Relationship Between Serum Uric Acid Levels and Kidney Allograft Function Within the First Year Post-Transplantation

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Abstract

Background: Hyperuricemia negatively impacts cardiovascular health in patients with chronic kidney disease and kidney transplant recipients. Lowering serum uric acid has been associated with improved outcomes following kidney transplantation. This study investigated the relationship between serum uric acid levels and kidney allograft function in transplant recipients.

Methods: This retrospective, single-center cohort study included kidney transplant recipients within 12 months post-transplantation. Hyperuricemia was defined as a serum uric acid level greater than 7 mg/dL. The primary outcomes were the difference in allograft function between the hyperuricemia and normal uric acid group and the correlation between serum uric acid levels and kidney allograft function, measured by estimated glomerular filtration rate (eGFR) at 6 and 12 months post-transplant.

Results: A total of 134 patients were included in the study. By 6 months post-transplant, 50% of patients had developed hyperuricemia, with the majority being male. Serum uric acid levels were significantly and inversely correlated with eGFR at both 6 and 12 months. Although the hyperuricemia group showed a trend toward greater eGFR decline compared to the normal uric acid group, the between-group differences did not reach statistical significance. Nevertheless, eGFR values at both 6 and 12 months were substantially lower in the hyperuricemia group

Conclusions: The association between hyperuricemia and reduced kidney allograft function within the first year after transplantation was suggested. Further research is needed to determine whether elevated uric acid directly contributes to graft dysfunction or reflects declining kidney function.

Keywords: allograft dysfunction; KT; renal transplantation; graft failure; allograft failure

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ความสัมพันธ์ระหว่างระดับกรดยูริกในเลือดกับการทำงานของไตที่ปลูกถ่ายช่วงหนึ่งปีแรกหลังการปลูกถ่ายไต

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บทคัดย่อ

บทนำ: ภาวะกรดยูริกในเลือดสูง (Hyperuricemia) ส่งผลเสียต่อสุขภาพหัวใจและหลอดเลือดในผู้ป่วยโรคไตเรื้อรังและผู้ที่ได้รับการปลูกถ่ายไต การลดระดับกรดยูริกในเลือดมีความเกี่ยวข้องกับผลลัพธ์ที่ดีขึ้นหลังการปลูกถ่ายไต งานวิจัยนี้ศึกษาความสัมพันธ์ระหว่างระดับกรดยูริกในเลือดกับการทำงานของไตที่ปลูกถ่ายในผู้รับการปลูกถ่ายไต

ระเบียบวิธีวิจัย: การศึกษาเชิงย้อนหลังแบบกลุ่มตัวอย่างในศูนย์เดียว โดยรวบรวมข้อมูลจากผู้ป่วยปลูกถ่ายไตที่มีระยะเวลาหลังการปลูกถ่ายไม่เกิน 12 เดือน กำหนดภาวะกรดยูริกในเลือดสูงว่าเป็นระดับกรดยูริกในเลือดมากกว่า 7 มก./ดล. ผลลัพธ์หลักที่ศึกษา คือ ความแตกต่างของการทำงานของไตที่ปลูกถ่ายระหว่างกลุ่มที่มีกรดยูริกในเลือดสูงกับกลุ่มที่มีกรดยูริกในเลือดปกติ และความสัมพันธ์ระหว่างระดับกรดยูริกในเลือดกับการทำงานของไตที่ปลูกถ่าย ซึ่งประเมินจากอัตราการกรองของไตโดยประมาณ (eGFR) ที่ 6 และ 12 เดือนหลังการปลูกถ่าย

ผลการวิจัย: มีผู้ป่วยรวมทั้งสิ้น 134 รายเข้าร่วมการศึกษา โดยภายใน 6 เดือนหลังการปลูกถ่าย พบว่าร้อยละ 50 ของผู้ป่วยมีภาวะกรดยูริกในเลือดสูง และส่วนใหญ่เป็นเพศชาย ระดับกรดยูริกในเลือดมีความสัมพันธ์ทางลบอย่างมีนัยสำคัญกับค่า eGFR ทั้งในช่วง 6 และ 12 เดือน แม้ว่ากลุ่มที่มีภาวะกรดยูริกในเลือดสูงจะแสดงแนวโน้มของการลดลงของ eGFR มากกว่ากลุ่มที่มีกรดยูริกในเลือดปกติ แต่ความแตกต่างระหว่างกลุ่มไม่ถึงระดับนัยสำคัญทางสถิติ อย่างไรก็ตาม ค่า eGFR ทั้งในช่วง 6 และ 12 เดือนในกลุ่มกรดยูริกในเลือดสูงต่ำกว่ากลุ่มกรดยูริกในเลือดปกติอย่างมีนัยสำคัญ

