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ORIGINAL ARTICLE

Low preoperative serum uric acid is associated with early acute kidney injury after living donor liver transplantation

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Abstract

Background: Liver transplantation is treatment option for patients with end-stage liver disease and hepatocellular carcinoma. Renal function deterioration significantly impacts the survival rates of liver recipients, and serum uric acid (SUA) is associated with both acute and chronic renal function disorders. Thus, our study aimed to assess the relationship and predictive value of preoperative SUA level and postoperative acute kidney injury (AKI) in living donor liver transplantation (LDLT).

Methods: We conducted a prospective observational study on 87 patients undergoing LDLT. Blood samples were collected immediately before LDLT, and renal function status was followed up for 3 consecutive days postoperatively.

Results: Low SUA levels (cutoff value 4.15 mg/dL) were associated with a high risk of early posttransplantation AKI. The area under the curve was 0.73 (sensitivity, 79.2%; specificity, 59.4%). Although not statistically significant, there were no deaths in the non-AKI group but two in the early AKI group secondary to liver graft dysfunction in addition to early AKI within the first month after LDLT.

Conclusion: AKI after liver transplantation may lead to a deterioration of patient status and increased mortality rates. We determined low preoperative SUA levels as a possible risk factor for early postoperative AKI.

Keywords: Acute kidney injury; Living donor liver transplantation; Serum uric acid

1. INTRODUCTION

Since the establishment of kidney transplantation in the 1950s, liver transplantation has gained wide attention among clinicians, and in 1967, the first liver transplant recipient to survive longer than 1 year was reported.[1] Living donor liver transplantation (LDLT) is an accepted therapeutic option for patients with end-stage liver disease and hepatocellular carcinoma, with a 1-, 3-, and 5-year survival rate of 86%, 72%, and 68%, respectively. For those who decide on LDLT, the waiting time is considerably shorter compared with those on the waiting list for liver grafts from deceased donors.[2] Nevertheless, LDLT still poses challenges to transplant surgeons postoperatively in the intensive care unit (ICU) and during long-term follow-up. Early posttransplantation complications include acute rejection, vascular thrombosis, anastomotic leakage, gastrointestinal hemorrhage, infection, prolonged encephalopathy, and acute kidney injury (AKI),[3] among which AKI is of significant concern.

A recent meta-analysis reported the pooled incidence of AKI after liver transplantation as 37.5%.[4] AKI after liver transplantation may lead to the deterioration of patient status secondary to a prolonged ICU stay, increased risk of graft failure, progression to chronic kidney disease (CKD), and most importantly, increased mortality rates.[5,6] Furthermore, patients who require renal replacement therapy due to AKI often exhibit a higher mortality rate.[7]

AKI is a multifactorial clinical condition that has undergone extensive study in various clinical settings. Of particular interest to liver transplantation, recipient-related and perioperative risk factors have been identified. Individual risk factors, such as overweight, anemia, hypoalbuminemia, high serum creatinine (SCr), hyponatremia, and high model for end-stage liver disease (MELD) and sequential organ failure assessment scores have been recognized. Preexisting conditions, such as nonalcoholic liver disease, liver cirrhosis, diabetes mellitus (DM), diuretic use (furosemide/spironolactone), and the presence of hepatic encephalopathy in addition to perioperative risk factors, including ABO incompatibility, intraoperative blood loss or hypotension requiring red blood cell (RBC) transfusion, and prolonged cold ischemia time (CIT) or

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warm ischemia time (WIT), are all considered significant risk factors.[4,6,8–10] Early allograft dysfunction (EAD) within 1 week after transplantation has also been identified as associated with postoperative AKI and a higher mortality rate.[11,12]

The association of serum uric acid (SUA) levels with AKI is controversial.[13] High preoperative SUA levels have been associated with the risk of postoperative AKI in patients undergoing cardiac and renal surgeries and in patients with severe burns.[14–19] In deceased donor liver transplantation, baseline SUA levels were significantly higher in recipients with newly developed CKD.[20] In contrast, several cohorts have shown that low SUA levels might lead to AKI development.[16,21] Individuals with initially normal kidney function but low SUA levels were reported to experience a time-dependent decline in estimated glomerular filtration rate (eGFR).[22] Matsukuma et al[23] demonstrated that low SUA was associated with a deterioration of renal function to end-stage renal disease in patients with IgA nephropathy. Because the significance of preoperative SUA levels on postoperative AKI remains unclear in LDLT, the aim of our study was to assess the relationship and predictive value of preoperative SUA levels with postoperative AKI in LDLT.

