

# Mortality risk stratification in heat stroke using cardiac troponin I and bedside parameters: a retrospective study

Rui Huang<sup>a,b</sup>, Xiangchao Wu<sup>c</sup>, Yunfei Xiang<sup>a,b</sup>, Junyu Jiang<sup>a,b</sup>, Shanmu Ai<sup>a,b</sup>, Wanshu Que<sup>a,b</sup>, Haojie Wu<sup>a,b</sup>, Sen Zhang<sup>a,b</sup>, Fating Zhou<sup>a,b,\*</sup> , Yu Ma<sup>a,b,\*</sup> 

## Abstract

**Background:** This study aimed to characterize fatal heat stroke (HS) and develop a risk-prediction model for early identification of high-risk patients.

**Methods:** Patients with HS were divided into survival and nonsurvival groups based on discharge outcomes. Baseline demographics and laboratory parameters were compared. After univariate logistic regression identified candidate variables ( $P < 0.05$ ), stepwise multivariate logistic regression was performed to select independent predictors of mortality for the final model. A combined predictive model was constructed and evaluated using the Hosmer–Lemeshow test and area under the receiver operating characteristic curve (AUC). Differences in AUCs between models were compared using DeLong test. The optimal cutoff value was determined using the Youden index. Internal validation of the final model was performed using the bootstrap method with 1000 resamples.

**Results:** A total of 209 patients with HS were analyzed, including 165 survivors (78.9%). Nonsurvivors were significantly older, had a higher proportion of females, and exhibited elevated respiratory rate, international normalized ratio, hydroxybutyrate dehydrogenase, cardiac troponin I (cTnI), neutrophil-to-lymphocyte ratio, systemic immune-inflammation index (SII), quick Sequential (Sepsis-related) Organ Failure Assessment (qSOFA), and Acute Physiology and Chronic Health Evaluation II scores, along with lower lymphocyte percentage (all  $P < 0.05$ ). Multivariate logistic regression identified age, cTnI, qSOFA, and SII as independent risk factors for mortality. The combined model demonstrated excellent discriminatory power (AUC = 0.955 vs. age = 0.897; qSOFA = 0.789; cTnI = 0.847; SII = 0.596; DeLong  $P < 0.05$ ), outperforming all individual predictors.

**Conclusion:** Older age, elevated cTnI, higher qSOFA, and SII scores independently predict mortality in HS patients. The combination of these factors offers superior predictive value.

**Trial Registration:** Chinese Clinical Trial Registry; www.chictr.org.cn; ChiCTR2400079671.

**Keywords:** Cardiac troponin I, Heat stroke, High-temperature, Mortality

RH and XW contributed equally to this article.

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

<sup>a</sup>Medical College, Chongqing University, Emergency department, Chongqing University Central Hospital (Chongqing Emergency Medical Center), Chongqing, China, <sup>b</sup>Chongqing Key Laboratory of Emergency Medicine, Chongqing, China, <sup>c</sup>Department of Emergency Medicine, Chongqing Wulong District People's Hospital, Chongqing, China.

\*Corresponding author. Address: Department of Emergency Medicine, Chongqing Emergency Medical Center, No.1 JianKang Road, Yuzhong District, Chongqing 400010, China. E-mail address: zhoulating@163.com (F. Zhou); Department of Emergency Medicine, Chongqing Emergency Medical Center, No.1 JianKang Road, Yuzhong District, Chongqing 400010, China. E-mail address: 81846846@qq.com (Y. Ma).

Copyright © 2026 The Author(s). Published by Wolters Kluwer Health, LLC.

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

*Emergency and Critical Care Medicine* (2026) 6:2

Received: 21 August 2025; Accepted: 6 April 2026

Published online: 14 July 2026

<http://dx.doi.org/10.1097/EC9.000000000000188>

## Introduction

Exacerbated by the urban heat island effect and global warming, heat stroke (HS) increases the prevalence of seasonal disorders. HS, the most severe heat-related illness, manifests clinically as a core body temperature exceeding 40 °C accompanied by central nervous system dysfunction, including seizures, altered awareness, and unconsciousness.<sup>[1]</sup> HS is clinically categorized into 2 groups based on the pathophysiology and age distribution: exertional heat stroke (EHS) and classical heat stroke (CHS). EHS typically occurs in younger individuals during intense physical exertion in hot environments, whereas CHS predominantly occurs in older adults with chronic comorbidities.<sup>[2]</sup>

HS frequently leads to multiorgan dysfunction, particularly involving the cardiac, hepatic, and pulmonary systems, thereby substantially increasing mortality risk.<sup>[3]</sup> Among affected organs, the heart is particularly the most thermolabile during heat stress.<sup>[4]</sup> Heat stress induces hemodynamic alterations, blood flow redistribution, and disruption of cardiac conduction. Patients may develop complications such as tachyarrhythmias, ischemic cardiac changes, and hemodynamic instability due to elevated temperatures. These changes increase cardiac conduction and accelerate impulse transmission through the atrioventricular node, bundle of His, right and left bundle branches, and Purkinje fibers.<sup>[5]</sup> In response to heat, the cardiovascular system increases

heart rate (HR), cardiac contractility, and cardiac output while reducing perfusion to noncutaneous tissues to dissipate heat.<sup>[6]</sup> Elevated temperatures directly stimulate the sinus node, whereas increased sympathetic activity and reduced parasympathetic activity due to heat stress further increase HR and cardiac output.<sup>[7]</sup> Myocardial ischemia, nonspecific ST-T wave alterations, and ST-segment elevation or depression can result in regional ventricular wall motion abnormalities in patients with HS.<sup>[8, 9]</sup> Notably, a recent study reported myocardial injury in 57.3% of EHS cases, with a significantly higher 90-day mortality among the affected patients than among those without injury.<sup>[10]</sup>

The severe complications associated with HS warrant further investigation into its risk factors, with the aim of reducing mortality and improving patient outcomes. Therefore, this study aimed to characterize the clinical profiles of mortality in patients with HS, identify the independent risk factors, and develop a risk-prediction model.

