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SII and SIRI Based pre-operative parametric Nomogram for Predicting All-Cause Mortality in Diabetic patients with the first Myocardial Infarction

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Article

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Abstract

Objective：This study sought to develop and validate a nomogram incorporating clinical factors and complete blood count (CBC)-derived inflammatory indices, designed to predict all-cause mortality in diabetic patients following their first acute myocardial infarction and primary percutaneous coronary intervention(pPCI).

Methods: This retrospective multicenter cohort study analyzed patients with concomitant diabetes and myocardial infarction who underwent pPCI, collected from 82 hospitals between 2010 and 2024. Patients were categorized into survival and non-survival groups. Restricted cubic spline (RCS) analysis was employed to examine the relationships between SII, SIRI, AISI, NLPR and mortality. The nomogram was constructed by integrating independent predictors identified through multivariable Cox regression, with the optimal model selected based on minimal AIC value. Subjects were stratified into high- and low-risk groups according to the median nomogram score. The model's performance, clinical utility, and validity were assessed using the C-index, calibration curves, decision curve analysis (DCA), and survival curves.

Results: A total of 3403 eligible patients were included with 1-year all-cause mortality of 4.06%.RCS curves revealed significant non-linear relationships between SII,SIRI,AISI,NLPR and 1-year mortality (*P*-nonlinear < 0.001). Multivariable Cox regression analysis identified 10 independent predictors for 1-year mortality after adjustment, including SII and SIRI. These predictors were incorporated into a nomogram model optimized by minimal AIC value. The model demonstrated excellent discriminative ability (C-index: 0.895) and good calibration (Hosmer-Lemeshow test: 0.6092). Decision curve analysis confirmed clinical utility with net benefit thresholds ranging from 0.01 to 0.78. Patients stratified into high-risk groups based on the median nomogram score showed significantly elevated mortality compared to low-risk groups.

Conclusion: Some biomarkers such as SII and SIRI were identified as independent predictors of 1-year mortality after pPCI in patients with diabetes and first myocardial infarction. An economical and robust nomogram integrating these novel inflammatory markers with conventional risk factors was developed and validated in Tianjin, China, and demonstrated satisfactory performance.

1 Introduction

Diabetes mellitus(DM), a pressing global public health issue, is projected to reach 643 million individuals by 2030 and surge to 783 million by 2045[1]. Diabetes is an established independent risk factor for acute myocardial infarction(AMI) [2].Unfortunately, as a leading cause of death worldwide, AMI is responsible for more than 100,000 deaths each year, constituting 16% of global mortality[3]. Notely, the mortality risk remains 1.5-2-fold higher in diabetic myocardial infarction patients compared to their non-diabetic counterparts[4]. Primary percutaneous coronary intervention (pPCI) is an effective method for reducing adverse cardiac events for AMI patients[5]. Although more aggressive pharmacotherapy and pPCI have considerably reduced

mortality in diabetes patients with AMI in recent years, the 1-year mortality rate remains persistently high[6-8].Therefore, pinpointing reliable prognostic indicators in this high-risk subgroup is essential to refine risk stratification and guide therapeutic interventions accordingly.

In recent decades, a growing body of evidence frames diabetes and its vascular complications as chronic low-grade inflammatory states[9-12].Targeting systemic inflammation presents a potential strategy to reduce mortality by an estimated 10-20% in people with dysglycemia (including DM and PreDM)[13].Although previous research into anti-inflammatory therapies for AMI has yielded preliminary positive outcomes, a significant limitation remains: the inflammatory biomarkers used to evaluate treatment response tend to be prohibitively expensive, methodologically outdated, or clinically inaccurate[14].To bridge this gap, the use of clinically reliable inflammatory biomarkers for systemic inflammation assessment would be valuable in optimizing anti-inflammatory therapy for diabetic patients after myocardial infarction(MI).

Evolving evidence has suggested that the blood cells such as lymphocytes, neutrophils, monocytes, and platelets are involved in systemic inflammation and are associated with the pathogenesis of atheroprogression and plaque Invulnerability in AMI[15].The characterization of inflammation through the ratios of various leukocyte subtypes in the complete blood count has recently found application in studies of diabetes-related cardiovascular diseases(CVD)[16-17].Since then, the systemic immune-inflammation index (SII) serves as a robust and independent prognostic marker for adverse outcomes, aiding in risk stratification among diabetic patients, particularly those with concurrent MI[18-20].Systemic Inflammation Response Index (SIRI) emerges as a pivotal and cost-effective prognostic tool, demonstrating its value both as an independent risk factor for major adverse cardiovascular events(MACE) in Acute Coronary Syndrome(ACS) patients post-PCI and as an accurate predictor of long-term outcomes when measured at the 12-hour mark following the procedure[21-23].Additionally, Aggregate Index of Systemic Inflammation (AISI) has been established as an independent predictor of adverse outcomes in ACS patients undergoing PCI[22-23]. Neutrophil-to-Lymphocyte Platelet Ratio (NLPR) is a novel inflammatory index derived from the NLR that reflects chronic inflammation. It has been shown to be independently and incrementally associated with long-term mortality risk among patients with non-ST-segment elevation myocardial infarction[24].While proven valuable elsewhere, whether these inflammatory markers predict 1-year mortality after pPCI in diabetics with the first MI is unclear.

Furthermore, existing studies often exclude individuals with chronic kidney disease (CKD) or other comorbidities, limiting model generalizability. Additionally, the indicators used are mostly postoperative or intraoperative parameters, reducing their utility for timely identification of high-risk individuals. Given this, our study sought to determine the prognostic role of pre-operative CBC-derived inflammatory markers and other variables in diabetic patients with the first MI treated by pPCI, and to utilize these inexpensive, readily available tests to construct a clinically useful nomogram. This model aims to empower clinicians with a readily calculable

mortality risk score, allowing for early high-risk patient identification and supporting evidence-based risk stratification and clinical decision-making.

2 Materials and methods

Study population and data source

The data for this study were sourced from the Coronary Angiography and Coronary Heart Disease Intervention database of the Tianjin Health and Medical Big Data Super Platform. The Platform collects and aggregates baseline clinical characteristics, diagnosis and treatment data from 82 hospitals in the Tianjin area from January 2010 to March 2024. Additionally, we acquired individuals' mortality data from the Tianjin Center for Disease Prevention and Control (CDC).This study was approved by the Ethics Committee of The Second Hospital of Tianjin Medical University (KY2025k012) and was carried out in accordance with the Declaration of Helsinki. The need for informed consent was waived by the ethics committee owing to the retrospective, observational nature of this study.The inclusion criteria included: aged ≥ 18 years; patients who were diagnosed for diabetes mellitus (E10, E11-14) and acute myocardial infarction (I21.9)for the first time treated with primary PCI. The exclusion criteria were as follows: White blood cell count greater than 20×10^9/L, severe liver disease, severe kidney disease based on the ICD-10 codes (Supplemental Table 1), CKD stage 5 or End-stage renal disease (ESRD)，incomplete blood routine information and missing follow-up records.Patients with a history of prior coronary revascularization.

