ORIGINAL ARTICLE

# Acute kidney injury after liver surgery: does postoperative urine output correlate with postoperative serum creatinine?

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## Abstract

**Background:** Acute kidney injury (AKI) after hepatectomy occurs in around 10% of cases. AKI is often defined based only on postoperative serum creatinine increase. This study aimed to assess if postoperative urine output (UO) correlated with serum creatinine after hepatectomy.

**Methods:** All consecutive hepatectomy patients (2010–2016) were assessed. AKI was defined according to KDIGO criteria: serum creatinine increase $\geq$26.5 μmol/l, creatinine increase $\geq$1.5x baseline creatinine, or postoperative oliguria. Oliguria was defined as daily mean UO <0.5 mL/kg/h. AKI was subdivided into creatinine-based or oliguria-based AKI according to the defining criterion.

**Results:** Out of 285 patients, AKI was observed in 79 cases (28%). Creatinine-based AKI occurred in 25 patients (9%) and oliguria-based only AKI in 54 patients (19%). Ten patients fulfilled both criteria (4%). Postoperative UO correlated poorly with postoperative serum creatinine level in both whole cohort (rho = –0.34, p <0.001) and AKI subgroup (rho = –0.189, p = 0.124). No association was found between postoperative oliguria and postoperative serum creatinine increase (HR = 0.5, 95%CI: 0.2–1.9, p = 0.341). On multivariable analysis, operation duration >360 minutes was the only predictor of creatinine increase (HR = 3.6, 95%CI: 1.1–11.4, p = 0.032).

**Conclusion:** Postoperative UO showed poor correlation with postoperative serum creatinine both in all patients and AKI patients. Surgery duration >360 minutes appeared as the only independent predictor of postoperative serum creatinine increase.

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## Introduction

Liver surgery is associated with substantial postoperative morbidity even though recent advances in anesthesia and surgical techniques have been made.[1] Postoperative overall complications after hepatectomy range from 30% to 60% depending on the extent of liver resection.[2–4]

Acute kidney injury (AKI) represents a known and frequent complication after liver surgery.[4] Incidence of AKI after hepatectomy varies considerably in the literature because of the use of different definitions (KDIGO, AKIN, Club ascites).[5] Based only on the serum creatinine increase, AKI occurs in 5–15% of the patients.[6–8] Laparoscopy has been associated in some articles with reduced postoperative AKI rate in comparison with open liver surgery.[9] Different perioperative risk factors for AKI after hepatectomy have been identified such as epidural analgesia, high MELD (model of end-stage liver disease) score, chronic kidney disease, preoperative biliary obstruction, hemodynamic instability, bleeding, or sepsis.[6,10,11]

Urine output (UO) has always been an important clinical postoperative measure after liver surgery to anticipate potential renal complications such as hepatorenal syndromes and to assess the intravascular volume requirements.[12,13] However, several

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recent reports showed that UO was not a reliable parameter for volume assessment and AKI during postoperative phase.[14] Moreover, data comparing serum creatinine level increase and UO as criteria to define post-hepatectomy AKI remain scarce.

The aim of the present study was to assess the degree of correlation between UO and serum creatinine level after hepatectomy.

## Methods

### Patients

All consecutive patients who underwent liver surgery in the department of visceral surgery of the Lausanne University Hospital (CHUV) from June 2010 to December 2016 were retrospectively included. Operations were anatomical resections, wedge resections, and pericystectomies. Minor hepatectomies were defined as resection of <3 Couinaud segments and major as resection of ≥3 segments. From July 2013, all patients followed an Enhanced Recovery After Surgery (ERAS) program.[15] Preoperative fitness was graded according to the American Society of Anesthesiologists (ASA) classification. In case of future liver remnant judged too small (<30% of total liver volume for normal liver, <40% for cirrhotic or post-chemotherapy liver), a preoperative portal vein embolization was performed.

