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Original Research Article

Correlation of the severity of chronic kidney disease with serum uric acid

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ABSTRACT

Background: Chronic kidney disease (CKD) is marked by kidney damage or a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for at least three months, regardless of the underlying etiology. When a variety of renal problems are present, albuminuria, defined as an albumin-to-creatinine ratio >30 mg/g in two out of three spot urine samples, can serve to determine kidney failure.The estimated global rate of CKD is 13.4%. In India, one of the most widespread illnesses that is not transmissible. CKD has a significant morbidity, mortality rate, and financial impact. Around 5.2 million people in India died from CKD-related causes in 2008 and that number could rise to 7.63 million by 2023. Some Indian states, including Puducherry, Andhra Pradesh, Maharashtra, and Odisha have been identified as CKD hotspots. Uric acid is a common component because purine nucleotides are biologically broken down into it. Gout and associated diseases, including diabetes and the formation of ammonium acid urate kidney stones, are all related to high blood levels of uric acid. Serum uric acid is eliminated principally by the kidneys and while there is a compensatory increased removal by the gut in the setting of renal insufficiency, this is not completely effective and serum uric acid increases as the GFR falls with approximately half of the subjects becoming hyperuricemic by the time dialysis is initiated.

Objectives: The aim of the present study was to assess the serum uric acid in CKD and study the correlation between eGFR (which is a marker of severity of CKD) and serum uric acid in CKD.

Materials and Methods: The present study was an observational study. The study was conducted over a period of six months on 180 patients. Blood samples were obtained in Becton Dickinson's commercially available red capped tubes vacutainers (BD). After that, blood samples were left undisturbed at room temperature for 15-30 minutes to coagulate. For 5 minutes, the tubes were centrifuged at 3000 rpm. After centrifugation, the sample solution (serum) was transferred to a fresh polypropylene tube with a Pasteur pipette. Serum uric acid and serum creatinine was done on fully automated SYSMEX BX-3010.

Results: Our results show that mean and standard deviation of serum uric acid with p value between males and females in the different stages of chronic kidney disease which shows a statistically significant difference between stage II, stage III B and stage IV. p-value was found to be statistically significant (p=0.0042, 0.0004 and 0.0487 respectively).

Conclusion: The present study highlights the progressive increase in serum uric acid levels as CKD advances through its stages.

1. Introduction

An international public health issue, kidney failure has a rising frequency and incidence, significant expenditures,

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and unfavorable consequences. Even more people have chronic kidney disease (CKD), which has side effects such as renal function loss, cardiovascular disease (CVD), and early death.[1]

CKD is marked by kidney damage or a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for at least three months, regardless of the underlying etiology. When a variety of renal problems are present, albuminuria, defined as an albumin-to-creatinine ratio >30 mg/g in two out of three spot urine samples, can serve to determine kidney failure.[2] The estimated global rate of CKD is 13.4%.[3] In India, one of the most widespread illnesses that is not transmissible. CKD has a significant morbidity, mortality rate, and financial impact. Around 5.2 million people in India died from CKD-related causes in 2008 and that number could rise to 7.63 million by 2023. Some Indian states, including Puducherry, Andhra Pradesh, Maharashtra, and Odisha, have been identified as CKD hotspots.[4]

The three most typical CKD causes are glomerulonephritis, diabetes mellitus and hypertension. One in three persons with diabetes and one in five adults with hypertension both have CKD.[5] The primary elements of the diagnosis of CKD are the estimated glomerular filtration rate (eGFR), serum creatinine level and patient's medical history.[6]

Chronic kidney disease is classified into different stages based on the eGFR:

Table 1: Chronic kidney disease stages based on eGFR[7]

Stages	GFR value ml/ min/1.73m ^{2}	Classification
I	>90	Normal or high
II	60-89	Slightly decline
III A	45-59	Mild to moderately decline
III B	30-44	Moderately to severely decline
IV	15-29	Severely decline
V	<15	Renal failure

