

### **Evaluation of some Biochemical and Hematological Parameters in Patients with Chronic Kidney Disease**

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#### Abstract:

Background: Chronic kidney disease is a condition that results from an indefinite change in the structure and function of the kidneys. A slow, steady progression characterizes it and is irreversible.

Objectives: This study aims to evaluate the findings of certain biochemical and hematological tests in samples from Iraqi CKD patients.

Methods: This study included 90 subjects, where 70 patients with chronic kidney disease and 20 healthy individuals. Blood samples were collected from the patients during their visits to Ghazi Al-Hariri Surgical Specialties' Hospital-Medical City, Baghdad, Iraq. Age, sex and body mass index were assessed for each participant followed by renal function tests [serum blood urea, creatinine, uric acid and estimated glomerular filtration rate], and complete blood count, Also, serum levels of uromodulin and cyst atin C were measured statistically studies were carried out using analysis of variance (ANOVA).

**Results:** the study demonstrated a highly significant (P < 0.001) increase in blood urea, serum creatinine and uric acid levels, while a significant (P < 0.05) decrease in estimated glomerular filtration rate levels in patients compared to the control group. On the other hand, it showed a highly significant (P<0.001) decrease in hemoglobin and hematocrit values and a significant (P < 0.05) decrease in the red blood cell count. Patients had revealed a significant (P < 0.05) increase in cystatin C level and a decrease in uromodulin level when compared to the control group.

conclusion: the present study shows that chronic kidney disease patients have upregulated renal function parameters blood urea, serum creatinine and with downregulated estimated glomerular filtration rate, while hematological disorder was more prevalent in patients. On the other hand, cystatin Clevel revealed an increase while uromodulin level showed a decrease in Iraqi patients.

Keywords: Chronic Kidney disease; Cystatin C; Hematological; Hemoglobin; Uromodulin.

### Introduction:

Chronic kidney disease (CKD) is a disorder that affects multiple systems and organs, the increased rates of cardiovascular morbidity and mortality, as well as bone disease development. The main risk factors for developing renal dysfunction into uremia include arterial hypertension (AH), diabetes mellitus (DM), dyslipidemia, and glomerular or congenital abnormalities (1). The kidney structure or function persists for more than three months abnormal are diagnosed according to the Kidney Disease Improving Global Outcomes (KDIGO) initiative as CKD (2)Since 1990, the incidence of CKD has risen by about 30%, mainly due to renal replacement therapy (RRT) and the long-term use of dialysis for patients with end-stage kidney disease (ESRD). CKD will develop a serious public health problem due to its prevalence, risk of death, recurrent hospitalizations, and economic burden (3). Anemia is a communal significance of CKD and its incidence increases as the eGFR falls. Several mechanisms have been proposed to explain

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CKD-related anemia, including relative erythropoietin insufficiency, shortened red cell life span. aberrant iron metabolism, chronic inflammation, metabolic abnormalities (3). Blood urea, creatinine, and eGFR are the typical methods for measuring kidney function (4). Cystatin C(Cys C) is a fairly reliable substance that can be examined quickly, correctly, and precisely by an automated analyzer. Furthermore, in CKD and lower renal filtration disorders, cys Clevels predict mortality and morbidity more strongly than S. Cr. Levels (5) Uromodulin (Umod) is separated as a highly glycosylated mucoprotein that inhibits viral hemagglutination, is expressed primarily in the kidney. Almost all uromodulin in the kidney is released from the luminal surface of tubular epithelial cells between the thick ascending limb (TAL) of Henle's loop and the early distal convoluted tubule. Uromodulin is cleaved by proteases and eliminated in the urine(6). It is the most abundant protein in urine, and kidney tubular epithelial cells manufacture and secrete it (7). The decline in Umod levels associated with the stages of CKD recommends reduced cell viability in the TAL segment (8).

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#### Material and Methods: Study subjects

Ninety subjects were analyzed in this study, who were divided into two groups. The first group comprised 70 CKD patients, while the second group consisted of 20 healthy individuals who served as the control group. The study was conducted between October 2022 and February 2023, and the study subjects were patients at Ghazi Al-Hariri Surgical Specialties Hospital - Medical City in Baghdad, Iraq. The study design included descriptive data such as (age, sex and BMI), as well as clinical data such as (disease duration and stages of the disease (G2, G3, G4).

### **Collection of blood samples**

Blood samples of five millilitres were collected from CKD patients and the controls through a venous blood draw and the samples were divided into two tubes: the first tube contained ethylene diamin tetraacetic acid (EDTA) for assessing complete blood count (CBC); the second tube contained gel, and was then centrifuged at 4000 rpm for 4 min to collect the serum used in the renal function tests.

### **Renal function tests**

The levels of B. urea and S.Cr. were measured by Siemens's diagnostic equipment to obtain the results for patients and control. The level of UA was measured by spectrophotometry with BioSystems kit. The CKD-EPI 2021 equations, on the other hand, are used to calculate the level of eGFR. It is possible to program the eGFR was creatinine equation for age's  $\geq 18$  years in a single sentence for eGFRcr:

eGFRcr =  $142 \times \min(\text{Scr}/\kappa, 1)^{(a)} \times \max(\text{Scr}/\kappa, 1)^{-1.20} \times 0.9938^{\text{Age}} \times 1.012$  [if female]

Where  $\kappa = 0.7$  (females) or 0.9 (males).

a = -0.241 (female) or -0.302 (male).