### Acute kidney injury

AKI was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria.[16] The criteria to define AKI were as follows: serum creatinine increase ≥26.5 μmol/L, serum creatinine increase ≥1.5x baseline creatinine, or postoperative oliguria for at least 6 hours. Regarding the 26.5 μmol/L serum creatinine increase, the change should have occurred over a 48-hour period. Postoperative oliguria was defined as daily mean UO <0.5 mL/kg/h. Daily UO for each patient were extracted by 3 investigators from electronic patient charts (scanned copies of the nurse notes). During the postoperative period after hepatectomy, nurses measured and collected UO every 4 hours. Therefore UO was calculated for a 24-hour period and then mean hourly UO divided by patient weight was calculated in mL/kg/h. Time frame for postoperative AKI was the first postoperative week.

### Collected data and outcomes

Diagnosis of liver cirrhosis was based on imaging (ultrasound, CT or MRI), ultrasound transient elastography (values >12.5 kPa), presence of portal hypertension (measured hepatic venous pressure gradient >10 mmHg), or liver biopsy (percutaneous or transjugular). Diabetes mellitus diagnostic criteria were as follows: hemoglobin A1C ≥6.5%, fasting plasma glucose level ≥7 mmol/l, 2-hour plasma glucose ≥11.1 mmol/l during an oral glucose tolerance test, or symptoms of hyperglycemia and a random plasma glucose ≥11.1 mmol/l.

Postoperative complications were defined according to Clavien classification.[17] Minor complications were graded as I-II and major as III-IV. Postoperative mortality (grade V) was defined as death during the hospital stay or during the 30 postoperative days. Comprehensive complication index (CCI) was used to quantify all complications that individual patients developed.[18] Readmissions were defined as rehospitalization during the 90 postoperative days.

### Statistics

Categorical data were compared using a chi-square test. Continuous variables were analyzed using a Mann–Whitney *U* or a Kruskal–Wallis test. Correlations were made using the Spearman coefficient as distributions were non-Gaussian. A fitted line was calculated using a quadratic regression model. To find independent risk factors for creatinine increase (or factors susceptible to trigger creatinine increase) and for 30-day mortality a multivariable logistic regression was performed. All items with p-value <0.1 on univariable analysis were included in the multivariable analysis. All statistical analyses were performed using SPSS 24 for Mac OS X. This study was approved by the local Ethics Committee (number 2017-01169). This study was registered on Research Registry (UIN: researchregistry3606) and followed the STROBE guidelines.

## Results

### Patients

Overall 306 patients were eligible for inclusion, but 21 patients refused to consent. Therefore 285 patients were included for analysis. Among these 285 patients, 79 presented AKI based on the KDIGO criteria (28%). Creatinine increase AKI occurred in 25 patients (9%) and oliguria only AKI in 54 patients (19%). Ten patients (4%) had both creatinine increase AKI and oliguria AKI.

Missing data for serum creatinine were 3 for preoperative values (1%), 17 for postoperative day (POD) 1 values (6%), 13 for POD 2 values (5%), and 23 for POD 3 values (8%). Regarding UO, 37 values on POD 1 (13%) and 23 values on POD 2 (8%) were missing. These missing data were therefore excluded from the analyses using creatinine levels and UO.

Among the 97 non-ERAS patients, 24 (25%) developed AKI and 18 (19%) developed postoperative oliguria. The 188 ERAS patients showed similar outcomes as non-ERAS patients with 55 patients who developed AKI (29%, p = 0.420) and 46 oliguria (24%, p = 0.257).

### Correlations between creatinine levels and urine outputs

Postoperative mean daily UO of all patients (median 0.9 mL/kg/h, 0.6–1.4) on POD 1 correlated poorly with postoperative serum creatinine on POD 2 (median 66 μg/L, 54–82, rho = −0.34, p < 0.001). Same results were found for mean UO on POD 2 (median 1 mL/kg/h, 1–1.5) and creatinine on POD 3

(median 63 μg/L, 52–78, rho = −0.2, p = 0.006). Fig. 1a,b shows these correlations graphically. The Spearman coefficient of correlation between the difference of postoperative serum creatinine on POD 2 and preoperative creatinine and UO on POD 1 was −0.289 (p <0.001).

