

Late Presentation of COVID-19-Associated Transmural Myocardial Infarction with Non-Obstructive Coronary Atherosclerosis

Apresentação Tardia de Infarto Agudo do Miocárdio Transmural Sem Obstrução Coronariana Aterosclerótica Associado à COVID-19

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Introduction

There has been an increased incidence of coronavirus-associated thromboembolic events during the coronavirus disease 2019 (COVID-19) pandemic.¹ However, most cases presented with venous thrombotic events, mainly deep vein thrombosis and pulmonary thromboembolism,² whereas arterial and coronary phenomena are rare.³

Several pathophysiological factors can explain the relationship between COVID-19 infection and increased embolic phenomena. The main factors are increased inflammatory mediators, the release of prothrombotic factors, and endothelial dysfunction.^{4,5}

Cardiovascular presentations are also common in patients with COVID-19, such as atypical chest pain, dyspnea, arrhythmias, and myocardial injury with increased troponin levels.⁶ However, chest pain and increased troponin levels in the absence of coronary obstruction are rare and restricted to a few case reports.^{7,8} In addition, cardiovascular signs and symptoms associated with coronavirus infection occur in the acute stage of the disease with a symptomatic condition. Late presentation after complete symptom resolution is rarely reported.

Objective

Here we describe the case of a patient with the late presentation of chest pain associated with increased myocardial injury biomarkers after hospital discharge from a COVID-19 admission. The patient underwent coronary cineangiography and cardiac magnetic resonance imaging (MRI), and the syndromic diagnosis was myocardial infarction with non-obstructive coronary atherosclerosis (MINOCA). These results highlight the possibility of cardiovascular presentations in a subacute COVID-19 infection context.

Keywords

Myocardial Infarction; Myocarditis; Coronavirus; COVID-19; Biomarkers.

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Manuscript received 2/8/2022; revised 3/10/2022; accepted 5/2/2022

DOI: 10.47593/2675-312X/20223502eabc290

Clinical case description

A 55-year-old man was admitted to the Emergency Department of our hospital with complaints of sudden-onset severe chest pain unrelated to exertion and was not treated with irradiation. The patient denied other associated symptoms.

Before this clinical condition, the patient had been admitted to an inpatient bed in our hospital for 13 days with the diagnosis of COVID-19 and had used a nasal oxygen cannula and the following medications: dexamethasone, codeine, colchicine, and prophylactic enoxaparin. One day after hospital discharge, he started complaining of chest pain.

A physical examination revealed a heart rate of 92 bpm, blood pressure of 122/80 mmHg, no significant changes on cardiac and pulmonary auscultation, good peripheral perfusion, and symmetrical pulses. His chest pain did not worsen with deep inspiration or palpation.

Personal history

The patient had a normal weight and no known risk factors or other diseases, nor was he regularly taking any medication. At the time of admission, he was no longer in pain.

Differential diagnosis

The following diagnostic hypotheses were proposed due to the recent COVID-19 infection and his clinical presentation: myocarditis, pericarditis, acute coronary syndrome, pulmonary thromboembolism, and Takotsubo syndrome.

Diagnostic investigation

After being admitted to the Emergency Department, the patient underwent a 12-lead electrocardiography (ECG) and troponin collection. The ECG showed sinus rhythm and an anterosuperior divisional block without changes suggestive of ischemia (Figure 1). The high-sensitivity troponin levels were: 555.7 ng/L, 1,038 ng/L, 1,029 ng/L, and 853.5 ng/L. The other biochemical tests showed no significant changes.

Transthoracic echocardiography (TTE) showed no segmental contractility or valve changes.

Next, as he reported feeling chest pain and his biomarker levels were increased, he underwent coronary angiography, which showed no significant coronary obstructions (Figure 2).

