Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

Role of procalcitonin levels in progression of staging of chronic kidney disease: A retrospective observational study.

Ayushi Chaudhari[1], Devopam Roy[2], Syed Yasrib Nahid Zaidi[1]*
[1]Assistant Professor, Department of General Medicine, Naraina Hospital & Research Center, Uttar Pradesh, India.
[2]Associate Professor, Department of General Medicine, Naraina Hospital & Research Center, Uttar Pradesh, India.

Abstract

Background:
Chronic kidney disease (CKD) is associated with chronic inflammation and progressive deterioration of renal function. Procalcitonin, traditionally used as a marker of bacterial infection, has been reported to be elevated in CKD patients even in the absence of infection.

Objectives:
To evaluate the association between serum procalcitonin levels and the severity and progression of chronic kidney disease.

Methods:
This retrospective observational study was conducted at Naraina Hospital & Research Center, Uttar Pradesh, India, over one year. Medical records of 200 adult CKD patients were reviewed. Patients with acute infections, sepsis, dialysis dependency, renal transplantation, or incomplete records were excluded. CKD staging was determined using the estimated glomerular filtration rate (eGFR). Associations between procalcitonin levels, CKD stage, and disease progression were analyzed using ANOVA, independent t-test, and Pearson correlation analysis.

Results:
Mean procalcitonin levels increased significantly across CKD stages, from 0.08±0.03 ng/mL in stages 1–2 to 0.85±0.26 ng/mL in stage 5 ($p<0.001$). Patients with CKD stage progression had significantly higher procalcitonin levels compared with patients without progression (0.64±0.21 vs. 0.22±0.10 ng/mL; $p<0.001$). Procalcitonin demonstrated a significant negative correlation with eGFR ($r=-0.71$, $p<0.001$) and a positive correlation with serum creatinine ($r=0.68$, $p<0.001$).

Conclusion:
Serum procalcitonin levels increase significantly with advancing CKD stage and are associated with disease progression. Elevated procalcitonin may reflect chronic inflammation and impaired renal clearance rather than infection alone.

Recommendation:
Procalcitonin should be interpreted cautiously in CKD patients and may be considered as an adjunct biomarker for monitoring disease severity and progression. Prospective multicenter studies are recommended to validate its prognostic utility.

Keywords: *Chronic Kidney Disease; Procalcitonin; Inflammation; Estimated Glomerular Filtration Rate; Disease Progression; Retrospective Observational Study.*
Submitted: *April 01, 2026* ***Accepted:*** *May 28, 2026* ***Published:*** *June 11, 2026*

Corresponding Author: *Syed Yasrib Nahid Zaidi*
Email: ***meetnahid15@gmail.com***
Assistant Professor, Department of General Medicine, Naraina Hospital & Research Center, Uttar Pradesh, India.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

INTRODUCTION

Chronic kidney disease (CKD) is a progressive and irreversible condition characterized by a gradual decline in renal function over time. It represents a major public health challenge worldwide due to its increasing prevalence, association with significant morbidity and mortality, and substantial economic burden on healthcare systems. CKD is commonly staged based on estimated glomerular filtration rate (eGFR), with advancing stages reflecting worsening renal function and higher risk of complications. Early identification of factors associated with disease progression is critical, as timely intervention can delay progression to end-stage renal disease and reduce cardiovascular and infectious complications [1].

Inflammation has emerged as a central mechanism in the pathogenesis and progression of CKD. Patients with CKD frequently exhibit a chronic low-grade inflammatory state, which contributes to endothelial dysfunction, accelerated atherosclerosis, protein-energy wasting, and increased susceptibility to infections. This inflammatory milieu is not only a consequence of declining renal function but also a driver of disease progression. As CKD advances, reduced renal clearance of inflammatory mediators, oxidative stress, and immune dysregulation further amplify systemic inflammation, creating a vicious cycle that accelerates renal damage [2].

Among the various inflammatory biomarkers studied in CKD, procalcitonin has gained increasing attention. Traditionally recognized as a marker of bacterial infection and sepsis, procalcitonin levels are now known to be influenced by non-infectious inflammatory states, including renal dysfunction. Understanding the role of procalcitonin beyond acute infection, particularly its association with CKD staging and progression, may provide valuable insight into disease monitoring and risk stratification.

