ELSEVIER

Contents lists available at ScienceDirect

Annals of Hepatology

journal homepage: www.elsevier.es/annalsofhepatology



Original article

Impact of neohepatic albumin-bilirubin scores on renal outcomes following living donor liver transplantation: a propensity score analysis



Hye-Won Jeong^a, Hye-Mee Kwon^b, Sung-Hoon Kim^b, Seong-Mi Yang^b, In-Gu Jun^b, Jun-Gol Song^{b,*}, Gyu-Sam Hwang^b

- a Department of Anesthesiology and Pain Medicine, Eunpyeong St. Mary's Hospital College of Medicine, The Catholic University of Korea, Seoul, Korea
- b Department of Anesthesiology and Pain Medicine, Laboratory for Cardiovascular Dynamics, Asan Medical Center, University of Ulsan College of Medicine, Seoul,

ARTICLE INFO

Article History: Received 2 April 2025 Accepted 1 July 2025 Available online 8 September 2025

Keywords:
Acute kidney injury
Albumin-bilirubin score
Chronic kidney disease
Living donor liver transplantation

ABSTRACT

Introduction and Objectives: Acute kidney injury (AKI) after liver transplantation (LT) impacts patient and graft outcomes. The Albumin-Bilirubin (ALBI) score, an objective and sensitive liver function index, may help predict post-LT outcomes. This study evaluated the association between neohepatic ALBI scores and renal outcomes in living donor LT (LDLT) recipients.

Patients and Methods: We examined 2171 adult LDLT recipients between 2012 and 2019. Outcomes included severe post-LT AKI, renal replacement therapy (RRT), chronic kidney disease (CKD) at 1 year, early allograft dysfunction (EAD), and overall graft failure. Multivariate logistic regression, Cox proportional hazards regression, and propensity score matched (PSM) analyses were performed to evaluate the association between neohepatic ALBI and post-LT outcomes.

Results: Severe AKI, RRT, CKD, EAD, and overall graft failure occurred in 21.6%, 2.2%, 41.9%, 5.9%, and 15.8% of patients, respectively. Higher neohepatic ALBI scores (\geq -1.615) were significantly associated with severe AKI (OR: 2.34, 95% CI: 1.79−3.04, P<0.001, multivariate analysis; OR: 2.18, 95% CI: 1.62−2.95, P<0.001, PSM analysis), RRT (OR: 3.80, 95% CI: 1.53−11.31, P=0.008, multivariate analysis; OR: 7.17, 95% CI: 1.61−31.89, P=0.010, PSM analysis), CKD (OR: 1.22, 95% CI: 1.00−1.47, P=0.044, multivariate analysis; OR: 1.43, 95% CI: 1.11−1.85, P=0.006, PSM analysis), and overall graft failure (HR: 1.30, 95% CI: 1.01−1.68, P=0.041, multivariate analysis; HR: 1.55, 95% CI: 1.08−2.23, P=0.018, PSM analysis).

Conclusions: Neohepatic ALBI scores are significantly associated with post-LT severe AKI, RRT, CKD, and graft failure, underscoring their prognostic value in LDLT recipients.

© 2025 Fundación Clínica Médica Sur, A.C. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/)

1. Introduction

Acute kidney injury (AKI) is a frequent and critical complication of liver transplantation (LT) [1,2]. Post-LT AKI has been consistently associated with adverse post-transplant outcomes, including chronic kidney disease (CKD) and decreased patient and graft survival rates

Abbreviations: ABOi, ABO incompatibility; AKI, Acute kidney injury; ALBI, Albumin-Bilirubin; BMI, Body mass index; CI, Confidence interval; CKD, Chronic kidney disease; CNI, Calcineurin inhibitor; CTP, Child-Pugh-Turcotte; DDLT, Deceased donor LT; EAD, Early allograft dysfunction; GFR, Glomerular filtration rate; HCC, Hepatocellular carcinoma; HR, Hazard ratio; IQR, Interquartile range; KDIGO, Kidney Disease Improving Global Outcome; LC, Liver cirrhosis; LDLT, Living donor LT; LT, Liver transplantation; MELD, Model for End-Stage Liver Disease; MELD-Na, MELD sodium; OR, Odds ratio; PRBC, Packed red blood cell; PRS, Post-reperfusion syndrome; PSM, Propensity score matched; PT-INR, Prothrombin time—international normalized ratio; ROC, Receiver operating characteristic; RRT, Renal replacement therapy; sCr, Serum creatinine

E-mail address: jungol.song@amc.seoul.kr (J.-G. Song).

[1—4]. Consequently, identifying and mitigating risk factors for renal function deterioration are critical to improve postoperative outcomes in LT recipients.

The Albumin-Bilirubin (ALBI) score, initially developed to evaluate liver function and predict outcomes in patients with hepatocellular carcinoma (HCC) [5], has had its application extended to encompass a wide range of chronic liver diseases [6–10]. Compared with the Child-Pugh-Turcotte (CTP) score or Model for End-Stage Liver Disease (MELD) score, the ALBI score is objective and can detect smaller changes in liver dysfunction [11]. Regarding chronic liver disease, numerous publications have demonstrated that the ALBI score is highly prognostic in patients across all types and stages of chronic liver disease [6–10].

According to recent research, a higher pre-transplant ALBI score has also been shown to be associated with adverse post-transplant complications and decreased survival rates in patients undergoing LT [12–15]. As the function of the transplanted liver graft is crucial for

^{*} Corresponding author.

determining the prognosis of LT recipients [11], we focused on postreperfusion (neohepatic) ALBI scores. Therefore, this study aimed to investigate the association between neohepatic ALBI scores and post-LT renal outcomes in living donor LT (LDLT) recipients. We hypothesized that higher neohepatic ALBI scores could adversely affect renal outcomes following LDLT.

2. Patients and Methods

2.1. Patients

We reviewed electronic medical records of patients who underwent LT at our center from January 2012 to December 2019. The study included adults (≥18 years) who underwent LDLT. Exclusion criteria were preoperative serum creatinine (sCr) level >1.4 mg/dL, diagnosis of CKD or hepatorenal syndrome, or hemodialysis at baseline.

2.2. Clinical data

Data, including demographic information, donor characteristics, perioperative laboratory results, intraoperative details, and postoperative outcomes, were collected from the electronic medical records system. Collected demographics included age, sex, body mass index (BMI), comorbidities like diabetes mellitus, hypertension, coronary artery disease, and congestive heart failure, liver disease details (etiology of liver cirrhosis [LC], MELD sodium [MELD-Na] score, and CTP score), and compatibility issues such as dual donor grafts and ABO incompatibility (ABOi). Donor-related variables included donor age, sex, and total fatty change. Preoperative laboratory data included albumin, total bilirubin, creatinine, prothrombin time-international normalized ratio (PT-INR), and sodium. Intraoperative data included post-reperfusion syndrome (PRS), volume and type of fluids (crystalloid and colloid), massive transfusion, vasopressor use, anesthetic time, cold ischemic time, warm ischemic time, total ischemic time, graft-to-recipient weight ratio, intraoperative embolization, and biopump use. PRS was defined as a decrease in mean arterial pressure by more than 30 % compared to the pre-reperfusion level, persisting for at least 1 min within the initial 5 min following liver graft reperfusion [16]. Massive transfusion was defined as the transfusion of ≥ 10 packed red blood cell (PRBC) units within 24 h, or ≥4 PRBC units within 1 hour [17]. Intraoperative laboratory values, including albumin and bilirubin were measured at the preanhepatic phase (1 h after skin incision), anhepatic phase (30 min after liver removal), and neohepatic phase (1 h after graft reperfusion), as per our standard protocol for LT patients. The ALBI score was calculated using the formula: $(log_{10}bilirubin \times 0.66)$ + (albumin $\times -0.085$), where bilirubin is measured in μ mol/L and albumin in g/L [5].

