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Relationship between Serum Uric Acid Levels and Inflammatory Markers in Patients with Chronic Kidney Disease

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ABSTRACT

Introduction: Chronic kidney disease (CKD) is a progressive condition characterized by gradual loss of renal function, often accompanied by metabolic disturbances and increased cardiovascular risk. Among the metabolic abnormalities observed in CKD, hyperuricemia has gained attention due to its potential role in promoting inflammation, endothelial dysfunction, and disease progression. Elevated serum uric acid (SUA) levels have been associated with increased levels of inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). This study aimed to evaluate the association between serum uric acid levels and inflammatory markers in patients with chronic kidney disease **Methods & Materials:** This study was a hospital, based cross-sectional observational one among 52 patients with chronic kidney disease (CKD) who were attending the Department of Biochemistry, Jalalabad Ragib-Rabeya Medical College and Hospital, Sylhet, Bangladesh, from January 2025 to December 2025. Patients (18 years) with a confirmed diagnosis of CKD based on KDIGO guidelines were included. Analysis of collected data was done by using SPSS version 26.0. **Result:** Serum uric acid levels rose progressively in line with the CKD stage, from 5.8 1.1 mg/dL in stage 2 to 8.3 1.6 mg/dL in stage 5 ($p < 0.001$). Inflammation markers were high throughout the group, with average values of CRP 8.6 4.2 mg/L, IL, 6 14.8 6.1 pg/mL, and TNF, 12.3 5.4 pg/mL. CRP, IL, 6, and TNF, levels were significantly higher in patients with hyperuricemia than in those with normal uric acid levels ($p < 0.01$). Besides, serum uric acid had moderate to strong positive associations with these markers (IL, 6 r = 0.61, CRP r = 0.58, TNF, r = 0.49). **Conclusion:** Serum uric acid levels not only increase progressively in line with the worsening of chronic kidney disease but also, they are highly related to, inflammatory markers such as CRP, IL, 6, and TNF, . Patients who suffer from hyperuricemia are characterized by a greater inflammatory state, and serum uric acid can be seen as strongly positively correlated with inflammatory markers, fomalting a direct linkage of hyperuricemia to systemic inflammation in CKD.

Keywords: *Serum Uric Acid, Chronic Kidney Disease, Inflammatory Markers*

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INTRODUCTION

Chronic kidney disease (CKD) is a progressive disorder leading to an irreversible decline in kidney function and is considered one of the leading public health problems globally [1]. Besides the conventional risk factors, chronic systemic inflammation has been identified as a key element in the pathology of CKD and its progression, which leads to endothelial dysfunction, faster atherosclerosis, and adverse clinical outcomes [2]. CKD patients are in a continuous pro, inflammatory state attributable to the decreased ability of the kidneys to clear inflammatory cytokines, oxidative stress, accumulation of uremic toxins, and stimulation of immune pathways. Consistently, raised levels of inflammatory markers like C, reactive protein (CRP), interleukin, 6 (IL, 6), and tumor necrosis factor, alpha (TNF alpha) have been reported in patients with CKD and these are linked to the extent of the disease and higher cardiovascular risk [3]. At present, these inflammatory markers are regarded as significant prognostic indicators in CKD. Serum uric acid (SUA), which is the end product of purine metabolism, is normally excreted mainly through the kidneys. In CKD, reduced glomerular filtration causes decreased uric acid excretion, thus resulting in hyperuricemia that is typically present even at the early stages of renal dysfunction [4]. Besides elevated SUA serving merely as a sign of renal impairment, it has also been increasingly identified as a factor causing kidney disease progression and cardiovascular complications [5]. Experimental and clinical studies have demonstrated that uric acid probably acts as a pro, inflammatory agent through generating oxidative stress, activating nuclear factor, kappa B (NF, B), causing endothelial dysfunction, and resulting in the release of inflammatory cytokines. Hyperuricemia is capable of stimulating the growth of vascular smooth muscle and of over, activation of the renin Angiotensin Aldosterone system, thus leading to further renal injury and systemic inflammation [6]. Several clinical studies have shown that there is a strong correlation between high levels of SUA and inflammatory markers in patients with CKD. The higher the SUA levels, the more it has been linked with the increase of CRP, IL, 6, and TNF, levels, thus indicating that uric acid may play a part in the chronic inflammatory state of CKD [7]. Research on subjects with moderate to severe CKD has revealed that hyperuricemia remains an independent predictor of raised inflammatory biomarkers after controlling for other variables such as age, diabetes, and renal function [8]. The association between serum uric acid and inflammatory markers in chronic kidney disease (CKD) has remained largely unclear, especially at the different stages of the disease, although there has been an increasing interest. There is a need to comprehend this association because hyperuricemia and inflammation are two separate factors that predict the worsening of renal and cardiovascular conditions. Hence, the present investigation will be focused on determining the link between serum uric acid levels and inflammatory markers in CKD patients.

