Coronavirus Myocarditis: Case Report

Miocardite por Coronavírus: Relato de Caso

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Introduction

The first cases of coronavirus 2019 disease (COVID-19) were described in Wuhan, China, in the end of December 2019.[1] The identified pathogen was called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), an RNA virus of the family Coronaviridae.[2] Due to its rapid spread, the World Health Organization declared COVID-19 a pandemic on March 11, 2020.[3] The main clinical presentation includes respiratory symptoms, such as fever, cough, myalgia, and dyspnea, that can progress to pneumonia or severe acute respiratory syndrome.[4] Among many other disorders, myocardial injury is widely described in the literature, especially associated with influenza and parvovirus B-19, but more clarifications are necessary with SARS-CoV-2 about the pathophysiology involved in the intense viral replication with systemic inflammatory involvement.[5,6]

SARS-CoV-2 is not only a cause of viral pneumonia, it also has important implications for the cardiovascular system, especially in men with risk factors, including older age, diabetes, hypertension, and obesity.[7] Cardiac injury was detected in 19.7% of cases; of the patients who died, 10.6% had coronary heart disease, 4.1% had heart failure, and 5.3% had cerebrovascular disease.[8] High cytokine concentrations are detected in the systemic inflammatory phase of COVID-19, such as interleukin 6 (IL-6), associated with an increase in troponin and other inflammatory biomarkers (D-dimer, ferritin, C-reactive protein, lactic dehydrogenase, procalcitonin, and leukocyte count), causing cardiovascular system injury.[8-10] Of these disorders, myocarditis comprises approximately 7.2% of the cardiovascular complications related to the novel coronavirus.[11] In this phase of disease progression, transthoracic echocardiography should be the method of choice to diagnose and monitor patients, improving the therapeutic management for providing hemodynamic data since patients with ventricular dysfunction are more likely to need mechanical ventilation, which consequently results in a worse prognosis.[3,12]

Keywords

Coronavirus; COVID-19; Echocardiography; Hospitalization; Myocarditis.

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This report presents the case of a patient with myocardial involvement induced by COVID-19 and its clinical and echocardiographic progression during hospitalization. The main objective is to demonstrate the degree of initial inflammatory myocardial impairment and its resolvability after clinical treatment demonstrated by echocardiography.

Case report

A 55-year-old hypertensive and diabetic woman was admitted to the emergency department of the authors' institution on April 10, 2020, reporting oppressive chest discomfort associated with nausea and vomiting that had started 4 hours earlier. The patient reported a 1-week history of a dry cough and runny nose and a fever peak of 38ºC during this period.

On physical examination, she was eupneic, conscious, and oriented, presenting a Glasgow Coma Scale score of 15, no fever and no changes on cardiac and pulmonary auscultation, blood pressure 150 mmHg/110 mmHg, heart rate of 84 beats per minute, wide and symmetrical peripheral pulses, respiratory rate of 18 breaths per minute, no abdominal abnormalities, no lower limb edema, and peripheral oxygen saturation of 98% on room air.

A 12-lead electrocardiogram showed anterolateral ST segment and J point elevation (Figure 1), and the patient was referred for cardiac catheterization, which evidenced the absence of coronary atheromatosis.

A sample for real-time polymerase chain reaction for respiratory virus research collected on April 11, 2020 tested positive for the novel coronavirus. However, despite a peripheral saturation greater than 94%, the patient progressed with dyspnea and required a nasal oxygen catheter at 3 L/min. A chest X-ray showed a slightly increased cardiac area, and the patient was referred for echocardiogram.

