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Prothrombin Time (Pt) and Partial Thromboplastin Time with Kaolin (Pttk) of Cigarette Smokers in Calabar, Cross-river State, Nigeria

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ABSTRACT

Smoking injures blood vessel walls by damaging endothelial cells, thus increasing permeability to lipids and other blood components. It is a major health problem that results in significant morbidity and mortality. Scanty information on haemostatic studies on smokers was observed in Nigerian population especially in this locality, hence the reason for the study. The objective is to assess the prothrombin time and partial thromboplastin time test values of cigarette smokers and to observe if there is any significant effect of cigarette smoking on these parameters. Prothrombin time (PT) and partial thromboplastin time with kaolin (PTTk) was investigated in 240 volunteers in Calabar, Cross-River state of Nigeria. The comprised 120 cigarette smokers and 120 age and sex matched non-cigarette smokers. Citrated blood samples were collected and their plasma analyzed using Standard methods within an hour of sample collection. The results show that mean prothrombin time and partial thromboplastin time values of smokers were significantly lower (P<0.001) when compared with the non-smokers. Duration of smoking significantly lowered the mean values of partial thromboplastin time test and prothrombin time test of smokers (P<0.001, P<0.05) respectively. PT and PTT (K) of smokers did not vary significantly (P>0.05) based on sex and age. It was concluded that cigarette intake as well as duration of intake, reduces PT and PTT (K) values thus may predispose cigarette smokers to rheologic abnormalities.

Key words: cigarette-smoking, Prothrombin time, partial thromboplastin time.

Introduction

Tobacco use is the single most preventable cause of death worldwide, responsible for the deaths of approximately half of all long-term users[12]. An estimated five million people died from tobacco use in 2000, representing about 12% of adult deaths[7]. Of these, about 30% (1.42 million deaths) resulted from cancer alone[8]. Among different types of tobacco use, cigarettes account for the largest share of manufactured tobacco products in the world. Smoke from the average cigarette contains around 4,000 chemicals, some of which are highly toxic and at least 60 cause cancer[1]. In 1964, the United States Surgeon General’s report publicized epidemiological research conclusively showing that cigarette smoking was a definite cause of cancers of the lung and larynx in men and chronic bronchitis in both men and women[13]. Since then, the number of diseases associated with smoking has continued to grow and because of this, the American Cancer Society and other organizations initiated studies comparing deaths among smokers and nonsmokers over a period of several years. All such studies found increased mortality among smokers, both from cancer and other causes[9,10,1].

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Studies on effects of cigarette smoking on red blood cells and coagulation factors have been reported. The studies showed that there is increased blood viscosity, elevated red cell density, increased aggregation of red cells, elevated haematocrit, increased synthesis of fibrinogen, hyper-coagulable state and reduction of albumin/fibrinogen ratio among smokers[4,6]. Chronic smoking has been reported to be associated with increased serum cholesterol, reduced high density lipoprotein, and other lipid effects that contribute to atherosclerosis[2]. Information observed from literature were from Countries outside Nigeria. It is the aim of this study to determine the PT and PTT (k) values of cigarette smokers in Nigerian population and compare these parameters with that obtained from nonsmokers.

Material and Methods

Blood samples were collected from consenting 240 subjects. They were made up of students of university of Calabar, blood donors and volunteers living in the city of Calabar. One hundred and twenty were smokers of which fourteen were females and 106 were males. The remaining 120 were nonsmokers (Control) who were 62 males and 58 females. The smokers and nonsmokers were age and sex matched.

Prothrombin time and partial thromboplastin test kits (Plasmascan and Hemosean respectively) from Qumica Clinka Applicada Spain were used in the study. Five milliliters of blood were collected by venupuncture from each volunteer. 4.5ml was dispensed into labeled plastic bottle containing 0.5ml of trisodium citrate and properly mixed. Within 1hr of collection; the samples were spun at 3000rpm for 10 minutes to obtain platelet poor plasma required for the analysis. The tests were carried out immediately, as instructed by the manufactures where it is not possible; the samples were stored at -20c till when required for analysis.

Results

The mean PT and PTT (k) of smokers were significantly (P<0.01,P<0.001) reduced when compared with non-smokers (Table 1). The PT and PTT (k) of smokers who have smoked for over 11 years and more were significantly (P<0.05, P<0.001) reduced when compared with smokers of 10 years and below (table 2). The relationship between four age groups viz (10-20 years), (21-30 years), (31-40 years) and (41-50 years) and PT and PTT (k) parameters showed no significant difference (P>0.05) (Table 3). Furthermore the PT and PTT (k) of smokers did not vary significantly base on their sex (Table 4). The data were analyzed by student’s “t” test and ANOVA. Unless otherwise stated the data were expressed as means +/- standard deviation. P<0.05 was considered significant in all statistical comparisons.

Discussion and conclusion

The study shows that PT and PTTK of smokers were significantly lower (p<0.01, 0.001) when compared with control subjects though the values were within the reference range of the manufactures as well as reference value of Dacie and Lewis[5]. Duration of smoking was grouped into two (1-10years) and (11-20years) and the PT and PTT (k) values compared. The PT and PTT (k) of those who have smoked for 11 years and more were significantly (P<0.05, 0.001) lower when compared with those who have smoked for 10 years and less. The hastened time observed in smokers of 11 years and more may be attributed to increased plasma viscosity and increased synthesis of fibrinogen in plasma as reported earlier by Cook and Ubben[4], and Eber, and Schumacher,[6]. The finding agrees with Pilgeram and Pickards report of 1968 that prolonged cigarette in-take causes an increased amount of fibrinogen in plasma which could lead to hastened bleeding arrest.

