

The relationship between hemoglobin-red blood cell distribution width ratio and short-term prognosis in patients with in-hospital cardiac arrest: a retrospective study

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Abstract

Background: Cardiac arrest is a critical clinical condition with a grave prognosis, imposing a substantial burden on healthcare resources and society. Its underlying pathophysiology is characterized by the abrupt cessation of cardiac pump function, leading to systemic ischemia and hypoxia, particularly in the brain. This ischemic insult, in turn, activates a potent systemic inflammatory response that exacerbates organ injury. Conventional clinical parameters often fall short in providing a multidimensional assessment that concurrently addresses both inflammatory and ischemic-hypoxic risks, thereby limiting the identification of high-risk patients. The hemoglobin-to-red cell distribution width ratio (HRR) has recently emerged as a novel and integrated biomarker capable of reflecting systemic inflammatory activity and ischemic-hypoxic status. Although its prognostic value has been validated in various diseases, evidence regarding its association with outcomes following in-hospital cardiac arrest resuscitation remains scarce. Furthermore, existing studies predominantly focus on 30-day outcomes, leaving its role in predicting short-term prognosis underexplored. Therefore, this study aimed to investigate the impact of HRR on the short-term (7-day) prognosis of patients after cardiac arrest.

Methods: This study retrospectively analyzed data from adult in-hospital cardiac arrest patients who underwent successful cardiopulmonary resuscitation and achieved the return of spontaneous circulation at Yantai Yuhuangding Hospital between 2023 and 2025. The first available laboratory data after return of spontaneous circulation were collected. Significant variables affecting 7-day mortality were identified using univariate and multivariable Cox regression analyses. Additionally, Kaplan–Meier curves were used to further explore the impact of the HRR on the short-term mortality of the patients. Finally, a nomogram model incorporating HRR was developed for clinical prediction of in-hospital mortality risk.

Results: A total of 216 patients who met the inclusion and exclusion criteria were enrolled in this study. The multivariate Cox regression analysis revealed that HRR (hazard ratio: 0.887; 95% confidence interval: 0.845–0.931) and other factors were associated with the short-term mortality of the patients. After adjusting for relevant factors, it was found that Kaplan–Meier curves demonstrated that a decrease in HRR was correlated with an increased short-term mortality rate in the patients ($P < 0.05$).

Conclusion: The initial postresuscitation HRR (<9.13) level was significantly associated with an increased risk of short-term mortality in cardiac arrest patients.

Keywords: Cardiac arrest, Hemoglobin-to-RDW ratio, Mortality, Nomogram, Prognosis

Introduction

Cardiac arrest (CA) is defined as the abrupt cessation of cardiac mechanical activity, resulting in profound systemic ischemia,

particularly in critical organs such as the brain.^[1] This ischemic insult triggers a cascade of detrimental inflammatory responses, which often leading to fatal outcomes if not immediately treated. According to studies, including by Martijon et al, CA claims millions of lives annually, with survival rates typically remaining below 10%.^[2, 3] The management of CA consumes substantial healthcare resources and imposes a significant economic burden on families and society. Current conventional biomarkers often fail to provide a comprehensive assessment of multidimensional risks, such as the degree of inflammatory response and ischemic-hypoxic injury, thereby limiting the implementation of personalized interventions (eg, anti-inflammatory therapy or ischemia-targeted strategies) for high-risk patients. Consequently, the identification of novel, readily available, and clinically significant prognostic biomarkers is crucial for improving risk stratification and guiding timely treatment in CA. The hemoglobin-to-red cell distribution width ratio (HRR) has recently emerged as a novel composite biomarker that reflects both systemic inflammatory status and ischemic-hypoxic burden.^[4] Its prognostic value has been reported in various conditions,^[5–8] including chronic obstructive pulmonary disease,^[9] acute ischemic stroke, epidural hemorrhage, and several malignancies.

QL and YH contributed equally to this article.

The datasets generated during and/or analyzed during the current study are not publicly available but are available from the corresponding author upon reasonable request.