2.3. Anesthetic technique

Standard monitoring, as recommended by the American Society of Anesthesiologists, was applied before the induction of anesthesia. Anesthetic management and patient care for LT were carried out according to our institutional protocol, as described previously [18 –20]. Anesthesia was induced with thiopental sodium, fentanyl, and rocuronium. General anesthesia was maintained with volatile anesthetics (sevoflurane or desflurane) combined with a continuous infusion of fentanyl and rocuronium or vecuronium. Radial and femoral artery cannulation was performed intraoperatively for continuous arterial monitoring and central venous catheter was inserted into the internal jugular vein for fluid infusion. A Swan-Ganz catheter was inserted via a 9Fr introducer sheath to monitor pulmonary arterial pressure and cardiac output. Intraoperative volume replacement involved a ratio of 1 L of crystalloid (Plasma solution A; CJ Pharmaceutical, Seoul, Korea) to 100–200 mL of 20 % human albumin (Green

Cross Co., Yong-In, Korea), aimed at maintaining albumin levels above 3 mg/dL, as guided by intraoperative albumin measurements. Mean arterial blood pressure was maintained either above the preoperative level or at least 65 mmHg. Incremental doses of inotropes, vasopressors, and additional fluids were administered according to the patient's hemodynamic status.

2.4. Surgical technique

The native liver of the recipient was dissected to expose the inferior vena cava after separating the portal vein, hepatic artery, and bile duct. After removing the native liver, the donor graft was transplanted into the recipient. An end-to-end anastomosis between the right portal vein of the donor and the portal vein of the recipient was performed after completing the hepatic vein anastomosis. Graft reperfusion was performed after completing portal vein anastomosis. Patients with postreperfusion syndrome received norepinephrine or epinephrine depending on the severity. A continuous infusion of norepinephrine was commenced to achieve a target mean arterial pressure above 70 % of the baseline. Sequential anastomosis of the hepatic artery and bile duct was performed after graft reperfusion. Postoperatively, the recipients were transferred to the intensive care unit without extubation. Detailed surgical procedures for LDLT have been described in previous studies [19]. The same type of immunosuppression protocol was used in all cases, which was comprised of tacrolimus and mycophenolate mofetil (500 mg twice daily) as the primary immunosuppressive agents after LDLT [20].

2.5. Outcomes

The primary outcome of this study was severe AKI (>stage 2), defined by the Kidney Disease: Improving Global Outcome (KDIGO) criteria [21]. AKI diagnosis was based on an increase in sCr level by ≥0.3 mg/dL within 48 h postoperatively or an increase in sCr level to ≥1.5 times the baseline value within 7 days postoperatively; as per our routine protocol for LDLT recipients, baseline sCr levels were measured the day before transplantation [22]. According to the KDIGO criteria, stage 1 AKI was defined as an increase in sCr level to 1.5-1.9 times the baseline level or by an increase in sCr level of ≥0.3 mg/dL. Stage 2 AKI was indicated by an increase in sCr level to 2.0-2.9 times the baseline level, whereas stage 3 AKI was defined as an increase in sCr level to 3.0 times the baseline level, an increase in sCr level to ≥4.0 mg/dL, or initiation of renal replacement therapy (RRT). Secondary outcomes included need for postoperative RRT, CKD, early allograft dysfunction (EAD), and overall graft failure. CKD was defined as a decrease in glomerular filtration rate (GFR) (<60 mL/min per 1.73 m²) persisting for more than 3 months at 1 year after LT [23]. EAD was defined by the presence of one or both of the following variables: bilirubin >10 mg/dL or PT (INR) >1.6 on postoperative day 7 [24]. Graft failure was defined as the necessity for retransplantation or death, determined through medical records and the Organ Transplantation registry of our center.

2.6. Statistical analysis

Continuous variables are presented as means \pm standard deviation or medians with interquartile ranges (IQR) and categorical variables as frequencies and percentages. Receiver operating characteristic (ROC) curves were used to determine the optimal neohepatic ALBI cutoff for predicting severe AKI. Patients were stratified based on this cutoff, and group comparisons were performed using the Student's t-test, Mann-Whitney U test, Chi-square test, or Fisher's exact test, as appropriate. Propensity score matching (PSM) was employed to balance baseline characteristics, with logistic regression used to calculate the propensity scores. Model discrimination was evaluated using C statistics (C-statistics = 0.858), and calibration was

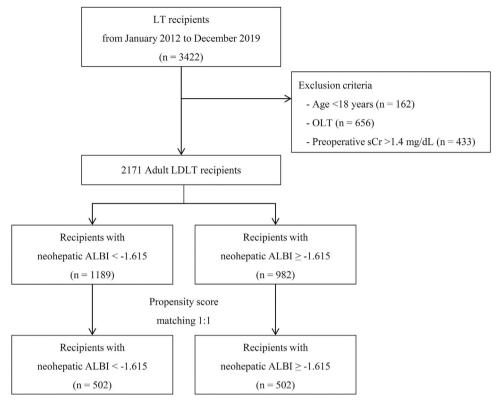


Fig. 1. Flowchart of the study population. Abbreviations: LT, liver transplantation; OLT, orthotopic liver transplantation; sCr, serum creatinine; LDLT, living donor liver transplantation: ALBI. Albumin-Bilirubin.

assessed using Hosmer-Lemeshow statistics (P = 0.109). Factors associated with severe AKI, postoperative RRT, CKD, and EAD were identified using multivariate logistic regression analysis. In addition, factors associated with overall graft failure were identified using multivariate Cox proportional hazards regression analysis. The level of statistical significance was established at a p-value <0.05. Data analyses were performed using SPSS version 22.0 (IBM Corp., Armonk, NY, USA) and R version 3.5.1 (R Foundation for Statistical Computing, Vienna, Austria).

2.7. Ethical statements

The Institutional Review Board of our center approved this study (protocol number: 2023–0561) and waived the requirement for informed consent due to its retrospective design.

