Journal of The Cardiovascular Thoracic Anaesthesia and Intensive Care Society

GKDA Derg 2023;29(1):7-14
DOI: 10.14744/GKDAD.2023.79664

Research Article

Systemic Immune-Inflammation Index Predicts Acute Kidney Injury after Cardiac Surgery: A Retrospective Observational Study

Aslıhan Aykut,[1] Emine Nilgün Zengin,[1] Bekir Boğaçhan Akkaya,[2] Nevriye Salman[1]

[1]Department of Anesthesiology and Reanimation, Ministry of Health Ankara City Hospital, Ankara, Türkiye
[2]Department of Cardiovasculer Surgery, Ministry of Health Ankara City Hospital, Ankara, Türkiye

ABSTRACT

Objectives: Inflammation plays an essential role in the development of postoperative acute kidney injury (AKI) in patients receiving cardiac surgery. The study aims to examine the predictive value of the systemic immune-inflammation index (SII), a new biomarker reflecting systemic inflammation, in the development of AKI following cardiac surgery in patients who had coronary artery bypass grafting (CABG).

Methods: Patients who received CABG operation in 2022 were retrospectively examined. The incidence of AKI 7 days postsurgery using Kidney Disease Improving Global Outcomes criteria was the primary outcome. The patients were classified into the AKI (n=160) and non-AKI groups (n=424). Patients were compared in terms of basic clinical features, operative characteristics, postoperative variables, and hematological indices derived from preoperative complete blood count analysis. The ability of SII to predict AKI was assessed using receiver-operating characteristic (ROC) curve analysis. Multivariate logistic regression analysis was used to determine the independent relationship between the development of SII and AKI.

Results: In this investigation, the incidence of AKI was 25.6%. eGFR, serum albumin, and hemoglobin were significantly lower in the AKI group, whereas body mass index, hypertension, serum creatinine, neutrophil–lymphocyte ratio, platelet–lymphocyte ratio, and SII were significantly greater. The preoperative SII cutoff of 651.7 predicted AKI with 65.0% sensitivity and 64.9% specificity. The area under the ROC curve was 0.718 (95% confidence interval 0.676–0.760).

Conclusion: Preoperative SII may be a simple, inexpensive, and useful prognostic biomarker in predicting postoperative AKI in patients undergoing CABG.

Keywords: Acute kidney injury, coronary artery bypass grafting, systemic immune-inflammation index

Please cite this article as: "Aykut A, Zengin EN, Akkaya BB, Salman N. Systemic Immune-inflammation Index Predicts Acute Kidney Injury after Cardiac Surgery: A Retrospective Observational Study. GKDA Derg 2023;29(1):7-14".

Introduction

Acute kidney injury (AKI) after cardiac surgery is one of the most frequent and significant complications found in patients undergoing open heart surgery. High mortality and morbidity are linked to AKI.[1,2] It has been revealed that the incidence of AKI after cardiovascular surgery is 5%–42%, and the incidence of renal replacement therapy (RRT) is 2%–8%.[3-5] There is no developed treatment for AKI after cardiovascular surgery An opportunity to create early identification and intervention techniques to improve outcomes may arise from an accurate assessment of AKI. Various risk-scoring models with independent risk factors have been created in previous studies to increase AKI predictability.[4-7] Due to the inconsistent risk factors found in this research or the expensive price of new biomarkers, there is still a need to discover a clinically meaningful and affordable risk factor for AKI.

Atherosclerosis is a major contributor to coronary artery disease and is highly linked with an ongoing inflammatory response.[8,9] The role of direct inflammatory injury in addition to intraoperative ischemia-reperfusion injury, endothelial cell dysfunction, and apoptosis in the pathogenesis of AKI is well known.[10,11] However, there are limited studies investigating the impact of inflammation due to preoperative ath-

Address for correspondence: Emine Nilgün Zengin, MD. Saglik Bakanligi Ankara Sehir Hastanesi, Anesteziyoloji ve Reanimasyon Klinigi, Ankara, Türkiye

Phone: +90 506 337 05 48 **E-mail:** nilbavullu@gmail.com

Submitted: January 18, 2023 **Revised:** January 30, 2023 **Accepted:** February 02, 2023 **Available Online:** March 29, 2023

The Cardiovascular Thoracic Anaesthesia and Intensive Care - Available online at www.gkdaybd.org

erosclerosis and other comorbid conditions on postoperative complications in patients who will be operated on for coronary artery bypass grafting (CABG). The neutrophil×platelet/lymphocyte ratio-based systemic immune-inflammation index (SII), which combines three inflammatory peripheral cell counts to take patients' inflammatory and immunological status into account, was recently developed.[12,13] This index has been reported to have a predictive value for mortality in patients with cardiovascular disease, including coronary artery disease and acute coronary syndrome.[14,15] In cardiac surgery, preoperative SII value has been linked with major negative outcomes such as postoperative atrial fibrillation (AF), prolonged mechanical ventilation, intra-aortic balloon pump (IABP) requirement, and mortality.[16-18] However, the literature on the usefulness of preoperative SII in predicting the occurrence of AKI after cardiac surgery, particularly in patients undergoing isolated CABG, is limited. As a result, this study mainly aimed to examine the predictive accuracy of SII for the formation of AKI in patients receiving CABG.

