: EFFECT OF CIGARETTE SMOKING ON T.L.C. AND D.L.C.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Smokers</th>
<th>Non Smokers</th>
<th>Z Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 35 Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. <em>T.L.C.</em></td>
<td>8640 ± 844.9</td>
<td>7442.8±1169.6</td>
<td>4.9</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>(Cells/cu.mm)</td>
<td></td>
<td></td>
<td></td>
<td>H.S.</td>
</tr>
<tr>
<td>2. **D.L.C. %)</td>
<td>59.71 ± 5.64</td>
<td>61.97 ± 4.32</td>
<td>1.91</td>
<td>N.S.</td>
</tr>
<tr>
<td>Neutrophil</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>38.37 ± 5.05</td>
<td>32.43 ± 3.95</td>
<td>5.48</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Monocyte</td>
<td>1.84 ± 0.75</td>
<td>1.48 ± 0.6</td>
<td>0.86</td>
<td>N.S.</td>
</tr>
<tr>
<td>Eosinophil</td>
<td>1.66 ± 1.04</td>
<td>1.74 ± 0.87</td>
<td>0.35</td>
<td>N.S.</td>
</tr>
<tr>
<td>Basophil</td>
<td>0.2 ± 0.4</td>
<td>0.2 ± 0.4</td>
<td>0</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

When P < 0.001 and Z value is > 2, the result is statistically considered as highly significant (H.S.). N.S. - Not Significant.

Thus the study depicts that there is a persistent effect of smoking on TLC. Chronic tissue damage may be a possible mechanism for the increased total leucocyte count smokers. Smoking has an irritant effect on the respiratory tree with resultant chronic inflammation. Prolonged smoking impairs ciliary movements, causes hypertrophy and hyperplasia of mucus secreting glands, hyper responsiveness of the airways and causes bronchiolar inflammation. Airway epithelium is regarded as a physical barrier which prevents the entry of inhaled noxious particles into the submucosa. Exposure to smoke causes increased release of inflammatory cytokines from the epithelial cells. All of them can influence the growth, differentiation and activation of leucocytes. This possibly explains the leucocytosis in smokers. Another mechanism put forward by some workers is that nicotine increases release of catecholamines which can increase the total leucocyte count. Hemoconcentration attributed to cigarette smoking can also be considered as a possible explanation for the elevation of total leucocyte count.
TABLE II: OTHER PARAMETERS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Smokers Mean± S.D.</th>
<th>Non-Smokers Mean± S.D.</th>
<th>Z value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.B.C.count (millions/cu.mm)</td>
<td>5.24 ± 0.45</td>
<td>5.08 ± 0.46</td>
<td>1.47</td>
<td>P &gt; 0.005 N.S.</td>
</tr>
<tr>
<td>Hb concen. (g/dl)</td>
<td>14.81 ± 1.06</td>
<td>14.44 ± 0.81</td>
<td>1.64</td>
<td>N.S.</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>41.74 ± 2.3</td>
<td>40.6 ± 2.67</td>
<td>1.91</td>
<td>N.S.</td>
</tr>
<tr>
<td>M.C.V. (fl)</td>
<td>79.93 ± 4.82</td>
<td>80.73 ± 5.27</td>
<td>0.67</td>
<td>N.S.</td>
</tr>
<tr>
<td>M.C.H. (pg)</td>
<td>29.12 1.89</td>
<td>28.7 ± 2.22</td>
<td>0.85</td>
<td>N.S.</td>
</tr>
<tr>
<td>M.C.H.C. (g/dl)</td>
<td>35.56 ± 2.98</td>
<td>35.68 ± 2.15</td>
<td>0.19</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

(N.S. = statistically not significant)

Now a days there is increasing evidence that apart from the known risk factors like cigarette smoking, diabetes & hypertension, inflammation also plays an important role in the progression of coronary heart disease. Elevated WBC counts as observed in smokers along with high C reactive proteins are associated with increased incidence as well as mortality from coronary heart disease.\(^{12-15}\)

Our study also aimed at Differential leucocyte counts because the association of cigarette smoking with total leucocyte count has been established by many but its effect on the differential leucocyte count is a matter of debate. According to some researchers effect of smoking on differential count is not uniform and is influenced by the current smoking behaviour.
Some studies have shown that neutrophil count rises and lymphocyte count shows a decrease, while few studies have shown that both these counts are increased. Hence in our study, the subjects chosen were all active smokers and casual blood samples were taken i.e. the subjects was not asked to smoke cigarettes or abstain from smoking prior to tests. Our findings reveal that the lymphocyte count increases from a mean value of 32.4% in non-smokers to 38.3% in smokers, which is found to be statistically significant. (Table I). Amongst other parameters, neutrophil count showed a slight fall in smokers than non-smokers but the difference is not statistically significant. No significant change is observed in eosinophil, basophil and monocyte counts.

The lymphocytosis can be attributed to chronic tissue damage & inflammation produced by toxic smoke products. This corresponds with the findings of Silvermann NA et al that leucocytosis in smokers is mainly attributable to an increased lymphocyte count and that too of the ‘T’ lymphocytes. Similar findings have been reported by some other researchers also. Alteration in the T lymphocytes may explain the increased risk of infections and neoplasia in smokers. The result obtained in case of rest of all parameters [RBC count, hemoglobin, hematocrit, and blood indices. (Table II) were statistically not significant.

According to Whithead TD et al, haemoglobin levels and PCV increase significantly in those smoking more than 10 cigarettes per day, but the effect on WBC count is seen even in subjects smoking less than 10 cigarettes per day. Smoking is also considered as a major cause of polycythemia and elevated hematocrit levels.

In the present study, though the mean values for all these parameters in smokers are found to be higher than non-smokers, the difference is not statistically significant. This can be possibly explained by the younger age group of the subjects with relatively less duration of smoking. In future we would like to extend our study in smokers of higher age group with duration of smoking more than 15 years.

**CONCLUSION:** Increased leucocyte count along with raised C reactive protein levels is shown to be associated with increased mortality rate in patients with ischemic heart disease. So, these can be considered as important prognostic markers in young smokers with ischemic heart disease. Alteration of lymphocyte count and an imbalance between T cell subsets contributes to the increased risk of infection and neoplasia in smokers. Hence smoking is considered as one of the major avoidable risk factors for cardiovascular diseases and death. Encouraging results have been observed in smokers who show a rapid return of many hematological abnormalities towards normal on abstention from smoking. Also, the risk of adverse effects starts to decline quite rapidly after cessation of smoking. This fact is of immense importance for the young smokers who are otherwise free from other predisposing factors like obesity, hypertension, diabetes etc. So they have a bright future provided they exercise their will to stop smoking.

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**REFERENCES:**


10. Eric G. Honig, Roland H. Ingram Jr. Chronic bronchitis, emphysema and airway obstruction. In- Harrison’s Principles of Internal Medicine, 14th edition, USA:


