

Assessing acute kidney injury following hepatectomy: Review

Saud Mohammed Saud Aleisa , Abdulaziz Fahad Altowairqi , Abdulaziz Saud Fahad Aljuaid , Abdulaziz Saleh Omar Khalid , Abdulrahman Ali Hadaidi , Abdullah Adel Alshaibi , Khalid Dhaifallah Hameed Althobaiti

Abstract— The aim of this study was to identify the pre and postoperative risk factors and estimate prognostic reasons of AKI, and highlight methods for preventing these events. on short- and long-term outcomes following hepatectomy for hepatocellular carcinoma (HCC). Comprehensive review of literature was performed using medical electronic databases such as MEDLINE, and EMBASE, articles concerned with acute kidney injury following hepatectomy published up to September, 2017 were recruited. In conclusion postoperative AKI occurs in 12% of patients after liver surgery. Some reasons like low preoperative kidney function, hypertension and blood transfusion can cause AKI. To reduce mortality and AKI cases it is important to carefully make operation plan and diagnose before, also carefully select patients.

Index Terms—Acute kidney injury following hepatectomy, Assessing kidney injuries, Hepatectomy complications.

INTRODUCTION

Presently, partial hepatectomy is the therapy option for a variety of primary liver tumors (benign or malignant), tumors of the bile ducts and also second malignant liver tumors. The partial liver resections could additionally be required in the management of complicated cystic liver illness, benign biliary systems, some situations of hepatic injury and also much more just recently with living donor liver hair transplant [1]. With the improvement of medical strategies, enhanced choice of patients to treatment, breakthroughs in anesthetic support and also perioperative care, this generally intricate and also been afraid procedure has actually come to be a regular treatment in the past 20 years, with appropriate death rates varying from 3.1% to 4.5% [2].

Amongst the potential complications of significant surgeries, involving the partial hepatectomy, acute kidney injury (AKI) need to be thought about as a vital reason for raised morbidity and also postoperative death [3], with an occurrence varying from 10% to 30% after significant procedures [4]. Literary works information report an occurrence of 1% of AKI in the postoperative significant non-cardiac surgical procedure without liver resection [5] regarding 20% after cardiac surgical treatment [6] and also 50% after liver transplantation [7].

In spite of this multiplicity of danger aspects for postoperative AKI after partial hepatectomy, there are primary factors that plainly trigger its occurrence. Initial aspect associates with huge blood losses with kidney hypoperfusion while the operation [8], that quite frequently may be correlated by the deleterious kidney results of red blood cell transfusion [9], as well as in some moments this kidney hypoperfusion takes place in patients with raised kidney vulnerability to ischemia, generally aged patients with underlying cardiac or kidney problems, or in the end it could be drug-induced [10] 2nd element associates with the incident of post-hepatectomy liver failure (PLF) with subsequent distributive circulatory modifications as well as hepatorenal syndrome (HRS) [8]. Later on, patients could have greater than one factor adding to post-operative AKI, and also regularly these combinations of acute disrespects can be worsened by sepsis [11] or direct exposure to nephrotoxic medications, such as aminoglycosides [12].

Acute kidney injury (AKI) following hepatectomy remains understudied in terms of diagnosis, severity, recovery and prognostic value. The aim of this study was to identify the pre and postoperative risk factors and estimate prognostic reasons of AKI, and highlight methods for preventing these events. on short- and long-term outcomes following hepatectomy for hepatocellular carcinoma (HCC).

METHODOLOGY:

Comprehensive review of literature was performed using medical electronic databases such as MEDLINE, and EMBASE, articles concerned with acute kidney injury following hepatectomy published up to September, 2017 were recruited. Restriction to only English language articles were applied in our search strategy.

DISCUSSION

Classifications for acute kidney injury:

AKI is defined by the degeneration of kidney function over a duration of hrs to days, leading to the failing of the kidney to eliminate nitrogenous waste items and also to keep liquid and also electrolyte homeostasis [13]. Over the last few years, numerous requirements have actually been recommended for the medical diagnosis of AKI generally populace, especially the "Risk, Injury, Failure, Loss of Renal Function and also End-Stage Renal Disease" (RIFLE) requirements [14] (Table 2), the "Acute Kidney Injury Network" (AKIN) requirements [15] (Table 1) and also much more just recently, the criteria recommended by a panel of professionals, which integrate the AKIN and also the RIFLE criteria, hence suggesting a brand-new category: The "Kidney Disease Improving Global Outcomes" criteria [16]. A rise in basal SCr of a minimum of 44.2 μmol/ L (0.5 mg/dL), a reduction in Cr clearance of a minimum of 50% or the requirement for kidney replacing treatment (RRT) were one of the most regular meanings applied for AKI in medical technique. Where UO has actually been applied to specify AKI, it is usually thought about that a value less than 400 - 500 mL/day could be an indicator.