## Materials and methods

### Study population

This multicenter retrospective cohort study consecutively enrolled patients with HS who were admitted to the emergency departments of 3 hospitals in Chongqing—Chongqing High-Tech District People's Hospital, Chongqing Fifth People's Hospital, and Chongqing Emergency Medical Center—between 2020 and 2023. Ethical approval was obtained from the institutional review boards (approval No. ChiCTR 2400079671), and the requirement for informed consent was waived due to the retrospective nature of the study. The study adhered to the guidelines of the Declaration of Helsinki. HS was diagnosed according to the Chinese Expert Consensus on the Diagnosis and Treatment of Heat Stroke.<sup>[11]</sup> Myocardial injury was defined as an elevation of blood cardiac troponin I (cTnI) levels above the 99th percentile upper reference limit.<sup>[12, 13]</sup> As these reference limits are assay-dependent, all measurements had to be performed using the ADVIA Centaur System (Siemens, Erlangen, Germany). In this study, the specific cutoff value was cTnI > 0.04 ng/mL. The inclusion criteria were (1) age ≥ 18 years and (2) fulfillment of the HS diagnostic criteria. The exclusion criteria were as follows: (1) a history of underlying cardiac disease (eg, acute coronary syndrome, myocarditis, aortic dissection, heart failure, and arrhythmia), (2) a history of infectious disease within 4 weeks before admission, and (3) patients with missing outcome variable or incomplete cTnI records.

### Data collection

To ensure methodological rigor, all data collectors underwent standardized training before study initiation. HS cases were identified using International Classification of Diseases, Tenth Revision (ICD-10) codes. The demographic and laboratory parameters were recorded using structured case reports. The collected variables included demographic data (ie, sex and age) and physiological measurements (ie, rectal temperature, HR, respiratory rate [RR], and blood pressure) at presentation, along with documented prodromal symptoms and preexisting comorbidities. Critical care interventions were classified according to the shock status and intensive care unit admission. The laboratory assessments included routine blood tests, coagulation profiles, and myocardial enzyme levels obtained during the initial evaluation. Outcome measures included the occurrence of multiorgan dysfunction syndrome and in-hospital mortality, with follow-up

data for disenrolled patients using validated telephone protocols. If patients could not be contacted directly, their family members were contacted instead. Incorrect phone numbers or an inability to contact patients after 3 attempts at different times were recognized as follow-up failures.

### Statistical analysis

Following data entry into EpiData software (version 3.1; Centers for Disease Control and Prevention, Atlanta, GA), statistical analyses were performed using SPSS Statistics (version 29.0; IBM Corporation, Armonk, NY). Continuous variables were assessed using the Shapiro–Wilk test. Normally distributed data were expressed as mean ± standard deviation and were compared using independent sample *t* tests. Nonnormally distributed variables were expressed as medians with interquartile ranges and analyzed using the Mann–Whitney *U* test. Categorical data were presented as frequencies (percentages), with between-group comparisons performed using the  $\chi^2$  tests or Fisher exact tests, as appropriate. To account for the wide range of systemic immune-inflammation index (SII) values and improve clinical interpretability, SII was divided by 1000 before inclusion in the logistic regression models; thus, the reported odds ratios (OR) reflect the mortality risk per 1000-unit increase in SII. Univariate logistic regression was first used to identify factors associated with mortality in HS patients. Variables with *P* < 0.05 were then entered into multivariate logistic regression to identify independent risk factors, which were subsequently used to construct a combined predictive model. To avoid model overfitting and satisfy the events per variable ≥10 principle, stepwise logistic regression was applied to select variables for the final multivariate model, and the number of variables retained in the final model was restricted to 4. Additionally, model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test, with *P* > 0.05 indicating good agreement between predicted and observed outcomes. Discriminative ability was evaluated using the receiver operating characteristic curve, and areas under the curve (AUCs) were compared across models using DeLong test. Meanwhile, optimal cutoff values for predicting mortality were determined using the Youden index, and the corresponding sensitivity and specificity were calculated. Internal validation of the final model was performed using the bootstrap method with 1000 resamples. Statistical significance was set at a *P* < 0.05.

## Results

### Clinical characteristics of survival and nonsurvival patients

A total of 209 patients were included in this study, of whom 165 (78.9%) survived and 44 (21.1%) died (Fig. 1). Baseline characteristics stratified by clinical outcomes are presented in Table 1.

Statistical analysis revealed that the patients in the nonsurvival group were significantly older than those in the survival group. Significant differences were also observed in the distribution of sex and HS type, with a higher proportion of females and CHS in the nonsurvival group. Additionally, the RR was significantly higher in the nonsurvival group than in the survival group.

