

A Nomogram for Predicting Early Mortality in Cardiac Arrest Patients Undergoing ECPR: A Multicenter Development and External Validation Study

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A Nomogram for Predicting Early Mortality in Cardiac Arrest Patients Undergoing ECPR: A Multicenter Development and External Validation Study

Background: Despite its growing use as a rescue therapy for refractory cardiac arrest (CA), extracorporeal cardiopulmonary resuscitation (ECPR) is plagued by persistently high early in-hospital mortality. While existing prediction tools focus on mid-term survival, few are designed for the critical initial 10-day period—a time window when clinical interventions are most impactful. This study sought to address this gap by developing and externally validating a nomogram for 10-day mortality prediction using routinely available laboratory parameters.

Methods: A training cohort of 162 ECPR-treated CA patients (Huai'an Hospital Affiliated to Xuzhou Medical University, 2018–2025) and an external validation cohort of 49 patients (Huai'an First Hospital Affiliated to Nanjing Medical University, same period) were retrospectively analyzed. Cox regression was used to identify independent predictors and build the nomogram, which was then assessed for calibration and discrimination in both cohorts.

Results: Among the 211 patients analyzed, five routine laboratory parameters were confirmed as independent predictors: D-dimer, international normalized ratio, serum potassium, serum sodium, and lymphocyte-to-monocyte ratio (LMR). The nomogram demonstrated good discriminative performance in the external validation set, with an area under the curve (AUC) of 0.793 (95% CI: 0.660–0.926).

Conclusions: We developed and externally validated a concise, routine blood test-based nomogram for early mortality risk stratification in the first 10 days post-ECPR. This readily applicable tool may facilitate timely risk assessment and individualized decision-making in critically ill CA patients receiving ECPR.

Keywords: Cardiac arrest; Extracorporeal cardiopulmonary resuscitation; 10-day in-hospital mortality

Introduction

Cardiac arrest (CA) is the leading cause of death among patients with cardiac disease. As documented by emergency medical services, the incidence of CA is 40.6 per 100,000 person-years in Europe, 47.3 in North America, 45.9 in Asia, and 51.1 in Australia, with a gradual upward trend over time¹⁻³. In China, the burden of CA is particularly severe, with approximately 544,000 new cases diagnosed annually. Specifically, the survival rate of in-hospital CA patients ranges from 6% to 26%, while that of out-of-hospital CA patients is below 10%, posing a substantial threat to human life and health⁴⁻⁶.

Extracorporeal cardiopulmonary resuscitation (ECPR) has emerged as a salvage therapy for refractory CA, yet its clinical application is resource-intensive and demands rapid decision-making in time-sensitive scenarios⁷. Numerous studies have investigated mid-term outcomes, such as 28-day and 30-day survival, in ECPR-treated patients⁸⁻¹⁰, the critical early phase—specifically the first 10 days post-ECPR—remains understudied. This early window is particularly consequential, as it encompasses the highest risk of multi-organ failure and hemodynamic instability, and timely identification of high-risk patients could profoundly influence resource allocation and therapeutic intensity.

To address these challenges, we conducted a two-center retrospective study, utilizing data from one hospital for model development and one from another for external validation. This study aims to identify potential factors associated with 10-day in-hospital mortality, thereby providing a reference for clinical decision-making. Additionally, the findings offer a scientific basis for optimizing the allocation of medical resources and reducing the burden on patients' families.

Materials and Methods

Study Population

A total of 167 consecutive patients with CA who received ECPR were enrolled from Huai'an Hospital Affiliated to Xuzhou Medical University between March 2018 and May 2025. These patients served as the initial training cohort. During the same period, 55 similar patients from Huai'an First Hospital Affiliated to Nanjing Medical

University were enrolled as the initial external validation cohort. After screening according to strict inclusion and exclusion criteria, 162 patients remained in the final training cohort and 49 patients in the external validation cohort. The detailed screening process is shown in Figure 1. This study was approved by the Ethics Committee of the Affiliated Huai'an Hospital of Xuzhou Medical University (Approval number: HEYLL202568), as well as the Ethics Committee of Huai'an First Hospital Affiliated to Nanjing Medical University (Approval number: KY-2026-129-01).

Inclusion Criteria

(I) The patient meets the indications for ECPR: failure to achieve return of spontaneous circulation after ≥ 30 minutes of conventional CPR, or severe cardiogenic shock/respiratory failure refractory to conventional therapy⁴; (II) Age ≥ 18 years; (III) Complete clinical data, including baseline demographic characteristics, laboratory results, treatment measures, and prognostic information.

Exclusion Criteria

(I) Diagnosed with advanced malignant tumors before ECPR; (II) Severe congenital cardiopulmonary diseases; (III) Missing key clinical indicators.

Data Collection

Gender, age, CRRT treatment, high-sensitivity C-reactive protein levels, history of hypertension, absolute neutrophil count, absolute monocyte count, white blood cell count, absolute lymphocyte count, red blood cell count, hemoglobin, platelet, aspartate aminotransferase, potassium, sodium, calcium, blood urea nitrogen, serum creatinine, history of diabetes, chlorine, lactate dehydrogenase, creatine kinase, history of hyperlipidemia, creatine kinase-MB, myoglobin, cardiac troponin T, partial pressure of oxygen, D-dimer, thrombin time, pH were collected before ECPR and outcome indicators, survival time were recorded.