On April 14, 2020, the fourth day of hospitalization, she underwent transthoracic echocardiography (Figure 2) according to the updated recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging. The test showed a normal-sized left ventricle with slightly increased diastolic wall thickness (left ventricular diastolic diameter, 45 mm; left ventricular systolic diameter, 38 mm; mass index, 120.21 g/m^2; interventricular septum, 12 mm; and posterior wall, 12 mm) and diffuse hypocontractility, significant systolic dysfunction (ejection fraction, 32.98%), and diastolic dysfunction due to impaired relaxation. The right ventricle had a normal cavity and reduced systolic function (tricuspid annular plane systolic excursion [TAPSE], 16 mm). The right and left atria presented cavities with normal volume. The mitral, aortic, tricuspid,

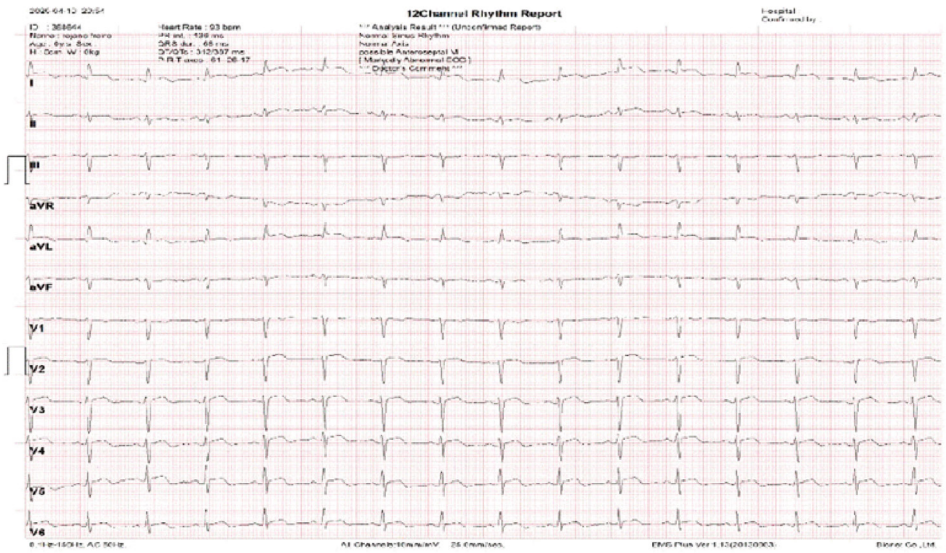

Figure 1 – *Admission electrocardiogram.*

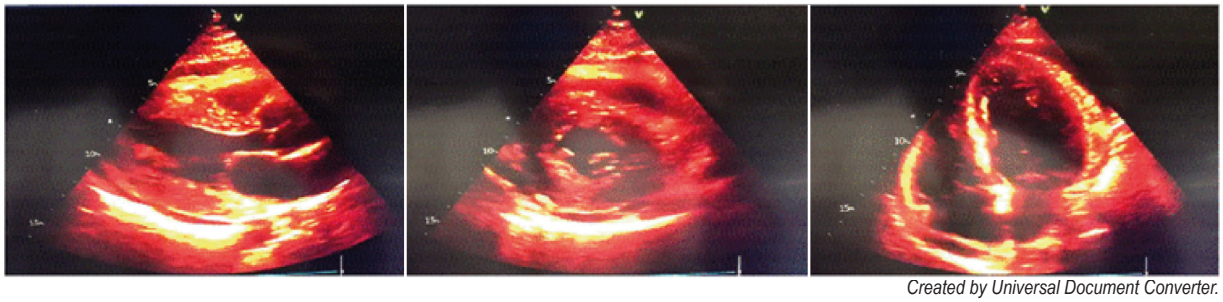

Figure 2 – *Initial transthoracic echocardiogram.*

and pulmonary valves were functionally and morphologically normal. A mild pericardial effusion was also observed (without tamponade signs).

Considering the clinical and laboratory context, the patient underwent the following therapeutic regimen: chloroquine (400 mg twice a day on D1 and 400 mg daily from D2 to D5) associated with azithromycin (500 mg daily from D1 to D5) in addition to acetylsalicylic acid 500 mg four times a day. On April 15, 2020, the patient's condition worsened to include a cough and purulent sputum, and Tazocin® was started on April 16, 2020. Figure 3 shows the chest X-ray performed on April 17, 2020, with good clinical and laboratory response (Table 1). During the use of chloroquine and azithromycin, the corrected QT interval ranged from 387 ms (April 10) to 437 ms (April 17), returning to 367 ms (April 20).