2. METHODS

2.1. Objectives

This prospective, observational, single-institution hospital-based study was performed in a tertiary medical center and analyzed 87 consecutively recruited patients who underwent LDLT from October 2018 to July 2022 at Chang Gung Memorial Hospital (Taoyuan, Taiwan). All subjects gave informed consent before study participation. Exclusion criteria were preoperative sepsis, shock status, pulmonary hypertension (defined as pulmonary artery wedge pressure >35 mmHg), and patient refusal. Patients with gout and taking uric acid-lowering medication, such as allopurinol, patients with moderate to severe CKD (defined as eGFR <45 mL/min/1.73 m^2 for more than 3 months, according to the KDIGO 2012 guideline), and patients who received renal replacement therapy before transplantation were also excluded (Fig. 1).[24,25]

This study was approved by the Institutional Review Board of Chang Gung Memorial Hospital (approval no.: IRB2106130012; CMRPG3L1831, CMRPG3L1311-3, CMRPG3L0611-3).

2.2. Data collection and variable definition

An indwelling arterial catheter was inserted for perioperative monitoring and blood sample collection before inducing general anesthesia. Laboratory test results, including SUA and SCr, were recorded before surgical incision and for 3 consecutive days following transplantation. The SCr was used to calculate the eGFR as follows: eGFR = 186 × $Cr^{-1.154}$ × $age^{-0.203}$ × (0.742 if women) mL/min/1.73 m^2. The patients' MELD scores were calculated on the basis of the data on the day of transplantation. The preinduction SCr level was used as the baseline value to analyze postoperative AKI. AKI was defined as an increase in SCr ≥0.3 mg/dL (26.5 μmol/L) within 48 hours or an increase in SCr ≥1.5 times the baseline value within 7 days postoperatively, as per KDIGO.[24,25] In our study, early AKI was defined as the development of AKI within 72 hours following LDLT,[6] and recipients who developed AKI within 72 hours postoperatively were allocated to the AKI group. All recipients were followed up for the total length of ICU stay, EAD development, and 1-year mortality. Hyponatremia, hypoalbuminemia, and anemia were defined as serum sodium <125 mmol/dL, serum albumin <3.5 g/dL, and hematocrit <36% for women and <41% for men, respectively.

2.3. Statistical analysis

The AKI incidence was initially plotted against the SUA quartiles. The receiver operating curve (ROC) was plotted to determine the cutoff value of SUA for AKI. According to the value, the patients were categorized into either the high or low SUA group, which was then utilized for further analysis as a categorical variable. Reported recipient-related risk factors for postoperative AKI after liver transplantation were analyzed between the AKI and the non-AKI groups using independent *t*

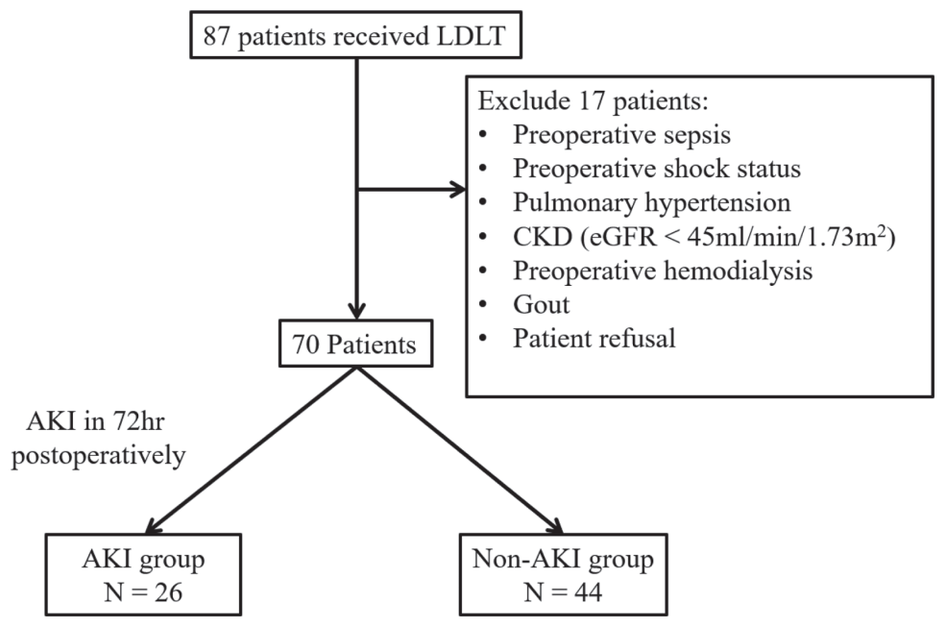

Fig. 1 Flow diagram of patient selection. AKI = acute kidney injury; CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; hr = hours; LDLT = living donor liver transplantation.

test for numerical variables and Chi-squared test for categorical variables. Multivariate logistic regression without elimination was then performed to examine low SUA as a potential risk factor. Adjustments were made for known risk factors for AKI, namely age, gender, body mass index (BMI), diuretic use, ABO incompatibility, DM, hypertension, viral hepatitis, alcoholism, MELD score, creatinine, blood urea nitrogen (BUN), anemia, hypoalbuminemia, hyponatremia, blood loss, RBC transfusion, CIT, and WIT. A p value <0.05 was considered statistically significant. All statistical analyses were performed using SPSS v24 (IBM Corp. Released 2019, IBM SPSS Statistics for Windows, Version 26.0; IBM Corp, Armonk, NY).

3. RESULTS

From October 2018 to July 2022, 87 patients underwent LDLT (Fig. 1). We excluded 17 patients secondary to their history of gout, moderate CKD, or requirement for renal replacement therapy before transplantation. The mean patient age was 54.9 ± 10.3 years, with 53 (75.7%) males and 17 (24.3%) females. There were 48 (68.6%) patients with hepatitis B virus- and hepatitis C virus-related cirrhosis (viral cirrhosis), and 22 (31.4%) had alcoholic liver cirrhosis. Early AKI (within 72 hours postoperatively) was observed in 26 (37.1%) patients and no AKI in the remainder (44, 62.9%). The average MELD score was 15.9 ± 8. There were 57 (81.4%) anemic, 50 (71.4%) hypoalbuminemic, and 16 (22.9%) hyponatremic patients. The average SCr, BUN, and SUA levels were 0.74 ± 0.32, 15.37 ± 7.22, and 4.7 ± 2.1 mg/dL, respectively (Table 1).

There were no statistically significant differences observed between patient gender, BMI, preoperative left ventricular ejection fraction, and comorbidity distribution between the AKI and the non-AKI groups, except that DM was more prevalent in the AKI group than the non-AKI group (34.6% vs 13.6%, p = 0.039; Table 2). Additionally, preoperative laboratory test results were similar between the two groups, with the exception of hypoalbuminemia, which was more prevalent in the AKI than the non-AKI group (88.5% vs 61.4%, p = 0.015). The SUA level was also significantly lower in the AKI group than in the non-AKI group (3.7 ± 1.5 vs 5.3 ± 2.2 mg/dL, p = 0.001). No statistical difference was observed in both CIT and WIT during transplantation. Although intraoperative blood loss did not significantly differ between the two groups, the AKI group received more RBC transfusions than the non-AKI group (10.4 ± 9.6 vs 5.7 ± 5.9 units, p = 0.014). No statistical significance was observed in intraoperative urine output between the two groups (1.17 ± 1.15 vs 1.35 ± 0.76 mL/kg/h, p = 0.416). Supplementary Table 1, http://links.lww.com/JCMA/A254, shows the nutritional status (BMI and albumin). A statistically significant difference among the SUA quartile groups was observed for albumin (p = 0.038) but not BMI.

3.1. Primary outcome: Association of SUA with early AKI

To determine the association of SUA with the risk of early AKI, the patients were divided into four groups by SUA quartiles (Fig. 2). A low SUA level appeared to be associated with a high risk of early posttransplantation AKI. By convention, hypouricemia is accepted as uric acid >7.0 mg/dL and >6.5 mg/dL for men and women, respectively. However, in our study, such a traditional definition of hyperuricemia failed to demonstrate the actual relationship between SUA level and early AKI. Thus, we used ROC analysis, and our results revealed that the area under the curve was 0.73 (Fig. 3), with an SUA value of 4.15 mg/dL identified as the optimal cutoff point (sensitivity, 79.2%; specificity, 59.4%). The patients were then classified into the high SUA or low SUA group. The low SUA group comprised 35.7% of transplant recipients (Table 1), and the prevalence of low SUA was higher in the AKI compared with the non-AKI group (61.5% vs 20.5%, p = 0.001; Table 2). Further analysis by univariate logistic regression demonstrated that preoperative DM, low SUA, hypoalbuminemia, and more intraoperative blood loss were significantly related to AKI, with an odds ratio (OR) of 3.35, 6.22, 4.83, and 1.09,

Table 1

Baseline characteristics of the enrolled patients

Characteristics	Mean ± SD N (%)	Preoperative lab data	Mean ± SD N (%)
Gender		Creatinine, mg/dL	0.74 ± 0.32
Male	53 (75.7%)	eGFR, mL/min/1.73 m^2	114.5 ± 45.9
Female	17 (24.3%)	BUN, mg/dL	15.37 ± 7.22
Age, y	54.9 ± 10.3	Hematocrit, %	43.3 ± 9.8
Height, cm	165.2 ± 7.4	Anemia	57 (81.4%)
Body weight, kg	68.5 ± 12.7	Albumin, g/dL	3.2 ± 0.7
BMI	25 ± 4	Hypoalbuminemia	50 (71.4%)
ABO incompatibility	7 (10%)	Serum sodium, mmol/L	135.5 ± 15.56
Diuretic use	36 (51.4%)	Hyponatremia	16 (22.9%)
Comorbidity		Uric acid, mg/dL	4.7 ± 2.1
DM	15 (21.4%)	Low SUA (<4.15 mg/dL)	25 (35.7%)
Hypertension	22 (31.4%)	Perioperative factors	
Viral hepatitis	48 (68.6%)	Cold ischemia time, min	38.6 ± 24.5
HBV	38 (54.3%)	Warm ischemia time, min	32.3 ± 7.2
HCV	11 (15.7%)	Intraoperative blood loss, mL	1943 ± 2139
HCC	32 (45.7%)	RBC transfusion, unit	7.5 ± 7.7
Alcoholism	22 (31.4%)		
MELD score	15.9 ± 8		

BMI = body mass index; BUN = blood urea nitrogen; cm = centimeter; dL = deciliter; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; g = gram; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; kg = kilogram; L = liter; LVEF = left ventricular ejection fraction; m = meter; MELD = model for end-stage liver disease; mg = milligram; min = minute; mL = milliliter; RBC = red blood cell; SUA = serum uric acid.

Table 2

A comparison of basic characteristics and biochemical data for AKI and non-AKI patients

	AKI	non-AKI	
	Mean ± SD	Mean ± SD	
	N (%)	N (%)	*p*
Gender			
Male	22 (84.6%)	31 (70.1%)	0.182
Female	4 (15.4%)	13 (29.5%)	
Age, y	58 ± 9	53 ± 10.7	0.050
Height, cm	164.9 ± 7.3	165.4 ± 7.6	0.762
Body weight, kg	67.8 ± 11.7	68.9 ± 13.4	0.718
BMI	24.9 ± 3.7	25.1 ± 4.2	0.821
ABO incompatibility	2 (7.7%)	5 (11.4%)	0.621
Diuretic use	15 (57.7%)	21 (47.7%)	0.420
Comorbidity			
DM	9 (34.6%)	6 (13.6%)	0.039[a]
Hypertension	9 (34.6%)	13 (29.5%)	0.659
Viral hepatitis	21 (80.8%)	27 (61.4%)	0.910
HBV	18 (64.3%)	20 (45.5%)	0.054
HCV	4 (15.4%)	7 (16.9%)	0.954
HCC	5 (19.2%)	17 (38.6%)	0.349
Alcoholism	10 (38.5%)	22 (50%)	0.091
MELD score	18.3 ± 9.2	14.5 ± 7	0.055
Preoperative lab data			
Creatinine, mg/dL	0.74 ± 0.3	0.75 ± 0.33	0.806
eGFR, mL/min/1.73 m^2	117.2 ± 61.1	113 ± 34.8	0.713
BUN, mg/dL	14.67 ± 6.02	15.79 ± 7.88	0.535
Anemia	24 (92.3%)	33 (75.0)	0.072
Hypoalbuminemia	23 (88.5%)	27 (61.4%)	0.015[a]
Hyponatremia	8 (30.8%)	8 (18.2%)	0.226
SUA, mg/dL	3.7 ± 1.5	5.3 ± 2.2	0.001[a]
Low SUA (<4.15 mg/dL)	16 (61.5%)	9 (20.5%)	0.001[a]
Perioperative factors			
Cold ischemia time, min	38 ± 21.6	39 ± 26.2	0.874
Warm ischemia time, min	33 ± 7.3	32 ± 7.2	0.575
Blood loss, mL	2419 ± 2453	1662 ± 1903	0.154
RBC transfusion, unit	10.4 ± 9.6	5.7 ± 5.9	0.014[a]
Intraoperative UO, mL/kg/h	1.17 ± 1.15	1.35 ± 0.76	0.416

AKI = acute kidney injury; BMI = body mass index; BUN = blood urea nitrogen; cm = centimeter; dL = deciliter; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; g = gram; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; kg = kilogram; L = liter; LVEF = left ventricular ejection fraction; m = meter; MELD = model for end-stage liver disease; mg = milligram; min = minute; mL = milliliter; RBC = red blood cell; SUA = serum uric acid; UO = urine output.

[a]*p* value < 0.05.

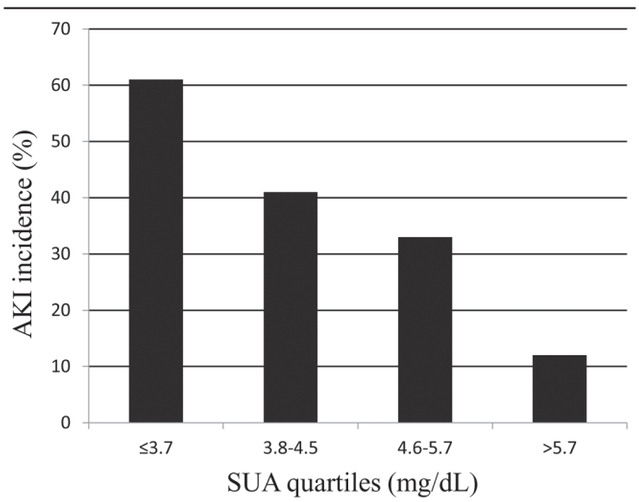

Fig. 2 Incidence of early AKI vs SUA level quartile. AKI = acute kidney injury; SUA = serum uric acid.

respectively (Table 3). After taking into consideration other risk factors described in the literature, multivariate logistic regression analysis was performed, which again showed that a low SUA level (<4.15 mg/dL) was a significant risk factor for AKI (OR, 9.37, confidence interval, 2.05-43.06, p = 0.004) (Table 3).

3.2. Correlation between AKI and preoperative SUA

AKI was classified into three stages according to the extent of increase in SCr, and the patients were stratified according to the AKI stages between the high and low SUA groups (Table 4). In patients without AKI and with less severe AKI (stage 1), a higher proportion had high preoperative SUA levels (>4.15 mg/dL), whereas more patients with a higher AKI stage (stages 2 and 3) were in the low SUA group.

3.3. Secondary outcomes: 1-, 3-, 6-, and 12-month survival, EAD, and ICU stay

There were no deaths within the first month after LDLT in the non-AKI group but two patients died in the AKI group due to

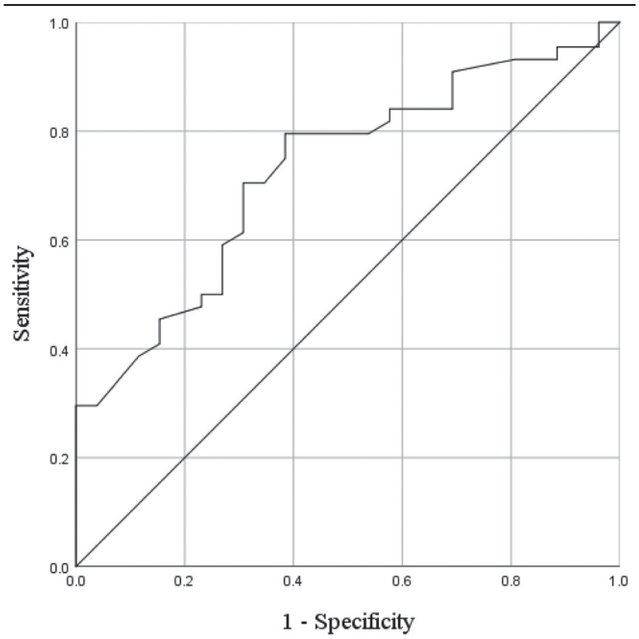

Fig. 3 ROC curve for the cutoff value of SUA. ROC = receiver operating characteristic; SUA = serum uric acid.

liver graft dysfunction in addition to early AKI. That said, there was no significant difference between the survival rates at 1, 3, 6, and 12 months for the AKI and the non-AKI groups (Fig. 4 and Table 5). Similarly, no significant differences in EAD and the length of ICU stay were observed between the two groups.

4. DISCUSSION

Uric acid is the final product of endogenous purine catabolism in humans. Ribose-5-phosphatase is converted into inosine monophosphate, which is further converted to adenosine monophosphate, guanosine monophosphate, and inosine. Purine nucleoside phosphorylase phosphorylates inosine, yielding hypoxanthine, which is further catalyzed by xanthine oxidase to generate xanthine and, finally, uric acid. These biochemical reactions mainly occur in the liver.[26] Approximately two-thirds of uric acid is excreted by the kidneys, and the remainder by the intestines.[27] Conditions affecting uric acid production or excretion may lead to SUA derangement. Uric acid is an intriguing entity that has been extensively studied for its antioxidative and prooxidative properties. Uric acid may potentiate intracellular and mitochondrial oxidative stress in obesity and hepatic steatosis.[28] Several mechanisms involving uric acid have been proposed as culprits for deteriorating renal function. Uric acid crystal precipitation in renal tubules leads to obstructive nephropathy, as observed in tumor lysis syndrome during leukemia treatment.[29,30] Uric acid may also impair renal vessel autoregulation and cause renal vasoconstriction secondary to interruption of the NO-mediated pathway.[31,32] Furthermore, the proinflammatory properties of uric acid stimulate chemoattractant protein-1 production and prompt chemotaxis of inflammatory cells into the renal parenchyma.[33]

Uric acid may act as a powerful antioxidant in hydrophilic environments and a scavenger for free radicals, such as peroxynitrite.[34,35] Uric acid also protects dopaminergic neurons from oxidative stress via an astrocyte-mediated pathway in the nervous system and plays a protective role in patients with multiple sclerosis, Parkinson disease, and experimental allergic encephalomyelitis.[36–40] Low SUA levels are also associated with AKI.[41] Patients with renal hypouricemia are prone to AKI development after vigorous exercise. Although the pathogenesis is unclear, it is possibly attributed to the antioxidative activity of uric acid. Oxygen free radicals decrease renal blood flow, contributing to renal vasoconstriction during exercise. When followed by an ischemia-reperfusion event after restoring renal blood flow, inadequate levels of antioxidative uric acid in patients with renal hypouricemia fail to protect their kidneys from oxidative stress damage, leading to AKI.[42,43]

