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Inflammatory Immune Responses and Biochemical Disorders in Chronic Kidney Disease Patients

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Abstract

Background

Chronic kidney disease (CKD) is decline of glomerular filtration rate (GFR) and the presence of albuminuria are important risk factors for the eventual development of kidney failure, which frequently progresses over time.

Methodology

A case-control study includes 50 patients with CKD and 50 age-matches healthy from male and female and between the ages of 20 and 65 year from September 2025 to November 2025.The controls were clinically evaluated by a specialized physician. This investigation was carried out in Karbala at the Imam Al-Hussein medical city.

Results

According to the results of biochemical indicators, chronic kidney disease patients had significantly higher levels of urea and creatinine and lower levels of eGFR when compared to the control group. According to the results of immunological parameters, chronic kidney disease patients had significantly higher levels of IL-17 and IL-10. Significant negative and positive correlations between anti-inflammatory cytokine levels and renal function markers were found via correlation analysis. Strong selective power for urea (AUC=0.95), creatinine (AUC=0.91) and eGFR(AUC=0.92) and excellent diagnostic performance for IL-10 (AUC= 0.98) and IL-17 (AUC=0.97) were demonstrated by ROC analysis. The present study aims to evaluate biochemical disorders such as urea, creatinine, eGFR and inflammatory cytokines such as IL-10, IL-17 in chronic kidney disease patients and their correlation with decrease of kidney functions.

Conclusion

Higher levels of urea and creatinine and lower levels of eGFR were identified as a risk factors for kidney disease progression and higher concentrations of pro inflammatory cytokines such as IL-17 and anti-inflammatory cytokines such as IL-10 were identified as inflammatory markers for renal functions disorders in patients with CKD, also age and BMI are determinant factors for CKD.

الاستجابات المناعية الالتهابية والاضطرابات الكيموحيوية في مرضى المرض الكلوي المزمن

قاسم حمادي عبد

الخلاصة

المقدمة :مرض الكلى المزمن (CKD) هو انخفاض في معدل الترشيح الكبيبي (GFR) ووجود الألبومين في البول هما عاملان خطران مهمان لتطور الفشل الكلوي في النهاية، والذي يتقدم غالبًا مع مرور الوقت.

العينات وطرق العمل :تشمل هذه الدراسة 50 مريضًا و50 شخصًا سليمًا من اعمار متطابقة من الذكور والإناث تتراوح أعمارهم بين 20 و65 عامًا من الشهر التاسع 2025 الى الشهر الحادي عشر 2025. تم تقييم الضوابط سريرياً من قبل طبيب متخصص. تم إجراء هذا التحقيق في كربلاء في مدينة الإمام الحسين الطبية.

النتائج :وفقًا لنتائج المؤشرات البيوكيميائية، كان لدى مرضى المرض الكلوي المزمن مستويات عالية من اليوريا والكرياتينين ومستويات واطئة من معدل الترشيح الكبيبي المقدر مقارنةً بمجموعة السيطرة. وفقًا لنتائج المعايير المناعية، كان لدى مرضى المرض الكلوي المزمن مستويات عالية من الانترلوكين 17 والانترلوكين 10 مقارنة بمجموعة السيطرة. تم العثور على ارتباطات سلبية كبيرة بين مستويات السيتوكينات المضادة للالتهابات وعلامات وظائف الكلى من خلال تحليل الارتباط. تم إثبات القوة الانتقائية القوية لليوريا حيث كانت منطقة المنحني 95و0 والكرياتينين حيث كانت منطقة المنحني 91و0ومعدل الترشيح الكبيبي المقدر حيث كانت منطقة المنحني92و0 والأداء التشخيصي الممتاز للانترلوكين 17 حيث كانت منطقة المنحني 97و0والانترلوكين 10 حيث كانت منطقة المنحني 98و0 من خلال تحليل المنحني تهدف الدراسة الحالية إلى تقييم الاضطرابات الكيميائية الحيوية مثل اليوريا، الكرياتينين، معدل الترشيح الكبيبي والسيتوكينات الالتهابية مثل الانترلوكين 17 والانترلوكين 10 لدى المرضى الذين يعانون من مرض الكلى المزمن وعلاقتها بانخفاض وظائف الكلى.

الاستنتاج :تم تحديد مستويات عالية من اليوريا والكرياتينين ومستويات واطئة من معدل الترشيح الكبيبي المقدر كعوامل خطر لتقدم مرض الكلى، وتم تحديد تراكيز عالية من السيتوكينات المؤيدة للالتهابات مثل الانترلوكين 17 والسيتوكينات المضادة للالتهابات مثل الانترلوكين 10 كعلامات التهابية لاضطرابات وظائف الكلى في المرضى الذين يعانون من مرض الكلى المزمن، كما أن العمر ومؤشر كتلة الجسم هما عوامل حاسمة لمرض الكلى المزمن.

1. Introduction

Chronic kidney disease (CKD) is one of the most important public health concerns impacting human health in the twenty-first century (Levin et al., 2017). Chronic kidney disease (CKD), a condition that is on the rise, affects roughly 10–12% of individuals globally (Yan and Shao, 2024). As the body's blood filter, the kidney removes excess or harmful substances like sodium and uric acid. As the glomerular filtration rate (GFR) gradually drops in chronic kidney disease (CKD), the body will store more filtered chemicals. Recent study suggests that many of these chemicals have proinflammatory effects (Webster et al., 2017). CKD is mostly caused by three risk factors: obesity, diabetes mellitus, and hypertension. Hematological and biochemical indicators are impacted by CKD and become increasingly apparent as the disease progresses. Chronic kidney disease (CKD) and end-stage renal disease (ESRD) are known to induce a number of problems, such as hyperkalemia, metabolic acidosis, and hyperphosphatemia, because the kidneys are essential for regulating body fluids, electrolytes, and acid-base balance. These conditions can lead to serious consequences such as vascular calcification, muscular atrophy, bone-mineral problems, and death. Because changes in biochemical parameters like sodium, potassium, calcium, magnesium, and chloride can be lethal, they should be kept within physiological ranges (Hossain et al., 2017). In contrast to many other CKD consequences, hypertension can exist from the very beginning of the illness and becomes more common as GFR gradually decreases(Mitsnefes, 2012). It was projected that there were 7.6 billion people on the planet. Through the use of representative samples from 18 high-income and 14 middle- or low-income nations, the prevalence of chronic kidney disease (stages G1–G5) was estimated to be 844 million patients worldwide. Of these, 3·9 million were receiving kidney replacement treatment (i.e., kidney transplantation or maintenance dialysis) for renal failure (Jager et al., 2019) .Adults with CKD are known to have far higher rates of cardiovascular morbidity and death than the general population(Gansevoort et al., 2013). Few therapies were demonstrated to decrease the progression of chronic renal disease or lower cardiovascular risk, and there were fewer trials on chronic kidney disease than in other specialties (Baigent et al., 2017) .However, diffuse and non-occlusive arterial stiffening in adolescents and young adults with end-stage renal disease (ESRD) is more frequently caused by medial calcification and is closely linked to certain uremia-related variables, including high serum phosphate levels, long-term dialysis, and hypertension(Briet et al., 2012).

