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ORIGINAL RESEARCH

Correlation Analysis of the New Inflammatory Composite Index with the Degree of Coronary Artery Lesions in Elderly Patients

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Background: Systemic inflammation plays a key role in the development and progression of coronary artery disease (CAD). However, the clinical relevance of hematologic inflammatory indices in elderly patients has not been fully clarified.

Objective: To investigate the association of five hematologic inflammatory indices—neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI)—with the presence and severity of CAD in an elderly population.

Methods: This retrospective single-center study included 582 consecutive patients aged ≥60 years who underwent coronary angiography between 2021 and 2023. Based on the extent of coronary artery involvement, patients were categorized into four groups: normal (n = 131), single-vessel disease (n = 133), two-vessel disease (n = 136), and three-vessel disease (n = 182). Inflammatory indices were calculated from routine blood counts. Group differences were assessed using the Kruskal–Wallis test. Multivariable logistic regression analysis was performed after adjustment for age, sex, body mass index, hypertension, and diabetes. Discriminatory performance was evaluated using receiver operating characteristic (ROC) curve analysis with comparisons conducted by the DeLong test.

Results: All five inflammatory indices were significantly associated with increasing angiographic severity of CAD (all $P < 0.05$). Among them, SIRI demonstrated the strongest independent association (OR = 4.91, 95% CI: 2.11–11.44) and the highest area under the ROC curve (AUC = 0.766), followed by SII (AUC = 0.749). The addition of inflammatory indices to conventional risk factors modestly improved model discrimination.

Conclusion: This study demonstrates that SIRI and SII are valuable inflammatory biomarkers for evaluating the severity of coronary artery disease in elderly patients, highlighting the role of systemic inflammation in the progression of age-related atherosclerosis. As the first systematic comparison of five hematologic composite indices in a geriatric cohort, our findings suggest that SII and SIRI—both cost-effective and readily available parameters—are closely associated with CAD severity, though prospective studies are needed to establish their clinical value.

Keywords: elderly patients, coronary artery disease, systemic inflammation response index, systemic immune-inflammation index, hematologic inflammatory indices

Introduction

The global population is aging rapidly, resulting in an increasing prevalence of coronary artery disease (CAD) among older adults.[1,2] CAD is a leading cause of morbidity and mortality in this population,[3] often accompanied by multiple comorbidities, endothelial dysfunction, and chronic low-grade inflammation. Traditional risk factors—including

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hypertension, diabetes, dyslipidemia, obesity, smoking, and genetic predisposition—remain important contributors,[4] yet mounting evidence underscores the critical role of systemic inflammation in atherosclerotic progression and plaque instability. Inflammatory biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and the neutrophil-to-lymphocyte ratio (NLR) have been extensively studied in relation to CAD risk and prognosis.[5] More recently, composite indices derived from peripheral blood counts—particularly the systemic immune-inflammation index (SII) and the systemic inflammation response index (SIRI)—have gained attention due to their ability to integrate multiple immune cell components and their routine clinical availability.[6] Emerging data suggest that these indices are associated with coronary atherosclerotic burden and adverse outcomes,[7–14] with SIRI showing especially strong correlations in preliminary analyses. Most existing studies focus on mixed-age cohorts, with limited evidence in elderly populations. Furthermore, prior work has generally examined inflammatory markers individually rather than comparatively. To date, no study has systematically assessed five indices—NLR, platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), SII, and SIRI—in a geriatric cohort to determine their relative association with angiographic CAD severity. Considering the unique immunosenescence and heightened inflammatory signaling in aging, findings from younger or heterogeneous populations may not generalize to elderly patients. Therefore, this study aimed to compare these five inflammatory indices in elderly individuals undergoing coronary angiography and to test whether composite indices (particularly SIRI) outperform single-cell ratios in reflecting coronary lesion severity. The results were further evaluated using ROC analysis to quantify their discriminative performance.

