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RESEARCH ARTICLE

Influence of chronic Cigarette Smoking on Serum Biochemical Profile among Sudanese smokers

Mutwakil H. Alhibrii [1], AbdElkarim A. Abdrabo*[2], Mohamed F. Lutfi[3]

[1] Department of Clinical chemistry, Faculty of Medical Laboratory Sciences, Sudan International University- Sudan

[2] Department of Clinical chemistry, Faculty of Medical Laboratory Sciences, Al Neelain University - Sudan

[3] Department of Physiology, Faculty of Medicine and Health Sciences, Al Neelain University- Sudan

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ABSTRACT

Context: Cigarette smoking is a risk factor for wide range of diseases; however, still there are no laboratory screening tests for early detection of the possible biochemical derangements induced by smoking.

Aim: to evaluate serum cholesterol, liver enzymes, uric acid and hemoglobin concentrations in smokers regardless of their present complain.

Materials and Methods: the study involved a control group of apparently healthy non-smokers (N = 105) matched for age with a test group of smokers (N = 105). The age range of both groups was 25-63 years. Random blood glucose (RBG), serum cholesterol, uric acid, liver enzymes and haemoglobin concentrations were measured according to the standards. Appropriate statistical tests were used to assess significant difference in the means of the studied concentrations between smokers and the control group.

Results: The smokers showed significantly higher RBG (M±SD = 143.7±27.0 mg/dl), aspartate transaminase (M±SD = 26.2±6.0 U/L), alanine transaminase (25.0±5.3 U/L) and haemoglobin (M±SD = 13.5±2.0 g/dl) levels compared to with non-smokers (M±SD = 127.9±26.4 mg/dl, 23.9±6.1 U/L, 22.6±10.1 U/L, 12.8±1.9 g/dl respectively, $P < 0.05$). In contrast, uric acid concentrations were less in smokers (M±SD = 4.9±0.8 mg/dl) compared with the control group (M±SD = 5.1±0.7 mg/dl and, $P = 0.048$). Cholesterol and alkaline phosphatase concentrations were not significantly different in the studied groups.

Conclusion: the study added further evidences for the possible harmful consequences of smoking including augmented oxidative stress as indicated by low serum uric acid levels and high liver transaminases concentrations, hyperglycaemia and high haemoglobin concentrations.

Keywords: Cigarette smoking, biochemical derangements, Random blood glucose, harmful consequences.

1. INTRODUCTION

The hazards of cigarette smoking are well recognized worldwide [1, 2], yet significant numbers of people continue to smoke in the developing countries [3, 4]. In Sudan, prevalence of cigarette smoking in the adult population reached 12% [4]. Alternatively in the some developed countries, although prevalence of cigarette smoking is almost double that of Sudan, it started to decrease over the last year [5].

Cigarette smoking is a known risk factor for respiratory [6, 7], cardiovascular [8, 9], neoplastic [10, 11] and other diseases [12, 13]. The common pathophysiologies of most smoking-related diseases are imbalance of systemic oxidants and antioxidants [14, 15], enhanced inflammatory reactions [16], insulin resistance [17-20], dyslipidemia [21] and others [22-24]. In spite of the growing knowledge regarding the detrimental effects of smoking, there are no routine laboratory tests used for early detection of biochemical derangements in cigarette smokers. Simple investigations like lipid profile, hepatic enzymes, inflammatory markers, uric acid and hematological counts may gives clues about

*Corresponding author: AbdElkarim A. Abdrabo | Department of Clinical chemistry, Faculty of Medical Laboratory Sciences, Al Neelain University - Sudan | Email: abdrabokarim@hotmail.com

the possible complications of smoking in the near future. For examples hypercholesterolemia puts smokers at risk of atherosclerosis, high liver enzymes and/or low uric acid may indicate enhanced oxidative stress, high hemoglobin may be a compensation for hypoxic hypoxia due to smoking-induced respiratory disease or low oxygen carrying capacity secondary to excess carboxyhemoglobin. In Sudan, previous studies assessing these simple parameters are scarce, if ever. This study aims to evaluate serum cholesterol, some liver enzymes, uric acid and hemoglobin concentrations in smokers regardless of their present complain.

2. MATERIALS AND METHODS

The study involved two groups: a control group of apparently healthy non-smokers (N = 105) matched for age with a test group of smokers (N = 105). The age range of both groups was 25-63 years. All volunteers were recruited from Omdurman military hospital – Khartoum - Sudan

Venous blood samples were collected from each volunteer in appropriate containers. Random blood glucose, serum cholesterol, uric acid, and liver enzymes concentrations, namely aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP), were measured using Spinreact reagents (Girona – Spain). Hemoglobin concentrations were determined using automated haematology analyzer (Sysmex - Japan).

Statistical evaluation was performed using the Microsoft Office Excel (Microsoft Office Excel for windows; 2007) and SPSS (SPSS for windows version 19). Normal distribution of the studied variables was examined using Kolmogorov-Smirnova and Shapiro-Wilk tests. Unpaired T-test and Mann-Whitney U test were used to assess significant difference in the means of the studied variables in smokers and non-smokers. Correlations between serum biochemical profile and the duration of smoking were assessed using bivariate correlations. P < 0.05 was considered statistically significant.