Methods

Ankara City Hospital's local ethics committee approval (approval no: E1-22-2983, date: 19.10.2022) was obtained for the study. The records of 660 patients who had cardiopulmonary bypass (CPB) during CABG in our hospital's operating theater for cardiovascular surgery between January 2022 and December 2022 were then retrospectively examined. Patients with preoperative evidence of acute or chronic infection, patients with systemic inflammatory or autoimmune dysfunction, patients with dialysis-dependent chronic kidney disease (CKD), patients with recurrent cardiac surgery, patients who need preoperative or postoperative IABP/extracorporeal membrane oxygenation (ECMO), and patients who were reoperated and/or died due to bleeding revision in the first week were removed from the study (Fig. 1). The files of the remaining 624 patients had the baseline complete blood count needed for inflammatory indices and all available clinical data. For the post hoc analysis, these patients were deemed fully evaluable. The need for informed consent was waived for this retrospective study. All procedures were conducted to the Declaration of Helsinki.

Patients' age, gender, body mass index (BMI), comorbidities, left ventricular ejection fraction, and preoperative laboratory data (blood urea, serum creatinine [sCr], determined glomerular filtration rate [eGFR], blood glucose level, glycosylated hemoglobin [HbA1c]), hemoglobin, neutrophils, lymphocytes, platelets, and C-reactive protein (CRP) were noted. Neutrophil–lymphocyte ratio (NLR), platelet–lymphocyte ratio (PLR), and SII (neutrophil×platelet/lymphocyte) values were determined. In the intraoperative period,

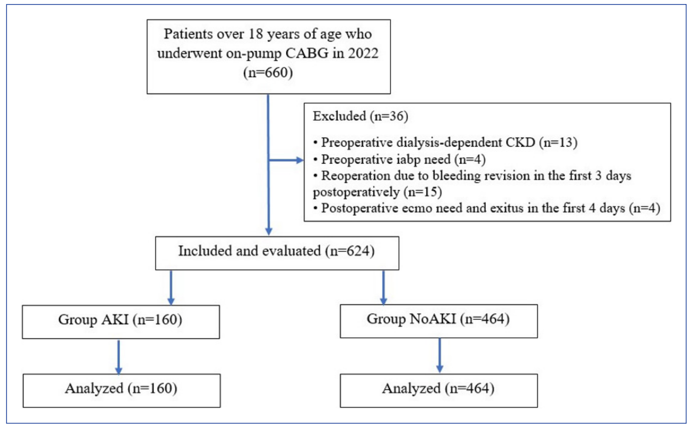

Figure 1. Flowchart of the patients.

CABG: coronary artery bypass grafting; CKD: chronic kidney disease; iabp, intra-aortic balloon pump; AKI: acute kidney injury; ECMO: extracorporeal membrane oxygenation.

cross clamp (CC), CPB, operation times, totally infused intravenous fluid (excluding blood product), blood and blood products, amount of urine, and need for diuretic and inotropic agents were recorded. The Kidney Disease Improving Global Outcomes (KDIGO) criteria, which classify AKI based on the highest change in sCr from preoperative baseline levels, were used to characterize the incidence of AKI.[19] All patients who met the KDIGO requirement for stages 1, 2, and 3 were considered to have AKI. A new requirement for dialysis following surgery is known as RRT. Postoperative result variables included extubation time, length of hospital and intensive care unit (ICU) stay, postoperative complications (AF, cerebrovascular accident), the requirement for erythrocyte suspension, and 30-day mortality.

Statistical Analysis

The IBM SPSS.25.0 software was employed for all dates examined. Categorical variables were defined as frequencies and percentages and analyzed with the Chi-squared test or Fisher's exact test. Continuous variables were presented as mean±SD (standard deviation) or medians (interquartile range) and to examine the differences between the two groups, the normality test (Kolmogorov–Smirnov) Mann–Whitney U test or Student's t-test were employed. To gather information on systemic inflammatory indices' capacity to predict AKI, a receiver-operating characteristic (ROC) curve was created. The area under the curve (AUC) was used to evaluate the ROC curve. Youden's index (J=sensitivity+specificity−1) was used to determine the most appropriate cutoff value. A model for multivariate logistic regression analysis was developed using factors that showed statistical significance in univariate logistic regression to identify the predictors of AKI. To prevent multicollinearity, NLR, PLR, neutrophil, lymphocyte, and platelet were excluded from the multivariate logistic regression model. The Hosmer–Lemeshow test was employed for model fit. $p<0.05$ was considered significant in all comparisons. The sample size was established by the number of cases between the given dates, and posthoc power analysis was done using the total number of patients. After the study was completed, SII (measurement with the difference between groups) values were used for power analysis. According to this; Power analysis was conducted with G*Power 3.1.9.7 statistical package program; n=624 (n1=160, n2=464), α=0.05, effect size (d)=0.3; power=93% was observed.

