

Development and Validation of a Prognostic Model for One-year Mortality in Older Adults with Acute Myocardial Infarction Incorporating Activities of Daily Living Impairment

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Purpose: Functional impairment, measured by Activities of Daily Living (ADL), is a major predictor of poor outcomes in older adults. However, among patients with acute myocardial infarction (AMI), a prognostic model that incorporates ADL status is lacking. This study aimed to develop and validate such a model for predicting one-year mortality in older adults with AMI.

Patients and Methods: This study included 43,308 AMI patients admitted to the Tianjin Health and Medical Big Data Superplatform from January 2010 to March 2024. Patients were categorized by Barthel Index into ADL normal (n=41,312), mildly impaired ADL (n=1,318), moderately impaired ADL (n=452), and severely impaired ADL (n=226). The cohort was randomly split 7:3 for model development and validation. A nomogram for 1-year all-cause mortality was constructed using predictors identified by Lasso regression, with model performance evaluated through receiver operating characteristic curve (ROC), decision curve analysis (DCA), and calibration curve.

Results: Baseline characteristics revealed that with increasing ADL impairment, patients were significantly older [median (IQR): 73·0 (68·0-78·0) vs 78·0 (73·0-83·0) vs 79·0 (73·0-84·0) vs 78·0 (73·0-83·0), $p < 0·001$], had higher Killip class, and received less percutaneous coronary intervention (PCI) (48·9% vs 25·0% vs 17·3% vs 8·9%, $p < 0·001$). Kaplan-Meier analysis showed significantly increased mortality risks ($p < 0·001$). The final model incorporated seven predictors: ADL status, age, Killip class, ST-elevation myocardial infarction (STEMI) diagnosis, history of stroke, valvular heart disease, and diabetes mellitus. The nomogram achieved area under the curve (AUC) values of 0·794 and 0·780 in the training and validation cohorts, respectively.

Conclusion: We developed and validated a clinically applicable nomogram for predicting one-year mortality in older adults with AMI, integrating ADL impairment into risk assessment. Incorporating functional evaluation into prognostic modeling may improve individualized management and early rehabilitation planning in geriatric cardiology.

Keywords: acute myocardial infarction, activities of daily living, prognosis, nomogram predictive model

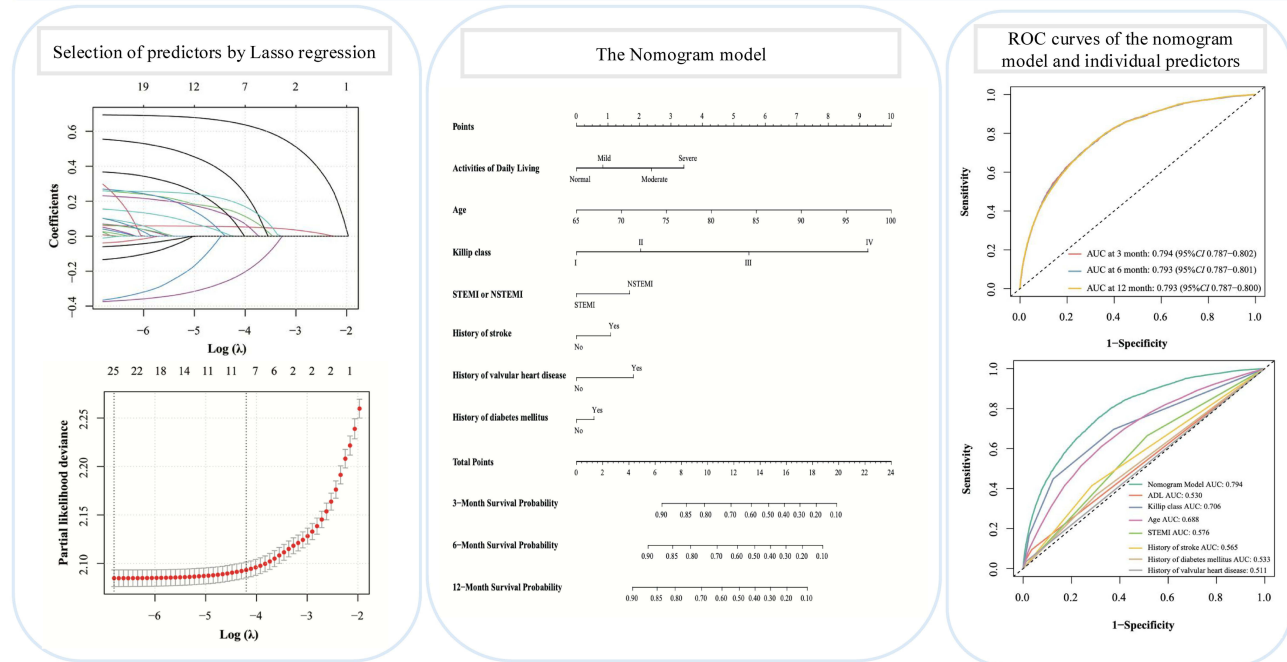
Introduction

Acute myocardial infarction (AMI) remains a major contributor to morbidity and mortality among older adults. According to the 2019 Global Burden of Disease study, the incidence of non-ST-elevation myocardial infarction (NSTEMI) is almost twice that of ST-elevation myocardial infarction (STEMI).¹ Although timely percutaneous coronary intervention (PCI) has well-established benefits for STEMI based on its underlying pathophysiology of plaque rupture or erosion,² invasive strategies are also effective in appropriately selected NSTEMI patients.³ However, NSTEMI—a presentation more common in older individuals—often requires individualized diagnostic and therapeutic strategies, including careful selection of



Graphical Abstract

Development and validation of a prognostic model for one-year mortality in older adults with acute myocardial infarction incorporating Activities of Daily Living impairment



antithrombotic regimens and tailored decisions regarding invasive management.^{4,5} Identifying high-risk older adults with AMI is therefore essential to optimize clinical intervention and improve survival outcomes.

Activities of daily living (ADL) serve as a key indicator of functional status in older adults and reflect underlying geriatric syndromes such as frailty, sarcopenia, and nutritional decline.^{6,7} Impaired ADL has been consistently associated with worse outcomes during acute illness.^{8,9} Among very old AMI patients, ADL impairment has been shown to be an independent predictor of mortality,¹⁰ Early recognition of functional limitation before hospital admission may therefore support risk stratification and guide treatment intensity, especially in emergency settings where older patients often present with heterogeneous clinical profiles.

