A Case of COVID-19-Induced Delayed-Onset Myocarditis

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Patient: Female, 38-year-old
Final Diagnosis: COVID-19 infection • myocarditis
Symptoms: Dyspnea • hypotension
Medication: —
Clinical Procedure: —
Specialty: Cardiology • Critical Care Medicine • Infectious Diseases

Objective: Rare coexistence of disease or pathology
Background: COVID-19 is a pandemic caused by a coronavirus that has only recently been discovered. The disorder is characterized by persistent respiratory system malfunction, which can range from modest difficulty breathing to potentially lethal complications such as acute respiratory distress syndrome. Additional organs are affected as a result of its presence. An adverse impact of COVID-19 infection is myocarditis, which is a condition that affects the heart muscle.

Case Report: We describe the case of a 38-year-old woman who was hospitalized at University Medical Center Ho Chi Minh City following a 15-day fever, a 3-day bout of dyspnea, and a positive nasal PCR SAR-CoV-2 test. The Lake Louise criteria were used to determine that the patient had a high probability of having myocarditis. She was then treated with oxygen treatment, vasoconstrictor medicines, inotropic therapy, and cornerstone heart failure medications, and was discharged 2 weeks later after a complete recovery.

Conclusions: Myocarditis has been identified as a cause of death in COVID-19, although it is not known how common the ailment is in the general population. Early detection and complete treatment, which should include support for the cardiovascular system, are consequently critical for successful outcomes. Magnetic resonance imaging (MRI) of the cardiovascular system (cardiac MRI) is the most important noninvasive method for diagnosing myocarditis.

Keywords: COVID-19 • Magnetic Resonance Imaging • Myocarditis

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Background

In December 2019, many instances of influenza-like disease were reported in Wuhan (China), prompting the designation of COVID-19 as the next pandemic (Siripanthong, Nazarian, Muser [1]). SARS-CoV-2 was the designation given to the newly discovered coronavirus (Severe Acute Respiratory Syndrome Coronavirus 2). The majority of deaths occur as a result of acute respiratory distress or multiorgan failure, which combined account for up to 1% of all deaths [1]. According to a recent analysis, patients with COVID-19 had a nearly 16-fold increased risk of myocarditis compared to those without COVID-19 [2]. At least 47.8% of COVID-19 patients exhibited myocardial damage, and around 2% had confirmed myocarditis, according to autopsy findings [3]. Additionally, myocardial injury can result in irregular heartbeats and, in severe cases, cardiac shock. As a result, acute myocarditis must be recognized and treated appropriately in this situation.

We describe a patient with SARS-CoV-2 who developed myocarditis and acute heart failure at University Medical Center Ho Chi Minh City. A consent form for publication was signed by the patient.

Case Report

A 38-year-old woman with no known cardiac abnormalities presented with dyspnea after a 15-day fever, a 5-day dry cough course, and a positive fast COVID-19 test. Three days before admission, she began having breathing problems while walking and developed paroxysmal nocturnal dyspnea later in the day. She was then admitted to a nearby hospital and treated with vasopressor medicines for severe dyspnea and hypotension. The admission ECG indicated a sinus rhythm of 158 beats per minute, a QS wave in the inferior, V1, and V2 leads, with a ST elevated segment in the V1, V2, and V3 leads (Figure 1). The echocardiogram indicated a 40% ejection fraction and hypokinesis of the septal wall. Chest computed tomography showed ground-glass opacification lesions in both lungs with no signs of pulmonary artery embolism. The laboratory test results revealed elevated hs-troponin T concentration at 30 670 ng/L, D-dimer was 7103 ng/mL, CRP was 91 mg/L, LDH was 677 U/L, and a negative fast COVID-19 test. Treatment began with a 15 L/min oxygen supplement, noradrenaline (0.04 mcg/kg/min) and dobutamine (5 mcg/kg/min). The diagnosis of acute anterior ST elevation myocardial infarction was made and the patient was then transferred to University Medical Center Ho Chi Minh City for primary PCI.