สรุป: การศึกษานี้ชี้ให้เห็นถึงความสัมพันธ์ระหว่างภาวะกรดยูริกในเลือดสูงกับการทำงานของไตที่ปลูกถ่ายที่ลดลงภายในหนึ่งปีหลังการปลูกถ่าย อย่างไรก็ตามยังจำเป็นต้องมีการวิจัยเพิ่มเติมเพื่อระบุว่า ระดับกรดยูริกที่สูงส่งผลต่อการเสื่อมของไตที่ปลูกถ่ายโดยตรง หรือเป็นเพียงผลลัพธ์จากการทำงานของไตที่ลดลง

คำสำคัญ: เปลี่ยนไต; ยูริก; ไตวาย; ไตเรื้อรัง; ปลูกถ่ายอวัยวะ

Introduction

The prevalence of hyperuricemia among kidney allograft recipients has been reported to range from 19% to 55%. Risk factors for hyperuricemia in this population include decreased estimated glomerular filtration rate (eGFR), use of diuretics, calcineurin= inhibitors—particularly cyclosporine—obesity, and metabolic syndrome.[1] Hyperuricemia contributes to the onset and progression of cardiovascular diseases by influencing molecular pathways involved in inflammation, oxidative stress, insulin resistance and diabetes, endoplasmic reticulum stress, and endothelial dysfunction.

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Several studies have demonstrated that elevated serum uric acid levels are associated with overall and death-censored graft failure.[2-4] In addition, some evidence suggests that hyperuricemia increases the risk of developing new-onset diabetes mellitus after transplantation in kidney transplant recipients.[5-7]

Hyperuricemia in kidney transplant recipients is linked to metabolic syndrome and may contribute to reduced graft function, thereby negatively affecting quality of life and potentially decreasing allograft survival due to related complications. The present study explored the correlation between hyperuricemia and kidney allograft function during the first year after transplantation.

Material and Methods

Study design and population

This retrospective, single-center cohort study was conducted at Rajavithi Hospital in Bangkok, Thailand, and included patients with end-stage kidney disease (ESKD) who underwent kidney transplantation between January 1997 and December 2022. Eligible participants were 18 years or older and had received either a living donor kidney transplant (LDKT) or a deceased donor kidney transplant (DDKT) at least one year prior to enrollment. All included patients had stable allograft function at three months post-transplantation.

Patients were excluded if they experienced graft loss or death during the study period, underwent multiple organ transplantation, or had comorbid conditions such as leukemia, psoriasis, or transfusion-dependent thalassemia. The study protocol is illustrated in Figure 1. Ethical approval was granted by the Rajavithi Hospital Ethics Committee (Approval No. 66207). Informed consent was not required for this study.