Prothrombin time and partial thromboplastin time tests of smokers and non-smokers did not significantly vary based on their age and sex (tables 3 and 4). While the highest number of people smoking was within the age range of 20-30years, fewer number of smokers participated in the study after the age of 30years (table 3). This agrees with CDC’s third national report on human exposure to environmental chemicals[3]. The implication of this finding is that cigarette smoking is still predominant among youths and in view of this, activities such as public education and enlightenment programme on the risk of cigarette smoking by the Clinicians and non-governmental organizations (NGOs) should be targeted more on youths to relinquish cigarette smoking.

Paucity of information on effects of cigarette smoking on coagulation factors particularly in developing countries such as Nigeria was observed from review of literature. This study has established base line values of PT and PTT (k) in smokers and in control subjects in this locality. It has also shown that there is significant reduction in PT and PTT (k) values in smokers. Again prolong intake of cigarette (up to 11years and more) further lowers the PT and PTT (k) values. In conclusion, the study observed hastened PT and PTTK values in smokers. Therefore it is the duty of everyone particularly clinicians and vascular surgeons to educating people and patients who smoke to quit smoking.
Table 1: Prothrombin time and partial thromboplastin time tests in 120 smokers compared with non-smokers

<table>
<thead>
<tr>
<th>Status</th>
<th>Parameters</th>
<th>Smokers (n=120)</th>
<th>Non-smokers (n=120)</th>
<th>critical t</th>
<th>calculated t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT (s)</td>
<td>11.4 ±1.7</td>
<td>12.0 ±2.0</td>
<td>2.52</td>
<td>2.5</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>PTT (s)</td>
<td>24.6 ± 4.3</td>
<td>29.5 ± 9.2</td>
<td>3.24</td>
<td>5.1</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

Results expressed as mean ± SD

PT = Prothrombin time (Reference value= 10-15 (s))
PTT = partial thromboplastin time with kaolin tests (Reference value =23-45(s))
s =seconds
n =number of subjects studied

Table 2: Effect of duration of smoking on PT and PTT (k) of smokers

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Duration (years)</th>
<th>Smokers (n=100)</th>
<th>Non-smokers (n=20)</th>
<th>critical t</th>
<th>calculated t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT (s)</td>
<td>11.5 ±1.7</td>
<td>10.8 ±1.4</td>
<td>1.96</td>
<td>2.0</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>PTT (s)</td>
<td>28.9± 3.7</td>
<td>23.9± 4.2</td>
<td>3.29</td>
<td>5.0</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

Results expressed as mean ± SD

PT = Prothrombin time
PTT = partial thromboplastin time with kaolin tests
s =seconds
n =number of subjects studied

Table 3: Age relationship of PT and PTTK of smokers and Non-smokers

<table>
<thead>
<tr>
<th>Age range</th>
<th>PT (s)</th>
<th>PTT (s)</th>
<th>PT (s)</th>
<th>PTT (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>years</td>
<td>Smokers</td>
<td>Non smoker</td>
<td>Smokers</td>
<td>Non smoker</td>
</tr>
<tr>
<td>10-20</td>
<td>10.3 ±1.4</td>
<td>10.5 ±2.1</td>
<td>22.2 ±2.8</td>
<td>31.5 ±2.1</td>
</tr>
<tr>
<td></td>
<td>(n=112)</td>
<td>(n=41)</td>
<td>(n=112)</td>
<td>(n=41)</td>
</tr>
<tr>
<td>21-30</td>
<td>11.6 ±1.5</td>
<td>11.9 ±2.4</td>
<td>24.3 ±4.8</td>
<td>30.7 ±4.7</td>
</tr>
<tr>
<td></td>
<td>(n=94)</td>
<td>(n=120)</td>
<td>(n=94)</td>
<td>(n=120)</td>
</tr>
<tr>
<td>31-40</td>
<td>11.4 ±2.1</td>
<td>12.7 ±2.8</td>
<td>28.0 ±4.2</td>
<td>29.3 ±3.8</td>
</tr>
<tr>
<td></td>
<td>(n=10)</td>
<td>(n=14)</td>
<td>(n=10)</td>
<td>(n=14)</td>
</tr>
<tr>
<td>41-50</td>
<td>11.5 ±0.7</td>
<td>--------</td>
<td>28.5 ±0.7</td>
<td>--------</td>
</tr>
<tr>
<td></td>
<td>(n=4)</td>
<td>(n=0)</td>
<td>(n=4)</td>
<td>(n=0)</td>
</tr>
</tbody>
</table>

ANOVA F=0.68
P>0.05
Results expressed as mean ± SD

PT = Prothrombin time
PTT = partial thromboplastin time with kaolin tests
S =seconds
n =number of subjects studied

Table 4: sex variation on PT and PTTK in smokers

<table>
<thead>
<tr>
<th>Sex</th>
<th>Male (n=106)</th>
<th>Female (n=14)</th>
<th>Calculated T</th>
<th>Critical T</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT (s)</td>
<td>11.4 ±1.9</td>
<td>11.9 ±1.9</td>
<td>0.15</td>
<td>1.96</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PTT (k)</td>
<td>24.5 ±4.0</td>
<td>29.4 ±5.3</td>
<td>0.29</td>
<td>1.96</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

Results expressed as mean ± SD

PT = Prothrombin time
PTT (K) = partial thromboplastin time with kaolin tests
s =seconds
n =number of subjects studied

Reference