Tikrit Journal of Pure Science
ISSN: 1813 – 1662 (Print) --- E-ISSN: 2415 – 1726 (Online)
Journal Homepage: http://tjps.tu.edu.iq/index.php/j

The effect of hypertension, diabetes, and smoking on the levels of some biochemical parameters in chronic renal failure patients

Noor H. Ali[1], Firas T. Maher[1], Mohammad M. F. Al-Halbosiy[2]
[1] Department of Chemistry, College of Science, University of Tikrit , Tikrit , Iraq
[2] Biotechnology Research Center, Al-Nahrain University , Baghdad , Iraq
DOI: http://dx.doi.org/10.25130/tjps.24.2019.049

ARTICLE INFO.
Article history:
-Received: / / 2017
-Accepted: / / 2017
-Available online: / / 2018
Keywords: hypertension chronic renal failure, diabetes chronic renal failure, Smoking chronic renal failure.
Corresponding Author:
Name: Noor H. Ali
E-mail: allynwr032@gmail.com
Tel:

ABSTRACT

This study has been acomblished between December 2016 and September 2017 which included 70 patients with chronic renal failure who attended at Al-yarmuke teaching hospital in Baghdad with25 healthy individual as control. The research aims to study the effect of hypertension, diabetes and smoking on the levels of some biochemical parameters of chronic renal failure patients comparing with healthy control .The results showed a significant increase in the concentrations of urea, creatine, uric acid, cholesterol, Triglycerides(TG), low density lipoproteins (LDL) and Very low density lipoproteins (VLDL), compared to healthy control. The results also showed that hypertension has an effect on the concentration of urea, uric acid, creatinine, cholesterol, Triglycerides (TG), low density lipoproteins (LDL) and Very low density lipoproteins (VLDL), the results indicated that the diabetes has effects on the concentration of urea, creatinine, cholesterol and Triglycerides (TG), High density lipoproteins (HDL), low density lipoproteins(LDL) and Very Low density lipoproteins (VLDL) with no effects on the concentration of uric acid. There was an effect of smoking on The concentrations of cholesterol and the Triglycerides(TG), low Density Lipoproteins(LDL), Very Low density Lipoproteins (VLDL), urea, uric acid, and creatinine .The gender has no effect on the concentration of all measured parameters except on TG concentration.

1. Introduction

Kidney insufficiency is divided into two categories based on either acute or chronic nature. Chronic insufficiency progresses slowly to an irreversibly decreased renal function, whereas in the acute condition, loss of kidney function is rapid and usually reversible. Kidney disease results in the loss or reduction of functioning nephrons. CKD is defined as the presence of kidney damage, or a decreased level of kidney function. It's usually a result of a complication from another serious medical condition. Unlike acute renal failure, which happens quickly and suddenly, chronic renal failure happens gradually – over a period of weeks, months, or years – as the kidneys slowly stop working, leading to end-stage renal disease (ESRD).[1] The progression is so slow that symptoms usually don't appear until major damage is done. In the United States, approximately 1 in 1,000 people are getting treated for ESRD, and greater than 19 million adults are living with some type of CKD. In Canada, approximately 1.3 to 2.9 million people suffer from CKD. It is manifested by abnormal albumin excretion or decreased kidney function, quantified by measured glomerular filtration rate (GFR), that persists for more than 3 months. Although creatinine clearances can be calculated from urine creatinine concentration measured in a 24-hour urine collection and the office is to estimate from the serum creatinine concentration. One of the complications resulting from diabetes or high blood pressure is the damage to the small blood vessels in the body. The blood vessels in the kidneys also become damaged, resulting in CKD.[2]

Urea is the major nitrogen- containing metabolic product of protein catabolism in human, accounting for more than 85% of the non-protein nitrogen eventually excreted. Urea biosynthesized from deamination of amino acids by hepatic enzymes of urea cycle.[3]

