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ORIGINAL RESEARCH

Correlation of Novel Inflammation Index with Postoperative Acute Kidney Injury in Patients with Joint Arthroplasty: A Retrospective Cohort Study

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Background: Acute kidney injury (AKI) is a frequent complication following joint arthroplasty. This study investigated the association between novel inflammation indices and postoperative AKI.

Methods: This retrospective cohort study included 1434 patients who underwent hip or knee arthroplasty, with 1225 patients comprising the complete case analysis dataset. The primary analysis was performed using the complete case analysis dataset, while sensitivity analyses were conducted in both the imputed dataset and the dataset excluding patients with abnormal preoperative creatinine. Inflammation indices, including the Systemic Immune-Inflammation Index (SII), Systemic Inflammatory Response Index (SIRI), Aggregate Index of Systemic Inflammation (AISI), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR), were derived from neutrophil, lymphocyte, monocyte, and platelet counts. Multivariate logistic regression and receiver-operating characteristic curve (ROC) analyses were performed to assess predictive performance. The area under the ROC (AUC) was compared using DeLong test.

Results: Among 1225 patients in the complete case analysis dataset, 116 (9.47%) developed AKI (Stage I: 59, Stage II: 55, Stage III: 2). Elevated SIRI and MLR were independently associated with increased AKI risk [adjusted OR (95% CI) for Quartile 4 vs Quartile 1: 1.961 (1.070–3.595) and 1.902 (1.043–3.468), respectively]. AISI showed marginal significance [adjusted trend OR (95% CI): 1.192 (0.995–1.428)]. The predictive performance of the basic model (AUC = 0.565) significantly improved after incorporating SIRI, AISI, and MLR (ΔAUC: +0.066, +0.065, +0.070, respectively; all DeLong $P < 0.05$). Monocyte count alone also enhanced prediction (ΔAUC: +0.065, DeLong $P < 0.05$). Sensitivity analyses confirmed robustness.

Conclusion: SIRI, MLR, AISI, and monocyte count may serve as predictive indicators for AKI following joint arthroplasty. Further randomized trials are needed to establish causality.

Keywords: acute kidney injury, arthroplasty, inflammation, retrospective studies

Introduction

Hip and knee arthroplasty are standard procedures for degenerative joint diseases, effectively improving function, alleviating pain, and enhancing quality of life.[1] It is estimated that the number of patients who have a demand for hip and knee arthroplasty will double by 2026.[2] A notable complication following joint arthroplasty is acute kidney injury (AKI), occurring in 2–20% of cases,[3–5] which exacerbates hospital stays, chronic kidney disease risk, readmission, and mortality.[6,7] However, early AKI detection and treatment remain challenging, underscoring the need for predictive risk factors.

Inflammation plays a pivotal role in kidney injury. Peripheral inflammatory cytokines migrate to renal tissue, inducing oxidative stress, microvascular obstruction, and structural damage, thereby promoting the progression of kidney injury.[8]

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Novel hematologic inflammation indices, including the Systemic Immune-Inflammation Index (SII), Systemic Inflammatory Response Index (SIRI), Aggregate Index of Systemic Inflammation (AISI), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR), quantify the interplay between inflammation and immunity, emerging as excellent predictors of adverse outcomes. For instance, NLR, PLR, and MLR correlate with AKI after cardiovascular surgery,[9,10] while SII predicts AKI post-craniotomy and liver resection.[11,12] Our prior work also linked NLR to AKI following digestive surgery.[13]

Unlike other non-cardiac and cardiac surgical procedures, joint arthroplasty carries a substantial risk of intra-articular infection secondary to prosthesis implantation, which may be exacerbated by preexisting systemic inflammation.[14–16] Thus, the preoperative inflammatory state may particularly important for patients undergoing joint arthroplasty. Despite this, no studies have examined the association between novel inflammation indices and AKI in arthroplasty patients. This study aimed to evaluate six composite and four single-cell indices (neutrophil, lymphocyte, monocyte, and platelet counts) as predictors of AKI after hip/knee arthroplasty, identifying the most clinically actionable markers.

Materials and Methods

Study Design and Patients

This retrospective cohort study adhered to the Declaration of Helsinki and was approved by the Medical Ethics Committee of Nanfang Hospital, Southern Medical University (approval number: NFEC-2023-573). Informed consent was waived due to the retrospective property of the study. All patient data were confidential and handled in compliance with ethical guidelines.

We extracted clinical records of 1513 adult patients (≥18 years) who underwent hip or knee arthroplasty at Nanfang Hospital between 2019 and 2022 from the Perioperative Data Warehouse. For patients with multiple surgeries, only the first procedure was included. Exclusion criteria comprised preoperative renal disease, missing serum creatinine data, or incomplete inflammation indices (neutrophil, lymphocyte, monocyte, or platelet counts), resulting in 79 exclusions. The final cohort included 1434 patients. Among these, 209 had missing covariate data (missingness <10% per variable). Primary analyses utilized a complete dataset of 1225 patients, while sensitivity analyses employed multiple imputation datasets (Figure 1).

