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Case Report

COVID-19 Infection Concomitant With Infective Tricuspid Valve Endocarditis

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ABSTRACT

The COVID-19 pandemic, together with its complications and management, has been a significant issue worldwide since March 2020. Concomitant infections in vulnerable patients with preexisting cardiovascular diseases are not uncommon. Sharing information about the diagnostic management and treatment of these comorbidities has a prominent role in unveiling some of this pandemic's challenges. We herein describe a young adult with a history of implantable cardioverter-defibrillator implantation diagnosed with COVID-19 infection and infective endocarditis. ***(Iranian Heart Journal 2022; 23(4): 102-108)***

KEYWORDS: COVID-19, Endocarditis, Tricuspid valve

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The world has been struggling with a new COVID-19 pandemic since March 2020. There is multisystem involvement of the disease; nonetheless, the diagnosis of COVID-19 must not imply the exclusion of other diseases. The cardiovascular outcomes of this infection are under study the world over. The recognition and management of related comorbidities in vulnerable patients are essential.

Case Report

A 35-year-old woman was brought to the emergency department by the emergency medical service (EMS) with the chief complaints of fatigue, shortness of breath, dry cough, and dyspnea on exertion, which had worsened the night before admission. The patient had experienced several episodes of fever (38.4 °C) and chills in the preceding few days. She had a history of permanent pacemaker (PPM) implantation 2 years previously for sick sinus syndrome. She also had undergone sleeve surgery for weight loss 4 years earlier.

On admission, the patient was ill but alert. She had a blood pressure of 120/80 mm Hg, a heart rate of 80 beats per minute, an oral temperature of 37.5 °C, a respiratory rate of 24 breaths per minute, and an oxygen saturation level of 96% with a face mask. On physical examination, she was pale. Chest auscultation revealed fine crackles at the base of the lungs and 4 out of 6 holosystolic murmurs at the lower left sternal border. Her extremities were cold.

Initial laboratory tests showed leukocytosis, thrombocytosis (platelet =651000), microcytic anemia, an elevated lactate dehydrogenase level (1039 U/L [normal range <480 U/L]), a high C-reactive protein level (200 mg/L), a high ferritin level (977 ng/mL [normal range =13–172]), an increased D-dimer level (4323 ng/ml [normal range = up to 1000), and an elevated NT-proBNP level (7302 pg/mL [normal range = up to 125]). Her troponin and procalcitonin levels were within the normal ranges. Electrocardiography showed normal sinus rhythm with a normal axis and no significant ST-T changes (Fig. 1). The findings of the patient's spiral lung high-resolution computed tomography were multiple ground-glass opacities, perifissural consolidation at the right upper lobe, and consolidation with cystic bronchiectasis at the posterior and lateral segments of the right lower lobe (Fig. 2). Transthoracic echocardiography showed an ejection fraction of 40% to 45% and a large mass (2.2 cm^2 in area) with a shaggy appearance on the tricuspid valve, producing severe tricuspid regurgitation. Given the patient's clinical setting, the mass was highly suspicious of vegetation, with thrombosis being less likely. Additionally, the PPM lead was seen on the right ventricle with no visible clot or mass on it (Fig. 3).

The findings suggested that infective endocarditis and concomitant COVID-19 infection were the most probable diagnosis. A nasopharyngeal swab test reverse-transcription polymerase chain reaction (RT-PCR) assay was performed to confirm COVID-19. While awaiting the results, the patient was quarantined in the isolation room. Transesophageal echocardiography (TEE) was performed to confirm the previous findings. TEE demonstrated a moderately sized shaggy mass (1.6×0.7 cm) on the tricuspid valve. The mass had perforated the posterior leaflet, leading to free severe tricuspid regurgitation (Fig. 4 & 5). Empirical antibiotic therapy was started including meropenem (1 g every 8 h) and vancomycin (1 g every 12 h). Loop diuretics, spironolactone (12.5 mg daily), bisoprolol (2.5 mg daily) for neurohormonal blockade, and heparin prophylaxis were added.

On day 3 of admission, COVID-19 PCR was positive, confirming the infection. The interleukin-6 level was high (11.7 pg/mL [reference range <5.9 pg/mL). The antiviral regimen, composed of remdesivir, interferon β-1, and N-acetylcysteine (600 mg every 12 h) for hepatoprotection, was prescribed.

