ORIGINAL RESEARCH

# Analysis of Prognostic Risk Factors of Sepsis Patients With Myocardial Injury: Six-month Survival Outcome

Guangqing Huang, MM; Wenzi Yang, MM; Xu Zhao, MM; Yong Bai, MM; Xing Jiang, MM; Jie Liu, MD


## ABSTRACT

**Context** • Sepsis is a systemic, comprehensive inflammatory response that can induce serious complications for patients. No uniform definition and diagnostic criteria exist internationally for sepsis related to myocardial injury. Studying factors affecting prognosis for sepsis patients with myocardial injury can be helpful in providing a theoretical basis for clinical diagnosis and treatment.

**Objective** • The study intended to explore the predictors of the short-term prognosis for septic patients with myocardial injury and to provide a theoretical basis for improving that prognosis.

**Design** • The research team performed a retrospective study.

**Setting** • The study took place at the Renmin Hospital at Hubei University of Medicine in Shiyan, Hubei, China.

**Participants** • Participants were 138 patients with sepsis and myocardial injury at the hospital between January 2018 and February 2021.

**Groups** • The research team divided participants into a survival group with 114 patients and a mortality group with 24 patients, based on their survival status at six months after being in the hospital.

**Outcome Measures** • The research team collected and analyzed the following data: (1) demographic and clinical characteristics, such as age, gender, underlying disease, and disease severity; (2) echocardiographic indicators, including left ventricular ejection fraction (LVEF), stroke volume (SV), left ventricular end systolic dimension (LVESD), and left ventricular end diastolic dimension (LVEDD); and (3) myocardial injury markers and inflammatory factors, including white blood cell (WBC) count and levels of cardiac troponin I (cTnI), N-terminal pro-brain natriuretic peptide (NT-proBNP), creatine kinase (CK), C-reactive protein (CRP), and procalcitonin (PCT).

**Results** • The six-month mortality rate for patients with sepsis with myocardial injury was 17.39%. Compared to the survival group at participants' initial visits to the hospital, the mortality group had a significantly greater age ($P = .046$), sepsis severity ($P < .001$), heart rate ($P < .001$), APACHE II score ($P < .001$), SOFA score ($P < .001$), and use of vasoactive drugs ($P = .002$), and its length of hospital stay was significantly shorter ($P < .001$). The mortality group's LVEF was significantly lower than that of the survival group ($P < .001$). The mortality group's levels of WBC, cTnI, NT-proBNP, CK, CRP, and PCT were significantly higher than those in survival group (all $P < .001$). The univariate analysis found that an age>64 years ($P < .001$), a high APACHE II score ($P < .001$), an elevated cTnI ($P = .017$), an elevated NT-proBNP ($P = .029$), an elevated CK ($P < .001$), an elevated CRP ($P = .031$), and an elevated PCT ($P < .001$) were risk factors for a poor prognosis for patients. Multifactor logistic regression analysis showed that the risk factors for death were an age>64 years ($P < .001$), a high APACHE II score ($P < .001$), and elevated levels of cTnI ($P = .013$), NT-proBNP ($P < .001$), CK ($P < .001$), CRP ($P < .001$), and PCT ($P = .009$).

**Conclusion** • In summary, risk factors for poor prognosis in septic patients with myocardial injury included age>64 years, high APACHE II, elevated cTnI, elevated NT-proBNP, elevated CK, elevated CRP, and elevated PCT. (*Altern Ther Health Med.* 2023;29(8):744-749).