2. Materials and Methods

2.1. Study Groups and Blood Samples Collection

To determine the impact of CKD on several immunological and biochemical parameters in patients and controls, a case-control study was carried out. In this study, 50 patients and 50 healthy controls (clinically evaluated by a specialist doctor) are drawn from males and females aged between 20 and 65 years from September 2025 to November 2025. The World Health Organization (WHO) method, which is weight in kilograms divided by length in meters squared (kg/m2), was used to calculate body mass index (BMI). Using age-appropriate WHO reference criteria, participants were split into four groups according to their BMI: underweight, normal weight, overweight, and obese. BMI categories were utilized to conduct comparative and statistical studies between chronic kidney disease patients and healthy, as well as to assess relationships between BMI and immunological and biochemical variables. After drawing five milliliters of blood and putting it in disposable plane tubes, the tubes are centrifuged for ten minutes at 3000 runs per minute, allowed to clot at room temperature for thirty minutes, immediately transferred to another tube, and then frozen at -20 degrees Celsius for additional analysis. To examine biochemical and immunological markers, the serum was isolated. This study was conducted at the Imam Al-Hussein medical city in Karbala.

2.2. Parameters of Study

2.2.1. Biochemical Parameters

2.2.1.1.Urea and Creatinine

A Mindray BS-240 automated chemistry analyzer (Mindray, China) equipped with BioSystems diagnostic kits (Spain) was used to measure the levels of serum urea and creatinine. The kinetic Jaffe reaction was used to test creatinine, while the enzymatic urease-GLDH technique was used to measure urea. The reference ranges for creatinine and urea were 0.6–1.3 mg/dL and 15–45 mg/dL, respectively.

2.2.1.2.Estimated Glomerular Filtration Rate (eGFR)

The CKD-EPI equation was used to determine the estimated glomerular filtration rate (eGFR) from serum creatinine levels, taking into account factors related to age and sex to improve the accuracy of estimating the glomerular filtration rate. Normal value of eGFR ≥ 90 mL/min/1.73 m².

2.2.2. Immunological Parameters

2.2.2.1.Serum Levels of IL-10 and IL-17

Sandwich ELISA kits (Elabscience Biotechnology Co., Ltd., Wuhan, China) were used to measure the serum IL-10 and IL-17 concentrations in accordance with the manufacturer's instructions. An ELISA microplate reader (Stat Fax 2100, Awareness Technology Inc., USA) was used to measure the amounts of cytokines. Cytokine concentrations were computed using standard curves and represented in pg/mL after absorbance was measured at 450 nm. For IL-10 and IL-17, the typical reference ranges were 5–15 pg/mL and 10–35 pg/mL, respectively.

2.2.2.2. Ethical Approval

The Institutional Ethics Committee of the College of Applied Medical Science's Pathological Analysis Department accepted the study protocol. PAAMSKU/04 in 25 August 2025 is the reference number. Every participant provided written informed permission.

2.3.Statistical Analysis

One-way ANOVA was used to evaluate the data, which was displayed as mean ± SD. ANOVA IBM SPSS Program version 20 was used to analyze the data, an independent t-test was performed to compare each group's means. Pearson correlation was employed to determine relationships between variables. The chi-square (χ^2) test was used to examine relationships between categorical data, such as age, sex, and BMI categories. Pearson's correlation coefficient (r) for parametric data was used to evaluate correlations between immunological and biochemical parameters. To assess the diagnostic efficacy of biochemical and immunological markers, ROC curve analysis was performed. (McKendrick, 2003).

2.5. Inclusion Criteria

1. Age range: 20–65
2. chronic kidney disease diagnosis
3. Acceptance of involvement voluntarily

2.6. Exclusion Criteria

1. Acute kidney damage, an ongoing infection, autoimmune conditions, cancer, liver illness, pregnancy, or recent surgery
2. Using anti-inflammatory or immunosuppressive medications.

3. Results

3.1. Distribution of Chronic Kidney Disease and Healthy According to Age

Results of the current study in Table1 showed there is significant distribution (P≤0.01) of ages (20-35,36-50,51-65) years in chronic kidney disease patients compared with healthy, Table1.

Table1: Distribution of Chronic Kidney Disease and Healthy According to Age

Age	Study groups N (%)		X^2	P value	Total
	Healthy	Chronic kidney disease patients			
20-35 year	15(30%)	9(18%)	9.30	0.009	24(24%)
36-50 year	22(44%)	13(26%)			35(35%)
51-65 year	13(26%)	28(56%)			41(41%)
Total	50(100%)	50(100%)			100(100%)
Statistical significance was set at p ≤ 0.01					

3.2.Distribution of Chronic Kidney Disease Patients and Healthy According to Sex

There is significant distribution (P≤0.01) of sex (male and female) in chronic kidney disease patients compared with healthy in Table2.