The Barthel Index (BI) is a widely used measure of basic ADL encompassing ten domains, including feeding, mobility, personal hygiene, dressing, toileting, and continence.^{11,12} It is simple, reliable, and feasible to administer through patient or caregiver interviews, making it particularly suitable for rapid assessment in the emergency department. Despite the BI's extensive use in geriatric practice, evidence regarding its prognostic value specifically in AMI—particularly among older adults—remains limited.

Existing studies suggest that functional impairment may play a crucial role in clinical decision-making and outcomes. Data from the Yamagata AMI registry demonstrated that pre-hospital ADL impairment was associated with lower PCI utilization and significantly higher mortality among older AMI patients.¹³ Similarly, the Chinese Retrospective Evaluation of Acute Chest Pain (REACP) study showed that ADL impairment independently predicted both all-cause and cardiovascular mortality in patients with acute coronary syndrome.¹⁴ However, there remains a lack of practical, easy-to-use tools to predict mortality risk in AMI patients with impaired ADL—an increasingly common population in aging societies.

Therefore, the objective of this study was to develop a clinically applicable nomogram to predict 1-year all-cause mortality among AMI patients with concomitant ADL impairment, with the goal of supporting more individualized and geriatric-focused clinical interventions.

Materials and Methods

Study Population

Data for this study were derived from the Coronary Artery Disease (CAD) Database within the Tianjin Health and Medical Big Data Super Platform (hereafter referred to as “the Platform”). Tianjin Health and Medical Big Data Co., Ltd., the data custodian, is authorized to manage data collection, governance, and application across the Platform. The Platform integrates clinical diagnosis and treatment information from 43 tertiary and 39 secondary hospitals in Tianjin, as well as public health records. Following standardized processing and de-identification, these data are transformed into a structured research database.

The CAD Database includes patients hospitalized at least once between January 2010 and March 2024 with a discharge diagnosis of CAD. Comprehensive healthcare information is available, including demographic characteristics, clinical diagnoses, medication and non-medication prescriptions, laboratory and imaging results, surgical procedures, healthcare utilization and cost data, community-based medication and health examination records, and public health mortality data. This study was approved by the Institutional Review Board of the Second Hospital of Tianjin Medical University (#KY2023052-01) and was registered at the China Clinical Trials Registry (ChiCTR2400094021) on 16 December 2024. The study was carried out in accordance with the Declaration of Helsinki, as well as national legislation and institutional guidelines.

Patients with AMI were identified using International Classification of Diseases, Tenth Revision (ICD-10) codes I21 and I22. Functional status was assessed using the Barthel Index (BI) obtained from the Tianjin Community Health Questionnaire prior to the AMI event. Participants were categorized into four groups based on BI scores: 100 (normal ADL, completely independent), 61–99 (mild impairment, mildly dependent), 41–60 (moderate impairment, moderately dependent), and 0–40 (severe impairment, fully dependent). We included adults aged ≥ 65 years and excluded individuals with conditions associated with an expected survival of less than one year (eg, prior malignancy with poor prognosis), ensuring a population representative of older adults with sufficient survival horizon for 1-year outcome evaluation. Comorbidities and complications were identified using ICD-10 codes. All ICD-10 codes applied in this study are provided [Table S1](#).

Clinical Outcomes

The primary outcome was all-cause mortality and cardiac deaths. The secondary outcomes included recurrent myocardial infarction, ischemic stroke, major bleeding events, and net adverse clinical events (NACE). All deaths were classified as cardiac deaths unless a non-cardiac cause could be confirmed. Recurrent myocardial infarction or ischemic stroke was defined as the occurrence of these events during the follow-up period that necessitated hospitalization. Major bleeding events were categorized according to the bleeding academic research consortium (BARC) types of 3–5.¹⁵ NACE were defined to include cardiac death, recurrent myocardial infarction, major bleeding events, and ischemic stroke.¹⁶

Statistical Analysis

For continuous variables, means with standard deviations (SD) were reported for normally distributed data, while medians with interquartile ranges were reported for non-normally distributed data. Group comparisons were conducted using either t-tests or Mann–Whitney *U*-tests, depending on data distribution. Categorical variables were presented as counts with percentages and were analyzed using χ^2 tests or Fisher’s exact tests, as appropriate. We used Kaplan–Meier (KM) curve to assess mortality differences across groups. Additionally, we assessed all-cause mortality and cardiac deaths in the subgroup of ADL impaired patients who underwent PCI.

The time interval between ADL assessment and hospital admission was calculated for each patient. Because ADL evaluations were derived from routine health examinations, we recorded the assessment-to-admission interval as a continuous variable. To evaluate the potential influence of long intervals on model performance, sensitivity analyses were conducted by restricting the cohort to patients with ADL assessments performed within 6 months, 1 year, and 2 years prior to admission.

Model Building and Validation

To identify robust predictors and reduce the risk of overfitting, the study sample was first randomly divided into a training set and a validation set using stratified sampling at a 7:3 ratio. We then applied the Least Absolute Shrinkage and Selection Operator (LASSO) regression with 100 bootstrap iterations to screen candidate variables. Treatment-related variables were not included in the LASSO selection or final nomogram, as the model was designed to estimate baseline risk at the time of initial clinical assessment, prior to treatment decisions. Vertical dashed lines were positioned at the values of the minimum lambda (λ min) and the lambda within one standard error (λ 1se). The λ 1se value was selected as the optimal penalization parameter, balancing model simplicity and predictive performance. Variables retained after LASSO selection were subsequently entered into a multivariable Cox proportional hazards model. Based on the final model, a nomogram was constructed to predict 1-year all-cause mortality among AMI patients with impaired ADL. Model performance was evaluated through multiple approaches. Discrimination was assessed using the receiver operating characteristic (ROC) curve and the corresponding area under the curve (AUC). Clinical utility was examined using decision curve analysis (DCA). Calibration was evaluated by plotting calibration curves to compare predicted and observed probabilities of 1-year survival.

To facilitate clinical application, the final model was saved as an RDS file and an interactive web-based calculator was implemented using the Shiny framework. The calculator allows users to input the aforementioned predictors via a graphical interface and provides individualized 1-year mortality risk estimates along with 95% confidence intervals. The calculator is publicly accessible at <https://yukunzhang.shinyapps.io/adlapp/>.

To further assess the discriminatory ability of each individual predictor included in the final model, we additionally plotted ROC curves for all single variables. The AUC values of each predictor were calculated to quantify their individual predictive performance for 1-year all-cause mortality.

All analyses were conducted as two-tailed tests, with clinical significance defined as $p < 0.05$. Statistical analyses were performed using R software version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria). We have adhered to the STROBE guidelines in our reporting ([Table S2](#)).

