ORIGINAL ARTICLE

INFLAMMATION INDICES PREDICT STENT THROMBOSIS IN GERIATRIC PATIENTS WITH ST-ELEVATION MYOCARDIAL INFARCTION

Turkish Journal of Geriatrics
2026; 29(3):285–295

DOI: 10.29400/tjgeri.2026.500

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Received : Jul 26, 2026
Accepted: Aug 23, 2026

Cite this article as:
Kafes H, Tekin Tak B, Ergün AC et al. Inflammation Indices Predict Stent Thrombosis in Geriatric Patients with ST-elevation Myocardial Infarction. Turkish Journal of Geriatrics 2026; 29(3):285–295. doi: 10.29400/tjgeri.2026.500

Abstract

Introduction: The objective of this investigation was to determine whether the systemic immune-inflammation index (SII) and pan-immune-inflammation value (PIV), which serve as comprehensive indicators of systemic inflammatory pathways, possess the prognostic capacity to identify future stent thrombosis (ST) in geriatric STEMI patients.

Materials and Method: We comprehensively screened a consecutive cohort of 1,683 elderly individuals (aged 75.3 ± 6.8 years; 64.6% male) who had been treated with primary percutaneous coronary intervention (PCI) for acute ST-elevation myocardial infarction (STEMI). Stratification was performed based on the clinical manifestation of angiographically confirmed ST during follow-up. Both SII and PIV scores were computed from standard complete blood counts obtained at the time of initial clinical presentation.

Results: Over an average follow-up duration of 30 ± 10 months, definite ST was documented in 104 individuals (6.2%). Patients experiencing ST exhibited distinct baseline profiles, characterized by a higher prevalence of tobacco use and hypertension, a greater frequency of early dual antiplatelet therapy (DAPT) cessation (<30 days), prolonged total stent lengths, and markedly elevated baseline high-sensitivity C-reactive protein (hs-CRP), SII, and PIV levels. In multivariable Cox proportional hazards modeling, both admission PIV and SII emerged as robust, standalone indicators of subsequent ST. Furthermore, correlation analyses demonstrated that hs-CRP levels fluctuated in tandem with both SII ($r=0.342$, $p<0.001$) and PIV ($r=0.268$, $p<0.001$) values.

Conclusion: In summary, these data demonstrate that baseline immuno-inflammatory indices serve as independent and highly reliable indicators of subsequent ST development within the high-risk geriatric cohort treated with primary PCI for STEMI.

Keywords: Aged; Biomarkers; Inflammation; Stent; Thrombosis; ST Elevation Myocardial Infarction.

INTRODUCTION

Chronological aging is closely mirrored by a rising incidence of cardiovascular conditions, which remain the most prominent cause of clinical complications and mortality among older adults (1). Elderly individuals constitute a rapidly expanding proportion of clinical presentations for acute coronary syndromes (ACS), most notably ST-elevation myocardial infarction (STEMI) (1). Emergency mechanical revascularization via primary percutaneous coronary intervention (PCI) is the most favored reperfusion modality for managing STEMI, a therapeutic priority that extends fully to the geriatric population (2). The clinical efficacy of primary PCI is frequently challenged by advancing age, with older patients demonstrating poorer procedural outcomes and an inherently higher susceptibility to intervention-related complications than younger individuals (3). Beyond their baseline vulnerability to adverse cardiac events, geriatric individuals consistently exhibit suboptimal outcomes following percutaneous coronary interventions (2). These include lower procedural success rates, reduced resolution of ST-segment changes, decreased myocardial blush grade, lower incidence of post-percutaneous coronary intervention (PCI) blood flow, and increased risk of complications such as stent thrombosis (ST) (4).

