Original Article

Comparative Analysis of Serum Uric Acid Levels in Hypertensive Patients With and Without Chronic Kidney Disease.

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ABSTRACT

Background: High blood pressure is associated with chronic kidney disease (CKD) due to elevated serum uric acid levels, which are a marker for kidney disease. Nonetheless, few studies compare blood pressure between CKD patients with and without hypertension.

Objective: To determine the difference in serum uric acid levels between hypertensive patients with CKD and those without, and to assess the role of each in the development of complications.

Methodology: A total of 180 hypertensive patients aged 30–65 years (mean age: 47.5 ± 8.2 years) were included in the study. The participants were divided into two groups: hypertensive patients with CKD (eGFR <60 mL/min/1.73 m²) and hypertensive patients without CKD. Subsequently, SUA, serum creatinine, and eGFR were analyzed. Statistical analyses involved the determination of the mean differences and correlations.

Results: out of 180 Patients with CKD and hypertension had a higher mean age of SUA (7.8 ± 1.3 mg/dL) compared to patients without CKD (5.6 ± 1.1 mg/dL, $p<0.001$). With higher levels of eGFR, SUA concentration drops ($r = -0.42$, $p<0.01$). No significant differences were noted in relation to sex. In the CKD group, the serum creatinine concentration was also higher (1.7 ± 0.4 mg/dL) compared to the control group (0.9 ± 0.2 mg/dL, $p < 0.001$).

Conclusion: Hypertensive patients with CKD demonstrate significantly elevated SUA levels, suggesting that SUA may contribute to renal dysfunction. Routine monitoring of SUA could facilitate earlier detection of kidney involvement in hypertensive patients.

Keywords: Uric acid, Hypertension,Chronic kidney disease, Renal function

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INTRODUCTION

Hypertension is one of the many contributors globally to chronic kidney disease, which is characterized by CKD-related illnesses, heavily advanced cases, and mortality associated with cardiovascular diseases [1]. The importance of identifying the CKD-related hypertension markers lies in the early disease identification and treatment [2]. Uridine acid is associated with the metabolism of purines, which is linked to hypertension and kidney dysfunction [3]. SUA and hyperuricemia are presumed to contribute to endothelial dysfunction and kidney injury through oxidative stress and the activation of the renin-angiotensin system [4,5]. Epidemiological studies have shown that individuals with hyperuricemia and decreased GFR have a higher likelihood of experiencing the condition, suggesting that urate accumulation may contribute to the progression of CKD [6,7]. Given the presence of diabetes, hypertension, and proteinuria, there is a considerable risk of advanced kidney disease in individuals with high SUA, as highlighted in long-term studies [8]. Urate-lowering therapy with allopurinol has been shown to improve cardiovascular and renal outcomes in patients with CKD [9,10]. However, routine guidelines for assessing the risk of chronic kidney disease do not currently include measures for SUA. Data on how often blood creatinine rises independently and how often it is simply a result of kidney impairment are not the same [11,12]. In this way, looking at SUA in hypertensive people with and without CKD may show its clinical importance among those who have hypertension. In parts of the world with low and middle incomes, where education about and access to CKD screening are not widespread, SUA could make diagnosis more practical [13]. However, information from local hypertensive groups that could help explain this is not widely available. The purpose of this study is to compare serum uric acid levels in hypertensive patients who have CKD with those who do not and investigate their relationship with renal function (eGFR). Having a well-defined connection could help use SUA as a usual metric in treating hypertension and detecting early CKD.

RESEARCH OBJECTIVES

To compare serum uric acid levels with hypertensive patients that also have chronic kidney disease and those that do not have chronic kidney disease, and examine the association with other renal function indicators, more specifically estimated glomerular filtration rate.

MATERIALS & METHODS

Study Design & Setting: This Cross Sectional Study Conducted at Department of Nephrology Unit, Khyber Teaching Hospital Peshawar from jan 2023 to jan 2024

Sample Size: A total of 180 hypertensive patients were enrolled, with 90 having CKD and 90 without CKD.

INCLUSION CRITERIA

Adults aged 30–65 years who attended outpatient clinics with or without chronic kidney disease.

EXCLUSION CRITERIA

Patients with diabetes mellitus, gout, malignancy, infections, or those using urate-lowering drugs (e.g., allopurinol, febuxostat) within the past three months were excluded.

