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EVALUATION OF ALCOHOLIC CONSUMPTION ON SERUM URIC ACID, UREA, AND CREATININE LEVELS

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ABSTRACT

Background: Several metabolic changes occurs in humans'body duo to alcoholic consumption which may lead to changes in biochemical substance such as uric acid, urea and creatinine. **Objective:** The objective of this study was to evaluate and compare serum uric acid, urea and creatinine in alcoholic and non-alcoholic abuse. **Methodology:** This is descriptive case control study carried in Khartoum state. One hundred (100) apparently healthy males (50 Alcoholic abuse and 50 non- alcoholic abuse) serum uric acid, urea and creatinine were estimated using endpoint and kinetic methods and established mathematical analysis. **Results:** The (mean ±SD in mg/dl) of serum uric acid urea and creatinine in alcoholic abuse respectively were (7.8 ±1.06, 39.5 ±6.9, 1.01 ±0.21) while the (mean ±SD) of serum uric acid, urea and creatininein non-alcoholic abuse respectively were (5.2 ±0.8, 32 ±7.2, 0.8 ±0.2)The results showed that there was significant difference in the uric acid between the two groups (P value < 0,005) there was no significant difference in the urea between the two groups (P Value > 0.005). There was no significant difference in the creatinine between the two groups (P value >0.005). **Conclusion:** The result was concluded that there was significant increase in serum uric acid in alcoholic abuse groups (value 0.00). While urea and creatinineremainedunchanged and no significant difference between alcoholic and non-alcoholic abuse (P value 0.077 and 0.64 respectively).

KEYWORD: serum uric acid, urea and creatininein.

INTEODUCTION

Chronic alcohol consumption is accompanied by various metabolic abnormalities.[1] Alcohol consumption cause direct damage to the kidneys with lead to elevating blood pressure and inducing electrolyte imbalance. Though one study showed that alcohol consumption was significant associated with lower blood urea nitrogen (BUN) and creatinine.[2] However in another study alcohol intake had no effect on glomerular filtration rate (GFR) and renal plasma flow[3]

Alcohol induces hyperuriceamia by several mechanisms, the diuresis and volume depletion induced by alcohol consumption result in decreased glomerular filtration rate (GFR) and increased tubular reabsorption of uric acid. The lactic acidosis that follows alcohol ingestion diminished the renal tubular secretion of uric acid by competitive inhibition between lactic acid and uric acid. Recently it has been shown that the production of uric acid for adenine nucleotide is increased when large amount of alcohol are consumed.[4]

Several studies have indicated that ethanol increase urate excretion and increases de-novo urate formation, probably by the between the two, however leads to hyperuricaemia.[5] The purine content of several alcohol (especially bear contain high amount of guano sire, which is probably the most readily absorbed dietary purine). It was concluded that the hyperuricaemia effect of alcohol mediated by digestion of purine and effect of ethanol on uric acid synthesis.[6] Alcohol can risk health will-being through three intermediate and linked ways, direct biochemical effect, intoxication and episodic heavy drinking[7] alcohol consumption is associated with a number of changes in cell function and the oxidant-antioxidant system, body weight, percent of body fat, body mass index, same hematological parameters are effect and hyparuriceamia are common feature of alcohol ingestion.[8]

The objective of this study was to evaluate and compare uric acid, urea and creatinine in alcohol and non -alcohol abuse.

MATERIAL AND METHODS

Study population

A descriptive case control study was carried in Khartoum in the period from March to May 2015, A group of 100 male involved in the study, 50 heavy alcoholic subjects

and other 50 nonalcoholic healthy subjects aged between 25-70 year.

Including criteria
Apparently healthy alcoholic abuse males.

Excluding criteria
Subject with following illness are excluded from the study (Gout, Known renal diseases, cardiovascular disease and hypertension).

Blood Sample
Venous blood sample (3ml) were collected in heparinized bottles. Sample was centrifuged and plasma separated and stored in plastic tubes of 4^0c. serum urea, uric acid was measured using established method with the mindery Kit, creatinine was measured end point using established method with the mindery kits, then concentration was measured by using BS-200 Chemistry analyzer (Mindery).

Statistical analysis
Statistical analysis was done using excel. Showing the mean and standard deviation.
Permission was obtained p value calculated by independent T test.

RESULTS
In table (1) Comparing between means and standard division of the two groups.Uric acid show significant increase in alcoholic groups when it compared with non-alcoholic groups. P value (0.00).Urea showed no significant difference between alcoholic and non-alcoholic groups P.value (0.077). Also Creatinineshowed no significant difference between alcoholic and non-alcoholic groups. P value (0.647).

Parameter	Alcoholic mean ±SD	Nonalcoholic mean ± SD	P value
Uric acid	7.85 ± 1.06	5.2 ± 0.85	0.00
Urea	39 ± 6.9	32.0±7.2	0.077
creatinine	1.01 ± 0.21	0.82±0.23	0.647

DISCUSSION
There are several change occurs in biochemical parameter in the body such as uric acid, urea and creatinine. Being alcohol induced person leads to potentially increased concentration of uric acid and leading to health problem.[8]

Hyperuricaemia has been observed in alcoholic abused in table (1) statistical analysis reveals significant increase in serum uric acid level in alcoholic abuse subject as compare to non alcoholicgroups P. value (0.00). Similar findings were observed by Faler et al (1982) in their study they found that alcohol intake has been show to increase uric acid production by increasing ATP degradation to AMP uric acid precursor.[9] Puig et al (1984) in their study with similar findings showed that ethanol increase in uric acid production involve acetate conversion to acetyl Co A the metabolism of ethanol.[10]

A number of mechanism have been implicated in the pathogenesis of alcoholic induced hyperuricemia inducing both decrease urate excretion[(11)(12)] and increased production[5][13], the former is via conversion of alcoholic to lactic acid which reduces renal acid excretion by competitively inhibiting uric acid secretion by proximal tubule[14][15][16].

When the alcohol intake exceed heavy, there is definite evidence of impaired renal function as indicated by increased serum creatinine levels. So, hyperuricemia observed in heavy alcoholics can be attribted to1) increased ATPdegradation, 2)Decreased urate excretion via conversion of alcohol to lactic acid ,which reduce renal uric acid excretion by competitively inhibiting uric acid secretion by the proximal tubules[14][5][16]. and 3) to some extent impaired renal function leading to decreased urate excretion.

Alcohol consumption cause direct damage to the kidneys with lead to elevating blood pressure and inducing electrolyte imbalance. Though one study showed that alcohol consumption was significant associated with lower blood urea nitrogen (BUN) and creatinine.[2] However in another study alcohol intake had no effect on glomerular filtration rate (GFR) and renal plasma flow.[3]

The result showed that there was no significant change in urea and creatinine between two groups. Which that means alcoholic abuse has no direct effects in urea and creatinine.

CONCLUSION
The results concluded that there was significant increase in serum uric acid in alcholic abuse group P. value (0.00).

Urea remainedunhorsed and no significant difference between alcholic and non alcholic abuse (P value 0.077).
Creatinine remained unchanged and no significant difference between alcholic and nonalcoholic abuse (P value 0.64).

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