

Research Article

Retrospective Evaluation of the Predictive Factors of Acute Kidney Injury after Liver Resection

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Abstract

Introduction: The incidence and risk factors of Postoperative Acute Kidney Injury (PO-AKI) after liver surgery are not well established. The purpose of the present study was to evaluate the incidence and perioperative risk factors of PO-AKI in patients undergoing liver resection.

Methods: We conducted a retrospective study on consecutive patients undergoing any type of liver resection between November 1, 2008 and May 31, 2012 at Nippon Medical School Hospital. Electronic medical records were abstracted for comorbid conditions and intraoperative predictors with the potential for association with PO-AKI. The development of AKI was defined according to the Kidney Disease: Improving Global Outcomes criteria definition.

Results: The study included 199 of 204 patients who underwent liver resection during the study period. The other five cases were excluded because they underwent renal replacement therapy before surgery. The AKI rate was 23.6% (47 of 199 patients), four of whom (8.5% of AKI patients) required renal replacement therapy. Multivariate analysis identified BMI (odds ratio: 1.20, 95% CI: 1.07-1.36, P=0.003), CKD (odds ratio: 3.31, 95% CI: 1.32-8.46, P=0.011), platelet count (odds ratio: 0.94, 95% CI: 0.89-0.99, P=0.022) and albumin (odds ratio: 0.32, 95% CI: 0.14-0.70, P=0.004) as independent predictors of PO-AKI, but not intraoperative data such as water balance and urine output.

Conclusion: This study suggests the importance of assessing patient characteristics preoperatively. Appreciation of risk factors for PO-AKI may help identify high-risk surgical populations.

ABBREVIATIONS

PO-AKI: Postoperative Acute Kidney Injury; RIFLE: the Risk, Injury, Failure, Loss, Endstage kidney disease; AKIN: the Acute Kidney Injury Network; sCr: Serum Creatinine; BMI: Body Mass Index; COPD: Chronic Obstructive Pulmonary Disease; CKD: Chronic Kidney Disease; KDIGO: the Kidney Disease: Improving Global Outcomes

INTRODUCTION

In recent years, Acute Kidney Injury (AKI) has gained attention as a significant clinical problem. In 2004, the Acute Dialysis Quality Initiative consensus group proposed the definition for AKI (the Risk, Injury, Failure, Loss, End-stage kidney disease, or RIFLE criteria) [1]; 3 years later, the Acute Kidney Injury Network (AKIN) Staging system was advocated [2]. Several recent adult studies revealed AKI as an independent

risk factor for poor outcome following cardiac surgery [3,4], and not cardiac surgery [5,6], despite which there are still no specific treatments for AKI. The 2005 American Society of Nephrology Renal Research Report listed early AKI diagnosis and treatment as a top priority research area [7]. They revealed that treatment of AKI must occur very early after the inciting injury in order to be effective. Despite the recognition of renal failure as a serious complication of hepatic surgery, the incidence and risk factors of Postoperative Acute Renal Injury (PO-AKI) after liver surgery are not well established [8-10]. The purpose of the present study was to evaluate the incidence and perioperative risk factors of PO-AKI in patients undergoing surgical resection of the liver.

MATERIALS AND METHODS

Study and setting

We conducted a retrospective study that included consecutive

patients undergoing any type of liver resection between November 1, 2008 and May 31, 2012 at Nippon Medical School Hospital. The ethical committee at our hospital has established that retrospective studies involving analysis of medical records can be conducted without authorization from the committee.

