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ORIGINAL RESEARCH

Retrospective Study of Risk Factors for Myocardial Damage in Patients With Critical Coronavirus Disease 2019 in Wuhan

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BACKGROUND: The novel severe acute respiratory syndrome coronavirus 2 threatens human health, and the mortality rate is higher in patients who develop myocardial damage. However, the possible risk factors for myocardial damage in patients with coronavirus disease 2019 (COVID-19) are not fully known.

METHODS AND RESULTS: Critical type patients were selected randomly from 204 confirmed COVID-19 cases occurring in Renmin Hospital of Wuhan University from February 1, 2020 to February 24, 2020. Univariate analyses were used to compare the 2 groups: the myocardial damage group and the non–myocardial damage group. A total of 82 critical patients with COVID-19 were recruited: 34 with myocardial damage and 48 without myocardial damage. A total of 30 patients died in the myocardial damage group, and 20 died in the non–myocardial damage group. In univariate analysis, the proportion of elderly patients (>70 years old, 70.59% versus 37.50%; *P*=0.003) and patients with cardiovascular disease (41.18% versus 12.50%; *P*=0.003) was higher among myocardial damage patients than among non–myocardial damage patients. Multivariate analysis showed that age >70 years old (hazard ratio [HR], 2.44; 95% CI, 1.01–5.40), CRP (C-reactive protein) >100 mg/L (HR, 1.92; 95% CI, 0.94–3.92), lactate dehydrogenase >300 U/L (HR, 2.67; 95% CI, 1.03–6.90), and lactic acid >3 mmol/L (HR, 3.25; 95% CI, 1.57–6.75) were independent risk factors for myocardial damage in patients with COVID-19.

CONCLUSIONS: Old age (>70 years old), CRP >100 mg/L, lactate dehydrogenase >300 U/L, and lactic acid >3 mmol/L are high-risk factors related to myocardial damage in critical patients with COVID-19.

Key Words: COVID-19 ■ critical type ■ myocardial damage ■ risk factors

The outbreak of a novel respiratory infection of coronavirus disease 2019 (COVID-19) was identified in China in late December 2019.[1,2] The common clinical manifestations included fever, nonproductive cough, dyspnea, myalgia, fatigue, normal or decreased leukocyte counts, and radiographic evidence of pneumonia.[3,4] At present, the novel severe acute respiratory syndrome coronavirus 2 has spread throughout the world. This disease was divided into 4 types according to the new coronavirus pneumonia prevention and control program in China (sixth edition): light, common, severe, and critical.[5] Fatalities associated with infections caused by this virus are of great public health concern. A previous study reported that the mortality rate was 62% among critically ill patients with COVID-19 in Wuhan.[6] In another study, Shi and colleagues reported that cardiac injury is a common condition among hospitalized patients with COVID-19, and it is associated with a higher risk of in-hospital mortality.[7] However, the possible risk factors associated with myocardial damage in critical patients with COVID-19 are not fully known. Hence, the aim of this study was to analyze the possible risk factors associated with myocardial damage in critical patients with COVID-19.

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CLINICAL PERSPECTIVE

What Is New?

- This study was first to analyze the possible risk factors of myocardial damage in patients with coronavirus disease 2019.

What Are the Clinical Implications?

- These findings suggest that old age (>70 years old), C-reactive protein >100 mg/L, and increased lactate dehydrogenase and lactic acid >3 mmol/L are high-risk factors that were related to myocardial damage of critical patients with coronavirus disease 2019.
- These results imply that myocardial damage is a common condition, and we should intervene related factors to reduce myocardial injury.

Nonstandard Abbreviations and Acronyms

COVID-19	coronavirus disease 2019
CRP	C-reactive protein

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Population

A retrospective review of medical records from 204 confirmed COVID-19 cases was performed in Renmin Hospital of Wuhan University from February 1 to February 24, 2020. All patients enrolled in this study were confirmed to have COVID-19 by using real-time polymerase chain reaction on samples from the respiratory tract. The diagnosis of COVID-19 was based on the World Health Organization's interim guidelines.[8] A total of 82 critical patients with COVID-19 were recruited according to the diagnostic criteria. Critical status was defined based on the new coronavirus pneumonia prevention and control program in China (sixth edition).[5] Informed consent of the subjects were waived. This study was reviewed and approved by the Medical Ethical Committee of Renmin Hospital of Wuhan University.

Data Collection

The demographic characteristics, clinical records, initial hospitalized laboratory findings, computed tomography findings, and outcome data were extracted from electronic medical records using a standardized data collection form. Two experienced clinicians entered and reviewed the data. The criteria[9] for the confirmed diagnosis of novel severe acute respiratory syndrome coronavirus 2 was that at least 1 amplified gene site was positive for the *NP* (nucleocapsid protein) gene and the *ORF1ab* (open reading frame) gene. Myocardial injury was defined as blood levels of cardiac biomarkers (high sensitive troponin I: Siemens Healthcare Diagnostics Inc) above the 99th percentile upper reference limit and increased B-type natriuretic peptide, regardless of new abnormalities in electrocardiography[7] (normal range of high sensitive troponin I, 0–0.04 ng/mL).