**Guangqing Huang, MM; Wenzi Yang, MM; Xu Zhao, MM; Yong Bai, MM; Jie Liu, MD**; Emergency and Critical Care Center, Renmin Hospital, Hubei University of Medicine, Shiyan, Hubei, China. **Xing Jiang, MM**, Reproductive Medicine Center, Renmin Hospital, Hubei University of Medicine and Biomedical Engineering College, Hubei University of Medicine, Shiyan, Hubei, China

*Corresponding author: Jie Liu, MD*
*E-mail: 28799898@qq.com*
*Corresponding author: Xing Jiang, MM*
*E-mail: odudk0@163.com*

Sepsis is a systemic, comprehensive inflammatory response that can induce serious complications for patients being treated for other diseases, such as coagulation abnormalities, multiple organ failure, and intestinal bacterial translocation.[1] Globally as of 2020, more than 19-million patients had developed sepsis each year, including about six-million deaths and a mortality rate of more than 25%, posing a serious threat to the health of patients.[2] Madorin et al found that the mortality rate in multicenter studies was 17.9%.[3]

However, some studies have reported significantly higher mortality rates than those described above. Mu et al and Sevilla et al found that the mortality rates from sepsis with myocardial injury were 44.1% and 52%, respectively.[4,5] Wang et al found

that about 40% of septic patients had varying degrees of myocardial dysfunction, while the mortality rate from sepsis with myocardial injury was as high as 70%.[6]

Many factors may have caused this difference, including different characteristics of the observed patients, different geographical environments, differences in the distribution and level of medical resources, and sample sizes. Xing et al pointed out that etiological characteristics, such as the site of the infection, can be associated with differences in disease mortality.[7]

The mortality rate for sepsis with myocardial injury is high, and the prevention, diagnosis and treatment of sepsis with myocardial injury in clinical work need continuous strengthening.

## Mechanisms

Xin and Lu indicate that researchers haven't comprehensively elaborated the mechanism of myocardial injury that sepsis causes.[8] Sepsis-induced myocardial injury is associated with such factors as hemodynamic influences, changes in cardiomyocyte metabolism, activation of inflammatory pathways, cardiomyocyte apoptosis, and autonomic dysfunction.

Wu et al found that myocardial ischemia, hypoxia, and increased vascular endothelin-1 secretion can occur in the early stage of sepsis, which can decrease vasoconstriction ability and lead to myocardial ischemia.[9] Those researchers also found that myocardial ischemia can hinder the scavenging of oxygen free radicals, that free radicals can burst after blood-supply recovery, resulting in an overload of calcium ions ($Ca^{2+}$), and that the vicious cycle can aggravate the injury of myocardial cells.

Patients with sepsis release large amounts of bacterial endotoxin in their blood, have activated inflammatory cells, secrete inflammatory mediators, and produce many free radicals. Oxygen free radicals, cytokines, and endotoxin could increase the permeability of the myocardial-cell membrane, and myocardial-cell necrosis may occur in severe cases, even leading to the death of patients.[10]

## Myocardial Enzymes

Myocardial enzymes are specific markers of myocardial cell injury and include cardiac troponin I (cTnI), creatine kinase (CK), N-terminal pro-brain natriuretic peptide (NT-proBNP), procalcitonin (PCT), and C-reactive protein (CRP). White blood cells (WBC) produce a number of enzymes that help fight disease.

**cTnI and CK.** In the early stage of myocardial injury, the body releases these enzymes into the blood, and they closely relate to the degree of myocardial injury.[11] CTnI is a contractile protein in myocardial tissue, which is very low in human blood under normal conditions, and only myocardial tissue expresses it. When myocardial injury occurs, the level of cTnI can be higher in the human body, and it's one of the most reliable markers of myocardial injury at present. CK is widely distributed in the human body and can peak in the short time of 2-6 hours after myocardial injury, making it an important indicator for assessing myocardial injury.

**NT-proBNP.** NT-proBNP is a neuroendocrine hormone that ventricular myocytes secrete, and its levels can increase with ventricular overload, which is a sensitive indicator of cardiac function.[12]

**PCT.** PCT is significantly increased in bacterial infections, severe shock, and multiple-organ-failure syndrome, with high sensitivity and specificity, and clinicians commonly use it for auxiliary diagnosis, disease stratification, efficacy monitoring, and prognosis evaluation in sepsis.[13]

**CRP.** CRP is a pro-inflammatory factor, and the aggravation of systemic infection dysregulates the responses of pro-inflammatory factors and anti-inflammatory factors. Circulating inflammatory mediators can act directly on cardiomyocytes and the peripheral vascular system, resulting in sepsis-associated myocardial injury.