The serum creatinine serves as the foundation for the eGFR, which is proportional to 1/creatinine, meaning that the two are inversely correlated, with the eGFR decreasing as creatinine increases.[8] Serum creatinine is a waste product of muscle metabolism that is easily measured and eliminated by the kidneys; it serves as an essential biomarker of renal function. In the body, creatine, phosphocreatine also referred to as creatine phosphate and adenosine triphosphate (ATP) work together to make creatinine.[9] Both the CKD-EPI equation and the Modified Diet in Renal Disease (MDRD) equation incorporate serum creatinine to determine GFR.[10] These eGFR equations are preferred over serum creatinine alone because they take into consideration variables including race, age, and gender.[11]

Uric acid is a common component because purine nucleotides are biologically broken down into it.[12] Gout and associated diseases, including diabetes and the formation of ammonium acid urate kidney stones, are all related to high blood levels of uric acid.[13] The production of uric acid from xanthine and hypoxanthine, which are then created from other purines and are both catalyzed by the enzyme xanthine oxidase. Additionally, under hypoxic circumstances (low oxygen saturation), uric acid is released. Elevated uric acid levels in the blood are referred to as hyperuricemia. In the pH range of physiological fluids, uric acid exists mostly as the ion urate.[14] Hyperuricemia is defined as serum uric acid concentrations beyond the normal range, which is 6 mg/dL for women, 7 mg/dL for men, and 5.5 mg/dL for children (under 18 years old). Low uric acid levels, or hypouricemia, are abnormal.

High intakes of sucrose, high-fructose corn syrup, and dietary purine can raise uric acid levels.[15] Increased serum uric acid levels may result from decreased renal excretion.

Serum uric acid is eliminated principally by the kidneys and while there is a compensatory increased removal by the gut in the setting of renal insufficiency, this is not completely effective and serum uric acid increases as the GFR falls with approximately half of the subjects becoming hyperuricemic by the time dialysis is initiated.[16] This makes it very difficult to assess the role of uric acid in the progression of renal disease in subjects with CKD.

So, the present study has been planned to study the severity of chronic kidney disease with serum uric acid.

2. Aims and Objectives

The aim of the present study was to assess the serum uric acid in CKD and study the correlation between eGFR (which is a marker of severity of CKD) and serum uric acid in CKD.

3. Materials and Methods

For serum uric acid testing, all blood samples taken in plain vacutainer tubes were sent to the Biochemistry laboratory of the Biochemistry Department, AIMSR. The investigation lasted six months (October 2022 to March 2023) after receiving clearance from the AIMSR Research Committee and the Ethics Committee of Biomedical and Health Research, Adesh University, Bathinda.

3.1. Sample size

The sample size was calculated by using Daniel's formula:

Z^2pq/e^2

The notion for the formula is:

Z=1.96

p=prevalence (prevalence of chronic kidney disease according to previous studies is 13% (p=0.13))

q=1-p

e=allowable error, and in this case, it is 10% of p

According to the formula, the calculation was done and the sample size was calculated to be 180.

3.2. Inclusion criteria

All the diagnosed cases of chronic kidney disease (all the patients were evaluated for chronic kidney disease as per the K/DOQI criteria by the National Kidney Foundation for diagnosis of CKD).[17]

3.3. Exclusion criteria

1. All HIV-positive individuals.
2. All the patients have a history of gout and /or hyperuricemia.
3. Patients who are taking anti-tubercular drugs and thiazide diuretics.

3.4. Methodologies and experimental strategy

3.4.1. Serum preparation

Blood samples were obtained in Becton Dickinson's commercially available red-capped tubes vacutainers (BD). After that, blood samples were left undisturbed at room temperature for 15-30 minutes to coagulate. For 5 minutes, the tubes were centrifuged at 3000 rpm. After centrifugation, the sample solution (serum) was transferred to a fresh polypropylene tube with a Pasteur pipette.

