

Case Series

Post COVID 19 Myocarditis: Clinical Case Series

Hanane ZOUZOU

Professor in cardiology, Department of Medicine Batna 2 university Algeria

Received: 01 April, 2023

Accepted: 04 May, 2024

Published: 09 May 2024

Abstract:

Corona virus disease 2019 infection exhibits a tropism for the respiratory tract, however several cardiac damages have been reported, such as coronary disease, cardiac arrhythmias and myocarditis; this latter has become more frequent after the outbreak of the COVID-19 pandemic, and may be the only manifestation of COVID 19 infection, the diagnosis is not always obvious especially for focal myocarditis and can be misdiagnosed as an acute coronary syndrome in some patients.

We reported five patients, admitted between June 2020 and January 2021, in our cardiology department for isolated acute focal myocarditis secondary to COVID 19 infection without respiratory or other damages, the diagnosis was not evident at first, but further investigations such as: electrocardiographic evolution, magnetic resonance imagery, coronary computed tomography angiography, and COVID 19 antibody testing, were in favor of the diagnosis of focal myocarditis secondary to COVID 19 infection. Cardiac magnetic resonance imaging showed, delayed contrast enhancement in the lateral wall of the left ventricle, for all five patients, so fibrosis is preferentially located in the lateral wall, and the outcome was favorable without hemodynamic or arrhythmic complications.

Key words: Focal myocarditis, Covid 19, subepicardial fibrosis

Introduction:

Corona virus disease 2019 infection exhibits a tropism for the respiratory tract, however several cardiac damages have been reported, such as coronary disease, cardiac arrhythmias, and myocarditis, this latter has become more frequent after the outbreak of the COVID-19 pandemic, and may be the only manifestation of COVID 19 infection.

The electrocardiographic evolution, coronary artery angiography and Cardiac magnetic resonance imaging, excluded coronary disease and allowed the diagnosis of focal myocarditis, and the serology tests may be the only proof that myocarditis is secondary to COVID 19 infection.

We reported a series of five patients admitted in our cardiology department for management of isolated myocarditis, more investigations were in favor of focal acute myocarditis secondary to COVID19 infection.

Case 1

A 23 year-old man, admitted on May 10th 2020, in cardiology department, for chest pain with tall T wave in precordial leads, (figure 1), the diagnosis of acute coronary syndrome was suspected and patient had received dual platelet and anticoagulation therapy, two weeks before hospitalization, the patient had presented asthenia and body aches, but the polymerase chain reaction (PCR) test was negative and chest computed tomography (CT) scan was normal, Echocardiography Doppler showed normal left ventricular ejection, Coro scan realized showed normal coronary arteries.

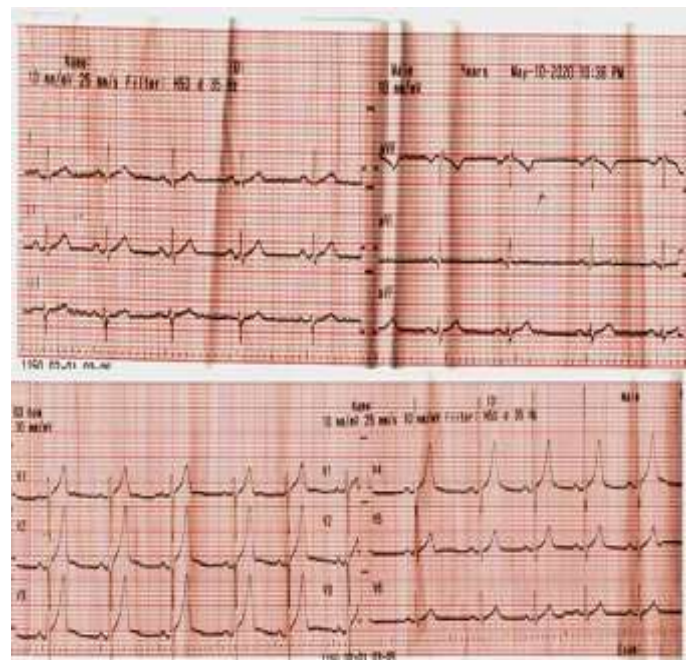


Figure 1: surface ECG of the first patient showed tall T wave in leads V2 to V4

But after Gadolinium administration, Cardiac magnetic resonance imaging (CMRI) showed, delayed contrast enhancement in the epicardial layer of lateral wall of the left ventricle. (Figure 2), serology tests for COVID 19 were realized in December 2020, showed positive test for IgG (IgG = 7.34 AU/ml) and negative test for IgM (IgM = 0.21 AU/ml), which proves that he had been in contact with COVID infection virus.