Scr = serum creatinine in mg/dL; divide by 88.4 for creatinine in mmol/L

Age (years) (9).

### Hematological tests

The complete blood count (CBC) was measured by a NIHON KOHDEN auto hematological analyzer to measure several hematological parameters namely hemoglobin (Hb) level, hematocrit (HCT), red blood cells (RBC) count, total white blood cells (WBC) count differential WBC (neutrophile, eosinophile, basophile, lymphocyte and monocyte), and platelets (PLT) count.

### Cystatin C and Uromodulin levels

Enzyme-linked immunosorbent assay (ELISA) was employed to estimate levels of cys C and Umod using the Cloud-Clone Crop kit from the USA.

### Statistical analysis

The data was then analyzed using the Statistical Package for Social Sciences (SPSS; Version 28) (IBM) program. The statistical studies were carried out using analysis of variance (ANOVA). The data were provided as mean standard error (M $\pm$ S.E.) and a *P*-value of (*P*<0.05) was considered significant.

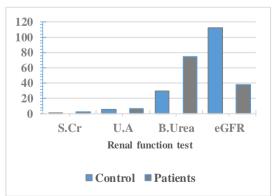
### Results: Descriptive data of the study groups

Table (1) present descriptive information about the study subjects. The results presented indicate that there were non-significant (P > 0.05) differences between patients with CKD and control in regards to age,  $(47.56\pm1.55 \text{ vs} 40.65\pm2.74 \text{ years}, \text{respectively})$ , and BMI values  $(31.79\pm4.21, \text{ vs} 30.79\pm1.64 \text{ kg/m}^2 \text{ respectively})$ . The sex comparison was made between the two groups as follows; patients [male (59%) and female (41%)], and control [male (55%) and female (45%)]. The findings revealed that the percentage of the males were significantly higher (p<0.05) than that of the percentage of females.

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Descriptive data (Mean ± SE)			
Age (year)	BMI	Gender	
	$(kg/m^2)$	No (%)	
		Male	Female
47.56±1.5	31.79±4.2	41 (59%)	29 (41%)
5	1		
40.65±2.7	30.79±1.6	11(55%)	9 (45%)
4	4		
0.902(NS)	0.036(NS)	0.985(NS	0.995(NS
		)	)
Significant			
	Age (year) 47.56±1.5 5 40.65±2.7 4 0.902(NS)	Age (year)         BMI (kg/m²)           47.56±1.5         31.79±4.2           5         1           40.65±2.7         30.79±1.6           4         4           0.902(NS)         0.036(NS)	(kg/m²)         No (%)           Male           47.56±1.5         31.79±4.2         41 (59%)           5         1           40.65±2.7         30.79±1.6         11(55%)           4         4           0.902(NS)         0.036(NS)         0.985(NS)

### Rena faction tests of study groups

Data presented in figure (1), shows the results of the renal function tests (B.U., S.Cr. UA, and eGFR) of patients with CKD and the control group. A highly significant (P<0.001) increase was found in the levels of B.U. and S.Cr. (74.26 $\pm$ 3.82, 2.31 $\pm$ 0.11 mg/dL, respectively) compared with values in the control group (30.0 $\pm$ 2.08, 0.76 $\pm$ 0.04 mg/dL, respectively). There was a significant (P<0.05) increase in UA levels in the patients (6.63 $\pm$ 0.19 mg/dL) as compared to the control group (5.73 $\pm$ 0.35 mg/dL). The eGFR value showed a highly significant (P<0.001) decrease in the patients (37.46 $\pm$ 2.26 ml/min/1.73m<sup>2</sup>) compared with the control group (112.25 $\pm$ 4.19 ml/min/1.73m<sup>2</sup>).



# Figure (1): The result of renal faction tests of study groups

### Hematological parameters of study groups

Table (2) presents the hematological data, which demonstrated a highly significant (P<0.001) decrease in Hb and HCT values in the patients (11.22 $\pm 0.23$  gm/dL, 34.03 $\pm 0.62$  %, respectively) compared with

the control  $(13.06 \pm 0.48 \text{ gm/dL}, 39.48 \pm 1.22 \%)$ , respectively). While there was a significant (P < 0.05) decrease in the RBC count in the patients (4.35±0.09 10<sup>6</sup>/ $\mu$ L) compared with the control (4.77± 0.14 10<sup>6</sup>/µL). Non-significant (P>0.05) differences in the MCV, MCH, and MCHC values with patients CKD (79.29±1.13 fL, 26.22±0.48 pg, and 32.55±0.34 g/dL, respectively); as compared with the control (82.10±1.21 fL, 27.42±0.52 pg, and 33.04±0.28 g/dL, respectively). Also, non-significant (P>0.05) differences were found in the numbers of total WBC  $(7.95\pm0.33 \ 10^{3}/ \mu L)$  and differential WBC (neutrophils, eosinophils, basophils, lymphocytes, monocytes), (58.83±1.27, 2.23±0.198, and 1.70±0.21, 31.00±1.13, 6.28±0.30 %, respectively). In addition, a non-significant (P>0.05) differences in platelet count was reported in a CKD patient  $(245.47\pm8.78 \ 103/ \mu L)$  compared with the control  $(260.35\pm17.05\ 10^3/\ \mu L)$ 

