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ORIGINAL ARTICLE

The Relationship Between Systemic Inflammatory Response Index and Systemic Immune-Inflammation Index and Diagnostic Rates of Myocardial Infarction, Pulmonary Thromboembolism, and Aortic Dissection in Patients Presenting to the Emergency Department with Chest Pain

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Abstract

Background: This study aims to investigate the association between the Systemic Inflammatory Response Index (SIRI) and the Systemic Immune-Inflammation Index (SII) and serious cardiovascular diagnoses such as myocardial infarction, pulmonary thromboembolism, and aortic dissection in patients presenting to the emergency department with chest pain. Additionally, it evaluates the potential role of these biomarkers in supporting clinical decision-making processes.

Methods: This retrospective cross-sectional study included 612 patients aged 18–100 years who presented with chest pain to the Emergency Department of Yozgat City Hospital between July 1, 2025, and July 31, 2025. Demographic characteristics, final diagnoses, and laboratory parameters were analyzed, and SII and SIRI values were calculated accordingly. Statistical analyses were conducted using SPSS 22 with appropriate statistical tests.

Results: In the STEMI group, the mean SII was 1725.90 ± 1274.25 and the mean SIRI was 4.50 ± 3.66. In the NSTEMI group, these values were 1410.94 ± 1341.51 and 3.28 ± 2.79, respectively. Patients with non-cardiovascular causes exhibited significantly lower SII and SIRI levels. Based on ROC analysis, the AUC for SII was 0.738 with a cut-off value of 658, while the AUC for SIRI was 0.723 with a cut-off of 1.92. A positive and significant correlation was found between SII/SIRI and troponin and D-dimer levels ($p < 0.001$).

Conclusion: SII and SIRI levels are markedly elevated in chest pain of cardiovascular origin and may serve as useful adjunct biomarkers in the diagnostic differentiation of these conditions.

Keywords: Chest Pain, Systemic Inflammatory Response Index (SIRI), Emergency Department Diagnostic Approach.

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INTRODUCTION

Chest pain is one of the most common reasons for presentation to emergency departments, and due to the wide spectrum of underlying pathologies, it requires a holistic evaluation that encompasses biological, psychological, and social dimensions. While some cases of chest pain may herald serious cardiovascular conditions, others are associated with benign disorders that can be managed with minimal intervention. This uncertainty compels clinicians to work against time, making rapid yet accurate decisions essential (1). Consequently, the importance of decision-support tools—particularly laboratory-based biomarkers—continues to grow within the diagnostic workflow (2).

In recent years, inflammation-based biomarkers such as the Systemic Inflammatory Response Index (SIRI) and the Systemic Immune-Inflammation Index (SII) have emerged as tools that allow a more comprehensive assessment of systemic inflammatory burden. These indices rely on simple mathematical combinations of complete blood count parameters and reflect the degree of inflammatory response by incorporating neutrophil, monocyte, lymphocyte, and platelet counts (3). Their ability to be easily calculated from routine laboratory tests and their cost-effectiveness provide significant advantages for clinical implementation (4).

Numerous studies have demonstrated that SIRI and SII possess prognostic value in cardiovascular diseases such as coronary artery disease, acute coronary syndrome, and myocardial infarction (5). In the literature, these indices have been reported to be effective in predicting disease presence, severity, and clinical outcomes. Notably, they have shown correlation with lesion severity in NSTEMI cases, suggesting their potential utility in risk stratification (6,7). Furthermore, elevated SIRI and SII levels have been documented in chronic inflammatory conditions such as obesity and hypertension, supporting the association between inflammatory burden and increased cardiovascular risk (8).

The role of inflammatory markers has also garnered growing attention in the diagnostic evaluation of pulmonary thromboembolism (PTE), a critical differential diagnosis in patients presenting with chest pain. Several studies have shown that SIRI and SII contribute to PTE diagnosis and risk stratification, and may be particularly valuable in predicting short-term adverse outcomes among high-risk patients (9,10,11).

These findings underscore the need to consider not only symptoms but also the underlying biochemical and pathophysiological processes in the evaluation of chest pain. In this context, the use of inflammation-based indices may facilitate early risk identification and enhance clinical decision-making.

The aim of this study is to retrospectively analyze the relationship between SIRI and SII levels and serious diagnoses such as myocardial infarction and pulmonary thromboembolism in patients presenting to the emergency department with chest pain, and to elucidate the potential diagnostic and prognostic value of these biomarkers within clinical decision-support processes.

MATERIALS AND METHODS

Study Design and Participants

This study was approved by the Yozgat Bozok University Non-Interventional Clinical Research Ethics Committee (2025-GOKAEK-2518_2025.15.10_687). The research included adult patients aged 18–100 years who presented with chest pain to the Emergency Medicine Clinic of Yozgat City Hospital between July 1, 2025, and July 31, 2025. The study employed a retrospective, descriptive, and cross-sectional design with no control group. Data were obtained exclusively from the Yozgat City Hospital digital medical records system; no patients from other centers were included. Researchers from the Departments of Cardiovascular Surgery, Pulmonology, and Cardiology contributed solely to data analysis and reporting.