Subjects and Methods

Research Subjects

This single-center retrospective study included 3158 consecutive patients who underwent coronary angiography for suspected CAD at our institution between January 2021 and December 2023. Exclusion criteria were: acute or chronic infections; severe hepatic or renal dysfunction; hematologic or hemorrhagic disorders; malignancies; autoimmune diseases; abnormal thyroid function; recent anticoagulant or thrombolytic therapy; other cardiac conditions such as rheumatic heart disease, cardiomyopathy, or myocarditis; and prior percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG).

After applying these criteria, 2576 patients were excluded. The primary reason for exclusion was age <60 years, followed by acute infections, previous PCI or CABG, malignancies, or incomplete data. Ultimately, 582 elderly patients (aged ≥60 years) met all eligibility criteria and were included in the final analysis. Written informed consent was obtained from all participants, and the study protocol was approved by the Institutional Ethics Committee of our hospital (approval number: K-2024-206-K01).

Based on coronary angiography, patients were classified into four groups. The non-CAD group (n = 131) included individuals with <50% luminal stenosis in all major coronary vessels. The CAD group (n = 451) comprised patients with ≥50% stenosis in at least one major coronary artery and was further stratified by the number of affected vessels: single-vessel (n = 133), double-vessel (n = 136), and triple-vessel disease (n = 182).

Methods

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Suzhou Hospital Affiliated to Nanjing Medical University (Approval No. K-2024-206-K01). Written informed consent was obtained from all participants prior to enrollment.

Baseline clinical data were collected for all patients, including demographic characteristics (age, sex), medical history, and comorbidities. Documented conditions included hypertension, type 2 diabetes, long-term smoking and alcohol use, and other underlying diseases. Fasting venous blood samples were collected in the early morning after admission. Laboratory assessments included complete blood counts, lipoprotein(a) [LP(a)], total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), fasting plasma glucose (FPG), fibrinogen (Fg), and creatinine (Cr).

Coronary angiography was performed by experienced interventional cardiologists using the Judkins technique. Multi-angle imaging was acquired via radial or femoral artery access. All procedures adhered to the American College of Cardiology/American Heart Association (ACC/AHA) guidelines for coronary angiography. Examinations were conducted using a PHILIPS V5000 angiography system with iopromide 370 as the contrast medium.

Diagnostic Criteria

The severity of coronary artery lesions was determined by angiography and classified as single-vessel, double-vessel, or triple-vessel disease according to involvement of the left main (LM), left anterior descending (LAD), left circumflex (LCX), and right coronary artery (RCA). Branch vessel lesions (eg, diagonal or marginal branches) were assigned to the corresponding main coronary arteries. In cases involving the left main coronary artery, the disease was classified as double-vessel disease regardless of concomitant LAD or LCX involvement. The presence of an additional lesion in the RCA upgraded the classification to triple-vessel disease.

Hypertension was defined based on the JNC VII criteria[15] as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg measured on at least two separate occasions, or a previously established diagnosis. Type 2 diabetes was diagnosed according to the American Diabetes Association criteria[16] as fasting (≥8 hours) venous plasma glucose ≥7.0 mmol/L, 2-hour postprandial plasma glucose ≥11.1 mmol/L, or a prior confirmed diagnosis.

Smoking status was classified in accordance with WHO guidelines: individuals who smoked at least one cigarette per day for at least one year, or those who had quit smoking within the preceding six months, were considered current smokers.

Inflammatory Indices

Peripheral blood cell counts were used to calculate five inflammatory indices according to the following formulas:

Neutrophil-to-lymphocyte ratio (NLR) = neutrophil count / lymphocyte count

Platelet-to-lymphocyte ratio (PLR) = platelet count / lymphocyte count

Monocyte-to-lymphocyte ratio (MLR) = monocyte count / lymphocyte count

Systemic immune-inflammation index (SII) = platelet count × neutrophil count / lymphocyte count

Systemic inflammation response index (SIRI) = neutrophil count × monocyte count / lymphocyte count

Compared with simple two-parameter ratios, SII and SIRI incorporate three immune cell lineages, reflecting interactions between innate immune cells (neutrophils and monocytes), adaptive immunity (lymphocytes), and platelet-related thrombotic activity. These composite indices may provide a more comprehensive representation of systemic inflammatory status.

