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A study of some biochemical parameters in blood serum of patients with chronic renal failure

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Abstract

This study was carried out on 55 patients(32males,23 females) with chronic renal failure aged (15-75)year admittedto al-dialysis unit in Kirkuk general hospital which compared with 35 healthy subjects as control group .The aim of this study is to investigate some biochemical parameters included urea ,creatinine ,uric acid, total protein ,triglycerides, total calcium ,inorganic phosphorus ,magnesium ,iron ,total iron binding capacity and ferritin in the serum of patients with chronic renal failure (CRF)before and after haemodialysis and to detect body mass index(BMI) and serum glucose in patientsbefore dialysisonly .

The results showed that there was significant increase($p<0.05$) in the serum urea, creatinine ,uric acid ,triglyceride, magnesium and ferritin while revealed significant decrease in the levels oftotal protein, iron and total iron binding capacity inpatients before dialysis compared to the control group.Non-significant increase($p>0.05$) is in serum glucose, phosphorus and body mass index in patients undergoing haemodialysis which was compared to the control group .There was non-significant decrease in the calcium in serum pre-dialysis patients than those in the control group.

In comparative study between pre and post haemodialysiscases,the serum urea,creatinine ,uric acid ,triglyceride, inorganic phosphorus and magnesium level showed significant decrease ($p<0.05$)in post dialysis compared to pre-dialysis ,while there was significant increase ($p<0.01$)in the serum total protein ,calcium ,iron ,total iron binding capacity and ferritin in post dialysis compared to pre dialysis .

Nosignificant changes($p>0.05$) were observed in the levels of magnesium ,iron, total iron binding capacity and total protein whereas significant increase($p<0.01$) observed in the levels of urea ,creatinine ,uric acid , triglyceride, inorganic phosphorus and ferritin in post dialysis compared with blood serum of normal individuals.There was significant decrease in serum calcium in post dialysis when compared with thecontrol group.

Keywords :chronic renal failure ,haemdialysis, elements

1-Introduction

Chronic renal failure (CRF) also called chronic kidney failure, chronic renal insufficiency, or uremia is a slowly progressive loss of renal function over a period of months or year and defined as an abnormally low glomerular filtration rates(GFR)[1]. CRF that leads to severe illness and requires some form of renal replacement therapy such as dialysis is called end-stage renal disease[2].

CRF occurs in 1.0 of every 5000 people, usually in middle-aged and older people, although children and pregnant women are also susceptible. CRF may be irreversible, and eventually leads to total kidney failure[3].Many people are unaware of the problem until more than 70% of kidney function has been lost [4].

When healthy, the kidney removes waste products and fluids from the blood stream and excreting them in the urine, also the kidneys maintain the body's internal equilibrium of water and minerals (sodium, potassium, chloride, calcium, phosphorus, magnesium and sulfate) . Dialysis may be used for very sick patients who have suddenly lost their kidney function (acute renal failure) or for quite stable patients who have permanently lost their kidney function (end stage renal failure)[5].

Dialysis is a procedure that removes excess fluids and toxic end products of metabolism such as urea from the plasma and corrects electrolytes balance by dialyzing the patient's blood against fluid containing no urea but has levels of minerals like potassium and calcium that are similar to their naturalconcentration in healthy blood[6].

In medicine, dialysis is a type of renal replacement therapy which is used to provide an artificial replacement for lost kidney function due to renal failure. It is a life support treatment and does not treat any kidney diseases[3].

CRF produces a number of abnormalities of calcium and phosphorus metabolism [7].Several studies have revealed irondeficiency as avery common cause of anemia in pre-dialysispatient,even assessed by reduced iron staining in the bone marrow [8].

Serum ferritin levels are measured in patients as part of the iron studies workup for anemia. The ferritin levels measured have a direct correlation with the total amount of iron stored in the body including cases of anemia of chronic disease. If ferritin is high, there is iron in excess [9].

The aim of this study is to investigate some biochemical changes inserumconsidering urea,creatinine,uric acid,total protein ,triglyceride, calcium, inorganic phosphorus, magnesium ,iron,total iron binding capacity and ferritin inpatients with CRF before and after dialysis,and to detect body mass index and serum glucose in patients undergoing haemodialysis only in patients before dialysis then compared them with the control group.