Results

The data of 624 patients included in the study were examined retrospectively. The patients were classified into two groups patients who developed AKI (all patients with KDIGO stages 1, 2, and 3, Group AKI) and patients who did not develop AKI (Group NoAKI). According to KDIGO criteria, stage 1 AKI was found in 134 patients in Group AKI within the first 7 days postoperatively, stage 2 in 21 patients, and stage 3 in 5 patients. RRT was required in 4 patients. The incidence of AKI in this investigation was 25.6% (n=160). There was no statistical difference between the groups in terms of age, gender, ejection fraction, and comorbidities (diabetes, hyperlipidemia, chronic obstructive pulmonary disease, and previous cerebrovascular disease) (p:>0.05). Hypertension rate and BMI were statistically considerably lower in the AKI group (p:0.047, p:0.002, respectively). sCr, blood urea, NLR, PLR, and SII values were observed to be statistically significantly greater in the AKI group (p:0.004, p:<0.001, p:<0.001, p:<0.001, p:<0.001, respectively) (Table 1). Albumin, eGFR, hemoglobin, and lymphocyte levels were statistically significantly lower in the AKI group (p:<0.001, p:<0.001, p:0.002, p:<0.001, respectively) (Table 1).

Table 1. Baseline characteristics and laboratory data

	NoAKI (n=464) Mean±SD	AKI (n=160) Mean±SD	p*
Gender (M), n(%)	380 (81.9)	127 (79.4)	0.481
Age (year)	60.49±9.4	62.13±8.0	0.051
BMI (kg/m^2)	28.25±3.4	29.29±3.66	**0.002**
EF (%)	52.99±6.8	52.07±7.2	0.162
Hypertension, n(%)	165 (35.6)	71 (44.4)	**0.047**
Diabetes, n(%)	182 (39.2)	64 (40.0)	0.863
Hyperlipidemia, n(%)	108 (23.3)	36 (22.5)	0.841
COPD, n(%)	51 (11.0)	20 (12.5)	0.604
Cerebrovascular disease, n(%)	19 (4.1)	6 (3.8)	0.848
Baseline laboratory data			
	Median (IQR)	**Median (IQR)**	
Serum creatinine (mg/dL)	0.90 (0.8–1.0)	0.94 (0.8–1.1)	**0.004**
Blood urea (mg/dL)	34.00 (28.0–41.0)	38.0 (32.0–45.0)	**<0.001**
eGFR (ml/min/1.73 m^2)	88.00 (75.0–99.0)	80.00 (66.0–95.0)	**<0.001**
Blood glucose level (mg/dL)	111.00 (93.0–154.5)	120.00 (101.5–156.5)	0.065
HbA1c (%)	7.00 (6.0–9.1)	7.05 (6.0–8.3)	0.728
Serum albumin (g/dL)	43.00 (40.0–45.0)	41.00 (38.0–44.0)	**<0.001**
CRP (mg/dL)	3.2 (1.5–7.6)	3.8 (1.9–9.0)	0.785
Hemoglobin (g/dL)	14.00 (12.9–14.9)	13.60 (12.0–14.6)	**0.002**
Neutrophil (10^3/µL)	4.93 (4.0–6.0)	4.76 (3.9–5.9)	0.402
Lymphocyte (10^3/µL)	2.18 (1.7–2.7)	1.60 (1.3–1.84)	**<0.001**
Platelet (10^3/µL)	243.00 (204.5–286.0)	259.50 (212.5–309.0)	0.051
NLR	2.25 (1.7–2.9)	2.99 (2.4–3.7)	**<0.001**
PLR	112.26 (86.7–142.5)	155.46 (132.1–199.9)	**<0.001**
SII	541.17 (388.5–758.8)	747.18 (577.9–1043.2)	**<0.001**

COPD: chronic obstructive pulmonary disease; SD: standard deviation; BMI: body mass ındex; EF: ejection fraction; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; Hb: hemoglobin; NLR: neutrophil–lymphocyte ratio; PLR: platelet–lymphocyte ratio; SII: systemic immune-inflammation index.

