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Evaluation of adropin and erythropoietin levels and study of biochemical markers in patients with chronic renal failure in Kirkuk city, Iraq

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Abstract

This study aimed to evaluate the levels of adropin, erythropoietin and biochemical parameters such as urea, creatinine, albumin and uric acid in patients with chronic renal failure in the end stage of the disease in Kirkuk province, Iraq. Adropin is considered an important variable in this study due to its important role in metabolic balance, which makes it an important biomarker in assessing variables in chronic kidney failure.

The study samples consisted of 90 individuals, divided into 60 patients and 30 healthy individuals, where the comparison between patients and healthy individuals was studied in terms of gender, age and overall. Adropin and erythropoietin were estimated by ELISA, while the remaining variables were estimated by spectroscopic methods used according to each diagnostic kit. The results showed that the levels of urea ($P \leq 0.0001$) and creatinine ($P \leq 0.0001$) in the patients were significantly higher than in the control group, indicating deterioration of kidney function. A slight decrease in albumin levels was observed in patients ($P \leq 0.0197$), while erythropoietin levels showed a significant increase ($P = 0.0101$). Adropin levels were not statistically significant ($P = 0.0688$), The value indicates that the relationship between adropin and chronic kidney disease did not reach the level of statistical significance. This result does not mean that there is no biological relationship in patients, as changes in metabolism, inflammation, and vascular function can occur, all of which are affected by adropin levels. Analysis of the effects of sex and age showed significant differences in several indices, including erythropoietin and albumin. The aim of study to indicates the importance of monitoring biochemical markers to assess the health status of CKD patients and guide appropriate therapeutic interventions.

Introduction

The dysfunction resulting from kidney disease affects the basic physiological processes in the body and will lead to a range of diseases. The kidney is essential to these processes. Numerous ischemia, toxic, or immunological issues can cause damage to kidney cells, resulting in inflammation and cell death. This can then cause damage to other organs and eventually total failure. It has been proposed that acute kidney injury (AKI) and chronic kidney disease (CKD) are inseparable, despite the fact that their underlying processes differ [1,2]. Regardless of the underlying cause, inflammation and immune system activation are fundamental pathways shared by AKI and CKD. Thus, a key pathogenic mechanism for both

AKI and CKD is inflammation, a process that, in theory, seeks to identify and eliminate dangerous bacteria [3]. Chronic kidney disease (CKD) is a global public health problem, affecting approximately 10% of the world's population [4]. CKD is defined as the presence of kidney damage or decreased kidney function for at least three months regardless of cause. Kidney damage generally refers to pathological abnormalities in the native or transplanted kidney, identified by imaging, biopsy, or clinical signs such as increased albuminuria—an albumin-to-creatinine ratio (ACR) of 30 mg/g (3.4 mg/mmol)—or changes in urinary sediment; decreased kidney function refers to decreased glomerular filtration rate (GFR)[5].

Adropin is mainly a hepatokine because of its strong expression in the liver, where dietary intake of fats and carbs controls its levels [6]. Adipose tissue and muscle also express adropin, despite the liver being its primary source. Particularly in adipose tissue, adropin levels can be impacted by obesity and insulin resistance [7]. Numerous physiological roles of adropin are known to be maintained, including neurological, renal, and pancreatic function as well as metabolic regulation [8, 9]. Adiponectin, an important adipokine, has been shown to interact with adropin.

AMP-activated protein kinase (AMPK) is a key regulator of energy balance and metabolic regulation. The ability of adrenaline to regulate glucose metabolism and support overall metabolic health is facilitated by the interaction between [10]. In addition, adropin has antioxidant properties, which are essential for maintaining cellular health and preventing many diseases. Increased oxidative stress is associated with a deficiency of adenosine, which can lead to cell damage and dysfunction [11,12].

