Original Article

Prevalence of Hyperuricemia in Chronic Kidney Disease Patients: A Descriptive Study

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Abstract

Background: Hyperuricemia is frequently observed in chronic kidney disease (CKD) patients and is often associated with a decrease in the renal excretion of uric acid. Increasing serum uric acid (SUA) could not be solely the result of renal dysfunction but act as a factor for the progression of renal dysfunction, as emerged evidence suggests. **Material and Methods:** To assess the frequency of hyperuricemia in patients with CKD and its relationship with renal function tests and associated co-morbidities. Design is a descriptive study was carried out among patients who were diagnosed with CKD (Stages 2-5) of 100 individuals. Serum uric acid, serum creatinine, blood urea and estimated glomerular filtration rate (eGFR) were assessed. One-way ANOVA was used to compare SUA among the various stages of CKD and Pearson correlation was used to evaluate the association between SUA and renal parameters. **Results:** Hyperuricemia was present in 68.0% of the people in the study. The mean SUA level was significantly different with different stages of CKD ($p < 0.001$). There was strong positive correlation with SUA and serum creatinine ($r = +0.62$) and blood urea ($r = +0.55$) and strong negative correlation with eGFR ($r = -0.68$). There was significant correlation between hyperuricemia and hypertension (79.4%) and diabetes mellitus (61.8%). **Conclusion**: Hyperuricemia is very common in persons with CKD, and is associated with clinically relevant cardiovascular comorbidities and the degree of renal impairment. In comprehensive treatment of CKD patients, it is possible that it is necessary to set up a uric acid monitoring and management plan.

Keywords: Hyperuricemia, Chronic Kidney Disease, Serum Uric Acid, Renal Function, Estimated Glomerular Filtration Rate (eGFR), Kidney Disease.

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Introduction

Chronic kidney disease (CKD) is a world-wide public health problem that involves progressive loss of renal function with a variety of metabolic and systemic complications.[1] Of these, there is hyperuricemia, which is the presence of abnormally high blood uric acid levels, which is commonly found due to the kidneys' decreased ability to excrete urate.[2] In human, uric acid is the end product of purine metabolism, with about two-thirds of uric acid daily excretion occurring through the renal pathway.[3] Thus, in the CKD patients, the level of serum uric acid (SUA) is usually increased as the glomerular filtration rate (GFR) decreases, and the hyperuricemia is burdensome for this patient population.[4] Recently, epidemiological studies have found that hyperuricemia does not only represent a decreased renal clearance, but could also have a pathogenic role in the pathogenesis and progression of KD.[5] It has been proposed that the effects of CPH are mediated by systemic and glomerular hypertension, activation of the renin-angiotensin system and renal inflammation and fibrosis.[6] Moreover, high SUA concentrations have also been independently linked to the risk of cardiovascular morbidity and mortality in subjects with abnormal kidney function.[7] The background frequency and clinical correlates for hyperuricemia in the various stages of CKD are therefore important for planning the best treatment approaches and for possibly slowing the course of CKD.

Materials and Methods

Study Design and Setting: The present descriptive study is carried out in a tertiary hospital for the purpose of studying the prevalence of hyperuricemia in chronic kidney disease patients. In total, 100 patients with clinical and laboratory diagnosis of CKD were enrolled in the study.

Inclusion and exclusion criteria are provided.

Inclusion criteria

Ages ≥ 18 years old, CKD (Stages 2 to 5) with eGFR based on the MDRD (Modification of Diet in Renal Disease) formula.

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Exclusion Criteria

Acute kidney injury, medications known to have significant effect on uric acid levels (such as high dose aspirin, diuretics, urate-lowering therapy), gouty arthritis before the appearance of CKD.

Data Collection and Laboratory Analysis

Demographic parameters (age, sex), comorbidities (hypertension, diabetes mellitus) were documented. All blood samples were taken from the veins under sterile conditions. Standard laboratory techniques were used to determine the following biochemical parameters:

1. Serum Uric Acid (SUA): By uricase-PAP method. Hyperuricemia was defined as SUA levels > 7.0 mg/dL in men and > 6.0 mg/dL in women.
2. Serum Creatinine: measured by the Jaffe's kinetic method.
3. Blood Urea: The urease-GLDH method is used to measure blood urea.
4. eGFR: Calculated using the MDRD equation.