Compared with the survival group, lymphocyte percentage was significantly lower in the nonsurvivor group, whereas the international normalized ratio and hydroxybutyrate dehydrogenase levels were significantly higher. Moreover, cTnI levels and the neutrophil-to-lymphocyte ratio were markedly elevated in the nonsurvival group than in the survival group. Scores of established

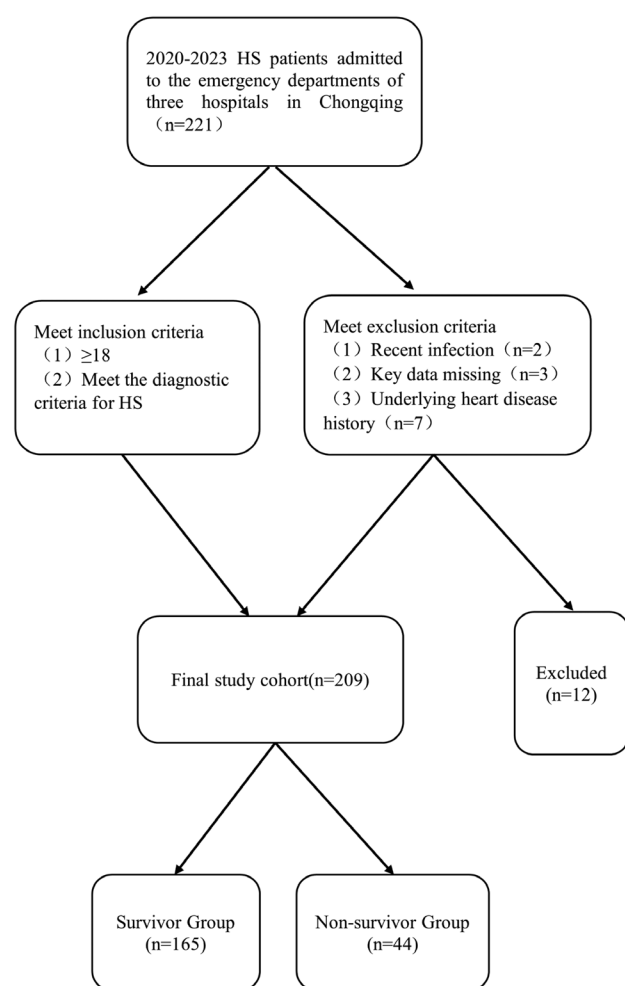


**Figure 1.** Flow diagram of participant selection and grouping in the retrospective study with HS patients. HS, heat stroke.

critical illness assessment tools were significantly worse in the non-survival group, with both quick Sequential (Sepsis-related) Organ Failure Assessment (qSOFA) and Acute Physiology and Chronic Health Evaluation II scores significantly higher than those in the survival group. No statistically significant differences were observed between the 2 groups regarding temperature (T), HR, blood pressure, monocyte and eosinophil counts, platelet count, or other coagulation and myocardial enzyme profile indicators such as creatine kinase and creatine kinase-myocardial band.

### Risk factors for nonsurvival patients with heat stroke

Univariate logistic regression analysis revealed that age, sex, type of HS, RR, cTnI, qSOFA score, Acute Physiology and Chronic Health Evaluation II score, SII, and neutrophil-to-lymphocyte ratio were significant risk factors for mortality in patients with HS (all  $P < 0.05$ ; Table 2). Multicollinearity diagnostics were performed on the variables with  $P < 0.05$  from the univariate analysis, and the results showed no serious multicollinearity among the variables (variance inflation factor  $< 5$ ) (Supplementary Table 1, <https://links.lww.com/ECCM/A126>). After stepwise multivariate logistic regression analysis, 4 variables, including age (OR: 1.18; 95% confidence interval [CI]: 1.11–1.26;  $P < 0.001$ ), cTnI (OR: 1.67; 95% CI: 1.24–2.24;  $P < 0.001$ ), qSOFA score (OR:

1.83; 95% CI: 1.02–3.28;  $P = 0.044$ ), and SII (OR: 1.34; 95% CI: 1.06–1.70;  $P = 0.016$ ), were retained in the final model (all  $P < 0.05$ ; Table 2).

### Development of a mortality prediction model for heat stroke based on independent risk factors

To construct a predictive model for mortality in patients with HS, the above 4 independent risk factors were incorporated into a combined predictive model. The AUC of this model was 0.955 (95% CI: 0.93–0.98; Fig. 2), significantly higher than that of the individual predictors. The AUC values for each individual indicator were as follows: age, 0.897 (95% CI: 0.86–0.94); cTnI, 0.847 (95% CI: 0.79–0.91); qSOFA score, 0.789 (95% CI: 0.72–0.86); and SII, 0.596 (95% CI: 0.50–0.69) (Fig. 2). DeLong test confirmed that the combined model had significantly superior predictive ability compared with age (AUC difference: 0.058; 95% CI: 0.02–0.09;  $P = 0.0015$ ), cTnI (AUC difference: 0.108; 95% CI: 0.06–0.16;  $P < 0.001$ ), qSOFA score (AUC difference: 0.166; 95% CI: 0.11–0.22;  $P < 0.001$ ), and SII (AUC difference: 0.359; 95% CI: 0.26–0.46;  $P < 0.001$ ) (Supplementary Table 2, <https://links.lww.com/ECCM/A127>). The Hosmer–Lemeshow goodness-of-fit test produced a chi-square statistic of 4.995 (degrees of freedom [ $df$ ] = 8,  $P = 0.758$ ), demonstrating no significant discrepancy between the predicted probabilities and the actual outcomes. This nonsignificant result confirms adequate model fit and good calibration. The optimal cutoff values for each indicator were identified using the Youden index: age  $> 72.5$  years predicted mortality with 93.3% sensitivity and 80.5% specificity; SII  $> 1623$  yielded 57.8% sensitivity and 61.0% specificity; qSOFA score  $> 1.5$  had 82.2% sensitivity and 64.6% specificity; and cTnI  $> 1.24$  ng/mL demonstrated 66.7% sensitivity and 90.2% specificity. The combined prediction model achieved a sensitivity of 91.1% and a specificity of 84.2% at the optimal cutoff value determined by the Youden index. Internal validation of the stepwise regression results was performed using the bootstrap method with 1000 resamples, yielding a mean AUC of 0.945 (95% CI: 0.856–0.986), which was comparable to the AUC of the original model (Supplementary Figure 1, <https://links.lww.com/ECCM/A128>).