Patients and methods

We obtained information about preoperative Serum Creatinine (sCr) concentrations (baseline values obtained within a month preceding surgery) from electronic medical records. The patients' charts were also reviewed for data during the first postoperative week. Data were abstracted for urine output and postoperative sCr concentrations (on postoperative days 0, 1, 3, 5 and 7). Of note, all pre- and postoperative sCr measurements were performed at the same biochemistry laboratory. All patients without baseline and postoperative sCr data, or those on dialysis therapy before surgery, were excluded. Electronic medical records were also abstracted for comorbid conditions with the potential for association with PO-AKI, including age, gender, Body Mass Index (BMI), presence of cardiovascular disease, medically-treated hypertension, Chronic Obstructive Pulmonary Disease (COPD), medically treated diabetes, preoperative Chronic Kidney Disease (CKD), cerebral infarction, Charlson index [11], underlying liver diseases, and preoperative laboratory data. Cardiovascular disease was defined by the presence of coronary artery disease (myocardial infarction, coronary stent placement, or cardiac bypass surgery) or congestive heart failure/cardiomyopathy (or ejection fraction <40 %), while respiratory disease was defined as moderate to severe chronic obstructive pulmonary disease. Additionally, CKD was defined as an estimated glomerular filtration rate of 60 mL/min/1.73 m². We also considered the following intraoperative predictors: surgery time, anesthesia time, major/minor liver resection, Pringle time (temporary total liver inflow obstruction time), and water balance.

The diagnosis of PO-AKI was made by comparing baseline and highest postoperative sCr concentrations, to determine the presence of significant predefined changes. The development of AKI was determined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria definition as an increase in sCr level by 0.3 mg/dL within 48 hours, or greater than 1.5 times baseline within 1 week, together with a urine volume <0.5 ml/kg/h for 6 hours [12].

Anesthesia for liver resection in all the patients consisted of general anesthesia using sevoflurane, with or without epidural anesthesia, at the anesthesiologist's discretion. Part of the perioperative analgesic regimen relied on 0.2% ropivacaine with fentanyl via the epidural catheter, or continuous intravenous injection of fentanyl, which was controlled by the attending anesthesiologist.

Statistical analysis

Clinical data were prospectively recorded and tabulated with Excel software (Microsoft Corp, Redmond, WA, USA). Continuous variables were compared between groups using the two-tailed unpaired Student t-test. In case of dichotomous variables, group differences were examined by Fisher's exact tests, as appropriate. Multiple logistic regressions were used to identify the risk factors associated with PO-AKI on multivariate analyses. The model was

built using variables that demonstrated a value of $P < 0.25$ on univariate analysis. Significance within the model was evaluated by the likelihood ratio test, and the strength of the association of variables with PO-AKI was estimated by calculating the Odds Ratio (OR) and 95% Confidence Interval (CI). Results are presented as mean values \pm Standard Deviation (SD). Statistical analysis was performed with JMP version 11 software (SAS Institute, Inc, Cary, NC, USA).

RESULTS

Of the 204 patients who underwent liver resection during the study period, 199 patients, comprising 141 men (70.9%), were included in the study. The remaining five cases were excluded because they underwent renal replacement therapy before surgery. The AKI rate was 23.6% (47 of 199 patients), four of whom (8.5% of AKI patients) required renal replacement therapy. The details of PO-AKI are shown in table 1. Patients were comparable in terms of demographic data, comorbidities (Table 2) and intraoperative data (Table 3). Mean age was 69.0 ± 10.1 years, 17.1% had preexisting CKD, and 96.5% suffered from malignant liver disease. Table 2 shows that patients with AKI included a higher proportion of men (85.1% vs. 66.4%, $P=0.016$), who were almost 4 years older (mean age: 72.1 ± 8.7 vs. 68.1 ± 10.4 , $P=0.016$) and had a higher BMI (24.7 ± 3.2 vs. 22.2 ± 3.4 , $P<0.001$) than patients without AKI. Patients with AKI suffered more often from CKD (31.9% vs. 12.5%, $P=0.004$). Preoperative platelet and albumin levels were lower in patients with AKI (15.9 ± 6.0 vs. 19.1 ± 8.6 , $P=0.021$), (3.5 ± 0.5 vs. 3.8 ± 0.5 , $P=0.006$). However, there were no significant differences in underlying liver diseases. In the intraoperative parameters, both water balance and urine output were lower (6.24 ± 3.12 ml/kg/hr vs. 7.50 ± 3.46 ml/kg/hr, $P=0.03$), (1.10 ± 0.67 ml/kg/hr vs. 1.56 ± 1.09 ml/kg/hr, $P=0.007$) in the AKI group compared with the non-AKI group. Although blood loss was not significantly different between the two groups, it tended to be higher in the AKI group. There were no differences in surgical data and anesthesia time between the two groups.

