Free Fatty Acids and Biochemical Changes in Iraqi patients with Chronic Renal Failure

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Abstract
Chronic renal failure (CRF) is progressive irreversible destruction of kidney tissue by disease which, if not treated by dialysis or transplant, will result in patient's death. This study was carried out on 30 patients (17 male and 13 female) with chronic renal failure. The aim of this research was studied the changes in the level of total protein ,albumin, calcium ,ionized calcium, phosphorous , iron ,ALP, LDH ,CK and FFA in patients with CRF before and after hemodialysis .The obtained results have been compared with 30 healthy subjects as control group (18male and 12 female). The results showed that there was significant increase in the level of calcium ,ionized calcium, phosphorous ,iron ,ALP,LDH,CK and FFA ,while there was a significant decrease in the level of total protein ,albumin before hemodialysis comparison to control group . Non significant changes was observed in the level of total protein ,albumin, calcium ,ionized calcium, phosphorous and significant increase in the level of iron ,ALP,LDH,CK and FFA after hemodialysis as compared to control group. This study shows significant positive correlation between FFA and each of albumin and total protein in pre and post-dialysis patients ,and a significant positive correlation with calcium and non significant with ionized calcium in pre-dialysis patients where as there were non significant correlation with calcium and a significant negative correlation with ionized calcium in post-dialysis patients. The conclusion of this study is hemodialysate composition (concentration of electrolytes, free –ionized calcium and some other plasma constituents), the increase concentration of other biochemical changes after renal dialysis because of amissibility a much of amounts of body fluids, and the change in acidosis status may be affect on the correlation between FFA and other parameters used in this study.
Key words :chronic renal failure, hemodialysis, free fatty acids

Introduction

Chronic renal failure (CRF) is defined as kidney damage for more than three months as evidenced by structural or functional abnormalities with or without decreased glomerular filtration rate (GFR) and manifested either as pathological abnormalities or kidney damage markers in blood or urine or in the imaging tests. Many people are unaware of the problem until more than 70% of kidney function has been lost (1, 2).

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 patients blood against fluid containing no   urea   but   with   appropriate concentrations of electrolytes ,free –ionized calcium   and   some   other plasma constituents (3).

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Hemodialysis relies on the principles of solute diffusion across a semi- permeable membrane. Movement of metabolic waste products takes place down a concentration gradient from the circulation into the dialysate, and in the reverse direction (4).

CRF produces a number of abnormalities of calcium and phosphorus metabolism (5, 6). Several studies have revealed iron deficiency as a very common cause of anemia in pre-dialysis patient, even when assessed by reduced iron staining in the bone marrow (7).During the last decade, for the clinical monitoring of bone turnover in patients with CRF, the determination of total ALP has been used as biochemical marker of bone disease for many years (8). During the past years, they have noted a remarkable tendency for elevations in serum LDH levels to occur in patients with far-advanced renal insufficiency, the etiology of LDH increase in uremia is obscure and this increase is found in all fives isoenzymes in a normal ratio in renal failure (9).

Malnutration occurs in a large proportion of maintenance hemodialysis patients (10), and low levels of serum albumin concentrations have been associated with increased morbidity and mortality (11).Low serum albumin in hemodialysis patients is due to reduction in the rate of albumin synthesis and external loss. Total serum free fatty acid (FFA) levels provide an important measure of the physiologic state (9). Low level of FFA occurs in all tissues but substantial amount can sometimes be found in the plasma, particularly during fasting or starvation. Plasma FFA (transport by serum albumin) is "en route" from their point of origin (triacylglycerol of adipose tissue or

circulating lipoproteins) to their site of consumption (most tissues) (10).

Plasma concentrations of FFA are very variable, being influenced by hormonal, metabolic and nutritional status. Abnormally high plasma concentrations of FFA are implicated in increased risk ventricular fibrillation (12) and sudden cardiac death (13) and more controversially of coronary heart disease (14, 15). The important of FFA in renal disease seems to have been neglected in the last years; nevertheless, many studies have in the past, established that FFA concentrations in end stage renal failure patients are increased following treatment by hemodialysis (16, 17).