Table2: Distribution of chronic kidney disease patients and healthy according to sex

Sex	Study groups N (%)		X^2	P value	Total
	healthy	Chronic kidney disease patients			
Male	18(36%)	29(58%)	4.86	0.027	47(47%)
Female	32(64%)	21(42%)			53(53%)
Total	50(100%)	50(100%)			100(100%)
Statistical significance was set at p ≤ 0.01					

3.3.Distribution of Chronic Kidney Disease Patients and Healthy According to BMI

There is significant distribution (P≤0.01) of BMI (underweight, normal weight, overweight and obese) in chronic kidney disease patients compared with healthy in Table3.

Table3: Distribution of Chronic Kidney Disease Patients and Healthy According to BMI

BMI	Study groups N (%)		x^2	P value	Total
	Healthy	Chronic kidney disease patients			
Underweight	3(6%)	7(14%)	13.4	0.004	10(10%)
Normal weight	26(52%)	10(20%)			36(36%)
Overweight	15(30%)	21(42%)			36 (36%)
Obese	5(10%)	12(24%)			17(17%)
Total	50 (100%)	50(100%)			100(100%)

3.4. Effect of Chronic Kidney Disease on Some of Biochemical Parameters

Results of biochemical parameters in Table4 showed that there is significant raise (p≤0. 01) in level of urea and creatinine in patients with chronic kidney disease compared with healthy, while notice that there is significant low (p≤0. 01) in eGFR in patients with chronic kidney disease compared with healthy.

Table4: Effect of Chronic Kidney Disease on Some of Biochemical Parameters (Means ± SD)

Parameters	Groups		P value
	Patients	Healthy	
Urea (mg/dL)	A 92.14±36.17	B 25.43±8.77	**< 0.001**
Creatinine (mg/dL)	A 2.58±0.78	B 1.36±0.45	**< 0.001**
eGFR mL/min/1.73 m²	A 47.56±18.22	B 94.49±27.16	**< 0.001**
Significant differences are represented by different letters at (p≤0. 01)			

3.5. Effect of Chronic Kidney Disease on Some of Immunological Parameters

Results of immunological parameters in Table5 appeared that there is significant increase (p≤0. 01) in level of IL-10 and IL-17 in patients with chronic kidney disease compared with healthy.

Table5: Effect of Chronic Kidney Disease on Some of Immunological Parameters (Means ± SD)

Parameters	Groups		P value
	Patients	Healthy	
IL-10 (pg/mL)	A 32.99±9.96	B 11.90±4.88	**p < 0.001**
IL-17 (pg/mL)	A 72.00±18.82	B 26.78±9.19	**p < 0.001**
Different letters represent a significant difference at (p≤0. 01)			

3.6. Correlation Coefficient Between Study Parameters (Urea, Creatinine, GFR, IL-10 And IL-17) In Chronic Kidney Disease Patients

Results of correlation in Table6 showed that there is negative significant correlation (p≤0. 01) between Urea and eGFR, urea and IL-17, while notice that there is positive significant correlation (p≤0. 05) between Creatinine and eGFR and positive significant correlation (p≤0. 01) between Creatinine and IL-17, eGFR and IL-17, IL-10 and IL-17 in patients with CKD

Table6: Correlation Coefficient Between Study Parameters (Urea, Creatinine, eGFR, IL-10 And IL-17) in Chronic Kidney Disease Patients

Variables	Urea	Creatinine	eGFR	IL-10	IL-17
Urea	1	0.063 (0.663)	−0.763** (<0.001)	−0.093 (0.522)	−0.242 (0.090)
Creatinine	0.063 (0.663)	1	0.295* (0.038)	0.044 (0.762)	0.598** (<0.001)
eGFR	−0.763** (<0.001)	0.295* (0.038)	1	0.048 (0.739)	0.488** (<0.001)
IL-10	−0.093 (0.522)	0.044 (0.762)	0.048 (0.739)	1	0.510** (<0.001)
IL-17	−0.242 (0.090)	0.598** (<0.001)	0.488** (<0.001)	0.510** (<0.001)	1

3.7. Diagnostic Performance of Urea, Creatinine, GFR, IL-10 And IL-17 Based on ROC Curve Analysis in Chronic Kidney Disease Patients and Healthy

Results in Table7 With an AUC of 0.95, urea showed outstanding diagnostic accuracy according to ROC curve analysis, demonstrating a very good capacity to differentiate between patients and controls. Urea demonstrated excellent clinical

dependability with a sensitivity of 98% and a specificity of 86% at a cut-off value of 33.50 mg/dL. Similarly, with an AUC of 0.91, creatinine demonstrated acceptable diagnostic accuracy. Creatinine showed good specificity (84%) and sensitivity (88%) with a cut-off value of 1.58 mg/dL, suggesting its utility as a diagnostic marker. AUC of eGFR is 0.08 because AUC<0.5 was obtained via ROC analysis since eGFR declines as the disease progresses. In order to reflect the actual diagnostic performance, the inverse AUC (1−AUC) was computed, hence validating eGFR's potent discriminative ability, therefore AUC of eGFR become 0.92 with very good sensitivity (90%) and good specificity (82%) at a cut-off value of 84 mL/min/1.73 m², on the other hand, eGFR showed very good diagnostic ability. While having a 98% very high sensitivity, IL-10 demonstrated excellent diagnostic efficacy with an AUC of 0.98, indicating excellent clinical dependability. Its diagnostic efficacy is further limited by good specificity (86%) at the chosen cut-off value 16.85 pg/mL. AUC of 0.97, IL-17 showed excellent diagnostic accuracy. With very good sensitivity of 98% and a specificity of 90% at a cut-off value of 37.50 pg/mL, IL-17 may be a valuable inflammatory biomarker for illness classification.

Table7: Diagnostic Performance of Urea, Creatinine, eGFR, IL-10 And IL-17 Based on ROC Curve Analysis in Chronic Kidney Disease Patients and Healthy

Parameters	AUC	Sensitivity (%)	Specificity (%)	Cut-off
Urea	0.95	98%	86%	33.50 mg/dL
Creatinine	0.91	88%	84%	1.58 mg/dL
eGFR	0.92	90%	82%	84 mL/min/1.73 m²
IL-10	0.98	98%	86%	16.85 pg/mL
IL-17	0.97	98%	90%	37.50 pg/mL

3.8. Estimated Glomerular Filtration Rate (eGFR) According to Sex and Age in Chronic Kidney Disease Patients

There is gradual decrease in level of eGFR with increasing age, also there is significant difference (p≤0. 05) in GFR between male and female in chronic kidney disease patients as shown in Table8.