Figure 2: Cardiac magnetic resonance imaging (CMRI) of the first patient showed, delayed contrast enhancement in the epicardial lateral wall of the left ventricle

Case 2:

A 28 year-old man, with no particular cardiovascular risk factor, admitted on August 21th, 2020, in cardiology department for chest pain with ST segment elevation in leads DII, DIII, aVF; and negative T waves in DI and aVL (Figure 3); high-sensitivity cardiac troponin level was very high 8410.3 ng/l then 9949.8 ng/l; the diagnosis of acute coronary syndrome with ST segment elevation was retained and patient had received dual platelet and anticoagulation therapy, the patient had also presented asthenia and fatigue, two months before, with positive serology test for COVID-19 (IgG = 8.49 AU/ml), and abnormal inflammatory tests (positive C protein reactive = 14.9 mg/l, high sedimentation rate: 40/50), chest computed tomography (CT) scan was normal, Echocardiography Doppler showed normal left ventricular ejection, Coro scan realized showed normal coronary arteries, but after Gadolinium administration, Cardiac magnetic resonance imaging (CMRI)

showed, delayed contrast enhancement in the epicardial layer of postero-lateral and posterior walls of the left ventricle in favor of subacute myocarditis.

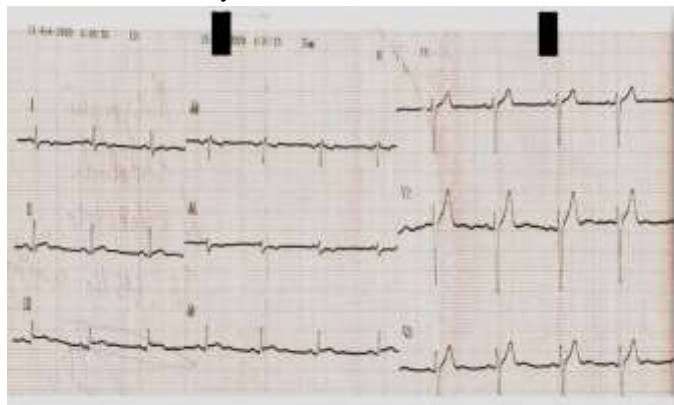


Figure 3: Surface ECG of the second patient showed ST segment elevation in DII, DIII, aVF, and negative T wave in DI, aVL

Case 3

A 44 year-old man, who is smoking, admitted on September 19th 2020, in cardiology department for chest pain with normal surface ECG at admission, but 2 days after, surface ECG showed flat T wave in leads DI and aVL; high-sensitivity cardiac troponin level was very high 2642.40 ng/l then 1904.6 ng/l, the diagnosis of acute coronary syndrome without ST segment elevation was retained and patient had received dual platelet and anticoagulation therapy, the patient had also presented asthenia, after a short trip aboard two months before, serology testing showed positive serology test for COVID-19, normal inflammatory tests (positive C protein reactive = 0.28 mg/l, sedimentation rate: 03), chest computed tomography (CT) scan was normal, Echocardiography Doppler showed normal left ventricular ejection, Coro scan realized showed normal coronary arteries, but Cardiac magnetic resonance imaging (CMRI) with Gadolinium administration, showed delayed contrast enhancement in the epicardial layer of medio-lateral wall of the left ventricle in favor of subacute myocarditis (Figure 4).



Figure 4: Cardiac magnetic resonance imaging (CMRI) of the third patient showed, delayed contrast enhancement in the epicardial layer of medio-lateral wall of the left ventricle.

Case 4:

A 26 year-old man, with no particular cardiovascular risk factor, admitted on December 15th 2020, in cardiology department for asthenia, fever and chest pain with ST segment elevation in leads DI and aVL with negative T wave in aVL (Figure 5); high-sensitivity cardiac troponin level was very high 10964.5 ng/l then 7339 ng/l, the diagnosis of acute coronary syndrome with ST segment elevation was retained and patient had received dual platelet and anticoagulation therapy, echocardiography exam showed preserved systolic ventricular function, serology testing showed positive serology test for COVID-19 (IgG = 2.74 AU/ml), anormal inflammatory tests (positive C protein reactive = 19.92mg/l), the polymerase chain reaction (PCR) test was negative and chest computed tomography (CT) scan was normal, Coro scan showed normal

coronary arteries, but after Gadolinium administration, Cardiac magnetic resonance imaging (CMRI) showed, delayed contrast enhancement in the epicardial layer of lateral and infero lateral walls of the left ventricle in favor of myocarditis (Figure 6).