*Bold values indicated p:<0.05, The independent samples t-test was used for continuous variables (mean±SD), the Mann–Whitney U test was used for continuous variables (median, IQR), and the Chi-square test was performed for categorical variables (n, %).

In the intraoperative period, the mean duration of CPB-packed red blood cell (pRBC) transfusion, and the proportion of patients in need of dobutamine and diuretics were observed to be statistically significantly higher in the AKI group. The proportion of patients in Group AKI who required only a pRBC transfusion during the postoperative phase was found to be statistically greater (p: 0.041) (Table 2).

We employed preoperative SII, NLR, and, PLR values for ROC analysis. The optimal value of the SII level in identifying AKI was 651.37 with a sensitivity of 65.0% and a specificity of 64.9%. Notably, the SII's AUC value (0.760) for detecting AKI was substantially greater than the NLR's and the PLR's combined AUC values (AUC: 0.556 vs. AUC: 0.554, respectively, Fig. 2).

A model was developed for multivariate regression analysis from the parameters reported to be potential risk factors for AKI in univariate logistic regression analysis (Table 3). According to the findings of multivariate logistic regression analysis, there was a higher risk of AKI in patients with BMI ≥ 30 kg/m2 (1.7 times), patients with preoperative sCr > 1.3 mg/dL (2.3 times), patients with preoperative serum albumin < 4 g/dL (1.9 times), in patients who require pRBC transfusion

Table 2. Intraoperative and postoperative variables

	No AKI (n=464) Mean±SD	AKI (n=160) Mean±SD	p*
Intraoperative Period			
Duration of CC (dk)	63.20±18.8	64.72±18.4	0.373
Duration of CPB (dk)	100.05±25.9	107.96±28.6	**0.002**
Duration of operation (dk)	312.21±48.7	320.47±37.2	0.051
pRBC transfusion, n (%)			
0 unit	359 (77.4)	96 (60.0)	
1–3 units	101 (21.8)	60 (37.5)	**<0.001**
4–6 units	4 (0.9)	4 (2.5)	
FFP transfusion, n(%)			
0 unit	405 (87.3)	131 (81.9)	
1–3 units	57 (12.3)	26 (16.3)	0.086
4–6 units	2 (0.4)	3 (1.9)	
PC transfusion, n(%)			
0 unit	462 (99.6)	158 (98.8)	0.263
1 unit	2 (0.4)	2 (1.3)	
Total intravenous fluid (ml)	1607.03±537.7	1559.17±429.5	0.382
Total urine output (ml)	915.11±498.5	958.26±461.8	0.392
Dopamine, n(%)	86 (18.5)	39 (24.4)	0.111
Dobutamine, n(%)	29 (6.3)	20 (12.5)	**0.011**
Steradine, n(%)	7 (1.5)	4 (2.5)	0.411
Adrenalin, n(%)	4 (0.9)	4 (2.5)	0.112
Diuretic, n(%)	50 (10.8)	32 (20.0)	**0.003**
Postraoperative Period			
	Median (IQR)	**Median (IQR)**	
CVD SVD, n(%)	30 (6.5)	9 (5.6)	0.705
AF, n(%)	52 (11.2)	17 (10.6)	0.840
pRBC transfusion, n(%)	150 (32.3)	66 (41.3)	**0.041**
Extubation time (>12 h), n(%)	8 (1.7)	2 (1.3)	0.680
ICU stay (h)	24 (20–24)	24 (22–48)	0.800
Hospital stay (day)	7 (6–12)	7 (6–12)	0.343
Mortalite, n(%)	6 (1.3)	2 (1.3)	0.967

SD: standard deviation; CC: cross clamp; CPB: cardiopulmonary bypass; pRBC: packed red blood cell; FFP: fresh frozen plasma; CVD: cerebrovascular diseases; AF: atrial fibrillation; PC: platelet concentrate.

*Bold values indicated p:<0.05. The independent samples t-test was used for continuous variables (mean±SD), the Mann–Whitney U test was used for continuous variables (median, IQR), and the Chi-square test was performed for categorical variables (n, %).