Among AKI patients, the correlation coefficient between serum creatinine levels on POD 2 and mean UO on POD 1 was low (rho = −0.189, p = 0.124, Fig. 1c). A similarly low correlation coefficient was found between creatinine levels on POD 3 and mean UO on POD 2 (rho = 0.222, p = 0.069, Fig. 1d). The correlation coefficient between the difference of postoperative creatinine on POD 3 and preoperative value and mean UO on POD 2 for AKI patients was 0.164 (p = 0.186).

### Patients with acute kidney injury

Demographics and preoperative characteristics of patients without AKI and with AKI (subdivided into creatinine increase AKI and oliguria only AKI) are shown in Table 1. Intraoperative and postoperative results are summarized in Table 2. Of note, more men were present in the creatinine increase AKI and oliguria only AKI groups compared to non-AKI patients. Creatinine increase AKI and oliguria AKI patients had longer operative time (p <0.001), more blood loss (p <0.001), and higher UO on POD 1 (p <0.001) than non-AKI patients. Patients with creatinine increase AKI received more IV fluids on POD 1 (p = 0.022), had higher creatinine level on POD 1 (p <0.001), more overall complications (p = 0.003), more major complications (p = 0.044), higher CCI (p = 0.001), and longer length of stay (p = 0.001) compared to oliguria AKI and non-AKI patients.

Of note, patients with AKI had significantly more pedicular clamping, more complications, higher CCI and received more IV fluids on POD 1 than patients without AKI (Supplementary Table 5). Intra- and postoperative outcomes comparing creatinine increase and oliguria AKI patients only are shown in Supplementary Table 6. Oliguria AKI patients had significantly

