DOI: 10.14744/ejmi.2021.89773
EJMI 2021;5(4):435–440

Research Article

Systemic Immune-Inflammation Index is a Novel Marker to Predict Contrast-Induced Acute Kidney Injury

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Abstract

Objectives: Systemic immune-inflammation index (SII) is a novel marker based on blood peripheral platelet, neutrophil, and lymphocyte counts. We aimed to evaluate the worth of a SII in predicting contrast-induced nephropathy.

Methods: A total number of 190 consecutive patients who underwent emergency percutaneous coronary intervention were included in the study. Patients were divided into two groups, according to contrast-induced acute kidney injury (CI-AKI) development. Patients with an increase in serum creatinine of ≥ 0.5 mg/dL or a 25% increase from baseline assessed 48-72 hours after angiography without an alternative explanation were considered CI-AKI. SII is calculated with a formula of total platelet count x total neutrophil count / total lymphocyte count.

Results: Two groups were assigned according to CI-AKI development. CI-AKI (+) group included 33 patients, and CI-AKI (-) group included 157 patients. CI-AKI developed patients with higher serum creatinine levels, NLR (Neutrophil-Lymphocyte Ratio), and SII levels. Whereas, in the non-CI-AKI group, e-GFR, serum albumin levels, and ejection fraction were higher. Multivariable logistic regression analysis revealed that higher SII (p=0.001, [OR]=1.002[1001-1004]) were independent risk factors for CI-AKI.

Conclusion: The current study demonstrated that SII, a novel marker based on blood peripheral cell counts, is a promising prognostic factor for predicting CI-AKI.

Keywords: Systemic immune-inflammation index, contrast-induced acute kidney injury, coronary artery disease

Cite This Article: *Gucun M, Akyuz O, Bayram E, Cakmak EO. Systemic Immune-Inflammation Index is a Novel Marker to Predict Contrast-Induced Acute Kidney Injury. EJMI 2021;5(4):435–440.*

Iodinated radiocontrast agents have been known for decades to cause acute kidney injury (AKI) when administered intravenously or intra-arterially. AKI that is judged to be caused by iodinated contrast material has historically been called contrast-induced nephropathy (CIN) but has since been termed contrast-induced AKI (CI-AKI). AKI developing after contrast material administration is reversible in most cases, but its development may be associated with adverse outcomes.[1] CI-AKI is generally determined as an increase of ≥ 0.5 mg/dL (≥44 µmol/L) or 25% from baseline in SCr (serum creatinine) evaluated 48-72 hours after contrast media exposure.[2]

There is little agreement in the medical literature regarding the incidence of CI-AKI. Incidence rates in patients without any risk factors range from 3% to 7%. However, they may be as high as 50% in patients with risk factors such as chronic kidney disease, diabetes mellitus, intravascular volume reduction, high osmolality contrast media, proteinuria, anemia, and advanced age.[3] It is the third most

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Submitted Date: September 30, 2021 **Accepted Date:** November 08, 2021 **Available Online Date:** December 29, 2021

common cause of hospital-acquired acute renal injury and represents about 11% of the cases.[4] Although CI-AKI has a transient nature, it is associated with a longer hospital stay, increased morbidity, mortality, and a higher financial cost.[5] There is little agreement in the medical literature regarding the incidence of CI-AKI. The incidence of contrast nephropathy is lower than expected.[6, 7]

Treatment of CI-AKI is based on supportive therapy, and specific treatment options are lacking. Thus, identifying high-risk patients and prevention strategies are a cornerstone of CI-AKI management.

Underlying mechanisms of CI-AKI are not completely described. The best data related to the nephrotoxicity of contrast media come from animal models. Studies show evidence of acute tubular necrosis (ATN), but the mechanism by which ATN occurs is not well understood.[8] Multiple factors such as the direct cytotoxic effect of the contrast agent, intrarenal vasoconstriction, renal medullary hypoxia, ischemic damage and oxidative stress have been implicated in the pathogenesis of CI-AKI.[9] Besides, the activation of the inflammatory process has been proposed as a contributing factor to CI-AKI development.[10, 11] Compared with other types of ATN, CI-AKI is usually characterized by relatively rapid recovery of kidney function. One likely possibility is that the degree of tubular necrosis is much less severe than seen in other settings. It is also possible that the decline in GFR is due to functional changes in tubule epithelial cells rather than necrosis. Even if other causes of AKI are identified, it is still possible that iodinated contrast exposure may have contributed to kidney injury.

