

## Assessment of Creatinine and Microalbuminuria in Sudanese Smoker

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**Abstract:** Chronic smoking adversely influences the prognosis of nephropathies. The objective of this study was to the relationship between renal damage and chronic smoking through parameters can bring a clear vision about kidney status. This study was a case control study, was conducted in Khartoum state. Microalbuminuria is defined as excretion of 30–300 mg of albumin per 24 hours (or 20–200 mcg/min or 30–300 mcg/mg creatinine) on 2 of 3 urine collections. Microalbuminuria is caused by glomerular capillary injury and so may be a marker for diffuse endothelial dysfunction. According to Steno hypothesis, albuminuria might reflect a general vascular dysfunction and leakage of albumin and other plasma macromolecules such as low density lipoproteins into the vessel wall that may lead to inflammatory responses and in turn start the atherosclerotic process. Apparent healthy subjects were randomly participate in this study, A fifty (50) subjects chronic smoker subjects were involved in this study and other ninety four (94) ones were considered as control group, statistical analysis for chemical results of creatinine, microalbuminuria (MIA) and calculated ACR (albumin/creatinine ratio) revealed significant different in creatinine in smokers group when compared to non-smokers group, and strongest relationship between creatinine and duration of smoking, and between MIA and ACR with number of smoked of packs per day.

**Keywords:** smoking, Microalbuminuria, albuminuria, creatinine.

### INTRODUCTION

Smoking affects vascular and hormonal systems and is also involved in the development of atherosclerosis, thrombogenesis and vascular occlusion [1]. Chronic smoking adversely influences the prognosis of nephropathies [2] although earlier reports [3] had indicated that smoking may alter renal function, it was not until 1978 that additional information was published clearly indicating that smoking is a renal risk factor. At that time [4], Microalbuminuria is defined as excretion of 30–300 mg of albumin per 24 hours (or 20–200 mcg/min or 30–300 mcg/mg creatinine) on 2 of 3 urine collections [5]. It is noteworthy that smoking increases the urinary albumin concentration, even in a range of albumin concentrations below the level of microalbuminuria [6]. Even in nondiabetic and nonhypertensive individuals, smoking was independently associated with microalbuminuria [7].

Until 2003, the data on the risk of smoking-associated CKD in patients of the general population were scarce. The Multiple Risk Factor Intervention Trial (MRFIT) investigated 332,544 men and documented that smoking was significantly associated with an increased risk for end stage renal disease (ESRD) [8]. Microalbuminuria is caused by glomerular capillary injury and so may be a marker for diffuse

endothelial dysfunction [9]. According to Steno hypothesis, albuminuria might reflect a general vascular dysfunction and leakage of albumin and other plasma macromolecules such as low density lipoproteins into the vessel wall that may lead to inflammatory responses and in turn start the atherosclerotic process [10].

Blood pressure (BP) and heart rate are increased by smoking, which for the major part is due to the action of nicotine [11]. Because increased BP is one of the most important factors promoting progression of CKD, it is likely to play an important role in mediating smoking-induced renal damage. The rise in BP is due to an increase in cardiac output and total peripheral vascular resistance. The BP rise appears immediately and occurs before any increase in circulating catecholamines [12]. Some data implicate an alteration of the diurnal rhythm of BP in smokers (e.g., a lower night/day ratio of systolic and diastolic BP in healthy smokers as compared with nonsmokers [13]. Because alterations of the day/night BP profile do have a notable impact on renal [14] and cardiovascular risk [15]. Besides the aforementioned changes in systemic hemodynamics, smoking alters intrarenal hemodynamics [11]. In brief, the data available led to the hypothesis of increased glomerular pressure induced by smoking as a result of impaired renal autoregulation, at least in patients with renal disease. In healthy

individuals, an increase in renal vascular resistance is observed. This is thought to be “physiologic” and to protect the glomeruli from the increase in systemic BP, resulting in unchanged intraglomerular pressure [16]. The objective of present study was the relationship between renal damage and chronic smoking through parameters can bring a clear vision about kidney status, creatinine and microalbuminuria in different locations of Khartoum state-Sudan.

## MATERIALS AND METHOD

A ninety nine (99) subjects were enrolled in this cross-sectional study, 50 (50.5%) were cigarette smoker males, and the others were non-smokers comprised 49 (49.5%), who considered as control, all of them voluntarily participated. 2.5 ml of blood collected in heparinized container, from each subject, for Creatinine assessment by means of spectrophotometer (Biosystem) its reference rang for Creatinine is 0.6-1.6mg/dl. Every blood sample accompanied with urine sample to detect value of microalbuminuria, by means of Nycocardimmuno chromatography device, its reference range for microalbuminuria is 30-300 mg/l. ACR calculated albumin/creatinine, its reference range  $\geq 2.5$ mg/mmol. Laboratory analysis was performed at the department of clinical chemistry, faculty of medical laboratory sciences, Alneelain University. Statistical analysis was performed by means of statistical package or social science (SPSS) software version 19.