2-Material and Methods

2-1 Subjects and Samples

The collection of samples was conducted during theperiodfromSeptember 2010 to May 2011.From 55 patients entered the artificial Kidney Unit in Kirkuk General Hospital in Kirkuk Governorate.The cases were diagnosed as renal failure for both sex based on the history, clinical examination and taking renalfunction tests.Blood serum of (55)patients with chronic renal failure (CRF) undergoing hemodialysis.32 of them were males and 23 were femalesand their age ranges between 15-75 years .Blood samples from (35) healthy control subjects were obtained for comparison,their ages ranged from 20to 70 years .The weight and length were measured for both groups to determine body mass index(BMI).BMI can

be calculated according the following equation [50].

BMI = Weight in kg/ [height in (m)]

Venous blood (10 ml) were drawn from each fasting patient(8-12 hours fasting) ,5ml before dialysis and 5ml after dialysis . Serum then was separated by centrifugation for (10) minutes, and then divided in aliquots each subject's serum was frozen at (-20 °C) before analysis to assess some biochemical tests.

2-2- Methods

Table (1):Methods used for determination of some biochemical parameters in blood serum.

Parameters measured in serum	Methods used	References
Uric acid	Uricase method	Fossati, etal.,[10]
Creatinine	Colorimetric reaction	jaffe[11]
Urea	enzymatic and colorimetric method	Tietz [12]
glucose	enzymatic method	Tietz[13]
Total protein	Colorimetricbiuret method	Gornal etal.,[14]
Triglyceride	Trinder reaction	Fossati and Prencipe[15]
Inorganic phosphorus	Daly and modified by Gamst and Try	Tietz [12]
Calcium	O-cresol phathalinecomplexone) method	Tietz [12]
magnesium	Ginder ,Heth and Khayam-Bashi method	Ginder and Heth[16].
Iron	colorimetric direct methods	Douglaset al.,[17]
Total iron binding capacity	colorimetric methods	Tietz[18]
Ferritin	Enzyme Linked Florescent Assay (ELFA)	Verentetal., [19]

2-3 Statistical analysis

(Excel) programswas used toanalyze data, using student unpaired and paired T-test , Chi-square test Regression and Correlation analysis. P value of less than 0.05 was considered significant.

3-Results and Discussion

The results showed that the enrolled patients were distributed according to gender.Table(2) patients were divided into two groups , the malegroup forming (58.2%) of them wasfound to be higher than the female group(41.8%) but, statistical analysis showed no significant difference between them.This study was incompatible withKadhim [20]but in accordance with that reported in western studies ,inwhich annual incidence was twice as high in males than in females [21].

Table-2: Association between chronic renal failure and gender.

Gender	no. of patients	%
Male	32	58.2
Female	23	41.8
Total	55	100%

P> 0.05(Chi-square=1.47)

In table (3) the age distribution in renal failure showed a peak level inthe age between (55-65) years represented by 15 patients and theleast was in the age group between (15-25) years represented by 4 patients.This study wascompared with the other studies in westerncountries in which peak incidence is older than 75 years[21] and compatible with local earlier study [20].Our explanation for this ,was due to the most of Iraqi people sufferfrom diabetes mellitusorhypertension (which are the mostcommon cause of chronic renal failure).They have either poorcompliance to the drugs or under controlled therapy. Limitedmedical services and delay of medical consultation may be anothercause for such incidence .

Table -3:The age distribution of chronic renal failure patients .

Age/year	no. of patients	%
15-25	4	7.3
26-35	7	12.7
36-45	13	23.6
46-55	10	18.2
56-65	15	27.3
66-75	6	10.9
Total	55	100

P< 0.05(Chi-square=9.89)

In table (4) the relation between CRF and BMI indicated an increase in BMI with 25.1 in patientswith CRF compared to the control group22.7.This study showed no significant difference between BMI and CRF.This may be explained that from measurement of BMI was at the time patients suffered weight loss as a consequence of morbidity related to CRF itself.

Ejerblad*etal*. showed that obesity was independently associated with increased risks for all major types of CRF , that is obesity entails an extra burden on the nephrons, which promotes the progression of renal failure[22].Obese individuals, compared with lean, are at higher risk for developing proteinuria and CRF after unilateral nephrectomy [23]. This supports the view that the coexistence of obesity and reduced number of functioning nephrons increases risk for CRF). Suggested contributing factors include hyperlipidemia, hyperleptinemia, a state of low-grade inflammation, hyperfiltration caused by insulin resistance, increased sympathetic activity, and activated renin-angiotensin system [24,25].

