European Journal of Case Reports in Internal Medicine

COVID-19 Myopericarditis in a Young Healthy Male

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Doi: 10.12890/2022_003361- European Journal of Case Reports in Internal Medicine - © EFIM 2022

Received: 16/04/2022
Accepted: 07/05/2022
Published: 23/05/2022

How to cite this article: Bandeira M, Serrazina J, Santos CA, Jacinto N, Pais de Lacerda A. COVID-19 myopericarditis in a young healthy male. *EJCRIM* 2022; **9**:doi:10.12890/2022_003361.

Conflicts of Interests: The Authors declare that there are no competing interest

ABSTRACT

SARS-CoV-2 infection can present in different clinical forms, most commonly as bilateral pneumonia, but also with pericardial/myocardial involvement. Cardiac involvement in COVID-19 is associated with worse outcomes.

The authors report a case of myopericarditis as the primary manifestation of SARS-CoV-2 infection in a 20-year-old male patient with no known cardiovascular (CV) disorders or risk factors.

The patient presented with pleuritic chest pain and high fever, with no respiratory symptoms. Electrocardiogram (ECG) and echocardiogram changes were consistent with pericarditis; concomitant elevation of cardiac enzymes revealed myocardial involvement. The patient had a slow but favourable evolution with no apparent impact on cardiac function. Other causes of myopericarditis were excluded and SARS-CoV-2 admitted as the most likely aetiological agent.

This case highlights possible cardiac involvement in SARS-CoV-2 infection with little or no pulmonary disease in a young healthy patient. Such systemic and potentially troublesome manifestations of COVID-19 are increasingly being described.

KEYWORDS

Myopericarditis, COVID-19, SARS-CoV-2

LEARNING POINTS

- Acute myopericarditis is a possible manifestation of SARS-CoV-2 infection.
- SARS-CoV-2 cardiac involvement may occur both in older and in younger previously healthy subjects, and could be more frequent than expected.
- Further investigation should address the prevalence of myocardium and pericardium involvement in COVID-19 patients, as well as its complications, sequelae and prognostic value for both older and young patients.

INTRODUCTION

Coronavirus disease 2019 (COVID-19) most frequently affects the lungs and can cause acute respiratory failure, although some patients are asymptomatic. SARS-CoV-2 binds to its receptor, angiotensin-converting-enzyme 2 (ACE2), which is highly expressed in the lungs, and also in the heart and kidneys [1, 2].

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Myopericarditis is sometimes seen in clinical practice as acute pericarditis and myocarditis share similar aetiologies, in particular cardiotropic viruses [3]. Diagnosis usually relies on evidence of pericarditis associated with markers of myocardial injury.

COVID-19-induced cardiac injury is a negative prognostic factor. The mechanisms responsible for cardiac involvement are not yet fully understood [4], but may include direct viral injury, systemic inflammation, plaque rupture or treatment side-effects [5, 6]. The absence of significant mononuclear infiltrates or other tissue lesions in histopathological reports suggests little direct viral involvement in myocardial injury [7].

CASE DESCRIPTION

A 20-year-old man presented to the emergency room with a 6-day history of myalgia, a high fever (39.5°C) and dysgeusia. His symptoms had recently worsened with retrosternal pain aggravated by inspiration. His previous medical history only included psoriasis, which was controlled with topical corticosteroids.

A PCR test was positive for SARS-CoV-2. Laboratory tests on admission showed a leukocyte count of 11.61×10^9 cells/l (4–11), neutrophils 8.37×10^9 cells/l and lymphocytes 2.08×10^9 cells/l, D-dimers 0.28 µg/ml (0–0.25) and elevated inflammatory parameters such as fibrinogen 770 mg/dl (200–400), ferritin 662 ng/ml (30–400) and C-reactive protein 28.3 mg/dl (<0.5), with an unremarkable chest x-ray. ECG showed PR-segment depressions on multiple leads (*Fig. 1*). Further blood work revealed troponin-T 144 ng/l (<14) and CK-MB 42.4 ng/ml (4.9–6.2), suggesting concomitant myocardial injury and the diagnosis of acute myopericarditis [8].

