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Incidence and Risk Factors of Acute Kidney Injury following Orthotopic Liver Transplantation

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Background: Acute kidney injury (AKI) commonly occurs during post-liver transplant period. However, the incidence varies largely between studies and the predisposing factors are not well understood.

Objectives: This study aims to identify the incidence and risk factors contributing to early AKI following liver transplantation.

Methods: Electronic medical records of 87 patients underwent orthotopic liver transplantation at King Chulalongkorn Memorial Hospital during January 2014 to October 2019 were retrospectively reviewed. Primary endpoint was the incidence of AKI which was defined according to Kidney Disease Improving Global Outcomes (KDIGO) criteria. Secondary endpoints were the need for renal replacement therapy (RRT), length of hospital stay, and factors associated with post-liver transplant AKI. Data were collected and analyzed using univariate and multiple logistic regression with stepwise variable selection to identify risk factors for AKI.

Results: AKI occurred in 43 patients (49.4%). Independent factors found to be associated with post-liver transplant AKI were decreased intraoperative urine output, longer duration of postoperative dopamine infusion, and higher dose of postoperative diuretics (P=0.002, P=0.019, and P<0.001, respectively). In AKI group, 2 patients required RRT (4.8%). When comparing AKI to non-AKI groups, duration of length of hospital stay was not significantly different (7.7 vs 11.1 days, respectively, P=0.86). There was no mortality in our study.

Conclusions: The incidence of AKI following orthotopic liver transplantation was 49.4%. Decreased intraoperative urine output, prolonged duration of postoperative dopamine infusion, and higher dose of postoperative diuretics were independently associated with the development of AKI. AKI was not associated with prolonged hospitalization.

Keywords: Acute kidney injury, Incidence, Liver transplantation, Risk factors

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Liver transplantation is one of the standard treatments in patients with end-stage liver disease (ESLD). Acute kidney injury (AKI) is a common complication following liver transplantation, with incidence varying from 5 to 94%[1-3] due to the application of different definitions and criteria.[4] The pathophysiology underlying AKI development is complicated and multifactorial in etiology. In ESLD patients, hyperdynamic circulation syndrome develops, causing splanchnic vasodilatation and arterial hypotension.[5] As a result, renal circulatory vasoconstriction, decreased renal perfusion, and AKI follow respectively.[6] The development of AKI increases morbidity, in-hospital mortality, and healthcare expenses.[7] Moreover, AKI is associated with poor graft survival[8,9], development of chronic kidney disease (CKD)[10,11], and need for renal replacement therapy (RRT) which lead to prolonged hospitalization.[12-14] During the past decades, a number of predisposing factors have been studied with varying results between institutions. Patient-related factors including female sex,

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higher body mass index (BMI), and higher severity of liver disease graded by Model for End-Stage Liver disease (MELD) and Child-Turcotte-Pugh (CTP) score are found associated with higher incidence of post-liver transplantation AKI.[15-19] Surgical and anesthesia-related factors have also been studied, due to the modifiability with resultant clinical impact. Duration of cold and warm ischemic time, surgical techniques, and donor age were long found significant for AKI occurrence.[20-22] Intraoperative and postoperative fluid and vasopressor management, blood product transfusion, urine output, and glycemic control have also been found associated with post-liver transplantation AKI.[17,21,23-27] Nowadays, with advancement in anesthesia, operative technique, and immunosuppression, AKI is still common post-liver transplantation surgery. Yet, factors associated with AKI remain largely unclear. Our study aims to identify the incidence and associated risk factors of AKI following orthotopic liver transplantation (OLT) in King Chulalongkorn Memorial Hospital and obtain its effect on RRT and length of hospital stay (LOS).

Material and Methods

The study was a retrospective observational study. Approval was obtained from the Institutional Review Board from Chulalongkorn University (number 360/63). Electronic medical records of ESLD patients more than 18 years of age who underwent OLT from cadaveric donor in King Chulalongkorn Memorial Hospital during January 2014 to October 2019 were reviewed. The exclusion criteria were simultaneous liver-kidney transplantation, history of previous liver or kidney transplantation, ESRD, preoperative AKI, mortality within 72 hours during postoperative period, and missing anesthesia record. The diagnosis of AKI followed Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guideline for Acute Kidney Injury.[28] Data were collected until 72 hours postoperatively.