3.4.2. Specimen storage and handling during testing

During testing, the temperature of the specimens was kept between 20 and 25 degrees Celsius. Specimens were kept at 4-8°C for a maximum of 8 days. If the samples required to be preserved for a longer amount of time, they were put in a deep freezer at -20°C

3.5. Procedure

All the samples were collected from the patients attending the IPD and OPD Department of Adesh Institute of Medical Science and Research, Bathinda. Serum uric acid and serum creatinine were done on fully automated SYSMEX BX-3010.

3.6. Renal parameters

The following renal parameters were assessed in the following study:

1. Serum creatinine
2. Serum uric acid
3. eGFR

Parameters	Methods	Biological reference
Creatinine	Jaffe's method	0.9-1.3 mg/dL (for males); 0.6-1.1 mg/dL (for females)
Serum uric acid	Enzymatic photometric test using TOOS	3.5-7.2 mg/dL (for males); 2.6-6.0 mg/dL (for females)
eGFR	MDRD equation	85 mL/min/1.73m^2

3.7. Statistically Analysis

The data analysis was done by using suitable software like MS EXCEL 2021. T-test was used for enumeration data, which were expressed as mean and standard deviation. Pearson's analysis was employed for correlation analysis between the variables. Statistical method $p<0.05$ was considered to indicate a statistically significant.

4. Results

This study was conducted in Adesh Institute of Medical Sciences and Research, Bathinda. During a six-month period, 180 serum samples of chronic kidney disease patients were tested for serum uric acid.

The five stages of CKD were determined by the estimated glomerular filtration rate (eGFR). The eGFR was calculated using the level of creatinine. In the present study, males and females were distributed in the stages based on the eGFR because of their different biological references of serum creatinine in males and females.

The study's findings are as follows:

Table 2: Age distribution amongst chronic kidney disease patients

Age Group	No. of Patients	Percentage
20-30	13	7.2
31-40	25	13.8
41-50	26	14.4
51-60	51	28.3
61-70	65	36.1
Total	**180**	**100**
Range	20-70	
Mean ± SD	52.8 ± 12.8	

The above table shows the age distribution among 180 chronic kidney disease patients. The age of patients ranged from 20-70 years. The mean± SD for age was 52.8±12.8 years. Only 7.2% of patients were in the age group of 20-30 years., 13.8% of the patients were the age between 31-40 years, and 28.3% of the patients were the age between 51-60. The patients between 41 and 50 years of age were 14.4%. Maximum patients, that is, 36.1%, were in the age group between 61-70 years.

As evident from the above table, there were 126 males (67.7%) and 54 females (32.2%) among a total of 180 chronic kidney disease patients. So, in the present study,

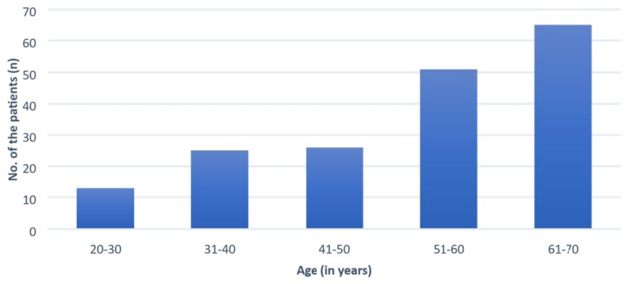

Figure 1: Age distribution in chronic kidney disease patients

Table 3: Gender distribution amongst chronic kidney disease patients

Gender	No. of Patients	Percentage
Male	126	67.7
Female	54	32.2
Total	**180**	**100**

the analysis according to gender showed marked male preponderance.

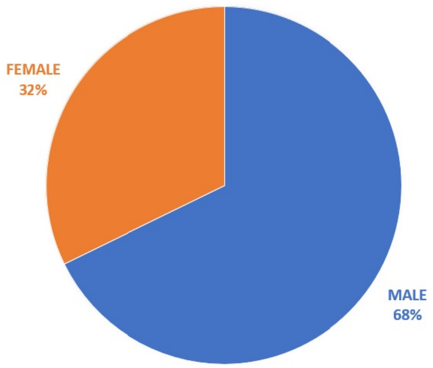

Figure 2: Gender distribution in chronic kidney disease patients

Table 4: OPD/IPD wise distribution

OPD/IPD	No. of Patients	Percentage
OPD	32	17.7
IPD	148	82.2
Total	**180**	**100**

IPD patients had a higher proportion of cases (82.2%) than IPD patients (17.7%).

