

Preoperative Hepatic Steatosis as a Postoperative Acute Renal Damage Predictor in Isolated Coronary Artery Bypass Grafting Surgery?

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Abstract

Aim: The aim of this study was to evaluate the prognostic effect of preoperative hepatic steatosis on the development of postoperative acute kidney injury (AKI) in patients undergoing isolated coronary artery bypass grafting (CABG). **Methods:** This retrospective, single-center cohort study included 146 patients who underwent isolated CABG between January 2020 and December 2022. Preoperative hepatic steatosis was assessed using non-contrast computed tomography (CT) images and quantitatively measured using the liver attenuation index (LAI). Patients were divided into three groups based on LAI values: mild, moderate, and severe hepatic steatosis. **Results:** Postoperative AKI developed in 26% of the patients included in the study. The distribution of hepatic steatosis was determined as follows: mild in 67.1% of patients, moderate in 21.09% of patients, and severe hepatic steatosis in 11.0% of patients. In multivariate logistic regression analysis, severe hepatic steatosis, age (OR: 1.09 per year; 95% CI: 1.02–1.14; $p=0.008$), and diabetes mellitus (OR: 2.35; 95% CI: 1.11–4.95; $p=0.025$) were identified as independent predictors of postoperative AKI. The effect of severe hepatic steatosis on the development of AKI was found to be strongly significant (OR: 5.87; 95% CI: 1.83–18.85; $p=0.003$). Hyperlipidemia showed an increased risk of AKI but did not reach statistical significance (OR: 2.49; 95% CI: 0.85–7.31; $p=0.097$). Analysis of the relationship between the variables showed a moderate level of association (Cramér's V: 0.354). **Conclusion:** It has been shown that preoperative hepatic steatosis may be independently associated with AKI after CABG surgery. Assessment of hepatic steatosis in the preoperative period may be clinically valuable in developing strategies to reduce the risk of AKI and improve surgical outcomes by contributing to the early identification of high-risk patient groups.

Keywords: hepatic steatosis; acute renal failure; coronary artery bypass graft; hepatic attenuation index; risk factors

1. Introduction

Coronary artery disease is among the most common cardiovascular diseases worldwide, and its incidence and mortality are increasing with the aging population and changing lifestyles^{1,2}. Coronary artery bypass grafting (CABG) is currently considered one of the most effective revascularization methods in the treatment of coronary artery disease³. However, complications developing after CABG significantly affect clinical outcomes, and acute kidney injury (AKI) stands out as one of the most important causes of morbidity and mortality among these complications^{2,4,5}.

The incidence of AKI after CABG varies between 5–30% according to studies, and mortality and hospital stay are significantly increased in these patients⁶. It has been shown that even mild changes in renal function have significant effects on clinical outcomes. The development of AKI is characterized by a sudden decrease in glomerular filtration rate, an increase in serum creatinine and/or a decrease in urine output, and is defined according to the KDIGO criteria^{5,7,8}. The pathogenesis of AKI after CABG is multifactorial, with mechanisms such as renal hypoperfusion, hemodynamic instability, ischemia-reperfusion injury, systemic inflammatory response, and oxidative stress playing a role. However, individual differences in the development of AKI in patients with similar clinical features have not been fully explained, and the identification of preoperative risk factors has become increasingly important^{9–11}.

In recent years, it has been shown that hepatic steatosis is not a disease limited to the liver, but a condition associated with systemic inflammation and metabolic dysfunction. Lipid accumulation in hepatocytes leads to endoplasmic reticulum stress and an increase in reactive oxygen species, triggering an inflammatory response. In addition, cytokine release from adipose tissue and TNF- α -mediated inflammation can affect renal microcirculation and contribute to kidney damage. Ectopic lipid accumulation and lipotoxicity can also negatively affect kidney function by increasing inflammation, fibrosis, and apoptotic processes in renal tubular cells^{12–15}.

In this context, it has been shown that there is a complex organ cross-interaction between the heart, liver, and kidney, and that dysfunction in one organ system can affect the functions of other organs. This axis of interaction is particularly important in terms of clinical outcomes in patients undergoing cardiac surgery¹⁶.

In this study, we aimed to evaluate the effect of preoperative hepatic steatosis on the development of postoperative AKI in patients undergoing isolated CABG surgery. The hypothesis that hepatic steatosis may be an independent risk factor for postoperative AKI was tested. Establishing this relationship may contribute to the early identification of high-risk patients and the development of individualized perioperative management strategies.