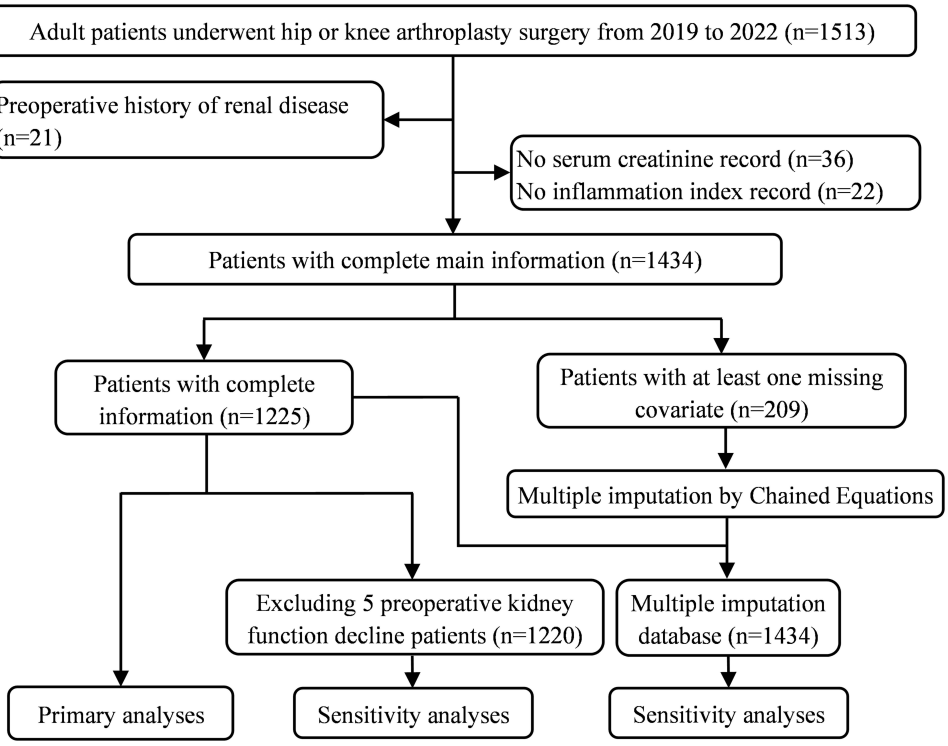


Figure 1 The flowchart of the study population.

Data Collection

Preoperative inflammatory markers, including complete blood count parameters (white blood cells, lymphocytes, neutrophils, monocytes, and platelet counts) and C-reactive protein levels, as well as pre- and postoperative serum creatinine levels within 7 days, were collected. The study incorporated covariates across multiple domains: (1) Demographics: Age, sex, smoking status, alcohol consumption, and body mass index (BMI); (2) Clinical characteristics: American Society of Anesthesiologists physical status (ASAPS), blood pressure, chronic comorbidities, and medication use; (3) Laboratory parameters: Preoperative serum hemoglobin, potassium, albumin, triglyceride, high-density lipoprotein cholesterol (HDL-C), glucose, urea, and uric acid; (4) Intraoperative variables: Anesthesia type, blood transfusion, vasopressor use, and fluid balance; (5) Postoperative information: duration of surgery and anesthesia, length of hospitalization, analgesic modality, and intensive care unit (ICU) admission.

Outcomes

AKI was diagnosed based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. The highest serum creatinine concentrations measured within 48 h and 7 days after surgery were extracted, respectively. AKI was defined as the highest serum creatinine level increased by ≥26.5 mmol/L within 48 h or increased by >1.5 times within 7 days postoperatively compared to the preoperative level. Subsequently, the severity of AKI was determined. Postoperative creatinine increased by ≥26.5 mmol/L within 48 hours, or increased by 1.5 to 1.9 times within 7 days than its preoperative level was defined as AKI in stage I. Increased by 2.0 to 2.9 times within 7 days was defined as AKI in stage II. An increase of ≥354 mmol/L within 48 h or more than three times within 7 days compared to the preoperative level was defined as AKI in stage III.

Novel Inflammation Index

Single inflammation indices used to calculate novel inflammation indices included neutrophil, lymphocyte, monocyte, and platelet counts. The definitions and calculation formulas for the six novel inflammation indices are as follows:

NLR, neutrophil count/lymphocyte count; SIRI, neutrophil count × monocyte count/lymphocyte count; AISI, neutrophil count × monocyte count × platelet count/lymphocyte count; SII, neutrophil count × platelet count/lymphocyte count; PLR, platelet count/lymphocyte count; MLR, monocyte count/lymphocyte count.

Statistical Analysis

This retrospective cohort study included all eligible patients meeting the predefined inclusion criteria. A priori power analysis conducted using PASS 2021 software (version 21.0.3) confirmed that the study achieved >90% statistical power with the current sample size (Appendix, Supplementary Files 1–6).

Analyses were performed according to a predefined statistical analysis plan comprising primary and sensitivity analyses. The primary analysis utilized the complete case dataset (n = 1225), while sensitivity analyses were conducted using multiply imputed data (n = 1434). Missing data were handled using five imputations via chained equations, with results reported as pooled odds ratios (ORs) with corresponding 95% confidence intervals (CIs). Additional sensitivity analyses excluded patients with preoperative renal dysfunction (defined as serum creatinine >132 μmol/L for males or >106 μmol/L for females) from the complete dataset. Comprehensive methodological details are available in the Appendix and Supplementary Methods section.

Continuous variables were assessed for normality using the Kolmogorov–Smirnov test. As all continuous variables demonstrated non-normal distributions, they were expressed as medians with interquartile ranges (IQRs) and compared using the Mann–Whitney *U*-test. Categorical variables were summarized as frequencies with percentages and analyzed using Pearson's chi-square test. Patients were stratified into quartiles based on preoperative inflammation index values. Trend analyses were performed to examine: (1) the incidence of postoperative AKI across inflammation index quartiles using the Cochran-Armitage trend test, and (2) the distribution of AKI stages across quartiles using the Mantel-Haenszel chi-square test for ordered categorical variables.

The association between inflammation index and AKI was evaluated through binary logistic regression, while its relationship with AKI severity was assessed using ordinal logistic regression. The inflammation index was analyzed both as quartiles and as a continuous variable (per standard deviation increase). Multivariable regression models were employed to adjust for potential confounding factors. Predictive performance was assessed using receiver operating characteristic (ROC) curve analysis. We first constructed a baseline model incorporating known confounders controlled in the logistic regression model. Subsequently, we evaluated the incremental predictive value of adding the inflammation index to this model by comparing the areas under the curves (AUCs) using DeLong test.

All analyses were performed using R statistical software (version 4.3.1; R Foundation for Statistical Computing, Vienna, Austria). A two-tailed *P*-value <0.05 was considered statistically significant throughout all analyses.