Multivariate analysis identified BMI (odds ratio: 1.20, 95%

Table 1: Details of postoperative acute kidney injury.

The incidence of PO-AKI	
AKI, number (%)	47 (23.6%)
Renal replacement therapy	4 (8.5)
Onset of AKI	
POD 0	20 (42.6%)
POD 1	15 (31.9%)
POD 2	3 (6.4%)
POD 3	3 (6.4%)
POD 5	2 (4.2%)
POD 7	4 (8.5%)
Stage of AKI	
1	25 (53.2%)
2	17 (36.2%)
3	5 (10.6%)

Abbreviations: PO-AKI: Postoperative Acute Kidney Injury; AKI: Acute Kidney Injury; POD: Postoperative Day

Table 2: Preoperative patient characteristics.

	All Patients	PO-AKI	Non PO-AKI	P value
	n = 199	n = 47	n = 152	
Age, mean ± SD	69.0±10.1	72.1±8.7	68.1±10.4	0.016
Gender, male/female, number (%)	141/58 (70.9%/29.1%)	40/7 (85.1%/14.9%)	101/51 (66.4%/33.6%)	0.016
Body mass index (kg/m ²), mean ± SD	22.8±3.47	24.7±3.2	22.2±3.4	<0.001
Hypertension, number (%)	101 (50.8%)	26 (55.3%)	75 (49.3%)	0.508
Cardiovascular disease, number (%)	12 (6.0%)	5 (10.6%)	7 (4.7%)	0.195
Chronic kidney disease, number (%)	34 (17.1%)	15 (31.9%)	19 (12.5%)	0.004
COPD, number (%)	19 (9.5%)	2 (4.3%)	17 (11.2%)	0.254
Diabetes, number (%)	50 (25.1%)	15 (31.9%)	35 (23.0%)	0.249
Cerebral infarction, number (%)	16 (8.0%)	5 (10.6%)	11 (7.2%)	0.5387
Charlson-index, mean ± SD	5.7±2.7	5.9±2.7	5.6±2.8	0.524
Viral hepatitis, number (%)	81 (40.7%)	22 (46.8%)	59 (38.8%)	0.396
Cirrhosis, number (%)	60 (30.2%)	18 (38.3%)	42 (27.6%)	0.203
Steatosis, number (%)	5 (2.5%)	1 (2.1%)	4 (2.6%)	1
Fibrosis, number (%)	32 (16.1%)	11 (23.4%)	21 (13.8%)	0.171
Benign/malignant disease, number (%)	7/192	0/47 (0%/100%)	7/145	0.202
Primary/secondary liver tumor, number (%)	148/51	38/9 (80.9%/19.1%)	110/42 (72.4%/27.6%)	0.339
AST (U/L), mean ± SD	40.0±34.7	35.8±18.5	41.3±38.3	0.342
ALT (U/L), mean ± SD	36.0±38.5	31.5±24.0	37.4±41.9	0.359
Bilirubin (μmol/L), mean ± SD	0.86±0.65	0.81±0.58	0.88±0.68	0.54
Hemoglobin (g/dl), mean ± SD	12.4±1.9	12.1±2.1	12.5±1.8	0.139
Platelet (×10 ⁴ /μl), mean ± SD	18.3±8.3	15.9±6.0	19.1±8.6	0.021
Albumin (g/dl), mean ± SD	3.8±0.5	3.5±0.5	3.8±0.5	0.006
Prothrombin (%), mean ± SD	88.9±16.1	90.0±15.4	88.7±16.4	0.517

Abbreviations: PO-AKI: Postoperative Acute Kidney Injury; SD: Standard Deviation; COPD: Chronic Obstructive Pulmonary Disease; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase

CI: 1.07-1.36, P=0.003), CKD (odds ratio: 3.31, 95% CI: 1.32-8.46, P=0.011), platelet count (odds ratio: 0.94, 95% CI: 0.89-0.99, P=0.022) and albumin level (odds ratio: 0.32, 95% CI: 0.14-0.70, P=0.004) (Table 4) as independent predictors of PO-AKI, but not intraoperative data, such as water balance and urine output.

