Incidence and Risk Factors of Acute Kidney Injury in Pediatric Liver Transplant Patients: A Retrospective Study

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Abstract

Background: Acute kidney injury (AKI) significantly contributes to the mortality and morbidity rates among pediatric liver transplant (LT) recipients.

Objective: Our study aimed to assess the potential factors contributing to AKI in pediatric LT patients and to analyze the impact of AKI on postoperative mortality and hospitalization duration.

Materials and methods: About 235 pediatric LT patients under the age of 18 between the years 2015 and 2021 were evaluated retrospectively. The relationship between preoperative and intraoperative variables of the patients and AKI developed when the early postoperatıve period was assessed.

Results: A correlation was found between the patients' preoperative age, albumin levels, and AKI. AKI was found to be associated with the duration of surgery and intraoperative blood transfusion.

Conclusion: Our findings revealed that the severity of AKI in pediatric LT patients is linked to extended surgical durations and increased blood transfusions resulting from hemodynamically compromised blood loss. Furthermore, independent risk factors for AKI were identified as prolonged warm ischemia and the overall duration of the operation.

Keywords: Acute kidney injury, Liver transplantation, Pediatric transplantation.

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Highlights

Acute kidney injury (AKI) is a complication that increases mortality and morbidity, which is frequently seen in the pediatric patient group undergoing liver transplant (LT). We aimed to investigate and emphasize the preventable causes of AKI.

Introduction

Liver transplantation is the preferred treatment for end-stage liver disease in both adult and pediatric populations.[1] Among hospitalized individuals, acute kidney injury is prevalent and is linked to a nearly tenfold rise in in-patient mortality rates. Acute kidney injury occurs in approximately 20–47% of cases, and when it progresses to stage 3, it is correlated with a substantial in-hospital mortality rate, reaching up to 71%.[2,3] After pediatric liver transplantation, the occurrence of AKI has been linked to extended periods of mechanical ventilation, prolonged stays in the intensive care unit, and an elevated risk of mortality. It has been highlighted that the development of AKI may result in chronic kidney disease, end-stage renal disease, and ultimately, mortality in affected patients.[4]

Research on AKI in adult patients has demonstrated that several factors, including female gender, obesity levels, diabetes mellitus, a high model for end-stage liver disease (MELD) score, extensive bleeding during surgery, the administration of hydroxyethyl starch solution, uncontrolled perioperative blood sugar, ischemia duration, and graft size, contribute to the occurrence of AKI.[5,6] The occurrence of AKI in pediatric patients is probable to stem from various factors, including patient-related characteristics, preoperative organ function features, donor-related elements, as well as intraoperative factors, and postoperative outcomes, indicating a multifaceted etiology. Identifying high-risk factors for AKI in pediatric patients is crucial, as it can impact the management of hemodynamic parameters during surgery, guide fluid management preferences, and influence protocols for immunosuppression and antibiotic treatment.[7,8] However, the number of patients in the literature for pediatric patients is small, we aimed to conduct a study with a larger number of patients and to examine the relationship between many factors in the preoperative and intraoperative process.

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Materials and Methods

This study was conducted retrospectively by analyzing the in-hospital data of patients who underwent LT in the İnönü

Table 1: Evaluation of the relationships between preoperative data and AKI

	No AKI (n = 175)	*AKI I (n = 26)*	*AKI II (n = 20)*	*AKI III (n = 14)*	*p-value*
Age (years)	4.2 ± 4.1	6.6 ± 4.8	2.9 ± 2.3	2.2 ± 2.7	$0.002^{\alpha,\beta,\Phi}$
Gender (Male, %)	105 (60.7)	19 (67.9)	8 (40.0)	9 (64.3)	0.241
Creatinine (mg/dL)	0.4 ± 0.2	0.5 ± 0.1	0.5 ± 0.1	0.4 ± 0.1	0.064
Albumin (gm/dL)	3 ± 0.6	3.1 ± 0.6	3 ± 0.6	2.6 ± 0.5	0.083
PT (second)	22.2 ± 11.0	23.6 ± 10.7	20.8 ± 8.3	24.2 ± 13	0.755
PTT (second)	38.9 ± 20.6	44.3 ± 27.4	35.8 ± 11.1	40. ± 12.8	0.530
Ascites (mL)	0.24 ± 0.4	0.4 ± 0.5	0.3 ± 0.4	0.1 ± 0.2	0.087
Tbil (mg/dL)	15.8 ± 13.5	14.8 ± 12	17.2 ± 16.6	18.4 ± 13.5	0.839