Variable extraction

The baseline clinical characteristics included age, gender, Body mass index (BMI) and smoking history. BMI was calculated using the standard formula (kg/m²). Smoking status was dichotomized into current smokers (coded as "yes") and non-smokers (coded as "no").Comorbidities were extracted using International Classification of Diseases-Tenth Revision (ICD-10) codes(Supplemental Table 1), which included hypertension, cerebrovascular accident history comprised of ischemic and hemorrhagic stroke, chronic kidney disease (CKD stage1-4) and diabetes target organ damage(DTOD)(including diabetic kidney, diabetic retinopathy, diabetic cataract, diabetic peripheral neuropathy, diabetic peripheral vascular disease, diabetic foot ulcers).The laboratory test indicators measured on the first day of admission included:complete blood count (CBC)-derived inflammatory indices：white blood cell (WBC), neutrophils(NE),lymPhocyte(LYM),monocyte(MO),platelet(PLT),low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), total cholesterol(TC), triglyceride (TG), UREA and creatinine(CR). The discharge medications β-Blockers and statins have also been recorded.The derived inflammatory markers based on the blood routine test are calculated using the following formula.SII = PlateletCount ×NeutrophilCount/LymphocyteCount;

SIRI = NeutrophilCount ×MonocyteCount/LymphocyteCount;AISI = Neutrophil Count×Platelet Count× Monocyte Count/Lymphocyte Count;NLPR=(Neutrophil×100)/(Lymphocyte×Platelet)

Endpoint

The endpoint was 1-year mortality, defined as the all-cause mortality of diabetic patients following their first AMI and primary PCI within 1 year.

Exploration of the correlation between SII,SIRI,AISI,NLPR and all-cause mortality

A comparison was performed between the survival and non-survival groups regarding the following CBC-derived inflammatory indices:SII, SIRI, AISI, and NLPR. Additionally, the dose-response relationships between these four inflammatory markers (treated as continuous variables) and 1-year all-cause mortality were evaluated using restricted cubic spline(RCS). To assess the robustness of the findings, sensitivity analysis was conducted through subgroup analyses based on key clinical characteristics: age (≤65 vs. >65 years), sex, and the presence of STEMI, CKD, diabetes with target organ damage, hypertension, or a history of cerebrovascular accident.Then, p-values were obtained for the interactions.

Model construction and assessment

Independent risk factors for mortality were identified using a stepwise multivariable Cox proportional hazards regression model, with variable selection guided by the Akaike Information Criterion (AIC) and the Concordance Index (C-index) to determine the optimal predictive model. Based on these results, a nomogram was subsequently constructed to visualize the prognostic model.

The receiver operating characteristic (ROC) curves and the area under the curve (AUC) of the full-variable nomogram were compared with those of the model excluding SII and SIRI, and the difference was assessed using DeLong’s test. Calibration was evaluated using the Hosmer-Lemeshow test and calibration curves. Decision curve analysis (DCA) was employed to assess the clinical usefulness and net benefit of the nomogram. Patients were stratified into two groups based on the median nomogram score from the entire cohort as the cutoff. Kaplan-Meier survival analysis was then performed to compare the two groups, thereby validating the predictive accuracy of the nomogram model.

Statistical analysis

SPSS 27.0 and R 4.3.1 are used for data statistics.Student’s t-test or Mann-Whitney test were used to compare continuous variables, which are presented as mean ± standard deviation or Median and quartiles. Categorical variables are presented as frequencies and percentages, and differences between groups were compared using chi-square test. Variables exceeding a 20% missing rate were excluded from analysis. For the remaining variables, incomplete data were handled using multiple imputation techniques. To evaluate the internal validity of the nomogram, we performed bootstrap resampling (n = 1,000) to estimate the area under the ROC curve (AUC). Restricted cubic splines with three knots (placed at the 5th, 35th, 65th, 95th, percentiles) were employed to examine the potential nonlinear associations between the continuous inflammatory indices (SII, SIRI, AISI, and NLPR) and 1-year mortality. Univariate

Cox regression analysis identified factors significantly associated with the endpoint ($p < 0.05$), all of which were incorporated into a multivariate model adjusted for potential confounding variables.Statistical significance was defined as $P < 0.05$ (two-tailed).

3. Results

Patient characteristics

A total of 3,403 eligible diabetic patients with the first AMI who underwent primary PCI were enrolled(Fig. 1).During the 1-year follow-up period, 138 patients died, corresponding to a mortality rate of 4.06%.Table 1 shows the baseline characteristics of the survival group and non-survival group. Survivors and non-survivors demonstrated significant differences in several clinical and laboratory parameters. Compared with the survival group, non-survivors were older and had a higher proportion of female patients. They also exhibited significantly higher rates of cerebrovascular events, diabetic target organ damage, and chronic kidney disease.In terms of laboratory findings, non-survivors showed elevated levels of white blood cells, neutrophils, lymphocytes, and SII, SIRI, AISI, and NLPR. Additionally, they had higher serum creatinine and urea levels.Notably, the use of statins and beta-blockers was significantly lower in non-survivors compared to survivors. All reported differences were statistically significant ($p < 0.05$).

Table 1
Baseline characteristics of participants in survival and non-survival groups.

Variables	Survival N = 3265	Non-survival N = 138	$Z/t/X^2$	P
Age(years)	64(57,70)	71(65.75,75.25)	8.063	< 0.001
Female gender,n(%)	1091(33.4)	60(43.5)	5.990	0.014
BMI(Kg/m^2)	25.25(23.38,27.68)	25.18(22.82,27.60)	-0.646	0.518
Smoking ,n (%)	643(19.7)	22(15.9)	1.185	0.276
STEMI,n (%)	1354(41.5)	60(43.5)	0.220	0.639
Killip_1_level	2123(65.0)	80(58.0)	1.698	0.089
Hypertension,n (%)	2349(71.9)	108(78.3)	2.632	0.105
Cerebrovascular accident history ,n (%)	750(23.0)	49(35.5)	11.582	< 0.001
CKD ,n (%)	150(4.6)	36(26.1)	118.371	< 0.001
Diabetes target organ damage,n (%)	162(5.0)	25(18.1)	44.116	< 0.001
Beta blockers ,n (%)	2587(79.3)	93(67.4)	11.237	< 0.001
Statins,n (%)	3257(99.8)	133(96.4)	39.705	< 0.001
WBC ($10^9/L$)	9.55(7.71,11.80)	10.27(7.80,13.43)	2.228	0.026
LYM ($10^9/L$)	1.64(1.30,1.97)	1.54(1.20,1.79)	-3.075	0.002
MO($10^9/L$)	0.52(0.38,0.68)	0.54(0.41,0.69)	0.711	0.477
PLT($10^9/L$)	216.00(182.00,258.00)	215.50(176.75,257.50)	-0.655	0.512
NE($10^9/L$)	6.45(4.80,8.87)	8.09(5.12,15.17)	4.209	< 0.001
Cr(umol/L)	68.81(56.69,82.70)	84.39(65.63,137.27)	7.411	< 0.001
UA(umol/L)	309.72(257.83,374.17)	339.57(263.42,443.03)	3.604	<