Chronic Kidney Disease Progression and the Role of Inflammation

The progression of CKD is influenced by multiple factors, including underlying etiology, hypertension, diabetes mellitus, proteinuria, and recurrent infections. In recent years, chronic inflammation has been increasingly recognized as an independent predictor of CKD progression. Elevated inflammatory markers have been associated with faster decline in eGFR, increased hospitalization, and higher mortality rates in CKD patients [3].

Inflammation contributes to CKD progression through several mechanisms. Pro-inflammatory cytokines promote glomerular and tubular injury, stimulate fibrotic pathways, and impair endothelial function. Persistent inflammation also enhances oxidative stress, leading to cellular damage and apoptosis within renal tissue. As kidney function deteriorates, the clearance of inflammatory mediators is reduced, resulting in sustained elevation of circulating inflammatory markers. This chronic inflammatory state is particularly pronounced in advanced stages of CKD and is associated with poor clinical outcomes.

Traditional inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate have been widely studied in CKD. However, these markers lack specificity and may be influenced by a variety of non-renal factors. There is a growing need for biomarkers that can more accurately reflect the inflammatory burden associated with renal dysfunction and provide prognostic information regarding disease progression.

Procalcitonin as a Biomarker in Renal Dysfunction

Procalcitonin is a 116-amino-acid precursor of the hormone calcitonin and is normally produced in negligible amounts by thyroid C cells. Under physiological conditions, circulating procalcitonin levels remain low. However, during systemic bacterial infections and inflammatory states, procalcitonin is produced by various extrathyroidal tissues and released into the circulation. Because of its rapid rise and relative specificity for bacterial infections, procalcitonin has been widely used as a diagnostic and prognostic marker in sepsis and severe infections [4].

In patients with chronic kidney disease, elevated procalcitonin levels have been observed even in the absence of overt infection. This elevation is thought to result from a combination of increased systemic inflammation and reduced renal clearance. As kidney function declines, the metabolism and elimination of procalcitonin may be impaired, leading to accumulation in the circulation. Additionally, CKD-associated inflammatory stimuli may trigger increased procalcitonin production independent of infection.

Several studies have reported higher baseline procalcitonin levels in patients with advanced CKD compared with those in earlier stages or with normal renal function. Importantly, these elevations may correlate with disease severity and inflammatory burden rather than acute infectious processes alone. This raises important clinical questions regarding the

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

interpretation of procalcitonin levels in CKD patients and their potential role as indicators of disease progression rather than solely markers of infection [5].

Rationale for Studying Procalcitonin in CKD Staging Progression

Given the complex interplay between inflammation and renal dysfunction, there is growing interest in evaluating procalcitonin as a biomarker in CKD beyond its traditional infectious indications. If procalcitonin levels are shown to correlate with CKD staging and progression, they could serve as a useful adjunct in risk stratification and monitoring of patients with chronic kidney disease.

Retrospective analyses provide an opportunity to evaluate real-world data across a broad patient population and assess associations between laboratory parameters and disease severity. In tertiary care settings such as Naraina Hospital & Research Center, CKD patients frequently present across all stages of disease, often with multiple comorbidities. Understanding the relationship between procalcitonin levels and CKD staging in such a population may help clarify whether elevated procalcitonin reflects progressive renal dysfunction, heightened inflammatory activity, or both.

Furthermore, distinguishing infection-related procalcitonin elevation from CKD-related elevation has important clinical implications. Misinterpretation of elevated procalcitonin levels may lead to unnecessary antibiotic use, increased healthcare costs, and antimicrobial resistance. Conversely, recognizing procalcitonin as a marker of disease progression could enhance clinical decision-making and prompt closer monitoring of high-risk patients.

In this context, the present retrospective study was undertaken to evaluate the role of procalcitonin levels in the progression of CKD staging. By analyzing procalcitonin values across different stages of chronic kidney disease over one year in a cohort of 200 patients, this study aims to explore the association between procalcitonin levels and CKD severity. The findings may contribute to improved understanding of inflammatory biomarkers in CKD and support more nuanced interpretation of procalcitonin levels in patients with chronic renal dysfunction [6].