Table8: Estimated Glomerular Filtration Rate (eGFR) According to Sex and Age in Chronic Kidney Disease Patients

Age	Sex		X2	p-value
	Male	Female		
20-35 year	34 mL/min/1.73 m²	25 mL/min/1.73 m²	**6**	**0.01**
36-50 year	31mL/min/1.73 m²	23 mL/min/1.73 m²		
51-65 year	28mL/min/1.73 m²	21 mL/min/1.73 m²		

3.9. Relationship Between Sex and BMI in Chronic Kidney Disease Patients

Results in Fig.1. showed that there is significant increase in percent of BMI in underweight and overweight in male compared with female, while notice that there is significant decrease in normal weight and obese in male compared with female in chronic kidney disease patients.

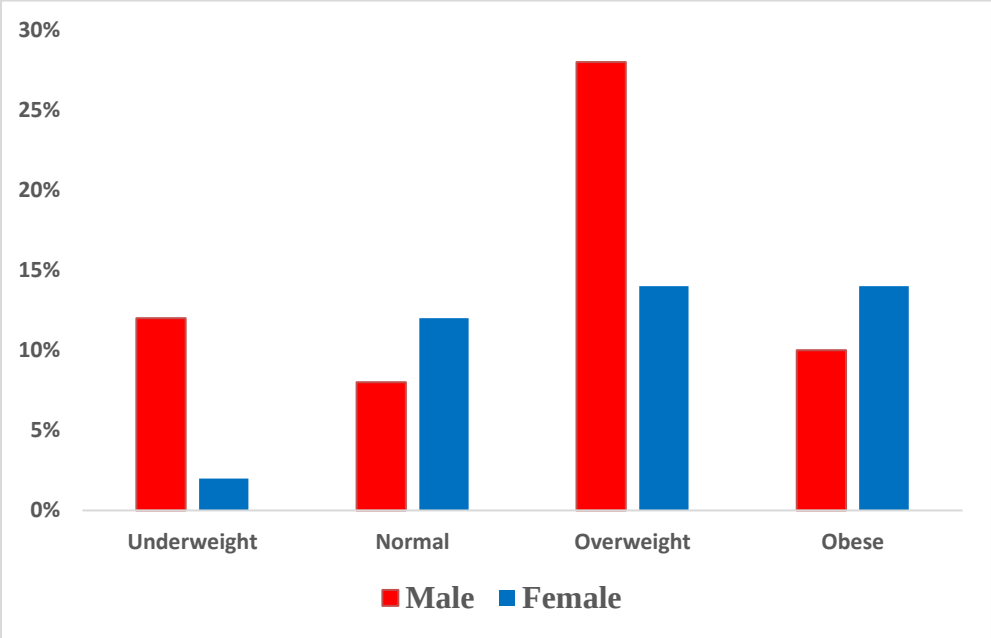

Figure1: Relationship Between Sex and BMI in Chronic Kidney Disease Patients

3.10. Relationship Between Age and BMI in Chronic Kidney Disease Patients

The results in Fig.2. showed that there is significant decrease in percent of BMI in underweight, overweight and obese compared normal weight in (20-35) year, while noted significant increase in percent of BMI in underweight compared with other ages in (36-50) year, while notice significant increase in percent of BMI in overweight and obese compared with underweight and normal weight in (51-65) year in chronic kidney disease patients.

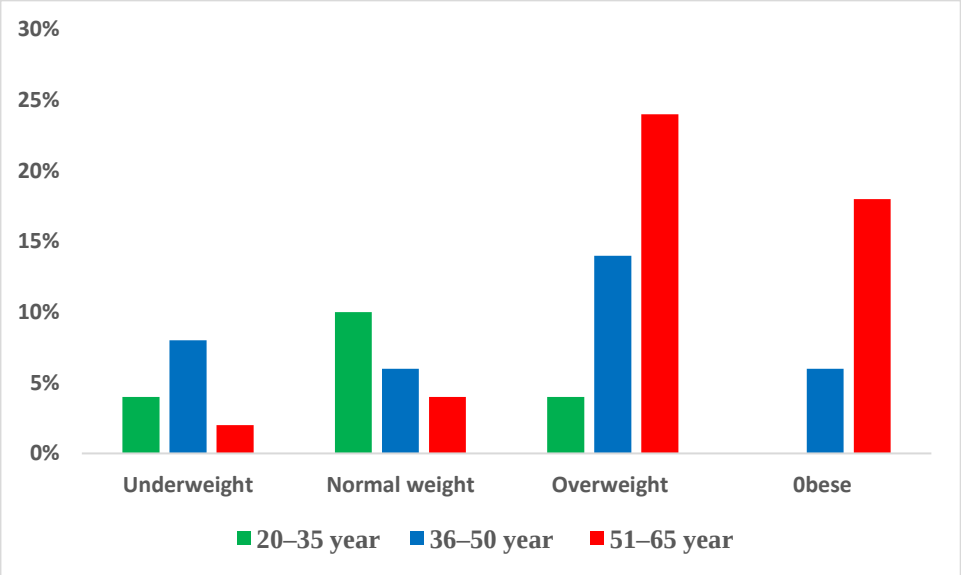

Figure2: Relationship Between Age and BMI in Chronic Kidney Disease Patients

3.11. Association Between Mean Urea, Creatinine, eGFR, IL-10 And IL-17 Level and Sex in Chronic Kidney Disease Patients

The results in Fig.3 showed that there is significant increase in mean of Urea, Creatinine, eGFR, IL-10 and IL-17 in male compared with female in chronic kidney disease patients.

