

تقييم بعض المؤشرات البيوكيميائية والدموية والمناعية لدى مرضى الكلى المزمنة في محافظة

واسط

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المستخلص :

الخلفية: مرض الكلى المزمن (CKD) هو اضطراب جهازى مركب، يتجاوز مجرد انخفاض كفاءة الترشيح الكلوي؛ فهو حالة مستمرة لأكثر من ثلاثة أشهر، تتميز بتدهور تدريجي في وظائف الكلى، حيث ينخفض مستوى الترشيح الكبيبي إلى أقل من ٦٠ مل/دقيقة/١.٧٣ م². الأهداف: أُجريت هذه الدراسة للتحقق من المؤشرات البيوكيميائية والدموية والمناعية الالتهابية في مجموعة ضابطة من الأفراد الأصحاء ومجموعة من الأفراد المصابين بمرض كلوي مزمن. **المنهجية:** أُجريت دراسة حالة-ضابطة، شملت ثلاثون مريضاً مصاباً بمرض كلوي مزمن وثلاثون شخصاً سليماً كمجموعة مرجعية. تم قياس المؤشرات التالية: اليوريا (Urea)، الكرياتينين (sCr)، حمض اليوريك (UA)، الهيموجلوبين (Hb)، عدد خلايا الدم البيضاء (WBC)، الصفائح الدموية (PLT)، حديد المصل (SI)، وفيريتين المصل (SF)، ثم تم تحليلها إحصائياً. **النتائج:** أظهرت النتائج انخفاض متوسط مستوى الحديد لدى مرضى القصور الكلوي المزمن (9.88 ± 64.13 ميكروغرام/ديسيلتر) مقارنةً بالمجموعة الضابطة (129.72 ± 15.6 ميكروغرام/ديسيلتر). وكان متوسط مستوى الحديد أقل بشكل ملحوظ لدى مرضى القصور الكلوي المزمن مقارنةً بالمجموعة الضابطة ($P < 0.05$). كما أظهرت النتائج ارتفاعاً ذا دلالة إحصائية في متوسط مستوى الفيريتين لدى مرضى القصور الكلوي المزمن مقارنةً بالمجموعة الضابطة (743.80 ± 76.3) مقابل 211.21 ± 33.5 على التوالي؛ $P < 0.05$). وأظهرت النتائج أيضاً ارتفاعاً في عدد خلايا الدم البيضاء، حيث بلغ 10.79 ± 8.01 لدى مرضى القصور الكلوي المزمن و 4.65 ± 0.46 في المجموعة الضابطة. وكان متوسط مستوى الفيريتين أعلى بشكل ملحوظ لدى مرضى القصور الكلوي المزمن مقارنةً بالمجموعة الضابطة



($P < 0.05$). مع ذلك، كان متوسط مستوى الهيموجلوبين وعدد الصفائح الدموية أقل بكثير لدى

المصابين مقارنةً بمجموعة الضبط السليمة. ($P < 0.05$)

الخلاصة: توضح هذه النتائج التفاعلات بين الاختلالات في المؤشرات البيوكيميائية والدموية والمناعية الالتهابية في الفيزيولوجيا المرضية لمرضى القصور الكلوي المزمن. قد تُستخدم هذه المؤشرات الحيوية كمؤشرات لتقييم التطور المرضي وتوجيه الخطط العلاجية المناسبة للمرضى. **الكلمات المفتاحية:** الفشل الكلوي المزمن؛ الحديد؛ فقر الدم؛ الفيريتين؛ اليوريا؛ الكرياتينين.

Evaluation of some biochemical, hematological, and immunological indicators in chronic kidney disease patients in Wasit Province

