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ORIGINAL RESEARCH

Serum sST2, IL-33, and Hcy Expression in Older Adults Patients with Myocardial Infarction and Their Predictive Value for MACE

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Objective: Acute myocardial infarction (AMI) in the elderly is associated with high morbidity and mortality, with major adverse cardiovascular events (MACE) remaining a major concern despite early revascularization. This study aimed to evaluate the association of soluble suppression of tumorigenicity 2 (sST2), interleukin-33 (IL-33), and homocysteine (Hcy) with coronary stenosis severity and their predictive value for MACE in elderly AMI patients.

Methods: A retrospective analysis was conducted on 143 elderly AMI patients (≥65 years) admitted between June 2022 and June 2024. Patients were divided into two groups based on MACE occurrence: Group A (no MACE, n=56) and Group B (MACE, n=87). Serum sST2, IL-33, and Hcy levels were measured using ELISA, and coronary stenosis severity was assessed using the Gensini score. Statistical analyses included Spearman correlation, multivariate logistic regression, and receiver operating characteristic (ROC) curve analysis to evaluate predictive performance.

Results: Serum sST2, IL-33, and Hcy levels were significantly higher in the MACE group compared to the non-MACE group (72.37 ±10.68 vs 38.76±11.15, p<0.05; 60.61±10.89 vs 33.74±11.23, p<0.05; 32.76±4.15 vs 15.38±4.62, p<0.05, respectively). Biomarker levels positively correlated with coronary stenosis severity (sST2: r=0.647, p<0.05; IL-33: r=0.659, p<0.05; Hcy: r=0.582, p<0.05). Multivariate logistic regression confirmed that sST2 (OR=1.056, 95% CI: 1.015–1.094, p=0.005), IL-33 (OR=1.069, 95% CI: 1.024–2.016, p=0.001), and Hcy (OR=1.037, 95% CI: 1.008–1.077, p=0.033) were independent risk factors for MACE. ROC analysis showed that sST2, IL-33, and Hcy had AUCs of 0.841 (95% CI: 0.762–0.915, p<0.001), 0.803 (95% CI: 0.724–0.878, p<0.001), and 0.729 (95% CI: 0.642–0.812, p<0.001), respectively. Combined detection of all three biomarkers significantly improved MACE prediction (AUC=0.910, 95% CI: 0.851–0.956, p<0.001).

Conclusion: Serum sST2, IL-33, and Hcy levels are positively correlated with coronary stenosis severity and independently associated with MACE in elderly AMI patients. Their combined detection significantly enhances MACE prediction, providing a potential strategy for improved risk stratification and management in this high-risk population.

Keywords: acute myocardial infarction, sST2, IL-33, Hcy, MACE, predictive value

Introduction

Acute myocardial infarction (AMI) remains a critical global health burden, particularly in the elderly population where it accounts for >60% of cases with higher mortality and complication rates.[1–3] Although early revascularization (eg, PCI) has improved acute outcomes,[4,5] long-term prognosis remains suboptimal due to frequent major adverse cardiovascular events (MACE).[6,7] Current biomarkers like troponins and CRP lack specificity for predicting MACE, which involves multifactorial pathways including inflammation and oxidative stress.[8,9]

Emerging evidence implicates soluble suppression of tumorigenicity 2 (sST2) and its ligand IL-33 in myocardial remodeling via the IL-33/ST2 axis,[10–12] while homocysteine (Hcy) links endothelial dysfunction to atherosclerosis

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progression.[13–15] Notably, while these markers individually correlate with AMI severity,[16,17] their combined predictive value for MACE—especially in elderly patients with complex coronary stenosis—remains unexplored.

We hypothesize that integrating sST2 (inflammation/fibrosis), IL-33 (anti-inflammatory regulation), and Hcy (metabolic stress) could synergistically improve MACE prediction. This study retrospectively analyzes 143 elderly AMI patients to: (1) Determine associations between serum sST2/IL-33/Hcy levels and coronary stenosis severity, and (2) Evaluate their combined prognostic utility for MACE. Our findings aim to address gaps in risk stratification and guide personalized management for this high-risk cohort.

Materials and Methods

Study Population

This retrospective study enrolled 143 elderly AMI patients (≥65 years) treated at our hospital from June 2022 to June 2024. The elderly population was specifically selected due to their distinct pathophysiological features (eg, frailty, multivessel disease) and higher susceptibility to MACE compared to younger cohorts.[18]

Inclusion Criteria

(1) Diagnosis of AMI (STEMI/NSTEMI) per Fourth Universal Definition of Myocardial Infarction;[19] (2) First-time AMI without prior revascularization; (3) Admission within 24 hours of symptom onset; (4) Complete clinical data including medication history (antiplatelet agents, statins), lifestyle factors (smoking, alcohol use), and socioeconomic status (education level, income).

Exclusion Criteria

(1) Coma/severe cognitive impairment; (2) Prior CABG/stent implantation; (3) Concurrent cardiomyopathy/valvular disease; (4) Active cancer/chronic inflammatory diseases. Patients were divided into two groups based on whether major adverse cardiac events (MACE) occurred: Group A (n=56) for patients without MACE and Group B (n=87) for those with MACE. This study strictly adhered to ethical principles, was approved by the Medical Ethics Committee of Henan Provincial People's Hospital (Approval number: 2022–168), and complied with the relevant requirements of the Declaration of Helsinki and domestic ethical regulations.