We collected demographic data, etiology, comorbidity, MELD and CTP score, and baseline serum creatinine (SCr) and glomerular infiltration rate (GRF) calculated by Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. Intraoperative parameters, including operative time, anesthetic time, cold and warm ischemic time, donor age, doses and duration of vasopressors and inotropes, blood loss and fluid management, and urine output were recorded. After completion of surgery, the duration of vasopressors and inotropes use, diuretic dose, urine output, and LOS were obtained.

Sample size was calculated using estimating proportion of one group with alpha error rate of 0.1. The incidence of post-liver transplantation AKI equals to 35% from previous study.[20] Thus, the minimum sample size required was 87 patients.

All continuous data were presented as mean with standard deviation (SD) or median with interquartile range (IQR). Categorical data were presented as counts with percentages. Imputation was not done on missing data. Statistical comparisons for continuous variables were compared by Student's unpaired t-test in normally distributed data or Mann-Whitney-U test in non-normally distributed data; categorical variables were compared by Pearson's chi-squared test. We fitted logistic regression models to examine the association between each predictor and AKI. Factors showing an association with AKI at a level of $P < 0.20$ were included in the stepwise regression analysis. All analyses were conducted using RStudio (version 3.6.1, RStudio, Inc., Boston, MA). Statistical significance level was accepted at two-sided p-values < 0.05.

Results

During the enrollment period, electronic medical records of 99 patients undergoing OLT were retrospectively reviewed. Of these, 12 patients were excluded from the study (1, 1, and 10 patients underwent simultaneous liver-kidney transplantation, previous liver transplantation, and were diagnosed with preoperative AKI, respectively). In total, data of 87 patients were collected and analyzed (Figure 1). The

baseline characteristics of the study population are shown in Table 1. The incidence of postoperative AKI was 49.4% (43 of 87 patients). Among these 43 patients, 62.8% (27 of 43), 23.2% (10 of 43), and 14% (6 of 43) were diagnosed with stage 1, 2, and 3 AKI respectively. There was no difference in the demographic data between the groups. The majority indications for OLT were viral hepatitis (N = 30, 34.5% for hepatitis C cirrhosis and N = 29, 33.3% for hepatitis B cirrhosis) and alcoholic cirrhosis (N = 23, 26.4%). Non-alcoholic steatohepatitis (N = 6, 6.9%), cryptogenic cirrhosis (N = 4, 4.6%), and miscellaneous causes (N = 6, 6.9%) comprised the rest. Severity of ESLD were assessed by MELD and CTP scores, which did not differ between the 2 groups. Baseline SCr were comparable. The surgery was performed using piggyback technique in all patients. During surgery, 7 patients (8%) received blood salvage and 2 patients (2.3%) received autologous blood donation. Intraoperative crystalloid use consisted of normal saline, 5% dextrose in 0.45% saline, acetated and lactated Ringer's solutions. Synthetic colloid use consisted of 4% Gelofusine and hydroxyethyl starch. While synthetic colloid use was 5% albumin. Furosemide was used for postoperative diuretic, both orally and intravenously.

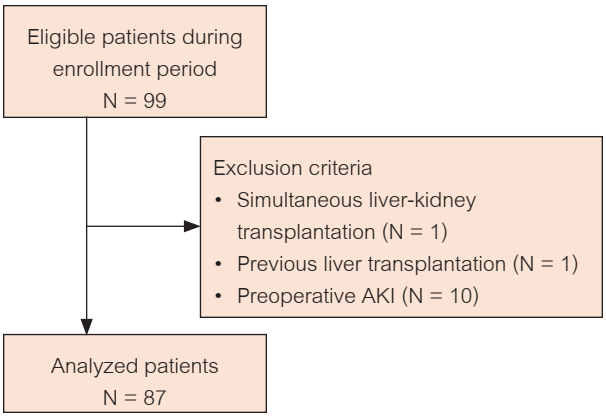

Figure 1 Study flow

There was no death in our study. The LOS was 7.77 days in AKI group and 11.11 days in non-AKI group with no statistical significance (P = 0.86). Two patients needed RRT, but none needed permanent dialysis (Table 2).

In univariate analysis, the increase in MELD score, low-dose intraoperative norepinephrine infusion, intraoperative and postoperative urine output, postoperative dopamine infusion time, and furosemide dose were found associated with the occurrence of AKI (Table 3). Multiple logistic regression with stepwise variable selection found association of AKI only with intraoperative urine output, postoperative dopamine infusion time, and furosemide dose (Table 4).