Stent thrombosis is a rare but dreaded complication that can arise after PCI and poses significant risks, with the potential for serious morbidity and mortality (5). The development of ST is driven by a multifaceted interplay of clinical variables, encompassing patient profiles, anatomical complexities, interventional strategies, and post-procedural management (5,6). This increased vulnerability is further driven by 'inflammaging,' a phenomenon characterized by chronic, low-grade systemic inflammation that develops during physiological aging. This age-related inflammatory shift not only promotes the progression of atherosclerosis but also predisposes aged individuals to an exaggerated prothrombotic state, potentially explaining the higher incidence of adverse mechanical events such as ST after percutaneous interventions. The specific pathophysiological mechanism underlying its occurrence remains unclear, with evidence suggesting that inflammation significantly contributes to the development of ST (6). The systemic immune-inflammation index (SII) and pan-immune-inflammation value (PIV) represent cost-effective and easily accessible biomarker combinations that have emerged as reliable proxies for mapping individual immune-thrombotic and inflammatory states (7,8). These leukocyte-derived markers combine information from both innate and adaptive immunity and avoid relying on the absolute levels of a single leukocyte subtype, which can be influenced by factors such as infection or dehydration (8). Increasing evidence from observational studies has demonstrated a significant relationship between these inflammatory indices and the presence and severity of several cardiovascular disorders (9,10). Previous studies have examined the relationship between inflammatory indices and primary and secondary outcomes in STEMI (11). However, no data are available on the importance of the novel immune-inflammatory index values in geriatric patients with acute STEMI. Accordingly, our primary objective was to investigate the predictive value of admission-derived PIV and SII values regarding the development of stent thrombosis complications in aged individuals sustaining a STEMI.

MATERIALS AND METHOD

Study population

The current single-center study employed a retrospective design to evaluate 1,683 consecutive older adults (aged ≥65 years) admitted with acute STEMI. Eligible participants were required to have reached our tertiary hospital within 12 hours of symptom onset and to have undergone primary PCI during the period spanning from June 2019 to December 2023. Patients with advanced kidney or liver dysfunction, prior fibrinolytic therapy, hemodynamically significant valvular pathologies,



previous coronary artery bypass grafting, cardiogenic shock, active infection, malignancy, or hematological/ inflammatory diseases (including autoimmune and connective tissue disorders), as well as those using corticosteroids or immunosuppressive agents, or possessing incomplete admission laboratory data, were excluded. In addition, patients with procedural complications or suboptimal outcomes—defined as coronary dissection or abrupt closure, residual stenosis >20%, post-procedural Thrombolysis in Myocardial Infarction (TIMI) flow <3, complex bifurcation lesions requiring advanced intervention, stent under-sizing, or residual thrombus—were also excluded. The protocol for this investigation adhered strictly to the ethical principles outlined in the Declaration of Helsinki and received formal approval from the institutional review board.

Definitions and study endpoints

In accordance with the Fourth Universal Definition of Myocardial Infarction, the diagnosis of STEMI required the presence of typical ischemic chest pain lasting over 30 minutes, combined with specific electrocardiographic criteria. These criteria included new ST-segment elevation measured at the J-point in at least two contiguous leads, with thresholds set at ≥2.0 mm in men and ≥1.5 mm in women for leads V2–V3, and/or ≥1.0 mm in all other contiguous leads (12).

Patients were classified as hypertensive if they exhibited a systolic blood pressure of ≥140 mm Hg or a diastolic blood pressure of ≥90 mm Hg during clinical assessment, or if they were currently receiving antihypertensive pharmacotherapy. Diabetes was clinically defined by either a fasting blood sugar measurement of ≥126 mg/dL or the current utilization of insulin therapy or oral hypoglycemic agents. The diagnosis of dyslipidemia was established based on the documentation of serum total cholesterol >200 mg/dL, LDL cholesterol >130 mg/dL, or triglyceride levels exceeding 150 mg/dL, or the current use of lipid-lowering medications. A positive family history of premature coronary artery disease (CAD) was established if a first-degree male relative was diagnosed with CAD before reaching 55 years of age, or a first-degree female relative before 65 years of age. Smoking status was defined by active tobacco consumption at the time of admission or a history of cessation within the preceding 12 months.

The primary clinical endpoint was the development of definite stent thrombosis, adjudicated according to the Academic Research Consortium (ARC) classifications, which necessitated angiographic or pathological documentation of a thrombus localized within or immediately adjacent to the scaffolded segment (13). Accordingly, the study population was subdivided into ST and no-ST groups. Definite ST was established when coronary angiography revealed a thrombus within the stent or within 5 mm of its proximal or distal borders, causing complete or incomplete vessel occlusion associated with signs of acute myocardial infarction.

