

Effect of smoking and obesity on renal functions in the middle-age men in Basra city

Ali Abdul Baqi Ali ^{1,*}, Tayseer Ali Talab ² and Mahdi M Thuwaini ³

¹ Department of Surgery, Faculty of Medicine, University of Thi-Qar, Iraq.

² Department of Pharmacology, Faculty of Medicine, University of Thi-Qar, Iraq.

³ Department of Physiotherapy, College of Medical and Healthy Techniques, Southern Technical University- Basra, Iraq.

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Abstract

The overweight and obesity were increased all over the world as a serious public health concern. BMI is the most common measure used to determine whether the individual was an overweight or obese. There were a wide range of health complications associated with obesity, such as fatigue, shortness of breath, difficulty sleeping, back and joint pain, diabetes, high levels of serum lipids, cardiovascular disease, and chronic kidney disease. On the other hand, tobacco which contained a lot of chemicals, approximately 100 of them were classified as causes or potential causes of smoking-related diseases such as respiratory diseases, lung cancer, cardiovascular disease etc. The current study was performed on 77 subjects (25 smokers, 22 obese and 20 obese smokers, and 10 healthy controls) to investigate the effect of smoking and obesity on renal functions in the in middle-age. The study revealed that both smoking and obesity didn't affected the level of serum creatinine, uric acid and urea, but both significantly elevated the serum level of sodium, potassium and calcium and significantly increased the prevalence of proteinuria. According to the results we can concluded that both obesity and smoking induced significant electrolyte disturbances, and increased the prevalence of proteinuria which consider as sensitive marker of glomeruli injury.

Keywords: Smoking; Obesity; Renal functions; Creatinine; Urea; Electrolytes

1. Introduction

The renal effects of cigarette smoking were recorded as two to three fold decrease of glomerular filtration rate and increased of albuminuria or proteinuria. It caused chronic renal disease in general population. ^[1-2]

Nicotine is one of a lot of substances which obtained by active and passive tobacco smoking. Nicotine, in man is consumed commonly by smoking of cigars, cigarettes, and pipes. The addiction liability and biological effects of smoking are mainly mediated by nicotine, the main alkaloid in the tobacco. ^[3]

An inverse relationship was recorded between serum tobacco alkaloids and the estimated glomerular filtration rate. ^[4] The mechanisms of renal damage induced by smoking are included increasing of intraglomerular pressure as an acute effect and endothelial cell dysfunction as chronic effect. ^[5]

Overweight and obesity are important risk factors for renal function deterioration. Overweight increases the kidneys above, the additional work will elevated the risk of kidney disease. ^[6] It was recorded that obesity and overweight significantly caused renal function impairment in both genders and in all ages. ^[7] The consequences of kidney injury of obesity included hyper-filtration, which resulted in histological changes, continuous loss of glomerular filtration rate, hypertrophy of glomeruli and focal segmental glomerulo-sclerosis. However, deterioration of pressure natriuresis, due

* Corresponding author: Ali Abdul Baqi Ali

to elevation of tubular reabsorption of sodium, is a key event linked hypertension and obesity. [8-9] The current study was designed to study the effect of obesity and smoking on renal functions in the in middle-age men in Basra city.

2. Subjects and methods

This study was carried out in Basra city from May 2022 to September 2011. It was performed on 77 subjects, 25 smokers, and 22 obese and 20 obese smokers. The studied parameters were also investigated in 10 healthy subjects of the same age group to serve as control. All subjects were males, and the subjects complained hypertension, cardiovascular problem, diabetes, urinary system disease, and all participants taking drugs for any purposes were excluded. [9-10] the study was permitted by the postgraduate studies ethical committee of Southern Basra- Technical University, and a written informed consent was taken from all the participants. BMI (body mass index) was estimated as [weight (kg)/ square height (m)]. BMI of the healthy range is 18.5 to 24.9 kg/m², while, ≤25.0 kg/m² was considered as overweight. [10]

The blood samples were collected after 12 hrs fasting, left at room temperature for 30 minutes, the clotted blood was centrifuged for 5 minutes at 5000 rpm. The serum was separated and stored for subsequent investigation at -20 °C. Serum uric acid and serum urea were determined by an enzymatic method (kit: bio Merieux/ France). [11-12] While, creatinine was determined by colorimetric method (kit: Syrbio/France). [13] The serum levels of sodium, potassium and calcium were determined by flame atomic absorption spectrophotometry. [14-15] Urine samples were taken to measure the proteinuria by a dipstick method. [11] The statistical analysis was performed by using student t – test for all parameters except the results of proteinuria which analyzed by *chi-squared* test. [16]

3. Results

As shown in table 1, the age of the control group was 18-42, obese group 22-39, smoking group 25-42 and obese smoker group 21-40 years. All the participants are middle age, and almost of the same age group (18-42 years). BMI of the control group was 23.34±0.11, obese group 37.89±8.01, smokers 23.11±0.07 and obese smoker was 27.71±1.01.

No significant changes were recorded among groups in the level of serum creatinine, uric acid and urea, while there was significant increase in the prevalence of proteinuria in obese (13.67%), smokers (12.00%), and obese smokers (20%) in comparison with control group (0.0 %).