Results

Characteristics of the Patients

The baseline characteristics of the study population are presented in Table 1. Of the 1225 patients included in the complete dataset, 576 (47.0%) underwent hip arthroplasty, while 649 (53.0%) underwent knee arthroplasty. Postoperative AKI occurred in 116 patients (9.47%), with the following stage distribution: stage I (n = 59, 50.86%), stage II (n = 55, 47.41%), and stage III (n = 2, 1.72%).

Compared to patients without AKI, those who developed AKI were significantly older (median [IQR]: 63.00 [54.75–70.25] vs 61.00 [52.00–68.00]) and had lower preoperative creatinine levels (59.00 [50.00–69.00] vs 63.00 [54.00–74.00]). Additionally, patients with AKI exhibited higher systemic inflammatory markers, including monocyte count (0.51 [0.40–0.62] vs 0.47 [0.37–0.57]), systemic inflammation response index (SIRI) (0.85 [0.64–1.28] vs 0.79 [0.54–1.14]), and monocyte-to-lymphocyte ratio (MLR) (0.23 [0.19–0.31] vs 0.22 [0.18–0.28]). Surgical duration was longer in the AKI group (125.50 [110.00–159.25] vs 122.00 [102.00–150.00]), and postoperative peak creatinine levels were significantly elevated (95.87 [86.45–113.79] vs 64.00 [53.00–75.69]).

Table 1 Characteristics of Patients Grouped by Postoperative AKI and Non-AKI

Characteristics	Total (n = 1225)	Non-AKI (n = 1109)	AKI (n = 116)	*P*
Age (years)	61.00 (52.00–68.00)	61.00 (52.00–68.00)	63.00 (54.75–70.25)	0.031
Males, n (%)	410 (33.47)	380 (34.27)	30 (25.86)	0.068
Smoking, n (%)	214 (17.47)	194 (17.49)	20 (17.24)	0.946
Drinking, n (%)	246 (20.08)	228 (20.56)	18 (15.52)	0.197
Preoperative creatinine (μmol/L)	63.00 (54.00–73.00)	63.00 (54.00–74.00)	59.00 (50.00–69.00)	0.006
BMI (kg/m^2)	24.96 (22.31–27.47)	24.89 (22.27–27.40)	25.45 (22.83–27.98)	0.181
Systolic pressure (mmHg)	126.00 (116.00–136.00)	126.00 (115.00–135.00)	126.00 (117.75–139.00)	0.524
Diastolic pressure (mmHg)	78.00 (70.00–84.00)	78.00 (70.00–84.00)	78.00 (71.50–86.25)	0.338
Preoperative hemoglobin (g/L)	130.00 (120.00–139.00)	130.00 (120.00–139.00)	128.00 (120.00–137.00)	0.348
Albumin (g/L)	38.80 (37.10–40.80)	38.80 (37.10–40.90)	38.55 (37.18–40.38)	0.473
Blood glucose (mmol/L)	4.94 (4.53–5.57)	4.93 (4.53–5.62)	5.05 (4.61–5.42)	0.671
Urea (mmol/L)	5.50 (4.60–6.40)	5.50 (4.60–6.40)	5.60 (4.50–6.93)	0.340
Triglyceride (mg/dL)	120.00 (87.00–173.00)	120.00 (86.00–173.00)	120.00 (89.75–172.50)	0.774
HDL-C (mg/dL)	47.00 (39.00–57.00)	47.00 (39.00–57.00)	46.50 (40.00–58.25)	0.823
Uric acid (μmol/L)	360.00 (304.00–425.00)	360.00 (304.00–425.00)	359.50 (305.75–426.50)	0.919
CRP (mg/L)	2.44 (1.01–6.31)	2.39 (1.01–6.34)	3.04 (1.01–5.82)	0.829
White blood cell count ($\times 10^9$/L)	6.39 (5.38–7.59)	6.38 (5.36–7.53)	6.67 (5.55–7.97)	0.063
Platelet count ($\times 10^9$/L)	236.00 (199.00–276.00)	235.00 (200.00–275.00)	242.50 (194.00–282.00)	0.451
Lymphocyte count ($\times 10^9$/L)	2.07 (1.68–2.52)	2.07 (1.68–2.52)	2.08 (1.60–2.53)	0.814
Neutrophils count ($\times 10^9$/L)	3.59 (2.81–4.47)	3.57 (2.81–4.44)	3.84 (2.86–4.72)	0.111
Monocytes count ($\times 10^9$/L)	0.47 (0.37–0.57)	0.47 (0.37–0.57)	0.51 (0.40–0.62)	0.023

(*Continued*)

Table 1 (Continued).