Otomo et al[21] studied the relationship between SUA and AKI in 59 219 hospitalized patients, revealing a U-shaped graph suggesting that both hyperuricemia and hypouricemia, with a nadir SUA level of approximately 4 to 5 mg/dL, led to an increased incidence of AKI. A similar U-shaped graph was obtained in patients undergoing cardiovascular surgery, suggesting that postoperative AKI was associated with preoperative hyperuricemia and hypouricemia.[16] Although we were unable to show a similar U-shaped relationship, we observed that recipients with preoperative SUA <4.15 mg/dL were nine times more likely to develop AKI in the first 72 hours after LDLT. Several hypotheses have attempted to explain how low SUA negatively affects renal function. First, malnutrition secondary to decreased protein and energy intake may be associated with low SUA, leading to AKI. Otomo et al. reported that hypouricemic patients were complicated with hypoalbuminemia and malnutrition status. Furthermore, higher all-cause mortality was reported in malnourished older adult patients with low SUA levels.[21,44] In the present study, the albumin levels in the four SUA quartiles all fell below the normal laboratory range (<3.5 mg/dL; Supplementary Table 1, http://links.lww.com/JCMA/A254). Explicitly, the group with the lowest SUA quartile was associated with the lowest albumin level and the highest AKI incidence, which was consistent with the aforementioned studies. Because malnutrition and hypoalbuminemia are known risk factors for AKI, we adjusted for albumin and BMI in our multivariate analysis. Despite such adjustment, low SUA persisted as an independent risk factor for postoperative AKI. Second, the antioxidant property of uric acid has been widely discussed. The SUA level was significantly lower in patients with chronic liver cirrhosis compared with the normal population. In liver transplantation, ischemia-reperfusion injury during the reperfusion stage is related to the release of reactive oxygen species, changes in micro-RNA expression, and autophagy regulation.[45] The relatively low uric acid level in liver recipients fails to generate sufficient antioxidant capacity to prevent the injury from placing excessive oxidative stress on the kidneys. Our study also showed a trend of patients without AKI or with mild AKI having high SUA levels, while those with more severe AKI had lower SUA levels, suggesting that low preoperative SUA may be a risk factor for severe AKI development.

We have presented the first cohort study to identify low pretransplantation SUA levels as a novel risk factor for early AKI after LDLT. However, this prospective study has some limitations. First, when diagnosing AKI, we used SCr instead of urine output according to the KDIGO guideline, although it may be debatable which one is preferable in reaching a diagnosis of AKI.[25] Second, Asian ethnicity and the small population size may have reduced the applicability of our findings, and validation using a larger data set is warranted.

In conclusion, AKI after liver transplantation leads to a deterioration of patient status secondary to prolonged ICU stay, increased risk of graft failure, progression to CKD, and, most importantly, increased mortality rates. We have demonstrated that low preoperative SUA may be a risk factor for early postoperative AKI.

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Table 3

Logistic regression analysis of the risk factors

	Univariate regression			Multivariate regression		
	OR	(95% CI)	*p*	OR	(95% CI)	*p*
Male sex	2.31	(0.66-8.02)	0.189			
Age, y	1.06	(1.00-1.12)	0.057			
Height, cm	0.36	(0.00-254.49)	0.758			
Body weight, kg	0.99	(0.96-1.03)	0.714			
BMI	0.99	(0.87-1.12)	0.818			
Diuretic use	1.49	(0.56-3.97)	0.421			
ABO incompatibility	0.65	(0.12-3.62)	0.623			
Comorbidity						
DM	3.35	(1.03-10.92)	0.045[a]			
Hypertension	1.26	(0.45-3.56)	0.659			
Viral hepatitis	2.64	(0.84-8.34)	0.097			
HBV	2.70	(0.97-7.51)	0.057			
HCV	0.96	(0.25-3.66)	0.954			
HCC	0.63	(0.23-1.68)	0.350			
Alcoholism	0.38	(0.12-1.19)	0.097			
MELD score	1.06	(1.00-1.13)	0.061			
Preoperative lab data						
Creatinine, mg/dL	0.87	(0.19-4.06)	0.857			
eGFR, mL/min/1.73 m^2	1.00	(0.99-1.01)	0.709			
BUN, mg/dL	0.98	(0.91-1.05)	0.530			
Anemia	4.00	(0.81-19.73)	0.089			
Hypoalbuminemia	4.83	(1.26-18.57)	0.022[a]			
Hyponatremia	2.00	(0.65-6.20)	0.230			
SUA, mg/dL	0.63	(0.46-0.86)	0.004[a]			
Low SUA (<4.15 mg/dL)	6.22	(2.12-18.28)	0.001[a]	[b]9.37	(2.05-43.06)	0.004[a]
Perioperative factors						
Blood loss, mL	1.09	(1.01-1.16)	0.022[a]			
RBC transfusion, unit	1.00	(1.00-1.00)	0.158			
Cold ischemia time, min	1.02	(0.95-1.09)	0.570			
Warm ischemia time, min	1.00	(0.98-1.02)	0.872			