The above table shows the distribution of patients in the stages of chronic kidney disease. The minimum number of males and females present was in stage I and the maximum number was in stage V.

Table 6able 6 shows the mean and standard deviation of serum uric acid *w*ith p- value between males and females in the different stages of chronic kidney disease, which

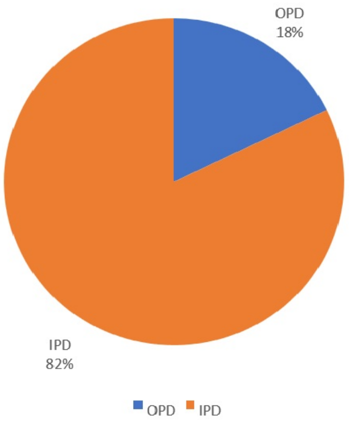

Figure 3: OPD/IPD wise distribution of chronic kidney disease patients

Table 5: Patients distribution in the stages of chronic kidney disease based on eGFR

Stages	eGFR value ml/ min/1.73m^2	No. of Patients	Male	Female
I	>90	10	7	3
II	60-89	21	13	8
III A	45-59	18	13	5
III B	30-44	27	20	7
IV	15-29	46	32	14
V	<15	58	41	17

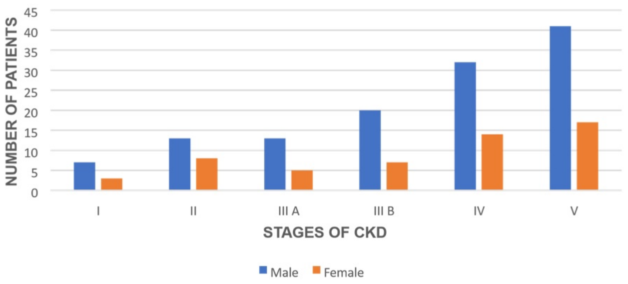

Figure 4: Patients distribution in the stages of chronic kidney disease

Table 6: Distribution of serum uric acid in the various stages of chronic kidney disease and their mean and standard deviation with *p*-value

Stages	Mean ± Standard deviation		*p* -value
	Males	Females	
I	6.76±0.53	6.57±0.32	=0.5866
II	7.22 ±0.36	6.63±0.47	=0.0042*
III A	8.20±0.47	7.88±0.75	=0.2881
III B	9.46±0.69	8.52±0.60	=0.0004*
IV	9.51±0.98	8.81±1.28	=0.0487*
V	9.63±2.24	9.21±2.01	=0.5498

*(p<0.05)=significant

Table 7: Correlation of eGFR with serum uric acid in the stages of CKD

Stages	Parameters	*r* -value of male	*p* -value	*r* -value of female	*p* -value
I	eGFR vs. Uric Acid	-0.84686	=0.016423*	-0.89599	=0.294351
II	eGFR vs. Uric Acid	-0.54436	=0.054623	-0.57084	=0.140178
III A	eGFR vs. Uric Acid	-0.59532	=0.031944*	-0.67548	=0.211233
III B	eGFR vs. Uric Acid	-0.76235	=0.000094*	-0.75836	=0.048332*
IV	eGFR vs. Uric Acid	-0.77531	<0.00001*	-0.73705	=0.002637*
V	eGFR vs. Uric Acid	-0.83286	<0.00001*	-0.82991	=0.00039*

*(p<0.05)=significant

shows a statistically significant difference between stage II, stage III B, and stage IV (p=0.0042, 0.0004, and 0.0487 respectively).

