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Original Research Article

Evaluation of Biochemical and Inflammatory Markers: Correlation of Serum Electrolytes, Urea, Creatinine, Uric Acid, and hsCRP in Chronic Renal Failure

Himanshu Kumar Sarathe[1], Savita Rathore[2], Seema Tamrakar[3]

[1]Phd Scholar, Department of Biochemistry, Index Medical College Hospital & Research Center, Indore, M.P, India

[2]Professor, Department of Biochemistry, Mahavir Institute of Medical Science & Research, Bhopal, M.P, India

[3]Assistant Professor, Department of Biochemistry, Mahavir Institute of Medical Science & Research, Bhopal, M.P, India

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Corresponding Author: Himanshu Kumar Sarathe

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Abstract:

Background: Renal function gradually declining is linked to CRF and disturbances in fluid, electrolyte, and metabolic parameters. Evaluation of biochemical markers such as creatinine, electrolytes, urea, uric acid and hsCRP can help assess disease severity and systemic inflammation.

Objective: To measure and investigate the connections and associations between serum levels of potassium, urea, creatinine, sodium, uric acid, chloride, and hsCRP and renal function in patients with CRF.

Methods: Over the course of 24 months, In collaboration with the Department of Biochemistry, the Department of Medicine at Index Medical College Hospital & Research Center in Indore (M.P.) carried out this cross-sectional study. A total of 120 patients aged 18–70 years, diagnosed with CRF, were enrolled after obtaining written informed consent. Patients receiving lipid-lowering or urate-lowering therapy, as well as pregnant and lactating women, were excluded. Fasting venous blood samples were analyzed for electrolytes (ion-selective electrode), urea and creatinine (enzymatic methods), uric acid (uricase–POD method), and hsCRP (high-sensitivity immunoturbidimetric assay). SPSS was used to analyze the data; Pearson's or Spearman's coefficients were used to determine correlations, and a p-value of less than 0.05 was deemed statistically significant.

Results: The participants' average age was 54.8 ± 11.6 years, with males comprising 64.2%. Hypertension (71.7%) and diabetes mellitus (48.3%) were the predominant comorbidities. Mean eGFR was 31.6 ± 14.2 mL/min/1.73 m². Mean serum sodium, potassium, and chloride were 137.2 ± 5.1, 5.04 ± 0.82, and 103.1 ± 6.0 mmol/L respectively. Mean urea and creatinine levels were 92 ± 35 mg/dL and 3.24 ± 1.41 mg/dL, while mean uric acid was 7.8 ± 2.1 mg/dL. Median hsCRP level was 3.8 mg/L (IQR: 2.1–6.9). Serum creatinine was negatively correlated with sodium ($r = -0.28$, $p = 0.003$) and positively correlated with potassium ($r = 0.42$, $p < 0.001$), uric acid ($r = 0.46$, $p < 0.001$), and hsCRP ($\rho = 0.33$, $p < 0.001$). Patients in advanced CKD stages exhibited significantly higher potassium, uric acid, and hsCRP levels ($p < 0.05$).

Conclusion: Declining renal function in CRF is accompanied by electrolyte imbalance, hyperuricemia, and elevated hsCRP, indicating both metabolic and inflammatory derangements. Monitoring these biochemical markers provides valuable insight into disease progression and may assist in guiding management strategies.

Introduction

A persistent and irreversible deterioration in renal function, CRF, also known as CKD, is defined by the kidneys' incapacity to maintain normal metabolic, fluid, and electrolyte balance. As functional nephrons are lost, waste materials like creatinine and urea build up in the blood, leading to metabolic acidosis, anemia, endocrine dysfunction, and a host of systemic complications. The illness progresses gradually and is the common ESRD, such as hypertensive nephrosclerosis, diabetic nephropathy, glomerulonephritis, and polycystic kidney disease. CKD is diagnosed when the eGFR remains below 60 mL/min/1.73 m² for three months or more, or when there is evidence of continuous kidney damage, such as albuminuria or structural abnormalities, according to the KDIGO guidelines.

The global burden of CKD is rising steadily, driven primarily by increasing rates of diabetes and hypertension. Both conditions contribute to vascular injury, glomerular sclerosis, and progressive loss of

renal function. The disease progression varies considerably among patients, influenced by genetic factors, comorbidities, lifestyle, and environmental exposures. In India, CRF has emerged as a major public health concern, with delayed diagnosis and limited access to renal replacement therapy contributing to poor outcomes. Understanding the biochemical alterations associated with CRF is therefore essential for early diagnosis, disease monitoring, and therapeutic decision-making.