AKI = acute kidney injury; BMI = body mass index; BUN = blood urea nitrogen; cm = centimeter; dL = deciliter; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; g = gram; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; kg = kilogram; L = liter; LVEF = left ventricular ejection fraction; m = meter; MELD = model for end-stage liver disease; mg = milligram; min = minute; mL = milliliter; RBC = red blood cell; SUA = serum uric acid.

[a]*p* value < 0.05.

[b]Adjusted for age, gender, BMI, diuretic use, ABO incompatibility, DM, hypertension, viral hepatitis, alcoholism, MELD score, creatinine, BUN, anemia, hypoalbuminemia, hyponatremia, blood loss, RBC transfusion, cold ischemia time, warm ischemia time.

Table 4

Staging of AKI in high and low SUA groups

	Low SUA (N = 25)	High SUA (N = 45)
No AKI	9 (20.5%)	35 (79.5%)
AKI stage 1	5 (35.7%)	9 (64.3%)
AKI stage 2	6 (100%)	0 (0.0%)
AKI stage 3	5 (83.3%)	1 (16.7%)

AKI = acute kidney injury; SUA = serum uric acid.

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://links.lww.com/JCMA/A254.

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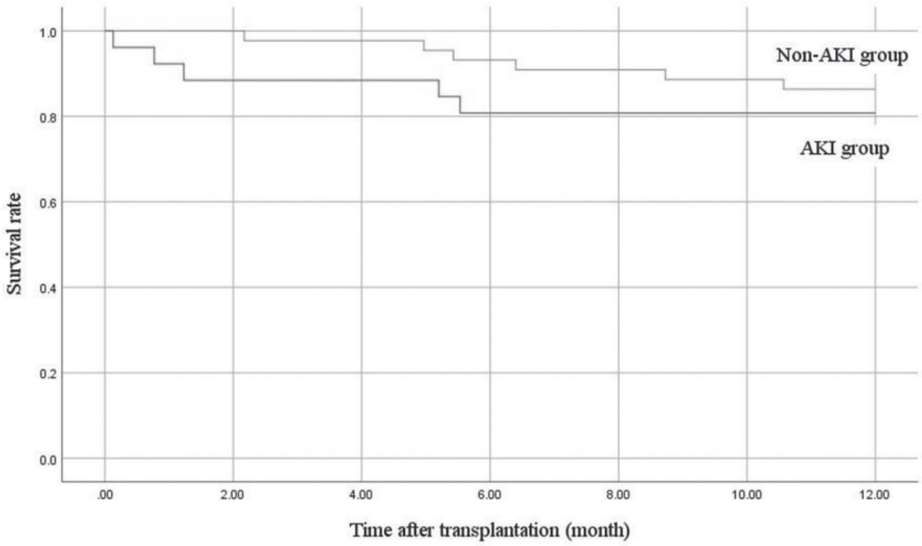

Fig. 4 Kaplan-Meier curves of 12-mo survival of AKI and non-AKI groups. AKI = acute kidney injury.

Table 5

Analysis for survival, EAD and ICU stay

	AKI, %	non-AKI, %	*p*
1-mo survival	92.3	100.0	0.063
3-mo survival	88.5	97.7	0.101
6-mo survival	80.8	93.2	0.108
12-mo survival	80.8	86.4	0.471
EAD	8 (30.1%)	6 (13.6%)	0.083
ICU stay, d	15.4 ± 9.2	17.5 ± 19.3	0.604

AKI = acute kidney injury; EAD = early allograft dysfunction; ICU = intensive care unit.

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