Laboratory parameters

Venous blood samples were collected at the time of hospital admission (emergency department), prior to the administration of any heparin or primary PCI procedure, to avoid procedural inflammatory bias. Baseline venous blood samples were drawn from a prominent antecubital vein in all participants; these specimens were subsequently allocated into additive-free tubes for biochemical assays and pre-cooled EDTA tubes for hematological evaluations. Complete blood counts were performed using an automated hematology analyzer XE-1200 (Sysmex, Kobe, Japan). The baseline SII level was calculated using the following equation: neutrophil (N)×platelet (P)/ lymphocyte (L). The baseline PIV level was calculated using the following equation: neutrophil (N)×platelet (P) ×monocyte (M) / lymphocyte (L). Calculation of admission-derived SII was performed by multiplying the absolute counts of neutrophils (N) and platelets (P), followed by division by the lymphocyte count (L). The calculation of baseline PIV expanded upon this framework by including the absolute monocyte count (M) in the numerator, yielding the ratio: (N)×platelet (P)/ lymphocyte.

Echocardiography

To quantify the left ventricular ejection fraction (LVEF), transthoracic two-dimensional and Doppler echocardiography was executed for all patients in the intensive coronary care unit within 24 to 48 hours of hospitalization, utilizing commercially available diagnostic ultrasound platforms operated by skilled operators.

Coronary angiographic evaluation

The primary PCI procedures for the entire study cohort were performed in the high-volume setting of our tertiary referral cardiology center. All interventions were handled by expert operators utilizing the center's 24/7 emergent PCI infrastructure and strictly following active guidelines. Coronary angiography and interventions were conducted via radial or femoral access, and all patients received intravenous heparin and dual antiplatelet therapy (aspirin plus a P2Y12 inhibitor) upon admission. Procedural strategies, including stent selection and adjunctive therapies, were left to the operator's discretion. Both bare-metal and drug-eluting stents were used. We used a cobalt chromium bare metal stent (BMS) (Commander™, Alvimedica, and Tango™, Microport) and drug-eluting stents (DESs) with everolimus and paclitaxel (Xience V Everolimus Eluting Coronary Stent System, Abbott Vascular, and TAXUS Paclitaxel-Eluting Coronary Stent System, Boston Scientific Corporation). Primary PCI aimed to restore flow in the infarct-related artery, while non-culprit lesions were not treated unless cardiogenic shock was present. The mean door-to-balloon time was <30 minutes. Following the intervention, optimal guideline-directed medical management was initiated for all patients, which encompassed a minimum 12-month regimen of dual antiplatelet therapy. Angiographic data, including TIMI flow and residual stenosis, were independently assessed by two blinded interventional cardiologists. In cases of stent thrombosis, dual antiplatelet therapy use and duration were verified through medical records and follow-up contact.

Statistical analysis

Data analysis was carried out through the application of SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as either mean ± standard deviation (SD) or median with interquartile range (IQR) based on normality, whereas categorical data were reported as absolute counts and percentages. The Kolmogorov–Smirnov test was employed to determine whether the continuous variables followed a normal distribution. Inter-group comparisons of continuous metrics were performed using the Student's t-test or Mann–Whitney U test, as appropriate for the underlying distribution. Proportions and categorical frequencies were compared across groups utilizing the chi-square or Fisher's exact methods. To identify independent determinants of stent thrombosis (ST), factors showing a significant trend ($p<0.05$) in the univariate assessments were progressively incorporated into multivariable Cox regression models. Separate multivariable Cox regression analyses were performed to avoid multicollinearity between the inflammation indices. We analyzed SII and PIV as continuous variables in multivariable Cox regression models to preserve all available information, avoid arbitrary cut-offs, and mitigate the inflation of hazard ratios and type I errors.

We evaluated the predictiveness of the admission SII and PIV values for the occurrence of ST with receiver operating characteristic (ROC) analysis instead of ROC-based cut-off selection and the associated risk of bias.

The correlation between inflammation indices and hs-CRP levels was assessed using Pearson's correlation test. Statistical significance was established at a two-tailed p-value of <0.05 for all analytical evaluations.

