Case study

ACUTE MYOCARDITIS AFTER COVID-19 INFECTION IN A YOUNG PATIENT: A CASE REPORT

ABSTRACT:

A 23 years-old male patient with a recent history of flu symptoms presented to the emergency department for acute chest pain. The electrocardiogram (ECG) found diffuse ST elevation in anterior territory. Transthoracic Echocardiography (TTE) showed left ventricular systolic dysfunction with changes in segmental contractions. Coronary angiography revealed normal coronary arteries. Due to the pandemic context, COVID-19 PCR test with serology was performed, it was positive. Cardiac Magnetic Resonance Imaging (MRI) confirmed acute myocarditis. The patient was discharged 7 days later with good clinical evolution.

Through this article, we learn to consider cardiac involvement of SARS-CoV19, even with poor respiratory symptomatology, and to insist on the importance of the cardiac evaluation even for young patients with a mild Covid-19 infection.

KEYWORDS:

Covid-19, Myocarditis, Coronavirus infections, Echocardiography.

INTRODUCTION:

Coronavirus disease 2019 (COVID-19), due to the SARS-CoV-2 virus (severe acute respiratory syndrome coronavirus 2), was described for the first time in China in December 2019. In March 2020, it was declared as worldwide pandemic. **(1, 2)** Clinical presentation is large, going from asymptomatic to severly sick patients, with fever, cough, myalgia, and/or fatigue, as most common symptoms. **(3)**

Cases of cardiac involvement by the COVID-19, developing with acute myocarditis have also been described, mostly in serious and severe cases, **(4, 5)** but still underdiagnosed in young population having mild COVID-19 infections.

We present the case of an acute myocarditis related to a mild COVID-19 infection in a young patient.

CASE REPORT:

A 23 year-old male, without any previous medical or surgical history, presented to the emergency department for an acute chest pain spreading to the neck and both arms, fever, fatigue and weakness. A family history was not contributory. There was no history of recent intake of any drug. He reported a history of recent flu-like symptoms fifteen days before, and was treated by a symptomatic treatment without clinical amelioration.

The physical examination found fever (38,6°C), SaO2= 98% hemodynamic stability (blood pressure = 116/75 mmHg, heart rate = 81 beats per minute), without signs of acute heart failure. ECG found regular sinus rhythm (81 bpm) with a diffuse ST-segment elevation in the anterior territory. **(Figure 1)**

Echocardiography revealed a left ventricular systolic dysfunction with a hypokinesia of the anterior, anteroseptal, anterolateral walls, with a left ventricular ejection fraction (LVEF) at 40%.

Myocardial injury markers showed elevated troponin (15431ng/ml).

Biochemical assessment **(Table 1)** revealed inflammatory reaction, with elevated C-reactive protein (CRP).

Coronary angiography was performed, being normal.

Cardiac MRI revealed the presence of late enhancement areas in 7 LV segments out of 17, confirming then, the diagnosis of an acute myocarditis. **(Figure 2)**

Immunological tests for autoimmune diseases and different viral serological tests were also negative (including hepatitis B and C viruses, Human immunodeficiency Virus (HIV) and Cytomegalovirus (CMV).)

The nasopharyngeal swab tested negative for COVID-19 by reverse transcription polymerase chain reaction (RT-PCR), but the serology testing was positive (for both Immunoglobulin G and M).

The diagnosis of Covid-19 infection was considered.

The patient was managed hemodynamically and treated with steroids, with significant amelioration of clinical symptoms and progressively normal biochemical tests, after 3 weeks of therapy. A TTE control showed a significant amelioration of the LVEF to 55%, with a normal wall motion.

	At Admission	**7 days later**	**Reference values**
Troponin us	15431	9679	<30 ng/ml
CRP	61.7	31	<5mg/l
White cell count	9300	7831	4000-10000/ul
Lymphocytes count	1391	1702	1500-4000/ul
Hemoglobin	14.8		13-18g/dl
Plaquet count	261000		150000-400000/ul
Creatinin	8.7	7.3	7-12mg/l
CPK	504	377	<30UI/l

Table 1: Laboratory tests at the admission, and 7 days later.

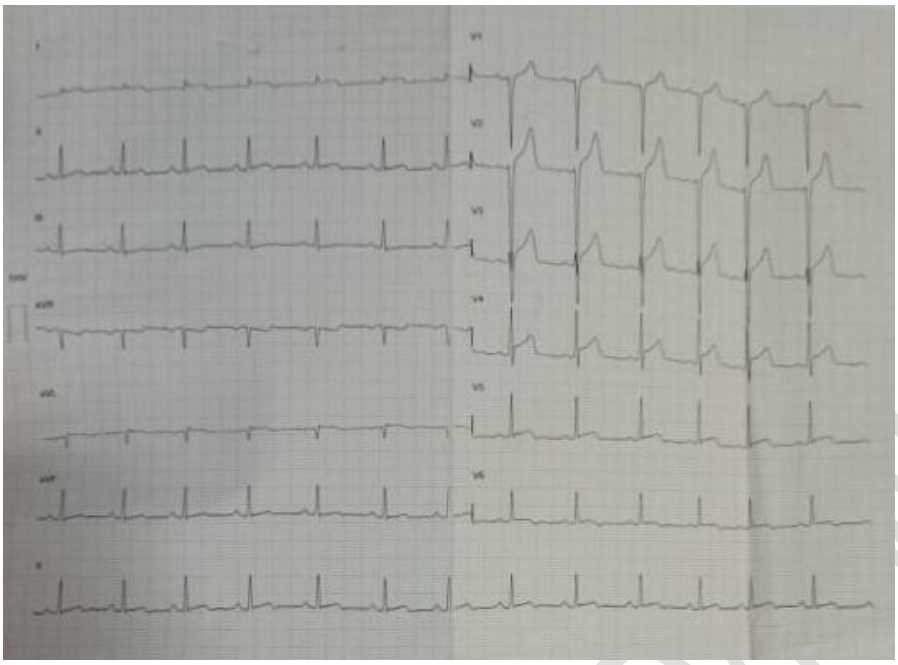

Figure 1 : ECG found diffuse ST-segment elevation in the anterior territory.