**WBC.** WBC can secrete various cytokines, such as interleukins (ILs), interferons (IFNs), and tumor necrosis factors (TNFs), that are involved in the regulation of inflammation and immune responses.[14]

## Prognostic Indicators

Li et al found that serum levels of cTnI, myoglobin, CK-myoglobin binding (CK-MB), and NT-proBNP were closely related to the degree of myocardial injury in patients with carbon monoxide poisoning and that clinicians can use them as indicators to assess clinical efficacy.[15] Peng et al used PCT, CK-MB, NT-proBNP, and cTnT-high-sensitive (hs) singly to determine the severity of sepsis in children.[16] Frencken et al found that NT-proBNP, cTnI, and hs-CRP were highly expressed in septic patients with myocardial injury.[17]

Xia et al pointed out that advanced age and elevated cTnI were high-risk factors for sepsis with cardiac injury and that myocardial injury was more severe in older patients with sepsis-related diastolic dysfunction.[18] Chronic diseases, a poor nutritional status, reduced multiple-organ-function reserve, and decreased immunity, often accompany aging, resulting in weak body resistance and compensatory ability and often in a poor prognosis.[10]

Wang and Chen found that levels of PCT, compared with those of CRP, had a stronger correlation with scores on the Acute Physiology and Chronic Health Evaluation II (APACHE II)[19] and the sequential organ failure assessment (SOFA)[20] and could better reflect a patient's condition and prognosis.[21] Chen et al's study concluded that the levels of cTnI and CK-MB and the APACHE-II[19] scores were significantly higher in patients who died of sepsis with cardiomyopathy.[22]

Mu et al concluded that the independent risk factors for septic patients with myocardial infarction were elevated PCT and decreased left ventricular ejection fraction (LVEF).[23] Chen et al suggested that 60% of patients with septic shock may have left ventricular hypodynamic changes, including diminished systolic function and that decreased LVEF indicated an impaired left ventricular systolic function and reduced ejection capacity.[24]

Li believed that the risk factors for septic patients with myocardial injury was NT-proBNP and cTnI.[28] Zhang et al

found that a high APACHE II[19] score, pulmonary infection and combined acute kidney injury were risk factors for death in the disease.[25] Zhao et al found that the main risk factors in patients with septic myocardial injury are advanced age, high levels of TNF alpha (TNF-α), and white medium diathesis IL-1 beta (IL-1β) and nitric oxide (NO).[26]

Olsen et al confirmed that clinicians can use APACHE II[7] to assess the condition and prognosis of critical patients and that a significant correlation existed between APACHE II[7] and death for those patients.[27] Li et al found that cTnI, NT-proBNP, and CK could effectively assess cardiac-function status and that their levels were closely related to the severity of myocardial injury in sepsis.[28]

### Diagnosis

Clearly, research has provided no consensus about prognostic indicators. Also, no uniform definition and diagnostic criteria exist internationally for sepsis related to cardiac-machine injury, which can adversely affect the prognosis of patients and lead to adverse outcomes.[29-31] Timely identification and intervention for septic patients with myocardial injury can effectively improve their prognosis. Therefore, studying factors affecting prognosis for sepsis patients with myocardial injury can be helpful in providing a theoretical basis for clinical diagnosis and treatment.

The prognosis for patients with sepsis with myocardial injury is very serious. Early identification of sepsis for patients with myocardial injury, identification of related risk factors, and performance of key interventions can effectively improve patients' prognosis and are of great significance in clinical practice.[32]

### Current Study

The current study intended to explore the predictors of the short-term prognosis for septic patients with myocardial injury and to provide a theoretical basis for improving that prognosis.

## METHODS

### Participants

The research team performed a retrospective study, which took place at the Renmin Hospital at Hubei University of Medicine in Shiyan, Hubei, China. Potential participants were patients with sepsis and myocardial injury at the hospital between January 2018 and February 2021.