A growing number of studies have demonstrated a close association between CI-AKI and circulating immune-inflammatory cells, such as platelets, neutrophils, and lymphocytes. In addition, inflammatory indices calculated by the counts of these cells, such as NLR and PLR, have been reported as useful prognostic markers predicting CI-AKI.[12,13]

SII is a novel marker based on blood peripheral platelet, neutrophil, and lymphocyte counts.[14] This integrated indicator can comprehensively reflect the balance of host immune and inflammatory status. In previous studies, SII has been shown as a reliable prognostic indicator in malignancy patients.[15] It has also been noted that SII may be associated with adverse outcomes in coronary artery disease and heart failure patients.[16, 17]

The medical use of predictive analysis is increasing day by day. We tried to find a predictive marker for the risk of acute kidney injury after the procedure in clinical practice. This study aims to evaluate the predictive value of CI-AKI after an emergency percutaneous coronary intervention.

Methods

Study Design and Study Population

A total of 232 consecutive patients who underwent PCI diagnosed with coronary artery disease (CAD) patients between June 2019 and April 2020 were retrospectively enrolled in the study. The diagnosis of CAD was made with positive results on a stress test, ischemic changes in ECG recordings accompanying a history of angina, or myocardial infarction (MI) attack.

Patients with acute kidney disease before the procedure, hypovolemic, dehydrated and congestive heart failure patients, state after kidney transplantation, hemodialysis patients, those known to have severe liver disease, active hepatitis B or C, Covid-19 PCR-test positive, mental disease, chronic inflammatory disease, malignancy and active infection disease, patients with a history of exposure to the nonsteroidal anti-inflammatory drug within 30 days, nephrotoxic antibiotics, and contrast agent within the last 30 days, patients who have recently undergone major surgery or trauma, patients using post-procedure angiotensin-converting enzyme inhibitors, angiotensin receptor blockers and diuretics were excluded study. Overall, a number of 42 patients were excluded from the study. Thirty-one 31 patients were excluded due to at least one exclusion criteria and 11 patients due to missing data.

Finally, 190 patients were included in the study. Two groups were assigned according to CI-AKI development after contrast agent administration. CI-AKI group included 33 patients, and the non-CI-AKI group included 157 patients.

Demographic and Clinical Data

A clinical history of risk factors, such as age, sex, hypertension, diabetes mellitus, chronic kidney disease, was determined from the hospital's medical database. Baseline laboratory findings were collected from venous blood samples obtained at hospital admission. Neutrophil, lymphocyte, and platelet counts were measured as part of the automated complete blood count. The SCr level was measured at admission and daily post-procedure until the patient was discharged.

All cases received hydration (0.9% normal saline at 0.5-1.0 mL/kg/hr) for up to 12 hours after coronary angiography, depending on the patient's volume status. N-acetylcysteine (>1200 mg/day) was administered from hospitalization until one day after coronary angiography. The contrast agent dose was adjusted according to patient weight and baseline renal function. The low-osmolar agent iohexol was used during the procedure. CI-AKI was defined as a 25% increase of 0.5 mg/dL absolute increase in serum cre-

atinine level above baseline within 48-72 hours of contrast exposure, without an alternative explanation.