Patients were included if they presented to the emergency department with chest pain, were between 18 and 100 years of age, had a definitive diagnosis, and had complete laboratory data available. Exclusion criteria included missing laboratory data, presentation for reasons other than chest pain followed by a later cardiovascular diagnosis, and conditions known to significantly influence inflammatory biomarkers, such as advanced infections, active malignancy, chronic inflammatory diseases, or a history of immunosuppressive therapy. This approach was chosen to minimize potential confounding effects on inflammation-based indices.

The primary outcome of the study was to evaluate the diagnostic performance of SIRI and SII in differentiating cardiac (STEMI, NSTEMI) and non-cardiac causes of chest pain. Secondary outcomes included: (1) assessing the association of SIRI and SII with specific diagnoses such as STEMI, NSTEMI, and pulmonary thromboembolism (PTE); (2) examining correlations between these indices and Troponin-I and D-dimer levels; and (3) determining cut-off values, sensitivity, and specificity through ROC analyses.

Clinical diagnoses were confirmed according to international guidelines. STEMI diagnosis was established based on the criteria of the 2023 European Society of Cardiology (ESC) guidelines, including typical ischemic symptoms, ST-segment elevation in at least two contiguous leads, and elevated troponin levels. NSTEMI was diagnosed by the presence of elevated troponin without ST-segment elevation. The diagnosis of pulmonary thromboembolism was confirmed using the 2019 ESC PTE guidelines, incorporating clinical suspicion, elevated D-dimer, and findings from CT pulmonary angiography.

Data Collection

Data were collected retrospectively from patient charts and the hospital information management system. Recorded variables included age, sex, hospitalization indication, definitive diagnosis, and laboratory parameters (white blood cell, neutrophil, lymphocyte, monocyte, and platelet counts; C-reactive protein; Troponin-I; and D-dimer). When necessary, ECG, echocardiography, and CT angiography reports were reviewed.

Using these data, inflammation-based indices were calculated as follows:

- **SIRI = (Neutrophil × Monocyte) / Lymphocyte**
- **SII = (Platelet × Neutrophil) / Lymphocyte**

Patients with missing data were excluded from the analysis, and no data correction or imputation procedures were performed.

Statistical Analysis

Data were analyzed using SPSS for Windows Version 22. The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Variables with normal distribution were compared using the Independent Samples t-test, while non-normally distributed variables were compared using the Mann–Whitney U test or the Kruskal–Wallis test. For correlation analyses, Pearson correlation was used for normally distributed variables and Spearman correlation for non-normally distributed variables.

Receiver operating characteristic (ROC) curve analysis was performed to assess the diagnostic performance of SIRI and SII in distinguishing cardiac diagnoses. Area under the curve (AUC), cut-off values, sensitivity, and specificity were calculated. A p-value of <0.05 was considered statistically significant for all analyses.

RESULTS

In this study, a total of 612 patients who presented with chest pain to the Emergency Medicine Clinic of Yozgat City Hospital between July 1, 2025, and July 31, 2025, were retrospectively evaluated. Of the included patients, 61.6% were male (n = 377) and 38.4% were female (n = 235). The mean age of male patients was 49.9 ± 17.8 years, whereas the mean age among female patients was 50.9 ± 20.2 years.

Evaluation of sex-based laboratory parameters provided valuable insights into the physiological responses of individuals presenting with chest pain. The mean white blood cell (WBC) count was 9.82 ± 6.62 $\times 10^9$/L in males and 8.93 ± 2.85 $\times 10^9$/L in females. Hemoglobin (HB) levels were measured as 15.15 ± 1.64 g/dL in males and 13.06 ± 1.63 g/dL in females. Platelet counts were higher in female patients, with a mean of 279.49 ± 72.29 $\times 10^9$/L compared with 247.86 ± 63.24 $\times 10^9$/L in males. Neutrophil counts were 6.25 ± 3.15 $\times 10^9$/L in males and 5.84 ± 2.70 $\times 10^9$/L in females, whereas lymphocyte counts were similar between groups (2.31 ± 0.99 $\times 10^9$/L in males; 2.23 ± 0.97 $\times 10^9$/L in females). Mean monocyte levels were 0.71 ± 0.34 $\times 10^9$/L in males and 0.64 ± 0.29 $\times 10^9$/L in females (Table 1).

Assessment of cardiac and thrombotic biomarkers revealed broad variability among patients presenting with chest pain. The mean Troponin level was 1031.76 ± 4450.83 ng/L. The mean D-dimer level was 0.59 ± 1.46 μg/mL, reflecting its critical role in evaluating or excluding pulmonary embolism and other thromboembolic conditions.

