EFFECT OF SMOKING ON HEMATOLOGICAL PARAMETERS IN HUMAN BEINGS

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Abstract: Fresh peripheral blood samples from healthy adult non-smokers and smokers (males) were collected and analysed for RBC count, hemoglobin (Hb) content, packed cell volume (PCV), MCV, MCH and MCHC, total and differential leucocytes (WBC) counts and total platelets count. The results revealed significant reduction in RBC count, Hb content and PCV in smokers. Study shows that total WBC count, neutrophils, eosinophils, basophils and monocytes were significantly increased in smokers; however lymphocytes count was significantly decreased. Platelets counts were significantly higher in smokers than that of non-smokers. It is concluded that smoking alters hematological parameters that is injurious to health.

Key words: Smoking, Hematology

INTRODUCTION

Cigarette smoking is one of the major causes of cancer and cardiovascular diseases leading to millions of premature deaths each year all over the world. Scientists have identified about 4,000 different substances in tobacco all of which have certain degree of toxic effects. At least 43 of them are known carcinogens. Cigarette smoke contains a variety of other compounds including oxidants and free radicals [1-3] that are capable of initiating or promoting oxidative damage [4-7] leading to various degenerative pulmonary and cardiovascular diseases as well as cancer [8-11]. Although the effect of smoking on haematological parameters has been studied previously, but the literature is limited and controversial. Moore [12] and Moore and Pearson [13] reported polycythemia in cigarette smokers. The effect of carbon monoxide on haemoglobin, its binding and dissociation with oxygen and occurrence of hypoxia have been reported by many workers [14-16]. A number of investigators found the mean WBC and neutrophils counts were significantly higher in habitual smokers and that the range was strongly skewed upward [17-21]. Smoking is also reported to cause eosinophilia [22]. Exposure to cigarette tar inhibits ribonucleotide reduction and blocks lymphocyte proliferation [23]. Smoking increases the activation of platelets by 100 times, which can lead to a significant increase in blood clots. The main aim of this study is to screen long-term effect of smoking on haematological parameters, particularly on the basis of frequency and duration of smoking.

MATERIALS AND METHODS

Fresh peripheral blood samples from healthy adult male non-smokers and chronic smokers (65 samples each) were drawn in sterile anticoagulant added syringe. Each sample was mixed gently to avoid clotting and used for various haematological parameters within 1 hour of collection. Total RBC, WBC and platelets were counted by hematocytometric method using Neubauer’s chamber [24]. Haemoglobin content was measured by Sahli’s method. Packed cell volume (PCV) was measured using Wintrobe’s hematocrit tubes. Mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) was calculated. For differential count of WBC, blood films were stained by Leishman’s stain [24]. For each parameter at least 10 replicates were carried out. Students’ t-test was used for statistical analysis of the data; p < 0.05 was considered as significant. The data obtained on smokers
RESULTS

RBC and its related parameters: Smoking resulted in a significant reduction in RBC count, Hb content and PCV, however MCHC reduction was insignificant. The MCV and MCH were insignificantly increased in smokers as compared to non-smokers (Table 1). A comparison of RBC count and MCHC in short-term and long-term smokers also revealed insignificant changes. Likewise, Hb content, PCV, MCV and MCH were insignificantly increased in long-term smokers (Table 1). In frequent smokers the RBC count was insignificantly reduced, but the Hb content, PCV, MCV, MCH and MCHC were higher (Table 1).

Total and differential WBC count: Results revealed a significant increase in total WBC counts, neutrophils, was also analysed based on the duration (short-term vs. long-term) and frequency (less frequent vs. frequent) of smoking.

Table 1: Erythrocytes (RBC), haemoglobin (Hb), packed cell volume and haematological indices in the blood of smokers and non-smokers.