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However, evidence regarding the association between HRR and outcomes after in-hospital cardiac arrest (IHCA) remains limited, and most existing studies in CA populations have focused on 30-day outcomes. Although 30-day mortality is commonly used as an endpoint in CA research, it may not fully capture the early clinical course after return of spontaneous circulation (ROSC). A substantial proportion of deaths occur during the early post-resuscitation period following IHCA,^[10] primarily due to severe hypoxic-ischemic brain injury, refractory shock, and early post-CA syndrome. Importantly, the first 7 days represent a critical window for intensive care management, prognostic assessment, and decisions regarding life-sustaining treatment. Moreover, previous studies have mostly focused on the correlation between a single indicator and patient prognosis,^[11, 12] biomarkers reflecting acute inflammatory and ischemic-hypoxic stress, such as HRR, may be more closely associated with early outcomes than with longer term mortality, which can be influenced by subsequent complications and comorbidities.^[13] Therefore, we aimed to investigate the relationship between HRR and 7-day mortality in patients with IHCA.

Methods

Database

This study utilized data from patients who experienced IHCA at Yantai Yuhuangding Hospital between 2023 and 2025. For all eligible patients meeting the predefined inclusion and exclusion criteria, we collected baseline characteristics and the first set of laboratory parameters obtained after the ROSC. The laboratory panel included cardiac troponin I, brain natriuretic peptide, plateletcrit, urea, C-reactive protein, HRR, red blood cell count (RBC), neutrophil count, albumin, lymphocyte count, potassium, pH, and lactate. Baseline clinical variables, including age, sex, smoking history, comorbidities (hypertension, arrhythmia, diabetes, heart failure, and stroke), the presence of a shockable initial rhythm, duration of cardiopulmonary resuscitation (CPR) in minutes, total adrenaline dosage (AD), and outcomes (survival to 7 days or more and nonsurvival within 7 days), were collected in accordance with the Utstein-style reporting guidelines.^[14, 15]

Inclusion and exclusion criteria

The inclusion and exclusion criteria of this study were as follows. Inclusion criteria: (1) patients who experienced IHCA and received CPR; (2) patients aged 18 years or older; and (3) patients with complete data. Exclusion criteria: (1) CA caused by trauma ($n = 34$); (2) CA caused by end-stage diseases such as brain herniation, malignant tumors, etc. ($n = 12$); and (3) Patients with previous hematological diseases or immune system diseases ($n = 11$). A total of 273 patients with CA were collected according to the inclusion criteria, and after exclusion, there were 216 cases of data. The patients were divided into the survival to 7 days or more group ($n = 56$) and the nonsurvival within 7 days group ($n = 160$) according to their prognosis (Fig. 1).

Data processing

Data were entered into Microsoft Excel 2021 (Microsoft Corporation, Redmond, WA) for data management and analyzed with R statistical software (version 4.4.1, R Core Team, Vienna, Austria). Continuous variables were handled based on distribution: normally distributed data were expressed as mean $\pm$ SD ($\bar{X} \pm SD$) and compared using independent-samples t -test;

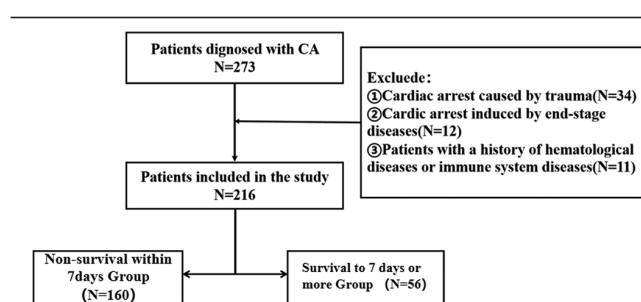


Figure 1. Extraction, inclusion, and exclusion of cardiac arrest data. CA, cardiac arrest.

nonnormally distributed data as median (interquartile range) (M [Q1, Q3]) with Mann–Whitney U test. Categorical variables were reported as n (%) and compared via χ^2 test. Cox proportional hazards regression (univariate and multivariate) was used to assess the association between HRR and prognosis in CA patients ($P < 0.05$ for significance).