The Acute Kidney Injury Network (AKIN) classification:

The AKIN classification (Table1) was presented in March 2007 in Critical Care [15], as well as it is a later variation of the RIFLE category with some changes: the medical diagnosis of AKI is just taken into consideration after accomplishing a sufficient standing of hydration as well as after omitting urinary system blockage; the AKIN category just depends on SCr; baseline SCr is not required in the AKIN category, as well as it needs a minimum of 2 values of SCr acquired within a duration of 48 h; AKI is specified by the unexpected reduction (in 48 h) of kidney function, specified by a rise in outright SCr of a minimum of 26.5 μmol/ L (0.3 mg/dL) or by a portion rise in SCr ≥ 50% (1.5 × baseline value), or by a reduction in the UO (recorded oliguria <0.5 mL/kg/h for greater than 6 h); Stage 1 represents the danger course, however it likewise takes into consideration an outright rise in SCr ≥ 26.5 μmol/ L (0.3 mg/dL); Stages 2 as well as 3 represent injury and also failing classes, specifically; Stage 3 also take into concentration patients who needs RRT of the stage (defined by SCr and/or UO) they are in at the point of RRT beginning; the two result classes (loss of kidney function and end-stage kidney disease) were removed from the classification.

Table.1. The AKIN classification/staging system of acute kidney injury [15].

Stage	SCr	UO
1	↑ SCr ≥26.5 μmol/L (≥0.3 mg/dL) or ↑SCr ≥150 a 200% (1.5 a 2×)	<0.5 mL/kg/h (>6 h)
2	↑ SCr >200 a 300% (>2 a 3×)	<0.5 mL/kg/h (>12 h)
3	↑ SCr >300% (>3×) or if baseline SCr ≥353.6 μmol/L (≥4 mg/dL) ↑SCr ≥44.2 μmol/L (≥0.5 mg/dL)	<0.3 mL/kg/h (24 h) or anuria (12 h)

SCr, serum creatinine; UO, urine output. RRT renal replacement therapy

These alterations were based upon the collective proof that even little rises in SCr are connected with a bad result, and also in the severe irregularity of sources as well as of the signs to begin RRT displayed in various regions and also health centers [17].

Strengths of the AKIN classification: initially, the AKI explanation is just taken into consideration after an appropriate condition of hydration is attained. As a result, the AKIN classification, not like RIFLE, includes essential etiological details. Second, the AKIN classification is based upon SCr and also not on GFR modifications. Third, the AKIN classification does not require baseline SCr to specify AKI, although it needs a minimum of 2 SCr resolutions within 48 h.

The RIFLE classification:

The RIFLE classification [14] (Table2) is based upon SCr and also UO components, and also takes into consideration 3 intensity classes of AKI (Risk, Injury and also Failure), depending on the variants in SCr and/or UO, as well as 2 result classes (loss of kidney function and also end-stage kidney illness). The patient must be categorized applying the criteria (SCr and/or UO) which results in the most awful category (optimum RIFLE), for example, if a patient remained in the Risk course depending on the UO yet in the Injury course basing on SCr variant, after that the most awful requirements (SCr) need to be utilized for identifying the extent of AKI in this patient.

Table 2: Risk, Injury, Failure, Loss of kidney function and

End-stage kidney disease (RIFLE) classification [14].

Class	GFR	UO
Risk	↑ SCr × 1.5 or ↓ GFR >25%	<0.5 mL/kg/h × 6 h
Injury	↑ SCr × 2 or ↓ GFR >50%	<0.5 mL/kg/h × 12 h
Failure	↑ SCr × 3 or ↓ GFR >75% or if baseline SCr ≥353.6 μmol/L(≥4 mg/dL) ↑ SCr >44.2 μmol/L(>0.5 mg/dL)	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss of kidney function	Complete loss of kidney function >4 weeks	
End-stage kidney disease	Complete loss of kidney function >3 months	

GFR, glomerular filtration rate; UO, urine output; SCr, serum creatinine.