<table>
<thead>
<tr>
<th></th>
<th>RBC X 10^6/mm³</th>
<th>Hb gm/dl</th>
<th>PCV (%)</th>
<th>MCV (μm³)</th>
<th>MCH (pg)</th>
<th>MCHC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-Smokers</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>4.7±0.05</td>
<td>13.8±0.22</td>
<td>44.6±1.86</td>
<td>78.1±2.89</td>
<td>25.5±7.20</td>
<td>32.1±1.69</td>
</tr>
<tr>
<td><strong>Smokers</strong></td>
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<tr>
<td>Short-term smokers</td>
<td>5.42±0.15 (19)</td>
<td>13.83±0.22 (28)</td>
<td>44.61±1.86 (19)</td>
<td>78.13±2.89 (19)</td>
<td>25.57±7.20 (19)</td>
<td>32.11±1.69 (19)</td>
</tr>
<tr>
<td>Long-term smokers</td>
<td>4.7±0.05</td>
<td>13.85±0.22</td>
<td>44.6±1.86</td>
<td>78.1±2.89</td>
<td>25.5±7.20</td>
<td>32.1±1.69</td>
</tr>
</tbody>
</table>

Values are mean ± SEM; Values in parentheses indicate number of replicates. Significant at: as compared with non-smokers *p<0.02, **p<0.01, ***p<0.001. Abbreviations used: RBC =Red blood cell; Hb =Haemoglobin; PCV=Packed cell volume; MCV=Mean corpuscular volume; MCH =Mean corpuscular haemoglobin; MCHC=Mean corpuscular haemoglobin concentration.

Table 2: Showing total and differential WBC counts in the blood of non-smokers and smokers.

<table>
<thead>
<tr>
<th></th>
<th>WBC (x10³cells/ml)</th>
<th>Neutrophils (%)</th>
<th>Eosinophils (%)</th>
<th>Basophils (%)</th>
<th>Lymphocytes (%)</th>
<th>Monocytes (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-smokers</strong></td>
<td>6.39±0.12 (29)</td>
<td>62.10±0.87 (20)</td>
<td>1.57±0.16 (28)</td>
<td>0.25±0.03 (28)</td>
<td>35.75±0.78 (28)</td>
<td>1.64±0.12 (28)</td>
</tr>
<tr>
<td><strong>Smokers</strong></td>
<td>8.36±0.30*** (65)</td>
<td>65.71±1.10*** (63)</td>
<td>2.31±0.29*** (46)</td>
<td>0.78±0.07*** (31)</td>
<td>25.65±0.98*** (60)</td>
<td>5.80±0.51*** (61)</td>
</tr>
</tbody>
</table>

Values are mean±SEM. Values in parentheses indicate number of replicates. Significant at: as compared with non-smokers *p<0.02, **p<0.01, ***p<0.001. As compared with short-term smokers ***p<0.001.

Table 3: Showing platelets count in the blood of smokers and non-smokers.

<table>
<thead>
<tr>
<th></th>
<th>Platelets (millions/mm³)</th>
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<tbody>
<tr>
<td><strong>Non-smokers</strong></td>
<td>234.9±16.09 (19)</td>
</tr>
<tr>
<td><strong>Smokers(Total)</strong></td>
<td>309.77±12.66*** (61)</td>
</tr>
</tbody>
</table>

Values are mean ± S.E.M. Values in parentheses indicate number of replicates. Significant at: As compared with non-smokers *p<0.02, **p<0.01, ***p<0.001. As compared with short-term smokers ***p<0.001.

cosinophils, basophils and monocytes in smokers. However, lymphocytes count was significantly decreased in smokers than that of non-smokers. Amongst smokers duration and frequency-dependent changes were also observed. The monocytes counts were higher in long-term smokers but the basophils count was reduced. The changes were significant in both the
cases. Total WBC count, neutrophils, eosinophils and lymphocytes content were slightly increased in long-term smokers (Table 2). Frequency wise too, the changes were insignificant. While total WBC count and neutrophils were reduced, the eosinophils, basophils, lymphocytes and monocytes counts were higher in frequent smokers but the changes were insignificant (Table 2).

**Platelets count:** Table 3 shows the changes in platelets counts in smokers and non-smokers. Results revealed a significant increase in platelets counts in smokers. Duration and frequency-dependent changes were also observed amongst smokers. As for example the increases in platelets counts in long-term smokers and decrease of platelets counts in frequent smokers were evident but the changes were insignificant.