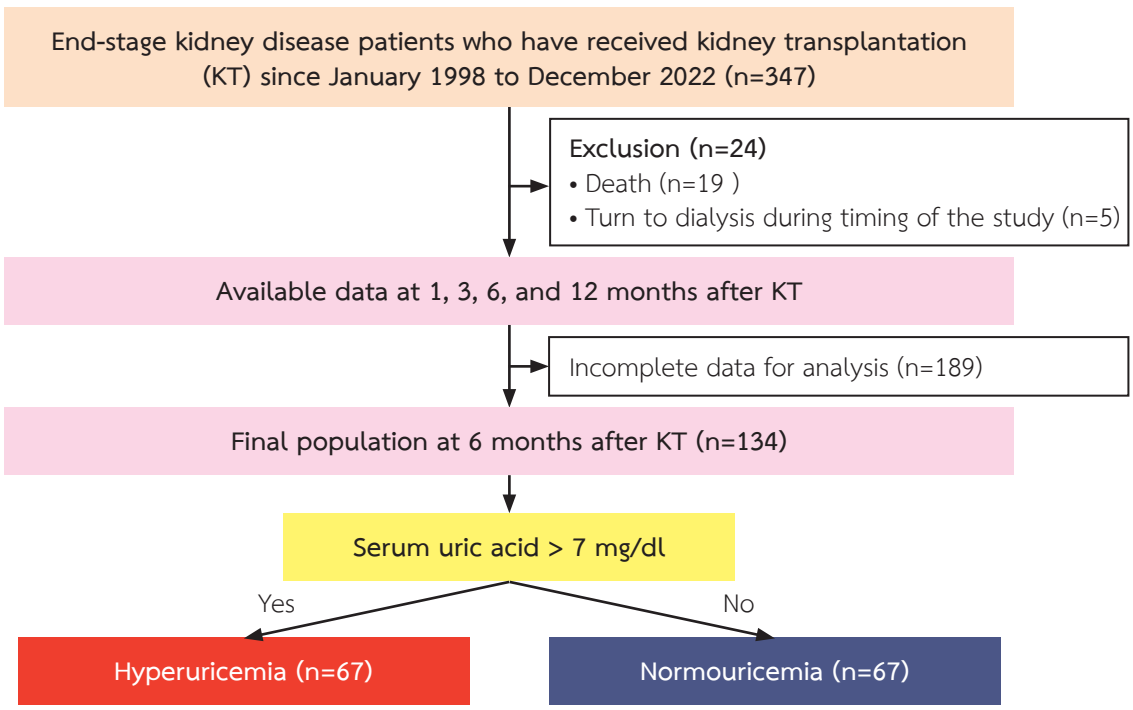

Figure 1 The study flow diagram

Definition and Outcomes

Hyperuricemia was defined as a serum uric acid level greater than 7.0 mg/dL. The primary outcomes were the difference in allograft function between the hyperuricemia and normal uric acid group and the correlation between serum uric acid levels and kidney allograft function, measured by estimated glomerular filtration rate (eGFR) at 6 and 12 months post-transplant.

Data Collection

Demographic and laboratory data were collected from the electronic medical records at 1 month, 3 months, 6 months, and 12 months after transplantation.

Sample Size Calculation

The sample size was calculated using the correlation coefficient estimation formula by Demirci B. Gurlek (1995)[8], with an expected correlation coefficient (r) of 0.59. For a significance level (α) of 0.05, the corresponding Z-score is 1.96, and for a test power of 80%, the Z-score is 0.842. Accounting for approximately eight potential confounding factors in the analysis, the final calculated sample size was 110 patients.

Statistical Analysis

Categorical baseline demographic data are presented as frequencies and percentages. Continuous variables are expressed as mean ± standard deviation for normally distributed data, and as median, minimum, maximum, and interquartile range (IQR) for non-normally distributed data. Differences in categorical variables were analyzed using the Chi-square test, Fisher's exact test, or McNemar test, as appropriate. For continuous variables, comparisons were made using the Student's t-test when the assumptions of normality and homogeneity of variances were met; otherwise, the Mann-Whitney U test was used. Correlation analyses included Pearson's correlation, linear regression, and repeated measures ANOVA where applicable. A p-value of < 0.05 was considered statistically significant. All analyses were conducted using SPSS version 29.0.2.0.

Results

A total of 337 patients underwent kidney transplantation between January 1997 and December 2022. Of these, 24 were excluded due to return to dialysis or death, and 189 were excluded due to insufficient data, leaving 134 patients for the final analysis. Baseline demographic and laboratory data at 6 months post-transplant are presented in **Table 1**.

Fifty percent of the patients had hyperuricemia. The hyperuricemia group had a higher proportion of male patients and a greater average height. There were no significant differences between the hyperuricemia and normouricemia groups in terms of blood pressure, dialysis vintage, underlying cause of ESKD, donor type, early post-transplant allograft function, immunosuppressive regimen, or prior history of gout. However, at 6 months post-transplant, the hyperuricemia group had significantly lower eGFR and higher serum creatinine levels. No significant differences were observed between the groups in immunosuppressive drug levels, fasting plasma glucose, or LDL cholesterol.