$\alpha = p < 0.05$ in group 0 vs group I, $\beta = p < 0.05$ in group 0 vs group II, $\Phi = p < 0.05$ in group I vs group III

University Liver Transplant Institute between 2015 and 2021, after receiving ethics committee approval (2022/3318). The hospital's database was searched for children aged under 18 years who had LT during the study period. Patients with pre-LT end-stage renal disease and preoperative creatinine elevation, patients with normal preoperative laboratory data and hepatorenal syndrome clinic, and patients with missing data in the electronic data system were excluded from the study. Files of 276 patients were reached. About 36 patients were excluded due to lack of data and five patients were excluded because of high preoperative creatinine levels. So 235 patients were evaluated.

Age, gender, and transplantation indication of the patients were recorded. Preoperative data included the presence of ascites, laboratory test results (serum creatinine, coagulation parameters [partial thromboplastin time (PTT), prothrombin time (PT), international normalized ratio (INR)], albumin values, total bilirubin (T BIL), intraoperative variables consisted of operation time, amount of total fluid, crystalloid fluid, colloid, albumin, blood transfusion, urine output, warm ischemia time (WIT) and cold ischemia time (CIT) and the use of diuretics. During the postoperative period, length of stay in intensive care unit and hospital, mortality, and creatinine values at postoperative 1, 2, and 7 days were recorded.

Acute kidney injury grading was made according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria.[6,9] The patients were classified as no AKI (Group 0) and grades I, II, and III (Groups I to III respectively) AKI according to the KIDIGO classification based on their postoperative creatinine values.

Grade I: It was defined as a 1.5–1.9-fold increase in the preoperative creatinine value compared with the peak creatinine value in the first 7 days or an increase of ≥0.3 mg/dL within 48 hours.

Grade II: It was defined as a 2–2.9-fold increase in the preoperative creatinine value compared with the peak creatinine value in the first 7 days.

Grade III: It was defined as the preoperative creatinine level ≥4.0 mg/dL higher than the highest creatinine level in the first 7 postoperative days or a 3-fold increase.

Anesthetic drugs used for liver transplantation have been standardized in our institution: anesthesia is induced using pentothal (3–8 mg/kg) fentanyl (1–2 μg/kg/min), and maintained with sevoflurane (0.5–1%) and remifentanil (0.1–0.3 μg/kg/min). The first choice used during surgery was norepinephrine, although the use of vasopressors was still decided by the anesthesiologist. In our clinic, intraoperative corticosteroid administration is applied at 10 mg/kg after arterial anastomosis. In the postoperative period, corticosteroid treatment is continued by titrating the dose and if there is no contraindication, tacrolimus is added to the treatment on the 3rd day. Maintenance doses were based on daily blood levels of tacrolimus.

Statistical Analysis

Statistical analyses were conducted using JASP software (version 0.14.1.0). Continuous variables are presented as mean ± standard deviation, while categorical variables are reported as numbers and percentages. We assessed the normality of variables using the Shapiro–Wilk test. To examine differences between continuous variables, either one-way ANOVA or the Kruskal–Wallis test was employed, with the Bonferroni test applied as a post hoc analysis. Categorical variables were compared using the Chi-square test. The associations between variables were examined through either Pearson or Spearman correlation analysis, chosen based on the nature of the data. We utilized multiple logistic regression analysis to ascertain the independent variables that predict the occurrence of both AKI and mortality. For univariate analysis, regression analysis was conducted for p-values < 0.10. Statistically significant results were defined as p-values < 0.05.

Results

Biliary atresia emerged as the predominant cause of liver transplantation, with primary sclerosing cholangitis, autoimmune hepatitis, and metabolic liver disease following as the subsequent most frequent causes, in that order. When preoperative data were evaluated, in the study including 235 patients, the mean age of the patients was 4.3 ± 4.2, and the mean age was significantly higher in patients with AKI I than in the other groups. ($p < 0.05$, for all). The other preoperative data, including gender, creatinine, albumin, PT, PTT, acid, and bilirubin were similar in all groups (for all, $p > 0.05$) (Table 1).