Statistical Methods

All statistical analyses were performed using SPSS version 27.0. Continuous variables are expressed as mean ± SD, and normality was assessed with appropriate tests. Two-group comparisons were performed using the independent samples *t*-test for normally distributed variables or the Mann–Whitney *U*-test for non-normally distributed variables. Multiple-group comparisons were conducted using one-way ANOVA (for homogeneous variances) or the Kruskal–Wallis test (when variances were unequal), followed by post-hoc pairwise comparisons with Bonferroni correction.

Spearman's rank correlation was used to evaluate associations between inflammatory indices and CAD severity. Multivariable logistic regression (enter method) was applied to identify factors independently associated with CAD; variables with $P < 0.10$ in univariable analysis or deemed clinically relevant were included. Results are reported as odds ratios (OR) with 95% confidence intervals (CI).

Receiver operating characteristic (ROC) curves were generated to assess the discriminative ability of each inflammatory index. Areas under the curve (AUC) were calculated, and pairwise comparisons were performed using the DeLong test.

Two-sided $P < 0.05$ was considered statistically significant, except for Bonferroni-adjusted pairwise comparisons ($P < 0.0083$).

Results

Baseline Characteristics

A total of 582 elderly patients (289 men and 293 women; mean age 66.70 ± 5.75 years) were included. According to coronary angiography, 451 patients had CAD and 131 were non-CAD. Compared with non-CAD patients, those with CAD were more often male and had higher prevalences of hypertension, diabetes, and smoking (all $P < 0.01$). Laboratory analyses showed significantly higher levels of fibrinogen, lipoprotein(a), creatinine, fasting plasma glucose, triglycerides, total cholesterol, LDL-C, neutrophil count, monocyte count, platelet count, and all five inflammatory indices (NLR, PLR, MLR, SII, and SIRI) in the CAD group (all $P < 0.01$). HDL-C and lymphocyte count were lower (both $P < 0.01$), while age did not differ significantly between groups ($P > 0.05$) (Table 1).

Inflammatory Indices According to Vessel Involvement

Among CAD patients, 133 had single-vessel, 136 had double-vessel, and 182 had triple-vessel disease. Greater numbers of affected vessels were associated with older age and higher prevalences of hypertension and diabetes. Fibrinogen, creatinine, lipoprotein(a), fasting plasma glucose, triglycerides, LDL-C, neutrophil count, and monocyte count increased progressively with vessel involvement (all P for trend < 0.05). All five inflammatory indices showed a significant stepwise increase from single- to triple-vessel disease (all P for trend < 0.001) (Table 2).

Table 1 Comparison of Clinical Characteristics Between CAD and Non-CAD Groups

Variable	CAD Group (n=451)	Non-CAD Group (n=131)	*P* Value
Age (years)	67.08±5.88	65.37±5.03	0.068
Sex (male/female)	250/201	39/92	<0.001
Hypertension, n (%)	238(52.8)	68(51.9)	<0.001
Diabetes, n (%)	97(21.5)	23(17.6)	<0.001
Smoking, n (%)	219(48.6)	25(19.1)	<0.001
Fg (g/L)	3.05±0.76	2.73±0.51	<0.001
LP(a) (g/L)	0.34±0.30	0.24±0.15	<0.001
Cr (μmol/L)	70.69±16.93	66.04±12.90	0.008
FPG (mmol/L)	6.85±2.89	5.74±1.24	<0.001
TC (mmol/L)	4.68±1.15	2.73±0.51	<0.001
TG (mmol/L)	1.72±0.97	1.52±0.81	0.025
LDL-C (mmol/L)	2.85±0.88	2.65±0.58	0.006
HDL-C (mmol/L)	1.15±0.27	1.21±0.27	0.017
NE ($\times 10^9$/L)	6.07±2.95	4.35±1.40	<0.001
LY ($\times 10^9$/L)	1.87±0.82	1.97±0.66	0.004
MO ($\times 10^9$/L)	0.43±0.18	0.37±0.14	<0.001
PLT ($\times 10^9$/L)	238.64±60.81	219.93±49.07	0.003
NLR	3.51±2.44	2.52±1.35	<0.001
PLR	144.82±62.65	123.01±40.71	<0.001
MLR	0.26±0.12	0.21±0.11	<0.001
SII	657.21±405.73	481.02±189.62	<0.001
SIRI	1.51±1.25	0.96±0.69	<0.001

Notes: Data are presented as mean ± SD or n (%). *P* values were calculated using independent *t*-test, Mann–Whitney *U*-test, or chi-square test as appropriate.