The study included potential participants if they: (1) met all of the clinical diagnostic criteria for sepsis, which a doctor-in-charge had diagnosed[33]; (2) had a significant or suspected infection, accompanied by a body temperature of >38°C or <36°C , a heart rate of >90 beats/min, shortness of breath, a respiratory rate of >30 beats/min, or changes in consciousness; (3) had a sequential organ failure assessment (SOFA) score of ≥2 points[20]; (4) met the clinical diagnostic criteria for a myocardial injury[34]: (5) had a left ventricular ejection fraction (LVEF) of <50% and a level of serum troponin T (cTnT) of >0.03 μg/L; (6) had no known heart disease, such as congenital heart disease, valvular heart disease, cardiomyopathy, coronary heart disease, or a history of other heart disease; and (7) were patients at the hospital for the first time.

The study excluded potential participants if they: (1) had primary liver or kidney disease; (3) had had no blood tests, cardiac ultrasounds, or ECG examinations during hospitalization; (3) had had a cardiac ultrasound of poor quality because they were uncooperative; (4) had had a cardiac ultrasound that revealed a moderate-to severe-mitral valve, active valve disease, or regional, myocardial, wall-movement abnormalities; (5) had a history of cardiothoracic surgery; or (6) were lactating or pregnant women.Participants signed written informed consent forms. The research team formulated the study's protocols in accordance with the requirements of the Declaration of Helsinki of the World Medical Association, and the hospital's Ethics Committee approved them.

### Procedures

**Groups.** The research team continuously recruited participants for the study.

**Participants' characteristics.** The research team recorded all of participants' demographic and clinical characteristics at their initial visits.

**Echocardiographic indicators.** Sonographers with more than 10 years of work experience at the hospital completed the assessment. At participants' initial visit, the sonographer measured LVEF, stroke volume (SV), left ventricular end systolic dimension (LVESD), and left ventricular end diastolic dimension (LVEDD) using a Philips iE33 ultrasound machine.

**Myocardial injury markers and inflammatory factors.** At the time of participants' initial admission, the hospital: (1) collected 5.0 mL of participants' peripheral venous blood in two tubes; (2) submitted one tube to hypothermia after centrifugation and extraction of supernatant and measured the WBC count using a Mindray BS-800 biochemical analyzer; (3) submitted the other tube to hypothermia after centrifugation and extraction of supernatant and measured levels of cTnI, NT-proBNP, CK, CRP, and PCT using an enzyme-linked immunosorbent assay (ELISA) from manufacturer.

### Outcome Measures

**Demographic and clinical data.** The research team compared the survival and mortality groups' characteristics—age, gender, underlying disease—hyperlipidemia, diabetes, hypertension, or chronic obstructive pulmonary disease, sepsis severity, site of infection, temperature, respiratory rate, heart rate, score on APACHE II,[19] score on SOFA,[20] length of hospital stay, smoking habits, alcohol consumption, need for mechanical ventilation, and use of vasoactive drugs.

**Echocardiographic indicators.** For all parameters, the research team averaged results from 3-5 cardiac cycles.

**Univariate analysis.** The research team excluded heart rate upon admission using multiple collinearity testing, with a correlation coefficient of >0.7. In the logistic regression analysis, the team included the independent variables of age, sepsis severity, APACHE II score, SOFA score, length of hospital stay, use of mechanical ventilation, use of vasoactive drugs, and levels of WBC, cTnI, NT-proBNP, CK, CRP, and PCT, and prognosis was the dependent variable.

**Multivariate analysis.** The research team included the independent variables set as outcome measures that had differences in the comparisons among single factors—age>64 years old, APACHE II score, and levels of cTnI, NT-proBNP, CK, CRP, and PCT, and prognosis was the dependent variable.

### Statistical Analysis

The research team analyzed all data using SPSS 21.0 software. The team: (1) tested the normality of continuous variables using the K-S test; (2) expressed normally distributed measurement data as means ± standard deviations (SDs) and measurement data not normally distributed as medians and quartiles (P25, P75) and compared the groups using the Student's *t* test and Mann-Whitney test for nonparametric distributions; (3) expressed categorical data as numbers (n) and percentages (%) and compared the groups using the Chi-square ($\chi^2$) analysis or Fisher's Exact Test; and (4) identified the risk factors affecting prognosis of septic patients with myocardial injury using logistic regression analysis. $P < .05$ for a two-sided test indicated statistical significance.