Table 1 Baseline demographics of all patients and laboratory data at 6 months after kidney transplantation

Parameters	All patients (N =134)	Hyperuricemia (N=67)	Normal uric acid (N=67)	P
Age, years	42.7±10.6	41.0±10.7	44.3±10.4	0.072
Male (N/%)	75 (56)	46 (68.7)	29 (43.3)	0.005
Body weight, kg	65.5±20.6	67.0±18.8	63.9±22.2	0.122
Height, cm	162.6±11	165.4±10	160.0±12	<0.001
Body mass index, kg/m^2	24.7±6.22	24.5±5.70	24.8±6.82	0.719
Systolic BP, mmHg	131±15	131±16	130±14	0.621
Diastolic BP, mmHg	76±13	75±14	77±11	0.334
Mode of dialysis (Hemodialysis, N/%)	121 (90.3)	62 (92.5)	59 (88.1)	0.562
Dialysis vintage, months	60 (5-300)	60 (11-300)	60 (5-204)	0.670

Table 1 Baseline demographics of all patients and laboratory data at 6 months after kidney transplantation (continue)

Parameters	All patients (N =134)	Hyperuricemia (N=67)	Normal uric acid (N=67)	P
Etiology of end-stage kidney disease (N/%)				0.765
Unknown	81 (60.45)	40 (59.70)	41 (61.19)	
IgA nephropathy	16 (11.94)	8 (11.94)	8 (11.94)	
Diabetes	11 (8.21)	5 (7.46)	6 (8.96)	
Polycystic kidney disease	10 (7.46)	7 (10.45)	3 (4.48)	
Others	16 (11.94)	7 (10.45)	9 (13.43)	
Deceased donor (N/%)	116 (86.6)	57 (85.1)	59 (88.1)	0.619
Graft function (N/%) Immediate graft function Slow graft function Delayed graft function	 86 (64.2) 27 (20.1) 21 (15.7)	 38 (56.7) 15 (22.4) 14 (20.9)	 48 (71.6) 12 (17.9) 7 (10.4)	0.173
Immunosuppressive drugs (N/%)				0.590
Tacrolimus + prednisolone + mycophenolic acid	116 (86.6)	60 (89.6)	56 (83.6)	
Cyclosporin prednisolone + mycophenolic acid	13 (9.7)	6 (9)	7 (10.4)	
Tacrolimus + everolimus + prednisolone	4 (3)	1 (1.5)	3 (4.5)	
Uric acid-lowering agents (N/%)				0.205
Yes	9 (6.7)	6 (9)	3 (4.5)	
Allopurinol	3 (2.2)	3 (4.5)	0 (0)	
Febuxostat	6 (4.5)	3 (4.5)	3 (4.5)	
Gout (N/%)	7 (5.2)	3 (2.2)	4 (3.0)	1
Laboratory data (6 months after kidney transplantation)				
Serum uric acid (mg/dl)	7.2±1.8	8.6±1.4	5.9±0.9	<0.001
Serum creatinine (mg/dl)	1.48±0.71	1.74±0.81	1.34±0.50	0.001
eGFR (ml/min/1.73m^2)	51.25±19.50	45.97±18.22	56.53±19.44	0.002
Drug levels				
Tacrolimus (ng/ml)	6.8 (1.4-39.1) (n=121)	6.8 (1.4-39.1) (n=61)	6.7 (1.9-15.7) (n=60)	0.779
Cyclosporin (ng/ml)	163.0±42.7 (n=13)	157.5±15.7 (n=6)	167.7±58.2 (n=7)	0.686
Fasting glucose (mg/dL)	100.4±26.9	97.1±22.3	103.7±30.6	0.155
LDL cholesterol (mg/dL)	118.6±35.5	116.3±36.8	120.8±34.4	0.469

eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein

Serum uric acid and eGFR of all patients at 1, 3, 6, and 12 months after kidney transplantation are shown in **Table 2** and **Figure 2**. During the 12-month follow-up, serum uric acid increased significantly. There were no significant changes in serum creatinine and eGFR.

