

The predictive value of four severity scores for 28-day mortality in patients with cardiac arrest in the ICU: a retrospective study based on MIMIC ☒

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Abstract

Survival of treated patients with cardiac arrest (CA) in the intensive care unit (ICU) remains low, and the Oxford Severity of Illness Score, Simplified Acute Physiology Score II (SAPS II), Systemic Inflammatory Response Syndrome criteria critically ill patients, but the cardiac arrest patients have not been studied. However, the results of studies in which different severity scores predict survival status in patients with cardiac arrest are unclear. The purpose of this study is to evaluate the effectiveness of the OASIS, SAPS II, SIRS, and SOFA scores in predicting 28-day mortality among patients with cardiac ogenic shock (CS) in the intensive care unit (ICU), and to identify the scoring system with the highest diagnostic value.

Methods: This study screened patients defined as CA from the MIMIC Ⅲ database. The OASIS, SAPS II, SIRS, and SOFA scores was divided into low, medium and high groups based on the Jorden Index. Statistical methods used included univariate and multivariate Cox regression analysis, ROC curves, propensity score matching, and generalized linear model analysis with inverse probability weighting, and patients identified with CA by ICD-9 codes were included in the sensitivity analysis.

Results: This study included a total of 2,535 ICU patients following cardiac arrest, of whom 1,068 (42.1%) died within 28 days. Univariate analysis revealed that age, sex, multiple laboratory parameters, and four severity scores (OASIS, SAPS II, SIRS, SOFA) were all significantly associated with 28-day mortality (all $P < 0.05$). Multivariate Cox regression analysis further confirmed that blood urea nitrogen (HR = 1.003, $P = 0.034$), lactate (HR = 1.076, $P < 0.001$), and bilirubin (HR = 1.021, $P = 0.044$) were independent risk factors, while albumin (HR = 0.817, $P < 0.001$) and PO_2 (HR = 0.999, $P < 0.001$) were independent protective factors. Among the four scoring systems, OASIS (HR = 1.017, $P < 0.001$) and SAPS II (HR = 1.018, $P < 0.001$) retained independent predictive ability in the multivariate model, whereas SIRS and SOFA lost statistical significance. Stratified analysis showed that the moderate and high groups of OASIS, SAPS II, and SIRS were all significantly associated with 28-day mortality (all $P < 0.001$), whereas none of the SOFA groups showed a significant association. Sensitivity analysis (patients with cardiac arrest) and inverse probability weighting analysis further confirmed the robustness of these results. ROC curve analysis indicated that the predictive performance of SAPS II was slightly better than that of OASIS ($P = 0.023$), but the AUC for both was below 0.7.

Dicussion:

Conclusion: The OASIS, SAPS II, and SIRS scores are robust independent predictors of 28-day mortality in ICU patients following cardiac arrest, whereas the predictive performance of the SOFA score has been inconsistent across different analytical methods. The predictive ability of SAPS II is slightly superior to that of OASIS, but both are only moderate. The clinical predictive value of a single scoring system is limited; therefore, it is recommended to combine multiple scoring systems with key laboratory parameters (particularly lactate and albumin) for a comprehensive prognostic assessment.

Introduction

Cardiac arrest (CA) is a major public health problem that threatens human life, with 26.7% of adults surviving to hospital discharge after in-hospital cardiac arrest (IHCA) and only 10.5% of out-of-hospital cardiac arrest (OHCA) patients, according to a recent report by the American Heart Association^[1]. Although many studies have reported on the treatment and prognosis of patients with OHCA and IHCA^[2-4], few have examined the survival and prognosis of patients with post-CA who are admitted to the intensive care unit (ICU) after treatment^[5-7]. Currently, despite updated international guidelines and continuous medical developments, most CA patients have a high short-term mortality rate even if they are resuscitated and survive until discharged from the ICU. The simple laboratory score obtained by CA patients at admission is a useful tool to assess their prognosis^[6]. Assessing the physiological status of patients based on early validated scores at ICU admission, predicting patient mortality risk, and using scoring systems to accurately guide patients' subsequent treatment is a major focus of current researchers^[8].

At present, there are many relevant scoring systems used in the ICU to diagnose sepsis and predict patient prognosis, but validated scoring systems specifically for patients with CA remain unstudied and undeveloped. For this situation, several studies have applied scoring systems commonly used in the ICU to the prediction of survival status of CA patients after treatment^[9-12]. Scoring systems: Oxford Severity of Illness Score (OASIS), which is highly correlated with ICU and hospital mortality, is a new score based on the Acute Physiology and Chronic Health Evaluation II (APACHE II) score-the most commonly used ICU ward disease severity scoring system^[13]-with an optimized number of parameters without loss of predictive accuracy^[14]. The Simplified Acute Physiology Score II (SAPS II) is calculated from some of the patient's physiological indicators, and it's often used in the ICU to assess the severity of illness in critically ill patients^[15]. Systemic inflammatory response syndrome (SIRS) criteria have been described as clinical manifestations of the host response to inflammation and have long been used as a screening tool for sepsis in hospitalized patients^[16]. The Sequential Organ Failure Score (SOFA) is a scoring system to assess the severity of multi-organ dysfunction^[17], and it was originally designed to be used in the assessment of patients with sepsis to help identify the risk of death in patients with sepsis^[18]. Currently, many researchers explore the assessment of these scoring systems for the prognosis of different types of ICU patient treatment, but most of them study a single scoring system or compare the value ability of two scoring systems in predicting the prognosis of different types of patients. Moreover, few studies have examined the application of different scoring systems in the ICU in assessing the prognosis and survival status of patients with CA. Therefore, the purpose of our study was to evaluate whether the four scores, OASIA, SAPS II, SIRS, and SOFA, were effective in predicting 28-day mortality in patients with CA in the ICU, as well as to compare the predictive accuracy of the different scoring systems in determining patient survival, and to screen the scoring system with the highest diagnostic value to guide and recommend the appropriate clinical therapeutic schemes.

Methods

2.1 Data source

The data for this study were provided from a large, freely available public database in the United States known as the Medical Information Mart for Intensive Care $\times$ (MIMIC $\times$) database^[19]. The database was developed by the Massachusetts Institute of Technology (MIT) Physiological Computing Laboratory, who collected and organized data on more than 60,000 patients with complete information from the intensive care unit at Beth Israel Deaconess Medical Center (BIDMC) in Massachusetts between 2001 and 2022. Data include : vital signs, medications, laboratory measurements, observations and records drawn by care providers, fluid balance, procedure codes, diagnosis codes, imaging reports, length of stay, and survival data. The MIMIC $\times$ database supports applications including academic and industrial research, quality improvement programs, and higher education courses. We obtained the permission from the PhysioNet website to gain access to this database and extract relevant patient information through PostgreSQL software^[20]. The project was approved by the Institutional Review Board of MIT and BIDMC and did not require additional ethics approval.

2.2 Subject

Diagnostic criteria for patients with CA in this project were defined as patients with a diagnosis of CA according to International Classification of Diseases, Ninth Revision (ICD-9) codes, and mean arterial pressure (MAP) below 20 mmHg within 24 hours of admission to the ICU. We included only patients who were admitted to the ICU for the first time. Inclusion criteria: Diagnosed as CA; MAP < 20 mmHg within 24 hours after entering ICU. Exclusion criteria: Age < 18 years old; ICU length of stay < 24 hours. Finally, a total of 2,535 cases were selected in this study.

2.3 Data extraction

The data was extracted using structured query language (SQL) through pgAdmin 4 PostgreSQL 9.6 software, including: age, gender, scores, laboratory parameters, patient entry and exit time of ICU, and time of death. The scores included OASIS, SAPS II, SIRS and SOFA. Laboratory parameters include: potassium ion, sodium ion, calcium ion, bicarbonate ion, anion gap, urea nitrogen, lactate, creatinine, albumin, glucose, glutamate transaminase, glutamic oxalacetic transaminase, troponin t, creatine kinase, bilirubin, PH, PO₂, PCO₂, white blood cells, red blood cells, hemoglobin, erythrocyte specific volume, platelets, and prothrombin time.