SII=Systemic Immune-Inflammation Index, SIRI=Systemic Inflammation Response Index, AISI=Aggregate Index of Systemic Inflammation, NLPR=Neutrophil-to-Lymphocyte Platelet Ratio

Variables	Survival N = 3265	Non-survival N = 138	Z/t/X^2	P
				0.001
UREA(mmol/L)	6.30(4.90,8.18)	7.63(5.87,12.53)	4.856	< 0.001
TC(mmol/L)	4.49(3.85,5.22)	4.67(3.80,5.53)	1.312	0.189
TG(mmol/L)	1.64(1.17,2.41)	1.55(1.04,2.72)	-0.603	0.546
LDL-C(mmol/L)	2.44(1.88,3.07)	2.70(2.05,3.35)	2.682	0.007
HDL-C(mmol/L)	1.03(0.81,1.24)	1.05(0.81,1.27)	0.393	0.694
SII	872.83(591.93,1180.69)	1345.00(1181.95,1525.25)	12.633	< 0.001
SIRI	2.03(1.22,3.46)	3.05(1.53,5.87)	5.217	< 0.001
AISI	434.88(255.78,692.03)	721.50(541.86,898.19)	9.179	< 0.001
NLPR	1.99(1.2,3.27)	3.00(1.85,6.00)	6.271	< 0.001
Random Plasma Glucose(mmol/L)	10.42±3.98	10.00±3.09	-1.547	0.124
Insulin Therapy,n (%)	2200(67.4)	100(72.5)	1.561	0.211

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SII,SIRI,AISI,NLPR and all-cause mortality

Non-survivors exhibited significantly higher levels of SII,SIRI,AISI and NLPR compared to survivors, as summarized in Table 1 and illustrated in Supplemental Fig. 1 .RCS analysis revealed significant nonlinear associations between these inflammatory markers and mortality risk. The bootstrap-derived confidence band indicated the precision of this nonlinear estimate(Fig. 2). The risk of all-cause mortality in diabetic patients with myocardial infarction undergoing percutaneous coronary intervention increases with elevated levels of SII, SIRI, AISI, and NLPR. Subgroup analysis demonstrated that the positive association between SII and all-cause mortality was consistent across all strata, with a significant dose-response relationship. (Supplemental Fig. 2). Notably, The prognostic effect of SIRI demonstrated significant heterogeneity across subgroups defined by age, sex, STEMI, CKD, diabetic target organ damage and cerebrovascular accident history except Hypertension(all P for interaction < 0.05, Supplemental Fig. 3).SII demonstrated a higher AUC than SIRI, AISI, NLPR(0.817 vs. 0.6309, 0.7303, 0.6574) for diagnosing all-cause mortality.(by DeLong method, area under $\text{curve}_{\text{SII}}$ vs. area under $\text{curve}_{\text{SIRI}}$,z test $= 7.376$, $P < .001$,(Fig. 3).

Selection of clinical variables and construction of a nomogram model

Univariable Cox regression analysis identified that ten variables were significantly associated with all-cause death (Table 2). Multivariate analysis identified that older age (HR = 1.060,95%CI, 1.038 to 1.081, p<.001), elevated Cr (HR = 1.003, 95%CI, 1.002 to 1.004, P<.001), increased LDL-C (HR = 1.192, 95%CI, 1.011 to 1.405, P = 0.037),SII (HR = 1.003, 95%CI, 1.002 to 1.003, P < 0.001) and SIRI (HR = 1.066, 95%CI, 1.027 to 1.107, P < 0.001), CKD (HR = 2.055, 95%CI, 1.223 to 3.451,P=.007), DTOD (HR = 2.526,95%CI, 1.526 to 4.184, p<.001),Killip class<2(HR = 0.293, 95%CI, 0.190 to 0.451,P<.001), use of beta-blockers(HR = 0.579, 95%CI, 0.400 to 0.838,P=.004), and use of statins(HR = 0.071, 95%CI, 0.028 to 0.184,P<.001) were independent predictors for all-cause mortality (Table 2).The one composed of age,Killip_classification DTOB,CKD,Cr,LDL-C,Beta,Tatin,SII and SIRI with AIC of 1880.27 and a concordance index (C-index) of 0.8947 was selected as the best model (Table 3).Based on these ten candidate predictors, a nomogram was developed to estimate the probability of 1-year all-cause mortality (Fig. 4).

Table 2
Univariate and multivariate cox regression analysis results for all-cause death.

Covariates	Univariate		Multivariable	
	HR(95%CI)	*P-value*	HR(95%CI)	*P-value*
Age(years)	1.056(1.077,1.097)	< 0.001	1.060(1.038,1.081)	< 0.001
Female gender,n(%)	1.522(1.087,2.131)	0.014		
BMI(Kg/m^2)	0.985(0.938,1.034)	0.541		
Smoking ,n (%)	0.774(0.491,1.221)	0.270		
STEMI,n (%)	1.088(0.777,1.524)	0.622		
Killip class<2	0.157(0.106,0.233)	< 0.001	0.293(0.190,0.451)	< 0.001
Hypertension,n (%)	1.399(0.934,2.097)	0.104		
stroke ,n (%)	1.825(1.288,2.586)	0.001		
CKD ,n (%)	6.771(4.630,9.903)	< 0.001	2.055(1.223,3.451)	0.007
Diabetes target organ damage ,n (%)	4.012(2.601,6.187)	< 0.001	2.526(1.526,4.184)	< 0.001
Beta blockers ,n (%)	0.545(0.382,0.777)	< 0.001	0.579(0.400,0.838)	0.004
Statins,n (%)	0.067(0.027,0.164)	< 0.001	0.071(0.028,0.184)	< 0.001
WBC ($10^{9}/L$)	1.044(1.016,1.072)	0.002		
LYM ($10^{9}/L$)	0.660(0.480,0.908)	0.011		
MO($10^{9}/L$)	0.992(0.468,2.103)	0.981		
PLT($10^{9}/L$)	1.000(0.997,1.003)	0.883		
NE($10^{9}/L$)	1.004(1.003,1.005)	< 0.001		
Cr(umol/L)	1.004(1.003,1.004)	< 0.001	1.003(1.002,1.004)	< 0.001
UA(umol/L)	1.004(1.003,1.005)	< 0.001		

SII=Systemic Immune-Inflammation Index, SIRI=Systemic Inflammation Response Index, AISI=Aggregate Index of Systemic Inflammation, NLPR=Neutrophil-to-Lymphocyte Platelet Ratio