Abbreviations: CAD, coronary artery disease; Fg, fibrinogen; Cr, serum creatinine; FPG, fasting plasma glucose; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; NE, neutrophil count; LY, lymphocyte count; MO, monocyte count; PLT, platelet count; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio; SII, systemic immune-inflammation index (platelet × neutrophil / lymphocyte); SIRI, systemic inflammation response index (neutrophil × monocyte / lymphocyte).

Table 2 Inflammatory Indices by Coronary Lesion Count in Elderly CAD Patients

Variable	SVD (n=133)	DVD (n=136)	TVD (n=182)	*P* value
Age (years)	65.71±5.26	66.82±5.66	67.91±7.86	<0.001
Sex (male/female)	67/66	79/57	105/77	0.358
Hypertension, n (%)	66(49.6)	70(51.4)	102(56.0)	0.034
Diabetes, n (%)	20(15.0)	28(20.6)	47(25.8)	0.001
Smoking, n (%)	60(45.1)	71(52.2)	88(48.4)	0.590
Fg (g/L)	2.67±0.52	2.93±0.73	3.21±0.87	<0.001
LP(a) (g/L)	0.25±0.14	0.31±0.35	0.39±0.34	<0.001
Cr (μmol/L)	66.23±14.15	69.87±15.49	72.61±17.07	0.005
FPG (mmol/L)	6.23±2.56	6.80±2.35	7.35±3.29	<0.001
TC (mmol/L)	4.48±0.97	4.64±0.87	4.86±1.39	0.091
TG (mmol/L)	1.66±0.83	1.66±0.99	1.74±1.08	0.871
LDL-C (mmol/L)	2.66±0.75	2.83±0.66	3.01±1.07	0.006
HDL-C (mmol/L)	1.19±0.29	1.18±0.30	1.14±0.28	0.256
NE ($\times 10^9$/L)	5.17±2.54	5.79±2.42	6.41±2.68	<0.001
LY ($\times 10^9$/L)	1.92±0.87	1.85±0.67	1.79±0.70	0.431
MO ($\times 10^9$/L)	0.39±0.16	0.44±0.16	0.46±0.18	0.005
PLT ($\times 10^9$/L)	220.3±56.50	225.8±46.80	235.23±61.99	0.205
NLR	2.63±1.20	3.36±1.98	3.96±2.23	<0.001
PLR	129.81±43.73	148.94±59.78	162.52±59.18	<0.001
MLR	0.22±0.08	0.25±0.11	0.28±0.13	<0.001
SII	544.13±224.04	605.22±249.83	684.11±329.63	<0.001
SIRI	1.05±0.58	1.38±0.92	1.77±1.22	<0.001

Notes: Data are presented as mean ± SD or n (%). *P* values were calculated using one-way ANOVA, Kruskal–Wallis test, or chi-square test as appropriate.
Abbreviations: SVD, single-vessel disease; DVD, double-vessel disease; TVD, triple-vessel disease. See Table 1 for other abbreviations.

Correlation Analysis

Spearman's rank correlation revealed positive associations of NLR, PLR, MLR, SII, and SIRI with CAD severity (all $P < 0.001$), with SIRI exhibiting the highest correlation coefficient ($r = 0.52$) (Table 3).

Multivariable Logistic Regression Analysis

After adjustment for conventional cardiovascular risk factors, male sex, hypertension, diabetes, smoking, PLR, SII, and SIRI were independently associated with CAD. Among the inflammatory indices, SIRI showed the highest odds ratio (OR = 4.91, 95% CI: 2.11–11.44), followed by SII and PLR (Table 4).