Figure 5: Surface ECG of the fourth patient showed ST segment elevation in leads DI and aVL with negative T wave in aVL

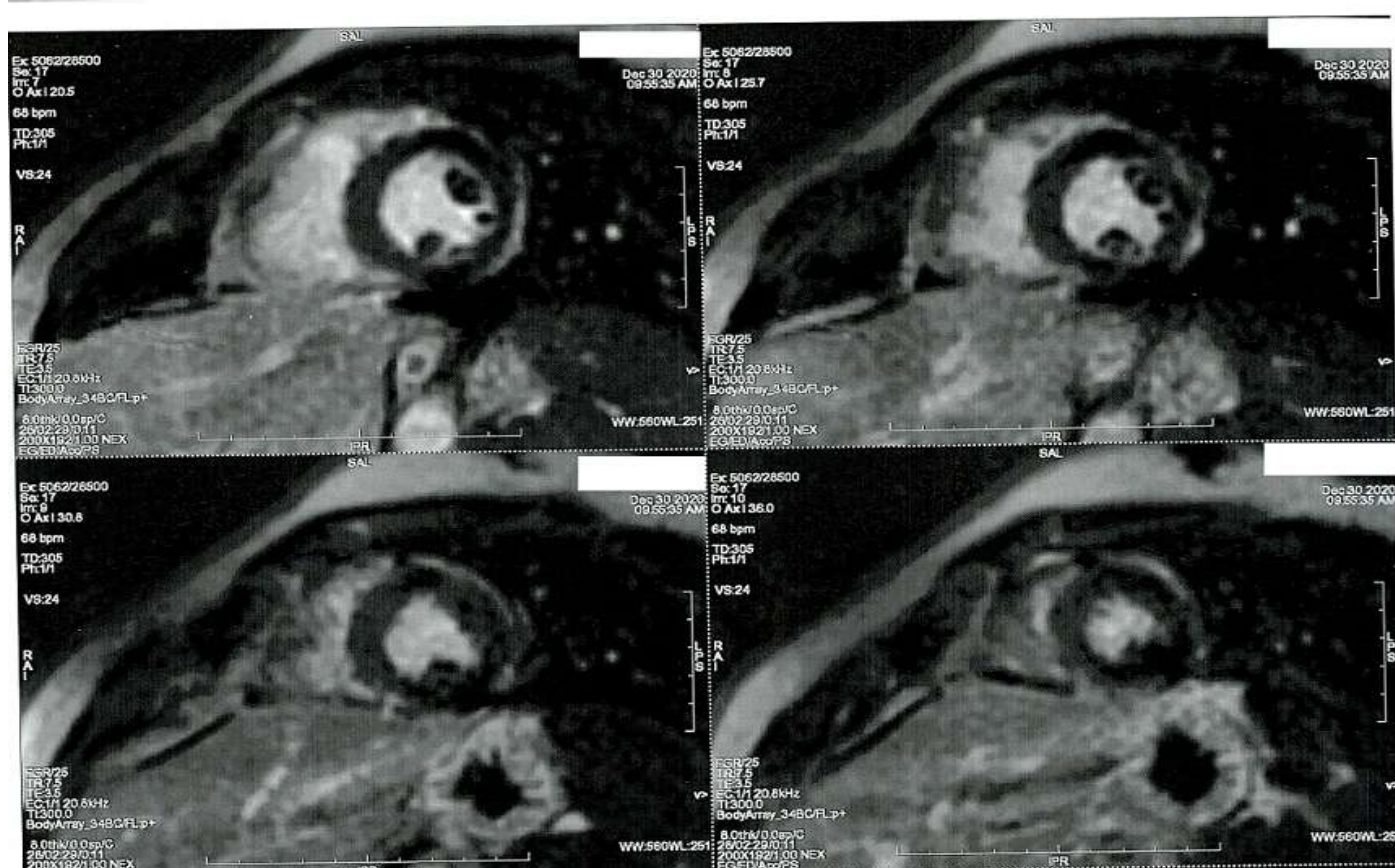


Figure 6: Cardiac magnetic resonance imaging (CMRI) of the fourth patient showed, delayed contrast enhancement in the epicardial layer of lateral and infero lateral walls of the left ventricle