SII is calculated with a formula of total platelet count (per mm^3) x total neutrophil count (per mm^3) / total lymphocyte count (per mm^3). The estimated glomerular filtration rate (e-GFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[18]

The study was approved by the hospital's local Ethics Committee (IRB approval number: 2020.4/03-308)

Statistical Analysis

The Statistical Package for Social Sciences Software (SPSS 17.0 Chicago, Illinois) was used to compare demographic and clinical variables. The Kolmogorov-Smirnov test was used to test the normality of distribution. Quantitative variables with a normal distribution were shown as mean (standard deviation), non-normal distributed variables were shown as median (minimum-maximum). Categorical variables were expressed as number and percentage of values. Student's t test was applied to compare groups for normally distributed continuous variables, and the Mann-Whitney U test was used in cases where the distribution was not normal. X^2 test or Fisher exact test was used for categorical variables. Receiver operating characteristic (ROC) curves were used to determine the optimum cutoff values for SII, NLR, and CRP to predict CI-AKI in clinical decision making. To assess AUC differences between classification models, we used DeLong's test for ROC curves. Binary logistic regression analysis was used to identify independent predictors of CI-AKI. P<0.05 level was considered statistically significant.

Results

In our study, we enrolled a number of 190 patients to explore the relationship between SII and CI-AKI's development. The median age was 64 (35-86) years, and males accounted for 73.2% (139/190). Diabetes mellitus was detected in 36.3% (69/190) of patients. Baseline demographic and clinical characteristics of the patients categorized according to CI-AKI development are given in Table 1. The incidence of CI-AKI was 17.3% (33/190). The patients in the CI-AKI group were significantly older, with a higher incidence of diabetes mellitus when compared with the non-CI-AKI group (p=0.001). CI-AKI developed patients had higher serum creatinine levels, C-reactive protein levels, NLR, BMI, and SII levels. Whereas, in the group without CI-

Table 1. Baseline characteristics of study population with and without CI-AKI

	CI-AKI (-) (n=157)	CI-AKI (+) (n=33)	p
Age (years)	63 (35-86)	70 (44-86)	0.002**
Gender (Female)	38 (24.2%)	13 (39.4%)	0.073
DM	49 (31.2%)	20 (60.6%)	0.001*
BMI (kg/m^2)	27.45 (18.36-42.22)	31.25 (26.12-36.32)	0.001**
Ejection Fraction (%)	55 (35-65)	45 (30-50)	<0.001**
e-GFR ($mL/min/1.73m^2$)	90.13 (34.45-107.8)	81.71 (47.22-110.2)	0.595
Pre-procedure creatinin (mg/dL)	0.84 (0.46-1.55)	1.05 (0.59-1.55)	0.001**
Troponin (ng/mL)	0.586 (0.02-20.00)	0.655 (0.02-22.00)	0.136
Neutrophil (K/μL) × 10^3	5.45 (2.20-6.85)	5.86 (2.54-6.82)	0.116
Lymphocyte (K/μL) × 10^3	1.83 (1.45-2.02)	2.03 (1.54-2.36)	0.001**
Monocyte (K/μL) × 10^3	0.52 (0.48-0.54)	0.53 (0.47-0.55)	0.386
Platelet (K/μL) × 10^3	236.8 (128.6-257.8)	271.3 (125.8-284.5)	0.001**
Albumin (g/L)	3.6 (2.9-4.4)	3.3 (2.7-3.8)	<0.001**
Hemoglobin (g/dL)	13.7 (9.4-17.7)	13.2 (8.1-16.3)	0.185
HDL-cholesterol (mg/dL)	40 (27-64)	33 (28-47)	0.004**
LDL-cholesterol (mg/dL)	121 (38-224)	118 (81-163)	0.418
Triglycerides (mg/dL)	119 (50-474)	133 (71-289)	0.349
Contrast media volüme (mL)	120 (20-320)	140 (60-310)	0.071
NLR	2.6 (0.125-13.8)	4.44 (1.142-6.6)	<0.001**
PLR	96.53 (5.16-604)	98.8 (53.21-266.3)	0.137
SII	549 (207.5-4167.6)	1096 (170.3-1758)	<0.001**

Bold values represent statistical signifcance with p<0.05; *Chi square test; **Mann Whitney U test; CI-AKI contrast-induced acute kidney injury; DM: diabetes mellitus; BMI: body mass index; e-GFR: estimated glomerular filtration rate; CRP: c-reactive protein; HDL: high-density lipoprotein; LDL: low-density lipoprotein; NLR: neutrophil to lymphocyte ratio; PLR: platelet to lymphocyte ratio; SII: systemic immune-inflammation index.