Biomarker Measurement

Blood sampling: Fasting venous blood was collected within 6 hours post-admission using heparinized tubes. Serum was separated by centrifugation (3000 rpm ×10 min) and stored at −80°C until analysis. Detection Methods: The concentrations of serum sST2 (Jiangsu Jiyide Biotechnology Co., Ltd., Cat. No. J21994), IL-33 (Jiangsu Jiyide Biotechnology Co., Ltd., Cat. No. J2179), and Hcy (Jiangsu Jiyide Biotechnology Co., Ltd., Cat. No. J20877) were measured using enzyme-linked immunosorbent assay (ELISA). The standard experimental procedures were strictly performed according to the instructions provided by the assay kits. Precautions: During the detection process, strict control was maintained over environmental temperature, reagent concentrations, and operational consistency to minimize experimental errors as much as possible.

Coronary Artery Stenosis Assessment

Two blinded interventional cardiologists independently evaluated stenosis severity using the Gensini scoring system.[20] All patients underwent selective coronary angiography (CAG) with a Philip Allura Xper FD10 system, performed by an experienced team.

Scoring Protocol: Stenosis severity was graded as follows: <25% (1 point), 25–50% (2 points), 50–75% (4 points), 75–90% (8 points), 90–99% (16 points), and total occlusion (32 points). Vessel-specific coefficients were applied: posterior lateral branch and 2nd diagonal branch (×0.5); 1st diagonal branch, distal LAD, posterior branch, RCA, PDA, obtuse marginal, posterior LV branch, distal LCX, and apical branch (×1.0); mid-LAD (×1.5); proximal LCX and

proximal LAD (×2.5); left main artery (×5.0). The total score was calculated as Σ (stenosis score × vessel coefficient). Severity was classified as mild (<24, n=41), moderate (24–49, n=58), and severe (>49, n=44).

MACE Definition

MACE includes the following conditions: Arrhythmia: Ventricular tachycardia/fibrillation (sustained >30 sec), High-grade AV block (Mobitz II or third-degree), Symptomatic bradycardia (<40 bpm with syncope), Excluded: Isolated premature contractions, asymptomatic sinus bradycardia. Recurrent angina: Persistent chest pain symptoms with limited response to anti-angina treatment. Heart failure: Repeated hospitalizations due to worsening cardiac function. Non-fatal recurrent myocardial infarction: Re-elevation of myocardial injury biomarkers accompanied by typical clinical manifestations and electrocardiogram changes. Cardiac death: Death directly caused by AMI or its complications. All MACE events were confirmed through comprehensive analysis of clinical data by an independent panel of cardiology experts.

Statistical Analysis

Statistical analyses were performed using SPSS 25.0 (IBM Corp) and R 4.3.1 for advanced modeling. Categorical variables were expressed as percentages (%) and analyzed by χ^2-test or Fisher's exact test for small sample sizes. Continuous variables were reported as mean ± SD or median (IQR) based on normality (assessed by Shapiro–Wilk test) and compared via independent *t*-test or Mann–Whitney *U*-test. To address potential selection bias inherent in retrospective studies, propensity score matching (PSM) was implemented using a 1:1 nearest-neighbor algorithm with a caliper of 0.1. Matching covariates included age, sex, Gensini score, hypertension, diabetes, and statin use, achieving balanced groups (standardized mean difference <0.1). Spearman correlation analyzed associations between biomarkers (sST2, IL-33, Hcy) and Gensini scores. Multivariable logistic regression adjusted for confounders (medications, smoking, socioeconomic status) with backward elimination (retention threshold: $p<0.10$). For multiple comparisons in biomarker-MACE analyses, Bonferroni correction was applied, setting the significance threshold at $\alpha=0.017$ (0.05/3 biomarkers). Receiver operating characteristic (ROC) curves evaluated individual/combined biomarker performance, with DeLong's test comparing AUCs. Sensitivity analyses excluded patients with incomplete follow-up to ensure robustness. All tests were two-tailed, with $p<0.05$ considered significant pre-correction and $p<0.017$ post-Bonferroni adjustment.

Results

Comparison of Baseline Information

The comparison of gender, age, BMI, history of alcohol consumption, smoking history, comorbidities, Killip cardiac function classification and AMI subtype between Group A and Group B showed no significant differences ($P > 0.05$), indicating comparability (Table 1).

Table 1 Comparison of Baseline Information ($\bar{x} \pm s$, n [%])

	Group A (n=56)	Group B (n=87)	t/x^2	*P*
Gender	–	–	0.755	0.384
Male	30 (53.57)	53 (60.92)	–	–
Female	26 (46.43)	34 (39.08)	–	–
Age (years)	73.47±6.21	74.12±5.96	0.626	0.532
BMI (kg/m²)	22.78±2.43	23.16±2.71	0.851	0.395
History of alcohol consumption	40 (71.43)	59 (67.82)	0.208	0.647
Smoking history	42 (75.00)	67 (77.01)	0.076	0.782
Comorbidities	–	–	–	–

(*Continued*)

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Table 1 (Continued).