Table 3 Inflammatory Indices and Coronary Lesion Severity in Elderly Patients

Inflammatory Index	Correlation Coefficient (r)	*P* value
NLR	0.42	<0.001
PLR	0.29	0.022
MLR	0.41	<0.001
SII	0.46	<0.001
SIRI	0.52	<0.001

Notes: Correlation coefficients were calculated using Spearman's rank correlation. Lesion severity was defined as the number of affected coronary vessels (1, 2, or 3).
Abbreviation: CAD, coronary artery disease.

Table 4 Logistic Regression Analysis of Factors Associated with CAD in Elderly Patients

Risk Factors	*β*	*OR*	95% CI	*P*
Age	−0.962	0.458	0.286–0.553	0.009
Hypertension	0.561	1.652	1.156–2.405	<0.001
Diabetes	0.296	1.428	1.322–1.563	<0.001
Smoking	1.155	3.174	2.307–4.366	<0.001
NLR	0.246	0.782	0.587–1.043	0.094
PLR	0.210	1.021	1.011–1.031	0.008
MLR	0.524	0.23	0.022–3.378	0.138
SII	0.668	1.868	1.478–2.361	0.007
SIRI	1.591	4.910	2.108–11.441	0.004

Notes: Variables were selected based on clinical relevance and significance in univariable analyses ($P < 0.10$). The enter method was used to retain all clinically important variables.

ROC Curve Analysis

Figure 1 shows ROC curves of NLR, PLR, and MLR for discriminating CAD. The AUCs were 0.718 (95% CI: 0.674–0.762) for NLR, 0.709 (95% CI: 0.663–0.756) for MLR, and 0.652 (95% CI: 0.603–0.702) for PLR. DeLong