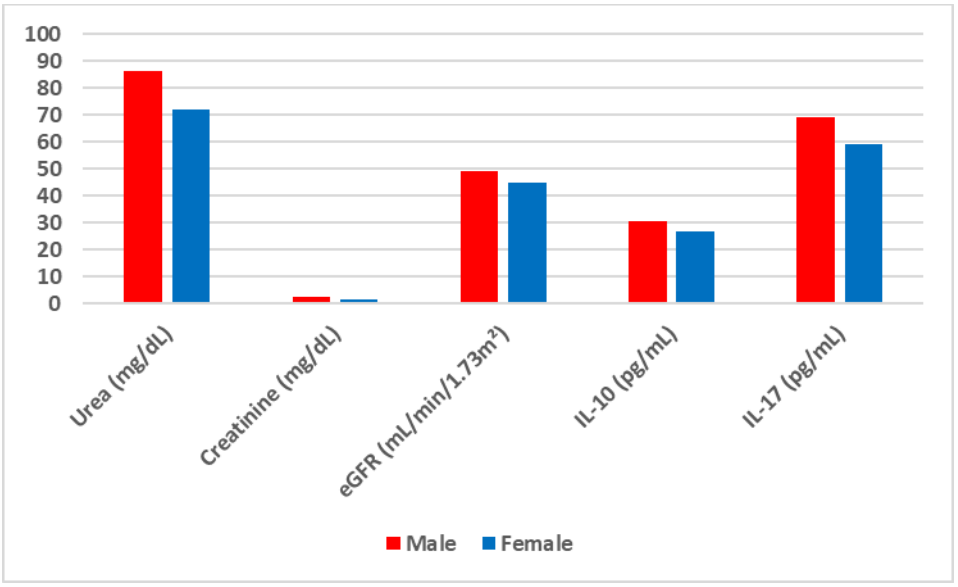

Figure3: Association Between Mean Urea, Creatinine, eGFR, IL-10 and IL-17 Level and Sex in Chronic Kidney Disease Patients

3.12. Association Between Mean Urea, Creatinine, eGFR, IL-10 And IL-17 Level and Age In Chronic Kidney Disease Patients

The results in Fig.4. showed that there is gradual increase in mean of urea, creatinine, IL-10 and IL-17 with progressive of age, while notice gradual decrease in mean of eGFR with increasing of age in CKD patients.

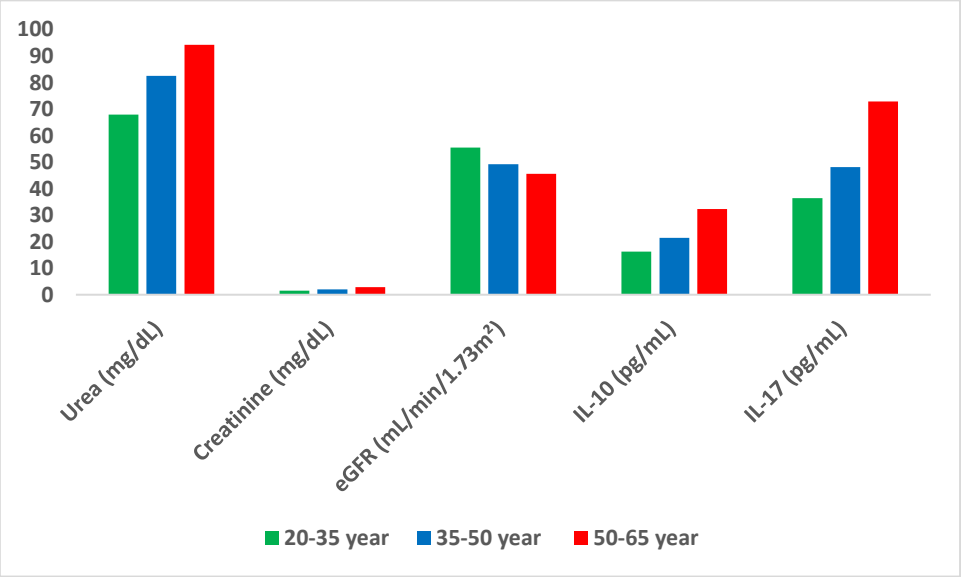

Figure4: Association Between Mean Urea, Creatinine, eGFR, IL-10 And IL-17 Level And Age In Chronic Kidney Disease Patients

3.13. Association Between Mean Urea, Creatinine, eGFR, IL-10 And IL-17 Level And BMI in Chronic Kidney Disease Patients

The results in Fig.5. showed that there is gradual increase in mean of urea, creatinine, IL-10 and IL-17 level with increasing BMI, while notice gradual decrease in eGFR with increasing BMI in chronic kidney disease patients.

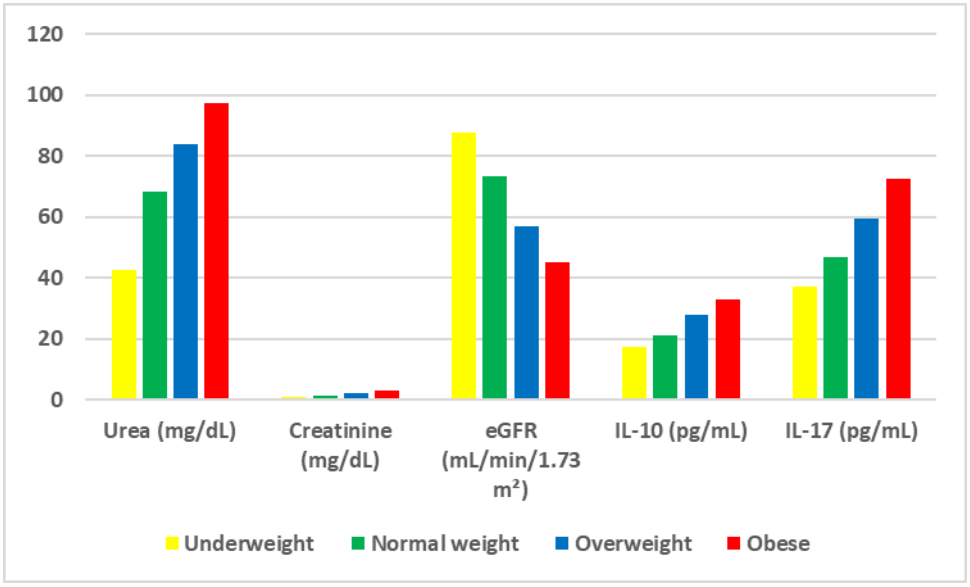

Figure5: Association Between Mean Urea, Creatinine, eGFR, IL-10 and IL-17 Level And BMI In Chronic Kidney Disease Patients

3.14. Percentage Some Of Clinical Symptoms and Signs in Chronic Kidney Disease Patients

The results in Fig.6. showed that there is significant distribtion in percent of clinical symptoms and signs such as Fatigue and weakness, Edema, Hypertension, Oliguria, Anorexia, Nausea and vomiting, Pruritus , Dyspnea, Anemia and Bone or joint pain that formed (74%), (62%), (78%), (34%), (50%), (36%), (26%), (30%) (66%) and (20%) respectively in chronic kidney disease patients.