AKI, GFR, serum albumin levels, and ejection fraction were higher. Even though the amount of contrast media used in patients was higher in the CI-AKI group, the relationship was not statistically significant (p=0.071).

Table 2 shows in univariable analysis model for determining risk factors of CI-AKI, SII (p=0.001, odds ratio [OR]=1.001[95%CI:[1.000-1.002]), serum albumin level (p<0.001, [OR]=0.700[0.605-0.808]), age (p=0.005, [OR]=1.054[1.016-1.093]), diabetes mellitus (p=0.002, [OR]=3.391[1.561-7.365]), body mass index (p=0.004, [OR]=1.134[1.040-1.237]), and ejection fraction (p<0.001, [OR]=0.872[0.827-0.918]) were associated with the development of CI-AKI.

Multivariable logistic regression analysis shown that higher BMI (p=0.001, [OR]=1.267[1.098-1.463]), low serum albumin level (p<0.001, [OR]=0.465[0.313-0.690]) lower ejection fraction (p=0.001, [OR]=0.794[0.695-0.908]), diabetes mellitus (p=0.006, [OR]= 10.330[1.967-54.25]), and higher SII (p=0.001, [OR]=1.002[1001-1004]) were independent risk factors for CI-AKI (Table 2).

The ROC curves of inflammatory factors to predict CI-AKI are shown in Figures 1-3. The area under curve (AUC) for SII was 0.825 (p<0.001, 95% CI: [0.752-0.898]), the AUC for NLR was 0.817 (p<0.001, 95% CI: [0.744-0.889]), the AUC for PLR was 0.583 (p=0.137, 95% CI: [0.454-0.711]), and the AUC for CRP was 0.694 (p<0.001, 95% CI: [0.591-0.798]). In figure 2, the AUCs between SII and NLR were compared with DeLong Test. No statistically significant superiority of SII against NLR was found (p=0.287). The optimum cut-off level of SII was 769.3 (sensitivity=87.9% and specificity=74.5%) for predicting CI-AKI.

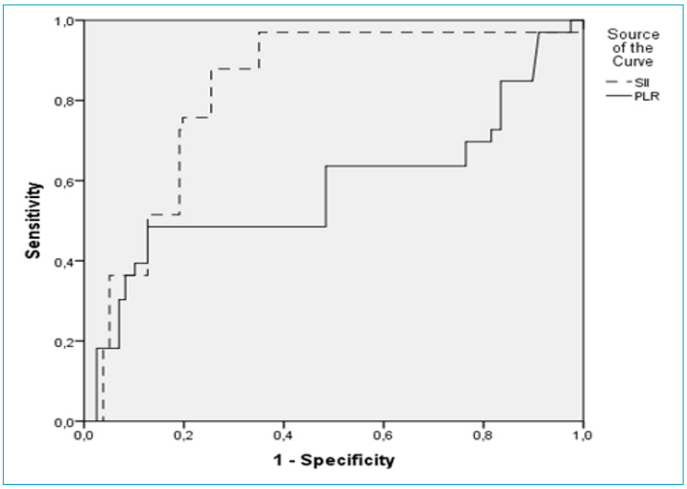

Figure 1. ROC curve for platelet to lymphocyte ratio and systemic immune-inflammation index for predicting contrast induced-acute kidney injury.

Discussion

In this study, we investigated the relationship between SII and the development of contrast nephropathy. The results of our study showed that increasing SII could predict the development of CI-AKI. Also, increased BMI decreased serum albumin level, decreased ejection fraction, and the presence of diabetes mellitus was associated with an increased risk of CI-AKI.