Characteristics	Total (n = 1225)	Non-AKI (n = 1109)	AKI (n = 116)	*P*
SIRI	0.80 (0.55–1.15)	0.79 (0.54–1.14)	0.85 (0.64–1.28)	0.047
SII	397.56 (297.27–555.10)	395.18 (294.98–551.27)	434.78 (308.77–576.14)	0.265
AISI	184.20 (118.82–283.22)	182.00 (118.07–280.52)	210.32 (134.62–346.76)	0.061
NLR	1.72 (1.34–2.22)	1.71 (1.34–2.21)	1.80 (1.38–2.25)	0.255
PLR	112.03 (91.63–142.99)	111.91 (91.96–142.51)	115.10 (88.23–146.85)	0.952
MLR	0.22 (0.18–0.28)	0.22 (0.18–0.28)	0.23 (0.19–0.31)	0.030
ASAPS, n (%)				0.369
I	127 (10.37)	111 (10.01)	16 (13.79)	
II	986 (80.49)	898 (80.97)	88 (75.86)	
III or IV	112 (9.14)	100 (9.02)	12 (10.34)	
ACEI/ARB, n (%)	181 (14.78)	159 (14.34)	22 (18.97)	0.181
Calcium channel blockers, n (%)	224 (18.29)	203 (18.30)	21 (18.10)	0.957
Hypertension, n (%)	464 (37.88)	417 (37.60)	47 (40.52)	0.538
Coronary heart disease, n (%)	30 (2.45)	25 (2.25)	5 (4.31)	0.295
Diabetes, n (%)	110 (8.98)	99 (8.93)	11 (9.48)	0.842
Dyslipidemia, n (%)	16 (1.31)	15 (1.35)	1 (0.86)	0.990
Stroke, n (%)	23 (1.88)	21 (1.89)	2 (1.72)	1.000
Duration of surgery (min)	122.00 (103.00–151.00)	122.00 (102.00–150.00)	125.50 (110.00–159.25)	0.042
Duration of anesthesia (min)	224.00 (189.00–268.00)	224.00 (189.00–268.00)	230.50 (189.50–272.00)	0.799
Anesthesia type, n (%)				0.131
General anesthesia	480 (39.18)	427 (38.50)	53 (45.69)	
Spinal anesthesia	745 (60.82)	682 (61.50)	63 (54.31)	
Blood transfusion, n (%)	272 (22.20)	246 (22.18)	26 (22.41)	0.954
Vasoactive drugs, n (%)	956 (78.04)	865 (78.00)	91 (78.45)	0.911
Balance of inflow and outflow (mL)	−1000 (−1300, −600)	−1000 (−1300, −600)	−1000 (−1300, −700)	0.470
The type of arthroplasty, n (%)				
Hip	576 (47.0)	515 (46.4)	61 (52.6)	0.207
Knee	649 (53.0)	594 (53.6)	55 (47.4)	
Analgesic pump, n (%)	1116 (91.10)	1007 (90.80)	109 (93.97)	0.255
Intensive care unit, n (%)	9 (0.73)	9 (0.81)	0 (0.00)	–
Postoperative highest creatinine (μmol/L)	66.00 (54.00–80.00)	64.00 (53.00–75.69)	95.87 (86.45–113.79)	<0.001
Stage of AKI, n (%)				
Stage I	59 (4.82)	–	59 (50.86)	–
Stage II	55 (4.49)	–	55 (47.41)	–
Stage III	2 (0.16)	–	2 (1.72)	

Abbreviations: ACEI, angiotensin-converting enzyme inhibitors; AISI, Aggregate Index of Systemic Inflammation; AKI, acute kidney injury; ASAPS, American Society of Anesthesiologists physical status; ARB, Angiotensin receptor antagonists; BMI, body mass index; CRP, C-reactive protein; HDL-C, High density lipoprotein cholesterol; MLR, monocyte-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SIRI, Systemic Inflammatory Response Index; SII, Systemic Immune Inflammatory Index.

Association of Novel Inflammation Index with the Incident AKI

The incidence of AKI gradually increased with quartiles of SIRI and MLR (Figure 2). Binary logistic regression analyses showed that the SIRI and MLR were positively associated with postoperative AKI. The adjusted OR and 95% CI of AKI for the fourth quartile of SIRI and MLR were 1.961 (1.070–3.595) and 1.902 (1.043–3.468), respectively, compared with the first quartile. The odds of AKI also increased with per SD change in SIRI and MLR, with adjusted OR and 95% CI of 1.229 (1.006–1.502) and 1.287 (0.999–1.658), respectively. For AISI, a marginal significance for the association with AKI was observed, with an adjusted OR and 95% CI of 1.192 (0.995~1.428) for the trend of quartiles (Table 2).

Given that the number of patients with stage III kidney injury was scarce, in ordinal logistic regression, patients with stages III and II were merged. SIRI and MLR were positively associated with postoperative AKI severity, with adjusted OR and 95% CI of 1.969 (1.075–3.604) and 1.929 (1.057–3.520), respectively. The severity of AKI also increased with