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Abstract

Background: Chronic Kidney Disease (CKD) it's a compound systemic disorder, more than just a decrease in renal filtration efficiency; it is a persistent condition lasting more than three months, characterized by a gradual decline in kidney function, with the glomerulars filtering level falling less than ($60 \text{ ml/Minut/ } 1.73 \text{ m}^2$). (IR). **Objectives:** This study was conducted to investigate biochemical, hematological, and immune-inflammatory markers in a control group of healthy individuals and a group of sick individuals with prolonged renal illness. **Methodology:** A case-control study was conducted, including thirty sick people with prolonged kidney disease and thirty healthy people as a control group. The following markers: urea (Urea), creatinine (sCr), uric acid (UA), hemoglobin (Hb), white blood cell count (WBC), platelets (PLT), serum iron(SI), and serum ferritin(SF) were measured and then statistically analyzed. **Results:** The results showed that the mean iron level was lower, $64.13 \pm 9.88 \text{ } \mu\text{g/dL}$, in the patients with prolonged renal illness and $129.72 \pm 15.6 \text{ } \mu\text{g/dL}$ in the control group. The mean appears to be significant lower in CKD patients, in contrast to the control group ($P < 0.05$). Furthermore, the current results showed a statistically significant increase in the mean ferritin level in CKD patients compared to the control group (743.80 ± 76.3 vs. 211.21 ± 33.5 ,



respectively; $P < 0.05$). The results also showed an increase in the white blood cell count, which was 8.01 ± 1.79 in CKD patients and 4.65 ± 0.46 in the control group. The mean was significantly higher in CKD patients compared to the control group ($P < 0.05$). However, mean hemoglobin (HGB) and platelet count were significantly lower in infected people than in the healthy control group ($P < 0.05$).

Conclusion: These results illustrate the interactions among abnormalities in biochemical, hematological, and immune-inflammatory markers in the pathophysiology of chronic kidney disease. These biomarkers may serve as indicators to assess disease progression and guide appropriate treatment plans.

Keywords: chronic renal failure; iron; anemia; ferritin; urea; creatinine.

Introduction

Chronic kidney disease (CKD) is an illness in which the kidneys cannot maintain the body's fluid balance, characterized by anatomical abnormalities or weakened renal function that persists for more than three months. This impairment results from internal damage to the renal glomeruli and blood vessels (Purnadianti, 2024). This functional decline leads to metabolic and hormonal imbalances, most notably disruption of the renal-hematopoietic axis. Damage to the kidney's interstitial tissue causes a decrease in erythropoietin production, and iron balance is disrupted due to chronic inflammation. This makes anemia a key clinical complication of the disease and a major indicator of the patient's deteriorating health (Babitt and Lin, 2012)

CKD affects people worldwide, but it hits low- and middle-income countries the hardest. These areas often do not have the resources to manage the disease well. (Iatriddi *et al.*, 2025) Globally, about 10% of people have CKD, but it is more common in older people, women, and racial minorities. In countries like Egypt, it is a very significant challenge, affecting a big portion of adult people, especially those with high blood pressure. (Nagib *et al.*, 2023) Also, this kidney dysfunction in patients with chronic kidney failure



leads to the accumulation of metabolic waste products in the body (Siregar, 2020).

Kidney disease is associated with a variety of hematological alterations with anaemia being the predominant feature. The etiology of anaemia in patients with chronic kidney disease is Multicausal, and causes include hemolysis and shortened red blood cell lifespan, iron deficiency, vitamin B12 and folic acid deficiency, blood loss (dialysis, sampling, gastrointestinal), aluminum poisoning, fibrocystic osteomyelitis, anemia of chronic disease due to recurrent infections, and erythropoietin (EPO) resistance (Shastry and Belurkar, 2019).

Anemia in CKD is related to reduced health-related quality of life, increased cardiovascular and all-cause mortality, and a great risk of progression to end-stage renal disease (ESKD). Several studies have examined the prevalence and consequences of iron deficiency anaemia in dialysis people. But limited data exist on the prevalence and relatedness of absolute and functional iron deficiency in non-dialysis-dependent CKD (Awan *et al.*, 2021).

Given the physiological nature of kidney function, the interaction between renal function biomarkers (urea, creatinine, and uric acid), complete blood count parameters (hemoglobin, white blood cells, and platelets), and iron status indicators (serum iron and ferritin) is crucial for improving diagnosis, risk classification, and targeted treatment. Therefore, to provide a comprehensive analysis of the interaction between renal function, hematological profile, and iron deficiency status characteristic of kidney failure patients, this study aims to analyze these biomarkers in kidney failure patients with iron deficiency anemia and compare them to a control group of healthy individuals.

Material and Method

The Design of Study for Individuals

the case control study were behave at Al-Zahraa Teaching Hospital, Iraq, from January 2026 to April 2026. The study included 30 patients of both sexes diagnosed with chronic kidney disease (CKD), clinically diagnosed by a specialist physician, and 30 healthy individuals (both sexes) as a control group.