Erythropoietin (EPO) is a nitrogen-linked glycoprotein produced in the kidneys during puberty, which stimulates the production of red blood cells (RBCs) in the bone marrow by acting as a peptide hormone and hematopoietic growth factor (HGF). [13] The synthesis and secretion of EPO depend on the N-linked polysaccharide side chains, which also help to stabilize the protein in the blood, lower hepatic clearance, and enable the systemic movement of EPO from the kidney to the bone marrow [14]. Relative erythropoietin deficiency is a well-known cause of renal anemia, Erythropoietin deficiency begins early in the onset of CKD [15]. This absolute erythropoietin deficiency can be caused by decreased erythropoietin production and/or by errors in EPO-sensing [14,16,17]. In patients with moderate to severe chronic renal disease, elevated urea levels are very prevalent, High levels of serum urea have both direct and indirect long-term harmful effects in addition to being linked to uraemia [18]. Blood urea levels are influenced by a complex balance between production, metabolism, and excretion. Low protein intake and liver disease patients have lower urea production, whereas high protein intake or higher endogenous or exogenous protein metabolism raises urea production. The measurement of urea is widely accessible and recognized as a kidney function indicator [19]. Additionally, skeletal muscle produces and releases creatinine, a waste product of NPN, at a steady pace. The body typically produces creatinine at a fairly consistent rate, depending on muscle mass, and it gives off energy. Creatinine is filtered by the glomerulus, and a small amount is also secreted into the glomerular filtrate by the proximal tubule. It can also serve as an indicator of kidney function. Creatine is synthesized in the liver, pancreas, and kidneys from the transfer of the amino acids arginine, glycine, and methionine [20]. Albumin is the most abundant protein in plasma, normally accounting for about 50% of

human plasma protein with a molecular weight of 66 kDa. The liver produces albumin; a protein whose primary functions include supplying nutrients to tissues and regulating the osmotic pressure of blood vessels. Albumin performs a number of tasks, including binding ligands, moving different chemicals (hormones, vitamins, medications, and calcium) throughout the body, and facilitating tissue growth, healing, and blood vessel integrity. Albumin levels in the human body typically range between 30 and 55 g/L. In critical illness, the rates of albumin synthesis and degradation are altered, resulting in abnormal distribution of albumin between intravascular and extravascular compartments [21,22]. The main byproduct of purine nucleoside metabolism (adenosine and guanosine) is uric acid (UA). Prior to being secreted into the renal lumen, the majority of the filtered uric acid is reabsorbed in the proximal convoluted tubules. reabsorbed in the distal tubules after being transported to the distal proximal tubules.

Physiologically, the concentration of uric acid in plasma may be affected by age, gender, race and even physical activity [23]. This study aims to study the levels of adropin, erythropoietin and some biochemical variables such as urea, creatinine, albumin and uric acid in patients with renal failure and compare them with healthy individuals with an analysis of the effect of gender and age groups in Kirkuk Governorate, Iraq.

Material and Method

Samples were collected from Al-Amal Center for Haemodialysis in Kirkuk Governorate, in 2024 AD. The study included 90 samples, including both the control group (healthy individuals) and the group of patients with chronic kidney disease in end stage of this disease. 30 people of both sexes, 17 men and 13 women, with ages ranging from 13 to 75 years, made up the control group, which served as a comparison group of people who appeared to be in good health. The patient group consisted of 60 individuals of the same age, 32 of whom were male and 28 of whom were female.

Blood samples were drawn at a rate of 5 ml from the person intravenously using a plastic syringe and collected in a plain tube at room temperature, then the serum was separated using a centrifuge at 3000 rpm for 10 minutes, after which the blood serum was divided into Eppendorf tubes and kept frozen at -20 degrees Celsius until laboratory analysis was performed.

The urease enzyme is employed in the diagnostic kit from the French company Biomerieux, which is part of the enzymatic approach (Urease-Modified Berthelot Reaction) used to estimate urea. The French business Biolabo supplied a diagnostic kit that used the color reaction method (Jaffe reaction) to measure the serum's creatinine levels. Using the diagnostic kit supplied by the French business (biolabo), the amount of uric acid in the serum was also calculated.

