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Effects of Smoking on Hepatic and Renal Biomarkers among Smokers in Brazzaville

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ABSTRACT

Background : Exposure to tobacco has serious consequences on many organs such as the liver and kidneys.
Objective. This study aimed to determine the effect of cigarette smoking on serum concentrations of uric acid, creatinine, and transaminases (AST and ALT).
Materials And Methods : A cross-sectional study was conducted during the period from July to November 2018, in Brazzaville (Republic of Congo). A total of 100 subjects were included, on the basis of inclusion criteria randomly allocated, 50 smokers and 50 non-smokers. The smokers were divided into three groups according to the intensity and duration of smoking (light smokers, moderate smokers and heavy smokers). The average age was 35.86 ± 11.65 and 31.88 ± 8.92, respectively for non-smokers and smokers. A pre-established questionnaire allowed the collection of demographic and clinical data. Urine samples were collected to verify cigarette smoking status. The biochemical assays were carried out by spectrophotometry, on the serum obtained after centrifugation of venous blood collected on a dry tube, using commercial kits. The average age and weight of smokers were lower than those of non-smokers. The levels of uric acid, creatinine and transaminases were significantly elevated in the groups of smokers, depending on the intensity and duration of smoking compared to the group of non-smokers.

Keywords: smokers, tobacco, uric acid creatinine, transaminases, Brazzaville

INTRODUCTION

Smoking is a major public health issue. Tobacco consumption is a scourge that kills around 6 million people each year, estimates the World Health Organization.(1) Illness and death caused by exposure to tobacco smoke is the most serious preventable public health problem today. The smoke of tobacco is made up of many chemicals, cytotoxic, carcinogens and free radicals that affect many organs.(2) Tobacco smoking is a well-known independent risk factor for cardiovascular diseases and type 2 diabetes. Many previous studies have suggested that smoking is also associated with an increased risk of insulin resistance and metabolic syndrome.(3-5) Smoking is associated with an increase in the frequency of pathologies such as myocardial infarction, strokes, type 2 diabetes, lung cancer and many other cancers.(6) Smoking also increases the risk of liver cirrhosis and also affects kidney function.(7) Cigarettes were not thought to cause direct liver injury or to increase the risk of developing a chronic liver disease such as non alcoholic fatty liver disease (NAFLD) or alcohol-associated cirrhosis.(8) Smoking is associated with increased

progression and severity of liver disease, particularly fibrosis and liver cancer. This is logical, given what is known about the carcinogenic and profibrogenic effects of smoking on the human body.[9,10] Data from the literature show that smokers tend to lose at least 10 years of life compared to individuals who have never smoked.[10-13] Moreover, several authors report that smoking is associated with an increased risk of CKD. [14-16]

The objective of this study was to determine the effects of smoking on renal and hepatic biomarkers among smokers in Brazzaville.

MATERIALS & METHODS

We use a non-randomized convenient sampling technique. 100 subjects were included in the study on the basis of inclusion criteria and were randomly allocated into 2 groups as Group A and B. Subjects were aged between 18 and 70 years old, recruited in five (05) districts of Brazzaville (Bacongo, Poto-poto, Moungali, Talangaï, Mfilou). All subjects included were volunteers and had signed informed consent. On inclusion, sociodemographic and clinical characteristics (Table I) were collected after questioning and routine clinical examination. Each subject included benefited from a clinical investigation, in search of underlying pathology. Thus, obese subjects, alcoholics, or subjects with risk factors such as hypertension and diabetes mellitus were excluded from this study. Each participant had given informed consent and the study was approved by the scientific committee of the Faculty of Health Sciences (Marien NGOUABI University).

The participants in this study were divided into 2 groups:[17]

- Group I - Non-smokers (Control) n= 50
- Group II - Cigarette smokers n = 50

Group II were divided into 3 subgroups according to duration and number of cigarettes smoked per day :

- Group II A (n=19) - Light smokers (Duration - 1 to 5 years, between 10 and 15 cigarettes/day)
- Group II B (n=18) - Moderate smokers (Duration 6 to 10 years, between 16 and 20 cigarettes/day)
- Group II C (n=13) - Heavy smokers (Duration - more than 10 years, beyond 20 cigarettes/day).

Epidemiological data were collected using a survey form, during the interview with the participants. The blood was collected in a dry tube, after fasting for about 12 hours, and the urine was collected in a sterile pot. After collection, the blood samples were centrifuged at 3500 rpm for 5 minutes and the serum was decanted and then stored at a temperature of – 20°C until the biological analyses.