### Discussion

HS is a life-threatening condition triggered by heat-induced bodily damage, with an exceptionally high mortality rate. Previous data analyses indicate that during summer heatwaves, the incidence of CHS was 17.6–26.5/100,000, with mortality rates among hospitalized patients ranging from 14% to 65%, and reaching up to 60% in patients admitted to intensive care units.<sup>[14, 15]</sup> While elevated cardiac biomarkers and electrocardiogram abnormalities typically indicate myocardial injury in HS, these findings can lead to the misdiagnosis of acute myocardial infarction, particularly in emergency settings.<sup>[16]</sup> Therefore, the high mortality rate associated with HS urgently requires effective early risk-warning tools. Using multivariate logistic regression analysis, this study identified advanced age, elevated cTnI levels, high qSOFA scores, and increased SII as independent risk factors for HS. By combining cTnI with age, qSOFA score, and SII, a strong predictive model was constructed.

Heat-induced cardiac injury progresses rapidly in HS and often manifests within the first day of the illness.<sup>[17]</sup> HS triggers an inflammatory storm through the TLR4/NF- $\kappa$ B signaling

**Table 1**  
**Clinical Data Comparison Between 2 Groups of Patients with Heat Stroke**

| Variables                | Survival Group (n = 165) | Nonsurvival Group (n = 44) | Z/ $\chi^2$ | P Value |
|--------------------------|--------------------------|----------------------------|-------------|---------|
| T (°C)                   | 39.65 (38.32–40.50)      | 39.8 (39.0–40.75)          | −1.060      | 0.289   |
| Age (yr)                 | 61 (50–70)               | 81 (75–88)                 | −8.159      | <0.001  |
| Sex                      |                          |                            |             |         |
| Male                     | 115 (69.70)              | 20 (45.50)                 | 8.926       | 0.003   |
| Female                   | 50 (30.30)               | 24 (54.50)                 |             |         |
| Comorbidities            |                          |                            |             |         |
| Yes                      | 67 (40.60)               | 24 (54.50)                 | 0.187       | 0.665   |
| No                       | 98 (59.40)               | 20 (45.50)                 |             |         |
| Heat stroke types        |                          |                            |             |         |
| CHS                      | 90 (54.50)               | 37 (84.10)                 | 12.719      | <0.001  |
| EHS                      | 75 (45.50)               | 7 (15.90)                  |             |         |
| HR (beats/min)           | 99 (80–120)              | 110 (87–126)               | −1.470      | 0.142   |
| RR (beats/min)           | 20 (20–23)               | 22 (20–27.5)               | −2.415      | 0.016   |
| SBP (mm Hg)              | 110 (123–134)            | 104 (119–135)              | 0.596       | 0.551   |
| DBP (mm Hg)              | 73 (62–81)               | 69 (59.50–84.50)           | 0.744       | 0.457   |
| WBCs ( $\times 10^9/L$ ) | 11.69 (8.25–15.25)       | 13.53 (9.95–16.69)         | −1.777      | 0.076   |
| NEUT ( $\times 10^9/L$ ) | 9.42 (6.07–13.11)        | 11.26 (8.07–14.90)         | −1.706      | 0.088   |
| NEUT%                    | 84.40 (75.50–89.30)      | 86.9 (80.65–90.15)         | −1.819      | 0.069   |
| MONO ( $\times 10^9/L$ ) | 0.60 (0.41–0.92)         | 0.57 (0.39–1.09)           | −0.287      | 0.774   |
| MONO%                    | 5.40 (4.20–7.10)         | 5.20 (3.85–7.60)           | 0.015       | 0.988   |
| LYM ( $\times 10^9/L$ )  | 1.06 (0.69–1.63)         | 0.81 (0.56–1.30)           | 1.898       | 0.058   |
| LYM%                     | 9.30 (5.60–17.20)        | 6.70 (4.70–11.45)          | 2.035       | 0.042   |
| EOS ( $\times 10^9/L$ )  | 0.01 (0.00–0.04)         | 0.01 (0.00–0.03)           | 0.646       | 0.518   |
| EOS%                     | 0.1 (0.00–0.38)          | 0.1 (0.00–0.25)            | 0.888       | 0.374   |
| PLT ( $\times 10^9/L$ )  | 159.00 (96.00–232.00)    | 135.00 (104.00–190.50)     | 0.641       | 0.521   |
| PT (s)                   | 14.70 (13.34–16.78)      | 15.80 (14.15–17.60)        | −1.795      | 0.073   |
| APTT (s)                 | 35.30 (31.30–39.10)      | 35.20 (32.80–43.00)        | −0.712      | 0.476   |
| INR                      | 1.19 (1.06–1.37)         | 1.29 (1.12–1.46)           | −2.012      | 0.044   |
| TT (s)                   | 17.40 (16.10–18.50)      | 17.20 (16.35–18.95)        | −0.555      | 0.579   |
| F1b (g/L)                | 2.74 (2.31–3.41)         | 3.21 (2.25–3.98)           | −1.418      | 0.156   |
| CK (U/L)                 | 408.65 (207.00–1223.00)  | 368.00 (188.00–1557.50)    | 0.206       | 0.837   |
| CK-MB (ng/mL)            | 9.13 (4.16–21.98)        | 9.00 (3.86–18.00)          | 0.061       | 0.951   |
| LDH (U/L)                | 300.50 (207.00–439.00)   | 349.00 (256.00–478.00)     | −1.604      | 0.109   |
| HBDH (U/L)               | 202.00 (154.25–329.50)   | 264.00 (190.00–426.00)     | −2.445      | 0.015   |
| Myoglobin (ng/mL)        | 253.10 (51.55–996.00)    | 463.60 (153.85–1333.20)    | −1.946      | 0.052   |
| cTnI (ng/mL)             | 0.02 (0.00–0.24)         | 2.10 (0.10–6.10)           | −7.122      | <0.001  |
| qSOFA                    | 1 (0–2)                  | 2 (2–3)                    | −6.148      | <0.001  |
| APACHE II                | 11 (7–15)                | 17 (12–25)                 | −5.221      | <0.001  |
| SII                      | 1248.90 (631.91–2464.24) | 1903.87 (903.05–2865.57)   | −1.962      | 0.05    |
| NLR                      | 8.41 (4.24–15.86)        | 14.20 (7.65–24.30)         | −3.334      | <0.001  |