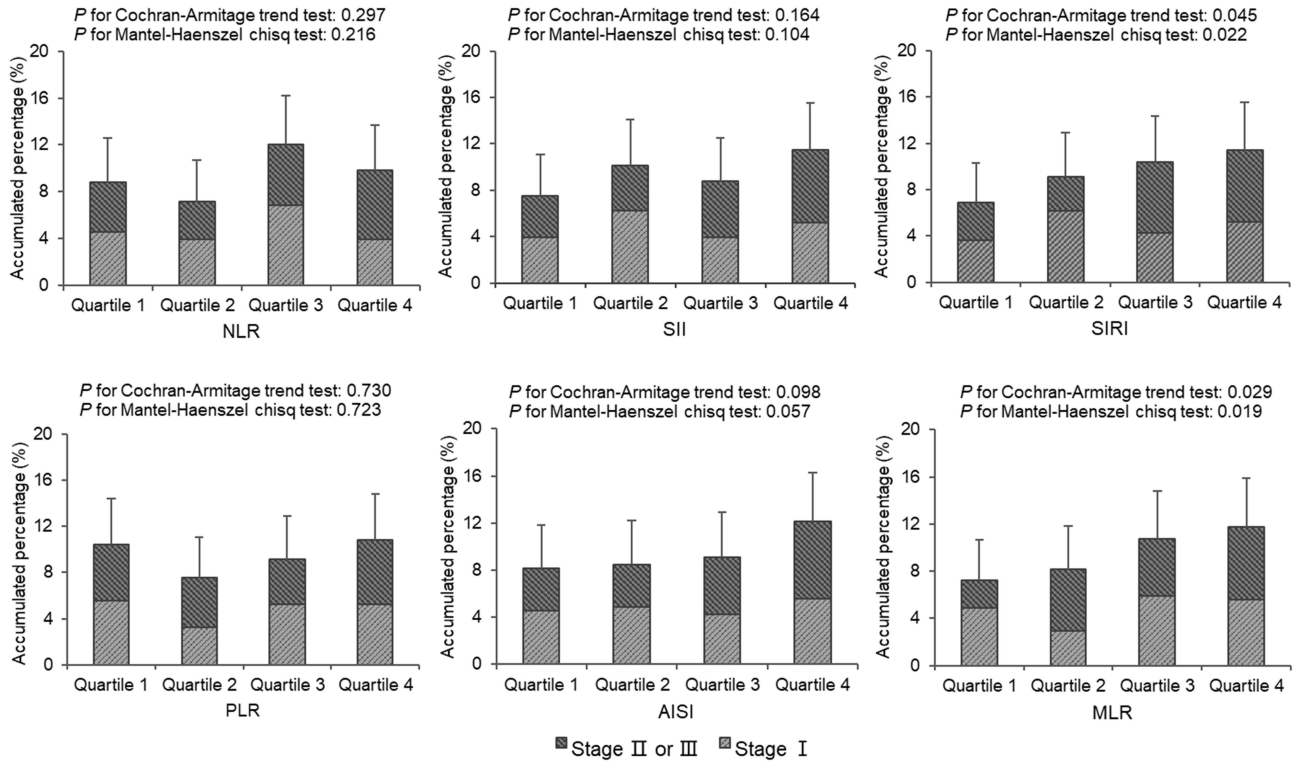


Figure 2 The incidence of AKI and AKI stage among the quartile of novel inflammation index.
Abbreviations: AISI, Aggregate Index of Systemic Inflammation; AKI, acute kidney injury; MLR, the monocyte-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SII, Systemic Immune Inflammatory Index; SIRI, Systemic Inflammatory Response Index.

the level of SIRI [adjusted OR and 95% CI: 1.252 (1.037–1.512) for the trend of SIRI quartiles, 1.253 (1.040–1.509) for per SD of SIRI] and MLR [adjusted OR and 95% CI: 1.254 (1.040–1.513) for MLR quartiles, 1.299 (1.025–1.647) per SD of MLR]. In addition, AISI was associated with AKI severity with marginal significance (adjusted OR and 95% CI: 1.196 [0.998–1.433] for the trend of quartiles) (Table 3). Sensitivity analyses after multiple imputations or exclusion of patients with preoperative kidney function decline showed similar results (Appendix, Supplementary Tables 1–4).

Table 2 Associations Between Novel Inflammation Indices and Postoperative AKI

Inflammation indices		AKI, n (%)	OR (95% CI) of postoperative AKI		
			Model 1	Model 2	Model 3
NLR	Quartile 1	27 (8.82)	1	1	1
	Quartile 2	22 (7.19)	0.800 (0.445~1.439)	0.805 (0.444~1.459)	0.783 (0.432~1.419)
	Quartile 3	37 (12.05)	1.416 (0.839~2.390)	1.411 (0.826~2.410)	1.423 (0.835~2.426)
	Quartile 4	30 (9.80)	1.123 (0.651~1.939)	1.083 (0.606~1.935)	1.143 (0.643~2.031)
	Trend		1.096 (0.923~1.301)	1.085 (0.904~1.302)	1.107 (0.923~1.328)
	Per SD		1.139 (0.968~1.339)	1.113 (0.927~1.337)	1.145 (0.951~1.379)
SII	Quartile 1	23 (7.52)	1	1	1
	Quartile 2	31 (10.13)	1.387 (0.789~2.438)	1.413 (0.795~2.512)	1.396 (0.788~2.474)
	Quartile 3	27 (8.79)	1.186 (0.664~2.119)	1.212 (0.670~2.192)	1.187 (0.658~2.141)
	Quartile 4	35 (11.44)	1.589 (0.915~2.759)	1.623 (0.913~2.883)	1.629 (0.922~2.878)
	Trend		1.130 (0.951~1.342)	1.136 (0.949~1.358)	1.137 (0.952~1.359)
	Per SD		1.067 (0.906~1.256)	1.038 (0.875~1.232)	1.038 (0.877~1.229)

(*Continued*)

Table 2 (Continued).

Inflammation indices		AKI, n (%)	OR (95% CI) of postoperative AKI		
			Model 1	Model 2	Model 3
SIRI	Quartile 1	21 (6.86)	1	1	1
	Quartile 2	28 (9.15)	1.367 (0.758~2.464)	1.331 (0.731~2.424)	1.343 (0.738~2.443)
	Quartile 3	32 (10.42)	1.579 (0.889~2.806)	1.623 (0.896~2.940)	1.661 (0.921~2.998)
	Quartile 4	35 (11.44)	1.753 (0.995~3.087)	1.851 (1.007~3.402)	1.961 (1.070~3.595)
	Trend		1.193 (1.003~1.418)	1.223 (1.012~1.477)	1.247 (1.032~1.505)
	Per SD		1.227 (1.033~1.458)	1.197 (0.999~1.434)	1.229 (1.006~1.502)
PLR	Quartile 1	32 (10.46)	1	1	1
	Quartile 2	23 (7.52)	0.696 (0.397~1.220)	0.657 (0.370~1.166)	0.678 (0.383~1.200)
	Quartile 3	28 (9.12)	0.859 (0.504~1.466)	0.821 (0.472~1.429)	0.806 (0.465~1.398)
	Quartile 4	33 (10.78)	1.035 (0.619~1.731)	0.993 (0.575~1.715)	1.001 (0.587~1.707)
	Trend		1.031 (0.868~1.223)	1.019 (0.850~1.221)	1.016 (0.851~1.214)
	Per SD		1.022 (0.848~1.232)	1.006 (0.826~1.226)	0.998 (0.821~1.214)
AISI	Quartile 1	25 (8.17)	1	1	1
	Quartile 2	26 (8.50)	1.044 (0.588~1.852)	1.022 (0.570~1.834)	1.051 (0.587~1.881)
	Quartile 3	28 (9.12)	1.128 (0.642~1.983)	1.179 (0.660~2.106)	1.202 (0.675~2.139)
	Quartile 4	37 (12.09)	1.546 (0.906~2.638)	1.678 (0.953~2.955)	1.688 (0.964~2.954)
	Trend		1.156 (0.973~1.374)	1.192 (0.992~1.431)	1.192 (0.995~1.428)
	Per SD		1.131 (0.995~1.285)	1.095 (0.956~1.255)	1.103 (0.960~1.268)
MLR	Quartile 1	22 (7.19)	1	1	1
	Quartile 2	25 (8.17)	1.148 (0.633~2.085)	1.233 (0.670~2.269)	1.201 (0.654~2.206)
	Quartile 3	33 (10.75)	1.555 (0.884~2.734)	1.664 (0.927~2.986)	1.648 (0.922~2.945)
	Quartile 4	36 (11.76)	1.721 (0.987~3.001)	1.868 (1.019~3.423)	1.902 (1.043~3.468)
	Trend		1.212 (1.019~1.441)	1.241 (1.028~1.499)	1.251 (1.037~1.510)
	Per SD		1.242 (1.015~1.518)	1.233 (0.982~1.548)	1.287 (0.999~1.658)

Notes: Model 1: Unadjusted; Model 2: Adjusted for age, sex, BMI, albumin, glucose, ASAPS, anesthesia type, blood transfusion, balance of inflow and outflow, duration of surgery, and use of vasoactive drugs; Model 3: Adjusted for Model 2 plus preoperative creatinine.