Statistical Analysis

We express descriptive data as the mean±SD or median (interquartile range) for continuous variables. Means of continuous variables were compared using independent-group *t* tests when the data were normally distributed; otherwise, the Mann–Whitney *U* test was used. Categorical variables were expressed as number (percentage) and compared by Pearson χ^2 test or Fisher exact test. Univariate analysis was used to evaluate the demographics and clinical factors associated with myocardial injury among patients with COVID-19. We used Kaplan–Meier survival analysis to estimate the non–myocardial damage cumulative proportion and the stratified log-rank test to compare the difference of non–myocardial damage cumulative proportion between different groups. Time to events (myocardial damage) was defined as the time from illness onset to events. Two groups were created to study the relationship between high-risk factors and myocardial damage associated with COVID-19 using hazard ratios (HRs) generated by the Cox proportional hazards regression model: myocardial damage and non–myocardial damage. A forward selection procedure was then used to construct a final mode. Proportional hazard assumptions were verified systematically for the proposed models. Hypothesis testing was conducted using a 2-sided test, with an α value of 0.05 indicating statistical significance. Multivariate Cox regression coefficients were used to generate the nomogram. All analyses were performed using SPSS (version 20.0) and R V.2.13.0. (http://www.R-project.org).

RESULTS

Baseline Characteristics and Symptoms of Patients With Critical COVID-19

A total of 82 patients with critical COVID-19 were included in this study, and 52 (63.4%) were men. The mean age was 67.9±14.6 years old, ranging from 29 to 95 years old. The baseline characteristics of the 82 confirmed cases are shown in Table 1.

Overall, the proportion of elderly patients (>70 years old, 70.59% versus 37.50%; *P*=0.003) and patients

Table 1. Baseline Characteristics and Symptoms of Study Population

Characteristic	All Cases, N (N/82)	Myocardial Damage Cases, N (N/34)	Non–Myocardial Damage Cases, N (N/48)	*P* Value
Sex				
Male	52 (63.41)	23 (67.65)	29 (60.42)	
Female	30 (36.59)	11 (32.35)	19 (39.58)	0.503
Age, y				
>70	42 (51.22)	24 (70.59)	18 (37.50)	
<70	40 (48.78)	10 (29.41)	30 (62.50)	0.003
Chronic diseases				
Hypertension	38 (46.34)	20 (58.82)	18 (37.50)	0.056
Cardiovascular disease	20 (24.39)	14 (41.18)	6 (12.50)	0.003
Diabetes mellitus	14 (17.07)	7 (20.59)	7 (14.58)	0.476
Cerebrovascular disease	6 (7.32)	4 (11.76)	2 (4.17)	0.266*
COPD	8 (9.76)	5 (14.71)	3 (6.25)	0.266*
Chronic kidney disease	4 (4.88)	2 (5.88)	2 (4.17)	1.000*
Chronic liver disease	4 (4.88)	1 (2.94)	3 (6.25)	0.638*
Malignancy	3 (3.66)	2 (5.88)	1 (2.08)	0.576*
First symptom				
Fever	77 (93.90)	31 (91.18)	46 (95.83)	0.644*
Temperature >39°C	47 (57.32)	20 (58.82)	27 (56.25)	0.816
Cough	48 (58.54)	14 (41.18)	34 (70.83)	0.007
Fatigue	38 (46.34)	16 (47.06)	22 (45.83)	0.913
Anorexia	38 (46.34)	17 (50.00)	21 (43.75)	0.576
Myalgia	8 (9.76)	3 (8.82)	5 (10.42)	1.000*
Dyspnea	47 (57.32)	21 (61.76)	26 (54.17)	0.493
Pharyngalgia	9 (10.98)	5 (14.71)	4 (8.33)	0.478*
Diarrhea	14 (17.07)	5 (14.71)	9 (18.75)	0.632
Vomiting	3 (3.66)	1 (2.94)	2 (4.17)	1.000*
Dizziness	4 (4.88)	2 (5.88)	2 (4.17)	1.000*

COPD indicates chronic obstructive pulmonary disease.
*Pearson χ^2 test with Fisher exact test.

with cardiovascular disease (41.18% versus 12.50%; *P*=0.003) was higher among myocardial damage patients than among non–myocardial damage patients, whereas the proportion of patients experiencing cough (41.18% versus 70.83%; *P*=0.007) was lower in myocardial damage patients than among non–myocardial damage patients. There was no significant difference in the proportion of hypertension, diabetes mellitus, cerebrovascular disease, chronic obstructive pulmonary disease, chronic kidney disease, or chronic liver disease between the myocardial damage group and the non–myocardial damage group. Furthermore, first symptoms such as fever, temperature, and fatigue showed no difference between the 2 groups.