## RESULTS

### Participants

The research team included and analyzed the data of 138 participants, 114 in the survival group and 24 in the mortality group. The six-month mortality rate from sepsis for participants with myocardial injury was 17.39% for 24 participants, with a range for time to death of 5-28 days and a mean of 17.09 ± 10.67 days . Moreover, all deaths happened within 30 days of treatment.

At the initial visit (Table 1), no significant differences existed between the groups in gender, underlying disease, site of infection, temperature, respiratory rate, use of mechanical ventilation, alcohol consumption, or smoking habits ($P > .05$).

The mortality group's mean age, at 72.28 ± 15.82 years, was significantly older than that of the survival group, at 65.02 ± 14.29, with $P = .046$, and its sepsis severity, at eight participants with shock (33.33%), was significantly higher than that of the survival group, at three participants with shock (2.63%), with $P < .001$.

The mortality group's mean heart rate, at 105.98 ± 4.98 beats/min, was significantly higher than that of the survival group, at 98.17 ± 3.29 beats/min, with p<0.001, and its mean APACHE II score, at 29.76 ± 2.28 points, was significantly higher than that of the survival group, at 22.92 ± 2.31 points, with $P < .001$.

The mortality group's mean SOFA score, at 9.21 ± 1.32 points, was significantly higher than that of the survival group, at 7.12 ± 1.23 points, with $P < .001$, and its use of vasoactive drugs, with 15 participants receiving them (62.50%), was significantly higher than that of the survival group, with 34 participants receiving them (29.82%), with $P = .002$.

The mortality group's mean length of hospital stay, at 10.37 ± 3.43, was significantly shorter than that of the survival group, at 13.29 ± 3.29, with $P < .001$.

**Table 1.** Comparison of Participants' Demographic and Clinical Characteristics at the Initial Visit

| Characteristic | Survival Group n = 114 Mean ± SD n (%) | Mortality Group n=24 Mean ± SD n (%) | $\chi^2/t$ | *P* value |
|---|---|---|---|---|
| Age, y | 65.02 ± 14.29 | 72.28 ± 15.82 | -2.077 | .046[b] |
| Gender | | | 0.277 | .599 |
| Male | 73 (64.04) | 14 (58.33) | | |
| Female | 41 (35.96) | 10 (41.67) | | |
| Underlying Disease[a] | | | 0.191 | .979 |
| Hyperlipidemia | 34 (29.82) | 8 (33.33) | | |
| Diabetes | 30 (26.32) | 6 (25.00) | | |
| Hypertension | 58 (50.88) | 14 (58.33) | | |
| COPD | 23 (20.18) | 6 (25.00) | | |
| Sepsis Severity | | | 24.476 | <.001[d] |
| No shock | 111 (97.37) | 16 (66.67) | | |
| Shock | 3 (2.63) | 8 (33.33) | | |
| Site of Infection | | | 0.152 | .902 |
| Lung | 78 (68.42) | 18 (75.00) | | |
| Abdominal cavity | 11 (9.65) | 1 (4.17) | | |
| Urinary tract | 17 (14.91) | 3 (12.49) | | |
| Hematologic | 5 (4.39) | 1 (4.17) | | |
| Other | 3 (2.63) | 1 (4.17) | | |
| Temperature, °C | 36.98 ± 0.81 | 37.32 ± 1.03 | -1.783 | .077 |
| Respiratory Rate, beats/min | 23.16 ± 2.09 | 23.67 ± 2.56 | -1.043 | .299 |
| Heart rate, beats/min | 98.17 ± 3.29 | 105.98 ± 4.98 | -9.576 | <.001[d] |
| APACHE II, points | 22.92 ± 2.31 | 29.76 ± 2.28 | -13.213 | <.001[d] |
| SOFA score, points | 7.12 ± 1.23 | 9.21 ± 1.32 | -7.132 | <.001[d] |
| Length of hospital stay, d | 13.29 ± 3.29 | 10.37 ± 3.43 | 3.923 | <.001[d] |
| Use of Mechanical Ventilation | | | 3.741 | .053 |
| Yes | 61 (53.51) | 18 (75.00) | | |
| No | 53 (46.49) | 6 (25.00) | | |
| Use of Vasoactive Drugs | | | 9.244 | .002[c] |
| Yes | 34 (29.82) | 15 (62.50) | | |
| No | 80 (70.18) | 9 (37.50) | | |
| Alcohol Consumption | | | 0.145 | .704 |
| Yes | 20 (17.54) | 5 (20.83) | | |
| No | 94 (82.46) | 19 (79.17) | | |
| Smoking | | | 2.948 | .086 |
| Yes | 24 (21.05) | 9 (37.50) | | |
| No | 90 (78.95) | 15 (62.50) | | |

