

ORIGINAL PAPER
CARDIOVASCULAR MEDICINE

Manifestation of cardiac injury in hospitalised patients with COVID-19

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Abstract

Background: COVID-19 has been sweeping the world since it emerged in late December 2019. However, little is known about cardiac injury in hospitalised COVID-19 patients. This study is to investigate the incidence and characteristics of myocardial injury in COVID-19 patients admitted in hospital.

Methods: Fifty-four COVID-19 patients were enrolled in one ward in Tongji Hospital, Wuhan, China, and 5 were excluded caused by missing cardiac troponin I levels. Forty-nine participants were included in the final analysis. The clinical manifestations of hospitalised patients were analysed. Patients were divided into two groups, cardiac injury group and non-cardiac injury group, based on whether cardiac troponin I was elevated. Epidemic characteristics and laboratory test results were analysed in these two group.

Results: The average age of patients in the cardiac injury group was older (68.0 years old) than that in the non-cardiac injury group (61.5 years old). The percentages of patients with diabetes and critically severe pneumonia in the cardiac injury group were 38.5% and 38.5% respectively. Lymphocytes were decreased in 53.1% of all enrolled patients, but this decrease was more prominent (76.9%) in the cardiac injury group than the non-cardiac injury group (44.4%). Patients in the cardiac injury group also had lower platelet counts.

Conclusions: COVID-19 can cause cardiac injury in many patients. It is more common in older patients and patients with diabetes and is associated with a significant decrease in lymphocytes.

What' known

A comprehensive understanding of COVID-19, the emergent hygeian issue, is essential for its treatment. COVID-19 can damage vital organs and systems, like cardiovascular system, in addition to the respiratory system.

What' new

Understanding the myocardial damage of COVID-19 is very important for clinicians to control this disease.

1 | BACKGROUND

COVID-19 has been spreading wildly since its outbreak in late December 2019 in Wuhan, China. With more and more studies reported, it has been found that COVID-19 can damage multiple systems and organs in addition to the respiratory system.¹⁻³ In hospital patients, Cardiac injury is a common condition and it is associated with higher risk of in-hospital mortality.⁴ One study found that cardiac injury occurred in 12% of COVID-19 patients,² and another has indicated that COVID-19 patients with cardiovascular disease are at greater risk for cardiac injury.⁵ The population with cardiovascular disease is a precisely defined, large base group.⁶ COVID-19 could also cause fulminant myocarditis.⁷ In our clinical observation, it was also found that COVID-19 caused serious cardiac injury resulting in death. Therefore, it is important to identify cardiac damage caused by COVID-19. At present, few studies have been reported cardiac injury by COVID-19, and the clinical manifestations and mechanisms of COVID-19 on cardiac injury are not completely clear, where a huge gap in our understanding exists. This study aimed to investigate the manifestation of cardiac injury in COVID-19 patients. The incidence and clinical characteristics of cardiac injury are described. At the moment, there is no effective treatment for COVID-19, only supportive treatment. Therefore, this study would help clinicians understand more about the disease and intervene in the disease process in advance.

2 | METHODS

2.1 | Patients

This was a single-centre retrospective study. In this study, COVID-19 patients were enrolled after being admitted to East Ward 11 of Tongji Hospital from February 10 to February 23, 2020. All COVID-19 patients in the ward were enrolled. Oral consent was obtained from the patients, and the patients' written consents were waived. Tongji Hospital is the national designated hospital for admission of COVID-19 patients. All patients were diagnosed according to WHO guidelines.⁸ The study has been approved by the ethics committees of Tongji Hospital and Beijing Hospital.