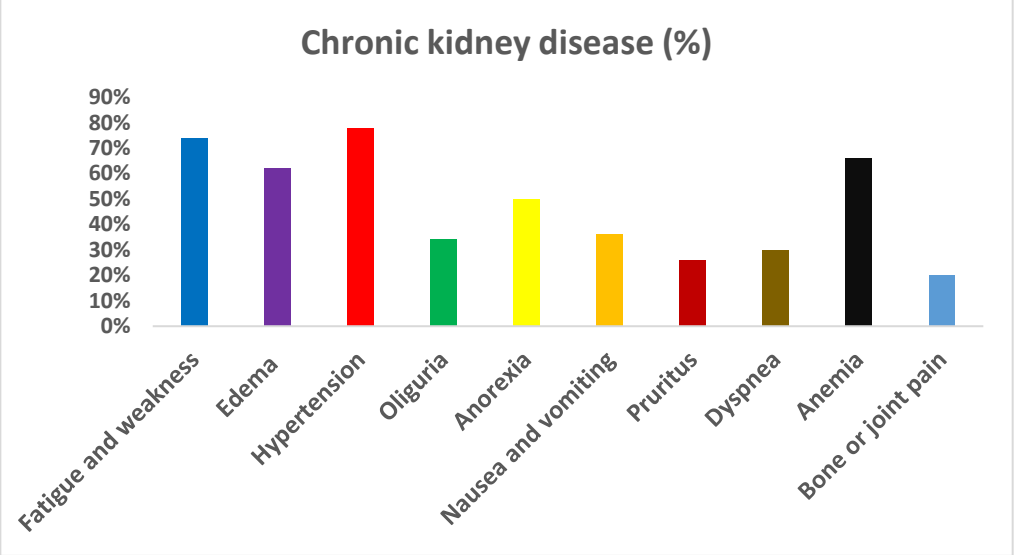

Figure6: Percentage Some of Clinical Symptoms and Signs in Chronic Kidney Disease Patients

3.15. ROC Curve Showing the Diagnostic Performance of Urea, Creatinine, eGFR, IL-10 and IL-17 in Chronic Kidney Disease Patients and Healthy

Results in Fig.7. of urea showed outstanding diagnostic accuracy according to ROC curve analysis, demonstrating excellent capacity to differentiate between patients and controls. Urea demonstrated excellent clinical dependability with excellent sensitivity of 98% and a specificity of 86%. Similarly, creatinine demonstrated acceptable diagnostic accuracy. Creatinine showed specificity (84%) and good sensitivity (88%). eGFR showed good diagnostic ability, good sensitivity (90%) and specificity (82%). IL-10 demonstrated excellent diagnostic efficacy having a 98% very high sensitivity. Its diagnostic efficacy is further limited by the specificity (86%). IL-17 showed excellent diagnostic accuracy. With very good sensitivity of 98% and good specificity of 90%, IL-17 may be a valuable inflammatory biomarker for illness classification.

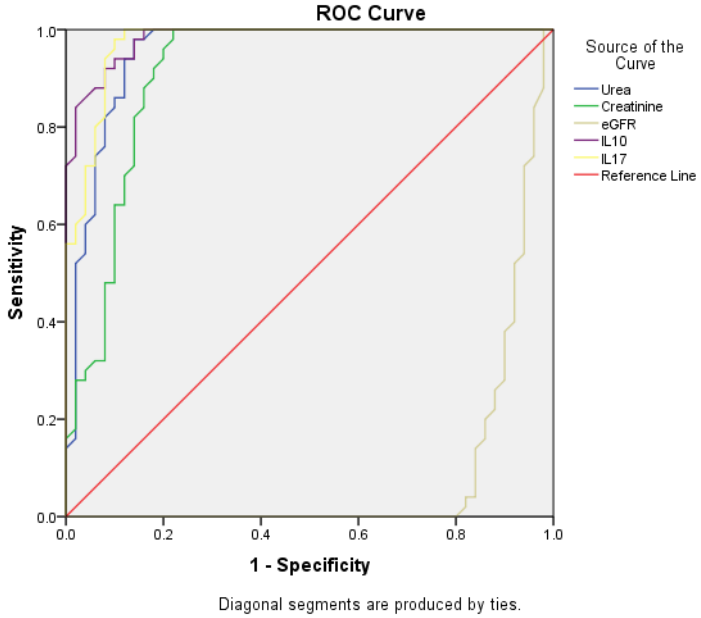

Figure6: ROC Curve Showing the Diagnostic Performance of Urea, Creatinine, eGFR, IL-10 And IL-17 In Chronic Kidney Disease Patients and Healthy