Table 2 Serum uric acid and kidney allograft function during the first year after kidney transplantation

N = 134	1 month	3 months	6 months	12 months	P-value
Uric acid (mg/dl)	5.9 ±1.6	6.8 ±1.6	7.2 ±1.8	7.1 ±1.8	< 0.001
eGFR (ml/min/1.73m^2)	56.34 ±24.04	50.99 ±19.97	51.25 ±19.5	54.21 ±19.8	0.604
Creatinine (mg/dl)	1.43 ±0.92	1.69 ±0.69	1.48 ±0.71	1.58 ±0.60	0.113

eGFR, estimated glomerular filtration rate

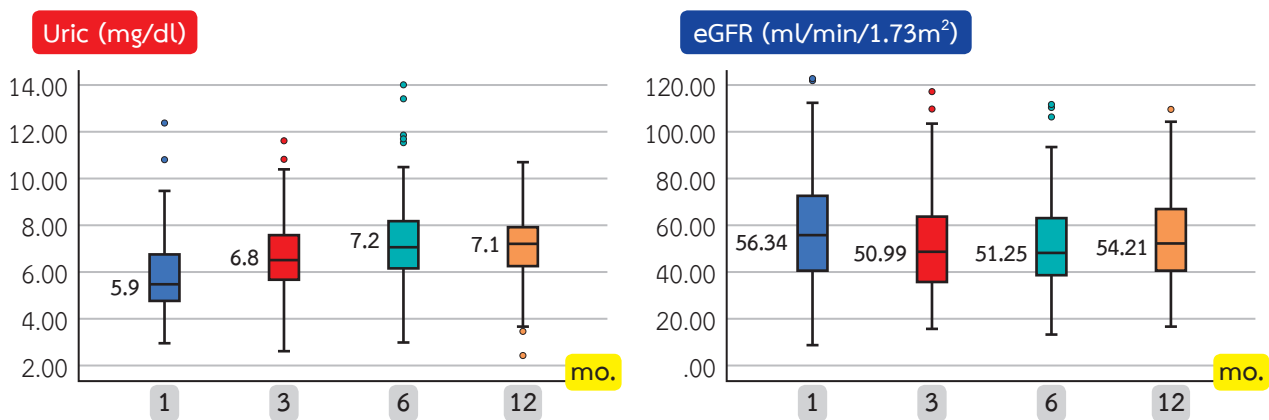

Figure 2 Serum uric acid and kidney allograft function during the first year after kidney transplantation
eGFR, estimated glomerular filtration rate

The correlations between serum uric acid with allograft function and other laboratory parameters are shown in **Table 3** and **Figure 3**. A negative correlation between serum uric acid and eGFR and a positive correlation with serum creatinine were observed at 6 months and 12 months after kidney transplantation. There were no correlations between serum uric acid with fasting glucose, LDL, tacrolimus, and cyclosporin levels at 6 months.

Table 3 Correlations between serum uric acid and other laboratory data

Parameters	6 months		12 months	
	r	p-value	r	p-value
eGFR	-0.448	<0.001	-0.437	<0.001
Serum creatinine	0.564	<0.001	0.444	<0.001
Fasting glucose	0.026	0.776	-	-
LDL	-0.112	0.197	-	-
Tacrolimus (N=121)	-0.002	0.984	-	-
Cyclosporin (N=13)	-0.158	0.607	-	-

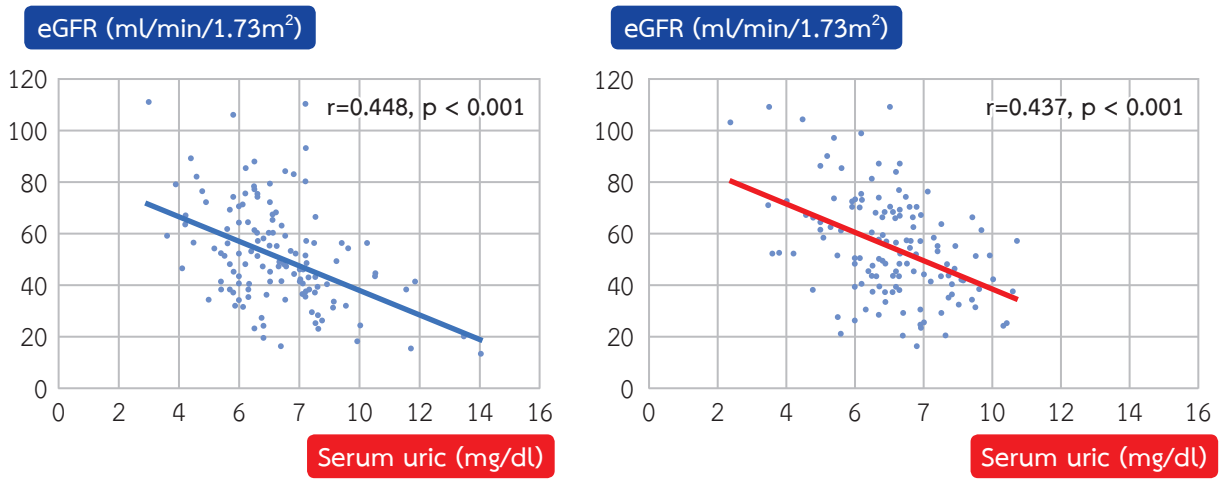

Figure 3 Correlations between serum uric acid and allograft function at 6 (left) and 12 months (right)