Statistical Analysis: The SPSS version 27.0 software package was used to analyse data. Results of continuous variables were presented as Mean ± Standard Deviation (SD) whereas categorical variables were presented as frequencies and percentages. For comparison, mean SUA levels among the various stages of CKD were compared using one-way ANOVA. Pearson's correlation coefficient (r) was used to assess correlation between SUA and renal function parameters (creatinine, urea and eGFR). A p value of < 0.05 were regarded as significant.

Results

[Table 1] depicts the prevalence of hyperuricemia among patients with chronic kidney disease. Hyperuricemia was observed in 68% of the study population, indicating a high burden of elevated serum uric acid levels among CKD patients.

Table 1: Frequency Distribution of Hyperuricemia among CKD Patients (n = 100)

Hyperuricemia Status	Frequency	Percentage (%)
Yes	68	68.0
No	32	32.0
Total	100	100.0

Table 2: Distribution of CKD Stages in the Study Population

CKD Stage	eGFR Range (ml/min/1.73 m²)	Number of Patients	Percentage (%)
Stage 2	60–89	14	14.0
Stage 3	30–59	36	36.0
Stage 4	15–29	28	28.0
Stage 5	<15	22	22.0
Total	—	100	100.0

[Table 2] shows the distribution of patients according to CKD staging based on estimated glomerular filtration rate (eGFR). The majority of patients were in stage 3 and stage 4 CKD.

Table 3: Serum Uric Acid Levels According to CKD Stage

CKD Stage	Mean Serum Uric Acid (mg/dL)	Standard Deviation
Stage 2	8.49	1.35
Stage 3	7.66	1.46
Stage 4	7.48	1.55
Stage 5	7.87	1.32

[Table 3] demonstrates the variation in mean serum uric acid levels across different stages of CKD. One-way ANOVA showed a statistically significant difference in serum uric acid levels across CKD stages ($p < 0.001$).

Table 4: Comparison of Renal Function Parameters Based on Hyperuricemia Status

Parameter	Hyperuricemia (n = 68) Mean ± SD	Normal Uric Acid (n = 32) Mean ± SD	p-value
Serum Creatinine (mg/dL)	4.6 ± 2.1	2.9 ± 1.4	<0.001
Blood Urea (mg/dL)	86.3 ± 28.5	61.4 ± 22.3	<0.01
eGFR (ml/min/1.73 m²)	26.4 ± 14.2	42.8 ± 18.6	<0.001

[Table 4] table compares renal function parameters between CKD patients with and without hyperuricemia. Patients with hyperuricemia had significantly higher serum creatinine and blood urea levels and significantly lower eGFR values.

Table 5: Correlation Between Serum Uric Acid and Renal Parameters

Parameter	Correlation Coefficient (r)	p-value
Serum Creatinine	+0.62	<0.001
Blood Urea	+0.55	<0.001
eGFR	−0.68	<0.001

[Table 5] shows the correlation analysis between serum uric acid and renal function parameters. Serum uric acid showed

a strong positive correlation with serum creatinine and blood urea, and a strong negative correlation with eGFR.

Table 6: Association of Hyperuricemia with Comorbidities

Comorbidity	Hyperuricemia n (%)	Normal Uric Acid n (%)	p-value
Hypertension	54 (79.4)	18 (56.2)	0.02
Diabetes Mellitus	42 (61.8)	14 (43.7)	0.04
Cardiovascular Disease	24 (35.3)	6 (18.7)	0.05

[Table 6] highlights the association between hyperuricemia and common comorbid conditions in CKD patients. Hyperuricemia was significantly associated with hypertension and diabetes mellitus.

Discussion

The present study comprehensively examines the prevalence, disease stage dependence, correlation with renal function parameters and association with common comorbidities of hyperuricemia in patients with chronic kidney disease (CKD). Our results highlight the important burden of high serum uric acid (SUA) levels in this population and the direct correlation with the severity of renal impairment.