Then its absorbance is measured at a wavelength of 510nm, where the absorbance intensity is proportional to the amount of uric acid present in the sample. Albumin has the ability to bind to the reagent 3,3,5,5-Tetrabromo-m-Cresol Sulphonphthalein (bromocresol green, BCG) in acidic medium, where the resulting complex of albumin-BCG absorbs at a wavelength of 628 nm and the Biolabo diagnostic kit was used. The hormones adropin and erythropoietin (EPO) were determined using the diagnostic kit from the Chinese company)

Sunlong). Following the steps specified by the company. The absorbance was measured at a wavelength of 450 nm and then the results were calculated through the relationship obtained from the calibration curves for both adropin Figure 1 and erythropoietin Figure 2

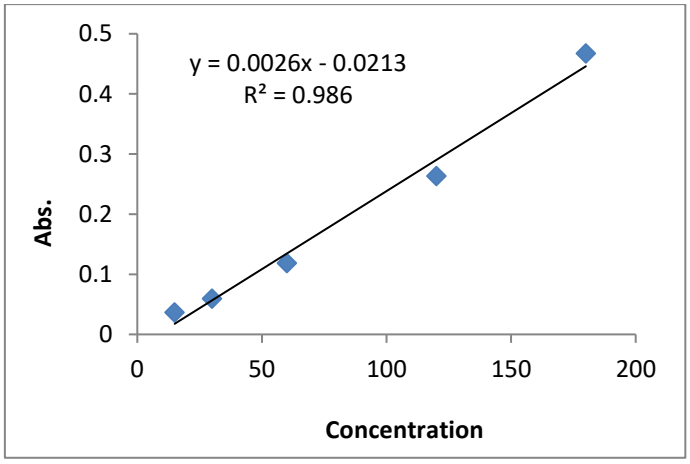

Fig. 1 *Calibration curve for the hormone adropin.*

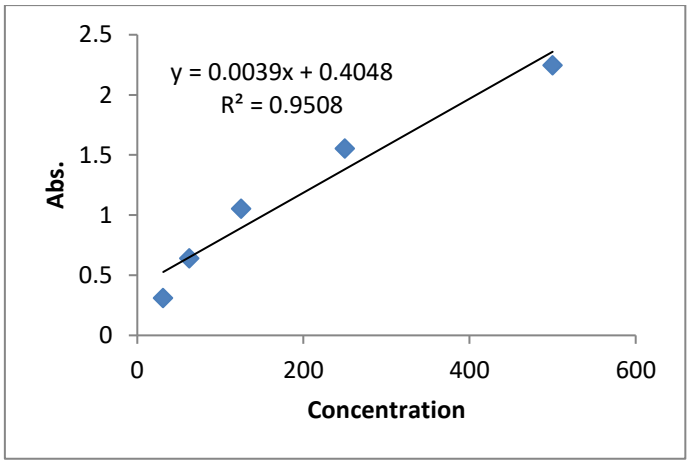

Fig. 2 *Calibration curve for erythropoietin hormone.*

Statistical analysis

Version 20 of SPSS was used to statistically evaluate the results. The arithmetic mean and standard deviation were taken out, two groups were compared using the T-test, and more than two groups were compared using the one-way anova test. The groups that showed significant differences were chosen at a probability level of $p \leq 0.05$.

Results and Discussion

Table 1 indicates significant differences in the levels of the studied indicators between CKD patients and the control group. The results showed a significant increase in urea levels (137.6 ± 38.46 vs. 33.48 ± 6.128, $P \leq 0.0001$) and creatinine (6.776 ± 2.118 vs. 0.7873 ± 0.1658, $P \leq 0.0001$), indicating a significant impairment in kidney function. In contrast, a slight but significant decrease in albumin was observed in patients (3.814 ± 0.4878 vs. 4.07 ± 0.4865, $P \leq 0.0197$). Erythropoietin levels were higher among patients (257066 ± 236913 vs. 141390 ± 56162, $P=0.0101$), while adropin levels were not statistically significant ($P=0.0688$) despite being higher among patients (541.9 ± 1196 vs. 138.2 ± 45.03).

Table 1: Comparison of biochemical parameters between CKD patients and control group.

