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Successful Treatment of a COVID-19 Patient with Fulminant Myocarditis: A Case Report and Review of Literature

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Abstract

Lung damage is the primary clinical concern when treating Coronavirus Disease 2019 (COVID-19) patients. However, not enough attention has been given to potential myocardial damage associated with COVID-19 infection. Here, we report a 65-year-old woman who diagnosed with COVID-19 and myocarditis based on 2019-nCoV nucleic acid test, electrocardiogram (ECG), and myocardial markers. The patient with fulminant myocarditis who presented with the following: (1) obvious symptoms of pulmonary infection without any cardiac-specific symptoms; (2) alleviation of pulmonary symptoms with worsening myocardial damage after treatment; and (3) sudden cardiac arrest caused by malignant arrhythmia. The patient was administered oral Ambroxol tablets, Arbidol Hydrochloride capsules, Lianhuaqingwen capsules (Traditional chinesemidicine), and Ibuprofen capsules, as well as intravenous Moxifloxacin, 20% human serum albumin, and Imipenem sildastatin sodium. On day 8 after hospitalization, the patient suddenly went into a cardiac arrest.After cardiopulmonary resuscitationand electric defibrillation, the patient's vital sign recovered and was immediately transferred to ICU for further treatment. The patient received high-dose methylprednisolone (500mg/d), immunoglobulin therapy (20g/d), ventilator-assisted ventilation, and extracorporeal membrane pulmonary oxygenation (ECMO).One month later, the patient was in stable condition and discharged home. This case study and the literature indicate that symptoms of myocardial injury caused by COVID-19 may be atypical and may develop prior to the onset of pulmonary symptoms. Therefore, active prevention and early adoption of various treatment measures, even in the absence of myocardial injury, may benefitCOVID-19 patients.

Keywords

Fulminant myocarditis, Corona Virus Disease 2019, SARS-CoV-2, COVID-19, Cardiac injury, infection

1. Introduction

The Coronavirus Disease 2019 (COVID-19) is an acute pandemic disease caused by novel coronavirus (2019-nCoV) infection. The incubation period of 2019-nCoVis 1-14 days, with the highest risk at 3-7 days, and the main symptoms are fever, fatigue, and dry cough. Other reported symptoms include nasal congestion, runny nose,

sore throat, myalgia, and diarrhea [1]. The main pathological features of COVID-19 are pulmonary exudative inflammation and alveolar damage [2]. Early intheCOVID-19 outbreak, clinicians were primarily concerned about the lung damage caused by the disease, but little attention has been given to the potential for myocardial damage. As the pandemic developed, subsequent studies reported that COVID-19 patients with myocardial injury had a worse prognosis [3]. Here, we report a COVID-19 patient with fulminant myocarditis, who was admitted to Leishenshan Hospital in Wuhan City, China. The patient's symptoms of pulmonary infection were obvious without any cardiac-specific symptoms. The pulmonary symptoms were alleviated after treatment, but myocardial damage related indicators continued to deteriorate. As a result of the cardiac injury, the patient experienced sudden cardiac arrest caused by malignant arrhythmia. After active treatment, the patient was successfully discharged and returned home. This case report provides insight for treating COVID-19 patients who are at risk of myocardial disease and for providing follow-up care.

2. Case Presentation

Chief complaints: A 65-year-old woman had a fever and presented with cough. **History of present illness:** One month before admission, the patient had a fever without obvious inducement. The patient's body temperature was 38.8°C and she presented with dyspnea, cough, and fatigue. **History of past illness:** No previous disease to report in anamnesis. **Personal and family history:** The patient had no significant past medical history. **Physical examination upon admission:** Due to the limitation of protective clothing, the cardiopulmonary examination could not be carried out. **Laboratory examinations:** The patient's 2019-nCoV nucleic acid test was positive. MB isoenzyme of creatine kinase (CK-MB) = 86.59 ng/ml (Normal reference: 0-4.97 ng/ml); troponin I (TNI) = 0.106 ng/ml (Normal reference: 0-0.04 ng/ml); type B natriuretic peptide (BNP) = 113.13 pg/ml (Normal reference: 0-100pg/ml); and additional markers are listed in Table 1. There are no bacteria in the blood cultures.