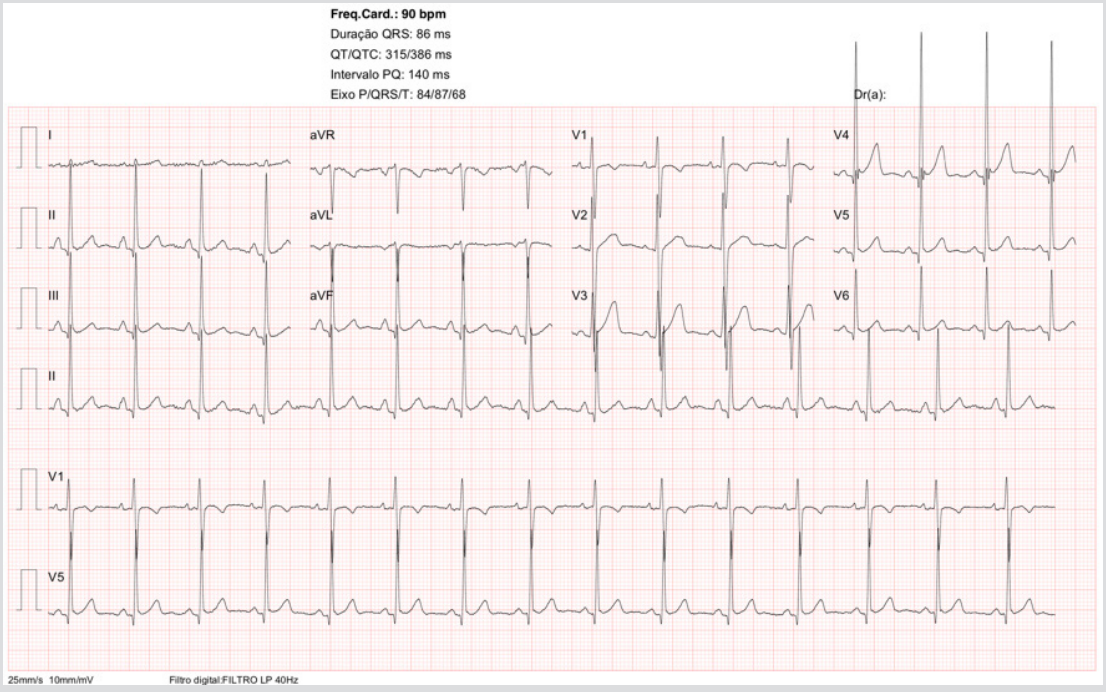

Figure 1. Admission ECG

The patient was admitted to a COVID-19 ward. Pericardium hyperechogenicity and a small right atrium pericardial effusion on echocardiogram were consistent with acute pericarditis.

Ibuprofen 600 mg three times a day and colchicine 0.5 mg once a day were added to his COVID-19 treatment protocol (lopinavir/ritonavir 400 mg/100 mg plus hydroxychloroquine 200 mg twice a day). Other common causes of myopericarditis were excluded, such as enteroviruses, herpesviruses, adenovirus and parvovirus. Bacterial cultures were negative as well as test results for *Mycobacterium tuberculosis*, *Pneumococcus sp.*, *Legionella pneumophila*, *Mycoplasma* and *Chlamydia pneumoniae*. Toxoplasmosis, brucellosis, hepatitis B and C, syphilis, HIV and autoimmune causes were likewise excluded.

The patient maintained high and frequent fever spikes, chest pain and elevated troponin-T rising to a maximum of 690 ng/l (<14) on day 4. Serial ECGs showed normalization of PR-segment depression and the presence of nearly-diffuse, concave-upwards ST-segment elevation (*Fig. 2*), consistent with pericarditis [9].

He later improved and was discharged asymptomatic after 10 days, with normal levels of cardiac enzymes and an echocardiogram showing pericardium thickening with no effusion. The ECG changes did not revert completely, showing a diffuse sustained ST-segment elevation.