Electrolyte disturbances are among the earliest and most clinically significant changes in patients with CRF. Sodium, potassium, and chloride play vital roles in maintaining fluid balance, nerve conduction, and acid–base homeostasis. With declining renal function, the kidneys lose their ability to regulate these ions effectively. Sodium levels may decrease due to fluid overload and impaired reabsorption, while potassium levels rise as excretion diminishes, predisposing patients to life-threatening arrhythmias. Chloride imbalance reflects accompanying metabolic acidosis and influences renal buffering mechanisms. Alongside these electrolyte changes, one indicator of renal insufficiency and a gauge of the severity of the condition is the buildup of nitrogenous waste products like urea and creatinine.

Another important biochemical alteration in CRF involves elevated serum uric acid and elevated levels of inflammatory indicators, including hsCRP. The kidneys are mainly responsible for excreting uric acid, which is the byproduct of purine metabolism. Impaired renal clearance leads to hyperuricemia, which in turn contributes to oxidative stress, endothelial dysfunction, and inflammation. Elevated hsCRP levels reflect low-grade systemic inflammation and are independently associated with the progression of CKD and cardiovascular risk. Together, these markers offer valuable insight into the complex interplay between metabolic derangement, inflammation, and renal decline.

Given the clinical significance of these biochemical and inflammatory markers, the present study was undertaken to evaluate the levels of urea, creatinine, uric acid, serum electrolytes, and hsCRP in patients with CRF and to assess their interrelationship. The study aimed to identify patterns of alteration across different stages of CKD and explore correlations between renal function and systemic inflammation. By analyzing these parameters in a defined patient population, the study seeks to enhance understanding of the biochemical landscape of chronic renal failure and its implications for disease management and prognosis.

Materials and Methods

Over a 24-month period, the study was carried out in the Department of Medicine and Department of Biochemistry at the Index Medical College Hospital & Research Center in Indore (M.P.). A total of 120 patients aged 18–70 years with established chronic renal failure were included. Patients on lipid-lowering or urate-lowering therapy, pregnant and lactating women, and individuals with acute infection or liver disease were excluded.

A detailed medical history and thorough clinical examination were carried out for all subjects. Demographic information, duration of kidney disease, comorbidities such as hypertension and diabetes, and ongoing treatments were recorded. Prior to analysis, approximately 5 mL of fasting venous blood was obtained, the serum was separated by centrifugation, and it was kept at -20 °C. Serum electrolytes (Na^+, K^+, Cl^-) were measured using ion-selective electrode methods; urea and creatinine by enzymatic colorimetric assays; uric acid by the uricase–peroxidase (POD) method; and hsCRP by high-sensitivity immunoturbidimetric assay on an automated analyzer.

Microsoft Excel was used to enter the data, and SPSS was used for analysis. The mean ± SD or median (IQR) were used to express quantitative variables. Correlations between renal function markers (urea, creatinine, eGFR) and serum electrolytes, uric acid, and hsCRP were assessed using either the Spearman's or Pearson's correlation coefficients, depending on the situation. The threshold for statistical significance was set at $p < 0.05$.

Results

The study, which was carried out at the Index Medical College Hospital & Research Center in Indore (M.P.), involved 120 patients who had been diagnosed with chronic renal failure. Among them, 77 (64.2%) were males and 43 (35.8%) were females, with a mean age of 54.8 ± 11.6 years (range: 25–70 years). Hypertension was present in 86 patients (71.7%), while 58 (48.3%) had diabetes mellitus. Nearly one-third of the subjects (39 patients, 32.5%) were on maintenance hemodialysis.

According to KDIGO classification, 22 patients (18.3%) were in stage 3a, 32 (26.7%) in stage 3b, 43 (35.8%) in stage 4, and 23 (19.2%) in stage 5 of CKD.