2. Materials and Methods

This study was conducted retrospectively using data from 146 patients who underwent isolated CABG surgery at Bursa Yuksek Ihtisas Training and Research Hospital between January 2020 and December 2022. Patients who had preoperative CT images within the specified date range were consecutively included in the study. The study protocol was approved by the institution's ethics committee. Since this is a retrospective study using anonymized clinical data, informed consent was not required. All clinical and radiological data for this study were obtained from the electronic medical record system of Bursa Yuksek Ihtisas Training and Research Hospital. Data include demographic characteristics, pre- and post-operative clinical test results, postoperative renal function parameters, and radiological images. All data were anonymized in accordance with confidentiality and data protection principles.

Inclusion Criteria: Patients aged 18 years and older, patients who underwent isolated CABG surgery, and patients who had a non-contrast CT scan for any reason before surgery to evaluate hepatic steatosis.

Exclusion Criteria: Patients with preoperative renal disease, patients undergoing emergency CABG, patients undergoing postoperative revision, patients with known chronic renal failure, patients using nephrotoxic agents, patients undergoing combined heart surgery, and patients with incomplete medical records.

2.1. Evaluation of Hepatic Steatosis

The presence of hepatic steatosis was evaluated using non-contrast CT images before CABG surgery. CT scans were performed by a radiologist with 15 years of experience in abdominal imaging. In non-contrast CT sections, Hounsfield unit (HU) measurements were performed at four different levels using 10 mm diameter regions of interest (ROIs). A total of 20 ROIs were placed in the liver parenchyma, 12 in the right lobe and 8 in the left lobe, avoiding major vascular structures, and the mean hepatic attenuation (MHA) was calculated by averaging the HU values obtained from these areas. In the same sections, the mean splenic attenuation (MSA) was determined by averaging the HU values obtained from 10 ROIs with a diameter of 10 mm placed in regions far from the main vascular structures of the spleen. The liver attenuation index (LAI) was calculated as the difference between MHA and MSA ($LAI = MHA - MSA$). Hepatic steatosis grading was done according to the LAI value; $LAI \geq 5$ was classified as <5% steatosis, $LAI > 5 > -10$ as 6–30% steatosis, and $LAI \leq -10$ as $\geq 30\%$ steatosis¹⁷.

2.2. Evaluation of Renal Damage

Patients with normal preoperative creatinine values (Also with normal Estimated Glomerular Filtration Rate) were included in the study. AKI was defined and its incidence calculated according to the “Kidney Disease: Improving Global Outcomes” (KDIGO) guidelines⁸. Assessment for AKI was made based on renal function at 24 and 48 hours postoperatively and upon hospital discharge.

2.3. Statistical Analysis

Data were analyzed using SPSS software (version 25.0; IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation (SD) or median (interquartile range, IQR), while categorical variables were expressed as frequency and percentage. Continuous variables were compared using Student's t-test or Mann-Whitney U test. Categorical variables were compared using chi-square test or Fisher's exact test. Logistic regression analysis was performed to identify independent determinants of postoperative AKI. Variables with a p-value < 0.1 in univariate analysis were included in the multivariate model. Adjusted odds ratios (OR) and 95% confidence intervals (CI) were calculated. The statistical significance level was set at $p < 0.05$.

3. Results

The study included 147 patients who underwent isolated CABG surgery and had preoperative non-contrast CT scans; 1 patient was excluded due to death. The ages of the patients ranged from 39 to 86 years, with an average age of 63.6 ± 8.6 years. The gender distribution of the patients was 78.8% male and 21.2% female. The majority of patients (67.1%) had mild steatosis ($LAI \geq 5 \leq 5\%$). Postoperative AKI did not develop in 74.0% of the patients. The demographic and clinical characteristics of the patients are summarized in Table 1.