METHODS & MATERIALS

This hospital-based cross-sectional observational study was conducted at the Department of Biochemistry, Jalalabad Ragib-Rabeya Medical College and Hospital, Sylhet, Bangladesh, and included 52 patients of chronic kidney disease (CKD) who visited the hospital between the period of January 2025 and December 2025. Diagnosis of chronic kidney disease (CKD)

that was confirmed, based on KDIGO guidelines, was one of the inclusion criteria for adult patients (> 18 years), who were sequentially enrolled; patients with acute kidney injury, active infection, autoimmune disease, malignancy, or recent usage of uric acid, lowering or anti, inflammatory drugs were excluded. Demographic and clinical data were obtained, and the CKD stages were determined by using the estimated glomerular filtration rate (eGFR). Venous blood samples were collected to measure serum uric acid, CRP, IL, 6, and TNF, alpha levels under sterile conditions using standard laboratory methods. The cut, off points for the diagnosis of hyperuricemia were used. Data analysis was performed using Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables were expressed as meanstandard deviation, while categorical variables were expressed as frequencies and percentages. The Students t test was used to compare groups, and Pearsons correlation coefficient to find the relation between serum uric acid and inflammatory markers. A $p<0.05$ was considered significant.

RESULTS

The average age of the individuals in the study was 49.6 12.8 years, with a male predominance (males 59.6%). Hypertension was the leading comorbidity, seen in 69.2% of patients, followed by diabetes mellitus at 53.8%. The average BMI was 24.9 3.6 kg/m, which means that most of the patients were from the normal to overweight category (*Table I*).

Table I
Baseline demographic and clinical characteristics of the study population (*n* = 52).

Parameter	Value
Mean age (years)	49.6 ± 12.8
Male: Female	31: 21
Mean BMI (kg/m²)	24.9 ± 3.6
Diabetes mellitus	28 (53.8%)
Hypertension	36 (69.2%)
Smoking history	14 (26.9%)

More than half of the patients were in the advanced stages of CKD, 67.3% of which were in stages 4 and 5. Stage 4 CKD accounted for the largest number (34.6%) thus the study population had a high proportion of advanced renal dysfunction cases (*Table II*).

Table II
Distribution of patients according to CKD stage (*n* = 52).

CKD Stage	Number (%)
Stage 2	6 (11.5%)
Stage 3	17 (32.7%)
Stage 4	18 (34.6%)
Stage 5	11 (21.2%)

Mean serum uric acid levels progressively rose with higher CKD stages, from 5.8 1.1 mg/dL at stage 2 to 8.3 1.6 mg/dL at stage 5. The increase was very statistically significant ($p < 0.001$), thus there is a very strong correlation between impairment of kidney function n and increase of uric acid levels (*Table III*).

Table III
Serum uric acid levels across different CKD stages (*n* = 52).

CKD Stage	Serum uric acid (mg/dL), Mean ± SD
Stage 2	5.8 ± 1.1
Stage 3	6.6 ± 1.3
Stage 4	7.5 ± 1.4
Stage 5	8.3 ± 1.6
p-value	<0.001

In the study group, all inflammatory markers measured were increased. Mean CRP was 8.6 4.2 mg/L, while IL, 6 and TNF, levels were 14.8 6.1 pg/mL and 12.3 5.4 pg/mL, respectively, thus, CKD patients are in a chronic inflammatory state (*Table IV*).

Table IV
Levels of inflammatory markers in the study population (*n* = 52).

Inflammatory marker	Mean ± SD
CRP (mg/L)	8.6 ± 4.2
IL-6 (pg/mL)	14.8 ± 6.1
TNF-α (pg/mL)	12.3 ± 5.4

Compared to patients with normal serum uric acid, those with hyperuricemia had substantially elevated levels of all the inflammatory markers. CRP, IL, 6, and TNF, levels were significantly increased in the hyperuricemic group with statistical significance ($p < 0.01$ for all comparisons) (*Table V*).

Table V
Comparison of inflammatory markers between normal uric acid and hyperuricemia groups (*n* = 52).

Parameter	Normal SUA (n = 21)	Hyperuricemia (n = 31)	p-value
CRP (mg/L)	6.1 ± 2.9	10.3 ± 4.1	<0.001
IL-6 (pg/mL)	11.2 ± 4.5	17.2 ± 5.8	<0.001
TNF-α (pg/mL)	9.4 ± 3.8	14.3 ± 5.6	0.002

Serum uric acid levels were found to be moderately to strongly positively correlated with inflammatory markers. The highest correlation was with IL, 6 (r = 0.61) followed by CRP (r = 0.58) and TNF, (r = 0.49). Since all correlations were statistically significant, hyperuricemia and systemic inflammation in CKD patients most likely closely correlate with one another (*Table VI*).