At the Emergency Department, the patient was conscious, with cold extremities, a weak 130 bpm pulse, 110/60 mmHg blood pressure, and a 96% oxygen saturation with a 15 L/m mask. The examination revealed moist coarse rales on both lungs. A routine chest radiograph showed evidence of peri-bronchial cuffing and an elevated cardiothoracic ratio (Figure 2). The ECG indicated a 120-bpm sinus rhythm, QS in V1-V3, D3, and aVF leads, elevated ST segment in V1-V4 leads, and a negative T wave in V2-V6 leads. The laboratory test results showed elevated hs-troponin T at 1716 ng/L, D-dimer was 10880 ng/mL, LDH was 782.80 U/L, ferritin was 7861 ng/mL, and IL-6 was 66.71 pg/mL. Her PCR SARS-CoV-2 test result was positive. We kept her on a 15 L/m oxygen mask, as well as noradrenaline, dobutamine, and intravenous furosemide. She was subsequently moved to the Interventional Cardiology Department.
myocarditis following COVID-19. Although the predominant

Discussion

was satisfactory, and a 2-week checkup found no irregularities.

no dilated chambers, no dyskinesis, a 57% left ventricular ejec
tion fraction, and no significant abnormalities. Her rehabilitation

performed after dobutamine was terminated, which indicated

proBNP, were within normal limits. Another echocardiogram was

appetite was normal. Other laboratory test results, such as NT-

We then started her on 100 mg sacubitril/valsartan and 2.5 mg

bisoprolol every day. There was no fever or dyspnea, and her

patient, an MRI indicated slight dilatation, a low left ventricu-

dial injury (IRI) (MRI). With regard to the left ventricle of our

Figure 3. CMR imagings with global hypokinesis of both

ventricles and pericardial effusion (arrow) in short-axis

Cine imagings.

where she was diagnosed with COVID-19-related acute heart

failure and severe pneumonia.

The patient remained conscious for the next 72 h, with a blood

pressure ranging between 85/50 and 120/70 mmHg. Oxygen

supplementation was continued nasally with a 5 L/m flow. A new

chest X-ray was taken, and the indicators of congestion were considerably reduced. She was kept on dobutamine for a few days after that, with concomitant oral digoxin, and intravenous furosemide and spironolactone. We ordered a card-

iac MRI (Figure 3), which revealed a mildly dilated left ven-

tricle, a severe reduction in left ventricular ejection fraction (18%), hypokinesis of the septal region and left free ventricu-

lar wall, myocardial hypertensity in the left free ventricular wall on T2W, and late gadolinium enhancement (LGE) of cardi-

ac muscles with non-ischemic pattern. An angiography of the
coronary arteries indicated no major stenoses in the main ar-

teries. The final diagnosis was COVID-19-associated myocarditis aggravating severe acute heart failure.

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endocardial biopsy should be used to confirm the diagnosis of
definite myocarditis. In the absence of diagnostic instruments

and experienced physicians to perform the biopsy, there are

alternative approaches to increase the likelihood of correctly
diagnosing myocarditis, such as cardiac MRI or echocardiog-

raphy [1]. Myocarditis is characterized by ischemia-reperfu-
sion injury (IRI) (MRI). With regard to the left ventricle of our

patient, an MRI indicated slight dilatation, a low left ventricu-

lar ejection fraction (18%), hypokinesis in the septum and left

free wall, late enhancement of cardiac muscles, and hyperin-
tensity in the left free wall on T2W. All of these signs point to

myocarditis. The Lake Louise criteria, which is typically used
to diagnose suspected myocarditis, states that if at least 1 of
the following criteria is positive in a patient with a high clini-
cal suspicion of myocarditis, a CMR scan gives strong evidence
for acute myocardial inflammation: T2-based marker for myocar-
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[7]. As previously described, our patient displayed myocardial edema on T2-turbo inversion recovery magnitude imaging (Figure 4) and a non-ischemic pattern on ventricular septum imaging (Figure 5), both of which are consistent with a high suspicion of myocarditis.

Myocarditis often manifests within a few hours or days af-
ter exposure to risk factors. In the case of novel, non-experi-
mental agents such as the SARS-CoV-2 virus, case reports and
series would provide a new perspective on the virus’s effect
on cardiac cells in addition to other organs. Cytokine storm
is the hallmark of the condition, resulting in endothelial inflam-
mation and micro- and macrovascular thrombosis [8].

