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CASE REPORT

Cardiac Manifestations Associated With Coronavirus Disease: A Case Report

Gabriela Ribeiro Prata Leite Barros, Raphaela Tereza Brigolin Garofo, Bernardo Noya Alves de Abreu, Adriano Camargo de Castro Carneiro, André Luiz Esteves Mendonça

Hospital do Coração, São Paulo, SP – Brazil

Abstract

This study presents a 47-year-old female patient, with a history of diabetes, who contracted SARS-CoV-2 and exhibited cardiovascular complications.

Methods

This case report information was obtained by analyzing medical records; the data was acquired by the medical team, recording images of complementary exams and reviewing the literature.

Introduction

Coronavirus disease 2019 (COVID-19) has been declared a worldwide public health emergency. According to data from the Brazilian Society of Cardiology (SBC), cardiovascular involvement related to the new coronavirus can manifest itself as arrhythmias (16%), myocardial ischemia (10%), myocarditis (7.2%), and shock (1-2%).[1] A study published in the Journal of the American Medical Association (JAMA), conducted in 02 North American hospitals, shows that cases of takotsubo increased during the COVID-19 outbreak. Before the pandemic, approximately 5 to 12 patients presented the takotsubo syndrome upon hospital admission; after an increase in SARS-CoV-2 cases, the number of patients with takotsubo reached 20.[2]

Keywords

Cardiovascular Diseases/complications; SARS-CoV-2, Betacoronavirus; Pandemics; Arrhythmia Cardiac; Myocardial Ischemia; Myocarditis; Takotsubo Cardiomyopathy.

Case Report

A 47-year-old female patient, from São Paulo, with controlled diabetes, sought out the emergency department of a tertiary hospital, reporting that she was admitted seven days earlier with a diagnosis of COVID-19 and myocarditis.

She returned reporting sudden, severe, tight chest pain, unrelated to physical exertion, associated with sweating and dyspnea. Upon initial assessment and physical examination, the patient was considered to be in good general condition and eupneic; the cardiac auscultation was rhythmic, with normal heart sounds; and no murmurs were observed. In pulmonary auscultation, a rhonchi in the right hemithorax was audible. The patient had a blood pressure of 170 x 100 mmHg, a heart rate of 53 bpm, and blood saturation of 96% in ambient air.

The electrocardiogram (EKG) performed upon hospital admission revealed a 1st degree atrio-ventricular block (AVB) (Figure 1). Laboratory tests showed an increase in troponin 0.99 (normal range (NR) < 0.034), NT pro-BNP 2470 (NR<100), and D-Dimer 1165 (NR < 500).

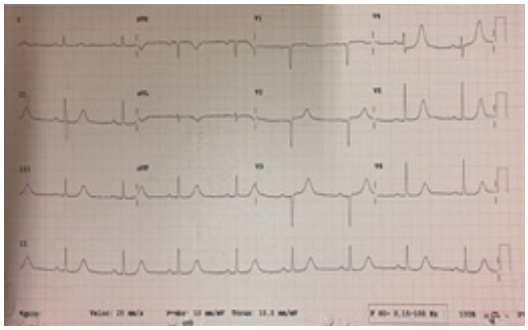

Figure 1 – EKG: 1st degree atrio-ventricular block.

Mailing Address: Gabriela Barros
R. Des. Eliseu Guilherme, 147 – Paraíso. Postal Code: 04004-030, São Paulo, SP – Brazil.
E-mail: dragabrielaprata@gmail.com

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A triple-rule-out computed tomography (CT) of the chest (Figure 2) revealed ground glass opacity, associated with thickening of the interlobular septal, together with signs of pulmonary congestion in the peribronchovascular interstitium; therefore, inflammatory/infectious processes could not be ruled out. The CT exam of the coronary and pulmonary arteries showed an absence of coronary calcifications and atherosclerotic luminal reduction (Figures 3 and 4). The presence of segmental systolic dysfunction of the left ventricle proved not to be related to the coronary territory. The existence of a non-ischemic pattern of myocardial fibrosis with lipomatous metaplasia was also found, suggesting a previous myocarditis (Figure 5), with no signs of pulmonary thromboembolism (Figure 6). The pattern of left ventricular segmental contractility found in the CT exam of the heart could suggest stress-induced cardiomyopathy (Takotsubo syndrome), since the changes in the myocardial fibrosis were not suggestive of an acute condition (lipomatous metaplasia) (Figure 7).