3. Results

This study included 2171 adult recipients of LDLT, with a median follow-up time of 6.6 years (IQR: 4.8-8.7 years) (Fig. 1). ROC curve analysis identified the optimal cutoff value of neohepatic ALBI for predicting severe AKI as -1.615, exhibiting a sensitivity of 0.701, specificity of 0.616, positive predictive value of 0.335, and negative predictive value of 0.882. The area under the ROC curve was 0.700 (95 % confidence interval [CI]: 0.674–0.726; *P* < 0.001) (Fig. 2). Based on this optimal cutoff value, the 2171 patients were stratified into two groups. Table 1 details the preoperative and intraoperative data of the study participants. Individuals with a higher ALBI score exhibited a lower incidence of hypertension but a higher prevalence of congestive heart failure and higher MELD-Na and CTP scores. Preoperatively, they had prolonged PT (INR), higher total bilirubin levels, and lower albumin, creatinine, and sodium levels. Their donors had a higher degree of fatty change in the liver tissue. In addition, these individuals had a higher incidence of dual donor LT and massive transfusions during LT. They experienced longer cold ischemic time, warm ischemic time, total ischemic time, and anesthesia duration, in addition to a higher graft-to-recipient weight ratio and a higher incidence of biopump use.

Among the 2171 LDLT recipients, 21.6 % (n = 469) developed severe AKI, and 2.2 % (n = 48) required postoperative RRT. Meanwhile, 72 of the 2171 patients lacked follow-up data on GFR. Among the remaining 2099 recipients, 41.9 % (n = 879) developed CKD. The incidences of EAD and overall graft failure were 5.9 % (n = 127) and

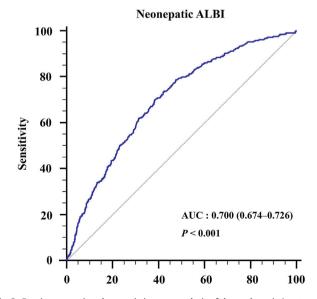


Fig. 2. Receiver operating characteristic curve analysis of the neohepatic (post-reperfusion) Albumin-Bilirubin score for predicting severe acute kidney injury. Abbreviations: ALBI, Albumin-Bilirubin; AUC, Area under the curve.

 Table 1

 Perioperative characteristics of the study population before and after propensity score matching.

| | | Unmatched (n = | Propensity score matched (n = 1004) | | | | |
|--------------------------------------|------------------|---|---|-------|------------------------------------|---|---------|
| | Total (n = 2171) | Neohepatic ALBI < -1.615 (n = 1189) | Neohepatic ALBI ≥ -1.615 $(n = 982)$ | SMD | Neohepatic ALBI < -1.615 (n = 502) | Neohepatic ALBI ≥ -1.615 $(n = 502)$ | SMD |
| Demographic variables | | | | | | | |
| Age (years) | 54.0 ± 8.4 | 54.3 ± 8.3 | 53.8 ± 8.6 | 0.059 | 54.3 ± 8.5 | 54.6 ± 8.1 | 0.036 |
| Sex, male | 1627 (74.9) | 913 (76.8) | 714 (72.7) | 0.094 | 372 (74.1) | 377 (75.1) | 0.023 |
| Body mass index (kg/m ²) | 24.5 ± 3.5 | 24.5 ± 3.4 | 24.4 ± 3.6 | 0.036 | 24.5 ± 3.7 | 24.4 ± 3.6 | 0.019 |
| Diabetes mellitus | 522 (24.0) | 289 (24.3) | 233 (23.7) | 0.014 | 125 (24.9) | 120 (23.9) | 0.023 |
| Hypertension | 397 (18.3) | 262 (22.0) | 135 (13.8) | 0.218 | 78 (15.5) | 79 (15.7) | 0.005 |
| Coronary arterial disease | 207 (9.5) | 110 (9.3) | 97 (9.9) | 0.021 | 40 (8.0) | 43 (8.6) | 0.022 |
| Congestive heart failure | 152 (7.0) | 60 (5.1) | 92 (9.4) | 0.168 | 31 (6.2) | 42 (8.4) | 0.084 |
| Etiology | (, | , , | () | 0.313 | () | () | 0.026 |
| Hepatitis B virus | 1296 (59.7) | 787 (66.2) | 509 (51.8) | | 288 (57.4) | 283 (56.4) | |
| Hepatitis C virus | 150 (6.9) | 76 (6.4) | 74 (7.5) | | 37 (7.4) | 38 (7.6) | |
| Alcoholic | 441 (20.3) | 185 (15.6) | 256 (26.1) | | 111 (22.1) | 116 (23.1) | |
| Other disease | 284 (13.1) | 141 (11.9) | 143 (14.6) | | 66 (13.2) | 65 (13.0) | |
| MELD-Na score | 13.0 ± 6.2 | 10.3 ± 4.3 | 16.3 ± 6.5 | 1.071 | 13.1 ± 5.2 | 13.1 ± 5.2 | 0.002 |
| Child-Pugh-Turcotte score | 7.6 ± 2.0 | 6.7 ± 1.6 | 8.6 ± 1.9 | 1.104 | 7.7 ± 1.8 | 7.7 ± 1.7 | < 0.001 |
| Dual donor grafts | 162 (7.5) | 71 (6.0) | 91 (9.3) | 0.124 | 37 (7.4) | 41 (8.2) | 0.030 |
| ABO incompatibility | 512 (23.6) | 300 (25.2) | 212 (21.6) | 0.086 | 127 (25.3) | 121 (24.1) | 0.028 |
| Donor-related variables | (, | , | (, | | () | (' ') | |
| Age (years) | 28.6 ± 8.3 | 28.6 ± 8.4 | 28.5 ± 8.3 | 0.008 | 28.6 ± 8.5 | 28.6 ± 8.2 | 0.005 |
| Sex, male | 1466 (67.5) | 780 (65.6) | 686 (69.9) | 0.091 | 350 (69.7) | 351 (69.9) | 0.004 |
| Total fatty change (%) | 3.8 ± 6.7 | 3.3 ± 6.0 | 4.3 ± 7.4 | 0.148 | 4.0 ± 7.3 | 3.8 ± 6.7 | 0.038 |
| Preoperative laboratory variables | | | | | | | |
| Albumin (g/dL) | 3.1 ± 0.6 | 3.3 ± 0.5 | 3.0 ± 0.6 | 0.593 | 3.1 ± 0.5 | 3.0 ± 0.5 | 0.133 |
| Total bilirubin (mg/dL) | 3.3 ± 5.7 | 1.6 ± 2.9 | 5.2 ± 7.3 | 0.648 | 2.8 ± 4.2 | 3.0 ± 5.0 | 0.043 |
| Creatinine (mg/dL) | 0.8 ± 0.2 | 0.8 ± 0.2 | 0.7 ± 0.2 | 0.183 | 0.7 ± 0.2 | 0.7 ± 0.2 | 0.077 |
| Prothrombin time (INR) | 1.4 ± 0.4 | 1.3 ± 0.3 | 1.6 ± 0.4 | 0.785 | 1.4 ± 0.5 | 1.4 ± 0.3 | 0.038 |
| Sodium (mmol/L) | 138.7 ± 4.6 | 139.9 ± 3.4 | 137.3 ± 5.5 | 0.568 | 139.0 ± 4.1 | 138.5 ± 4.9 | 0.106 |
| Intraoperative variables | 13017 ± 110 | 130.0 ± 3.1 | 137.13 ± 0.10 | 0.000 | 13010 ± 111 | 15015 ± 110 | 0.100 |
| Post-reperfusion syndrome | 1402 (64.6) | 769 (64.7) | 633 (64.5) | 0.005 | 322 (64.1) | 324 (64.5) | 0.008 |
| Albumin (L) | 3.7 ± 3.3 | 3.6 ± 3.4 | 3.9 ± 3.2 | 0.088 | 4.2 ± 2.5 | 4.0 ± 3.7 | 0.061 |
| Massive transfusion | 672 (31.0) | 208 (17.5) | 464 (47.3) | 0.671 | 168 (33.5) | 173 (34.5) | 0.021 |
| Vasopressor use | 1878 (86.5) | 1040 (87.5) | 838 (85.3) | 0.062 | 439 (87.5) | 437 (87.1) | 0.012 |
| Anesthetic time (h) | 13.8 ± 2.3 | 13.2 ± 2.0 | 14.6 ± 2.4 | 0.641 | 14.0 ± 2.1 | 14.0 ± 2.2 | 0.001 |
| Cold ischemic time (min) | 85.7 ± 26.5 | 83.9 ± 26.1 | 87.9 ± 26.9 | 0.150 | 86.4 ± 26.0 | 86.9 ± 26.0 | 0.019 |
| Warm ischemic time (min) | 42.0 ± 16.0 | 41.1 ± 16.1 | 43.1 ± 15.7 | 0.127 | 42.6 ± 15.2 | 42.9 ± 16.0 | 0.015 |
| Total ischemic time (min) | 127.7 ± 32.4 | 125.0 ± 31.8 | 131.0 ± 32.9 | 0.127 | 128.9 ± 31.7 | 129.7 ± 32.6 | 0.023 |
| Graft-to-recipient weight ratio | 1.1 ± 0.2 | 1.1 ± 0.2 | 1.1 ± 0.3 | 0.173 | 1.1 ± 0.2 | 1.1 ± 0.2 | 0.023 |
| Intraoperative embolization | 359 (16.5) | 193 (16.2) | 166 (16.9) | 0.018 | 101 (20.1) | 92 (18.3) | 0.046 |
| Biopump use | 276 (12.7) | 112 (9.4) | 164 (16.7) | 0.217 | 58 (11.6) | 79 (15.7) | 0.122 |

