A case of fulminant myocarditis associated with a novel coronavirus infection

Bixia Yan
Dali University
Zhengjiang Liu (✉️ 29542006j@sina.com)
Youjiang Medical University for Nationalities, Affiliated Hospital of Youjiang Medical University for Nationalities

Case Report

Keywords: SARS-CoV-2, fulminant myocarditis, post-acute phase, intra-aortic balloon inversion, extracorporeal membrane oxygenation

Posted Date: May 25th, 2023

DOI: https://doi.org/10.21203/rs.3.rs-2888970/v1

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Abstract

Background

COVID-19 patients mainly have the high incidence and mortality of major cardiovascular diseases in the acute phase. However, fulminant myocarditis and cardiogenic shock are rare in the post-acute phase, and the prognosis is very poor.

Case presentation

Here, we report a case of a 43-year-old young man with fulminant myocarditis and cardiogenic shock in the acute phase after coronavirus infection, with timely extracorporeal membrane oxygenation and intra-aortic balloon application in respiratory failure, which improved the prognosis of fulminant myocarditis and saved the lives of COVID-19 patients. After active anti-shock, mechanical ventilation, anti-inflammation, and organ support and other treatments, the patient was discharged after a stable condition and stable vital signs. The patient was followed up weekly after discharge and showed good cardiopulmonary recovery.

Conclusions

In critically ill patients with severe myocarditis, cardiogenic shock, arrhythmia, respiratory and circulatory failure, ECMO and IABP in patients with explosive myocardial inflammatory cardiogenic shock have achieved better results.

Background

In December 2019, COVID-19 (COVID-19) was first detected in Wuhan, China, and the patient developed influenza-like symptoms. The virus was isolated and identified as a new coronavirus strain, now named SARS-CoV-2 (Severe acute respiratory syndrom coronavirus 2). The mortality rate for COVID-19 is estimated to be 1%, mainly due to severe acute respiratory syndrome and multiple organ dysfunction. COVID-19; however, the first case of myocarditis has been reported in COVID-19 patients, Coronavirus disease can cause fulminant myocarditis, cardiogenic shock, and myocarditis has been identified as the cause of death in some COVID-19 patients. Now we report a case of fulminant myocarditis and cardiogenic shock in the acute phase after coronavirus infection.

case presentation

The patient, a male patient, was 43 years old. Due to "chest pain, shortness of breath for more than 6 hours", he was admitted to our hospital. None of breath 6 hours before admission, Sudden chest pain on the walking hospital, Located behind the sternum, Persistent, pressing samples, The range is about a
slap-sized, Blood pressure measurement: 82 / 50mmHg, To the static drip of dopamine, The ECG shows an uplift change in the ST segment, CK-MB 42.31ng/ml, cTnI 16.66ng/ml, Myo < 30.0ng/ml, Consider "acute myocardial infarction", "Polivine 300mg, aspirin 300mg, low molecular weight heparin 4250iu" and referral to our hospital emergency, Emergency coronary angiography was performed through a green channel, Coronary angiography was performed without any abnormality. Novel coronavirus infection for more than 20 days. Smoking for more than 20 years, an average of 20 cigarettes per day, did not quit smoking, no special rest. Physical examination on admission: blood pressure 90 / 53mmHg, pulse 101 times / minute, fingertip blood oxygen about 88–93%. Clear, a little wet rales can be heard in the left floor of the left lung, heart rate 101 times / min, heart rhythm, no third and fourth heart sounds, no pathological noise in the auscultation area of each valve, no pericardial friction sounds, and no abnormal signs. After admission: blood cell analysis: white blood cell count 17.25 (10 ^ 9 / L), Hemoglobin concentration of 124 (g / L); Blood biochemistry: total protein 61.1 (g / L), Creatine kinase 403 (U / L), Total bilirubin 1.6 (umol/L), C-Reactive protein 7.65 (mg/L), Albumin, 36.6 (g / L), Creatine kinase Isozyme 66 (U / L), Lactate dehydrogenase 282 (U / L), Myoglobin 144.2 (ug / L), Troponin I 18.023 (ug / L), D dimer 1.11 (mg/L), Pro-BNP 4942pg/ml. All the other blood parameters were within the normal range. Chest CT: considering the possibility of interstitial pulmonary edema; admitted cardiac ultrasound: left atrial enlargement, segmental wall motion abnormalities, mitral regurgitation (mild); pericardial effusion (small), reduced left ventricular systolic function (EF: 40%); recommended review after treatment. Combined with the above history, physical examination and examination results, patients with young men, no basic history, will be coronavirus infection more than 20 days, before the onset of fever, symptoms of chest pain, elevated troponin, patients with blood pressure to maintain adrenaline, consider will be coronavirus infectious myocarditis, acute heart failure, cardiogenic shock, with respiratory failure, serious illness.