SII, a novel immune-inflammatory index, has been firstly proposed to predict the prognosis of patients after curative resection for hepatocellular carcinoma.[14] In subsequent studies, it has been reported that the increased SII score is

Table 2. Univariable and multivariable predictors of contrast induced-acute kidney injury in the study population

	Univariable		Multivariable	
	OR (95% CI of OR)	p	OR (95% CI of OR)	p
Age (years)	1.054 (1.016-1.093)	0.005	0.994 (0.936-1.056)	0.850
Gender (female)	2.036 (0.926-4.476)	0.077		
DM	3.391 (1.561-7.365)	0.002	10.33 (1.967-54.25)	0.006
BMI (kg/m2)	1.134 (1.040-1.237)	0.004	1.267 (1.098-1.463)	0.001
HDL cholesterol (mg/dL)	0.925 (0.877-0.976)	0.005		
Ejection Fraction (%)	0.872 (0.827-0.918)	<0.001	0.794 (0.695-0.908)	0.001
e-GFR (mL/min/1.73m^2)	0.987 (0.967-1.008)	0.987		
CRP (mg/L)	1.013 (0.964-1.066)	0.604		
Albumin (g/L)	0.700 (0.605-0.808)	<0.001	0.465 (0.313-0.690)	<0.001
Pre-procedure creatinin (mg/dL)	20.960 (3.091-142.1)		<0.002	
Contrast media volüme (mL)	1.004 (0.999-1.009)	0.146		
NLR	1.456 (0.935-2.432)	0.115		
SII	1.001 (1.000-1.002)	0.001	1.002 (1.001-1.004)	0.001

Bold values represent statistical signifcance with p<0.05; DM: diabetes mellitus; BMI: body mass index; e-GFR: estimated glomerular filtration rate; CRP: c-reactive protein; SII: systemic immune-inflammation index.

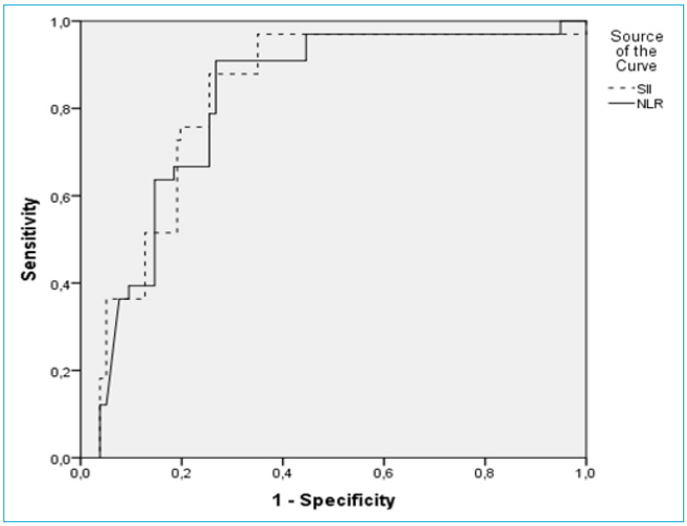

Figure 2. ROC curve for neutrophil to lymphocyte ratio and systemic immune-inflammation index for predicting contrast induced-acute kidney injury.

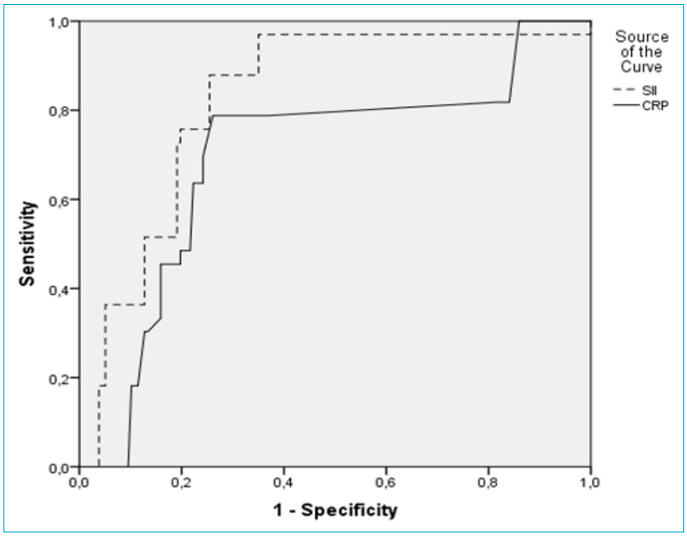

Figure 3. ROC curve for C-reactive protein and systemic immune-inflammation index for predicting contrast induced-acute kidney injury.