Covariates	Univariate		Multivariable	
	HR(95%CI)	*P-value*	HR(95%CI)	*P-value*
UREA(mmol/L)	1.044(1.025,1.063)	< 0.001		
TC(mmol/L)	1.085(0.957,1.230)	0.203		
TG(mmol/L)	1.056(0.957,1.165)	0.280		
LDL-C(mmol/L)	1.227(1.041,1.447)	0.015	1.192(1.011,1.405)	0.037
HDL-C(mmol/L)	1.223(0.810,1.847)	0.339		
SII	1.002(1.002,1.003)	< 0.001	1.003(1.002,1.003)	< 0.001
SIRI	1.022(1.017,1.028)	< 0.001	1.066(1.027,1.107)	< 0.001
AISI	1.002(1.001,1.002)	< 0.001		
NLPR	1.099(1.078,1.121)	< 0.001		
SII=Systemic Immune-Inflammation Index, SIRI=Systemic Inflammation Response Index, AISI=Aggregate Index of Systemic Inflammation, NLPR=Neutrophil-to-Lymphocyte Platelet Ratio				

Table 3
Model selection and comparison with minimum AIC

Model	AIC	C-index	AUC	Delong
AGE+DTOD + CKD+Cr+killip-class+ Beta+Tatin + LDL	1998.27	0.8342	0.839(0.807−.871)	Z=−6.528,P < 0.001
AGE+DTOD + CKD+Cr+killip-class+Beta+Tatin + LDL-C+SIRI	1987.67	0.8366		
AGE + TangB + CKD + Cr + KILLIP_level + Beta + Tatin + LDL + SII	1885.52	0.8949		
AGE+DTOD + CKD+Cr+killip-class+Beta+Tatin + LDL-C + SII+SIRI	1880.27	0.8947	0.901(0.878–0.923)	

Model selection with stepwise approach and AIC, then comparing C-index between the model of

baseline charicteristics and baseline charicteristics + SII+SIRI. DTOB,diabetes target organ damage;CKD,chronic kidney disease;Cr,creatinine;LDL-C,low density lipoprotein

cholesterol;SII,systemic immune-inflammation index;SIRI,Systemic Inflammation Response Index; AIC, Akaike information criterion; CI, confidence interval; C-index, concordance index.

Validation of the all-cause mortality nomogram

The nomogram incorporating SII and SIRI demonstrated a significantly higher AUC compared to the model without these indices (0.901 vs. 0.839), with the difference being statistically significant (DeLong's test, Z = -6.528, $P < 0.001$)(Fig. 5,Table 3). The optimal threshold was identified as 103.9, yielding a sensitivity of 0.865 and a specificity of 0.776. The average AUC generated by bootstrap resampling was 0.900. Furthermore, calibration curve analysis showed good overall agreement between observed and predicted outcomes, despite a slight deviation from the ideal reference line. (Fig. 6).The decision curve showed that using the nomogram provided a net benefit for threshold probabilities between 1% and 78%, supporting its potential for clinical use. (Fig. 7).

Risk stratification based on the nomogram

To evaluate the effectiveness of stratifying the study patients into the two proposed risk groups using the nomogram, we calculated the total points for each individual and classified them into low- and high-risk groups based on the median nomogram score (85.86).Subsequent Kaplan-Meier survival analysis demonstrated that patients in the high-risk group had significantly shorter survival times compared to those in the low-risk group, thereby validating the rationality of the nomogram model .($P < 0.0001$, Fig. 8)

4. Discussion

Diabetes may increase the risk of adverse outcomes after MI, potentially through dysregulated inflammatory processes such as leukocyte and platelet activation, myelopoiesis, and hyperglycaemia-induced trained immunity[12]. Chronic inflammation, which drives atherosclerosis, induces insulin resistance, and triggers endothelial dysfunction, critically underpins the pathophysiology of both metabolic dysfunction and CVD[25]. Given that atherosclerosis forms the pathological foundation of ACS[16], and considering the significant co-prevalence and synergistic worsening between diabetes and AMI, inflammation is recognized as pivotal to the prognosis of this comorbid state[10, 17, 26].Although the impact of inflammatory responses on post-AMI outcomes is well-established, the utility of SII and SIRI for risk stratification remains unclear in the high-risk subgroup of diabetic patients presenting with the first AMI treated with pPCI.

To investigate this uncertainty, we conducted a study to evaluate the prognostic value of SII and SIRI for 1-year all-cause mortality in this specific population and developed a practical nomogram incorporating these indices. Each of our key findings merits further elaboration. First, both SII and SIRI were identified as independent predictors of mortality, exhibiting a pronounced non-linear relationship with risk as delineated by RCS analysis. Second, SII

demonstrated superior discriminative ability compared to SIRI for predicting mortality. Furthermore, the positive association between SII and mortality was consistent across all examined subgroups, whereas the association for SIRI showed heterogeneity in most. Third, the integration of SII and SIRI with baseline clinical variables significantly improved predictive performance, enabling the development of a robust nomogram. This model incorporates ten key predictors: two inflammatory indices (SII and SIRI), age, Killip class, diabetic target organ damage (DTOD), CKD, creatinine level, LDL-C level, and the use of beta-blockers and statins. It demonstrated strong discriminative ability (AUC 0.901, 95% CI 0.796–0.851). Although calibration showed modest deviation from ideal, its clinical utility was further supported by DCA, which indicated a consistent net benefit across a range of probability thresholds.These results offer a morepractical, economical, and reliable method for evaluating the long-term outcomes of diabetic patients with AMI, while also informing the design of personalized post-discharge management strategies in clinical practice.

Emerging evidence highlights the SII and SIRI as integrative biomarkers of systemic inflammation with significant prognostic value in CVDs[27–29]. The SII has been established as a reliable predictor of inflammation, coagulation risk, and adverse outcomes (including mortality) in patients with AMI, and may demonstrate superior predictive performance compared to conventional risk factors[30–31]. Similarly, the SIRI, derived from neutrophil, monocyte, and lymphocyte counts, serves as an independent risk factor for MACE and disease severity in patients with ACS or AMI, offering a predictive advantage over traditional two-parameter indices[32–33].

Our analysis within a diabetic cohort presenting with first AMI confirms and extends these findings. Critically, both SII and SIRI remained independent predictors of all-cause mortality after comprehensive multivariable adjustment. However, a direct comparative evaluation revealed that SII demonstrated superior predictive performance to SIRI, evidenced by a higher AUC for mortality. To the best of our knowledge, this is the first study to validate the superior predictive value of the SII over the SIRI for 1-year mortality in diabetic patients following pPCI for the first time MI. In contrast to our findings, Liu et al. reported in a single-center study with a relatively small sample size that the AUC values for SIRI (0.764) and AISI (0.738) at 12 hours post-PCI were higher than that for SII (0.678) in predicting long-term adverse outcomes in STEMI patients[22]. It is important to note that their study population did not specifically consist of diabetic patients with MI, and the assessment time points differ (12 hours post-PCI vs. pre-procedure in our study).Xu et al. reported that the SII demonstrated a stronger association with total cardiovascular disease in diabetic patients, suggesting that their metabolic profile may differ from that of the general population[34].The potential mechanism underlying SII's enhanced predictive capacity may lie in its unique composition, specifically through the inclusion of platelet count. Platelets are active participants in both thrombosis and inflammation —a synergistic process often termed "thrombo-inflammation," which is central to the pathogenesis of atherothrombotic events like AMI. By integrating signals from the cellular immune response (via neutrophils and lymphocytes) and the pro-thrombotic state (via platelets),

SII may provide a more holistic capture of the pathophysiological interplay driving adverse outcomes, particularly in high-risk populations such as those with diabetes.