4.1. *Stage I*

Figure 5

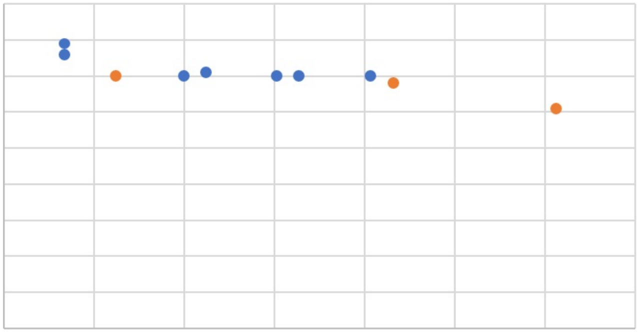

Figure 5: Showing correlation of eGFR and serum uric acid in stage I

4.2. *Stage II*

Figure 6

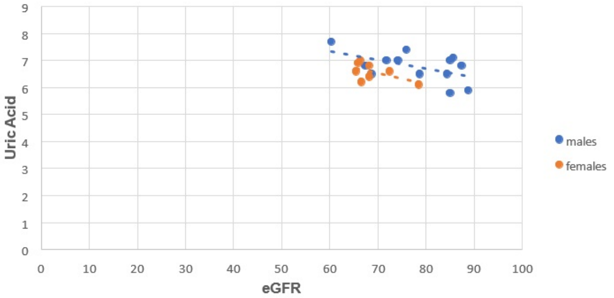

Figure 6: Showing correlation of eGFR and serum uric acid in stage II

4.3. *Stage III A*

Figure 7

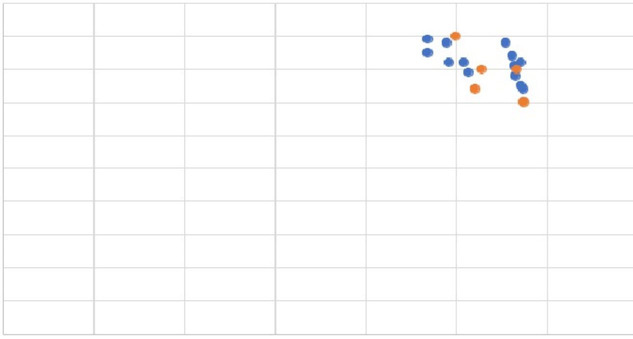

Figure 7: Showing correlation of eGFR and serum uric acid in stage III A

4.4. *Stage III B*

Figure 8

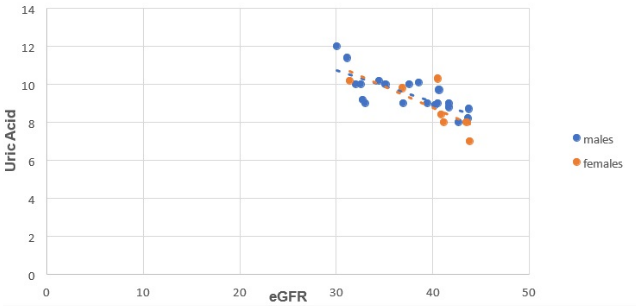

Figure 8: Showing correlation of eGFR and serum uric acid in stage III B

4.5. *Stage IV*

Figure 9

4.6. *Stage V*

Figure 10

5. Discussion

Chronic kidney disease (CKD) is a prevalent health condition that affects millions of individuals worldwide.[18]