2.2 | Data collection

All patients were under the charge of Professor Wang Fang's team. We recorded the patients' clinical features: age, sex, disease history, onset duration, exposure history, severity (mild, severe and critically severe), diabetes, hyperlipidaemia and cardiovascular disease. We also recorded the patients' symptoms and results of physical examination: fever, cough, sore throat, fatigue, myalgia, headache, diarrhoea, chest congestion, shortness of breath, palpitation, blood pressure, heart rate and respiratory rate. The following laboratory tests were also recorded: routine blood test, liver and kidney

function, coagulation function, D-dimer, C-reactive protein, erythrocyte sedimentation rate (ESR) and procalcitonin. In particular, we recorded the test findings closely relating to cardiac injury: cardiac troponin I (cTn I), N terminal brain natriuretic peptide (NT-BNP) and CT scans. Pericardial effusion and heart shadow enlargement were observed by CT scan with the patient in the supine position. Onset duration was defined as the date from symptom discovery to hospitalisation. Cardiac injury was defined as serum level of troponin I above the 99th percentile of the reference limit. Severity was defined as: mild: non-pneumonia or mild pneumonia; severe: respiratory frequency ≥ 30 /minute, blood oxygen saturation $\leq 93\%$, PaO₂/FiO₂ ratio [the ratio between arterial oxygen partial pressure and fractional inspired oxygen] < 300 and/or lung infiltrates $> 50\%$ within 24 to 48 hours; or critically severe: respiratory failure, septic shock and/or multiple organ dysfunction.⁹

Throat swab specimens were obtained from all patients, and COVID-19 was tested for by RT-PCR according to the instructions.² The presence of the COVID-19 virus was confirmed in four different institutions, the Chinese Center for Disease Control and Prevention, Chinese Academy of Medical Sciences, Chinese Academy of Military Medical Sciences and Wuhan Institute of Virology. RT-PCR detection reagents were provided by these four institutions.

2.3 | Statistical analysis

Continuous variables are presented as mean (SD) if normally distributed and median (IQR) if not normally distributed. Categorical variables are presented as number (%). A χ^2 test or Fisher's exact test was applied to compare parameters in the cardiac injury and non-cardiac injury groups. The corresponding statistical method was used to deal with the few missing values. Boxplots were drawn to indicate plasma concentration of cardiac injury markers. A *P* value less than .05 was considered statistically significant. All statistical analyses were done using the software SPSS (version 26.0).

3 | RESULTS

Fifty-four patients were enrolled, and 5 were excluded because of missing cardiac troponin I levels. The study ended up with 49 patients in the final analysis. The average age of patients was 63 years old. Patients in the cardiac injury group were significantly older than patients in the non-cardiac injury group. The mean age of the cardiac injury group was 68 years old, and that of the non-cardiac injury group was 61.5 years old. There was no difference between the groups in onset duration, gender or history of exposure. In terms of severity of the disease, the proportion of patients with critically severe pneumonia was significantly higher in the cardiac injury group than that in the non-cardiac injury group. Critically severe COVID-19 pneumonia occurred in 5 of 13 patients in the cardiac injury group (38.5%) and in 3 of 36 patients in the non-cardiac injury group (11.1%). There was no difference between the two groups in

Characteristic	All patients (n = 49)	Cardiac injury (n = 13)	No cardiac injury (n = 36)	P value
Age, years	63 (50-68)	68 (59.5-76)	61.5 (48-66)	.007
Onset duration, days	10 (5-14.5)	7 (4-14.5)	10 (7-14.5)	.375
Men	31 (63.3%)	9 (69.2%)	22 (61.1%)	.743
Women	18 (36.7%)	4 (30.8%)	14 (38.9%)	
Exposure history	18 (36.7%)	3 (23.1%)	15 (41.7%)	.322
COVID-19 severity				
Mild	20 (40.82%)	5 (38.5%)	15 (41.7%)	-
Severe	21 (42.9%)	3 (23.1%)	18 (50%)	.454
Critical severe	8 (16.3%)	5 (38.5%)	3 (8.3%)	.023
Other disease	14 (28.6%)	6 (46.2%)	8 (22.2%)	.152
Diabetes	9 (18.4%)	5 (38.5%)	4 (11.1%)	.043
Hyperlipidaemia	2 (4.1%)	1 (7.7%)	1 (2.8%)	.464
Hypertension	22 (44.9%)	8 (61.5%)	14 (38.9%)	.279
Cardiovascular disease	4 (8.2%)	1 (7.7%)	3 (8.3%)	1.000