Creatinine is a chemical waste molecule (M.W 113 daltons) that is generated during muscle cells metabolism, through nonenzymatic dehydration of creatine and phosphocreatine. Creatine itself synthesized in the liver and pancreas from three amino acid (Arginine, Glycine and Methionine) and this molecule is a major importance in energy production in muscle. Approximately 2-3% of the body creatine is converted to creatinine every day. Kidneys are responsible for maintaining blood creatinine within the normal range. So creatinine has been found to be a fairly reliable indicator of kidney function, and as kidneys become impaired, creatinine level in the blood is raised. Abnormal high levels creatinine is an indicator of possible malfunction, or failure of both kidneys, sometimes even before a patient reports any symptoms.[4]

Uric acid (UA) is the major product of the catabolism of the purine nucleosides(adenosine and guanosine), the large part of the filtered uric acid reabsorbed in the proximal convoluted tubules followed by secretion of uric acid in the lumen of distal protein of the proximal tubules and further reabsorbed in the distal tubules.[5].

Lipids are hetrogeneous group of compounds which are relatively insoluble in water but dissolve in non-polar organic solvents such as chloroform. The major lipids found in the blood are cholesterol, triacylglycerol, and nonesterified fatty acids. They have many structural, metabolic and nutrient functions in the biological system. Lipids are found in circulation combined with specific carrier protein to form the lipoprotein molecules, in the form of complexes with weak forces to enable exchange of lipid molecules between the plasma and cells. Hyperlipidemia is an independent risk factor. Basic research has provided strong evidence that oxidation of low density lipoprotein (LDL) plays an important role in the pathogenesis of atherosclerosis. Oxidative stress, alterations in lipid metabolism, hyperhomocysteinaemia observed in hemodialysis patients could increase LDL oxidation.[6]

2. Materials and working methods:

A. Samples collection

Blood samples were collected from 70 patients (40 males and 30 females) suffering from chronic renal failure, diabetes and hypertension. Ages of the involved subjects ranged from (18–73) years. The samples from the control group included healthy persons (25 samples: 14 male and 11 female). Samples were collected during the period 11/2016 to 2/2017. Patients ' samples were collected before dialysis.

The samples divided into three groups of patients:

- Group 1:- it included a total of 49 patients suffering from hypertension and chronic renal disability.
- Group 2 :- it included a total of 58 patients suffering from chronic renal and diabetes.
- Group 3:- it included a total of (40) patients smokers (10 males) and (30) non-smokers males.

B. Collection Of Blood Samples

Five milliliters were collected disposable plastic syringes from venous blood for each patient and control (healthy individuals), then 2.5 ml of blood was taken in a plain tube. it was left for 30 to 40 minutes, after which the serum was separated from the part of the centrifuge at (5000) RPM for 5 minutes and the serum was kept at -10 c^{o} .

C. Materials and Methods:

Specific chemicals used in this work are listed in Table (2-1) with their suppliers:

N	Supplier	Chemicals
1	Kit creatinin	AFLO-Italy
2	Kit Urea	AFLO- Italy
3	Kit Ureic aced	AFLO- Italy
4	Kit Cho	AFLO- Italy
5	Kit TG	AFLO- Italy
6	Kit HDL	AFLO- Italy

Determination of LDL-C & VLDL-C: Serum level of LDL-C was calculated according to friedewald formula, which was based on the assumption that VLDL-C was present in serum at a concentration equal to one fifth (or 0.2) of the triglyceride concentration. The formula was only valid at triglyceride concentration of less than 400 mg/dl144 .[7]

VLDL-C = TG/ 5 (in mg/dl) or VLDL= TG/ 2.2 (in mmol/L)

LDL-C = TC – (HDL-C + VLDL-C).

Statistical analysis:

All values ae displayed as (SD ± mean). Statistical analysis System-SAS (2012) has been used to analyze data to examine the impact of transactions on considered qualities. Differences with ($P \leq 0.001$) were considered significance, while $P > 0.05$ = non-significant (N.S) .

Results and discussion

The mean (±SD) of urea concentration in serum of control group (healthy individuals), serum of kidney disease, kidney disease with hypertension and kidney disease with diabetic are illustrated in tables (3-1) . The results indicated a significant increase ($p \leq 0.01$) in the serum levels of urea concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the urea concentration of patients and no significant effect of sex and smoking on level of urea concentration in patients group.