Results

Baseline Characteristics

The baseline characteristics of the study population are presented in [Table 1](#). After excluding patients with a history of malignancy or other conditions associated with an expected survival of less than one year, a total of 43,308 patients were included in the final analysis. Compared with patients with normal ADL, those with increasing levels of ADL impairment were significantly older [median (IQR): 73.0 (68.0–78.0) vs 78.0 (73.0–83.0) vs 79.0 (73.0–84.0) vs 78.0 (73.0–83.0), $p < 0.001$], and were less frequently diagnosed with STEMI (45.8% vs 40.5% vs 29.6% vs 29.6%, $p < 0.001$). Patients with greater ADL impairment also showed a substantially higher prevalence of intermediate-to-high frailty risk¹⁷ (45.1% vs 63.8% vs 76.5% vs 85.8%, $p < 0.001$) and a higher Charlson Comorbidity Index¹⁸ [median (IQR): 1.0 (0.0–2.0) vs 1.0 (0.0–3.0) vs 2.0 (0.0–4.0) vs 2.0 (1.0–4.0), $p < 0.001$]. The proportion of patients presenting with Killip Class III and IV was also highest in the severe ADL impairment group, at 27.0% and 12.8%, respectively ($p < 0.001$).

The median interval between ADL assessment and hospital admission was 226 days (IQR 115–355), and approximately 80% of patients were assessed within 1 year prior to admission. Importantly, the timing distribution was comparable across ADL groups, indicating no systematic imbalance (225 [116; 348] vs 228 [115; 377] vs 198 [73; 365] vs 226 [125; 349] days, $p=0.243$) ([Table 1](#)).

The use of PCI and evidence-based medications after admission is summarized in [Table 1](#). Compared with patients with normal ADL, those with impaired ADL were markedly less likely to undergo PCI (48.9% vs 25.0% vs 17.3% vs 8.9%, $p < 0.001$). The use of guideline-recommended therapies—including aspirin, clopidogrel/ticagrelor, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers (ACEIs/ARBs), angiotensin receptor–neprilysin inhibitors (ARNI), β -blockers, and statins—was significantly lower among patients with ADL impairment, while diuretic use was higher.

Table 1 Baseline Clinical Characteristics and Clinical Outcomes of 1-Year Follow-Up Time

Variable	ADL Normal N=41312	ADL Mild Impaired N=1318	ADL Moderate Impaired N=452	ADL Severe Impaired N=226	P Overall	P for Trend
Demographic variables						
Male, N (%)	23,182 (56.1)	592 (44.9)	225 (49.8)	115 (50.9)	<0.001	<0.001
Age, years	73.0 [68.0;78.0]	78.0 [73.0;83.0]	79.0 [73.0;84.0]	78.0 [73.0;83.0]	<0.001	<0.001
STEMI, N (%)	18,921 (45.8)	534 (40.5)	134 (29.6)	67 (29.6)	<0.001	<0.001
Killip class, N (%)					<0.001	<0.001
I	23371 (56.6)	614 (46.6)	198 (43.8)	83 (36.7)		
II	10420 (25.2)	366 (27.8)	128 (28.3)	53 (23.5)		
III	5466 (13.2)	238 (18.1)	87 (19.2)	61 (27.0)		
IV	2055 (5.0)	100 (7.6)	39 (8.6)	29 (12.8)		
Intermediate to High Frailty Risk, N (%)	18,615 (45.1)	841 (63.8)	346 (76.5)	194 (85.8)	<0.001	<0.001
Time Interval Between ADL Assessment and AMI Admission, days	225 [116;348]	228 [115;377]	198 [73;365]	226 [125;349]	0.243	0.902
Complications, N (%)						
Ventricular Fibrillation	726 (1.8)	25 (1.9)	3 (0.7)	5 (2.2)	0.259	0.602
Ventricular Tachycardia	1404 (3.4)	49 (3.7)	14 (3.1)	7 (3.1)	0.898	0.956
III° Atrioventricular Block	2034 (4.9)	68 (5.2)	21 (4.7)	9 (4.0)	0.883	0.705
Medical histories, N (%)						
Charlson Comorbidity Index	1.0 [0.0;2.0]	1.0 [0.0;3.0]	2.0 [0.0;4.0]	2.0 [1.0;4.0]	<0.001	<0.001
Diabetes Mellitus	13140 (31.8)	493 (37.4)	181 (40.0)	97 (42.9)	<0.001	<0.001
Hypertension	23909 (57.9)	835 (63.4)	295 (65.3)	154 (68.1)	<0.001	<0.001
Atrial Fibrillation	2794 (6.8)	115 (8.7)	43 (9.5)	25 (11.1)	<0.001	<0.001
Valvular heart disease	740 (1.8)	28 (2.1)	13 (2.9)	9 (4.0)	0.026	0.003
Hyperlipidemia	14268 (34.5)	489 (37.1)	176 (38.9)	92 (40.7)	0.012	0.001
Chronic Obstructive Pulmonary Disease	1542 (3.7)	67 (5.1)	21 (4.7)	12 (5.3)	0.033	0.009
Parkinson's Disease	281 (0.7)	16 (1.2)	9 (2.0)	5 (2.2)	<0.001	<0.001
Prior PCI	2019 (4.9)	45 (3.4)	11 (2.4)	1 (0.4)	<0.001	<0.001
Ischemic Stroke	12715 (30.8)	574 (43.6)	240 (53.1)	134 (59.3)	<0.001	<0.001
Cerebral Hemorrhage	776 (1.9)	48 (3.6)	24 (5.3)	20 (8.9)	<0.001	<0.001
Renal Insufficiency	3426 (8.3)	194 (14.7)	80 (17.7)	39 (17.3)	<0.001	<0.001
Peripheral Vascular Disease	4495 (10.9)	158 (12.0)	55 (12.2)	20 (8.9)	0.343	0.604
Medication at discharge, N (%)						
PCI	20198 (48.9)	329 (25.0)	78 (17.3)	20 (8.9)	<0.001	<0.001
Aspirin	36048 (87.3)	1035 (78.5)	309 (68.4)	139 (61.5)	<0.001	<0.001
Clopidogrel/Ticagrelor	37771 (91.4)	1130 (85.7)	353 (78.1)	167 (73.9)	<0.001	<0.001
Anticoagulant	1415 (3.4)	43 (3.3)	13 (2.9)	6 (2.7)	0.826	0.352

(Continued)

Table I (Continued).