TABLE 1 Baseline demographic characteristics of participants

Characteristic	All patients (n = 49)	Cardiac injury (n = 13)	No cardiac injury (n = 36)	P-value
Fever	38 (77.55%)	9 (69.23%)	29 (80.56%)	.451
Cough	34 (69.39%)	7 (53.85%)	27 (75%)	.178
Sore throat	5 (10.2%)	2 (15.38%)	3 (8.33%)	.598
Fatigue	29 (59.18%)	10 (76.92%)	19 (52.78%)	.191
Myalgia	13 (26.53%)	2 (15.38%)	11 (30.56%)	.467
Headache	6 (12.24%)	3 (23.08%)	3 (8.33%)	.321
Diarrhoea	11 (22.45%)	1 (7.69%)	10 (27.78%)	.246
Chest congestion	16 (32.65%)	3 (23.08%)	13 (36.11%)	.502
Shortness of breath	16 (32.65%)	3 (23.08%)	13 (36.11%)	.502
Palpitation	2 (4.08%)	0 (0%)	2 (5.56%)	1.000
Heart rate	94.0 (81.0-102.0)	92.0 (82.0-112.5)	94.5 (80.3-100.8)	.482
Respiratory rate >24 breaths per minutes	5 (10.20%)	3 (23.07%)	2 (5.56%)	.109

TABLE 2 Baseline demographic characteristics of signs and symptoms

occurrence of chronic diseases such as hyperlipidaemia, hypertension or cardiovascular disease. In the cardiac injury group, the rate of diabetes was higher than in the other group. Of the 13 patients with cardiac injury, 5 were diabetic (38.5%) (Table 1).

The symptom of fever appeared in 77.55% of enrolled patients, and cough and fatigue appeared in 69.39% and 59.18% of enrolled patients respectively. Chest tightness appeared in 32.65% and palpitation appeared in 32.65% of the study population, with no difference between the two groups for either of these symptoms. There was no significant difference in the percentage of patients with any other symptoms between the two groups (Table 2).

Table 3 (at the end of the text) shows the laboratory test results. The rate of abnormal leucocyte levels was 38.8% in the whole

population, and lymphocytic decline happened in 53.1% of patients. Moreover, reduction in lymphocyte levels was found significantly more frequently in the cardiac injury group (76.9%) than the non-cardiac injury group (44.4%). Patients with cardiac injury also had significantly fewer platelets than patients without cardiac injury. The coagulation function of fibrinogen increased in 71.4% of all patients with no difference between the groups. Thromboplastin time decreased in 98% of all patients. D-dimer increased in 79.6% of the population, with no difference between the two groups. C-reactive protein and blood sedimentation rate increased in patients with and without cardiac injury in 95.9% and 77.6% of cases respectively. Serum electrolytes and blood glucose showed no significant differences between the two groups.