Table (3-1): Mean ±SD of Urea mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±3.44[b] 20.04	±3.29[a] 156.93**	Males	18.33±3.29[b]	Control	Urea mmol/l
±1.86[b] 16. 48	±3.09[a] 152.77**	Female	155.20[**] ±3.24[a]	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension , diabetes
151.00±3.33[b]	163.15**±7.43[a]	Males	159.62*±11.28[b]	P + Hypertension	
139.87±3.60[b]	160.20**±9.40[a]	Female	142.80± 6.79[a]	P+ No Hypertension	
154.35±3.11[a]	P+ No Smoking	164.50±3.02[a]	P+ Smoking	Smoking	

*=P≥0.05 **=P≥0.001 n.s= Non Significant

The increase of urea to the severity and progress of renal failure and the rate of intake of proteins and their rate of impairment is due to dysfunction and lack of kidney function results in the concentration and accumulation of urea in the blood and is concentrated. This increased mean value showed that there was an impairment of renal function either due to reduction of GFR or obstruction that interferes with urinary excretion. Blood urea levels are quite sensitive indicators of renal disease, becoming elevated when renal function drops to around 25-50% of normal. .[8]

The results in table (3-2) indicated a significant increase (p≤0.01) in the serum levels of creatinine concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the urea concentration of patients and no significant effect of sex and smoking on level of creatinine concentration in patients group.

Table (3-2): Mean ±SD of Creatinin mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±0.97[b] 0.74	7.31**±1.90[a]	Males	0.82±0.14[b]	Control	Creatinin (mmol/l)
±0.15[b] 0.9	7.03**±2.16[a]	Female	7.19±1.02[a]	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension , diabetes
7.09±1.99[a]	7.54[n.s]±1.87[a]	Males	9.98**±0.42[b]	P + Hypertension	
6.83±2.48[a]	7.25[n.s]±1.79[a]	Female	6.35±0.70[a]	P+ No Hypertension	
7.29[n.s]±1.85[a]	P+No Smoking	7.44±2.35[a]		P+ Smoking	Smoking

*=P≥0.05 **=P≥0.001 n.s= Non Significant

Our results are in agreement with other researchers , like *Noor Al Amin et al.(2014)* who found that CKD patients have higher serum urea and creatinine levels, leading to various other dangerous diseases. Urea and creatinine are good indicators of a normal functioning of the kidney and increase of the substances in the serum are indications kidney dysfunction.[9]. *Moses and John kennedy* (2013) found that creatinine levels slightly increased due to damage to the kidney and with this; there was reduced glomerular filtration rate due to inflammation of the kidney. .[10]

The results in table (3-3) indicated a significant increase (p≤0.05) in the serum levels of ureic aced concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the ureic aced concentration of patients and no significant effect of sex and smoking on level of creatinine concentration in patients group.

Table (3-1): Mean ±SD of Uric acid mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±0.95[b] 4.79	6.81**±1.82[a]	Males	4.38±0.85[b]	Control	Uric acid
±0.45[b] 3.94	7.98**±1.14[a]	Female	7.26[*] ±0.76[a]	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension, diabetes
6.54 ±1.48[a]	7.09[n.s]±1.23[a]	Males	7.94*±0.69[b]	P + Hypertension	
7.07 ±0.528[a]	8.82[n.s]±2.10[a]	Female	5.78±0.42[a]	P+ No Hypertension	
6.93[n.s]±1.80[a]	P+No Smoking	6.76±1.34[a]	P+Smoking		Smoking

*=P≥0.05 **=P≥0.001 n.s= Non Significant

These results disagrees with the results of other researchers who reported that urate was not elevated or a risk factor for cardiovascular events. [11] However, our result were in agreement with that of *Mehmet Eren et al.(2014)* who found that hyperuricemia in association with various inflammatory diseases, and elevated serum uric acid, has been found to be a graded marker of risk for the development of coronary heart disease, cerebrovascular disease, stroke, and acute renal

failure.[12]. Although uric acid is an antioxidant responsible for 60% of the total antioxidant capacity in humans, the rise in serum uric acid in patients with cardiovascular disease might represent a compensatory mechanism to the increased oxidative stress that occurs in this condition. Epidemiological studies have revealed that uric acid concentrations predict the progression of chronic kidney disease the development of stroke, and a recent meta-analysis reported that uric acid is associated with the presence of hypertension diabetes and metabolic syndrome.[13]