Case 5:

A 17 year-old man, who is smoking, and he has already had a COVID 19 infection four months before, admitted on January 21st 2021, in cardiology department for fever and chest pain with slight ST segment elevation in inferior and precordial leads (figure 7), high-sensitivity cardiac troponin level was very high 833.1 ng/l then 445.3 ng/l, the diagnosis of acute myocarditis was suspected, inflammatory tests were normal (positive C protein reactive = 2.96 mg/l, sedimentation rate: 6, fibrinogen level : 3.64 g/l), serology testing showed positive serology test for COVID-19 (IgG positive), the chest computed tomography (CT) scan was normal, Coro scan showed normal coronary arteries, but after Gadolinium administration, Cardiac magnetic resonance imaging (CMRI) showed, delayed contrast enhancement in the epicardial layer of lateral and infero lateral walls of the left ventricle especially in the basal and median left ventricular regions in favor of acute myocarditis (Figure 8).

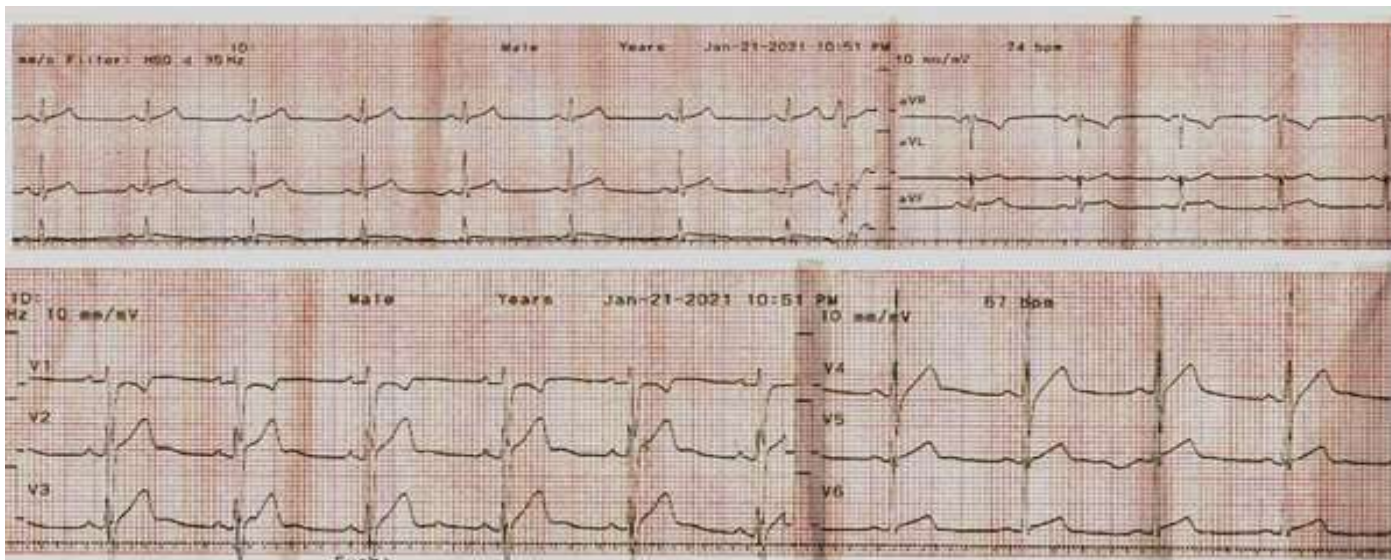


Figure 7: Surface ECG of the fifth patient showed slight ST segment elevation in inferior and precordial leads



Figure 8: Cardiac magnetic resonance imaging (CMRI) of the fifth patient showed, delayed contrast enhancement in the epicardial layer of lateral and infero lateral walls of the left ventricle especially in the basal and median left ventricular regions

Discussion:

The SARS-CoV-2 infection (COVID-19) started in China in late 2019, then spread all over the world and was considered as pandemic infection by the World Health Organization (WHO) in March 2020.

Corona virus disease 2019 infection exhibits a tropism for the respiratory tract, however several cardiac damages have been reported, such as coronary disease, cardiac arrhythmias and myocarditis; this latter has become more frequent after the outbreak of the COVID-19 pandemic, and may be the only manifestation of COVID 19 infection.