 Table (2): The results of hematological parameters of study groups

study groups			
Hematological	Groups (Mean ± SE)		
tests	Patients	Control	P-value
Hb (gm/dL)	11.22±0.23	13.06±0.48	< 0.001*
HCT (%)	34.03±0.62	39.48±1.22	< 0.001*
RBC (10 <sup>6</sup> /µL)	4.35±0.09	$4.77 \pm 0.14$	0.031*
MCV (fL)	79.29±1.13	82.10±1.21	0.100 NS
MCH (pg)	26.22±0.48	27.42±0.52	0.205 NS
MCHC(g/dL)	32.55±0.34	33.04±0.28	0.462 NS
WBC (10 <sup>3</sup> /	7.95±0.33	8.07±0.45	0.846 NS
μL)			
Ne. (%)	58.83±1.27	55.94±1.77	0.257 NS
Eo.(%)	2.23±0.198	2.04±0.49	0.684 NS
Ba. (%)	1.70±0.21	1.32±0.22	0.365 NS
Lym. (%)	31.00±1.13	34.33±1.62	0.150 NS
Mon. (%)	6.28±0.30	6.41±0.49	0.833 NS
PLT (10 <sup>3</sup> / μL)	$245.47 \pm 8.78$	$260.35 \pm 17.05$	0.430 NS
NS: Non-signif	ïcant, *: Sign	ificant (P≤0.05)	), **: High

significant (P< 0.001)

### Levels of Uromodulin and Cystatin C in the study groups

The data in figure (2), shows the levels of Urom and cys C in the studied groups. Statistically, there was a significant (P < 0.001) decrease in Uomd level in patients ( $5.45\pm0.14$  ng/mL) when compared with the control group ( $21.84\pm1.13$  ng/mL). While the level of cys C revealed a significant (P < 0.001) increase in the patients ( $18.5\pm0.39$  ng/mL) when compared with the control group ( $9.37\pm0.48$  ng/mL).

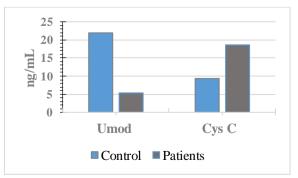


Figure (2) Levels of uromodulin and cystatin  $\boldsymbol{C}$  in the study groups

# Correlation between Uromodulin and studied parameter

The data presented in table (3) shows that the correlation analysis between the uromodulin level and the other parameters tested. Current study reported a significant negative correlation between uromodulin and the following parameters: B. urea (r= -.0321, P=.015), S.Cr. (r= -.341, P=.014), UA (r= -.294, P=.014) and cys C (r= -.452, P=.000). While a significant positive correlation was found between the levels of Umod and eGFR (r=.425, P=.003). On the other hand, a non-significant (P>0.05) correlation was found between Umod and the other parameter.

 Table (3):
 Correlation analyses between the level of uromodulin and studied parameters

Parameters	R	P-value
Blood urea	0321*	.015
(mg/dL)		
Serum creatinine	341*	.014
(mg/dL)		
Uric acid (mg/dL)	294*	.014
eGFR	.425*	.003
ml/min/1.73m <sup>2</sup>		
Hb (gm/dL)	103	.397
HCT (%)	069	.569
RBC (10 <sup>6</sup> /µL)	.037	.764
MCV (fL)	130	.283
MCH (pg)	147	.226
MCHC (g/dL)	135	.263
WBC (10 <sup>3</sup> / µL)	017	.886
Ne. (%)	086	.477
Es. (%)	008	.946
Ba. (%)	.042	.728
Lym. (%)	.090	.460
Mon. (%)	.012	.919
PLT (10 <sup>3</sup> / μL)	.047	.697
Cystatin C	452**	.000
(ng/mL)		

### Correlation between Cystatin C and studied parameters

The data presented in table (4) shows the results of the correlation between of the levels of cys C and the other studied parameters. The current finding revealed a significant positive correlation between the levels cys C and S.Cr. (r = .440, P = .000), while a significant negative correlation was found between the level of cys C and those of eGFR (r = -.399, P = .001) and Umod (r = -.452, P = .000). On the other hand, a non-significant (P > 0.05) correlation was found between the level cyst C and the rest of parameters.