associated with poor patient prognosis in various types of malignancies such as hepatocellular carcinoma, gastrointestinal tract cancers, urinary cancers, small cell, lung cancer, and acral melanoma.[15] Recently, SII was linked to poor outcomes in patients with coronary artery disease, heart failure patients, and elective off-pump coronary artery bypass grafting patients.[19, 20]

The relation between radiocontrast administration and AKI is highly confounded and usually unpredictable. CI-AKI is an important cause of hospital-acquired acute kidney injury. It can be defined as acute deterioration in kidney functions following contrast media administration. Decreased renal medulla's perfusion due to vasoconstriction and direct toxic effects of contrast media on renal tubular cells are accused as the main pathophysiological factors in CI-AKI's development. In recent years, there has been growing data regarding the relationship between inflammation and CI-AKI's development. In a prospective, single-center study, 343 patients were enrolled to investigate the value of serum high-sensitive CRP and procalcitonin level to predict AKI development. Pre-procedural high-sensitive CRP and pro-calcitonin levels were predictors of CI-AKI development.[21] Also, inflammatory peripheral cells (neutrophils, lymphocytes, and platelets) have been linked to the development of CI-AKI. Besides, inflammatory indexes derived from these cells, such as NLR and PLR, have been explored as simple and useful predictors of CI-AKI development.[12, 13] However, the mechanism by which inflammation contributes to CI-AKI development is not fully understood. Multiphoton intravital microscopy studies have been shown that uptake of contrast media by perivascular resident renal phagocytes drives nod-like receptor pyrin containing 3 (Nlrp3) inflammasome activation and IL-1–dependent leukocyte recruitment, respectively.[11] Besides, contrast media administration has been linked to pyroptosis and epithelial cell lysis following inflammatory caspase activation.[10] As a result, extensive tubular epithelial cell lysis, disruption of tubules and epithelial cells, and the release of proinflammatory cytokines develop, and these changes drive an increase in serum urea and creatinine levels, which are all hallmarks of AKI.

SII can be calculated with a formula of "platelet count x neutrophil count/lymphocyte count" as an extension of NLR and PLR. Both NLR and PLR are pointed out as strong predictors of CI-AKI in prior studies. A total number of 1432 patients were enrolled in a retrospective study to evaluate the association between NLR and CI-AKI in patients with ST-segment elevation myocardial infarction. NLR and lesion length were independent predictors of CI-AKI development.[13] In addition, Acikgoz and colleagues had shown the role of PLR to predict the CI-AKI in a study, including 3352 patients undergone primary PCI for ST-elevation myocardial infarction.[12] However, to the best of our knowledge, the value of SII has not been previously evaluated in the prediction of CI-AKI development. In the current study, it has been shown that the SII score was more sensitive and specific to predict CI-AKI development compared with both NLR and PLR. This result can be attributed to the additive effect caused by the SII content, which involves both NLR and PLR components. To avoid multicollinearity, we did not include NLR and PLR into the regression models. However, ROC curve analyzes showed a higher AUC value of SII and NLR compared to PLR, leading us to the conclusion that SII and NLR better predicted CI-AKI.

Limitations

This study has some limitations. First, some patients were excluded from the study population due to insufficient data regarding the study's retrospective design. Second, the present study is a single-center study, and the result of this study should not be generalized to the whole population. Third, only data were available to calculate SII prior to the procedure, so there is no information about the trend of SII scores during and after the procedure.