RESULTS

A detailed comparison of baseline clinical status, laboratory markers, echocardiographic metrics, and angiographic findings between patients

Table 1. Baseline clinical and laboratory characteristics of the study population stratified by stent thrombosis

Characteristic	Total (n = 1683)	Stent Thrombosis (+) (n = 104)	Stent Thrombosis (-) (n = 1579)	P value
Age (years)	75.3 ± 6.8	75.4 ± 6.6	75.3 ± 6.8	0.974
Gender, Male n (%)	1088 (64.6)	70 (67.3)	1018 (64.5)	0.558
Hypertension, n (%)	691 (41.1)	55 (52.9)	636 (40.3)	**0.011**
Dyslipidemia, n (%)	1010 (60.0)	70 (67.3)	940 (59.5)	0.117
Smoking, n (%)	692 (41.1)	53 (51.0)	639 (40.5)	**0.035**
Diabetes, n (%)	653 (38.8)	40 (38.5)	613 (38.8)	0.942
Family history of CAD, n (%)	501 (29.8)	29 (27.9)	472 (29.9)	0.664
LVEF (%)	44.2 ± 5.0	44.5 ± 5.0	44.2 ± 5.0	0.528
Beta Blocker, n (%)	1614 (95.9)	99 (95.2)	1515 (95.9)	0.707
Statin, n (%)	1495 (88.8)	89 (85.6)	1406 (89.0)	0.277
ACEi/ARB, n (%)	1515 (90.1)	93 (90.3)	1422 (90.1)	0.939
Aspirin, n (%)	1502 (89.2)	90 (86.5)	1412 (89.4)	0.358
Clopidogrel, n (%)	1374 (81.6)	78 (75.0)	1296 (82.1)	0.071
Prasugrel, n (%)	48 (2.9)	4 (3.8)	44 (2.8)	0.529
Ticagrelor, n (%)	46 (2.7)	4 (3.8)	42 (2.7)	0.472
GpIIb/IIIa inhibitor, n (%)	564 (33.5)	39 (37.5)	525 (33.2)	0.374
DAPT interruption <30 days, n (%)	66 (3.9)	9 (8.7)	57 (3.6)	**0.010**
DAPT interruption <6 months, n (%)	91 (5.4)	9 (8.7)	82 (5.2)	0.131
DAPT adherence (1 year), n (%)	1338 (79.5)	83 (79.8)	1255 (79.5)	0.946
Stent Diameter (mm)	2.9 ± 0.3	2.9 ± 0.3	2.9 ± 0.3	0.742
Stent length† (mm)	22 (21-25)	24 (21-26)	22 (21-25)	**0.013**
Number of stents	1.6 ± 0.6	1.6 ± 0.5	1.6 ± 0.6	0.432
BMS, n (%)	202 (12.0)	18 (17.3)	184 (11.7)	0.086
Pain to balloon time † (hours)	4.0 (3.0-4.0)	4.0 (3.0-4.0)	4.0 (3.0-4.0)	0.783
Urea† (mg/dL)	36 (28-46)	35 (28-46)	37 (28-46)	0.706
Creatinine† (mg/dL)	0.9 (0.7-1.1)	0.9 (0.7-1.1)	0.9 (0.7-1.0)	0.398
Total cholesterol (mg/dL)	177 ± 42	177 ± 44	177 ± 42	0.974
Triglyceride (mg/dL)	150 ± 76	156 ± 62	149 ± 77	0.365
LDL-C (mg/dL)	109 ± 34	110 ± 37	109 ± 34	0.764
HDL-C (mg/dL)	41 ± 11	40 ± 10	41 ± 11	0.105
Albumin (g/dL)	3.6 ± 0.7	3.6 ± 0.2	3.6 ± 0.7	0.443
hs-CRP†(mg/L)	5.0 (2.2-12.0)	8.0 (2.0-16.0)	5.0 (2.4-10.6)	0.088
Hemoglobin (g/dl)	13.8 ± 1.7	13.6± 1.5	13.8± 1.7	0.475
WBC (×10³/µL)	7.6 ± 1.6	7.8 ± 1.4	7.6 ± 1.6	0.221
Neutrophil (×10³/µL)	4.6 ± 1.2	4.9 ± 1.2	4.6 ± 1.2	**0.028**
Lymphocyte (×10³/µL)	2.4 ± 0.7	2.1± 0.5	2.4 ± 0.7	**<0.001**
Monocyte (×10³/µL)	0.5 ± 0.2	0.6 ± 0.1	0.5 ± 0.2	0.122
SII	507 ± 170	640 ± 210	498 ± 164	**<0.001**
PIV	287 ± 145	391 ± 156	280 ± 142	**<0.001**