Patient was hospitalized with broad-spectrum antibiotic therapy that was initiated due to recent hospitalization. Diuretic therapy, angiotensin-converting enzyme inhibitors (ACE inhibitors), and beta-blockers were also prescribed, followed by complementary investigation.

Cardiac magnetic resonance (CMR) showed moderate left ventricular systolic dysfunction (ejection fraction of 41%) and an aspect compatible with previous myocarditis without signs of current inflammatory process. There were also changes in contractility of the left side ventricle (LV), with anterior and mediobasal akinesia with localized feature, preserving the apex and more basal portions, which did not allow us to rule out adrenergic cardiomyopathy, Takotsubo syndrome (Figure 8).

The patient also presented frequent ventricular extrasystoles, solved using beta-blockers. After improvements in the condition, the patient was discharged, using ACE inhibitors, beta-blockers, and loop diuretics.

After 45 days, the patient remained asymptomatic and underwent CMR (Figure 9), which showed preserved biventricular systolic function and the presence of non-ischemic inferior and inferobasal fibrosis, although nonspecific, which could correspond to previous old inflammatory cardiomyopathy. Compared to the first CMR, there was an improvement in the left ventricular segmental contractility. The patient also underwent a new chest CT scan that showed a resolution of the previous pulmonary condition (Figure 10).

This case report aims to demonstrate the association between COVID-19 and cardiac involvement, most likely causing takotsubo cardiomyopathy.

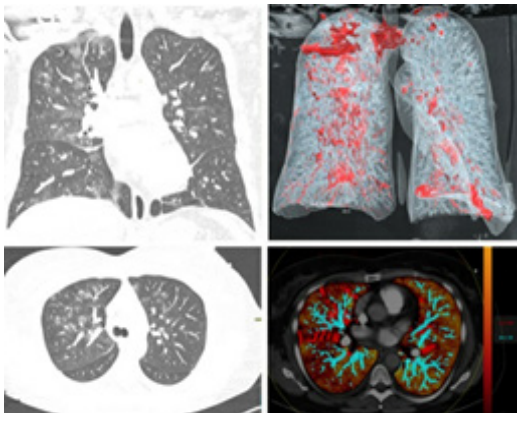
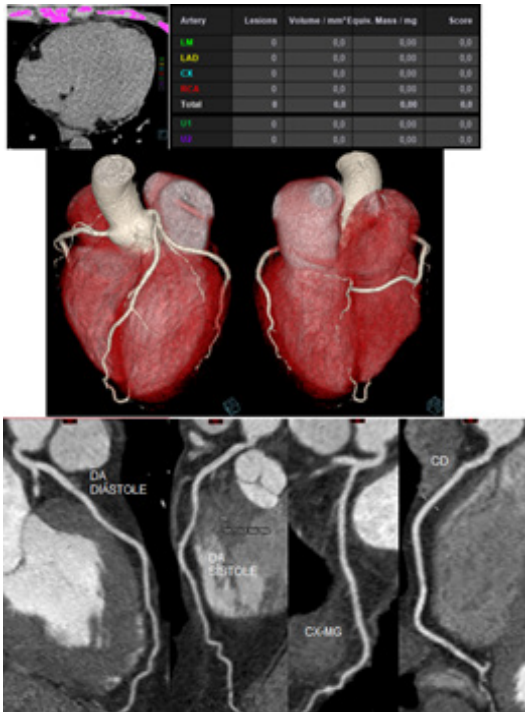


Figure 2 – Chest CT showing ground glass opacity associated with thickening of the interlobular septal and signs of pulmonary congestion.



Figures 3 and 4 – Coronary CT scan with 3D reconstruction showing absence of lesions.

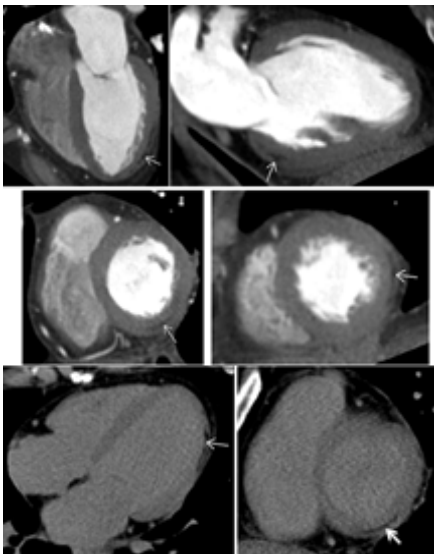


Figure 5 – Segmental systolic dysfunction of the left ventricle that suggested a previous myocarditis.