4.Discussion

Males and females with chronic renal disease have significantly higher urea and creatinine levels than the control groups, according to a case control study. These findings corroborated those of (Mehdi, 2011), who noted that individuals with chronic renal failure had higher serum creatinine levels, which are commonly understood to be only indicative of renal function. Additionally,(Sathishbabu and Suresh, 2012) shown a marked rise in the amounts of creatinine and urea in chronic renal disease. Urea, a consequence of protein metabolism, is commonly used in clinical settings as a proxy for the degree of chronic renal disease and the adequacy of dialysis. For a long time, urea was believed to be a relatively inactive, safe substance. Numerous investigations have shown that urea is a direct and indirect uremic toxin (Massy et al., 2016). Urea and creatinine levels rise as a result of nitrogenous wastes building up in the blood due to the kidneys' inability to eliminate them from the blood in renal failure (Norris, 2019). Moreover, elevated urea concentrations in endothelial cell progenitor cultures have been connected to accelerated senescence and the generation of free radicals (D'Apolito et al., 2017). Because the kidneys cannot eliminate urea and creatinine from the body, they gradually return to the bloodstream. As the kidneys fail in a series of stages, blood toxin levels increase. End-stage renal disease (ESRD) is the term used to describe a condition in which renal function is less than 10%. One could consider ESRD to be a more severe kind of CKD. The condition is characterized by a bilateral, chronic, gradual degradation of the kidney's functional units, or nephrons (Maleki et al., 2021). Urea transporter inhibition was associated with improvements in heart function and reductions in myocardial fibrosis and hypertension in a study conducted on nephrectomized mice (Kuma et al., 2020). In the present study there are significant decrease in eGFR in CKD patients in comparison with control, these results accompanied with(Gupta et al., 2012) inflammatory cytokines also build up as a result of the lower eGFR. Reduced urine phosphate excretion and anomalies in the control of calcium and parathyroid hormones are the outcomes of decreased eGFR (Inker et al., 2019). CKD–mineral bone abnormalities is the collective term for these anomalies (Ketteler et al., 2025). The current study appeared that there are significant increase in IL-10 in CKD patients in contrast to control, These findings were supported by(Jin et al., 2013), they discovered that IL-10 loss exacerbated renal fibrosis and severe renal inflammation in mice with unilateral ureteral obstruction (UUO).(Andres-Hernando et al., 2017) shown that IL-10 could inhibit the local or systemic inflammatory response triggered by ischemia AKI. found that the activation of the angiotensin-II type 2 receptor in mice with acute renal damage brought on by endotoxin infection. (AT2R) raised IL-10, an anti-inflammatory cytokine. The kidneys showed a considerable decrease in TNF-α and IL-6, indicating that IL-10 plays a function in preventing inflammation and protecting the kidneys from AKI. (Soranno et al., 2014) based on the anti-fibrotic and anti-inflammatory qualities of IL-10 in CKD, employed injectable and self-assembling Dock-and-Lock hydrogels to deliver IL-10 to the kidneys of mice with unilateral ureteral obstruction (UUO), which were sustained in the mice for 30 days. Another study shown that IL-10 greatly decreased kidney ischemia-reperfusion (IR) damage by inhibiting the inflammatory mediators TNF-α and IL-6. Therefore, it was expected that IL-10 would be an important target for the therapy of IR damage (Wan et al., 2014). The current study appeared that there are significant raise in IL-17 in CKD patients in comparison with control, the increase in concentrations of IL-17 may be associated with medication resistance, disease severity, or disease phenotype, according to our findings and a prior investigation that examined interleukins in relation to lupus nephritis histology and therapeutic response (Zickert et al., 2015).Numerous illnesses, including metabolic syndrome (Mohammadi et al., 2017)and various forms of cancer(Sahana et al., 2017), have been linked to elevated interleukin levels. Similarly, the aforementioned cause most likely contributed to the current study's higher serum IL-17 and IL-2 concentrations(Akchurin and Kaskel, 2015). The pathophysiological balance between pro- and anti-inflammatory cytokines determines the overall effect of an inflammatory response(Akchurin and Kaskel, 2015). Both acute and chronic renal disorders activate these cytokines(Koelman et al.,

2019). The most well-known pro-inflammatory cytokines include tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), interleukin-2 (IL-2), interleukin-6 (IL-6), interleukin-8 (IL-8), and interleukin-17 (IL-17). (Fasoulakis et al., 2018).

Conclusion

Higher levels of urea and creatinine and lower levels of eGFR were identified as a risk factors for kidney disease progression and higher concentrations of pro inflammatory cytokines such as IL-17 and anti-inflammatory cytokines such as IL-10 were identified as inflammatory markers for renal functions disorders in patients with CKD, also high percents of patients with CKD on older ages and prevalence of CKD with overweight patients indicator on decrease in number of nephrons, increase in glomerulosclerosis and lack ability of kidney in regulation of fluids and salts, hyperfiltration of glomeruli and development of obesity-related glomerulopathy therefore age and BMI are determinant factors for CKD. According to sensitivity and specificity of urea is considered power diagnostic parameter for CKD patients. Finally disorders in biochemical and immunological markers lead to metabolic abnormalities in kidney and sometimes death in patients with CKD, therefore early identification for these disorders lead to reduce renal complications.

Conflicts of Interest

No conflicts of interest are disclosed by the authors.

References

AKCHURIN, O. M. & KASKEL, F. 2015. Update on inflammation in chronic kidney disease. *Blood purification,* 39**,** 84-92.

ANDRES-HERNANDO, A., OKAMURA, K., BHARGAVA, R., KIEKHAEFER, C. M., SORANNO, D., KIRKBRIDE-ROMEO, L. A., GIL, H.-W., ALTMANN, C. & FAUBEL, S. 2017. Circulating IL-6 upregulates IL-10 production in splenic CD4+ T cells and limits acute kidney injury–induced lung inflammation. *Kidney international,* 91**,** 1057-1069.

Baigent C, Herrington WG, Coresh J, et al. 2017.Challenges inconducting clinical trials in nephrology: conclusions from a Kidney Disease-Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int, 92: 297–305.

BRIET, M., BOUTOUYRIE, P., LAURENT, S. & LONDON, G. M. 2012. Arterial stiffness and pulse pressure in CKD and ESRD. *Kidney international,* 82**,** 388-400.

D'Apolito M, Colia AL, Lasalvia M et al. 2017. Urea-induced ROS accelerate senescence in endothelial progenitor cells. Atherosclerosis,263: 127–136.

FASOULAKIS, Z., KOLIOS, G., PAPAMANOLIS, V. & KONTOMANOLIS, E. N. 2018. Interleukins associated with breast cancer. *Cureus,* 10**,** e3549.

FATIMA, N., PATEL, S. & HUSSAIN, T. 2021. Angiotensin AT2 receptor is anti-inflammatory and reno-protective in lipopolysaccharide mice model: role of IL-10. *Frontiers in pharmacology,* 12**,** 600163.