•	-	
Parameters	R	P-value
Blood urea (mg/dL)	.234	.051
Serum	.440**	.000
creatinine(mg/dL)		
Uric acid (mg/dL)	208	.083
eGFR ml/min/1.73m <sup>2</sup>	399**	.001
Hb (gm/dL)	031	.800
HCT (%)	011	.931
RBC (10 <sup>6</sup> /µL)	.121	.317
MCV (fL)	179	.139
MCH (pg)	176	.144
MCHC (g/dL)	107	.380
WBC (10 <sup>3</sup> / µL)	077	.526
Ne. (%)	129	.289
Es. (%)	020	.873
Ba. (%)	061	.616
Lym. (%)	.117	.334
Mon. (%)	.126	.299
PLT (10 <sup>3</sup> / μL)	039	.746
Uromodulin (ng/mL)	452**	.000

 Table (4): Correlation analyses between the level cystatin C and the other studied parameters

### Discussion: -

The current study showed no age disparity among the subjects involved,, which agreed with a previous study (10) which revealed that age has no statistically significant effect on the presence of CKD. However, it disagreed with other studies (11, 12) which discovered that the average age of CKD patients was older than the control population. The current study also discovered a non-significant difference in BMI values between studied group. which disagreed with other authors (13) who reported that increased unhealthy BMI levels were pointedly related to higher risk of CKD. On the other hand, the study found a non-substantial difference in the sex between the patients and control groups. These findings were in agreement with previous studies (14, 15) which found non- significant sex differences association with the frequency and distribution of the main causes of CKD. Nevertheless, the finding in the present study that male were a majority in both study groups agreed with the results of an earlier study (16). This could be due to premenopausal women having a lower incidence of hypertension, less diabetic microvascular disease and a slower damage in renal function (17).

Regarding the findings of the increased levels of B. urea and S.Cr., a similar study was previously conducted (18) showed that B. urea and S. Cr levels were considerably advanced in CKD patients. The reasons may be due to the fact that patients lose their ability to effectively filter waste products from the blood by the kidneys. Additionally, increased protein intake contributes to higher levels of S.Cr. and B. urea due to muscle wasting and protein metabolism abnormalities (19, 20). Furthermore, this study showed an increased level of UA in CKD patients, which is consistence with previous reports (21). These finding may be due to the fact that the kidneys are impaired by reduced uric acid excretion with subsequent accumulation of UA in the bloodstream, which was already reported (22,23). On the other hand, The decrease in GFR indicated in the current study was consistent with the findings of a previous study (24) which found that the S.Cr. levelincreased whereas the level of eGFR decreased due to abnormal kidney function that led to lower efficiency in filtering creatinine and its accumulation in the bloodstream.

The other findings of current study were also in agreement with other studies (25, 26) which found significant decreases in the values of RBCs, Hb, and HCT among CKD patients leading to symptoms of anemia. These values of hematological parameters are reduced due to the decreased synthesis rate of the hormone erythropoietin caused by kidney failure. Increased breakdown of RBCs in chronic renal disease due to reduced erythropoietin production leads to a drop in red blood cell count, which decreases Hb concentration and HCT in those suffered from CKD with mild to moderate renal injury (27). The present finding also showed nonsignificant differences in MCV, MCH, and MCHC values, which agreed with previously published findings (28). This was, however, in disagreement with other data (29) who found that the values of MCV, MCH, and MCHC decrease significantly as the disease progresses in more advanced stages of chronicity. The present results found no significant differences in the levels of PLT, which is in agreement with earlier data(30) which found that platelets function remained constant or even improved as CKD progressed. Also, non-significant changes in counts of total and differential WBCs presented in the current study, were in agreement with other authors (31) who revealed that WBC, neutrophils, lymphocytes, monocytes, eosinophils, and basophils numbers were found to have no correlation with CKD progression. The current finding of decreased Umod value in CKD patients was in agreement with those published by other researcher (32) who found that reduced levels of Umod are reflected indirectly as impairments in renal function. This finding may be due to the tubular cells of the kidney being damaged or undergoing structural changes that lead to a decrease in Umod production, which may be due to fibrosis within kidney tissue (8, 33). Moreover, the present results showed an increase in cys C levels in CKD patients, which was in agreement with earlier finding (34) which found serum cys C is a reliable biomarker for CKD. It is especially useful in patients where traditional methods of measuring creatinine and GFR are ineffective. CKD patients usually have increased levels of cys C due to inflammation, renal tubular failure, and decreased muscle mass. Additionally, smoking, diabetes, hypertension, and cardiovascular disease are risk factors that contribute to higher levels of serum cys C (35). The current study showed negative correlations between the level of Umod and B. urea, S.Cr. and Cys C, while showed a positive correlation between

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Umod and eGFR. These findings were in agreement with previously published reports (36) which stated the positive correlation between Urom and eGFR and the negative correlation of Umod with B. urea, S.Cr., and cys C may predict that Umod level may be a marker of renal function with similarly high diagnostic accuracy. Umod measurements may become a method for estimating the number of functional nephrons that is independent of nonrenal variables and thus greater to GFR calculation-based S.Cr. Alternatively, it may be used to supplement GFR in the assessment of total renal function (33, 37). On the other hand, there was negative correlation between Umod and UA value, which was in agreement with finding of other authors (38) who stated an inverse relationship between Umod and uric acid. The current study revealed a positive correlation between Cys C and S.Cr, which was consistent with previous research (39). The findings of the present study can be explained on the ground that the levels of Cys C and S.Cr. increased as kidney function declined. The findings indicated that Cys C could be a dependable marker of GFR. This is especially useful when S.Cr. may not accurately reflect kidney function. A combination of Cys C and S.Cr can provide a more comprehensive evaluation of kidney function (40, 41). The result regarding the negative correlation between cyst C and eGFR was in agreement with earlier findings (42) which stated the precision, sensitivity, and specificity of cys C to detect GFR relative to creatinine are valuable in clinical research, according to the study. The primary rationale for the increased use of cys C tests has been their ability to predict the effects of declining GFR. The present finding of no correlation between cys C and B.urea was in disagreement with another previous study (43) which stated a significant positive correlation between cys C level with B. urea and uric acid.