To assess multicollinearity among the predictors included in the multivariable Cox regression model, we calculated variance inflation factors (VIFs) for each variable. A VIF greater than 5 is typically considered indicative of significant collinearity.^[16] All variables in the final model had VIF values between 1.0 and 1.4, suggesting that multicollinearity did not pose a significant issue in the analysis. The proportional hazards assumption was evaluated using Schoenfeld residuals with the `cox.zph` function.^[17] This test was performed for each predictor in the multivariable Cox model, and the global test was conducted to assess the overall model fit. A P value greater than 0.05 for a predictor indicates that the proportional hazards assumption holds for that variable. Variables with significant P values ($P \leq 0.05$) were examined further to assess potential violations of the proportional hazards assumption. Patients who did not experience the event (eg, death) during the study period were treated as right-censored. The analysis used the Kaplan–Meier method to estimate survival curves and the Cox proportional hazards model to evaluate risk factors, with censoring accounted for in both methods.^[18, 19] Kaplan–Meier curves visualized the impact on HRR of 7-day survival. The optimal cutoff value for HRR was determined using the `surv_cutpoint` function from the survival package. This method identifies the cutoff point that best differentiates patient survival outcomes based on 7-day mortality. HRR was calculated as hemoglobin concentration divided by red blood cell distribution width (RDW, %), using the first laboratory measurements obtained after ROSC. HRR was treated as a dimensionless variable in all analyses.^[20]

Results

This study enrolled a total of 216 patients with CA (Table 1). Among them, 160 patients (74%) died within 7 days of hospitalization (nonsurvivors), while 56 patients (26%) survived to 7 days or more (survivors). The proportion of males was higher in nonsurvivors (65.8%) than in survivors (50.0%). Nonsurvivors were older, with a median age of (77.00 [73.00–81.00]) years, compared with years (73.00 [69.00–77.00]) in survivors. A history of smoking was more common in nonsurvivors (50.5%) than in survivors (25.0%). By contrast, a shockable rhythm was observed in 94.3% of survivors, substantially higher than the 36.9% rate in nonsurvivors. Regarding resuscitation details, the median duration of CPR

Table 1
Baseline Characteristics of the Study Group

Characteristic	All patients (n = 216)	Death (n = 160)	Survival (n = 56)	P*
Sex (male), n (%)	133 (61.5)	105 (65.8)	28 (50.0)	0.045*
Age (years), median (IQR)	75.00 (71.00–79.00)	77.00 (73.00–81.00)	73.00 (69.00–77.00)	0.039*
Smoking, n (%)	95 (43.9)	81 (50.5)	14 (25.0)	0.042*
Hypertension, n (%)	104 (48.1)	73 (45.9)	31 (56.2)	0.171
Arrhythmia, n (%)	94 (43.5)	74 (46.2)	20 (35.7)	0.225
Diabetes, n (%)	93 (43.0)	73 (45.9)	20 (35.2)	0.144
Heart failure, n (%)	100 (46.3)	75 (46.9)	25 (44.6)	0.894
Stroke, n (%)	78 (36.1)	53 (33.3)	25 (45.7)	0.085
ICU admission (%)	156 (72.2)	120 (74.8)	36 (64.8)	0.146
shockable rhythm (%)	113 (52.3)	60 (36.9)	53 (94.3)	0.023*
CPR duration, min, median (IQR)	13.00 (9.00–18.00)	16.00 (12.00–21.00)	7.00 (5.00–10.00)	0.036*
AD (mg), median (IQR)	5.00 (3.00–8.00)	8.00 (6.00–10.00)	3.00 (2.00–5.00)	0.034*
cTnl (pg/mL), median (IQR)	66.21 (28.43–102.34)	93.21 (45.61–138.49)	30.2 (18.94–45.36)	0.081
BNP (pg/mL), median (IQR)	274.58 (210.31–356.82)	334.75 (260.41–412.71)	388.05 (320.21–455.61)	0.501
Pct (ng/L), median (IQR)	0.53 (0.30–0.82)	0.51 (0.28–0.75)	0.56 (0.34–0.90)	0.221
UREA (mmol/L), median (IQR)	5.98 (4.91–7.30)	6.60 (5.32–8.21)	5.46 (4.43–6.32)	0.127
CREA (umol/L)	63.21 ± 10.79	68.09 ± 14.78	50.87 ± 9.81	0.254
Alb (g/L)	39.19 ± 3.27	38.13 ± 2.13	40.05 ± 1.87	0.059
CRP (mg/L), median (IQR)	9.50 (5.21–14.61)	13.50 (8.41–18.71)	10.64 (6.18–15.23)	0.582
HRR median (IQR)	9.10 (8.56–9.64)	6.79 (6.16–7.42)	9.96 (8.98–10.94)	0.019*
RBC (g/L)	3.98 ± 0.45	3.96 ± 0.98	4.01 ± 1.01	0.809
Neu (10 ⁹ /L)	7.34 ± 2.21	7.60 ± 2.30	7.33 ± 1.51	0.107
Lym (10 ⁹ /L)	1.36 ± 0.61	1.61 ± 0.51	1.05 ± 0.46	0.568
K (mmol/L)	5.10 ± 0.45	5.21 ± 1.26	4.10 ± 1.56	0.229
pH median (IQR)	7.33 (7.28–7.39)	7.19 (7.16–7.22)	7.32 (7.28–7.36)	0.054
Lac (mmol/L), median (IQR)	2.20 (1.51–3.19)	2.85 (2.08–3.89)	2.25 (1.59–3.02)	0.012*