Table 1. Mean ± SD Values of Hematological and Biochemical Parameters By

Parameter	Male (n=377)	Female (n=235)	p-value
White Blood Cells (10^9/L)	9.82 ± 6.62	8.93 ± 2.85	0.028
Hemoglobin (g/dL)	15.15 ± 1.64	13.06 ± 1.63	<0.001
Platelets (10^9/L)	247.86 ± 63.24	279.49 ± 72.29	<0.001
Neutrophils (10^9/L)	6.25 ± 3.15	5.84 ± 2.70	0.048
Lymphocytes (10^9/L)	2.31 ± 0.99	2.23 ± 0.97	0.332
Monocytes (10^9/L)	0.71 ± 0.34	0.64 ± 0.29	0.014

Based on final diagnoses, the most common condition was ST-segment elevation myocardial infarction (STEMI), observed in 180 patients (29.41%). This was followed by non–ST-segment elevation myocardial infarction (NSTEMI) in 104 patients (16.99%), pulmonary thromboembolism (PTE) in 43 patients (7.03%), and arrhythmia in 36 patients (5.88%). The remaining 249 patients (40.69%) were diagnosed with non-cardiovascular causes of chest pain. No cases of aortic dissection were identified during the study period.

The high numbers of STEMI and NSTEMI cases may be attributed to Yozgat City Hospital serving as the only center in the region providing emergency coronary angiography and interventional cardiac procedures, resulting in the referral of all STEMI/NSTEMI cases to this institution.

Analysis of SII and SIRI values across diagnostic groups showed that patients diagnosed with STEMI had a mean SII of 1725.90 ± 1274.25 and a mean SIRI of 4.50 ± 3.66. In the NSTEMI group, the mean SII was 1410.94 ± 1341.51 and the mean SIRI was 3.28 ± 2.79. Among patients with PTE, the mean SII was 1857.95 ± 1640.83 and the mean SIRI was 6.52 ± 8.10. Patients presenting with non-cardiovascular causes exhibited significantly lower values, with a mean SII of 731.12 ± 654.18 and a mean SIRI of 2.09 ± 3.79 (Table 2).

Table 1. Mean ± SD Values of SII and SIRI Across Diagnostic Groups

Diagnosis Group	SII (Mean ± SD)	SIRI (Mean ± SD)
STEMI	1725.90 ± 1274.25	4.50 ± 3.66
NSTEMI	1410.94 ± 1341.51	3.28 ± 2.79
Pulmonary Embolism (PTE)	1857.95 ± 1640.83	6.52 ± 8.10
Non-cardiovascular Chest Pain	731.12 ± 654.18	2.09 ± 3.79

Comparison of SII and SIRI values among diagnostic groups using the Mann–Whitney U test demonstrated significant differences between patients with non-cardiovascular causes and those diagnosed with STEMI for both SII ($p < 0.001$) and SIRI ($p < 0.001$). Similarly, SII ($p < 0.001$) and SIRI ($p < 0.001$) values differed significantly between non-cardiovascular presentations and NSTEMI cases.

Additionally, a statistically significant difference in SII ($p < 0.001$) and SIRI ($p < 0.001$) values was observed between patients with non-cardiovascular causes and those diagnosed with PTE. However, no significant differences were found between the STEMI and NSTEMI groups for either SII ($p = 0.18$) or SIRI ($p = 0.09$). Similarly, comparisons between the STEMI and PTE groups revealed no significant differences for SII ($p = 0.34$) or SIRI ($p = 0.48$) (Figure 1).

When WBC, Troponin, and D-dimer levels were analyzed across diagnostic categories, notable variations were identified. In the STEMI group, the mean WBC level was $10.58 \pm 3.29 \times 10^9$/L, the mean Troponin level was 3342.30 ± 7646.92 ng/L, and the mean D-dimer level was 0.48 ± 0.77 μg/mL. For the NSTEMI group, these values were $9.67 \pm 2.82 \times 10^9$/L, 902.34 ± 2443.31 ng/L, and 0.46 ± 0.64 μg/mL, respectively. Among patients with non-cardiovascular causes, the mean WBC level was $8.13 \pm 2.49 \times 10^9$/L, the Troponin level was 19.56 ± 48.12 ng/L, and the D-dimer level was 0.68 ± 1.74 μg/mL.

Comparisons of key laboratory parameters among STEMI, NSTEMI, and non-cardiovascular groups were performed using the Kruskal–Wallis test because the variables did not conform to a normal distribution. The analysis demonstrated that several biomarkers differed significantly between diagnostic groups. WBC levels showed a marked difference ($p < 0.001$), and Troponin levels exhibited a highly significant variation as well ($p < 0.001$). D-dimer levels also differed significantly across groups ($p < 0.001$). In contrast, Hemoglobin (HB) levels did not differ significantly between diagnostic categories ($p = 0.073$) (Figure 2). Due to low sample numbers, patients diagnosed with PTE and arrhythmia were excluded from this subset analysis.