By days 6 to 10, the patient's symptoms, including dyspnea and fatigue, were mostly resolved. Her oxygen saturation level was 97% in room air, and her blood culture revealed no growth after 7 days. Follow-up TEE examinations were done on days 7, 14, and 21 of admission, and they revealed a noticeable decrease in the size of the mass (Fig. 6). The C-reactive protein level exhibited a fall from 200 mg/L to 12 mg/L on the discharge day. The lactate dehydrogenase level, the white blood cell count, and the platelet level returned to the normal reference range. A nasopharyngeal swab re-examination was performed on day 10 of hospitalization, and the results were negative. After 3 weeks of standard antibiotic therapy, the patient was discharged on day 21.

The patient was referred to a cardiac surgery center, where the endocardial leads were removed, and the vegetation on the tricuspid valve was debrided. In addition, the valve was replaced with a 31 Hancock II bioprosthesis. The bipolar epicardial right atrial and ventricular leads were placed properly and connected to its generator on the right hypochondriac abdomen. The culture of the valve biopsies and the pacemaker lead was positive for methicillin-resistant *Staphylococcus aureus* (MRSA), and the patient continued the vancomycin therapy for 6 weeks.

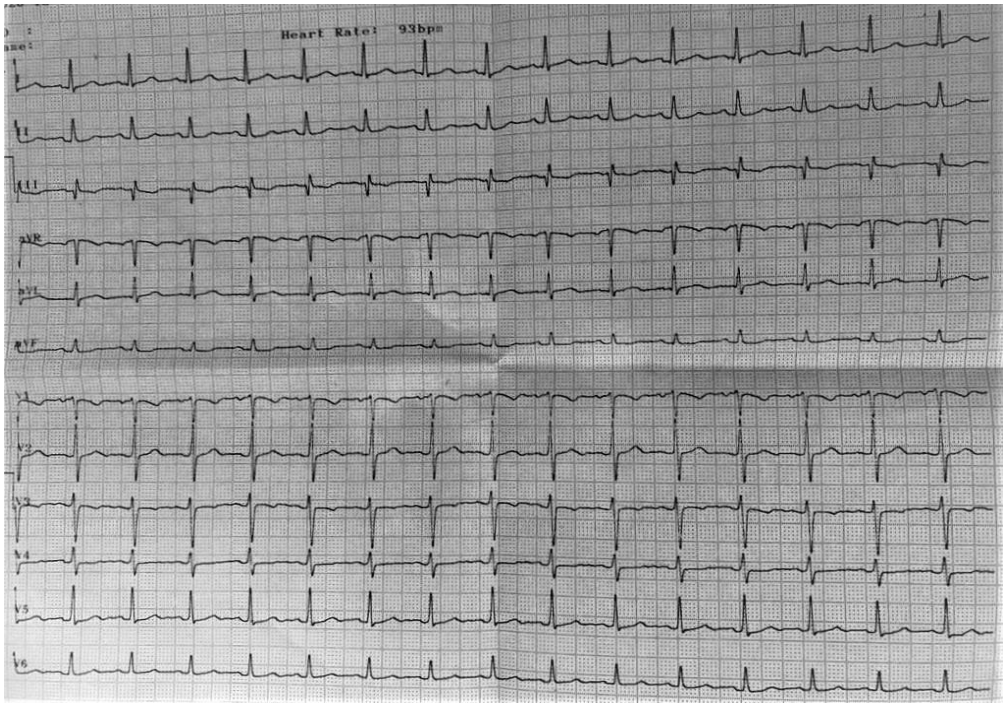

Figure 1: Electrocardiography shows normal sinus rhythm with a normal axis and no significant ST-T changes.