Into intensive care unit via endotracheal intubation, continuous ventilator assisted ventilation treatment, high-dose vascular active drugs pump to maintain blood pressure and heart rate, blood pressure is still unstable, respiratory failure, oliguria, limbs cold tissue low perfusion performance, consider patient myocardial edema, will be coronavirus infection myocarditis progression, accord with mechanical heart auxiliary indications. Decided to perform extracorporeal membrane lung (ECMO) V-A mode assistance. The arterial cannula (17 Fr) and venous cannula (21 Fr) were placed in the peripheral femoral artery and femoral vein, respectively, allowing drainage from the femoral vein and reflux to the femoral artery. The cardiopulmonary bypass was successfully established, and the patient was successfully connected to the ECMO machine. The ratio of blood flow and gas flow is 1:1, the rotation speed is 3000rpm, and the flow rate is 2.8-3.0 l. During the period of continuous ECMO assistance, Pro-BNP 11267 pg/ml, electrocardiogram indicated high atroventricular block (see Fig. 2), reexamination of cardiac ultrasound showed no effective contraction, EF: 17%, no open aortic valve, aggravated pulmonary edema, and LV hypertension. In accordance with IABP indications, the IABP pacemaker was combined in a 1:1 ratio. The patient had continuous diuresis, obvious metabolic alkalosis, combined with bedside color ultrasound monitoring: the inferior vena cava was still full, and there were indications for bedside CRRT treatment. In order to optimize capacity and adjust the internal environment, CRRT treatment was received. According to the development of the patient's condition, With empirical anti-bacterial infection treatment with
cefuroxime / piperacillin tazobactam / cefoperazone sulbactam / in combination with voriconazole plus compound sulfamethoxazole, Add β blockers according to blood pressure and heart rate, diuretic, Neoretin resistance against heart failure, Spironolactone discharges sodium, preserves potassium improves cardiac remodeling\(^7\) and reduces pulmonary edema\(^8\), Blood pressure has leveled off, Regulation of immunity, With vitamin C, coenzyme Q10 / trimetazidine, Patient general condition, oxygenation and liver and kidney function, Cardiac enzymes, troponin became normal, Repeat cardiac color ultrasound: EF53%, Pro-BNP 495pg/ml, Improvement and discharged. After outpatient follow-up, the condition is stable.

**Discussion**

Since the start of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) war, as of December 2021, COVID-19 has spread to 222 countries, resulting in more than 260 million confirmed cases and more than 5 million deaths worldwide\(^9\). The most common long-term effects and adverse consequences of SARS-CoV-2 infection are sepsis, pneumonia, and other pulmonary infections. In fact, in addition to the possible direct infection of the heart muscle and other types of cardiovascular cells, the novel coronavirus can also mediate immune responses and inflammation, thus causing severe damage and dysfunction of the cardiovascular system. In the acute phase, the incidence of major all-cause cardiovascular diseases and mortality of COVID-infected patients increased significantly. Compared with uninfected patients, the risk of various cardiovascular diseases after "Yang" increased significantly by 330%, and the risk of all-cause death was 81.1 times that of the former. And after the acute phase ("Yang" after 21 days to 18 months), the incidence of cardiovascular sequelae and all-cause death risk is still "high", compared with not infected new crown, even has "Yang kang", the risk of major cardiovascular disease is still 40% higher, all-cause mortality is 4.5 times of the healthy controls\(^10\).