In this study, ROC curve analysis was conducted to assess whether SII and SIRI values could discriminate between cardiovascular and non-cardiovascular causes of chest pain. The analysis included patients with STEMI, NSTEMI, and non-cardiovascular presentations. The area under the curve (AUC) for SII was 0.738, indicating a moderate discriminative ability. The identified cut-off value of 658 yielded 73.8% sensitivity and 66.2% speci-

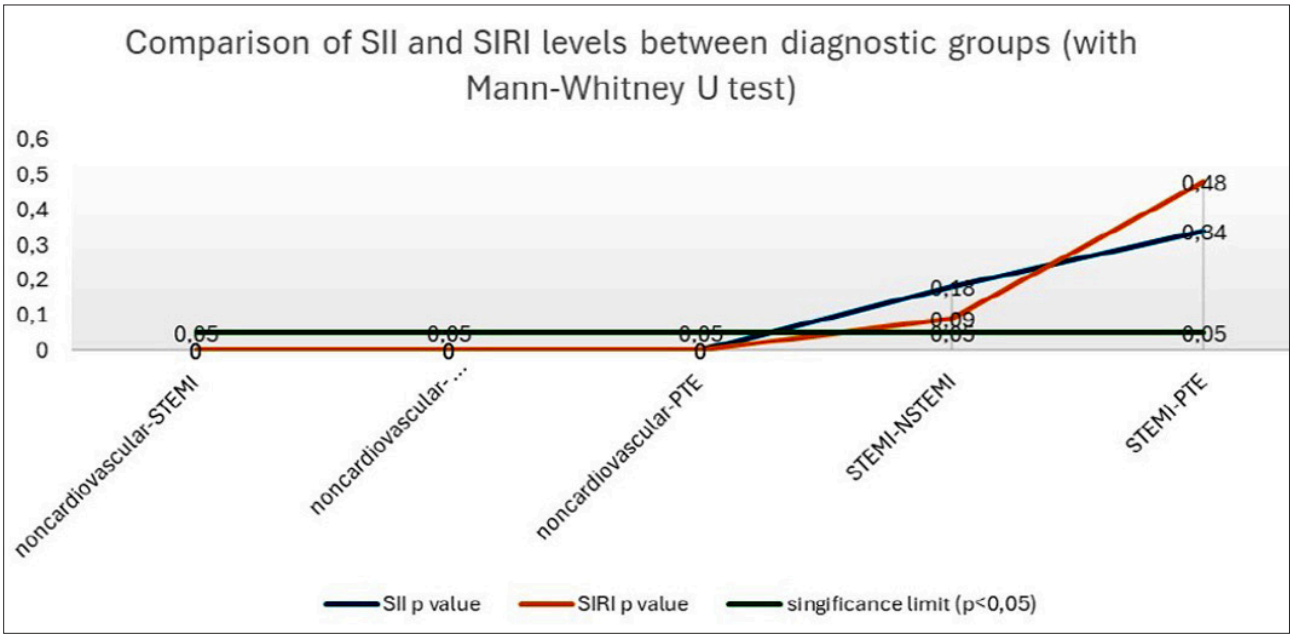

Figure 1: p-values of SII and SIRI comparisons among diagnostic groups (Mann–Whitney U test).