The database changed the birth dates of patients aged > 89 years old to protect patient privacy, which were advanced to 300 years old before admission, and we adjusted these patients' ages back to their true ages before conducting statistical analyses. The length of ICU stay is equal to the time spent leaving the ICU minus the time spent entering the ICU. The Jorden index for each score was calculated to determine their optimal threshold and grouped according to the optimal threshold, with each of the four scores being classified into three groups: Low (OASIS, < 39; SAPS II, < 43; SIRS, < 3; SOFA, < 6), Medium (OASIS, 39–47; SAPS II, 43–62; SIRS, = 3; SOFA, 6–10) and High (OASIS, > 47; SAPS II, > 62; SIRS, = 4; SOFA, > 10).

2.4 Outcomes

The outcome of our study was the 28-day mortality rate of CA patients after entering the ICU.

2.5 Statistical analysis

Continuous variables in the results of this study are presented as mean $\pm$ standard deviation if they conform to a normal distribution and are compared between groups using t-test, median and interquartile spacing if they do not conform to a normal distribution, Wilcoxon rank sum test for comparison between groups and numbers and percentages for categorical variables, as well as chi-square test or Fisher exact test for comparison between groups. Correlation analyses were performed on the four scores to compare the strength of their correlations with patients' 28-day mortality, and the results are shown as R-squared. Univariate cox regression analysis was performed to test the effect of various covariates on final patient outcomes. We excluded covariates that were not statistically significant in the univariate cox regression model, and the remaining variables continued to be included in the multivariate cox regression analysis model to compare scores of different severities to assess the predictive power of patients' 28-day mortality, with results expressed as hazard ratios and 95% confidence intervals. ROC curves for the two scores were plotted and the area under the curve was compared to determine the association between scores and outcomes, after which Kaplan-Meier curves for different subgroups of each score were plotted to observe the change in patient survival. We included covariates that had an impact on patients' survival outcomes in a propensity score matching model based on a multifactorial cox regression analysis to reduce the impact of data bias and confounding variables on the results, and we subsequently used the scores generated after matching as weights to perform an inverse probability weighted generalized linear model analysis. This approach combines a generalized linear model with a propensity score model to more accurately assess the individual score's association with the outcome, requiring only one of the two models to be correctly specified to obtain unbiased results.

To further test the robustness of our findings, we performed a sensitivity analysis on the data, it was a multifactorial cox regression model analysis as well as a ROC curve analysis in which we extracted patients with a diagnosis of CA separately according to ICD-9 codes. P value less than 0.05 was considered statistically significant and statistical software used in this study included SPSS software (V26, IBM), RStudio and Stata (V14).

Results

3.1 Basic characteristics

As shown in Fig. 1, the patient data were drawn from the MIMIC π public database, which included a total of 65,366 patients admitted to the ICU for the first time. After excluding 62,303 patients defined as having non-cardiogenic shock (non-CA), 258 patients under the age of 18, and patients with an ICU stay of less than 24 hours, a total of 2,535 patients were ultimately included in this study. All of these patients

had clear and complete outcome data; among them, 1,467 were recorded as survivors and 1,068 as deceased.

3.2 Demographic Characteristics

There were significant differences between the two groups in terms of age and gender distribution ($P < 0.01$). The median age of the non-survivors was significantly higher than that of the survivors (69.00 years vs. 65.00 years). Regarding gender composition, the proportion of males was slightly higher in the survivor group than in the non-survivor group (64.76% vs. 59.18%).

Most laboratory parameters showed significant differences between the two groups ($P < 0.01$) in Table 1, specifically:

- Electrolytes and Renal Function:** Non-survivors had slightly higher serum sodium levels, significantly lower serum calcium and bicarbonate levels, and significantly higher anion gap, blood urea nitrogen, creatinine, and lactate levels.
- Liver Function and Cardiac Enzymes:** Non-survivors had significantly lower serum albumin levels than survivors, while blood glucose, troponin T, total bilirubin, ALT, AST, and creatine kinase levels were all significantly higher than in survivors.
- Arterial Blood Gas Analysis:** Non-survivors had significantly lower pH and PO_2 levels than survivors, indicating more severe acidosis and hypoxemia.
- Complete blood count:** The white blood cell count was significantly elevated in non-survivors, while there were no statistically significant differences between the two groups in red blood cell count, hemoglobin, hematocrit, and platelet count ($P > 0.05$).
- Coagulation function:** Prothrombin time was significantly prolonged in non-survivors.

Table 1
Comparison of basic characteristics of survivors and non-survivors in patients with cardiac arrest

Variable	Total (n = 2535)	Survivors (n = 1467)	Non-survivors (n = 1068)	P value
Age	67.00 (56.00–78.00)	65.00 (55.00–76.00)	69.00 (58.00–80.00)	< 0.001
Male	1582 (62.41%)	950 (64.76%)	632 (59.18%)	0.005
Laboratory tests				
Potassium	4.20 (3.80–4.70)	4.20 (3.80–4.70)	4.20 (3.80–4.80)	0.806
Sodium	138.00 (135.00–141.00)	138.00 (135.00–141.00)	139.00 (135.00–142.00)	0.006
Calcium	8.30 (7.80–8.80)	8.40 (7.90–8.90)	8.20 (7.70–8.80)	< 0.001
Bicarbonate	21.00 (18.00–24.00)	22.00 (19.00–25.00)	20.00 (17.00–24.00)	< 0.001
Anion Gap	15.00 (13.00–19.00)	15.00 (12.00–18.00)	17.00 (14.00–21.00)	< 0.001
Urea nitrogen	23.00 (16.00–40.00)	21.00 (14.00–35.00)	27.00 (18.00–45.00)	< 0.001
Lactate	2.30 (1.50–4.20)	2.00 (1.30–3.30)	3.00 (1.70–5.50)	< 0.001
Creatinine	1.20 (0.90–2.10)	1.10 (0.80–1.80)	1.40 (1.00–2.30)	< 0.001
Albumin	3.10 (2.60–3.50)	3.20 (2.70–3.60)	2.90 (2.40–3.30)	< 0.001
Glucose	152.00 (115.00–217.00)	142.00 (112.00–198.50)	168.00 (125.00–241.00)	< 0.001
Troponin T	0.18 (0.06–0.58)	0.15 (0.05–0.50)	0.21 (0.07–0.70)	< 0.001
Bilirubin	0.60 (0.40–1.00)	0.60 (0.40–0.90)	0.60 (0.40–1.20)	< 0.001
ALT	47.00 (21.00–130.50)	40.00 (19.00–97.00)	63.00 (26.00–190.75)	< 0.001
AST	72.00 (32.00–197.00)	56.00 (27.00–138.00)	101.50 (41.00–299.00)	< 0.001
CK	7.00 (3.00–21.00)	6.00 (3.00–17.00)	9.00 (4.00–25.00)	< 0.001
PH	7.34 (7.25–7.40)	7.35 (7.28–7.41)	7.31 (7.20–7.39)	< 0.001
PO2	94.00 (52.00–202.00)	103.00 (55.50–221.00)	87.00 (49.00–173.00)	< 0.001
PCO2	42.00 (36.00–50.00)	43.00 (36.00–49.00)	42.00 (35.00–50.00)	0.207

Variable	Total (n = 2535)	Survivors (n = 1467)	Non-survivors (n = 1068)	P value
RBC	3.63 (3.03–4.30)	3.67 (3.03–4.33)	3.58 (3.01–4.29)	0.226
WBC	12.60 (8.70–18.00)	12.00 (8.50-16.65)	13.70 (9.20-19.52)	< 0.001
Hemoglobin	10.80 (8.90–12.80)	10.90 (9.00-12.90)	10.60 (8.80–12.70)	0.066
Hematocrit	33.30 (28.10–39.20)	33.50 (28.10–39.30)	33.05 (28.00-39.20)	0.707
Platelet	197.00 (143.00-263.00)	196.00 (146.00-257.00)	198.00 (135.00-271.25)	0.536
PT	32.50 (28.10-43.85)	31.70 (27.80-42.15)	33.60 (28.48–47.02)	< 0.001
Scores				
OASIS	36.00 (30.00–43.00)	34.00 (28.00–40.00)	39.00 (33.00–46.00)	< 0.001
SAPS II	43.00 (33.00–55.00)	38.00 (30.00–49.00)	49.00 (39.00–62.00)	< 0.001
SIRS	3.00 (2.00–3.00)	3.00 (2.00–3.00)	3.00 (2.00–4.00)	< 0.001
SOFA	2.00 (0.00–4.00)	1.00 (0.00–4.00)	3.00 (0.00–5.00)	< 0.001
OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; RBC, red blood cell; WBC, white blood cell; PT, prothrombin time. P < 0.05 was statistically significant.				

