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Early Prediction of Acute Kidney Injury in Living Donor Liver Transplantation by Serum Cystatin C Concentration at the End of the Surgery

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Introduction. Acute kidney injury (AKI) is a prevalent complication of liver transplantation, leading to prolonged hospital or intensive care unit stay and significant morbidity. Recently, biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL) and cystatin C have been investigated for their potential role in the early detection of AKI in liver transplantation patients.

Method. Our study comprised 60 patients with end-stage liver disease undergoing living donor liver transplantation. Based on the postoperative development of AKI, the patients were categorised into two groups: the AKI group comprising 22 patients and the non-AKI group comprising 38 patients. Serum cystatin C and urine NGAL levels were measured twice: immediately after induction of anaesthesia (baseline) and at the end of the surgery.

Results. The overall incidence of AKI was 36.66%. The mean cystatin C level measured at the end of the surgery was significantly higher in the AKI group (1.12 ± 0.40 mg/L) than in the non-AKI group (0.82 ± 0.27 mg/L) [P = .001]. The receiver operating characteristic curve for the postoperative cystatin C biomarker demonstrated a significant difference between the AKI and non-AKI groups [area under the curve: 0.71, P = .007]. However, baseline cystatin C and urine NGAL levels did not significantly differ between the groups.

Conclusion. Cystatin C levels measured at the end of the surgery showed a better predictive value and higher accuracy in identifying post-liver transplantation patients with AKI than baseline cystatin C and urine NGAL.

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INTRODUCTION

Orthotopic liver transplantation (OLT) is the treatment of choice for end-stage liver disease (ESLD) although it is associated with significant morbidity and mortality.[1] Acute kidney injury (AKI) is a common complication of liver transplantation, resulting in prolonged intensive care unit or hospital stay and substantial morbidity, which may progress to chronic kidney disease in liver transplant recipients.[1,2] A variety of factors contribute to AKI after liver transplantation (LT), including hepatic decompensation in recipients, diabetes mellitus, suboptimal donor graft quality, intraoperative hemodynamic instability, blood loss, blood product transfusions, and the administration of nephrotoxic drugs.[3-6] The estimated incidence rates for post-liver transplant AKI and severe AKI requiring renal replacement therapy (RRT) are

40.8% and 7.0%, respectively.[7]

The conventional diagnostic tool for assessing renal function is measurement of serum creatinine, which has a few limitations, including insensitivity to minor changes in renal function, lack of specificity due to the impact of non-renal factors (e.g., muscle mass and diet), and a delayed response to kidney injury. These challenges are exacerbated in cases of ESLD, where creatinine has been demonstrated to be an inaccurate indicator of renal function.[8] In this regard, patients in the perioperative period of LT may benefit from more sensitive and specific biomarkers of AKI to detect early and mild renal impairment, thereby significantly improving patient care by facilitating early therapeutic or preventive intervention for acute kidney injury after LT. Over the last decade, several novel kidney biomarkers have been extensively studied and introduced into clinical practice. Among them, serum cystatin C and urine neutrophil gelatinase-associated lipocalin (NGAL) have shown potential for the early detection of AKI in patients with cirrhosis.

Cystatin C is a 13-kD endogenous cysteine protease inhibitor that is produced by all nucleated cells at a constant rate.[9] In contrast to serum creatinine levels, cystatin C levels are less influenced by sex, hepatic function, or muscle mass.[10] Due to its small size, cystatin C is freely filtered by the glomeruli and is completely reabsorbed and destroyed by the cells in the proximal renal tubules without reaching the systemic circulation.[11] Accordingly, serum cystatin C levels indirectly reflect glomerular filtration rate and aids in assessing renal function.[11]

NGAL is a member of the lipocalin family of proteins and is a low-molecular-weight protein present in neutrophils.[12] It is also produced by the cells of the thick ascending loop and collecting ducts of the kidneys, as well as by hepatocytes the lung and gastrointestinal tract cells.[13] Its production is considerably elevated following renal tubular ischaemia, septicaemia, or nephrotoxic damage,[14] making it a marker of renal tubular damage.[15] Both cystatin C and urine NGAL have been identified as early predictors of AKI in various clinical scenarios, including critical illness, after cardiopulmonary bypass surgery, heart failure, and contrast-induced kidney injury.[16-19] However, only limited studies have explored the role of these biomarkers in detecting AKI in patients undergoing OLT. Therefore, this study aimed to analyse the predictive ability of serum cystatin C and urine NGAL in detecting immediate AKI after living donor related LT (LDLT).