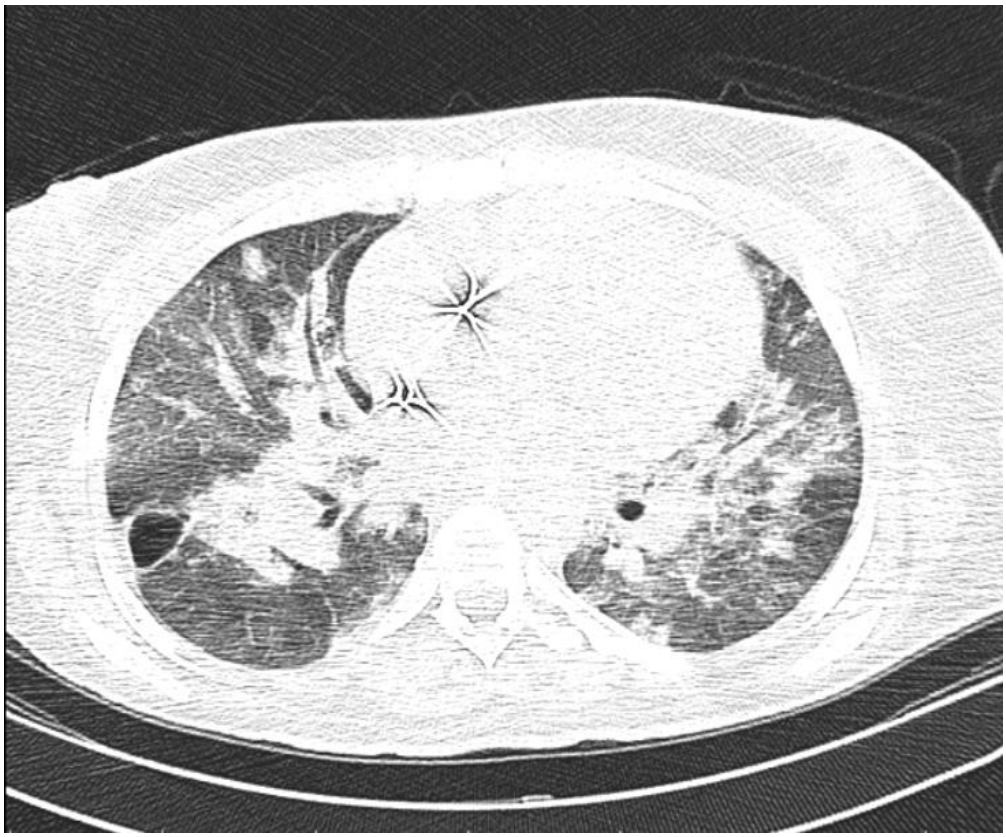

Figure 2: The patient's spiral lung high-resolution computed tomography shows multiple ground-glass opacities, perifissural consolidation at the right upper lobe, and consolidation with cystic bronchiectasis at the posterior and lateral segments of the right lower lobe.