Table 1. Demographic Characteristics and Clinical Status of Patients

Variable	n	%	Mean ± SD	Mean (Min-Max)
Gender (Male)	115	78.8		
Gender (Female)	31	21.02		
Age (years)			63.6 ± 8.6	39-86
Smoking	49	33.6		
HT				
DM	61	41.8		
HL	18	12.03		
Hepatosteatosi Grade				
Mild (LAI ≥ 5 ≤ %5 steatoz)	98	67.1		
Moderate (5 > LAI > -10 %6 - %30 steatoz)	32	21.09		
Severe (LAI ≤ -10 ≥ %30 steatoz)	16	11.0		
KDIGO Stage 0 (No AKI)	108	74.0		
KDIGO Stage 1	28	19.02		
KDIGO Stage 2	6	4.01		
KDIGO Stage 3	4	2.07		

DM - Diabetes Mellitus, HT- Hypertension, HL-hyperlipidemia, KDIGO - Kidney Disease: Improving Global Outcomes, LAI - Liver Attenuation Index, AKI - Acute Kidney Injury

The incidence of AKI was lowest in patients with mild hepatic steatosis, while it was highest in those with severe hepatic steatosis, especially in mild and moderate stages. A significant majority of patients with mild hepatic steatosis (82.65%) did not develop post-operative AKI. Severe AKI was observed in 3.07% of patients with mild hepatic steatosis. Only 31.25% of patients with severe hepatic steatosis did not develop AKI. Severe hepatic steatosis caused severe AKI in 7.25% of patients. The distribution of postoperative AKI according to hepatic steatosis is shown in Table 2 and Figure 1.

Table 2. Hepatosteatosi Grade distribution by KDIGO Stage

Hepatosteatosi Grade	KDIGO Stage	n	%
Mild (LAI ≥ 5 ≤ %5 steatoz)	Stage 0	81	82.65
Mild (LAI ≥ 5 ≤ %5 steatoz)	Stage 1	10	10.20
Mild (LAI ≥ 5 ≤ %5 steatoz)	Stage 2	4	4.08
Mild (LAI ≥ 5 ≤ %5 steatoz)	Stage 3	3	3.07
Moderate (5 > LAI > -10 %6 - %30 steatoz)	Stage 0	22	68.75
Moderate (5 > LAI > -10 %6 - %30 steatoz)	Stage 1	10	31.25
Moderate (5 > LAI > -10 %6 - %30 steatoz)	Stage 2	0	0
Moderate (5 > LAI > -10 %6 - %30 steatoz)	Stage 3	0	0
Severe (LAI ≤ -10 ≥ %30 steatoz)	Stage 0	5	31.25
Severe (LAI ≤ -10 ≥ %30 steatoz)	Stage 1	8	50.00
Severe (LAI ≤ -10 ≥ %30 steatoz)	Stage 2	2	12.50
Severe (LAI ≤ -10 ≥ %30 steatoz)	Stage 3	1	7.25