Patients with: active infection/inflammation, recent blood transfusion, bleeding, malignant tumors, blood disorders, liver disease, those taking iron supplements, or erythropoietic stem cell stimulators (ESAs) may be excluded.

Sample Collection and Analysis

Methods for Samples Collection and analysis: (5) ml of vein blood was withdrawn from both healthy individuals and CKD patients. Each sample was immediately divided into two parts: the first part (1 milliliters of blood) was placed in an EDTA K3 tube for hematological tests, including a complete blood count (CBC), and the second part (4 milliliters of blood) was placed in gel tubes to obtain serum. The serum was used to measure iron and ferritin levels, as well as kidney function indicators (creatinine, urea, and uric acid).

Biochemical parameters were measured using reagents and equipment from Abbott (USA), in accordance with the manufacturer's recommendations. Iron stores were measured using a Mini VIDAs analyzer (Germany). Additionally, complete blood count parameters were measured using reagents from a Sysmex corporation and Sysmex analyzer (Germany).

Statistical analysis.

All statistical analyses were performed with the help of SPSS software (Version 26, IBM Corp., Armonk, NY, USA). Values of each parameter were presented in the form of Mean $\pm$ Standard Deviation (Mean $\pm$ SD). All study group were compared through independent t-test of variance. A statistically significant difference was considered as $p > 0.05$ and a highly significant difference was considered as $p \leq 0.001$. Numeric data were presented as mean, standard deviation after performance of **Kolmogorov-Smirnov normality** test and making decision about normally distributed variables.

Results

Hematological parameters of patients and controls

The comparison of some blood parameters (WBC, HGB and Platelets) in CKD patients and controls it was carried out and we observed the results was given a proven in the table (1). Average level of white blood cells (WBC)



count were 8.01 ± 1.79 and 4.65 ± 0.46 , in CKD individuals and control group separately the average level stayed highest in CKD patients in compared to control and the difference remained significant ($P < 0.05$). But the means of both Hemoglobin (HGB) and platelets count were significantly lower in peoples with CKD when compared to control, ($P < 0.05$).

Table (1): Comparison of some blood parameters (WBC, HGB and Platelets) in chronic kidney failure patients and healthy controls.

Groups		WBC count (10^3 / μ L)	HGB (g/dL)	Platelets
CKD patients	Mean $\pm$ SD	8.01 ± 1.79	8.06 ± 1.51	156.97 ± 31.2
	Range	5.20-11.00	3.70-9.80	51.00-230
Control	Mean $\pm$ SD	4.65 ± 0.46	14.26 ± 1.17	260.23 ± 39.2
	Range	4.00-5.80	11.90-16.10	168.00-412.00
p-value		0.001**	0.001**	0.001**

SD: standard deviation; †: Independent T test; **: significant at $P < 0.05$

Iron profile (Iron and Ferritin) of patients and controls

The comparison of iron (Fe) profile (the Iron and the Ferritin) in CKD patients and controls it was completed and the results obtained were demonstrated in table (2). Mean levels of Iron were $64.13 \pm 9.88 \mu\text{g/dL}$ and $129.72 \pm 15.6 \mu\text{g/dL}$, in chronic kidney disease patients and (control group) correspondingly the mean level was lower in CKD patients in compared to non-affected peoples(control), while the change stayed significant ($P < 0.05$). Also, the current finding show significant rise of mean Ferritin level



in CKD patients in compared to control (743.80 ± 76.3 versus 211.21 ± 33.5) respectively, ($P < 0.05$).