Hypertension and diabetes are the two most important CKD risk factors. Overall, diabetes is prevalent in 44% of the ESRD population. Together, these two disorders constitute 72% of the causes of ESRD. Recently, insulin resistance, obesity, and the metabolic syndrome have been implicated as risk factors. Increased blood pressure, which in turn generates additional damage to the kidneys and additional increases in blood pressure, causes the renal artery stenosis that helps in the occurrence of renal sclerosis nephrosclerosis, which is diagnosed with the presence of fish in the small capillary arteries leading to high creatinine in high pressure patients The blood gets as a result of the regression rate of the nomination glomerular filtration Rate (GFR) .[14],[15]

Kidneys play a pivotal role in blood pressure control through several mechanisms, natriuresis and diuresis, neuro-hormonal factors such as RAAS, and the regulation of sympathetic nervous system activity. Kidneys are one of the organs affected during hypertension, resulting in functional and structural damage with consequent renal dysfunction, in turn inducing an exacerbated hypertension phenotype. Therefore, managing only blood pressure is insufficient to treat hypertension-associated end-organ damage. Hypertension, like anemia, is almost universal in CKD patients and often is the first sign of CKD. The coincidence of CKD and high blood pressure is particularly important because hypertension contributes to the development of cardiovascular disease, the leading cause of morbidity and mortality in CKD patients. Hypertension in CKD patients is mainly the result of an expanded extracellular volume from a salt-rich diet and a decreased capacity for excretion of sodium. As discussed earlier, the normal response to an increase in the extracellular volume is a rise in blood pressure that stimulates sodium excretion to achieve a balance between sodium intake and the excretion of salt.[16]

Another mechanism for hypertension in CKD patients is activation of the renin angiotensin-aldosterone (RAA) system and the sympathetic nervous system. Evidence for activation of the RAA system in CKD patients includes circulating levels of renin and aldosterone that are too high for individuals who are hypertensive, suggesting that the vasoconstrictive action of angiotensin II and the salt retention induced by aldosterone contribute to hypertension. Diabetes is one of the most leading causes of chronic renal failure, causing kidney dysfunction, so that the blood is not filtered properly and becomes scarcer and implemented. Blood proteins and other components of blood serum that should be kept in blood and allowed to go down with the generation and keep the materials you have to dispose of. Like a cricket. This leads to chronic renal failure of diabetics. [17] ,[22]

The results in table (3-4) indicated a significant increase ($p \leq 0.01$) in the serum levels of cholesterol concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the cholesterol concentration of patients and no significant effect of gender and smoking in patients group.

Table (3-4): Mean ±SD of cholesterol mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±1.23^{b} 155.31	245.39**±4.06^{a}	Males	152.52±11.35^{b}	Control	Cho (mmol/l)
±2.46^{b} 149.50	278.05±2.70^{a}	Female	259.20**±10.34^{a}	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension , diabetes
222.14±7.93^{c}	269.60**±4.85^{a}	Males	240.71±19.60^{b}	P + Hypertension	
217.20±9.20^{c}	**±8.52^{b} 347.60	Female	279.98±11.80^{a*}	P+ No Hypertension	
230.80±3.76^{a}	P+No Smoking	290.74**±5.62^{b}	P+ Smoking	Smoking	

*=P≥0.05 **=P≥0.001 n.s= Non Significant

The results in table (3-5) indicated a significant increase ($p \leq 0.01$) in the serum levels of triglycerides concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the triglycerides and no significant effect of gender and smoking in patients group.