Variable	ADL Normal N=41312	ADL Mild Impaired N=1318	ADL Moderate Impaired N=452	ADL Severe Impaired N=226	P Overall	P for Trend
ACEIs/ARBs	26513 (64.2)	761 (57.7)	242 (53.5)	95 (42.0)	<0.001	<0.001
ARNI	6065 (14.7)	115 (8.7)	53 (11.7)	20 (8.9)	<0.001	<0.001
β-Blockers	27159 (65.7)	791 (60.0)	247 (54.6)	104 (46.0)	<0.001	<0.001
CCB	9156 (22.2)	324 (24.6)	121 (26.8)	42 (18.6)	0.01	0.114
Diuretic	25486 (61.7)	979 (74.3)	359 (79.4)	173 (76.5)	<0.001	<0.001
Statins	37808 (91.5)	1128 (85.6)	370 (81.9)	161 (71.2)	<0.001	<0.001
Ivabradine	905 (2.2)	29 (2.2)	15 (3.3)	3 (1.3)	0.354	0.681
Levosimendan	891 (2.2)	21 (1.6)	5 (1.1)	10 (4.4)	0.026	0.916
Outcomes, N (%)						
In-Hospital Deaths	1347 (3.3)	73 (5.5)	29 (6.4)	25 (11.1)	<0.001	<0.001
All-cause Mortalities	7190 (17.4)	420 (31.9)	204 (45.1)	135 (59.7)	<0.001	<0.001
Cardiac Deaths	5581 (13.5)	323 (24.5)	159 (35.2)	102 (45.1)	<0.001	<0.001
Recurrent MI	2313 (5.6)	78 (5.9)	21 (4.7)	6 (2.7)	0.193	0.139
Ischemic Stroke	1337 (3.2)	34 (2.6)	10 (2.2)	4 (1.8)	0.193	0.031
Major Bleeding Events	1665 (4.0)	64 (4.9)	22 (4.9)	6 (2.7)	0.247	0.548
NACE	9994 (24.2)	456 (34.6)	197 (43.6)	116 (51.3)	<0.001	<0.001

Abbreviations: STEMI, ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; ACEIs/ARBs, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers; ARNI, angiotensin receptor-neprilysin inhibitor; CCB, calcium channel blocker; NACE, net adverse clinical events.

To ensure model reliability, baseline characteristics between the training and validation cohorts were also compared (Table 2). No significant differences were observed across demographic variables, comorbidities, or treatment patterns (all $p > 0.05$), indicating that the two datasets were well balanced and suitable for subsequent model development and validation.

One-Year Outcomes of Patients

The clinical outcomes during 1-year follow-up are shown in Table 1. Compared to patients with normal ADL, those with ADL impairment had a significantly higher in-hospital mortality rate (3.3% vs 5.5% vs 6.4% vs 11.1%, $p < 0.001$), all-cause

Table 2 Training vs Validation Cohorts Baseline Comparison

Variable	Testing N=12991	Training N=30317	P value
Demographic variables			
Male, N (%)	7226 (55.6)	16,888 (55.7)	0.884
Age, years	73.0 [69.0;79.0]	73.0 [69.0;79.0]	0.256
STEMI, N (%)	5887 (45.3)	13,769 (45.4)	0.855
Killip class, n (%)			0.587
I	7270 (56.0)	16,996 (56.1)	
II	3336 (25.7)	7631 (25.2)	
III	1736 (13.4)	4116 (13.6)	
IV	649 (5.0)	1574 (5.2)	
Intermediate to High Frailty Risk, N (%)	6004 (46.2)	13,992 (46.2)	0.843
Complications, N (%)			
Ventricular Fibrillation	241 (1.9)	518 (1.7)	0.305
Ventricular Tachycardia	459 (3.5)	1015 (3.4)	0.344
III° Atrioventricular Block	640 (4.9)	1492 (4.9)	1.000
Medical histories, N (%)			
Charlson Comorbidity Index	1.0 [0.0;2.0]	1.0 [0.0;2.0]	0.689
Diabetes Mellitus	4197 (32.3)	9714 (32.0)	0.595
Hypertension	7594 (58.5)	17,599 (58.0)	0.439
Atrial Fibrillation	883 (6.8)	2094 (6.9)	0.694
Valvular heart disease	222 (1.7)	568 (1.9)	0.257
Hyperlipidemia	4537 (34.9)	10,488 (34.6)	0.516
Chronic Obstructive Pulmonary Disease	505 (3.9)	1137 (3.8)	0.512
Parkinson's Disease	92 (0.7)	219 (0.7)	0.922
Prior PCI	607 (4.7)	1469 (4.9)	0.455
Ischemic Stroke	4077 (31.4)	9586 (31.6)	0.636
Cerebral Hemorrhage	270 (2.1)	598 (2.0)	0.495
Renal Insufficiency	1108 (8.5)	2631 (8.7)	0.625
Peripheral Vascular Disease	1363 (10.5)	3365 (11.1)	0.066
Medication at discharge, N (%)			
PCI	6168 (47.5)	14,457 (47.7)	0.700
Aspirin	11270 (86.8)	26,261 (86.6)	0.725
Clopidogrel/Ticagrelor	11881 (91.4)	27,540 (90.8)	0.45
Anticoagulant	440 (3.4)	1037 (3.4)	0.883
ACEIs/ARBs	8311 (64.0)	19,300 (63.7)	0.540
ARNI	1820 (14.0)	4433 (14.6)	0.100
β-Blockers	8424 (64.8)	19,877 (65.6)	0.153
CCB	2909 (22.4)	6734 (22.2)	0.688
Diuretic	8115 (62.5)	18,882 (62.3)	0.725
Statins	11866 (91.3)	27,601 (91.0)	0.325
Ivabradine	292 (2.3)	660 (2.2)	0.671
Levosimendan	271 (2.1)	656 (2.2)	0.634

(Continued)

Table 2 (Continued).

Variable	Testing N=12991	Training N=30317	P value
Outcomes, N (%)			
In-Hospital Deaths	445 (3.4)	1029 (3.4)	0.892
All-cause Mortalities	2384 (18.4)	5565 (18.4)	1.000
Cardiac Deaths	1828 (14.1)	4337 (14.3)	0.532
Recurrent MI	693 (5.3)	1725 (5.7)	0.146
Ischemic Stroke	420 (3.2)	965 (3.2)	0.810
Major Bleeding Events	487 (3.8)	1270 (4.2)	0.360
NACE	3163 (24.3)	7600 (25.1)	0.114

Abbreviations: STEMI, ST-elevation myocardial infarction; PCI, percutaneous coronary intervention; ACEIs/ARBs, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers; ARNI, angiotensin receptor-neprilysin inhibitor; CCB, calcium channel blocker; NACE, net adverse clinical events.

mortality (17.4% vs 31.9% vs 45.1% vs 59.7%, $p < 0.001$), cardiovascular mortality (13.5% vs 24.5% vs 35.2% vs 45.1%, $p < 0.001$), and incidence of NACE (24.2% vs 34.6% vs 43.6% vs 51.3%, $p < 0.001$). KM curve analysis revealed that the risk of both all-cause and cardiovascular mortality increased significantly with the degree of ADL impairment ($p < 0.001$) (Figure 1).