Laboratory Findings of Patients With COVID-19

In Table 2, most of the laboratory findings were significantly different between the myocardial damage group and the non–myocardial damage group, so we converted the data into a classification variable. As shown in Table 3, the following factors were associated with a high risk of myocardial damage associated with COVID-19: CRP (C-reactive protein) >100 mg/L (*P*=0.020), procalcitonin >0.5 ng/mL (*P*=0.002), D-dimer >20 mg/L (*P*=0.002), creatine kinase-MB >5 ng/mL (*P*=0.014), blood urea nitrogen >8 mmol/L (*P*<0.001), creatinine >100 μmol/L (*P*=0.001), lactate dehydrogenase >300 U/L (*P*=0.013), and lactic acid >3 mmol/L (*P*<0.001). However, there were no differences in increased alanine aminotransferase, increased aspartate aminotransferase, albumin <30 g/L, increased creatine kinase, or blood electrolytes such as potassium, sodium, chlorine, and calcium between the myocardial damage group and the non–myocardial damage group.

ECG Outcome

ECG data were available for 48 patients, of whom 41.7% were in the myocardial damage group and 58.3% in

Table 2. Mean or Median of Study Population

Characteristic	All Cases, Mean±SD or Median (IQR)	Myocardial Damage Cases, Mean±SD or Median (IQR)	Non–Myocardial Damage Cases, Mean±SD or Median (IQR)	*P* Value
C-reactive protein, mg/L	73.2 (36.0–167.1)	107.8 (61.1–199.3)	62.5 (27.3–100.0)	0.002
Procalcitonin, ng/mL	0.13 (0.07–0.40)	0.25 (0.10–1.04)	0.10 (0.06–0.20)	0.001
D-dimer, mg/L	3.20 (1.23–9.69)	4.56 (2.55–22.56)	2.08 (0.96–5.24)	0.004
Creatine kinase-MB, ng/mL	2.01 (0.97–4.73)	4.52 (2.24–7.52)	1.20 (0.75–2.13)	<0.001
Alanine aminotransferas, U/L	36 (23–79)	36 (25–80)	35 (23–76)	0.889
Aspartate aminotransferase, U/L	41 (29–76)	62 (29–123)	35 (29–60)	0.031
Albumin, g/L	33.11±3.86	31.64±4.06	34.12±3.41	0.004
Blood urea nitrogen, mmol/L	6.60 (4.50–9.50)	10.30 (7.40–18.90)	5.15 (4.15–7.25)	<0.001
Creatinine, μmol/L	67 (51–89)	87 (59–135)	61 (49–74)	0.002
Creatine kinase, U/L	86 (41–163)	133 (67–258)	66 (37–109)	0.011
Lactate dehydrogenase, U/L	435 (287–641)	580 (416–830)	339 (265–493)	<0.001
Hs-TnI, ng/mL	0.026 (0.006–0.456)	0.675 (0.073–3.161)	0.010 (0.006–0.020)	<0.001
BNP, pg/mL	768 (218–2724)	2834 (1337–7020)	249 (147–678)	<0.001
Lactic acid, mmol/L	2.82±1.34	3.44±1.44	2.26±0.97	<0.001
Potassium, mmol/L	4.02±0.70	4.27±0.86	3.85±0.52	0.017
Sodium, mmol/L	140.38±6.03	141.76±6.07	139.44±5.89	0.089
Chlorine, mmol/L	105.28±5.40	106.92±5.47	104.16±5.10	0.023
Calcium, mmol/L	2.03±0.12	2.03±0.13	2.04±0.12	0.700

BNP indicates B-type natriuretic peptide; Hs-TnI, high sensitive troponin I; IQR, interquartile range; and creatine kinase-MB.

the non–myocardial damage group. Table 4 shows the ECG characteristics. There was a significant difference in ECG (P=0.01) and ST-T abnormalities (P=0.007) between the myocardial damage group and the non–myocardial damage group. The proportion of patients with ventricular arrhythmia was higher in the myocardial damage group than in the non–myocardial damage group (30.0% versus 3.6%; P=0.016). However, the other arrhythmic events, such as sinus tachycardia, atrioventricular block, and atrial arrhythmia showed no difference between the myocardial damage group and the non–myocardial damage group.