[a]Participants could have more than one underlying disease
[b]$P < .05$, indicating that the mortality group's age was significantly older than that of the survival group
[c]$P < .01$, indicating that the mortality group's use of vasoactive drugs was significantly greater than that of the survival group
[d]$P < .001$, indicating that the mortality group's sepsis severity, heart rates, Apache II scores, and SOFA scores were significantly higher than those of the survival group and length of hospital stay was significantly shorter than that of the survival group

**Abbreviations:** APACHE II, Acute Physiology and Chronic Health Evaluation II; COPD, chronic obstructive pulmonary disease; SOFA, sequential organ failure assessment.

### Echocardiographic Indicators

Table 2 shows that no significant differences existed in SV, LVESD, or LVEDD between the groups ($P > .05$). The mortality group's LVEF, at 32.32 ± 4.04, was significantly lower than that of the survival group, at 40.53 ± 3.03 ($P < .001$).

### Myocardial Injury Markers and Inflammatory Factors

Table 3 shows that the mortality group's median level of WBC, at 10.54 (6.68, 14.63) × $10^9$/L, was significantly higher than that of the survival group, at 8.62 (5.92, 12.09) × $10^9$/L

**Table 2.** Comparison of Echocardiographic Indicators

| Indicators | Survival Group n = 114 Mean ± SD | Mortality Group n = 24 Mean ± SD | *t* | *P* value |
|---|---|---|---|---|
| LVEF, % | 40.53 ± 3.03 | 32.32 ± 4.04 | 35.176 | <.001[a] |
| SV, ml | 44.91 ± 5.87 | 43.98 ± 6.03 | 0.702 | .484 |
| LVESD, mm | 65.87 ± 7.83 | 63.98 ± 7.92 | 1.073 | .285 |
| LVEDD, mm | 53.67 ± 5.98 | 54.27 ± 6.01 | -0.446 | .656 |

[a]*P* < .001, indicating that the mortality group's LVEF was significantly lower than that of the survival group

**Abbreviations:** LVEF, left ventricular ejection fraction; LVEDD, left ventricular end diastolic dimension; LVESD, left ventricular end systolic dimension; SV, stroke volume.

(*P* < .001), and its median level of cTnI, at 0.06 (0.02, 0.12) μg/L, was significantly higher than that of the survival group, at 0.02 (0.01, 0.05) μg/L (*P* < .001).

The mortality group's median level of NT-proBNP, at 3042.09 (13.02, 12076.97) ng/L, was significantly higher than that of the survival group, at 891.65 (263.19, 2848.09) ng/L (*P* < .001) and its median level of CK, at 147.98 (57.32, 638.91) U/L, was significantly higher than that of the survival group, at 88.38 (45.28, 203.81) U/L (*P* < .001).

The mortality group's median level of CRP, at 89.18 (53.29, 129.37) mg/L, was significantly higher than that of the survival group, at 62.18 (19.39, 103.29) mg/L (*P* < .001), and its median level of PCT, at 2.15 (0.29, 11.65) μg/L, was significantly higher than that of the survival group, at 0.27 (0.09, 1.27) μg/L (*P* < .001).

