Case report

A 38-year-old man was taken to the emergency room complaining of chest pain, dyspnea, myalgia, and a significant decline in his general condition. He had a history of hyperthyroidism controlled with thiamazole 5 mg/day and depressive disorder treated with escitalopram 10 mg/day and bupropion 150 mg/day. He had no previous history of infarction, established cardiovascular diseases, or familial sudden death.

The patient reported symptoms of prostration and a dry cough for a few days for which he was tested for severe acute respiratory syndrome coronavirus 2 (Sars-CoV-2) infection at another institution. On admission, the patient was sweating, had cold extremities, and was anxious. His blood pressure was 128/58 mmHg, heart rate was approximately 140 bpm, and peripheral oxygen saturation was 99% on room air. His initial electrocardiogram (Figure 1) revealed an elevated ST-segment in the inferior and lateral wall but no clear evidence of reciprocal changes.

The patient was admitted to the emergency department for continuous monitoring and hemodynamic support measures since he had clinical signs of shock. It was necessary to obtain central venous access and start vasopressors to improve his mean arterial pressure and tissue perfusion. Initial ultrasensitive troponin T test showed results of 0.065 ng/mL and 0.077 ng/mL (reference value, <0.014 ng/mL). Although viral myopericarditis was the probable diagnosis, the patient underwent coronary angiography to rule out the possibility of myocardial infarction, established cardiovascular diseases, or familial sudden death.

Cardiac catheterization showed normal epicardial coronary arteries. The patient was sent to the emergency room, where bedside transthoracic echocardiography showed a left ventricular ejection fraction (LVEF) of 31% with diffuse left ventricular wall hypokinesia. A minor pericardial effusion and a deficit in the inspiratory collapse of the vena cava caused by systemic venous congestion were identified, corroborating the diagnosis of acutely decompensated heart failure.

The patient was transferred to the intensive care unit, where he continued to receive hemodynamic support measures. During the hospitalization, he underwent two chest computed tomography scans (Figure 2). Antibiotics were started due to the possibility of a bacterial infection associated with a viral condition in a patient at risk of unfavorable progression. Initially, ceftriaxone and azithromycin were administered, but the scheme was scaled to piperacillin and tazobactam due to radiological and clinical worsening. Dexamethasone 6 mg/day was administered for 10 days. The patient also developed renal dysfunction and hyperkalemia, which were managed with glucose and insulin and the optimization of his hemodynamic/volemic condition. Since the patient was hemodynamically stable, cardiac magnetic resonance imaging (MRI) was possible, which confirmed the diagnosis of acute viral myopericarditis (Figure 3). MRI showed an LVEF of 18%, late pericardial enhancement near the diaphragmatic face of the heart, increased myocardial signaling in the lateral and posterior walls, and mesocardiac fibrosis in the lateral and posterior walls.

Reverse transcriptase polymerase chain reaction (RT-PCR) findings were positive for Sars-CoV-2 infection. Other serological findings such as hepatitis B, hepatitis C, and Coxsackievirus were negative. As the patient’s condition progressed despite clinical and hemodynamic stability, heart failure therapy was started, initially with bisoprolol and enalapril. At the end of his hospital stay, transthoracic echocardiography showed a significantly improved LVEF (71%), and the patient was discharged from the hospital for outpatient follow-up on bisoprolol 10 mg/day and enalapril 5 mg every 12 hours.

Discussion

The novel coronavirus infection has surprised the entire world with its huge range of presentations, including those affecting the cardiovascular system and ranging from mild to severe. Potential mechanisms of myocardial injury include direct damage due to hemodynamic instability and/or hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction, thrombosis due to hypercoagulability, and systemic inflammation, which can lead to coronary plaque instability.

This report corroborates other studies demonstrating the occurrence of an elevated ST segment in COVID-19 patients, such as the case series published by Bangalore et al., who reported that, of 18 patients with ST elevation, half underwent angiography for suspected infarction, and two-thirds had obstructive disease. This reinforces the challenge of deciding whether to subject the patient to cardiac catheterization in cases of diagnostic uncertainty between obstructive coronary artery disease and differential diagnoses such as viral myocarditis.

Keywords
Cardiogenic shock; Coronavirus infections; COVID-19; Myocarditis.
Echocardiography performed in the emergency room can provide additional information to help guide decisions, such as the presence of regional deficits in myocardial contractility, but such findings are also likely to occur in myocarditis or other conditions (previous infarction, left bundle branch block, pre-excitation due to accessory pathway, and previous cardiac surgery). Most infarctions in Sars-CoV-2 are type 2, but type 1 infarctions can occur and, when suspected, their management is indicated by the guidelines.

As for the use of antibiotic therapy, most coronavirus infection cases are not associated with concomitant bacterial infection, but antibiotics can be used in appropriate cases, especially in patients with associated leukocytosis and/or elevated pro-calcitonin levels, although these findings are not specific and may be elevated in more severe cases.

Serological screening for viruses other than Sars-CoV-2 that can cause myocarditis presents no relevant results and should not be performed routinely; however, it is interesting in certain cases, especially for viruses that cause chronic diseases such as hepatitis C or HIV, as it provides the diagnosis of important public health diseases.

Authors' contributions
Research conception and design: MZF and CPJ; data collection: MZF and DRRR; data analysis and interpretation: MZF, CPJ, and DSS; manuscript writing: MZF and CPJ.

Conflict of interest
The authors have declared that they have no conflict of interest.
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Figure 3 – Cardiac magnetic resonance image. (A) Pericardial effusion and right atrial collapse. (B) Pericardial hypersignaling. (C) Interstitial fibrosis.

References