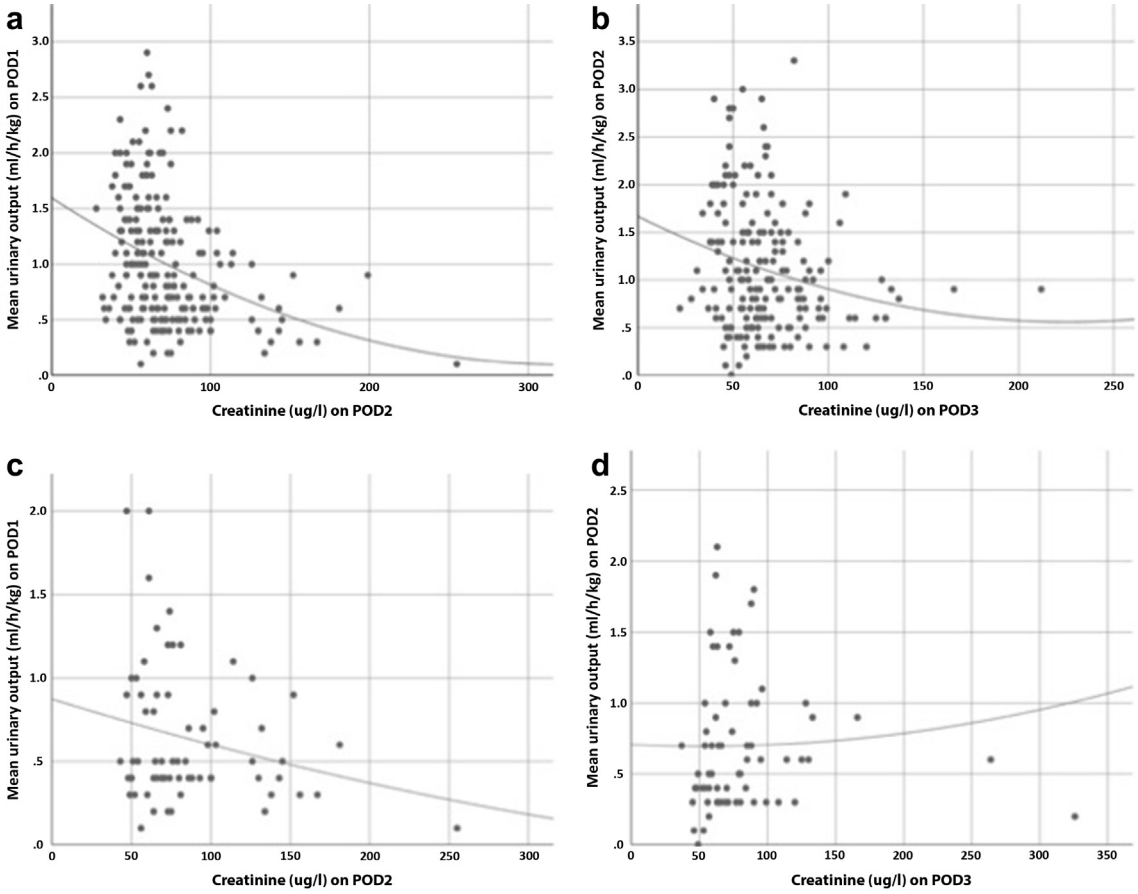


**Figure 1** Scatter plots. **a.** Creatinine levels on POD 2 and mean urine output on POD 1 for the entire cohort (rho = –0.34, p <0.001). The fitted line was calculated using a quadratic regression model ($R^2$ = 12%). **b.** Creatinine levels on POD 3 and mean urine output on POD 2 for the entire cohort (rho = –0.2, p = 0.006). The fitted line was calculated using a quadratic regression model ($R^2$ = 6%). **c.** Creatinine levels on POD 2 and mean urine output on POD 1 for patients with acute kidney injury (n = 79, rho = –0.189, p = 0.124). The fitted line was calculated using a quadratic regression model ($R^2$ = 8%). **d.** Creatinine levels on POD 3 and mean urine output on POD 2 for patients with acute kidney injury (n = 79, rho = 0.222, p = 0.069). The fitted line was calculated using a quadratic regression model ($R^2$ = 3%)

**Table 1** Demographics and preoperative details of operated patients with and without acute kidney injury (AKI). AKI patients are subdivided into AKI based on creatinine increase (Cr-AKI) and oliguria (O-AKI)[a]

| | Patients without AKI (n = 206) | Patients with Cr-AKI (n = 15) | Patients with O-AKI (n = 54) | P-value |
|---|---|---|---|---|
| Age, *years* | 64 (54–69) | 67 (62–73) | 61 (51–69) | 0.200 |
| Men/women | 110 (53%)/96 (47%) | 13 (87%)/2 (13%) | 41 (76%)/13 (24%) | **<0.001** |
| BMI, $kg/m^2$ | 25 (22–28) | 25 (22–28) | 27 (24–31) | 0.077 |
| ASA score, 1/2/3 | 4 (2%)/148 (72%)/54 (26%) | 0/9 (60%)/6 (40%) | 0/40 (74%)/14 (26%) | 0.628 |
| Smokers | 60 (29%) | 2 (13%) | 16 (30%) | 0.413 |
| Cancers | 165 (80%) | 11 (73%) | 45 (83%) | 0.677 |
| Alcohol use | 88 (43%) | 5 (33%) | 23 (43%) | 0.775 |
| Cirrhosis | 18 (9%) | 1 (7%) | 5 (9%) | 0.952 |
| Diabetes mellitus | 38 (18%) | 4 (26%) | 7 (13%) | 0.422 |
| PVE | 42 (20%) | 4 (26%) | 13 (24%) | 0.741 |

BMI: body-mass index; ASA: American Society of Anesthesiologists; PVE: portal vein embolization; AKI: acute kidney injury.
Data are presented as median and interquartile range or number and percentage. Significant p-values appear in bold.
[a] The 10 patients who had both Cr-AKI and O-AKI were excluded.

**Table 2** Intra- and postoperative results of operated patients with and without acute kidney injury (AKI). AKI patients are subdivided into AKI based on creatinine increase (Cr-AKI) and oliguria (O-AKI)[a]