Parameters	Patients	Control	P value
Urea	137.6 ± 38.46	33.48 ± 6.128	≤ 0.0001 ****
Creatinine	6.776 ± 2.118	0.7873 ± 0.1658	≤ 0.0001 ****
Albumin	3.814 ± 0.4878	4.07 ± 0.4865	≤ 0.0197 *
Epo	257066 ± 236913	141390 ± 56162	0.0101 *
Adropen	541.9 ± 1196	138.2 ± 45.03	0.0688

Table 2 shows the effect of gender on the studied indicators, as male patients showed higher levels of creatinine (7.38 ± 2.32) compared to females (6.45 ± 1.95), while females had higher levels of erythropoietin (286980 ± 282257 vs. 201513 ± 282257, P = 0.0356). Albumin was lower among males than females, reflecting possible effects of malnutrition or greater protein loss in males.

Table 2:Effect of gender on biochemical parameters.

Parameters	Gender	Patients	Control	P value
Urea	Male	132.6 ± 36.51	33.9 ± 6.84	≤0.0001****
	Female	140.3 ± 39.67	32.92 ± 5.26	≤0.0001 ****
Creatinine	Male	7.38 ± 2.32	0.882 ± 0.149	≤0.0001****
	Female	6.45 ± 1.95	0.663 ± 0.083	≤0.0001****
Albumin	Male	3.68 ± 0.539	4.305 ± 0.394	0.0003***
	Female	3.886 ± 0.448	3.77 ±0.435	0.424
Epo	Male	201513 ± 282257	160475 ± 63909	0.1379
	Female	286980 ± 282257	116433 ± 131401	0.0356+
Adropen	Male	141.1 ± 47.87	145.9 ± 48.97	0.7609
	Female	757.7 ± 1444	128.1 ± 38.85	0.1247

Table 3 indicates that there were no significant differences in urea and creatinine levels between the different age groups (G1, G2, G3), suggesting that these changes are related to disease severity rather than age. However, erythropoietin levels showed a decrease in G2 compared to G1 and G3, which may reflect differences in disease stages or physiological response.

Table 3: Effect of age on biochemical parameters.

Parameters	G1	G2	G3
Urea	139.8 ± 35.91	128.8 ± 16.89	139.5 ± 43.84
	A	B	A
Creatinine	7.319 ± 2.207	6.245 ± 2.852	6.758 ± 1.849
	A	A	A
Albumin	3.891 ± 0.5001	3.912 ± 0.5965	3.760 ± 0.4548
	A	A	A
Epo	168066 ± 63822	144041 ± 43567	262648 ± 16015
	A	B	A

Adropen	766.8 ± 1551 A	861.9 ± 1600 A	370.6 ± 905.4 B

The results of the study indicate that there are clear and significant changes in the studied biochemical parameters in CKD patients compared to the control group, reflecting the significant impact of this disease on various vital body functions. The results highlight changes in the levels of urea, creatinine, albumin, erythropoietin (Epo), and adropin, with a focus on gender and age as additional determinants to explain these changes.

As for urea levels, the results showed a significant increase in patients, with an average of (137.6 ± 38.46) compared to the control group, which recorded (33.48 ± 6.128), with a very high statistical significance (P≤0.0001). This increase is attributed to the decreased efficiency of the kidneys in eliminating nitrogenous waste, which is one of the main indicators of impaired kidney function and disease progression. Protein metabolism produces urea, which builds up in the blood as a sign that the kidneys are not doing a good job of getting rid of waste. The urea levels in male patients were slightly lower than those in female patients (132.6 ± 36.51), but both were considerably higher than those in the control group when the data are broken down by gender.

Creatinine was also significantly higher among patients, with a mean of (6.776 ± 2.118) compared to the control group (0.7873 ± 0.1658), with a probability value of (P≤0.0001). Creatinine is a normal metabolic product of muscle, and its accumulation in the blood reflects a decrease in the glomerular filtration rate (GFR). When comparing the results between the sexes, males showed higher levels (7.38 ± 2.32) than females (6.45 ± 1.95), suggesting that males may be more affected by the deterioration of kidney function.