Values are expressed as mean \pm standard deviation, median (interquartile range), or number of patients (%), as appropriate. Abbreviations: ALBI, Albumin-Bilirubin score; SMD, standardized mean difference; MELD-Na score, Model for End-Stage Liver Disease sodium score; INR, international normalized ratio.

15.8 % (n = 292), respectively. A significant difference was noted in the incidence of severe AKI between the two groups (33.5 % in the higher ALBI group vs. 11.8 % in the lower ALBI group, P < 0.001) (Table 3).

Table 1 also presents preoperative and intraoperative characteristics of PSM patients (1:1 matched set, n=1004; n=502 in the low ALBI group and n=502 in the high ALBI group). After PS matching, individuals with a higher ALBI score exhibited lower preoperative levels of albumin and sodium. Additionally, they had a higher frequency of intraoperative biopump use. The incidence of severe AKI was 29.9 % (n=150) in the higher ALBI group compared with the 16.3% (n=82) in the lower ALBI group (P<0.001) (Table 3).

Univariate logistic regression analysis linked a higher ALBI score with an increased risk of severe AKI (odds ratio [OR]: 3.78, 95 % CI: 3.03-4.71, P<0.001), a finding that remained significant after adjusting for AKI-related variables (OR: 2.34, 95 % CI: 1.79-3.04, P<0.001) as shown in Table 2. Additionally, female sex, higher BMI and CTP score, alcoholic liver disease, and longer anesthetic time were significantly associated with severe AKI. In the PSM analysis, a higher ALBI score was significantly associated with severe AKI (OR: 2.18, 95 % CI: 1.62-2.95, P<0.001), as indicated in Table 4.

Furthermore, the group with higher ALBI scores exhibited significantly greater incidences of post-RRT (4.3 % vs. 0.5 %, P < 0.001), CKD (45.2 % vs. 39.2 %, P = 0.006), EAD (9.5 % vs. 2.9 %, P < 0.001), and overall graft failure (18.2 % vs. 13.0 %, P = 0.021) (Tables 3 and 4). After PS matching, the higher ALBI score group still showed significantly higher incidences of post-RRT (2.8 % vs. 0.4 %, P = 0.003), CKD (46.9 % vs. 38.2 %, P = 0.007), and overall graft failure (16.8 % vs. 10.6 %, P = 0.019). Higher ALBI was significantly associated with post-RRT (OR: 3.80, 95 % CI: 1.53–11.31, P = 0.008, multivariate analysis; OR 7.17, 95 % CI 1.61–31.89, P = 0.010, PSM analysis), CKD (OR: 1.22, 95 % CI: 1.00–1.47, P = 0.044, multivariate analysis; OR: 1.43, 95 % CI: 1.11–1.85, P = 0.006, PSM analysis), and overall graft failure (hazard ratio [HR]: 1.30, 95 % CI: 1.01–1.68, P = 0.041, multivariate analysis; HR: 1.55, 95 % CI: 1.08–2.23, P = 0.018, PSM analysis) (Table 4).

4. Discussion

In this large retrospective study of LDLT recipients, a high neohepatic ALBI score (\geq -1.615) was independently associated with post-LT AKI, need for RRT, CKD, and overall graft failure. Even after PS matching to adjust for important preoperative and intraoperative

Table 2Multivariate logistic regression analysis for severe acute kidney injury after living donor liver transplantation.