Data are n (%) or median (IQR).

APACHE II, Acute Physiology and Chronic Health Evaluation II; APTT, activated partial thromboplastin time; CHS, classical heat stroke; CK, creatine kinase; CK-MB, creatine kinase-myocardial band; cTnI, cardiac troponin I; DBP, diastolic blood pressure; EHS, exertional heat stroke; EOS, eosinophils; F1b, fibrinogen degradation products; HBDH, hydroxybutyrate dehydrogenase; HR, heart rate; INR, international normalized ratio; IQR, interquartile range; LDH, lactate dehydrogenase; LYM, lymphocytes; MONO, monocytes; NEUT, neutrophils; NLR, neutrophil-to-lymphocyte ratio; PLT, platelets; PT, prothrombin time; qSOFA, quick Sequential (Sepsis-related) Organ Failure Assessment; RR, respiratory rate; SBP, systolic blood pressure; SII, systemic immune-inflammation index; T, temperature; TT, thrombin time; WBCs, white blood cells.

pathway, leading to systemic inflammatory response syndrome and endothelial cell injury, while suppressing the antioxidant defense system and inducing ferroptosis in cardiomyocytes, resulting in a marked elevation of troponin levels.<sup>[18]</sup> Audet et al.<sup>[19]</sup> demonstrated in an experimental model that cTnI concentration rises sharply when the core body temperature peaks, with cTnI levels in severely affected rats being 676 times higher than those in controls, showing a strong correlation with disease severity over the following 24 hours. Similarly, in a large cohort study conducted during the 2003 heat wave in France, Hausfater et al.<sup>[20]</sup> found that elevated cTnI was common in patients with CHS and severely elevated cTnI (>1.50 ng/mL) served as an independent predictor of mortality. Our observations were consistent with these findings. Our study further substantiates that cTnI, as a key independent risk factor, has

significant discriminative value for mortality risk. Data analysis revealed that cTnI levels were markedly elevated in the deceased group, with the median value substantially exceeding the clinical diagnostic threshold (>0.04 ng/mL). At the optimal cutoff of 1.24 ng/mL, cTnI demonstrated a sensitivity of 66.70% and a specificity of 90.20% in identifying fatal outcomes. These findings suggest that this cutoff has meaningful clinical utility for risk stratification. Therefore, early and rapid measurement of cTnI may aid in the emergency assessment and risk management of severe HS.

This study revealed that advanced age was an independent risk factor for mortality in patients with HS. The data showed that the median age of nonsurvivors was significantly higher than that of survivors. Stafoggia et al.<sup>[21]</sup> clearly demonstrated in a multicity study that heat-related mortality from noninjury causes

**Table 2**  
**Univariate and Multivariate Logistic Regression Analyses of the Risk Factors for Heat Stroke Mortality**

| Variables         | Univariate Logistic |            |         | Multivariate Logistic |           |         |
|-------------------|---------------------|------------|---------|-----------------------|-----------|---------|
|                   | OR                  | 95% CI     | P Value | OR                    | 95% CI    | P Value |
| Sex               |                     |            |         |                       |           |         |
| Male              |                     | Reference  |         |                       |           |         |
| Female            | 2.91                | 1.48–5.72  | 0.002   |                       |           |         |
| Comorbidities     |                     |            |         |                       |           |         |
| No                |                     | Reference  |         |                       |           |         |
| Yes               | 1.18                | 0.60–2.29  | 0.634   |                       |           |         |
| Heat stroke types |                     |            |         |                       |           |         |
| CHS               |                     | Reference  |         |                       |           |         |
| EHS               | 0.27                | 0.12–0.61  | 0.002   |                       |           |         |
| T                 | 1.15                | 0.93–1.44  | 0.204   |                       |           |         |
| Age               | 1.15                | 1.10–1.21  | <0.001  | 1.18                  | 1.11–1.26 | <0.001  |
| HR                | 1.00                | 1.00–1.00  | 0.694   |                       |           |         |
| RR                | 1.06                | 1.01–1.11  | 0.012   |                       |           |         |
| SBP               | 1.00                | 1.00–1.01  | 0.282   |                       |           |         |
| DBP               | 1.00                | 0.99–1.02  | 0.696   |                       |           |         |
| WBCs              | 1.05                | 0.99–1.11  | 0.076   |                       |           |         |
| NEUT              | 1.04                | 0.99–1.10  | 0.150   |                       |           |         |
| NEUT%             | 1.02                | 0.99–1.06  | 0.139   |                       |           |         |
| MONO              | 1.54                | 0.90–2.63  | 0.117   |                       |           |         |
| MONO%             | 1.02                | 0.94–1.11  | 0.555   |                       |           |         |
| LYM               | 0.74                | 0.50–1.11  | 0.147   |                       |           |         |
| LYM%              | 0.96                | 0.92–1.01  | 0.090   |                       |           |         |
| EOS               | 0.39                | 0.00–33.45 | 0.677   |                       |           |         |
| EOS%              | 0.99                | 0.61–1.59  | 0.958   |                       |           |         |
| PLT               | 1.00                | 1.00–1.00  | 0.522   |                       |           |         |
| PT                | 0.99                | 0.96–1.03  | 0.742   |                       |           |         |
| APTT              | 1.01                | 0.99–1.03  | 0.316   |                       |           |         |
| INR               | 1.17                | 0.64–2.15  | 0.613   |                       |           |         |
| TT                | 0.98                | 0.93–1.03  | 0.415   |                       |           |         |
| F1b               | 1.00                | 0.97–1.02  | 0.722   |                       |           |         |
| CK                | 1.00                | 1.00–1.00  | 0.929   |                       |           |         |
| CK-MB             | 1.00                | 0.99–1.00  | 0.605   |                       |           |         |
| LDH               | 1.00                | 1.00–1.00  | 0.671   |                       |           |         |
| HBDH              | 1.00                | 1.00–1.00  | 0.421   |                       |           |         |
| Myoglobin         | 1.00                | 1.00–1.00  | 0.119   |                       |           |         |
| cTnI              | 1.85                | 1.45–2.35  | <0.001  | 1.67                  | 1.24–2.24 | <0.001  |
| qSOFA             | 3.06                | 2.06–4.54  | <0.001  | 1.83                  | 1.02–3.28 | 0.044   |
| APACHE II         | 1.16                | 1.10–1.22  | <0.001  |                       |           |         |
| SII*              | 1.17                | 1.03–1.32  | 0.013   | 1.34                  | 1.06–1.70 | 0.016   |
| NLR               | 1.03                | 1.01–1.05  | 0.009   |                       |           |         |