3.3 Multivariate Analysis and Prognostic Factors

Risk Factors

Age was significantly associated with 28-day mortality; for every 1-year increase in age, the risk of death increased by 0.8% (HR = 1.008, 95% CI 1.004–1.012, $P < 0.001$), which shown in Table 2. Male patients had a 5.7% higher risk of death than female patients (HR = 1.057, 95% CI 1.050–1.063, $P < 0.001$). Elevated levels of several laboratory markers were significantly associated with an increased risk of death. For every 1 mmol/L increase in serum sodium, the risk of death increased by 1.8% (HR = 1.018, 95% CI 1.007–1.030, $P = 0.001$); For every 1-unit increase in anion gap, the risk of death increased by 6.5% (HR = 1.065, 95% CI 1.055–1.075, $P < 0.001$); For every 1 mg/dL increase in blood urea nitrogen, the risk of death increased by 0.7% (HR = 1.007, 95% CI 1.005–1.009, $P < 0.001$); For every 1 mmol/L increase in lactate, the risk of death increased by 12.7% (HR = 1.127, 95% CI 1.111–1.144, $P < 0.001$); For every 1 mg/dL increase in creatinine, the risk of death increased by 3.3% (HR = 1.033, 95% CI 1.006–

1.062, $P = 0.018$); For every 1 mg/dL increase in blood glucose, the risk of death increased by 0.2% (HR = 1.002, 95% CI 1.001–1.002, $P < 0.001$); For every 1 ng/mL increase in troponin T, the risk of death increased by 2.4% (HR = 1.024, 95% CI 1.003–1.045, $P = 0.025$); For every 1 mg/dL increase in bilirubin, the risk of death increased by 4.9% (HR = 1.049, 95% CI 1.032–1.065, $P < 0.001$); For every 1×10^9 /L increase in white blood cell count, the risk of death increased by 2.0% (HR = 1.020, 95% CI 1.014–1.026, $P < 0.001$); For every 1-second increase in prothrombin time, the risk of death increased by 0.2% (HR = 1.002, 95% CI 1.000–1.004, $P = 0.013$); ; for every 1 U/L increase in creatine kinase, the risk of death increased by 0.1% (HR = 1.001, 95% CI 1.001–1.002, $P < 0.001$). Although ALT and AST showed statistical significance, the effect sizes were extremely small.

Protective Factors

Elevated levels of certain markers were associated with a reduced risk of mortality. For every 1 mg/dL increase in serum calcium, the risk of mortality decreased by 9.0% (HR = 0.910, 95% CI 0.856–0.967, $P = 0.002$); for every 1 mmol/L increase in bicarbonate, the risk of death decreased by 6.3% (HR = 0.937, 95% CI 0.926–0.948, $P < 0.001$); For every 1 g/dL increase in albumin, the risk of death decreased by 34.9% (HR = 0.651, 95% CI 0.595–0.712, $P < 0.001$); For every 0.1-unit increase in pH, the risk of death decreased by 89.4% (HR = 0.106, 95% CI 0.068–0.165, $P < 0.001$); For every 1 mmHg increase in PO_2 , the risk of death decreased by 0.1% (HR = 0.999, 95% CI 0.998–0.999, $P < 0.001$).

Disease Severity Scores

All four disease severity scores were significantly positively associated with 28-day mortality. For every 1-point increase in the OASIS score, the risk of death increased by 5.7% (HR = 1.057, 95% CI 1.050–1.063, $P < 0.001$); For every 1-point increase in the SAPS II score, the risk of death increased by 3.2% (HR = 1.032, 95% CI 1.029–1.036, $P < 0.001$); For every 1-point increase in the SIRS score, the risk of death increased by 25.9% (HR = 1.259, 95% CI 1.177–1.347, $P < 0.001$); For every 1-point increase in the SOFA score, the risk of death increased by 6.4% (HR = 1.064, 95% CI 1.043–1.086, $P < 0.001$).

Indicators with no significant correlation

Serum potassium ($P = 0.364$), pCO_2 ($P = 0.643$), red blood cell count ($P = 0.808$), hemoglobin ($P = 0.595$), hematocrit ($P = 0.344$), and platelet count ($P = 0.573$) were not significantly associated with 28-day mortality.

Table 2
Univariate Cox regression analysis of different severity scores and other factors and 28-day mortality of patients

Variables	Beta	SE	Z	P	HR
Age	0.008	0.002	3.948	< 0.001	1.008 (1.004 ~ 1.012)
Gender	0	0	0	0	0
Female	0	0	0	0	1.000 (Reference)
Male	0.055	0.003	16.721	< 0.001	1.057 (1.050 ~ 1.063)
Laboratory tests					
Potassium	0.032	0.035	0.909	0.364	1.033 (0.963 ~ 1.107)
Sodium	0.018	0.006	3.267	0.001	1.018 (1.007 ~ 1.030)
Calcium	-0.094	0.031	-3.025	0.002	0.910 (0.856 ~ 0.967)
Bicarbonate	-0.065	0.006	-10.875	< 0.001	0.937 (0.926 ~ 0.948)
Anion Gap	0.063	0.005	12.967	< 0.001	1.065 (1.055 ~ 1.075)
Urea nitrogen	0.007	0.001	7.31	< 0.001	1.007 (1.005 ~ 1.009)
Lactate	0.12	0.008	15.801	< 0.001	1.127 (1.111 ~ 1.144)
Creatinine	0.033	0.014	2.359	0.018	1.033 (1.006 ~ 1.062)
Albumin	-0.429	0.045	-9.434	< 0.001	0.651 (0.595 ~ 0.712)
Glucose	0.002	0	6.998	< 0.001	1.002 (1.001 ~ 1.002)
Troponin T	0.023	0.01	2.243	0.025	1.024 (1.003 ~ 1.045)
Bilirubin	0.047	0.008	6.021	< 0.001	1.049 (1.032 ~ 1.065)
ALT	0	0	7.013	< 0.001	1.000 (1.000 ~ 1.000)
AST	0	0	7.282	< 0.001	1.000 (1.000 ~ 1.000)
CK	0.001	0	3.491	< 0.001	1.001 (1.001 ~ 1.002)
PH	-2.248	0.228	-9.873	< 0.001	0.106 (0.068 ~ 0.165)
PO2	-0.001	0	-4.97	< 0.001	0.999 (0.998 ~ 0.999)
pCO2	-0.001	0.002	-0.463	0.643	0.999 (0.994 ~ 1.004)
RBC	-0.009	0.035	-0.243	0.808	0.992 (0.926 ~ 1.062)
WBC	0.019	0.003	6.584	< 0.001	1.020 (1.014 ~ 1.026)
Hemoglobin	-0.006	0.012	-0.531	0.595	0.994 (0.971 ~ 1.017)

Variables	Beta	SE	Z	P	HR
Hematocrit	0.004	0.004	0.946	0.344	1.004 (0.996 ~ 1.012)
Platelet	0	0	0.564	0.573	1.000 (1.000 ~ 1.001)
PT	0.002	0.001	2.491	0.013	1.002 (1.000 ~ 1.004)
Scores					
OASIS	0.055	0.003	16.721	< 0.001	1.057 (1.050 ~ 1.063)
SAPS II	0.032	0.002	18.591	< 0.001	1.032 (1.029 ~ 1.036)
SIRS	0.23	0.034	6.68	< 0.001	1.259 (1.177 ~ 1.347)
SOFA	0.062	0.01	6.123	< 0.001	1.064 (1.043 ~ 1.086)
OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; RBC, red blood cell; WBC, white blood cell; PT, prothrombin time. $P < 0.05$ was statistically significant.					