Table VI
Correlation between serum uric acid and inflammatory markers (*n* = 52).

Inflammatory marker	Correlation coefficient (r)	p-value
CRP	0.58	<0.001
IL-6	0.61	<0.001
TNF-α	0.49	0.001

DISCUSSION

This study showed that the average age was 49.6 12.8 years, most patients were male (59.6%), and the great majority were in CKD stages 35 (88.5%). Such a breakdown is biologically relevant, as the inflammatory burden is known to increase with the worsening of the disease. In the Chronic Renal Insufficiency Cohort (CRIC) study, Goncalves et al. showed that the average age was 58 11 years with 52% males and the authors demonstrated that the levels of inflammatory markers in patients with eGFR <45 mL/min/1.73 m were significantly higher than in earlier stages [9]. Even though our sample was a little younger, the fact that the majority of the patients were in the advanced stages explains the high inflammatory burden seen in our patients. It was statistically significant that mean SUA increased from 5.8 1.1 mg/dL in stage 2 to 8.3 1.6 mg/dL in stage 5 ($p < 0.001$). Jalal et al. demonstrated equivalent results in a study where mean SUA levels in CKD stage 3 were 6.2 1.4 mg/dL and in stages 45 were 8.1 1.7 mg/dL, and SUA was an independent predictor of CKD progression [8]. The close agreement between this study's values and those reported by Jalal et al. clarifies the idea that deterioration of renal function causes an increase in uric acid which is a result of decreased renal excretion. The patients showed a mean CRP level of 8.6 4.2 mg/L, IL, 6 was 14.8 6.1 pg/mL, and TNF, was 12.3 5.4 pg/mL, all reflecting an inflammatory state of great intensity. Stenvinkel et al. found patients with advanced CKD had mean CRP levels of around 912 mg/L, and IL, 6 levels of over 15 pg/mL in stages 45 disease [2]. Similarly, Gupta et al. recorded TNF, levels of 11.9 4.8 pg/mL in non, dialysis CKD patients [10]. These similar data indicate that the inflammatory pattern seen in this research is typical in moderate to severe stages of CKD. The patients having hyperuricemia in this research showed remarkably higher levels of CRP (10.3 4.1 vs 6.1 2.9 mg/L), IL, 6 (17.2 5.8 vs 11.2 4.5 pg/mL), and TNF, (14.3 5.6 vs 9.4 3.8 pg/mL) compared with the normouricemic ones. Kuwabara et al. found that CKD patients with SUA 7.0 mg/dL had average CRP levels of 9.8 3.6 mg/L which was higher than 5.9 2.4 mg/L in patients with the lower SUA, along with IL, 6 levels being significantly elevated [7]. The size and sign of the differences in this study correspond quite closely to these results and we interpret these as supportive of uric acid being involved in the systemic inflammation. We observed moderate to strong positive associations between SUA and CRP (r = 0.58), IL, 6 (r = 0.61), and TNF, (r = 0.49) in the present study. Ruggiero and colleagues in the CoLaus study conducted on a population sample, found less strong but still significant correlations between SUA and CRP (r = 0.200.30) as well as IL, 6 (r 0.10) [11]. The greater strength of the correlations seen in present study probably mirror the heightened inflammatory response and decreased renal clearance in CKD as compared to the general population. Besides, Johnson et al. also revealed that uric acid could trigger pro, inflammatory pathways like NF, B, thus explaining the link between uric acid and inflammation found in the study [4]. Feig et al. gathered the evidence that CKD patients generally have serum uric acid levels between 7.0 and 9.0 mg/dL in later stages, and at the same time, they have higher inflammatory markers, particularly CRP values which often exceed 8 mg/L and IL, 6 levels above 1015 pg/mL. Our results are very similar to these documented ranges, both accounts showing that the mean values of SUA, CRP and IL, 6 in stage 5 CKD patients were 8.3 1.6 mg/dL, 8.6 4.2 mg/L, and 14.8 6.1 pg/mL, respectively [12].

LIMITATIONS

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSION

Serum uric acid concentration increases as the severity of chronic kidney disease progresses. Uric acid is also significantly related to higher levels of inflammatory markers such as CRP, IL, 6, and TNF alpha. Those who are hyperuricemic have a higher level of inflammation than the normal range individuals. Besides, serum uric acid is highly correlated with inflammatory markers, which denotes that hyperuricemia and systemic inflammation in CKD are tightly connected.

RECOMMENDATION

Patients suffering from chronic kidney disease, especially those with advanced stages, should be regularly checked for serum uric acid and inflammatory markers. Early detection and proper treatment of hyperuricemia may be useful in decreasing the inflammation load and subsequently the progression of the disease. More studies over time and of interventional nature are needed to establish whether treatment to lower uric acid levels could enhance the inflammatory condition and health outcomes in CKD patients.

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CONFLICT OF INTEREST

None declared.

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