The formulas for calculating the selected indicators are presented as follows:

$$\text{LMR(Lymphocyte-Monocyte Ratio}^{11}) = \frac{\text{lymphocyte count}}{\text{monocyte count}}$$

$$\text{INR(International Normalized Ratio}^{12}) = \frac{\text{Prothrombin time}}{\text{Mean Normal Prothrombin time}}$$

×International Sensitivity Index

Outcome

The primary endpoint was all-cause in-hospital mortality within 10 days after admission for ECPR. The 10-day time-point was selected for several reasons: First, prior real-world cohorts demonstrated that a large proportion of deaths among ECPR-treated cardiac arrest patients occur within the early 10-day hospitalization period, representing the peak phase of post-arrest ischemia-reperfusion injury, coagulopathy and multiple-organ failure¹³. Second, the 10-day window extends beyond the 72-hour neuro-prognostication period after cardiac arrest, reducing confounding from early withdrawal-of-life-support decisions made solely based on incomplete neurological assessment¹⁴. Third, we compared model discrimination across 5-, 10- and 15-day endpoints in our dataset; the 10-day endpoint yielded the optimal AUC performance, while predictive efficacy declined for events occurring beyond day-10. Finally, most existing ECPR risk scores were designed for discharge or 28-/30-day outcomes; a 10-day endpoint addresses the unmet clinical need for early risk stratification during the resource-intensive initial ICU course after ECPR^{15,16}. Patients discharged alive within 10 days were censored at the time of hospital discharge.

Variable processing

The cutoff values for serum potassium, serum sodium, high-sensitivity C-reactive protein (hs-CRP), D-dimer, and LMR were determined based on the AUC and Youden index.

Statistical Analysis

Statistical analysis was performed using R software. Normally distributed measurement data were expressed as mean $\pm$ standard deviation (Mean $\pm$ SD) and analyzed using the t-test; non-normally distributed data were expressed as M (Q₁, Q₃) and analyzed using the Wilcoxon rank-sum test; count data were expressed as n (%) and analyzed using the chi-square test or Fisher's exact test. Multiple imputation with chained equations (MICE) was performed to handle missing data. With 10-day in-hospital mortality as the outcome, survival curves were plotted using the Kaplan-Meier method, and the log-rank test was performed. Univariate Cox regression was used to screen for potential influencing factors, and indicators with $P < 0.05$ were included in multivariate Cox regression to

identify independent risk factors affecting patient prognosis. Hazard ratios (HR) and 95% confidence intervals (CI) were calculated. The P -value < 0.05 was considered statistically significant.

Results

Baseline Characteristics of Enrolled Patients

A total of 211 CA patients treated with ECPR were included in the final analysis, consisting of 162 patients in the training cohort and 49 patients in the validation cohort. The baseline clinical characteristics of all patients are summarized in Table 1. No statistically significant inter-group differences were observed.

Variables associated with a poor outcome

Univariate Cox regression analysis was performed on clinical variables of the 162 patients in the training cohort, with detailed results presented in Table 2. Univariate Cox regression analysis identified several prognostic factors for CA patients undergoing ECPR. High LMR (> 8.86) was associated with an increased risk (HR = 1.958, 95%CI: 1.347 - 2.846, $P < 0.001$). Elevated serum potassium (HR = 1.286, 95%CI: 1.089 - 1.519, $P = 0.003$), serum sodium (HR = 1.054, 95%CI: 1.030 - 1.079, $P < 0.001$), and INR (HR = 1.620, 95%CI: 1.225 - 2.143, $P = 0.001$) were also identified as risk factors. D-Dimer level elevated (> 8.07) was also associated with an increased risk of adverse outcomes (HR = 1.917, 95%CI: 1.331 - 2.761, $P < 0.001$).

Kaplan-Meier survival curves demonstrated significant inter-group survival differences stratified by these five laboratory indicators at their optimal cut-off values (Figure 3).

Independent predictors of a poor outcome

Multivariable Cox regression analysis was conducted to identify independent prognostic factors for 10-day in-hospital all-cause mortality in ECPR-treated CA patients, with key results illustrated in Figure 2. D-Dimer > 8.07 was identified as an independent risk factor (HR = 1.487, 95%CI: 1.009 - 2.194, $P = 0.045$), indicating that mortality risk of patients with D-Dimer > 8.07 increased 48.7% compared with those with D-Dimer ≤ 8.07 . LMR > 8.86 (HR = 1.561, 95%CI: 1.033 - 2.358, $P = 0.034$) was also identified as an independent risk factor. Additionally, elevated serum potassium (HR = 1.267, 95%CI: 1.065 - 1.508, $P = 0.008$), serum sodium (HR = 1.033, 95%CI:

1.007 - 1.060, $P = 0.011$) and high INR (HR = 1.227, 95%CI: 1.009 - 1.492, $P = 0.041$) were also independent risk factors.

Development and External Validation of the Prognostic Nomogram

Based on the five independent prognostic factors screened via multivariate Cox regression analysis (D-Dimer, serum potassium, serum sodium, INR, and LMR), a visual prognostic nomogram was constructed to predict the short-term survival probability of CA patients after ECPR treatment. Among 5-, 10- and 15-day endpoints, the nomogram achieved optimal discrimination for 10-day in-hospital mortality (AUC=0.793, 95%CI 0.660-0.926), while the predictive performance declined for 5-day (AUC=0.703) and 15-day (AUC=0.777) mortality(Figure 4). External validation using an independent cohort demonstrated good predictive performance of the established nomogram for 10-day in-hospital mortality, confirming its robust clinical utility for prognostic assessment. The calibration curves demonstrated agreement between the predicted and observed survival probabilities in both the training and external validation cohorts at 5, 10, and 15 days (Figure 5).

Sensitivity analysis

Imputation was performed on the original data in this study, followed by sensitivity analysis on the imputed dataset. The results revealed no statistically significant differences between the data before and after imputation, suggesting that they could be considered derived from the same population, as demonstrated in Supplementary Tables 1 and 2.