DATA COLLECTION

Demographic data, clinical history, and laboratory values (SUA, serum creatinine, and eGFR) were collected. Blood pressure was recorded as the average of two seated measurements.

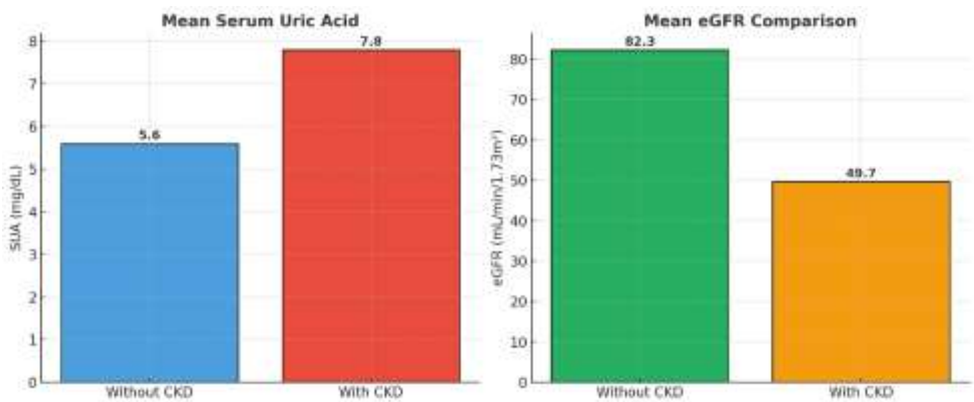
STATISTICAL ANALYSIS

Data were analyzed using SPSS version 24.0 (IBM Corp, Armonk, NY). Continuous variables were expressed as mean ± SD. Independent *t*-tests compared group means, and Pearson correlation assessed associations between SUA and eGFR. A *p*-value <0.05 was considered statistically significant.

RESULTS

Among the 180 included hypertensive patients, we analyzed 90 hypertensive patients with and 90 without chronic kidney disease (CKD). The mean ages were comparable (54.1 ± 7.9 and 52.4 ± 8.3 years). The distributions of gender were also similar. The mean serum urate concentration (SUA) of patients with CKD was much higher (7.8 ± 1.3 mg/dL) than of those without CKD (5.6 ± 1.1 mg/dL, $p<0.001$). Of the four definitions of CKD used, the mean serum creatinine rule (with 1.7 ± 0.4 mg/dL) and other unmentioned criteria indicated elevated status of the CKD group, whereas the mean eGFR was lower (49.7 ± 6.3) to CKD non affected group (with 82.3 ± 5.6 mL/min/1.73 m², $p<0.001$). In patients with CKD, there was an inverse correlation of eGFR ($r = -0.42$, $p<0.01$). No differences were found between the sexes and urate levels.

Fig 1. Comparison of Mean Serum Uric Acid and eGFR Levels in Hypertensive Patients With and Without CKD



The bar charts show mean serum uric acid (SUA, mg/dL) and estimated glomerular filtration rate (eGFR, mL/min/1.73 m²) among hypertensive patients with and without chronic kidney disease (CKD). Patients with CKD had significantly higher SUA levels (7.8 ± 1.3 mg/dL vs. 5.6 ± 1.1 mg/dL, $p<0.001$) and lower eGFR (49.7 ± 6.3 vs. 82.3 ± 5.6 mL/min/1.73 m², $p<0.001$). Error bars represent standard deviation.

Table 1. Baseline Characteristics of Hypertensive Patients With and Without CKD

Variable	Hypertension without CKD (n=90)	Hypertension with CKD (n=90)	*p*-value
Mean Age (years)	52.4 ± 8.3	54.1 ± 7.9	0.21 (NS)
Male, n (%)	48 (53.3%)	50 (55.6%)	0.77 (NS)
Female, n (%)	42 (46.7%)	40 (44.4%)	
Mean SBP (mmHg)	142 ± 12	145 ± 13	0.12 (NS)
Mean DBP (mmHg)	89 ± 9	90 ± 10	0.38 (NS)

Table 1 shows baseline demographic and clinical characteristics of hypertensive patients stratified by CKD status. There were no significant differences between groups regarding age, gender distribution, or blood pressure values.