MATERIALS AND METHODS

Study Design and Setting

This retrospective observational study was conducted at Naraina Hospital & Research Center, Agra, Uttar Pradesh, India, a tertiary-care multispecialty teaching hospital providing nephrology, internal medicine, surgery, critical care, emergency, and diagnostic services to patients from Agra and neighboring districts. The study reviewed medical records from January 2024 to December 2024.

Participants selection

A consecutive sampling method was employed. All eligible CKD patients meeting the inclusion criteria during the study period were included until the required sample size was achieved.

Study Population and Sample Size

A total of **200 patients** diagnosed with chronic kidney disease were included in the study. Patients were identified from hospital medical records, including inpatient and outpatient databases, during the study period. CKD diagnosis was based on documented evidence of reduced kidney function persisting for at least three months, as indicated by estimated glomerular filtration rate (eGFR) measurements.

Adult patients aged 18 years and above with available records of serum procalcitonin levels and renal function parameters were considered eligible for inclusion. Patients were included across all stages of CKD, allowing evaluation of procalcitonin levels in relation to disease severity and staging progression.

Sample size was calculated using Cochran's formula:

$n = Z^2P(1-P)/d^2$

Assuming a prevalence of elevated procalcitonin among CKD patients of 15%, 95% confidence level, and 5% precision, the minimum required sample size was 196. To improve statistical power, 200 patients were included.

Inclusion and Exclusion Criteria

Inclusion criteria consisted of patients with a confirmed diagnosis of chronic kidney disease, documented CKD staging based on eGFR, and at least one recorded serum procalcitonin measurement during the study period. Patients with sufficient laboratory and clinical data to assess disease stage and inflammatory status were included.

Exclusion criteria included patients with acute kidney injury, renal transplant recipients, and those undergoing dialysis at the time of data collection. Patients with documented acute bacterial infections, sepsis, recent major surgery, trauma, or inflammatory conditions that could significantly elevate procalcitonin levels were excluded to minimize

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

confounding. Records with incomplete laboratory data or missing key variables were also excluded from analysis.

Data Collection and Variables

Patient data were extracted from hospital records using a structured data collection format. Demographic variables included age and sex. Clinical data collected included the underlying etiology of CKD, comorbid conditions such as diabetes mellitus and hypertension, and documented CKD stage at the time of procalcitonin measurement.

Renal function parameters included serum creatinine levels and estimated glomerular filtration rate, calculated using standard equations as documented in patient records. CKD staging was categorized according to established staging criteria based on eGFR values. Progression of CKD staging was assessed by comparing documented CKD stages over the study period, where follow-up data were available. Serum procalcitonin levels were recorded as measured in the hospital's central diagnostic laboratory. Procalcitonin measurements were performed using standardized immunoassay techniques as part of routine clinical evaluation. The timing of procalcitonin testing in relation to renal function assessment was noted wherever available.

Assessment of CKD Staging Progression

CKD staging progression was defined as advancement to a higher CKD stage during the study period, based on documented changes in eGFR. Patients were categorized into groups based on CKD stage at baseline. For patients with serial renal function assessments, changes in staging were documented and analyzed. Procalcitonin levels were evaluated across different CKD stages to assess their association with disease severity. Mean and median procalcitonin values were compared between CKD stages. In patients with available longitudinal data, changes in procalcitonin levels were correlated with progression or stability of CKD staging.

Bias Control

Patients with documented infections, sepsis, recent surgery, trauma, or inflammatory disorders were excluded to minimize confounding. Standardized data extraction forms were used, and records with missing key variables were excluded to reduce information bias.

Statistical Analysis

Statistical analysis was performed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Comparison of procalcitonin levels among CKD stages was performed using one-way ANOVA. An independent sample t-test was used to compare progression and non-progression groups. Pearson correlation analysis assessed relationships between procalcitonin, eGFR, and serum creatinine. A p-value <0.05 was considered statistically significant.

Ethical Considerations

As this was a retrospective study based on a review of existing medical records, no direct patient interaction was involved. Patient identifiers were removed before data analysis to ensure confidentiality. The study was conducted in accordance with ethical standards for research involving human data, and institutional guidelines were followed throughout the study process.