**Conclusions:** The present study revealed that CKD patients with impaired kidney function show elevated values of renal function parameters (B. urea and S. Cr) and a decrease in eGFR, in addition to the development of anemia. Furthermore. The study revealed that cys C level was increased while the Umod level was decreased in our patients with CKD. Also, a significant correlation was shown between uromodulin and cystatin C, on one side, and other studied parameters, on the other side. Finally, the level of Umod appears to be in better correlation with the results of renal function test than the level of cys C in CKD patients.

### Limitation:

The study was based and only on center (Kidney Disease and Transplant Center) at Ghazi Ai-Hariri Hospital For surgical hence, the findings don't represent the whole population.

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This research was self-funded and no external entities were involved **Author contributions:** 

Study conception & design: (Hadeel T.A. AL-Ani and Makarim Q. D. Al-Lami.). Literature search: (Hadeel T.A. AL-Ani). Data acquisition: (Hadeel T.A. AL-Ani and Makarim Q. D. Al-Lami.). Data analysis & interpretation: (Hadeel T.A. AL-Ani and Makarim Q. D. Al-Lami). Manuscript preparation: (Hadeel T.A. AL-Ani and Makarim Q. D. Al-Lami.). Manuscript editing & review: (Hadeel T.A. AL-Ani and Makarim Q. D. Al-Lami.).

### Authors' declaration

Conflicts of Interest: None.

We confirm that all the tables in the manuscript are ours. Authors sign on ethical consideration's approval-Ethical Clearance: accepted by the Researcher Ethical Committee and Scientific Committee designated by Biology Department, College of Science, University of Baghdad under the reference number (No. CSEC/0922/0105).

### References:

1. Tinti F, Lai S, Noce A, Rotondi S, Marrone G, Mazzaferro S, et al. Chronic kidney disease as a systemic inflammatory syndrome: update on mechanisms involved and potential treatment. Life. 2021; 11 (5): 419. https://doi.org/10.3390/life11050419

2. Evenepoel P, Cunningham J, Ferrari S, Haarhaus M, Javaid MK, Lafage-Proust M-H, et al. European Consensus Statement on the diagnosis and management of osteoporosis in chronic kidney disease stages G4-G5D. Nephrology Dialysis Transplantation. 2021; 36(1):42-59. https://doi.org/10.1093/ndt/gfaa192.

3. Wong MM, Tu C, Li Y, Perlman RL, Pecoits-Filho R, Lopes AA, et al. Anemia and iron deficiency among chronic kidney disease Stages 3-5ND patients in the Chronic Kidney Disease Outcomes and Practice Patterns Study: often unmeasured, variably treated. CKJ. 2020; 13(4):613-24. https://doi.org/10.1093/ckj/sfz091

4. Bidin MZ, Shah AM, Stanslas J, Seong CLT. Blood and urine biomarkers in chronic kidney disease: An update. Clin. Chim. Acta. 2019; 495: 239-50. https://doi.org/10.1016/j.cca.2019.04.069.

5. Min B, Yun S-R, Yoon S-H, Kim J-D, Hwang WJ, Hwang WM, et al. Comparison of the association intensity of creatinine and cystatin C with hyperphosphatemia and hyperparathyroidism in patients with chronic kidney disease. Scientific reports. 2023; 13(1):3855. https://doi.org/10.1038/s41598-023-31048-2.

6. Usui R, Ogawa T, Takahashi H, Iwasaki C, Koike M, Morito T, et al. Serum uromodulin is a novel renal function marker in the Japanese population. CEN. 2021; 25: 28-36. <u>https://doi.org/10.1007/s10157-020-01964-y</u>

7. Albayati AS, Al Jowari SA. Study the Association of Uromodulin Gene rs13332878 with Chronic Kidney Disease. IJSci. 2023:1071-8. https://doi.org/10.24996/ijs.2023.64.3.4

8. Lv L, Wang J, Gao B, Wu L, Wang F, Cui Z, et al. Serum uromodulin and progression of kidney disease *in patients with chronic kidney disease. Journal Translational Medicine.* 2018; 16:1-9. *https://doi.org/10.1186/s12967-018-1693-2.* 

9. Scherberich JE, Gruber R, Nockher WA, Christensen EI, Schmitt H, Herbst V, et al. Serum uromodulin-a marker of kidney function and renal parenchymal integrity. Nephrology Dialysis Transplantation. 2018; 33(2):284-95. https://doi.org/10.1093/ndt/gfw422