KDIGO - Kidney Disease: Improving Global Outcomes, LAI - Liver Attenuation Index

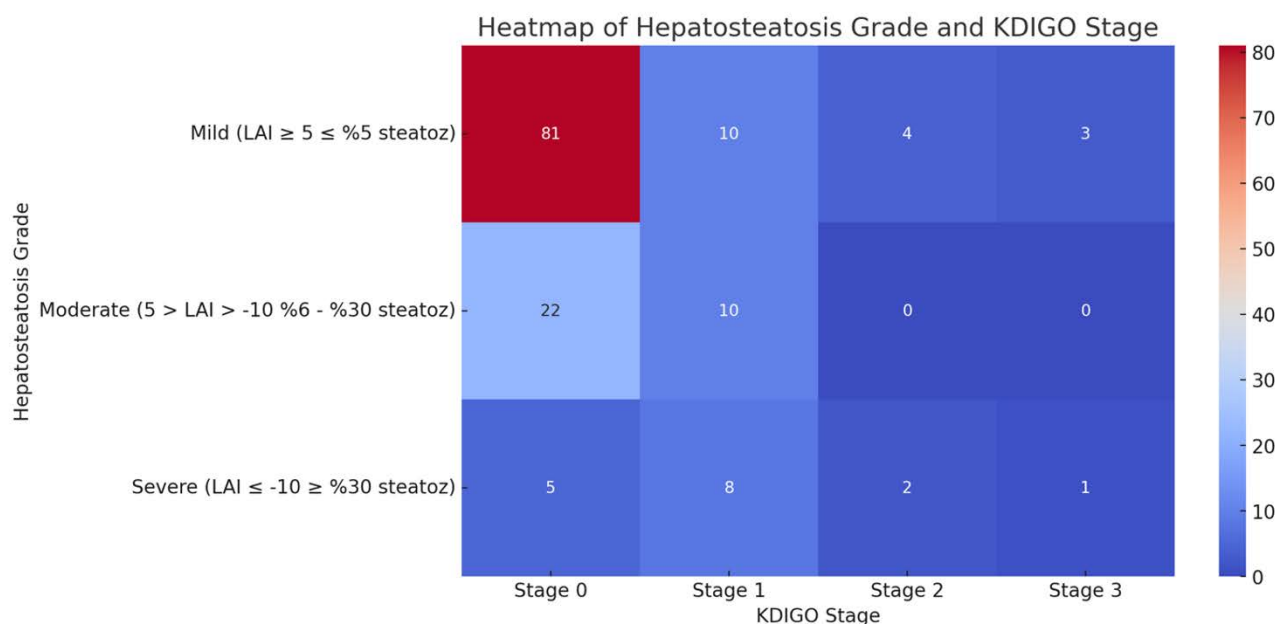


Figure 1. Hepatosteatosis Grade distribution by KDIGO Stage

Univariate analysis identified several important risk factors associated with the development of postoperative AKI in patients undergoing isolated CABG surgery. The mean age of patients who developed AKI (67.4 ± 8.2 years) was statistically significantly higher than that of those who did not develop AKI (62.2 ± 8.3 years) ($p < 0.001$). The rate of diabetes in patients who developed AKI (57.9%) was significantly higher than in those who did not develop AKI (36.1%) ($p = 0.014$). Hyperlipidemia was significantly more common in patients who developed AKI (21.1%) than in those who did not develop AKI (9.3%) ($p = 0.04$). Patients with mild hepatic steatosis were less likely to develop AKI (44.7% in the AKI group, 75.0% in the non-AKI group), a statistically significant difference ($p = 0.001$). Severe hepatic steatosis was significantly more common in the AKI group (26.3%) than in the non-AKI group (5.6%), with a highly significant p-value ($p < 0.001$). These findings suggest that advanced age, diabetes mellitus, hyperlipidemia, and severe hepatic steatosis are important risk factors for developing AKI after CABG surgery (Table 3).

Table 3. Univariate Analysis of Risk Factors for Postoperative AKI

Variable	AKI (n=38)	No AKI (n=108)	P-value
Age (years)	67.4 ± 8.2	62.2 ± 8.3	0.00
Gender (Male), n(%)	30 (78.9%)	85 (78.7%)	0.98
DM, n(%)	22 (57.9%)	39 (36.1%)	0.01
HT, n(%)	26 (68.4%)	57 (52.8%)	0.08
HL, n(%)	8 (21.1%)	10 (9.3%)	0.04
Smoking, n(%)	15 (39.5%)	34 (31.5%)	0.36
Hepatosteatosis Grade, n(%)			
Mild (LAI $\geq 5 \leq 5$ steatoz)	17 (44.7%)	81 (75.0%)	0.00
Moderate (5 > LAI > -10 %6 - %30 steatoz)	11 (28.9%)	21 (19.4%)	0.20
Severe (LAI $\leq -10 \geq 30$ steatoz)	10 (26.3%)	6 (5.6%)	<0.001

AKI: Acute kidney injury, DM - Diabetes Mellitus, HT- Hypertension, HL-hyperlipidemia, LAI - Liver Attenuation Index

Multivariate logistic regression analysis identified significant independent risk factors associated with the development of postoperative AKI in patients undergoing

isolated CABG surgery. Each additional year of age increased the probability of developing postoperative AKI by 9% (OR, 1.09; 95% CI: 1.02-1.14; $p=0.01$). Diabetic patients were more than twice as likely to develop AKI compared to non-diabetic patients (OR: 2.35; 95% CI: 1.11-4.95; $p=0.03$). Although not statistically significant, hyperlipidemia showed a tendency to increase the risk of AKI (OR: 2.49; 95% CI: 0.85-7.31; $p=0.10$). This finding points to a potential association requiring further investigation. Patients with severe hepatic steatosis were almost six times more likely to develop AKI than those with mild hepatic steatosis (OR, 5.87; 95% CI, 1.83-18.85; $p=0.01$), indicating a strong and significant association. The results showed that advanced age, diabetes mellitus, and severe hepatic steatosis are important independent risk factors for the development of postoperative AKI in patients undergoing CABG surgery, as shown in Table 4. A Cramér V value of 0.354 indicates a moderate to high effect size association between the degree of hepatosteatosi and the severity of AKI development. The compression of the distribution of KDIGO stages among different degrees of hepatic steatosis in patients undergoing isolated CABG surgery is presented in Table 5.