In the future, we also plan to conduct dynamic monitoring of these inflammatory indices to further analyze their optimal predictive value at different time points.Notably, the association between elevated SII and higher mortality risk was robust and consistent across all patient subgroups, whereas the prognostic value of SIRI showed less consistency across populations, further supporting the superior predictive ability of SII. Our RCS analysis revealed nonlinear associations of both SII and SIRI with all-cause mortality. This pattern is consistent with observations from previous longitudinal cohort studies[17, 35].Ultimately, the inclusion of both inflammatory markers provided a model with superior fit and incremental predictive value over a baseline model containing only traditional risk factors.In summary, our findings highlight their potential utility in refining risk stratification strategies for patients with diabetes who experienced a first MI.

The AISI (also termed SIIRI or PIV), calculated from neutrophil, monocyte, platelet, and lymphocyte counts, is recognized as a comprehensive marker of inflammatory-thrombotic interplay and an independent predictor of adverse outcomes in coronary artery disease[36–37]. Similarly, the NLPR, which combines NLR with platelet count, has emerged as a stable independent predictor of long-term mortality in NSTEMI or ACS patients following PCI cohorts[38]. In our cohort, however, both AISI and NLPR demonstrated significant univariate associations with 1-year mortality but failed to retain independent prognostic significance following multivariable adjustment. In terms of discriminative ability, AISI was inferior to SII but superior to SIRI, while NLPR performed worse than SII and similarly to SIRI. The diminished predictive power of AISI in our study may be methodologically explained by the very early timing of blood sampling post-MI, which likely preceded the full development of reactive thrombocytosis and the later peak of severe inflammatory-platelet response typically seen with larger infarcts[39]. Xu et al. demonstrated that associations of neutrophil or lymphocyte counts with cardiovascular events persisted even after stratifying participants by blood lipid levels; however, no significant association was observed between monocyte counts and any cardiovascular events. Nevertheless, monocytes contribute to the progression of atherosclerosis by producing inflammatory cytokines in the circulation and differentiating into long-lived macrophages[34].These findings highlight that while composite indices capture broader inflammatory pathways, their prognostic utility is context-dependent and may be significantly influenced by the clinical timing of assessment and different cohort.

Collectively, the evidence linking inflammation to diabetes and its atherosclerotic cardiovascular disease (ASCVD) is now compelling and clinically actionable..The time for taking action has now arrived.Emphasizing anti-inflammatory therapy as an independent pillar of management, the 2025 ACC Scientific Statement recommends routine inflammation assessment for prevention and treatment of ASCVD. It advocates for targeted anti-inflammatory strategies in patients with residual inflammatory risk, independent of LDL cholesterol levels, representing a pivotal evolution in clinical guidelines[11].Our understanding remains limited, as inflammation is likely only one component of the death mechanisms post-MI in diabetes, and the potential

differences in the patterns of fatal manifestations compared to non-diabetic individuals have not been specifically investigated. This underscores a substantial need for further study.

As expected, well-established prognostic factors such as advanced age[16, 40–43] and higher Killip[22, 44] class were confirmed as significant determinants of mortality in our cohort and were incorporated into the nomogram. Notably, this study innovatively incorporates diabetic target organ damage (DTOD) as a categorical variable into a multivariable prediction model, establishing it as an independent risk factor for post-infarction mortality. Its prognostic utility stems from its role as a composite marker, which not only reflects the impact of diabetes duration, treatment response, and microvascular dysfunction but, more importantly, integrates the systemic pathophysiological disturbances in diabetic patients. These include the severity of macrovascular disease, chronic inflammatory states, autonomic dysfunction, and multi-system interactions, thereby enabling a more comprehensive identification of high-risk phenotypes.It is important to acknowledge that while our assessment of diabetes status was advanced by the inclusion of DTOD, it remained constrained by the limited availability of key clinical data. Detailed metrics of glycemic control (HbA1c levels), specific glucose-lowering therapies (particularly newer agents such as SGLT2 inhibitors), and other potential influencing factors like treatment duration were not fully accounted for due to data scarcity. This omission underscores the substantial clinical heterogeneity within the diabetic population and highlights a critical direction for future research: to refine risk stratification by building upon the robust foundation of DTOD through the integration of more granular data on treatment specifics and metabolic control.

The model also reinforces the critical importance of renal function in risk stratification. Elevated creatinine levels and the presence of CKD were both independently associated with increased mortality, aligning with their consistent inclusion in major risk scores such as GRACE, CADILLAC and New York State Risk Scores [45–48]. This underscores the necessity of renal assessment in this patient population.A direct, head-to-head comparison with established scores like GRACE was not feasible in this study due to extensive missing data for key GRACE components (e.g., heart rate and blood pressure) in our retrospective registry. Nonetheless, the discriminative ability of our model (AUC 0.901) appears numerically favorable compared to the typical performance range of the GRACE score (AUC 0.75–0.80)in ACS populations [49], though such indirect comparison must be interpreted with caution.Regarding metabolic and inflammatory pathways, the nomogram includes LDL-cholesterol, reflecting its established role in cardiovascular risk[50–51, 16]. Furthermore, statin therapy, with its dual lipid-lowering and anti-inflammatory properties[52], was retained in the model.

Limitations

This study has several limitations. First, its retrospective, cross-sectional, and observational design precludes the establishment of causal relationships. Second, while multicenter, all participating hospitals were within Tianjin, and the limited sample size may constrain the generalizability of our findings to other geographic and demographic populations. Third, substantial missing data were noted for several key variables (e.g., intraprocedural PCI details,

left ventricular ejection fraction, and specific inflammatory markers like high-sensitivity C-reactive protein), a common challenge in retrospective multicenter studies involving varied clinical practice patterns. Fourth, we did not perform a comparative analysis between patients with and without diabetes, which limits insight into diabetes-specific effects. Fifth, the proposed models require external validation in an independent cohort, which was not feasible here due to the limited number of available cases. Consequently, the predictive performance observed here should be considered preliminary, and prospective validation is warranted. Future work should prioritize enhancing data completeness, conducting the aforementioned diabetes-focused analysis, and developing robust predictive models that integrate the GRACE score with the inflammatory indices evaluated here.

Conclusion

In conclusion, this study identifies SII and SIRI as robust and independent risk factors for all-cause mortality in diabetic patients following their first MI after pPCI. Their addition to conventional clinical parameters provides incremental predictive value, underscoring their utility as superior inflammatory markers. Based on these findings, a simple-to-use prognostic nomogram incorporating SII and SIRI was developed and validated to predict 1-year all-cause mortality in this population. The model demonstrated excellent discriminative ability and was well-calibrated, offering a practical and personalized tool for risk stratification. This nomogram may assist clinicians in identifying high-risk individuals and tailoring follow-up interventions to improve outcomes.