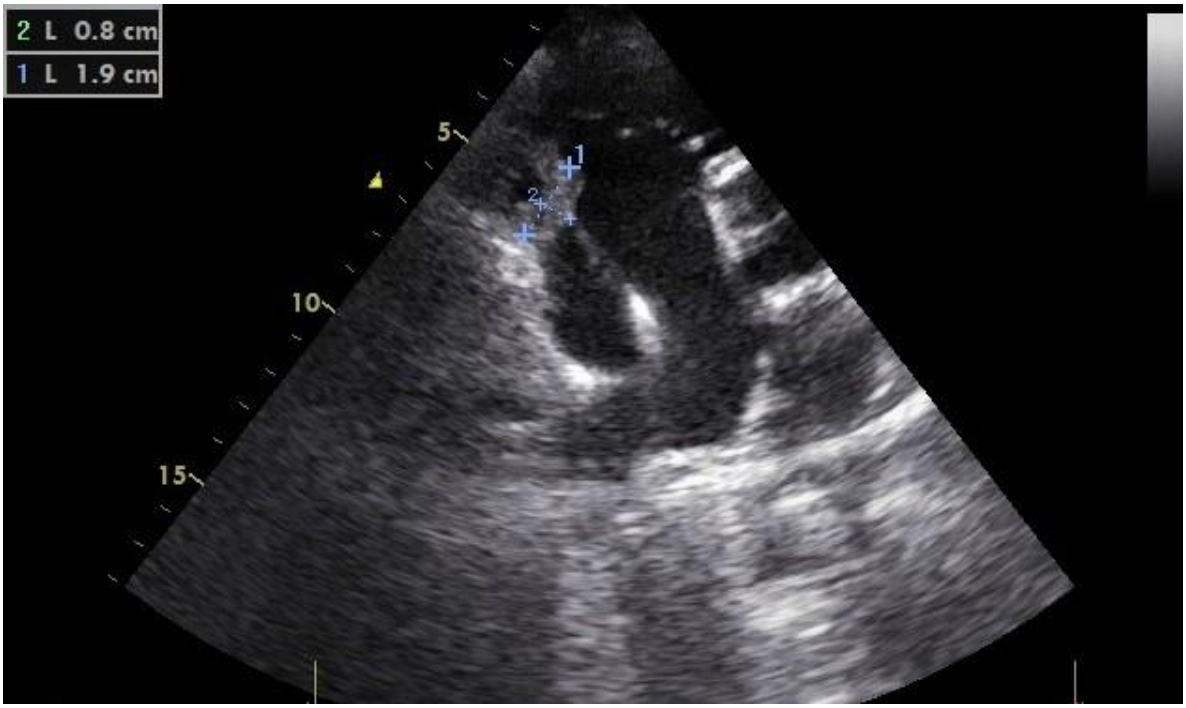

Figure 3: The patient's permanent pacemaker lead can be seen on the right ventricle with no visible clot or mass on it.

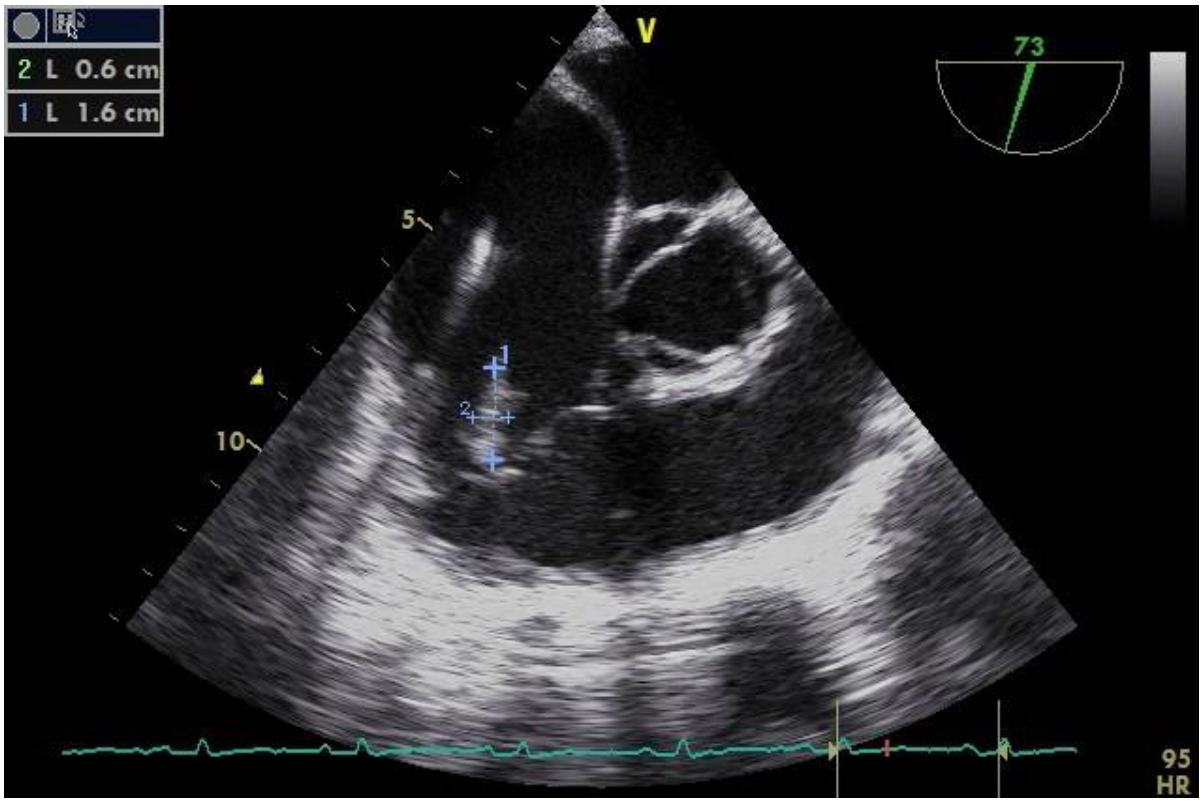

Figure 4: The image shows the perforation of the posterior leaflet by the mass.

