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Indication of laboratory parameters for kidney diseases at King Fahad Medical City, Riyadh, Saudi Arabia

Yousef Sultan Alobaisi[1*], Firoz Anwar[2], Khaled Dabour[3], Khalid Almatham[4], Sultan Alshahrani[1], Khalid A. Alzahrani[5] Yasir Awad Ahmed[1], Maher Mohammed Alobaysi[6]

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ABSTRACT

End-stage renal disease (ESRD), chronic kidney disease (CKD), and acute kidney failure (AKF) are serious global health issues that contribute to high sickness and death rates. This King Fahad Medical City study investigated the importance of reliable biomarker monitoring and early treatment to enhance patient outcomes. 220 people had their serum levels of albumin, C-reactive protein (CRP), urea, creatinine, salt and phosphate assessed. Creatinine ($\chi^2 = 60.73$, $p < 0.001$), urea ($\chi^2 = 48.66$, $p < 0.001$), phosphate ($\chi^2 = 19.20$, $p = 0.004$), sodium ($\chi^2 = 14.10$, $p = 0.029$) and body weight ($\chi^2 = 13.24$, $p = 0.039$) showed significant differences across diagnostic groups, according to the Kruskal-Wallis test. Significant activity was indicated by the highest CRP values (103.9 mg/L; 95% CI: 83.9–123.9 mg/L) in AKF patients, whereas the highest average creatinine levels (428.6 µmol/L; 95% CI: 330.0–527.2 µmol/L) were seen in ESRD patients. The above results show that every disease has an unique biomarker signature. Also a gender study revealed that, on average, male patients weighed 7.29 kg heavier than female patients ($t = 2.739$, $p = 0.007$). The study shows that kidney-related illnesses may be effectively treated with biomarker-based diagnostics. Important biomarkers for CKD, ESRD and AKF detection include creatinine, urea, CRP and sodium. This highlights how helpful these indicators are for supporting timely diagnosis, improving clinical intervention techniques and directing customized treatment plans.

INTRODUCTION

Chronic kidney disease (CKD) and end-stage kidney disease (ESRD) continue to be major global health challenge, significantly contributing to the overall burden of morbidity, mortality, and the cost of health care with CKD affecting approximately 13.4% of the worldwide population which together is estimated to be over 700 million people living with some form of kidney disease (Kovesdy, 2022). ESRD Prevalence varies but is increasing, with approximately 2 million people currently on dialysis and expected to increase in the coming years (Filipska *et al.,* 2021). The high and growing rates of diabetes mellitus, hypertension and the aging population are the main causes of the worrying rise in kidney disease frequency, according to recent epidemiological data (Major *et al.,* 2018; Webster *et al.,* 2017). Chronic kidney disease (CKD) is the third most commonly seen and biggest disorder globally, behind heart disease and cancer, according to clinical data analysis (Jiang *et al.,* 2023). According to Al-Sayyari and Shaheen (2011), the high incidence of risk factors such as obesity and metabolic syndrome worsens the increasing rate of renal disease in Saudi Arabia.

Reducing the burden of illness still demands thorough analysis of renal functions, timely identification of kidney disease, and progress in the disease. Indicators such as serum creatinine and urea are crucial to conventional methods. A increase in serum creatinine, a sign of a low glomerular filtration rate (GFR), remains one of the most significant markers of renal impairment (Wang *et al.,* 2019). However, these standard markers are either too general or too specific to identify the kind of kidney damage or in the early stages of the illness (Mizdrak *et al.,* 2022). Therefore, the need for stronger biomarker panels is growing in order to provide rapid, accurate, sensitive and comprehensive diagnostic medical information.

Between 2022 and 2024, new biomarkers and the improvement of current ones have become more popular in an effort to improve diagnostic and prognostic capacities. According to Gremese *et al.* (2023), there is potential for improving the detection of renal illness by using indicators of inflammation, problems with electrolytes and nutritional status. Due to their high relationship with the course of the illness and risk for heart disease, biomarkers that show inflammation and mortality in patients with CKD and ESRD, including C-reactive protein (CRP), are now among the most significant (Li *et al.,* 2023; Stenvinkel *et al.,* 2021).

Electrolyte issues involving sodium, phosphate, calcium and albumin are among the primary causes of CKD

[1] King Fahad Medical City, Pathology and Clinical Laboratory Medicine Administration, Riyadh, 11525, Saudi Arabia
[2] King Abdulaziz University, Department of Biochemistry, Faculty of Science, Jeddah, Saudi Arabia
[3] Tanta University, Department of Entomology, Faculty of Science, Tanta, 31527, Egypt
[4] King Fahad Medical City, Department of Nephrology, Riyadh, 11525, Saudi Arabia
[5] King Fahad Medical City, Department of Electronic Medical Records (EMR), Riyadh, Saudi Arabia
[6] King Fahad Medical City, Simulation Development Department, Academic and Training Affairs, Riyadh, 11525, Saudi Arabia
[*] Corresponding author's e-mail: yalobaisi@kfmc.med.sa

impacts, including mineral and bone diseases and cardiovascular diseases. As chronic kidney disease (CKD) is becoming more common and has a significant impact on healthcare systems, better methods of diagnosis and treatment must be developed. Early identification, treatment of underlying causes and secondary prevention measures can stop or even prevent the development of the condition (Bektay *et al.,* 2024). More complex methods are encouraged by recent KDIGO guidelines, which stress the active continual tracking of these biomarkers and concentrate on therapy based on trends rather than single values (Valson *et al.,* 2020). Despite the guidelines, there is a lack of more thorough research that look at how kidney disease handles change when markers alter in a clinical environment and commitment to these recommendations differs.