Table 4: Multivariate Logistic Regression Analysis of Risk Factors for Postoperative AKI

Variables	OR	95% CI	P
Age	1.09	1.02 - 1.14	0.01
DM	2.35	1.11 - 4.95	0.01
HL	2.49	0.85 - 7.31	0.10
Hepatosteatosi Grade			
- Mild (Reference)	1.00		
- Moderate	1.75	0.64 - 4.79	0.28
- Severe	5.87	1.83 - 18.85	0.01

Multivariate Logistic Regression, OR: Odds Ratio, CI: Confidence Interval, DM - Diabetes Mellitus, HL-hyperlipidemia, LAI - Liver Attenuation Index

Table 5. Compression KDIGO Stages by Hepatosteatosi Grade

Hepatosteatosi Grade	Stage 0	Stage 1	Stage 2	Stage 3	P	Cramér's V
Mild ($LAI \geq 5 \leq \%5$ steatoz)	81	10	4	3		
Moderate ($5 > LAI > -10 \%6 - \%30$ steatoz)	22	10	0	0	<0.001	0.35
Severe ($LAI \leq -10 \geq \%30$ steatoz)	5	8	2	1		
Mild vs. Moderate	$p1 = 0.00$	$p2 = 0.50$	$p3 = 0.01$	$p3 = 0.01$		
Mild vs. Severe	$p1 = 0.00$	$p2 = 0.02$	$p3 = 0.01$	$p3 = 0.02$		
Moderate vs. Severe	$p1 = 0.01$	$p2 = 0.02$	$p3 = 0.01$	$p3 = 0.02$		

chi-square test

KDIGO - Kidney Disease: Improving Global Outcomes, LAI - Liver Attenuation Index

Logistic regression analysis was used to model the probability of AKI development depending on different degrees of hepatic steatosis. The effect of gender on the probability of developing AKI was not statistically significant ($p=0.48$). The probability of developing AKI increased significantly with age. For each additional year of age, the logarithm of the probability of developing AKI increased by 0.0913 ($p<0.001$), indicating a strong association between advanced age and the risk of AKI. The degree of hepatic steatosis was found to be independently associated with AKI ($p<0.001$) (Table 6).

Table 6. Probability of AKI Occurrence Based on Hepatosteatoses Grades

Coefficient	Estimate	SE	z	P	95% CI
Gender	0.32	0.45	0.70	0.48	[-0.57, 1.21]
Age	0.09	0.03	3.53	0.00	[0.04, 0.14]
Diabetes	0.71	0.44	1.63	0.10	[-0.14, 1.57]
Hypertension	0.61	0.42	1.46	0.14	[-0.21, 1.42]
Hyperlipidemia	0.44	0.53	0.84	0.40	[-0.60, 1.49]
Hepatosteatoses Grade	0.77	0.07	11.25	0.00	[0.64, 0.91]

SE: standard error, CI: confident interval, AKI: acute kidney injury

4. Discussion

This study demonstrated a significant association between preoperative hepatic steatosis and postoperative AKI in patients undergoing isolated CABG surgery. In retrospective analysis, it was found that the incidence and severity of AKI increased significantly with increasing degree of hepatic steatosis. This finding suggests that fatty liver disease may be independently associated with renal outcomes after cardiac surgery.

In our study, the incidence of postoperative AKI was found to be 26%, which is consistent with previous CABG series. In the literature, it is reported that the development of AKI after cardiac surgery varies between 5–30%, and it is emphasized that this situation is affected by patient characteristics, surgical techniques, and differences in perioperative management⁶. In this context, it has been shown that AKI is a multifactorial complication and that factors such as advanced age, diabetes mellitus, prolonged cardiopulmonary bypass time, and transfusion play an important role^{2,18}.