Association Between PCI Implementation and Mortality

We performed subgroup analyses involving 1,996 AMI patients with impaired ADL (Figure S1). The multivariable Cox regression analysis revealed that PCI treatment was associated with a reduced risk of all-cause mortality (aHR: 0.45, 95% CI: 0.35–0.58, $p < 0.001$) and cardiovascular mortality (aHR: 0.38, 95% CI: 0.28–0.52, $p < 0.001$) in AMI patients with impaired ADL. Therefore, PCI strategy should be considered for AMI patients with ADL impairment.

Development and Validation of the Nomogram Model

Variables including baseline characteristics and medical history of the AMI cohort were subjected to Lasso regression analysis (Figure S2). This process ultimately identified 7 predictors with non-zero coefficients: ADL status, age, Killip class, STEMI diagnosis, history of stroke, valvular heart disease, and diabetes mellitus (Figure 2A). Using the online

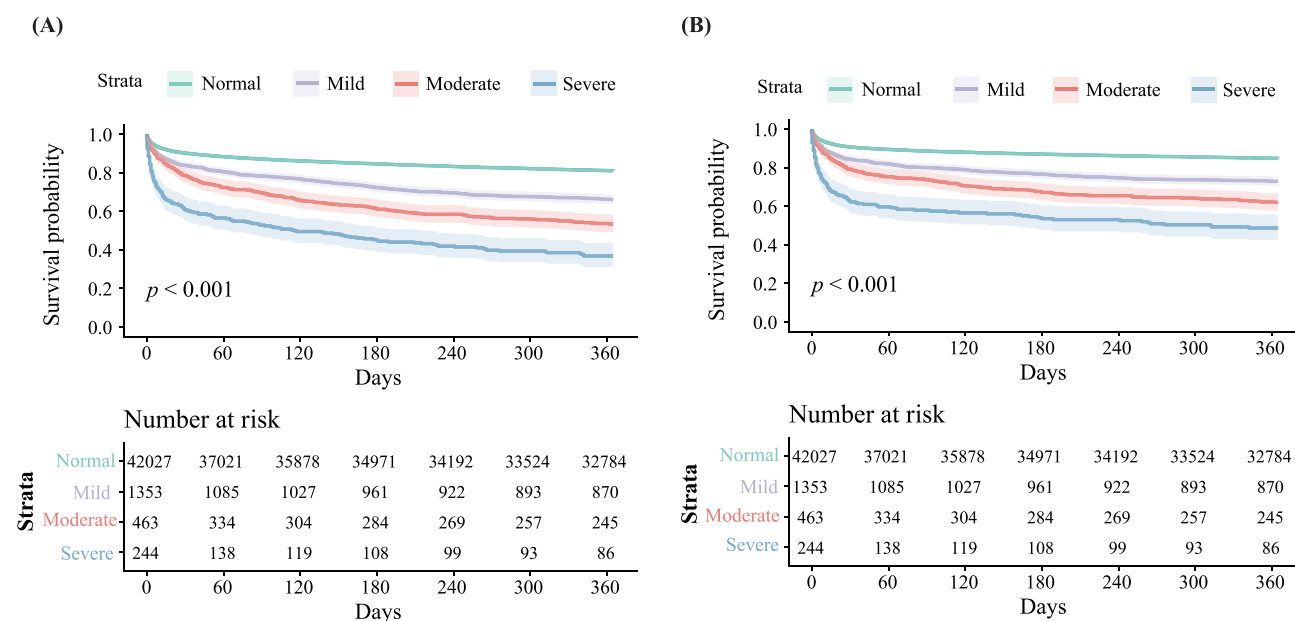
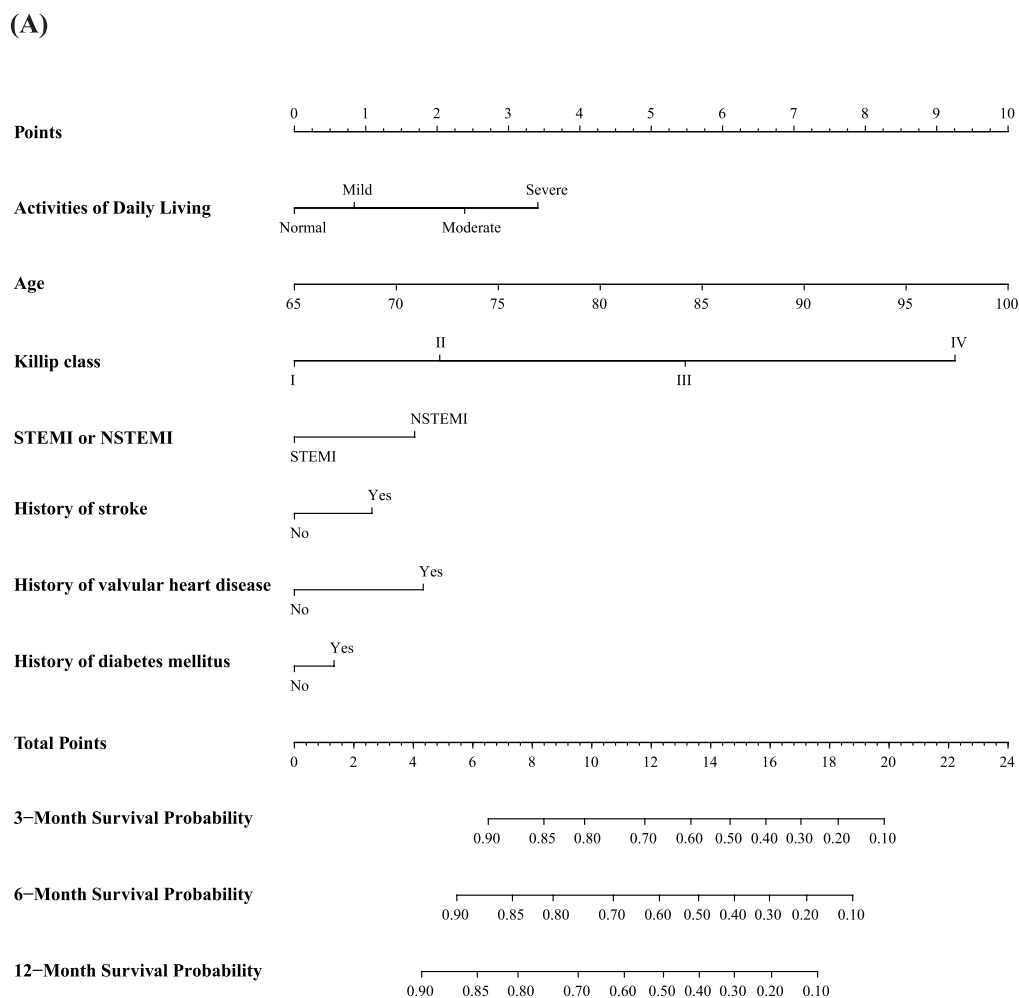


Figure 1 Kaplan-Meier survival curves for AMI patients stratified by ADL status.
Notes: (A) Kaplan-Meier survival curves of all-cause mortality; (B) Kaplan-Meier survival curves of cardiovascular mortality.