Discussion

CA remains a critical global public health challenge, and ECPR is currently recognized as a beneficial intervention for patients with refractory CA unresponsive to conventional cardiopulmonary resuscitation. Nevertheless, contemporary evidence indicates that post-ECPR survival remains suboptimal, ranging from 29.0% to 33.6%⁴. In this context, we identified several readily available laboratory parameters that enable early prognostic assessment in this population, which holds practical significance for risk stratification and the formulation of individualized treatment strategies.

Given that the 10-day in-hospital mortality endpoint differs from conventional

endpoints widely-used in previous ECPR investigations, its rationale deserves further elaboration. In contrast to widely-used endpoints including 28-day, 30-day or hospital-discharge mortality in ECPR research, the present study adopted a 10-day in-hospital mortality endpoint. The first 10 days constitute the highest-risk phase after ECPR, dominated by post-cardiac-arrest syndrome including coagulation derangement, myocardial stunning, acute kidney injury and hypoxic-ischemic brain injury¹³. Deaths occurring after day-10 are more likely driven by secondary complications such as hospital-acquired infection, which are less well predicted by admission baseline laboratory parameters. The comparative ROC analysis in our cohort showed that baseline D-dimer, INR, serum potassium, serum sodium and LMR achieved the best discriminative ability for 10-day rather than 5- or 15-day mortality. Therefore, this nomogram is intended for early risk stratification at the time of ECPR initiation, to support clinical decision-making for the initial 10-day critical period, rather than to predict long-term or discharge outcomes.

Our study identified elevated D-dimer as an independent risk factor for 10-day in-hospital mortality in CA patients. A study by Szymanski et al. on patients with CA confirmed that high D-dimer levels are an independent predictor of 30-day all-cause mortality, and D-dimer levels in the death group were significantly higher than those in the Non-survival group¹⁷. Asano et al. further demonstrated a significant positive correlation between D-dimer levels and duration of ischemia-hypoxia and the severity of arrest¹⁸. Our study aligns with previous investigations. The underlying mechanism likely involves systemic ischemia-reperfusion injury following CA, which triggers extensive endothelial damage and coagulation cascade activation, resulting in a sharp elevation of D-dimer. Prolonged CA leads to insufficient perfusion of vital organs, including the heart and brain, ultimately culminating in death.

This study found that an elevated INR is also an independent risk factor for early mortality in patients with CA (HR = 1.620, 95%CI: 1.225 - 2.143, $P = 0.001$) with each 1-unit increase conferring a 62.0% increment in mortality risk.

Increased INR directly reflects impaired hepatic synthetic function, insufficient systemic perfusion, and coagulation disorders, which significantly increase the risks of hemorrhage, thrombosis, and multiple organ failure^{19–22}. Several prognostic models for cardiovascular diseases have also incorporated INR into scoring systems. High INR level is associated with poor outcomes in patients with acute vertebrobasilar artery occlusion and significantly increase mortality following thrombectomy^{23,24}. Our findings reinforce the value of INR as a pre-ECPR prognostic indicator and underscore the importance of early coagulation monitoring and optimization.

In addition, we found that elevated serum potassium (HR = 1.336, 95%CI: 1.173 - 1.522, $P < 0.001$) and high serum sodium (HR = 1.064, 95%CI: 1.036 - 1.092, $P < 0.001$) emerged as independent mortality predictors, with each 1 mmol/L increase associated with a 33.6% and 6.4% increase in death risk, respectively. Tissue ischemia, rhabdomyolysis, and impaired renal function after CA are important causes of elevated serum potassium and sodium, which can directly induce malignant arrhythmias and circulatory collapse^{25,26}. Increased potassium and sodium in the early stage of acute kidney injury are significantly associated with poor prognosis, indirectly supporting this view^{27–29}.

Notably, LMR was identified as an independent predictor of all-cause mortality in our cohort. LMR is a composite marker reflecting both the inflammatory and immune status of the host. Previous studies have shown that higher LMR levels are associated with an 8.3% reduction³⁰ in the probability of return of spontaneous circulation after CA and an increased risk of early mortality. Our data demonstrated that elevated LMR was associated with increased mortality risk. Excessive lymphocyte activation and monocyte depletion or compensatory reduction can exacerbate tissue damage, impair the body's ability to clear inflammation, and increase the risk of secondary immune paralysis, infection, and multiple organ dysfunction, ultimately leading to increased mortality. Accumulating evidence has established the prognostic value of LMR in various cardiovascular diseases, including acute coronary syndrome, heart failure, and post-cardiopulmonary bypass populations³¹.

Several ECPR prognostic models have been developed to date. The RESCUE-IHCA score incorporates age, initial rhythm, duration of CA, and a history of renal insufficiency to predict in-hospital mortality, with an AUC of 0.72 in the derivation cohort and 0.68 in external validation³². Another Pre-ECPR score combines age, initial rhythm, CA duration, pupil dilation, and arterial pH to predict favorable neurological outcomes, achieving an AUC of 0.87³³.

However, most existing models are oriented toward discharge survival or 30-day neurological prognosis, with limited attention to the early in-hospital mortality window. Our study fills this research gap and constructed a risk prediction model for 10-day early in-hospital mortality in patients using only five routinely available clinical indicators. This model could serve as a useful reference for making real-time treatment decisions.

Finally, this study has several limitations. First, the retrospectively observational design may introduce selection bias; despite the implementation of strict inclusion and exclusion criteria, confounding factors may still influence the stability of the study results. Second, limited sample size may restrict the extrapolation of the findings. Third, further large-scale multicenter prospective studies are required to externally validate the model and confirm its widespread clinical applicability. Fourth, the primary endpoint of 10-day in-hospital mortality is not a universally standardized endpoint for ECPR studies. Although we provided both clinical and internal statistical rationale for this choice, external validation in independent cohorts is required to confirm the generalizability of the model for this specific early-phase endpoint. The model should not be extrapolated to predict 28-day or discharge survival without further validation.