10. Miller WG, Kaufman HW, Levey AS, Straseski JA, Wilhelms KW, Yu HY, et al. National Kidney Foundation Laboratory Engagement Working Group recommendations for implementing the CKD-EPI 2021 race-free equations for estimated glomerular filtration rate: practical guidance for clinical laboratories. Clinical chemistry. 2022; 68(4):511-20. https://doi.org/10.1093/clinchem/hvab029

11.Sabri NW, Rashid BA, Shwiehk RS, Mahdy AH. Assessment of Risk Factors of Chronic Kidney Disease among Patients Attending Medical City Complex. MTU. 2023; 5(2):147-54.

https://doi.org/10.51173/jt.v5i2.885.

12. Chu CD, McCulloch CE, Banerjee T, Pavkov ME, Burrows NR, Gillespie BW, et al. CKD awareness among US adults by future risk of kidney failure. AJKD. 2020; 76(2):174-83. https://doi.org/10.1053/j.ajkd.2020.01.007

13. Salim IK, Diajil AR. Assessment of salivary immunoglobulin A, interleu-kin-6 and C-reactive protein in chronic kidney dis-ease patients on hemodialysis and on conservative treatment. JBCD. 2022; 34(2):62-73.

https://doi.org/10.26477/jbcd.v34i2.3146.

14. Hill NR, Fatoba ST, Oke JL, Hirst JA, O'Callaghan CA, Lasserson DS, et al. Global prevalence of chronic kidney disease-a systematic review and meta-analysis. PloS one. 2016;11(7):e0158765.

https://doi.org/10.1371/journal.pone.0158765

15. Duan J-Y, Duan G-C, Wang C-J, Liu D-W, Qiao Y-J, Pan S-K, et al. Prevalence and risk factors of chronic kidney disease and diabetic kidney disease in a central Chinese urban population: a crosssectional survey. BMC nephrology. 2020; 21(1):1-13. https://doi.org/10.1186/s12882-020-01761-5

16. Kang A, Sukkar L, Hockham C, Jun M, Young T, Scaria A, et al. Risk factors for incident kidney disease in older adults: an Australian prospective population-based study. Int. Med J. 2022; 52(5):808-17. https://doi.org/10.1111/imj.15074.

17. AL-Qadhi HI, AL-Kinani TA, Obeed AA. Effect of Dapagliflozin on Hemoglobin Level in Heart Failure Patients with Chronic Kidney Disease and/or Diabetes. JFac Med Baghdad. 2022; 4(64). <u>https://doi.org/10.32007/jfacmedbagdad.6441973</u>. 18.Harris RC, Zhang M-Z. The Role of Gender Disparities in Kidney Injury. ATM. 2020; 8(7). <u>https://doi.org/10.21037/atm.2020.01.23</u>

19. Ahmed SS, Saleh MA-D. Evaluation of Serum Complement Proteins and Biochemical Parameters in Patients with Chronic Kidney *Disease-associated Pruritus. DJPS. 2022; 18(4).* <u>https://doi.org/10.24237/djps.1804.588B</u>

20. Chang H-L, Wu C-C, Lee S-P, Chen Y-K, Su W, Su S-L. A predictive model for progression of CKD. Medicine. 2019; 98(26). https://doi.org/10.1097/MD.00000000016186.

21. Ferguson MA, Waikar SS. Established and Emerging Markers of Kidney Function. ClinChem. 2012; 58(4):680-9.

https://doi.org/10.1373/clinchem.2011.167494 22.Oh TR, Choi HS, Kim CS, Bae EH, Ma SK, Sung S-A, et al. Hyperuricemia has increased the risk of progression of chronic kidney disease: propensity score matching analysis from the KNOW-CKD study. Scientific reports. 2019; 9(1):6681. https://doi.org/10.1038/s41598-019-43241-3

23. Zhou Q, Ke S, Yan Y, Guo Y, Liu Q. Serum Uric Acid is Associated with Chronic Kidney Disease in Elderly Chinese Patients with Diabetes. Renal Failure. 2023; 45(1):2238825. https://doi.org/10.1080/0886022X.2023.2238825

24. Srivastava A, Kaze AD, McMullan CJ, Isakova T, Waikar SS. Uric Acid and the Risks of Kidney Failure and Death in Individuals with CKD. AJKD. 2018; 71(3):362-70. https://doi.org/10.1053/j.ajkd.2017.08.017

25.Ammirati AL. Chronic kidney disease. Revista da Associação Médica Brasileira. 2020; 66:s03s9.https://doi.org/10.1590/1806-9282.66.s1.3

26. Alsodani MH, Alhashemi WKH. The Effect of Allopurinol on Serum Uric Acid, Blood Urea and Serum Creatinine in Chronic Kidney Disease Patients with High Uric Acid. World Bulletin of Public Health. 2022; 15: 174-81. https://www.scholarexpress.net.