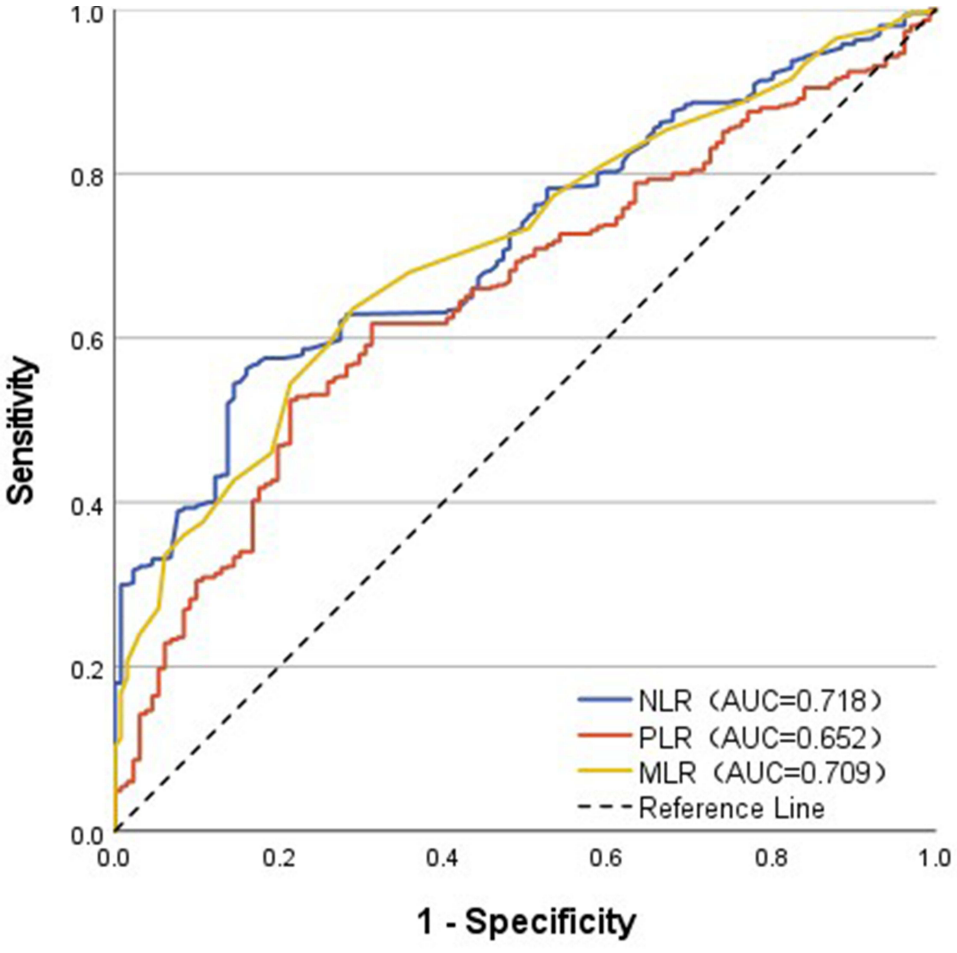

Figure 1 Receiver operating characteristic (ROC) curves of NLR, PLR, and MLR for discriminating coronary artery disease (CAD) in elderly patients. AUCs were 0.718 (95% CI: 0.674–0.762) for NLR, 0.709 (95% CI: 0.663–0.756) for MLR, and 0.652 (95% CI: 0.603–0.702) for PLR. DeLong test showed a higher AUC for NLR than PLR ($P = 0.010$), whereas NLR and MLR did not differ significantly ($P = 0.655$). MLR showed a marginally higher AUC than PLR ($P = 0.049$).

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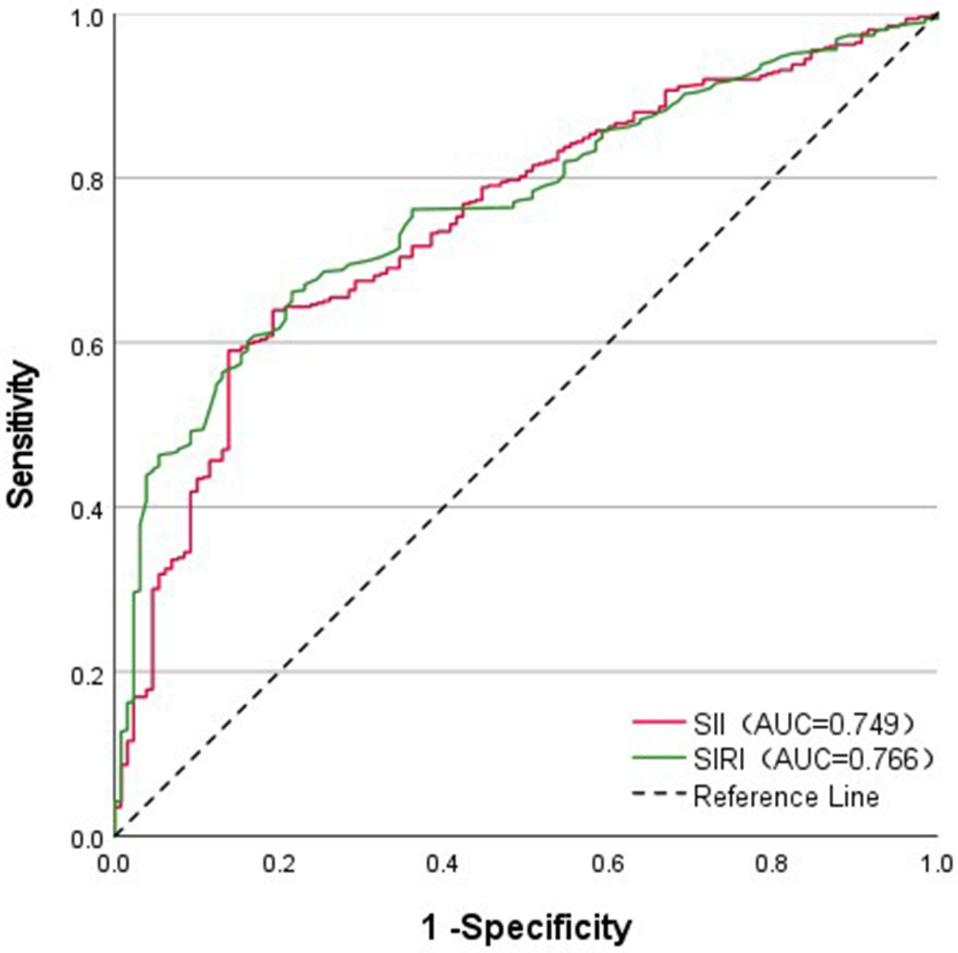