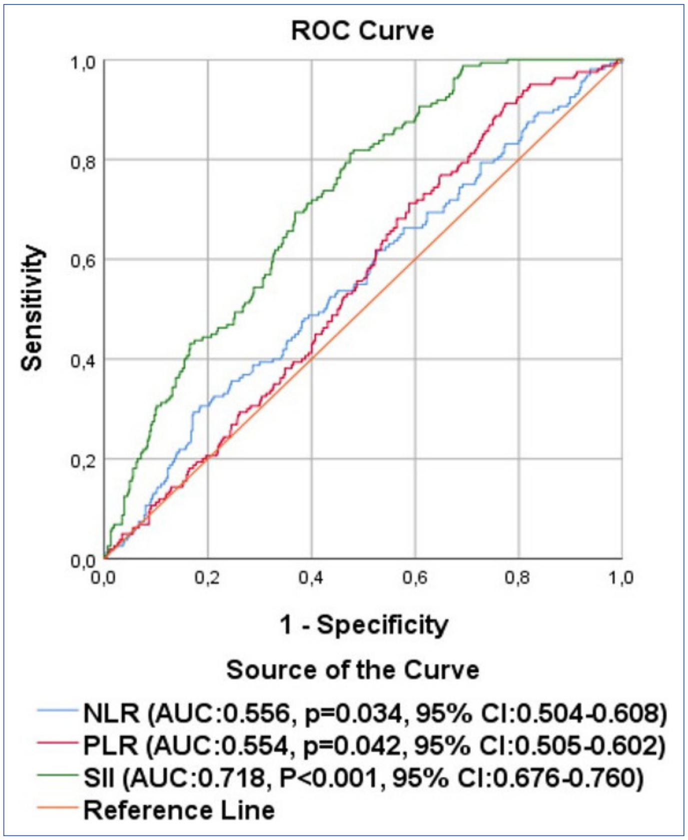

Figure 2. ROC curve analysis to establish the predictive value of SII, NLR, and PLR for AKI.

ROC: receiver-operating feature; AUC: area under the curve; NLR: neutrophil–lymphocyte ratio; PLR: platelet–lymphocyte ratio; SII: systemic immune-inflammation index.

during surgery (1.8 times), who need intraoperative diuretics (1.8 times), in patients with CPB time > 120 min (1.7 times), patients with preoperative SII > 651.37 (3.3 times) (p:<0.05).

Discussion

In this retrospective study, it was revealed that preoperative SII value may be a significant and independent predictor of postoperative AKI in patients receiving isolated CABG. However, obesity, preoperative creatinine elevation, hypoalbuminemia, intraoperative pRBC transfusion, diuretic requirement, and long CPB duration were validated as independent risk factors among other predictive risk factors in previous studies. Moreover, compared to NLR and PLR, the preoperative SII value demonstrated a better effective discrimination ability for AKI. Postcardiovascular surgery AKI is an independent risk factor that increases cost and morbidity and in-hospital mortality.[1,2] However, positive results can be attained if therapeutic interventions are undertaken within 24–48 h.[20] Thus, early risk factor identification by anesthesiologists and surgeons is crucial to lowering mortality and improving prognosis in patients with AKI.

Inflammation has been associated with many diseases, including chronic heart failure, cancer, metabolic problem, and cardiovascular disease.[21-23] In patients with coronary artery disease, atherosclerosis is now recognized not only as a cholesterol disorder accumulating on the vessel walls but also as a continuous, dynamic, and inflammatory process in the vascular system.[8,9] It has been demonstrated that endothelial damage and occlusive intravascular platelet aggregation contribute to the pathophysiology of atherosclerosis and acute coronary syndrome.[24] This platelet activation adheres to the endothelium during the coronary artery disease process, releasing a variety of proinflammatory cytokines. Moreover, it leads to neutrophil adhesion. Inflammatory mediators are secreted by neutrophils, which can control inflammatory respons-

Table 3. Univariate and multivariate logistic regression

	Univariate			Multivariate		
	p	OR	%95 CI	p	OR	%95 CI
BMI ≥ 30 kg/m^2	**0.012**	1.676	1.122–2.503	0.013	1.757	1.127–2.741
Hypertension	0.048	1.446	1.003–2.083			
Preoperative hemoglobin ≤ 10 g/dL	0.019	3.436	1.226–9.633			
Preoperative blood urea ≥ 40 mg/dL	0.005	2.077	1.241–3.474			
Preoperative sCr > 1.3 mg/dL	**<0.001**	2.713	1.598–4.608	0.006	2.303	1.278–4.151
Preoperative eGFR < 90 ml/min/1.73 m^2	0.004	1.721	1.185–2.501			
Preoperative serum albumin < 4 g/dL	**<0.001**	2.095	1.439–3.051	0.002	1.929	1.276–2.916
Preoperative SII > 651.37	**<0.001**	3.397	2.331–4.950	<0.001	3.345	2.251–4.973
pRBC transfusion during surgery	**<0.001**	2.279	1.553–3.346	0.004	1.839	1.209–2.796
Intraoperative dobutamin use	0.013	2.143	1.175–3.907			
Intraoperative furosemid use	**0.003**	2.070	1.273–3.365	0.030	1.800	1.057–3.062
CPB time > 120 min	**0.003**	1.906	1.238–2.936	0.029	1.706	1.055–2.759

OR: odds ratio; CI: confidence interval; BMI: body mass index; sCr: serum creatinine; pRBC: packed red blood cells; CPB: cardiopulmonary bypass; eGFR: estimated glomerular filtration rate; SII: systemic immune-inflammation index.