Data are presented as mean ± SD or n (%); †Comparisons were made using the Mann-Whitney U test at P <0.05, and these values were described by median with inter-quartile range (25th and 75th percentile); ACE inh: angiotensin-converting enzyme inhibitor, ARB: Angiotensin II receptor blocker, BMS: bare metal stent, CAD: Coronary artery disease, hs-CRP: high-sensitivity C-reactive protein, DAPT: Dual antiplatelet therapy, GpIIb/IIIa inhibitor: Glycoprotein IIb/ IIIa inhibitor, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, LVEF: left ventricular ejection fraction, PIV: pan-immune-inflammation value , SII: systemic immune-inflammation index, WBC: white blood cell. Bold data displays a statistically significant difference (p<0.05)

with and without stent thrombosis is outlined in Table 1. Of the 1683 patients, 64.6% were male, and the mean age was 75.3 ± 6.8 years. After a mean follow-up period of 30 ± 10 months, stent thrombosis was observed in 104 patients (6.2%) (Table 1). A comparative analysis revealed that total stent length, alongside baseline values of SII and PIV, were significantly higher in patients with stent thrombosis than in those without, whereas baseline hs-CRP levels showed a non-significant trend toward higher values in the thrombosis group (p=0.088). Moreover, the rates of hypertension, smoking, and early discontinuation of DAPT (<30 days) were also elevated in the thrombosis cohort.

ROC curve analysis was used to assess the discriminatory capability of the admission SII and PIV values for stent thrombosis. ROC curve analysis showed that admission SII and PIV values significantly predicted stent thrombosis, with an AUC of 0.70 (p <0.001) for SII and 0.71 (p <0.001) for PIV.

The associations between potential risk factors and stent thrombosis were assessed using univariate and multivariate regression analyses. In the univariate Cox proportional hazards analysis, a significant association with the development of stent thrombosis (ST) was observed for hypertension, smoking, and premature DAPT interruption of less than 30 days (p<0.05). Furthermore, increased stent length, alongside elevated baseline SII and PIV scores, also emerged as significant predictors within this model (Table 2). The stent type was not statistically significant for ST in the univariate analysis (p=0.094). Subsequently, two different models were created to assess the predictive value of both the SII and PIV in the multivariate analysis (Table 3). In multivariate models adjusted for hypertension, smoking, DAPT interruption of less than 30 days, and stent length, SII (HR, 1.004; 95% CI, 1.003–1.006; p<0.001) and PIV (HR, 1.003; 95% CI, 1.002–1.004; p<0.001) were confirmed as independent predictors of ST.