The sera were used to assay the levels of uric acid, creatinine and transaminases (AST and ALT), by colorimetric or enzymatic methods, using the *BIOMATE 3S spectrophotometer* and commercial kits.

The dosage of urinary cotinine was made in order to objectify tobacco exposure in our study population. We used the commercial kit *Narco Check*®.

Statistical Analysis

The quantitative variables are presented in mean ± standard deviation and the qualitative variables in numbers and percentages. Data were processed with Microsoft Excel 2016 software for database design and Graph Pad software version 8 (California, USA) for statistical analyses. The ANOVA test was used to compare the different groups. The value of $p < 0.05$ was considered significant.

RESULT

The average age was 35.86 ± 11.65 and 31.88 ± 8.92 respectively for the non-smoking and smoking groups. The smoking group was almost exclusively made up of men (49 men against one (01) woman). The average weight was 59.06 ± 7.40 and 65.44 ± 10.23 in the groups of smokers and non-smokers respectively, with a significant difference ($p=0.0006$). Table I summarizes the socio-demographic characteristics of the study population.

Table I: socio-demographic characteristics of the study population

Variable	Non smokers	Smokers	*p* -value
Age (years)			
Mean ± SD	35.86±11.65	31.88±8.92	*p=0.06*
Median (Min – max)	48 (22-70)	33 (18-51)	
Gender			
Men n(%)	23 (46)	49 (98)	
Women n(%)	27 (54)	01 (02)	
Sex ratio (M /F)	0.8	49	
Weight			
Mean ± SD	65.44±10.23	59.06 ±7.40	*p=0.0006*
Median (Min – max)	35 (45-102)	57 (43-78)	
BMI			
Mean ± SD	23.37 ±0.37	22.72 ±0.32	*p=0.02*
Median (Min – max)	18.20 (17.64-35.84)	11.38 (15.38-26.76)	

n: number; SD: Standard deviation; Min: minimum; Max: maximum; %: percentage

Urinary cotinine concentrations increased with duration and intensity of smoking. Figure 1 illustrates the comparison of parameters in different test groups (group IIA, I I B and IIC).

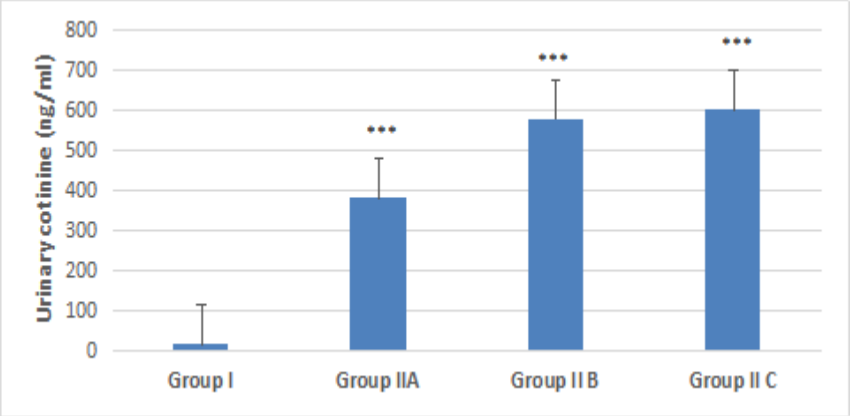

Figure 1 : distribution of participants according to categories. Group I (non-smokers) , Group II A (light smokers), Group II B (moderate smokers), Group II C (heavy smokers). The results are expressed as mean ± standard deviation.

Table II gives the levels of uric acid, creatinine and transaminases (AST, ALT) in non-smokers (Group-I), in light smokers (Group-II A), in moderate smokers (Group-II B) and in heavy smokers (Group–II C). Analysis of these results show an increase in the concentrations of the various variables depending on the duration and degree of smoking. The difference between the groups is significant.