Table 1. Laboratory test results

Indicator	2nd day	4th day	6th day	Normal reference
Leucocyte count (*10^9/L)	13.26	16.64	16.85	3.5-9.5
Neutrophile granulocytecount (*10^9/L)	11.09	14.44	14.83	1.8-6.3
Neutrophile granulocyte percentage (%)	83.6	86.7	88.0	40-75
Leukomonocytecount (*10^9/L)	1.19	1.21	1.10	1.1-3.2
Leukomonocyte percentage (%)	9.0	7.3	6.5	20-50
MB isoenzyme of creatine kinase (ng/ml)	86.59	100.10	127.24	0-4.97
Myohemoglobin (ng/ml)	2585.37	1017.88	1360.28	0-65
Troponin I (ng/ml)	0.106	0.055	0.073	0-0.04
Type B natriuretic peptide (pg/ml)	113.13	265.40	302	0-100
D-dimer (mg/L)	1.31	1.20	1.11	0-0.55
Alanine aminotransferase (IU/L)	196	193	188	7-45
Aspertate aminotransferase (IU/L)	239	236	189	13-35
Total protein (g/L)	63.2	62.8	63.1	65-85
Albumin (g/L)	27.8	27.2	27.6	40-55
Globulin (g/L)	35.4	35.6	35.5	20-30
Urea nitrogen (mmol/L)	3.5	4.2	4.7	3.1-8.8
Creatinine (umol/L)	45.7	47.1	41.4	49-90
Uric acid (umol/L)	216	208	204	156-357

Imaging examinations: Chest computed tomography (CT) examination (Figure 1) showed a high-density patchy infiltration shadow scattered in both lungs and an unclear lesion boundary that was distributed along the subpleural and lung marking. The electrocardiogram (ECG) examination (Figure 2) showed that paroxysmal atrial tachycardia, right bundle branch block, and low voltage of chest conduction. The left ventricular wall dyskinesia,

left ventricular hypertrophy, and decreased ventricular systolic function was diagnosed via Cardiac echocardiography (Figure 3). Because of the conditions available in the hospital at that time and the feasibility of practical operation, the coronary artery CT (CTA) scan was performed instead of coronary angiography, and the result indicated tiny stenosis in local lumen of right coronary artery (RCA), left anterior descending branch (LAD), and left circumflex branch (LCX) (Figure 4).