Declarations

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Author contributions

Study conception and design:Zhi-Qin Fang and Kang-Yin Chen; data collection: Shao-Qiang Qin, Tian-Shu Gu;analysis and interpretation of results: Zhi-Qin Fang, Su-Tao Hu, draft manuscript preparation:Zhi-Qin Fang, Yu-Kun Zhang,Xian Shao, Kang-Yin Chen, Tong-Liu. All authors reviewed the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Informed consent

Given the retrospective design of the study, the requirement for informed consent was waived by the Institutional Review Board at the Second Hospital of Tianjin Medical University.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Figures

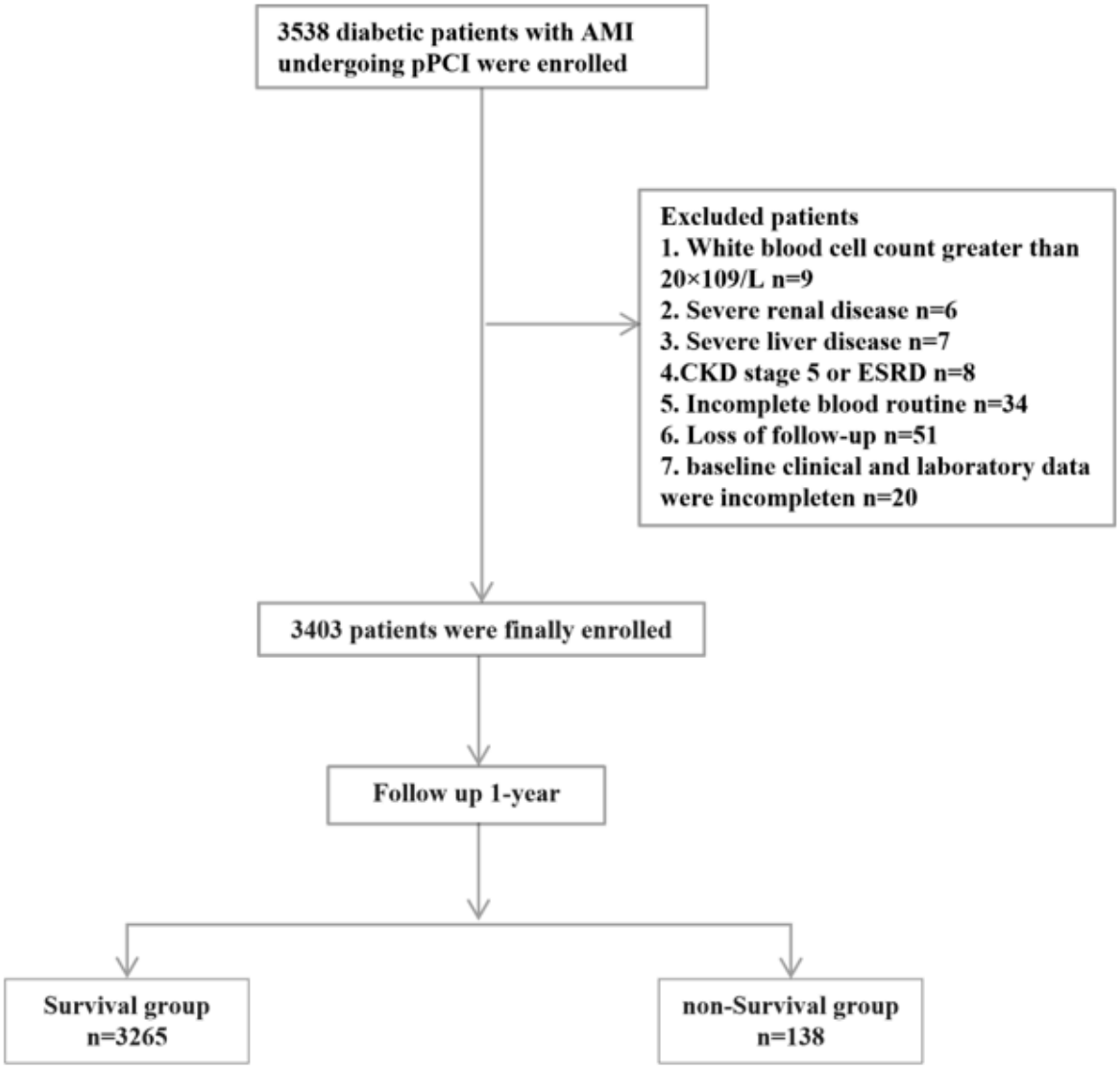


Figure 1

Participant screening flowchart. AMI, acute myocardial infarction□pPCI,primary Percutaneous Coronary Intervention;CKD,chronic kidney disease; ESRD,End-Stage Renal Disease.