This study addresses explicitly these existing limitations by examining and profiling several critical biomarkers, including creatinine, urea, C-reactive protein (CRP), albumin, sodium, calcium, and phosphate in patients with chronic kidney disease stages 3 to 5, end stage renal disease, acute kidney failure, acute nephritic syndrome, chronic renal failure-associated anemia, and septic shock. The primary goal of this study is to identify distinct patterns and variations in indicators associated with specific renal disorders, thereby aiding in the further refinement of diagnostic precision, tailoring management levels, and optimizing clinical outcomes for patients.

The primary hypothesis is that there is a notable variation in biomarkers among different renal disorders, corresponding to distinct underlying pathophysiological processes. In addition, we also hypothesize that systematically profiling biomarkers will add more value in predicting superior diagnostic and prognostic information compared to traditional isolated biomarker assessments.

The study's findings are meant to help individuals by providing explanation on some choices regarding the implementation of early treatment interventions, the customization of therapies, and the use of pertinent biomarker trends and variations across various kidney disease types to track the progression of the disease. In the end, these discoveries may lead to better clinical procedures and established care models for kidney disease management, which would greatly improve nephrology patient care and results.

LITERATURE REVIEW

Chronic kidney disease (CKD) poses an increasingly prevalent global health concern, creating problems for healthcare systems due to the chronic health subjects, the costly nature of treatment, and progressive deterioration of renal functions (Kuo & Chapman, 2020; Major *et al.,* 2018). Kidneys are of great physiological homeostasis maintenances through removing metabolic waste like urea, creatinine, uric acid, and regulates fluid volune and sodium level, serum osmolality and secretes hormones like erythropoietin, vitamin D, and renin (Okoro & Farate, 2019; Webster *et al.,* 2017).

The increase prevalence of chronic kidney disease (CKD) is closely associated with the elderly population alongside the increase in chronic illnesses, such as diabetes and hypertension which are May risk factors for CKD progression (Major *et al.,* 2018). Recently some studies have highlighted lifestyle factors such as obesity and smoking towards the development and progression of CKD (Li *et al.,* 2023), Such factors lead to a higher population of patients progressing towards CKD stage 5 which necessitates renal replacement therapies, such as dialysis or transplantation (Schrauben *et al.,* 2022). The economic burden of population suffering from CKD is considerable because of the expenditures associated with its management and complications (Zhang & Parikh, 2019).

Moreover, latest insights in nephrology emphasize the importance of proactive chronic kidney disease (CKD) management, including early diagnosis and stratified interventions. This includes the new digital health assets and technologies that support enhanced patient engagement and interface as well as newer biomarkers for prompt diagnosis (Chen *et al.,* 2021; Wang *et al.,* 2019; Zhang & Parikh, 2019). The interconnections between diverse risk factors and various biomarkers are complex, and their understanding helps design efficient control methods for CKD and alleviating its global health challenges.

As CKD progresses, its complications widen systemically, foremost in importance is CKD-Mineral Bone Disorder (CKD-MBD). Along with bone and vascular problems CKD-MBD is caused by disruptions in the metabolism of calcium, phosphate, parathyroid hormone (PTH) and vitamin D (Valson *et al.,* 2020). The burden of illness and death increases significantly by these chronic alterations. Hence, their continuous monitoring is important because of the precision needed.

Management of CKD-MBD is directed towards managing each facet of the enduring mineral bone relationship. Recent publications focus more on the need for cardiovascular complication and bone disease detection and intervention at earlier stages (Kuo & Chapman, 2020). In CKD patients, serum creatinine and urea are used to track electrolytes such calcium, phosphate, potassium and sodium as well as kidney function (Wang *et al.,* 2019). Also, C-reactive protein (CRP), which is more prominent in chronic kidney disease (CKD) and is associated with increased cardiovascular risk, can be employed as a sign of inflammation (Stenvinkel *et al.,* 2021). The chances of renal illnesses, cardiovascular problems and disease progression are all closely associated with high levels of CRP (Li *et al.,* 2023; Tang *et al.,* 2018).

Another recent emphasis has been placed on the value of albumin as more than a nutritional status measure but as a prognostic marker of patients with renal disease. In case of CKD, serum albumin levels are decreased on account of malnutrition and inflammation, which are risk factors closely related to poor clinical outcomes, higher mortality rates, and poor life expectancy (Gremese *et al.,* 2023).