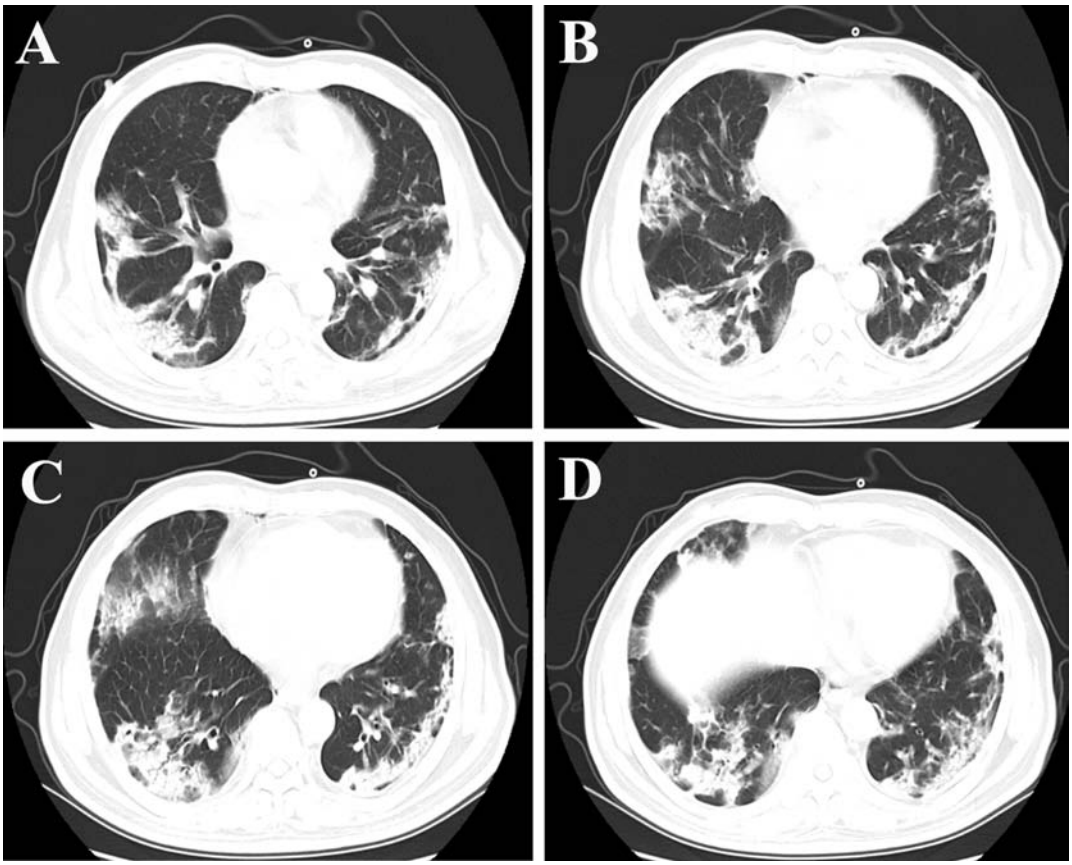

Figure 1. CT imaging of the patient (A-D). Thehigh-density patchy infiltration shadow was scattered in both lungs, and the lesion boundary was unclear and distributed along the subpleural and lung marking. Infectious lesions and viral pneumonia were considered.

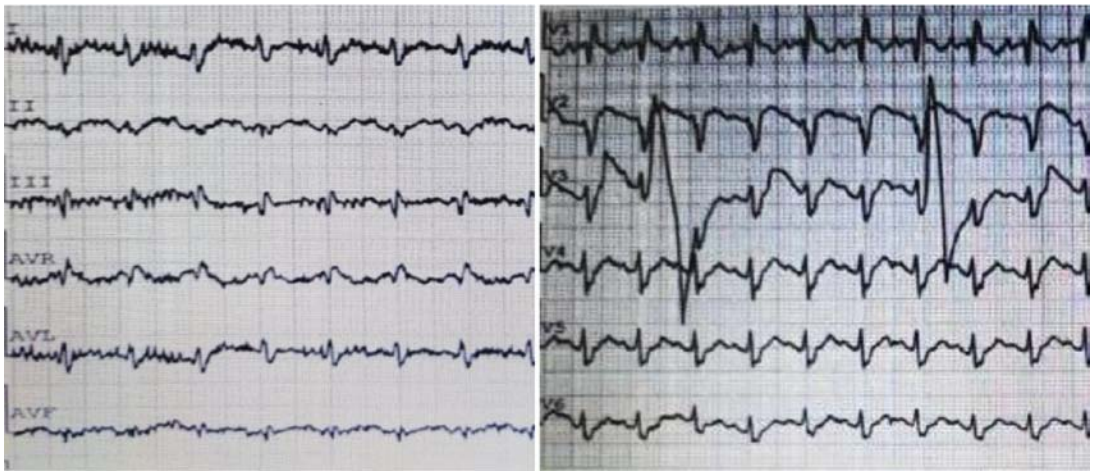

Figure 2. ECG characteristics of the patient. The ECG showed the following: the P'-P' spacing was regular, the QRS wave groups demonstrated anrSr' type, the QRS wave group period was<0.12 s, the P'-R was ≥ 0.12 s, the R-R was < 1/2, the HR ≥160 bpm, and the voltage of the QRS wave groups of each chest conduction was≤1.0 mV. The diagnosis was paroxysmal atrial tachycardia, right bundle branch block, and low voltage of chest conduction.