Materials and Methods:
Chemicals
The concentration of urea , creatinine , calcium , phosphorus, total serum protein, Iron, LDH, creatine kinase which determine in the serum samples were supplied by Randox kits, while serum uric acid and albumin were determined using kit supplied by (Giesse Diagnostics, Italy, Globalemed, LLC). Serum electrolytes (Na,K) were estimated by Ion Selective Electroid (ISE) method using Electrolyte analyzer .Serum total iron binding capacity , ALP were measured by routine colorimetric assay using kit supplied (Biomaghreb,bioMerieux). Free fatty acid was estimated by colorimetric assay using soap formation(18).

Calculations:
Ionized calcium (iCa) was calculated according to following equations(19)

$$iCa = \frac{60 \times \text{total Ca} - (k' / 12)}{k' + 60}$$

k´=0.19 × total protein (gm/L) + albumin (gm/L)

Sample Collection

Thirty patients (17 male and 13 female) were involved in this study. The patients were referred to Baghdad Teaching Hospital, Al-Kadhimiya Teaching Hospital, and Al -Hakeem Hospital, Baghdad, Iraq. All patients with CRF were diagnosed by clinical examination by (urea & creatinine). The mean age of the patients was 45±10 years. All those patients were treated with hemodialysis (twice in a week). Control group consisted of 30 healthy subjects (18 male and 12 female) with mean age (40±10).

Preparation of Blood Samples

Ten milliliters samples of venous blood were taken from all fasting patients before and after hemodialysis (Frequency of dialysis in those patients was twice in a week and duration of dialysis was 1.5 year). Blood samples were left for 20 minutes at room temperature. After blood coagulation, the sera were separated by centrifugation at 3000 rpm for 15 minutes and then sera stored at -20 °C. Hemolyzed samples were discarded.

Statistical Analysis

The data was analyzed on the computer statistical programme SPSS version10. The mean ±SD was also computed for the comparison of results. The comparison of mean between two groups was tested by Student's 't' test. Results were considered statistically significant if P value is less than 0.05.

Results and Discussions

The results in table I shows highly significant increase in the serum levels of (urea, creatinine) in pre and post-dialysis patients with CRF compared with those of normal individuals and highly significant decrease in the levels of these parameters in post dialysis

patients with CRF compared with pre-dialysis patients with CRF(p<0.001).

Table(I):Biochemical parameters in the two groups patients (pre-and post dialysis)and control group.

parameters	control	Pre-dialysis	Post-dialysis
Urea(mmol/L)	4.095±0.73	31.89 ± 5.51***	19.89± 6.25***
creatinine(mmol/L)	0.100 ± 0.016	0.953 ± 0.151***	0.656 ± 0.185***
Uric acid (mmol/L)	0.268 ± 0.026	0.459± 0.084***	0.316± 0.055**
Total Protein (g/L)	72.6 ± 4.317	69.106 ± 7.428*	71.574±3.548
Albumin(g/L)	44.893 ± 3.452	40.973 ± 6.165**	44.58 ± 3.649

values significantly different from the controls *$p<0.05$, **$p<0.01$, ***$p<0.001$

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 a higher production of other waste substances of protein catabolism (20) while the increase in creatinine level in serum of CRF patients is attributed to the decrease in the number of functioning nephrons, which would reduce the GFR, which causes major decreases in renal excretion of water and solutes (21).

The decreased of the level of urea and creatinine in post dialysis patients with CRF compared with pre-dialysis patients with CRF is due to hemodialysis removes toxins from the blood by a closed – loop process where the blood of the patient is continuously being withdrawn, dialyzed, and returned to the patient. These findings are also supported by other studies Nappi *et al* and Maruyama et *al* (22,23) .