| | Patients without AKI (n = 206) | Patients with Cr-AKI (n = 15) | Patients with O-AKI (n = 54) | P-value |
|---|---|---|---|---|
| Minor/major hepatectomy | 113 (55%)/93 (45%) | 5 (33%)/10 (67%) | 26 (48%)/28 (52%) | 0.215 |
| Laparoscopy | 31 (15%) | 1 (7%) | 8 (15%) | 0.672 |
| Pedicular clamping | 93 (45%) | 9 (60%) | 28 (52%) | 0.406 |
| Operative time, *min* | 279 (193–359) | 346 (220–486) | 319 (241–400) | **<0.001** |
| Blood loss, *mL* | 500 (300–1000) | 1000 (575–1500) | 900 (500–1500) | **<0.001** |
| IV fluids on POD1, *mL* | 1100 (630–1890) | 2130 (1500–2850) | 1140 (630–2095) | **0.022** |
| UO on POD1, *mL/kg/h* | 0.9 (0.6–1.4) | 0.7 (0.6–1) | 0.5 (0.4–0.9) | **<0.001** |
| Creatinine level on POD1, *mg/L* | 72 (57–90) | 110 (91–131) | 69 (59–80) | **<0.001** |
| Complications | 93 (45%) | 13 (87%)[b] | 32 (59%)[b] | **0.003** |
| Minor (I–II) | 62 (30%) | 7 (47%) | 22 (41%) | 0.173 |
| Major (III–V) | 31 (15%) | 6 (40%) | 10 (18%) | **0.044** |
| CCI | 0 (0–22.2) | 22.6 (20.9–47.6) | 20.9 (0–29.5) | **0.001** |
| Readmissions | 10 (5%) | 1 (7%) | 3 (6%) | 0.502 |
| Length of stay, *days* | 9 (7–14) | 18 (12–32) | 9 (7–16) | **0.001** |

IV: intravenous; POD: postoperative day; UO: urine output; CCI: comprehensive complication index; AKI: acute kidney injury.
Data are presented as median and interquartile range or number and percentage. Significant p-values appear in bold.
[a] The 10 patients who had both Cr-AKI and O-AKI were excluded.
[b] Excluding acute kidney injury.

fewer overall complications than creatinine increase AKI patients. Moreover, creatinine increase AKI patients received more IV fluids on POD 1 and had a longer length of stay compared to oliguria AKI patients.

## Risk factors for postoperative serum creatinine increase

Uni- and multivariable analyses are shown in Table 3. On univariable analysis, ASA score, diabetes mellitus, major hepatectomy, operation time, blood transfusion, UO on POD 1 <0.5 mL/kg/h, and UO on POD 1 <0.3 mL/kg/h were associated with serum creatinine increase. On multivariable analysis, only operation time >360 minutes was an independent risk factor for creatinine increase after hepatectomy (HR = 3.6, p = 0.032).

## Risk factors for postoperative 30-day mortality

Table 4 summarized the uni- and multivariable analyses of potential predictors of 30-day mortality after hepatectomy. On multivariable analysis, development of creatinine increase AKI

**Table 3** Uni- and multivariable analyses of potential pre- and intraoperative risk factors for postoperative creatinine increase

| | HR univariable (95% CI) | P-value | HR multivariable (95% CI) | P-value |
|---|---|---|---|---|
| Age >60 | 1.0 (0.9–1.1) | 0.740 | | |
| BMI >25 kg/m$^2$ | 1.0 (1.0–1.2) | 0.357 | | |
| ASA score >2 | 3.2 (1.4–7.4) | **0.006** | 2.1 (0.8–6.1) | 0.153 |
| Malignancy | 1.3 (0.5–3.7) | 0.580 | | |
| Cirrhosis | 1.7 (0.5–6.4) | 0.400 | | |
| Diabetes | 3.1 (1.3–7.6) | **0.013** | 1.8 (0.6–5.3) | 0.312 |
| PVE | 2.2 (0.8–5.6) | 0.109 | | |
| Pedicular clamping | 0.9 (0.4–2.1) | 0.805 | | |
| Laparoscopy | 0.2 (0.1–1.4) | 0.105 | | |
| Major hepatectomy | 2.9 (1.2–6.8) | **0.019** | 1.2 (0.4–4.0) | 0.781 |
| OP time >360 min | 5.5 (2.3–13.2) | <**0.001** | 3.6 (1.1–11.4) | **0.032** |
| Epidural | 1.7 (0.5–6.0) | 0.388 | | |
| Blood transfusion | 2.7 (1.1–6.6) | **0.035** | 1.8 (0.5–6.0) | 0.305 |
| UO on POD 1 <0.5 mL/h/kg | 3.7 (1.4–9.4) | **0.006** | 0.5 (0.2–1.9) | 0.341 |
| UO on POD 1 <0.3 mL/h/kg | 9.5 (2.3–38.7) | **0.002** | 3.7 (0.5–28.7) | 0.203 |