Earlier studies have led to progress regarding biomarkers'

usefulness in renal diseases. A study by Dublin Acute Biomarker Group Evaluation (DAMAGE) was done as proof of progressive potential of different urinary biomarkers in critically ill patients at ICU as predictor of acute kidney injury (AKI). Urinary markers such as NGAL, cystatin C, KIM-1 and albumin were able to predict progression of AKI seven days after ICU admission, which is an important prognosticator as well as a marker associated with earlier development of AKI (McMahon *et al.,* 2019). Similarly, another researcher performed follow-up secondary analysis also found interleukin-18, NGAL, cystatin C and monocyte chemotactic protein 1 to be important predictors of progression to AKI with the consequences of AKI. This underscores the possibility of these biomarkers to be used in clinical risk stratification (Duff *et al.,* 2022).

Inflammatory markers such as CRP have been emphasized by different studies to be related to disease processes beyond inflammatory markers. For example, this second evidence adds to the fact that CRP is more than a passive marker and contributory to renal pathology since it confirmed its role in preserving insulin resistance, metabolic disorders and renal fibrosis (Tang *et al.,* 2018).

While a lot work has been done for the progression of biomarker research to highlight their usefulness in the early detection of kidney disease, there still remain substantial gaps in research especially for diverse populations of kidney diseases. Few prior attempts have been made to only on single biomarker and did not take into account a multifaceted approach. In addition to this, many of the existing literature lacks essential cross comparative studies on multiple renal diseases and therefore the specificity and clinical impact of the diagnosis remains relatively lacking. Still, limitations in precision medicine due to deficiency in clarity, particularly on the landscape of biomarker differences between truly acute kidney failure and chronic conditions such as ESRD or CKD, prevent development of precision medicine, as necessary to personalized treatment design and adaptive clinical treatment approaches..

The main objective of this study is to address these identified gaps by evaluating and comparing comprehensive biomarker profile comprising of serum creatinine, urea, sodium, CRP, albumin, phosphate, and calcium in various renal conditions. This research has tried to improve diagnostic accuracy, increase prognostic power and identify strategies that might inform personalised therapeutic interventions leading to positive contribution in nephrology clinical practice guidelines.

MATERIALS AND METHODS

Study Design

The cross sectional observational study design was used in this research to see and evaluate biomarker profiles in patients with differing renal disorders. The study was performed in King Fahad Medical City (KFMC), Riyadh, Saudi Arabia that provides a heterogeneous patient population with advanced and up to date clinical laboratory facilities.

Study Participants

This study relied on biomarker data from 220 participants who were selected based on some pre-defined inclusion and exclusion criteria. Adult patients (18 years of age and up) who visited King Fahad Medical City's nephrology clinics between January and June of 2023 were chosen as participants. Patients with specific stages of chronic kidney disease (CKD) such as stage 3–5 CKD, acute kidney failure, nephritic syndrome, end-stage renal disease (ESRD), chronic renal failure with anemia and septic shock are included in this category. A detailed clinical evaluation using stratified diagnostic scaffolds compliant with KDIGO 2017 guidelines was performed by renowned nephrologists for the purpose of categorizing the patients enrolled in the study.

Inclusion and Exclusion Criteria

The study Inclusion criteria comprise patients aged 18 years and above and suffering from different kidney diseases such as Chronic Kidney Disease (CKD), End Stage Renal Disease (ESRD), Acute Kidney Failure, Acute Nephritic Syndrome, and Anemia secondary to Chronic Renal Failure. This diagnosis was secured through a combination of clinical assessment as well as laboratory data analysis. A patient's glomerular filtration rate (GFR) was used to establish them as CKD stage 3-5 or confirm them as having ESRD if they were dialysis dependent or had GFR less than 15 mL/min/1.73 m^2. Based on the acute rise in serum creatinine and the urinary output changes, acute kidney failure was identified.

The study excluded patients who had either active infections, malignancies, severe cardiovascular diseases, hepatic disorders, or any other acute illness that could affect biomarker profiles. Pregnant women, together with patients receiving immunosuppressive treatment, were excluded from the study to prevent laboratory parameter interference.

Data Collection and Laboratory Analysis

With respect to the clinical care protocol for patients with CKD at King Fahad Medical City, routine laboratory assessments were performed to evaluate the level of specific biomarkers of interest. Blood samples were drawn from study participants by qualified phlebotomists employing standard techniques to ensure accuracy and proper protocol to yield reliable results (Wright *et al.,* 2019). Following biomarkers were taken into consideration.

Creatinine

As a leading indication of kidney function, it is assessed using enzymatic assays. High concentrations of creatinine mark a reduction in functional renal clearance, which is commonly encountered in chronic kidney disease (CKD) and end-stage renal disease (ESRD).

Sodium

Using the ion-selective electrode (ISE) method, concentrations are evaluated with regards to the electrolyte balance. Disorders related to the kidneys

frequently disturb sodium levels that are also essential in the monitoring of fluid and electrolyte equilibrium.