## RESULTS

Blood and urine samples of 99 subjects were tested for Creatinine and microalbuminuria(MIA) respectively, the mean  $\pm$  SD of smoker group of Creatinine, MIA, duration of smoking, number of packs per day, albumin/creatinine ratio (ACR) and age as  $1.05 \pm 0.77$  mg/dl,  $25.19 \pm 65.01$  mg/l,  $19.54 \pm 40.42$  mg/mmol,  $11.13 \pm 8.16$  years and  $2.22 \pm 1.05$  respectively. While mean  $\pm$  SD for control group as  $0.7906 \pm 0.21$  mg/dl for creatinine,  $12.40 \pm 6.25$  mg/l for microalbuminuria and  $17.2 \pm 10.5$  mg/mmol for Albumin/creatinine ratio. Statistical analysis for data obtained revealed a significant difference with creatinine while no difference obtained with MIA, as in table 1.

**Table-1: Independent t- test**

	Smoker Mean $\pm$ SD	Non-smoker Mean $\pm$ SD	P value
creatinine	$1.05 \pm 0.77$	$0.7906 \pm 0.21$	0.026
MIA	$25.19 \pm 65.01$	$12.40 \pm 6.25$	0.167
(ACR)	$19.54 \pm 40.42$	$17.2 \pm 10.5$	0.689

While correlation analysis revealed a relationship between creatinine, microalbuminuria and (ACR) with duration of smoking and number of cigarette packs per day as in table 2 as Pearson's correlation is significant in between 1(+ve correlation) and -1 (-ve correlation), the strongest significance found

between creatinine and duration, while MIA and ACR both have strongest significance with number of cigarette packs per day, as in table 2.

**Table-2: Correlation**

	duration	Number of packs
Creatinine Sig(2-tailed)	0.028	0.86
MIA Sig(2tailed)	.078	0.008
ACR Sig(2tailed)	.642	.004

Correlation is significant at the 0.05 level (2-tailed).

## DISCUSSION

Ninety nine (99) Sudanese subjects were involved in this cross-sectional study, 50.5% were smokers and 45.5% were none-smokers as control. All are enrolled in this study to assess creatinine, microalbuminuria and calculation of albumin/creatinine ratio to find out the effect of smoking habit in there renal function, chemical analysis for targeted parameters showed mean+STD for smokers as  $1.05 \pm 0.77$ mg/dl,  $25.19 \pm 65.01$  mg/land  $19.54 \pm 40.42$ mg/l respectively and for none smoker  $0.7906 \pm 0.21$  mg/dl,  $12.40 \pm 6.25$  mg/mmol and  $17.2 \pm 10.5$  mg/mmol.

4 (8%) of smokers showed increased level of creatinine one of them accompanied with increased level of microalbuminuria. Creatinine showed a significant difference when statistical analysis performed in compare with control group as p value 0.026, while strong relationship obtained when correlation statistical analysis conducted between creatinine and duration of smoking, and between microalbuminuria and albumin/creatinine ratio with number of packs per day.

Generally increased urinary albumin excretion (UAE) has been related to unfavorable cardiovascular outcomes in the general population [17, 18]. Increased UAE could be the consequence of an augmented intraglomerular capillary pressure, it could reflect the existence of intrinsic glomerular damage that causes changes in glomerular barrier filtration, or it could be the consequence of a tubular alteration that impedes the normal reabsorption of filtered albumin. However, it has been suggested that microalbuminuria may represent the renal manifestation of generalized, genetically-conditioned vascular endothelial dysfunction that may underlie the link between an increased UAE and an elevated risk for cardiovascular disease [19, 20]

Renal dysfunction is a common finding in patients with hypertension and is associated with an increased risk for cardiovascular events (CVEs) [21, 22] as well as with progression to ESRD [23]. It has

been pointed out that cardiovascular risk progressively increases as renal function declines and that it is already significantly elevated at the earliest stages of renal damage [24].

Our findings in agreement with study revealed that the effect of smoking irreversible renal damage in men involved in their study [25]. And with agreement with other one assumed that the effects of smoking beyond renal failure to other system breakdown [26].

## CONCLUSION

This study revealed the effects of smoking, even apparent healthy individual may develop several defects that can be irreversible in some point, so good awareness of smoking and its consequences should be considered.

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