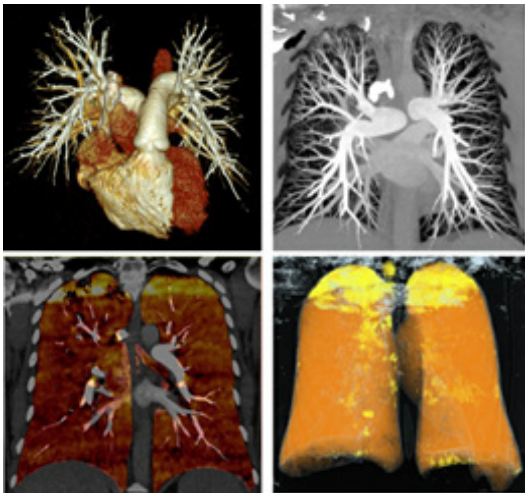


Figure 6 – Absence of pulmonary thromboembolism

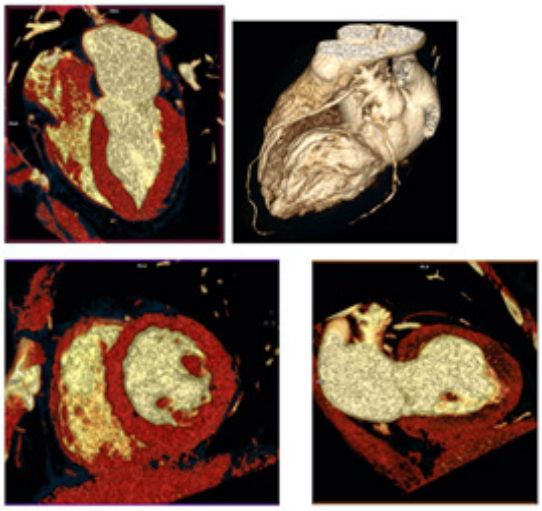


Figure 7 – CT of the heart could suggest stress-induced cardiomyopathy.

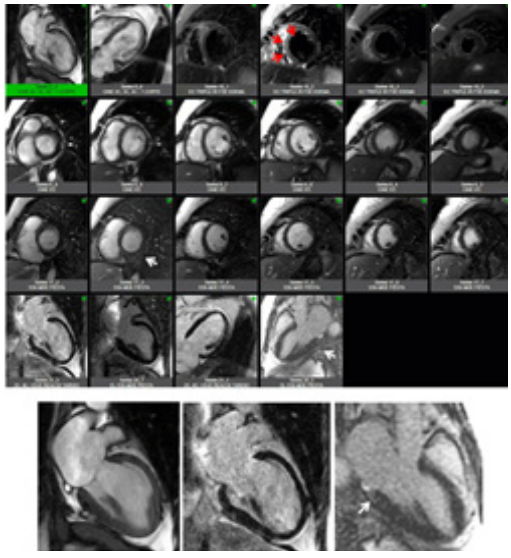


Figure 8 – CMR: anterior and mediobasal akinesia with localized feature, preserving the apex and more basal portions.

Discussion

Myocarditis and COVID- 19

Since December 2019, a type of coronavirus (SARS-CoV-2) is responsible for the current pandemic, known as COVID-19. Among the complications related to this type of virus, we can include heart attack. Thus, like some previously mentioned viruses, SARS-CoV-2 is believed to be related to cases of myocarditis.[3]

The pathophysiology is still unknown. SARS-CoV-2 infection is caused by binding the spike protein from the viral surface to the receptor of the angiotensin-converting enzyme 2 (ECA-2) after the activation of the spike protein by the transmembrane

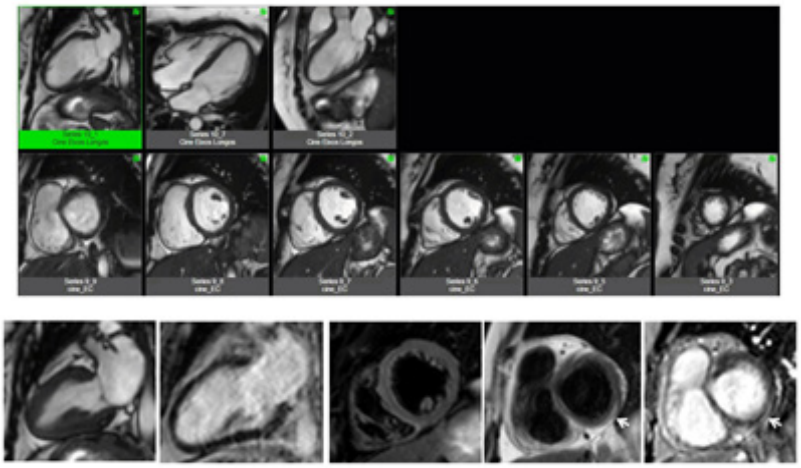


Figure 9 – CMR showing evolutionary improvement of left ventricular segmental contractility.