3.4 Multivariate Analysis and Prognostic Factors

Sensitivity analysis

As shown in Table 3, variables with $P < 0.05$ in the univariate analysis were included in the multivariate Cox regression model to assess the independent association of each indicator with 28-day mortality after adjusting for confounding factors.

Age was no longer statistically significant in the multivariable model (HR = 1.002, 95% CI 0.998–1.006, $P = 0.356$), suggesting that the effect of age on mortality risk may be explained by other, more direct pathophysiological indicators. Compared with females, the difference in male mortality risk was not statistically significant (HR = 0.894, 95% CI 0.787–1.016, $P = 0.087$), indicating that after adjusting for other factors, sex was not an independent risk factor for 28-day mortality.

Independent risk factors among laboratory indicators (HR > 1, $P < 0.05$)

Serum urea nitrogen was an independent risk factor for 28-day mortality; for every 1 mg/dL increase in serum urea nitrogen, the risk of death increased by 0.3% (HR = 1.003, 95% CI 1.000–1.006, $P = 0.034$). Lactate is one of the most significant independent predictors of 28-day mortality; for every 1 mmol/L increase in lactate, the risk of death increases by 7.6% (HR = 1.076, 95% CI 1.050–1.102, $P < 0.001$). Bilirubin was independently associated with 28-day mortality; for every 1 mg/dL increase in bilirubin, the risk of death increased by 2.1% (HR = 1.021, 95% CI 1.001–1.041, $P = 0.044$).

Independent protective factors (HR < 1, $P < 0.05$)

Creatinine acted as a protective factor in the multivariable model; for every 1 mg/dL increase in creatinine, the risk of death decreased by 5.6% (HR = 0.944, 95% CI 0.903–0.988, $P = 0.012$). This finding may be related to active interventions for patients with renal impairment in clinical practice and should be interpreted with caution. Albumin was an independent protective factor for 28-day mortality; for every 1 g/dL increase in albumin, the risk of death decreased by 18.3% (HR = 0.817, 95% CI 0.737–0.904, $P < 0.001$). PO_2 was associated with a reduced risk of mortality; for every 1 mmHg increase in PO_2 , the risk of mortality decreased by 0.1% (HR = 0.999, 95% CI 0.998–0.999, $P < 0.001$).

Disease Severity Scores

The OASIS score was an independent risk factor for 28-day mortality; for every 1-point increase in the score, the risk of death increased by 1.7% (HR = 1.017, 95% CI 1.007–1.027, $P < 0.001$). The SAPS II score was an independent risk factor for 28-day mortality; for every 1-point increase in the score, the risk of death increased by 1.8% (HR = 1.018, 95% CI 1.012–1.024, $P < 0.001$). The SIRS score was no longer statistically significant in the multivariable model (HR = 1.034, 95% CI 0.959–1.113, $P = 0.385$), suggesting that its impact on mortality risk may be masked by other more precise scoring systems or laboratory markers. The SOFA score was no longer statistically significant in the multivariate model (HR = 0.982, 95% CI 0.960–1.005, $P = 0.118$), indicating that the SOFA score is not an independent predictor of 28-day mortality after adjusting for other factors.

Variables that lost statistical significance

The following variables were significant in univariate analysis but lost statistical significance after multivariate adjustment: Serum sodium ($P = 0.179$), serum calcium ($P = 0.787$), bicarbonate ($P = 0.613$), anion gap ($P = 0.325$), blood glucose ($P = 0.229$), troponin T ($P = 0.334$), ALT ($P = 0.164$), AST ($P = 0.699$), creatine kinase ($P = 0.979$), pH ($P = 0.395$), white blood cell count ($P = 0.715$), and prothrombin time ($P = 0.781$). The predictive value of these parameters for 28-day mortality may depend on other, more important clinical factors and does not represent independent risk factors.

Table 3

Multivariate Cox regression analysis of different severity scores and other factors and 28-day mortality of patients

Variables	Beta	SE	Z	P	HR	Significant
Age	0.002	0.002	0.923	0.356	1.002 (0.998 ~ 1.006)	
Gender						
Female						1.000(Reference)
Male	-0.112	0.065	-1.71	0.087	0.894 (0.787 ~ 1.016)	
Laboratory tests						
Sodium	0.007	0.005	1.344	0.179	1.007 (0.997 ~ 1.018)	
Calcium	-0.008	0.03	-0.271	0.787	0.992 (0.936 ~ 1.051)	
Bicarbonate	-0.004	0.008	-0.506	0.613	0.996 (0.980 ~ 1.012)	
Anion Gap	0.008	0.008	0.985	0.325	1.008 (0.992 ~ 1.025)	
Urea nitrogen	0.003	0.002	2.114	0.034	1.003 (1.000 ~ 1.006)	*
Lactate	0.073	0.012	5.891	< 0.001	1.076 (1.050 ~ 1.102)	**
Creatinine	-0.057	0.023	-2.498	0.012	0.944 (0.903 ~ 0.988)	*
Albumin	-0.203	0.052	-3.897	< 0.001	0.817 (0.737 ~ 0.904)	**
Glucose	0	0	1.203	0.229	1.000 (1.000 ~ 1.001)	
Troponin T	0.015	0.015	0.965	0.334	1.015 (0.985 ~	

Variables	Beta	SE	Z	P	HR	Significant
					1.045)	
Bilirubin	0.021	0.01	2.01	0.044	1.021 (1.001 ~ 1.041)	*
ALT	0	0	1.392	0.164	1.000 (1.000 ~ 1.000)	
AST	0	0	-0.386	0.699	1.000 (1.000 ~ 1.000)	
CK	0	0.001	-0.026	0.979	1.000 (0.999 ~ 1.001)	
PH	0.245	0.288	0.851	0.395	1.278 (0.726 ~ 2.249)	
PO2	-0.001	0	-4.466	< 0.001	0.999 (0.998 ~ 0.999)	**
WBC	0.001	0.004	0.365	0.715	1.001 (0.994 ~ 1.008)	
PT	0	0.001	-0.278	0.781	1.000 (0.998 ~ 1.002)	
Scores						
OASIS	0.017	0.005	3.407	< 0.001	1.017 (1.007 ~ 1.027)	**
SAPS II	0.018	0.003	5.923	< 0.001	1.018 (1.012 ~ 1.024)	**
SIRS	0.033	0.038	0.868	0.385	1.034 (0.959 ~ 1.113)	
SOFA	-0.018	0.012	-1.564	0.118	0.982 (0.960 ~ 1.005)	
OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; RBC, red blood cell; WBC, white blood cell; PT, prothrombin time. $P < 0.05$ was statistically significant.						

3.5 Analysis of the Association Between Different Severity Scores and 28-Day Mortality

Results of the multivariable Cox regression analysis (Table 4) showed that, compared with the low-score group, both the moderate- and high-score groups for the OASIS, SAPS II, and SIRS scores were significantly associated with an increased risk of 28-day mortality (all $P < 0.001$). In contrast, neither the moderate group (HR = 0.96, 95% CI: 0.81–1.13, $P = 0.600$) nor the high group (HR = 0.72, 95% CI: 0.46–1.15, $P = 0.167$) of the SOFA score showed statistical significance.