DISCUSSION

PO-AKI is a major complication after abdominal surgery, associated with substantial mortality, morbidity and costs [5,13,14]. Although no specific treatments exist to modify the clinical course of PO-AKI once it is established, early identification of patients at risk of PO-AKI would help to allocate appropriate resources to high-risk patients, and to initiate appropriate clinical management strategies to prevent PO-AKI.

Despite recognition of PO-AKI as a serious complication of hepatic surgery, the incidence and risk factors of PO-AKI after liver surgery are not well established [9,10]. Slankamenac [9] and Edwards [15] reported a 15.1% and 13% incidence of renal compromise after hepatic surgery, respectively. The incidence of PO-AKI in our study was 23.6%, which was different from the incidences reported in these studies. Comparison of PO-AKI across different studies is difficult due to lack of a standardized definition for AKI. There are certain differences between the RIFLE criteria and the more recent AKIN definition, with a

reportedly higher detectability of AKI by the AKIN definition than the RIFLE criteria [16]. Therefore, new KDIGO criteria, based on new evidence, were adopted for determining PO-AKI in this report. The observation period in this study was 7 days postoperatively, and the number of PO-AKI cases in the late phase, after 48 hours postoperatively, was 9 (19.1% of PO-AKI) in this study. This is a rare study using KDIGO criteria for PO-AKI, which revealed that 7 days after operation is an appropriate observation period for PO-AKI after liver resection.

One of the most important features of our study is the accurate evaluation of AKI risk factors using the KDIGO criteria. Only one other study has examined the risk factors associated with PO-AKI following liver resection. In that study, however, PO-AKI was defined according to the RIFLE criteria [9]. In multivariate analysis, four variables were identified as risk factors for PO-AKI after liver resection, namely BMI, preoperative CKD, platelet count and serum albumin. Preoperative CKD is not a surprising result. Pisimisis et al. [17] revealed that a preoperatively depressed estimated GFR (eGFR) correlated with deterioration of renal function in the postoperative period. Therefore, in view of the role of CKD (basal eGFR) in PO-AKI development, renal function should be assessed preoperatively.

Hypoalbuminemia has been shown to be an important risk factor for AKI following various surgeries [18-21]. In addition,

Table 3: Intraoperative patient characteristics.

	All Patients	PO-AKI	Non PO-AKI	P value
	n = 199	n = 47	n = 152	
Surgery time (min), mean ± SD	352.1±139.7	328.9±136.9	359.4±140.1	0.192
Anesthesia time (min), mean ± SD	448.9±149.1	433.0±156.2	453.8±147.1	0.406
Major/minor liver resection, number (%)	61/138 (30.7%/69.3%)	11/36 (23.4%/76.6%)	50/102 (32.9%/67.1%)	0.278
Pringle method, number (%)	179 (89.9%)	43 (91.4%)	136 (89.5%)	
Pringle time (min), mean ± SD	57.5±36.3	53.4±31.3	58.7±37.7	0.388
Water balance (ml/kg/hr), mean ± SD	7.15±3.52	6.24±3.12	7.50±3.46	0.03
Blood loss (ml/kg/hr), mean ± SD	2.06±2.24	2.31±2.97	1.99±1.97	0.383
Urine volume (ml/kg/hr), mean ± SD	1.50±1.21	1.10±0.67	1.56±1.09	0.007
Red blood cell transfusion (unit), median (range)	2 (0-40)	2 (0-40)	2 (0-34)	0.715

Abbreviations: PO-AKI: Postoperative Acute Kidney Injury; SD: Standard Deviation

Table 4: Risk factors on multiple logistic regression.