Table (3-5): Mean ±SD of TG mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±2.39[b] 61.23	160.88±3.09[a]	Males	60.4±2.2[b]	Control	TG (mmol/l)
±2.96[b] 59.42	191.3±3.33[a]	Female	173.70**±6.58[a]	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension , diabetes
131.67±2.40[c]	**±9.30[a] 191.60	Males	135.36±17.84[b]	P + Hypertension	
142.69±7.38[c]	**±9.54[b] 246.90	Female	198.28±10.74[a**]	P+ No Hypertension	
149.61±4.17[a]	P+No Smoking	195.81*±5.19[b]	P+ Smoking	Smoking	

*=P≥0.05 **=P≥0.001 n.s= Non Significant

The results in table (3-6) indicated a significant decrease (p≤0.01) in the serum levels of HDL concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the HDL and no significant effect of gender and smoking in patients group.

Table (3-6): Mean ±SD of Urea mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±3.28[b] 44.15	18.46±2.19[a]	Males	41.16±3.09[b]	Control	HDL(mmol/l)
±3.78[b] 37.92	21.33±2.88[c]	Female	19.67**±2.57[a]	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension , diabetes
20.38±7.13[b]	16.45**±6.85[a]	Males	23.96±2.01[b]	P + Hypertension	
27.06±8.53[c]	14.77**±8.54[a]	Female	16.16±1.21[a**]	P+ No Hypertension	
19.26[n.s]±2.35[a]	P+No Smoking	16.00±2.36[a]	P+ Smoking	Smoking	

*=P≥0.05 **=P≥0.001 n.s= Non Significant

The results in table (3-7) indicated a significant decrease (p≤0.01) in the serum levels of LDL concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the LDL and no significant effect of gender and smoking in patients group.

Table (3-7): Mean ±SD of Urea mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±4.11[b] 98.91	194.73±4.25[a]	Males	99.31±4.00[b]	Control	LDL(mmol/l
±4.58[b] 99.75	218.95±5.9[a]	Female	204.90**±5.78[a]	Patients	
SD± Mean P+NoSugar	SD± Sugar Mean P+	Gender	SD± Mean	Item	Hypertension , diabetes
284.40±7.7[c]	214.99**±8.37[a]	Males	190.57±18.28[b]	P + Hypertension	
161.60±9.30[c]	175.43**±4.43[b]	Female	224.06±11.01[a*]	P+ No Hypertension	
181.88±5.28[a]	P+No Smoking	234.50*±4.10[b]	P+ Smoking	Smoking	

*=P≥0.05 **=P≥0.001 n.s= Non Significant

The results in table (3-7) indicated a significant decrease (p≤0.01) in the serum levels of VLDL concentration in serum patients chronic renal failure compared to the control group (healthy individuals), and a significant effect of pressure and sugar on the VLDL and no significant effect of gender and smoking in patients group.

Table (3-8): Mean ±SD of Urea mmol/l centration in studied groups.

SD± Mean Control	SD± Mean Patients	gender	SD± Mean		Variable
±1.28[b] 12.25	32.18±1.62[a]	Males	12.07±2.25[b]	Control	VLDL(mmol/l)
±1.39[b] 11.88	38.26±1.65[a]	Female	34.74**±3.72[a]	Patients	
SD± Mean P+ No diabetes	SD± Mean P + diabetes	Gender	SD± Mean		Hypertension , diabetes
26.33±4.28[c]	38.31**±8.54[a]	Males	27.07±3.56[b]	P + Hypertension	
28.54±5.48[c]	49.37**±8.29[b]	Female	39.65±2.14[a**]	P+ No Hypertension	
29.92±2.83[a]	P+No Smoking	39.16*±3.01[b]	P+ Smoking	Smoking	

*=P≥0.05 **=P≥0.001 n.s= Non Significant

Dyslipidemia is a common complication of progressive kidney disease, which is characterized by high triglyceride and low high-density lipoprotein (HDL) cholesterol levels, accumulation of remnant particles, a predominance of small dense low-density lipoprotein (LDL) particles, and increased levels of lipoprotein A. In patients with advanced chronic kidney disease (CKD), LDL and HDL particles undergo oxidative modification, resulting in the formation of small lipoproteins and enhanced production of oxidized LDL.[23] ,[24]

The evidence for dyslipidemia as an independent risk factor for renal disease development and/or progression is not as strong in clinical human studies as it is in experimental studies. [25]