RESULTS

A total of 200 patients with chronic kidney disease (CKD) were included in the retrospective analysis. The mean age of the study population was 56.8 ± 13.2 years, with males accounting for 63.0% (n=126) of participants. Hypertension (71.0%) and diabetes mellitus (54.0%) were the most common comorbidities. The mean serum creatinine level was 3.12 ± 1.58 mg/dL, while the mean estimated glomerular filtration rate (eGFR) was 38.4 ± 22.7 mL/min/1.73m².

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

Table 1. Baseline Characteristics of Study Participants (n=200)

Variable	Value
Age (years)	56.8 ± 13.2
Male	126 (63.0%)
Female	74 (37.0%)
Hypertension	142 (71.0%)
Diabetes Mellitus	108 (54.0%)
Diabetic Nephropathy	82 (41.0%)
Hypertensive Nephropathy	64 (32.0%)
Other Causes of CKD	54 (27.0%)
Serum Creatinine (mg/dL)	3.12 ± 1.58
eGFR (mL/min/1.73m²)	38.4 ± 22.7

Distribution of CKD Stages

Patients were distributed across all stages of CKD. Stage 3 CKD constituted the largest proportion of the study population (36.0%), followed by Stage 4 (28.0%), Stage 1–2 (19.0%), and Stage 5 (17.0%). The distribution of patients across CKD stages was statistically significant ($\chi^2 = 18.42$, $p < 0.001$), indicating a predominance of moderate-to-severe disease within the study cohort.

Table 2. Distribution of Patients According to CKD Stage

CKD Stage	Number of Patients (n)	Percentage (%)
Stage 1–2	38	19.0
Stage 3	72	36.0
Stage 4	56	28.0
Stage 5	34	17.0
Total	200	100

Procalcitonin Levels Across CKD Stages

Mean serum procalcitonin levels increased progressively with advancing CKD stage. Patients with Stage 1–2 CKD demonstrated the lowest mean procalcitonin concentration (0.08 ± 0.03 ng/mL), whereas patients with Stage 5 CKD exhibited the highest levels (0.85 ± 0.26 ng/mL). One-way analysis of variance (ANOVA) revealed a statistically significant difference in procalcitonin levels among CKD stages ($F = 82.71$, $p < 0.001$).

Figure 1 illustrates the trend of increasing mean procalcitonin levels across CKD stages. The rise in procalcitonin appeared proportional to the severity of renal dysfunction, suggesting a strong association between declining kidney function and elevated procalcitonin levels, even in the absence of documented infection.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

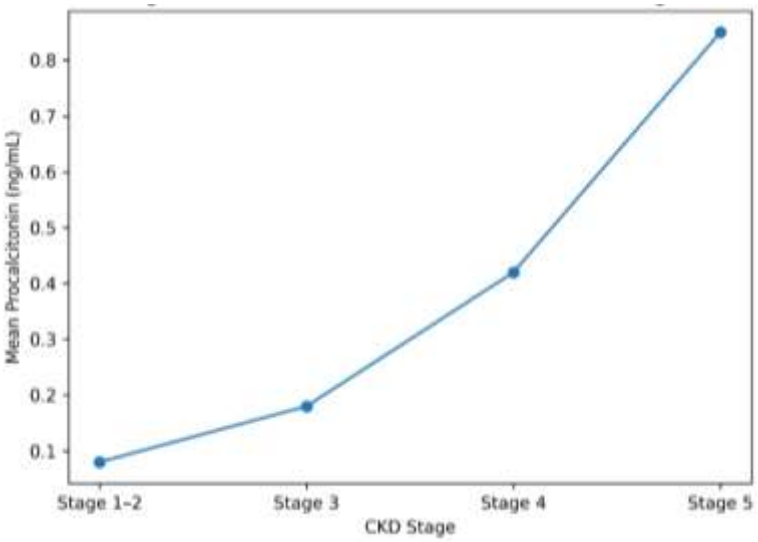

Figure 1: Mean Procalcitonin Levels Across CKD Stages

Table 3. Comparison of Procalcitonin Levels Across CKD Stages

CKD Stage	Number of Patients (n)	Mean Procalcitonin (ng/mL)
Stage 1–2	38	0.08 ± 0.03
Stage 3	72	0.18 ± 0.06
Stage 4	56	0.42 ± 0.14
Stage 5	34	0.85 ± 0.26

ANOVA: F = 82.71, **p < 0.001**

The results demonstrate a significant upward trend in serum procalcitonin concentrations with deterioration of renal function.