The temporal pattern of the SCr and/or UO variation is also relevant for defining AKI: the deterioration of renal function must be sudden (1-7 days) and sustained (persisting >24 h).

Predictive factors that are associated with PHLF:

Numerous patient-related aspects are related to raised danger of PHLF (Table 3). Personnel death in patients with diabetic issues going through curative-intent hepatic resection for therapy of colorectal metastases has actually been revealed to be greater than similar patients without diabetes mellitus [17]. Since set, operative death was 8% in diabetics compared with 2% in non-diabetics (P<0.02). In addition, 80% of peri-operative fatalities in diabetic patients were secondary to PHLF. Excess death seen in diabetic patients going through significant hepatic resection is most likely multi-factorial, with modifications in liver metabolic rate, lowered immune function, and also hepatic steatosis resulting in post-operative liver disorder [18].

Chemotherapy-associated steatohepatitis (CASH) is a raising obstacle in the period of unique chemotherapeutic and also biologic representatives. Numerous commonly-used chemotherapy representatives trigger damages to hepatocytes, involving 5-fluorouracil, irinotecan, oxaliplatin, cituximab, and also bevacizumab [20]. In addition, pre-operative poor nutrition or kidney insufficiency, hyperbilirubinemia, thrombocytopenia, existence of co-morbidities (lung illness), and also advanced age are connected with boosted danger of PHLF [21].

Along with patient-specific aspects, the performance of the surgery itself affects danger of PHLF. Elements related to

raised danger are displayed in (Table 3) and also involve personnel approximated blood loss > 1,200 mL [22], intra-operative transfusion criteria, require for vena caval or various other vascular resection [23], personnel time > 240 mins [24], resection of > 50% of liver quantity, significant hepatectomy involving appropriate lobe [25], as well as skeletonization of the hepatoduodenal tendon in cases of biliary hatred. In patients for who <25% of the pre-operative liver quantity is left post-resection, the danger of PHLF is 3 times that of patients with ≥ 25% of liver quantity staying [26].

Problems of post-operative management affect the danger of PHLF, with post-operative hemorrhage [27] and also incident of intra-abdominal infection [28] providing raised danger. (Table 3).

Table 3. Predictive factors associated with increased risk of PHLF

Surgery related:	Patient related:
EBL >1,200 mL	Obesity
Intra-operative transfusions	Chemotherapy-associated steatohepatitis
Need for vascular resection	Hepatitis B, C
>50% liver volume resected	Malnutrition
Major hepatectomy including right lobectomy	Renal insufficiency
Skeletonization of hepatoduodenal ligament	Hyperbilirubinemia
<25% of liver volume remaining	Diabetes mellitus
Post-operative management:	Lung disease
Post-operative hemorrhage	Cirrhosis
Intra-abdominal infection	Age >65 years

An additional element associates with the incident of PLF with its distributive circulatory modifications, which is a significant reason of mortality after hepatic resection, and also finally could advance to HRS [29].

The existence of biliary blockage was a self-determining predictor of postoperative AKI in accordance with the authors' outcomes, and also the system whereby bilirubin might be harmful to the kidneys appears to be inflammatory along with obstructive [30], as well as hemodynamic adjustments could additionally contribute in biliary actors nephropathy [31]. Along with the abovementioned results, patients that are candidates for surgical treatment in the existence of biliary blockage with congestive cholestasis in the liver [32] could go through significant hepatic resections, with ensuing reduction

in the quantity of a functionally lacking liver parenchyma, inclining for PHLF.

Patients could have combinations of kidney insults that may be worsened by sepsis [33], which was an individual predictor in the authors' evaluation. The septicemia and its hemodynamic and also systemic consequences could finally exist together with liver failure, usually being the last case of PHLF [34].

Age likely contributes in survivability complying with partial hepatectomy. Fortner and Lincer [35] observed a considerable rise in death after age 65 (11.1%) and also discovered an additional rise in death in this age with prolonged hepatic resection (30.7%).

Chronic liver illness has actually been connected with greater danger of liver failure adhering to partial hepatectomy. Cirrhosis as well as recurring inflammatory action have actually been mentioned as adding aspects [36].

