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-like symptoms presented to the emergency department for acute chest pain. The electrocardiogram showed a diffuse ST elevation in anterior territory. Transthoracic Echocardiography (TTE) showed left ventricular dysfunction with changes in segmental contractions, with serological diagnosis of Covid-19 infection. Invasive coronary angiography was performed, revealing angiographically normal coronary arteries. Due to the pandemic context, a covid PCR test with serology was performed, the serology being positive. A cardiac Magnetic Resonance Imaging (MRI) examination, confirmed acute myocarditis, indicating cardiac involvement by coronavirus disease 2019. The patient was discharged 7 days later with good clinical evolution.

This article can help in considering cardiac affection due to SARS-CoV2, even with poor respiratory symptomatology, and to insist on the importance of the cardiac evaluation for young patients with a mild Covid-19 infection.

KEYWORDS:

Covid-19, Myocarditis, Coronavirus infections, Echocardiography.

INTRODUCTION:

COVID- 19 (Coronavirus disease 2019), caused by the SARS-CoV-2 virus (severe acute respiratory syndrome coronavirus 2), was described for the first time in Whuan, China in December 2019. In March 2020, it was declared as worldwide pandemic. **(1, 2)** The clinical presentation spectrum is wide, from asymptomatic patients to critically ill cases. The first described, and most common symptoms of this infection are fever, cough, myalgia, and/or fatigue. **(3)**

Cases of heart involvement by the coronavirus 2019 disease (COVID-19), developing with acute myocarditis have also been described, mainly in severe cases, **(4, 5)** but still underdiagnosed in young patients with mild COVID-19 infections.

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

CASE REPORT:

A 23 year-old male with no medical, surgical or family, nor drug history, presented to the emergency department for an acute chest pain spreading to the neck and both arms, fever, fatigue and weakness. He reported a history of recent flu-like symptoms 15 days before his consultation that was treated for, by a symptomatic treatment without clinical amelioration.

The physical examination in the emergency room, found fever (38,6°C), SaO2= 98% hemodynamic stability with a blood pressure at 116/75 mmHg, a heart rate at 81 beats per minute, without signs of acute heart failure. The rest of the cardiopulmonary examination was normal. The Electrocardiogram showed a regular sinusal rythm at 81 bpm and a diffuse ST-segment elevation in the anterior territory. **(Figure 1)**

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

Markers of myocardial injury showed elevated troponin us (15431ng/ml).
Biological assessment **(Table 1)** revealed also an inflammatory syndrome, a high level of C-reactive protein (CRP).

Invasive coronary angiography was performed, revealing angiographically normal coronary arteries.

A cardiac Magnetic Resonance Imaging (MRI) examination was performed, revealing the presence of late enhancement areas in 7 LV segments out of 17, confirming then, the diagnosis of an acute myocarditis. **(Figure 2)**

All immunological tests for autoimmune diseases were negative. Other serological tests were, also, negative, including those for hepatitis B and C viruses, Human immunodeficiency Virus (HIV) and Cytomegalovirus (CMV).

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

The diagnosis of Covid-19 infection was considered.

The patient was treated with steroids and hemodynamic monitoring with increasing improvement of clinical symptoms and progressively normal laboratory tests, after 3 weeks of therapy. A TTE control showed an 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.