Figure 2 ROC curves of SII and SIRI for discriminating CAD in elderly patients. AUCs were 0.766 (95% CI: 0.724–0.807) for SIRI and 0.749 (95% CI: 0.704–0.794) for SII. SIRI demonstrated significantly higher AUCs than NLR ($P = 0.020$), PLR ($P < 0.001$), and MLR ($P = 0.005$), but did not differ from SII ($P = 0.514$). SII showed a higher AUC than PLR ($P = 0.001$) and was comparable to NLR ($P = 0.190$) and MLR ($P = 0.168$).

tests indicated that the AUC of NLR was higher than that of PLR ($P = 0.010$), whereas NLR and MLR did not differ significantly ($P = 0.655$). The AUC of MLR was higher than that of PLR ($P = 0.049$).

Figure 2 presents ROC curves for SII and SIRI. SIRI showed the highest AUC (0.766, 95% CI: 0.724–0.807), followed by SII (0.749, 95% CI: 0.704–0.794). DeLong comparisons indicated that SIRI had significantly higher AUCs than NLR ($P = 0.020$), PLR ($P < 0.001$), and MLR ($P = 0.005$), but did not differ from SII ($P = 0.514$). SII had a higher AUC than PLR ($P = 0.001$) but was comparable to NLR (P = 0.190) and MLR ($P = 0.168$). Overall, AUCs ranged from 0.652 to 0.766.

Discussion

To our knowledge, this study is the first to systematically compare the associations of five routine blood-derived inflammatory composite indices with the severity of coronary artery lesions in an elderly, disease-specific cohort. Our findings indicate that SIRI (AUC = 0.766) and SII (AUC = 0.749) exhibited better discriminative ability compared to traditional markers such as NLR, PLR, and MLR ($P < 0.01$). These results are consistent with those reported by Xu et al,[13] who found SIRI to be associated with in-stent restenosis in a mixed-age population. However, direct comparison should be interpreted with caution given the differences in study design (cross-sectional vs. cohort), outcome definition (lesion severity vs. restenosis), and population (elderly vs. mixed-age). Notably, the association of SIRI with CAD severity was more pronounced in our elderly cohort (OR = 4.91, 95% CI: 2.108–11.441) compared to that reported in

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a mixed-age population (OR = 1.194, 95% CI: 1.087–1.313),[12] suggesting a potential "downward shift" in the inflammation-sensitive threshold among elderly patients, likely linked to aging-associated immune remodeling. Supporting this, de Mol et al[17] demonstrated in an animal model that age-related pro-inflammatory immune changes accelerate atherosclerosis progression. However, the AUC values observed in this study ranged from 0.65 to 0.77, indicating modest discriminative ability. These findings should therefore be interpreted with caution, and the clinical utility of these indices requires further validation in prospective studies.

The core components of SII and SIRI include neutrophils, lymphocytes, monocytes, and platelets.[18] Neutrophils contribute defensively via mechanisms such as degranulation, phagocytosis, reactive oxygen species generation, and neutrophil extracellular trap (NET) formation.[19–21] Concurrently, neutrophils initiate sterile inflammatory responses, enhance interactions between vascular endothelial cells and platelets, and promote immunothrombosis formation, thus driving atherosclerotic progression.[22,23] A large cohort study linked elevated neutrophil counts to increased risks of various cardiovascular diseases, including heart failure (HR: 2.04, 95% CI: 1.82–2.29), peripheral arterial disease (HR: 1.95, 95% CI: 1.72–2.21), sudden coronary death (HR: 1.78, 95% CI: 1.51–2.10), abdominal aortic aneurysm (HR: 1.72, 95% CI: 1.34–2.21), and non-fatal myocardial infarction (HR: 1.58, 95% CI: 1.42–1.76).[24]

Monocytes play a critical role in inflammation and atherosclerosis pathogenesis.[25,26] Upon migration into the intima of injured vessels and differentiation into macrophages, they facilitate oxidized LDL uptake and release pro-inflammatory mediators, thereby advancing atherogenesis.[27,28] Additionally, monocytes impede macrophage migration and LDL oxidation, promote cholesterol accumulation in vessel walls, lower HDL levels, and consequently increase cardiovascular risk.[29–31]