The increase in serum uric acid in pre-dialysis patients with CRF in

table (1)may be attributed to declining GFR which leads to hyperuricemia due to reduced urinary clearance of urate in addition to hyperparathyroidism, is a common complication of moderate – to advanced CRF, can also promote, hyperuricemia via enhanced urate absorption(24). Sombolos K. *etal*, reported that Hemodialysis has a significant impact on the lowering of uric acid level and the correction of hyperuricemia, especially when high-flux membranes are used (25).

This study revealed , significantly decreased in serum level of albumin and total protein in pre - dialysis patients with CRF compared to control as shown in table I .These results may be attributed to the either changes in the structure of basement membrane of glomeruli which consequent lead to the leakage of albumin and some low molecular weight proteins or restriction of protein intake (26)and protein malnutrition (27).Our finding supported by other investigators which found a significant increase in serum of albumin and protein in post –dialysis patients as compared to pre-dialysis patients p<0.05 as shown in table I and explained this result according to haemoconcentration following dialysis procedure and metabolic acidosis which increase whole body protein degradation(16) .

The results in table II show highly significant decrease in level of sodium in pre dialysis patients comparison to control and significantly increase in post-dialysis patients compared to pre-dialysis patients p<0.001. The results also revealed high significantly increase in the level of potassium in pre-dialysis patients compared to control , significantly decreased in post-dialysis patients compared to pre-dialysis patients p<0.001.

Table(II):The level of some ions in two groups with CRF and control group.

parameters	Control (mean±SD)	Pre-dialysis (mean±SD)	Post- dialysis (mean±SD)
calcium(mmol/L)	2.24±0.11	2.46±0.29*	2.24±0.2
Ionized calcium(mmol/L)	1.09± 0.068	1.257 ± 0.142 ***	1.098±0.107
Phosphorous (mmol/L)	1.203 ± 0.120	1.792 ± 0.481***	1.343 ± 0.422
Potassium (mmol/L)	4.013 ± 0.241	5.92 ± 1.194***	4.513± 0.603
sodium(mmol/L)	142.533 ± 2.503	134.733 ± 4.2***	141.473± 5.596**
Iron(mmol/L)	0.018±0.003	0.033±0.003***	0.035±0.003***
Iron binding capacity (mmol/L)	0.07 ± 0.003	0.0628 ± 0.013*	0.0629 ± 0.009*

values significantly different from the controls *$p<0.05$, **$p<0.01$, ***$p<0.001$

Sodium transport activity is regulated by many factors, including protein kinase–dependent phosphorylation , which can increase both activity and channel numbers. Distal tubular Na and K^+/H^+ transport is regulated by the action of aldosterone , which increases the synthesis of apical Na^+ and K^+ channels, the Na^+-K^+ ATPase ,along with the activity of Na^+ –H^+ exchange and H^+ – ATPase (28).

The increase in the level of potassium can be explained by potassium homeostasis is largely regulated by the kidney accounting for excretion of 90% of daily potassium loss(29).Therefore patients with renal failure, acute or chronic, have impaired regulatory mechanisms and are prone to hyperkalaemia. However the decreased of potassium ion and increased of sodium ion after dialysis may be attributed to hemodialysis procedure. This result is agreement with other studies reported by and Heguilen *et al* (30), who suggested that this reduction in the level of potassium after hemodialysis was due to the enhanced shifting of K^+ from the extracellular to the intracellular fluid compartment rather than its removal by dialysis.

In this study all patients were treating with calcium carbonate therefore, there were significantly increased in the level of calcium and ionized calcium but after dialysis there were significantly decreased $p<0.001$ in the level of these parameters (table II), and this result consistent with Katzir et *al* (31)who suggested , a role for low calcium dialysis in treating acute serum calcium elevation and post-dialysis hypertension in patients receiving maintenance hemodialysis.