### Univariate Analysis

An age>64 years (*P* < .001), a high APACHE II score (*P* < .001), an elevated cTnI (*P* = .017), an elevated NT-proBNP (*P* = .029), an elevated CK (*P* < .001), an elevated CRP (*P* = .031), and an elevated PCT (*P* < .001) were all factors associated with a poor prognosis (Table 4).

### Multivariate Analysis

Table 5 shows that the risk factors for mortality were an age>64 years (*P* < .001), a high APACHE II (*P* < .001), an elevated cTnI (*P* = .013), an elevated cTnI NT-proBNP (*P* < .001), an elevated CK (*P* < .001), an elevated CRP (*P* < .001), and an elevated PCT (*P* = .009).

## DISCUSSION

The current study's mortality rate for patients with sepsis was 17.39% for 24 out of 138 participants, which was close to the mortality rate reported in a prior multicenter study, at 17.9%.[14] Significant differences existed between the survival and mortality groups in demographic and clinical characteristics at the initial visit. After logistic regression analysis, the current study found that an age>64 years and a high APACHE II, and elevated levels of cTnI, NT-proBNP, CK, CRP, and PCT may be risk factors for prognosis in patients with sepsis combined with myocardial injury.

The current study had some limitations. It was a single-center retrospective study with small sample size, which may weaken the generalizability of the results. Another limitation

**Table 3.** Comparison of Myocardial Injury Markers and Inflammatory Factors

| Outcome Measures | Survival Group n = 114 Median (P25, P75) | Mortality Group n = 24 Median (P25, P75) | *t* | *P* value |
|---|---|---|---|---|
| WBC, x10$^9$/L | 8.62 (5.92, 12.09) | 10.54 (6.68, 14.63) | -23.173 | <.001[a] |
| cTnI, μg/L | 0.02 (0.01, 0.05) | 0.06 (0.02, 0.12) | -26.903 | <.001[a] |
| NT-proBNP, ng/L | 891.65 (263.19, 2848.09) | 3042.09 (13.02, 12076.97) | -144.299 | <.001[a] |
| CK, U/L | 88.38 (45.28, 203.81) | 147.98 (57.32, 638.91) | -38.567 | <.001[a] |
| CRP, mg/L | 62.18 (19.39, 103.29) | 89.18 (53.29, 129.37) | -19.994 | <.001[a] |
| PCT, μg/L | 0.27 (0.09, 1.27) | 2.15 (0.29, 11.65) | -43.353 | <.001[a] |

[a]*P* < .001, indicating that the mortality group's WBC, cTnI, NT-proBNP, CK, CRP, and PCT were significantly higher than those of the survival group

**Abbreviations:** CK, creatine kinase; CRP, C-reactive protein; cTnI, cardiac troponin I; NT-proBNP, N-terminal pro-brain natriuretic peptide; PCT, procalcitonin; WBC, white blood cell count.

**Table 4.** Logistic Regression Analysis of Factors Affecting Prognosis in Sepsis With Myocardial Injury

| Factors | β | SE | Wald | OR (95% CI) | *P* value |
|---|---|---|---|---|---|
| Age, y | | | | | |
| 50-64 | 0.632 | 0.432 | 2.087 | 1.872 (0.793-3.872) | .156 |
| >64 | 1.563 | 0.516 | 10.187 | 3.298 (1.287-8.982) | <.001[b] |
| Severity, n | | | | | |
| No shock | 1.209 | 0.918 | 1.821 | 3.317 (0.545-8.932) | .189 |
| Shock | 1.055 | 0.901 | 1.351 | 2.847 (0.467-12.987) | .256 |
| APACHE II, points | 0.876 | 0.179 | 22.187 | 2.387 (1.721-3.281) | <.001[b] |
| SOFA score, points | 0.972 | 0.0509 | 3.398 | 2.267 (1.097-1.387) | .053 |
| Length of hospital stay, d | 0.643 | 0.467 | 1.326 | 1.908 (0.736-5.067) | .182 |
| Mechanical ventilation | 0.543 | 0.871 | 0.459 | 1.728 (0.563-2.653) | .092 |
| Use of vasoactive drugs | 0.278 | 0.279 | 1.187 | 0.708 (0.438-1.579) | .302 |
| WBC>10×109/L | 1.589 | 1.809 | 0.819 | 3.287 (1.198-6.587) | .378 |
| cTnI>0.6 μg/L | 0.612 | 0.261 | 5.321 | 1.783 (1.102-2.987) | .017[a] |
| NT-proBNP>3000.09 | 0.398 | 0.317 | 3.098 | 1.387 (1.065-1.879) | .029[a] |
| CK>145 U/L | 0.839 | 0.291 | 4.876 | 1.643 (1.019-2.876) | <.001[b] |
| CRP>85 mg/L | 0.301 | 0.328 | 2.791 | 1.537 (0.786-2.035) | .031[a] |
| PCT>2.10 μg/L | 0.876 | 0.179 | 22.187 | 2.387 (1.721-3.281) | <.001[b] |