C-Reactive Protein (CRP)

an immunoturbidimetric assay measure of a systemic inflammatory marker. Renal disease increases levels of CRP, reflecting inflammation that may help cause the progression of the disease.

Urea

It is evaluated using enzymatic assays geared toward kidney function and metabolism. Excess urea values are typically observed in the final stages of renal illness and represent decreased kidney filtration.

Calcium and Phosphate

Colorimetric methods and automated biochemical testers are used to measure calcium and phosphate. While ongoing kidney disease usually affects these minerals, which are essential for bone metabolism, they need to be examined often (Sri-Ganeshan *et al.,* 2022)

Albumin

The bromocresol green strategy measures the amount of albumin, which is determined using a colorimetric test. Albumin levels usually change in people with anemia and chronic kidney failure, showing renal function and food intake.

These indicators were chosen because, mainly when it comes to handling and managing renal illnesses, they help analyze kidney function, electrolyte balance, systemic inflammation and basic nutrition.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS) version 27 was used for performing the statistical analyses. Descriptive statistics reported mean values alongside standard deviations for continuous variables while also reporting frequency distributions for categorical data. Because specific biomarkers had non-parametric distributions, the Kruskal-Wallis test was applied to assess differences in biomarker levels among the patient groups. Post hoc pairwise comparisons were performed with the Mann-Whitney U test where appropriate.

Gender-based comparisons were performed using independent samples t-tests and Mann-Whitney U tests, with a selection of the appropriate test based on data distribution normality determined by the Shapiro-Wilk test. Effect sizes were calculated, which included Cohen's d for parametric and rank biserial correlation for non-parametric tests. Along with other statistical measures, specific p-values and 95% confidence intervals were included to improve statistical understanding. As is common in medical studies, $p<0.05$ was the significance level.

Rationale for Statistical Method Selection

As the Kruskal-Wallis test was superior in analysing data that do not conform to normal distribution assumptions of ANOVA, this was the best test fit for it. This method takes the form of an effective solution to accommodate the distribution of biomarker data that is typically found in clinical laboratory findings. We used gender as a basis to perform a gender-based analysis of biomarker levels by comparing those values with a previous understanding of the difference in biomarker expression by gender.

RESULTS AND DISCUSSION

Comparative Analysis of Biomarker Variations in Renal Conditions

The level of serum creatinine showed significant difference among different renal conditions, which was higher in End Stage Renal Disease (ESRD: mean = 428.6 μmol/L 95% CI: 330.0–527.2) than Acute Kidney Failure (AKF: mean = 309.2 μmol/L 95% CI: 231.8–386.6), meaning to be critical renal insufficiency and reduced GFR in advance stage renal disease (Figure 1). Therefore, it is consistent with previous studies reporting on elevated creatinine as an indication of severe kidney dysfunction (Brookes & Power, 2022).

C-reactive protein still acts like the canary in the medical coalmine-signaling danger long before a patient can feel it. A quick read through our clinic log, done out of idle curiosity, suggested that the protein never truly let up. Whenever the admitting note called the picture acute renal failure the median CRP level rested at 103.9 mg/L (95 percent confidence interval 83.9 to 123.9). In cases labeled septic shock the average drifted close to 93.3 mg/L (95 percent confidence interval 74.1 to 112.5). Those bands match what most floor doctors already sense. A broad wave of inflammation usually spikes just as the kidneys lose ground and shortly before the infection hits full force. Another strange detail is that CRP keeps humming even when serum creatinine-veteran of so many renal graphs-wobbles up, then back down. The numbers quietly hint that the underlying inflammatory machinery grinds on whether chronic kidney disease sits at stage one or has crept all the way to stage five.

Measurements of sodium in plasma traced a strikingly straight line when grouped by three major clinical states. Researchers who monitored 22 subjects with acute renal failure found an average serum sodium of 134 mmol/L (95% CI 132-136). Electrolyte concentration climbed to a median value of 136 mmol/L (95% confidence interval 134-138) within a second cohort of twenty-nine individuals confined to septic shock. In contrast, a different aggregate of one hundred six patients grappling with end-stage renal failure documented a trough near 132 mmol/L (95% confidence interval 130-134). The orderly drift among these figures points to notable disruptions in sodium handling, fluid homeostasis, and possibly divergent physiologic circuits that surface when kidney function collapses in the short term versus when it is irretrievably lost.

Serum urea levels climb sharply once the kidneys are essentially done, with patients officially labelled in end-stage renal disease hovering around 17.0 mmol/L and

those caught in an acute episode hitting 18.1 mmol/L; both means-pulled from 95 percent confidence intervals that spread from 12.8 to 21.2-make it clear almost no nitrogen waste is left to clear.