Table 1: Baseline characteristics of study participants (n = 120)

Parameter	Mean ± SD / n (%)
Age (years)	54.8 ± 11.6
Gender (Male/Female)	77 (64.2%) / 43 (35.8%)
Hypertension	86 (71.7%)
Diabetes mellitus	58 (48.3%)
On maintenance dialysis	39 (32.5%)
Mean BMI (kg/m²)	24.6 ± 3.8
Mean eGFR (mL/min/1.73 m²)	31.6 ± 14.2
CKD Stages (3a / 3b / 4 / 5)	22 (18.3%) / 32 (26.7%) / 43 (35.8%) / 23 (19.2%)

Biochemical Parameters: The mean serum sodium, potassium, and chloride concentrations were 137.2 ± 5.1 mmol/L, 5.04 ± 0.82 mmol/L, and 103.1 ± 6.0 mmol/L, respectively. Mean blood urea was 92 ± 35 mg/dL, while serum creatinine averaged 3.24 ± 1.41 mg/dL, indicating substantial impairment of renal excretory function. Mean serum uric acid was 7.8 ± 2.1 mg/dL, with 61% of patients showing levels above 7 mg/dL. The median hsCRP level was 3.8 mg/L (interquartile range: 2.1–6.9 mg/L), signifying mild to moderate inflammation in a majority of participants.

Patients in stage 5 CKD had markedly elevated mean creatinine and potassium levels with reduced sodium compared to those in earlier stages. Likewise, hsCRP and uric acid levels showed progressive increase with worsening renal function, a pattern that was statistically significant ($p < 0.05$).

Table 2: Biochemical parameters of study subjects

Parameter	Mean ± SD / Median (IQR)	Reference Range	Significance (Stage trend, p)
Sodium (mmol/L)	137.2 ± 5.1	135–145	↓ with stage, p = 0.01
Potassium (mmol/L)	5.04 ± 0.82	3.5–5.1	↑ with stage, p < 0.001
Chloride (mmol/L)	103.1 ± 6.0	98–107	NS
Urea (mg/dL)	92 ± 35	15–45	↑ with stage, p < 0.001
Creatinine (mg/dL)	3.24 ± 1.41	0.6–1.3	↑ with stage, p < 0.001
Uric acid (mg/dL)	7.8 ± 2.1	3.4–7.0	↑ with stage, p < 0.001
hsCRP (mg/L)	3.8 (2.1–6.9)	< 3	↑ with stage, p = 0.004

↑ = increasing trend; ↓ = decreasing trend; NS = not significant.

Correlations Between Biochemical Parameters: Significant correlations between a number of biochemical variables were shown by correlation analysis. Potassium ($r = 0.42$, $p < 0.001$), uric acid ($r = 0.46$, $p < 0.001$), and hsCRP ($\rho = 0.33$, $p < 0.001$) all showed positive correlations with serum creatinine, suggesting that deteriorating renal function was linked to elevated potassium retention, hyperuricemia, and inflammation. Conversely, serum sodium showed a negative correlation with creatinine ($r = -0.28$, $p = 0.003$), suggesting mild hyponatremia with disease progression. No significant association was found between chloride and renal markers. eGFR correlated inversely with urea, creatinine, and hsCRP, confirming that lower filtration capacity coincided with higher inflammatory and metabolic derangement.

