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Impact of Cigarette Smoking on Kidney Functions of Adults in Kosti, Sudan

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KEYWORDS

Smokers, Non-smokers, Duration, Creatinine, Urea, Uric acid, Kidney functions

ABSTRACT:

Objectives: To determine the impact of cigarette smoking on renal parameters among Sudanese adult subjects in Kosti –White Nile State in the period from September to December 2021.

Method: This is a cross-sectional case-control study conducted at Kosti City located in the White Nile State of Sudan. General data from the subjects were collected by using a self-administrated pre-coded questionnaire specifically designed to obtain information that helped in the study. 5ml of blood samples were collected from the cephalic vein of 50 healthy smokers and 50 healthy no-smokers respectively. The serum was separated in sterile containers and was analyzed for creatinine, urea, and uric acid concentration. The 2-tailed T-test and P value are obtained to assess the significance of the results.

Results: A positive relation was observed between the urea level and the duration of smoking per year. The urea level was significantly higher and a significant correlation between urea and uric acid was also observed in the study group.

Conclusion: There is a substantial correlation between urea, uric acid, and smoking. Smoking for years modulates kidney functions.

Introduction

Smoking, a global tobacco epidemic, as referred to by WHO, is reported to be one of the primary reasons for early death worldwide. The prevalence of cigar smoking differs based on location, the historical period, the population surveyed, and also on the method of survey.

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The United States is considered to have the foremost position in terms of smoking, closely followed by Germany and the United Kingdom [1]. In a gender basis study, 4.3% of men and 0.3% of women were found to smoke cigars [2].

Smoking is studied to affect an individual's health irrespective of his or her age. Recently, there has been a rise in morbidity and mortality associated with cardiovascular and lung diseases related to smoking [3]. Smoking is known to induce kidney damage as well [4]. Among the several toxic and noxious components of tobacco, nicotine is established as the key element introduced through active and passive smoking [5,6,7]. The active substances in tobacco are rapidly absorbed into the bloodstream through the alveoli in the lung. Cigarette smoking is observed to reduce the effectiveness of hemoglobin (Hb). Hb attaches to carbon monoxide (CO) to form carboxyhemoglobin, which is an inactive form that fails to transport oxygen. This disturbs the oxygen delivery mechanism to tissues as the capability of the Hb molecules is reduced [8]. At the same time, the bone marrow is stimulated for erythropoiesis which increases hemoglobin concentration and hematocrit value. [9]. This poses a threat to intravascular clotting [10].

Raised levels of serum cadmium and lead levels in smokers are reported in some studies resulting in glomerular dysfunction. Nephropathies are accelerated by nicotine with an increased incidence of microalbuminuria progressing to proteinuria [11]. The vasoconstrictive quality of nicotine is responsible for the transient increase in blood pressure and a decline in the glomerular filtration rate and renal blood flow [12]. Smoking induces the activation of fibroblasts thereby, accelerating kidney fibrosis which along with the damage of the epithelial and endothelial cell linings leads to a decline in kidney function [13,14]. Research also showed that glycotoxins levels such as advanced glycation end products (AGEs) are considerably higher in cigarette smokers [15] and these are responsible for the rapid advancement of chronic kidney diseases (CKD), particularly in diabetic cases [16]. In a study including middle-aged Japanese men, smoking was found to elevate the chances of glomerular hyperfiltration [17]. Many studies reported non-smokers to have considerably low GFR compared to cigarette smokers [18-21].

Hence, the present study was conducted to determine the impact of cigarette smoking and the duration of smoking on the renal functions of adult Sudanese.

Methods

Study Design:

This is a cross-sectional case-control study conducted at Kosti City located in the White Nile State of Sudan. Overall, 100 samples were collected for this study, of which 50 samples comprised healthy smokers without any history of ailments. The remaining 50 samples included healthy individuals aged 18 – 60 years, which is considered the control group. Data was collected using a self-administrated pre-coded questionnaire designed to obtain information that helped the study.

Sample collection:
5ml of blood samples were collected from the cephalic vein of the controls and the smokers using sterile syringes. This blood was allowed to clot at room temperature, and serum was separated in sterile containers and was analyzed for creatinine, urea, and uric acid concentration.

Estimation of Creatinine: Creatinine was estimated by Modified Jaffe's method (Alkaline Picrate Method)

CALCULATION OF ESTIMATED GFR (MDRD equation): the following formulae were used for the calculation in the Sudanese population:

eGFR (ml/min/1.73m^2) = 186 x (S. Creatinine in mg/dl)$^{-1.154}$x (Age in yrs)$^{-0.203}$ (for female x 0.742)

Estimation of Uric Acid: Uric acid was estimated by Uricase –Trinder – Enzymatic and colorimetric method and was calculated as follows:

URIC ACID (mg/dl) = Absorbance of test X Concentration of standard (mg/dl)

Estimation of Urea: Urea was estimated by the Diacetyl Monoxime Method and was calculated as follows:

Blood urea (mg%) = $\frac{\text{O.D. Test}}{\text{O.D. Std.}}$ x $\frac{\text{Amount of Std.}}{\text{Vol. of blood.}}$ x 100

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Statistical analysis:

The SPSS tool was used to analyze the results obtained. The 2-tailed T-test and P value were obtained to assess the significance of the results.