3. RESULTS

The males constitute 82.9% and 93.4% of the control and the test groups respectively; reflecting the significant association between male gender and smoking (Pearson's chi-square = 6.77, *P* = 0.009). The age of the smokers (M±SD = 42.1±9.9 years) was comparable with the control group (M±SD = 39.9±12.0 years, *P* = 0.148). The M±SD of the duration of the smoking = 16.5±9.0 years with an average of 4.5 cigarettes/day.

Although Cholesterol concentrations were higher in the smokers (M±SD = 169.5±13.0 mg/dl) compared with non-smokers (M±SD = 165.8±15.1 mg/dl), this difference did not reach statistical significance (*P* = 0.057, figure 1). In contrast, random blood concentrations were significantly higher in smokers (M±SD = 143.7±27.0 mg/dl) compared with the control group (M±SD = 127.9±26.4 mg/dl, *P* = 0.000) (figure 1).

As shown in figure 2, the only liver enzyme that did not achieve significant concentration difference between studied groups was alkaline phosphatase. The concentrations of AST and ALT were significantly higher in smokers (M±SD = 26.2±6.0 U/L, 25.0±5.3 U/L respectively) compared to non-smokers (M±SD = 23.9±6.1 U/L and 22.6±10.1 U/L, *P* = 0.000 and 0.005 respectively). In addition, uric acid concentrations were lower (M±SD = 4.9±0.8 mg/dl) while haemoglobin levels were higher (M±SD = 13.5±2.0 g/dl) in smokers compared to the control group (M±SD = 5.1±0.7 mg/dl and 12.8±1.9 g/dl, *P* = 0.048 and 0.001 respectively).

There was significant positive correlation between the duration of smoking and aspartate transaminase concentrations (CC = 0.391, *P* = 0.000). However, there were no other significant correlations between duration of smoking and the other studied parameters (*P* > 0.05).

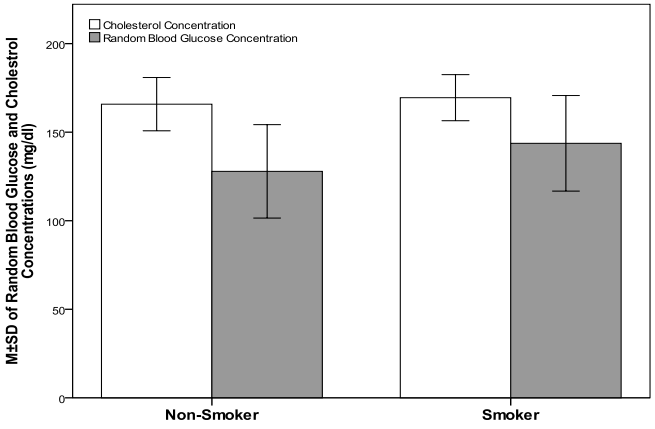

Figure 1: Random blood glucose and cholesterol concentrations in the studied groups

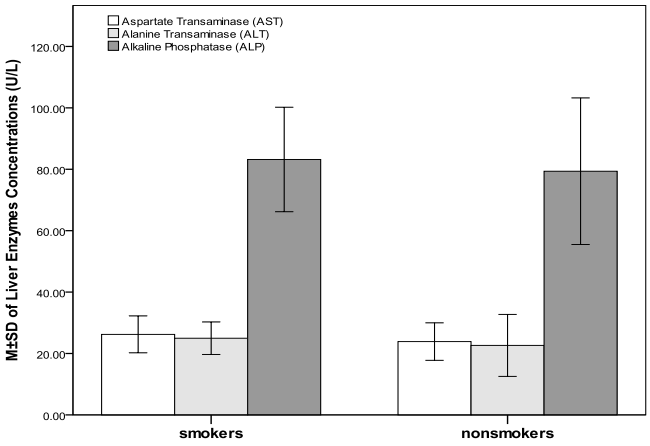

Figure 2: Liver enzymes concentrations in the studied groups .