es. They may also demonstrate vigorous chemotaxis and phagocytosis. An increased neutrophil count shows an overactivated inflammatory response. Lymphocytes play a major role in specialized immunity. Inflammation and reduced lymphocyte count are two characteristics of weakened immunity. Hematological parameters such as neutrophils, platelets, and lymphocytes acquired from the complete blood count are potential markers of this inflammation. For instance, NLR is an indicator of critical stenosis and has been linked with both the severity and plaque morphology of coronary atherosclerotic disease.[25] Similar to NLR, PLR has also been shown to be a reliable indicator of advanced atherosclerosis.[26] For these reasons, coronary artery patients are operated on with varying degrees of inflammatory load, depending on the severity of the disease. As a result, we sought to assess preoperative inflammation and determine the prognostic value of AKI, one of the problems that follow heart surgery that is frequently reported.

In this study, SII was employed as a measure of systemic inflammatory response for AKI. In a noninvasive, widely accessible full blood count, SII is easily collected. It is an innovative inflammatory biomarker that combines neutrophil, lymphocyte, and platelet counts to represent the overall inflammatory state of the body. The predictive value of this indicator has been investigated in studies on a variety of disorders. It was initially investigated for poor outcomes in cancer patients.[27,28] Subsequently, the predictive accuracy of SII for mortality in chronic heart failure cases was demonstrated.[29] Similar findings have been made with coronary artery patients who have received the percutaneous coronary intervention, showing it to be an independent predictor for significant cardiovascular events, including abrupt cardiac death.[30] There are also studies analyzing the association between preoperative SII and postoperative complications in patients undergoing cardiac surgery. In a study performed on patients operated on for acute type A aortic dissection, it was found that preoperative SII was predictive for predicting postoperative multiorgan failure and 30-day mortality.[18] In another study, preoperative SII in patients who received on-pump CABG; has been linked to longer postoperative mechanical ventilation and ICU stay, AF, increased requirement for inotropic support, IABP support, and other infections other than sepsis.[17] To our knowledge, our study is the first to assess SII particularly in predicting AKI after CABG and isolated showed that SII was independently associated with AKI in patients undergoing CABG. According to our multivariate analysis result, we revealed that the risk of AKI increased 3.3 times with a cutoff value of SII>651.37 (65.0% sensitivity, 64.9% specificity).

It has been demonstrated that preoperative NLR elevation is determinative for both postoperative AKI and RRT requirements.[31] However, in patients receiving CABG, a high preoperative NLR was linked to poor baseline renal function.[32] Similar to NLR, preoperative PLR can be used as an independent indicator of AKI following cardiac surgery.[33] In comparison to either of these markers alone, SII can more accurately reflect the body's immunological and inflammatory condition.[34] Furthermore, several previous studies have contrasted the prognostic importance of the index with its components, including NLR or PLR. For instance, SII values have been demonstrated to predict postoperative AF more efficiently than NLR or PLR in patients undergoing isolated CABG.[16] Remarkably, we also discovered in our study that SII has a higher AUC for AKI compared to the AUC of NLR or PLR. This could be justified by the idea that calculating SII by adding platelet count to the NLR would simultaneously assess the aggregation route and the inflammatory pathway.[35,36]

In addition to SII, our study also showed that obesity, preoperative creatinine elevation, hypoalbuminemia, intraoperative pRBC transfusion, diuretic need, and prolonged CPB duration may all be risk factors for postoperative AKI. The secondary findings of our study are mostly consistent with previous studies analyzing risk factors for cardiac surgery-related AKI.[4,37-39] Adding preoperative SII to these risk factors may also enhance the selection of high-risk patients. The patient's risk for AKI can help doctors prepare for intraoperative and postoperative follow-up/management. In the high-risk patient group, such prevention of intraoperative nephrotoxic agents, optimization of intravascular volume, improvement perfusion during CPB, and prophylactic renal protective measures like mannitol/fenoldopam can be taken. Moreover, RRT can be initiated early or anti-inflammatory medication can be used, albeit there isn't enough evidence to support either one yet.[20,40]

Limitations

This research has several limitations. First, it was a single-center retrospective analysis. Some patients were excluded because of missing variables. The correlation between AKI and SII was found using spot laboratory data. Unfortunately, data on postoperative SII were not obtained. Inflammation caused by the CPB pump can cause bias. These results will need to be confirmed by a larger sample size prospective research.

In conclusion, the risk stratification method in cardiac surgery cohorts should adequately address the inflammatory profile of patients. As a simple, easily calculable, and reproducible inflammatory marker, SII is independently associated with an elevated risk of AKI in patients receiving CABG.