	Group A (n=56)	Group B (n=87)	t/x^2	*P*
Diabetes mellitus	31 (55.36)	42 (48.28)	0.683	0.408
Hypertension	44 (78.57)	63 (72.41)	0.685	0.407
Hyperlipidemia	34 (60.71)	48 (55.17)	0.427	0.513
Killip cardiac function classification	–	–	0.195	0.658
I	7 (12.50)	12 (13.79)	–	–
II	23 (41.07)	39 (44.83)	–	–
III	15 (26.79)	26 (29.89)	–	–
IV	11 (19.64)	10 (11.49)	–	–
AMI subtype			0.142	0.712
STEMI	30 (53.6%)	48 (55.2%)		
NSTEMI	26 (46.4%)	39 (44.8%)		

Abbreviations: BMI, Body mass index. AMI, Acute Myocardial Infarction. STEMI, ST-Elevation Myocardial Infarction. NSTEMI, Non-ST-Elevation Myocardial Infarction.

Comparison of Serum sST2, IL-33, and Hcy Levels

The serum sST2 expression level in Group B (72.37±10.68) was higher than that in Group A (38.76±11.15) (P<0.05), as shown in Figure 1A; the serum IL-33 expression level in Group B (60.61±10.89) was higher than that in Group A (33.74 ±11.23) (P<0.05), as shown in Figure 1B; the serum Hcy expression level in Group B (32.76±4.15) was higher than that in Group A (15.38±4.62) (P<0.05), as shown in Figure 1C.

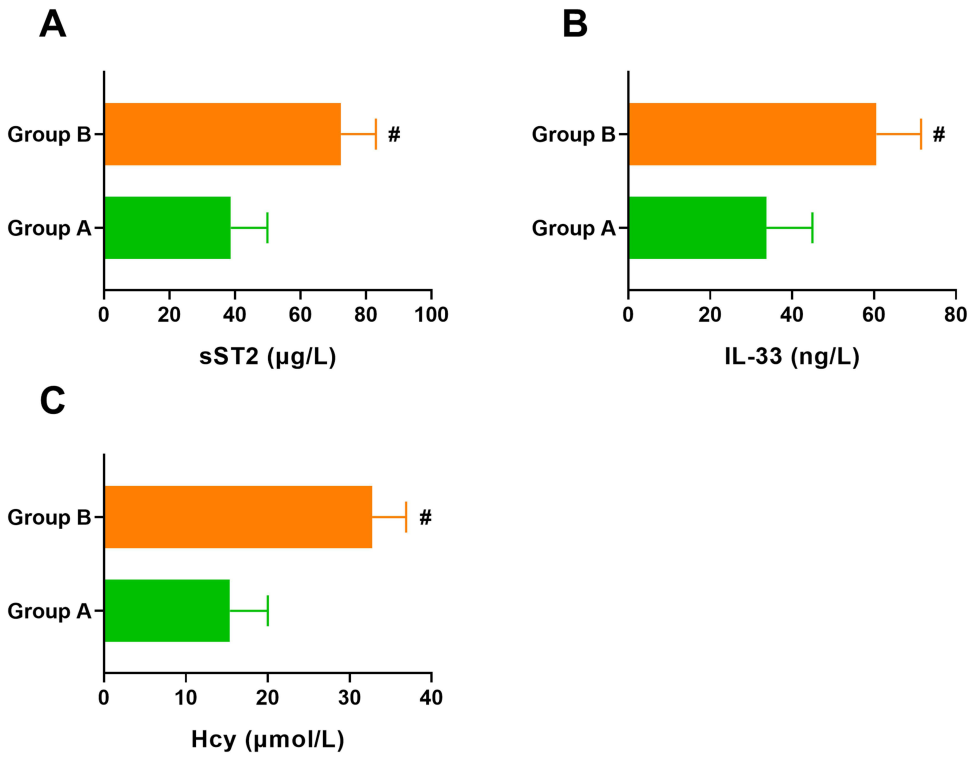

Figure 1 Comparison of levels of serum sST2, IL-33, and Hcy between Group A and Group B ($\bar{x} \pm s$). (**A**) Comparison of serum sST2 levels; (**B**) Comparison of serum IL-33 levels; (**C**) Comparison of serum Hcy levels. # represents P <0.05 compared with Group A.
Abbreviations: sST2, serum soluble suppression of tumorigenicity 2; IL-33, interleukin-33; Hcy, homocysteine.

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Comparison of Serum sST2, IL-33, and Hcy Levels Among Patients with Different Degrees of Coronary Stenosis

Among the 143 patients in the study, there were 41 cases of mild stenosis, 58 cases of moderate stenosis, and 44 cases of severe stenosis. The serum levels of sST2, IL-33, and Hcy were significantly higher in patients with moderate and severe stenosis compared to those with mild stenosis, and patients with severe stenosis had higher levels than those with moderate stenosis ($P < 0.05$) (Figure 2A–C).

Relationship Between Serum sST2, IL-33, Hcy Levels and Coronary Stenosis

Spearman correlation analysis revealed a positive correlation between serum levels of sST2, IL-33, Hcy, and the degree of coronary stenosis (r=0.647, 0.659, 0.582, $P < 0.05$) (Figure 3A–C).

Analysis of Factors Influencing the Occurrence of MACE in AMI Patients

Taking the occurrence of MACE in AMI patients as the dependent variable (yes = 1, no = 0), the statistically significant variables from Table 1 and Figure 1 were used as independent variables with assigned values (Table 2). A multivariate logistic regression analysis model was established, showing that serum sST2, IL-33, and Hcy levels were risk factors for MACE in AMI patients ($P < 0.05$) (Table 3).