APACHE II, Acute Physiology and Chronic Health Evaluation II; APTT, activated partial thromboplastin time; CHS, classical heat stroke; CI, confidence interval; CK, creatine kinase; CK-MB, creatine kinase-myocardial band; cTnI, cardiac troponin I; DBP, diastolic blood pressure; EHS, exertional heat stroke; EOS, eosinophils; F1b, fibrinogen degradation products; HBDH, hydroxybutyrate dehydrogenase; HR, heart rate; INR, international normalized ratio; LDH, lactate dehydrogenase; LYM, lymphocytes; MONO, monocytes; NEUT, neutrophils; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; PLT, platelets; PT, prothrombin time; qSOFA, quick Sequential (Sepsis-related) Organ Failure Assessment; RR, respiratory rate; SBP, systolic blood pressure; SII, systemic immune-inflammation index; T, temperature; TT, thrombin time; WBCs, white blood cells.  
\*SII values were divided by 1000 for analysis; thus, OR represents the risk per 1000-unit increase.

increases significantly with age. The strong association between age and the risk of fatal HS stems from the fact that human adaptation to high temperatures relies on the body’s natural cooling capacity.<sup>[22]</sup> However, older adults face age-related physiological disadvantages, including reduced cardiovascular reserve leading to impaired redistribution of skin blood flow, diminished sweat gland function, a blunted thirst response contributing to chronic dehydration tendency, and weakened thermal perception.

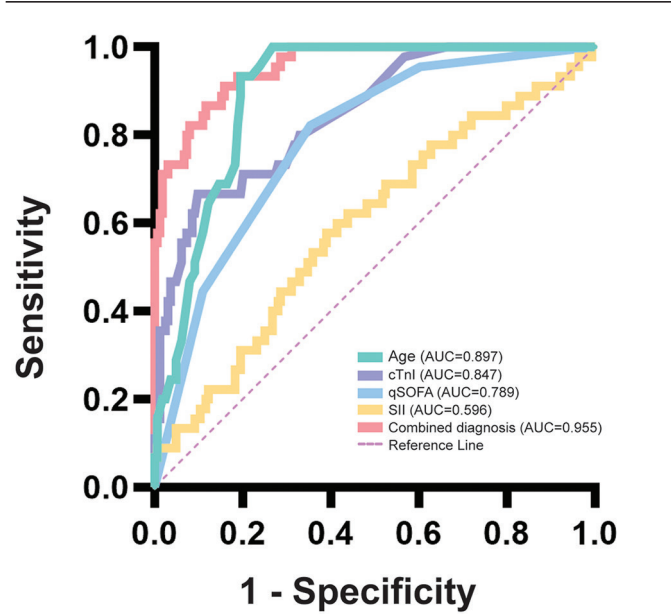


**Figure 2.** ROC curves of the combined prediction model, cTnI, age, qSOFA, and SII for predicting mortality in patients with heat stroke. AUC, area under the receiver operating characteristic curve; cTnI, cardiac troponin I; qSOFA, quick Sequential (Sepsis-related) Organ Failure Assessment; ROC, receiver operating characteristic; SII, systemic immune-inflammation index.

Together, these factors compromise the thermoregulatory ability of older individuals under heat stress, making them the most vulnerable to high-temperature environments.<sup>[23, 24]</sup> Consequently, older adult patients with HS have a markedly elevated risk of fatal outcomes. Advanced age should be considered as a central risk factor in the clinical management and prevention of HS and proactive interventions are essential to improve patient prognosis.