Risk Factors Associated With Myocardial Damage Associated With COVID-19

Based on Kaplan–Meier curves, the cumulative probability of non–myocardial damage was lower in patients >70 years old than in patients <70 years old (P=0.002) (Figure 1). Figure 2 shows that the cumulative probability of non–myocardial damage in patients with CRP >100 mg/L was lower than that in patients with CRP <100 mg/L (P=0.008). Figure 3 shows that the cumulative probability of non–myocardial damage in patients with lactate dehydrogenase >300 mmol/L was lower than that in patients with lactate dehydrogenase <300 mmol/L (P=0.002). The cumulative probability of non–myocardial damage was lower in cases with lactic acid >3 mmol/L than that in patients with lactic acid <3 mmol/L (P<0.001) (Figure 4).

The Cox proportional hazards regression model confirmed the independent predictors of myocardial damage with COVID-19 as shown in Table 5. The independent myocardial damage predictors of COVID-19 indicated by the Cox proportional hazards regression model were age >70 years old (HR, 2.44; 95% CI, 1.01–5.40), CRP >100 mg/L (HR, 1.92; 95% CI, 0.94–3.92), lactate dehydrogenase >300 mmol/L (HR, 2.67; 95% CI, 1.03–6.90), and lactic acid >3 mmol/L (HR, 3.25; 95% CI, 1.57–6.75).

The nomogram derived based on the multivariate Cox regression coefficients is shown in Figure 5. To use the nomogram, the first variable is located. A line is then drawn straight upward to the points axis to determine the number of points received for the variable. This process is repeated for the other 3 variables, and the points obtained for each variable are summed. The sum of these numbers is located on the total points axis, and a line is drawn downward to the survival axes to determine the likelihood of 14-day, 21-day, and 35-day non–myocardial damage. Calculation of non–myocardial damage can be automated through computer programming.

DISCUSSION

This present retrospective study identified several risk factors for myocardial damage in critical patients with COVID-19. In particular, older age, increased CRP

Table 3. Laboratory Findings of Study Population

Characteristic	All Cases, N (N/82)	Myocardial Damage Cases, N (N/34)	Non–Myocardial Damage Cases, N (N/48)	*P* Value
Blood electrolyte				
Potassium				
Increased	4 (4.88)	3 (8.82)	1 (2.08)	
Normal	62 (75.61)	27 (79.41)	35 (72.92)	
Decreased	16 (19.51)	4 (11.77)	12 (25.00)	0.173*
Sodium				
Increased	14 (17.07)	7 (20.59)	7 (14.58)	
Normal	58 (70.73)	25 (73.53)	33 (68.75)	
Decreased	10 (12.20)	2 (5.88)	8 (16.67)	0.317*
Chlorine				
Increased	14 (17.07)	9 (26.47)	5 (10.42)	
Normal	61 (74.39)	23 (67.65)	38 (79.16)	
Decreased	7 (8.54)	2 (5.88)	5 (10.42)	0.140*
Calcium				
Increased	0 (0)	0 (0)	0 (0)	
Normal	11 (13.41)	6 (17.65)	5 (10.42)	
Decreased	71 (86.59)	28 (82.35)	43 (89.58)	0.512*
C-reactive protein >100 mg/L	29 (35.37)	17 (50.00)	12 (25.00)	0.020
Procalcitonin >0.5 ng/mL	16 (19.51)	12 (35.29)	4 (8.33)	0.002
D-dimer >20 mg/L	16 (19.51)	12 (35.29)	4 (8.33)	0.002
Creatine kinase-MB >5 ng/mL	18 (21.95)	12 (35.29)	6 (12.50)	0.014
Alanine aminotransferase >50 U/L	32 (39.02)	14 (41.18)	18 (37.50)	0.737
Aspartate aminotransferase >40 U/L	44 (53.66)	20 (58.82)	24 (50.00)	0.430
Albumin <30 g/L	19 (23.17)	11 (32.35)	8 (16.67)	0.097
Blood urea nitrogen >8 mmol/L	32 (39.02)	23 (67.65)	9 (18.75)	<0.001
Creatinine >100 μmol/L	15 (18.29)	12 (35.29)	3 (6.25)	0.001
Creatine kinase >200 U/L	18 (21.95)	11 (32.35)	7 (14.58)	0.055
Lactate dehydrogenase >300 U/L	55 (67.07)	28 (82.35)	27 (56.25)	0.013
Lactic acid >3 mmol/L	25 (30.49)	18 (52.94)	7 (14.58)	<0.001
Hs-TnI >0.04 ng/mL	34 (41.46)	34 (100.00)	0 (0)	<0.001
BNP >900 pg/mL	43 (52.44)	34 (100.00)	9 (18.75)	<0.001

BNP indicates B-type natriuretic peptide; and Hs-TnI, high sensitive troponin I.
*Fisher exact test was used.

levels, lactate dehydrogenase, and lactic acid levels were associated with higher odds of myocardial damage. However, there was no difference in sex or first symptoms between the myocardial damage group and the non–myocardial damage group.