MATERIALS AND METHODS

After receiving approval from the institutional ethics committee (IEC/2021/83/MA10), we prospectively observed all consecutive adult patients (age: 18–65 years) with chronic liver disease who underwent LDLT at our institute from January 2021 to May 2022. Patients with a recent history of AKI (< 30 days), chronic kidney disease requiring RRT, acute liver failure, acute-on-chronic liver failure, severe cardiac dysfunction, or paediatric patients were excluded from the study. Written informed consent was obtained from all participants. A standard anaesthesia protocol was followed, which involved the administration of fentanyl (2 µg/kg), propofol (2 mg/kg), and rocuronium (1 mg/kg) to induce anaesthesia and facilitate endotracheal intubation. We maintained anaesthesia was maintained with a 50% oxygen-air mixture, sevoflurane at 0.8–1.0 minimum alveolar concentration, and infusions of fentanyl and atracurium. Intravenous fluids (PlasmaLyte A ™) and vasopressors were titrated to maintain a mean arterial pressure ≥ 65 mmHg and a stroke volume variation of < 13%. Monitoring and correction of coagulopathy were guided by Thromboelastography. ™ Arterial blood gas, blood sugar, and ionised calcium levels were serially monitored and treated as needed. Postoperatively, all patients were transferred to the intensive care unit (ICU) for further management. Immunosuppression was provided per our institute's protocol using triple immunosuppressive regimens. Methylprednisolone (100 mg) was administered before reperfusion of the graft, followed by a gradual taper until 20 mg by the end of the first week. The oral dose for mycophenolate mofetil was 1 g twice a day in combination with tacrolimus, which was initiated within the first 24 h post-LT. The initial oral dose of tacrolimus was 0.5 mg twice a day, which was initiated within the first 24 h post-LT if renal functions were normal. Subsequent dosing was guided by daily blood trough levels, aiming for a targeted tacrolimus level of 7–10 µmol/L.

Definition of postoperative AKI

Early AKI after LDLT was determined according

to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria.[20] The maximum serum creatinine level during the first week after surgery was compared to baseline levels before surgery. AKI was defined as an increase in serum creatinine level of ≥0.3 mg/dL within 48 h or an increase in serum creatinine level of ≥1.5 times that at baseline within the last 7 days. The serum creatinine level closest to the date of the surgery was considered the baseline serum creatinine level.

AKI severity was classified as follows:

Stage 1: Increase in serum creatinine of ≥0.3 mg/dl within 48 h or increase in serum creatinine level 1.5–1.9 times from baseline within 7 days.

Stage 2: Increase in serum creatinine level 2.0–2.9 times from baseline within 7 days.

Stage 3: Increase in serum creatinine level 3 times from baseline within 7 days or ≥4.0 mg/dL, with an acute increase of at least 0.5 mg/dL within 48 h or started on RRT.[20]

Based on the development of postoperative AKI, the patients were divided into two groups: AKI and non-AKI.

Biomarker assessment

Urine samples were collected from 60 patients immediately after the induction of anaesthesia (baseline) and at the end of the surgery. These samples were processed, stored at −80°C, and analysed for urine NGAL using sandwich enzyme immunoassay with a commercially available kit (Immunotag, Human NGAL ELISA kit).

Blood samples were collected from 60 patients at baseline (immediately after the induction of anaesthesia) and at the end of the surgery, and serum cystatin C level was measured using nephelometry (ATELLICA NEPH-630).

Data collection

Recipient characteristics, including demographic data, the Model for End-Stage Liver Disease (MELD) Score, baseline serum creatinine level, and other preoperative laboratory variables, along with donor characteristics, surgery-related variables (duration of the an-hepatic phase, duration of surgery, total blood loss, and total blood products transfused), and graft-related variables, were recorded.

Statistical analysis

Data were analysed using Statistical Package for Social Sciences (SPSS) software, version 21.0 (IBM, Chicago, USA). Continuous data were represented as mean ± SD or median (interquartile range). Categorical data were represented as numbers (percentages). The Kolmogorov–Smirnov test was used to determine the normality of the data. Continuous data were analysed using an independent t-test or Mann–Whitney U-test based on the normality of the data. Categorical data were analysed using chi-square or Fisher's tests when appropriate. P-values < 0.05 were considered significant. The receiver operating characteristic (ROC) curve was used to show the sensitivity and specificity and to determine the optimal cut-off value. Correlations between measured parameters and laboratory data were analysed using Spearman's correlation coefficient.