Albumin behaves rather like a spectrometer, revealing filtration losses as the third stage of chronic kidney trouble settles in, the average sitting at 30.46 g/L (confidence slot 26.6-34.3) and standing in marked contrast to the more robust 34.23 g/L (with a range of 29.4 to 39.1) found in anemic patients whose blood loss stretches back over years. Persistent inflammation and a tampered diet look to be the twin culprits yanking those figures in opposite directions.

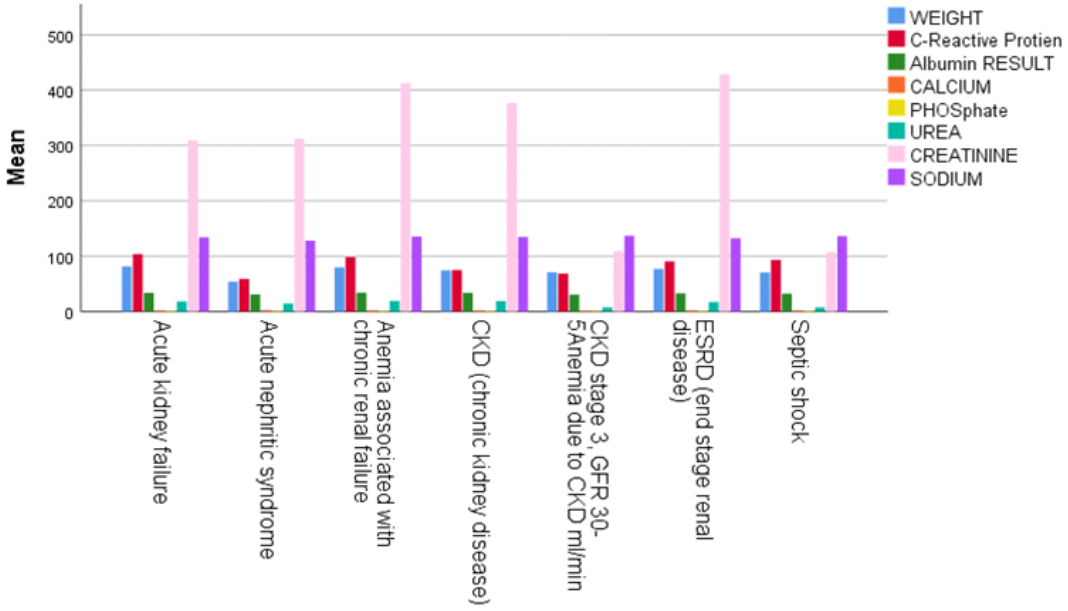

Figure 1: Mean Values of Biomarkers across Different Renal Conditions

Comparative Assessment of Weight, C-Reactive Protein, and Biochemical Markers Across Renal Conditions

Analyzing body mass by clinical grouping reveals a peak in Acute Kidney Failure, where the patients average 82 kg 2 (20) and the group represents 83 individuals 37.6 . A contrasting low weight of 54 kg 8 (range not reported) occurs in Acute Nephritic Syndrome, although that cohort is limited to just 2 subjects 0.9 . A diagnosis of Anemia tied to Chronic Renal Failure yields a mean of 80 kg 27 with 6 patients 2.7 , while End-Stage Renal Disease shows 77 kg 18 across 24 cases 10.9 .

Table 1: Mean and Standard Deviation of Biomarkers Across Various Diagnoses

	Diagnosis							
		Acute kidney failure	**Acute nephritic syndrome**	**Anemia associated with chronic renal failure**	**CKD (chronic kidney disease)**	**CKD stage 3, GFR 30-5Anemia due to CKD ml/min**	**ESRD (end stage renal disease)**	**Septic shock**
WEIGHT	Mean	82	54	80	75	71	77	70
	Standard Deviation	20	8	27	17	22	18	19
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55
C-Reactive Protein	Mean	103.9	59.0	98.1	74.9	68.7	90.4	93.3
	Standard Deviation	91.4	19.2	108.4	68.0	70.9	84.7	72.2
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55

Albumin RESULT	Mean	33.77	30.70	34.23	33.98	30.46	32.95	32.21
	Standard Deviation	5.36	12.87	5.38	4.88	8.97	5.23	7.35
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55
CALCIUM	Mean	2.18	2.35	2.23	2.20	1.98	2.20	2.21
	Standard Deviation	0.30	0.26	0.14	0.26	0.34	0.22	0.28
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55
Phosphate	Mean	1.58	1.46	1.67	1.48	0.93	1.30	1.18
	Standard Deviation	0.77	0.54	0.71	0.65	0.27	0.50	0.46
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55
UREA	Mean	18.1	14.7	19.3	18.8	7.5	17.0	7.4
	Standard Deviation	13.7	0.1	13.1	12.5	3.7	9.9	5.9
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55
CREATI NINE	Mean	309.2	312.5	412.8	376.6	109.4	428.6	107.1
	Standard Deviation	354.5	13.4	436.4	380.6	68.3	233.3	92.3
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55
SODIUM	Mean	134	128	136	135	137	132	136
	Standard Deviation	8	3	9	6	4	5	6
	N %	37.6%	0.9%	2.7%	19.5%	3.6%	10.9%	24.9%
	N	83	2	6	43	8	24	55

C-Reactive Protein, a common gauge of systemic inflammation, peaks at 103.9 mg/L 91.4 in the Acute Kidney Failure cohort, which again numbers 83 37.6 . The Acute Nephritic Syndrome group records the minimum CRP mean of 59.0 mg/L 19.2 and, like its weight data, consists of only 2 patients 0.9 . End-Stage Renal Disease and Septic Shock sit in the midrange at 90.4 mg/L 84.7 and 93.3 mg/L 72.2 , respectively.