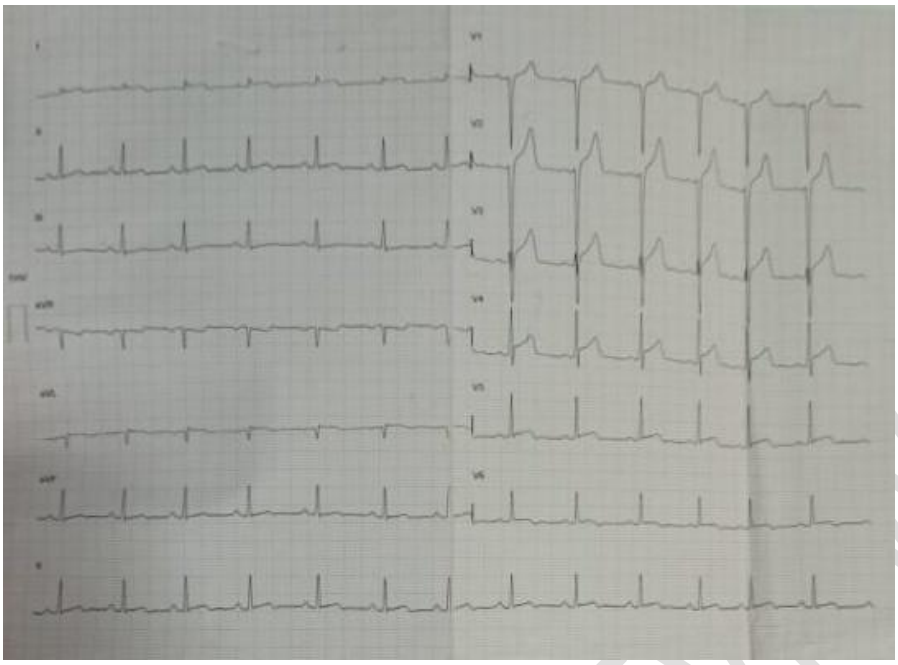

Figure 1 : Electrocardiogram showing an ST-segment elevation diffuse in the anterior territory.

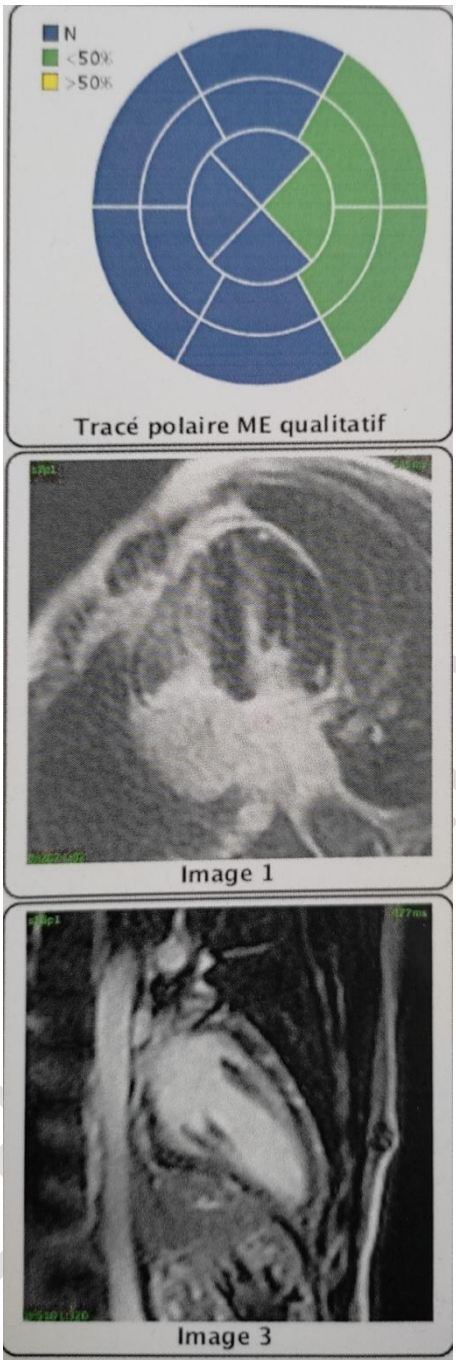

Figure 2 : MRI revealing the presence of late enhancement areas in 7 out of 17 LV segments confirming the diagnosis of an acute myocarditis.

DISCUSSION:

Myocarditis is defined as an inflammatory disease affecting the heart, characterized by an inflammatory infiltrates and myocardial injury apart from an ischemic cause. But the exact physiopathology and mechanisms of COVID-19 related to myocarditis is not clearly understood.

It is possibly due to direct viral infection to the myocardium or by the indirect toxicity causedby the systemic infection, and can trigger vascularitis or hypersensitivity reaction. **(6)**

The analysis of 44,672 confirmed cases of COVID-19 in Wuhan pointed out cardiovascular complications, such as myocarditis (10% of cases), myocardial injury (20%), arrhythmias, (16%) and heart failure andshock (5%). **(7, 8)**

Some studies suggest that severe COVID 19 infections with cardiovascular complications are mostly observed 8–15 days after the beginning of the symptomatology **(9)**, (which is consistent with our case). This duration of 8 days can explain, also, the negative nasopharyngeal swab test.