The prevalence of myocarditis is about 2.4 cases/1000 hospitalized patients for COVID 19 infection [1] the risk of myocarditis increases in the month after a positive SARS-CoV-2 test, by about 10 times; more frequent in men (60%) [2]

Myocarditis is defined as inflammation of the cardiac muscle with or not myocardial damage; with normal coronary perfusion.

The physiopathology of myocarditis related to COVID 19 infection is controversial, some mechanisms were incriminated such as direct cell injury by virus infection or toxins, but the most likely hypothesis is autoimmune processes, this latter is related to cytotoxicity due to hyper immune response, explained

by excessive release of inflammatory mediators from immune cells including the T lymphocytes which in turn causes T lymphocyte activation and myocardial damage related to Cardio tropism of T lymphocytes. [3-5]

The interaction between T lymphocytes and myocardial is explained essentially by heart production of Hepatocyte Growth Factor (HGF) and presence of an HGF receptor on T lymphocytes. [6]

In this our first series, between June 2020 and January 2021 between, all patients are men and have isolated focal myocarditis without hemodynamic or arrhythmic complications, or other damages, like respiratory infection.

Our patients had experienced symptoms several weeks after contact with the virus, this hypothesis was confirmed with serology testing which showed positive IgG but not IgM, which in favor of late occurrence of myocarditis and hyper immune response hypothesis.

The diagnosis was not evident at first, and misdiagnosed as an acute coronary syndrome in all five patients, surface ECG showed ST segment elevation in lateral leads, except for the first patient who had tall T waves in precordial leads.

Coronary computed tomography angiography excluded coronary disease for all patients and showed normal coronary arteries.

Further investigations such as: electrocardiographic evolution, magnetic resonance imagery and COVID 19 antibody testing, were in favor of the diagnosis of focal myocarditis secondary to COVID 19 infection.

Cardiac magnetic resonance imaging showed, delayed contrast enhancement in the lateral wall of the left ventricle, for all five patients, so fibrosis is preferentially located in the lateral wall; may be the production of Hepatocyte Growth Factor (HGF) is important in the lateral wall of the left ventricle.

Some studies, reported that 7% of COVID-19 death, was related to myocarditis [7], in our series, the evolution of our patients was favorable without complications.

Conclusion:

Post COVID 19 myocarditis is not rare; the diagnosis is not obvious and can be misdiagnosed as an acute coronary syndrome in some patients, but Coronary computed tomography angiography, magnetic resonance imagery and COVID 19 antibody testing, allow the correct diagnosis of myocarditis. Hyper immune response is the most likely hypothesis; focal form is common, especially in the lateral wall of left ventricle.

Bibliography

1. Ammirati E, Lupi L, Palazzini M, et al. Prevalence, Characteristics, and Outcomes of COVID-19-associated Acute Myocarditis. *Circulation*. 2022;145:1123–1139.
2. Patone M, Mei XW, Handunnetthi L, et al. Risks of myocarditis, pericarditis, and cardiac arrhythmias associated with COVID-19 vaccination or SARS-CoV-2 infection. *Nat Med*. 2022;28:410–422.

3. Kawakami R, Sakamoto A, Kawai K, et al. Pathological Evidence for SARS-CoV-2 as a Cause of Myocarditis: JACC Review Topic of the Week. *J Am Coll Cardiol*. 2021;77:314–325.
4. Yuki K, Fujiogi M, Koutsogiannaki S. COVID-19 pathophysiology: A review. *Clin Immunol*. 2020;215:108427.
5. Castiello T, Georgiopoulos G, Finocchiaro G, et al. COVID-19 and myocarditis: a systematic review and overview of current challenges. *Heart Fail Rev*. 2022;27:251–261.
6. Komarowska I, Coe D, Wang G, et al. Hepatocyte growth factor receptor c-Met instructs T cell cardiotropism and promotes t cell migration to the heart via autocrine chemokine release. *Immunity*. 2015; 42: 1087-1099
7. Driggin E, Madhavan M.V, Bikdeli B, et al. Cardiovascular considerations for patients, health care workers, and health systems during the coronavirus disease 2019 (COVID-19) pandemic. *J Am Coll Cardiol*. 2020; 75: 2352-2371

Copyright (c) 2024 The copyright to the submitted manuscript is held by the Author, who grants the Clinical Medicine and Health Research Journal a nonexclusive license to use, reproduce, and distribute the work, including for commercial purposes.



This work is licensed under a [Creative Commons Attribution 4.0 International License](https://creativecommons.org/licenses/by/4.0/)