Table 1 The characteristics of subjects in smoking and **obese** groups

	Control	Obese	Smokers	Obese smokers
No	10	22	25	20
Age (years)	18-42	22-39	25-42	21-40
BMI kg/m ²	23.34±0.11	37.89±8.01	23.11±0.07	27.71±1.01

Table 2 Effect of smoking and obesity on renal function parameters

	Control	Obese	Smokers	Obese smokers
Creatinine (mg/dl)	1.09±0.01	1.12±0.01	1.12±0.02	1.18±0.06
Urea (mg/dl)	30.00±2.09	30.91±1.24	32.62±1.01	31.01±1.68
Uric acid (mg/dl)	5.06±0.04	5.15±0.51	5.66±0.34	4.99±0.33
Prevalence of proteinuria	0/10 (0%) ^a	3/22 (13.67%) ^b	3/25 (12.00%) ^b	4/20 (20%) ^c
Na ⁺ (mmol/l)	147.65±9.02 ^a	149.01±7.00 ^a	166.05±3.29 ^b	150.50±3.01 ^c
K ⁺ (mmol/l)	4.00±0.01 ^a	4.92±0.60 ^b	5.41±0.51 ^c	5.89±0.71 ^c
Ca ²⁺ (mmol/l)	7.99±0.07 ^a	9.01±0.11 ^b	9.12±0.34 ^b	10.19±0.41 ^c

Different letter in the same row means significant difference between means

However, there was a significant elevation in the serum sodium in smokers (166.05 ± 3.29 mmol/l) and in obese smokers (150.50 ± 3.01 mmol/l) than in control (147.65 ± 9.02 mmol/l) ($p < 0.01$ and $p < 0.05$, respectively). Serum potassium was also elevated in obese (4.92 ± 0.60 mmol/l), smokers (5.41 ± 0.51 mmol/l) and obese smokers (5.89 ± 0.71 mmol/l) compared with control (4.00 ± 0.01 mmol/l) ($p < 0.05$, $p < 0.01$ and $p < 0.01$, respectively), while calcium was elevated in obese (9.01 ± 0.11 mmol/l), smokers (9.12 ± 0.34 mmol/l) and obese smokers (10.19 ± 0.41 mmol/l), compared with control (7.99 ± 0.07 mmol/l) ($p < 0.05$, $p < 0.05$ and $p < 0.01$ respectively) (table 2).

4. Discussion

Although this study did not prove that smoking and obesity have any effect on the level of creatinine, urea and uric acid, but we do not rule out these effects because our study did not include the smoking period and the time of occurrence of overweight and obesity, where the effect on kidney functions was increased over time. However, smoking is a common risk factor for many diseases included renal disease.^[17] It is recorded that smoking increased urine volume, sodium and chloride excretion, aldosterone and vasopressin secretion, impaired endothelial cell-dependent dilatation of vessels, altered proximal tubular function and decreased glomerular filtration rate.^[18-20]

Nicotine reduced the cell survival and enhanced generation of reactive oxygen in the cells of human kidney. It elevated significantly the $\alpha 7$ nicotinic acetylcholine receptor expression ($\alpha 7$ nAChR). Nicotine enhanced NLRP6 inflammasome and caused endoplasmic reticulum oxidative stress. It induced necrosis, mild apoptosis and triggered kidney cells autophagy significantly, with histopathological alterations. The results showed that the expression of $\alpha 7$ nAChR, IRE1 α , NLRP6 and LC3 in kidney sections was significantly elevated by nicotine. These results clearly confirmed that nicotine induced renal disease by modulating $\alpha 7$ nAChR, NLRP6 inflammasome, endoplasmic reticulum oxidative stress and autophagy.^[21] The micro-albuminuria showed double incidence in smokers in comparison with non-smokers. Albuminuria is a sensitive biomarker of the damage of glomeruli; its elevation indicated that smoking caused kidney damage either directly or indirectly.^[22]

On the other hand, many works revealed the relationship between overweight and obesity and the development of chronic renal diseases and its progression. Overweight and obesity were linked to occurrence and progression of low estimated glomerular filtration rate.^[23-26] Higher BMI was correlated with the occurrence and development of proteinuria. Increased adipose tissue in men was positively linked with a high incidence of albuminuria.^[23-24, 27-28]

Our study showed that smoking and obesity elevated the serum levels of sodium, potassium and calcium. These results were in agreement with many authors who found that smoking had significantly increased levels of serum sodium, potassium and calcium, and serum and urinary creatinine values.^[29-30] It was postulated that these effects could be attributed to positive association of smoking with alcohol and coffee consumption and its inverse relation to physical activity.^[29-30] Furthermore, the prevalence of electrolytes disturbance is higher in obese than in the general population. Increased serum electrolytes in obese may reflect plasma hyper-tonicity.^[31-32]

5. Conclusion

The current study revealed that smoking and obesity associated with electrolyte disturbances and renal impairment. Because the study was conducted on Middle Ages, smoking and obesity are expected to lead to further deterioration in renal function by time. Therefore, continuous checking of kidney functions for smokers and obese people is necessary.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors confirm that this paper's content has no conflict of interests.

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Author's contributions

The authors drafted the and approved the manuscript.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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