According to the intraoperative data (Table 2), cold and WITs were found to be similar between groups ($p > 0.05$). However, there was a statistically significant difference between the groups in terms of operative time and total amount of fluid and crystalloids. (for all, $p < 0.05$). In the group of patients with AKI III, the duration of surgery was found to be significantly longer in patients who developed AKI III than in other patients. Again, there was a difference between the groups in terms of blood transfusion, albumin, furosemide, and total urine amount (for all, $p < 0.05$) (Table 2).

Upon examining the patients' duration of stay in the intensive care unit and mortality rates (Table 3), it was determined that the overall mortality rate stood at 26%. Even though group III exhibited

Table 2: Evaluation of the relationships between intraoperative data and AKI

	No AKI (n = 175)	*AKI I (n = 26)*	*AKI II (n = 20)*	*AKI III (n = 14)*	*p-value*
CIT (min)	107.5 ± 101.5	101.7 ± 73.1	94 ± 51.9	103.2 ± 51.9	0.972
WIT (min)	55.8 ± 17.5	56.6 ± 15.1	56.7 ± 17.3	67 ± 20.6	0.181
Op time (min)	421.1 ± 107.4	470.3 ± 111.3	509.2 ± 103.9	521.8 ± 154.3	<0.001$^{\beta,¥}$
Total fluid (mL/kg/h)	19.42_{a} ± 16.70	42.86_{b} ± 89.32	55.68_{b} ± 38.96	$30.41_{a,b}$ ± 44.96	0.001
Total crystalloid (mL/kg/h)	16.17_{a} ± 16.40	35.31_{b} ± 84.41	48.44_{b} ± 33.40	$25.49_{a,b}$ ± 45.68	0.001
Total colloid (mL/kg/saat)	1.14_{a} ± 1.87	$1.94_{a,b}$ ± 2.50	2.52_{b} ± 3.95	$0.95_{a,b}$ ± 1.68	0.249
Blood transfusion (mL/kg)	7.83_{a} ± 13.14	35.13_{b} ± 72.97	25.17_{b} ± 52.98	31.17_{b} ± 52.14	0.001
Albumin (%20) (mL/kg)	5.64_{a} ± 5.28	$12.87_{a,b}$ ± 37.06	10.67_{b} ± 11.30	$9.30_{a,b}$ ± 4.97	0.014
Furosemide (mg/kg)	0.73_{a} ± 0.87	0.67_{a} ± 0.29	1.51_{a} ± 0.94	0.83_{a} ± 0.26	0.002
Mannitol (mL/kg)	2.93_{a} ± 2.06	8.39_{b} ± 5.81	5.36[1]	2.22[1]	0.148
Urine output (mL/kg/h)	2.63_{a} ± 2.19	$5.26_{b,c}$ ± 9.28	9.03_{b} ± 6.40	$1.77_{a,c}$ ± 1.31	0.004

β = $p < 0.05$ in group 0 vs group II, ¥ = $p < 0.05$ in group 0 vs group III; Values in the same row and subtable not sharing the same subscript are significantly different at $p < 0.05$ in the two-sided test of equality for column means. Cells with no subscript are not included in the test. Tests assume equal variances.[2] This category is not used in comparisons because the sum of case weights is less than two.[1] Tests are adjusted for all pairwise comparisons within a row of each innermost subtable using the Bonferroni correction.[2]

Table 3: Effects of AKI on length of stay in intensive care unit and mortality

	No presence of AKI	*AKI I*	*AKI II*	*AKI III*	*p-value**
ICU stay (days)	26.6 ± 31.1	22.7 ± 18.1	26.4 ± 20.5	12.5 ± 21.1	0.334
Hospital stay (days)	53.2 ± 41.8	45.8 ± 32.1	40.2 ± 19.3	14 ± 21.3	0.002¥
Mortality	34 (19.7%)	4 (14.3%)	11 (55.0%)	12 (85.7%)	<0.001

¥ = $p < 0.05$ in group 0 vs group III

Table 4: Predictors of AKI

	Univariate analysis		*Multivariate analysis*		
Parameters	*r*	*p-value*	*OR*	*95% CI*	*p-value*
Age	0.024	0.718			
Gender	–0.024	0.716			
Prealbumin	–0.016	0.806			
WIT	0.123	0.060	1.023	1.004–1.043	0.020
Operation time	0.261	<0.001	1.005	1.002–1.009	0.004