The qSOFA score and SII at emergency admission were also identified as independent risk factors for mortality in patients with HS, with both markers being significantly elevated in the nonsurvivor group compared with those in the survivor group. From a pathophysiological perspective, Bouchama and Knochel<sup>[3]</sup> noted that HS essentially represents a “sepsis-like” state characterized by the combined effects of systemic inflammatory response, oxidative stress, coagulopathy, and multiorgan cell death. In this process, damage-associated molecular patterns released from injured cells can further activate inflammatory and coagulation cascades, creating a “second hit” that perpetuates the progression of multiple organ dysfunction syndrome.<sup>[25]</sup> This mechanism is directly reflected in the 3 components of the qSOFA score: tachypnea indicates early compensatory response to systemic inflammation, hypotension reflects circulatory failure and organ ischemic risk, and altered mental status represents core central nervous system impairment in HS. As a comprehensive index of systemic immune inflammation, an elevated SII indicates an imbalanced state characterized by the co-existence of acute inflammatory activation and stress-induced immunosuppression. Such immunoinflammatory dysregulation not only correlates with acute disease severity but may also have long-term consequences. Epidemiological studies have shown that discharged patients with HS continue to face a significantly elevated risk of death from heart, liver, and kidney failure over the years to decades.<sup>[26]</sup> Zhang et al.<sup>[27]</sup> further proposed that

severe heat stress may induce persistent alterations in immune cell function through “epigenetic reprogramming,” leading to long-term immune dysfunction. Therefore, the qSOFA score and SII, as readily obtainable indicators at emergency admission, can promptly reflect both the critical condition of patients and their potential long-term risk, providing important evidence for improving patient prognosis.

Collectively, the findings of this study suggest that for patients with HS admitted to the emergency department, clinicians may incorporate cTnI along with age, qSOFA score, and SII to perform rapid risk stratification. This approach allows high-risk patients to be identified more promptly and provides an objective basis for initiating more aggressive treatments to improve long-term patient prognosis.

## Limitations

This is a multicenter retrospective analysis with certain inherent limitations. Although multicenter data enhances representativeness to some extent, retrospective studies cannot fully avoid issues such as selection bias and incomplete clinical data. Furthermore, while the sample size was relatively moderate, a larger cohort would further improve the model's stability and generalizability. Additionally, it is possible that not all potential confounding factors were fully accounted for. Despite internal validation using bootstrap resampling suggesting good model stability, the risk of overfitting cannot be entirely excluded, and the model remains in the developmental phase. Therefore, the model's robustness and clinical utility must be confirmed through rigorous external validation using independent cohorts and prospective studies.

## Conclusion

This study established that age, cTnI level, qSOFA score, and SII are independent risk factors for mortality in patients with HS. The AUC of the predictive model constructed based on these 4 indicators is 0.955. By integrating key markers reflecting patients' baseline status, cardiac injury, organ dysfunction, and inflammatory response, this model offers a more objective approach for clinical risk assessment of HS. Further steps will require external validation of the model through prospective multicenter studies and its translation into a more user-friendly clinical scoring tool.

## Conflict of interest statement

The authors declare no conflict of interest.

## Author contributions

Huang R contributed to writing the original draft, software, methodology, formal analysis, data curation, conceptualization, and review and editing. Wu X contributed to writing, review and editing, software, methodology, formal analysis, and data curation. Xiang Y and Jiang J contributed to software, resources, methodology, formal analysis, and data curation. Ai S, Que W, Wu H, and Zhang S contributed to software, methodology, investigation, and data curation. Zhou F contributed to writing, review and editing, visualization, validation, supervision, resources, project administration, data curation, conceptualization, and funding acquisition. Ma Y contributed to writing,

review and editing, visualization, validation, supervision, project administration, methodology, and conceptualization.

## Funding

This study was supported by an Eagle Talent Project of Chongqing Emergency Medical Center (SYRCCY20230312) and a key project co-organized by the Health Commission and Science & Technology Bureau of Chongqing Province (2024ZDXM024). This study was also supported by the General Project of the Chongqing Province Natural Science Foundation (CSTB2024NSCQ-MSX0873) and Chongqing Key Laboratory of Emergency Medicine (2023-KFKT-03).

## Ethical approval of studies and informed consent

The study followed the principles of the Declaration of Helsinki as revised in 2013. This multicenter study was approved, and written informed consent was waived by a steering ethics committee, Ethical Committee of Chongqing Emergency Medical Center (approval number: 2023120701, 2023-11-28), which provided approval for all participating sites.

## Acknowledgments

We thank all the patients who participated in this study, as well as all the clinicians and staff involved in data collection and analysis. We thank Editage for the language polishing.