These studies indicate an association between hyperlipidemia and renal disease development and/or progression, suggesting that hyperlipidemia is a risk factor for renal disease, although no definitive conclusions can be drawn. It is also not clear whether dyslipidemia increases the renal risk in those without other risk factors for kidney disease, because most studies that have evaluated the effects of dyslipidemia on renal disease have been performed with patients with pre-existing renal disease or other risk factors for renal disease, such as hypertension and diabetes. The risk of loss of renal function associated with dyslipidemia seems to be highest in those with moderate-to-severe renal disease and other renal risk factors, such as hypertension and diabetes. Another aspect that remains unanswered is which lipoprotein or lipoproteins better predict renal disease development and/or progression, because the data are inconsistent regarding the ability of cholesterol (or its fractions) and/or triglycerides to predict the progression of renal disease.[26] Finally, in some of these studies, the possible role of unmeasured confounders, such as the metabolic syndrome, cannot be ruled out. In fact, the metabolic syndrome, which is associated with high triglyceride and low HDL cholesterol levels, has been recently associated with the development of CKD during follow-up in the Atherosclerosis Risk in Communities study.[27].

HDL levels are inversely associated with cardiovascular risk both in the general population and in patients with chronic kidney disease (CKD). Although HDL levels may in part be genetically determined, both insulin resistance and CKD are associated with low HDL levels. As glomerular filtration rate declines, insulin sensitivity decreases, providing the possibility that insulin resistance may contribute to dyslipidemia in CKD.[28]

Apo AI, which is the activator of lecithin cholesterol acyl transferase (LACT), is reduced in CKD due to down regulation of hepatic apo AI genes leads to decline in the activity of LACT, which causes reduced cholesterol esterification and impairment of HDL maturation. The activity of LACT is consistently diminished in CKD, so there is decrease in HDL levels.[29]

Both conditions are also associated with a shift in the size distribution of LDL to increased content of small dense LDL. This similarity may be confounded by the known associations between diabetes and hypertension and the subsequent development of renal failure. Indeed low apo A I levels are a risk factor for loss of renal function,14 and may simply report an abnormality that leads both to renal failure and imparts cardiovascular risk. The question is further confounded by the facts that aspects associated with insulin resistance, type II diabetes, hypertension, adiposity, are all associated with the development of renal failure leading to the possibility of selection bias within a population of patients with CKD.[29,[32]

It is well known that patients with impaired renal function exhibit significant alterations in lipoprotein metabolism, which in their most advanced form may result in the development of severe dyslipidaemia[33]. The an increase in VLDL cholesterol and their remnants in CKD are mainly due to their reduced clearance as well as insulin resistance driven overproduction of VLDL [25].

References

[1] Mannangi, N. and Jayasree, S. (2014). Lipoprotein (a) & Lipid profile in chronic kidney disease. Case control study, *Web med Central biochemistry*, **5(2):** WMC004568.

[2] Katrin, U. et al. (2005). Factors Associated With Lipoporotein (a) in Chronic Kidney Disease. *American Journal of Kidney Diseases*, **45:** 28-38.

[3] Azra, K. (2014). Estimation of blood urea (BUN) and serum creatinine level in patient of renal disorder. *Indian Journal of Fundamental and Applied Life Sciences ISSN.*, **4(4)**.

[4] Dwinnell, B.G. and Anderson, R.J. (2012). Diagnostic Evaluation of the Patient with Acute Renal Failure, Atlas of Diseases of the Kidney. ISN Informatics Commission and NKF cyber Nephrology.

[5] Zoccali, C. and Mallamaci, F. (2013). Uric Acid, hypertension, and cardiovascular & renal complications. *Curr Hypertens Rep.*, **15(6):** 531-7.

[6] Nitin, S.N. and Jayshree, V.G. (2011). Lipid Profile and Serum Paraoxonase1 Activity in CRF Patients Pre and Posthemodialysis, *Al Ameen Journal Med Sci.*, **4(1):** 61-68.

[7] Warnick, G.R.; Knopp, R.H.; Virginia, F. and Lance, B. (1990). Estimating low– density lipoprotein cholesterol by Friedewald equation is adequate for classifying patients on basis of nationally recommended cut points, *Clin Chem.,* **36(1):** 15-19.