In our study, the prevalence of hyperuricemia was 68.0% (Table 1), with more than two-third of the patients who have CKD being affected by this metabolic anomaly. This is similar to the prevalence reported by Yirdaw et al,[8] which was 66.1% in one center hospital based study in Ethiopia. Likewise, Alsultan et al,[9] reported the prevalence of 55.5% in 3–5 stages of CKD and the severity of burden was greater with the advancement of stage 5 renal failure (ESRD). In the context of this high prevalence of hyperuricemia in our cohort, the progressive reduction in renal excretion of urate in the presence of decrease in glomerular filtration rate (GFR) was identified as an important factor contributing to urate accumulation, which was also observed by Takata et al. in their recent review of urate handling in kidney disease.[10]

In our study, the distribution of the CKD stages demonstrated that the majority of patient population was in the CKD stage 3 (36.0%) and stage 4 (28.0%) [Table 2]. Interestingly, the mean SUA level was significantly different during these stages ($p < 0.001$), and the mean level was the highest at 8.49 ± 1.35 mg/dL during Stage 2 [Table 3]. This report differs slightly with the study of Rizwan and Akhter,[11] employing one-way ANOVA where they demonstrated that SUA levels were generally found to be progressive in line with an increase in stage of the CKD. But the high SUA in early stages (Stage 2) is in line with the hypothesis of Yang et al,[12] that hyperuricemia could develop at an early stage of renal disease and represent a principal factor of renal fibrosis and of subsequent functional renal impairment.

A critical outcome of our study was the comparison of renal function parameters based on hyperuricemia status [Table 4]. Patients with hyperuricemia had significantly higher serum creatinine (4.6 ± 2.1 mg/dL vs. 2.9 ± 1.4 mg/dL; $p < 0.001$) and blood urea (86.3 ± 28.5 mg/dL vs. 61.4 ± 22.3 mg/dL; $p < 0.01$), along with significantly lower eGFR values (26.4 ± 14.2 vs. 42.8 ± 18.6 ml/min/1.73 m²; $p < 0.001$). Similarly Silva et al,[13] showed that SUA levels were significantly higher in patients with advanced renal failure (ARF), and that SUA was a good predictor of renal function. The very high eGFR difference of the 2 groups, hyperuricemic and normouricemic, confirms the metabolic role of SUA as a parameter of reduced renal function, as pointed by Perrenoud et al.[14]

Our correlation analysis [Table 5] showed a strong positive correlation between SUA and blood urea ($r = +0.55$, $p < 0.001$) and serum creatinine ($r = +0.62$, $p < 0.001$) and a strong negative correlation with eGFR ($r = -0.68$, $p < 0.001$). The correlation values obtained here are consistent with the values reported by Pandya et al,[15] who reported a correlation between the serum uric acid level and the rise in other nitrogenous wastes such as urea and creatinine in patients with CKD. Additionally, our study showed the inverse relationship of SUA to eGFR, which also was reported by Nie et al,[16] in their study after adjusting for multiple covariates, indicating that SUA is an independent factor related to renal functional decline.

Lastly, we found that hyperuricemia was significantly associated with comorbidities [Table 6]. Hyperuricemia was significantly associated with hypertension (79.4% vs. 56.2%; $p = 0.02$) and diabetes mellitus (61.8% vs. 43.7%; $p = 0.04$). The interdependency between hyperuricemia, hypertension and progression to CKD has been previously described by Ohno,[17] in which the presence of hyperuricemia leads to systemic hypertension via activation of the renin-angiotensin system. Moreover, as an important finding, the association of cardiovascular disease (35.3% vs. 18.7%; $p = 0.05$) was found in our study at the borderline of significance. Yokose et al,[18] stressed the importance of hyperuricemia as a potentially modifiable risk factor for the development of cardiovascular morbidity in patients with metabolic syndrome and renal insufficiency.

The high correlations with the widely used indicators of renal function like creatinine, urea and eGFR, as well as with hypertension and diabetes, indicates that SUA is an important parameter in the clinical management of the CKD. The results here confirm Takata et al,[10] proposal to introduce urate-lowering therapies into the treatment of CKD patients as a routine treatment to potentially reduce the progression of the disease and any associated comorbidities.

Conclusion

Conluded as Hyperuricemia' is a very common condition among patients with CKD, and it is strongly associated with the severity of renal failure and other cardiovascular comorbidities. The control and management of uric acid level could be important parts of the total management of a CKD patient, indirectly affecting the progress of the disease and the incidence of secondary complications.

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Conflicts of interest

There are no conflicts of interest.

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