In slightly more than 3 months, novel severe acute respiratory syndrome coronavirus 2 has spread worldwide and caused far greater morbidity and mortality than either severe acute respiratory syndrome or Middle East respiratory syndrome.[10] Analysis showed that both novel severe acute respiratory syndrome coronavirus 2 and the novel severe acute respiratory syndrome coronavirus shared a common ancestor that resembled the bat coronavirus HKU9-1.[11,12] The number of cases increased rapidly, and the mean mortality rate was higher in myocardial damage cases.[4] Myocardial injury has been described in many patients with COVID-19, and mortality has been associated with an increase in troponin levels.[7,13] However, the possible risk factors for myocardial damage associated with COVID-19 are not clear. In the present study, to the best of our knowledge, we first analyzed the possible risk factors of myocardial damage with COVID-19. We found that the proportion of elderly patients and patients with cardiovascular disease was higher among myocardial damage patients than among the non–myocardial damage patients, whereas other comorbidities and clinical symptoms did not show significant differences between the 2 groups. A previous study showed that many elderly

Table 4. Characteristics of ECG Outcome in the Study Population

Characteristic	All Cases (n=48)	Myocardial Damage Cases (n=20)	Non–Myocardial Damage Cases (n=28)	*P* Value
Abnormal ECG	28 (58.33)	16 (80.00)	12 (42.86)	0.010
Abnormal ST-T	18 (37.50)	12 (60.00)	6 (21.43)	0.007
Anterior ST-T changes	4 (8.33)	4 (20.00)	0 (0)	
Inferior ST-T changes	4 (8.33)	3 (15.00)	1 (3.57)	
All lead ST-T changes	10 (20.83)	5 (25.00)	5 (17.86)	
Prolonged QT	6 (12.50)	4 (20.00)	2 (7.14)	0.218*
Sinus tachycardia	6 (12.50)	4 (20.00)	2 (7.14)	0.218*
Sinus bradycardia	1 (2.08)	0 (0)	1 (3.57)	1.000*
Atrioventricular block	4 (8.33)	2 (10.00)	2 (7.14)	1.000[a]*
RBBB	2 (4.17)	2 (10.00)	0 (0)	
LBBB	3 (6.25)	1 (5.00)	2 (7.14)	
First degree A-V block	1 (2.08)	1 (5.00)	0 (0)	
Pathological Q wave	4 (8.33)	2 (10.00)	2 (7.14)	1.000*
Atrial arrhythmia	4 (8.33)	4 (20.00)	0 (0)	0.025*
Atrial premature beat	1 (2.08)	1 (5.00)	0 (0)	
Atrial tachycardia	2 (4.17)	2 (10.00)	0 (0)	
Atrial fibrillation	1 (2.08)	1 (5.00)	0 (0)	
Ventricular arrhythmia	7 (14.58)	6 (30.00)	1 (3.57)	0.016*
VPB	6 (12.50)	5 (25.00)	1 (3.57)	
Ventricular tachycardia	1 (2.08)	1 (5.00)	0 (0)	

One case combined with first-degree A-V block, complete RBBB, left anterior fascicular block, and prolonged QT. LBBB indicates left bundle branch block; RBBB, right bundle branch block; and VPB, ventricular premature beat.

*Pearson χ^2 test with Fisher exact test.

individuals have a compromised immune system, leading to increased susceptibility to infectious diseases and decreased responses to vaccination.[14] The effect of age on the immune system can be demonstrated by the low protective titers among 50% of adults >65 years old receiving an influenza vaccine.[15]

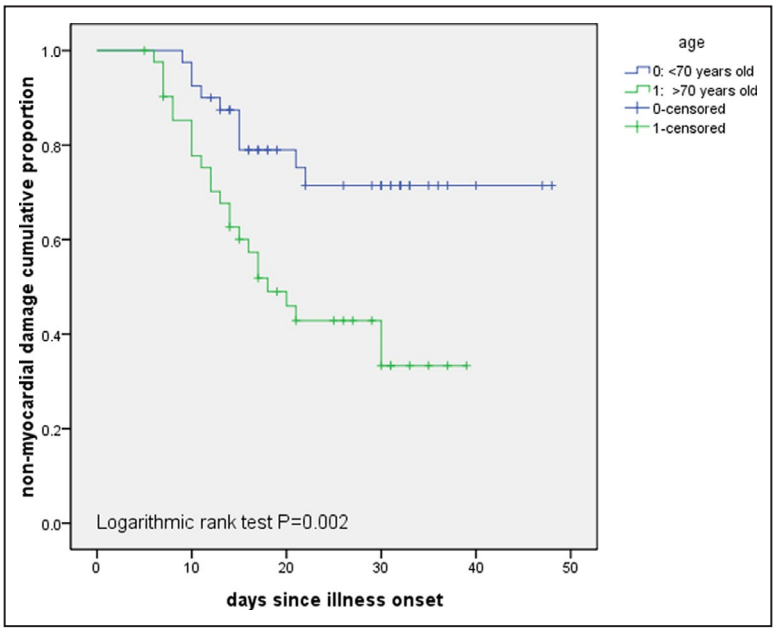

Figure 1. Non–myocardial damage cumulative proportion among patients >70 years old (green line) compared with that among younger patients <70 years old (blue line), duration since illness onset.
Logarithmic rank test *P*=0.002. 0, <70 years old; 1, >70 years old.