TABLE 3 Baseline demographic characteristics of laboratory tests

Detecting item	All patients (n = 49)	Cardiac injury (n = 13)	No cardiac injury (n = 36)	P value
Leucocytes (10 ⁹ /L) (3.5-9.5)	5.7 (3.5-7.8)	5.7 (3.8-7.8)	5.7 (3.5-7.9)	.892
Increased	7 (14.3%)	2 (15.4%)	5 (13.9%)	
Decreased	12 (24.5%)	2 (23.1%)	9 (25.0%)	
Lymphocyte (10 ⁹ /L) (1.1-3.2)	1.1 (0.7-1.4)	0.9 (0.6-1.2)	1.16 (0.8-1.5)	.045
Decreased	26 (53.1%)	10 (76.9%)	16 (44.4%)	
Platelet (10 ⁹ /L) (125-350)	231 (163-357.5)	161.0 (96.5-237.9)	294.5 (197.0-394.3)	.004
Increased	14 (28.6%)	1 (7.7%)	13 (36.1%)	
Decreased	6 (12.2%)	4 (30.8%)	2 (5.6%)	
Prothrombin time (s) (11.5-14.5)	14 (13.4-14.6)	14.3 (13.5-18.8)	13.9 (13.2-14.4)	.054
Increased	12 (24.5%)	5 (38.5%)	7 (19.4%)	
Fibrinogen (g/L) (2.0-4.0)	5.0 (3.7-6.0)	4.0 (2.6-5.7)	5.3 (4.2-6.1)	.098
Increased	35 (71.4%)	6 (46.2%)	29 (80.6%)	
Decreased	4 (8.2%)	3 (23.1%)	1 (2.8%)	
Thromboplastin time (seconds) (75-125)	41.4 (36.8-46.2)	44.5 (36.2-48.0)	40.9 (36.7-45.8)	.667
Increased	1 (2.0%)	1 (7.7%)	0	
Decreased	48 (98.0%)	12 (92.3%)	36 (100%)	
D-dimer (mg/L) (<0.5)	1.1 (0.6-3.3)	1.03 (0.6-5.0)	1.31 (0.6-3.4)	.91
Increased	39 (79.6%)	11 (84.6%)	28 (77.8%)	
C-reactive protein (mg/L) (<1)	33.2 (7.3-67.65)	30.9 (5.65-95.5)	33.6 (7.25-61.63)	.571
Increased	47 (95.9%)	12 (92.3%)	35 (97.2%)	
ESR (mm/hour) (0-20)	57.8 (21-72)	32 (16-64.9)	60 (25.75-80)	.108
Increased	38 (77.6%)	8 (61.5%)	30 (83.3%)	
Procalcitonin, (ng/mL) (0.02-0.05)	0.09 (0.03-0.24)	0.19 (0.04-0.5)	0.07 (0.03-0.21)	.146
Increased	32 (65.3%)	10 (76.9%)	22 (61.1%)	
AST (U/L) (<33)	30 (20.5-43.5)	37.13 (21.5-74)	29 (20.3-40.0)	.153
Increased	21 (42.9%)	8 (61.5%)	13 (36.1%)	
ALT (U/L) (<33)	29 (15.5-50)	37 (13.5-57.5)	26 (17.5-48.8)	.76
Increased	24 (49.0%)	8 (61.5%)	16 (44.4%)	
Lactate dehydrogenase (U/L) (135-214)	251 (191-312)	306 (182-434.5)	224 (191-293.1)	.253
Increased	30 (61.2%)	9 (69.2%)	21 (58.3%)	-
Decreased	1 (2.0%)	0	1 (2.8%)	.196
Serum creatinine (μmol/L) (45-84)	70.0 (61.5-83.0)	74.42 (64-111)	70 (61.3-78.6)	.12
Increased	11 (22.4%)	5 (38.5%)	6 (16.7%)	
Decreased	4 (8.2%)	2 (15.4%)	2 (5.6%)	
Serum potassium (mmol/L) (3.5-5.1)	4.3 (4.0-4.5)	4.12 (3.8-4.3)	4.32 (4.0-4.5)	.167
Increased	5 (10.2%)	2 (15.4%)	3 (8.3%)	
Decreased	2 (4.1%)	0	2 (5.6%)	
Serum sodium (mmol/L) (136-145)	140.3 (138.2-142.0)	140.3 (138.8-146.5)	140.2 (137.5-141.8)	.377
Increased	5 (10.2%)	4 (30.8%)	1 (2.8%)	
Decreased	8 (16.3%)	2 (15.4%)	6 (16.7%)	
Serum calcium (mmol/L) (2.2-2.5)	2.1 (2.1-2.2)	2.1 (2.1-2.2)	2.14 (2.1-2.2)	.964
Decreased	35 (71.4%)	9 (69.2%)	26 (72.2%)	
Fasting glucose (mmol/L) (4.11-6.05)	6.3 (5.3-7.4)	7.1 (5.9-10.5)	6.15 (5.3-7.1)	.054
Increased	29 (59.2%)	10 (76.9%)	19 (52.8%)	
TC (mmol/L) (<5.18)	3.4 (3.0-4.0)	3.3 (2.7-3.8)	3.44 (3.1-4.4)	.164
Increased	4 (8.2%)	0	4 (11.1%)	

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ESR, erythrocyte sedimentation rate; TC, total cholesterol.

Table 4 shows laboratory tests relating to cardiac injury. Of 49 patients, 12 had elevated cardiac troponin I (24.5%). The proportion of patients with increased creatine kinase-MB (CK-MB) was 8%, the percentage with increased CK was 19.2%, the proportion with elevated myoglobin was 11.1% and the rate of elevated NT-BNP was 46.94%. The CT results indicated that 8 of the 49 patients (16.32%) had an enlarged heart shadow. Pericardial effusion was present in 1 patient (Figure 1). The histogram in Figure 2 shows the proportion

of indicators associated with cardiac injury. The box plot in Figure 3 shows the range of parameters associated with cardiac injury in both groups.