Conclusion

In summary, we constructed and externally validated a concise nomogram incorporating five routinely available laboratory indicators—D-dimer, INR, serum potassium, serum sodium, and LMR—for predicting 10-day in-hospital mortality in CA patients treated with ECPR. This tool facilitates early risk stratification during the critical 10-day post-ECPR period and may assist clinicians in making timely, individualized decisions. Further validation in multicenter cohorts is needed to confirm the generalizability of these findings.

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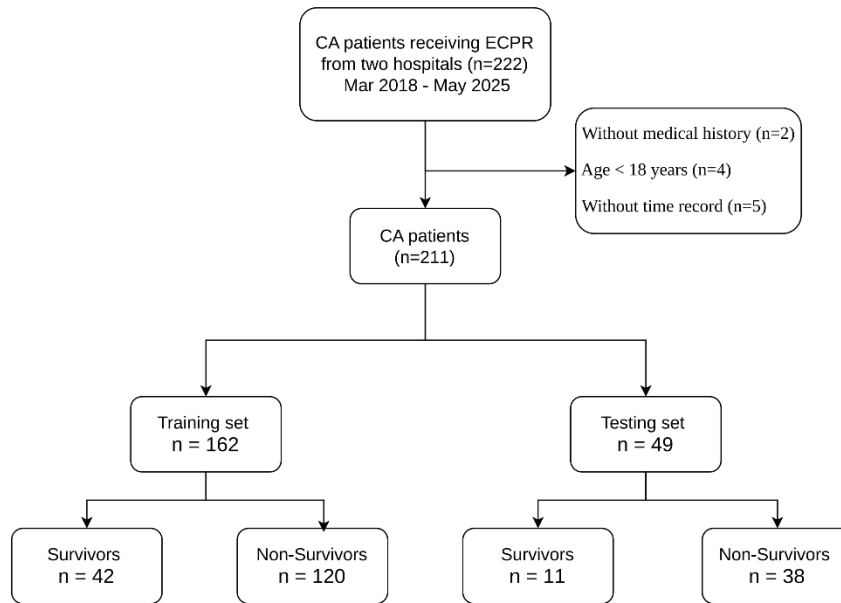


Figure 1: Flowchart of data extraction and study design.

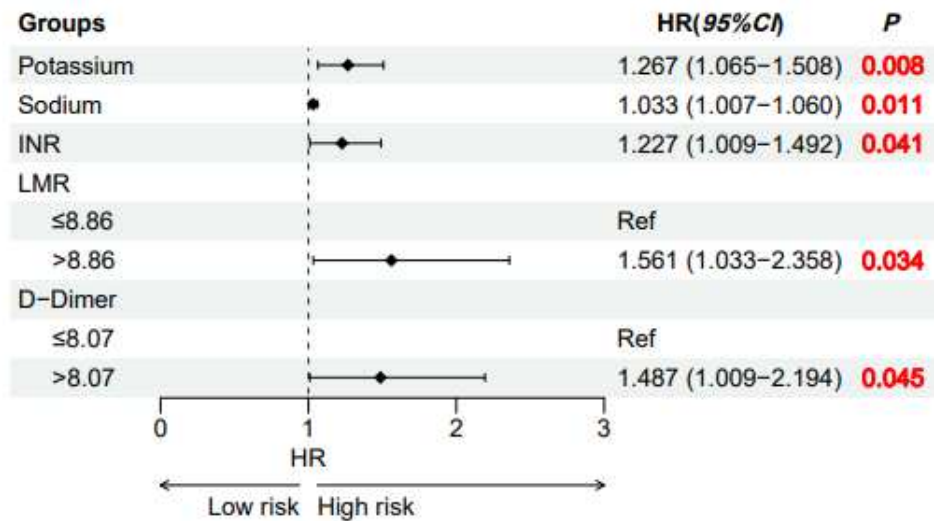


Figure 2: Multivariate Cox analysis regression results.