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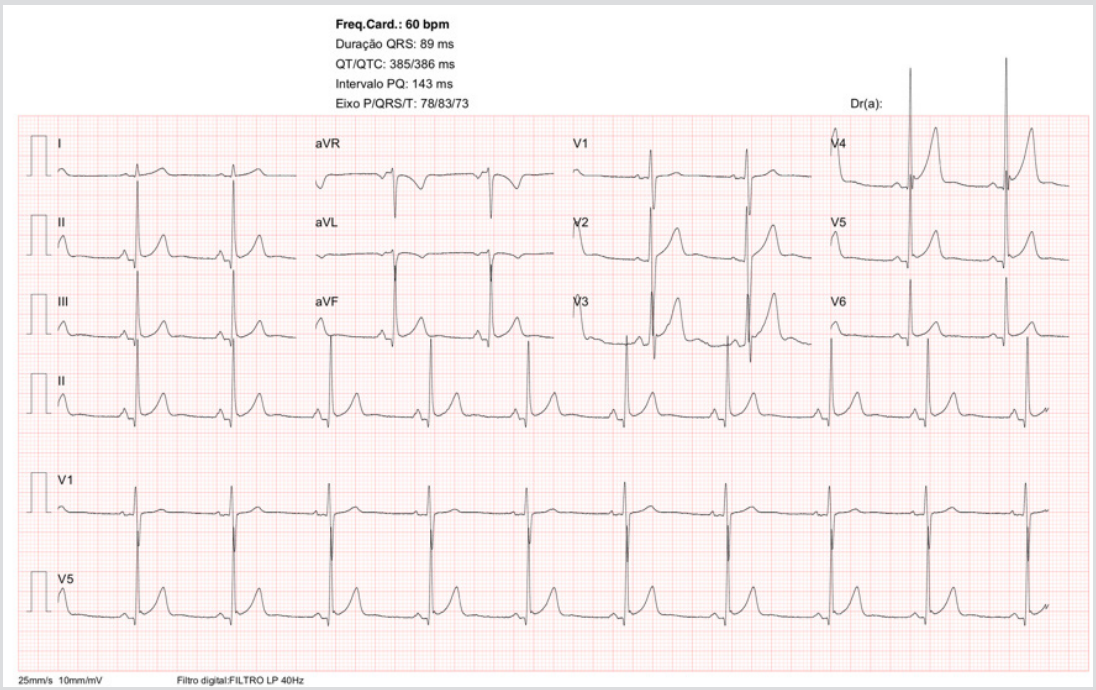

Figure 2. Discharge ECG

Four weeks after admission, the patient underwent cardiac MRI (*Fig. 3*) which showed late subepicardial enhancement on the lateral-inferior and median wall, consistent with acute myocarditis with no effect on ventricular function (ejection fraction 63.2%). It also demonstrated a small pericardial effusion, with no signs of current inflammation. The patient showed normalization of ECG and echocardiogram changes after 6 months.