| | | Un | ivariate | | | | | |
|--------------------------------------|------|------|----------|---------|------|------|-----------------|---------|
| | OR | 95 | 95 % CI | | OR | 95 5 | <i>P</i> -value | |
| Neohepatic ALBI ≥ −1.615 | 3.78 | 3.03 | 4.71 | < 0.001 | 2.34 | 1.79 | 3.04 | < 0.001 |
| Age (years) | 0.99 | 0.98 | 1.00 | 0.035 | 0.99 | 0.98 | 1.00 | 0.157 |
| Sex, male | 0.68 | 0.54 | 0.85 | 0.001 | 0.67 | 0.52 | 0.87 | 0.003 |
| Body mass index (kg/m ²) | 1.10 | 1.07 | 1.13 | < 0.001 | 1.11 | 1.07 | 1.14 | < 0.001 |
| Diabetes mellitus | 1.10 | 0.87 | 1.39 | 0.447 | | | | |
| Hypertension | 0.97 | 0.74 | 1.26 | 0.812 | | | | |
| Coronary arterial disease | 1.11 | 0.79 | 1.56 | 0.560 | | | | |
| Etiology | | | | | | | | |
| Hepatitis B virus | 1 | | | < 0.001 | 1 | | | 0.189 |
| Hepatitis C virus | 1.76 | 1.20 | 2.59 | 0.004 | 1.26 | 0.83 | 1.93 | 0.281 |
| Alcoholic | 1.92 | 1.49 | 2.46 | < 0.001 | 1.36 | 1.02 | 1.81 | 0.036 |
| Other disease | 1.59 | 1.18 | 2.15 | 0.003 | 1.09 | 0.77 | 1.54 | 0.618 |
| MELD-Na score | 1.09 | 1.07 | 1.10 | < 0.001 | | | | |
| Child-Pugh-Turcotte score | 1.34 | 1.27 | 1.40 | < 0.001 | 1.18 | 1.11 | 1.26 | < 0.001 |
| Dual donor grafts | 1.92 | 1.36 | 2.71 | < 0.001 | 0.84 | 0.53 | 1.35 | 0.471 |
| ABO incompatibility | 0.93 | 0.73 | 1.19 | 0.571 | | | | |
| Donor Age (years) | 1.01 | 0.99 | 1.02 | 0.385 | | | | |
| Donor Sex, male | 1.02 | 0.82 | 1.27 | 0.885 | | | | |
| Donor total fatty change (%) | 1.01 | 0.99 | 1.02 | 0.380 | | | | |
| Post-reperfusion syndrome | 0.97 | 0.78 | 1.20 | 0.754 | | | | |
| Albumin (L) | 1.06 | 1.02 | 1.09 | 0.001 | 1.00 | 0.96 | 1.04 | 0.975 |
| Massive transfusion | 2.25 | 1.83 | 2.78 | < 0.001 | 1.04 | 0.79 | 1.37 | 0.802 |
| Vasopressor use | 1.29 | 0.94 | 1.77 | 0.117 | | | | |
| Anesthetic time (h) | 1.22 | 1.17 | 1.27 | < 0.001 | 1.14 | 1.07 | 1.21 | < 0.001 |
| Cold ischemic time (h) | 1.00 | 1.00 | 1.01 | 0.481 | | | | |
| Warm ischemic time (h) | 1.01 | 1.00 | 1.02 | 0.005 | 1.00 | 0.99 | 1.01 | 0.880 |
| Graft-to-recipient weight ratio | 0.84 | 0.54 | 1.29 | 0.416 | | | | |
| Intraoperative embolization | 1.05 | 0.80 | 1.38 | 0.731 | | | | |
| Biopump use | 1.82 | 1.38 | 2.40 | < 0.001 | | | | |

Abbreviations: OR, odds ratio; CI, confidence interval; ALBI, Albumin-Bilirubin score; MELD-Na score, Model for End-Stage Liver Disease sodium score.

confounders, high neohepatic ALBI scores were significantly associated with post-LT AKI, RRT, CKD, and overall graft failure.

Previous research on the impact of pre-transplant ALBI scores on post-transplant AKI has yielded inconsistent results. A retrospective study of deceased donor LT (DDLT) recipients associated high preoperative ALBI scores (>-1.48) with an increased incidence of postoperative AKI [12]. In contrast, a study focusing on LDLT recipients reported no significant difference in renal failure rates among different ALBI grades [14]. These investigations, primarily focusing on pretransplant ALBI scores, relied on Pearson's chi-square test without adjustments for potential confounders. However, post-LT renal outcomes are significantly influenced by multiple factors, including not only preoperative status of LT recipients, which can be partially indicated by their preoperative ALBI scores, but also functionality of the transplanted graft and various perioperative factors. In our study, we selected the ALBI scores measured 1 h after graft reperfusion to

reflect both the status of the recipient and the functionality of the transplanted graft, thereby enabling early detection of postoperative AKI. Additionally, we accounted for important preoperative and intraoperative confounders using multivariate logistic regression and PSM analyses. This approach enabled a clearer elucidation of the association between neohepatic ALBI scores and post-LT renal outcomes such as AKI, RRT, and CKD.

The relationship between the ALBI score and AKI has also been investigated beyond LT, including in patients with HCC and those undergoing non-liver-related interventions or surgeries. In a retrospective study of patients undergoing platinum-based transcatheter arterial chemoembolization or transarterial infusion chemotherapy for HCC, high ALBI grades (II and III) were identified as independent risk factors for an increase in sCr level [25]. Furthermore, in the context of cardiac surgery, patients with high ALBI scores (>-2.44) had a greater incidence of postoperative AKI following cardiac valvular

Table 3Renal outcomes of the study population before and after propensity score matching.

| | Unma | tched (n = 2171) | Propensity score matched (n = 1004) | | | | | |
|--|--|--|-------------------------------------|---------------------------------------|---|-----------------|--|--|
| | Neohepatic ALBI < -1.615 (n = 1189) | Neohepatic ALBI ≥ -1.615 P-va $(n = 982)$ | | Neohepatic ALBI < -1.615 (n = 502) | Neohepatic ALBI ≥ -1.615 ($n = 502$) | <i>P</i> -value | | |
| Acute kidney injury | 626 (52.7) | 729 (74.2) | < 0.001 | 301 (60.0) | 352 (70.1) | < 0.001 | | |
| Acute kidney injury stage (1/2/3) | | | < 0.001 | | | < 0.001 | | |
| Stage 1 | 486 (40.9) | 400 (40.7) | | 219 (43.6) | 202 (40.2) | | | |
| Stage 2 | 115 (9.7) | 260 (26.5) | | 65 (13.0) | 131 (26.1) | | | |
| Stage 3 | 25 (2.1) | 69 (7.0) | | 17 (3.4) | 19 (3.8) | | | |
| Severe acute kidney injury | 140 (11.8) | 329 (33.5) | < 0.001 | 82 (16.3) | 150 (29.9) | < 0.001 | | |
| Post-renal replacement therapy | 6 (0.5) | 42 (4.3) | < 0.001 | 2 (0.4) | 14(2.8) | 0.003 | | |
| Chronic kidney disease (unmatched, $n = 2099$, matched, $n = 973$) | 454 (39.2) | 425 (45.2) | 0.006 | 186 (38.2) | 228 (46.9) | 0.007 | | |

Abbreviation: ALBI, Albumin-Bilirubin score

Table 4 Predictive value of neohepatic Albumin-Bilirubin score ≥ -1.615 for renal and graft outcomes after living donor liver transplantation.

| | | Crude | | | Multivariable adjusted ^a | | | Propensity score-matched | | | | |
|---|-------------------------------|----------|------|------------|-------------------------------------|------|------------|--------------------------|---------|------|------------|-----------------|
| | | Event/n | OR | 95 % CI | P-value | OR | 95 % CI | P-value | Event/n | OR | 95 % CI | <i>P</i> -value |
| Severe acute kidney injury | Neohepatic ALBI ≥ -1.615 | 329/982 | 3.78 | 3.03-4.71 | <0.001 | 2.34 | 1.79-3.04 | <0.001 | 150/502 | 2.18 | 1.62-2.95 | <0.001 |
| | Neohepatic ALBI < -1.615 | 140/1189 | 1 | | | 1 | | | 82/502 | 1 | | |
| Postoperative renal replacement therapy | Neohepatic ALBI \geq -1.615 | 42/982 | 8.81 | 4.03-23.18 | <0.001 | 3.80 | 1.53-11.31 | 0.008 | 14/502 | 7.17 | 1.61-31.89 | 0.010 |
| | Neohepatic ALBI < -1.615 | 6/1189 | 1 | | | 1 | | | 2/502 | 1 | | |
| Chronic kidney disease | Neohepatic ALBI \geq -1.615 | 425/940 | 1.24 | 1.05-1.47 | 0.013 | 1.22 | 1.00-1.47 | 0.044 | 228/486 | 1.43 | 1.11-1.85 | 0.006 |
| | Neohepatic ALBI < -1.615 | 454/1159 | 1 | | | 1 | | | 186/487 | 1 | | |
| Early allograft dysfunction | Neohepatic ALBI \geq -1.615 | 93/982 | 3.55 | 2.38-5.31 | <0.001 | 1.48 | 0.92-2.37 | 0.109 | 30/502 | 1.39 | 0.79-2.47 | 0.256 |
| ., | Neohepatic ALBI < -1.615 | 34/1189 | 1 | | | 1 | | | 22/502 | 1 | | |
| | | Event/n | HR | 95 % CI | P-value | HR | 95 % CI | P-value | Event/n | HR | 95 % CI | P-value |
| Overall graft failure | Neohepatic ALBI ≥ -1.615 | 156/982 | 1.31 | 1.04-1.65 | 0.021 | 1.30 | 1.01-1.68 | 0.041 | 74/502 | 1.55 | 1.08-2.23 | 0.018 |
| | Neohepatic ALBI < -1.615 | 136/1189 | 1 | | | 1 | | | 47/502 | 1 | | |