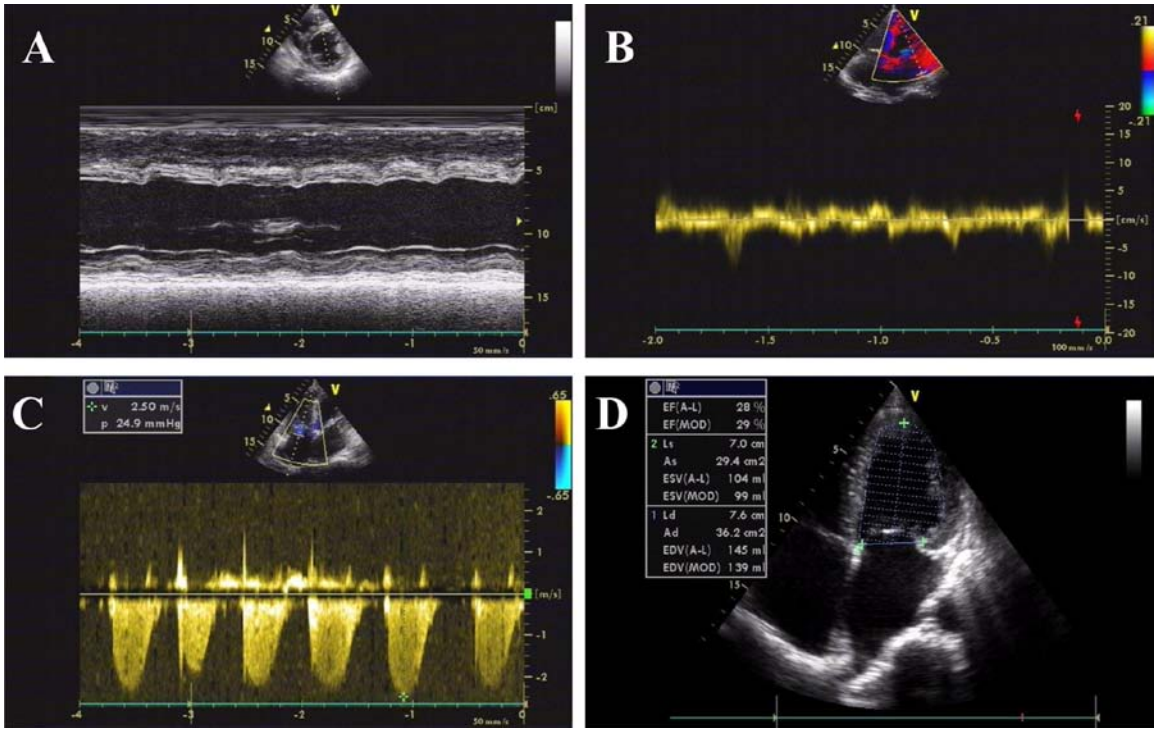

Figure 3. Characteristics of Cardiac echocardiography. The left ventricular wall dyskinesia, left ventricular hypertrophy, and decreased ventricular systolic function. Ejection fraction was 30%.