Abbreviations: AISI, Aggregate Index of Systemic Inflammation; AKI, acute kidney injury; CI, confidence interval; MLR, monocyte-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; PLR, platelet-to-lymphocyte ratio; SD, standard deviation; SII, Systemic Immune Inflammatory Index; SIRI, Systemic Inflammatory Response Index.

Table 3 Associations Between Novel Inflammation Indices and Postoperative AKI Stage

Inflammation indices		AKI of stage I, n (%)	AKI of stage II or III, n (%)	OR (95% CI) of postoperative AKI stage		
				Model 1	Model 2	Model 3
NLR	Quartile 1	14 (4.58)	13 (4.25)	1	1	1
	Quartile 2	12 (3.92)	10 (3.27)	0.799 (0.445~1.437)	0.794 (0.439~1.438)	0.785 (0.433~1.424)
	Quartile 3	21 (6.84)	16 (5.21)	1.405 (0.832~2.371)	1.407 (0.827~2.395)	1.412 (0.828~2.407)
	Quartile 4	12 (3.92)	18 (5.88)	1.137 (0.660~1.960)	1.135 (0.641~2.010)	1.160 (0.654~2.057)
	Trend			1.099 (0.926~1.305)	1.102 (0.920~1.321)	1.111 (0.927~1.333)
	Per SD			1.152 (0.982~1.351)	1.138 (0.951~1.361)	1.157 (0.965~1.388)
SII	Quartile 1	12 (3.92)	11 (3.59)	1	1	1
	Quartile 2	19 (6.21)	12 (3.92)	1.371 (0.779~2.412)	1.381 (0.780~2.447)	1.388 (0.782~2.463)
	Quartile 3	12 (3.91)	15 (4.89)	1.195 (0.670~2.131)	1.229 (0.683~2.210)	1.216 (0.675~2.189)
	Quartile 4	16 (5.23)	19 (6.21)	1.600 (0.922~2.774)	1.604 (0.910~2.828)	1.639 (0.928~2.894)
	Trend			1.134 (0.955~1.347)	1.137 (0.952~1.357)	1.142 (0.956~1.365)
	Per SD			1.070 (0.911~1.258)	1.034 (0.875~1.221)	1.040 (0.881~1.226)

(*Continued*)

Table 3 (Continued).

Inflammation indices		AKI of stage I, n (%)	AKI of stage II or III, n (%)	OR (95% CI) of postoperative AKI stage		
				Model 1	Model 2	Model 3
SIRI	Quartile 1	11 (3.59)	10 (3.27)	1	1	1
	Quartile 2	19 (6.21)	9 (2.94)	1.345 (0.745~2.428)	1.310 (0.720~2.384)	1.319 (0.724~2.404)
	Quartile 3	13 (4.23)	19 (6.19)	1.599 (0.901~2.836)	1.681 (0.934~3.024)	1.678 (0.931~3.025)
	Quartile 4	16 (5.23)	19 (6.21)	1.764 (1.003~3.104)	1.886 (1.033~3.444)	1.969 (1.075~3.604)
	Trend			1.199 (1.009~1.425)	1.236 (1.025~1.491)	1.252 (1.037~1.512)
	Per SD			1.251 (1.064~1.471)	1.233 (1.037~1.466)	1.253 (1.040~1.509)
PLR	Quartile 1	17 (5.56)	15 (4.90)	1	1	1
	Quartile 2	10 (3.27)	13 (4.25)	0.703 (0.402~1.230)	0.680 (0.385~1.201)	0.687 (0.388~1.214)
	Quartile 3	16 (5.21)	12 (3.91)	0.857 (0.502~1.461)	0.829 (0.479~1.436)	0.815 (0.470~1.413)
	Quartile 4	16 (5.23)	17 (5.56)	1.041 (0.623~1.739)	1.007 (0.592~1.712)	1.012 (0.595~1.724)
	Trend			1.031 (0.869~1.223)	1.020 (0.855~1.218)	1.019 (0.854~1.217)
	Per SD			1.026 (0.852~1.235)	0.996 (0.824~1.204)	1.001 (0.825~1.214)
AISI	Quartile 1	14 (4.58)	11 (3.59)	1	1	1
	Quartile 2	15 (4.90)	11 (3.59)	1.042 (0.587~1.850)	1.044 (0.584~1.867)	1.052 (0.587~1.885)
	Quartile 3	13 (4.23)	15 (4.89)	1.138 (0.648~1.999)	1.205 (0.678~2.139)	1.218 (0.685~2.166)
	Quartile 4	17 (5.56)	20 (6.54)	1.562 (0.916~2.663)	1.667 (0.955~2.911)	1.701 (0.973~2.975)
	Trend			1.161 (0.977~1.379)	1.189 (0.993~1.423)	1.196 (0.998~1.433)
	Per SD			1.145 (1.013~1.293)	1.111 (0.976~1.266)	1.117 (0.979~1.274)
MLR	Quartile 1	15 (4.90)	7 (2.29)	1	1	1
	Quartile 2	9 (2.94)	16 (5.23)	1.179 (0.650~2.140)	1.248 (0.681~2.290)	1.231 (0.670~2.262)
	Quartile 3	18 (5.86)	15 (4.89)	1.567 (0.890~2.761)	1.688 (0.944~3.017)	1.664 (0.929~2.980)
	Quartile 4	17 (5.56)	19 (6.21)	1.751 (1.003~3.055)	1.909 (1.049~3.474)	1.929 (1.057~3.520)
	Trend			1.215 (1.022~1.445)	1.249 (1.037~1.505)	1.254 (1.040~1.513)
	Per SD			1.260 (1.044~1.521)	1.265 (1.020~1.569)	1.299 (1.025~1.647)

Notes: Model 1: Unadjusted; Model 2: Adjusted for age, sex, BMI, albumin, glucose, ASAPS, anesthesia type, blood transfusion, balance of inflow and outflow, duration of surgery, and use of vasoactive drugs; Model 3: Adjusted for Model 2 plus preoperative creatinine.

