

STUDY OF SOME BIOCHEMICAL MARKERS FOR PATIENTS WITH CHRONIC KIDNEY DISEASES IN NINEVEH GOVERNORATE

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Abstract

According to several studies, chronic kidney disease (CKD) is highly prevalent worldwide. Patients with chronic kidney diseases on hemodialysis are more likely to have changes in some biochemical variables due to their life style and complications. Our study aimed to study C-reactive protein (CRP) levels, albumin, urea, and creatinine. Also, a study of CRP/Albumin and Urea / Creatinine ratios and glomerular filtration rate (GFR) values to investigate the progression chronic kidney diseases. The study included 159 individuals divided into two groups; first group included (80) patients with chronic kidney disease undergoing dialysis. And second group , included (79) of healthy people as the control group; the ages of both groups ranged from 18-55 years. significantly increase ($P \leq 0.01$) had been observed in urea and creatinine levels. Also, a significantly increase was established in the urea/creatinine ratio at ($P \leq 0.05$) in the patient's group when compared to the control group. While a significant decrease ($P \leq 0.01$) was observed in the level of albumin concentration and glomerular filtration rate. While there was a non-significant difference in the concentration of C-reactive protein and the ratio of CRP / albumin in patients compared to controls Also, negative significant correlation of GFR with creatinine, urea / creatinine ratio and urea concentration at ($r = -0.739, p = 0.00$, $r = -0.793, p = 0.00$, $r = -0.269, p = 0.02$ respectively). We can conclude that chronic kidney disease is related to the high ratios of urea/creatinine, which are more sensitive than biomarkers correlate with disease activity in CKD. While CRP and CRP / albumin ratios are not related directly to kidney dysfunction. Furthermore, GFR values declined considerably ($P \leq 0.01$) between the same groups.

Keywords: Chronic kidney diseases, C-reactive protein, haemodialysis, Albumin, Urea.

INTRODUCTION

Chronic kidney Disease (CKD) is a systemic condition that leads to a gradual decline in kidney function. Its incidence has increased during the past four decades so that some have called it a pandemic. It is a multifactorial disease caused by HIV, high blood pressure, diabetes, and autoimmune diseases (Batubo *et al.* ,2022). Despite being substantial predictors of CKD risk, cardiovascular disease, a family history of the condition, and several ethnic and racial backgrounds do not significantly contribute to the illness's development beyond diabetes, hypertension, and advanced age. The growing older population and rising numbers of people with diabetes and hypertension will cause the number of people with CKD to keep rising(Alkhaqani ., 2022) CKD occurs gradually in periods extending from weeks to years, which eventually leads to end-stage renal disease. At this point, Which can only be treated by peritoneal dialysis, hemodialysis, or kidney transplantation, Hemodialysis is one of the most widely used treatments to reduce the

increased risk of disease complications (Orantes-Navarro *et al.*, 2019). The amount of fluid filtered by the renal glomerular capillaries into the Bowman's capsule per unit of time is known as the glomerular filtration rate (GFR) (Momtaz *et al.*, 2016). The types of kidney impairment can be distinguished by biochemical indications, which can also be utilized to make a diagnosis. Due to the active role the kidneys play in maintaining the body's equilibrium, some of these markers are used alone (Al-Ali *et al.*, 2020).