[8] Sharma, A.; Hirulkar, NB.; Wadel, P. and Das, P. (2011). Influence of Hyperglycemia on Renal Function Parameters in Patients with Diabetes Mellitus. *International Journal of Pharmaceutical & Biological Archives*.

[9] Noor, U.A.; Raja, T.M.; Javaid, M.A.; Mudassar, Z. and Asad, M.R. (2014). Evaluating Urea and Creatinine Levels in Chronic Renal Failure Pre and Post Dialysis: A Prospective Study. *Journal of cardiovascular disease*, **2(2)**.
[10] Moses, O. and Johnkennedy, N. (2013). The Alteration of Serum Glucose Urea and Creatinine Level of Malaria Patients in Obowo Local Government Area of Imo State Nigeria. International Journal of Advanced Medicine, **(1):** 1-6.
[11] Christopher, E. and Ernest, A. (2014). Abnormal serum uric acid levels in health and disease: A double-edged sword. *American Journal of Internal Medicine*, **2(6):** 113-130.
[12] David, G. and Robert, U. (2013). The pathophysiology of hyperuricaemia and its possible relationship to cardiovascular disease, morbidity and mortality, *BMC Nephrology*, **14:** 164.
[13] Mehmet, E. (2014). The association between serum uric acid level and heart failure and mortality in the early period of STEMI. *Turkish Society of Cardiology*, **42(6):** 509-510.
[14] Guyton, A.C. et al. (1996). The dominant role of the Kidneys in long-term Arterial pressure regulation in normal and hypertensive states. In Laragh J. H., and Brenner, B.M.: Pathophysiology, Diagnosis and Management of hypertension. New York, Raven Press: p921.
[15] Prospective studies collaboration, (2002), "Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies", Lancet, **360:** p1903-1913.
[16] Lubanski, M.S.; McCullough, P.A., (2009). Kidney's role in hypertension. Minerva Cardioangiol. **(57):**743–759.
[17] Mennuni, S. et al. (2014). Hypertension and kidneys: Unraveling complex molecular mechanisms underlying hypertensive renal damage. *Journal Hum. Hypertens*, **(28):**74–79.
[18] Kurts, C.; Panzer, U.; Anders, H.J. and Rees, A.J. (2013). The immune system and kidney disease: Basic concepts and clinical implications. *Nat. Rev. Immunol*, **(13):**738–753.
[19] Watatnabe, S.; Kang, D.; Heng, L.; Nakagawa, T. and Kanellis, J. (2002). Uric acid, hominoid. Evaluation, and the pathogenesis of Sahsensitivity, *hypertension*, **(40):**355-360.
[20] Ansari, A.G. (2003). Hypertensive kidney disease. *JAMA*, **13(2):**110.
[21] Kik, k.R.; Koya, D. and Haneda, M. (2003), Progression of Diabetic nephropathy. *AM.J. kidney Disease*, **(41):**21.
[22] Rajapurkar, M. and Dabhi, M. (2010). Burden of disease-prevalence and incidence of renal disease in India. *Clinical Nephropathy*, **(74):**9-12.
[23] Stefanovic, V. and Milojkovic, M. (2004). Treatment of dyslipidemia in chronic kidney disease. *Int Journal Artif Organs*, **(27):**821–827.
[24] Moorehead, J.F.; Chan, M.K.; EL-nahas, M. and Varghese, Z. (1982). Lipid nephrotoxicity in chronic progressive glomerular and tubulointerstitial disease. *Lancet* **(II):**1309–1311.
[25] Aleix, C. and Elisabet, C. (2005). Dyslipidemia and the progression of renal disease in chronic renal failure patients, *Kidney International*, **68, Supplement (99):** S87–S93.
[26] Crook, E.D. et al. (2003). Lipid abnormalities and renal disease: Is dyslipidemia a predictor of progression of renal disease. *Am Journal Med Sci.*, **(325):**340–348.
[27] Kurella, M.; LO, JC. and Chertow, GM. (2005). Metabolic syndrome and the risk for chronic kidney disease among nondiabetic adults. *Journal Am Soc. Nephrol*, **(16):**2134–2140.
[28] Vaziri, N.D. and Moradi. H. (2006). Mechanisms of Dyslipidemia of chronic renal failure. *Hemodialysis International*, **(10):**1–7.
[29] Vaziri, ND.; Liang, K. and Parks, JS.; (2001). Down regulation of hepatic lecithin: cholesterol acyltransferase gene expression in chronic renal failure. *Kidney Int*, **(59):**2192-6.
[30] Vaziri, N.D. (2006). Dyslipidemia of chronic renal failure: the nature, mechanisms, and potential consequences. *Am Journal Physiol Renal Physiol*, **(290):**F262–F272.
[31] Lokesh, R.M.S.; Anwar M.M. and Sandhya A.S. (June. 2016). Study of Lipid Profile in Chronic Renal Failure Patients Undergoing Hemodialysis, *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, **15(6) Ver. II:** 01-03.
[32] Rao, A.M.; Bitla, A.R.; Reddy, E.P.; Sivakumar, V. and Srinivasa, R.P. (2010). Lipid abnormalities, lipoprotein (a) and apoprotein pattern in non-dialyzed patients with chronic kidney disease, *Indian Journal of Clinical Biochemistry*, **25(1):**47-50.
[33] Bhagwat, R.; Joshi, SP.; Satgia, P.; Rajesh, B. and Pradeep, S. (1997). Lipid abnormalities in CRF. *Ind. Journal Clin. Biochem.*, **12(1):**81-5.