Results:

Based on the data, it was found that 76% of our study group was within the age group of 20-30 years rest 22% was more than 30 years and only 2% was found to be less than 20 years as shown in Fig.1. Fig.2 represents the duration of smoking; 28% of the group was found to smoke for 3-5 years, 30% was found to smoke for less than 3 years whereas the majority (42%) was found to smoke for more than 5 years. Again 52% of the subjects were found to smoke less than 10 cigarettes per day, 40% were found to smoke 10-15 cigarettes per day, and the rest 8% were found to smoke more than 15 cigarettes per day as shown in Fig 3.

To continue with the results, this study also revealed that urea level was significantly higher in the smoker group when compared to the non-smoker group, and the increase in the level of urea is proportional to the duration of smoking per year. While the creatinine level remains steady with a slight fluctuation in uric acid levels as depicted in Tables 1,2 and 3.

Likewise, this research showed a significant correlation between urea and uric acid in the study group. Additionally, a substantial correlation was observed between urea and creatinine as well as among urea and uric acid respectively in the control group as represented in Tables 4.5 and 6.

Figuer1: Distribution of the study population according to age:

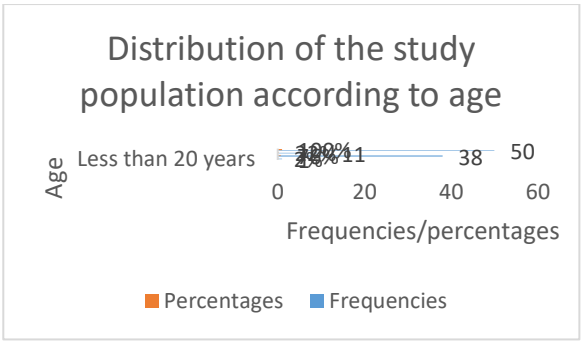

Figure 2: Distribution of the population according to the duration of smoking

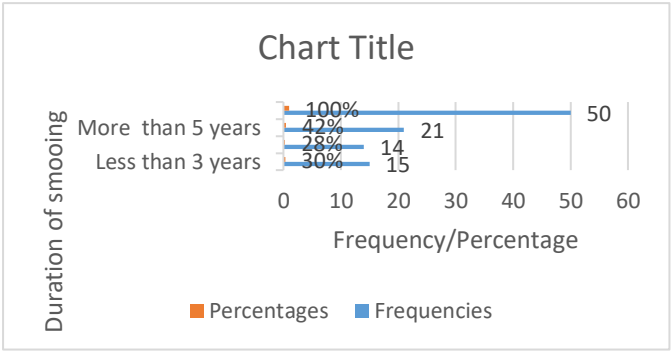

Figure 3: Distribution of the population according to cigarette consumption (cigarette/day)

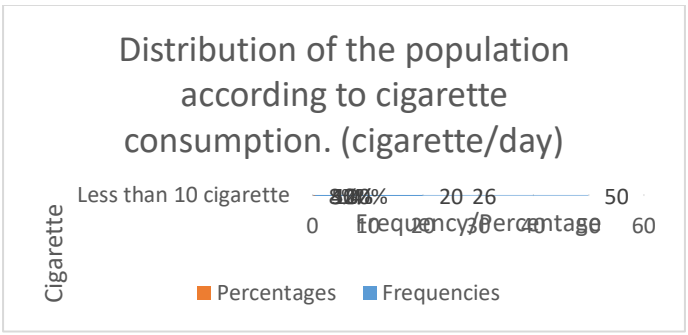

Figure 4: Distribution of renal parameters among the study groups

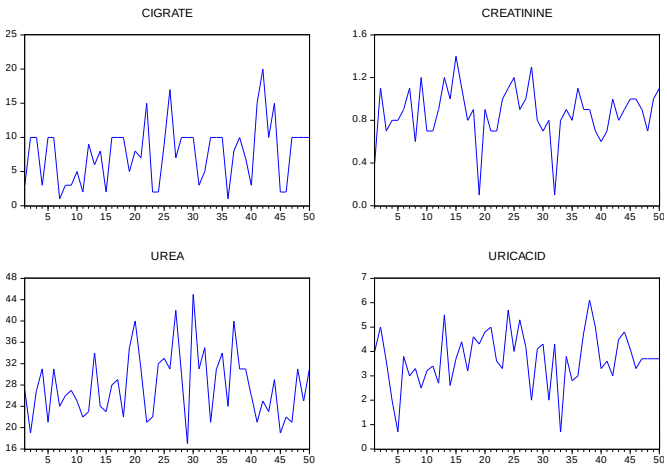

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Table 1: Comparison of renal function test parameters between smokers and controls according to the duration of smoking (Less than 3 years).