Differences in eGFR between the hyperuricemia and normal uric acid groups are presented in **Table 4** and **Figure 4**. Although the hyperuricemia group exhibited a trend toward a continued decline in eGFR, the between-group differences did not reach statistical significance. However, eGFR values at 6 and 12 months were notably lower in the hyperuricemia group compared to the normal uric acid group. Additionally, the decline in eGFR corresponded with an increase in serum uric acid in the hyperuricemia group (**Figure 5**).

Table 4 Estimated glomerular filtration rate in the hyperuricemia and normal uric acid groups

Groups	1 month	3 months	6 months	12 months	Between-Group Changes Mean Difference (95% CI)
Hyperuricemia	53.48±21	48±19.68	45.97±18.22	49.30±17.34	-4.18 (-8.52, 0.15)
Normal uric acid	59.21±24.74	53.99±19.95	56.52±19.44	59.13±20.98	-0.07 (-4.56, 4.41)
p-value	0.168	0.083	0.002	0.004	0.191

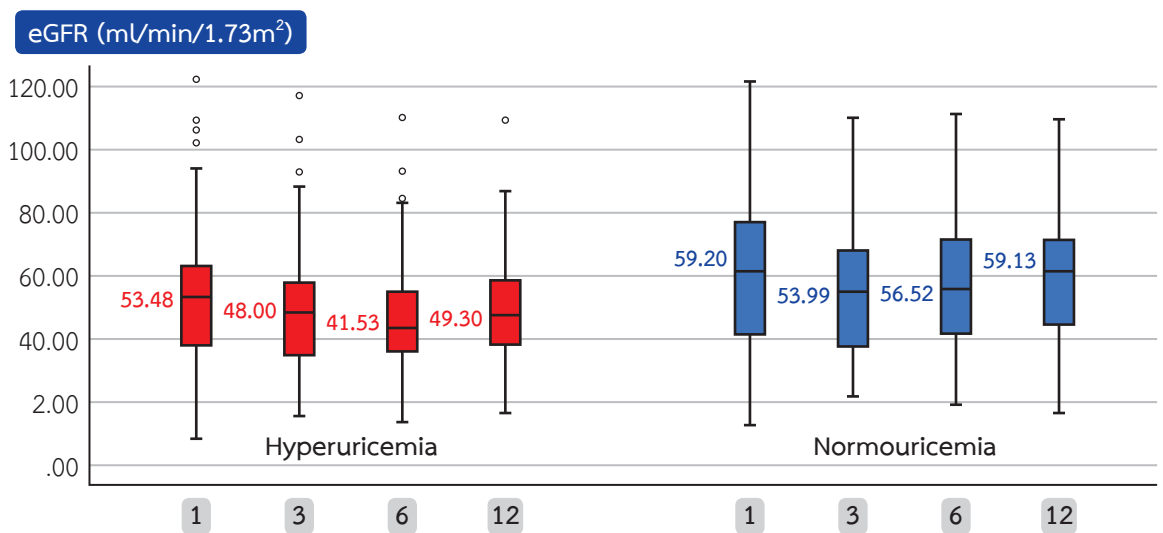

Figure 4 The changes in estimated glomerular filtration rate in the hyperuricemia and normal uric acid groups