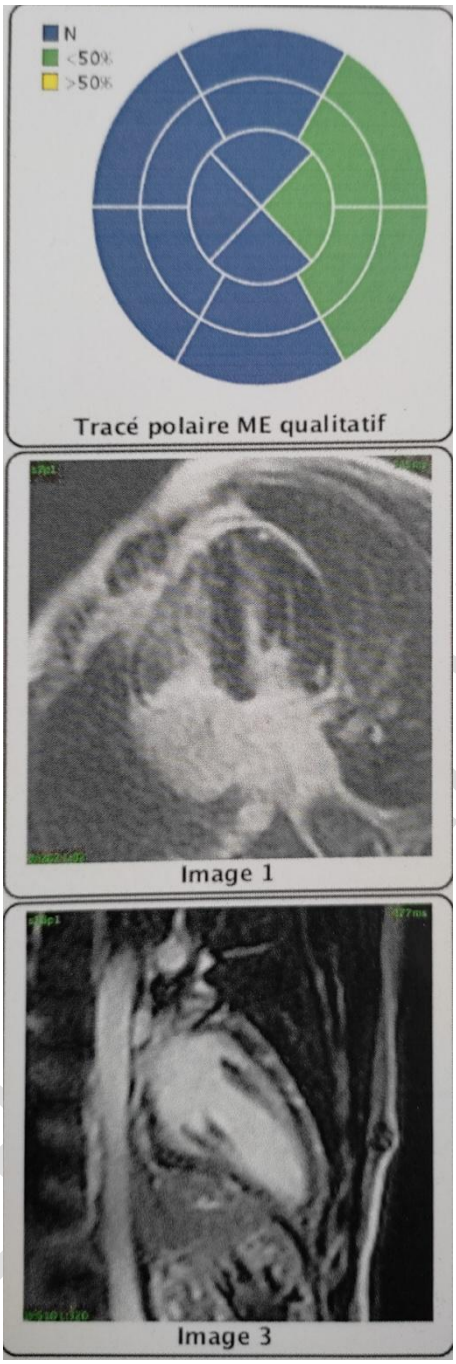

Figure 2 : MRI revealing the presence of late enhancement areas in 7 out of 17 LV segments confirming the diagnosis of an acute myocarditis.

DISCUSSION:

Myocarditis is defined as an inflammation affecting the heart, characterized by a myocardial injury and necrosis with, inflammatory infiltrates (ischemic cause non considered). Exact mechanisms and physiopathology of myocarditis related to COVID-19 are still not yet clearly understood. Most probably, it is the result of direct viral infection to the myocardium or

by the indirect toxicity caused by the systemic reaction, generating hypersensitivity reaction or vascularitis. **(6)**

The analysis of around 45,000 confirmed cases of COVID-19 in China found out cardiovascular involvement, such as myocardial injury (20%), arrhythmias (16%), myocarditis (10% of cases), and heart failure. **(7, 8)**

Other studies suggest that severe COVID 19 infections with cardiovascular complications are mostly observed around 10 to 15 days after the beginning of symptoms **(9)**, (which is convenient with our case). This explains also the negative COVID-19 PCR test.

Several cases of acute myocarditis were described with different levels of severity (from total recovery after the first week of treatment (like in our case), to more serious cases recovering in several weeks, or to cardiogenic shock and death. **(3, 10)**
Increased troponin I was reported in almost all the cases COVID-19 related myocarditis (like in our case). Some studies suggested that the high troponin level was observed in severe cases much more than others. **(11)**

Inciardi and al. **(12)** and the majority of myocarditis related to COVID-19 cases, figured out left ventricular systolic dysfunction with changes in segmental contractions on the transthoracic echocardiography (TTE). This was reported also in our case.

Although MRI remains the gold standard diagnostic test to look for compatible signs with myocarditis, (Non-ischemic late enhancement pattern, like in our case), and this, from a practical point of view, it is clearly difficult and burdensome to perform in COVID-19 patients with the associated risk of infection and contamination. Echocardiography may be helpful in assessing ventricular function. Interpreting BNP (or pro BNP), in this context, might be difficult to assess myocardial degradation. **(13)**

There is still no clear therapeutic consensus in the literature for myocarditis related to COVID-19. Different treatments were proposed after the first reported cases (steroids, intravenous human immunoglobulin, antiviral therapy, inotropic support, interferon alpha-1b, methylprednisone), with almost similar results. **(3, 10, 12)**

In this pandemic context, it is important to think of the hypothesis of cardiac involvement, especially in patients with unexplained increase in myocardial necrosis markers, with a new dysfunction documented by Echocardiography or with abrupt deterioration of symptoms despite respiratory support measures, those and in patients

CONCLUSION:

Despite the poor respiratory symptomatology presented by our patient, a severe cardiac complication was diagnosed. This raises the possibility of a cardiac involvement due to COVID-19 even with no alarming respiratory symptomatology, and the necessity of a systematic cardiac evaluation even for young patients with a mild Covid-19 infection.

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