The cyclic pentameric protein known as C-reactive protein is made up of five identical units that can bond together. The molecular weight of each unit is 2323 kDa, and each unit has an intra-disulfide bond (Ngwa *et al.*, 2019). The pentraxin family of plasma protein-dependent proteins includes CRP. The human CRP molecule is made up of five identical, glycosylated polypeptide-free components, each with 206 amino acid residues. The cyclic pentameric symmetry's yearly configuration is not shared by the protomers (Shrivastava *et al.*, 2015). is a heterogeneous protein synthesized in the liver, concentrations of CRP change during acute and chronic inflammatory conditions (Lewandowska *et al.*, 2020). Vanholder *et al.* noted that urea (a main metabolic waste) levels significantly increased in CKD patients, reaching (in patients with end-stage renal disease and pre-dialysis) up to 10 times or more above the normal range (Vanholder *et al.*, 2018). Creatinine is another metabolic waste that is a biomarker of the kidneys and is produced by non-enzymatic changes of creatine and muscle phosphocreatine (Pathan *et al.*, 2020). creatinine was first identified in 1847 and suggested as a filter marking in 1926 (Levey *et al.*, 2014). The level of serum creatinine is a crucial sign of renal health. It has a low recurrence rate for absorption and secretion, making it simple to monitor. In cases of severe nephron injury, serum creatinine levels rise. Consequently, it is not a proper analysis of the initial stages of renal failure (Tamadon *et al.*, 2018). Our study aimed to study C-reactive protein (CRP) levels, albumin, urea, and creatinine. Also, a study of CRP/Albumin and Urea / Creatinine ratios to investigate the progression of patients with chronic kidney diseases.

MATERIALS AND METHODS

2.1. Study subject:

Iraqi Ministry of Health /Research Committee of Ninavaha Health Directorate has approved the research methodology. Samples have been collected throughout eight months, which began in November of 2020, from the visitors of the unit of kidney dialysis in Ibn- Sena teaching hospital in Nineveh governorate, while the healthy group were volunteers. Each one of the participants has declared their permission to perform bio-medical via signing a written contract. The participants have filled questioner papers for their full names, age, Whereas the weights and heights of the participants have been measured with the use of the electronic balance and measuring body mass index (BMI). The study included 159 individuals divided into two groups; first group included eighty patients with chronic kidney disease undergoing dialysis and suffering from anemia in Nineveh governorate. And second group , included Seventy-nine of healthy people as the control group; the ages of both groups ranged from 18-55 years.

2.1. Sample collection:

Five millilitres of venous blood were drawn from the patients and the control group using a disposable syringe. The blood was centrifuged at 3000 x g for 10 minutes to extract the sera. Sera aliquots were put in Eppendorf tubes and kept frozen under (-20°C) until they were needed. The study excluded patients with cardiovascular problems and those on cytotoxic medications.

2.3. Estimated the biochemical markers:

The human serum C-Reactive Protein (CRP) concentration was measured by Enzyme-Linked Immuno-sorbent Assay (ELISA) kit according to manufactures instruction (My BioSource /USA). Albumin was measured in the serum based on measuring the activity of the alkaline phosphatase in humans (Biolabo/ France) kit. Serum urea was measured using the photometric method (Biosystem /Spain) kit. Serum Creatinine was determined by Colorimetric Method (With Deproteinization) by using Manual Kit assayed according to the manufactured procedure (Randox/ United kingdom)) and GFR calculated from the equation (Wang *et al.* , 2017):

$$\text{GFR} = 175 \times \text{SCr} - 1.234 \times \text{Age} - 0.179 \times 0.79 \text{ (if female)}$$

$$\text{GFR} = 175 \times \text{SCr} - 1.234 \times \text{Age} - 0.179 \text{ (if male)}$$

(Serum Creatinine : mg/dL; age : Year).

2.3 Statistical analysis

Statistical analysis of the data was done using SPSS version 19. using t-test independent student, the level of p-value was considered significant when equal or less than 0.05 ($p \leq 0.05$), and using Person correlation to confirm the potential correlation of GFR.