Association Between Procalcitonin Levels and CKD Stage Progression

During the one-year study period, 78 patients (39.0%) exhibited progression to a more advanced CKD stage, while 122 patients (61.0%) remained stable. Mean procalcitonin levels were significantly higher among patients who experienced CKD progression compared with those whose disease remained stable (0.64 ± 0.21 ng/mL vs. 0.22 ± 0.10 ng/mL). An independent sample t-test demonstrated a highly significant difference between the two groups (t = 17.34, p < 0.001).

Figure 2 depicts the comparison of mean procalcitonin levels between patients with and without CKD stage progression. The difference between the two groups suggests that elevated procalcitonin levels may be associated with ongoing inflammatory activity contributing to disease advancement.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

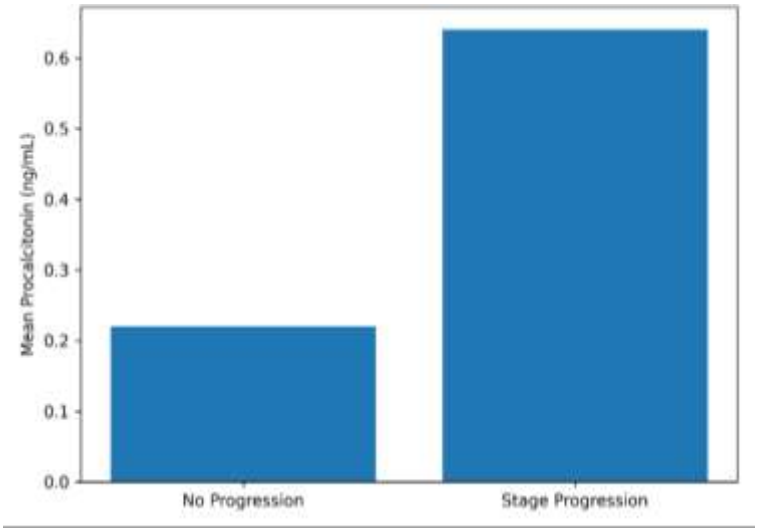

Figure 2: Procalcitonin Levels and CKD Stage Progression

Table 4. Association Between Procalcitonin Levels and CKD Stage Progression

Progression Status	Number of Patients (n)	Mean Procalcitonin (ng/mL)
No Progression	122	0.22 ± 0.10
CKD Progression	78	0.64 ± 0.21

Independent t-test: t = 17.34, **p < 0.001**

Patients with elevated procalcitonin levels were significantly more likely to demonstrate CKD progression during follow-up.

Correlation Between Procalcitonin and Renal Function Parameters

Pearson correlation analysis demonstrated a strong negative correlation between serum procalcitonin levels and eGFR (r = -0.71, p < 0.001), indicating that higher procalcitonin concentrations were associated with poorer renal function. Conversely, a strong positive correlation was observed between serum procalcitonin and serum creatinine levels (r = 0.68, p < 0.001).

Table 5. Correlation of Procalcitonin with Renal Function Parameters

Variable	Correlation Coefficient (r)	p-value
eGFR	-0.71	<0.001
Serum Creatinine	0.68	<0.001

These findings suggest that increasing procalcitonin levels are closely associated with declining renal function and worsening disease severity.

DISCUSSION

The present study demonstrated a significant increase in serum procalcitonin levels with advancing CKD stage (F=82.71, p<0.001). Patients with CKD progression had significantly higher procalcitonin concentrations than those without progression (0.64±0.21 vs. 0.22±0.10 ng/mL;

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

p<0.001). Furthermore, procalcitonin showed a strong inverse correlation with eGFR (r=-0.71, p<0.001).

This retrospective study evaluated the role of serum procalcitonin levels in relation to chronic kidney disease staging and progression over a one-year period in patients attending a tertiary care center. The findings demonstrate a clear and progressive increase in procalcitonin levels with advancing stages of CKD and significantly higher levels among patients who exhibited progression in CKD staging. These observations support the concept that procalcitonin elevation in CKD is not solely related to infection but may reflect underlying inflammatory activity and reduced renal clearance associated with disease progression.