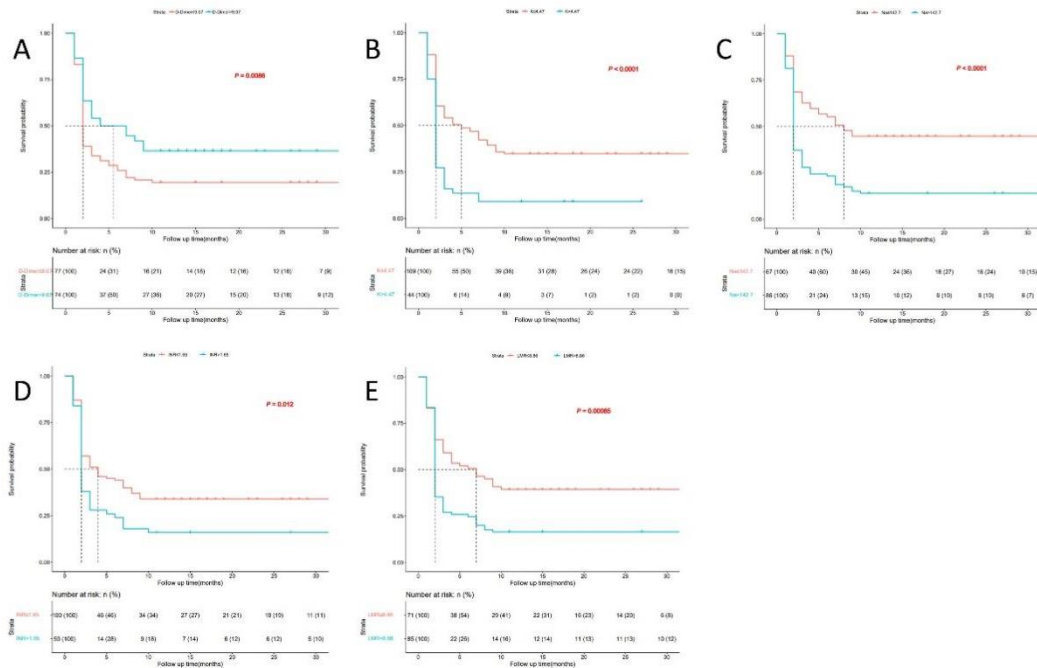


Figure 3: Kaplan-Meier survival curves for the independent prognostic factors. (A) D-dimer; (B) Serum potassium; (C) Serum sodium; (D) INR; (E) Lymphocyte-to-monocyte ratio (LMR).

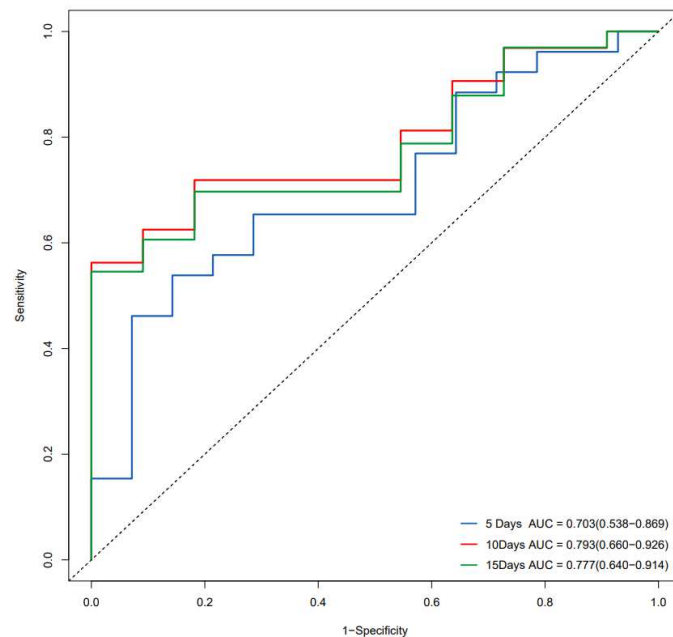


Figure 4: ROC curves of the nomogram for predicting 5-day, 10-day and 15-day in-hospital mortality in the external validation cohort.

5-day: AUC = 0.703 (95% CI 0.538 - 0.869); 10-day: AUC = 0.793 (95% CI 0.660 - 0.926); 15-day: AUC = 0.777 (95% CI 0.640 - 0.914).

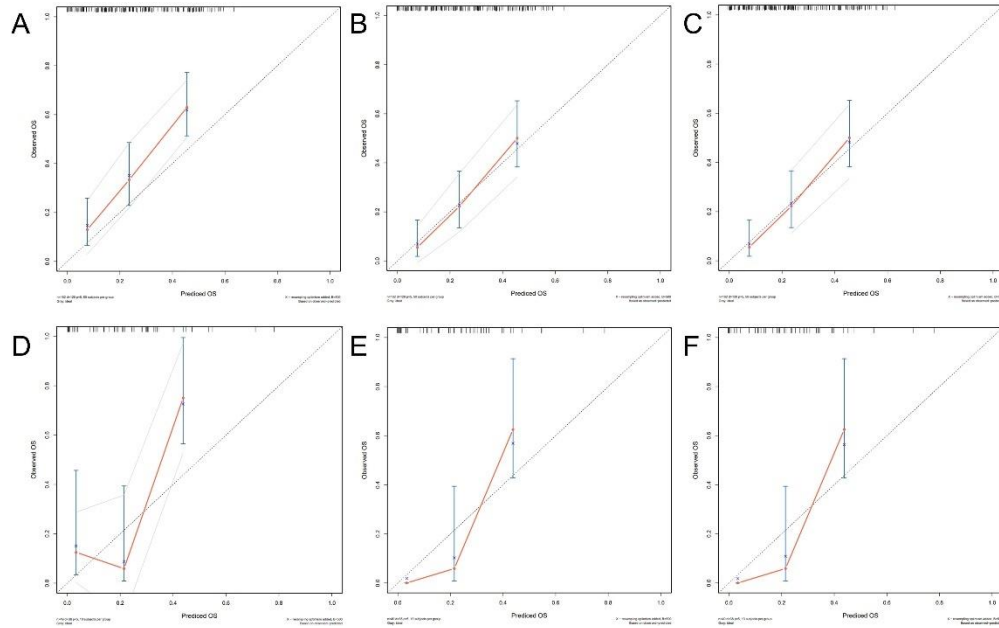


Figure 5. Calibration curves of the prognostic nomogram for predicting survival at different time points. Panels A, B, and C show the calibration curves for the training set at 5, 10, and 15 days, respectively. Panels D, E, and F show the calibration curves for the test set at 5, 10, and 15 days, respectively. The diagonal dashed line represents ideal prediction, and the solid line indicates the actual model performance.