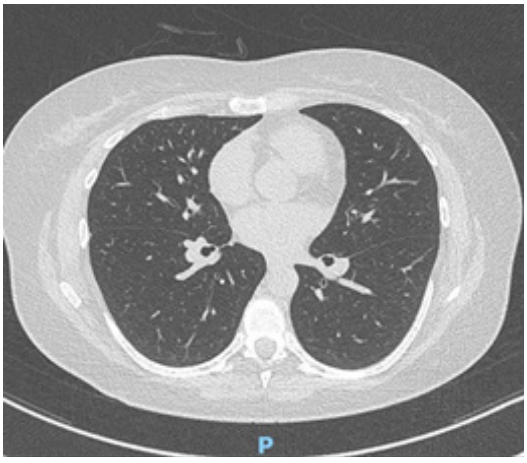


Figure 10 – Chest CT showing resolution of the pulmonary condition.

protease serine 2 (TMPRSS2). ECA-2 is found in several organs, like the heart, in which SARS-CoV-2 causes the deregulation of ECA, causing direct myocardial injury. Another hypothetical mechanism of direct myocardial viral lesion is mediated vasculitis through infection.[4]

The clinical presentation of myocarditis caused by COVID-19 is broad, ranging from fatigue and mild dyspnea to fulminant myocarditis. Patients usually have high levels of troponin and ECG abnormalities. AHA recommends doing at least 01 imaging exam, in the face of suspected myocarditis, such as an echocardiogram, which is more available and practical, or a cardiac magnetic resonance imaging. It is important to emphasize that if coronary angiography is indicated, endomyocardial biopsy (gold standard for diagnosis of myocarditis) can be added without adding much time or increasing the risk of viral dissemination. The initial treatment of fulminant myocarditis should follow the cardiogenic shock protocol, which includes the use of inotropes or vasopressors and mechanical ventilation. Arrhythmias can be managed by temporary cardiac stimulation or antiarrhythmic medications. Then, depending on the severity, the patient may need mechanical circulatory support. Precautions should be taken for the use of NSAIDs and drugs that prolong QTc in patients with COVID-19, because these drugs can exacerbate cardiac symptoms.[5]

Takotsubo and COVID-19

Takotsubo syndrome (TTS) or stress-induced cardiomyopathy is characterized by transient regional systolic dysfunction of the left ventricle and alterations in the electrocardiogram that imitate acute myocardial infarction in the absence of angiographic evidence of coronary obstruction. [6] TTS is responsible for 1.8-2.2% of all acute coronary syndromes. There are suggestions that TTS may be associated with COVID-19.[7] Myocardial injury is relatively common in patients with COVID-19, representing 7% to 23%

of cases, and it is associated with a higher morbidity and mortality rates.[8]

Conclusion

The cardiovascular outcome is a real possibility in the clinical experience of the new coronavirus pandemic. The clinical signs should always guide us to put forward such possibilities, thus continuing the alert for myocarditis and Takotsubo syndrome. The patient of this case report presented myocardial injury both at an early stage of the disease and at a later stage. Therefore, cardiovigilance is recommended in these patients, since, in view of the current knowledge, it is not clear if myocardial dysfunction or other complications may occur at a later point in time.

Author contributions

Conception and design of the research: Barros GRPL, Garofo RTB, Mendonça ALE, Abreu BNA. Acquisition of data: Barros GRPL, Garofo RTB, Mendonça ALE, Carneiro ACC, Abreu BNA. Analysis and interpretation of the data: Barros GRPL, Garofo RTB, Mendonça ALE, Carneiro ACC, Abreu BNA. Writing of the manuscript: Barros GRPL, Garofo RTB, Mendonça ALE, Abreu BNA. Critical revision of the manuscript for intellectual content: Barros GRPL, Garofo RTB, Mendonça ALE, Abreu BNA.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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