The results of the epidemiological investigation show that\(^11\): Out of novel coronavirus patients, the incidence of acute myocarditis was 2.4–4.1‰, The median age was 38 years, Chest pain and dyspnea were the most common symptoms (55.5% and 53.7%, respectively).39% had fulminant myocarditis findings and 70.4% were admitted to the intensive care unit. In all patients with myocarditis associated with COVID-19, the incidence of cardiogenic shock and distributive shock was 27% and 12%, respectively\(^12\). Mortality or temporary mechanical circulatory support during hospitalization was 20.4%. The 120-day mortality rate was 6.6%. This patient was a young man who developed chest pain for more than 20 days, was in the postacute stage, and had elevated myocardial injury markers, abnormal electrocardiogram and echocardiography, which was in line with the expert consensus of the American College of Cardiology (ACC) in 2022\(^13\), Novel coronavirus-associated myocarditis can be classified as possible, extremely likely and definite. Clear myocarditis has clinical manifestations of acute myocarditis, elevated myocardial injury markers, abnormal electrocardiogram and (or) echocardiography, so the diagnosis of fulminant myocarditis associated with novel coronavirus infection.
Guidelines for the National Institute of Health and Care Excellence in the UK\textsuperscript{14} Long novel coronavirus was defined as symptoms lasting more than 4 weeks after SARS-CoV-2 infection. The prevalence of long novel coronavirus in China is 49–76\%\textsuperscript{15, 16}. Although myocarditis cases following SARS-CoV-2 infection are rare, they largely follow this pattern, with the onset delayed by several days to weeks after viral infection. In some patients with myocarditis, elevated cTn and abnormal CMR are observed early after infection; however, more common are delayed onset of cardiac symptoms, elevated cardiac biomarkers, and abnormal cardiac imaging. At present, the mechanism of myocarditis after novel coronavirus infection is not completely clear, and the mechanism of myocardial injury includes include\textsuperscript{11, 13}: Direct cytotoxic injury, dysregulation of the renin-angiotensin-aldosterone system, endotheliitis and thrombotic inflammation, and dysregulation of immune response with cytokine release. The mechanisms of persistent cardiac injury following an acute infection remain poorly understood. One possible explanation is the chronic inflammatory response caused by the persistent viral reservoir in the heart after acute infection\textsuperscript{17}, In perivascular adipose tissue through the release of adipokines (such as monocyte chemotaxis protein-1) and activation of normal T cell expression and possible secretion of chemokines, through endothelial nitric oxide synthesis enzyme and coupling and reactive oxygen species aggravate endothelial dysfunction, causing obesity related inflammation signal, endothelial dysfunction\textsuperscript{18}, Induced coronary vasospasm, slow blood flow, myocardial ischemia changes, which may be the reason why the clinical manifestations of myocardial infarction, while coronary angiography is normal. An unintended consequence of these processes is the underlying tissue damage, followed by chronic myocardial fibrosis, resulting in impaired ventricular compliance, impaired myocardial perfusion, increased myocardial stiffness, reduced contractility, and underlying arrhythmias. Pathology is usually confined to the myocardium, but there is a risk of cardiac arrhythmia, progression to fulminant heart failure, and cardiogenic shock.

The combined application of IABP and V-A ECMO can convert the non-pulsatile perfusion of V-A ECMO from IABP to beat perfusion, make the perfusion closer to the physiological state of human body, improve the perfusion effect of organs, and promote the recovery of patients. IABP can provide higher diastolic pressure that oring coronary perfusion and improve oxygen supply to the myocardium. This study also showed that the systolic blood pressure and diastolic blood pressure increased significantly, and the central venous pressure decreased significantly, which improved the myocardial oxygen supply of the myocardium and reduced the volume load of the heart. The combination of IABP and V-A ECMO, which can reduce the time of ECMO operation, is stable in the patient, and restore cardiac function to a certain level, assisted by IABP alone. This can not only reduce the complications caused by long ECMO operation, but also avoid the fluctuations of circulation and cardiac function after ECMO evacuation\textsuperscript{19}. In this case, the coronavirus outbreak of severe myocarditis, cardiogenic shock, arrhythmia, extracorporeal membrane oxygenation and intra-aortic balloon antibo, improved peripheral organs and coronary blood flow, to maintain circulation stability, the treatment effect was obvious, and saved the patient.

**Conclusion**
This case report suggests that ECMO and IABP are combined with explosive myocardial shock, and in critically ill patients with cardiogenic shock, arrhythmia and respiratory and circulatory failure. Severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) infection with cardiac magnetic resonance imaging and (or) biopsy showed active myocarditis. The insufficiency of this case is that the patient was in critical condition, without cardiac magnetic resonance imaging, and endocardial biopsy further clarified the condition of myocardial edema and myocardial necrosis.

**Abbreviations**

ECMO  Extracorporeal membrane oxygenation
IABP  intra-aortic balloon pump
ECG  Electrocardiogram
V-A ECMO  Veno-arterial ECMO

**Declarations**

**Acknowledgements**

Not applicable.

**Author contributions**

ZL conceived the investigation, reviewed and edited the paper. BY analyzed the data, interpreted the patient data and wrote the manuscript. All authors read and approved the final manuscript.

**Funding**

Not applicable.

**Availability of data and materials**

All data in the study are included in this published article.

**Ethics approval and consent to participate**

Not applicable.

**Consent for publication**

Informed consent of clinical detail and image publication was obtained from the patient.
Competing interests

The authors declare that they have no competing interests

Author details

1Dali University, Dali 671000, Yunnan, China. *† Department of Cardiology, Youjiang Medical University for Nationalities, Affiliated Hospital of Youjiang Medical University for Nationalities, Baise 533000, China.

References


**Figures**

![Figure 1](image_url)

**Figure 1**

shows the ECG taken on admission
Figure 2

shows the ECMO assisted with high atrioventricular block