Table 1. Patient characteristics

Variables	Total (N = 211)	Test (N = 49)	Train (N = 162)	Statistics	P
Outcome				$\chi^2 = 0.092$	0.761
Survival	53 (25.12)	11 (22.45)	42 (25.93)		
All-cause mortality	158 (74.88)	38 (77.55)	120 (74.07)		
Survival time, M (Q ₁ , Q ₃)	2.00 (2.00 - 10.50)	3.00 (2.00 - 7.00)	2.00 (2.00 - 11.00)	W = 3962.5	0.987
Gender, n (%)				$\chi^2 = 0.116$	0.733
Male	161 (76.3)	36 (73.47)	125 (77.16)		
Female	50 (23.7)	13 (26.53)	37 (22.84)		
Age(year), Mean $\pm$ SD	58.67 $\pm$ 13.58	60.00 $\pm$ 15.20	58.27 $\pm$ 13.07	t = 0.783	0.435
CRRT, n (%)				$\chi^2 = 0.640$	0.424
No	59 (27.96)	11 (22.45)	48 (29.63)		
Yes	152 (72.04)	38 (77.55)	114 (70.37)		
hs-CRP (mg/L), n (%)				$\chi^2 = 0.352$	0.838
≤ 5	131 (62.09)	30 (61.22)	101 (62.35)		
5-10	30 (14.22)	7 (14.29)	23 (14.2)		
≥ 10	44 (20.85)	12 (24.49)	32 (19.75)		
Hypertension				$\chi^2 = 0.376$	0.540
No	92 (43.6)	19 (38.78)	73 (45.06)		
Yes	119 (56.4)	30 (61.22)	89 (54.94)		
ANC, M(Q ₁ , Q ₃)	8.23 $\pm$ 7.38	7.52 $\pm$ 5.35	8.45 $\pm$ 7.91	t' = -0.929	0.355
AMC, M(Q ₁ , Q ₃)	0.44 (0.26 - 0.69)	0.45 (0.28 - 0.86)	0.44 (0.26 - 0.65)	W = 4202.5	0.294
WBC (10 ⁹ /L), Mean $\pm$ SD	12.20 (9.62 - 17.02)	11.63 (9.84 - 15.08)	12.89 (9.60 - 17.94)	W = 3455.5	0.312
ALC (10 ⁹ /L), Mean $\pm$ SD	4.31 $\pm$ 2.63	4.47 $\pm$ 2.66	4.26 $\pm$ 2.63	t = 0.503	0.616
RBC (10 ¹² /L), Mean $\pm$ SD	4.05 $\pm$ 0.92	3.95 $\pm$ 1.00	4.09 $\pm$ 0.89	t = -0.912	0.363
LMR, M (Q ₁ , Q ₃)	9.27 (4.62 - 17.67)	7.52 (4.83 - 14.41)	9.79 (4.32 - 19.03)	W = 3539	0.435
Hb (g/L), Mean $\pm$ SD	123.56 $\pm$ 29.62	120.28 $\pm$ 32.20	124.59 $\pm$ 28.80	t = -0.889	0.375
PLT (10 ⁹ /L), Mean $\pm$ SD	166.04 $\pm$ 80.00	156.81 $\pm$ 79.00	168.94 $\pm$ 80.35	t = -0.925	0.356
AST (U/L), M(Q ₁ , Q ₃)	151.50 (61.00 - 322.50)	110.00 (56.00 - 238.00)	162.00 (69.00 - 367.30)	W = 3408	0.340
Potassium (mmol/L), Mean $\pm$ SD	4.26 $\pm$ 1.35	4.71 $\pm$ 1.98	4.12 $\pm$ 1.05	t' = 1.988	0.052
Sodium (mmol/L), Mean $\pm$ SD	143.98 $\pm$ 7.42	144.66 $\pm$ 7.42	143.77 $\pm$ 7.43	t = 0.734	0.464
Calcium (mmol/L), Mean $\pm$ SD	2.05 $\pm$ 0.29	2.06 $\pm$ 0.29	2.04 $\pm$ 0.28	t = 0.366	0.714
BUN (mmol/L), M	6.58 (5.38 - 8.51)	6.49 (5.69 - 8.49)	6.73 (5.24 - 8.55)	W = 3757	0.982

Variables	Total (N = 211)	Test (N = 49)	Train (N = 162)	Statistics	P
(Q ₁ , Q ₃)					
SCr (μmol/L), M (Q ₁ , Q ₃)	110.40 (88.31 - 140.78)	111.00 (89.00 - 137.10)	110.10 (88.00 - 143.20)	W = 3710.5	0.916
Diabetes mellitus, n (%)				χ ² = 0.604	0.437
No	141 (66.82)	30 (61.22)	111 (68.52)		
Yes	70 (33.18)	19 (38.78)	51 (31.48)		
Chlorine (μmol/L), Mean ± SD	101.52 ± 5.70	100.80 ± 4.75	101.76 ± 5.96	t = -1.022	0.308
LDH (U/L), M (Q ₁ , Q ₃)	492.50 (286.50 - 1086.00)	491.00 (264.00 - 959.00)	494.00 (287.50 - 1302.50)	W = 3311.5	0.271
CK (U/L), M (Q ₁ , Q ₃)	178.00 (105.00 - 534.00)	152.00 (83.00 - 276.00)	202.50 (106.25 - 704.25)	W = 3012	0.058
Hyperlipidemia, n (%)				-	0.464
No	200 (94.79)	48 (97.96)	152 (93.83)		
Yes	11 (5.21)	1 (2.04)	10 (6.17)		
CK-MB (U/L), M (Q ₁ , Q ₃)	168.00 (99.50 - 527.00)	146.00 (73.25 - 270.25)	175.00 (102.25 - 569.50)	W = 2885	0.093
Myo, M (Q ₁ , Q ₃)	434.00 (181.00 - 1194.00)	332.30 (200.30 - 843.68)	515.25 (176.97 - 1553.25)	W = 3029	0.139
CTnT (pg/mL), Mean ± SD	60.74 (11.96 - 566.05)	105.40 (17.35 - 567.20)	47.02 (10.21 - 562.95)	W = 3820	0.307
PO ₂ (mmHg), Mean ± SD	179.93 ± 170.57	172.00 ± 162.60	182.68 ± 173.73	t = -0.377	0.707
INR, M (Q ₁ , Q ₃)	1.34 (1.15 - 1.76)	1.25 (1.15 - 1.51)	1.38 (1.15 - 1.80)	W = 2866	0.186
D-dimer, Mean ± SD	18.80 ± 24.94	16.94 ± 23.13	19.40 ± 25.54	t = -0.599	0.550
TT (S), Mean ± SD	15.40 (13.30 - 20.45)	14.62 (13.30 - 17.50)	15.95 (13.30 - 20.88)	W = 3269.5	0.247
pH, Mean ± SD	7.14 ± 0.23	7.15 ± 0.25	7.14 ± 0.23	t = 0.346	0.729