27.Al-Lami MQ, Al-Tai QH, Al-Ani IY. Prevalence of Anemia among Iraqi Patients after Renal Transplantation. JFac Med Baghdad. 2011; 53(2):121-5.

https://doi.org/10.32007/ifacmedbagdad.532850 28. Rumano M, Elezi B, Rumano E. Biochemical and Hematological Parameters in a Group of Chronic Kidney Disease Patients in Albania. The Eurasia Proceedings of Health, Environment and Life Sciences. 2021:39-45.

https://doi.org/10.55549/ephels.7 29.Shastry I, Belurkar S. The spectrum of red blood cell parameters in chronic kidney disease: A study of 300 cases. JAHT. 2019; 10(2):61. https://doi.org/10.4103/joah.joah\_13\_19.

30. Shrestha O, Lamichhane A, Thapa TB, Timilsina A, Khanal PR, Singh V, et al. A Comparative Study of Biochemical and Hematological Parameter in Non Dialysis Dependent Chronic Kidney Disease and Dialysis Dependent Chronic Kidney Disease Patients. IJHSR. 2021; 11(3). https://doi.org/10.52403/ijhsr

31.Baaten CC, Sternkopf M, Henning T, Marx N, Jankowski J, Noels H. Platelet function in CKD: a systematic review and meta-analysis. JASNeph: JASN. 2021; 32(7):1583. https://doi.org/10.1681/ASN.2020101440.

32. Rahman MA, Shanjana Y, Ahmed MS, Dhama K, Hasan Fahim M, Mahmud T, et al. Hematological Abnormalities and Comorbidities Are Associated With the Severity of Kidney Disease: A Hospital-Based Cross-Sectional Study in Bangladesh. Clinical Pathology. 2022;15: 26. https://doi.org/10.1177/2632010X221114807

33. Fedak D, Kuźniewski M, Fugiel A, Wieczorek-Surdacka E, Przepiórkowska-Hoyer B, Jasik P, et al. Serum uromodulin concentrations correlate with glomerular filtration rate in patients with chronic kidney disease. Polskie Archiwum Medycyny Wewnętrznej. PAMW. 2016; 126 (12). https://doi.org/10.20452/pamw.3712.

34. Steubl D, Schneider MP, Meiselbach H, Nadal J, Schmid MC, Saritas T, et al. Association of serum uromodulin with death, cardiovascular events, and kidney failure in CKD.: CJASN. 2020; 15(5):616. https://doi.org/10.2215/CJN.11780919

35.Benoit SW, Ciccia EA, Devarajan P. Cystatin Cas a Biomarker of Chronic Kidney Disease: Latest Developments. Expert Review of Molecular Diagnostics. 2020; 20(10):1019-26.

https://doi.org/10.1080/14737159.2020.1768849 36.Lees JS, Rutherford E, Stevens KI, Chen DC, Scherzer R, Estrella MM, et al. Assessment of cystatin C level for risk stratification in adults with chronic kidney disease. JAMA Network Open. 2022; 5(10):e2238300-e.

<u>https://doi.org/10.1001/jamanetworkopen.2022.38300</u> 37. Genov D, Kundurdgiev A, Ivanova I, Nikolova M, Pencheva V, Hristova M, et al. Role of Serum Uromodulin in the Early Diagnosis of Chronic Kidney Disease. Acta Medica Bulgarica.48(1):13-6. <u>https://doi.org/10.2478/amb-2021-0002</u>. 38.Tan F, Zeng Y, Yan L, Zhang D. Low plasma uromodulin is a predictor of early stage chronic kidney disease progression. Int J Clin Exp Med. 2017;10(5):8055-9. https://www.ijcem.com.

Vukmirović Papuga M, Bukumirić Z, Ilinčić 39 B, Mijović R, Šašić Ostojić T, Žeravica R. Serum Potential Biomarker Uromodulin. а of Tubulointerstitial Damage, Correlates Well with Measured GFR and ERPF in Patients with Medicina. Obstructive Nephropathy. 2022. 58(12):1729. https://doi.org/10.3390/medicina58121729 40.DSa J, Shetty S, Bhandary RR, Rao AV. Association between serum cystatin C and creatinine in chronic kidney disease subjects attending a tertiary health care centre. Journal of Clinical and Diagnostic Research: JCDR. 2017; 11(4):BC09. https://doi.org/10.7860/JCDR/2017/26655.9655.

41. .Lin YL, Chen SY, Lai YH, Wang CH, Kuo CH, Liou HH, et al. Serum creatinine to cystatin C ratio predicts skeletal muscle mass and strength in patients with non-dialysis chronic kidney disease. JCINu.2020; 39(8):2435-41. https://doi.org/10.1016/j.clnu.2019.10.027.