4 | DISCUSSION

It is known that many viruses can cause cardiac injury, including adenoviruses, enteroviruses, herpesviruses and influenza A, among others.¹⁰ In our clinical practice, we found that COVID-19 could also cause cardiovascular damage. In early studies of the outbreak, COVID-19 was reported to cause cardiac injury in 12% of patients,² arrhythmia occurred in 16.7% of patients and 7.2% had cardiac injury.¹ In this study, 24.5% of enrolled patients had cardiac injury, a much higher rate than previous studies. Patients with cardiovascular disease might be at greater risk of severe infection than patients without. Moreover, these patients were associated with worse prognosis.⁵ In Yang et al's study, 10% of critically severe COVID-19 patients had cardiovascular disease and died within 28 days.¹¹ In a study of 76 deaths by COVID-19, the proportions of hypertension, coronary heart disease and heart failure were 59.0%, 14.5% and 10.5% respectively.¹² The 76 patients who died of COVID-19 were in a specific group, and this study was a relatively large study in the early stage of the pandemic. In the final phase of the disease, cardiac damage is easy to spot. In a study of 138 confirmed COVID-19 patients, 40% of inpatients had cardiovascular disease.¹ The incidence of cardiac injury varied from study to study. There are several possible reasons. First, there were differences between the studied groups in different studies. Second, the proportion of severe pneumonia varied from study to study. Third, there were different test methods to detect cardiac injury. In our study, the cardiac injury group had a higher occurrence of diabetes, suggesting that patients with diabetes are more vulnerable to damage to heart tissue. Li et al's observations also revealed a greater risk and poorer prognosis for patients with metabolic disease.⁵ Diabetes patients are more prone to infection of coronavirus because of their low immunity,¹³ and diabetes is an important risk factor for poor outcomes.^{14,15} For such patients, cardiac injury might be a promoting factor in disease severity. In our

TABLE 4 Laboratory findings of patients

Detecting item	All patients
CK-MB (n = 25) (ng/mL)	0.60 (0.30-0.85)
<3.4	23/25 (92%)
≥3.4	2/25 (8%)
CK (n = 26) (ng/mL)	62.5 (33.0-150.8)
<190	21/26 (80.8%)
≥190	5/26 (19.2%)
Myoglobin (n = 27) (ng/mL)	38.8 (32.9-53.5)
<106	24/27 (88.9%)
≥106	3/27 (11.1%)
cTnI (n = 49) (pg/mL)	3.8 (1.9-13.9)
<15.6	37/49 (75.5%)
≥15.6	12/49 (24.5%)
NT-BNP (n = 49) (pg/mL)	176.0 (57.0-459.0)
<247	26/49 (53.06%)
≥247	23/49 (46.94%)
Heart shadow enlargement (n = 49)	
Heart shadow enlargement	8/49 (16.32%)
Normal	41/49 (83.68%)
Pericardial effusion (n = 49)	
Hydropericardium	1/49 (2.04%)
Normal	48/49 (97.96%)

Abbreviations: CK, creatine kinase; CK-MB, creatine kinase isoenzyme-MB; cTnI, cardiac troponin I; NT-BNP, N-terminal brain natriuretic peptide.



FIGURE 1 Chest CT images from a male COVID-19 patient showing high-density, bright bronchograms in both lungs (left panel). Enlargement of the heart shadow and pericardial effusion are also seen (right panel)

FIGURE 2 Histogram of percentage statistics of different detection indicators. Blue is the percentage of the population whose detection indicators are within the reference range, while red is the percentage of the population whose detection indicators are outside the reference range. CK, creatine kinase; CK-MB, creatine kinase isoenzyme-MB; cTnI, cardiac troponin I; NT-BNP, N-terminal brain natriuretic peptide