Notes: SD: Standard Deviation; M: Median; Q₁: 1st Quartile; Q₃: 3rd Quartile; t: Student's t test; t': Satterthwaite t test; W: Wilcoxon rank sum test; χ²: Chi-square test; -: Fisher's exact test; F: Analysis of Variance; H: Kruskal-Wallis H test; χ²: Chi-square test; -: Fisher's exact test.

Abbreviations: CRRT, continuous renal replacement therapy; hs-CRP, high-sensitivity C-reactive protein; ANC, Absolute neutrophil count; AMC, absolute monocyte count; WBC, white blood cell count; ALC, Absolute lymphocyte count; RBC, red blood cell count; LMR, lymphocyte-to-monocyte ratio; Hb, hemoglobin; PLT, platelet; AST, aspartate aminotransferase; BUN, Blood urea nitrogen; SCr, serum creatinine; LDH, lactate dehydrogenase; CK, creatine kinase; CK-MB, Creatine kinase-MB; Myo, myoglobin; cTnT, Cardiac Troponin T; PO₂, partial pressure of oxygen; INR, international normalized ratio; TT, thrombin time.

Table 2. Univariate COX regression analysis

Variables	Outcome/Total	HR (95% CI)	P
Gender			
Male	N=93/125	Ref	
Female	N=27/37	0.978 (0.637 - 1.501)	0.917
Age	N=120/162	1.005 (0.992 - 1.019)	0.431
hs-CRP			
≤5	N=84/106	Ref	
5-10	N=16/24	0.741 (0.433 - 1.266)	0.272
≥10	N=20/32	0.685 (0.420 - 1.117)	0.129
Hb	N=120/162	0.995 (0.989 - 1.001)	0.076
AST	N=120/162	1.000 (1.000 - 1.000)	0.871
Potassium	N=120/162	1.286 (1.089 - 1.519)	0.003
Sodium	N=120/162	1.054 (1.030 - 1.079)	<0.001
Calcium	N=120/162	0.821 (0.419 - 1.607)	0.565
BUN	N=120/162	0.983 (0.942 - 1.026)	0.424
SCr	N=120/162	1.001 (0.999 - 1.002)	0.365
LMR			
≤ 8.86	N=45/73	Ref	
> 8.86	N=75/89	1.958 (1.347 - 2.846)	<0.001
Diabetes			
No	N=85/111	Ref	
Yes	N=35/51	0.876 (0.591 - 1.299)	0.510
Hypertension			
No	N=58/73	Ref	
Yes	N=62/89	0.878 (0.614 - 1.256)	0.477
Hyperlipidemia			
No	N=113/152	Ref	
Yes	N=7/10	0.818 (0.381 - 1.757)	0.607
Chlorine	N=120/162	1.001 (0.970 - 1.032)	0.967
LDH	N=120/162	1.000 (1.000 - 1.000)	0.240
CK	N=120/162	1.000 (1.000 - 1.000)	0.725
INR	N=121/162	1.620 (1.225 - 2.143)	0.001
CK-MB	N=120/162	1.000 (1.000 - 1.000)	0.909
cTnT	N=120/162	1.000 (1.000 - 1.000)	0.123
PO ₂	N=120/162	1.001 (1.000 - 1.002)	0.248
pH	N=120/162	0.501 (0.218 - 1.149)	0.103
D-Dimer			
<8.07	N=52/82	Ref	
>8.07	N=68/80	1.917 (1.331 - 2.761)	<0.001

Notes: HR: Hazard ratio; CI: Confidence intervals; Ref: reference

Supplementary Tables 1 Missing data and Sensitivity analysis in the training set

Variables	Missing data		Sensitivity analysis			
	Missing, n	Missing, %			Statistics	P
hs-CRP	6	3.7%	156 (96.30)	162 (100)	$\chi^2 = 0.029$	0.986
WBC	6	3.7%	12.89 (9.60 - 17.94)	12.89 (9.69 - 17.54)	W = 12637	1.000
ANC	6	3.7%	8.45 $\pm$ 7.91	8.38 $\pm$ 7.78	t = 0.073	0.942
AMC	6	3.7%	0.44 (0.26-0.65)	0.43 (0.26-0.64)	W = 12661.5	0.976
ALC	6	3.7%	4.26 $\pm$ 2.63	4.26 $\pm$ 2.60	t = -0.027	0.978
RBC	6	3.7%	4.09 $\pm$ 0.89	4.07 $\pm$ 0.88	t = 0.127	0.899
Hb	6	3.7%	124.59 $\pm$ 28.80	124.55 $\pm$ 28.36	t = 0.013	0.990
PLT	6	3.7%	168.94 $\pm$ 80.35	168.17 $\pm$ 79.28	t = 0.085	0.932
AST	9	5.56%	162.00 (69.00 - 367.30)	169.70 (71.38 - 399.00)	W = 12088.5	0.707
Calcium	9	5.56%	2.04 $\pm$ 0.28	2.04 $\pm$ 0.28	t = -0.009	0.993
BUN	9	5.56%	6.73 (5.24 - 8.55)	6.74 (5.30 - 8.54)	W = 12335.5	0.944
SCr	9	5.56%	110.10 (88.00 - 143.20)	113.05 (89.00 - 142.90)	W = 12121.5	0.737
Chloride	10	6.17%	101.76 $\pm$ 5.96	101.76 $\pm$ 5.83	t = -0.008	0.993
LDH	11	6.79%	494.00 (287.50 - 1302.50)	522.50 (300.90 - 1265.75)	W = 12081.5	0.852
CK	12	7.41%	202.50 (106.25 - 704.25)	215.50 (112.50 - 771.00)	W = 11801	0.662
CK-MB	12	7.41%	175.00 (102.25 - 569.50)	201.80 (104.50 - 642.25)	W = 11835	0.693
Myo	18	11.11%	-	-	-	-
cTnT	20	12.35%	-	-	-	-
PO ₂	21	12.96%	-	-	-	-
TT	22	13.58%	-	-	-	-
pH	22	13.58%	-	-	-	-