RESULTS AND DISCUSSION

The data between the two groups are shown in table (1), Results showed that there were a non-significant differences in mean age between all age of the studied group ($P \leq 0.05$), Body mass index (BMI) of CKD patients was shown to be lower than that of non-CKD, which may be explained by the fact that CKD patients may receive special attention and treatment from the health care system, and their adherence to a balanced diet and lifestyle may result in a reduction in excess calories and carbohydrates. Another factor is that individuals with CKD frequently develop anaemia, and their nutritional condition can deteriorate (Zaman *et al.* , 2018). In serum CRP concentration, there were no-significant differences at ($p \leq 0.05$) between CKD patients and the control group. This finding agreed with the study by Lee *et al.*, CRP concentrations in some CKD patients in our study were within normal limits (<6 mg/L). This suggests that there is no correlation between CKD and CRP, which is consistent with a study by (Lee *et al.* , 2015), which hypothesized that CRP was not a risk factor on its own for developing CKD. Taylor and Taylor support as they provide that high CRP concentrations are a significant predictor for the future risk of long-term mortality at the beginning of hemodialysis (Taylor *et al.* , 2013). However, it was explained by Abdel-Messeih *et al.* The immune system may become overactive as a result of uremia, which would raise cytokines and inflammatory markers. Cytokines are known as the main controllers of

the host's response to infection and inflammation, and they contribute to vascular disease and death in hemodialysis patients. Pro-inflammatory cytokines can also boost the production and release of acute-phase proteins like CRP from hepatocytes during an inflammatory response (Abdel-Messeih *et al.*, 2020). When compared to the control group, albumin in the patient group considerably decreased ($P \leq 0.01$). However, the same groups' urea and creatinine levels considerably rose ($P \leq 0.01$). In order to maintain oncotic pressure, microvascular permeability, acid-base balance, and stop platelet aggregation, albumin, a substantial plasma protein component, is needed (Akirov *et al.*, 2017). According to the findings, the level of albumin in the serum of patients with CKD was significantly decrease when compared to control group. This result is also consistent with Al-Khafaji *et al.* who demonstrated that the nephron and glomerular filtration barrier are damaged in CKD patients, resulting in a high GFR; hyper-filtration causes an increase in intraglomerular pressure, resulting in CKD as seen by hypoalbuminemia (Al-Khafaji *et al.*, 2020).

The processes that underpin these correlations are unknown, furthermore there is no direct physiological reason for low serum albumin causing the development of CKD, for various reasons serum albumin levels decrease such as liver injury, liver disease, in reaction to inflammation, albuminuria, as well as to malnutrition can cause deficient blood albumin levels, although the only impoverished state that regularly results in decreased serum albumin is kwashiorkor, an uncommon disease in the developing world (Lang *et al.*, 2018).

The study by Aljebory *et al.* which explained that the continued decrease in renal clearance or glomerular filtration rate leads to the gathering of urea in the CKD pateints refers to Containing Significant Irreversible Nephron Loss, also showed a significant increase in Urea concentration in the CKD patients group compared with the control group. The progression of the disease is connected to the increase in serum urea in CKD. However, a catabolic condition or excessive protein consumption, which increases the production of other catabolic waste products, has a significant impact on urea concentration (Aljebory *et al.*, 2019). And similar to the study by Hassen *et al.*, reported that this rise in serum urea is linked to a decrease in the number of nephrons in the body. Because the kidneys lose their ability to remove nitrogenous wastes from the blood in CKD, urea levels rise, resulting in increase of these chemicals in the blood (Hassen *et al.*, 2018).

According to the results of this study, shows an increase in creatinine concentration when comparing the patient group with control group, these results correspond to Amin *et al.* It is usually observed that leafy green vegetables and meat might lead to an increasing burden on kidneys and cause an increase in creatinine levels (Amin *et al.*, 2014). A similar study done by Singh *et al.* stated that creatinine is continuously generated in our bodies and eliminated by the glomerular filtration unit of the kidneys. As well as kidney function can be measured by how quickly creatinine is filtered through the kidneys. Kidney function may be deteriorated as a result of the kidney's failure to eliminate creatinine by urine excretion, resulting in higher amounts of creatinine in blood serum (Singh *et al.*, 2018).