RESULTS

A total of 74 patients underwent OLT during the study period. Out of these, 14 individuals were not included in the study. This group consisted of four patients with acute liver failure, six kids who were undergoing pediatric liver transplantation, and four patients with a recent history of acute kidney injury. Consequently, we prospectively assessed 60 adult patients who underwent LDLT during the study period. Their ages ranged from 20 to 62 years, with a mean value of 44.72 ± 9.00 years. The cohort included 53 males (88.33%) and 7 females (11.67%). The etiologies of ESLD were alcoholic liver disease (53.30%), NASH (11.70%), viral (10%), and others (25%)—including hepatocellular carcinoma, autoimmune, primary biliary sclerosis, primary sclerosing cholangitis, and hepatic epithelioid haemangioma.

According to the KDIGO criteria, 22 patients (36.66%) experienced AKI immediately after LDLT. The majority [17 (77.27%)] had stage 1AKI, and the remaining [5 (22.72%)] had stage 2 AKI. None of the patients received RRT within the first week after LT. The patients were divided into two groups: AKI (n = 22) and non-AKI (n = 38). Both groups were comparable with respect to basic parameters such as age, sex, body mass index (BMI), comorbidities, aetiology, and severity of liver disease (Table 1). Donor characteristics, including age, sex, and BMI, were also comparable. Among the intraoperative and anaesthesia-related variables, patients who developed AKI received significantly more fresh

Table 1. Data are presented as mean ± SD or n (%) as indicated. *Independent t-test, §Chi-square test. Abbreviations: BMI: Body mass index, MELD: Model for end-stage liver disease.

Variables	AKI group (n = 22)	Non-AKI group (n = 38)	*P*
Age (years)	46.67 ± 9.42	43.60 ± 8.68	.212*
Sex men (n (%))	21 (95.45)	32 (84.21)	.191§
BMI (kg/m^2)	26.01 ± 4.10	24.53 ± 3.35	.134*
Preoperative MELD	21 ± 5.84	21 ± 5.94	.753*
serum creatinine (mg/dL)	0.74 ± 0.17	0.72 ± 0.21	.713*
Haemoglobin (g/dL)	9.5 ± 1.8	9.0 ± 1.8	.258*
Aetiology of liver disease			
Alcohol related (n (%))	10 (45.45)	22 (57.89)	.352§
NASH (n (%))	4 (18.18)	3 (7.89)	.232§
Viral (n (%))	3 (13.64)	3 (7.89)	.475§
Others (n (%))	5 (22.72)	10 (26.32)	.757§
Comorbidities			
Hypertension (n (%))	2 (9.09)	1 (2.63)	.270§
Diabetes (n (%))	9 (40.90)	13 (34.21)	.604§
Hypothyroid (n (%))	7 (31.81)	10 (26.32)	.768§

frozen plasma (FFP) and single donor platelet concentrate transfusions intraoperatively (*P* = .033, *P* = .019, respectively). The median duration of ICU stay was significantly higher in the AKI group than in the non-AKI group [14.5 days (interquartile range [IQR] = 8, 30.25) vs 9 days (IQR = 6.75, 15), respectively, *P* = .03] (Table 2).

Serum cystatin C and urine NGAL (uNGAL) levels were measured at baseline (immediately after induction of anaesthesia) and at the end of the surgery. The mean cystatin C level at baseline was 1.49 ± 0.44 mg/L in the AKI group and 1.34 ± 0.45 mg/L in the non-AKI group, with no significant difference (*P* = .198). However, the mean cystatin C level at the end of the surgery was significantly higher in the AKI group (1.12 ± 0.40 mg/L) than in the non-AKI group (0.82 ± 0.27 mg/L; *P* = .001) (Table 3). The median uNGAL level at baseline was 2.38 ng/mL (IQR = 1.12, 5.32) in the AKI group and 1.92 ng/mL (IQR = 1.17, 5.17) in the non-AKI group, with no significant difference (*P* = .765). The uNGAL level at the end of surgery was also comparable between the groups (Table 4).

There was a statistically significant positive correlation between baseline cystatin C and baseline serum creatinine levels (r = 0.44, *P* = .0001) and

Table 2. Data are presented as mean ± SD, median (Q1, Q3) or n (%) as indicated.