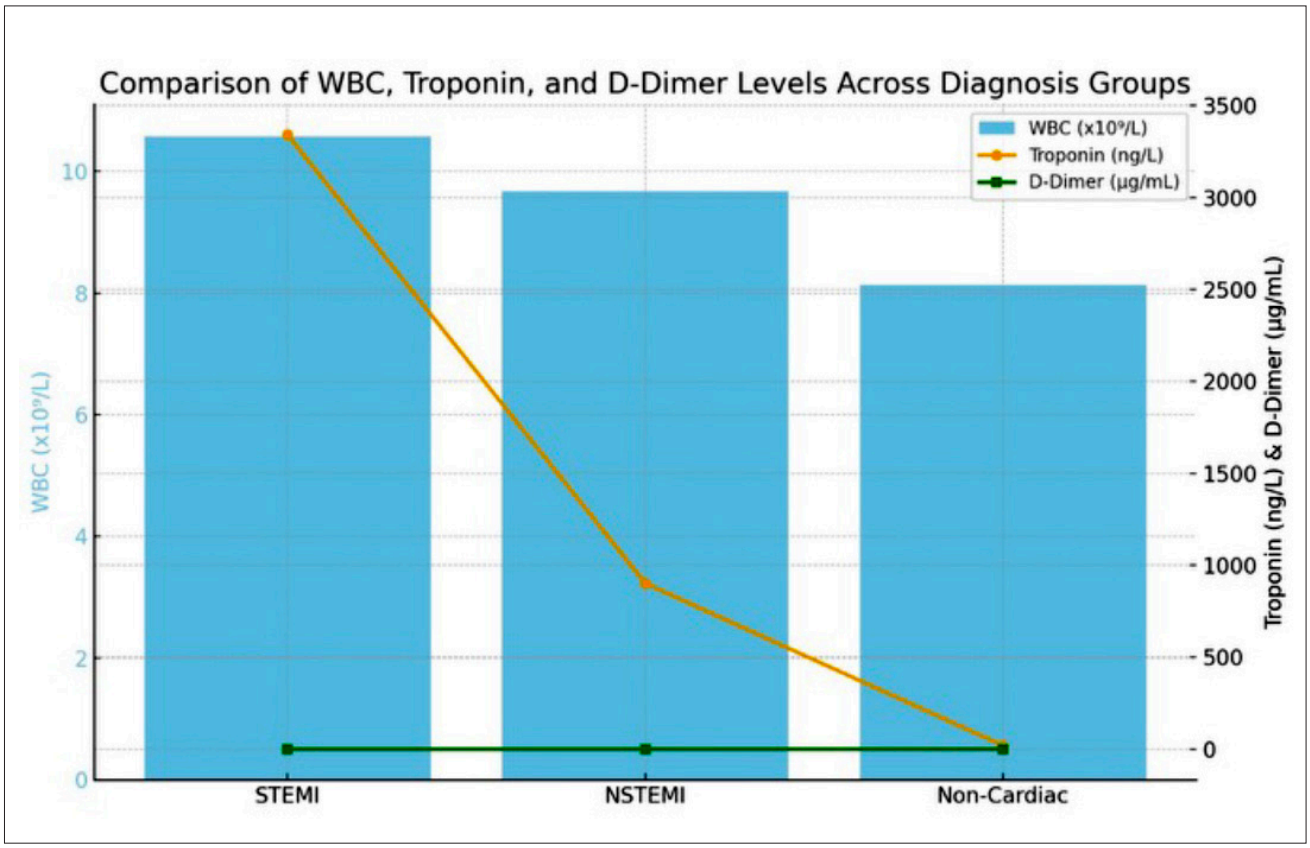

Figure 2: Comparison of mean WBC, Troponin, and D-dimer levels in STEMI, NSTEMI, and non-cardiovascular groups.

ficity. Similarly, SIRI demonstrated a moderate diagnostic performance, with an AUC of 0.723. The calculated cut-off value of 1.92 corresponded to 65.6% sensitivity and 73.2% specificity. These findings suggest that although neither index alone provides high diagnostic accuracy, both may serve as meaningful adjuncts to clinical evaluation (Figure 3).

Spearman correlation analysis was performed to examine the relationships between SII/SIRI and Troponin and D-dimer levels. This analysis offered important insights into the connection between systemic inflammation, myocardial injury, and thromboembolic processes. Significant positive correlations were observed between both SII and SIRI and Troponin levels (Spearman $r = 0.355$ for each, $p < 0.001$). Similarly, a positive and significant correlation was found between SII and D-dimer ($r = 0.286$) and between SIRI and D-dimer ($r = 0.331$) ($p < 0.001$ for both). These findings demonstrate that inflammation-based indices are associated with D-dimer, a biochemical indicator of thromboembolic activity.

DISCUSSION

In this retrospective evaluation of 612 patients presenting to the emergency department with chest pain, males constituted a clear majority of the study population (61.6%), while the mean ages of male and female patients were relatively similar. This finding highlights that chest pain is a more common reason for presentation among men; however, women of comparable age groups also seek emergency care for clinical conditions requiring cardiovascular evaluation. Consistent with previous literature, men have been reported to present more frequently with acute chest pain, a pattern attributed to both biological and behavioral factors (1,2). Nonetheless, the slightly higher mean age observed among female patients in our study may reflect sex-specific clinical characteristics, such as delayed presentation or a tendency toward atypical symptoms. These observations further underscore the importance of incorporating sex-based considerations into cardiovascular risk assessment.