Supplementary Tables 2 Missing data and Sensitivity analysis in the testing set

Variables	Missing data		Sensitivity analysis			
	Missing, n	Missing, %			Statistics	P
hs-CRP	1	1.85%	53 (98.15)	54 (100.00)	$\chi^2 = 0.006$	0.997
Diabetes	1	1.85%	53 (98.15)	54 (100.00)	$\chi^2 = 0.000$	1.000
Hyperlipidemia	1	1.85%	53 (98.15)	54 (100.00)	-	1.000
AMC	2	3.7%	0.57 $\pm$ 0.37	0.57 $\pm$ 0.36	t = -0.051	0.959
AST	2	3.7%	105.50 (47.00-227.50)	108.00 (47.75-231.20)	W = 1382	0.892
WBC	3	5.56%	13.04 $\pm$ 6.35	12.97 $\pm$ 6.18	t = 0.055	0.956

Variables	Missing data		Sensitivity analysis			
	Missing, n	Missing, %			Statistics	P
ANC	3	5.56%	7.65 ± 5.36	7.60 ± 5.23	t = 0.043	0.966
ALC	3	5.56%	4.51 ± 2.73	4.47 ± 2.67	t = 0.071	0.944
Hb	3	5.56%	119.98 ± 32.24	119.62 ± 31.41	t = 0.058	0.954
PLT	3	5.56%	157.27 ± 78.78	159.34 ± 77.38	t = -0.136	0.892
Potassium	3	5.56%	4.64 ± 1.97	4.61 ± 1.93	t = 0.076	0.940
Sodium	3	5.56%	143.45 ± 9.65	143.24 ± 9.44	t = 0.112	0.911
Calcium	3	5.56%	2.06 ± 0.30	2.06 ± 0.29	t = 0.035	0.972
BUN	3	5.56%	6.37 (5.55 - 7.86)	6.41 (5.62 - 7.98)	W = 1360	0.916
SCr	3	5.56%	110.50 (83.60 - 136.75)	107.75 (85.07 - 135.25)	W = 1380.5	0.985
Chloride	3	5.56%	99.96 ± 7.17	99.92 ± 6.99	t = 0.032	0.975
LDH	3	5.56%	485.00 (226.50 - 955.00)	439.00 (236.25 - 919.10)	W = 1369.5	0.964
CK	3	5.56%	151.00 (76.50 - 283.00)	151.50 (80.00 - 279.75)	W = 1373.5	0.985
CK-MB	3	5.56%	141.00 (73.50 - 278.50)	141.00 (73.50 - 278.50)	W = 1300.5	1.000
RBC	4	7.41%	3.91 ± 1.00	3.93 ± 0.97	t = -0.092	0.927
TT	7	12.96%	-	-	-	-
PO ₂	7	12.96%	-	-	-	-
pH	7	12.96%	-	-	-	-
Myo	8	14.81%	-	-	-	-
cTnT	10	18.52%	-	-	-	-

Full Title

A Nomogram for Predicting 10-Day In-Hospital Mortality in Cardiac Arrest Patients Treated With ECPR: Development and External Validation

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Ethics Approval

This retrospective study obtained ethical approval from two participating medical institutions:

The Affiliated Huai'an Hospital of Xuzhou Medical University (Approval number: HEYLL202568)

Huai'an First Hospital Affiliated to Nanjing Medical University (Approval number: KY-2026-129-01) All procedures followed the Declaration of Helsinki. Written informed consent was waived due to anonymized retrospective data analysis, and all patient information was strictly confidential.

Data Availability Statement

The data sets analyzed during the current study are available from the corresponding author upon reasonable request.

Patient and Public Involvement

Patients and/or the public were not involved in the design, conduct, reporting, or dissemination plans of this research.

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Author Contributions

All authors made substantial contributions to this study. Yong Liang conceptualized the study, designed the research protocol, supervised the entire project and finalized the article. Guanyu Xiao performed data collection, statistical analyses (including LASSO regression and multivariate Cox regression), and drafted the initial manuscript. Chang Li participated in model development, validated the nomogram, and interpreted the clinical implications. Jie Wang contributed to data curation, reviewed laboratory parameters and clinical records, and assisted in revising the manuscript. Yan Shi, Haichen Yang and Ji Fu critically revised the manuscript for important intellectual content, verified the rationality of data calibration, validated the reliability of scoring system applications. All authors have read and agreed to the published version of the manuscript, and are accountable for all aspects of the work to ensure that questions related to the accuracy or integrity of any part of the study are appropriately investigated and resolved.

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The authors report no conflicts of interest in this work.