Table 5: Predictors of mortality

	Univariate analysis		*Multivariate analysis*		
Parameters	*r*	*p-value*	*OR*	*95% CI*	*p-value*
Age	–0.217	0.001			
Gender	0.048	0.468			
Prealbumin	–0.206	0.002	0.415	0.239–0.721	0.002
Warm ischemia time	0.063	0.336			
Operation time	0.116	0.077			
AKI	0.240	<0.001	3.134	1.355–7.250	0.008

the shortest hospital stay, it also experienced the highest mortality rate within the groups. Groups II and III had higher mortality rates compared with the remaining groups.

Operation time and the duration of warm ischemia were incorporated as independent variables in the multiple logistic regression analysis conducted to identify factors independently predicting AKI. We found that all of these variables were independent predictors of AKI (Table 4).

Independent variables including age, prealbumin, and anhepatic time were subjected to the multiple logistic regression analysis performed to determine the independent predictors affecting mortality. Within these factors, prealbumin ($p = 0.002$) and AKI ($p = 0.008$) emerged as independent predictors of mortality (Table 5).

Discussion

Our study findings suggest that the severity of AKI is linked to prolonged operation time and increased blood transfusion necessitated by intraoperative blood loss. Prolonged cold ischemia and duration of surgery have been identified as risk factors for AKI. Furthermore, mortality risk in pediatric LT patients is heightened by factors, such as younger age, lower preoperative albumin levels, and the occurrence of AKI in the early postoperative phase, all identified as independent risk factors. Upon reviewing the existing literature, studies examining risk factors for AKI following pediatric LT were found to have a restricted number of cases. Our single-center analysis stands out for reporting the highest number of cases in this context.

Acute kidney injury is an important postoperative complication of pediatric LT. In a study by Erdost et al.[10] with 440 adults who had an LT, RIFLE criteria were found to be less sensitive than AKIN and KDIGO criteria in the diagnosis of AKI. Therefore, we used KDIGO criteria in the diagnosis and classification of AKI in our study. In a retrospective study evaluating the development of AKI after pediatric LT, when the frequency of AKI was studied by Hamada et al.,[11] AKI developed in 46.2% of the patients. When evaluated according to AKI staging, it was emphasized that 21.8% were stage I, 20.5% were stage II and 3.8% were stage III. In a similar study conducted by Şahintürk et al., they reported the incidence of AKI

as 34.2% and emphasized that while stage I AKI was 18%, stage III AKI was observed at a rate of 6%.[7]

Similarly, Nahum et al. reported the total incidence of AKI as 43–57%, with severe AKI in approximately 23% of cases.[6] In our study, while 26.4% of the patients developed AKI, stage I was found in 11.9%, stage II in 8.5%, and stage III in 6% of the patients. We think that the low rates in our study are due to the higher number of patients compared with other studies. In addition, we excluded patients with preoperative renal dysfunction, since we aimed to investigate the factors causing AKI.

Studies in pediatric patient populations have indicated that renal function does not fully mature until 2 years of age, indicating a period of relatively limited renal capacity.[12] Our observations revealed a correlation between stage III AKI and younger age, with a mean age of 2.2 ± 2.7 years in stage III. This association is believed to stem from the ongoing development of renal function during this phase.

There was a notable association observed in normal adult patients, linking preoperative creatinine levels with an elevation in preoperative INR and an increased risk of AKI. This outcome indicates a connection between the extent of liver disease severity and the likelihood of developing AKI post-surgery.[13] In our study, no correlation was found between the preoperative INR, APTT, and PT time and AKI. In a study conducted in pediatric patients, an increase in preoperative total bilirubin value was underlined as a risk factor for AKI.[11] In studies involving adults, a noteworthy association was observed between a higher MELD score and the occurrence of AKI. Additionally, bilirubin levels were found to exhibit a significant correlation with AKI in the adult population. Our interpretation of these findings is influenced by the pediatric nature of the patient group under investigation, coupled with the substantial number of patients included in our study.

Preoperative hypoalbuminemia was independently associated with postoperative AKI in non-cardiac surgeries. Li et al.[14] reported in a retrospective study of 729 patients following non-cardiac surgery that preoperative hypoalbuminemia was an independent risk factor for the development of AKI. Similarly, hypoalbuminemia has been reported as a risk factor for AKI after LT in adults.[13,15] In our study, we observed that preoperative hypoalbuminemia was low in all patient groups, it was not associated with the severity of AKI, but it was independently correlated with mortality.