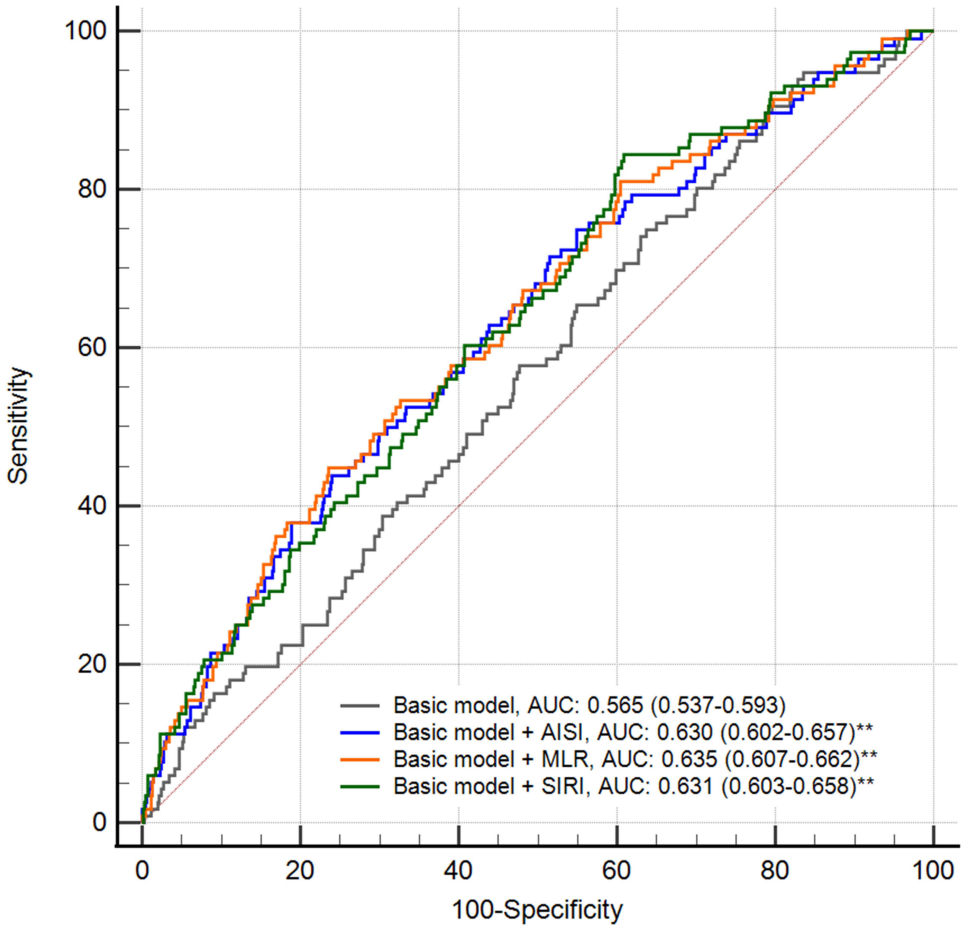
Abbreviations: AISI, Aggregate Index of Systemic Inflammation; AKI, acute kidney injury; CI, confidence interval; MLR, monocyte-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; PLR, platelet-to-lymphocyte ratio; SD, standard deviation; SII, Systemic Immune Inflammatory Index; SIRI, Systemic Inflammatory Response Index.

Association of Single Inflammation Indices with the Incident AKI

Among the four inflammation indices, only monocyte count was significantly associated with increased incidence of postoperative AKI (Appendix and Supplementary Figure 1). The odds of postoperative AKI were higher in the fourth quartile than in the first quartile of monocyte count (adjusted OR and 95% CI, 2.121 [1.194–3.768]). The odds of postoperative AKI increased with monocyte count [adjusted OR and 95% CI: 1.272 (1.055–1.532) for the trend of quartiles, and 1.410 (1.104–1.802) for per SD] (Appendix and Supplementary Table 5). Ordinal logistic regression analysis showed similar results for the association between monocyte count and postoperative AKI stage (Appendix and Supplementary Table 6).

Predictive Ability of Inflammation Indices for the Incident AKI

These results showed that the SIRI, AISI, MLR, and monocyte count were significantly associated with postoperative AKI. We further validated their predictive abilities. The AUC of postoperative AKI based on confounders was 0.565 (0.537–0.593). After adding SIRI, AISI, and MLR to the ROC model, the AUC significantly increased by 0.066, 0.065, and 0.070, respectively (Figure 3). The AUC significantly increased by 0.065 after adding monocyte count to the ROC model (Appendix and Supplementary Figure 2). However, the AUCs for the SIRI, AISI, MLR, and monocyte count were not significantly different (Appendix, Supplementary Table 7 and Supplementary Figure 3).



	Basic model		
	Difference between areas	95% CI	*P*
Basic model + AISI	0.065	0.008–0.121	0.025
Basic model + MLR	0.070	0.011–0.129	0.020
Basic model + SIRI	0.066	0.010–0.122	0.021

Figure 3 ROC analyses of AISI, MLR and SIRI and postoperative AKI. Basic model included age, sex, BMI, albumin, glucose, ASAPS, anesthesia type, blood transfusion, balance of inflow and outflow, duration of surgery, the use of vasoactive drugs and preoperative creatinine. **Significant different in AUC compared with basic model.
Abbreviations: AISI, Aggregate Index of Systemic Inflammation; AKI, acute kidney injury; AUC, area under curve; CI, confidence interval; MLR, the monocyte-to-lymphocyte ratio; SIRI, Systemic Inflammatory Response Index.