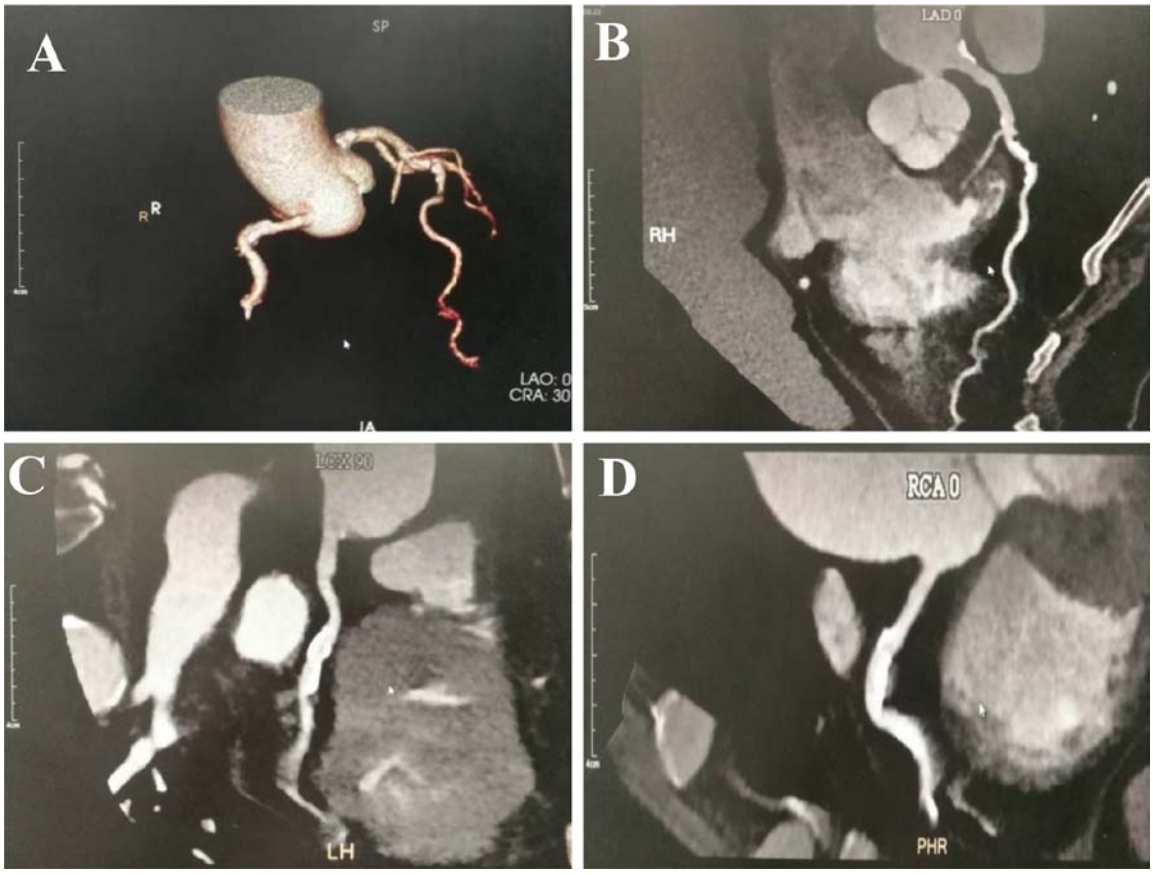

Figure 4. The coronary artery CT (CTA) scan indicated tiny stenosis in local lumen of right coronary artery (RCA), left anterior descending branch (LAD), and left circumflex branch (LCX). Atherosclerosis is diagnosed.

Final diagnosis: Based on above examinations, the patient was diagnosed with COVID-19 and Myocarditis caused by 2019-nCoV. **Treatment:** The patient was administered oral Ambroxol tablets (30mg,t.i.d), Arbidol Hydrochloride capsules (0.2g,t.i.d), Lianhuaqingwen capsules (1.4g,t.i.d), and Ibuprofen capsules (300mg,p.r.n),as well asintravenous Moxifloxacin (0.4g,s.i.d), 20% human serum albumin (10g,s.i.d), Methylprednisolone (40mg,s.i.d), and Imipenem sildastatin sodium (1.0g,q.8h). Following treatment, the patient's body temperature gradually decreased to normal, and symptoms of cough and dyspnea were significantly alleviated; however, there was no improvement in routine blood examinations, myocardial markers, and BNP levels. On day 6 after admission, CK-MB = 127.24 ng/ml(Normal reference: 0-4.97 ng/ml), TnI= 0.073 ng/ml (Normal reference: 0-0.04 ng/ml), and BNP = 302.00 pg/ml (Normal reference: 0-100pg/ml).