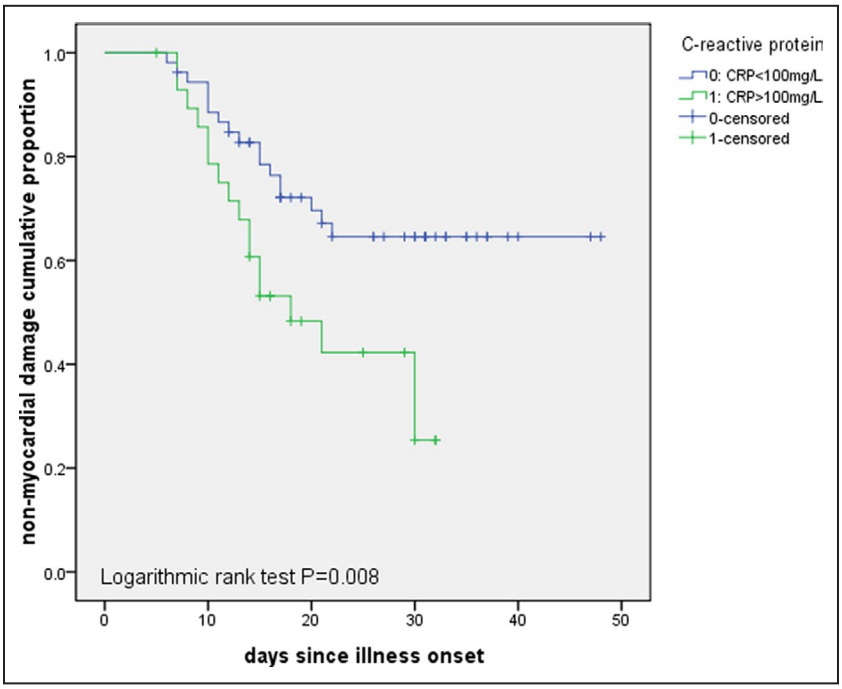

Figure 2. Non–myocardial damage cumulative proportion among patients with CRP (C-reactive protein) >100 mg/L (green line) compared with that among patients with CRP <100 mg/L (blue line), duration since illness onset.
Logarithmic rank test *P*=0.008. 0, CRP <100 mg/L; 1, CRP >100 mg/L.

There are at least 2 mechanisms of injury, including direct myocardial injury by the virus and cytokine/inflammation-mediated damage. Therefore, as one of the independent risk factors, an age-related compromised immune system still plays an important role in the outcome of critical cases.

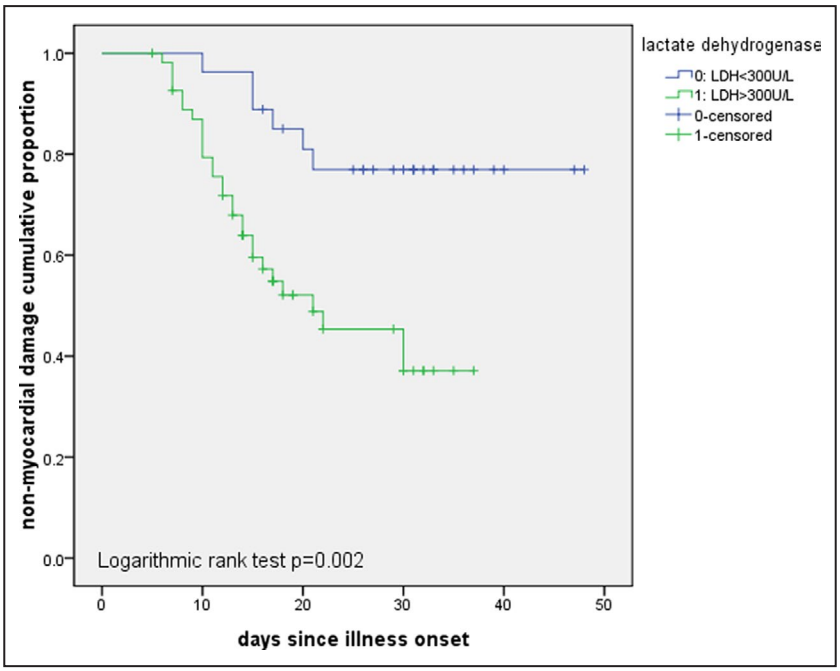

Figure 3. Non–myocardial damage cumulative proportion among patients with increased lactate dehydrogenase (LDH) (green line) compared with that among patients without high LDH (blue line), duration since illness onset.
Logarithmic rank test *P*=0.002. 0, LDH <300 U/L; 1, LDH >300 U/L.