When comparing the CKD group to the control group, the results show an increase in the urea/creatinine ratio at ($P \leq 0.05$). This is consistent with the findings of Tufan *et al.*, who identified a high urea/creatinine ratio as a sign of malnutrition in dialysis patients. The reason for the increase

in this ratio is correlated with protein intake at all levels of renal function. In theory, if creatinine production is reduced due to muscle loss, urea/creatinine ratio would be higher at a given protein intake (Tufan *et al.*, 2015). On the other hand study by Kouki *et al.*, who reported that urea / Cr ratio decrease in patients group when compared control group due to if the patients take mannitol or dextran (Kouki *et al.*, 2010) . Also there was non significant differences between the two groups at ($P \leq 0.05$) in CRP / Albumin ratio , the CRP / Albumin ratio was first proposed by Fairclough (Wang *et al.*, 2016) . While the GFR values declined considerably at ($P \leq 0.01$) between the same groups, this is consistent with the study conducted by (Heerspink *et al.*, 2014 , Yuste *et al.*, 2013) . This decrease can be explained by the progressive decline in GFR, which represents the irreversible loss of nephrons. In humans, neither nephron number nor single-nephron GFR can be determined in vivo. The GFR, on the other hand, is frequently regarded as the most accurate overall indicator of renal function. The current GFR and the rate at which it declines determine the time it takes to reach renal failure. As a result, the rate of GFR decline over time is regarded as a reliable predictor of kidney disease progression (Levey *et al.*, 2014). in contrast, several clinical studies show that hyperfiltration, or higher GFR, is a pathway for renal injury. The Brenner theory supposed that some people are prone to progressive renal impairment later in life when other risk factors become active because of a low nephron number at birth. In another sense, albuminuria at the single-nephron level is caused by intraglomerular hypertension, which is a beginning to hyperfiltration. Changes in efferent and afferent arteriolar resistances as well as changes in systemic arterial pressure can lead to increased glomerular capillary hydraulic pressure. Without treatment, albuminuria rises and GFR decreases over time, which could eventually result in end-stage renal failure. Human models of kidney injury support this pathogenetic viewpoint. Patients with unilateral renal agenesis, congenitally reduced nephron number, and acquired reduction in renal mass have been found to have glomerular hyperfiltration; these patients are more prone to proteinuria in infancy, which is associated with glomerular sclerosis. This model of renal damage may be applicable to the kidney in early diabetes or hypertension (Palatini ., 2012).

In table (2) showed negative significant correlation of GFR with creatinine and urea / creatinine ratio at ($r = -0.739, p = 0.00$, $r = -0.793, p = 0.00$ respectively) . and negative significant correlation with urea concentration at ($r = -0.269, p = 0.02$), but non significant correlation with other parameters.

Table (1) Comparison between patients and control groups regarding Age ,BMI,C-reactive protein (CRP), Albumin, Urea, Creatinine concentrations, CRP /Albumin ratio, Urea / Creatinine ratio, and GFR value.

Parameters	Groups	Mean + SD	P- value
Age (Years)	Patient Control	32.06 \pm 7.302 33.18 \pm 11.339	0.318 NS
BMI (Kg/m ²)	Patient Control	22.41 \pm 2.54 27.61 \pm 4.86	.000 **
CRP mg / L	Patient Control	6.71 \pm 2.93 5.38 \pm 0.31	.16NS
Albumin g/dl	Patient Control	3.43 \pm 0.53 4.38 \pm 0.31	.000 **
Urea mg/ dl	Patient Control	164.39 \pm 36.01 13.61 \pm 3.35	.000 **
Creatinine mg / dl	Patient Control	8.34 \pm 2.16 0.86 \pm 0.11	.000 **
CRP /Albumin $\times 10^4$	Patient Control	20406.59 \pm 3689 12290.20 \pm 162.93	.320 NS
Urea / Creatinine	Patient Control	18.03 \pm 4.52 15.28 \pm 3.93	.000 *
GFRml/min/1.73m ²	Patient Control	6.12 \pm 2.77 100.49 \pm 25.12	.000 **

** refer to high significant differences between the groups at (P \leq 0.01)

NS refer to no significant differences between the groups at (P \leq 0.05)

Table (2) Correlation of GFR with parameters under study

Parameters	GFR ml/min/1.73m ²	
	R	P
CRP mg / L	0.07-	0.57
Albumin g/dl	0.20	0.08
Urea mg/ dl	-0.269*	0.02
Creatinine mg / dl	-0.739**	0.00
CRP /Albumin $\times 10^4$	0.11-	0.35
Urea / Creatinine	0.793** -	0.00

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** . Correlation is significant at ($P \leq 0.01$) .