(B)
1-Year Mortality Risk Calculator

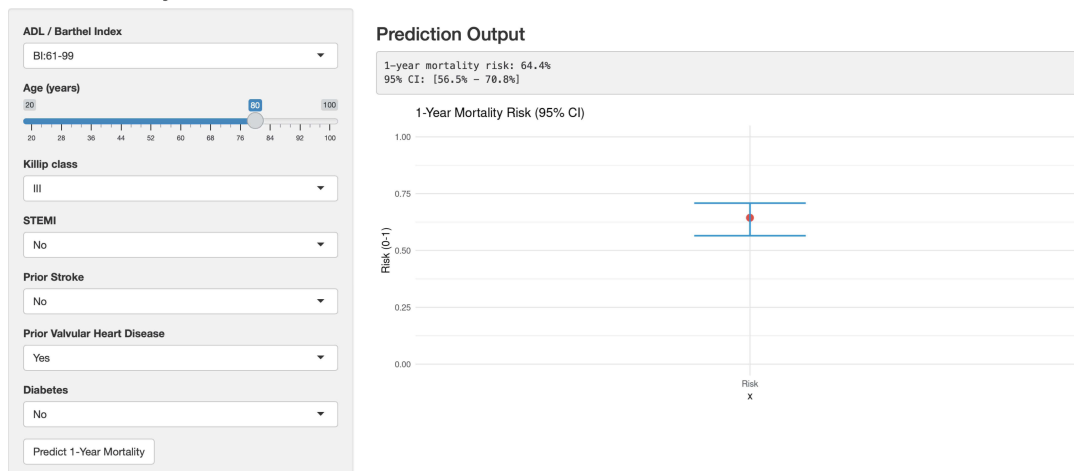


Figure 2 Nomogram for predicting 1-year mortality risk in AMI patients with impaired ADL.
Notes: (A) The Nomogram model identified 7 predictors with non-zero coefficients: ADL status, age, Killip class, STEMI diagnosis, history of stroke, valvular heart disease, and diabetes mellitus. (B) The online calculator. For example, a 80-year-old patient with an ADL score of 61–99, Killip class III, non-STEMI, valvular heart disease, without prior stroke, or diabetes, was predicted to have a 1-year mortality risk of 64.4%, with a 95% confidence interval of 56.5%–70.8%.

calculator, clinicians can rapidly estimate 1-year mortality risk based on patient-specific characteristics. For example, a 80-year-old patient with an ADL score of 61–99, Killip class III, non-STEMI, valvular heart disease, without prior stroke, or diabetes, was predicted to have a 1-year mortality risk of 64.4%, with a 95% confidence interval of 56.5%–70.8% (Figure 2B). The predictors selected by Lasso regression were incorporated into a multivariable Cox proportional hazards analysis to construct a nomogram for predicting 1-year all-cause mortality in AMI patients with ADL impairment (Table S3).

In the training cohorts, the nomogram demonstrated AUCs of 0.794 (95% CI: 0.787–0.802), 0.793 (95% CI: 0.787–0.801) and 0.793 (95% CI: 0.787–0.800) for predicting 3-month, 6-month, and 12-month mortality, respectively (Figure 3A). Corresponding AUCs in the validation cohorts were 0.780 (95% CI: 0.768–0.791), 0.779 (95% CI: 0.768–0.790), and 0.779 (95% CI: 0.770–0.790) (Figure 3B). The clinical utility of the nomogram, as assessed by DCA in Figure S3, showed a high net benefit in both cohorts, supporting its potential for guiding interventions. Furthermore, the calibration curves demonstrated excellent agreement between predicted and observed mortality risks in the training and validation cohorts (Figures S4A and S4B).

Individual ROC analyses were performed for each variable included in the final model (Figure 4). Although several predictors demonstrated moderate discriminatory capacity, all single-factor AUC values were inferior to that of the multivariable model, indicating that the integrated nomogram provides substantially improved predictive performance compared with any single predictor.

In sensitivity analyses restricted to patients with shorter assessment-to-admission intervals (≤ 6 months, ≤ 1 year, and ≤ 2 years), the predictive performance of the nomogram remained stable, with AUC values ranging from 0.789 to 0.803 (Figure S5). These findings indicate that variation in assessment timing did not materially influence model discrimination.

Discussion

In this study, we made several important observations relevant to the care of older adults with AMI. First, patients with pre-hospital ADL impairment were significantly older, and presented with more severe clinical conditions, including a higher prevalence of Killip class III–IV. They were less likely to receive PCI and experienced substantially higher risks of all-cause and cardiac mortality. Second, the graded relationship between ADL impairment and mortality observed in the KM analyses underscores the prognostic relevance of functional status in elderly AMI patients. Third, PCI therapy remained independently associated with reduced mortality in patients with impaired ADL, suggesting that functional