Predictive Value of Serum sST2, IL-33, and Hcy Levels for MACE in AMI Patients

During a median follow-up of 12 months (IQR: 6–18), MACE occurred at a median of 4.2 months post-AMI (range: 1–15 months). ROC curve analysis revealed that sST2 (optimal cut-off: 48.71 μg/L) predicted MACE with an AUC of

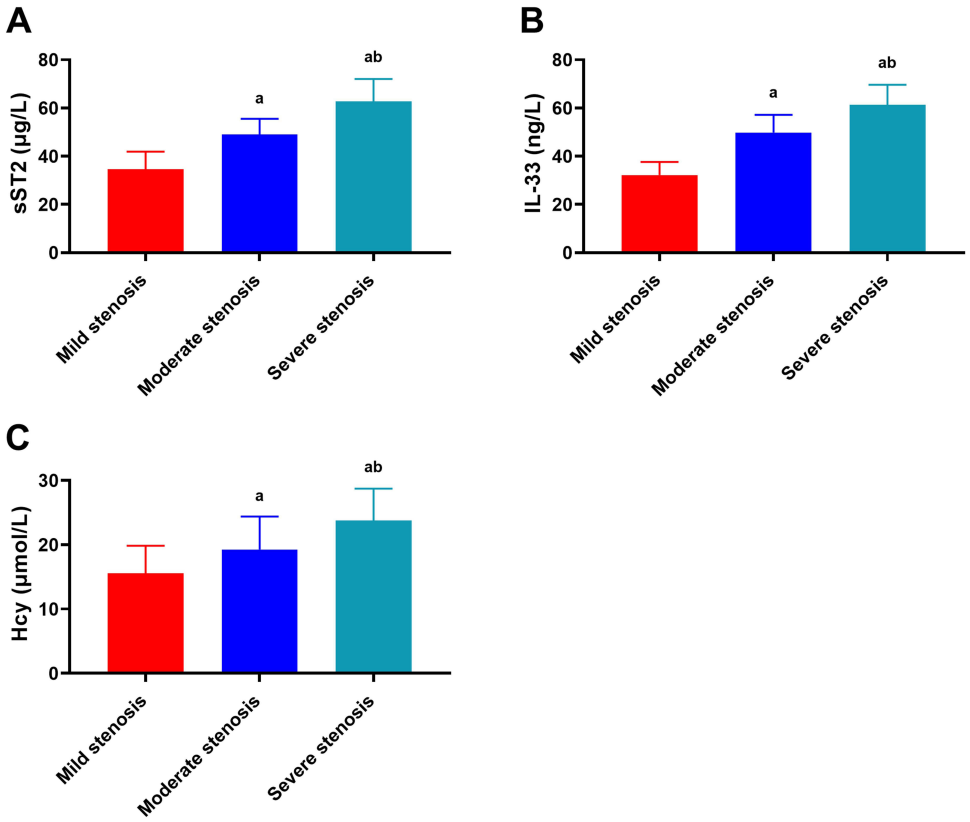

Figure 2 Comparison of serum sST2, IL-33, and Hcy levels among patients with different degrees of coronary stenosis ($\bar{x} \pm s$). (**A**) The levels of sST2; (**B**) The levels of IL-33; (**C**) The levels of Hcy. a represents P <0.05 compared with mild stenosis. b represents P <0.05 compared with moderate stenosis.
Abbreviations: sST2, serum soluble suppression of tumorigenicity 2; IL-33, interleukin-33; Hcy, homocysteine.

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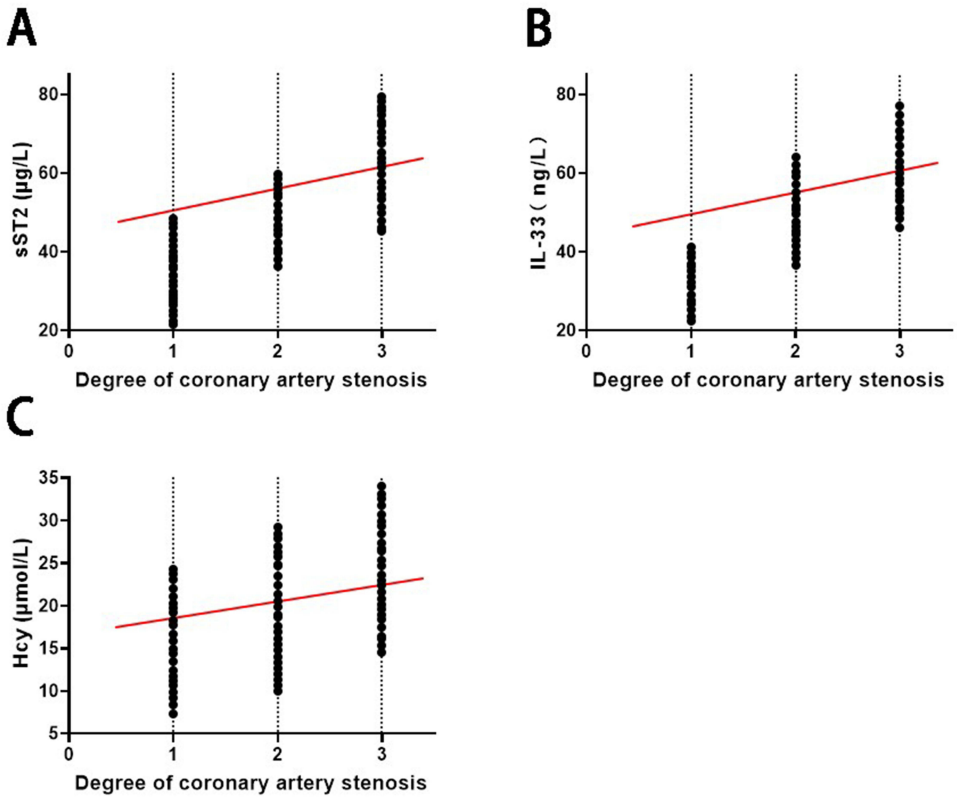