[a]*P* < .05, indicating that a cTnI>0.6 μg/L, an NT-proBNP>3000.09, and a CRP>85 mg/L were significantly associated with a poor prognosis
[b]*P* < .001, indicating that an age >64, high APACHE II score, CK>145 U/L, and PCT>2.10 μg/L were significantly associated with a poor prognosis

**Abbreviations:** APACHE II, Acute Physiology and Chronic Health Evaluation II; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end diastolic dimension; LVESD, left ventricular end systolic dimension; SOFA, sequential organ failure assessment; SV, stroke volume.

**Table 5.** Multivariate Analysis of Factors Affecting Prognosis in Sepsis With Myocardial Injury

| Factors | β | SE | Wald $\chi^2$ | OR (95%CI) | *P* value |
|---|---|---|---|---|---|
| Age>64 years | 0.913 | 0.176 | 23.981 | 0.562 (0.372-0.781) | <.001[c] |
| APACHE II≥29 | 0.892 | 0.176 | 25.008 | 0.652 (0.378-0.897) | <.001[c] |
| cTnI>0.6μg/L | 0.983 | 0.451 | 5.009 | 2.113 (1.372-3.981) | .013[a] |
| NT-proBNP>3000.09 | 0.997 | 0.276 | 12.187 | 2.687 (1.576-3.281) | <.001[c] |
| CK>145U/L | 0.967 | 0.309 | 9.761 | 2.651 (1.562-3.981) | <.001[c] |
| CRP>85mg/L | 0.839 | 0.167 | 28.761 | 2.287 (1.726-3.137) | <.001[c] |
| PCT>2.10μg/L | 0.365 | 0.145 | 3.091 | 1.387 (1.238-1.897) | .009[b] |

[a]*P* < .05, indicating that cTnI >0.6μg/L was significantly associated with mortality
[b]*P* < .01, indicating that PCT>2.10μg/L was significantly associated with mortality
[c]*P* < .001, indicating that an age>64 years, APACHE II≥29, NT-proBNP>3000.09, CK>145U/L, CRP>85mg/L were significantly associated with mortality

**Abbreviations:** APACHE II, Acute Physiology and Chronic Health Evaluation II; CK, creatine kinase; CRP, C-reactive protein; cTnI, cardiac troponin I; NT-proBNP, N-terminal pro-brain natriuretic peptide; PCT, procalcitonin.

was the large difference in the sample size of the two groups, which may had led to large errors.

In a subsequent study, the current research team plans to conduct a multicenter, case-control, cohort study and perform enrollment in a matched manner to ensure the comparability of sample size and baseline data to obtain a more reliable conclusion.

## CONCLUSIONS

The risk factors for poor prognosis in septic patients with myocardial injury include an age >64 years, a high APACHE II score, and elevated levels of cTnI, NT-proBNP, CK, CRP, and PCT.

### AUTHORS' DISCLOSURE STATEMENT

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### AUTHOR CONTRIBUTIONS

Guangqing Huang and Wenzi Yang contributed equally to this paper and are regarded as co-first authors.

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