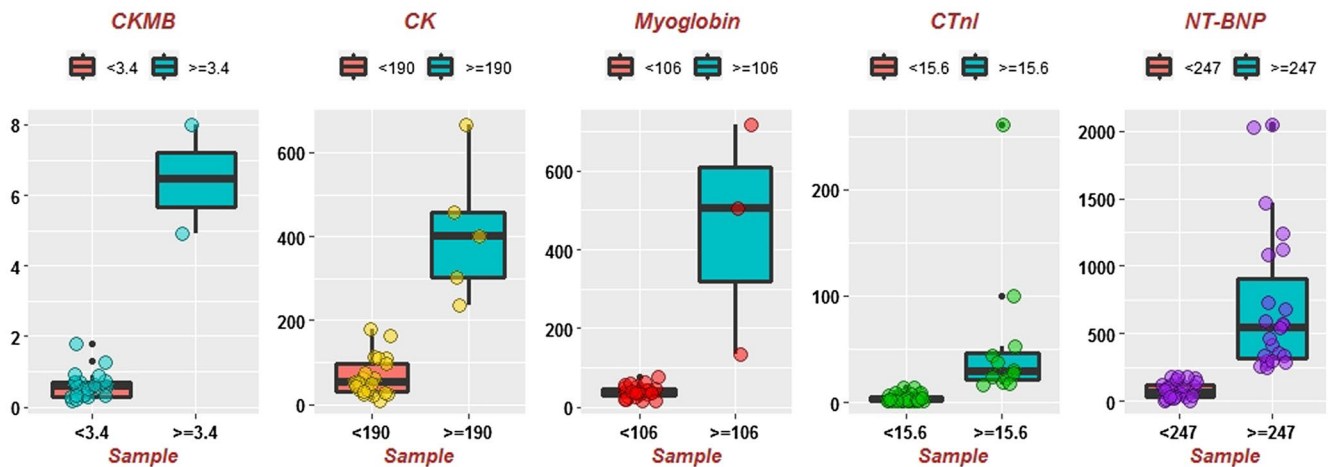
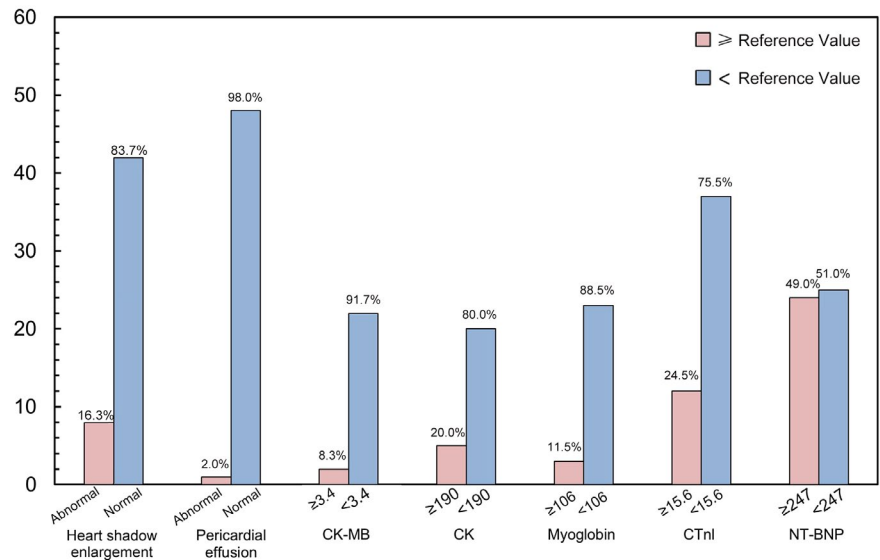


FIGURE 3 Distribution comparison of different parameters represented by boxplot. In the distribution diagram of each detection indicator, the box on the left represents the population distribution within the reference range, while the box on the right represents the population distribution outside the reference range. CK, creatine kinase; CK-MB, creatine kinase isoenzyme-MB; cTnI, cardiac troponin I; NT-BNP, N-terminal brain natriuretic peptide

study, elders were at higher risk of cardiac injury, which is consistent with previous studies.^{2,16} Wu et al showed that fatal cases were primarily in elderly patients,⁹ and cardiac injury might contribute to the severity of COVID-19 pneumonia in old patients.