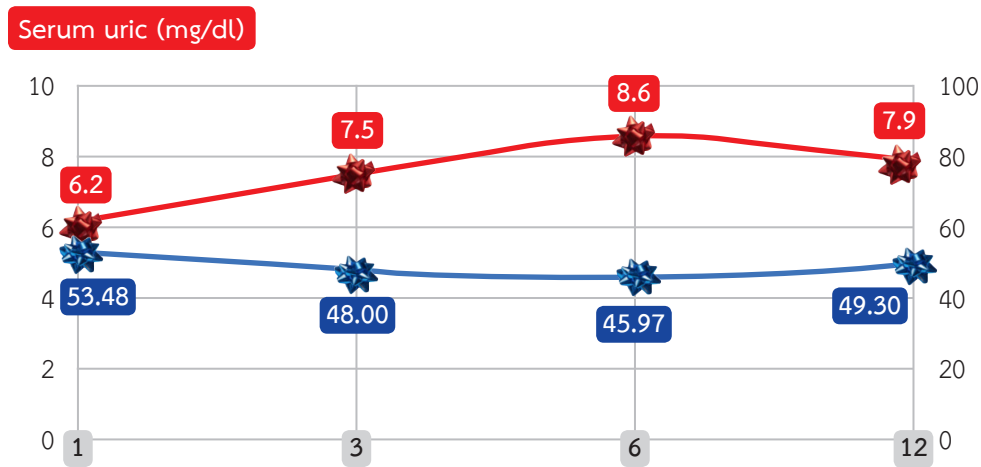

Figure 5 Serum uric acid and estimated glomerular filtration rate in the hyperuricemia group

Discussion

The present study observed a 50% prevalence of hyperuricemia at 6 months after kidney transplantation. The hyperuricemia group showed a higher proportion of male patients with increased height. There was a negative correlation between serum uric acid and kidney allograft function at 6 and 12 months after kidney transplantation. There were no correlations between serum uric acid with fasting glucose, LDL, tacrolimus, and cyclosporin levels at 6 months. Although the hyperuricemia group exhibited a trend toward a continued decline in eGFR, the between-group differences did not reach statistical significance. However, eGFR values at 6 and 12 months were notably lower in the hyperuricemia group compared to the normal uric acid group.

The 50% incidence of hyperuricemia observed in this study aligns with previously reported rates, though prevalence varies across countries. For example, Erkmen Uyar M et al. reported a prevalence of 37%[9] while Clive, David M et al. noted rates as high as 55%.[10] In our cohort, hyperuricemia was more common in males and associated with lower eGFR values. The lower incidence in females may be hormonally mediated, as estrogen is known to downregulate uric acid transporters, reducing uric acid reabsorption.[11]

Hyperuricemia was more frequent among patients with reduced eGFR, likely due to decreased renal clearance of uric acid. Serum uric acid levels demonstrated a moderate inverse correlation with allograft function at 6 and 12 months post-transplant. Additionally, serum uric acid levels increased over time at 1, 3, and 6 months post-transplant, while eGFR declined during the same period. Similar trends have been reported in other studies.[8,12] Several mechanisms may explain the detrimental effects of uric acid on kidney allograft outcomes. These include glomerular hypertrophy and tubulointerstitial fibrosis due to hyperuricemia, as well as altered renal plasma flow, arterial stiffness, and endothelial dysfunction resulting from impaired nitric oxide production in vascular endothelial cells.[1,13,14]

In this study, serum uric acid levels were not significantly associated with fasting glucose or LDL cholesterol, possibly due to the short follow-up period. A longer-term study by Sotomayor CG et al. found that hyperuricemia was associated with an increased risk of post-transplant diabetes mellitus after 5.3 years of follow-up.[5]

No correlation was observed between serum uric acid levels and tacrolimus or cyclosporin levels in this study. While no prior research has explored the relationship between uric acid and tacrolimus, a study by Einollahi B et al. found an association between hyperuricemia and higher cyclosporin trough levels.[15] Similarly, Marcén R et al. reported a higher prevalence of hyperuricemia in patients receiving cyclosporin, with a correlation to cyclosporin levels.[16] This is likely due to cyclosporin's effect in reducing uric acid clearance.[10,17] The absence of such a relationship in the current study

may be attributable to the small number of patients using cyclosporin.

The limitations of our study include its single-center design, short follow-up period, and the small number of patients receiving uric acid-lowering therapy. As a result, the effectiveness of uric acid-lowering therapy in slowing the decline of eGFR remains uncertain.

In conclusion, the association between hyperuricemia and reduced kidney allograft function within the first year after transplantation was suggested. However, whether this association reflects a direct detrimental effect of elevated uric acid on the allograft or is simply a consequence of declining allograft function remains unclear and warrants further investigation.

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