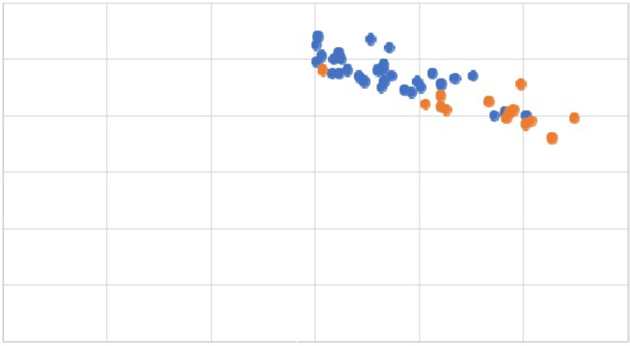

Figure 9: Showing correlation of eGFR and serum uric acid in stage IV

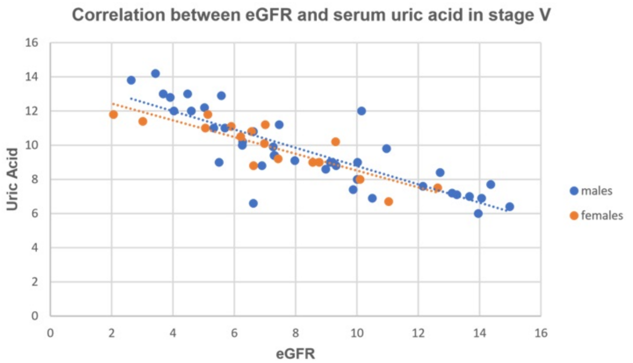

Figure 10: Showing correlation of eGFR and serum uric acid in stage V

It is a progressive and irreversible disease that results in the gradual deterioration of kidney function over time. CKD can have various causes, the most common being diabetes and high blood pressure.[19] The progression of CKD occurs in stages, each characterized by the level of kidney function and the presence of symptoms. The five stages of CKD are determined by the estimated glomerular filtration rate (eGFR) which measures how well the kidneys are filtering waste. Diagnosing CKD involves various tests and assessments. The eGFR mentioned earlier is calculated using a blood test that measures the level of creatinine, a waste product produced by muscle metabolism.[20]

This might be the first observational study conducted to assess the serum uric acid, serum C-reactive protein and lipid levels in the chronic kidney disease patients. To achieve the objectives of this study, 180 participants attending to outpatient departments (OPD) and inpatient department (IPD) of medicine, AIMSR Hospital, Bathinda were enrolled. The participants were further classified into the stages of chronic kidney disease for correlation and to identify status of biochemical parameters.

Chronic Kidney Disease (CKD) can develop at any age and various conditions can lead to CKD (Mary Mallappallilet al., 2014).[21] Previously study, Ji ChengLv et al., 2019have reported, CKD becomes more common with increasing age.[22] For every decade above age 40 years, GFR declines by 10 ml/min such that by age 70 years, the GFR has declined by about 30 ml/min. The aging process compounded by risk factors as well as well as hemodynamic and nonhemodynamic consequences of activation of the renin angiotensin system make the elderly susceptible to CKD. Therefore, in this present study, in order to verify the impact of age for the outcomes of chronic kidney disease participants were categorized into different age groups, which indicate; 7.2% of cases were in the age groups of 20 to 30 years,13.8% of cases were in the age groups of 31 to 40 years,14.4% of cases were in the age groups of 41 to 50, 28.3% of cases were in age groups of 51 to 60 years, while 36.1% of the cases were in the age groups of 61 to 70 years suggested that highest number of cases were in the older age groups (Table 2). This clearly indicated that increase in the age may have direct impact on the chronic kidney disease. The age of patients in the present study ranged from 20-70 years. The mean± SD for age was 52.8±12.8 years. Maximum patients, that is, 36.1% were in the age group between 61-70 years. The other studies like those done by Vandana Menon et al., 2003, Madero et al., 2009 and Zhibin li et al., 2013 were having the age of the patients as 52±12, 52±12 and 58.7± 12 respectively which was almost similar to the present study.[23–25] On the contrary, study by Kentaro Kohagura et al., 2013 was having mean± SD of the age of patients as 42.4± 18.5 which was lesser than the present study.[26] The study by Liu et al., 2021, Sebastjan bevc et al., 2017, Ailing zahang et al., 2021 and Yin et al., 2013 was having mean± SD of age as 63.5 ±13.5, 72.5± 5, 73.63 ±10.26, 73.63 ±10.26 respectively which was higher than the present study group.[17,27–29]