Increase inserum glucose in CRF compared with control group may depend on individuals with diabetes ,have increased chance of developing kidney disease. Diabetes causes the body to be unable to properly control the metabolism of glucose from food sources such as carbohydrates. If left untreated, a glucose buildup in the bloodstream can lead to severe degradation of kidney function. The filtering units of

the kidney are filled with tiny blood vessels. Over time, high sugar levels in the blood can cause these vessels to become narrow and clogged .Without enough blood, the kidneys become damaged and albumin (a type of protein) passes through these filters and ends up in the urine but this increase was not significant.

Table -4:Relationship between chronic renal failure andserum glucose ,BMI

Parameters	Patient n=55 (Mean ± SD)	Control n=35 (Mean ± SD)
BMI (Kg/m^2)	25.1±4.3	22.7±2.7
Serum glucose(mmol/L)	6.5±2.8	4.76±1.03

Table (5) showed highly significant increase ($p<0.01$) in the serum levels of urea ,creatinine in pre and post dialysis patients with CRF compared with those of normal individuals and highly significant decreasein the levels of these parameters in post dialysis patients compared with predialysis patients with CRF($p<0.01$).
In CRF the increase of serum urea is proportional to the progression of the disease,but it is highly influenced by a catabolic state or an excessive protein ingestion,leading to ahigher production of other waste substances of protein catabolism [26]while the increase in creatinine level in the serum of patients with CRF is attributed to the decrease in the number of functioning nephrons, which would reduce the GFR,which causes major decrease in renal excretion of water and solutes[27].

The decrease in the level of urea,creatinine in post dialysis patients compared with predialysis patients with CRF is due to thathaemodialysiswhich removes toxins from the blood by aclosed – loop process where the blood of the patient and is continuously being withdrawn,dialyzed,and returned to the patient. These findings also supported by other studies[28,29].

Significant increase in serum uric acid appeared before dialysis is compared to post dialysis and control the group($p<0.01$)because patients with chronic kidney disease (CKD) often manifest endothelial dysfunction, which is usually defined as a defect in endothelial nitric oxide (NO) bioavailability . At the same time, endothelial dysfunction has emerged as an important risk factor for the progression of kidney disease[30].

The endothelial dysfunction was independently associated with reduced eGFR that leads to the retention of substances such as uric acid and asymmetric dimethylarginine, the latter was considered as a circulating uremic toxin that competes with L-arginine for endothelial NO synthase[31].This suggests that uric acid might affect endothelial function distinct from the effect of reduced GFR. This study wasin accordance with Al-Rubae*et al*.[32].

Plasma triglyceride concentration was elevated significantly inpatients with CRF compared to post dialysis and control group. This Elevation is accompanied by increasedplasma concentration and impaired clearance of VLDL-C, which isassociated with the accumulation of

atherogenic VLDL-C remnants,commonly known as IDL-C [33] and[34]were originally found bothincreased synthesis and decreased clearance of very low densitylipoprotein (VLDL-C), in uremic patients as a cause ofhypertriglyceridemia. In earlier study it has been found that down regulation of skeletal muscle and adipose tissue LPL,hepatic lipase, VLDL receptor and hepatic LRP iscollectively were responsible for hypertriglyceridemia, impairedclearance, and elevated plasma levels of VLDL, IDL, andchylomicron remnants[35].Other studies [20]recorded the same results.

The concentration of calcium remains low after post hemodialysis in CRF patients when compared with the control groups but higher than the pre hemodialysis stage while the concentration of phosphorus becomes low in post dialysis compared with predialysis and the control group. Intestinal calcium absorption appears to be decreased in renal failure[36]. Fractional absorption of calcium is inversely related with plasma concentration of blood urea nitrogen[36]. Patients with chronic renal failure tend to ingest less calcium in their diets than normal subjects.This result is compatible with [32]. While increase serum phosphorus before dialysis due to the inorganic phosphate in the plasma is filtered in therenal glomeruli and 85 - 90% of the filtered load isreabsorbed by active transport in the proximal tubule. Thisactive transport is powerfully inhibited by parathyroidhormone. As GFR falls and phosphorus retention was increasedue to decreased excretion, serum phosphorus levels rise.Serious changes are apparent when the GFR falls to < 30ml/min[37]. Our study was compatible with [38].

In table (5) the results revealed significant decrease in the serum total protein in pre dialysis patients with CRF compared to post dialysis . This may be attributed to the either changes in the structure of basement membrane of glomeruli which consequently leads to the leakage of albumin and some low molecular weight proteins or restriction protein intake[39] and protein malnutrition[40].This was compatible with [32]who recorded significant decrease in total protein predialysis patients compared to the control group .