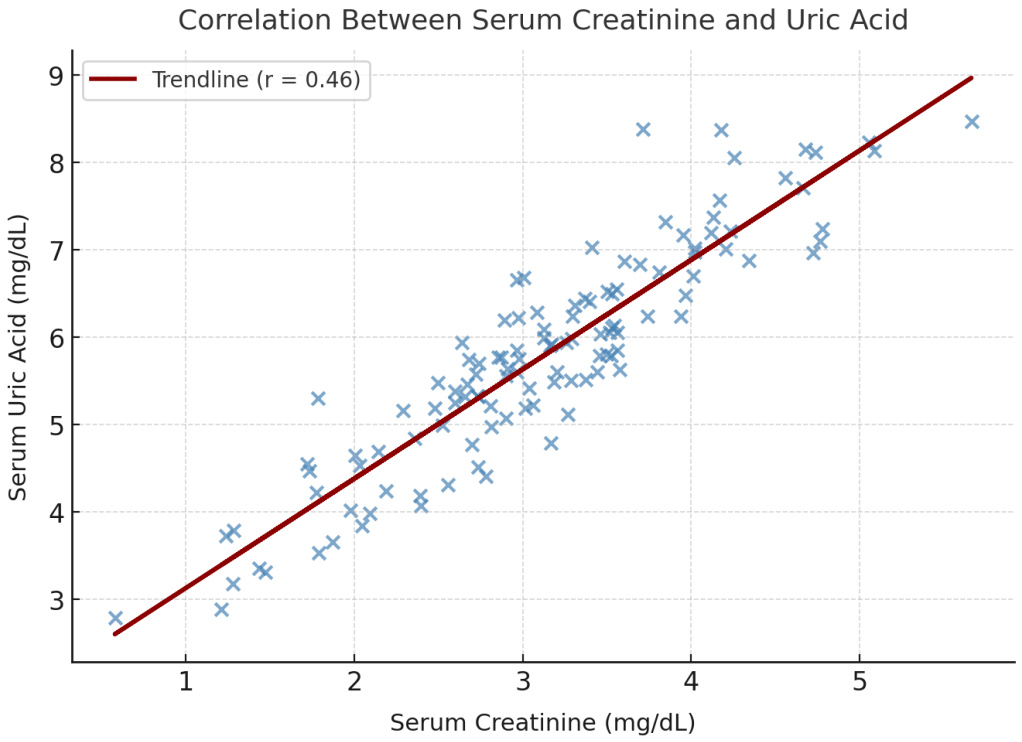

Figure 1: Correlation between serum creatinine and uric acid levels

Inflammatory Marker Trends: Analysis of hsCRP revealed that patients with diabetes and those undergoing hemodialysis had notably higher hsCRP values compared to non-diabetic and non-dialysis groups ($p < 0.05$).

Mean hsCRP was 5.7 ± 2.8 mg/L in dialysis patients versus 3.2 ± 1.6 mg/L in non-dialysis cases. A moderate inverse correlation was observed between eGFR and hsCRP ($r = -0.38$, $p < 0.001$), supporting the role of chronic inflammation in the progression of renal impairment.

Table 3: Correlation coefficients between major biochemical parameters

Parameters	Correlation Coefficient (r / ρ)	p-value	Relationship
Creatinine vs Potassium	r = 0.42	< 0.001	Positive
Creatinine vs Sodium	r = −0.28	0.003	Negative
Creatinine vs Uric Acid	r = 0.46	< 0.001	Positive
Creatinine vs hsCRP	ρ = 0.33	< 0.001	Positive
eGFR vs hsCRP	r = −0.38	< 0.001	Negative

Discussion

The results of this study, which was carried out at the Index Medical College Hospital & Research Center in Indore, showed that patients with CRF had unique biochemical and inflammatory changes. The mean age of the 120 participants was 54.8 years, and the majority (64.2%) were male. Hypertension and diabetes mellitus were the predominant etiological factors, affecting more than two-thirds of the study group. This pattern is consistent with the growing global evidence that these two conditions are the principal causes of chronic kidney disease. Ferro et al. (2018) and Cobo et al. (2016) similarly observed that diabetic and hypertensive nephropathies together account for over 70% of CKD cases worldwide. The predominance of advanced stages of CKD in this study suggests delayed diagnosis, which remains a challenge in resource-limited settings like India where awareness and screening programs are limited.

Electrolyte analysis demonstrated that serum sodium levels were slightly below normal in many patients and showed a declining trend with disease progression. Hyponatremia in CKD can occur as a consequence of fluid retention, reduced free-water clearance, and inappropriate antidiuretic hormone activity. Lim (2001) and Polska et al. (2012) reported similar findings, highlighting that the renal ability to handle sodium and water becomes increasingly impaired as kidney function deteriorates. Conversely, serum potassium levels showed a marked increase, positively correlating with creatinine ($r = 0.42$, $p < 0.001$). Hyperkalemia is a serious metabolic complication of CKD

resulting from impaired distal tubular secretion, acidosis, and certain medications such as ACE inhibitors. Persistent hyperkalemia can lead to cardiac arrhythmias and demands prompt management. Chloride values largely remained within normal limits but tended to fluctuate with metabolic acidosis, reflecting compensatory mechanisms in maintaining acid–base equilibrium.

Serum urea and creatinine concentrations were markedly elevated and demonstrated a strong negative correlation with eGFR. The mean urea level in this study was 92 mg/dL, and mean serum creatinine was 3.24 mg/dL. These values increased progressively with advancing stages of CKD, confirming impaired excretory function. Although urea and creatinine are influenced by factors such as dietary protein intake and muscle mass, their consistent elevation across CKD stages reinforces their diagnostic reliability. The results align with observations from Voskamp et al. (2018) and Attman and Samuelsson (2009), who emphasized the predictive value of rising nitrogenous waste levels for disease progression and prognosis. Clinically, monitoring these parameters remains essential for assessing renal function and determining the appropriate timing for dialysis initiation.