## References

- [1] Peiris AN, Jaroudi S, Noor R. Heat stroke. *JAMA*. 2017;318(24):2503. doi:10.1001/jama.2017.18780.
- [2] Bouchama A, Abuyassin B, Lehe C, et al. Classic and exertional heatstroke. *Nat Rev Dis Primers*. 2022;8(1):8. doi:10.1038/s41572-021-00334-6.
- [3] Bouchama A, Knochel JP. Heat stroke. *N Engl J Med*. 2002;346(25):1978–1988. doi:10.1056/NEJMra011089.
- [4] Nzvere FP, Tariq E, Nishanth K, Arshid A, Cancarevic I. Long-term cardiovascular diseases of heatstroke: a delayed pathophysiology outcome. *Cureus*. 2020;12(8):e9595. doi:10.7759/cureus.9595.
- [5] Marchand M, Gin K. The cardiovascular system in heat stroke. *CJC Open*. 2022;4(2):158–163. doi:10.1016/j.cjco.2021.10.002.
- [6] Crandall CG, González-Alonso J. Cardiovascular function in the heat-stressed human. *Acta Physiol (Oxf)*. 2010;199(4):407–423. doi:10.1111/j.1748-1716.2010.02119.x.
- [7] Wilson TE, Crandall CG. Effect of thermal stress on cardiac function. *Exerc Sport Sci Rev*. 2011;39(1):12–17. doi:10.1097/JES.0b013e318201eed6.
- [8] Chao CM, Wang LY, Huang CC, et al. Myocardial structure and functional alterations in a preclinical model of exertional heat stroke. *Life Sci*. 2023;323:121640. doi:10.1016/j.lfs.2023.121640.
- [9] Akhtar MJ, al-Nozha M, al-Harthi S, Nouh MS. Electrocardiographic abnormalities in patients with heat stroke. *Chest*. 1993;104(2):411–414. doi:10.1378/chest.104.2.411.
- [10] Zhong L, Ji J, Wang C, Liu Z. Clinical characteristics and risk factors of male exertional heatstroke in patients with myocardial injury: an over 10-year retrospective cohort study. *Int J Hyperthermia*. 2021;38(1):970–975. doi:10.1080/02656736.2021.1941312.
- [11] Liu SY, Song JC, Mao HD, Zhao JB, Song Q; Expert Group of Heat Stroke Prevention and Treatment of the People's Liberation Army, and People's Liberation Army Professional Committee of Critical Care Medicine. Expert consensus on the diagnosis and treatment of heat stroke in China. *Mil Med Res*. 2020;7(1):1. doi:10.1186/s40779-019-0229-2.
- [12] Thygesen K, Alpert JS, Jaffe AS, et al; Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition

- of Myocardial Infarction. Fourth universal definition of myocardial infarction (2018). *J Am Coll Cardiol*. 2018;72(18):2231–2264. doi:10.1016/j.jacc.2018.08.1038.
- [13] Sandoval Y, Apple FS, Saenger AK, Collinson PO, Wu AHB, Jaffe AS. 99th percentile upper-reference limit of cardiac troponin and the diagnosis of acute myocardial infarction. *Clin Chem*. 2020;66(9):1167–1180. doi:10.1093/clinchem/hvaa158.
- [14] Hart GR, Anderson RJ, Crumpler CP, Shulkin A, Reed G, Knochel JP. Epidemic classical heat stroke: clinical characteristics and course of 28 patients. *Medicine (Baltim)*. 1982;61(3):189–197. doi:10.1097/00005792-198205000-00005.
- [15] Argaud L, Ferry T, Le QH, et al. Short- and long-term outcomes of heatstroke following the 2003 heat wave in Lyon, France. *Arch Intern Med*. 2007;167(20):2177–2183. doi:10.1001/archinte.167.20.10147.
- [16] Chen WT, Lin CH, Hsieh MH, Huang CY, Yeh JS. Stress-induced cardiomyopathy caused by heat stroke. *Ann Emerg Med*. 2012;60(1):63–66. doi:10.1016/j.annemergmed.2011.11.005.
- [17] Dervišević E, Hasić S, Katika M, Dervišević L, Ajanović Z, Salihbegović A. Heat-related biomarkers: focus on the correlation of troponin I and 70 kDa heat shock protein. *Heliyon*. 2023;9(4):e14565. doi:10.1016/j.heliyon.2023.e14565.
- [18] Xia R, Sun M, Li Y, et al. The pathogenesis and therapeutic strategies of heat stroke-induced myocardial injury. *Front Pharmacol*. 2023;14:1286556. doi:10.3389/fphar.2023.1286556.
- [19] Audet GN, Quinn CM, Leon LR. Point-of-care cardiac troponin test accurately predicts heat stroke severity in rats. *Am J Physiol Regul Integr Comp Physiol*. 2015;309(10):R1264–R1272. doi:10.1152/ajpregu.00286.2015.
- [20] Hausfater P, Doumenc B, Chopin S, et al. Elevation of cardiac troponin I during non-exertional heat-related illnesses in the context of a heatwave. *Crit Care*. 2010;14(3):R99. doi:10.1186/cc9034.
- [21] Stafoggia M, Forastiere F, Agostini D, et al. Vulnerability to heat-related mortality: a multicity, population-based, case-crossover analysis. *Epidemiology*. 2006;17(3):315–323. doi:10.1097/01.ede.0000208477.36665.34.
- [22] Hajat S, O'Connor M, Kosatsky T. Health effects of hot weather: from awareness of risk factors to effective health protection. *Lancet*. 2010;375(9717):856–863. doi:10.1016/S0140-6736(09)61711-6.
- [23] Worfolk JB. Heat waves: their impact on the health of elders. *Geriatr Nurs*. 2000;21(2):70–77. doi:10.1067/mgn.2000.107131.
- [24] Kenney WL, Munce TA. Invited review: aging and human temperature regulation. *J Appl Physiol (1985)*. 2003;95(6):2598–2603. doi:10.1152/japplphysiol.00202.2003.
- [25] Wang Z, Zhu J, Zhang D, Lv J, Wu L, Liu Z. The significant mechanism and treatments of cell death in heatstroke. *Apoptosis*. 2024;29(7-8):967–980. doi:10.1007/s10495-024-01979-w.
- [26] Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response. *J Appl Physiol (1985)*. 2010;109(6):1980–1988. doi:10.1152/japplphysiol.00301.2010.
- [27] Zhang Z, Wu X, Zou Z, et al. Heat stroke: pathogenesis, diagnosis, and current treatment. *Ageing Res Rev*. 2024;100:102409. doi:10.1016/j.arr.2024.102409.

---

**How to cite this article:** Huang R, Wu X, Xiang Y, et al. Mortality risk stratification in heat stroke using cardiac troponin I and bedside parameters: a retrospective study. *Emerg Crit Care Med*. 2026;6(2):110–116. doi: 10.1097/EC9.000000000000188