Outcome and follow-Up: On day 8 after hospitalization, the patient presented with chest stuffiness and dyspnea symptoms, and unexplained hypotension. ECG monitoring indicated ventricular fibrillation waveform, and the patient suddenly went into a coma with cardiac and respiratory arrest. After cardiopulmonary resuscitation and electric defibrillation, the patient's vital sign recovered and was immediately transferred to ICU for further treatment. The patient received high-dose methylprednisolone (500mg/d), immunoglobulin therapy (20g/d), ventilator-assisted ventilation, and extracorporeal membrane pulmonary oxygenation (ECMO).One month later, the patient was in stable condition and discharged home.

3. Discussion

In addition to typical respiratory complications, 2019-nCoVappears to cause cardiac damage in a certain patient population. It has been reported that approximately a third of COVID-19 patients have a concomitant elevation in myocardial injury markers or new abnormalities, as evidenced by ECG [4], which can manifest as acute myocardial injury, arrhythmia, and heart failure. Among these, the incidence of acute myocardial injury is 7.2%, the incidence of arrhythmia is 16.7% [5], and for patients with existing cardiovascular diseases, the mortality rate is as high as 10.5%. Furthermore, the mortality rate of patients with fulminant myocarditis can reach 50% to 70% [6].

Fulminant myocarditis is characterized by rapid clinical progression with acute left heart failure, cardiac shock, or severe arrhythmia several days after viral infection [7]. In this case, the patient typically has no history of heart disease, no chest pain, or other specific manifestations, and no specific ST-T changes in the ECG. The coronary artery CT scan showed no vascular obstruction. Consequently, the possibility of myocardial infarction can be excluded. There are no bacteria in the blood cultures. Based on the patient’s precursory symptoms of 2019-nCoV infection, various laboratory indicators, chest imaging, ECG, and Echocardiographyin this case report, the patient's condition was characteristic of fulminant myocarditis. The patient went a sudden cardiac arrest as a result of malignant arrhythmia, which was induced by fulminant myocarditis subsequent to the 2019-nCoV infection.

The successful treatment in this case provides insight into prognoses for COVID-19 patients with myocardial injury, such that clinicians should be vigilant about monitoring signs of myocardial injury caused by virus infection and implement myocardial protection measures as early as possible. Fulminant myocarditis caused by 2019-nCoV infection is similar to myocarditis caused by other viral infections. However, it is worth noting that the symptoms of COVID-19 maybeatypical [8], since the onset may not be accompanied by any acute signs and may appear insidiously. Two case reports from China and Italy reported that 2019-nCoV infection could cause fulminant myocarditis even without symptoms and signs of interstitial pneumonia [9, 10]. Therefore, early detection of laboratory indicators associated with fulminant myocarditis is extremely important. These examinations should include testing for Erythrocyte Sedimentation rate, C-reactive protein, BNP, CK-MB, TnI,andTnT [11, 12]. Dynamic monitoring of these indicators in the treatment of patients with fulminant myocarditis can reflect disease progression. However, since myocardial marker levels can be affected by infection, hypoxia, renal function, and other factors, myocarditis cannot be diagnosed solely by assaying circulating myocardial injury markers. Diagnosis and clinical monitoring should be more comprehensive and fully consider the clinical situation of the patient, as well as results of ECG, imaging, pathology, and other auxiliary examinations.

Dynamic ECG monitoring is a powerful strategy to diagnose fulminant myocarditis. ECGs of patients with fulminant myocarditis are variable and have low specificity, including sinus tachycardia, supraventricular tachycardia, ventricular tachycardia, nonspecific ST-segment depression, T wave changes, I-III degree atrioventricular block, and bundle branch block [13]. Therefore, repeatedly reviewing ECGs to observe dynamic changes and compare differences is recommended. Persistent changes in ST segments, R on T waveforms, and high degree atrioventricular blocks are suggestive of a poor prognosis [14]. Paroxysmal atrial tachycardia, low voltage of chest conduction, and severe systolic dysfunction were observed in this case report, indicating that the myocardium was damaged by the 2019-nCoV infection.

