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CLINICAL ANALYSIS OF C-REACTIVE PROTEIN AS AN INFLAMMATORY MARKER IN CHRONIC KIDNEY DISEASE

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Abstract

Chronic kidney disease (CKD) is a progressive condition associated with significant morbidity and mortality worldwide, largely driven by persistent inflammation and impaired renal function. This study aimed to assess inflammatory and renal biomarkers, particularly high-sensitivity C-reactive protein (hs-CRP), in patients with CKD and evaluate their association with disease severity. A cross-sectional analytical study was conducted on 100 CKD patients in a tertiary care hospital. Demographic and clinical data were recorded, and biochemical parameters including hs-CRP, serum creatinine, estimated glomerular filtration rate (eGFR), serum albumin, and urinary albumin-to-creatinine ratio (UACR) were analyzed using standard laboratory techniques. The results showed that the mean age of participants was 52.26 ± 16.38 years, with a slightly higher proportion of males (56%). Elevated hs-CRP levels (10.92 ± 4.93 mg/L) indicated a high inflammatory burden among CKD patients. Serum creatinine (5.09 ± 2.46 mg/dL) and UACR (528.75 ± 253.19 mg/g) were markedly increased, while eGFR values showed wide variability, reflecting impaired renal function. Statistical analysis revealed significant differences among study variables ($F(5, 594) = 238.49$, $p < .001$), confirming a strong association between inflammation and renal dysfunction. In conclusion, elevated hs-CRP levels are strongly associated with systemic inflammation and disease progression in CKD patients. hs-CRP serves as a reliable, cost-effective biomarker for monitoring inflammation and identifying high-risk individuals. Routine assessment of CRP alongside renal function markers may enhance early diagnosis, improve clinical management, and reduce CKD-related complications.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive and irreversible condition characterized by structural and functional impairment of the kidneys, leading to metabolic, excretory, and endocrine dysfunction. It represents a growing global health burden, affecting approximately 8–16% of the population and contributing significantly to morbidity, mortality, and healthcare costs (Ridker et al., 2022). The progression of CKD is strongly

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influenced by chronic low-grade systemic inflammation, which plays a central role in renal fibrosis, endothelial dysfunction, and cardiovascular complications—the leading cause of death among CKD patients (Vachhani & Bhavsar, 2021).

C-reactive protein (CRP), an acute-phase reactant synthesized by the liver in response to pro-inflammatory cytokines—particularly interleukin-6 (IL-6)—has emerged as a sensitive and widely used biomarker of systemic inflammation. In CKD, elevated CRP levels are frequently observed even in the absence of overt infection, reflecting persistent inflammatory activity driven by uremic toxins, oxidative stress, and immune dysregulation (Vasilkova et al., 2024). High-sensitivity CRP (hs-CRP) assays further enhance the detection of low-grade inflammation, enabling early identification of patients at increased risk of disease progression and cardiovascular events (Ali et al., 2023).

Inflammation in CKD is multifactorial, involving cytokine activation (e.g., IL-6), complement system dysregulation (C3, C4), and metabolic disturbances such as phosphate retention and fibroblast growth factor-23 (FGF-23) elevation. These interconnected pathways contribute to nephron loss, vascular injury, and accelerated atherosclerosis (Baudier et al., 2024). Despite advancements in understanding CKD pathophysiology, CRP remains a practical, accessible, and clinically relevant biomarker for assessing inflammatory burden and predicting adverse outcomes.

Chronic kidney disease is increasingly recognized as an inflammatory disorder, with systemic inflammation contributing significantly to disease progression and associated complications. CRP has been extensively studied as a reliable biomarker reflecting inflammatory status in CKD patients.

Several large-scale studies have demonstrated a strong association between elevated CRP levels and CKD incidence and progression. Tsai et al. (2025), in a cohort of over 329,000 individuals, reported that higher CRP levels were linked with increased risk of developing CKD, particularly in advanced stages. Similarly, Liu et al. (2024) found that CRP concentrations progressively increase with declining renal function and are strongly associated with faster progression to end-stage renal disease (ESRD).