In the present study, particularly severe hepatic steatosis was shown to be independently associated with AKI development (OR: 5.87; 95% CI: 1.83–18.85; $p=0.003$). Furthermore, the increased rate of severe AKI at the KDIGO stage 3 level in these patients indicates that hepatic steatosis is associated not only with incidence but also with disease severity. These results support the view that hepatic steatosis, as a systemic metabolic disease, can have significant effects on renal function. Among the possible underlying mechanisms of this relationship between hepatic steatosis and renal dysfunction, inflammatory activation and metabolic dysfunction are prominent. It has been reported that with increasing hepatic steatosis, pro-inflammatory cytokines such as tumor necrosis factor- α (TNF- α) are elevated, which can trigger oxidative stress, endothelial dysfunction, and apoptotic processes, contributing to renal damage. It is also suggested that a decrease in peroxisome proliferator-activated receptor- δ (PPAR- δ) expression in kidney tissue can increase the inflammatory response and predispose to structural damage in kidney tissue. As a result of these processes, fibrotic changes can develop at the glomerular and tubulointerstitial levels^{19–21}.

However, it has been shown that hepatic steatosis can contribute to renal damage not only through inflammatory pathways but also through ectopic lipid accumulation and lipotoxicity. Lipotoxicity can lead to renal cell dysfunction by activating pro-inflammatory, profibrotic, and proapoptotic signaling pathways. Experimental studies have revealed the development of renal dysfunction, oxidative stress, and mitochondrial dysfunction in steatosis models induced by high-fat diets. These findings support the idea that hepatic steatosis is a systemic lipotoxic and inflammatory condition^{22–27}.

Our study also showed that age, diabetes mellitus, and other metabolic comorbidities are associated with the development of AKI. These findings are consistent with well-defined risk factors in the literature and reaffirm the multifactorial nature of AKI after

cardiac surgery^{2,18}. Gültekin et al. In the study conducted by [author's name], it was shown that the AST/ALT ratio can predict AKI after cardiac surgery, and the importance of liver function markers in perioperative risk assessment was emphasized. Our study expands on these findings, revealing that not only biochemical markers but also the degree of fatty liver disease can be an important risk determinant²⁸.

Although there are studies in the literature evaluating the relationship between hepatic steatosis and renal function, data examining the relationship between preoperative hepatic steatosis and postoperative AKI in isolated CABG patients are quite limited. In this respect, our study contributes to the existing literature by addressing liver-kidney interaction in cardiac surgery from a different clinical perspective. However, the findings need to be confirmed by prospective and multicenter studies. In conclusion, it has been shown that preoperative hepatic steatosis may be independently associated with AKI after CABG surgery. This suggests that considering fatty liver disease in risk-stratification processes may be clinically beneficial and may contribute to the development of individualized perioperative strategies in high-risk patients.

4.1. Limitations

This study, while shedding light on the relationship between hepatic steatosis and AKI after CABG surgery, has some limitations. Due to its retrospective design, selection bias and limitations in making causal inferences due to the observational data structure should be considered. In addition, obtaining data from electronic medical records may carry the risk of information bias, such as incomplete or erroneous data entries. Furthermore, the single-center nature of the study may limit the generalizability of the findings by restricting the patient population to a specific center. Therefore, larger sample sizes, multicenter, and prospective cohort studies are needed to validate the obtained results and to elucidate the underlying pathophysiological mechanisms in more detail. Also, hepatic steatosis was assessed using the LAI calculated from CT in our study. Nowadays, Magnetic Resonance Imaging-Proton Density Fat Fraction, transient elastography, Controlled Attenuation Parameter score, liver biopsy are considered much more reliable methods. These methods could not be used due to the study design. In particular, the investigation of inflammatory markers and related molecular pathways may contribute to the identification of new therapeutic targets for preventing the development of AKI after cardiac surgery in patients with hepatic steatosis.

5. Conclusion

This study demonstrated an independent association between severe hepatic steatosis and the development of postoperative AKI. We could perform more comprehensive preoperative assessment and individualized perioperative management strategies in high-risk patients. The findings support the growing literature on the relationship between liver disease and adverse renal outcomes and draw attention to the clinical significance of liver-kidney interaction in patients undergoing cardiac surgery. In this context, it is thought that a multidisciplinary approach in the presence of concomitant hepatic and renal risk factors may play an important role in improving surgical outcomes.

Ethics Approval: The study was approved by Bursa Yuksek Ihtisas Training and Research Hospital Clinical Research Ethics Committee (Date: 15-06-2022, protocol number: 2011-KAEK-25 2022/06-22)

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Data availability: The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request

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