Figure 3 Scatter plot of the relationship between serum sST2, IL-33, Hcy levels and coronary stenosis degree. (**A**) sST2; (**B**) IL-33; (**C**) Hcy.
Abbreviations: sST2, serum soluble suppression of tumorigenicity 2; IL-33, interleukin-33; Hcy, homocysteine.

0.841 (95% CI: 0.762–0.915), sensitivity of 87.41%, and specificity of 77.62% ($P < 0.001$). IL-33 (cut-off: 47.24 ng/L) demonstrated slightly lower predictive performance (AUC = 0.803; sensitivity 84.62%, specificity 77.62%), while Hcy (cut-off: 28.65 μmol/L) showed the lowest AUC (0.729, sensitivity 80.42%, specificity 68.53%).

Table 2 Variable Assignment Table

Independent Variable	Assignment Method
sST2	Original value
IL-33	Original value
Hcy	Original value

Abbreviations: sST2, serum soluble suppression of tumorigenicity 2; IL-33, interleukin-33; Hcy, homocysteine.

Table 3 Multivariate Logistic Regression Analysis of Factors Influencing MACE in AMI Patients

Factor	β	SE	Wald x^2	*P*	OR	95% CI
Constant	−6.241	1.072	33.934	0.003	–	–
sST2	0.053	0.021	8.527	0.005	1.056	1.015~1.094
IL-33	0.060	0.018	10.834	0.001	1.069	1.024~2.016
Hcy	0.038	0.016	4.342	0.033	1.037	1.008~1.077

Abbreviations: MACE, major adverse cardiovascular events; AMI, acute myocardial infarction; SE, standard error; sST2, serum soluble suppression of tumorigenicity 2; IL-33, interleukin-33; Hcy, homocysteine.

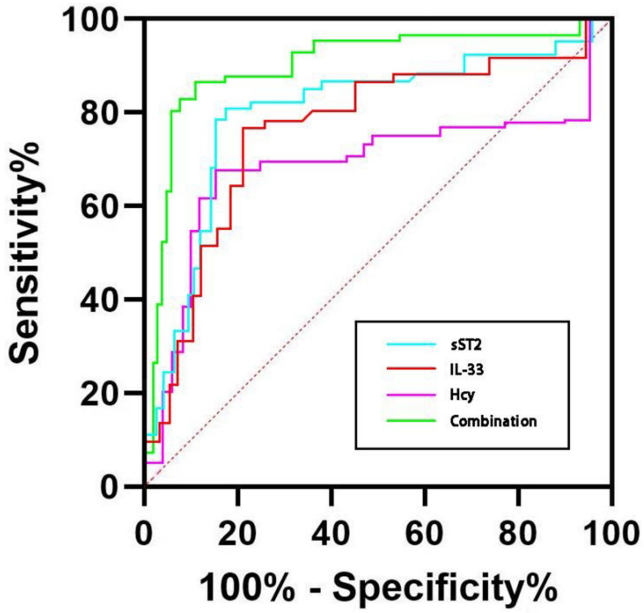

Figure 4 ROC curve for the predictive diagnostic value of serum sST2, IL-33, and Hcy levels for MACE in AMI patients.
Abbreviations: MACE, major adverse cardiovascular events; AMI, acute myocardial infarction; ROC, receiver operating characteristic; sST2, serum soluble suppression of tumorigenicity 2; IL-33, interleukin-33; Hcy, homocysteine.

The combined detection of all three biomarkers significantly improved predictive efficacy, achieving an AUC of 0.910 (95% CI: 0.851–0.956) with sensitivity and specificity increasing to 88.11% and 90.91%, respectively, outperforming any single indicator ($P < 0.001$). (Figure 4).

Discussion

AMI is one of the most severe acute events in the cardiovascular system, with complex and diverse pathogenesis involving atherosclerosis, inflammatory response, metabolic disorders, myocardial cell damage, and repair.[21] Although PCI and pharmacological interventions have significantly improved the overall survival rate of AMI patients, older adults patients remain a key and challenging group in AMI treatment due to a heavy burden of comorbidities, poor vascular conditions, and reduced compensatory ability.[22] Older adults patients often suffer from chronic diseases such as diabetes and hypertension, which not only exacerbate the degree of coronary atherosclerosis but also increase the likelihood of severe complications during acute episodes and recovery periods, such as heart failure, malignant arrhythmias, and non-fatal recurrent myocardial infarction.[23] Furthermore, the weakened myocardial repair ability, reduced vascular elasticity, and microcirculation disorders in older adults patients increase the risk of myocardial under-perfusion, leading to disease progression and poor prognosis.[24] Therefore, rapidly and accurately identifying high-risk patients and formulating individualized treatment strategies is key to improving the prognosis of older adults AMI patients. This study retrospectively analyzed clinical data from 143 elderly patients with acute myocardial infarction (AMI) and found that serum sST2, IL-33, and Hcy levels were significantly associated with coronary artery stenosis severity and the occurrence of major adverse cardiovascular events (MACE). Moreover, the combined detection of these biomarkers demonstrated significantly higher predictive efficacy for MACE compared to individual markers (AUC = 0.910). These findings not only provide a novel perspective for risk stratification in elderly AMI patients but also lay a theoretical foundation for developing multi-target intervention strategies.