Variables	AKI group (n = 22)	Non-AKI group (n = 38)	*P*
CIT (min)	85.5 (70.75, 118)	82.5 (65, 94.75)	.220†
WIT (min)	25.5 (21.75, 33.25)	26 (22, 31)	.830†
Duration of an-hepatic phase (min)	80.5 (62.75, 135.0)	88 (57, 156)	.908†
Temporary Portocaval shunt (n (%))			
Yes	12 (54.54)	27 (71.05)	.196§
No	10 (45.45)	11 (28.95)	
Graft lobe (n (%))			
Right	19 (86.36)	32 (84.21)	.822§
Left	3 (13.64)	6 (15.79)	
GRWR	0.94 ± 0.21	0.98 ± 0.22	.431*
Blood loss	2000 (1375, 3500)	1825 (1387, 2325)	.504†
PRBC (unit)	3.5 (2, 7.25)	4 (2, 5)	.744†
FFP (unit)	2 (0, 3)	0 (0, 2)	.033†
Cryoprecipitate (unit)	3.5 (0, 5.25)	0 (0, 4.5)	.069†
SDPC (unit)	0 (0, 1)	0 (0, 0)	.019†
Duration of surgery(min)	730 (700, 760)	737.5 (700, 765)	.799†
ICU Stay (days)	14.5 (8, 30.25)	9.5 (6.75, 15)	.030†

*Independent t-test, †Mann Whitney test, §Chi-square test
Abbreviations: CIT: Cold ischemia time, WIT: Warm ischemia time, GRWR: Graft recipient weight ratio, PRBC: Packed red blood cells, FFP: Fresh frozen plasma, SDPC: Single donor platelet, ICU: Intensive care unit.

Table 3. Data are presented as mean ± SD, *Independent t-test

Serum cystatin C (mg/L)	AKI group (n = 22)	Non-AKI group (n = 38)	*P*
Baseline	1.49 ± 0.44	1.34 ± 0.45	.198*
End of surgery	1.12 ± 0.40	0.82 ± 0.27	.001*

Table 4. Data are presented as median (Q1, Q3), †Mann Whitney test

Urine NGAL (ng/ml)	AKI group (n = 22)	Non-AKI group (n = 38)	*P*
Baseline	2.38 (1.12, 5.32)	1.92 (1.17, 5.17)	.765†
End of surgery	1.65 (0.79, 2.42)	1.43 (0.96, 2.07)	.679†

between serum cystatin C at the end of surgery and the corresponding serum creatinine levels (r = 0.52, *P* = .0001). No such correlations were observed between urine NGAL and serum creatinine levels.

An ROC curve analysis of cystatin C levels at the end of surgery was conducted to establish the accuracy and determine the optimum cut-off for predicting post-transplant AKI. ROC curves above the diagonal line are considered to have a reasonable discriminating ability to predict AKI. The discriminatory power of serum cystatin C level (mg/L) at the end of surgery was acceptable (area under the curve [AUC] 0.71; *P* = .007; 55% sensitivity and 85% specificity at a cut-off level of 1.1) (Figure 1).

DISCUSSION

In this study, we investigated early post-transplant AKI in patients undergoing LDLT, focusing on the predictive roles of biomarkers of serum cystatin C and uNGAL. Our findings highlighted that AKI is a significant clinical