Table 4

Multivariate Cox regression analysis of different severity scores and 28-day mortality of patients

Variable		HR	95%CI	P value
OASIS	Low	Reference		
	Median	1.34	1.15–1.56	< 0.001
	High	1.76	1.43–2.17	< 0.001
SAPS II	Low	Reference		
	Median	1.97	1.69–2.30	< 0.001
	High	2.53	2.04–3.14	< 0.001
SIRS	Low	Reference		
	Median	1.3	1.12–1.51	< 0.001
	High	1.34	1.14–1.57	< 0.001
SOFA	Low	Reference		
	Median	0.96	0.81–1.13	0.6
	High	0.72	0.46–1.15	0.167
OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score. The variables ($P > 0.05$ except for scores) in the single-factor Cox regression model were excluded, and the remaining variables were included in the multi-factor Cox regression model for analysis. Low groups as reference. $P < 0.05$ was statistically significant.				

Table 5

Sensitivity analysis: Multivariate cox regression analysis of different severity scores and 28-day mortality of patients To further control for potential confounding factors, this study conducted an inverse probability-weighted generalized linear model analysis (Table 6). The results confirmed that the OASIS, SAPS II, and SIRS scores were all independently associated with 28-day mortality (all $P < 0.001$). Consistent with the primary analysis, the moderate group of SOFA scores showed a significant association ($P < 0.001$), but the high-score group remained non-significant ($P = 0.381$). Taken together, OASIS, SAPS II, and SIRS scores are robust independent predictors of 28-day mortality, whereas the predictive performance of SOFA scores was inconsistent across different analytical methods and patient subgroups.

Variable		HR	95%CI	P value
OASIS	Low	Reference		
	Median	1.97	1.72–2.26	< 0.001
	High	3.28	2.79–3.86	< 0.001
SAPS II	Low	Reference		
	Median	2.3	2.00–2.64	< 0.001
	High	3.72	3.16–4.38	< 0.001
SIRS	Low	Reference		
	Median	1.46	1.26–1.68	< 0.001
	High	1.67	1.42–1.95	< 0.001
SOFA	Low	Reference		
	Median	1.35	1.14–1.59	< 0.001
	High	1.3	0.82–2.05	0.259
Patients diagnosed as cardiac arrest according to International Classification of Diseases (ICD-9) were included in sensitivity analysis, and the variables with $P < 0.05$ in univariate cox analysis were also included in multivariate cox regression model for analysis. Low groups as reference. OASIS, Oxford acute disease severity score; SAPS II, simplified acute physiology score 2; SIRS, systemic inflammatory response syndrome standard; SOFA, Sequential Organ Failure Score. $P < 0.05$ was statistically significant.				

Table 6

Analysis of inverse probability-weighted generalized linear model with different severity scores

Variable		Sx	T value	P value
OASIS	Low	Reference		
	Median	0.85	9.04	< 0.001
	High	1.49	10.93	< 0.001
SAPS II	Low	Reference		
	Median	1.06	11.14	< 0.001
	High	1.64	12.46	< 0.001
SIRS	Low	Reference		
	Median	0.51	5.38	< 0.001
	High	0.72	6.63	< 0.001
SOFA	Low	Reference		
	Median	0.41	3.39	< 0.001
	High	0.28	0.88	0.381
The scores of different severity levels were weighted by tendency score, and the variables that affected the survival of patients except for the score in multi-factor cox regression analysis were treated. The inverse probability-weighted generalized linear model analysis was also performed. Low groups as reference. OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score. $P < 0.05$ was statistically significant.				

3.6 Relationship between scores of different severity levels and 28-day mortality rate of patients.

This study analyzed the distribution trends of four clinical scoring systems (OASIS, SAPS II, SIRS, and SOFA) across different severity categories (Show in Fig. 2), and the results are as follows. OASIS scores show an upward trend as objective disease severity increases. From mild to severe, scores rise gradually, indicating consistency between subjective assessment and objective severity. The SAPS II score also increases with rising disease severity. At lower severity levels, the scores are lower; as severity gradually increases, the SAPS II scores exhibit a gradual upward trend, with relatively stable changes. SIRS scores are at lower levels when disease severity is low, and they increase accordingly as severity increases. The overall trend shows that SIRS scores are positively correlated with disease severity, but the rate of increase is relatively gentle. The SOFA score shows a continuous upward trend as disease severity increases. At lower levels of severity, the SOFA score remains low; as severity gradually increases, the SOFA score rises steadily, demonstrating a strong positive correlation.

3.7 Kaplan-Meyer survival curve: association between different severity scores and a 28-day mortality rate for patients, shown in Fig. 3

1. Trends in OASIS Scores Over Time Across Severity Groups

When stratified by OASIS scores, the low, moderate, and high severity groups exhibited distinct trends in their dynamic changes over the analysis period.

The low-severity group showed the slowest decline over time, consistently maintaining the highest level among the three groups, suggesting a better prognosis for patients in this group. The moderate severity group showed an intermediate rate of decline, with values at each time point consistently lower than those of the low severity group but higher than those of the high severity group. The high severity group declined the fastest; from the early stages of the analysis, its values were significantly lower than those of the other two groups, and by the end of the analysis, it had the lowest values, suggesting that patients in this group had the highest risk of events and the poorest prognosis. Throughout the entire observation period, the values across the three groups consistently maintained a stable gradient of “low > moderate > high,” with no crossover observed.

2. Trends in SAPS II Scores by Severity Group Over Time

The trends across the three groups stratified by SAPS II scores were similar to those observed for MEWS. The low-severity group showed the most gradual decline over time, consistently maintaining a relatively high level. The moderate severity group showed a moderate rate of decline, remaining between the low and high groups at all time points. The high severity group exhibited the most significant decline, remaining below the low and moderate groups from the early stages, with the gap widening further as the analysis period extended. Throughout the entire analysis window, the three groups consistently maintained a clear hierarchical order, indicating that the SAPS II score possesses good discriminatory ability regarding patient prognosis.

3. Trends in SIRS Scores Across Severity Groups Over Time

The three groups stratified by SIRS scores also exhibited a gradual decline over the course of the analysis. The low-severity group declined the slowest and consistently had the highest values among the three groups. The moderate-severity group showed an intermediate rate and magnitude of decline. The high-severity group declined the fastest, with values lower than those of the low- and moderate-severity groups at all time points, and became the lowest group by the end of the analysis. The three groups maintained a stable order of “low > moderate > high” throughout the entire observation period, with no crossover or overlap, suggesting that the SIRS score can effectively distinguish patients with different prognostic risks.

4. Trends in SOFA Scores Across Severity Groups Over Time

The trends in the three groups stratified by SOFA score (self-assessed severity) were consistent with those of the previous three scores. The low-severity group declined most slowly over time and consistently maintained the highest level. The rate of decline in the moderate severity group fell between that of the low and high groups. The high severity group declined the fastest, remaining significantly lower than the low and moderate groups from the early stages of the analysis and reaching the lowest level by the end. Throughout the entire analysis window, the three groups consistently maintained a stable gradient of “low > moderate > high,” with no crossover observed.

3.8 ROC Curve Analysis

The Log-rank test was used to compare the differences in AUC between the two scoring systems, which is shown in Fig. 4. The results showed that the predictive performance of the SAPS II score was significantly superior to that of the OASIS score ($\chi^2 = 5.20$, $P = 0.023$). The AUC of the SAPS II score was approximately 0.019 higher than that of the OASIS score, and this difference was statistically significant. Both scoring systems demonstrated moderate predictive ability for 28-day mortality in patients admitted to the ICU following cardiac arrest. The predictive performance of the SAPS II score was slightly superior to that of the OASIS score, which may be attributed to its more comprehensive inclusion of physiological parameters. However, the AUC for both scores did not reach the threshold of 0.7 or higher, indicating that a single scoring system has limited predictive value for individual prognosis. In clinical practice, it is recommended to conduct a comprehensive assessment by combining multiple scoring systems and laboratory indicators.