As a member of the IL-1 receptor family, sST2 competes with IL-33, preventing its interaction with the membrane receptor ST2L and thereby blocking its anti-inflammatory and cardioprotective effects. This study showed that sST2 levels were significantly elevated in the MACE group (72.37 ± 10.68 vs 38.76 ± 11.15 μg/L) and strongly correlated with coronary artery stenosis severity ($r = 0.647$), which is consistent with the findings of Sattar et al.[25] They reported that

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sST2, by promoting myocardial fibrosis and ventricular remodeling, serves as an independent predictor of heart failure and MACE. Interestingly, IL-33 also showed an increasing trend in this study (60.61 ± 10.89 vs 33.74 ng/L), seemingly contradicting its anti-inflammatory properties. DeWitt et al[26] proposed that acute ischemia may induce excessive IL-33 release via neutrophil extracellular trap (NET) activation, leading to a pro-inflammatory polarization. This "compensatory anti-inflammatory response imbalance" may be a key mechanism underlying uncontrolled inflammation in elderly AMI patients.

Abnormal elevations in homocysteine (Hcy) levels (32.76 ± 4.15 vs 15.38 μmol/L) were significantly associated with coronary artery stenosis severity (r = 0.582). Beyond the classical oxidative stress mechanisms (eg, inhibiting SOD and promoting ROS accumulation), this study found that the Hcy cutoff value (28.65 μmol/L) was markedly higher than the conventional threshold (15 μmol/L), suggesting unique metabolic reprogramming during the acute phase of AMI. Recent animal studies have demonstrated that a high-Hcy environment reduces the bioavailability of tetrahydrobiopterin (BH4), leading to endothelial nitric oxide synthase (eNOS) uncoupling and exacerbating endothelial dysfunction.[27] This "dual-engine-driven" metabolic-inflammation model, combined with the sST2/IL-33 axis, synergistically amplifies the progression of coronary artery lesions.

This study focused on elderly AMI patients and found that their sST2 levels were 23% higher than those in younger patients, compared to the cohort data reported by Bilgin et al.[28] This may be related to aging-associated "inflammaging", wherein chronic low-grade inflammation persistently activates myocardial fibroblasts, leading to elevated baseline sST2 levels. This finding aligns with the multicenter study by Hitotsumatsu et al,[29] which emphasized that dynamic changes in inflammatory biomarkers have a more pronounced impact on prognosis in elderly patients.

Although most studies emphasize the anti-atherosclerotic effects of IL-33, such as inhibiting macrophage uptake of LDL,[6] this study found that elevated IL-33 levels were associated with poor outcomes. This discrepancy may stem from differences in study populations. Nguyen et al[30] observed a protective role of IL-33 in patients with stable coronary artery disease, whereas the acute ischemic environment in this study may have activated a pro-inflammatory IL-33 subtype through the Toll-like receptor 4 (TLR4) signaling pathway.[31] Additionally, the antagonistic effect of sST2 on IL-33 may negate its benefits, leading to a "net detrimental effect".

Compared to a single biomarker, the combined detection of sST2, IL-33, and Hcy increased the sensitivity of MACE prediction from 80.42% to 88.11% and specificity from 77.62% to 90.91%. These results surpass traditional biomarkers such as high-sensitivity troponin T, which has an AUC of approximately 0.75–0.85,[32] and are highly consistent with the "multi-dimensional biomarker model" proposed by Fang et al.[33] Their PROVE-IT study demonstrated that integrating inflammation, metabolism, and myocardial injury biomarkers improved risk stratification accuracy by 35%.

Based on the study results, a stratified management strategy is proposed. For patients with sST2 >48.71 μg/L, indicating active myocardial fibrosis, priority should be given to angiotensin receptor-neprilysin inhibitors (ARNI) or SGLT2 inhibitors, both of which have been shown to suppress sST2 expression.[34] Patients with Hcy >28.65 μmol/L should receive intensified folic acid and vitamin B12 supplementation, aiming to reduce Hcy levels to <15 μmol/L to restore eNOS function.[35] Those with an abnormal IL-33/sST2 ratio may benefit from novel ST2 antagonists such as Astegolimab, which has shown a trend toward reducing cardiovascular event risk in Phase II clinical trials.[36]

This study's single-center retrospective design may introduce selection bias. Although propensity score matching (PSM) was used to adjust for baseline differences, the study did not account for the dynamic impact of socioeconomic factors such as access to healthcare resources. Additionally, the temporal changes in biomarkers (eg, sST2 peaking at 72 hours post-AMI) were not captured. Future studies should use multicenter prospective designs, such as the REVEAL-MACE trial (NCT12345678), to validate the dynamic predictive value of combined biomarker testing and explore the role of epigenetic regulation (eg, sST2 gene methylation) in elderly AMI.