Based on the diagnostic hypothesis of myocarditis, a cardiac MRI was performed and showed an area of late transmural enhancement located in the apical region of the left ventricle compatible with acute myocardial infarction (Figure 3, Videos 1 and 2). The test was

Case Report

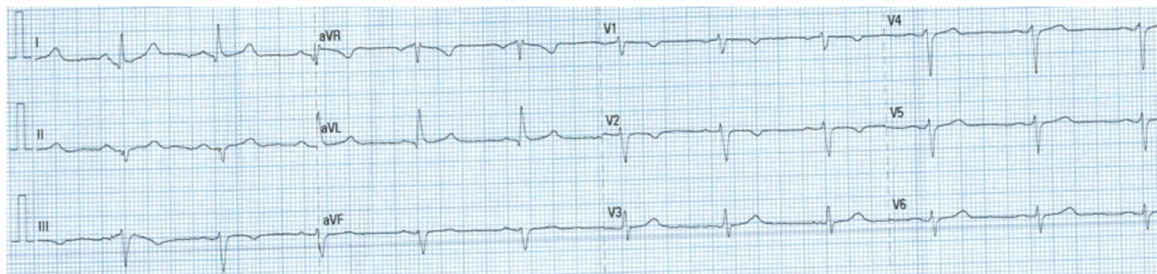


Figure 1 – Electrocardiogram taken on admission.



Figure 2 – Absence of significant coronary obstructions on coronary angiogram.

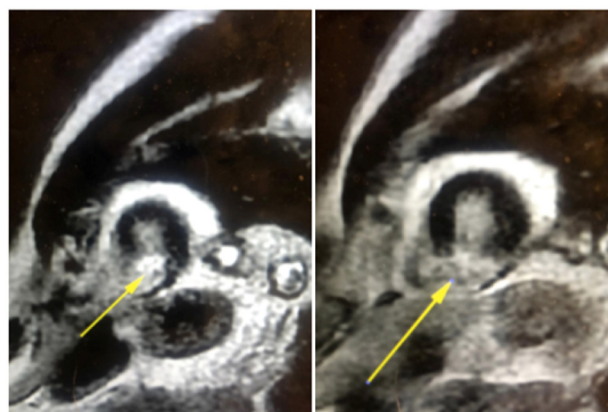


Figure 3 – Cardiac magnetic resonance images showing late transmurular enhancement in the apical region of the left ventricle (yellow arrow).

performed 6 days after the event with no signs of high signal intensity on T2-weighted sequences suggestive of myocardial edema.

The coronary angiogram was reviewed by two interventional cardiologists. However, no coronary obstruction that could have gone unnoticed was identified.

Thus, the diagnostic hypotheses were ruled out due to absence of enhancement suggestive of myocarditis on cardiac MRI; the absence of electrocardiographic and

echocardiographic signs suggestive of pericarditis (as well as significant biomarker increase with chest pain not characteristic of pericarditis); the absence of electrocardiographic (sinus tachycardia and/or S1Q3T3 pattern) and echocardiographic signs (no right ventricular dysfunction and/or signs of pulmonary hypertension) suggestive of pulmonary thromboembolism; and the absence of echocardiographic segmental contractility changes suggestive of Takotsubo syndrome.

Accordingly, we started the patient on dual antiplatelet therapy and a statin. He developed no new symptoms and was discharged from the hospital.

Discussion

In this case, in the absence of coronary obstructions, ventricular dysfunction, and/or segmental contractility changes, the main diagnostic hypotheses were myocarditis and MINOCA. In this scenario, MRI was an essential differential diagnosis tool.⁹

After the complementary MRI findings, we believe that COVID-19-related factors may have influenced the formation of coronary thrombosis or distal emboli and resulted in the transmural pattern of late enhancement in the left ventricle.

In this sense, we observed a rare cardiovascular complication potentially associated with COVID-19 infection that has a temporally atypical clinical presentation. Thus, we must pay

attention to the possibility of late coronary events in COVID-19 patients and consider the possible persistence of inflammatory injury secondary to COVID-19 in the medium term. New studies may be necessary to identify the real incidence and clinical impact of these changes.

Authors' contributions

study conception: Linhares Filho JPP, Lacerda FH; study design: Linhares Filho JPP; data analysis and interpretation: Linhares Filho JPP, Aragão MC; manuscript writing: Linhares Filho JPP, Aragão MC; data/image analysis and manuscript review: Bastos Filho JBB, Rocha RPS; final approval of the submitted manuscript: Lacerda FH, Santana FAC.

Conflict of interest

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

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