Serum uric acid levels were elevated in a majority of patients, showing a significant positive correlation with serum creatinine ($r = 0.46$, $p < 0.001$). Hyperuricemia, though often considered a secondary outcome of renal failure, has increasingly been recognized as a pathogenic factor that exacerbates kidney damage. Experimental evidence suggests that uric acid promotes glomerular hypertension, vascular smooth muscle proliferation, and oxidative stress. Fan et al. (2016) and Hameed (2020) observed similar trends, demonstrating that elevated uric acid levels are associated with faster progression of CKD and higher cardiovascular risk. In the present study, the co-occurrence of high uric acid and raised hsCRP levels further supports the hypothesis that metabolic and inflammatory pathways are closely linked in chronic kidney disease.

hsCRP levels were significantly raised, particularly in patients at later stages of CKD and in those undergoing dialysis. The median hsCRP was 3.8 mg/L, with diabetic and dialysis patients showing the highest values. Elevated hsCRP reflects ongoing systemic inflammation, likely due to persistent exposure to uremic toxins, oxidative stress, and impaired immune regulation. These findings are comparable to those of Song et al. (2022) and Pearce (2012), who emphasized that CRP elevation correlates with renal dysfunction and increased cardiovascular mortality. The observed negative correlation between eGFR and hsCRP ($r = -0.38$, $p < 0.001$) underscores the inflammatory component of renal failure. Chronic inflammation not only accelerates kidney damage but also contributes to the atherogenic lipid profile commonly observed in CKD, explaining the high cardiovascular morbidity among these patients.

The interrelationship among various biochemical parameters in this study highlights the complex interplay between metabolic retention and inflammation in CRF. The positive correlations between creatinine, potassium, uric acid, and hsCRP suggest that metabolic and inflammatory processes progress hand in hand as renal function declines. Sodium showed a negative relationship with creatinine, consistent with dilutional hyponatremia seen in advanced disease. These findings reinforce the view that CKD is not merely a disease of impaired excretion but a systemic disorder involving inflammation, oxidative stress, and endothelial dysfunction. Moradi and Vaziri (2018) described similar mechanisms, proposing that uric acid and CRP act as key mediators linking inflammation, dyslipidemia, and vascular injury in kidney disease.

From a clinical standpoint, the combined assessment of electrolytes, nitrogenous wastes, uric acid, and hsCRP provides a comprehensive picture of renal and systemic health. Hyperkalemia warrants immediate attention to prevent cardiac complications, while mild hyponatremia signals fluid imbalance that must be corrected cautiously. Elevated urea and creatinine levels guide disease staging and indicate when renal replacement therapy should be considered. Monitoring uric acid and hsCRP offers insight into inflammatory activity and cardiovascular risk, both of which strongly influence patient prognosis. Early detection and regular monitoring of these markers can help delay disease progression and improve outcomes through targeted interventions such as dietary control, medication optimization, and inflammation management.

In summary, this study demonstrated that chronic renal failure is accompanied by consistent and predictable biochemical and inflammatory changes. Elevated potassium, urea, creatinine, uric acid, and hsCRP levels, coupled with mild hyponatremia, reflect the combined effects of impaired renal clearance and systemic inflammation. The observed correlations among these parameters affirm that metabolic derangement and inflammation are central to the progression of kidney disease. Regular monitoring of these markers can enhance early diagnosis, optimize treatment strategies, and ultimately improve the quality of life for patients living with CRF.

Conclusion

In this study of patients with CRF, progressive deterioration of kidney function was associated with significant biochemical and inflammatory

alterations. Serum urea, creatinine, potassium, uric acid, and hsCRP levels increased in parallel with declining glomerular filtration rate, whereas serum sodium showed a modest decline. These findings highlight the combined impact of metabolic retention and systemic inflammation in the pathophysiology of CKD. Elevated uric acid and hsCRP concentrations suggest that oxidative stress and chronic inflammation contribute to disease progression and cardiovascular risk. Routine monitoring of these parameters provides valuable prognostic information and supports early, comprehensive management strategies aimed at preventing complications and slowing renal decline. Integrating metabolic and inflammatory marker assessment into standard clinical evaluation may enhance outcome prediction and improve long-term care for patients with CRF.

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