Table 2. Univariate Cox regression analysis

Variable	HR	95% CI	P value
Age	1.001	0.973–1.029	0.947
Gender	1.134	0.753–1.708	0.548
Hypertension	1.625	1.105–2.388	**0.014**
Diabetes	0.988	0.665–1.466	0.952
Dyslipidemia	1.373	0.911–2.068	0.130
Family history of CAD	0.915	0.596–1.404	0.684
Smoking	1.513	1.030–2.223	**0.035**
LVEF	1.011	0.974–1.050	0.562
GpIIb/IIIa inhibitor	1.202	0.808–1.787	0.364
DAPT interruption <30 days	2.336	1.179–4.628	**0.015**
DAPT interruption <6 months	1.646	0.831–3.261	0.153
DAPT adherence (1 year)	1.023	0.634–1.650	0.927
Stent diameter (mm)	0.922	0.533–1.593	0.770
Stent length (mm)	1.076	1.042–1.111	**<0.001**
Number of stents	1.124	0.822–1.537	0.463
BMS	1.544	0.929–2.566	0.094
Pain to balloon time (min)	0.970	0.881–1.069	0.542
Urea (mg/dL)	1.008	0.998–1.019	0.135
Creatinine (mg/dL)	1.643	0.923–2.925	0.091
Total cholesterol (mg/dL)	1.000	0.996–1.005	0.998
Triglyceride (mg/dL)	1.001	0.999–1.003	0.385
LDL-C (mg/dL)	1.001	0.995–1.006	0.772
HDL-C (mg/dL)	0.985	0.968–1.003	0.098
hs-CRP (mg/L)	1.035	1.010–1.061	**0.007**
Albumin (g/dL)	1.049	0.920–1.195	0.475
Hemoglobin (g/dL)	0.962	0.861–1.074	0.487
WBC ($\times 10^3/\mu L$)	1.078	0.962–1.209	0.196
Neutrophil ($\times 10^3/\mu L$)	1.186	1.019–1.381	**0.028**
Lymphocyte ($\times 10^3/\mu L$)	0.546	0.400–0.745	**<0.001**
Monocyte ($\times 10^3/\mu L$)	1.536	0.893–2.643	0.121
SII	1.004	1.003–1.005	**<0.001**
PIV	1.004	1.003–1.005	**<0.001**

BMS: bare metal stent, CAD: Coronary artery disease, CI: confidence interval, hs-CRP: high-sensitivity C-reactive protein, DAPT: Dual antiplatelet therapy, GpIIb/IIIa inhibitor: Glycoprotein IIb/ IIIa inhibitor, HDL-C: high-density lipoprotein cholesterol, HR: hazard ratio, LDL-C: low-density lipoprotein cholesterol, LVEF: left ventricular ejection fraction, PIV: pan-immune-inflammation value, SII: systemic immune-inflammation index, WBC: white blood cell. Bold data displays a statistically significant difference (p<0.05)



Table 3. Cox regression analysis to examine the association between inflammation indices and stent thrombosis

Variables	Adjusted HR	95%CI	p-value
MODEL 1			
Hypertension	1.474	0.986–2.202	0.058
Smoking	1.742	1.175–2.583	**0.006**
Stent length	1.043	1.007–1.081	**0.019**
DAPT interruption <30 days	1.363	0.664–2.799	0.399
SII	1.004	1.003–1.006	**<0.001**
hs-CRP	0.977	0.942–1.013	0.209
MODEL 2			
Hypertension	1.820	1.216–2.724	**0.004**
Smoking	1.528	1.019–2.291	**0.040**
Stent length	1.057	1.021–1.094	**0.002**
DAPT interruption <30 days	1.527	0.743–3.139	0.250
PIV	1.003	1.002–1.004	**<0.001**
hs-CRP	1.002	0.971–1.033	0.917

CI: confidence interval, hs-CRP: high-sensitivity C-reactive protein, DAPT: Dual antiplatelet therapy, HR: Hazard ratio, SII: systemic immune-inflammation index, PIV: pan-immune-inflammation value

In the correlation analysis, the SII had a statistically significant positive correlation with hs-CRP levels ($r = 0.342$, $p < 0.001$), and PIV had a mild but statistically significant positive correlation with hs-CRP levels ($r = 0.268$, $p < 0.001$).

DISCUSSION

This study showed that elevated SII and PIV levels are independently associated with a higher risk of stent thrombosis in geriatric patients with STEMI. These inflammatory indices could reflect the level of systemic inflammation, as indicated by their positive correlation with the serum hs-CRP values.

Geriatric patients with STEMI are at a higher risk than younger patients due to frailty and extensive coronary artery disease. Multiple comorbidities, atypical presentations, and delays in medical care contribute to increased risks of mortality and morbidity (2). Aged individuals are often selectively excluded from trials of acute coronary syndrome and revascularization owing to their potentially high-risk features. Consequently, available data have largely been drawn from cohorts of younger individuals, thereby restricting the generalizability of these insights to elderly patients, who fundamentally suffer from the most severe cardiovascular outcomes.