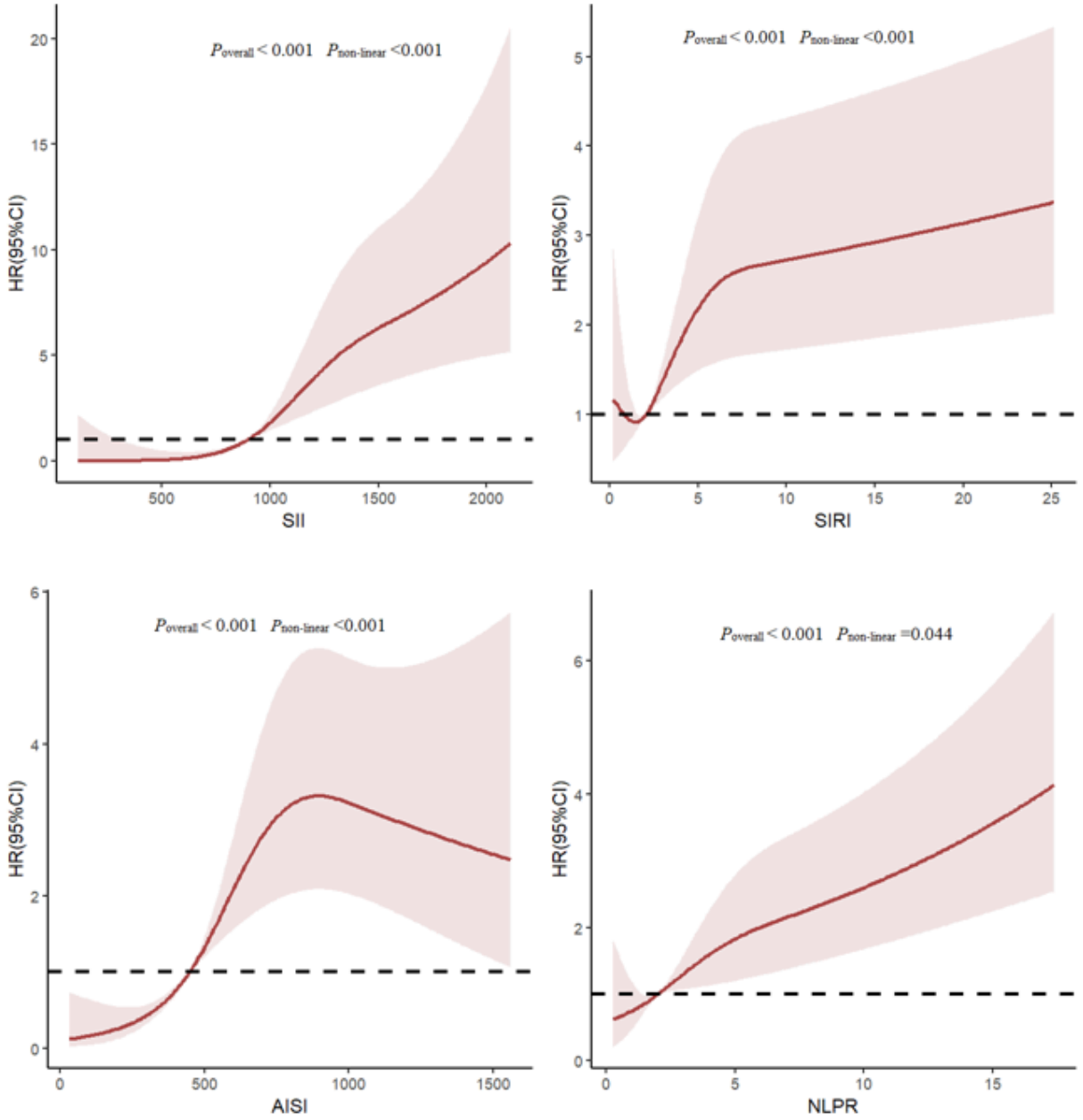


Figure 2

Dose–response relationships between all-cause mortality and SII、SIRI、AISI、NLPR. HRs (solid lines) and 95% confidence levels (shaded areas) were adjusted for age (as a continuous variable), Diabetes target organ damage(DTOD), chronic kidney disease (CKD), creatine(as a continuous variable), LDL-C(as a continuous variable). SII=Systemic Immune-Inflammation Index, SIRI=Systemic Inflammation Response Index, AISI=Aggregate Index of Systemic Inflammation, NLPR=Neutrophil-to-Lymphocyte Platelet Ratio.

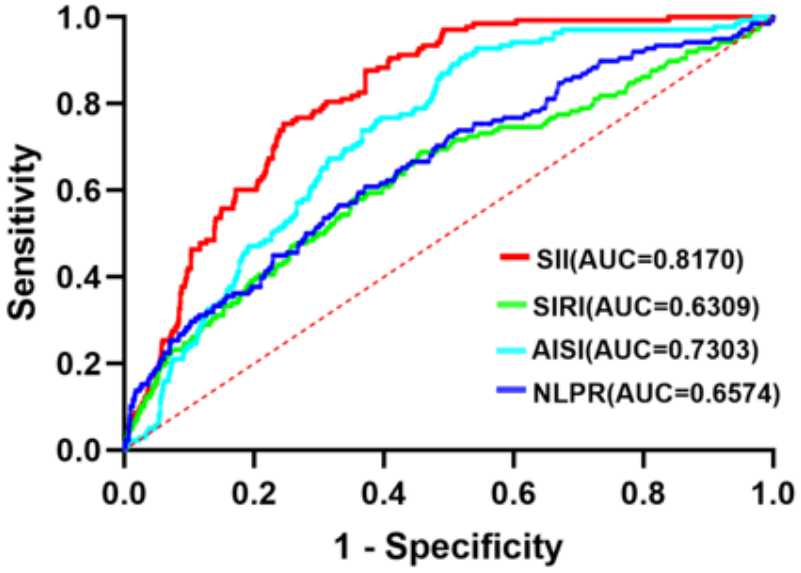


Figure 3

ROC Curves of the SII, SIRI, AISI and NLPR for predicting 1-year mortality

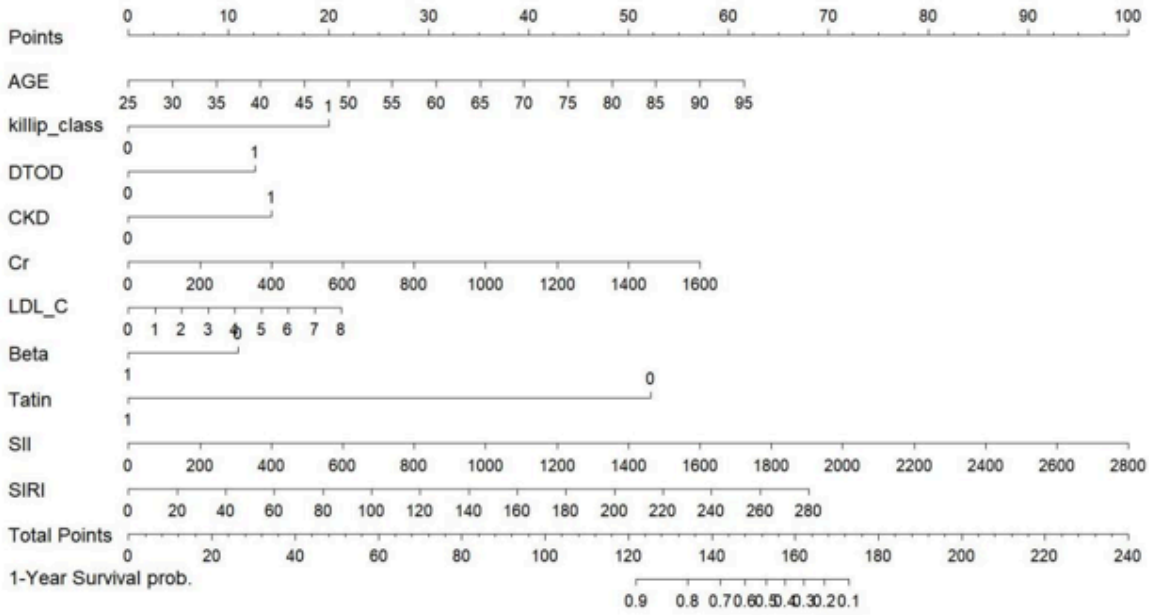


Figure 4

Nomogram based on age, killip class≥2, Diabetes target organ damage(DTOD), chronic kidney disease(CKD) stage 1-4, creatinine(Cr), Low-Density Lipoprotein Cholesterol(LDL-C), β-blocker, tatin,SII and SIRI in prediction for 1-year survival probability in diabetic patients with AMI after pPCI.

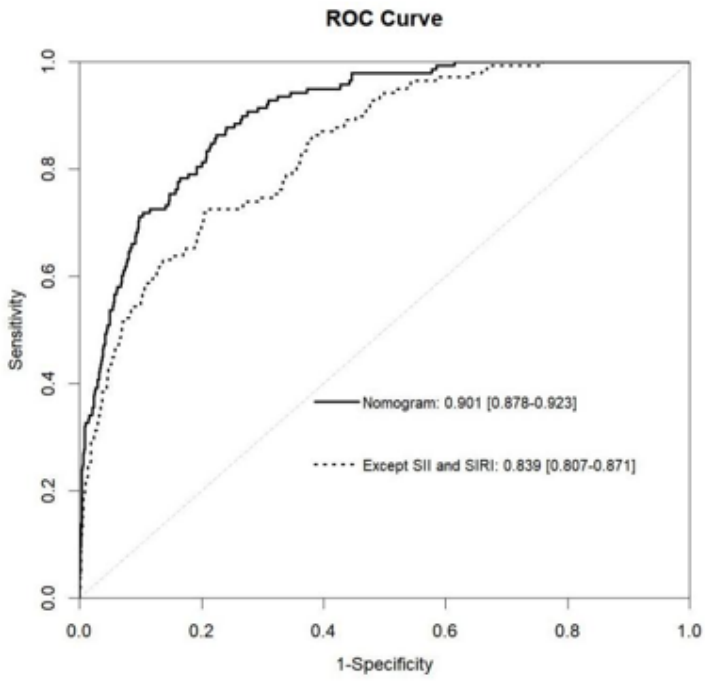


Figure 5

ROC curves of Nomogram models with versus without SII and SIRI for predicting 1-Year All-Cause Mortality.