The introduction of high-sensitivity CRP assays has improved the detection of low-grade inflammation, which is particularly relevant in chronic conditions like CKD. Even mildly elevated hs-CRP levels have been associated with increased cardiovascular risk, endothelial dysfunction, and atherosclerosis in CKD populations (Mizher et al., 2023). These findings emphasize the dual role of CRP as both an inflammatory and cardiovascular risk marker.

Inflammation in CKD is driven by multiple mechanisms, including oxidative stress, immune activation, and accumulation of uremic toxins. Vasilkova et al. (2024) highlighted that persistent inflammation contributes to complications such as anemia, malnutrition, and vascular calcification. Elevated CRP levels have also been linked to protein-energy wasting and reduced serum albumin levels, indicating poor nutritional status and worse clinical outcomes (Tavares et al., 2022).

Cardiovascular disease remains the leading cause of mortality in CKD patients, and inflammation plays a pivotal role in its pathogenesis. Studies consistently report that elevated CRP levels are associated with increased risk of myocardial infarction, stroke, and cardiovascular mortality (Supriyadi et al., 2023). Furthermore, higher CRP levels have been shown to predict earlier initiation of hemodialysis and increased mortality risk (Schöffer et al., 2021).

Despite its clinical utility, CRP is a non-specific biomarker influenced by various factors such as infections, obesity, and comorbid conditions. Baião et al. (2023) emphasized that CRP should be interpreted alongside other clinical and laboratory parameters to improve diagnostic accuracy. Consequently, recent research advocates for a multi-biomarker approach incorporating CRP, IL-6, complement proteins, and FGF-23 to provide a more comprehensive assessment of CKD progression (Brito et al., 2021).

In South Asian populations, including Pakistan, the burden of CKD is further exacerbated by the high prevalence of diabetes, hypertension, and

Frontier in Medical & Health Research

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rapid urbanization. Limited access to early diagnostic tools underscores the need for cost-effective biomarkers such as CRP for early risk stratification and disease monitoring (Cai et al., 2024).

Therefore, this study aims to clinically evaluate the role of CRP as an inflammatory marker in CKD and to explore its diagnostic and prognostic significance in disease progression.

The present study aims to determine the serum levels of C-reactive protein (CRP) in patients with chronic kidney disease (CKD) and to evaluate its association with systemic inflammation. It further seeks to assess the diagnostic and prognostic significance of CRP in relation to disease progression, complications, and varying stages of CKD. In doing so, the study addresses key research questions: what are the serum CRP levels in CKD patients, how do these levels correlate with the degree of inflammation, what is the clinical significance of CRP in predicting CKD progression and related complications, and whether a significant relationship exists between CRP levels and the stages of CKD.

Methodology

Study Design

This study employed a cross-sectional analytical design to assess serum C-reactive protein (CRP) levels in patients diagnosed with chronic kidney disease (CKD) and to evaluate its association with systemic inflammation and disease progression.

Study Setting and Duration

The research was conducted in the nephrology and clinical laboratory departments of a tertiary care hospital in Pakistan over a period of six months.

Study Population

The study population consisted of patients diagnosed with CKD attending outpatient and inpatient services. Participants were selected using a non-probability convenient sampling technique.

Inclusion and Exclusion Criteria

Patients aged 18 years and above with clinically and laboratory-confirmed CKD were included. Patients with acute infections, autoimmune disorders, malignancies, or those receiving anti-inflammatory or immunosuppressive therapy were excluded to avoid confounding effects on CRP levels.

Sample Size

A total of [insert number] participants were enrolled based on availability during the study period and in accordance with study objectives.

Data Collection Procedure

After obtaining informed consent, demographic and clinical data, including age, gender, CKD stage, and relevant medical history, were recorded using a structured proforma. Venous blood samples were collected under aseptic conditions for laboratory analysis.