Despite major technological leaps in coronary intervention and contemporary antiplatelet pharmacology, ST remains a critical clinical concern with severe prognostic implications. The multifactorial etiology of this phenomenon encompasses a complex combination of periprocedural factors, patient-related risks, and specific stent properties. The likelihood of ST increases with the acuity of the patient's condition, being higher in those with STEMI and increasing with age (5).

Beginning with the stent implantation procedure, baseline and post-procedural inflammatory status, which may lead to endothelial dysfunction and a hypercoagulable state, have been postulated to be the most important predictors of ST (6,7). This process is characterized by the interaction of platelets, endothelial cells, and leukocytes. Circulating monocytes and neutrophils are sources of several factors, and their receptors interact mainly with platelets and endothelial cells, causing increased inflammatory activity and an aggravated prothrombotic status (14). Increased platelet counts are closely correlated with acute-phase reactants, pro-inflammatory mediators, increased thrombocyte activation, and prothrombotic status. Conversely, lymphocytes are characterized by a homeostatic and regulatory inflammatory signaling cascade, reflecting a down-regulated or blunted adaptive immune response (14). Inflammatory responses to stenting lead to infiltration of macrophages and monocytes as well as antigen-specific adaptive immune hypersensitivity reactions and subsequent thrombus accumulation, causing inflammatory cell infiltration and insufficient reendothelialization in coronary arteries treated with a stent (15).

Investigating systemic inflammatory biomarkers offers critical insights into predicting ST development. Baseline C-reactive protein (CRP) levels, which quantify systemic inflammatory activity, have been firmly established as key indicators linked to recurrent myocardial infarction, stent-related thrombosis, and poor survival outcomes in acute coronary syndrome populations (16). Although baseline hs-CRP levels did not achieve formal statistical significance between the ST and non-ST groups ($p=0.088$), our study demonstrated a significant positive correlation between hs-CRP concentrations and both novel inflammatory indices ($r=0.342$ for SII and $r=0.268$ for PIV, $p<0.001$). This correlation reinforces the hypothesis that these composite hematological parameters effectively reflect systemic inflammatory signaling in the acute STEMI setting.

To date, some researchers have used white blood cells to predict the severity, complications, and prognosis of coronary artery disease and the occurrence of stent thrombosis; however, their sensitivities and specificities are limited (16). One may hypothesize that one- or two-component markers may be insufficient to predict the prognosis of patients with ACS. Recently, some novel composite inflammatory hematological indices, including the SII and, more recently, the PIV, have emerged to better reflect the overall inflammatory and immune-thrombotic status compared to the use of any single leukocyte subtype in isolation. Evidence from many observational studies has shown a significant relationship between these composite inflammation indices and the incidence and severity of several cardiovascular disease states, such as myocardial infarction, heart failure, and atrial fibrillation (10). Ocal et al. demonstrated that a high baseline SII actively represents an independent risk factor for short-term mortality and major adverse cardiac events among individuals presenting with STEMI (17). Liu et al. evaluated 216 patients with STEMI and demonstrated that PIV 12 h after PCI was a significant predictor of long-term prognosis (14). Zheng et al. evaluated 887 patients with STEMI and found that increased SII values during coronary stent implantation for primary PCI were associated with the development of stent thrombosis, consistent with our findings (11). However, this study differed from our study in that the patients were limited to younger patients and had a smaller sample size. Recently, Ma and Geng evaluated the prognostic utility of another composite inflammatory metric—the systemic inflammation response index (SIRI)—in a cohort of 960 elderly patients with acute coronary syndrome (ACS) undergoing stent implantation, demonstrating that SIRI independently predicted 3-year major adverse cardiac events (MACE) (18). While their findings underscore the heightened immune-inflammatory burden in older adults with ischemic heart disease, our study uniquely extends this concept in several key aspects. First, instead of a broad ACS cohort receiving both elective and primary revascularization, our investigation was restricted specifically to geriatric patients presenting with acute STEMI managed via primary PCI. Second, we assessed the predictive efficacy of admission SII and PIV—the latter providing a more comprehensive snapshot of both innate (neutrophils, monocytes), thrombotic (platelets), and adaptive (lymphocytes) pathways—specifically for the occurrence of definite stent thrombosis rather than composite MACE. Thus, our data refine the current understanding by confirming that multi-cell immuno-inflammatory parameters (SII and PIV) are specifically tied to acute mechanical-thrombotic complications in the highest-risk geriatric STEMI setting.