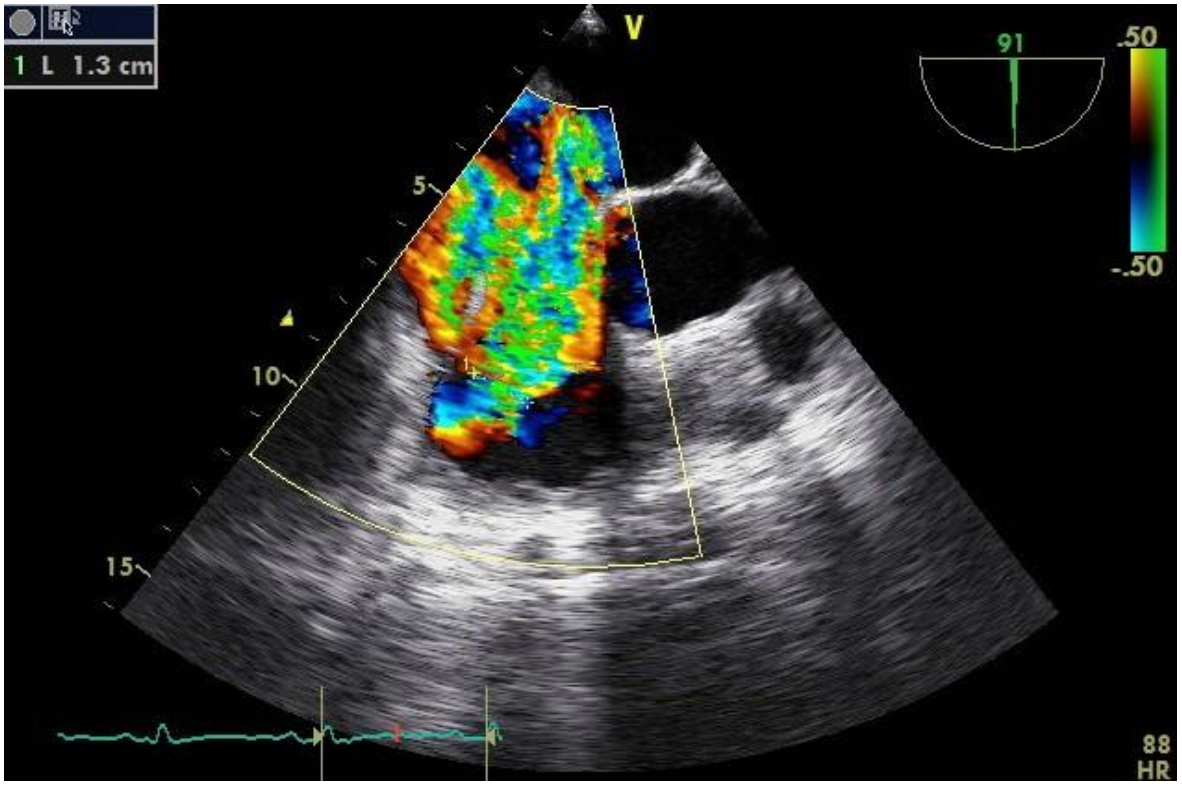

Figure 1: The image shows severe tricuspid regurgitation with a 1.3 cm vena contracta.