^a Adjusted by all variables in Table 2. Abbreviations: ALBI, Albumin-Bilirubin score; OR, odds ratio; CI, confidence interval; HR, hazard ratio.

surgery [26]. Additionally, a large study of individuals with coronary artery disease found the ALBI score to be an independent predictor of contrast-associated AKI after elective percutaneous coronary intervention, demonstrating a linear relationship between ALBI scores and the risk of developing this condition [27]. These findings underscore the ALBI score's utility as a predictor of renal outcomes not only in liver disease patients but also in broader clinical scenarios.

A high post-reperfusion ALBI score, marked by low levels of albumin and high levels of bilirubin, may be causally associated with the development of postoperative AKI in LDLT recipients. Albumin improves renal perfusion and glomerular filtration through prolonged potent renal vasodilation, a result of its reaction with oxides of nitrogen to form S-nitroso-albumin [28]. Additionally, albumin inhibits apoptosis of renal tubular cells by scavenging reactive oxygen species and transporting protective lysophosphatidic acid [29,30], while also stimulating renal tubular cell DNA synthesis [31]. Concurrently, an excess of bilirubin negatively impacts renal function through direct bile and bilirubin toxicity, as well as tubular obstruction [32,33]. Thus, the combined effects of hypoalbuminemia and hyperbilirubinemia may compromise tubular integrity and function, contributing to the development of postoperative AKI in LDLT recipients.

As demonstrated in previous literature, we propose that AKI led to an increased risk of RRT and CKD in our patients [2,34,35]. In addition, the ALBI score has been established as a sensitive marker of liver dysfunction with strong prognostic value in patients with chronic liver disease [5–11]. We suggest that this score may also serve as a surrogate marker of early graft function in the post-reperfusion period. Suboptimal graft function may initiate a cascade of adverse physiological processes, including persistent systemic inflammation, impaired immunosuppressant metabolism, hyperbilirubinemia, and deleterious hepatorenal interactions [33,36–38], collectively increasing the risk of CKD.

Further, our findings underscore the impact of neohepatic ALBI on overall graft failure in LDLT recipients. These findings are consistent with results of previous studies investigating the relationship between pre-transplant ALBI scores and post-transplant graft function. Specifically, a higher preoperative ALBI grade (grade III) and ALBI scores (\geq -1.28) were associated with increased risks of EAD

and graft dysfunction after LT, respectively [13,14]. Compared to previous studies, our study included advanced statistical analyses to adjust for confounding variables, thereby more robustly validating the association between neohepatic ALBI scores and EAD or graft failure. We demonstrated that high neohepatic ALBI scores are associated with overall graft failure, both in multivariate Cox proportional hazards regression and PSM analyses.

The significance of our study lies in its findings of the prognostic value of the ALBI score in LDLT recipients, who typically have relatively lower CTP and MELD scores than DDLT recipients. The ALBI score, due to its sensitivity in detecting minor changes in liver function that could not be discerned through the CTP score [11], has proven to be more effective in predicting outcomes in LDLT [13,14]. Our multivariate analysis supported these findings, demonstrating that only the ALBI score, but not MELD-Na score, was independently associated with post-LT AKI. In addition, compared to previous studies that used higher pre-transplant ALBI cutoffs (>-1.48 for AKI [12], >-1.39 for EAD [14], and ≥ -1.28 for graft dysfunction [13]), our study employed a lower cutoff value (≥ -1.615) for the neohepatic ALBI score. This lower cutoff demonstrates enhanced sensitivity in identifying patients at risk for poor postoperative outcomes. Moreover, our study found that neohepatic ALBI was an independent risk factor for AKI and critical long-term outcomes such as CKD and graft failure. Hence, our study contributes to the expanding evidence supporting the prognostic value of the ALBI score in LDLT recipients.

A high neohepatic ALBI score (≥−1.615) may enable early identification of high-risk patients, guiding targeted perioperative management to improve outcomes. In our practice, patients with elevated scores undergo intensified monitoring of renal function (e.g., serial measurements of serum creatinine and estimated GFR) and prompt evaluation of persistent hyperbilirubinemia to assess early graft dysfunction or biliary complications. Fluid balance is optimized to maintain hemodynamic stability, avoiding both hypotension and fluid overload, which can impair renal perfusion or cause renal congestion, respectively [39]. Blood products, crystalloids, albumin, vasopressors, or inotropes are used as needed to achieve this balance. Immunosuppressive regimens, particularly tacrolimus dosing, are carefully adjusted to minimize nephrotoxicity, especially in patients with high neohepatic ALBI scores. Strategies include delaying calcineurin

inhibitor (CNI) initiation or reducing CNI exposure [40–42]. Additional measures include avoiding nephrotoxic medications and initiating early nephrology consultation [39,40]. By facilitating these individualized interventions, the neohepatic ALBI score enhances intraoperative and postoperative care in high-risk patients, potentially reducing the incidence of short- and long-term complications.

Post-LT AKI has been reported to be associated with female sex, higher BMI, alcoholic LC, higher CTP scores, and longer anesthetic time [2,43]. Therefore, the results of our multivariate analysis align with these previously established associations, emphasizing the importance of these factors in contributing to the incidence of post-LT AKI.

In this study, the neohepatic ALBI score was measured 1 h after portal vein reperfusion, before hepatic arterial reperfusion, as LDLT involves portal vein anastomosis followed by hepatic artery and bile duct anastomoses. Consequently, the score reflects early graft function under portal venous flow, capturing the initial response to reperfusion, potentially influenced by ischemia-reperfusion injury and recipient status. While hepatic arterial reperfusion, which occurs later, is critical for long-term graft function, the prognostic value of the neohepatic ALBI score lies in its ability to assess graft performance in the early post-reperfusion period, which may predict subsequent renal and graft outcomes. Future studies could explore the prognostic value of the ALBI score measured after arterial reperfusion, to evaluate the combined effects of portal and hepatic arterial flows.