**DISCUSSION**

The present study clearly indicates significant reduction in RBC count and haemoglobin content among the smokers. Exact reasons of decreased RBC count, Hb concentration and PCV are not yet clearly understood, however it could be due to inhibition of haematopoiesis, increased rate of destruction of RBC or a combination of both. Jaffe and Kangler [25] have reported that cigarette smoke contains 2-20 mg of carbon monoxide. Exposure to a gas mixture containing about 0.1% carbon monoxide in air would result in 50% carboxyhaemoglobin at equilibrium at the sea level. Non-smoking human adults normally do not have more than 1% of their total circulating haemoglobin in the form of carboxyhaemoglobin but heavy smokers may show values as high as 5-10% saturation [26]. Thus oxygen carrying capacity of blood gets significantly reduced. Peripheral vasodilatation may develop in response to a slowly developing hypoxia, necessitating an increase in cardiac output. Tachycardia and electrocardiographic (ECG) changes suggestive of hypoxia may be observed at 30% or greater carboxyhaemoglobin saturation [27,28]. Non-significant increase in haemoglobin content and MCH in long-term and frequent smokers could be a response to rising carbon monoxide concentration.

The literature reveals number of factors, other than simple hypoxia, that contribute to carbon monoxide poisoning [29]. Differential reflectance spectroscopy allows direct measurements of brain energy metabolism in intact animals. Such studies show the inhibitory effect of carbon monoxide on cytochrome C oxidase that contributes to the intoxication syndrome. Impairment of energy metabolism can continue in smokers despite the elimination of carboxyhaemoglobin from the blood which may be an important factor for their sluggishness [16].

The results revealed a decrease in PCV, which could be due to reduction in RBC count. Thus the patient may have the possibility of developing anaemia (deficiency in the number of RBC or their haemoglobin content or both). The clinical feature of such persons may show easy fatigue, breathlessness, exertion, giddiness and loss of appetite.

Significant increases in total WBC count, as revealed in present study, could be due to increase in neutrophils, basophils, eosinophils and monocytes. An enormous amount of data gleaned from multiphase screening of thousands of normal people denotes that the mean WBC and neutrophil count are significantly higher in habitual cigarette smokers [12,17-19].

Many of the toxicants present in smoke like pyridine, benzene derivatives etc. may result in leukocytosis and neutrophilia [30]. Variation in the blood concentration of each leukocyte type can result from changes in (i) the flow of cells into the blood, (ii) the egress of cells from the blood, (iii) the distribution of cells within the vascular system or (iv) combinations of these factors [31,32].

The elevation of WBC counts is proportional to the number of cigarettes smoked daily, and the duration of which smoke is inhaled. Cigarette smoking is most important and prevalent perturbed of resting basal WBC count. The mean WBC count in adult non-smokers is 6400 cells/µl and it is increased by over 1000 cells/µl per each pack of cigarettes smoked daily [33].

The data presented in table 2 shows that there is a significant change in lymphocytes, eosinophils and monocyte count. While lymphocytes count are decreased significantly, the eosinophils and monocytes count are significantly higher. Smoking is also a reported cause of eosinophilia [22].

McCue et al. [34] and Ouyang et al. [35] have demonstrated that a component of cigarette tar is an extremely potent inhibitor of cytokine (IL-1β, TNFα, IL-2 and IFNγ) production. A 10% solution of cigarette tar derived from a single low-tar cigarette completely blocks production of these cytokines by human peripheral blood mononuclear cells. Thus, cigarette smoke blocks both cytokine production and lymphocyte proliferation and thus acts like a combination of the best
clinical immnosuppressive agents. Exposure to cigarette tar inhibits ribonucleotide reductive and blocks lymphocyte proliferation [23].

The present results also show a rise in platelets count in smokers. Smoking affects many of the steps involved in the clotting process. In particular, smoking just two cigarettes increases the activation of platelets by 100 times. Activated platelets can lead to a significant increase in blood clots. Platelet activation appears to be caused by elements in the smoke unrelated to nicotine. Nicotine patches (where nicotine is absorbed in the skin and delivered to the blood stream) do not cause these changes.

Smokers are more likely to develop a cerebral thrombosis (stroke). About 1% of all stroke deaths are estimated to be smoking related, with the overall relative risk of stroke in smokers being about 1.5 times to that of non-smokers [36]. Heavy smokers (consuming 20 or more cigarettes a day) have 2-4 times greater risk of stroke than non-smokers [37]. Bonita et al. [38] showed that both active and passive smoking increase the risk of stroke in men and women, and the present results are the clear indication of the same.

REFERENCES