Table 1 Baseline characteristics of study population

	Total (N = 87)	Patients with AKI (N = 43)	Patients without AKI (N = 44)	P-value
Preoperative parameters:				
Age (year)	55.43 (10.53)	56.02 (8.75)	54.84 (12.09)	0.60
Male (N, %)	60 (69.0)	31 (72.1)	29 (65.9)	0.70
BMI (kg/m^2)				0.02
≤20	10 (11.5)	6 (14.0)	4 (9.1)	
20-30	62 (71.3)	25 (58.1)	37 (84.1)	
>30	15 (17.2)	12 (27.9)	3 (6.8)	
Comorbidity (N, %)				
Diabetes mellitus	37 (42.5)	21 (48.8)	16 (36.4)	0.34
Hypertension	28 (32.2)	11 (25.6)	17 (38.6)	0.28
CKD	7 (8.0)	5 (11.6)	2 (4.5)	0.41
Etiology (N, %)				
Alcoholic cirrhosis	23 (26.4)	15 (34.9)	8 (18.2)	0.13
Hepatitis B cirrhosis	29 (33.3)	14 (32.6)	15 (34.1)	1
Hepatitis C cirrhosis	30 (34.5)	11 (25.6)	19 (43.2)	0.13

Table 1 Baseline characteristics of study population (cont.)

	Total (N = 87)	Patients with AKI (N = 43)	Patients without AKI (N = 44)	P-value
Severity				
MELD score	17.64 (6.71)	19.00 (6.02)	16.32 (7.15)	0.06
CTP score	8.84 (2.15)	9.08 (2.04)	8.66 (2.25)	0.46
Pre-transplant SCr (mg/dL)	0.91 (0.31)	0.94 (0.32)	0.88 (0.30)	0.35
Intraoperative parameters:				
Operative parameters (min)				
Operative time	409.05 (75.03)	423.58 (86.39)	394.84 (59.58)	0.07
Anesthetic time	509.15 (86.42)	525.23 (103.25)	493.07 (62.66)	0.08
Cold ischemic time	394.67 (105.39)	391.69 (80.35)	397.58 (126.08)	0.80
Warm ischemic time	41.80 (8.20)	42.62 (6.17)	41.00 (9.79)	0.37
Donor age (year)	32.28 (13.71)	31.39 (12.24)	33.11 (15.05)	0.57
Blood loss (ml)	4393.10 (5580.76)	5083.72 (7033.22)	3718.18 (3609.82)	0.26
Blood product transfusion (units)				
Packed red cells	5.11 (7.17)	5.95 (8.17)	4.30 (6.03)	0.28
Fresh frozen plasma	5.60 (5.94)	6.09 (7.19)	5.11 (4.41)	0.45
Platelet concentrate	7.60 (7.37)	8.44 (7.49)	6.77 (7.25)	0.29
Cryoprecipitate	2.92 (7.55)	3.53 (8.91)	2.32 (5.98)	0.46
Inotrope/vasopressor duration (min)				
Dobutamine <5 mcg/kg/min	3.49 (23.20)	6.98 (32.63)	0 (0.0)	0.17
Dobutamine ≥5 mcg/kg/min	0 (0.0)	0 (0.0)	0 (0.0)	N/A
Dopamine <5 mcg/kg/min	149.48 (142.90)	143.72 (144.91)	155.23 (142.35)	0.71
Dopamine ≥5 mcg/kg/min	110.35 (145.95)	132.44 (178.91)	88.26 (100.51)	0.16
Norepinephrine <0.5 mcg/kg/min	204.13 (183.57)	236.28 (187.98)	171.98 (175.33)	0.11
Norepinephrine ≥0.5 mcg/kg/min	13.60 (67.54)	13.95 (66.87)	13.26 (68.99)	0.96
Epinephrine <0.5 mcg/kg/min	39.65 (80.69)	40.70 (80.06)	38.60 (82.25)	0.91
Epinephrine ≥0.5 mcg/kg/min	2.27 (21.03)	4.53 (29.74)	0 (0.0)	0.32
Fluid administration (ml)				
Crystalloid	2281.99 (1910.61)	2644.12 (2437.54)	1928.28 (1114.20)	0.08
Synthetic colloid	179.07 (576.22)	237.21 (645.87)	120.93 (497.88)	0.35
Natural colloid	2405.62 (1379.43)	2484.84 (1511.28)	2326.40 (1246.65)	0.60
Urine output (ml/kg/h)	1.09 (0.77)	0.87 (0.70)	1.32 (0.79)	<0.01
Postoperative parameters:				
Inotrope/vasopressor duration (min)				
Dobutamine	12.71 (90.28)	7.14 (46.29)	18.14 (118.95)	0.58
Dopamine	342.29 (719.24)	552.74 (945.07)	136.74 (274.20)	<0.01
Norepinephrine	290.24 (539.20)	348.21 (550.97)	233.60 (527.71)	0.33
Epinephrine	48.53 (368.36)	97.86 (522.56)	0.35 (2.29)	0.23
Diuretic dose (mg)	183.51 (181.60)	253.77 (224.67)	114.88 (83.23)	<0.001
Urine output (ml/kg/h)	1.50 (0.59)	1.34 (0.48)	1.65 (0.66)	0.01