BMI: body-mass index; ASA: American Society of Anesthesiologists; PVE: portal vein embolization; OP: operation; UO: urine output; POD: postoperative day; HR: hazard ratio; CI: confidence interval.
Significant p-values appear in bold.

was found as an independent predictor of 30-day mortality after hepatectomy (HR = 13.3, p = 0.044).

## Discussion

The main results of the present study showed that postoperative UO correlated poorly with serum creatinine after hepatectomy. Furthermore, postoperative oliguria correlated weakly with postoperative serum creatinine increase. Moreover, oliguria was not found as a trigger or predictor of postoperative serum creatinine increase on multivariable analysis. The only independent factor associated with postoperative serum creatinine increase was an operation time >6 hours, and development of creatinine increase AKI was independently associated with 30-day postoperative mortality.

The correlations between mean daily postoperative UO and postoperative serum creatinine values were weak during the first postoperative days in all patients and in AKI patients. This suggests that UO during the first postoperative days after liver resection does not reflect precisely the filtration function of the kidneys. A transient oliguria might happen postoperatively without impeding the kidney function. It is possible that low UO

**Table 4** Uni- and multivariable analyses of potential predictors of 30-day mortality

| | HR univariable (95% CI) | P-value | HR multivariable (95% CI) | P-value |
|---|---|---|---|---|
| Age >60 | 1.8 (0.2–17.6) | 0.609 | | |
| ASA score >2 | 5.9 (0.5–65.6) | 0.152 | | |
| Cirrhosis | 6.0 (0.5–68.7) | 0.151 | | |
| Major hepatectomy | 5.0 (0.6–45.6) | 0.151 | | |
| OP time >360 min | 0.2 (0–1.7) | 0.123 | | |
| Blood transfusion | 4.5 (0.6–32.3) | 0.140 | | |
| Overall complication | 6.4 (0–12.9) | 0.996 | | |
| Major complication | 7.8 (0.3–22.5) | 0.994 | | |
| Presence of AKI | 10.9 (1.2–99.4) | **0.034** | 1.1 (0.1–48.4) | 0.959 |
| Presence of Cr-AKI | 17.1 (2.7–108.3) | **0.002** | 13.3 (1.1–163.5) | **0.044** |
| Presence of O-AKI | 5.4 (0.9–33.0) | 0.069 | 3.5 (0.3–45.0) | 0.336 |

ASA: American Society of Anesthesiologists; OP: operation; AKI: acute kidney injury; Cr-AKI: creatinine increase acute kidney injury; O-AKI: oliguria acute kidney injury; HR: hazard ratio; CI: confidence interval.
Significant p-values appear in bold.

might be a physiological response to hepatic surgery. Explanations for UO decrease or transient oliguria could be related to epidural analgesia and its vasodilatation effects, intraoperative fluid loss, urinary retention, temporary hypotension, or transitory kidney ischemia.[19] In their retrospective study on hepatectomy for hepatocellular carcinoma, Lim *et al.* found that oliguria was not a risk factor for AKI after hepatectomy.[11] Similar results were found in pancreatic surgery by Goren *et al.*[14] They showed that oliguria was not a predictive factor of AKI.[14] It is important to mention that UO was measured in the present study based on the daily volume and adjusted to the patient weight. Short duration oliguria (e.g., during 4–6 hours) might therefore not drastically influence the postoperative outcomes and might be tolerated in clinical practice early after hepatectomy. Response to oliguria with aggressive IV fluid therapy should not be the rule if no other reason imposes IV fluid replacement (e.g., hypotension, hypovolemia, dehydration).[12] In addition, these weak correlations between UO and serum creatinine were measured in different postoperative days without relevant changes (Fig. 1a,b). Bressan *et al.* found that UO overestimated the proportion of AKI after liver surgery, corroborating our results.[12] They concluded that definition of AKI should be revised in order not to take into account oliguria as a definition criterion.[12]