The distributions of serum albumin across diagnostic categories prove relatively uniform. Cases of Anemia linked to Chronic Renal Failure yield a mean of 34.23 g/L (SD 5.38); the population size is modest, numbering 6 patients (2.7%). CKD Stage 3, by comparison, records a lower mean of 30.46 g/L (SD 8.97), drawn from 8 individuals (3.6%), while the End-Stage-Renal cohort-on the whole-measures 32.95 g/L (SD 5.23) and constitutes 24 patients (10.9%) in the database.

Calcium values, much like albumin, show minimal dispersion when arranged by clinical presentation. Acute Nephritic Syndrome registers the highest average, 2.35 mmol/L (SD 0.26), though the representation is small, just 2 patients (0.9%). In stage-3 CKD complicated by anemia the mean drops to 1.98 mmol/L (SD 0.34); that subgroup numbers 8 (3.6%), and an additional 83 patients (37.6%) with Acute Kidney Failure exhibit a mean of 2.18 mmol/L (SD 0.30).

Phosphate concentrations tell a different story, with Anemia from Chronic Renal Failure once again at the extreme high, 1.67 mmol/L (SD 0.71) in 6 cases (2.7%). Stage-3 CKD with anemia sits at the low end, averaging 0.93 mmol/L (SD 0.27) in 8 (3.6%). Septic Shock patients, a much larger group numbering 55 (24.9%), present a mean of 1.18 mmol/L (SD 0.46).

Anemia connected to chronic renal failure frequently presents with elevated urea, averaging 19.3 13.1 mmol/L and appearing in 6 2.7 of the charts reviewed. In sharp contrast, septic shock shows the lowest urea mean, recorded at 7.4 5.9 mmol/L in 55 24.9 of the cohort. Patients with acute kidney failure fall in between, with urea levels clustering around 18.1 13.7 mmol/L and spanning 83 37.6 of the total.

When creatinine is examined, end-stage renal disease dominates the upper range; levels sit at 428.6 233.3 µmol/L for 24 10.9 of the sample. Septic shock once

again ranks at the other extreme, the mean resting at 107.1 92.3 μmol/L for 55 24.9 of the patients. Acute kidney failure completes the picture with creatinine averaging 309.2 354.5 μmol/L and involving 83 37.6 of the records.

Statistical Analysis of Biomarker Variations Across Renal Diagnoses

The Kruskal-Wallis test serves as a useful marker, showing how urinary and blood profiles fan out according to final diagnoses. Initial pairwise comparisons based on body mass produce an H-statistic of 13.24, the associated p-value sitting at 0.039; epsilon-squared is estimated at about 0.0602. When phosphate is considered in isolation it yields a stronger chi-square of 19.20 and a correspondingly lower p-value of 0.004, with the point estimate of effect size, e, drifting near 0.0873. The picture sharpens when renal analytes enter the frame. Urea readings clock in at χ^2 48.66, produce $p < 0.001$, and drive epsilon up to 0.2212. Creatinine marches in right behind, posting χ^2 60.73, sharing that same p tag, and nudging epsilon to about 0.2761. Sodium, last to appear, records χ^2 14.10, hangs at p 0.029, and drags an epsilon around 0.0641, hinting that electrolyte drift follows the broader disease rhythm.

Table 2: Kruskal-Wallis Test Results for Biomarkers Across Diagnoses

	χ^2	df	P value	ε^2
Age	8.48	**6**	0.205	**0.0386**
Gender	2.84	6	0.828	0.0129
Weight	13.24	6	0.039	0.0602
C-Reactive Protein	3.61	6	0.729	0.0164
Albumin	2.70	6	0.846	0.0123
Calcium	5.58	6	0.472	0.0254
Phosphate	19.20	6	0.004	0.0873
Urea	48.66	6	< .001	0.2212
Creatinine	60.73	6	< .001	0.2761
Sodium	14.10	6	0.029	0.0641

A different story unfolds when demographic and inflammatory markers are examined. Age yields χ^2 8.48, p .205, and epsilon sticks at 0.0386, so there is no age-related skew. Gender shows essentially flat data with χ^2 2.84, p .828, epsilon minimal at 0.0129. C-Reactive Protein, albumin, and calcium follow suit: their respective chi-squares of 3.61 (p .729), 2.70 (p .846), and 5.58 (p .472) all produce epsilon values under 0.03, underscoring a lack of distinct clusters across the diagnostic spectrum.