Data were presented as mean with standard deviation (SD) for continuous variables; counts with percentage for categorical variables.
Abbreviations: AKI, acute kidney injury; BMI, body mass index; CKD, chronic kidney disease; CTP Child-Turcotte-Pugh; MELD, Model for End-Stage Liver Disease; SCr, serum creatinine.

Table 2 Outcomes of study population

	Total (N = 87)	Patients with AKI (N = 43)	Patients without AKI (N = 44)	P-value
Death (N, %)	0 (0.0)	0 (0.0)	0 (0.0)	N/A
LOS (day)	9.46 (90.08)	7.77 (115.08)	11.11 (57.33)	0.86
RRT (N, %)	2 (2.3)	2 (4.8)	0 (0.0)	0.45

Data were presented as mean with standard deviation (SD) for continuous variables; counts with percentage for categorical variables.
Abbreviations: AKI, acute kidney injury; LOS, length of hospital stay; RRT, renal replacement therapy.

Table 3 Univariate analysis for associated factors of AKI

	Crude OR	95% CI	P-value
Preoperative parameters:			
Age (every 10 years increase)	1.06	0.72-1.59	0.70
Female	0.75	0.29-1.85	0.50
BMI (kg/m^2)			
≤20	1	1-1	
20-30	0.45	0.11-1.74	0.20
>30	2.67	0.45-17.66	0.30
Comorbidity			
Diabetes mellitus	1.67	0.71-3.98	0.20
Hypertension	0.55	0.21-1.35	0.20
CKD	2.76	0.56-20.09	0.20
Etiology			
Alcoholic cirrhosis	2.41	0.91-6.75	0.08
Hepatitis B cirrhosis	0.93	0.37-2.28	0.90
Hepatitis C cirrhosis	0.45	0.18-1.11	0.09
Severity			
MELD (every 10 scores increase)	1.91	1.07-3.57	0.03
CTP (every 1 score increase)	1.1	0.86-1.42	0.50
SCr (every 1 mg/dL increase)	1.96	0.50-8.31	0.30
Intraoperative parameters:			
Operative parameters			
Operative time (every 60 min increase)	1.43	1.01-2.12	0.05
Anesthetic time (every 60 min increase)	1.29	0.94-1.82	0.12
Cold ischemic time (every 10 min increase)	0.99	0.77-1.25	0.90
Warm ischemic time (every 10 min increase)	0.99	0.60-1.61	1
Donor age (every 10 years increase)	0.95	0.68-1.30	0.70
Blood loss (every 1000 ml increase)	1.05	0.97-1.17	0.30
Blood product transfusion			
Packed red cells (every 1 unit increase)	1.04	0.97-1.12	0.30
Fresh frozen plasma (every 1 unit increase)	1.03	0.96-1.12	0.40
Platelet concentrate (every 10 units increase)	1.54	0.91-2.74	0.10
Cryoprecipitate (every 10 units increase)	1.24	0.71-2.42	0.50

Table 3 Univariate analysis for associated factors of AKI (cont.)