The steatotic liver is extra at risk to ischemia/reperfusion injury, maybe because of a transformed sinusoidal microcirculation [37].

Scientifically, there seems a powerful connection in between sepsis and post-hepatectomy liver failure. In one collection of 19 patients establishing intraperitoneal septic difficulties after hepatectomy, 13 passed away of liver failure [38].

Both preoperative reduced kidney function and also hypertension have actually been revealed to be danger elements for postoperative AKI in different medical setups [39]. Although the relationships in between red cell transfusion and postoperative AKI are not completely know, there are numerous structures that might be considered. Initially, due to a deficit in 2,3-biphosphoglycerate (likewise called 2,3-diphosphoglycerate) in saved donated blood, oxygen dumping from hemoglobin might have suffered. Second, due to much less deformability of saved red cell, they might have obstructed smaller sized capillaries and also resulted in disability of oxygen distribution to cells. Furthermore, because transfused red cell have actually a reduced life expectancy, they might have created hemolysis and also caused a rise in flowing cost-free iron. Considering that free iron is a very powerful factor to oxidative tension, a raised amount of free plasma iron could have played a crucial function in triggering postoperative AKI in transfused patients.

Angiotensin II receptor blockers have actually been revealed to be among one of free-lance danger elements for postoperative AKI in some medical setups, involving among one of our previous research studies [40]. They are regarded to lower the glomerular capillary pressure as well as the GFR by lowering the resistance of the efferent arterioles.

Identification and management:

When existing, PHLF appears by dynamic multi-system body

organ failure, consisting of kidney insufficiency, encephalopathy, require for ventilator support, and also require for pressor support. As hepatic function worsens, patients establish consistent hyperbilirubinemia and also coagulopathy [41]. The growth of coagulopathy is a specifically inadequate prognostic sign [42]. Daily dimension of serum C-reactive protein (CRP) could assist with the very early recognition of patients that are creating hepatic insufficiency after hepatectomy. A research study by Rahman as well as coworkers revealed that patients that established PHLF had a reduced CRP degree on post-operative day 1 compared to patients that did not establish PHLF. A serum CRP <32 g/dL was an independent forecaster of PHLF in multivariate regression analysis. Other devices for forecasting PHLF consist of the '50-50 requirements', MELD system, and also Acute Physiology and also Chronic Health Evaluation (APACHE) III. While the MELD system has a sensibility of 55% for morbidity and also 71% for death, the ISGLS criteria for PHLF execute specifically well in examining the danger of raised death after hepatectomy [43].

In order to decrease the occurrence of postoperative AKI after partial hepatectomy, a cautious patient choice as well as pre-operative resection preparation are compulsory, specifically when it comes to inclining CKI, biliary obstruction and also underlying cirrhosis, where MELD rating estimation can be incredibly worthfull. MELD rating [43], a usefully and also thoroughly verified device for anticipating liver failure development, was a forecaster of postoperative AKI, and also one of the most crucial, it can be used in the preoperative duration.

One of the most reliable therapy for PHLF is liver transplantation, however this is generally preserved for patients that have actually failed all various other encouraging treatments [44]. Primary therapy of PHLF consists of supporting care of failing systems, consisting of intubation, pressors, or dialysis. Therapy consists of infusion of albumin, fibrinogen, fresh frozen plasma, blood transfusion, and also initiation of dietary supplements [45].

CONCLUSION

In conclusion postoperative AKI occurs in 12% of patients after liver surgery. Some reasons like low preoperative kidney function, hypertension and blood transfusion can cause AKI. To reduce mortality and AKI cases it is important to carefully make operation plan and diagnose before, also carefully select patients.

REFERENCES

- (1) Clavien PA, Petrowsky H, DeOliveira ML, Graf R. Strategies for safer liver surgery and partial liver transplantation. *N Engl J Med.* 2007;356:1545-1559.