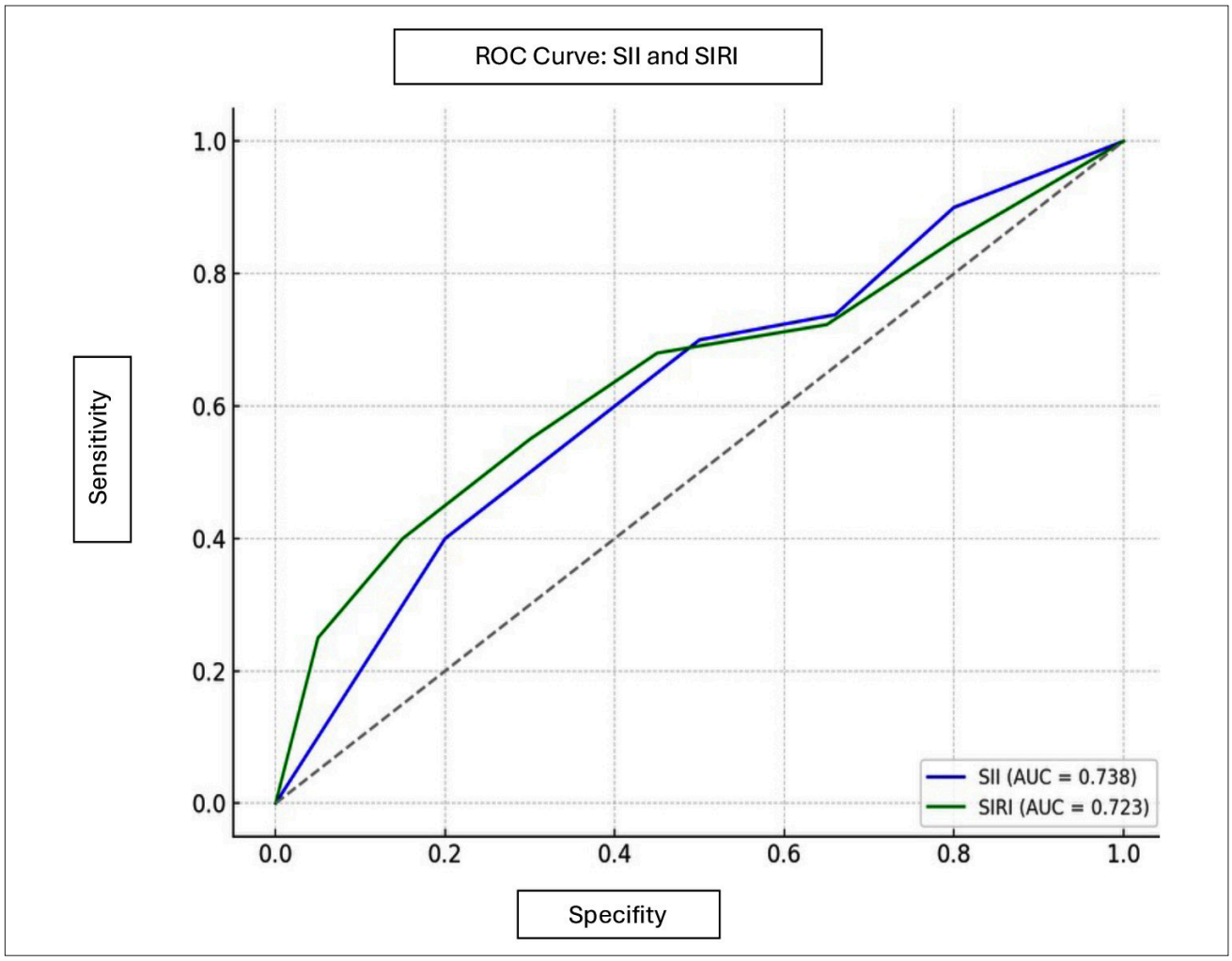

Figure 3: ROC analysis of SII and SIRI in distinguishing cardiac from non-cardiovascular diagnoses.

Comparison of laboratory parameters by sex provided meaningful insights into physiological responses among individuals presenting with chest pain. The finding that male patients had higher mean white blood cell (WBC) counts than females suggests that acute inflammatory responses may be more pronounced in men. Similarly, the significantly higher hemoglobin (Hb) levels observed in men reflect expected physiological differences and emphasize the need to consider sex-related hematologic variations during emergency evaluations. Conversely, the higher platelet counts recorded in female patients align with prior studies suggesting that estrogen may enhance platelet production (3). Elevated neutrophil and monocyte levels in male patients further support the hypothesis that systemic inflammatory activity may be more prominent in men. This is consistent with existing literature describing a stronger neutrophil-dominant inflammatory profile in males (4). Collectively, these findings indicate that sex-based interpretation of laboratory biomarkers may enhance diagnostic accuracy and improve risk stratification in clinical practice.

In the patient cohort evaluated in this study, substantial variability was observed in cardiac and thromboembolic biomarkers. The mean Troponin level of 1031.76 ± 4450.83 ng/L, accompanied by a notably large standard deviation, reflects considerable heterogeneity in the severity and extent of myocardial injury among patients. The literature consistently highlights the high sensitivity of troponin in diagnosing myocardial dam-

age and predicting clinical outcomes (3). In this regard, the markedly elevated troponin levels identified in the STEMI cases—representing the most frequent diagnostic category in our study—further reinforce the clinical reliability of this biomarker.