Lymphocytes have a distinctive regulatory role in inflammation. B lymphocyte deficiency accelerates atherosclerosis,[32] while clinical studies associate lymphopenia with worse prognoses in heart failure and acute coronary syndrome patients,[33–35] reinforcing the biological relevance of SIRI as a comprehensive marker of systemic inflammation. In our study, elevated release of pro-inflammatory microparticles from neutrophils coincided with significantly reduced lymphocyte counts and function in the CAD group, indicating immune dysregulation. The SIRI index (NE × MO / LY) demonstrated high sensitivity to these immunological shifts. A 20-year cohort study involving 42,875 American adults reported significant associations of elevated SII and SIRI with increased cardiovascular and all-cause mortality risks. In individuals aged ≥60 years, those in the highest quartiles of SII and SIRI showed a 1.33-fold and 1.39-fold increase in cardiovascular and all-cause mortality risk, respectively, highlighting the promise of inflammation-targeted strategies in elderly populations.[36] Moreover, recent evidence indicates significantly higher SII and SIRI levels in CAD patients versus hypertensive controls without CAD, with these associations persisting after adjustment for confounders.[37] Additional studies have reported associations of SII with cardiovascular diseases in middle-aged and elderly cohorts.[38] Beyond cardiovascular disease, systemic inflammation plays a critical role in various clinical settings. Uluc[39] recently reported that shock indices derived from routine vital signs are associated with mortality in septic patients, further supporting the potential value of easily obtainable inflammatory parameters in risk stratification.

Consistently, our findings revealed significantly elevated SII and SIRI levels in elderly CAD patients compared to non-CAD counterparts, with progressive increases corresponding to the number of affected coronary vessels. Spearman correlation confirmed positive associations between these indices and lesion severity. Multivariate logistic regression identified SII and SIRI as independent factors associated with CAD, with SIRI showing the strongest association (OR = 4.910), in agreement with recent reports.[12] ROC analysis further supported the discriminative ability of these composite indices (AUC > 0.74) compared to individual inflammatory markers, suggesting they may be relevant in risk assessment for elderly CAD patients.

While SII and SIRI were independently associated with CAD after adjusting for traditional risk factors, whether these indices offer incremental predictive value beyond established clinical factors remains to be determined. Formal assessment using metrics such as net reclassification improvement in prospective studies is needed to clarify their added clinical utility.

Limitations

Several limitations should be considered when interpreting our findings. The single-center retrospective design may have introduced selection bias. Moreover, most screened patients (2576 of 3158) were excluded because they were younger than 60 years; although this ensured a homogeneous elderly cohort, it may restrict the generalizability of the results to younger populations. The absence of longitudinal follow-up limited assessment of the long-term prognostic value of the inflammatory indices. In addition, residual confounding cannot be ruled out, as factors such as medication use (eg, statins, antiplatelet agents, anti-inflammatory drugs) and nutritional status were not fully accounted for. Furthermore, this study did not establish clinically applicable thresholds or provide absolute risk estimates for these inflammatory indices, which limits their direct use in clinical decision-making. Future prospective, multicenter studies with standardized methodologies are warranted to confirm these observations, derive optimal cut-off values, and further clarify their clinical significance.

Conclusion

In summary, this study demonstrates that SII and SIRI are associated with coronary lesion severity in elderly patients and exhibit good discriminative ability, with SIRI showing the strongest association. These hypothesis-generating findings suggest that SIRI warrants further investigation as a potential risk marker, though prospective validation is required before clinical application.

Data Sharing Statement

The datasets analyzed during the current study are available from the corresponding author (Dr. Xiang Lu and Dr. Liya Mo) on reasonable request.

Ethical Approval

This study was approved by the Ethics Committee of Suzhou Hospital Affiliated to Nanjing Medical University (Approval No. K-2024-206-K01). The study was conducted in accordance with the Declaration of Helsinki.

Informed Consent

CAD patients and their families signed informed consent forms.

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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 no conflicts of interest in this work.

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