Disclosures

Ethics Committee Approval: Ankara City Hospital's local ethics committee approval (approval no: E1-22-2983, date: 19.10.2022) was obtained for the study.

Informed Consent: Written informed consent was obtained from all patients.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Financial Disclosure: The authors declared that this study has received no financial support.

Authorship Contributions: Concept – A.A., E.N.Z., B.B.A., N.S.; Design – A.A., E.N.Z.; Supervision – A.A., N.S.; Fundings – A.A., E.N.Z., B.B.A., N.S.; Materials – A.A., E.N.Z., B.B.A.; Data collection &/or processing – A.A., E.N.Z.; Analysis and/or interpretation – A.A., B.B.A., N.S.; Literature search – A.A., E.N.Z., B.B.A., N.S.; Writing – A.A., E.N.Z., B.B.A., N.S.; Critical review – A.A., E.N.Z.

References

1. Wang Y, Bellomo R. Cardiac surgery-associated acute kidney injury: Risk factors, pathophysiology and treatment. Nat Rev Nephrol 2017;13:697–711.
2. Ried M, Haneya A, Kolat P, Potzger T, Puehler T, Schmid C, et al. Acute renal dysfunction does not develop more frequently among octogenarians compared to septuagenarians after cardiac surgery. Thorac Cardiovasc Surg 2012;60:51–6.
3. Wu B, Chen J, Yang Y. Biomarkers of acute kidney ınjury after cardiac surgery: A narrative review. Biomed Res Int 2019;2019:7298635.
4. Parolari A, Pesce LL, Pacini D, Mazzanti V, Salis S, Sciacovelli C, et al. Risk factors for perioperative acute kidney injury after adult cardiac surgery: Role of perioperative management. Ann Thorac Surg 2012;93:584–91.
5. Rahmanian PB, Kwiecien G, Langebartels G, Madershahian N, Wittwer T, Wahlers T. Logistic risk model predicting postoperative renal failure requiring dialysis in cardiac surgery patients. Eur J Cardiothorac Surg 2011;40:701–7.
6. Siew ED, Ware LB, Ikizler TA. Biological markers of acute kidney injury. J Am Soc Nephrol 2011;22:810–20.
7. Wagener G, Jan M, Kim M, Mori K, Barasch JM, Sladen RN, et al. Association between increases in urinary neutrophil gelatinase-associated lipocalin and acute renal dysfunction after adult cardiac surgery. Anesthesiology 2006;105:485–91.
8. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med 2005;352:1685–95.
9. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation 2002;105:1135–43.
10. Sharfuddin AA, Molitoris BA. Pathophysiology of ischemic acute kidney injury. Nat Rev Nephrol 2011;7:189–200.
11. Bonventre JV, Yang L. Cellular pathophysiology of ischemic acute kidney injury. J Clin Invest 2011;121:4210–21.
12. Yang R, Chang Q, Meng X, Gao N, Wang W. Prognostic value of Systemic immune-inflammation index in cancer: A meta-analysis. J Cancer 2018;9:3295–302.
13. De Giorgi U, Procopio G, Giannarelli D, Sabbatini R, Bearz A, Buti S, et al. Association of systemic inflammation index and body mass index with survival in patients with renal cell cancer treated with nivolumab. Clin Cancer Res 2019;25:3839–46.
14. Candemir M, Kiziltunç E, Nurkoç S, Şahinarslan A. Relationship between systemic immune-inflammation index (SII) and the severity of stable coronary artery disease. Angiology 2021;72:575–81.
15. Huang J, Zhang Q, Wang R, Ji H, Chen Y, Quan X, et al. Systemic immune-inflammatory index predicts clinical outcomes for elderly patients with acute myocardial infarction receiving percutaneous coronary intervention. Med Sci Monit 2019;25:9690–701.
16. Selcuk M, Cinar T, Saylik F, Dogan S, Selcuk I, Orhan AL. Predictive value of systemic immune inflammation index for postoperative atrial fibrillation in patients undergoing isolated coronary artery bypass grafting. Medeni Med J 2021;36:318–24.
17. Dey S, Kashav R, Kohli JK, Magoon R, ItiShri, Walian A, et al. Systemic immune-inflammation index predicts poor outcome after elective Off-Pump cABG: A retrospective, single-center study. J Cardiothorac Vasc Anesth 2021;35:2397–404.
18. Xu H, Li Y, Wang H, Yuan Y, Chen D, Sun Y, et al. Systemic immune-inflammation index predicted short-term outcomes in ATAD patients undergoing surgery. J Card Surg 2022;37:969–75.
19. Thomas ME, Blaine C, Dawnay A, Devonald MA, Ftouh S, Laing C, et al. The definition of acute kidney injury and its use in practice. Kidney Int 2015;87:62–73.
20. García-Fernández N, Pérez-Valdivieso JR, Bes-Rastrollo M, Vives M, Lavilla J, Herreros J, et al. Timing of renal replacement therapy after cardiac surgery: A retrospective multicenter Spanish cohort study. Blood Purif 2011;32:104–11.
21. Multhoff G, Molls M, Radons J. Chronic inflammation in cancer development. Front Immunol 2012;2:98.
22. Hotamisligil GS. Inflammation and metabolic disorders. Nature 2006;444:860–7.
23. Danesh J, Whincup P, Walker M, Lennon L, Thomson A, Appleby P, et al. Low grade inflammation and coronary heart disease: Prospective study and updated meta-analyses. BMJ 2000;321:199–204.
24. Handin RI. Platelets and coronary artery disease. N Engl J Med 1996;334:1126–7.
25. Ateş AH, Aytemir K, Koçyiğit D, Yalcin MU, Gürses KM, Yorgun H, et al. Association of neutrophil-to-lymphocyte ratio with the severity and morphology of coronary atherosclerotic plaques detected by multidetector computerized tomography. Acta Cardiol Sin 2016;32:676–83.
26. Yüksel M, Yıldız A, Oylumlu M, Akyüz A, Aydın M, Kaya H, et al. The association between platelet/lymphocyte ratio and coronary artery disease severity. Anatol J Cardiol 2015;15:640–7.
27. Zhong JH, Huang DH, Chen ZY. Prognostic role of systemic immune-inflammation index in solid tumors: A systematic review and meta-analysis. Oncotarget 2017;8:75381–8.