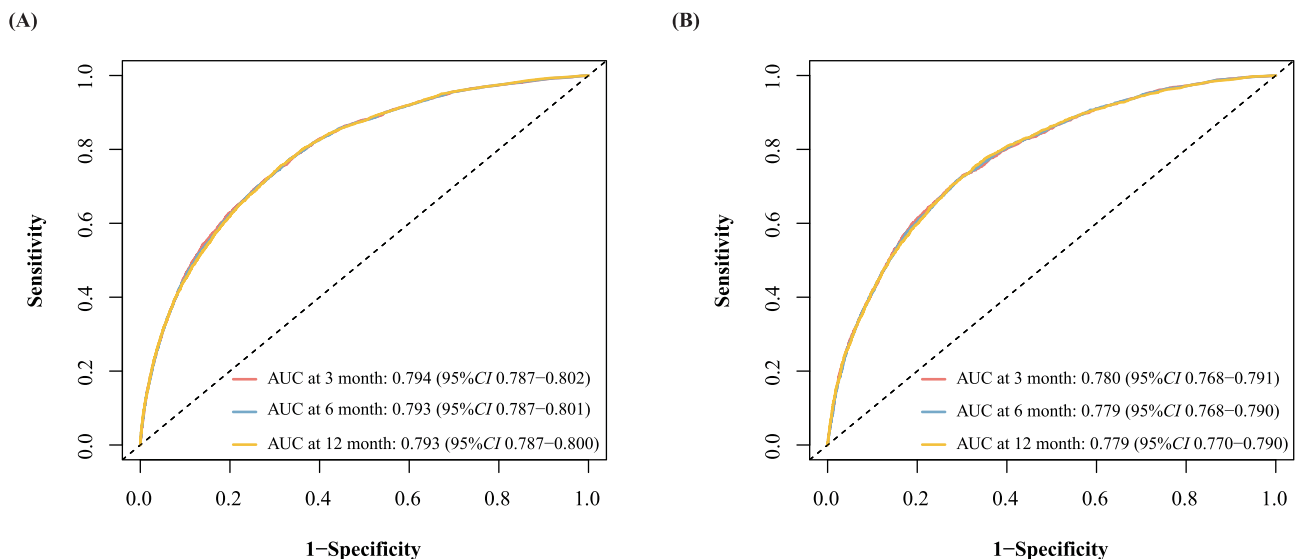


Figure 3 Receiver operating characteristic (ROC) curves of the nomogram for predicting mortality risk at 3, 6, and 12 months in the training and validation cohorts. **Notes:** (A) ROC curves of the nomogram in the training cohorts; (B) ROC curves of the nomogram in the validation cohorts.

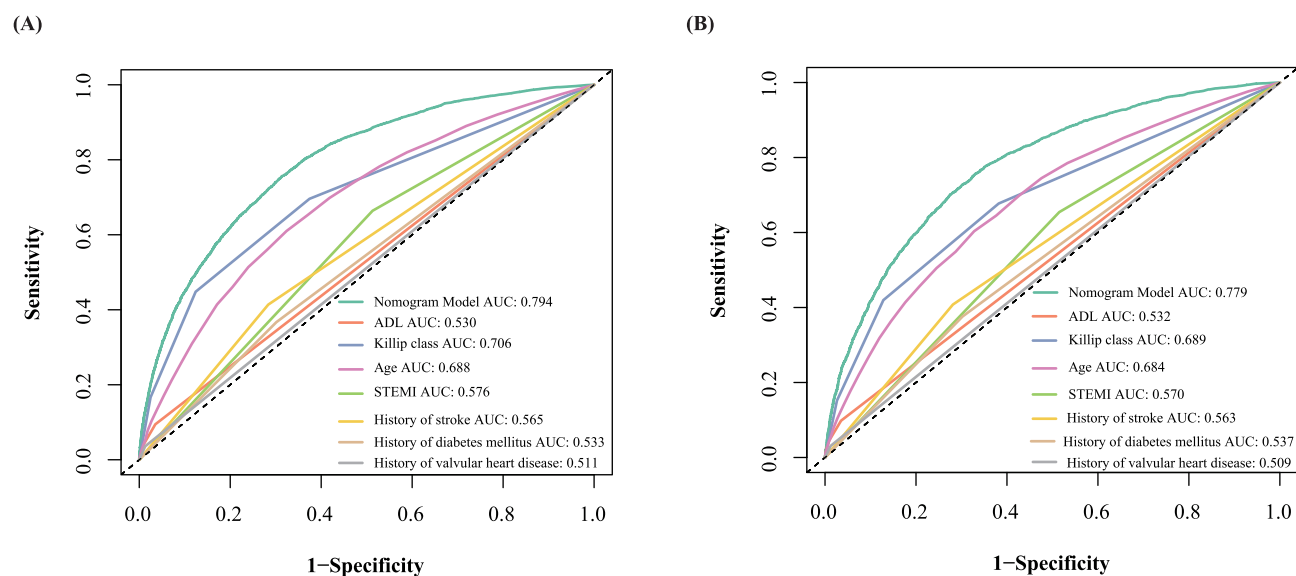


Figure 4 Receiver operating characteristic (ROC) curves for individual predictors included in the final model.

Notes: (A) ROC curves for individual predictors in the training cohorts; (B) ROC curves for individual predictors in the validation cohorts.

vulnerability should not preclude the use of evidence-based invasive therapies. Finally, the nomogram incorporating ADL impairment demonstrated strong predictive performance for 1-year mortality, with consistent discrimination, calibration, and clinical utility across both training and validation cohorts.

Pre-admission ADL status was assessed using the Barthel Index. In our cohort, approximately 4.6% of patients had impaired ADL. According to the frailty risk score developed by Gilbert et al¹⁷ the prevalence of moderate to severe frailty reached 69.2% among patients with ADL impairment, compared with 45.1% among those with preserved ADL. ADL impairment is widely viewed as a clinical manifestation of frailty and may accelerate frailty progression. Prior studies have shown that frailty is independently associated with all-cause mortality in AMI patients, and incorporating frailty measures into risk models enhances prognostic accuracy.¹⁹ Furthermore, among AMI patients who survived the first month, frailty was associated with a markedly increased risk of 1-year MACE.²⁰ These findings align with our results: both short-term in-hospital mortality and 1-year all-cause and cardiovascular mortality increased steadily with worsening ADL impairment.

ADL impairment also correlates strongly with the burden of comorbidities. Hypertension and diabetes contribute to accelerated functional decline, manifesting clinically as impaired ADL.^{21,22} Hypertension combined with impaired ADL significantly elevates the risks of cardiovascular disease and stroke.²³ Diabetes contributes to progressive muscle weakness and sarcopenia, with worse glycemic control and longer disease duration accelerating disability.²⁴ A meta-analysis showed that compared with non-diabetic individuals, those with diabetes had a 50–80% higher risk of ADL impairment.²⁵ In a cohort of 3530 older adults with atrial fibrillation, ADL independence declined 4.4% annually, and 31.9% developed new ADL dependence after stroke.²⁶ Preventive cardiovascular risk management—including lipid control—may help maintain physical function in older individuals.²⁷ In our study, the incidence of comorbidities rose in parallel with ADL impairment, suggesting a synergistic effect wherein multimorbidity and functional decline jointly contribute to excess mortality risk.