The exact pathophysiological mechanism of cardiac injury caused by COVID-19 has not been fully understood. Inflammatory storms may be one of the main causes of cardiac injury. A previous study found decreased CD4 and CD8 T-lymphocyte cells in peripheral blood with excessive activation.¹⁷ Our study also revealed that most patients had decreased lymphocyte levels, and lymphocyte levels were reduced more significantly in patients with cardiac injury. Lymphocyte depletion has a poor prognosis,¹⁸ so patients with myocardial injury might be at a higher risk of severe infection. Previous pathological results found a large number of inflammatory

cells in the body tissues of patients who died of COVID-19, and few inflammatory cells were seen in the myocardium.¹⁷ Biopsy indicates that the pathological features of COVID-19 greatly resemble those of SARS and Middle East respiratory syndrome (MERS) coronavirus infections. In genetic terms, the genome of this novel coronavirus had 82% nucleotide identity with that of human SARS coronavirus.¹⁹ SARS and MERS may cause myocarditis, acute myocardial infarction and acute heart failure, resulting from systemic inflammatory immune response.^{20,21} COVID-19 also causes oedema in the lung tissue, leading to malfunction of oxygen and carbon dioxide exchange, resulting in hypoxemia.¹⁷ Hypoxemia could stimulate the increase in excessive free oxygen radicals, which promote the release of inflammatory cytokines. On the other hand, lung infection can also increase the right ventricular load, aggravating cardiac dysfunction.

Meanwhile, inflammatory cell infiltration can aggravate myocyte apoptosis, leading to arrhythmia. The release of inflammatory cytokines can cause severe myocardium damage, which causes a decline or stop in systolic function. In the end, a patient presenting with hypotension or hemodynamic instability may end in a state of multi-organ failure. Hu reported that fulminant myocarditis leading to acute heart enlargement happened even in young patient.⁷ In critically severe cases, inflammatory destruction of the myocardium would lead to arrhythmia or shock, and eventually to death.

There is currently no specific antiviral treatment method for the cardiac injury caused by the virus. The strategy remains supportive treatment.^{10,22} For patients whose conditions may or have become severe, active clinical life support should be given, including mechanical ventilation and circulatory support of extracorporeal membrane oxygenation (ECMO). In patients with cardiac injury, it is necessary to prevent potential arrhythmias and heart failure. COVID-19 is a worldwide pandemic, and treatment has become a serious issue for all countries. Physicians should be concerned about cardiac injury by COVID-19, considering that in all influenza epidemics except for the 1918 flu, cardiovascular events outnumbered all other causes of death.²³ Early identification and timely treatment of cardiac injury in COVID-19 patients is critical to control the disease.

In conclusion, COVID-19 may cause cardiac injury in many patients, with elders and patients with diabetes being more vulnerable to such complications. The reduction in lymphocytes was more significant in patients with cardiac injury. Physicians should be vigilant to the risk of cardiac injury during treatment.

5 | LIMITATIONS

The study population was limited to those admitted to a particular ward. Patients transferred directly to the intensive care unit were not included in the study, and some mild cases were not observed because they were not admitted to the hospital, leading to missing data. A larger sample size and a larger scope of study can provide more intact data on cardiac injury caused by COVID-19.

6 | PERSPECTIVE

In our daily clinical practice, we found that patients with cardiac injury are also prone to arrhythmia and heart failure. Based on our experience, we used a large dose of vitamin C in addition to anti-arrhythmia drugs and anti-heart failure drugs. The patients' symptoms improved. Yet the main goal remains the comprehensive treatment of COVID-19 itself.

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ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Oral consent was obtained from the patients, and the patients' written consents were waived. The study has been approved by the ethics committees of Tongji Hospital and Beijing Hospital.

DISCLOSURES

The authors declare that they have no competing interests.

AUTHOR CONTRIBUTIONS

YZ collated the patients' data. ZP and YG contributed to the writing. CY contributed to the data analysis. YS, XG and FW were in charge of the patients. FW conceived and supervised the study. HZ was a major contributor in writing the manuscript. All authors read and approved the final manuscript.

DATA AVAILABILITY STATEMENT

Data are available from the corresponding author upon reasonable request.

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