Procalcitonin Levels and Severity of Chronic Kidney Disease

One of the key findings of this study is the stepwise increase in serum procalcitonin levels with advancing CKD stage. Patients in early stages of CKD demonstrated relatively low procalcitonin levels, while those in advanced stages showed markedly elevated values. This trend suggests a strong association between declining renal function and accumulation of procalcitonin in the circulation.

Several mechanisms may explain this relationship. As kidney function deteriorates, the renal clearance of procalcitonin is reduced, leading to its accumulation. Additionally, advanced CKD is characterized by persistent low-grade inflammation driven by uremic toxins, oxidative stress, endothelial dysfunction, and immune dysregulation. These factors may stimulate increased production of procalcitonin independent of infection. Similar observations have been reported in previous studies, where elevated baseline procalcitonin levels were noted in patients with advanced CKD even in the absence of overt sepsis or bacterial infection [7].

The progressive rise in procalcitonin across CKD stages observed in this study suggests that procalcitonin may serve as a marker of disease severity rather than a binary indicator of infection in this population. This finding has important clinical implications, particularly in advanced CKD patients, where elevated procalcitonin levels may be misinterpreted as evidence of infection.

Association Between Procalcitonin and CKD Stage Progression

Another important observation from this study is the significantly higher procalcitonin levels in patients who demonstrated progression of CKD staging during the study period compared with those whose disease remained stable. This association suggests that procalcitonin may be linked not only to static disease severity but also to dynamic disease progression.

Chronic inflammation is a well-recognized driver of CKD progression. Pro-inflammatory cytokines promote glomerulosclerosis, tubular injury, and interstitial fibrosis, leading to irreversible loss of nephron mass. Elevated procalcitonin levels may reflect heightened inflammatory activity that accelerates these pathological processes. The higher procalcitonin concentrations observed in patients with CKD progression in this study support the hypothesis that inflammatory burden plays a key role in advancing renal dysfunction [8]. While causality cannot be established due to the retrospective design, the observed association raises the possibility that procalcitonin could serve as a surrogate marker for identifying patients at higher risk of CKD progression. Such patients may benefit from closer monitoring and more aggressive management of modifiable risk factors.

Interpretation of Procalcitonin in CKD Without Infection

Procalcitonin is widely used as a biomarker for bacterial infection and sepsis; however, its interpretation in CKD patients is challenging. Elevated procalcitonin levels in the absence of infection may lead to unnecessary antibiotic administration, increased healthcare costs, and contribute to antimicrobial resistance. The findings of this study highlight the need for cautious interpretation of procalcitonin values in patients with renal dysfunction.

In advanced CKD, baseline procalcitonin levels may exceed conventional cut-off values used to indicate infection in the general population. This elevation reflects reduced renal clearance and chronic inflammatory activation rather than acute bacterial infection. Previous studies have emphasized the importance of adjusting diagnostic thresholds or interpreting procalcitonin levels in conjunction with clinical findings and other inflammatory markers in patients with CKD [9]. The present study reinforces the concept that elevated procalcitonin in CKD should not automatically be equated with infection. Clinicians should consider CKD stage, trend of procalcitonin levels, and the overall clinical context when making diagnostic and therapeutic decisions.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

Clinical Implications in Tertiary Care Practice

The findings of this study have significant implications for clinical practice in tertiary care settings, where CKD patients frequently present with advanced disease and multiple comorbidities. Understanding the relationship between procalcitonin levels and CKD staging can aid clinicians in differentiating inflammation related to renal dysfunction from true infectious processes.

Incorporating procalcitonin trends rather than single absolute values may enhance its utility in CKD patients. Rising procalcitonin levels over time in the absence of infection may signal worsening inflammatory burden and potential disease progression. This could prompt clinicians to intensify management of contributing factors such as poor blood pressure control, glycemic dysregulation, or ongoing proteinuria. Furthermore, recognizing procalcitonin as a marker associated with CKD progression may help refine risk stratification strategies. Patients with persistently elevated procalcitonin levels may represent a subgroup at higher risk for rapid decline in renal function and adverse outcomes, warranting closer follow-up and early nephrology intervention [10].