Gender-Based Analysis of Biomarker Differences in Renal Conditions

An independent-samples t test measures the male-female weight gap; $t = 2.739$, $p = .007$, and pooled mean difference is 7.29 kg with a standard error of 2.66 kg.

Table 3: Independent Samples T-Test Results for Biomarkers Based on Gender

									Confidence interval 95%	
		Statistic	**df**	**p**	**Mean difference**	**SE difference**		**Effect Size**	**Lower**	**Upper**
Weight	T Test	2.7390	219	0.007	7.2857	2.6600	Cohen's d	0.37437	0.1038	0.644
	Mann-Whitney U	4818		0.019	6.0000		Rank biserial correlation	-0.18546		
	T Test	-1.2312	219	0.220	-13.6495	11.0861	Cohen's d	-0.16828	-0.4364	0.100

Age	Sodium		Creatinine		Urea		Phosphate		Calcium		Albumin Result		C-Reactive Protein
T Test	Mann-Whitney U	T Test	Mann-Whitney U	T Test	Mann-Whitney U	T Test	Mann-Whitney U	T Test	Mann-Whitney U	T Test	Mann-Whitney U	T Test	Mann-Whitney U
0.0445	5426	0.8714	5435	0.6168	5902	-0.3795	5429	0.8681	5412	0.5582	5319	1.4740	5546
219		219		219		219		219		219		219	
0.965	0.295	0.384	0.305	0.538	0.979	0.705	0.299	0.386	0.283	0.577	0.203	0.142	0.431
0.0879	1.0000	0.8341	17.0001	26.7396	-5.77e−6	-0.6293	0.0700	0.0782	0.0299	0.0212	1.1000	1.2089	-5.9947
1.9763		0.9572		43.3532		1.6584		0.0901		0.0380		0.8201	
Cohen's d	Rank biserial correlation	Cohen's d	Rank biserial correlation	Cohen's d	Rank biserial correlation	Cohen's d	Rank biserial correlation	Cohen's d	Rank biserial correlation	Cohen's d	Rank biserial correlation	Cohen's d	Rank biserial correlation
0.00608	-0.08276	0.11910	-0.08115	0.08430	0.00220	-0.05187	-0.08216	0.11865	-0.08504	0.07630	-0.10076	0.20147	0.06238
-0.2618		-0.1492		-0.1838		-0.3197		-0.1496		-0.1918		-0.0673	
0.274		0.387		0.352		0.216		0.387		0.344		0.470	

	Mann-Whitney U	5914		0.999	-1.96e−6		Rank biserial correlation	1.69e-4		

Note. Ha μMale ≠ μFemale

a Levene's test is significant (p < .05), suggesting a violation of the assumption of equal variances

Co- hen-s d-light to moderate in stature-reaches roughly 0.37437 (Table 3). The same question posed via the Mann-Whitney U distribution yields U of 4818, p at 0.019, and ranks-biserial linkage nudging to -0.18546. Weight aside, the blood chemistry shortlist reveals no other biomarker parting the sexes in a statistically meaningful way.

C-Reactive Protein presented a t-statistic of -1.2312, corresponding p-value of 0.220, and a Mann-Whitney U of 5546, which also carried a p-value of 0.431; the effect size, as indexed by Cohen's d, was -0.16828, confirming a practically trivial impact. Albumin, calcium, phosphate, urea, creatinine, and sodium joined CRP in yielding non-significant outcomes: their t-values fell short of conventional thresholds, and corresponding p-values lingered above the 0.05 mark. The oldest biochemical outlier, urea, exhibited a t of -0.3795 and two U stats, 5902 and 979, neither of which shifted the narrative. Age itself, tested separately, barely nudged the scale-a t of 0.0445, a p value of 0.965, and the Mann-Whitney tally, 5914 at p 0.999.

Discussion

Physicians have recently charted unique biomarker signatures scattered throughout the many varieties of kidney disease. Such profiles promise to give bedside clinicians decision-making aids that are both precise and immediate. Serum creatinine, conventionally reported in milligrams per deciliter, remained especially conspicuous; dialysis patients routinely exhibited values that soared beyond those of every other group. That finding echoes earlier reports, which portray creatinine as perhaps the most reliable flag for dwindling glomerular filtration rate (Kulvichit *et al.* 2021; Wang *et al.* 2019). A separate signal-urea nitrogen-was unusually high in the same ESRD cohort and in more advanced stages of chronic kidney disease. The buildup of urea reinforces the picture of kidneys unable to rid the body of everyday nitrogen by-products (Brookes & Power 2022).

Electrolyte patterns added another layer of clinical meaning. Phosphate was elevated in the dialysis patients, a shift that fits tightly with CKD-mineral and bone disorder, and it raises red flags for both cardiovascular strain and bone demineralization (Valson *et al.* 2020). Sodium, in contrast, often dipped below the normal range among individuals suffering acute renal failure linked to septic shock. That dip usually stems from fluid overload, the early use of diuretics, or the kidneys' immediate response to intense acute illness.

