ABSTRACT

Objective:
Smoking is one of the major public health problems especially in young adults of developing countries. Smoking causes structural abnormalities in the airway due to inflammations leading to increased airway resistance which is associated with high differential count. The aim of the present study was to assess the association between smoking and changes in peripheral differential leukocyte count (DLC).

Methods:
Our study was conducted on 100 male student volunteers (both UG & PG) under the age group of 18-30 years from a tertiary care teaching institution, Tamilnadu. Among the 100 students, 50 were smokers (study group) and 50 were non-smokers (control group). Blood smears were prepared from both groups by spread technique using Leishman stain and the DLC results were compared and analysed.

Results:
The results of our study shows, in DLC, the neutrophil count showed a definite increase for smokers (70.1 ± 5.2) when compared to non-smokers (59.7 ± 4.4) and the ‘p’ value for neutrophil count (<0.001) was of high statistical significance.

Conclusion:
It was observed that the neutrophil count was increased in smokers indicating active airway inflammation.

INTRODUCTION:
The air we breathe is rarely a simple mixture of oxygen, nitrogen and water vapour. It also contains varied collection of other noxious gases and particles. In addition, some people further contaminate the air with cigarette smoke.

In the 16th century, king James I described the habit of smoking as “a custom loathsome to the eye, hateful to the nose, harmful to the brain and dangerous to the lungs”. However, firm evidence to support his last conclusion was delayed for about 350 years.

One third of the global population aged fifteen years and over (approx. 1.1 billion people) are current smokers. Most of them are living in developing countries (800 million) and most of them are males (700 million) [1]. It has been estimated that by 2025, 75% of early death in developing countries will be due to smoking related illness [2].

Cigarette smoke contains (per cigarette) various constituents like carbon monoxide (12-20ml), nicotine (0.05-2.5mg), tar (0.5-35mg), potential free radical, nitric oxide and traces of concentration of nitrogen di oxide, hydrocyanic acid, cyanogens, aldehydes, ketones, polynuclear aromatic hydrocarbons & nitrous amine [3,4]. These compounds cause direct and cell mediated oxidative damage to the lungs by alveolar macrophages and neutrophils [5,6].

Also, Smoking has been implicated as a potential cause of chronic idiopathic neutrophilia in smokers. However, it gets reverted back to normal after cessation of smoking [7].
The peripheral differential leukocyte count was performed by manual method (spread technique): Under proper aseptic precautions, a drop of capillary blood was collected by finger prick to prepare the blood smear. A drop of blood was placed on a clean, dry, grease free glass slide and the blood film was made with rapid movement using a smooth glass spreader. 5-8 drops of Leishman stain was added to the dried smear. After 2 minutes (fixation time), double the amount of distilled water was added and the smear felt undisturbed for the next 7 minutes (staining time). The smear was washed, dried and focused under oil immersion objective. 2-3 such blood smears were prepared from the same subject. The first 200 cells were counted by 2 independent observers to ensure adequate precision [8]

**RESULT:**

The effect of cigarette smoking was assessed on the peripheral differential leukocyte count in healthy male smokers and the data was compared with non-smokers.

**Table 1: Differential leukocyte count in smokers and non-smokers.**

<table>
<thead>
<tr>
<th>LEUKOCYTE subsets</th>
<th>STUDY GROUP</th>
<th>CONTROL GROUP</th>
<th>WILCOXON SIGNED RANK TEST-‘p’ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean±SD</td>
<td>Mean±SD</td>
<td></td>
</tr>
<tr>
<td>Neutrophils</td>
<td>70.1 ± 5.2</td>
<td>59.7 ± 4.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>2.1 ± 0.8</td>
<td>2.7 ± 1.2</td>
<td>0.003</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.4 ± 0.6</td>
<td>0.5 ± 0.5</td>
<td>0.555</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>23.7 ± 3.8</td>
<td>32 ± 4.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Monocytes</td>
<td>3.8 ± 1.8</td>
<td>5.1 ± 1.5</td>
<td>0.001</td>
</tr>
</tbody>
</table>

The neutrophil count for the study group and control group was 70.1 ± 5.2 and 59.7 ± 4.4 respectively. The ‘p’ value was <0.001 which was of high statistical significance. This proves that neutrophil shows a definite increase in smokers when compared to non-smokers.

The other leukocytes Like Eosinophil, Basophil, Lymphocyte and Monocyte were decreased in proportionate to the increase in neutrophil count in smokers but still they were in the normal range.

The mean and SD of Eosinophil, Basophil, Lymphocytes and Monocyte in smokers were 2.1 ± 0.8, 0.4 ± 0.6, 23.7 ± 3.8, 3.8 ± 1.8 respectively. For non-smokers it was 2.7 ± 1.2, 0.5 ± 0.5, 32 ± 4.6, 5.1 ± 1.5 respectively. [Table 1]

The data was analysed statistically using Wilcoxon signed rank test and the results obtained. The ‘p’ value of <0.05 was accepted a statistically significant.

**DISCUSSION:**

Cigarette smoking increases the total peripheral leukocyte count but its effect on the differential leukocyte count in the peripheral blood is largely unexplored.

The present study was done to study the effect of smoking on peripheral differential leukocyte count. In our study, we observed that of all the different leukocyte subset, the neutrophil count was increased in smokers when compared to non-smokers. The Eosinophil, Basophil, Lymphocyte and Monocyte were within the normal range.

Similar increase in Neutrophil count was noticed in a study conducted by Schwartz J and Weiss ST on smokers between the age group of 30-74 years [9] and also by Merlin et al on healthy smoking pregnant women [10].

In 1998, a study done by Jensen et al found that there was increase in the total leukocyte count and in DLC, increase in the number of neutrophil, lymphocyte and basophil in smokers when compared to non-smokers [11]. In an Indian study by Kumar et al, the neutrophil and the eosinophil count were increased [12]. But in our study only the neutrophil count was increased.

To combat the inflammatory reaction caused by the cigarette smoke in the bronchial airway, the neutrophils produces proteolytic enzymes extracellularly which on excess damages the normal resident cell [13]. In our future study, we have planned to assess these enzyme levels in smokers which will show the definite relationship between the neutrophils and cigarette smoke.

**CONCLUSION:**

In the present study, the Neutrophil count was significantly increased in smokers when compared to non-smokers indicating that there was an inflammatory reaction caused by the cigarette smoke.

**ACKNOWLEDGEMENT:**

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**REFERENCES**