Conclusion

SII is a novel marker that can be calculated easily from a complete blood count test. It can be an important indicator in determining the balance between systemic inflammation and immune status. Besides being a cheap and noninvasive test. SII levels can predict CI-AKI better than NLR and PLR. Consequently, increasing evidence suggests a close relationship between patients' inflammation status and the development of CI-AKI. Therefore, determining patients' inflammation status becomes crucial to establishing an adequate risk stratification. In this regard, inflammatory markers can be useful for predicting the risk of developing CI-AKI.

Disclosures

Ethics Committee Approval: The study was approved by the hospital's local ethics committee (IRB approval number: 2020.4/03-308) and followed the principles of the Declaration of Helsinki.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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

References

1. Rudnick M, Feldman H. Contrast-induced nephropathy: What are the true clinical consequences? Clin J Am Soc Nephrol 2008;3:263–72.
2. Kellum J, Lameire N, Aspelin P, Barsoum RS, Burdmann EA, Goldstein SL, et al. Kidney disease: Improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:1–138.
3. Bettmann AM. Contrast medium-induced nephropathy: Critical review of the existing clinical evidence. Nephrol Dial Transplant 2005;20:12–17.
4. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. Am J Kidney Dis 2002;39:930–6.
5. Mohammed NM, Rafie IM, Mahfouz A, Achkar K, Hajar R. Contrast-induced nephropathy. Heart Views 2013;14:106–16.
6. Wilhelm-Leen E, Montez-Rath ME, Cherto G. Estimating the risk of radiocontrast-associated nephropathy. J Am Soc Nephrol 2017;28:653–9.
7. McDonald RJ, McDonald JS, Bida JP, Carter RE, Fleming CJ, Misra S. Intravenous contrast material–induced nephropathy: Causal or coincident phenomenon? Radiology 2013;267:106–18.
8. Detrenis S, Meschi M, Musini S, Savazzi G. Lights and shadows on the pathogenesis of contrast-induced nephropathy: State of the art. Nephrol Dial Transplant 2005;20:1542–50.
9. McCullough PA, Adam A, Becker CR, Davidson C, Lameire N, Stacul F, et al. Epidemiology and prognostic implications of contrast-induced nephropathy. Am J Cardiol 2006;98:5–13.
10. Zhang Z, Shao X, Jiang N, Mou S, Gu L, Li S, et al. Caspase-11-mediated tubular epithelial pyroptosis underlies contrast-induced acute kidney injury. Cell Death Dis 2018;9:983.
11. Lau A, Chung H, Komada T, Platnich JM, Sandall CF, Choudhury SR, et al. Renal immune surveillance and dipeptidase-1 contribute to contrast-induced acute kidney injury. J Clin Invest 2018;128:2894–913.
12. Acikgoz E, Acikgoz S, Cicek G. Platelet to lymphocyte ratio is associated with contrast induced nephropathy after primary percutaneous coronary intervention for ST-elevation myocardial infarction section. Ann Med Res 2019;26:913–7.
13. Gulcu O. The association between neutrophil to lymphocyte ratio and contrast ınduced nephropathy in patients with ST segment elevation myocardial infarction. Ann Med Res 2019;26:1184–8.
14. 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.
15. 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.
16. 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.
17. Zhang Z, Navarese EP, Zheng B, Meng Q, Liu N, Ge H, et al. Analytics with artificial intelligence to advance the treatment of acute respiratory distress syndrome. J Evid Based Med 2020;13:301–12.
18. Levey AS, Stevens LA, Schmid CH, Zhang Y, Castro AF 3rd, Feldman HI, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med 2009;150:604–12.
19. Yang Y, Wu C, Hsu P, Chen S, Huang S, Chan WL, et al. Systemic immune-inflammation index (SII) predicted clinical outcome in patients with coronary artery disease. Eur J Clin Invest 2020;50:13230.
20. Dey S, Kashav R, Kohli JK, Magoon R, ItiShri WA, Grover V. 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.
21. Gu G, Yuan X, Zhou Y, Liu D, Cui W. Elevated high-sensitivity C-reactive protein combined with procalcitonin predicts high risk of contrast-induced nephropathy after percutaneous coronary intervention. BMC Cardiovasc Disord 2019;19:152.