The only risk factor for postoperative serum creatinine increase was an operation time >360 minutes. Lim *et al.* found in a retrospective study of 457 consecutive patients that an operation time >300 minutes was an independent predictive factor of AKI, as well as age, MELD score, and extent of liver resection.[11] Moreover, a long operation time was found in several studies in liver surgery as risk factor for postoperative complication.[3,11] Tzeng *et al.* in a large registry study found that an operation time >240 minutes was a risk factor for severe complications and mortality in elderly patients.[2] A long operation leads to higher insensitive fluid losses and higher IV fluid infusion. Moreover, transient intraoperative hypotension or need for vasopressors may disturb glomerular perfusion through vasoconstriction of the afferent renal artery.[12]

Patients with AKI had on univariable analysis more complications compared to patients without AKI. The same results were found for creatinine-based AKI patients compared to oliguria-based AKI patients (higher overall complication rate). Moreover, patients with creatinine increase had longer length of stay compared to patients with oliguria. AKI was shown in several articles to be associated with longer length of stay[12] or increased morbidity.[20] The prolonged length of stay induced by AKI is usually due to the necessity of intensive care unit management, additional exams, and/or specific treatment.[20]

The threshold of oliguria is usually set at 0.5 mL/h/kg.[16] This threshold might be different in liver surgery. In the present study, UO <0.5 mL/h/kg was not a risk factor for creatinine increase. Furthermore, oliguria was not associated with higher postoperative complications unlike patients with creatinine increase. These findings speak in favor of defining postoperative AKI in liver surgery based on creatinine increase only and not on UO <0.5 mL/h/kg. Bressan *et al.* from Calgary University also suggested to define AKI after liver surgery based on creatinine and not on oliguria.[12] Interestingly, a study from Kyoto University Hospital showed that intraoperative UO <0.3 mL/h/kg was associated with postoperative AKI after major abdominal surgery (including liver surgery).[21] Even though intraoperative UO is related to several specific factors that need to be taken into account (e.g., anesthesia type, vasopressor use, low central venous pressure during resection), this threshold for oliguria might remain relevant during the postoperative period and should be further evaluated. Of note, in the present cohort, postoperative UO <0.3 mL/h/kg was not predictive of postoperative creatinine increase on multivariable analysis (HR = 3.7, p = 0.203). Intraoperative UO were not available in the present study.

Serum creatinine level not only reflects the kidney function, but is also influenced by the skeletal muscle mass of patients.[22] To level this inter-patient variation, difference between preoperative and postoperative serum creatinine was correlated to postoperative UO. It is important to mention that correlation coefficients remained low, confirming that patient muscle mass did not influence the relationship between serum creatinine and postoperative UO.

The main strength of this study lies on the systematic measure and collection of UO during the first postoperative days, which is rarely done. These important data were and are routinely collected in our division thanks to the implementation of ERAS program in liver surgery. However, this study has several limitations that need to be addressed. First, the retrospective design of the study induced missing data and possible collection biases. Second, this study did not enable to take into account how response to oliguria (surveillance, diuretic use or IV fluid therapy) played a role. Moreover, fluid management was defined by the ERAS protocol in case of oliguria, but it was not possible to determine how doctors were compliant to the protocol regarding IV fluids and oliguria. Another point is that the ERAS protocol was implemented halfway through the study time period. It is possible that ERAS implementation could have influenced the results. However, no difference in terms of AKI, oliguria AKI and creatinine increase AKI was found between ERAS and non-ERAS patients. Finally, only experience from one single center was reported which may preclude generalization of the findings.

In this cohort of patients after liver surgery, postoperative UO correlated weakly with postoperative serum creatinine. Moreover, patients with creatinine increase had a higher rate of complications compared to patients with oliguria and creatinine increase was a risk factor for postoperative 30-day mortality. Finally, a long operation time was found as independent risk factor for AKI development after liver surgery.

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### Conflicts of interest

The authors have no conflicts of interest to disclose.

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### Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.hpb.2019.06.016.