Table II : profile of serum biomarkers in smokers and non-smokers, according to the duration and intensity of smoking

Variables	Non smokers	Smokers		
	Group I (n=50)	Group II-A (n=19)	Group II-B (n=18)	Group II-C (n=13)
Uric acid (mg/l)	37.09 ± 1.89	51,88 ± 2,288[a]	53,95 ± 1,92[a]	60,75 ± 1,94[a]
Creatinine (mg/l)	14,42 ± 0,39	16,58 ± 0,43[b]	18,12 ± 0,49[b]	18,85 ± 0,42[b]
GOT (UI/l)	18,18 ± 1,14	36.37 ± 3,26[a]	36,72 ± 3,53[a]	47,85 ± 2,75[a]
GPT (UI/l)	27,79 ± 1,59	50,28 ± 2,24[a]	55,99 ± 1,04[a]	59,43 ± 1,38[a]

(a):p<0.0001; (b):p<0.001

DISCUSSION

This study aimed to determine the effects of smoking on liver and kidney biomarkers in smokers, depending on the duration and intensity of smoking. Cotinine is a marker of tobacco exposure and is used to classify smokers according to duration and exposure to smoking.[18] The present study involved a total of 100 participants (50 smokers and 50 non-smokers). The average ages were 35.86 and 31.88, respectively among non-smokers and smokers. In addition, the body mass index of smokers was lower than that of non-smokers.

Cotinine is widely used as a biomarker of average daily nicotine intake. In this study, it made it possible to confirm smoking or exposure to tobacco, to better appreciate the

impact of the latter on the concentrations of the biomarkers studied.[19] Concentrations of relatively low levels of cotinine found in the group of non-smoking participants could be attributed to a form of passive smoking. Therefore, smoking assessed solely based on participants' self-reports is likely to underestimate the number of active and passive smokers. However, they were not associated with elevated uric acid, creatinine or transaminase values.

In this study, smoking was associated with increased levels of uric acid, creatinine and transaminases. The concentrations of these biomarkers increased proportionally with the intensity of smoking. A study conducted in Japan showed that smoking was associated with increased serum uric acid levels, regardless of gender.[20] Younkyung and Jihun[21] reported a dose-response relationship between cotinine-verified smoking and the risk of hyperuricemia only in women, in a general population study.[22]

Kidney function tests (including creatinine) are sufficient to determine if a patient has kidney disease.[23] In this study, the group of smokers had significantly higher creatinine values than those of the group of non-smokers. The increase in serum creatinine concentrations could be explained by the presence in tobacco of nicotine, free radicals and other nephrotoxic substances, likely to cause alterations in renal function in smokers.[24] Renal damage, assessed by the serum creatinine level, was proportional to the intensity and duration of exposure. Several mechanisms could explain this nephrotoxicity. Authors have reported that the consumption of 20 cigarettes per day leads to the inhalation of doses of cadmium (a nephrotoxic substance) of the order of 3.6 to 6.0 µg, of Cadmium, which is a cumulative nephrotoxic.[25] Cadmium nephrotoxicity would cause changes in proximal tubular function, characterized by increased excretion of beta 2-microglobulin and giving rise to the classic tubular proteinuria and glomerular dysfunction revealed by increased serum creatinine concentrations.

The present study found that AST and ALT liver enzymes were significantly elevated in the smoking groups compared to the non-smoking group. The literature review reports a controversy regarding the effects of tobacco exposure on liver enzymes.[2,26] Caution is therefore warranted in interpreting the results.

However, this study has some limitations. Indeed, the use of urinary cotinine revealed that a small part of the participants, considered non-smokers, corresponded rather to the definition of moderate passive smokers. Furthermore, failure to take into account other factors modifying the biomarkers studied could constitute a bias in the interpretation of the results. Despite these observations, the results of this preliminary study could constitute a basis for carrying out studies on a larger population, with more rigorous selection criteria and a more exhaustive questionnaire, taking into account various known factors having an impact on hepatic and renal biomarkers.

CONCLUSION

This study showed that smokers had significantly higher values of uric acid, creatinine and transaminases (AST and ALT) than non-smokers. This elevation was proportional to the intensity and duration of exposure. These results reveal that smoking would have effects on serum levels of uric acid, creatinine and transaminases. These effects depended on the duration and intensity of smoking. Studies on a larger population would make it possible to better characterize the effects of tobacco on biochemical parameters.

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Conflict of Interest: None

Source of Funding: None

Ethical Approval: The study was approved by the scientific committee of the Faculty of Health Sciences (Marien NGOUABI

University) and each participant had given informed consent.

Availability of data and materials: The data sets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Authors' contributions: LMM, AT– study design, acquisition, analysis and interpretation of data. LMM, CRDD - drafting the article. DM, AAA – revising the manuscript. All authors revised the article critically for important intellectual content, and approved the final version of the manuscript.

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