تأثير الضغط والسكر والتدخين على مستوى بعض المتغيرات الكيموحيوية في مرضى الفشل الكلوي المزمن

نور حسن علي[1] ، فراس طاهر ماهر[1] ، محمد محمود فرحان الحلبوسي [2]

[1] قسم الكيمياء ، كلية العلوم ، جامعة تكريت ، تكريت ، العراق

[2] مركز بحوث التقنيات الاحيائية ، جامعة النهرين ، بغداد ، العراق

الملخص

اجريت الدراسة للفترة من بداية كانون الاول 2016 الى نهاية ايلول 2017 شملت الدراسة 70 مريض من الذين يعانون الفشل الكلوي المزمن من اللذين يراجعون مستشفى اليرموك التعليمي في محافظة بغداد. اضافة الى 25 من الاشخاص الاصحاء ظاهريا. هدفت الدراسة الى معرفة تأثير الضغط والسكر والتدخين على مستوى تركيز وفعالية بعض المتغيرات الكيموحيوية لمرضى الفشل الكلوي المزمن ومقارنتها مع الاصحاء. بينت النتائج وجود ارتفاع معنوي في مستوى تركيز كل من (اليوريا والكرياتينين وحامض اليوريك والكولسترول وثلاثي الكليسيرايد والبروتينات الدهنية واطئة الكثافة والبروتينات الدهنية واطئة الكثافة جداً عند مقارنتهم مع الاصحاء، كما بينت النتائج ان للضغط تأثير على مستوى تركيز كل من اليوريا وحامض اليوريك والكرياتينين والكولسترول والبروتينات الدهنية عالية الكثافة والبروتينات الدهنية واطئة الكثافة والبروتينات الدهنية واطئة الكثافة جداً مع عدم وجود تأثير للضغط على مستوى تركيز ثلاثي الكليسيرات، كما بينت النتائج ان مرض السكر له تأثير على مستوى تركيز كل من اليوريا والكرياتينين والكولسترول وثلاثي الكليسيرايد والبروتينات الدهنية واطئة الكثافة والبروتينات الدهنية واطئة الكثافة وعدم وجود تأثير لمرض السكر على مستوى تركيز حامض اليوريك، كما بينت النتائج ان للتدخين تاثير على مستوى تركيز كل من الكولسترول وثلاثي الكليسيرايد والبروتينات الدهنية واطئة الكثافة والبروتينات الدهنية واطئة الكثافة وعدم وجود تاثير على مستوى تركيز كل من اليوريا وحامض اليوريك والكرياتينين والبروتينات الدهنية عالية الكثافة كما بينت النتائج ان ليس للجنس تأثير على مستوى تركيز جميع المتغيرات المقاسة.