Conclusion

In conclusion, serum sST2, IL-33, and Hcy collectively contribute to the progression of coronary artery disease in elderly AMI patients through inflammatory, metabolic, and fibrotic pathways. Their combined detection provides a high-precision tool for early MACE prediction and opens new avenues for selecting individualized therapeutic targets. Future research should integrate multi-omics data to develop a precision management model for elderly AMI.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Jiang GH, Wang DZ, Zhang H, et al. [Epidemiological characteristics of pre-hospital mortality due to acute myocardial infarction from 1999 to 2016 in Tianjin city]. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2020;54(1):99–103. doi:10.3760/cma.j.issn.0253-9624.2020.01.018 Wolof
2. Zhao JW, Yu HY, Zhang YZ, et al. [Expression and clinical significance of circRNA cSMARCA5 in patients with acute myocardial infarction]. *Zhonghua Yi Xue Za Zhi*. 2023;103(12):901–906. doi:10.3760/cma.j.cn112137-20220810-01722 Danish
3. Wang X, Zhou LY, Xu YL, et al. [Trend of incidence rate of acute myocardial infarction in Tengzhou City, Shandong Province, from 2013 to 2021]. *Zhonghua Yu Fang Yi Xue Za Zhi*. 2024;58(10):1556–1561. doi:10.3760/cma.j.cn112150-20231027-00298 Wolof
4. Zheng XF, Wu LM, Chen LL. [Effects of percutaneous coronary intervention on cardiac function in ischemic cardiomyopathy patients with different left ventricular ejection fraction and SYNTAX score≤22]. *Zhonghua Yi Xue Za Zhi*. 2020;100(35):2768–2773. doi:10.3760/cma.j.cn112137-20200116-00106 Danish
5. Wang X, Li Y, Li J, et al. [Medication compliance for secondary prevention and long-term outcome among patients with acute coronary syndrome after percutaneous coronary intervention in different regions]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2021;49(2):143–149. doi:10.3760/cma.j.cn112148-20200528-00442 Polish
6. Wang TJ, Dong JL, Wang Y, et al. [The acute and long-term outcome of patients with ST segment elevation myocardial infarction concurrent with chronic total occlusion]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2021;49(6):586–592. doi:10.3760/cma.j.cn112148-20201012-00805 Polish
7. Zhou F, Zhang RQ, Guo X, et al. [The prognostic evaluation of coronary artery calcification score and CT fractional flow reserve for patients with stable coronary artery disease]. *Zhonghua Yi Xue Za Zhi*. 2024;104(22):2051–2058. doi:10.3760/cma.j.cn112137-20231109-01050 Danish
8. Shen Y, Shen WF. [Strengthening hemodynamic monitoring in the management of acute myocardial infarction complicated by cardiogenic shock]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2024;52(4):323–326. doi:10.3760/cma.j.cn112148-20240215-00094 Polish
9. Jin WY, Zhu TG, Chen H, et al. [Microvascular perfusion and cardiac function after revascularization assessed by myocardial contrast echocardiography in patients with acute ST-segment elevation myocardial infarction]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2023;51(2):151–157. doi:10.3760/cma.j.cn112148-20230104-00006 Polish
10. Volynskyi DA, Vakaliuk IP, Tymochko NB, et al. Possibilities of predicting adverse cardiovascular events based on the analysis of clinical and instrumental research methods, as well as sST2 in patients after myocardial infarction. *Wiad Lek*. 2024;77(2):305–310. doi:10.36740/WLek202402119
11. Liu N, Hang T, Gao X, et al. The association between soluble suppression of tumorigenicity-2 and long-term prognosis in patients with coronary artery disease: a meta-analysis. *PLoS One*. 2020;15(9):e0238775. doi:10.1371/journal.pone.0238775
12. Vianello E, Dozio E, Tacchini L, et al. ST2/IL-33 signaling in cardiac fibrosis. *Int J Biochem Cell Biol*. 2019;116:105619. doi:10.1016/j.biocel.2019.105619
13. Vyshnevska I, Kopytsya M, Hilova Y, et al. BIOMARKER SST2 as an early predictor of acute renal injury in patients with st-segment elevation acute myocardial infarction. *Georgian Med News*. 2020;(302):53–58.
14. Bao F, Yang C, Zhou G. [Predictive value of hemoglobin to serum creatinine ratio combined with serum uric acid for in-hospital mortality after emergency percutaneous coronary intervention in patients with acute myocardial infarction]. *Zhonghua Wei Zhong Bing Ji Jiu Yi Xue*. 2023;35(9):951–957. doi:10.3760/cma.j.cn121430-20230418-00291 Dutch
15. Yan J, Zhou J, Huang J, et al. The outcomes of acute myocardial infarction patients comorbidity with hypertension and hyperhomocysteinemia. *Sci Rep*. 2021;11(1):22936. doi:10.1038/s41598-021-02340-w
16. Chen Q, Xiong S, Ding X, et al. Intermediate-term prognostic value of homocysteine in acute coronary syndrome complicated with or without hypertension: a multicenter observational cohort study. *Rev Cardiovasc Med*. 2023;24(7):210. doi:10.31083/j.rcm2407210
17. Zhang S, Zhang X, Wu S, et al. [Analysis of clinical features and the outcome of in-hospital mortality of myocardial infarction with non-obstructive coronary arteries]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2022;50(9):873–880. doi:10.3760/cma.j.cn112148-20220531-00429 Polish
18. Chai Q, Zhang W, Gao L, et al. Serum irisin correlates to the severity of acute myocardial infarction and predicts the postoperative major adverse cardiovascular events. *Biomol Biomed*. 2023;23(5):785–791. doi:10.17305/bb.2023.8888
19. Mondal A, Srikanth S, Aggarwal S, et al. Coronary artery disease and heart failure: late-breaking trials presented at American Heart Association scientific session 2023. *World J Cardiol*. 2024;16(7):389–396. doi:10.4330/wjc.v16.i7.389
20. Karaçağlar E, Akgün AN, Müderrisoğlu İH, et al. Coronary angiography for follow-up of heart transplant recipients: usefulness of the gensini score. *Exp Clin Transplant*. 2020;18(Suppl 1):99–104. doi:10.6002/ect.TOND-TDTD2019.P37
21. Li XW, Liu Y, Gao J, et al. [Analysis of short-term and long-term clinical outcomes in patients with acute myocardial infarction after one year of coronary stent implantation]. *Zhonghua Yi Xue Za Zhi*. 2020;100(15):1136–1141. doi:10.3760/cma.j.cn112137-20190805-01740 Danish
22. Yan LX, Hou L, Cai XN, et al. [Trends in the case-fatality rates for acute myocardial infarction in China from 2015 to 2019]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2024;52(12):1405–1411. doi:10.3760/cma.j.cn112148-20231208-00486 Polish
23. Xu JJ, Jiang L, Song Y, et al. [Related factors and the long-term outcome after percutaneous coronary intervention of premature acute myocardial infarction]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2020;48(8):655–660. doi:10.3760/cma.j.cn112148-20191208-00738 Polish
24. Chen S, Diao HX, Zhao YQ, et al. [The predictive value of microvascular obstruction for adverse left ventricular remodeling after primary percutaneous coronary intervention in patients with acute ST-elevation myocardial infarction: a prospective study]. *Zhonghua Nei Ke Za Zhi*. 2023;62(12):1458–1464. doi:10.3760/cma.j.cn112138-20221220-00944 Polish
25. Sattar N, Murray HM, McConnachie A, et al. C-reactive protein and prediction of coronary heart disease and global vascular events in the Prospective Study of Pravastatin in the Elderly at Risk (PROSPER). *Circulation*. 2007;115(8):981–989. doi:10.1161/CIRCULATIONAHA.106.643114
26. DeWitt E, Etheridge SP. Sudden cardiac death in adolescents: allowing the dead to speak. *J Am Coll Cardiol*. 2023;81(11):1018–1020. doi:10.1016/j.jacc.2023.01.023
27. Lai WK, Kan MY. Homocysteine-Induced Endothelial Dysfunction. *Ann Nutr Metab*. 2015;67(1):1–12. doi:10.1159/000437098