*Compared between group with death and group survival.

AD, adrenaline dosage; Alb, albumin; BNP, brain natriuretic peptide; CRP, C-reactive protein; cTnl, cardiac troponin I; HRR, hemoglobin-to-red cell distribution width ratio; K, potassium ion; Lac, lactic acid; Lym, lymphocyte; Neu, neutrophil; Pct, procalcitonin.

duration was longer in nonsurvivors (16.00 [12.00–21.00]) than in survivors 7.00 (5.00–10.00). Similarly, the median dose of epinephrine administered during CPR 8.00 (6.00–10.00) was higher in nonsurvivors compared with survivors (3.00 [2.00–5.00]). Finally, The HRR value was notably higher in the survival (9.96 [8.98–10.94]) compared with the nonsurvival group (6.79 [6.16–7.42]).

Cox data analysis

After performing univariate Cox regression analysis on the aforementioned data, we identified several variables notably associated with the 7-day mortality of CA patients, including sex (male) (hazard ratio [HR]: 1.642; 95% confidence interval [CI]: 1.192–2.273), shockable rhythm (HR: 0.821; 95% CI: 0.747–0.983); AD (HR = 1.053; 95% CI: 1.022–1.113), HRR (HR = 0.861; 95% CI: 0.766–0.968), and lactate (HR: 1.134; 95% CI: 1.051–1.198) (Table 2). The results demonstrated that sex (male) (HR: 1.718; 95% CI: 1.159–2.403), HRR (HR: 0.887; 95% CI: 0.845–0.931), LAC (HR: 1.126; 95% CI: 1.046–1.215), and presence of shockable rhythm (HR: 0.522; 95% CI: 0.350–0.787) remained statistically significantly associated with the 7-day mortality of CA patients (Table 3). In contrast, no significant associations were observed between 7-day mortality and either AD (HR: 1.034; 95% CI: 0.977–1.094) or Age (HR: 0.998; 95% CI: 0.963–1.024). Results of collinearity analysis: VIF were calculated for each variable included in the multivariable Cox regression model. The VIF values for all predictors ranged from 1.01 to 1.41 (HRR: 1.028, LAC: 1.413, age: 1.091, sex: 1.326, AD: 1.223), indicating minimal

multicollinearity among the variables. These results suggest that multicollinearity did not significantly affect the regression model, ensuring the stability and reliability of the model's parameter estimates. The proportional hazards assumption was tested using Schoenfeld residuals for each predictor. The assumption was violated for HRR ($P = 0.01$), indicating that HRR's effect on survival may vary over time. However, we chose to retain HRR in the model due to its clinical importance. The assumption was not violated for the other predictors (LAC, age, sex, AD), all of which had P values greater than 0.05, indicating consistent effects over time. The global test for the model ($P = 0.126$) suggested that the overall proportional hazards assumption was satisfied. Of the 216 patients included in the study, 160 patients (74%) died within the 7-day follow-up period, while 56 patients (26%) survived to 7 days or more. These patients were included in the Kaplan–Meier survival analysis, with their survival probability calculated up to the 7-day time point. The survival rates at 1, 3, and 7 days were 89%, 65%, and 39%, respectively. The median survival time for the cohort was 11 days, indicating that half of the patients survived longer than 11 days. The median survival rate has been indicated on the Kaplan–Meier survival curve, with a dotted gray line marking the median survival time. The red line indicates the 7-day survival rate for the two groups.