42.Nori Al-Nori MK. A comparative study between Cystatin C based equations in relation to other estimated Glomerular Filtration Rate equations for patients with chronic kidney disease in Mosul city. MJN. 2014; 2(2):107-14. https://doi.org/10.33899/mjn.2021.167937

43. Fang WC, Chen HY, Chu SC, Wang PH, Lee CC, Wu IW, et al. Serum Cystatin C Levels Could Predict Rapid Kidney Function Decline in A Community-Based Population. Biomedicines. 2022; 10(11):2789.<u>https://doi.org/10.3390/biomedicines10112789</u>

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### تقييم بعض المعلمات الكيموحيوية والدموية في المرضى الذين يعانون من مرض الكلى المزمن هديل طلال عياش1 مكارم قاسم داود1 أسم علوم الحياة, كلية العلوم, جامعة بغداد. بغداد. العراق

### الخلاصة:

**خلفية البحث :** مرض الكلى المزمن هو متلازمة تنتج عن تغيير غير محدد في هيكلية الكلى و / أو وظيفتها. يتميز بتطوره البطيء والمطرد و عدم روجو عه. يصنف المريض البالغ مع ظهور مرض الكلى المزمن ، لفترة تساوي أو تزيد عن ثلاثة أشهر ، معدل ترشيح كبيبي (GFR) أقل من 60 مل / دقيقة / 1.73 م 2. مرض السكري وارتفاع ضغط الدم وأمر اض الكلى المتعدد الكيسات و التهاب كبيبات الكلى المزمن وأمر اض الكلى الحادة لفتر ات طويلة هي الأسباب الرئيسية لمرض الكلى المزمن.

الاهداف : الهدفّ من هذه الدراسة هو تقييم نتائج بعض المعلمات الكيموحيوية و الدموية في عينات من المرضى العر اقبين الذين يعانون من مرض الكلي المزمن.

المواد وطرق العمل: تم استخدام تصميم در اسة الحالات حيث تم جمع 90 عينة, حيث 70 مريضا منهم يعانون مرض الكلى المزمن و 20 شخصا سليم. تم جمع عينات الدم من الرضى خلال زيار اتهم من مستشفى غازي الحريري للتخصصات الجر احية- المدينة الطبية, بغداد, العراق. تم تقييم العمر و الجنس ومؤشر كتلة الجسم (BMI) لكل مشارك متبوعا باختبار ات وظائف الكلى [ اليوريا في الدم (Burea), الكرياتينين (S.Cr), حمض اليوريك (UA) و GGFR J, و(CBC) تحليل الدم الكامل . ايضا تم قياس مستويات المصل للسيستاتين سى و اليور ومودولين.

النتائج بينت هذاك زيادة مُعنوية في مستويات .Burea, S.Cr و UA ، بينما تم الكشف عن انخفاض معنوي في مستوى RGFB في المرضى مقارنة بالمجموعة السليمة. من ناحية أخرى, تم تسجيل انخفاض في قيم الهيموغلوبين (Hb)و الهيماتوكريت (HCT)وفي عدد خلايا الم مقارنة بالمجموعة السليمة. من ناحية أخرى, تم تسجيل انخفاض في قيم الهيموغلوبين (Hb)و الهيماتوكريت (HCT)وفي عدد خلايا الم مقارنة بالمجموعة السليمة. من ناحية أخرى, تم تسجيل انخفاض في قيم الهيموغلوبين (Hb)و الهيماتوكريت (HCT)وفي عدد خلايا الم مقارنة بالمجموعة السليمة. من ناحية أخرى, تم تسجيل انخفاض في قيم الهيموغلوبين (Hb)و الهيماتوكريت (HCT)وفي عدد خلايا الم الحمراء (RBC). لم يتم الكشف عن فرق بين المرضى والمجموعة السليمة فيما يتعلق باختبار ات الدم الاخرى. كانت هناك زيادة معنوية في مستوى المسيتاتين سي و انخفاض معنوي في مستوى على ذلك, كشغت النتيجة الحالية عن وجود علاقة سلبية بين مستوى السيستاتين سي وانخفاض معنوي في مستوى يور ومودولين في المرضى. علاوة على ذلك, كشغت النتيجة الحالية عن وجود علاقة سلبية بين مستوى السيستاتين سي انخواض معنوي في مستوى يور ومودولين في المرضى. علاوة على ذلك, كشغت النتيجة الحالية عن وجود علاقة سلبية بين مستوى اليور ومودولين و العور وي الموضى. علاوة على ذلك, كشغت النتيجة الحالية عن وجود علاقة سلبية بين مستوى اليور ومودولين و مستوى يور ومودولين في المرضى. علاوة على ذلك, كشغت النتيجة الحالية عن وجود علاقة سلبية بين مستوى اليور ومودولين و مستوى يور مودولين و الحضافة الى ارتباط سلبى مع GFR.

الأستنتاجات: تظهر الدراسة الحالية أن مرضى CKD لديهم ارتفاع في معايير وظائف الكلى B. urea و S. Cr وانخفاض في eGFR، إلى جانب ظهور فقر الدم. من ناحية أخرى ، كشف مستوى السيستاتين سي عن زيادة بينما أظهر مستوى يورومودولين انخفاضا في المرضى العراقيين الذين يعانون من مرض الكلى المزمن. أيضا ، تم الكشف عن علاقة كبيرة بين اليورمودلين وسيستاتين سي من جانب ومع معايير در اسية اخرى من جانب اخر.

الكلّمات المفتاحية: يورومودولين ، سيستاتين سي ، أمراض الكلى المزمنة ، أمراض الدم.