Discussion

Based on the MIMIC-IV database, this study systematically evaluated the predictive value of various disease severity scores and laboratory markers for 28-day mortality in ICU patients following cardiac arrest. The main findings are as follows.

First, in univariate analysis, age, sex, and multiple laboratory parameters were significantly associated with mortality; however, after multivariate adjustment, only blood urea nitrogen, lactate, bilirubin, albumin, PO_2 , and creatinine (in a protective direction) retained independent significance. Notably, for every 1 mmol/L increase in lactate, the risk of death increased by 7.6%, making it one of the strongest independent predictors in this study. This finding is consistent with previous evidence showing that elevated lactate levels are associated with poor survival and neurological outcomes after cardiac arrest, likely reflecting the severity of systemic hypoperfusion and metabolic disturbance^[21]. Albumin, as an independent protective factor, may reflect the patient's nutritional status and inflammatory burden, and overall physiological status rather than exerting a direct protective effect. Recent post-resuscitation guidance emphasizes multimodal prognostication after cardiac arrest, integrating clinical examination, biomarkers, neurophysiology, and imaging rather than relying on a single score^[22]. Hypoalbuminemia is

common in critically ill patients and may reflect a combination of systemic inflammation, increased vascular permeability, altered albumin synthesis, and reduced nutritional reserve. Its association with mortality is therefore more likely to represent global disease severity and physiological vulnerability than a direct causal protective effect of albumin itself^[23].

Furthermore, among the four severity scores, OASIS and SAPS II consistently maintained independent predictive power in the multivariate models, whereas SIRS and SOFA lost statistical significance. This finding was based on the results of the present study. The persistent association of OASIS and SAPS II with mortality is biologically plausible because both scores summarize multiple physiological and demographic dimensions of acute illness. OASIS was developed as a reduced-variable severity score using a subset of APACHE II data elements while maintaining comparable predictive accuracy^[14]. SAPS II was developed from a large European and North American multicenter cohort and incorporates multiple physiological and clinical variables to estimate disease severity and mortality risk in critically ill patients^[15]. The different performance of these scores may be related to differences in their construction and the populations in which they were developed. SIRS primarily reflects systemic inflammatory response; however, SIRS criteria have limited specificity because they may be triggered by infection, ischemia-reperfusion injury, trauma, surgery, and other noninfectious stress states^[24]. SOFA focuses on organ dysfunction and was originally developed for critically ill patients with sepsis-related organ failure^[17, 18, 25]. In patients after cardiac arrest, neurological injury, ischemia-reperfusion injury, myocardial dysfunction, and cardiogenic shock may coexist. These complex and interacting processes may not be fully captured by a single inflammatory or organ-dysfunction score^[25].

Further subgroup analysis revealed that when OASIS, SAPS II, SIRS, and SOFA were categorized into low, moderate, and high groups, the moderate and high subgroups of OASIS, SAPS II, and SIRS were all significantly associated with mortality, whereas SOFA showed no significant association at any stratification level. Sensitivity analyses (restricted to patients with cardiac arrest) and inverse probability weighting analyses consistently confirmed the robustness of OASIS, SAPS II, and SIRS, whereas SOFA showed sporadic significance only in the moderate group and remained non-significant in the high group, this pattern indicates that the association between SOFA and mortality was not consistent across severity strata in this cohort. Because the number of patients in the high-SOFA group was relatively small, insufficient statistical power and imprecision may also have contributed to the lack of statistical significance.

ROC curve analysis showed that the predictive performance of SAPS II was slightly better than that of OASIS ($P = 0.023$), but the AUC of both scores remained below 0.70, indicating that the discriminatory ability of a single scoring system was limited. This finding has important clinical implications. Prognosis after cardiac arrest is influenced by multiple factors, including ischemia-reperfusion injury, neurological injury, myocardial dysfunction, systemic metabolic disturbances, systemic inflammation, and subsequent organ failure. These processes cannot be fully captured by a single simplified score. Current post-resuscitation recommendations therefore support a multimodal prognostic strategy that integrates

serial neurological examination, biomarkers, electrophysiology, neuroimaging, and clinical course rather than relying on one score or test^[22, 26, 27]. Accordingly, OASIS or SAPS II may be useful for initial risk stratification, but they should be combined with key laboratory indicators, such as lactate, albumin, and bilirubin, and interpreted within the broader clinical context.

Limitation

This study has the following limitations. First, the data were derived from a retrospective database; although multivariate and weighted analyses were used to control for confounders, unmeasured biases may still exist. Second, the sample size in the high-score group of the SOFA score was relatively small (Tables 5 and 6), which may have affected statistical power. Third, the study population was drawn from a single database (MIMIC-IV), and external validation still needs to be conducted in other independent cohorts. Fourth, information on the specific etiology of cardiac arrest (cardiac vs. non-cardiac) and prehospital management was not included in the analysis; these factors may modulate the predictive performance of the scoring systems.

There were significant differences in the dynamic trends over the analysis period among the low-, moderate-, and high-severity groups. The low-severity group showed the slowest decline and the highest final value; the high-severity group showed the fastest decline and the lowest final value; and the moderate-severity group fell between the two. The three groups maintained a clear hierarchical gradient throughout the entire observation period, suggesting that this severity stratification index has good predictive and discriminatory value for patient prognosis.

The four scoring systems (OASIS, SAPS II, SIRS, and SOFA) all exhibited a clear gradient pattern across the three severity categories (low, moderate, and high): the low-severity group showed the slowest decline over time and the highest final scores; the high-severity group showed the fastest decline and the lowest final scores; and the moderate-severity group fell between the two. No overlap was observed between groups across the entire observation period, suggesting that all four scoring systems can effectively distinguish prognostic risks among patients of different severity levels.

Future studies should validate these findings in prospective cohorts and explore the possibility of combining OASIS, SAPS II, and key biomarkers (such as lactate and albumin) in a unified model to develop more accurate prognostic prediction tools.

Conclusions

This systematic analysis of 2,535 post-cardiac arrest ICU patients from the MIMIC-IV database yielded the following conclusions: Independent predictors: Blood urea nitrogen, lactate, and bilirubin are independent risk factors for 28-day mortality; albumin, PO₂, and creatinine (with a protective effect) are independent protective factors. Among these, lactate is the single laboratory parameter with the highest predictive power. Performance of scoring systems: OASIS and SAPS II remained independent predictors

after multivariate adjustment; SIRS and SOFA lost their independence in multivariate models, but stratified and weighted analyses showed that the high-score group of SIRS remained robustly associated. Specificity of SOFA: The SOFA score showed no significant association in the primary analysis; it was only occasionally significant in the cardiac arrest subgroup and in the moderate-score group of the weighted analysis, while the high-score group remained consistently non-significant, suggesting that it is not suitable for prognostic stratification in this population. Limited predictive performance: Although the predictive performance of SAPS II was slightly superior to that of OASIS ($P = 0.023$), the AUC for both was below 0.7, indicating that a single scoring system has limited value for assessing individual prognosis. Clinical recommendations: It is recommended to combine the OASIS or SAPS II scores with key laboratory indicators such as lactate and albumin to comprehensively assess the 28-day mortality risk in ICU patients following cardiac arrest. Future multicenter prospective studies are needed to further validate and optimize the combined predictive model.