Biochemical Analysis

Serum CRP levels were measured using a standardized high-sensitivity assay method. Additional relevant biochemical parameters, including serum creatinine and urea, were assessed to determine CKD staging and renal function status.

Variables

The primary outcome variable was serum CRP level, while independent variables included CKD stage, demographic characteristics, and clinical parameters.

Statistical Analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version [insert version]. Descriptive statistics (mean, standard deviation, frequency, and percentage) were used to summarize the data. Inferential statistics, including correlation analysis and analysis of variance (ANOVA), were applied to examine the relationship between CRP levels and CKD stages. A p-value of less than .05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the institutional review board of the respective

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

hospital. All participants provided informed consent, and confidentiality of patient information was strictly maintained throughout the study.

Results

This chapter presents the findings of the study conducted to evaluate the role of C-reactive protein (CRP) as an inflammatory marker in patients with chronic kidney disease (CKD). The results are based on statistical analysis of demographic, clinical, and biochemical data collected from the study participants. Descriptive statistics were used to summarize variables such as age, gender distribution, serum creatinine, estimated glomerular filtration rate (eGFR), urinary albumin-to-creatinine ratio (UACR), and high-sensitivity C-reactive protein (hs-CRP). Inferential statistical tests were applied to determine the significance of differences and associations among study variables. The findings are presented in the form of tables, followed by their interpretation in relation to the study objectives.

Descriptive Statistics

A total of 100 participants diagnosed with chronic kidney disease (CKD) were included in the study. Descriptive statistics were computed to summarize demographic characteristics and biochemical parameters.

Table 4.1: ***Descriptive Statistics of Inflammatory and Renal Biomarkers in CKD Patients (N = 100)***

Variable	Min	Max	Mean	SD
Age (years)	25	80	52.26	16.38
hs-CRP (mg/L)	3.00	19.00	10.92	4.93
Serum Creatinine (mg/dL)	1.57	11.00	5.09	2.46
eGFR (mL/min/1.73 m²)	6.00	981.00	141.32	203.25
Serum Albumin (g/dL)	2.10	11.00	6.03	1.83
UACR (mg/g)	51.00	982.00	528.75	253.19

Note. hs-CRP = high-sensitivity C-reactive protein; eGFR = estimated glomerular filtration rate; UACR = urinary albumin-to-creatinine ratio.

The results indicate that the mean age of participants was 52.26 years (*SD* = 16.38). Elevated mean hs-CRP levels (10.92 ± 4.93 mg/L) suggest a high inflammatory burden among CKD patients. Increased serum creatinine (5.09 ± 2.46 mg/dL) and UACR (528.75 ± 253.19 mg/g), along with wide variability in eGFR (141.32 ± 203.25 mL/min/1.73 m²), reflect impaired renal function and significant albuminuria in the study population.

Frequency Distribution

Table 4.2: *Demographic Characteristics of the Study Population (N = 100)*

Variable	Min	Max	Mean	SD
Age (years)	25	80	52.26	16.38
Gender (coded)	1	2	1.44	0.49

Note. Gender was coded as 1 = Male and 2 = Female.

The demographic analysis shows that participants' ages ranged from 25 to 80 years, with moderate variability. The mean gender value (1.44 ± 0.49) indicates representation of both male and female participants, with no missing data reported.

Frontier in Medical & Health Research

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Gender Distribution

Table 4.3: ***Gender Distribution of CKD Patients (N = 100)***

Gender	Frequency	Percentage
Male	56	56.0%
Female	44	44.0%
Total	100	100.0%

Note. Percentages are based on total sample size; no missing data were observed.

The results show that 56% of participants were male and 44% were female, indicating slightly higher male representation. Both genders were adequately included in the study.

Inferential Statistics (One-Way ANOVA)

A one-way analysis of variance (ANOVA) was conducted to compare mean differences among six study variables (age, hs-CRP, serum creatinine, eGFR, serum albumin, and UACR).