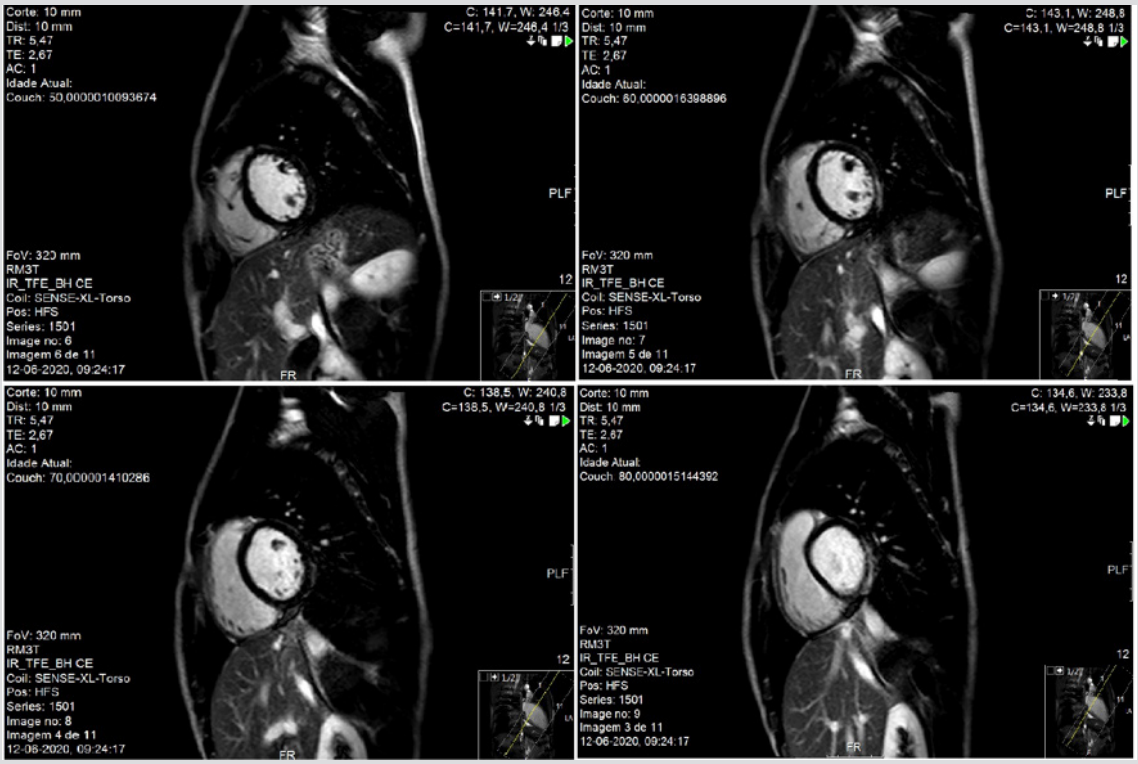

Figure 3. Cardiac MRI

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DISCUSSION

Uncommonly reported as manifestations of SARS-CoV-2 infection, myocardial and pericardial involvement should be considered by the healthcare professional when ECG abnormalities are seen in a COVID-19 patient [9].

The diagnosis of pericarditis was made as three out of four criteria were met; pericardial rub was missing. Myocardial involvement was evidenced by the significant elevation of myocardial enzymes [8]. Delayed MRI may be a limitation for detecting more typical findings.

Pericarditis has been reported during the pandemic [10]. A recent systematic review included 34 patients with COVID-19 of whom 21 had concomitant myocardial involvement [11]. These patients were mostly male with a mean age of 53 years. Comorbidities were common, particularly hypertension, and the majority (63%) displayed abnormalities in lung parenchyma imaging. We describe significant cardiac involvement in a young healthy man with no history of cardiovascular disease who responded slowly to both non-steroidal anti-inflammatory drugs and colchicine. The patient reported no respiratory symptoms and there were no abnormalities on chest imaging, in contrast to most cases of myopericarditis reported in the literature.

In addition to conveying a higher mortality rate, underlying CV disease has also proved to be the most common comorbidity affecting patients hospitalized for COVID-19 [5]. Interestingly, patients with underlying CV disease with normal troponin had a better prognosis than patients without CV who showed elevated troponin levels [12]; however, CV involvement in COVID-19 is mainly reported in older patients with multiple comorbidities.

Myocardial involvement in acute pericarditis with the classic aetiologies has no effect on prognosis [13]. However, in COVID-19, elevated troponin levels were correlated with more severe outcomes [4].

The imbalanced ACE/ACE2 ratio seen in psoriasis, with higher ACE tissue concentrations and activity as well as a tendency for higher ACE serum levels, correlates with higher CV comorbidity [14, 15]. On the other hand, binding of the virus to ACE2 can result in ACE2 downregulation, which could lead to excessive production of angiotensin by the ACE enzyme, the opposing physiological homologue of ACE2. This apparent interaction between these two diseases could have made this particular COVID-19 patient more susceptible to cardiac involvement [16].

CONCLUSION

This case highlights cardiac involvement in SARS-CoV-2 infection with no lung affects. Physicians should include COVID-19 in the differential diagnosis of patients presenting with myopericarditis. Prompt diagnosis and therapy may improve prognosis.

Further studies should address the prevalence and prognostic relevance of cardiac involvement in young patients with COVID-19, who are usually expected to have a mild course of disease.

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