Significant increase in the serum magnesium before dialysis compared with post dialysis and control group because renal excretion is the major route of magnesium elimination from the body and a positive magnesium balance would be expected under conditions of renal insufficiency. However, a compensatory decrease in tubular reabsorption is operating to maintain an adequate urinary magnesium excretion even when glomerular filtration rates are very low. The limited ability of the kidney to excrete an increased magnesium load may result in toxic concentrations of the ion in serum[41] .Our study is incompatible with [42] as it recorded a decrease in serum magnesium in patient with CRF compared to the control group.

The results recorded in table (6) shows that there was a significant increased in post dialysis compared with pre dialysis patients in iron and TIBC concentration . Iron and TIBC concentration was found to be significantly decreased ($p<0.001$) in serum of pre and post hemodialysis patients compared with the control group as shown in Table (5). The concentration of iron and TIBC was about (13.24,18.2 µmol/L and 39.7,54.88 µmol/L) respectively compared with the control group. These results were conformable to other results which are recorded [32,43,44,45].

In chronic renal failure, anemia can be a result of any of the mechanisms

“anemia of chronic renal insufficiency” is a result of a decreased production of red blood cells by the bone marrow .This defect in red blood cell production is largely explained by the inability of the failing kidneys to secrete erythropoietinhormone. This hormone is a necessary stimulus for normal bone marrow to produce red blood cells. In addition, other factors associated with renal failure, including the accumulation of the so-called uremic toxins, may play a role in depressing bone marrow function. Excess stores of aluminum may accumulate in the bone marrow of long term dialysis patients and can contribute to anemia as well[46].

The effectiveness of dialysis in reversing any complication of uremia depends on the nature of that complication. Those disturbances which are due to the accumulation of a uremic toxin may be reversible if that toxin is dialyzable and if the removal rate by dialysis outstrips its generation rate. Some improvement in red blood cells production is seen with the initiation of dialysis, probably by decreasing the toxic effect of uremia on the bone marrow. Dialysis, however, does not replace the hormone produce on functions of the kidney and therefore does not by itself correct the main cause of anemia, namely deficient production of erythropoietin [45].

The concentration of ferritin increased in pre and post hemodialysis in CRF patients when compared with control groups and increased in post hemodialysis when compared with pre hemodialysis stage. This result is compatible with[47].

In anemia of chronic disease , ferritin levels are normal or high, reflecting the fact that iron is stored within cells, and ferritin is being produced as an acute phase reactant but the cells are not releasing their iron. Iron is not utilized because there is less erythropoiesis (formation of red blood cells & hemoglobin). Thus, to store the unused iron, ferritin protein that binds this iron is produced in increased amount[48]. As well as in response to the inflammatory cytokines, increasingly IL-6, the liver produces increased amounts of hepcidin. Hepcidin in turn causes increased internalisation of ferroprotin molecules on cell membranes which prevents its release from iron stores. Inflammatory cytokines also appear to affect other important elements of iron metabolism, including decreasing ferroprotein expression, and probably directly blunting erythropoiesis by decreasing the ability of the bone marrow to respond to erythropoietin[49].

Table -5: Concentration of the parameters in bloodserum of hemodialysis patients and control subjects.

Mean ± SE			
Parameters	**Pre hemodialysis patients n = 55**	**Post hemodialysis patients n = 55**	**Control n=35**
Urea(mmol/L)	20.6±7.15***	8.23±3.27**	5.38±1.67
Creatinine (µmol/L)	246.28±34.78***	103.71±11.67**	93.52±9.5
Uric acid (*µmol/L*)	347±68.7***	255.29±40.9**	219.8±53.5
Triglyceride(mmol/L)	1.68 ±0.38**	0.7 ±0.2**	1±0.26
Protein(g/dl)	5.69 ±0.73**	7.07±2.2	7.16 ± 0.56
Calcium (mmol/L)	1.44 ±0.46	1.6±0.35**	2.33 ±0.11
Phosphorus(mmol/L)	2.1±0.05	1.47± 0.4**	1.12± 0.17
Magnesium(mmol/L)	1.58 ± 0.5**	1.24 ±1.01**	0.8±0.12
Iron(µmol/L)	13. 24 ±4.5**	18.28±2.9	22.38±5.4
TIBC(µmol/L)	39.7 ±13.6**	54.88±8.8	67± 16.4
Ferritin(ng/ml)	146.47±69*	177.09±87**	117.4±51

***Significant difference between pre hemodialysisat ($p < 0.001$).
** Significant difference between pre hemodialysis at ($p < 0.01$).
* Significant difference between pre hemodialysis at ($p < 0.05$).