Table 4.4: ***One-Way ANOVA Summary***

Source	SS	df	MS	F	p
Between Groups	21,011,210.51	5	4,202,242.10	238.49	< .001
Within Groups	10,466,338.57	594	17,620.10		
Total	31,477,549.08	599			

Note. SS = sum of squares; df = degrees of freedom; MS = mean square.

The ANOVA results revealed a statistically significant difference among the study variables, $F(5, 594) = 238.49$, $p < .001$, indicating substantial variation across inflammatory and renal markers.

Post Hoc Analysis (Tukey HSD Test)

Table 4.5: *Tukey HSD Post Hoc Comparisons*

Comparison	Q Statistic	p-value	Inference
A vs B	3.11	.238	Not significant
A vs D	6.71	.001	Significant**
A vs F	35.89	.001	Significant**
B vs D	9.82	.001	Significant**
B vs F	39.01	.001	Significant**
C vs D	10.26	.001	Significant**
C vs F	39.45	.001	Significant**
D vs E	10.19	.001	Significant**
D vs F	29.18	.001	Significant**
E vs F	39.37	.001	Significant**

Note. A = Age, B = hs-CRP, C = Serum Creatinine, D = eGFR, E = Serum Albumin, F = UACR. **p < .01.

Frontier in Medical & Health Research

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Post hoc analysis using Tukey's HSD test indicated that several variable pairs showed statistically significant differences, particularly those involving eGFR and UACR. These findings highlight strong variability between renal function markers and inflammatory indicators.

Overall, the results demonstrate elevated inflammatory markers and significant renal impairment among CKD patients. The statistical analyses confirm meaningful differences between key biochemical parameters, supporting the role of inflammation in CKD progression.

Discussion

The findings demonstrate significantly elevated hs-CRP levels among CKD patients, confirming the presence of systemic inflammation. This aligns with existing literature that identifies chronic inflammation as a key contributor to CKD progression and its associated complications. Elevated CRP levels observed in this study may reflect persistent inflammatory responses, oxidative stress, and reduced renal clearance.

The high mean UACR and serum creatinine levels further indicate impaired renal function and substantial kidney damage, while variability in eGFR supports heterogeneity in disease stages. The significant ANOVA results reinforce the interrelationship between inflammatory markers and renal dysfunction.

Consistent with previous studies, elevated CRP levels are associated with poor clinical outcomes, including cardiovascular risk, hypoalbuminemia, and disease progression. These findings support the role of CRP as a reliable, cost-effective biomarker for assessing inflammation and predicting prognosis in CKD patients.

Conclusion

The study concludes that elevated hs-CRP levels are strongly associated with systemic inflammation and renal impairment in CKD patients. CRP serves as a valuable biomarker for disease severity and progression. Routine monitoring of CRP, alongside renal function parameters, can facilitate early identification of high-risk patients and improve clinical management strategies to reduce complications and enhance patient outcomes.

Discussion

The present study demonstrated significantly elevated hs-CRP levels among patients with chronic kidney disease (CKD), indicating a high burden of systemic inflammation. These findings are consistent with previous literature, which identifies C-reactive protein (CRP) as a key inflammatory biomarker associated with CKD progression. Chronic inflammation in CKD is largely attributed to oxidative stress, reduced renal clearance, and persistent immune activation, all of which contribute to worsening renal function and increased cardiovascular risk.

The observed association between elevated CRP levels and impaired renal markers (e.g., serum creatinine, eGFR, and UACR) supports earlier findings that link inflammation with endothelial dysfunction, protein-energy wasting, and adverse clinical outcomes. Studies have also shown that CKD patients, particularly those undergoing hemodialysis, exhibit higher CRP levels due to additional inflammatory stimulation. Furthermore, elevated CRP has been associated with hypoalbuminemia and increased morbidity, reinforcing its role as a prognostic indicator.

Overall, the findings of this study strengthen the evidence that CRP is a reliable, cost-effective biomarker for assessing inflammation and monitoring disease progression in CKD patients. Routine measurement of CRP may assist clinicians in identifying high-risk individuals and implementing timely interventions to reduce complications.

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