Before hospital discharge, the patient underwent a repeat control echocardiogram on May 11, 2020, which showed a normal left ventricular cavity with normal wall thickness and contractility, normal systolic and diastolic function (left ventricular diastolic diameter, 47 mm; left ventricular systolic diameter, 28 mm; interventricular septum, 8 mm; posterior wall, 8 mm; and ejection fraction, 61%; global longitudinal strain, 20%; no pericardial effusion), right ventricle with normal thickness and normal systolic function recovery (TAPSE, 31 mm) (Figure 4). Table 2 shows the changed initial echocardiogram measurements compared to controls.

Discussion

Cardiovascular system injuries are probably multifactorial and may be caused by direct cardiac damage by the virus or by systemic inflammation and thrombosis, causing an imbalance between high metabolic demand and low reserves.[12] Myocarditis can be associated with acute heart failure in COVID-19 patients, such as fulminant myocarditis, with rapid progression and severe ventricular dysfunction associated with diffuse myocardial edema.[12] Therefore, some mechanisms have been proposed to explain the pathophysiology of

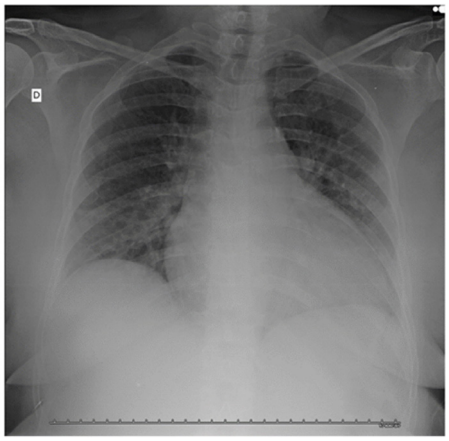

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Figure 3 – *Chest radiograph taken during hospitalization.*

Table 1 – Laboratory tests during hospitalization.

	RV	April 11, 2020	April 14, 2020	April 17, 2020	April 20, 2020
Troponin, ng/mL	< 0.014	0.975	1.080	0.982	0.141
Creatinine, mg/dL	0.5-0.9	0.94	0.66	0.91	0.86
Urea, mg/dL	< 50	60.9	46.2	38.6	34.8
Sodium, mmol/L	136-145	139.1	124.6	138.1	135.4
Potassium, mmol/L	3.5-5.1	4.32	4.57	4.19	5.59
Chlorine, mmol/L	98-107	103.2	93.0	102.7	101
Total calcium, mg/dL	8.6-10.2				9.88
Albumin, g/dL	3.4-4.8				4.18
Lactic dehydrogenase, U/L	135-225	-	513.98	338.13	-
Oxaloacetic transaminase, U/L	10-35	105.71	167.61	43.3	31.85
Pyruvic transaminase, U/L	10-35	125.0	325.0	200.82	110.95
C-reactive protein, mg/L	< 5	7.29	40.62	10.13	3.48
Hemoglobin, g/dL	14-17	11.5	10.5	10.7	12
Hematocrit, %	40-54	34	31	32.5	36.3
Leukocyte, UL	3.600-11.000	5.290	6.830	5.860	5.511
Lymphocyte, %	20-50	25.1	34.6	35.5	33.8
Platelets, UL	150.000-450.000	270.000	289.000	322.000	372.000

RV, reference value.

myocarditis caused by the novel coronavirus. One form of aggression would be direct cell damage when the virus enters the cell by binding to angiotensin II enzyme receptors found in myocytes. Another mechanism would be via infected antigen-presenting cells, which activate CD8 T cells that would migrate to myocytes due to a hepatocyte growth factor cardiac tropism, causing cytotoxic inflammation. This inflammation could also be amplified by the cytokine storm syndrome, with IL-6 being its main mediator.[13] Patients with myocardial injury had a higher rate of intensive care hospitalization than those without it (22.2% versus 2.0%), progressing with a higher incidence of heart failure (52% versus 12%) and, consequently, a higher mortality rate (59% versus 1%).[14,15]