Abbreviations

Cardiac arrest

CA

Intensive care unit

ICU

Simplified Acute Physiology Score II

SAPS II

Systemic Inflammatory Response Syndrome criteria

SIRS

Sequential Organ Failure Score

SOFA

Cardiac ogenic shock

CS

In-of-hospital cardiac arrest

IHCA

Out-of-hospital cardiac arrest

OHCA

Medical Information Mart for Intensive Care

MIMIC-III

Massachusetts Institute of Technology

MIT

Beth Israel Deaconess Medical Center

BIDMC

Mean arterial pressure

MAP

Alanine aminotransferase

ALT
Aspartate aminotransferase
AST
Creatine kinase
CK
Red blood cell
RBC
White blood cell
WBC
Prothrombin time
PT

Declarations

Ethical Approval and Informed Consent Statement

This study did not involve human subjects or animal experiments; all data were obtained through independent searches of publicly available sources and subsequently processed. Therefore, no ethical approval or informed consent from participants is required.

Consent to Publication

All authors agree to the publication of this manuscript. Availability of Data and materials. All data and materials in this study were independently retrieved, organised and analysed by the authors' team and are available upon reasonable academic request from the corresponding author.

Conflict of Interest

Zhang Yin was responsible for data collection and analysis; Wu Yifan and Ren Shuting conceived and designed this study; Sui Xin and Zhang Jian were responsible for data interpretation and developed the new software used in this study; Zhao Jingshun, Zhou Runfeng, Xu Wenjia and Wu Tong wrote this paper or made substantial revisions to it. The authors declare that there are no conflicts of interest.

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Author Contributions

All authors made substantial contributions to the design of this study, data collection and analysis, manuscript drafting and finalisation, and agree to take responsibility for all aspects of this work.

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Figures

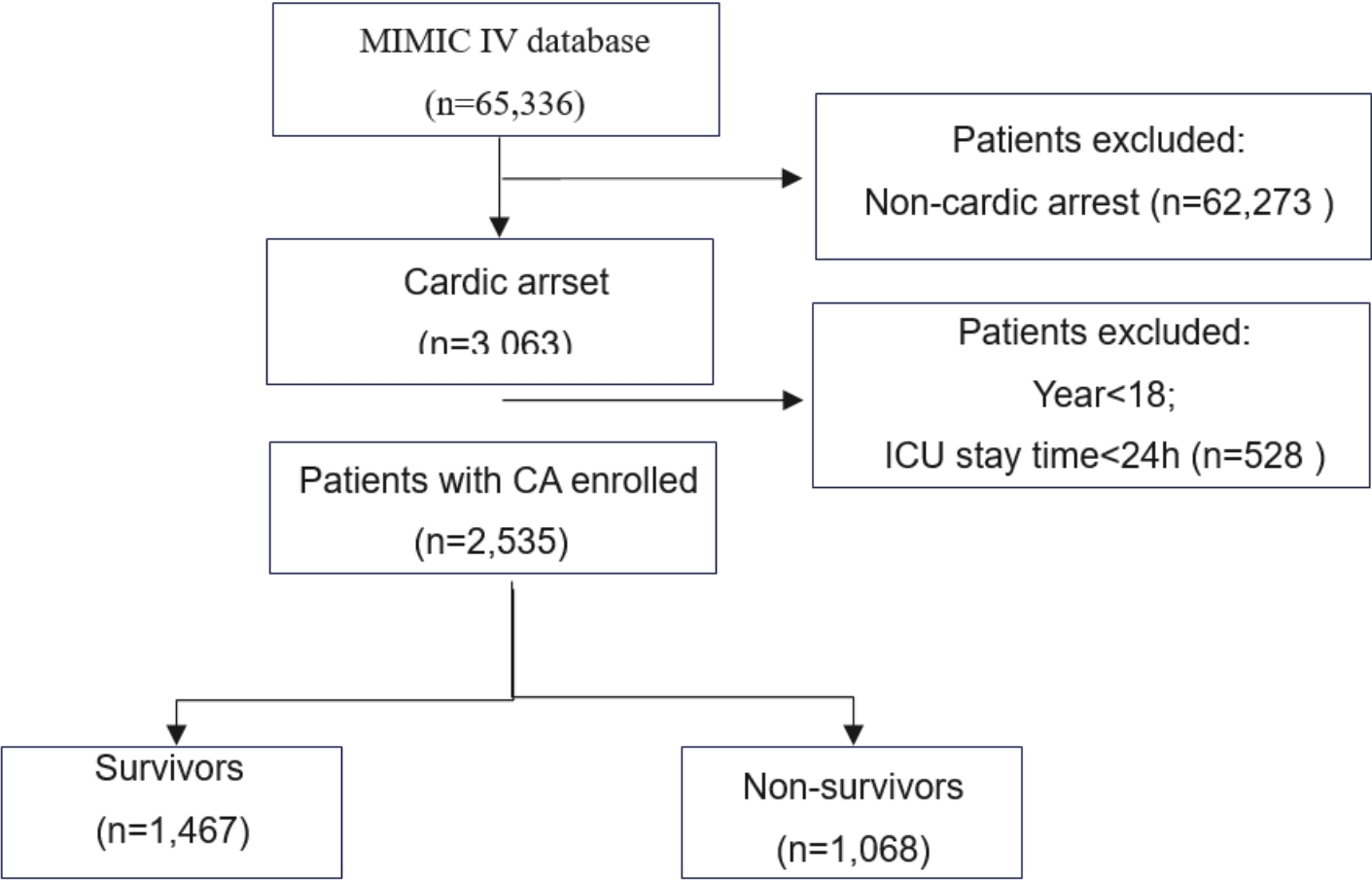


Figure 1

Flow chart of research object incorporation. MIMIC IV, Medical Information Market for Intensive Care IV.