Table (2): Comparison Iron profile (the Iron and Ferritin) in CKF patients, and healthy controls

Groups		Iron ($\mu\text{g/dL}$)	Ferritin (ng/mL)
CKF patients	Mean $\pm$ SD	64.13 ± 9.88	743.80 ± 76.3
	Range	29.00-97.00	384.00-1200.00
Control	Mean $\pm$ SD	129.72 ± 15.6	211.21 ± 33.5
	Range	95.00-173.00	102.00-331.00
p-value		0.001**	0.001**

SD: deviation of the mean; †: Self-determining T test; **: significant at $P < 0.05$

Kidney function Patient testing criteria and control group

Comparison of renal function tests parameters, (Urea, Uric acid and Creatinine) data between patients and controls as Table 3. CKD patients exhibited significantly elevated all renal function tests parameters, (Urea, Uric acid and Creatinine) levels compared with healthy controls. Blood urea mean level in the cases group was 162.63 ± 28.2 mg/dL whereas in the control group was 17.06 ± 4.68 mg/dL. These alterations had a statistical significance ($P < 0.001$). Likewise, serum Creatinine level was markedly increased in CKD patients (8.34 ± 2.31 mg/dL) compared to controls (0.94 ± 0.18 mg/dL; $P = 0.001$). Also, there was significant increase of Uric acid levels in CKD patients (8.23 ± 0.94) in compared to healthy controls (4.49 ± 0.86 ; $P = 0.001$).



Table (3): Comparison of Kidney function tests parameters (Urea , Uric acid and Creatinine) in CKF patients and controls

Groups		Urea (Mg/dl)	Creatinine (mg/dL)	Uric acid (mg/dL)
CKD patient s	Mean $\pm$ SD	162.63 $\pm$ 28.2	8.34 $\pm$ 2.31	8.23 $\pm$ 0.94
	Range	90.00-348.00	3.20-13.30	6.80-10.20
Control	Mean $\pm$ SD	17.06 $\pm$ 4.68	0.94 $\pm$ 0.18	4.49 $\pm$ 0.86
	Range	9.00-24.00	0.67-1.35	3.00-6.80
p-value		0.001**	0.001**	0.001**

SD: standard deviation; †: Independent T test; **: significant at $P < 0.05$

Correlation between Kidney function tests parameters (Urea, Uric acid and Creatinine) and other parameter in CKD patients.

The correlations between Kidney function tests parameter (Urea , Uric acid and Creatinine) plus other variables. in CKF patients were displayed in (Table No. 4). There are significant negative correlation between Uric acid and Iron levels ($r=-0.538$ and $p=0.001$), in CKF patients, but there are were non-significant correlation between all other parameters.



Table (4): Correlation between Kidney function tests parameters (Urea, Uric acid and Creatinine) and other parameters in CKD patients

Parameter s	Urea (mg/dL)		Creatinine (mg/dL)		Uric acid (mg/dL)	
	<i>r</i>	<i>P</i>	<i>r</i>	<i>P</i>	<i>r</i>	<i>P</i>
Iron	-0.047	0.806	-0.265	0.158	-0.538	0.001*
Ferritin	0.146	0.443	0.003	0.989	0.039	0.837
WBC	0.101	0.595	0.172	0.365	0.299	0.108
HGB	-0.049	0.796	-0.147	0.442	-0.071	0.708
Platelets	-0.043	0.822	-0.276	0.139	-0.218	0.246