28. Hu B, Yang XR, Xu Y, Sun YF, Sun C, Guo W, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. Clin Cancer Res 2014;20:6212–22.
29. Lerman DA, Diaz M, Peault B. Changes in coexpression of pericytes and endogenous cardiac progenitor cells from heart development to disease state. Eur Heart J 2018;39(Suppl 1):P1850.
30. Yang YL, Wu CH, Hsu PF, Chen SC, Huang SS, Chan WL, et al. Systemic immune-inflammation index (SII) predicted clinical outcome in patients with coronary artery disease. Eur J Clin Invest 2020;50:e13230.
31. Wheatley J, Liu Z, Loth J, Plummer MP, Penny-Dimri JC, Segal R, et al. The prognostic value of elevated neutrophil-lymphocyte ratio for cardiac surgery-associated acute kidney injury: A systematic review and meta-analysis. Acta Anaesthesiol Scand 2023;67:131–41.
32. Gibson PH, Croal BL, Cuthbertson BH, Small GR, Ifezulike AI, Gibson G, et al. Preoperative neutrophil-lymphocyte ratio and outcome from coronary artery bypass grafting. Am Heart J 2007;154:995–1002.
33. Parlar H, Şaşkın H. Are pre and postoperative platelet to lymphocyte ratio and neutrophil to lymphocyte ratio associated with early postoperative AKI following CABG? Braz J Cardiovasc Surg 2018;33:233–41.
34. Aziz MH, Sideras K, Aziz NA, Mauff K, Haen R, Roos D, et al. The systemic-immune-inflammation index independently predicts survival and recurrence in resectable pancreatic cancer and its prognostic value depends on bilirubin levels: A retrospective multicenter cohort study. Ann Surg 2019;270:139–46.
35. Magoon R. Evolving spectrum of prognostic inflammatory markers in infective endocarditis. J Cardiothorac Vasc Anesth 2020;34:2001–2.
36. Koo CH, Eun Jung D, Park YS, Bae J, Cho YJ, Kim WH, et al. Neutrophil, lymphocyte, and platelet counts and acute kidney injury after cardiovascular surgery. J Cardiothorac Vasc Anesth 2018;32:212–22.
37. Kim WH, Park MH, Kim HJ, Lim HY, Shim HS, Sohn JT, et al. Potentially modifiable risk factors for acute kidney injury after surgery on the thoracic aorta: A propensity score matched case-control study. Medicine (Baltimore) 2015;94:e273.
38. Lee EH, Baek SH, Chin JH, Choi DK, Son HJ, Kim WJ, et al. Preoperative hypoalbuminemia is a major risk factor for acute kidney injury following off-pump coronary artery bypass surgery. Intensive Care Med 2012;38:1478–86.
39. Tinica G, Brinza C, Covic A, Popa IV, Tarus A, Bacusca AE, et al. Determinants of acute kidney injury after cardiac surgery: A systematic review. Rev Cardiovasc Med 2020;21:601–10.
40. Scrascia G, Guida P, Rotunno C, de Luca Tupputi Schinosa L, Paparella D. Anti-inflammatory strategies to reduce acute kidney injury in cardiac surgery patients: A meta-analysis of randomized controlled trials. Artif Organs 2014;38:101–12.