- (2) Belghiti J, Hiramatsu K, Benoist S, Massault P, Sauvanet A, Farges O. Seven hundred forty-seven hepatectomies in the 1990s: an update to evaluate the actual risk of liver resection. *J Am Coll Surg*. 2000;191:38-46.
- (3) Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *J Am Soc Nephrol*. 2005;16:3365-3370.
- (4) Bihorac A, Yavas S, Subbiah S, Hobson CE, Schold JD, Gabrielli A, Layon AJ, Segal MS. Long-term risk of mortality and acute kidney injury during hospitalization after major surgery. *Ann Surg*. 2009;249:851-858.
- (5) Kheterpal S, Tremper KK, Englesbe MJ, O'Reilly M, Shanks AM, Fetterman DM, Rosenberg AL, Swartz RD. Predictors of postoperative acute renal failure after noncardiac surgery in patients with previously normal renal function. *Anesthesiology*. 2007;107:892-902.
- (6) Andersson LG, Ekroth R, Bratteby LE, Hallhagen S, Wesslén O. Acute renal failure after coronary surgery--a study of incidence and risk factors in 2009 consecutive patients. *Thorac Cardiovasc Surg*. 1993;41:237-241.
- (7) Rimola A, Gavaler JS, Schade RR, el-Lankany S, Starzl TE, Van Thiel DH. Effects of renal impairment on liver transplantation. *Gastroenterology*. 1987;93:148-156.
- (8) Saner F. Kidney failure following liver resection. *Transplant Proc*. 2008;40:1221-1224.
- (9) Tomozawa A, Ishikawa S, Shiota N, Cholvisudhi P, Makita K. Perioperative risk factors for acute kidney injury after liver resection surgery: an historical cohort study. *Can J Anaesth*. 2015;62:753-761.
- (10) Armstrong T, Welsh FK, Wells J, Chandrakumaran K, John TG, Rees M. The impact of pre-operative serum creatinine on short-term outcomes after liver resection. *HPB (Oxford)* 2009;11:622-628.
- (11) Abuelo JG. Normotensive ischemic acute renal failure. *N Engl J Med*. 2007;357:797-805.
- (12) Moore RD, Smith CR, Lipsky JJ, Mellits ED, Lietman PS. Risk factors for nephrotoxicity in patients treated with aminoglycosides. *Ann Intern Med*. 1984;100:352-357.
- (13) Thadhani R, Pascual M, Bonventre JV. Acute renal failure. *N Engl J Med*. 1996;334:1448-1460.
- (14) Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care*. 2004;8:R204-R212.
- (15) Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care*. 2007;11:R31.
- (16) KDIGO Board Members. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl*. 2012;2:1-138.
- (17) Chertow GM, Burdick E, Honour M, et al. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *J Am Soc Nephrol*. 2005;16:3365-3370.
- (18) Eguchi H, Umeshita K, Sakon M, et al. Presence of active hepatitis associated with liver cirrhosis is a risk factor for mortality caused by posthepatectomy liver failure. *Dig Dis Sci* 2000;45:1383-8.
- (19) Little SA, Jarnagin WR, DeMatteo RP, et al. Diabetes is associated with increased perioperative mortality but equivalent long-term outcome after hepatic resection for colorectal cancer. *J Gastrointest Surg* 2002;6:88-94.
- (20) Fong Y, Bentrem DJ. CASH (Chemotherapy-Associated Steatohepatitis) costs. *Ann Surg* 2006;243:8-9.
- (21) Jarnagin WR, Gonen M, Fong Y, et al. Improvement in perioperative outcome after hepatic resection: analysis of 1,803 consecutive cases over the past decade. *Ann Surg* 2002;236:397-406; discussion 406-7.
- (22) Tomuş C, Iancu C, Bălă O, et al. Liver resection for benign hepatic lesion: mortality, morbidity and risk factors for postoperative complications. *Chirurgia (Bucur)* 2009;104:275-80.
- (23) Melendez J, Ferri E, Zwillman M, et al. Extended hepatic resection: a 6-year retrospective study of risk factors for perioperative mortality. *J Am Coll Surg* 2001;192:47-53.
- (24) Fernandez FG, Ritter J, Goodwin JW, et al. Effect of steatohepatitis associated with irinotecan or oxaliplatin pretreatment on resectability of hepatic colorectal metastases. *J Am Coll Surg* 2005;200:845-53.
- (25) Nanashima A, Yamaguchi H, Shibasaki S, et al. Comparative analysis of postoperative morbidity according to type and extent of hepatectomy. *Hepatogastroenterology* 2005;52:844-8.
- (26) Shoup M, Gonen M, D'Angelica M, et al. Volumetric analysis predicts hepatic dysfunction in patients undergoing major liver resection. *J Gastrointest Surg* 2003;7:325-30.
- (27) Jarnagin WR, Gonen M, Fong Y, et al. Improvement in perioperative outcome after hepatic resection: analysis of 1,803 consecutive cases over the past decade. *Ann Surg* 2002;236:397-406; discussion 406-7.
- (28) Tzeng CW, Cooper AB, Vauthey JN, et al. Predictors of morbidity and mortality after hepatectomy in elderly patients: analysis of 7621 NSQIP patients. *HPB (Oxford)* 2014;16:459-68.
- (29) Angeli P, Gines P, Wong F, Bernardi M, Boyer TD, Gerbes A, Moreau R, Jalan R, Sarin SK, Piano S, et al. Diagnosis and management of acute kidney injury in patients with cirrhosis: revised consensus recommendations of the International Club of Ascites. *Gut*. 2015;64:531-537.
- (30) Ozturk H, Terzi A, Ozturk H, Kukner A. Effect of sirolimus on renal injury induced by bile duct ligation in rats. *Acta Cir Bras*. 2010;25:401-406.
- (31) Padillo FJ, Cruz A, Espejo I, Barcos M, Gómez-Alvarez M, Muntané J. Alteration of the renal regulatory hormonal pattern during experimental obstructive jaundice. *Rev Esp Enferm Dig*. 2009;101:408-412.
- (32) Cohnert TU, Rau HG, Buttler E, Hernandez-Richter T, Sauter G, Reuter C, Schildberg FW. Preoperative risk assessment of hepatic resection for malignant disease. *World J Surg*. 1997;21:396-400; discussion 401.
- (33) Armstrong T, Welsh FK, Wells J, Chandrakumaran K, John TG, Rees M. The impact of pre-operative serum creatinine on short-term outcomes after liver resection. *HPB (Oxford)* 2009;11:622-628.
- (34) Slankamenac K, Breitenstein S, Held U, Beck-Schimmer B, Puhan MA, Clavien PA. Development and validation of a prediction score for postoperative acute renal failure following liver resection. *Ann Surg*. 2009;250:720-728.
- (35) Fortner JG, Lincer RM. Hepatic resection in the elderly. *Ann Surg*. 1990;211:141-5.
- (36) Takenaka K, Kanematsu T, Fukuzawa K, Sugimachi K. Can hepatic failure after surgery for hepatocellular carcinoma in cirrhotic patients be prevented? *World J Surg*. 1990;14:123-7.