Similarly, the mean D-dimer level of 0.59 ± 1.46 μg/mL indicates that a considerable proportion of patients were evaluated for thromboembolic conditions. D-dimer is widely used as a diagnostic tool in thromboembolic events, particularly acute pulmonary embolism, and previous studies have emphasized its high negative predictive value in conditions such as PTE (9,10). The identification of 43 patients (7.03%) with PTE in our cohort supports the continued relevance of D-dimer in early risk stratification.

From a diagnostic distribution standpoint, the predominance of STEMI cases (29.41%) underscores the significance of acute myocardial infarction among patients presenting with chest pain. The subsequent proportions of NSTEMI (16.99%) and PTE (7.03%) highlight the necessity of considering a wide range of cardiovascular etiologies in the differential diagnosis of chest pain. Furthermore, the presence of 249 patients (40.69%) with non-cardiovascular causes illustrates that chest pain is not exclusively cardiac in origin but may arise from diverse systemic conditions. These findings align with prior research and support the essential role of a multidisciplinary approach in the evaluation of acute chest pain (1,2).

The variability observed in systemic inflammatory markers across diagnostic groups further underscores the relationship between inflammatory responses and the pathophysiology of cardiovascular diseases. Notably, patients diagnosed with acute coronary syndromes such as myocardial infarction, as well as those with pulmonary thromboembolism, exhibited more pronounced systemic inflammatory activity. These findings suggest that these non-infectious yet inflammation-driven clinical conditions can be meaningfully differentiated using inflammation-based biomarkers. In contrast, patients presenting with non-cardiovascular causes had lower levels of these indices, indicating a more limited inflammatory burden.

These results reinforce the concept that inflammatory markers play a significant role in the pathophysiology of cardiovascular diseases. A prospective cohort study by Pengfei et al. (2025) demonstrated that SII and SIRI were strongly associated with the development of cardiovascular disease in patients with cardiovascular–renal–metabolic syndrome (12). Similarly, Zheng et al. (2025) reported that elevated SII values were associated with both short- and long-term clinical outcomes in patients with acute myocardial infarction accompanied by hypertension (13).

Broadening this perspective, Liu and colleagues (2025) conducted a systematic review and meta-analysis and provided robust support for the utility of immune–inflammatory indices in cardiovascular risk stratification (14). Furthermore, a study by Luo et al. (2025) showed that complete blood count–based inflammatory markers were significantly associated with atrial fibrillation, heart failure, and cardiovascular mortality (15). When considered collectively alongside the findings of the present study, the literature strongly supports the potential diagnostic and prognostic value of SII and SIRI.

Comparisons among diagnostic groups revealed that the inflammatory indices SII and SIRI were significantly associated with cardiovascular diagnoses. In particular, patients presenting with cardiac and thromboembolic conditions such as STEMI, NSTEMI, and PTE demonstrated substantially higher levels of these markers compared with those presenting with non-cardiovascular causes. These findings once again highlight the integral role of inflammatory processes in acute cardiovascular events.

Similarly, Pengfei et al. (2025) emphasized the predictive value of SII and SIRI in the incidence of cardiovascular disease (12), while Zheng and colleagues (2025) reported significant correlations between these indices and clinical outcomes in hypertensive patients presenting with acute myocardial infarction (13). In a comprehensive systematic review, Liu et al. (2025) noted that these inflammation-based indices are valuable biomarkers for prognostic evaluation (14). Luo and colleagues (2025) further expanded the clinical relevance of such parameters by demonstrating their association with atrial fibrillation and cardiovascular mortality (15).

In this context, a meta-analysis by Ye et al. (2022) also identified SII as a strong prognostic indicator across various cardiovascular diseases (16), while the study by Karadeniz et al. (2024) supported the utility of these

indices in predicting clinical outcomes in acute coronary syndrome (17). Similarly, Tuzimek and colleagues (2024) reported that novel inflammatory indices such as SII, SIRI, and AISI were strongly associated with acute coronary syndrome in both diabetic and prediabetic patients, suggesting that these parameters may contribute to clinical assessment regardless of glycemic status (19). Regarding pulmonary embolism specifically, the review by Yang et al. (2021) highlighted the diagnostic value of inflammatory biomarkers in this population (18).

In our study, key laboratory parameters of patients presenting with STEMI, NSTEMI, and non-cardiovascular causes were compared across diagnostic groups. Since the variables did not show normal distribution, analyses were conducted using the Kruskal–Wallis test. The findings demonstrated significant differences in several biomarkers. WBC levels differed notably across groups ($p < 0,001$), while the marked difference in Troponin levels ($p < 0,001$) reflected the biochemical manifestation of myocardial injury. D-dimer levels also varied significantly between groups ($p < 0,001$). In contrast, HB levels did not differ significantly ($p = 0.073$).