Although substantial progress has been made over the past decade in utilizing biomarkers to elucidate how inflammatory pathways drive the pathogenesis of stent thrombosis (11,19), few studies have specifically investigated this phenomenon in geriatric populations. Data from another recent study by Del Turco et al. on 625 STEMI cases highlighted that age significantly influences the baseline inflammatory response, showing varying patterns between young and elderly groups during



initial admission (20). Their study substantiated that elderly patients exhibit a more intense acute systemic inflammatory response following STEMI. Concurrently, these patients carry a chronic, low-grade inflammatory burden-an architectural feature of aging shaped by multiple comorbidities, poor nutritional status, and a general decline in both adaptive and innate immunological defenses. Therefore, the current study aimed to investigate whether the clinical benefits of novel inflammation indices as predictors of stent thrombosis in geriatric patients could be validated in the STEMI setting.

Our findings are consistent with those reported in the literature, demonstrating that inflammation and immune-thrombotic alterations significantly contribute to stent thrombosis pathophysiology, and that indices that indirectly reflect inflammation can reflect the success rate of primary PCI in geriatric patients with STEMI. In clinical practice, these contemporary inflammatory biomarkers, SII and PIV, may offer potential utility in recognizing patients predisposed to ST. In the specific context of elderly STEMI cases, we hypothesize that utilizing these tools could theoretically inform future therapeutic decisions, such as the deployment of intensive monitoring, early intensified therapies, or personalized adjustments to antiplatelet protocols. However, given the retrospective nature of our data, these observations remain hypothesis-generating. Moreover, as the cohort of geriatric STEMI patients treated with primary PCI continues to climb, addressing their elevated susceptibility to mortality and morbidity becomes critical. Consequently, these high-risk individuals could achieve more pronounced clinical benefits from tailored anti-inflammatory therapeutics in the future.

This study has several limitations. Although this study included a relatively large cohort, it was limited by its single-center retrospective design. Additionally, several risk factors for ST were not assessed, which may have influenced the multivariate test results. We evaluated the SII and PIV values only once during admission, rather than examining temporal changes throughout the follow-up period. Furthermore, patients with cardiogenic shock, prior fibrinolytic therapy, or periprocedural mechanical complications were intentionally excluded to minimize procedural confounders. Because these excluded subgroups represent the highest-risk spectrum of geriatric STEMI patients, our findings may not be fully generalizable to the broader, more clinically heterogeneous real-world elderly population encountered in routine emergency practice. Finally, the absence of other inflammatory markers, except hs-CRP, is another limitation. It is important to note that the present study was designed to demonstrate an association rather than a direct mechanistic cause-and-effect link between contemporary inflammatory markers and ST. Prospectively designed studies on larger geriatric cohorts are needed to confirm our findings and determine the utility of these indices in this specific population.

Conclusions

Ultimately, SII and PIV represent highly accessible, cost-neutral biomarkers that can be easily calculated during initial bedside evaluation via routine blood testing, offering valuable insights into the underlying inflammatory and immuno-thrombotic status. This study is the first to examine the relationship between admission SII and PIV values and stent thrombosis in geriatric patients with STEMI. According to our data, higher values of both SII and PIV carry a statistically significant and independent association with subsequent stent thrombosis events, serving as hypothesis-generating risk indicators. Nevertheless, these observations necessitate validation through future prospective, large-scale trials before any definitive clinical or therapeutic recommendations can be established. Incorporating a broader spectrum of inflammatory biomarkers will be essential to definitively clarify the mechanistic role of these novel inflammatory metrics within the geriatric STEMI cohort.

Conflicts of interest: The authors of this article state that they have no conflict of interest.

Funding Statement: No funding information was available.

Abbreviations: ACS, acute coronary syndrome; CAD, coronary artery disease; hs-CRP, high-sensitivity C-reactive protein; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention; PIV, pan-immune-inflammation value; SII, systemic immune-inflammation index; ST, stent thrombosis; STEMI, ST-segment elevation myocardial infarction; TIMI, thrombolysis in myocardial infarction.

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