Li et al. investigated the impact of sevoflurane and propofol infusion on AKI incidence in their study involving 120 pediatric LT patients. They observed a reduced occurrence of AKI during the maintenance of anesthesia with sevoflurane.[16] At our clinic, sevoflurane is used for the maintenance anesthesia in all LT patients, necessitating additional studies to compare and evaluate this effect.

The duration of ischemia (both cold and WIT) is influenced by a range of factors, encompassing donor characteristics and the overall quality of the graft. Cold ischemia longer than 12 hours and warm ischemia longer than 1 hour are considered risk factors for graft failure. Iskender Ijtsma et al.[17] emphasized that the duration of the anhepatic phase independently graft dysfunction. They also mentioned that this finding does not completely account for the association between the anhepatic phase duration and the occurrence of AKI. Prior research has highlighted that an extended anhepatic phase may lead to renal congestion and impairment of renal function. In a study involving 24 patients without preexisting renal disease, a marked reduction in urine output was observed for all patients after the clamping of the inferior vena cava and portal veins in the operating room. In addition, hemodynamic parameters are significantly affected during the anhepatic phase when performing LT.[18–20] Decreased renal functions and reduced glomerular filtration rates may cause kidney function damage. In our investigation, while no significant correlation was noted between the severity of AKI and the durations of cold and warm ischemia, WIT emerged as an independent risk factor for AKI development, aligning with findings from other studies. We think that hemodynamic changes and decrease in renal functions due to hypotension during an anhepatic period would cause AKI.

In addition to other factors, operative time and bleeding severity were associated with AKI in patients undergoing LT. Blood loss seems to serve as an indicator of adverse events in surgery. Hemodynamic instability or anemia arising from significant bleeding may decrease oxygen delivery to the kidneys, leading to kidney injury. The administration of blood products during surgery has been suggested as a factor contributing to the risk of developing AKI.[11] It is emphasized that intraoperative blood transfusions increase the risk of AKI in LT patients. In a review of 100 cadaver orthotopic liver transplantations, it was noted that the need for more than 10 units of packed red cells and decreased diuresis were important predictors of renal dysfunction.[21] Koo et al. showed that 2.36 ± 2.4 units of packed RBC transfusion was significantly associated with postoperative renal dysfunction with a relative risk of 1.25.[22] Similarly, Ferah et al.[23] emphasized the relationship between operative time, prolonged anhepatic phase time, and intraoperative blood loss among these intraoperative values and AKI. Similar to previous studies in adult LT patients, in our study, intraoperative blood transfusion was found to be an independent risk factor for AKI. In patients requiring blood products, fluid, and albumin requirements were also increased. We think that blood product transfusion may trigger a systemic inflammatory response and deterioration in hemodynamic parameters may cause AKI. Since retrospective data were evaluated in our study, hemodynamic data could not be evaluated and prospective data on this subject are needed.

Diuretic-induced volume reduction is likely to exacerbate kidney damage. In the pediatric LT study conducted by Ferah et al.[23] high-dose furosemide use was related to AKI. In our clinic, furosemide is administered at a dose of 1–2 mg/kg as a diuretic to the LT patient group whose intraoperative hourly urine output is below 0.5 mL/kg. Similarly, in our study, the use of furosemide was found to be lower in the group that did not develop AKI. We think that oliguria and not using the diuretic group have a common effect on the development of AKI. We think that the significant difference in the fluid therapy given to the patients is related to the length of the case duration.

Ehab et al.[24] stated in their study that the duration of surgery is a risk factor for AKI in LT. Likewise, in our study, we concluded that the operation time is a predictive factor for AKI. We think that this is due to the increased duration of renal hypoperfusion and oliguria caused by prolonged cold and warm ischemia.

Exposure to a nephrotoxic drug is an important risk factor for AKI and treatment is administered according to institutional protocol.[25] It could not be analyzed in our study due to the lack of data on additional doses in treatment changes. Additional research is needed in this domain, as these factors are likely influential in the occurrence of AKI among patients undergoing liver transplantation.