Analysis of mortality rate

A total of 216 patients were included in the study, of whom 160 patients (74%) died within the 7-day follow-up period, while 56 patients (26%) survived to 7 days or longer. These

Table 2**Univariate Cox Regression Analysis of 7-Day Mortality after Cardiac Arrest**

Variable	β	HR	95% CI	P*
Age	0.071	1.073	0.952–1.151	0.067
Sex (male)	0.497	1.642	1.192–2.273	0.041*
shockable rhythm	−0.197	0.821	0.747–0.983	0.037*
AD	0.051	1.053	1.022–1.113	0.028*
BNP	−0.132	0.876	0.832–1.144	0.679
Pct	−0.053	0.948	0.894–1.102	0.075
UREA	−0.009	0.991	0.971–1.019	0.359
CRP	−0.003	0.997	0.989–1.006	0.705
HRR	−0.150	0.861	0.766–0.968	0.012*
RBC	0.033	1.033	0.887–1.213	0.668
Neu	−0.042	0.958	0.934–1.021	0.102
Alb	−0.016	0.984	0.980–1.017	0.054
Lym	0.017	1.017	0.999–1.036	0.741
K	0.126	1.134	0.859–1.553	0.375
Lac	0.126	1.134	1.051–1.198	0.026*
pH	−0.236	0.790	0.694–1.107	0.053

*Compared between group with death and group survival was statistically significant.

AD, adrenaline dosage; Alb, albumin; BNP, brain natriuretic peptide; CI, confidence interval; CRP, C-reactive protein; HR, hazard ratio; HRR, hemoglobin-to-red cell distribution width ratio; K, potassium ion; Lac, lactic acid; Lym, lymphocyte; Neu, neutrophil; Pct, procalcitonin.

Table 3**Multivariate Cox Regression Analysis of 7-Day Mortality after Cardiac Arrest**

Variable	Coef	HR	95% CI	P*
Age	−0.002	0.998	0.963–1.024	0.864
Sex (male)	0.545	1.718	1.159–2.403	0.035*
HRR	−0.120	0.887	0.845–0.931	0.001*
Lac	0.118	1.126	1.046–1.215	0.029*
AD	0.033	1.034	0.977–1.094	0.247
Shockable rhythm	−0.650	0.522	0.350–0.787	0.001*

AD, adrenaline dosage; CI, confidence interval; HR, hazard ratio; HRR, hemoglobin-to-red cell distribution width ratio; Lac, lactic acid.

data were incorporated into the Kaplan–Meier survival analysis. Kaplan–Meier survival curves were constructed to compare the predictive performance of survival rates stratified by different groups.^[18] The log-rank test was employed as the primary statistical approach to assess intergroup differences in survival outcomes. A two-tailed *P* value <0.05 was defined as statistically significant, indicating a meaningful distinction in survival rates between the compared groups.^[19, 21] Through the aforementioned univariate and multivariate Cox regression analyses, it was identified that the HRR serves as a protective factor against the 7-day in-hospital mortality of CA patients. Additionally, Kaplan–Meier survival curves were plotted (Fig. 2) to further explore the prognostic value of HRR. The results revealed that there was a statistically significant difference in the short-term in-hospital survival rate between patients in the HRR-High (>9.13) group and those in the HRR-Low (<9.13) group.

Based on these findings, we developed and validated a prognostic nomogram that incorporates HRR along with other significant predictors. This integrative model provides a quantifiable and readily accessible tool for clinicians to estimate individual patient risk, potentially enabling earlier identification of high-risk individuals and informing tailored treatment strategies. By integrating multiple independent predictors, the nomogram demonstrated superior predictive performance compared with any single parameter alone (Fig. 3).