Table -6: concentration of the parameters in bloodserum of pre and post hemodialysis patients.

Mean ± SE		
Parameters	Pre hemodialysis patients n = 55	Post hemodialysis patients n = 55
Urea(mm0l/L)	20.6±7.15***	8.23±3.27
Creatinine (µmol/L)	246.28±34.78***	103.71±11.67
Uric acid (*µmol/L*)	347±68.7***	255.29±40.9
Triglyceride (mmol/L)	1.68 ±0.38**	0.7 ±0.2**
Protein(gm/dl)	5.69 ±0.73**	7.07±2.2
Calcium (mmol/L)	1.44 ±0.46**	1.6±0.35
Phosphorus(mmol/L)	2.1±0.05**	1.47± 0.4
Magnesium(mmol/L)	1.58 ± 0.5*	1.24 ±1.01
Iron(µmol/L)	13.24 ±4.5**	18.28±2.9
TIBC(µmol/L)	39.7 ±13.6**	54.88±8.8
Ferritin (ng/ml)	146.47±69**	177.09±87

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* Significant difference between pre hemodialysis at ($p < 0.05$).

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دراسة بعض المتغيرات الكيموحيوية في مصل دم المرضى المصابين بالعجز الكلوي المزمن

اسراء انور محمد جمعة

كلية التقنية-كركوك-قسم التحليلات المرضية

الخلاصة

اجريت الدراسة الحالية على 55مريضا(32ذكر ,23 انثى)مصابين بالعجز الكلوي المزمن بعمر (15-75)سنة الراقدين في وحدة الديلزة في مستشفى كركوك العام مقارنة مع 35 شخص سليم كمجموعة سيطرة. هدفت هذه الدراسة الى متابعة بعض المتغيرات الكيموحيوية التي تضمنت اليوريا ,الكرياتنين حامض البوليك ,البروتين الكلي ,الدهون الثلاثية ,الكالسيوم ,الفوسفور اللاعضوي ,المغنسيوم,الحديد ,سعة ارتباط الحديد الكلي ,الفيراتين في مصل دم الاشخاص المصابين بالعجز الكلوي المزمن قبل وبعدالديلزة الدموية وللكشف عن مؤشر كتلة الجسم ومستوى الكلوكوز في المرضى المصابين قبل الديلزةفقط .

.اظهرت النتائج وجود زيادة معنوية قي مستوى اليوريا,الكرياتنين ,حامض البوليك,الدهون الثلاثية ,المغنسيوموالفيراتينفي المصل , بينما اظهرت انخفاضا معنويا ($p<0.05$) في مستوىالبروتين الكلي, الحديد ,وسعة ارتباط الحديد الكلي فيمصل المرضى قبل الديلزة مقارنة مع مجموعة السيطرة.

كما اظهرت النتائج زيادة غير معنوية ($p>0.05$)في مستوى الكلوكوز,الفوسفور اللاعضوي ومؤشر كتلة الجسم في الاشخاص المصابين بالعجز الكلوي مقارنة مع الاشخاص الاصحاء .بينما اظهرت انخفاض غير معنوي ($p>0.05$)في مستوى الكالسيوم في مصل المرضى قبل الديلزة مقارنة مع الاشخاص الاصحاء.

عند اجراء مقارنة بين المرضى قبل حالات الديلزة الدموية وبعدها اظهرت انخفاضا معنويا($p<0.05$)في مستوى اليوريا ,الكرياتنين ,حامض البوليك, الفوسفور اللاعضوي والمغنسيوم بعد الديلزة مقارنة مع قبل الديلزة بينما أظهرت ارتفاعا معنويا ($p<0.05$)في مستوى البروتين الكلي , الكالسيوم ,الحديد ,سعة ارتباط الحديد الكلي والفيراتين بعد الديلزة مقارنة مع قبل الديلزة

لوحظ فروق غير معنوية في مستوىالمغنسيوم, الحديد ,سعة ارتباط الحديد الكليوالبروتين الكليبينما لوحظت زيادة معنوية في مستوى اليوريا,الكرياتنين ,حامض البوليك,الدهون الثلاثي ,فوسفور اللاعضوي والفيراتين في مصل دم المرضى بعد الديلزة مقارنة مع مصل دم للاشخاص الاصحاء,ووجود انخفاض معنوي في مستوى الكالسيوم مقارنة مع مجموعة السيطرة.

الكلمات المفتاحية:العجز الكلوي المزمن,الديلزة الدموية,المعادن