The observed decrease in albumin, although statistically significant, was small, as the results showed a significant decrease in patients (3.814 ± 0.4878) compared to the control group (4.07 ± 0.4865) with a probability value (P≤0.0197). Albumin is a major plasma protein produced by the liver, and its decrease reflects the possibility of malnutrition or protein loss through the urine (proteinuria), which is common among patients with chronic renal failure. In terms of gender, albumin levels were lower in male patients than in females, which may indicate a greater effect of malnutrition or protein loss among males.

Elevated Epo levels in patients indicate compensatory erythropoiesis, and were significantly higher in patients (257,066 ± 236,913) than in controls (141,390 ± 56,162), with statistical significance (P = 0.0101). This increase reflects a compensatory response to CKD-induced anemia, whereby erythropoietin production is reduced in the affected kidney, stimulating its secretion in response to hypoxia. However, when stratified by sex, higher erythropoietin levels were observed in female patients than in males, suggesting a greater response in females to cope with CKD-induced anemia.

The non-significant trend for adropin (P=0.0688) requires cautious interpretation, although its levels were higher in patients (541.9 ± 1196) than in controls (138.2 ± 45.03). Adropin is a protein involved in the regulation of energy metabolism and vascular function, and these results may be indicative of metabolic changes associated with kidney disease, but the large

variability in values may reflect individual differences between patients and the small sample size. When stratified by sex, female patients showed significantly higher levels of adropin than males, which may indicate hormonal effects or metabolic differences.

In terms of age, urea and creatinine levels did not show significant differences between the three age groups (G1, G2, G3), suggesting that these changes are mainly related to disease severity rather than age. However, erythropoietin levels showed significant variation between age groups, being lower in G2 than in G1 and G3, reflecting a differential response of the body based on the stage of the disease or general health status.

The results obtained indicate that chronic kidney disease affects a wide range of physiological functions, so a comprehensive analysis to assess the health status of patients is imperative. The deterioration of kidney function and the progression of the disease are clearly associated with changes in the levels of urea, creatinine, albumin and erythropoietin. These results emphasize the need for individualized treatment strategies that take into account gender and age differences in order to provide more effective treatment for these patients.

Conclusions

In this study, patients with chronic renal failure showed significantly elevated urea and creatinine, reflecting decreased renal function, as well as decreased albumin, indicating malnutrition and low protein. Elevated erythropoietin levels reflect a compensatory response to anemia and differ between men and women. The results also showed that disease severity had a greater impact than age, emphasizing the importance of analyzing biochemical markers to accurately guide treatment.

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تقييم مستويات الأدروبين والإريثروبويتين والعوامل الكيميائية الحيوية في مرضى الفشل الكلوي المزمن في محافظة كركوك، العراق

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الخلاصة:
هدفت هذه الدراسة إلى تقييم مستويات هرموني الأدروبين والإريثروبويتين وبعض المتغيرات البيوكيميائية الأخرى مثل اليوريا والكرياتينين والألبومين وحمض البوليك لدى مرضى الفشل الكلوي المزمن في محافظة كركوك بالعراق. شملت العينة 90 فردًا موزعين على 60 مريضًا و30 فردًا سليمًا كمجموعة ضابطة .أظهرت النتائج ارتفاعًا معنويًا في مستويات اليوريا (P≤0.0001) والكرياتينين (P≤0.0001) لدى المرضى مقارنة بمجموعة الضبط، مما يشير إلى تدهور في وظائف الكلى. لوحظ انخفاض طفيف في مستويات الألبومين لدى المرضى(P≤0.0197) ، بينما أظهرت مستويات الإريثروبويتين زيادة معنوية(P=0.0101) ، مما يعكس استجابة تعويضية لفقر الدم. وعلى الرغم من أن مستويات الأدروبين لم تكن ذات دلالة إحصائية(P=0.0688) ، إلا أن زيادتها قد تعكس تغيرات في العمليات الأيضية المرتبطة بالمرض .وتم تحليل تأثير الجنس والعمر وأظهرت النتائج وجود فروق معنوية في بعض المؤشرات مثل الإريثروبويتين والألبومين وتشير هذه الدراسة إلى أهمية مراقبة المؤشرات البيوكيميائية لتقييم الحالة الصحية لمرضى الفشل الكلوي المزمن وتوجيه التدخلات العلاجية المناسبة.