r: Pearson correlation

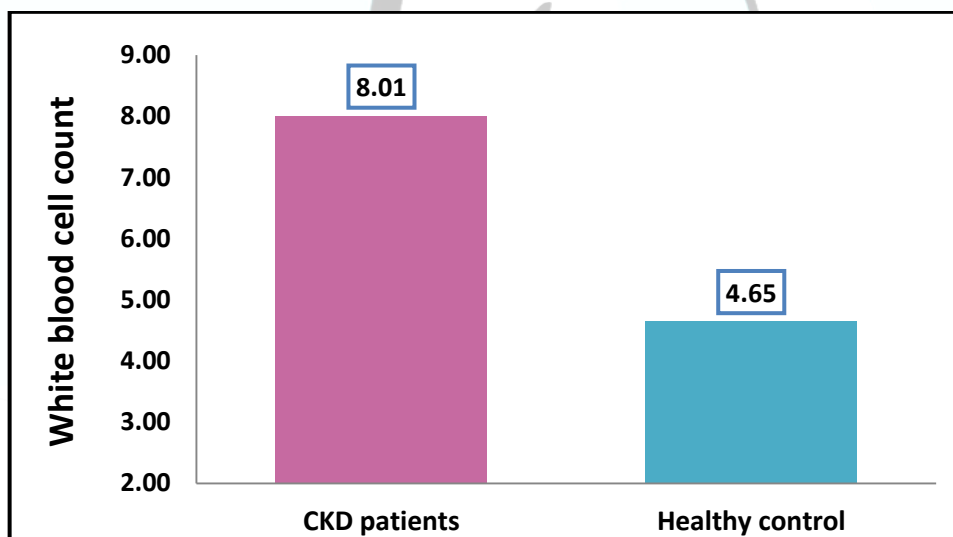


Figure (1): The means level of WBC in patients and control groups

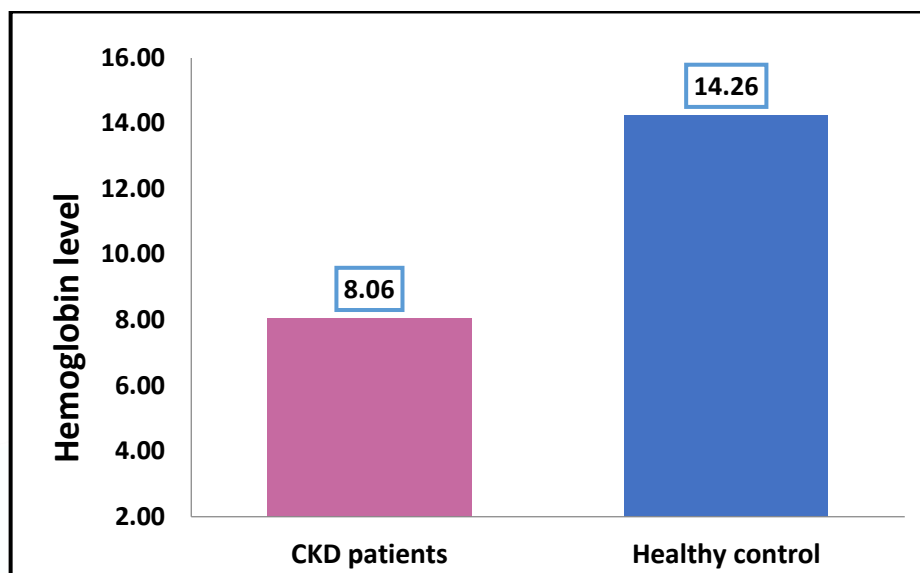


Figure (1): The means level of HGB in patients and control groups

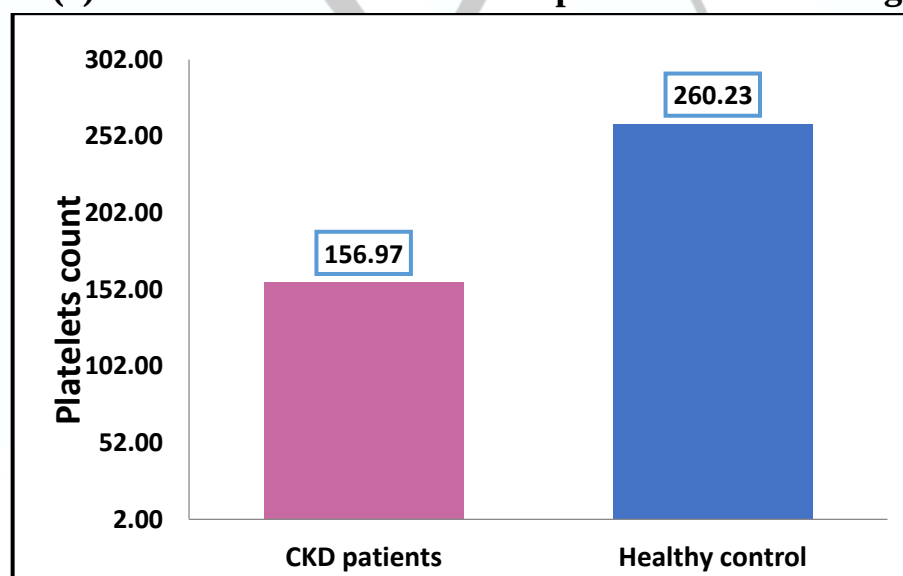


Figure (1): The means level of Platelets in patients and control groups

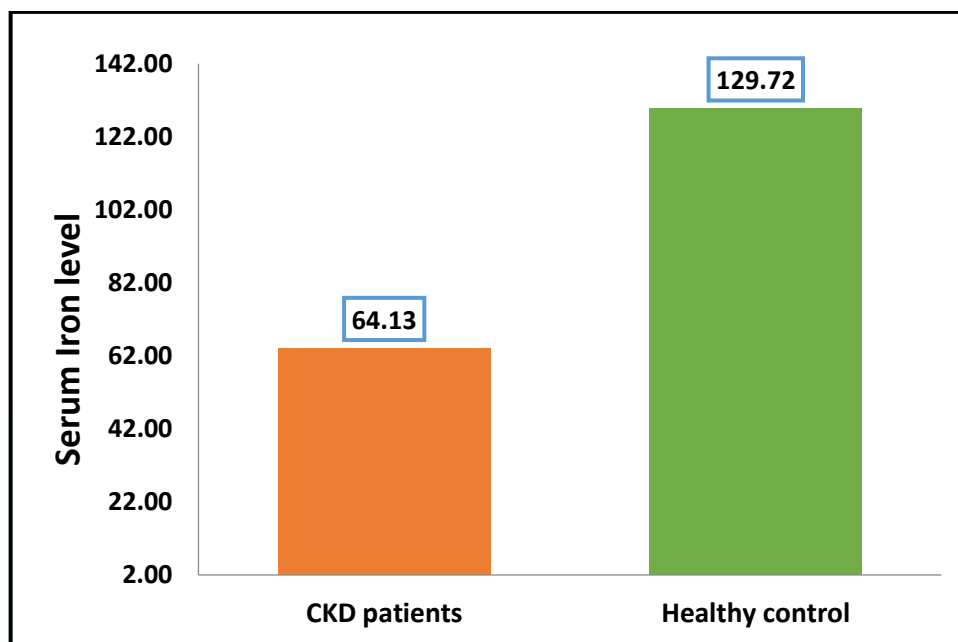


Figure (1): The means level of Iron in patients and control groups

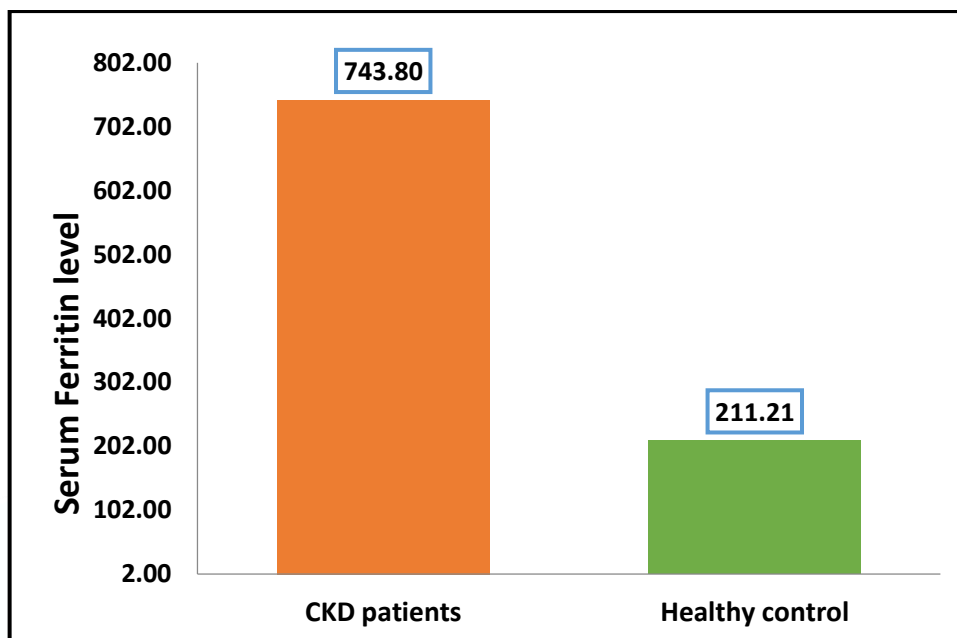


Figure (1): The means level of Ferritin in patients and control groups

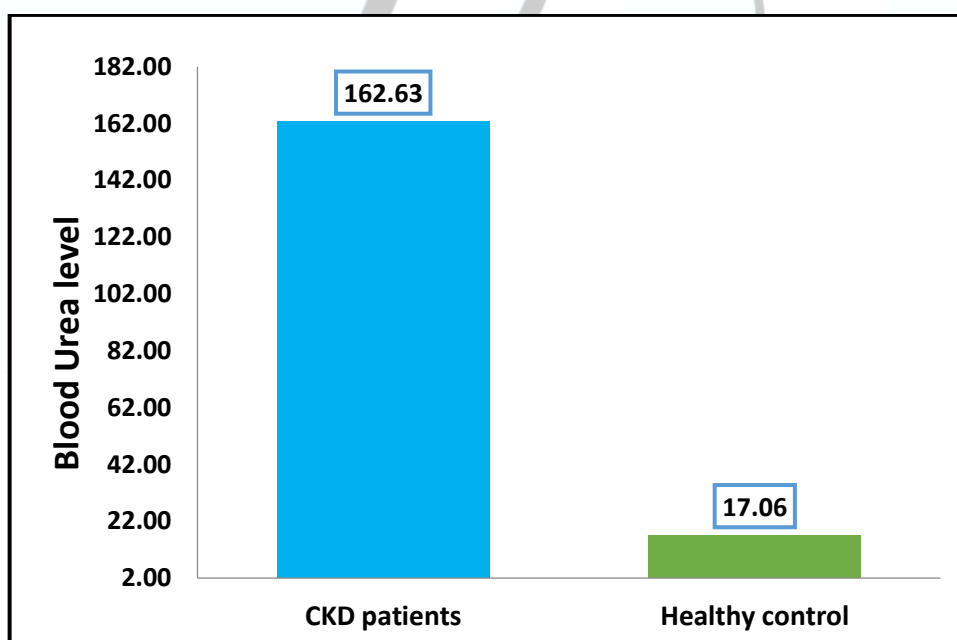


Figure (1): The means level of Urea in patients and control groups

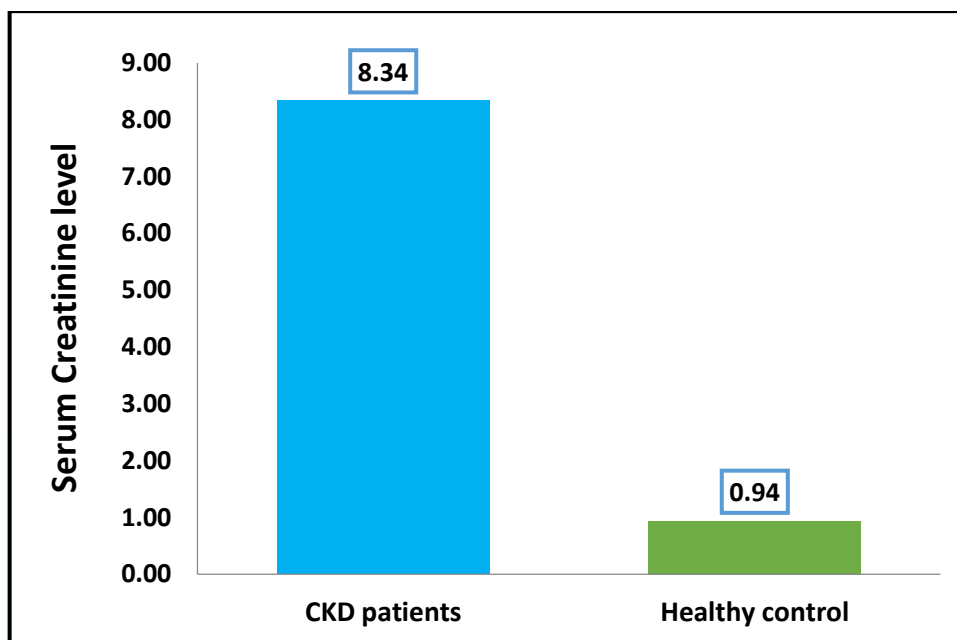


Figure (1): The means level of Creatinine in patients and control groups

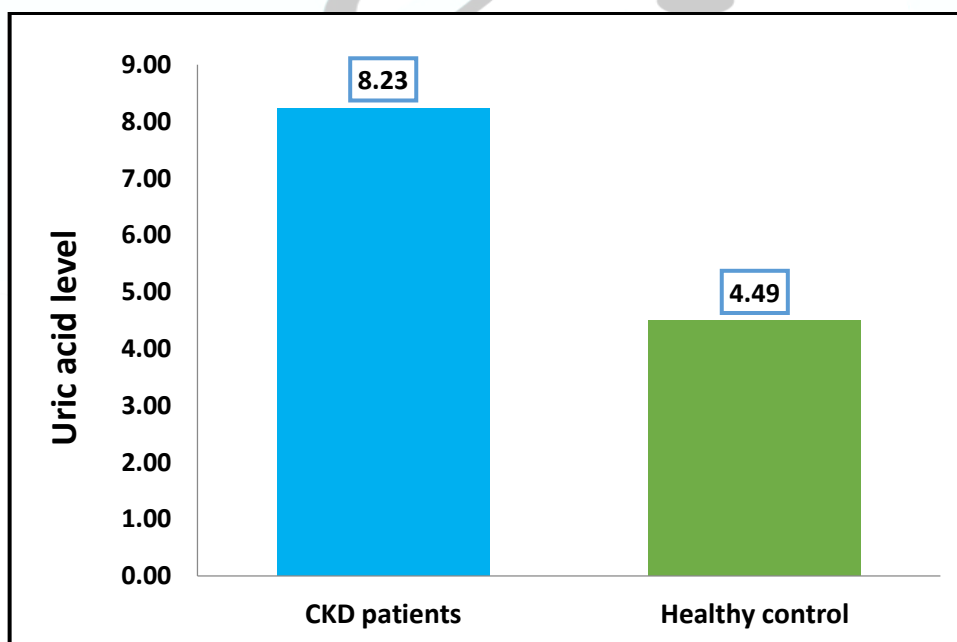


Figure (1): The means level of Uric acid in patients and control groups



Discussion