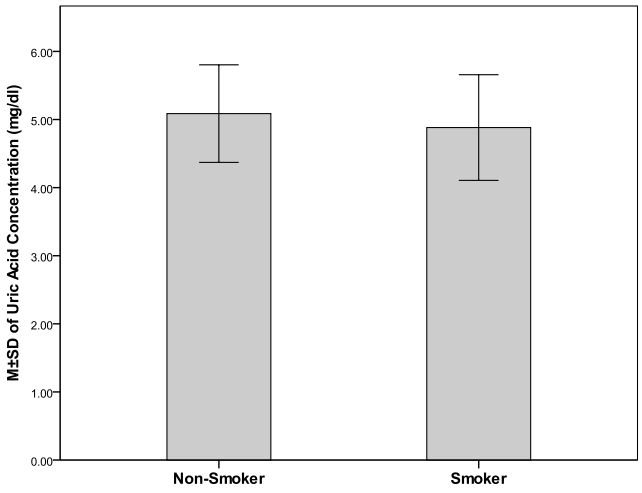

Figure 3: Uric acid concentrations in the studied groups

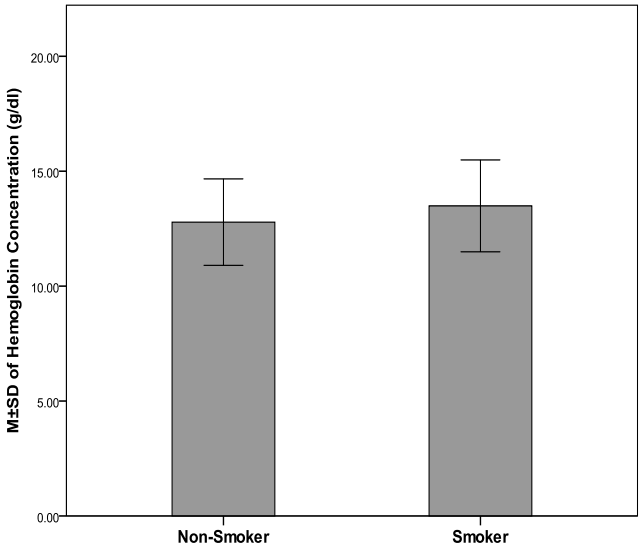

Figure 4: Hemoglobin concentrations in the studied groups.

4. DISCUSSION

The current results uncover some of the detrimental effects of smoking including hyperglycaemia, hypouricemia, increased liver transaminases and haemoglobin concentrations. These findings give venerable explanation for the increasing number of deaths among smokers all over the world [25].

The hyperglycaemic effect of smoking can partly be explained by its negative effects on insulin [26]. Short-term effects of smoking include augmentation sympathetic activity, elevation of catecholamines levels and consequently insulin resistance [17]. Actually, previous reports demonstrated a significant dose-response trend for higher risk of non-insulin dependent diabetes mellitus (NIDDM) among heavy smokers [18-20]. Insulin resistance in smokers also explains their tendency for hyperlipidemia and consequently coronary heart diseases [19, 26]. In the current study, results revealed higher cholesterol concentrations in the smokers compared with non-smokers; however, the difference in the means did not achieve statistical significance. However, according to Tucker *et al*, heavy smokers were 2 times and mild/moderate smokers were 1.5 times more likely to have hypercholesterolemia than non-smokers [21].

In the current results serum uric acid level in smokers did not reach the lower reference range (3.5 mg/dl); however, it was significantly lower than the non-smoker group. Physiologically, uric acid is the most abundant aqueous antioxidant, accounting for up to 60% of serum free radical scavenging capacity and hence its serum level can reflect body antioxidant capacity. Dietrich *et al* reported an intimate association between smoking and reduction of antioxidants including uric acid [27]. A recent study conducted by Haj Mouhamed and others confirmed the significantly low plasma levels of uric acid in smokers [28]. The same study attributed hypouricemia to the enhanced oxidative stress induced by chronic cigarette smoking and recommended plasma uric acid estimation as biological tobacco marker [28].

The effects of smoking on liver enzymes are controversial. Some previous reports suggest that cigarette smoking does not cause liver injury, but may enhance the effects of alcohol on liver damage in heavy drinkers [22]. Alternatively, other reports demonstrate that the activity of both AST and ALT were significantly elevated in heavy smokers, but not in mild to moderate smokers [23]. A possible explanation for increased transaminases level in smokers is the synergistic effects between smoking and oxidative stress. This assumption is further supported by the significantly higher transaminases but low uric acid levels in the smokers of this study. However, additional researches are desirable to clarify the possible cause(s) for enhanced transaminases activity in smokers.

The results of the present study also add further prove for the tendency of smokers to have higher hemoglobin concentrations. By the end of the last century, the data of the second national health and nutrition examination survey (NHNES) revealed generalized upward shift of the hemoglobin distribution curve in cigarette smokers [24]. The same survey showed no significant difference in mean hemoglobin when ex-smokers were compared with never-smokers. In contrast, more recent reports were able to demonstrate significantly higher hemoglobin levels in the current smokers compared with the never smokers [29] especially in those smoking more than 10 cigarettes per day [30].

It seems logical to mention that the current study was limited by two main shortcomings: firstly, evaluation of lipid profile in the studied subjects was not complete because most of the volunteers were not fasting at the

time of blood sampling. Only serum cholesterol was measured to assess dyslipidemia because its serum level would not significantly be affected by diet [31, 32]. Secondly, the majority of studied subjects with smoking habits are males. This is simply because smoking habit is not common among Sudanese females. The male gender predominance in the test group might act as a physiological confounding factor that influences the association between smoking habits and the serum concentrations of some studied variable like haemoglobin concentrations.

In conclusion, the present study elucidates a number of possible harmful consequences of smoking. Augmented oxidative stress as indicated by low serum uric acid levels and high liver transaminases concentrations, hyperglycaemia and increased haemoglobin concentrations were obvious in smokers. These pathological derangements will definitely put smokers at a higher risk of premature death.

5. ACKNOWLEDGEMENT

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Conflict of Interest: None Declared

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