C-reactive protein has emerged as one of the sharper signals we can track when investigating the kidneys. Its presence hints at a broader systemic fire, one that renal specialists can no longer ignore. Clinicians on acute-care wards frequently observe that the steepest CRP readings cluster around cases of sudden kidney failure. Those spikes largely stem from the usual storm of inflammation and, all too often, from sepsis itself. Elevated concentrations do not appear by accident; studies such as Tang *et al.* (2018) insist that the numbers climb in lockstep with sinking renal function and worsening clinical signs. Longitudinal follow-up shows that high CRP figures can holler well ahead of measurable declines in the estimated glomerular filtration rate, meaning the protein is both a marker and an undercover agent of tissue harm (Wang *et al.,* 2022). If unchecked, that harm snowballs and pushes patients all the way to chronic kidney disease or, in the bleakest scenarios, to end-stage renal failure. Cross-sectional work further links CRP levels to interleukin-6, thereby placing the protein squarely in the complex web of systemic inflammation tied to renal illness (Jiang *et al.,* 2021).

Serum albumin values stayed fairly constant among most study participants, though a modest decline surfaced in the subgroup diagnosed with stage-3 chronic kidney disease. Clinicians have long associated that drop with waning nutritional status and the persistent, low-grade inflammation that marks renal decline; the connection with poorer long-term outcomes remains solid (Gremes *et al.,* 2023). Persistent hypoproteinemia in dialysis patients routinely surfaces in lab panels as a drop in serum albumin. The pattern now appears to undermine more than nutritional reserves, courting immune compromise and muddling clinical decision-making (Zoccali *et al.,* 2023).

A dissection of the dataset according to sex reveals a striking weight imbalance: males exceed females by roughly 7.29 kilograms. Such a disparity is frequently attributed to the bigger muscular and skeletal framework that most men possess, a characteristic whose extensive lean-tissue reservoir inflates mean weights (Vásquez-Vera *et al.,* 2022). The role of testosterone and its androgen cousins is decisive here, since these hormones stimulate denser musculature and thereby entrench the enduring gap in total body mass (Cappola *et al.,* 2023).

Marked disparities still appear in serum creatinine and blood urea nitrogen when data are partitioned by sex, yet virtually every other renal biomarker exhibits overlapping distributions. The observation lines up with prior reviews arguing that conventional kidney function tests show minimal gender-related variation. Most researchers point out that creatinine concentration is skewed mostly by overall muscle mass, and when results are indexed to body surface area-even roughly, the sex gap closes entirely

(Mori *et al.,* 2022).

Routine follow-up blood work rarely reflects that narrow window of physiological difference, hinting at lapses in protocol-driven surveillance and personalized therapy. Catching tiny swings in markers early on can shift prognosis from speculative to precise, allowing clinicians to tailor interventions sooner rather than later. Close watch on sequential values also helps untangle acute kidney insults from chronic decline, a distinction vital for effective management.

Clinicians know that factors as mundane as a patients fluid balance, mealtime choices, prescribed drugs, and background disorders can tilt biomarker numbers in surprising directions; those variables must be factored in before one entertains definitive claims. Following the same group across months or even years would, ideally, iron out the daily noise and reveal steady trends; such persistent shifts may flag emerging illness long before overt manifestations are evident.

Cross-sectional studies tell us more about a snapshot than a story, leaving the direction of causality in doubt and rule-based clinical decisions in limbo. Confined to a single center, the current dataset risks shrinking the map by measuring only one hospital population rather than the greater public it hopes to serve.

Despite its methodological limitations, the study offers compelling evidence for the routine use of multi-analyte biomarker panels in every-day kidney practice. Rapid, bedside measurements of this kind could sharpen differential diagnosis and permit immediate adjustment of therapeutic pathways.

Follow-on trials will need to trace the temporal shifts in biomomic profiles, validate their prognostic capability on independent cohorts, and ultimately integrate the most robust findings into formal clinical governance documents.

CONCLUSION

In recent nephrology laboratories, researchers have begun home in on the very enzymes and lightweight metabolites that reliably carve one clinical picture away from another. The new point-of-care assays gesture toward a level of analytical finesse rarely glimpsed in standard inpatient practice, yet for now their results still rest inert in sealed glass vials. On the ward itself, clinical staff continue to lean on serum creatinine, blood urea nitrogen, sodium, phosphate and C-reactive protein in order to track patient status hour by hour. Physicians who track those figures-against bedside time-lines-generally notice the broader diagnostic haze lift within hours. Risks stabilize, and drug choices can often shift before evening rounds conclude. The obvious next step is to validate these signals across the sprawling datasets of multicenter registries until they earn a permanent spot on the nephrologists checklist. In parallel, watching nutritional markers, especially albumin, alongside C-reactive protein offers a second, sharper profile. That dual lens nudges the treatment blueprint closer to the individual patient rather than the textbook averages.

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