The results in table II revealed there was a highly significant increase in the serum levels of phosphorus in pre-dialysis patients with CRF compared with those of control subjects and a highly significant decrease $p<0.001$ in the levels of phosphorus in post - dialysis patients compared with pre-dialysis patients (32) . This increase is consequence of diminished phosphorus filtration and excretion with progression of CRF. Gutzwiller *et al* reported that current control of hyperphosphatemia is focused on reducing dietary phosphate intake and diminishing absorption using phosphate binders, where as control and quantification of phosphate removal by hemodialysis is undervalued (32). In table II there were a highly significant increase in the serum levels of iron and a significant decrease in the serum levels of total iron binding capacity in pre- dialysis patients with CRF compared with controls group .The results also revealed a highly significant increase in the serum levels of iron and non significant increase in serum levels of serum levels of total iron binding capacity in serum levels of total iron binding capacity in post- dialysis patients with CRF comparison with pre- dialysis cases .This result can be explained by continuous infusion of intravenous iron to correct iron deficiency in hemodialysis patients

.Intravenous iron has become an accepted therapy for iron deficiency in patients on maintains dialysis (33). Salahudeen *et al*, reported that large rapid infusion of iron is associated with supersaturation of transferrin , leading to the possibility of highly reactive unbound iron transiently present in the circulation(34) .

The results in table III revealed highly significant increase in the serum level of ALP,LDH,CK in pre- dialysis patients with CRF compared with those of control individuals and non significant changes $p > 0.05$ in these enzymes in post-dialysis patients as compared with pre- dialysis patients.

Table(III):Enzymes and FFA levels in pre-and post dialysis patients and control group.

parameters	control	Pre-dialysis	Post-dialysis
ALP(IU/L)	34.7 ± 3.634	178.56±74.522***	174.42±78.32***
LDH(IU/L)	112.267±9.565	138.653±31.463**	131.307±26.798*
CK(IU/L)	41. 93 ± 11.30	86.133±50.241**	86.26 ± 48.272**
FFA (mmol/L)	0.353 ± 0.08	1.016± 0.717***	1.514± 0.626***

values significantly different from the controls *$p<0.05$, **$p<0.01$, ***$p<0.001$

Tibi *et al*,found both bone and liver disease are common in dialysis patients; hence, an elevated unfractionated serum ALP level is often difficult to interpret .In dialysis patients, "intestinal-derived" alkaline phosphatase often is elevated, and the source of this enzyme may be the kidney(35), w hile Schoots *et al,* found the increase in the level of LDH in patients with CRF can be attributed to two possible causes first, it might be a consequence due to hemolysis due to the short survival time of red blood cells in uremic patients and a second possibly is that the reduced clearance of this enzyme may lead to its increase activity in the blood(36) . Lipshultz *et al* , reported low elevation of CK in hemodialysis patients, these findings have not been attributed to myocardial injury (because of factors such as lack

of assay specificity), or they have been attributed to injury caused by risk factors other than uremia(37).

In this study there was nonsignificant increase in the serum level of these enzymes(ALP,LDH) in post–dialysis patients compared with pre- dialysis patients. These results can be attributed to high molecular weights of these enzymes therefore can not permeable across through membrane.

In table III, there was a highly significant increase in the serum levels of FFA in pre- and post-dialysis patients with CRF as compared to control group. The results also revealed highly significant increase in the levels of FFA in post -dialysis patients with CRF compared with pre-dialysis patients with CRF (p< 0.001).The increase of FFA in blood may affect metabolic pathway and endocrine disturbance, it may lead to metabolic syndrome and insulin resistance .It may also affect the levels of total different type of lipoproteins, is associated with increased risk of vascular disease (16). Gillett *et al*, explained this result by heparinization of patients during hemodialysis and the consequent release into the circulation of lipoproteins and hepatic lipases has been thought to cause raised level of FFA concentrations. Other factors such as carnitine deficiency and the presence of acetate in the dialysis buffer solutions may lead to those subsequent changes in FFA (13).

Table (I) shows a significant positive correlation between FFA and each of albumin and total protein in pre and post-dialysis patients and a significant positive correlation with calcium and non significant with ionized calcium in pre-dialysis patients where as there were non significant correlation with calcium and a significant negative correlation ionized calcium in post-dialysis patients.