Several cases of acute myocarditis were reported with different degrees of severity ranging from a total recovery as of the first week after initiation of treatment (like in our case), to more severe cases recovering in 3–4 weeks, or to death due to cardiogenic shock. **(3, 10)**
Increased troponin I was reported in almost all the cases COVID-19 related myocarditis (like in our case). And some studies suggest that the increase troponin level was observed in severe cases much more than others. **(11)**

Inciardi et al. **(12)** and the vast majority of cases presenting COVID-19 related myocarditis reported a left ventricular dysfunction with changes in segmental contractions on the transthoracic echocardiography (TTE). That was reported also in our case.

Although MRI remains the gold standard diagnostic test to search for signs compatible with myocarditis, (such as the presence of 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 are no evidences in the literature about a clear and unique therapeutic consensus for the COVID-19 related myocarditis. Different treatments were proposed after the first case reported of acute myocarditis caused by a COVID-19 (steroids, intravenous human

immunoglobulin, antiviral therapy, inotropic support, interferon alpha- 1b, methylprednisone), with almost similar results. **(3, 10, 12)**

In the scenario of the SARS-CoV-2 pandemic, it is important to consider the hypothesis of cardiac involvement, mainly in patients with abrupt deterioration of symptoms despite respiratory support measures, those with unexplained increase in myocardial necrosis markers and in patients with a new dysfunction documented by Echocardiography.

CONCLUSION:

Regardless the poor respiratory symptomatology presented by our patient, a severe cardiac complication was diagnosed. This raises the possibility of a cardiac affection due to SARS-CoV2 even with no alarming respiratory symptomatology, and the necessity of a systematic cardiac evaluation even for young patients with a mild Covid-19 infection.

REFERENCES:

1- Cascella M, Rajnik M, Cuomo A, Dulebohn SC, Di Napoli R. Features, Evaluation and Treatment Coronavirus (COVID-19). 2020 Jul 4. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 [cited 2020 Jul 20].

2- Ekinci A, Yücel Uçarkuş T, Okur A, Öztürk M, Doğan S. MRI of pneumonia in immunocompromised patients: comparison with CT. Diagn Interv Radiol. 2017;23(1):22-8.

3- J.H. Zeng, Y.X. Liu, J. Yuan, et al., First case of COVID-19 complicated with fulminant myocarditis: a case report and insights, Infection 48 (2020) 773–777.

4- Hu H, Ma F, Wei X, Fang Y. Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin. Eur Heart J. 2020 Mar 16:ehaa190.

5- Rente A, Uezato Junior D, Uezato KM. Coronavirus and the heart | A case report on the evolution of

COVID-19 Associated with Cardiological Evolution. Arq Bras Cardiol. 2020;114(5):839-42.

6- Pagnoux C, Cohen P, Guillevin L. Vasculitides secondary to infections. Clin Exp Rheumatol. 2006;24(2 Suppl 41):S71-81. Review.

7- Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. JAMA. JAMA. 2020;323(11):1061-9.

8- Shi S, Qin M, Shen B, Cai Y, Liu T, Yang F, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA Cardiol. 2020;5(7):802-10.

9- A. Magadum, R. Kishore, Cardiovascular manifestations of COVID-19 infection, Cells 9 (11) (2020) 2508.

10- H. Hu, F. Ma, X. Wei, Y. Fang, Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin, Eur. Heart J. (2020).

11- F. Zhou, T. Yu, R. Du, G. Fan, Y. Liu, Z. Liu, et al., Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study, Lancet (2020).

12- R.M. Inciardi, L. Lupi, G. Zaccone, L. Italia, M. Raffo, D. Tomasoni, et al., Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19), JAMA Cardiol 5 (7) (2020) 819–824.

13- Zeng, J.H.; Liu, Y.; Yuan, J.; Wang, F.; Wu, W.; Li, J.; Wang, L.; Gao, H.; Wang, Y.; Dong, C.; Li, Y.; Xie, X.; Feng, C.; Liu, L. First Case of COVID-19 Infection with fulminant Myocarditis Complication: Case Report and Insights.