In the present study, there were 122 males (67.7%) and 58 females (32.2%) among total of 180 chronic kidney disease patients. So, in the present study, the analysis according to the gender showed marked male preponderance. As the enrolment of patients in the present study was random so a greater number of males could be due to high prevalence of kidney disease in males as compare to females because of differences in hormones levels. Higher testosterone levels in men may cause a loss in kidney function. On the other hand, men's kidney may not be protected by estrogen, which is higher in women until menopause. This was in accordance with other studies by Mohamed E. Suliman et al., 2006 and Oluseyi A. Adejumo et al., 2016who were also having a greater number of males in their studies in their studies as compared to females.[30,31] Also, H.K. Aggarwal et al., 2018 were having a greater number of males as was there in the present study.[32] Only the study by Sebastjan Bevc et al., 2017 were having a greater number of females as compare to males.[28]

An elevated serum uric acid level independently predicts the development of CKD (Jialing Zhang et al., 2021).[33] In the previous studies, Madero et al., 2009 suggested that elevated uric acid might contribute to the progression of kidney disease and Qimei Luo et al., 2019 investigated the relationship between elevated uric acid levels and the development of CKD and hypertension.[24,34] It found that higher uric acid levels were associated with an increased risk of both conditions. In this study, we investigated the variations in serum uric acid levels among different CKD stages and analysed the potential gender-based differences in these levels. Our findings revealed a notable trend in the serum uric acid levels as CKD progressed through its various stages. The serum uric acid levels showed an upward trajectory, with statistically significant differences observed between certain stages. Specifically, stages II, III B, IV, and V exhibited significantly higher serum uric acid levels compared to the earlier stages (Stage I and Stage III A) (Table 6). This observation suggests a potential association between advanced CKD stages and elevated uric acid levels. In agreement to the current study, Johnson et al 2013., have also discussed the mechanisms by which elevated uric acid levels might contribute to kidney disease development and progression, including inflammation and oxidative stress.[35] Interestingly, our study also evaluated genderbased differences in serum uric acid levels within each CKD stage. While there were variations, statistical significance was not consistently observed. This suggests that gender might not be a primary determinant of serum uric acid levels in CKD, at least within the context of this study. In the context of CKD, higher uric acid levels could potentially exacerbate renal function decline and contribute to the overall burden of the disease. The increase in serum uric acid concentrations in current study might due to high intake of meat and other foods that contain purines can lead to high uric acid levels and also there are other causes.

In this study, we explored the correlations between eGFR and uric acid in Stages of CKD patients. Our findings indicate a negative correlation between eGFR and uric acid levels in males and females. This suggests that in Stages of CKD, lower eGFR values are associated with higher uric acid levels. However, study by Ron T Gansvoort et al., 2013 assessed the association between serum uric acid levels and the risk of developing kidney disease in the general population.[36] Higher uric acid levels in males were found to be associated with an increased risk of incident CKD.

It's important to acknowledge some limitations of our study, including the relatively small sample size and the observational design. The observational design restricts our ability to establish causal relationships between serum uric acid levels and CKD progression.

Additionally, our study did not consider factors such as dietary habits, lifestyle, infections, comorbidities and medication use, which could influence serum uric acid levels. The link between elevated serum uric acid and CKD progression carries clinical implications. Elevated uric acid are common comorbidities in CKD patients. The negative correlations between eGFR and uric acid suggest that even in Stages of CKD, there are indication of altered renal function. Future research could benefit from longitudinal designs that track changes in serum uric acid levels as CKD progresses.

6. Conclusion

Chronic kidney disease is one of the non-communicable diseases that have had an increase in deaths related to them over the previous two decades. The present study highlights the progressive increase in serum uric acid levels as CKD advances through its stages. This finding shows the possible importance of uric acid in relation to the development of CKD. The correlations between eGFR and serum uric acid in the stages of CKD shed light on the complex interactions between renal function. The negative correlations with uric acid suggest that even in early CKD stages, there are indications of altered physiological processes. The correlations underscore the importance of considering multiple biomarkers in assessing CKD progression and cardiovascular risk.

7. Ethics Declaration

This study was approved by AIMSR Research Committee and the Ethics Committee of Biomedical and Health Research, Adesh University, Bathinda.

8. Consent

Written informed consent was obtained from all participants for the use of their blood sample for this research and the publication of any findings in the scientific literature.

9. Source of Funding

None.

10. Conflict of Interest

None.

11. Acknowledgment

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