Discussion

In this retrospective cohort of patients resuscitated from IHCA, HRR was independently associated with 7-day mortality. Patients with lower HRR values exhibited a higher risk of early death, suggesting that HRR may serve as a pragmatic marker for early risk stratification in this setting. Most prior CA studies evaluating hematological indices have focused on RDW as a single parameter and have commonly used longer term outcomes such as 30-day or in-hospital mortality.^[10, 22] While RDW has been linked to adverse outcomes, it does not incorporate hemoglobin concentration and, therefore, does not directly capture variation in oxygen-carrying capacity. HRR integrates hemoglobin and RDW into a single index, which may better reflect the combined burden of impaired oxygen transport and systemic stress. Evidence specifically addressing HRR in IHCA—particularly for early outcomes—remains limited; thus, our findings add to the emerging literature by examining HRR in relation to short-term mortality. The biological basis for the observed association is plausible but should be interpreted cautiously. RDW has been reported to correlate with systemic inflammation, oxidative stress, and disrupted erythropoiesis, and has also been associated with impaired oxygenation in prior studies. In the post-CA state, ischemic-hypoxic injury and inflammatory activation could contribute to anisocytosis and an increase in RDW, whereas

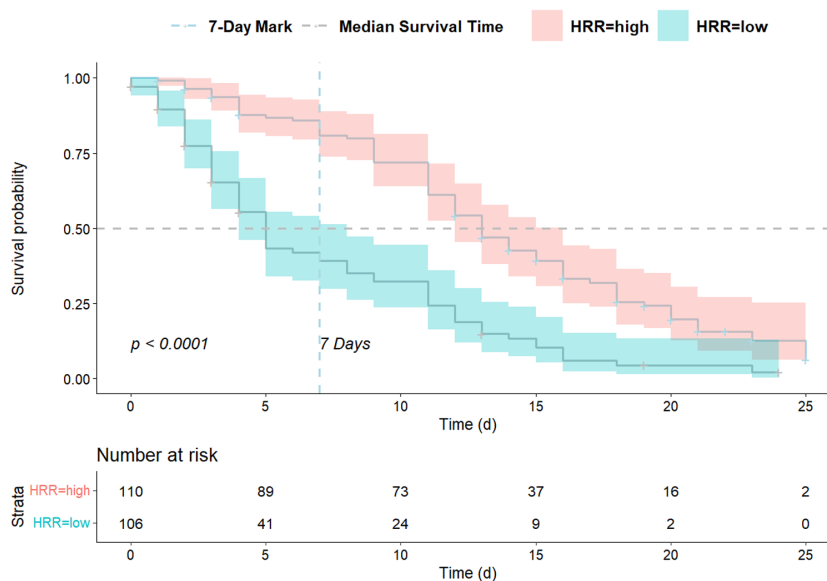


Figure 2. Kaplan—Meier survival curve for HRR-high versus HRR-low groups. The HRR-high group included patients with an HRR >9.13, while the HRR-low group included patients with an HRR <9.13. Shaded areas represent 95% confidence interval. The gray line marks the median survival time intervals, and the light blue line indicates the 7-day survival rate. HRR, hemoglobin-to-red cell distribution width ratio.

hemoglobin concentration reflects oxygen-carrying capacity that is directly relevant to tissue perfusion during the early postresuscitation period. Accordingly, a low HRR may represent the concurrence of reduced oxygen transport capacity and heightened systemic stress, which could be linked to worse early outcomes. Previous studies have established an inverse correlation between RDW and oxygen saturation.^[23–25] Decreased oxygen saturation impairs erythrocyte membrane integrity and reduces deformability,^[26] which not only compromises oxygen delivery but also stimulates the bone marrow to release immature RBCs, ultimately elevating RDW.^[27–29] Moreover, the immediate ischemic-hypoxic insult following CA can further increase RDW by disrupting red cell membranes and inducing inflammatory oxidative stress.^[11, 30] These pathophysiological processes collectively contribute to a reduction in HRR. These

considerations are hypothesis-generating; mechanistic studies are required to clarify causal pathways.

In previous studies on the prognosis of CA, age and adrenaline dose have also been validated as significant variables.^[31, 32] We included them as variables in the nomogram. On the basis of these results, we constructed a nomogram incorporating HRR alongside established clinical predictors, including age, sex, lactate, defibrillation, and CPR duration. The nomogram demonstrated acceptable discrimination, with a Harrell's C-index of 0.67. In addition, receiver operating characteristic analysis for 7-day mortality yielded an area under the curve of 0.84, indicating good short-term predictive performance. This tool may facilitate individualized estimation of short-term risk after IHCA in a clinically interpretable format. Nevertheless, given the retrospective single-center design and the limited