In previous studies involving all patient groups, the relationship between AKI and morbidity has been emphasized.[4–8] In a study including pediatric patients, it was reported that patients who developed AKI had prolonged length of stay on mechanical ventilation as well as prolonged NICU stay.[20] Findings from a study

involving 150 pediatric patients yielded comparable outcomes, demonstrating extended durations of hospitalization and intensive care stays.[9]

AKI also increases the ICU length of stay, the total length of hospital stay, and the mortality rate. In our study, it was observed that the mortality rate was 85.7% in patients who developed AKI stage III AKI, and this rate was significantly lower in patients without AKI and in patients with AKI stage I. In our study, unlike other studies, the length of hospital stay was the longest in the group without AKI stage I, while it was the shortest in AKI stage III. We think that this situation is related to the mortality rate of the patients.

Conclusion

In our study, prolongation of WIT and operation time are predictive factors for AKI. Long operation time and intraoperative blood transfusion increase the severity of AKI. We think that knowing the causes of AKI will be effective in preventing AKI. It should be supported by a multicenter prospective study investigating possible risk factors.

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References

1. Rawal N, Yazigi N. Pediatric Liver Transplantation. Pediatr Clin North Am 2017;64(3):677–684. DOI: 10.1016/j.pcl.2017.02.003.
2. Wong F, O'Leary JG, Reddy KR, Garcia-Tsao G, Fallon MB, Biggins SW, et al. Acute kidney ınjury in cirrhosis: Baseline serum creatinine predicts patient outcomes. Am J Gastroenterol 2017;112(7):1103–1110. DOI: 10.1038/ajg.2017.122.
3. Belcher JM, Garcia-Tsao G, Sanyal AJ, Bhogal H, Lim JK, Ansari N, et al. Association of AKI with mortality and complications in hospitalized patients with cirrhosis. Hepatology 2013;57(2):753–762. DOI: 10.1002/hep.25735.
4. Rahman S, Davidson BR, Mallett SV. Early acute kidney injury after liver transplantation: Predisposing factors and clinical implications. World J Hepatol 2017;9(18):823–832. DOI: 10.4254/wjh.v9.i18.823.
5. Sahinturk H, Ozdemirkan A, Zeyneloglu P, Gedik E, Pirat A, Haberal M. Early postoperative acute kidney ınjury among pediatric liver transplant recipients. Exp Clin Transplant 2021;19(7):659–663. DOI: 10.6002/ect.2018.0214.
6. Nahum E, Kadmon G, Kaplan E, Weissbach A, Hijazi H, Haskin O, et al. Prevalence of acute kidney injury after liver transplantation in children: Comparison of the pRIFLE, AKIN, and KDIGO criteria using corrected serum creatinine. J Crit Care 2019;50:275–279. DOI: 10.1016/j.jcrc.2019.01.010.
7. Jun IG, Lee B, Kim SO, Shin WJ, Bang JY, Song JG, et al. Comparison of acute kidney injury between ABO-compatible and ABO-incompatible living donor liver transplantation: A propensity matching analysis. Liver Transpl 2016;22(12):1656–1665. DOI: 10.1002/lt.24634.
8. Kim JM, Jo YY, Na SW, Kim SI, Choi YS, Kim NO, et al. The predictors for continuous renal replacement therapy in liver transplant recipients. Transplant Proc 2014;46(1):184–191. DOI: 10.1016/j.transproceed.2013.07.075.
9. Selewski DT, Cornell TT, Heung M, Troost JP, Ehrmann BJ, Lombel RM, et al. Validation of the KDIGO acute kidney injury criteria in a pediatric critical care population. Intensive Care Med 2014;40(10):1481–1488. DOI: 10.1007/s00134-014-3391-8.
10. Aksu Erdost H, Ozkardesler S, Ocmen E, Avkan-Oguz V, Akan M, Iyilikci L, et al. Acute renal ınjury evaluation after liver transplantation: With RIFLE criteria. Transplant Proc 2015;47(5):1482–1487. DOI: 10.1016/j.transproceed.2015.04.065.