In this case, echocardiographic acute myocardial impairment changes caused by the novel coronavirus are relevant. Older patients are the most vulnerable to complications, as are those with cardiovascular diseases, diabetes, and obesity. According to data from the Brazilian Society of Cardiology, arrhythmias (16%), myocardial ischemia (10%), myocarditis (7.2%), and shock (1–2%) are some of the cardiological complications related to COVID-19.[12] Patients with cardiovascular risk factors (older age, hypertension, and diabetes), coronary artery disease, cardiomyopathies, and cerebrovascular diseases are more susceptible to developing the severe form of the disease, being classified as a risk group for COVID-19 complications.[11]

Transthoracic echocardiography proved to be a useful tool for evaluating the cardiac function of these patients, identifying systolic and/or diastolic left ventricular dysfunction and, most importantly, presenting relevant hemodynamic data, which is important to its clinical management. The echocardiogram can be used daily, or when necessary, as a tool for monitoring

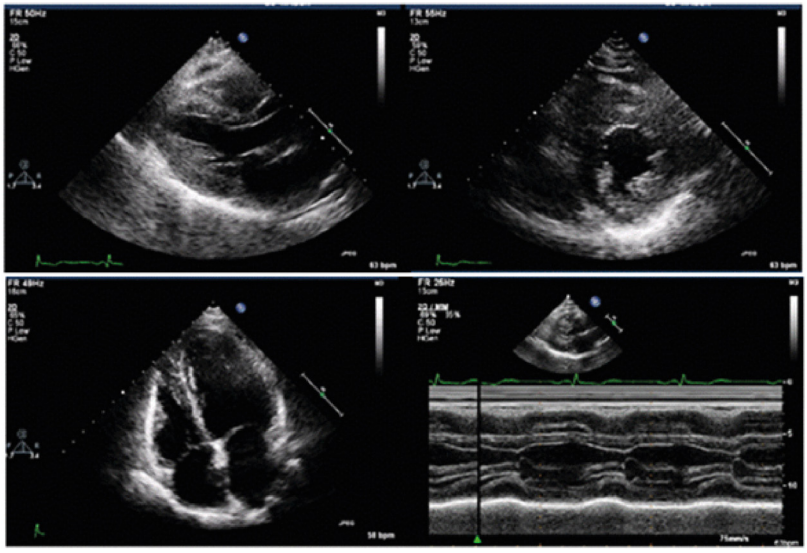

Figure 4 – *Second transthoracic echocardiogram.*

Table 2 – Comparison of the changed echocardiogram measurements during myocarditis and after its resolution.

	Echocardiogram April 14, 2020	Echocardiogram May 11, 2020
Interventricular septum, mm	12	8
Posterior wall, mm	12	8
Left ventricular diastolic diameter, mm	45	47
Left ventricular systolic diameter, mm	38	28
Fractional shortening, %	15.55	40.42
Ejection fraction, %	32.98	61
Left ventricular mass, g	197.5	121.66
Tricuspid annular plane systolic excursion, mm	16	31

hemodynamic parameters in critical patients, guiding their treatment with inotropic and/or circulatory support. In addition to the diagnosis, patients with ventricular dysfunction have a worse prognosis.[14]

Conclusion

This case report presented a form of myocarditis with diffuse myocardial involvement, transient left ventricular wall thickening, important initial impairment of the systolic function, and normalization of the left ventricular wall thickening and systolic function after approximately 1 month of treatment.

Authors' contributions

Research concept and design: Rocha AFB, Barros JLA, Canejo Sá M, Longo ACMS, Monteiro Júnior JGM, Silveira CAM; data collection: Rocha AFB, Barros JLA, Canejo Sá M, Longo ACMS, Monteiro Júnior JGM, Silveira CAM; manuscript writing: Rocha AFB, Monteiro Junior JGM, Silveira CAM, Del Castillo JM.; critical review of the manuscript for important intellectual content: Rocha AFB, Barros JLA, Canejo Sá M, Longo ACMS, Monteiro Júnior JGM, Silveira CAM; data analysis and interpretation: Rocha AFB, Barros JLA, Canejo Sá M, Monteiro Júnior JGM, Silveira CAM.

Conflict of interest

The authors have declared that they have no conflict of interest.

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