	Crude OR	95% CI	P-value
Inotrope/vasopressor duration (every 60 min increase)			
Dopamine <5 mcg/kg/min	0.96	0.81-1.14	0.70
Dopamine ≥5 mcg/kg/min	1.11	0.94-1.34	0.20
Norepinephrine <0.5 mcg/kg/min	1.12	0.98-1.29	0.01
Norepinephrine ≥0.5 mcg/kg/min	1	0.69-1.46	1
Fluid administration			
Crystalloid (every 1000 ml increase)	1.06	1-1.16	0.06
Synthetic colloid (every 500 ml increase)	1.04	0.95-1.14	0.36
Natural colloid (every 250 ml increase)	1.01	0.99-1.03	0.35
Urine output (every 0.5 ml/kg/h increase)	0.59	0.37-0.84	<0.01
Postoperative parameters:			
Inotrope/vasopressor duration (every 60 min increase)			
Dopamine	1.08	0.53-1.24	0.02
Norepinephrine	1.02	1.02-1.17	0.30
Furosemide dose (every 100 mg increase)	2.12	1.39-3.59	<0.01
Urine output (every 0.5 ml/kg/h increase)	0.66	0.43-0.95	0.04

Abbreviations: AKI, acute kidney injury; BMI, body mass index; CKD, chronic kidney disease; CTP Child-Turcotte-Pugh; MELD, Model for End-Stage Liver Disease; SCr, serum creatinine.

Table 4 Multivariate analysis for associated factors of AKI

	Crude OR	95% CI	P-value
Intraoperative urine output (every 0.5 ml/kg/h increase)	0.36	0.18-0.65	<0.01
Postoperative dopamine infusion duration (every 60 min increase)	1.09	1.02-1.20	<0.01
Postoperative furosemide dose (every 100 mg increase)	2.87	1.80-5.39	<0.001

Discussion

AKI is a common complication with the incidence of 49.4% in our study, which is comparable to previous studies.[13,16,29] We did not find any difference in preoperative variables between the AKI and non-AKI groups, unlike other studies. Patients with preoperative AKI were excluded from our study, making baseline demographic data including MELD score, CTP score, and pre-transplant SCr more homogeneously balanced. Our study focused on early postoperative AKI within 72 hours. During this time, major factors found to be attributed to the incidence are prerenal causes or ischemic acute tubular necrosis.[2,30] While etiology of late postoperative AKI is more multifactorial.[1] This explains the effect of prolonged duration of dopamine infusion during postoperative period. Patients who need longer duration of dopamine infusion are believed to be more hemodynamically unstable. In previous study, Bilbao et al.[31] found that patients who needed vasoactive agents required more frequent post-liver transplantation RRT. This finding correlates with results from previous studies by Zongyi et al.[21] and Cabezuelo et al.[1] However, both studies indicated that risk of AKI increases when postoperative dopamine use is longer than 6 days. To our knowledge, our study is the first study to find the association between each hour of dopamine infusion and the occurrence of AKI. Our finding is more consistent and practical in current perioperative post-liver transplant care. With advancement in anesthesia and postoperative practice, patients are unlikely to need intensive care stay longer than 3 days in our institution. Thus, making duration dopamine

usage rarely exceeds 6 days. Intraoperative urine output is a protective factor from our finding. The reduction in urine flow rate associates with the increased likelihood of the development of AKI, because it is directly related to the urine output criteria for the diagnosis. We found that higher dose of furosemide is independently associated with AKI. This may be both the cause and effect. Furosemide may be the result of oliguria management in patients with early AKI. On the other hand, the use of furosemide may convert oliguric to non-oliguric AKI[32] which causes harmful oxidative stress to the kidneys[33] and causes renal toxicity.[34] It is also possible that furosemide can aggravate severe oxidative stress secondary to ischemic reperfusion injury.[35]

Our study has several limitations. Since our study is a retrospective study, we were challenged with the incompleteness of the medical records. Secondly, our study has a small number of sample population. The sample size was calculated to identify the incidence of AKI in post-liver transplant patients. Thus, we can only find associations between the studied factors and the occurrence of AKI. Further study is needed with larger sample size to obtain the risk factors of AKI to be more of clinical usefulness. Lastly, our anesthetic records were manually documented without uniform hemodynamic monitoring. We were unable to obtain data of hemodynamic parameters which might be of clinical significance regarding the aforementioned etiology of early postoperative AKI.

The success in this study demonstrated the incidence of early postoperative AKI post-orthotopic liver transplantation surgery which is the primary outcome of the study. However, according to the small sample size and limitation in data collection, for example, unstable hemodynamic profiles which are probably important variables, or long-term outcomes which are related to early postoperative AKI and may be of clinical significance to implication in clinical practice.

Conclusion

The incidence of AKI following orthotopic liver transplantation was 49.4%. Decreased intraoperative urine output, prolonged duration of postoperative dopamine infusion, and higher dose of postoperative diuretics were independently associated with the development of AKI. AKI was not associated with prolonged hospitalization.

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