Our study has some limitations. First, its retrospective nature may inherently introduce bias and confounding factors. However, we used rigorous statistical methods, including multivariate and PSM analyses, to address this limitation. Second, as the research was conducted at a single, highly specialized medical center renowned for its extensive experience in LT [19], the outcomes, including the rates of AKI and graft failure, might not be representative of other institutions with different levels of expertise or patient populations. This necessitates cautious interpretation when generalizing our findings beyond the studied cohort. Lastly, our study's cutoff points for the ALBI score in predicting AKI, derived from ROC curve analyses, were based on data from a population of a single ethnicity at a single medical center. These cutoff values may require adjustment when applied to other ethnic groups. Therefore, to validate our findings, further research involving diverse patient populations across various ethnicities is needed.

5. Conclusions

Our study demonstrates that neohepatic ALBI scores are significantly associated with postoperative AKI, the need for RRT, CKD, and overall graft failure in LDLT recipients. These findings highlight the critical prognostic value of neohepatic ALBI scores in predicting renal and graft outcomes following LDLT.

Funding

This research was supported by a grant from the Korea Health Technology R&D Project through the Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (grant number: HR20C0026), and was also supported by grants (2023IE0008 and 2023IP0134) from the Asan Institute for Life Sciences, Asan Medical Center, Seoul, Korea.

Author contributions

Name: Hye-Won Jeong, M.D.; Contribution: This author helped in Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software,

Supervision, Validation, Visualization, and Writing - original draft and revision; Name: Hye-Mee Kwon, M.D., Ph.D.; Contribution: This author helped in Data curation and Formal analysis; Name: Sung-Hoon Kim, M.D., Ph.D.; Contribution: This author helped in Data curation and Formal analysis; Name: Seong-Mi Yang, M.D.; Contribution: This author helped in Data curation and Formal analysis; Name: In-Gu Jun, M.D.; Contribution: This author helped in Data curation and Formal analysis; Name: Jun-Gol Song, M.D., Ph.D.; Contribution: This author helped in Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, and Writing - original draft and revision; Name: Gyu-Sam Hwang, M.D., Ph.D.; Contribution: This author helped in Conceptualization, Resources and Supervision.

Data availability statement

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Declaration of interests

None.

References

- Thongprayoon C, Kaewput W, Thamcharoen N, Bathini T, Watthanasuntorn K, Lertjitbanjong P, et al. Incidence and impact of acute kidney injury after liver transplantation: a meta-analysis. J Clin Med 2019;8:372. https://doi.org/10.3390/ icm8030372.
- [2] Hilmi IA, Damian D, Al-Khafaji A, Planinsic R, Boucek C, Sakai T, et al. Acute kidney injury following orthotopic liver transplantation: incidence, risk factors, and effects on patient and graft outcomes. Br J Anaesth 2015;114:919–26. https://doi. org/10.1093/bja/aeu556.
- [3] Zongyi Y, Baifeng L, Funian Z, Hao L, Xin W. Risk factors of acute kidney injury after orthotopic liver transplantation in China. Sci Rep 2017;7:41555. https://doi. org/10.1038/srep41555.
- [4] Barri YM, Sanchez EQ, Jennings LW, Melton LB, Hays S, Levy MF, et al. Acute kidney injury following liver transplantation: definition and outcome. Liver Transpl 2009;15:475–83. https://doi.org/10.1002/lt.21682.
- [5] Johnson PJ, Berhane S, Kagebayashi C, Satomura S, Teng M, Reeves HL, et al. Assessment of liver function in patients with hepatocellular carcinoma: a new evidence-based approach-the ALBI grade. J Clin Oncol 2015;33:550–8. https:// doi.org/10.1200/jco.2014.57.9151.
- [6] Wang J, Zhang Z, Yan X, Li M, Xia J, Liu Y, et al. Albumin-bilirubin (ALBI) as an accurate and simple prognostic score for chronic hepatitis B-related liver cirrhosis. Dig Liver Dis 2019;51:1172–8. https://doi.org/10.1016/j.dld.2019.01.0111.
- [7] Fujita K, Oura K, Yoneyama H, Shi T, Takuma K, Nakahara M, et al. Albumin-bilirubin score indicates liver fibrosis staging and prognosis in patients with chronic hepatitis C. Hepatol Res 2019;49:731–42. https://doi.org/10.1111/hepr.13333.
- [8] Chan AW, Chan RC, Wong GL, Wong VW, Choi PC, Chan HL, et al. New simple prognostic score for primary biliary cirrhosis: albumin-bilirubin score. J Gastroenterol Hepatol 2015;30:1391–6. https://doi.org/10.1111/jgh.12938.
- [9] Oikonomou T, Goulis L, Doumtsis P, Tzoumari T, Akriviadis E, Cholongitas E. ALBI and PALBI grades are associated with the outcome of patients with stable decompensated cirrhosis. Ann Hepatol 2019;18:126–36. https://doi.org/10.5604/01.3001.0012.7904.
- [10] Chen B, Lin S. Albumin-bilirubin (ALBI) score at admission predicts possible outcomes in patients with acute-on-chronic liver failure. Med (Baltim) 2017;96: e7142. https://doi.org/10.1097/md.0000000000007142.
- [11] Toyoda H, Johnson PJ. The ALBI score: from liver function in patients with HCC to a general measure of liver function. JHEP Rep 2022;4:100557. https://doi.org/ 10.1016/j.jhepr.2022.100557.
- [12] Ma T, Li QS, Wang Y, Wang B, Wu Z, Lv Y, et al. Value of pretransplant albuminbilirubin score in predicting outcomes after liver transplantation. World J Gastroenterol 2019;25:1879–89. https://doi.org/10.3748/wjg.v25.i15.1879.
- [13] Tai K, Kuramitsu K, Kido M, Tanaka M, Komatsu S, Awazu M, et al. Impact of albumin-bilirubin score on short- and long-term survival after living-donor liver transplantation: a retrospective study. Transpl Proc 2020;52:910–9. https://doi.org/10.1016/j.transproceed.2020.01.020.
- [14] Zhang W, Liu C, Tan Y, Tan L, Jiang L, Yang J, et al. Albumin-bilirubin score for predicting post-transplant complications following adult-to-adult living donor liver transplantation. Ann Transpl 2018;23:639–46. https://doi.org/10.12659/aot.910824.
- [15] Bernardi N, Chedid MF, Grezzana-Filho TJM, Chedid AD, Pinto MA, Leipnitz I, et al. Pre-transplant ALBI grade 3 is associated with increased mortality after liver transplantation. Dig Dis Sci 2019;64:1695–704. https://doi.org/10.1007/s10620-019-5456-6.