We also observed that the proportion of AMI patients classified as Killip III–IV increased with worsening ADL impairment, indicating a strong association between impaired ADL and more severe heart failure. This finding is consistent with existing evidence. A longitudinal cohort of 1128 heart failure patients showed that ADL impairment significantly increased mortality and the risk of all-cause and non-cardiovascular hospitalization.²⁸ Roy et al reported a 73.9% prevalence of ADL impairment among patients with advanced heart failure, and these patients remained at high mortality risk regardless of ADL status.²⁹ Early cardiac rehabilitation, when clinically feasible, can improve ADL levels

and reduce rehospitalization and cardiovascular events.³⁰ Moreover, nearly half of patients recovering from acute cardiovascular events experience persistent declines in quality of life and self-perceived health status, which may negatively impact long-term functional recovery.³¹ Early identification of functional vulnerability, timely management of heart failure severity, and initiation of cardiac rehabilitation may therefore improve recovery of ADL and reduce mortality.

Evidence from the National Acute Coronary Syndrome Registry in England and Wales shows that older patients are less likely to receive medications and invasive strategies, while mortality rises sharply with age. Although the survival benefit of intensive therapy diminishes somewhat in very elderly patients, invasive treatment still provides measurable survival advantages.³² Among patients aged ≥ 80 years, invasive treatment was associated with lower mortality and reduced risk of heart failure hospitalization, regardless of frailty status.^{33,34} These results mirror our findings and reinforce that age or functional impairment alone should not preclude guideline-directed invasive management in appropriately selected older AMI patients.

An additional noteworthy finding is the relatively low utilization of PCI in the overall elderly AMI cohort, including patients without ADL impairment. Prior studies have shown that chronological age itself remains an independent predictor of conservative management, even in the absence of overt frailty.³⁵ Despite evidence from randomized trials supporting invasive strategies in selected elderly patients, real-world practice often reflects concerns regarding bleeding risk, renal dysfunction, multimorbidity, and perceived procedural vulnerability.³⁶ This phenomenon has been described as the “treatment–risk paradox,” whereby higher-risk elderly patients are paradoxically less likely to receive guideline-recommended invasive therapies. Furthermore, the lack of systematic integration of geriatric assessments, including frailty and cognitive evaluation, may contribute to variability and potential bias in clinical decision-making.³⁷

Nearly one-fifth of older AMI patients are readmitted within 30 days,³⁸ and early readmission substantially affects quality of life and health-care burden. Yet predicting readmission and long-term prognosis remains challenging.³⁹ Our prediction model provides a quantitative tool for evaluating 1-year mortality risk in AMI patients with impaired ADL. By integrating multiple risk factors and weighting them by their relative contributions, the nomogram outputs individualized risk estimates that support targeted interventions focusing on modifiable risk factors.

Prior studies have emphasized the prognostic value of functional status in AMI. The SILVER-AMI study, which enrolled 3,006 older AMI survivors across 94 US hospitals, evaluated 72 candidate variables. The final model—comprising 15 variables including mobility impairment—achieved strong predictive performance for 6-month mortality (AUC = 0.84), and adding functional and mobility measures substantially enhanced model discrimination.⁴⁰ Within the same cohort, ADL impairment emerged as the strongest predictor of 30-day readmission in elderly AMI patients.⁴¹ Our findings extend this body of evidence by demonstrating the prognostic relevance of ADL impairment for predicting 1-year mortality in AMI patients. Although treatment strategies influence patient prognosis, this study aimed to develop a baseline risk prediction model rather than to estimate treatment effects. Similar to established risk stratification tools such as the GRACE score,⁴² which rely solely on baseline characteristics available at presentation. Our model was designed to estimate intrinsic risk rather than treatment-modified outcomes. Treatment decisions occur downstream of baseline risk assessment and may act as intermediate variables. Excluding these variables helps avoid post-treatment bias and preserves the clinical interpretability and transportability of the model.

This study has several limitations that should be noted, particularly in the context of an older AMI population. First, as a multicenter, retrospective, observational study, the findings are subject to potential selection bias and residual confounding, which may limit the generalizability of the results to broader elderly populations with diverse functional and clinical characteristics. Second, ADL status was assessed based on pre-admission reports, which may not fully capture the patient’s functional trajectory at the time of hospitalization. Changes in ADL during follow-up—especially meaningful in older adults whose functional status can fluctuate—were not evaluated and therefore could not be incorporated into the analysis. Third, although we applied broad exclusion criteria to reduce potential confounding, this approach may have unintentionally omitted certain clinically relevant subgroups. Further research focusing on the relationship between ADL impairment and adverse outcomes across specific elderly subpopulations is warranted. Additionally, the Barthel Index primarily reflects chronic physical functional impairment and does not directly assess cognitive function. Cognitive impairment is common in elderly patients with AMI and may independently affect

prognosis and treatment decisions. The lack of standardized cognitive assessment in our registry should therefore be acknowledged when interpreting the findings. Future studies could integrate cognitive assessments alongside ADL evaluations, potentially providing a more comprehensive risk stratification model for older AMI patients. Finally, although the prediction model was internally validated using a multicenter cohort, external validation in independent elderly populations from different geographic or healthcare settings is warranted to further assess its transportability before broader clinical application.

Conclusion

ADL impairment is a strong and independent predictor of all-cause and cardiovascular mortality among older adults with AMI. We developed a practical and clinically applicable nomogram to predict 1-year mortality in AMI patients with impaired ADL. This tool may support clinicians in early risk identification, individualized treatment planning, and improved management of vulnerable older adults with acute myocardial infarction.

Abbreviations

ADL, activities of daily living; AMI, Acute myocardial infarction; BI, Barthel Index; ROC, receiver operating characteristic curve; AUC, area under the curve; DCA, decision curve analysis; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; CAD, Coronary Artery Disease; ICD, International Classification of Diseases; NACE, net adverse clinical events; BARC, bleeding academic research consortium; SD:standard deviations; KM, Kaplan-Meier; LASSO, Least Absolute Shrinkage and Selection Operator; ACEIs/ARBs, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers; ARNI, angiotensin receptor-neprilysin inhibitor; CCB, calcium channel blocker; aHR, adjusted hazard ratio; CI, confidence interval; MACE, major adverse cardiovascular events.

Data Sharing Statement

The data supporting the findings of this study can be obtained from the corresponding author upon reasonable request.

Ethics Approval and Informed Consent

The study protocol was reviewed and approved by the Clinical Research Ethics Committee of the Second Hospital of Tianjin Medical University (Approval No. KY2023052-01). As this retrospective study used anonymized and de-identified data extracted from the Tianjin Health and Medical Big Data Superplatform, the requirement for written informed consent was waived by the ethics committee. The study was carried out in accordance with the Declaration of Helsinki, as well as national legislation and institutional guidelines. The study is registered with the China Clinical Trials Registry (ChiCTR2400094021) on 16 December 2024.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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