11. Hamada M, Matsukawa S, Shimizu S, Kai S, Mizota T. Acute kidney injury after pediatric liver transplantation: Incidence, risk factors, and association with outcome. J Anesth 2017;31(5):758–763. DOI: 10.1007/s00540-017-2395-2.
12. Schwartz GJ, Work DF. Measurement and estimation of GFR in children and adolescents. Clin J Am Soc Nephrol 2009;4(11):1832–1843. DOI: 10.2215/CJN.01640309.
13. Sanchez EQ, Gonwa TA, Levy MF, Goldstein RM, Mai ML, Hays SR, et al. Preoperative and perioperative predictors of the need for renal replacement therapy after orthotopic liver transplantation. Transplantation 2004;78(7):1048–1054. DOI: 10.1097/01.tp.0000137176.95730.5b.
14. Li N, Qiao H, Guo JF, Yang HY, Li XY, Li SL, et al. Preoperative hypoalbuminemia was associated with acute kidney injury in high-risk patients following non-cardiac surgery: A retrospective cohort study. BMC Anesthesiol 2019;19(1):171. DOI: 10.1186/s12871-019-0842-3.
15. Erdost HA, Ozkardesler S, Akan M, Iyilikci L, Unek T, Ocmen E, et al. Comparison of the RIFLE, AKIN, and KDIGO Diagnostic Classifications for Acute Renal Injury in Patients Undergoing Liver Transplantation. Transplant Proc 2016;48(6):2112–2118. DOI: 10.1016/j.transproceed.2016.03.044.
16. Li H, Weng Y, Yuan S, Liu W, Yu H, Yu W. Effect of sevoflurane and propofol on acute kidney injury in pediatric living donor liver transplantation. Ann Transl Med 2019;7(14):340. DOI: 10.21037/atm.2019.06.76.
17. Ijtsma AJ, van der Hilst CS, de Boer MT, de Jong KP, Peeters PM, Porte RJ, et al. The clinical relevance of the anhepatic phase during liver transplantation. Liver Transpl 2009;15(9):1050–1055. DOI: 10.1002/lt.21791.
18. Li SR, Shen N, Yi HM, Gan XL, Hei ZQ. [Changes of systemic and pulmonary hemodynamics and plasma levels of inducible nitric oxide synthase and endothelin-1 in patients with hepatopulmonary syndrome]. Nan Fang Yi Ke Da Xue Xue Bao 2009;29(10):2030–2032. PMID: 19861258.
19. Li ZX, Wang MC, Zhang YC, Mao J, Chen M, Ni R, et al. Hemodynamics and vasoactive substance levels during renal congestion that occurs in the anhepatic phase of liver transplantation. World J Gastroenterol 2015;21(18):5482–5487. DOI: 10.3748/wjg.v21.i18.5482.
20. Zhang Y, Xiang B, Wu Y, Xie X, Wang J, Jin S. Risk factors and associated outcomes of early acute kidney injury in pediatric liver transplant recipients: A retrospective study. J Pediatr Surg 2020;55(3):446–450. DOI: 10.1016/j.jpedsurg.2019.07.019.
21. Guitard J, Cointault O, Kamar N, Muscari F, Lavayssière L, Suc B, et al. Acute renal failure following liver transplantation with induction therapy. Clin Nephrol 2006;65(2):103–112. DOI: 10.5414/cnp65103.
22. Koo M, Sabaté A, Ramos E, Dalmau A, León E, Fabregat J, et al. Factores relacionados con la disfunción renal postoperatoria en pacientes trasplantados hepáticos con función preoperatoria normal [Factors related to renal dysfunction after liver transplantation in patients with normal preoperative function]. Rev Esp Anestesiol Reanim 2006;53(9):538–544. PMID: 17297829.
23. Ferah O, Akbulut A, Açik ME, Gökkaya Z, Acar U, Yenidünya Ö, et al. Acute kidney ınjury after pediatric liver transplantation. Transplant Proc 2019;51(7):2486–2491. DOI: 10.1016/j.transproceed.2019.01.179.
24. Abdel-Khalek EE, Alrefaey AK, Yassen AM, Monier A, Elgouhari HM, Habl MS, et al. Renal dysfunction after living-donor liver transplantation: Experience with 500 cases. J Transplant 2018;2018(5):1–9. 5910372. DOI: 10.1155/2018/5910372.
25. Kwong A, Kim WR, Lake JR, Smith JM, Schladt DP, Skeans MA, et al. OPTN/SRTR 2018 annual data report: Liver. Am J Transplant 2020;20 Suppl s1:193–299. DOI: 10.1111/ajt.15674.