Limitations and Directions for Future Research

Despite its strengths, including a sizable sample and representation across all CKD stages, this study has several limitations. The retrospective design limits the ability to establish temporal or causal relationships between procalcitonin elevation and CKD progression. In addition, residual confounding from subclinical infections or unmeasured inflammatory conditions cannot be entirely excluded. The study relied on single or limited procalcitonin measurements rather than serial assessments in all patients, which may limit evaluation of longitudinal trends. Moreover, inflammatory markers such as C-reactive protein or interleukin levels were not uniformly available for comparison, which could have strengthened the interpretation of the inflammatory component.

Future prospective studies with serial procalcitonin measurements and longer follow-up durations are needed to better elucidate the role of procalcitonin in predicting CKD progression. Integration of procalcitonin with other inflammatory and fibrosis biomarkers may provide a more comprehensive risk assessment model. Additionally, defining CKD-specific reference ranges for procalcitonin could improve its clinical utility and reduce misinterpretation in this patient population [11,12].

Overall Interpretation

In summary, this study demonstrates that serum procalcitonin levels increase progressively with advancing stages of chronic kidney disease and are significantly higher in patients who experience CKD stage progression. These findings suggest that procalcitonin reflects the inflammatory and metabolic burden associated with declining renal function rather than infection alone. Careful interpretation of procalcitonin levels in CKD patients is essential to avoid diagnostic errors and to harness its potential role as a marker of disease severity and progression.

Generalizability

The study included patients across all CKD stages from a tertiary-care center, improving applicability to similar hospital-based CKD populations. However, findings should be interpreted cautiously when extrapolating to community settings or different geographic regions due to the single-center retrospective design.

Recommendation

Routine interpretation of procalcitonin in CKD patients should consider renal function status and CKD stage. Serial monitoring may assist in identifying patients at risk of disease progression. Large prospective multicenter studies are required to establish CKD-specific procalcitonin reference ranges and prognostic thresholds.

Abbreviation Full Form

CKD Chronic Kidney Disease
eGFR Estimated Glomerular Filtration Rate
PCT Procalcitonin
IEC Institutional Ethics Committee
SPSS Statistical Package for the Social Sciences

Source of Funding

No external funding was received for this study. The funders had no role in study design, data collection, analysis, manuscript preparation, or publication decisions.

Conflict of Interest

The authors declare no conflict of interest.

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

Data Availability

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Author Contributions

Ayushi Chaudhari: Conceptualization, data collection, manuscript drafting.
Devopam Roy: Data analysis, interpretation, critical revision.
Syed Yasrib Nahid Zaidi: Study supervision, manuscript review, final approval.

Author Biography

Dr. Ayushi Chaudhari is an Assistant Professor in the Department of General Medicine at Naraina Hospital & Research Center, Uttar Pradesh, India, with research interests in nephrology and internal medicine.
Dr. Devopam Roy is an Associate Professor in the Department of General Medicine with expertise in chronic disease management and clinical research.
Dr. Syed Yasrib Nahid Zaidi is an Assistant Professor in the Department of General Medicine whose research focuses on renal diseases, inflammatory biomarkers, and evidence-based clinical practice.

CONCLUSION

This retrospective study highlights the significant association between serum procalcitonin levels and both the severity and progression of chronic kidney disease in patients managed at a tertiary care center. The findings demonstrate a clear and progressive rise in procalcitonin levels with advancing stages of CKD, as well as significantly higher levels among patients who exhibited progression in CKD staging over the study period. These observations suggest that procalcitonin elevation in CKD is not solely indicative of acute bacterial infection but may reflect underlying chronic inflammation and reduced renal clearance associated with worsening renal function. Recognizing this distinction is particularly important in advanced CKD, where misinterpretation of elevated procalcitonin levels may lead to unnecessary antimicrobial therapy. The association between higher procalcitonin levels and CKD stage progression further supports the role of inflammation as a key driver of renal disease advancement. From a clinical perspective, procalcitonin may serve as a useful adjunct biomarker for risk stratification and monitoring of patients with CKD when interpreted in conjunction with clinical findings and renal function parameters. However, its use should be cautious and contextual, especially in the absence of overt infection. Although the retrospective design and limited availability of serial measurements restrict causal inference, the study provides valuable real-world evidence from a tertiary care setting. Future prospective studies with larger cohorts, serial biomarker assessment, and longer follow-up are warranted to clarify the prognostic utility of procalcitonin and to establish CKD-specific interpretive thresholds. Overall, the findings emphasize the potential role of procalcitonin as a marker of disease severity and progression in chronic kidney disease, underscoring the need for careful interpretation and further investigation.