	Odds ratio	95% confidence interval	P value
Age	1.04	0.99-1.09	0.118
Gender	2.59	0.96-7.81	0.059
Body mass index	1.20	1.07-1.36	0.003
Chronic kidney disease	3.31	1.32-8.46	0.011
Platelet	0.94	0.89-0.99	0.022
Albumin	0.32	0.14-0.70	0.004
Water balance	0.92	0.78-1.05	0.240
Urine volume	0.81	0.47-1.31	0.405

a recent meta-analysis found that hypoalbuminemia was not only an independent risk factor for AKI, but was associated with an increased risk of death following the development of AKI [22]. Despite the strong link between hypoalbuminemia and AKI, few studies have assessed the impact of preoperative hypoalbuminemia on PO-AKI in patients undergoing liver resection. Hypoalbuminemia may be causally linked to the development of postoperative AKI. Several studies suggest that serum albumin may have renoprotective effects at both the cellular and molecular level. Albumin has been reported to improve renal perfusion and glomerular filtration through prolonged potent renal vasodilation, which is caused by a reaction of serum albumin with oxides of nitrogen to form S-nitroso-albumin [23]. Another study found that albumin inhibited apoptosis of renal tubular cells through its capacity to scavenge reactive oxygen species and carry protective lysophosphatidic acid [24]. Furthermore, albumin stimulates the proliferation of renal tubular cells through the activation of phosphatidylinositide 3-kinase [25]. This observation indicates that albumin plays a crucial role in the maintenance of tubular integrity and function. Thus, a reduced serum albumin level may contribute to an increased risk of PO-AKI in patients undergoing liver resection.

Our multivariate analysis suggests that increasing BMI is a risk factor for AKI, independent of other risk factors. A large study in over 300,000 patients undergoing noncardiac surgery found that the risk of postoperative AKI was increased by three- to sevenfold in obese patient when compared to normal weight patients [26]. Further, this risk increased with BMI [26]. Volume resuscitation in obese patients is often more challenging than in

non-obese patients. Unfortunately, although under-resuscitation can result in hypotension and oliguria, volume overload can lead to cardiopulmonary and wound complications, which are also more common in obese patients.

The mechanisms of PO-AKI in patients undergoing major surgery are complex and multifactorial, including hemodynamic factors such as hypotension leading to ischemia, inflammatory factors, and nephrotoxin exposure from medications [27]. AKI following liver resection can occur secondary to development of hepatorenal syndrome. Hepatic vascular isolation during the surgery typically causes a decrease in venous return with a resulting decrease in cardiac output and an increase in systemic vascular resistance. The anesthetic management for maintenance of renal blood flow during this period is complex, and many patients require arterial lines and central venous catheters during the operation. MAP is maintained by infusing fluids and vasopressors under continuous hemodynamic monitoring. Low central venous pressure (less than or equal to 5 cmH₂O) anesthesia is a safe technique that facilitates operative control of hemorrhage, in particular from the major hepatic veins, and contributes to a lower incidence of PO-AKI [11]. In this report, although hypovolemia (low water balance, low fluid volume and high blood loss) was not significant, it tended to cause PO-AKI.

LIMITATIONS

Several limitations inherent to our study warrant careful consideration. First, our findings are based on a retrospective evaluation and reflect the practice at a single center with a relatively homogenous surgical population, which limits its wide

generalizability to other populations. Secondly, the study is also statistically limited because of the small number of patients enrolled.

CONCLUSION

The rate of PO-AKI following liver resection is approximately 23.6%. This study suggests the importance of preoperative assessment of patient characteristics. Appreciation of the risk factors for PO-AKI may help to identify high-risk surgical populations. Strategies to preserve renal function have the potential to improve the prognosis in these patients. These high-risk patients may be ideal candidates for recruitment into clinical trials on renal-protective therapies. To reduce the risk of PO-AKI and to achieve optimal treatment in the event of occurrence of PO-AKI, further studies with a larger patient population need to be performed to identify the risk factors for the development of PO-AKI.

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