Parameters		N	Mean	Std. Deviation	P-Value
Urea	Control	50	23.48	7.32	.289
	Case >3Yrs	15	25.60	4.06	
Creatinine	Control	50	.90	.25	.938
	Case >3Yrs	15	.90	.26	
Uric Acid	Control	50	4.74	3.96	.162
	Case >3Yrs	15	3.26	1.15	
Age	Control	50	27.70	8.96	.014
	Case >3Yrs	15	21.80	1.37	

Table 2: Comparison of renal function test parameters between smoker people and controls according to the duration of smoking (from 3 – 5 Years)

Parameters		N	Mean	Std. Deviation	P-Value
Urea	Control	50	23.48	7.32	.008
	Case 3-5Yrs	14	29.50	7.14	
Creatinine	Control	50	.90	.25	.870
	Case 3-5Yrs	14	.89	.28	
UricAcid	Control	50	4.74	3.96	.602
	Case 3-5Yrs	14	4.17	.94	
Age	Control	50	27.70	8.96	.623
	Case 3-5Yrs	14	26.35	9.12	

Table 3: Comparison of renal function test parameters between smoker people and controls according to the duration of smoking (more than 5 years)

Parameters		N	Mean	Std. Deviation	P-Value
Urea	Control	50	23.48	7.32	.010
	Case <5Yrs	21	28.38	6.69	
Creatinine	Control	50	.90	.25	.232
	Case <5Yrs	21	.82	.21	
UricAcid	Control	50	4.74	3.96	.253
	Case <5Yrs	21	3.72	1.12	
Age	Control	50	27.70	8.96	.166
	Case <5Yrs	21	31.38	12.49	

Table 4: Comparison between means of Urea, Creatinine, Uric Acid, and levels in smokers and non-smokers

Parameters	Case		Control		P-Value
	Mean	St.D	Mean	St.D	
Urea	27.86	6.24	23.48	7.32	0.002
Creatinine	0.86	0.25	0.90	0.25	0.457
Uric Acid	3.71	1.12	4.74	3.96	0.081
Age	27.10	10.15	27.70	8.98	0.755

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Table 5: Correlations between urea, creatinine, uric acid, age, and cigarette in smokers

Parameter	Uric acid	Age	Cigarette	
Urea	-.141-	.339*	-.193-	.020
	.329	.016	.179	.889
		.855	.205	.937
* Correlation is significant at the 0.05 level (2-tailed).				

Table 6: Correlations between urea, creatinine, uric acid, age, and cigarette in non-smokers

Parameter	Creatinine	Uric.Acid	Age
Urea	.451**	.355*	.015
	.001	.011	.918
** Correlation is significant at the 0.01 level (2-tailed).			
* Correlation is significant at the 0.05 level (2-tailed).			

Discussion:

The present study revealed that the urea level was significantly higher in smokers when compared to non-smokers. These findings agree with the results of other studies [22-26]. This rise in the urea level of smokers indicates renal dysfunction.

Besides, the study also indicated that the rise in the urea level is proportional to the duration of smoking per year. Though hardly any remarkable changes were observed in the creatinine levels with slight fluctuation in uric acid readings, a significant correlation was well established between the urea and creatinine levels of the control group. A significant association was also observed between the urea and uric acid levels of the said group. Likewise, the study group also confirmed a significant positive correlation between urea and uric acid levels. The changes in the overall readings imply the modulations of the kidney functions.

Nicotine, being a key component of cigarette smoke known to have a devastating effect on different tissues of the human body and thereby leads to the development of many diseases [27]. It is a potent contributor of free radicals and reactive oxygen species causing heart, lung, liver, and kidney damage [28].

Research showed nicotine modifies vascular endothelial structures modulating the vasoreactivity of the vasculature and thereby contributing to the clinical manifestations of smokers. Additionally, it was also found to alter the functions of smooth muscle cells [29].

The possible mechanism can be associated with an increase in the renovascular resistance of smokers. This causes a substantial decrease in the glomerular filtration rate (GFR), filtration fraction, and renal plasma blood, which substantiates the study by Ritz et al in the year 1998[23]. The decrease in GFR reduces the rate of distal tubular flow leading to an increase in urea reabsorption [30].

Thus, this present study successfully validates the outcomes of the previous studies and establishes the deteriorating effect of smoking on kidneys.

Conclusion:

This research aims to detect the impact of smoking on kidneys in adults. Based on our study, we can conclude that smoking has adverse effects on the kidneys. Duration of smoking affects the serum urea level by hindering the excretion of urea. Hence, it is advisable that smokers should drink an adequate amount of water to prevent dehydration and should estimate renal function consistently to avoid kidney damage.

Acknowledgment:

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Conflict of Interest:

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The authors state that they do not have any conflicts of interest

Ethical clearance:

The Standing Committee for Scientific Research – University of El Imam El Mahdi, School of Medicine granted ethical approval on February 24, 2021, and all methods were performed in accordance with the provisions of the approval.

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