REFERENCES

1. Levey AS, Coresh J. Chronic kidney disease. The Lancet. 2012 Jan 14;379(9811):165-80. https://doi.org/10.1016/S0140-6736(11)60178-5
2. Stenvinkel P, Ketteler M, Johnson RJ, Lindholm B, Pecoits-Filho R, Riella M, Heimbürger O, Cederholm T, Girndt M. IL-10, IL-6, and TNF-α: central factors in the altered cytokine network of uremia-the good, the bad, and the ugly. Kidney International. 2005 Apr 1;67(4):1216-33. https://doi.org/10.1111/j.1523-1755.2005.00200.x
3. Tonelli M, Sacks F, Pfeffer M, Jhangri GS, Curhan G. Biomarkers of inflammation and progression of chronic kidney disease. Kidney International. 2005 Jul 1;68(1):237-45. https://doi.org/10.1111/j.1523-1755.2005.00398.x
4. KL B. Clinical review 167: Procalcitonin and the calcitonin gene family of peptides in inflammation, infection, and sepsis: a journey from calcitonin back to its precursors. J Clin Endocrinol Metab. 2004;89:1512-25. https://doi.org/10.1210/jc.2002-021444
5. Herget-Rosenthal S, Marggraf G, Pietruck F, Hüsing J, Strupat M, Philipp T, Kribben A. Procalcitonin for accurate detection of infection in haemodialysis. Nephrology Dialysis Transplantation. 2001 May 1;16(5):975-9. https://doi.org/10.1093/ndt/16.5.975
6. Meisner M. Update on procalcitonin measurements. Annals of laboratory medicine. 2014 Jun 19;34(4):263. https://doi.org/10.3343/alm.2014.34.4.263

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i2.2350
Original Article

7. AA D. Procalcitonin and C-reactive protein plasma concentrations in nonseptic uremic patients undergoing haemodialysis. Intensive Care Med. 2003;29:579-83. https://doi.org/10.1007/s00134-003-1664-8
8. Stenvinkel P. Inflammation in end-stage renal disease: The hidden enemy. Nephrology. 2006 Feb;11(1):36-41. https://doi.org/10.1111/j.1440-1797.2006.00541.x
9. Schuetz P, Briel M, Mueller B. Clinical outcomes associated with procalcitonin algorithms to guide antibiotic therapy in respiratory tract infections. Jama. 2013 Feb 20;309(7):717-8. https://doi.org/10.1001/jama.2013.697
10. Carrero JJ, Stenvinkel P. Inflammation in end-stage renal disease-what have we learned in 10 years?. In Seminars in dialysis 2010 Sep (Vol. 23, No. 5, pp. 498-509). Oxford, UK: Blackwell Publishing Ltd. https://doi.org/10.1111/j.1525-139X.2010.00784.x
11. Rhee C. Using procalcitonin to guide antibiotic therapy. In Open Forum Infectious Diseases 2017 (Vol. 4, No. 1, p. ofw249). US: Oxford University Press. https://doi.org/10.1093/ofid/ofw249
12. Meisner M, Schmidt J, Hüttner H, Tschaikowsky K. The natural elimination rate of procalcitonin in patients with normal and impaired renal function. Intensive care medicine. 2000 Mar;26(Suppl 2): S212-6. https://doi.org/10.1007/s001340051146

PUBLISHER DETAILS:

Student's Journal of Health Research (SJHR)
(ISSN 2709-9997) Online
(ISSN 3006-1059) Print
Category: Non-Governmental & Non-profit Organization
Email: studentsjournal2020@gmail.com
WhatsApp: +256 775 434 261
Location: Scholar's Summit Nakigalala, P. O. Box 701432, Entebbe Uganda, East Africa