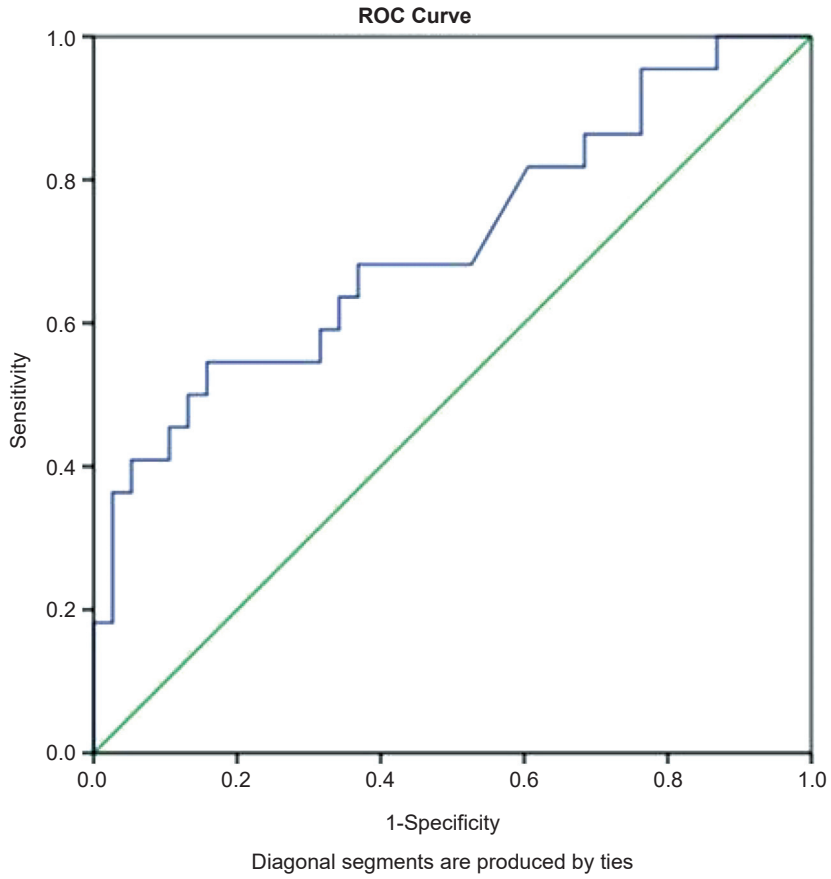

Figure 1. ROC curve analysis showing the diagnostic power of serum cyatatin C (end of the surgery) in the prediction of acute kidney injury.

challenge in the early postoperative period for LT patients, with nearly 36.66% experiencing some level of AKI. This aligns with the incidence of AKI estimated in prior studies across larger populations (17%–95%).[3-5] Notably, the majority (72.27%) of patients in our study had stage 1 AKI, and no patients had stage 3 AKI or required RRT within the first week post-LT.

We found that serum cystatin C level at the end of surgery was significantly associated with early AKI post-LT. Additionally, a statistically significant positive correlation was noted between serum cystatin C and corresponding serum creatinine levels. However, no association was observed between pretransplant serum cystatin C levels and early postoperative AKI. The ROC curve for the end-of-surgery cystatin C level showed a borderline significant change between the AKI and non-AKI groups (AUC: 0.71, P = .007), with a cut-off of 1.1 mg/L, with 55% sensitivity, and 85% specificity. This is in accordance with various previous studies evaluating the role of cystatin C as a predictor of AKI in patients with decompensated cirrhosis.[21,22]

In contrast, uNGAL levels could diagnose AKI in early stage, as no significant differences were observed in the median levels of this marker between the two groups. This finding is consistent with the findings reported by Mogawer *et al.*, who observed no difference in urine NGAL levels between AKI and non-AKI groups in patients with ESLD undergoing LDLT.[23] Similarly, Cezar *et al.* prospectively analysed uNGAL levels in 46 patients who underwent LDLT and observed no significant difference between the AKI and non-AKI groups.[24] While some studies have suggested NGAL as an early marker of renal damage after LT in adult patients,[25,26] the variable predictive performance may be explained, at least partly, by the non-uniform definitions of AKI used across studies, the severity of AKI, and differences in the timing of biomarker measurement. Baron-Stefaniak *et al.* demonstrated that uNGAL is a better predictor of severe AKI than mild AKI in LDLT patients.[27]

By analyzing different preoperative and intraoperative factors, we found that patients who experienced acute kidney injury (AKI) received significantly greater amounts of fresh frozen plasma (FFP) and platelet transfusions. Poor preoperative hepatic function has been linked to an increased risk of post-OLT AKI.[28,29] In the present study, while the MELD score did not differ between the AKI and non-AKI groups, patients with early AKI post-LT experienced a significantly longer ICU stay. This aligns with previous studies by Park *et al.* and Karapanagiotou *et al.*, underscoring the strong association between the occurrence of AKI and adverse outcomes, including prolonged ICU and hospital stays.[30,31]

CONCLUSION

This study demonstrates that serum cystatin C levels at the end of the surgery serve as a better predictive indicator of mild AKI in patients after LDLT than baseline cystatin C and urine NGAL levels. The significance of uNGAL in predicting less severe degrees of AKI among living donor patients remains debatable.

LIMITATIONS

First, this study is a single-centre trial with a small sample size, and the majority of patients exhibited mild renal impairment. Second, serum creatinine changes were utilised to diagnose AKI, although it was possible that some individuals had some degree of renal impairment without having elevated creatinine levels.

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