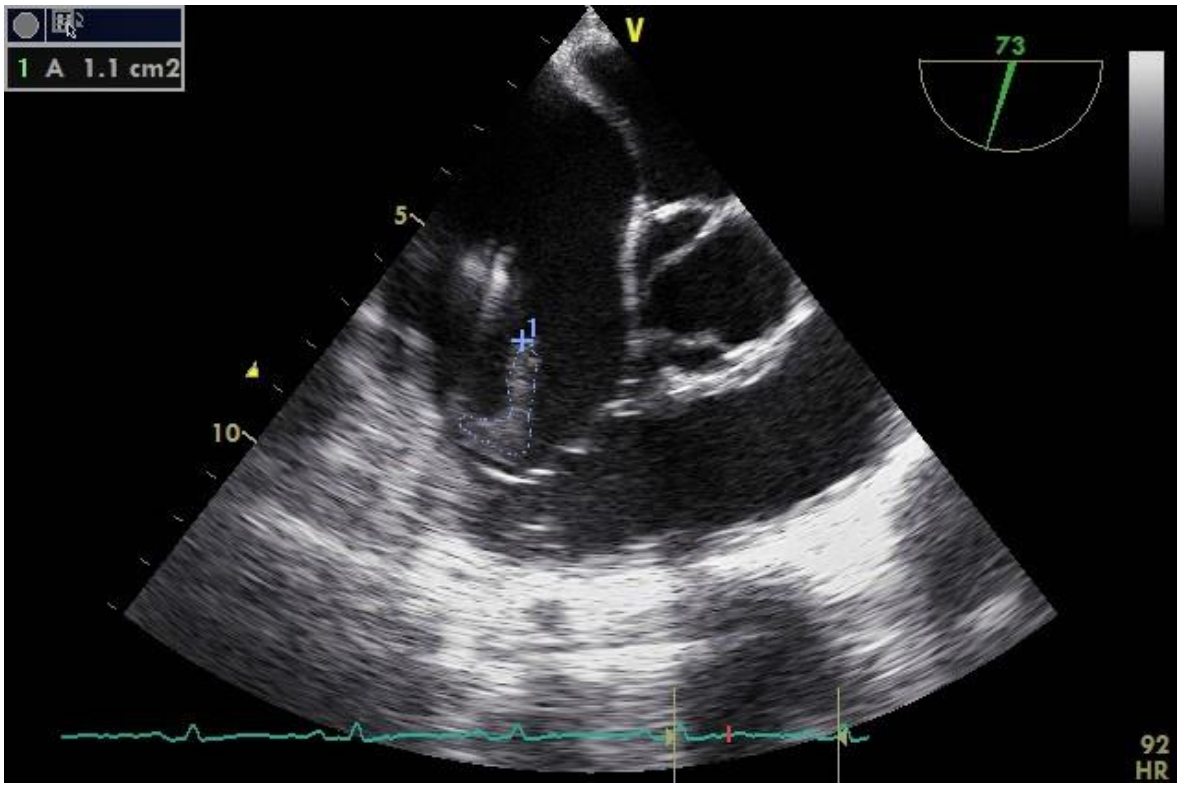

Figure 6: Transesophageal echocardiography shows a noticeable decrease in the size of the mass.

DISCUSSION

This is a report of a SARS-COV-2 interstitial pneumonia case complicated with infective endocarditis in a young adult patient. To date, only a few similar cases have been reported. COVID-19 has resulted in considerable morbidity and mortality worldwide since December 2019. Still, information on cardiac injury in patients affected by COVID-19 is limited.[1]

SARS-CoV-2 infection is associated with interstitial pneumonia and respiratory failure up to acute respiratory distress syndrome, especially in patients with previous cardiovascular diseases.[5] Cardiac injury is a common condition among patients hospitalized with COVID-19, and it is associated with a higher risk of in-hospital mortality.[1] Although the prevalence of COVID-19 in patients with cardiovascular diseases is under-reported, there is evidence that pre-existing cardiac diseases can render individuals vulnerable.[2] In our patient, an underlying cardiac disease (sick sinus syndrome) necessitated PPM implantation a few years earlier.

In COVID-19 patients with high suspicion of infective endocarditis, TEE represents the gold-standard examination for diagnosis.[4] Accordingly, we performed TEE on our patient. According to the Duke clinical criteria,[6] the major criteria by TEE findings, including an intracardiac mass on the tricuspid valve, valve perforation, and new regurgitation, and the minor criteria, including fever and predisposing heart conditions, directed us toward a diagnosis of infective endocarditis. The microorganisms demonstrated by culture in the patient's vegetation confirmed infective endocarditis according to the Duke pathological criteria.

The initial choice of empirical treatment in community-acquired infective endocarditis should cover the most common pathogens, including staphylococci, streptococci, and enterococci. Four intravenous antimicrobial agents, namely amoxicillin, vancomycin, gentamicin, and amphotericin B, are also recommended.[3] We started vancomycin while waiting for the culture results, which were positive for MRSA sensitive to vancomycin. We, therefore, continued vancomycin to reach an acceptable blood level of it.

According to our patient's positive COVID-19 PCR test, we commenced antiviral and anti-inflammatory medications. We also began remdesivir as treatment with remdesivir may prevent the progression of the disease to a more severe respiratory disease. It has been reported that the treatment of such patients with remdesivir is associated with fewer patients needing higher levels of respiratory support.[7] Moreover, we prescribed interferon β-1 simultaneously because our patient was in the early stages of infection. Based on *in vitro* studies, SARS-CoV-2 could be substantially more sensitive to interferon-I than other coronaviruses.[8]

Our case highlights the noticeable cardiovascular manifestations of COVID-19 infection and their management with a heart team, including a cardiac surgeon. During this pandemic, we should be aware of the conditions that are concomitant with COVID-19 infection rather than its complications to decrease patient mismanagement.

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