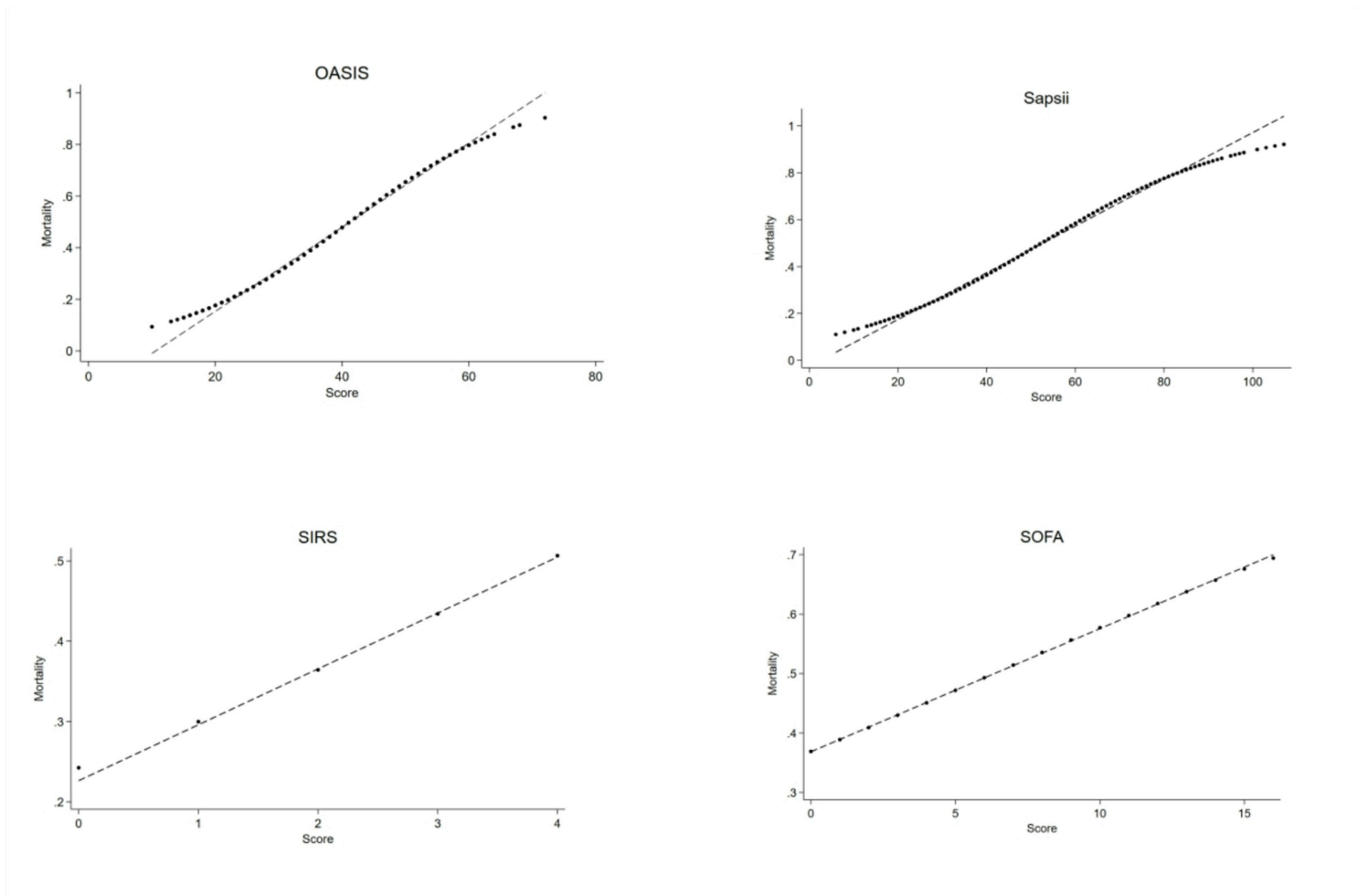


Figure 2

Relationship between scores of different severity levels and 28-day mortality rate of patients. $P < 0.05$ was statistically significant. OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score.

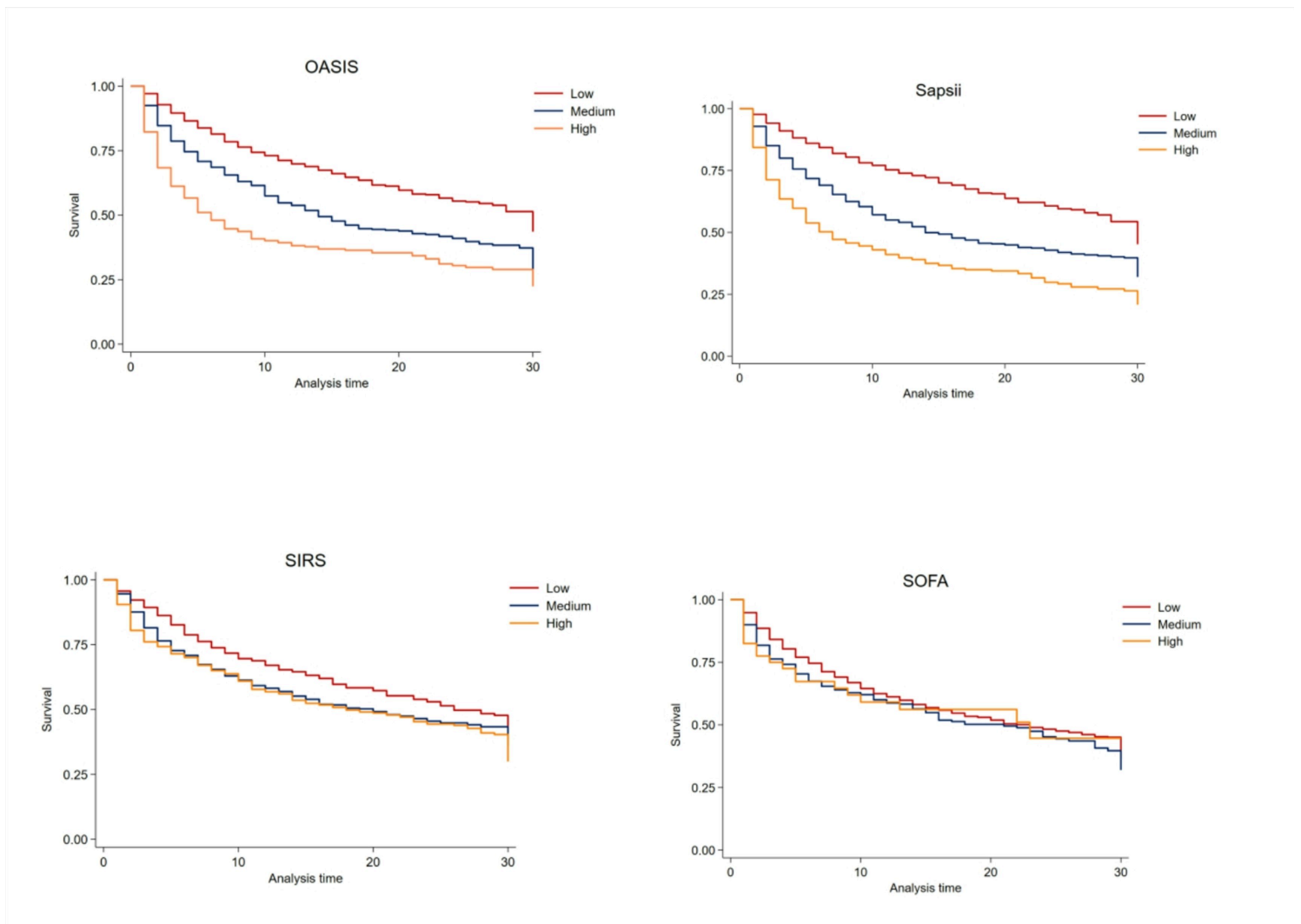


Figure 3

Kaplan-Meier survival curve: association between different severity scores and a 28-day mortality rate for patients. Low groups as reference. $P < 0.05$ was statistically significant. OASIS, Oxford Acute Disease Severity Score; SAPS II, simplified acute physiological score two; SIRS, standard for systemic inflammatory response syndrome; SOFA, sequential organ failure score.

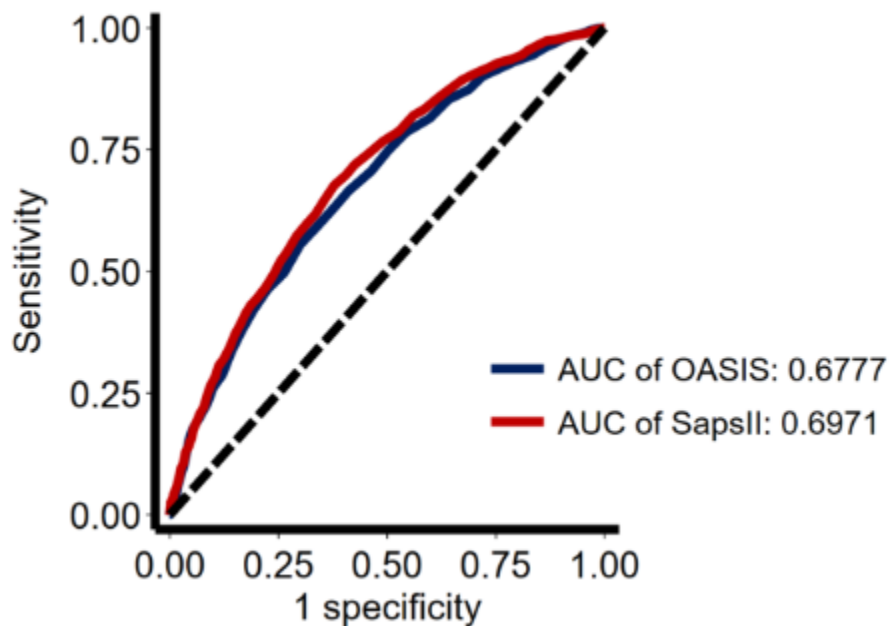


Figure 4

ROC curves for the treatment performance the Sensitivity of the OASIS score and Sapsii score for patients

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [PatientInformationMICEInterpolation.csv](#)