These findings are consistent with existing literature. Dziedzic et al. (3) demonstrated the association of SII and SIRI levels with the severity of coronary artery disease and acute coronary syndrome. Samedov et al. (20) highlighted the prognostic significance of systemic inflammation indices in patients diagnosed with acute coronary syndrome. Wang et al. (21) further showed that SII was associated with coronary stenosis severity, particularly among diabetic individuals. When considered alongside our findings, the literature strongly suggests that inflammation-based parameters may possess diagnostic and prognostic value.

The results obtained in this study contribute to the understanding of the relationship between inflammatory response and cardiovascular diseases. The demonstration that systemic inflammatory indices such as SII and SIRI are associated with myocardial injury and thromboembolic processes in patients presenting with chest pain suggests that these biomarkers may support clinical evaluation. However, the moderate discriminative power of both indices in ROC analysis and their insufficiency as stand-alone diagnostic tools indicate that they should not be used independently. Although SII and SIRI showed significant positive correlations with D-dimer and Troponin levels, these relationships do not imply causality and cannot replace core diagnostic parameters in clinical decision-making.

The potential role of SII and SIRI as biomarkers for predicting cardiovascular disease is increasingly emphasized in recent literature. In a prospective cohort study, Jiang et al. (2025) demonstrated significant associations of both SII and SIRI with cardiovascular and all-cause mortality (22). Similarly, a cross-sectional analysis by Fu and Deng (2025) revealed a strong relationship between SIRI and structural cardiovascular changes such as vascular calcification (23). In a multicenter study focusing on acute coronary syndromes, Marchi et al. (2024) reported that both SII and SIRI were independent predictors of mortality in STEMI patients (24).

Taken together, the correlations observed between inflammatory indices and cardiac biomarkers in our study, along with the classification performance demonstrated in ROC analysis, support the notion that SII and SIRI may serve as useful parameters in diagnostic evaluation and risk stratification. Nevertheless, the limited sensitivity and specificity values indicate that these indices are better suited as components of multiparametric assessment algorithms rather than standalone diagnostic tools. Such an approach may help reduce diagnostic uncertainty, particularly in heterogeneous clinical presentations such as chest pain.

Despite these findings, our study has several important limitations. First, its retrospective and single-center design limits the generalizability of the results. Additionally, the relatively small sample sizes in the pulmonary thromboembolism and arrhythmia groups precluded their inclusion in certain statistical analyses and restricted the scope of intergroup comparisons. The reliance on single-time-point measurements of inflammation markers prevented evaluation of temporal dynamic changes and their relationship with disease progression. Furthermore, the absence of follow-up data—such as mortality, complication rates, and length of hospital stay—limited the ability to assess the long-term prognostic value of the inflammation indices. Finally, the lack of multivariate analyses hindered evaluation of whether SII and SIRI have independent effects beyond other clinical and biochemical variables. Therefore, multicenter,

prospective studies with long-term follow-up are needed to corroborate these findings.

CONCLUSION

The findings of this study indicate that the systemic inflammation indices SII and SIRI may provide clinically meaningful information in differentiating cardiovascular from non-cardiovascular causes among patients presenting to the emergency department with chest pain, as well as in risk stratification. The significant correlations observed between these indices and established cardiac and thrombotic biomarkers such as troponin and D-dimer, together with their statistically notable discriminative performance in ROC analysis, suggest that SII and SIRI may serve as complementary tools in the diagnostic process.

Furthermore, the more pronounced inflammatory response observed in high-risk groups such as STEMI, NSTEMI, and PTE reinforces the role of systemic inflammation in the pathophysiology of cardiovascular events. However, considering the limited sensitivity and specificity of these indices, it is essential that SII and SIRI be evaluated not in isolation but within a multidisciplinary framework that incorporates clinical findings, laboratory parameters, and imaging modalities.

In conclusion, inflammation-based indices may offer potential value in early risk stratification and diagnostic guidance for patients presenting with chest pain in the emergency setting. Nevertheless, this potential requires validation through larger-scale, prospective studies to establish the broader applicability and prognostic strength of these biomarkers.

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Abbreviations List

SIRI: Systemic Inflammatory Response Index
SII: Systemic Immune-Inflammation Index
NSTEMI: non–ST-segment elevation myocardial infarction
STEMI: ST-segment elevation myocardial infarction
PTE: pulmonary thromboembolism
ROC: Receiver operating characteristic
AUC: Area under the curve

Ethics Approval and Consent to Participate

All authors declare that the study was conducted in accordance with the Declaration of Hesinki and followed the ethical standards of Türkiye. Approval was granted by the Yozgat Bozok University Non-Interventional Clinical Research Ethics Committee (2025-GO-KAEK-2518_2025.15.10_687).

Consent for Publication

This study was designed as a retrospective review of hospital data; thus, ethics committee approval was obtained, while patient informed consent was not deemed necessary.

Data Availability Statement

The study data are digitally archived in the personal repositories of the authors.

Competing Interests

All authors certify that they have no affiliation with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

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