Echocardiography in patients with fulminant myocarditis is characterized by severe systolic dysfunction, increased wall thickness, and reactive myocardial edema, whereas left ventricular dilatation and abnormal wall thickness appear in acute non-fulminant myocarditis [15]. Right ventricular dysfunction is more common in patients with fulminant myocarditis, which is indicative of a poor prognosis [16]. Dynamic monitoring of echocardiography can reflect the recovery process of fulminant myocarditis. The echocardiography of this patient suggested a severe myocardial injury accompanied by left ventricular hypertrophy, and decreased ventricular systolic function with ejection fraction 30%.

The pathophysiological mechanisms of cardiac injury caused by 2019-nCoV remain unclear. At present, there are three possible mechanisms [17, 18] (Figure 5). (1) 2019-nCoV directly invades the cardiomyocytes, causing cardiomyocyte injury and viral myocarditis. (2) Hypoxemia, respiratory failure, shock, or hypotension induced by pulmonary infection lead to an insufficient myocardial oxygen supply, resulting in vigorous metabolic demands following infection. As a consequence, the cardiac burden increases and the imbalance of oxygen supply leads to myocardial injury. (3) A disordered immune response, which may lead to a cytokine storm, underlies the myocardial injury [19]. Some studies have shown that patients with 2019-nCoV infections have high levels of inflammatory factors, such as interleukin-β (IL-β), interferon γ (IFN-γ), and monocyte chemoattractant protein-1 (MCP-1) [20]. A multi-site puncture histopathology study of minimally invasive autopsies of patients with COVID-19 revealed a severe cardiomyocyte injury with inflammatory cell infiltration. However, no 2019-nCoV components were detected in the myocardial tissue, as evidenced by electron microscopy, immunohistochemical staining, and PCR, further supporting the cytokine storm hypothesis [20].