GANSEVOORT, R. T., CORREA-ROTTER, R., HEMMELGARN, B. R., JAFAR, T. H., HEERSPINK, H. J. L., MANN, J. F., MATSUSHITA, K. & WEN, C. P. 2013. Chronic kidney disease and cardiovascular risk: epidemiology, mechanisms, and prevention. *The Lancet,* 382**,** 339-352.

GUPTA, J., MITRA, N., KANETSKY, P. A., DEVANEY, J., WING, M. R., REILLY, M., SHAH, V. O., BALAKRISHNAN, V. S., GUZMAN, N. J. & GIRNDT, M. 2012. Association between albuminuria, kidney function, and inflammatory biomarker profile in CKD in CRIC. *Clinical journal of the American Society of Nephrology,* 7**,** 1938-1946.

HOSSAIN, M. S., ZAMAN, K. M. A. U., AMIN, M. N., AHAMMED, R., HAQUE, M. E., IBRAHIM, M., HAKIM, M. L., MOHAMMAD, D., KABIR, M. A. & ARMAN, M. S. I. 2017. Association of biochemical parameters with renal functions of end stage renal disease (ESRD) patients of Bangladesh. *Journal of Bioanalysis & Biomedicine,* 9.

Inker LA, Grams ME, Levey AS, et al. 2019. Relationship of estimated GFR and albuminuria to concurrent laboratory abnormalities: an individual participant data meta-analysis in a global consortium Am J Kidney Dis, 73: 206–17.

Jager KJ, Kovesdy C, Langham R, Rosenberg M, Jha V, Zoccali C. A. 2019.single number for advocacy and communication-worldwide more than 850 million individuals have kidney diseases.Nephrol Dial Transplant; 34: 1803–05.

JIN, Y., LIU, R., XIE, J., XIONG, H., HE, J. C. & CHEN, N. 2013. Interleukin-10 deficiency aggravates kidney inflammation and fibrosis in the unilateral ureteral obstruction mouse model. *Laboratory investigation,* 93**,** 801-811.

Ketteler M, Evenepoel P, Holden RM, et al. 2025.Chronic kidney diseasemineral and bone disorder: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int, 107: 405–23.

KOELMAN, L., PIVOVAROVA-RAMICH, O., PFEIFFER, A. F., GRUNE, T. & ALEKSANDROVA, K. 2019. Cytokines for evaluation of chronic inflammatory status in ageing research: reliability and phenotypic characterisation. *Immunity & Ageing,* 16**,** 11.

Kuma A, Wang XH, Klein JD et al. 2020. Inhibition of urea transporter ameliorates uremic cardiomyopathy in chronic kidney disease. FASEB J, 34: 8296–8309.

LEVIN, A., TONELLI, M., BONVENTRE, J., CORESH, J., DONNER, J.-A., FOGO, A. B., FOX, C. S., GANSEVOORT, R. T., HEERSPINK, H. J. & JARDINE, M. 2017. Global kidney health 2017 and beyond: a roadmap for closing gaps in care, research, and policy. *The Lancet,* 390**,** 1888-1917.

Massy ZA, Pietrement C, Touré F. 2016. Reconsidering the lack of urea toxicity in dialysis patients. Semin Dial,29: 333–337.

MALEKI, R., JAHROMI, A. M., MOHAGHEGH, S., REZVANTALAB, S., KHEDRI, M. & TAYEBI, L. 2021. A molecular investigation of urea and creatinine removal in the wearable dialysis device using Two-Dimensional materials. *Applied Surface Science,* 566**,** 150629.

MCKENDRICK, J. H. 2003. 24 Statistical Analysis using SPSS. *Key Methods in Geography***,** 425.

MEHDI, W. A. 2011. Determination of resistin and several antioxidants in sera of patients with chronic renal failure. *Australian Journal of Basic and Applied Sciences,* 5**,** 196-203.

MITSNEFES, M. M. 2012. Cardiovascular disease in children with chronic kidney disease. *Journal of the American Society of Nephrology,* 23**,** 578-585.

MOHAMMADI, M., GOZASHTI, M. H., AGHADAVOOD, M., MEHDIZADEH, M. R. & HAYATBAKHSH, M. M. 2017. Clinical significance of serum IL-6 and TNF-α levels in patients with metabolic syndrome. *Reports of biochemistry & molecular biology,* 6**,** 74.

NORRIS, T. 2019. *Porth's Essentials of Pathophysiology*, Lippincott Williams & Wilkins.

SAHANA, K. R., AKILA, P., PRASHANT, V., CHANDRA, B. S. & SUMA, M. N. 2017. Quantitation of vascular endothelial growth factor and interleukin-6 in different stages of breast cancer. *Reports of biochemistry & molecular biology,* 6**,** 33.

SATHISHBABU, M. & SURESH, S. 2012. A study on correlation of serum prealbumin with other biochemical parameters of malnutrition in hemodialysis patient. *Int J Biol Med Res,* 3**,** 1410-1412.

SORANNO, D. E., LU, H. D., WEBER, H. M., RAI, R. & BURDICK, J. A. 2014. Immunotherapy with injectable hydrogels to treat obstructive nephropathy. *Journal of Biomedical Materials Research Part A,* 102**,** 2173-2180.

WAN, X., HUANG, W. J., CHEN, W., XIE, H.-G., WEI, P., CHEN, X. & CAO, C.-C. 2014. IL-10 deficiency increases renal ischemia-reperfusion injury. *Nephron Experimental Nephrology,* 128**,** 37-45.

WEBSTER, A. C., NAGLER, E. V., MORTON, R. L. & MASSON, P. 2017. Chronic kidney disease. *The lancet,* 389**,** 1238-1252.

YAN, Z. & SHAO, T. 2024. Chronic inflammation in chronic kidney disease. *Nephron,* 148**,** 143-151.

ZICKERT, A., AMOUDRUZ, P., SUNDSTRöM, Y., RöNNELID, J., MALMSTRöM, V. & GUNNARSSON, I. 2015. IL-17 and IL-23 in lupus nephritis-association to histopathology and response to treatment. *BMC immunology,* 16**,** 7.