The term “delayed-onset myocarditis” was coined in a recent
study [9]. The authors found 12 cases of myocarditis associ-
ated with COVID-19, with onset times ranging from several

clinical manifestation of COVID-19 is in the respiratory system,
some patients experience cardiac symptoms [4]. COVID-19’s
cardiac involvement in other coronavirus disorders is hypoth-
esized to be caused by the direct viral impact on myocardial
cells or indirect toxicity via systemic infection, inflammatory
arteries, and the host’s hypersensitive reactivity [5].

Symptoms of COVID-19-associated myocarditis include tired-
ness, dyspnea, chest discomfort, tachycardia, sudden cardiac
failure, and cardiac shock [1]. Ventricular failure can be utilized
to diagnose acute myocarditis patients 2-3 weeks after viral
infection, and this has been shown by 22 studies on COVID-19
myocarditis. Symptoms included fever, fatigue, and myalgia
in 77% of articles (18%) [6]. In 12 instances, chest discomfort
(50%), tachycardia (25%), and hypotension (50%) were record-
ed. Troponin, CRP, CK-MB, and NT-proBNP were also high in
most cases. T wave inversion, tachycardia, and ST abnor-
malities were identified in 47% of patients (18%). In this research,
77% of patients exhibited worsened left ventricular function.

Discussion

We describe the case of a 38-year-old woman who experienced
myocarditis following COVID-19. Although the predominant

Endocardial biopsy should be used to confirm the diagnosis of

definite myocarditis. In the absence of diagnostic instruments

and experienced physicians to perform the biopsy, there are

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ated with COVID-19, with onset times ranging from several
days to weeks and the longest period since SARS-CoV-2 infection being 1 month. Numerous factors can contribute to a delayed diagnosis or realization, spanning from the rarity of severe myocardial injuries to the limited diagnostic value of antibody tests, systolic dysfunction secondary to previous cardiac conditions, and the difficulty of conducting several investigations during the COVID-19 period. Although consensus has not been reached on the definition of “delayed-onset myocarditis”, recent findings indicate that COVID-19-related myocarditis that manifests several days to weeks later, as in our case, 13 days later, should be called “delayed-onset myocarditis”.

Throughout the diagnostic process for viral myocarditis, it is important to rule out the presence of bacteria. Aside from culture specimens, supporting variables for distinguishing between the 2 illnesses include the symptoms of presentation and laboratory test results. Throughout the course of treatment, our patient showed no indicators of acute infection, and procalcitonin levels and blood cultures were all negative throughout the process. The diagnosis for viral myocarditis became more likely when the SARS-CoV-2 PCR test revealed a positive result.

Using cardiac MRI, Inciardi et al identified COVID-19-related myocarditis by elevated troponin and impaired left ventricular function [5]. After 1 week of inotropic therapy, this patient fully recovered. Hu et al found a similar pattern in their investigation [10]. Due to hyperacute myocarditis and severe left ventricular failure, the 37-year-old patient required immediate surgery. After 3 weeks of corticosteroid and immunoglobulin treatments, he recovered completely. COVID-19-related myocarditis requires ICU care, oxygen, vasoconstrictor medications, and other bridging therapy [11]. In some cases, ECMO, intra-aortic balloon pumps, and ventricular assist devices have been employed. Despite this, the European Society of Cardiology does not recommend immunoglobulins or corticosteroids for advanced myocarditis [1]. COVID-19 patients with early signs of hypoxia can be effectively managed with Remdesivir 200 mg on day 1 and 100 mg on the next 9 days [12]. Dexamethasone is also used in this group of COVID-19 patients, with a dose of 6 mg per day for up to 10 days [13]. COVID-19-related myocarditis can be successfully treated with inotropic medications, renin-angiotensin system inhibitors, and diuretics.

**Conclusions**

Myocarditis is a serious cardiac consequence of SARS-CoV-2 infection, made even more problematic by the virus’s proclivity for delayed onset. Although its incidence is unknown, it is one of the causes of cardiac mortality in patients with COVID-19. As a result, early discovery and comprehensive treatment, including cardiac support, are crucial. Due to the difficulty of performing endocardial biopsy during the COVID-19 pandemic, cardiac magnetic resonance imaging is a noninvasive approach that can be utilized to identify myocarditis.

**Ethics Approval**

The publication of this case report was approved by the Ethics Committee for Biomedical Research at University Medical Center Ho Chi Minh City, and a consent form was signed by the patient.
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