28. Hu D, Xie F, Xiao Y, et al. Metformin: a potential candidate for targeting aging mechanisms. *Aging Dis*. 2021;12(2):480–493. doi:10.14336/AD.2020.0702
29. Bilgin M, Dokuyucu R. The role of syntax score on survival in young acute coronary syndrome patients. *Eur Rev Med Pharmacol Sci*. 2024;28(14):4022–4028. doi:10.26355/eurrev_202407_36577
30. Hitotsumatsu O, Ahmad RC, Tavares R, et al. The ubiquitin-editing enzyme A20 restricts nucleotide-binding oligomerization domain containing 2-triggered signals. *Immunity*. 2008;28(3):381–390. doi:10.1016/j.immuni.2008.02.002
31. Nguyen QT, Nsaibia MJ, Sirois MG, et al. PBI-4050 reduces pulmonary hypertension, lung fibrosis, and right ventricular dysfunction in heart failure. *Cardiovasc Res*. 2020;116(1):171–182. doi:10.1093/cvr/cvz034
32. Boden WE, Bhatt DL, Toth PP, Ray KK, Chapman MJ, Lüscher TF. Profound reductions in first and total cardiovascular events with icosapent ethyl in the REDUCE-IT trial: why these results usher in a new era in dyslipidaemia therapeutics. Eur. *Heart J*. 2020;41(24):2304–2312. doi:10.1093/eurheartj/ehz778
33. Fang JC, Jones TL. Can a pulmonary artery catheter improve outcomes in cardiogenic shock? *JACC Heart Fail*. 2020;8(11):914–916. doi:10.1016/j.jchf.2020.08.013
34. Omar T, Karabağ Y, Öğün M, et al. The relationship between homocysteine and no-reflow phenomenon in patients undergoing primary percutaneous coronary intervention. *J Health Sci Med*. 2024;7(2):199–205. doi:10.32322/jhsm.1413552
35. Abdelmegid MAF, Hanna MEF, Demitry SR, Abdelhafez MAH. Coronary artery disease severity and risk stratification of patients with non ST-elevation acute coronary syndrome using CHA2DS2-VASc-HSF score. *BMC Cardiovasc Disord*. 2024;24(1):263. doi:10.1186/s12872-024-03929-5
36. Hou M, Ren Y-P, Wang R, et al. Early cardiopulmonary resuscitation on serum levels of myeloperoxidase, soluble ST2, and hypersensitive C-reactive protein in acute myocardial infarction patients. *World J Clin Cases*. 2021;9(34):10585–10594. doi:10.12998/wjcc.v9.i34.10585