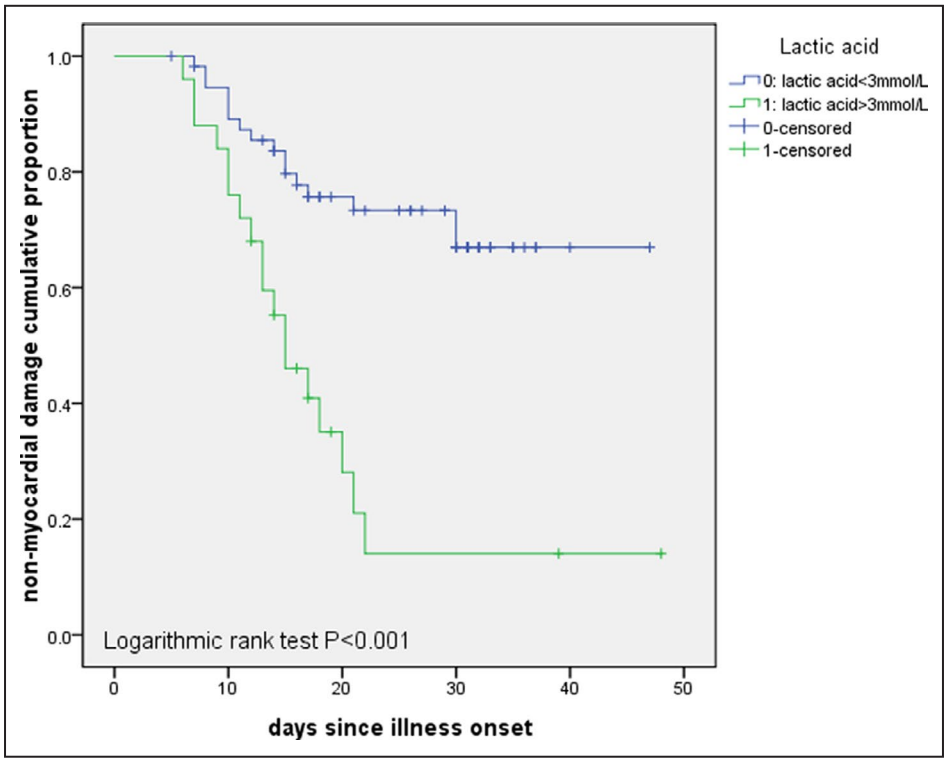

Figure 4. Non–myocardial damage cumulative proportion among patients with lactic acid >3 mmol/L (green line) compared with that among patients with lactic acid <3 mmol/L (blue line), duration since illness onset.
Logarithmic rank test *P*<0.001. 0, lactic acid <3 mmol/L; 1, lactic acid >3 mmol/L.

Table 5. Cox Proportional Hazards Regression Model of Risk Factors for Myocardial Damage With COVID-19

Characteristic	Coefficient	SE (Coefficient)	Wald	*P* Value	HR (95% CI)
Age >70 y	0.891	0.406	4.810	0.028	2.44 (1.01–5.40)
Lactate dehydrogenase >300 U/L	0.981	0.485	4.091	0.043	2.67 (1.03–6.90)
C-reactive protein >100 mg/L	0.650	0.365	3.165	0.075	1.92 (0.94–3.92)
Lactic acid >3 mmol/L	1.179	0.373	10.022	0.002	3.25 (1.57–6.75)

COVID-19 indicates coronavirus disease 2019; and HR, hazard ratio.

As a gauge of inflammation, CRP was identified as a risk factor related to myocardial injury.[16] Severe inflammatory storm events were observed in critical patients with COVID-19, which can promote thrombosis and myocardial infarction.[3,17] On the other hand, viruses or inflammation can directly lead to myocardial injury.[18,19] The increase in lactic acid indicates the existence of anoxia and acidosis in the body. When hypoxia occurs, increased reactive oxygen species beyond the capacity of antioxidants could cause a number of changes in cell components, such as proteins, lipids, and nucleotides, causing myocardial cell damage and even cell death both through apoptosis mechanisms or as a result of necrosis or autophagia.[20] Therefore, lactic acid is the early predictor of myocardial damage that needs to be focused on. In the present study, we found that there was higher CRP, higher procalcitonin, higher D-dimer, increased creatine kinase-MB, and increased creatinine in the myocardial damage patients than that in the non–myocardial damage patients. Furthermore, older age, CRP >100 mg/L, increased lactate dehydrogenase, and lactic acid >3 mmol/L are the independent myocardial damage predictors in patients with COVID-19. Lactate dehydrogenase, creatine kinase, and creatine kinase-MB localized in myocytes are released during the development of myocardial injury.[21] In the present study, we also found that the ventricular arrhythmia incidence and abnormal ST-T were higher in the myocardial damage group than in the