Anemia is among the most significant complications of chronic renal disease (CKD), adversely impacting life quality and increasing the morbidity and also the mortality rates. The mechanism of anemia related with CKD are complex and varied, including decreased endogenous erythropoietin production, absolute and/or functional iron deficiency, and inflammation accompanied by elevated hepcidin levels (Portoles et al.,2021).

Furthermore, several risk factors for anemia in CKD have been identified. A progressive decline in endogenous erythropoietin (EPO) levels is a major risk factor for anemia in CKD. However, other contributing factors to anemia in individuals with CKD have been described, including complete deficiency of iron is because of blood loss or impaired iron absorption, impaired iron utilization due to elevated hepcidin levels, and systemic inflammation resulting from CKD and its associated conditions. a reduced bone marrow response to EPO due to uremic toxins, a reduced red cell life span, or vitamin B12 or folic acid deficiencies (Anemii,2012) (Sanghani, and Haase,2022).

The results of the current study showed a significant decrease in the level of the hemoglobin and platelet in people patients with C.K.D. compared to healthy individuals. This decrease is primarily attributed to reduced erythropoietin production due to damage to the renal interstitial tissue, which is accountable for stimulating the bone marrow to manufacture red blood cells. Furthermore, the accumulation of uremic toxins leads to a shortened lifespan of red blood cells and inhibits platelet function (Babitt and Lin, 2012). In addition, the results revealed a paradoxical finding: a decrease in serum iron levels alongside a sharp and significant increase in ferritin levels in patients. This elevation in ferritin does not indicate iron overload but rather reflects a state of "inflammatory anemia," where ferritin acts as an acute-phase protein, rising as a result of the chronic inflammation associated with kidney failure. Inflammation leads to an increase in the hormone hepcidin, which binds iron to stores (ferritin) and prevents its use in hematopoiesis (Ganz, 2019; Hanna *et al.*, 2020). The study also recorded a



remarkable increase in urea, creatinine, and uric acid levels in patients. This elevation is a direct indicator of a decreased glomerular filtration rate (GFR) and kidney failure to eliminate nitrogenous waste. The accumulation of these wastes contributes to a toxic environment that negatively impacts all vital processes, including hematopoiesis and electrolyte balance (Webster *et al.*, 2017) (Elewa *et al.*, 2020).

Conclusion

The study demonstrates that abnormalities in hematological and immunological markers, as well as kidney function markers, are more p conspicuous in patients with chronic renal disease. Furthermore, these findings highlight the necessity of considering inflammatory markers and iron levels when diagnosing and treating chronic kidney disease patients to improve clinical outcomes.

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