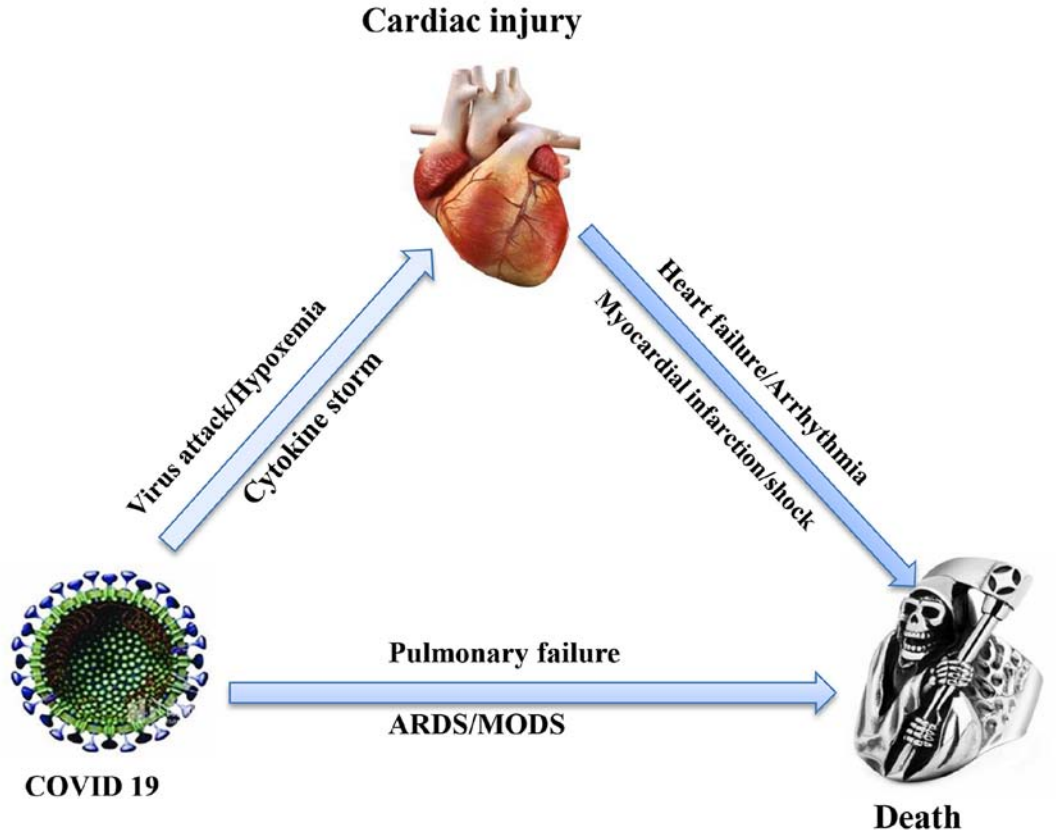

Figure 5. Mechanism of 2019-nCoV-induced myocardial damage and death. The mechanism underlying pneumonia leading to myocardial injury appears to involve direct virus invasion of the cardiomyocytes, secondary myocardial injury caused by a cytokine storm, and hypoxia.

2019-nCoV infection complicated by fulminant myocarditis progresses rapidly, with various unexpected difficulties encountered during treatment. Active comprehensive life support treatment should be initiated under intensive care, including rigorous bed rest, hemodynamic monitoring, continuous oxygen therapy, enhanced energy supply, and early prophylactic use of myocardial protective andantiarrhythmic drugs [21, 22]. Although early initiation of combined antiviral therapy is essential, there are currently no specific drugs for 2019-nCoV. The existing antiviral

drugs primarily include α-interferon, lopinavir/ritonavir, ribavirin, and chloroquine phosphate. According to the World Health Organizing guidelines, antimicrobials are recommended to treat all possible pathogens. For patients with sepsis, antimicrobials should be administered empirically within 1 hour of the initial evaluation. However, for patients with an accompany ingbacterial infection, poor immune function, and chronic severe basic diseases, antibiotics should be used as early as possible [23].

Immunotherapy for fulminant myocarditis includes administration of glucocorticoids and immunoglobulins, but the efficacy of this treatment strategy is not clear, and relevant clinical studies are still in progress. Despite the uncertainty, immunotherapy should be actively considered for patients with severe heart failure and patients with no improvement or deterioration following routine treatment [10]. If a patient shows signs of cardiac failure, advanced life support therapy should be given immediately, such as ventilator-assisted ventilation, blood purification and continuous renal replacement therapy (CRRT), intra-aortic balloon counterpulsation (IABP), and extracorporeal membrane pulmonary oxygenation (ECMO) [24]. CRRT and ECMO can remove some cytokines from the blood, increase blood oxygen saturation, reduce the excessive cytokine-induced immune response, and further reduce myocardial injury [25].

4. Conclusion

Symptoms of myocardial injury caused by 2019-nCoV infection may be atypical and may occurearlier than the appearance of pulmonary symptoms. This case report provides the following insights for treating COVID-19 patients: (1) Improvements in virus infection and pulmonary symptoms are not necessarily indicative of improvements in all COVID-19 disease-related symptoms. Changes in cardiac-related indicators and ECGs should be dynamically monitored. (2) Clinicians should be aware of myocardial injury induced by the virus in the treatment of patients with COVID-19, and early prophylactic use of myocardial protective drugs should be considered to avoid the occurrence of fulminant myocarditis even without evidence of myocardial injury. (3) If conditions continue to deteriorate, early combined advanced life support therapy, such as blood purification and CRRT, IABP, and ECMO, may positively affect the prognosis of COVID-19 patients.

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The patient's family has provided informed consent for publication of the case.

Conflict Of Interest

The authors have no conflicts of interest to declare.

Data Availability Statement

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

Consent

The patient's family provided written informed consent regarding the publication of the case details and any associated images.

Competing interests

The authors declare that they have no competing interests.

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