Table 2. Serum Uric Acid and Renal Function in Hypertensive Patients With and Without CKD

Parameter	Hypertension without CKD (n=90)	Hypertension with CKD (n=90)	*p*-value
Serum Uric Acid (mg/dL)	5.6 ± 1.1	7.8 ± 1.3	<0.001 (HS)
eGFR (mL/min/1.73 m²)	82.3 ± 5.6	49.7 ± 6.3	<0.001 (HS)
Serum Creatinine (mg/dL)	0.9 ± 0.2	1.7 ± 0.4	<0.001 (HS)
BUN (mg/dL) [if tested]*	—	—	—

Table 2 compares mean serum uric acid, eGFR, and serum creatinine between hypertensive patients with and without CKD. Patients with CKD had significantly higher SUA and creatinine, and lower eGFR values. (*BUN included if data available*).

Table 3. Correlation between Serum Uric Acid and Renal Function Parameters

Parameter	Correlation Coefficient (*r*)	*p*-value	Strength of Association
SUA vs. eGFR	–0.42	<0.01 (S)	Moderate Negative
SUA vs. Serum Creatinine	+0.39	<0.01 (S)	Moderate Positive

Table 3 shows correlation analysis between serum uric acid (SUA) and renal function markers. SUA correlated negatively with eGFR and positively with serum creatinine, both statistically significant.

DISCUSSION

This study demonstrates that CKD with hypertension will result in higher levels of SUA as compared to hypertensive patients without CKD. This further cements our hypothesis that SUA may be responsible for some of the pathologic processes in renal impairment in CKD hypertensive individuals.This is in line with the findings of Jalal et al. as they noted higher levels of SUA in patients with hypertension and early CKD, further adding to the argument of SUA serving as an early biomarker [14-15]. In the same Study, Kanbay et al. mentioned an inverse relationship of SUA with eGFR in the hypertensive subpopulation [16-19]. The negative correlation noted herein reinforces the indicator role of SUA in renal dysfunction.SUA may worsen renal impairment pathophysiological due to its effects of inducing renal glomerular and vascular structural changes such as the endothelium, and vascular smooth muscle and progressive system hyperplasia or growth due to the activation of the renin-angiotensin system, oxidative stress, and glomerular hyperfiltration [20]. These processes lead to nephron loss and glomerular injury further propagating the damaged kidney cycle.Some argue that higher levels of SUA indicate a lack of renal clearance and is not a risk factor of disease in and of itself [21-23]. The evidence from interventional studies are conflicting- the FEATHER trial suggested that renal decline was slowed with the use of febuxostat [24] while other studies reported little change.The practical value of SUA testing in resource-limited settings cannot be overstated. Given the low cost of SUA measurement, it can be a valuable supportive test in the management of hypertension to detect early renal impairment. Longitudinal studies to establish causality, as well as the effect of urate-lowering therapy on long-term renal outcomes in non-gout hypertensive patients, are still needed [25].

LIMITATIONS

This study was conducted at only one center, with a small group of participants and limited application. The study did not look at changes in data over time. Diet, medications and genetic background were not controlled for in this experiment. Because of the study's design, it is not possible to confirm whether SUA causes CKD progression.

CONCLUSION

SUA levels are significantly higher in hypertensive patients with CKD compared to those without. SUA may serve as an early risk marker for renal impairment in hypertension. Routine monitoring of SUA in hypertensive patients could aid in earlier detection and prevention of CKD progression.

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AUTHORS CONTRIBUTION

Concept & Design of Study: Noor Mohammad

Data Collection: Muhammad Waqas

Drafting:Zareen Ullah

Data Analysis:Noor Mohammad

Critical Review:Noor Mohammad

Final Approval of version: All Authors Approved The Final Version.

Accountability: All authors agree to be accountable for all aspects of the work.

All authors contributed significantly to the study's conception, data collection, analysis,

Manuscript writing, and final approval of the manuscript as per **ICMJE criteria.**

RESEARCH ETHICS STATEMENT

There were no animal studies conducted. This study was approved by the **Institutional Review Board (IRB No:1123/MTI/KTH/09/2022**) and conducted in accordance with the ethical principles of the Declaration of Helsinki (2013).All participants or legal guardians signed written informed consent. No recognizably identifiable human data were included. As described in the article and supplementary materials, data that that under or findings are held in online repositories.

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