Table (VI): Correlation coefficients and the significance levels of different serum chemical components in patients with CRF.

	Pre-Dialysis		Post-Dialysis	
	r	P	r	P
Calcium	0.482**	0.007	-0.188	0.319
Ionized calcium	0.106	0.576	- 0.523**	0.003
albumin	0.646**	< 0.01	0.787**	< 0.01
Total protein	0.643**	<0.01	0.708	< 0.01

* Correlation is significant at the level 0.05
**Correlation is significant at the level 0.01

The results outlined above revealed changes in the biochemical components in pre-dialysis and post-dialysis patients; there changes may be attributed to the changes due to hemodialysate composition, haemoconcentration. and change in acidosis status which is commonly associated with CRF cases where acidosis has greater effect on the levels of ionized calcium.

Conclusion

FFA status is ordinarily susceptible to changes in total protein and albumin .The latter component may subsequently affect the levels of total and ionized calcium levels in the sera of the corresponding patients .Therefore hemodialysate composition, haemoconcentration. and change in acidosis status may effect on the correlation between FFA and those parameters

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الاحماض الدهنية الحرة والتغيرات البايوكيميائية في المرضى العراقيين المصابين بالفشل الكلوي المزمن

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الخلاصة:

إن مرض الفشل الكلوي هو الهدم غير العكسي في أنسجة الكلية وفي حالة عدم معالجة المريض بغسيل أو زرع الكلية فان النتيجة سوف تؤدي إلى موت المريض. تضمنت هذه الدراسة 30 مريضاً (17 ذكراً و 13 أنثى) مصابين بمرض الفشل الكلوي المزمن . يهدف البحث إلى دراسة التغير الحاصل في مستوى كل من البروتين الكلي والألبومين و الكالسيوم وايون الكالسيوم والفسفور والحديد و الأنزيمات مثل ALP)   و LDH وCK) وقياس الحامض ألدهني الحر في مرضى الفشل الكلوي المزمن قبل وبعد عملية غسيل الكلية. قورنت النتائج المُستحصلة مع ثلاثون شخصاً سليماً كمجموعة سيطرة (18 ذكراً و12 أنثى). أشارت نتائج الدراسة إلى وجود زيادة معنوية في مستوى كل من الكالسيوم وايون الكالسيوم والفسفور والحديد وALP و LDH وCK والأحماض الدهنية الحرة ،ونقصان معنوي في مستوى كل من البروتين الكلي والألبومين قبل الغسيل الدموي مقارنة بمجموعة السيطرة. لم يلاحظ وجود تغير معنوي في مستوى كل من البروتين الكلي والألبومين و الكالسيوم وايون الكالسيوم والفسفور مع وجود زيادة معنوية في مستوى كل من الحديد وALP وLDH و CK والأحماض الدهنية الحرة بعد الغسيل الدموي مقارنة بمجموعة السيطرة.أوضحت الدراسة وجود علاقة موجبة معنوية بين الأحماض الدهنية الحرة مع كل من البروتين الكلي والألبومين في المرضى قبل وبعد الغسيل الدموي ، و علاقة موجبة معنوية مع الكالسيوم وغير معنوية مع ايون الكالسيوم قبل الغسيل الدموي للمرضى. و أشارت النتائج إلى وجود علاقة غير معنوية مع الكالسيوم و سالبة معنوية مع ايون الكالسيوم في المرضى بعد الغسيل الدموي .أوضحت نتائجنا بان مكونات محلول الغسيل (تركيز الالكتروليتات ، وايون الكالسيوم الحر، ومكونات البلازما الاخرى) وزيادة تركيز بعض المتغيرات البايوكيميائية بعد غسل الكلية نتيجة لفقدان كميات كبيرة من السوائل ,اضافة الى التغير في الحالة الحامضية قد يؤثر على العلاقة بين الأحماض الدهنية الحرة وتلك المتغيرات التي تضمنتها هذه الدراسة .