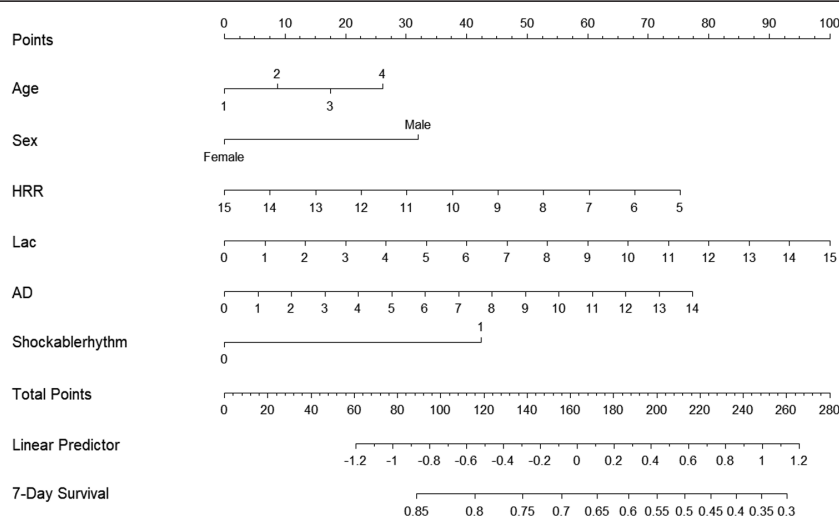


Figure 3. Nomogram for predicting short-term mortality in in-hospital patients. The Age variable was divided into four categories: 1, 50–60; 2, 60–70; 3, 70–80; 4, 80–90 years. AD, adrenaline dosage; HRR, hemoglobin-to-red cell distribution width ratio.

number of events relative to candidate predictors, the model should be regarded as exploratory and requires external validation in independent cohorts. In addition, exclusions of trauma-related arrest, end-stage disease, and preexisting hematological/immune disorders may restrict generalizability to broader critically ill populations. In conclusion, HRR was associated with 7-day mortality after IHCA and may provide incremental prognostic information as an easily obtainable hematological index. Future multicenter studies with external validation and mechanistic investigations are warranted to confirm its utility and to better define the biological underpinnings of this association.

Limitations

This study is subject to several limitations inherent in its single-center, observational design. The findings may lack generalizability, and residual confounding cannot be ruled out despite statistical adjustments. The modest sample size also limits statistical power for detecting weaker associations, while the retrospective nature introduces potential information bias from missing data or inadequate follow-up. Several exclusions in the present study may affect the generalizability of our findings. Patients with trauma-related CA, end-stage disease, or preexisting hematological or immune disorders were excluded, which may limit the applicability of the results to certain critically ill populations. These exclusions were implemented to reduce clinical heterogeneity and potential confounding, as the mechanisms and prognosis of trauma-related CA and terminal illnesses differ substantially from those of medical in-hospital CA. In addition, hematological and immune disorders can directly alter hemoglobin levels and RDW, potentially confounding the interpretation of HRR as a marker of acute ischemic-hypoxic and inflammatory stress. As a result, the findings of this study are most applicable to adult patients with medical in-hospital CA without preexisting conditions that markedly affect RBC indices. Future multicenter studies including broader patient populations are warranted to determine whether the prognostic value of HRR can be extended to other critically ill subgroups.

Conclusion

This study suggests that a decrease in the HRR is associated with an increased risk of short-term mortality in patients with CA. Therefore, the nomogram constructed using HRR can be used to predict the incidence of short-term mortality in CA, and it holds potential clinical predictive value.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Liu Q contributed to methodology, software, validation, formal analysis, data curation, visualization, and writing the original draft. Hu Y contributed to validation and visualization. Li Q contributed to conceptualization, investigation, resources, writing, review, and editing. Kang H contributed to supervision, writing, review, and editing. All authors have read and agreed to the published version of the manuscript.

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Ethical approval of studies and informed consent

The study followed the principles of the Declaration of Helsinki as revised in 2013. This study was approved and written informed consent was waived by the Ethics Committee of Yantai Yuhuangding Hospital Affiliated to Qingdao University (registration number: K2026-063, date: 2026-02-05) owing to the anonymized retrospective nature of the analysis.

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