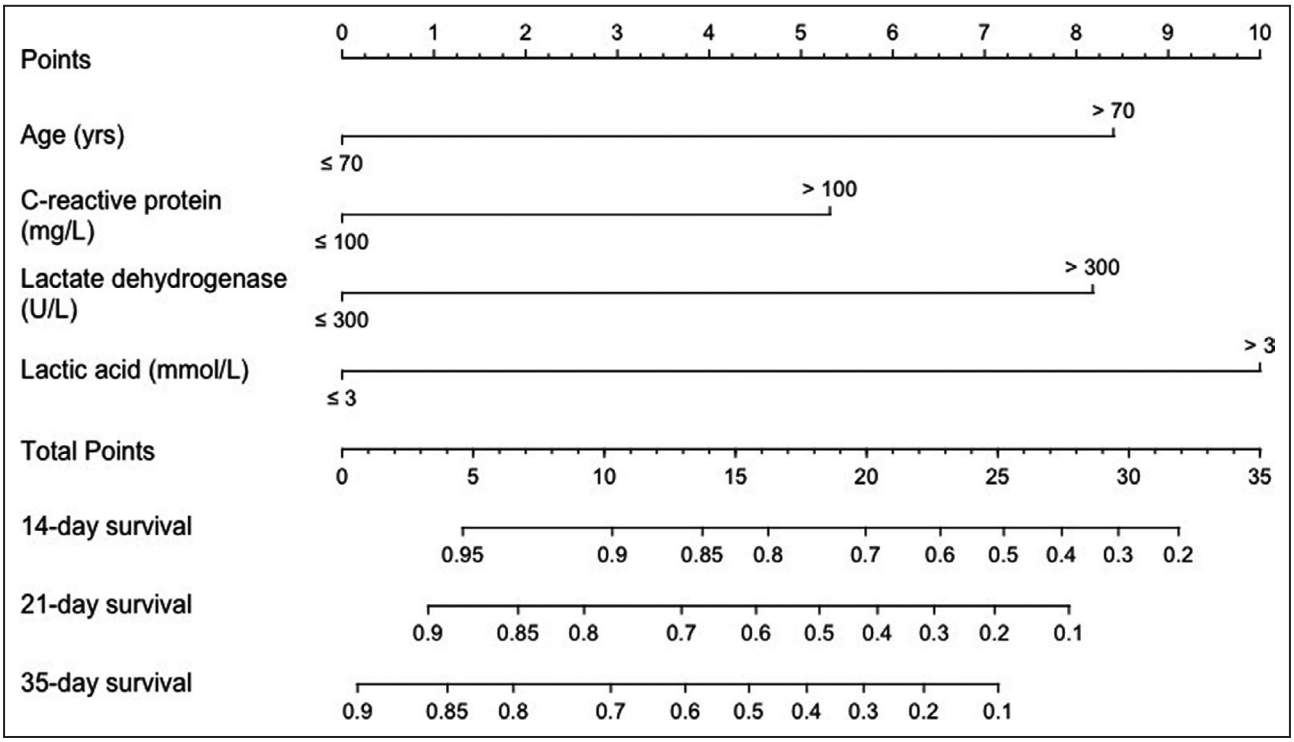

Figure 5. Nomogram for predicting the non–myocardial damage probability of patients with coronavirus disease 2019. Survival is defined as absence of myocardial damage.

non–myocardial damage group. These results imply that ST changes and ventricular arrhythmias may easily occur in patients with myocardial injury. Taken together, these results suggest that myocardial damage is a common condition, and we should address the related factors to reduce myocardial injury.

Limitations

There are several limitations to this study. First, this is a retrospective and observational study, and most of the patients were seriously ill at the time of admission. Few patients had echocardiographic data, and the patient's height and weight data were also missing, so we could not obtain the results of echocardiography or body mass index. In this study, 34 patients had no ECG data, so we only analyzed the results of ECG in 48 patients. Second, because of the retrospective study design and the limited number of patients, data from larger populations and multiple centers are warranted to further confirm the risk of mortality during hospitalization. Third, in this retrospective and observational study, there were significant differences in the type, timing, and dose of medication used in these patients. Furthermore, the aim of the study was to analyze the possible risk factors for myocardial damage in critical patients with COVID-19. Therefore, we did not collect the treatment data. This is a limitation of this study.

In conclusion, old age (>70 years old), CRP >100 mg/L, lactate dehydrogenase >300 U/L, and lactic acid >3 mmol/L are high-risk factors that were related to myocardial damage in critical patients with COVID-19.

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Disclosures

None.

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