- (37) Sun CK, Zhang XY, Zimmerman A, Davis G, Wheatley AM. Effect of ischemia-reperfusion injury on the microcirculation of the steatotic liver of the Zucker rat. *Transplantation*. 2001;72:1625-31.
- (38) Mochita S, Ogata I, Hirata K, Ohta Y, Yamada S, Fugiwara K. Provocation of massive hepatic necrosis by endotoxin after partial hepatectomy in rats. *Gastroenterology*. 1990;99:771-7.
- (39) Jafari SM, Huang R, Joshi A, Parvizi J, Hozack WJ. Renal impairment following total joint arthroplasty: who is at risk? *J Arthroplasty* 2010; 25(6 Suppl): 49-53.
- (40) Ishikawa S, Griesdale DE, Lohser J. Acute kidney injury after lung resection surgery: incidence and perioperative risk factors. *Anesth Analg* 2012; 114: 1256-62.
- (41) Roberts KJ, Bharathy KG, Lodge JP. Kinetics of liver function tests after a hepatectomy for colorectal liver metastases predict post-operative liver failure as defined by the International Study Group for Liver Surgery. *HPB (Oxford)* 2013;15:345-51.
- (42) Jin S, Fu Q, Wuyun G, et al. Management of post-hepatectomy complications. *World J Gastroenterol* 2013;19:7983-91.
- (43) Rahbari NN, Reissfelder C, Koch M, et al. The predictive value of postoperative clinical risk scores for outcome after hepatic resection: a validation analysis in 807 patients. *Ann Surg Oncol* 2011;18:3640-9.[
- (44) Chan SC, Sharr WW, Chan AC, et al. Rescue Living-donor Liver Transplantation for Liver Failure Following Hepatectomy for Hepatocellular Carcinoma. *Liver Cancer* 2013;2:332-7.
- (45) Chiarla C, Giovannini I, Giuliani F, et al. Plasma bilirubin correlations in non-obstructive cholestasis after partial hepatectomy. *Clin Chem Lab Med* 2008;46:1598-601

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