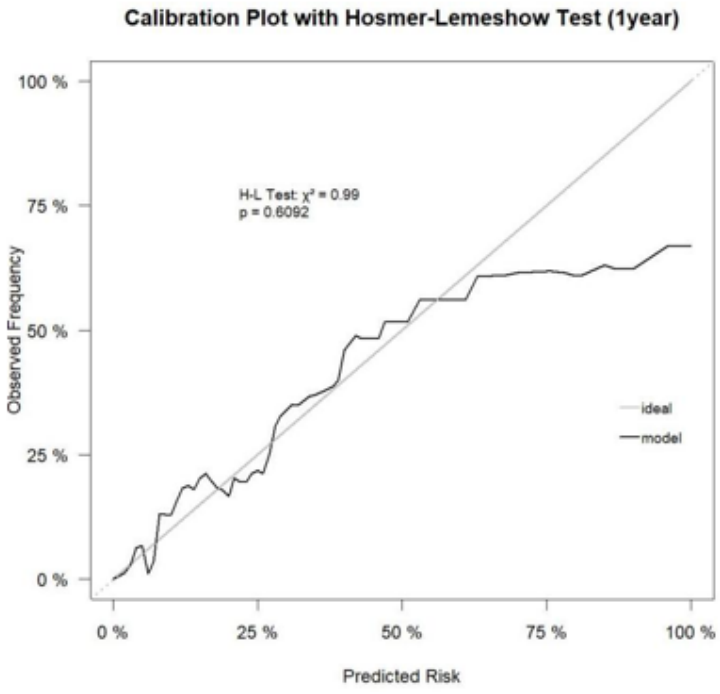


Figure 6

The calibration curve for predicting all-cause death probability. The x-axis was the survival rate by the nomogram, and the Y-axis was the actual survival rate. The gray line represents the predicting survival rate completely fits the actual survival rate by an ideal model. The black line

shows that the nomogram's predictions are close to the actual results, indicating good agreement of the calibration curve.

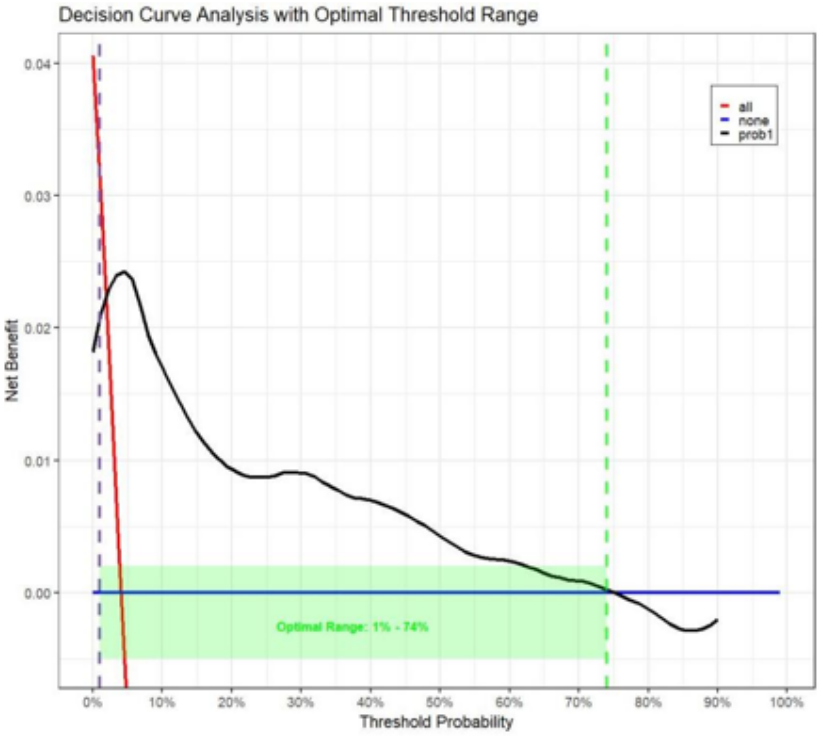


Figure 7

The DCA shows the clinical usefulness of the 1-year all-cause death nomogram. This DCA could provide a larger net benefit than “all ” and “none” with ranges of 1% to 78%. DCA: decision curve analysis .

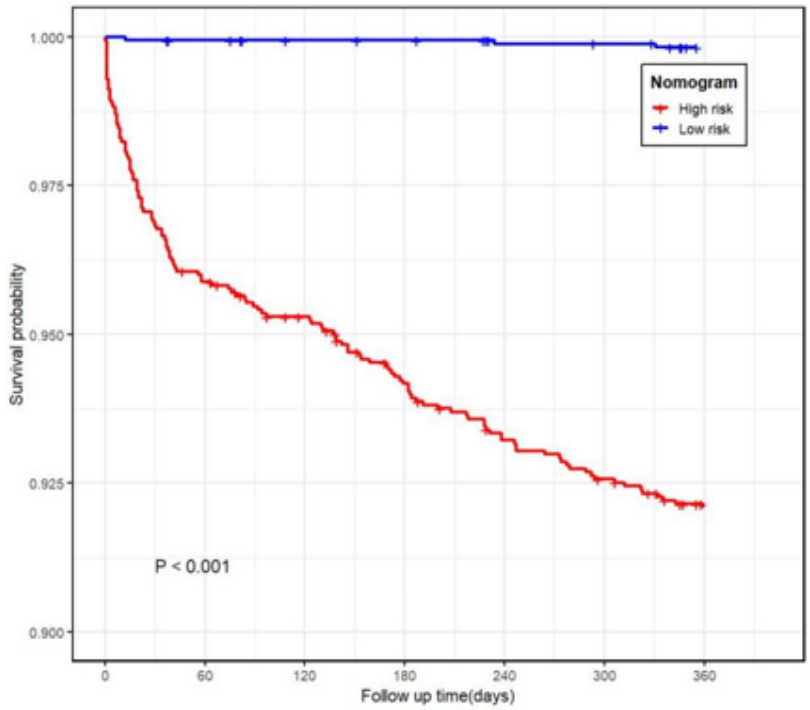


Figure 8

Kaplan–Meier curves for all-cause mortality according to median of nomo score 85.86. High risk:nomo score 85.86low risk:nomo score 85.86.

Supplementary Files

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