*. Correlation is significant at ($P \leq 0.05$)

r. correlation coefficient

CONCLUSIONS

Chronic kidney disease is related to the high concentration of urea and creatinine. This dependent biochemical parameter is closely associated with the progression of the disease. High ratios of urea/creatinine are more sensitive biomarkers for correlation with disease activity in CKD patients. While CRP and CRP / albumin ratios are not related directly to kidney dysfunction. Follow-up of renal kidney patients undergoing dialysis to reduce access to kidney transplantation.

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دراسة بعض المتغيرات الكيموحيوية لدى مرضى الكلى المزمنة في محافظة نينوى
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الملخص

وفقاً للعديد من الدراسات ، فإن مرض الكلى المزمن (CKD) منتشر بشكل كبير في جميع أنحاء العالم. المرضى الذين يعانون من أمراض الكلى المزمنة مع غسيل الكلى هم أكثر عرضة للتغيرات في بعض المتغيرات البيوكيميائية بسبب نمط حياتهم ومضاعفات المرض . هدفت دراستنا إلى تقدير مستويات البروتين التفاعلي (CRP) والألبومين واليوريا والكرياتينين. أيضا ، دراسة عن نسب CRP / الألبومين واليوريا / الكرياتينين للتحقيق من تطور مرض الكلى المزمنة . شملت الدراسة 159 فرداً مقسمين إلى مجموعتين. شملت المجموعة الاولى ثمانين مريضاً يعانون من مرض الكلى المزمن يخضعون لغسيل الكلى في محافظة نينوى. والمجموعة الثانية ، تم تضمين تسعة وسبعين من الأشخاص الأصحاء كمجموعة سيطرة ؛ تراوحت أعمار كلا المجموعتين بين 18-55 سنة. أظهرت النتائج زيادة معنوية ($P \leq 0.01$) في مستويات اليوريا والكرياتينين. أيضاً ، لوحظ زيادة كبيرة في نسبة اليوريا / الكرياتينين عند ($P \leq 0.05$) في مجموعة المرضى عند مقارنتها بمجموعة السيطرة. بينما لوحظ انخفاض معنوي ($P \leq 0.01$) في مستوى تركيز الألبومين ومعدل الترشيح الكبيبي. بينما كان هناك فرق غير معنوي في تركيز البروتين المتفاعل C ونسبة CRP / الألبومين في المرضى مقارنة بمجموعة السيطرة .

ايضا لوحظ هناك ارتباط معنوي سلبي لـ GFR مع الكرياتينين ونسبة اليوريا / الكرياتينين . وارتباط معنوي سلبي مع تركيز اليوريا عند ($p = 0.02$ ، $r = -0.269$).

نستنتج من ذلك ارتباط مرض الكلى المزمن بنسب عالية من اليوريا / الكرياتينين ، وهي أكثر حساسية من المؤشرات الحيوية للارتباط بنشاط المرض في مرض الكلى المزمن. بينما لا ترتبط نسب CRP و CRP / الألبومين ارتباطاً مباشراً باضطراب وظائف الكلى . علاوة على ذلك ، انخفضت قيم GFR بشكل كبير ($P \leq 0.01$) بين نفس المجموعات.

الكلمات المفتاحية: أمراض الكلى المزمنة ، بروتين سي التفاعلي ، غسيل الكلى ، الألبومين ، اليوريا.