Discussion

The present study demonstrated that preoperative SIRI, AISI, MLR, and monocyte count were significantly associated with an increased risk of postoperative acute kidney injury (AKI) in patients undergoing joint arthroplasty. While these four indices enhanced the predictive capacity for AKI, their individual predictive performances did not show statistically significant differences.

The pathophysiological link between inflammation and AKI is well-established. Inflammatory responses can induce renal vascular dysregulation and functional impairment through cytokine-mediated immune cell infiltration into renal parenchyma.[17,18] This mechanistic understanding supports the potential utility of preoperative inflammatory markers as predictors of postoperative AKI. Recent literature has highlighted the superior predictive value of novel inflammation indices (including NLR, SIRI, AISI, SII, PLR, and MLR) over single inflammation parameters, as they more comprehensively reflect systemic inflammation and immune status.[19,20] These indices offer additional clinical advantages as they

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are derived from routine laboratory tests, making them cost-effective and readily available. However, their specific utility for predicting postoperative AKI across different surgical contexts remains unclear.

Our findings present interesting contrasts with previous research. While our prior study identified NLR as a predictor of AKI in digestive surgery but not musculoskeletal surgery patients,[13] other reports have shown NLR and PLR to be independent predictors of AKI following coronary artery bypass grafting,[10] and SII is associated with AKI in neurosurgical patients.[12] In addition, SII is also associated with chronic kidney disease in the general population.[20,21] The absence of significant associations between NLR, PLR, SII and AKI in our arthroplasty cohort suggests possible procedure-specific variations in inflammatory sensitivity, warranting further investigation across surgical disciplines.

Current evidence regarding SIRI, AISI, and MLR shows considerable heterogeneity. While some studies found no association between these indices and AKI in craniotomy or valve replacement patients,[12,19] others reported MLR as a cost-effective AKI predictor in cardiac surgery and ICU settings,[22–24] and SIRI as a potential biomarker in acute pancreatitis.[25] Our results position SIRI, AISI, and MLR as novel predictive markers specifically for AKI in joint arthroplasty patients.

The cellular basis of these indices merits consideration. Neutrophils, as rapid responders to inflammatory stimuli, serve as early inflammation markers,[26–28] while monocytes amplify inflammatory cascades through cytokine release and immune cell activation.[20] Lymphocytes, crucial for adaptive immunity, reflect immune competence through their depletion.[29] In our cohort, among individual leukocyte subpopulations, only monocyte count demonstrated a significant association with postoperative AKI. This observation aligns with the known role of monocytes in orchestrating inflammatory responses, potentially through neutrophil and lymphocyte activation. However, composite indices such as SIRI and MLR, which integrate multiple leukocyte-derived parameters, are generally regarded as more robust indicators of low-grade chronic inflammation due to their ability to capture broader immune dysregulation. The AISI further incorporates platelet-to-white blood cell ratios, with prior evidence suggesting its utility in risk stratification for prolonged hospitalization in thoracic surgery patients.[30] Notably, our findings indicate that monocyte count alone exhibited predictive performance comparable to that of composite indices (SIRI, AISI, MLR) for AKI risk. This may reflect the central role of monocytes in initiating and sustaining inflammatory cascades relevant to AKI pathogenesis. However, the evidence supporting monocyte-driven mechanisms in joint arthroplasty–associated AKI remains less established compared with other surgical contexts. Nevertheless, our study confirms that both novel inflammation indices (SIRI, AISI, MLR) and monocyte count are independently associated with an elevated risk of postoperative AKI in this patient population.

The current evidence demonstrates significant heterogeneity in the associations between novel inflammation indices and AKI across different surgical procedures and disease states. This variability may be attributed to several factors: (1) differential surgical trauma and tissue injury patterns; (2) procedure-specific inflammatory responses; and (3) variations in renal susceptibility to inflammatory insults. Joint arthroplasty presents unique characteristics that may amplify inflammatory responses, including prosthetic implantation-induced foreign body reactions and more pronounced postoperative pain compared with other surgical interventions. These factors likely enhance renal sensitivity to preoperative inflammatory states. Our findings support this hypothesis, demonstrating higher AKI incidence in hip/knee arthroplasty patients relative to general non-cardiac surgery populations.[13] This observation underscores the importance of considering procedure-specific risk profiles when interpreting inflammatory markers.

The biological properties of these indices may further explain their variable performance. While SIRI, AISI, and MLR incorporate monocyte counts in their calculations, and activated monocytes/macrophages are known to release cytotoxic mediators (eg, interferon-γ, TNF-α, IL-1β) that promote renal injury, our data surprisingly showed no superior predictive value of these composite indices over monocyte count alone. This suggests that in arthroplasty patients, monocyte-driven inflammation may be particularly salient for AKI pathogenesis. These findings emphasize the need for context-specific application of inflammatory biomarkers in perioperative risk assessment.

This study has several limitations. First, this retrospective study could not determine a causal relationship between inflammation and postoperative AKI, and a potential selection bias was hard to avoid. A randomized controlled trial may be helpful for disclosure. Secondly, this study did not clarify the mechanism of inflammation in postoperative AKI. Third, postoperative biochemical examinations were missing, and dynamic changes in inflammation were not explored. Fourth, small sample size and absence of external data lead to incapable of internal and external verification. Finally, we could only

adjust for several important covariates in the multivariable models due to the small sample size of patients with AKI. Thus, the residual confounding in this study is concerning.

Conclusions

This study suggests that higher inflammation indices, including SIRI, AISI, MLR, and monocyte count, can predict a higher risk of postoperative AKI in patients undergoing joint arthroplasty. These indices may have potential applications as simple, inexpensive, and easily accessible markers in the clinical setting. However, other inflammation indices may not be able to predict postoperative AKI, which need to be validated in a larger prospective study.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no conflicts of interest.

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