- [16] Aggarwal S, Kang Y, Freeman JA, Fortunato Jr. FL, Pinsky MR. Postreperfusion syndrome: hypotension after reperfusion of the transplanted liver. J Crit Care 1993;8:154–60. https://doi.org/10.1016/0883-9441(93)90021-c.
- [17] Pham HP, Shaz BH. Update on massive transfusion. Br J Anaesth 2013;111(Suppl 1):i71–82. https://doi.org/10.1093/bja/aet376.
- [18] Jeong HW, Jung KW, Kim SO, Kwon HM, Moon YJ, Jun IG, et al. Early postoperative weight gain is associated with increased risk of graft failure in living donor liver transplant recipients. Sci Rep 2019;9:20096. https://doi.org/10.1038/s41598-019-56543-3.
- [19] Lee SG. A complete treatment of adult living donor liver transplantation: a review of surgical technique and current challenges to expand indication of patients. Am J Transpl 2015;15:17–38. https://doi.org/10.1111/ajt.12907.
- [20] Hwang S, Ahn CS, Kim KH, Moon DB, Ha TY, Song GW, et al. A cross-sectional analysis of long-term immunosuppressive regimens after liver transplantation at Asan Medical Center: increased preference for mycophenolate mofetil. Ann Hepatobiliary Pancreat Surg 2018;22:19–26. https://doi.org/10.14701/ahbps.2018.22.1.19.
- [21] Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pr 2012;120:c179–84. https://doi.org/10.1159/000339789.
- [22] Jun IG, Kwon HM, Jung KW, Moon YJ, Shin WJ, Song JG, et al. The impact of postre-perfusion syndrome on acute kidney injury in living donor liver transplantation: a propensity score analysis. Anesth Analg 2018;127:369–78. https://doi.org/10.1213/ane.000000000003370.
- [23] Kidney disease: improving global outcomes (KDIGO) CKD work group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int 2024;105:S117–314. https://doi.org/10.1016/ji.kint.2023.10.018.
- [24] Olthoff KM, Emond JC, Shearon TH, Everson G, Baker TB, Fisher RA, et al. Liver regeneration after living donor transplantation: adult-to-adult living donor liver transplantation cohort study. Liver Transpl 2015;21:79–88. https://doi.org/ 10.1002/lt.23966.
- [25] Hayashi M, Abe K, Fujita M, Okai K, Takahashi A, Ohira H. Acute kidney injury after platinum-based transcatheter arterial chemoembolization and transarterial infusion chemotherapy in patients with hepatocellular carcinoma. Jpn J Clin Oncol 2020;50:36–43. https://doi.org/10.1093/jjco/hyz129.
- [26] Duman ZM, Timur B. Albumin-bilirubin score: a novel mortality predictor in valvular surgery. Braz J Cardiovasc Surg 2023;38:271–7. https://doi.org/10.21470/ 1678-9741-2022-0008.
- [27] Chen JH, Zhang LW, Lin ZJ, Chen XF, Chen LC, Wang CX, et al. The association between the albumin-bilirubin score and contrast-associated acute kidney injury in patients undergoing elective percutaneous coronary intervention. Angiology 2024;33197241228051. https://doi.org/10.1177/00033197241228051.
- [28] Kaufmann MA, Castelli I, Pargger H, Drop LJ. Nitric oxide dose-response study in the isolated perfused rat kidney after inhibition of endothelium-derived relaxing factor synthesis: the role of serum albumin. J Pharmacol Exp Ther 1995;273:855– 62 https://www.ncbi.nlm.nih.gov/pubmed/7752090.
- [29] Iglesias J, Abernethy VE, Wang Z, Lieberthal W, Koh JS, Levine JS. Albumin is a major serum survival factor for renal tubular cells and macrophages through scavenging of ROS. Am J Physiol 1999;277:F711–22. https://doi.org/10.1152/ ajprenal.1999.277.5.F711.

- [30] Levine JS, Koh JS, Triaca V, Lieberthal W. Lysophosphatidic acid: a novel growth and survival factor for renal proximal tubular cells. Am J Physiol 1997;273:F575– 85. https://doi.org/10.1152/ajprenal.1997.273.4.F575.
- [31] Lee YJ, Han HJ. Albumin-stimulated DNA synthesis is mediated by Ca2+/PKC as well as EGF receptor-dependent p44/42 MAPK and NF-kappaB signal pathways in renal proximal tubule cells. Am J Physiol Ren Physiol 2008;294:F534–41. https:// doi.org/10.1152/aiprenal.00408.2007.
- [32] Fickert P, Krones E, Pollheimer MJ, Thueringer A, Moustafa T, Silbert D, et al. Bile acids trigger cholemic nephropathy in common bile-duct-ligated mice. Hepatology 2013;58:2056–69. https://doi.org/10.1002/hep.26599.
- [33] van Slambrouck CM, Salem F, Meehan SM, Chang A. Bile cast nephropathy is a common pathologic finding for kidney injury associated with severe liver dysfunction. Kidney Int 2013;84:192-7. https://doi.org/10.1038/ki.2013.78.
- [34] Barreto AG, Daher EF, Silva Junior GB, Garcia JH, Magalhães CB, Lima JM, et al. Risk factors for acute kidney injury and 30-day mortality after liver transplantation. Ann Hepatol 2015;14:688–94.
- [35] Velidedeoglu E, Bloom RD, Crawford MD, Desai NM, Campos L, Abt PL, et al. Early kidney dysfunction post liver transplantation predicts late chronic kidney disease. Transplantation 2004;77:553–6. https://doi.org/10.1097/01.tp.0000114609.99558.41.
- [36] Wadei HM, Lee DD, Croome KP, Mai ML, Golan E, Brotman R, et al. Early allograft dysfunction after liver transplantation is associated with short- and long-term kidney function impairment. Am J Transpl 2016;16:850-9. https://doi.org/ 10.1111/ajt.13527.
- [37] Wilkinson GR. Drug metabolism and variability among patients in drug response. N Engl J Med 2005;352:2211–21. https://doi.org/10.1056/NEJMra032424.
- [38] Ott R, Rupprecht H, Born G, Müller V, Reck T, Hohenberger W, et al. Plasma separation and bilirubin adsorption after complicated liver transplantation: a therapeutic approach to excessive hyperbilirubinemia. Transplantation 1998;65:434-7. https://doi.org/10.1097/00007890-199802150-00025.
- [39] Dong V, Nadim MK, Karvellas CJ. Post–liver transplant acute kidney injury. Liver Transpl 2021;27:1653–64. https://doi.org/10.1002/lt.26094.
- [40] Durand F, Francoz C, Asrani SK, Khemichian S, Pham TA, Sung RS, et al. Acute kidney injury after liver transplantation. Transplantation 2018;102:1636–49. https://doi.org/10.1097/tp.000000000002305.
- [41] Neuberger JM, Mamelok RD, Neuhaus P, Pirenne J, Samuel D, Isoniemi H, et al. Delayed introduction of reduced-dose tacrolimus, and renal function in liver transplantation: the 'ReSpECT' study. Am J Transpl 2009;9:327–36. https://doi. org/10.1111/j.1600-6143.2008.02493.x.
- [42] Kong Y, Wang D, Shang Y, Liang W, Ling X, Guo Z, et al. Calcineurin-inhibitor minimization in liver transplant patients with calcineurin-inhibitor-related renal dysfunction: a meta-analysis. PLoS One 2011;6:e24387. https://doi.org/10.1371/ journal.pone.0024387.
- [43] Park MH, Shim HS, Kim WH, Kim HJ, Kim DJ, Lee SH, et al. Clinical risk scoring models for prediction of acute kidney injury after living donor liver transplantation: a retrospective observational study. PLoS One 2015;10:e0136230. https:// doi.org/10.1371/journal.pone.0136230.