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COVID 19 myocarditis: Myth or reality?

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COVID 19 Myocarditis: Myth or Reality?

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Abstract

COVID 19 so far is not a known cardiotropic virus, and the term "myocarditis" should be exclusively used after EMB or autopsy proven diagnosis. We report a case of 26-year-old man admitted for COVID 19 infection and symptoms leading to myocarditis. We describe the workup that led to the potential diagnosis.

Keywords: COVID19, Myocarditis, Myocardial injury, Endomyocardial biopsy, Arrhythmias

1. Background

Coronavirus disease has quickly become a global pandemic. Beside acute respiratory distress syndrome, patients with cardiovascular diseases are more likely to develop severe symptoms. Therefore, myocardial injury is one of the important pathogenic features of COVID-19.

The proposed mechanisms of myocardial injury are direct damage to the cardiomyocytes, systemic inflammation, myocardial interstitial fibrosis; interferon mediated immune response, coronary plaque destabilization, and hypoxia.

This report describes a case of a 25 years old man, admitted for dyspnea and chest pain focusing on workup that led to the potential diagnosis.

2. Case presentation

In March 2020, the patient presented to the Emergency with a fever, chest pain, and dyspnea.

Physical examination revealed O_2 saturation was 96% with the nasal cannula at 10 L/min, respiratory rate at 36 breaths/min, blood pressure at 100/76 mmHg and temperature at 38, 6 °C.

The electrocardiogram demonstrated ST-segment elevation in lateral and posterior territories with Q wave (Fig. 1).

CT scan showed an atypical form of viral pneumonia and alveolar hemorrhage (Fig. 2).; Blood gas analysis after admission showed a pH: 7.43, PCO_2: 53.4 mmHg, PO_2: 66.2 mmHg. Pharyngeal swabs and sputum were tested positive for COVID 19.; Markers of myocardial injury included elevated troponin I (50.37 ng/mL), aspartate aminotransferase (752 U/L), Creatine phostokinase (7564 U/L), Lactate Deshydrogénase (3239U/L) and D dimer at 9600 μg/L. The immunological assessment was negative.

Bedside TTE showed myocardial wall-motion abnormalities along with apical akinesia and apical thrombosis, low LVEF (23%), restrictive mitral profile, and pulmonary hypertension (PAPS: 44 mmHg) (Fig. 3).

The diagnosis was considered severe pneumonia and myocarditis, with no obvious etiology beside a COVID 19 infection. The treatment regimen was ventilatory support, association Hydrochloroquine, Azithromycin and unfractionated heparin anticoagulation therapy due to apical thrombosis.

However, the patient's lung lesions continued to progress. The LVEF reminded the same at 25%, and the infection-related markers increased gradually. Moreover, the rest of myocardial injury biomarkers stayed elevated after 3 days of admission. Vasoactive drugs were needed. Multiple organs;

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dysfunction gradually developed. The patient died on the 15th day of hospitalization.

3. Discussion

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a novel betacoronavirus described among patients with pneumonia symptoms in Wuhan, China in December of 2019 [1]. Over the past few months, coronavirus has become a worldwide pandemic, with over 5,000,000 cases globally.

Several studies have shown that cardiac complications are not uncommon. Yet, there are controversial opinions about the combination of COVID 19 and myocarditis.

Pathological changes in the heart tissue may be due to the virus replication in the myocardium or to harmful immune responses caused by a viral infection. Autopsy studies showed inflammatory infiltration in the heart tissue, but no clear viral inclusion body was observed [2].

Here are some hypotheses of cardiac injury in the COVID 19 infection (Fig. 4).

Abbreviation

COVID 19	CORONA virus disease 2019
LVEF	left ventricle ejection fraction
ECG	electrocardiogram
TTE	transthoracic echVocardiography
UFH	unfractionned heparin
BNP	brain natriuric peptid
CMR	cardiovascular magnetic resonane
EMB	endomyocardial biopsy
ECMO	extracorporeal membrane oxygenation
VAD	ventricular assist device
IABP	intra-aortic balloon pump
PAPS	pulmonary artery systolic pressure

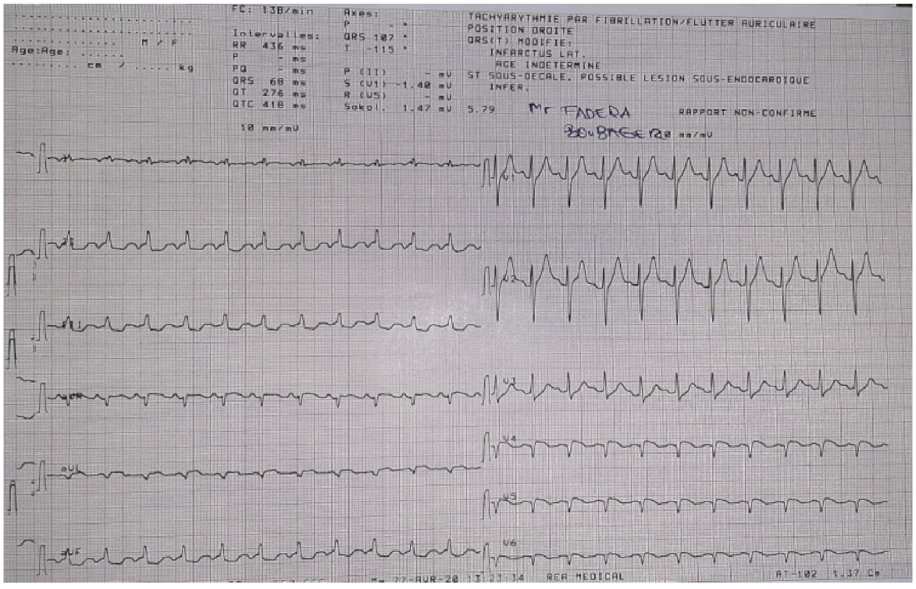

Fig. 1. Electrocardiogram showing ST segment elevation in lateral and posterior territories with Q waves.

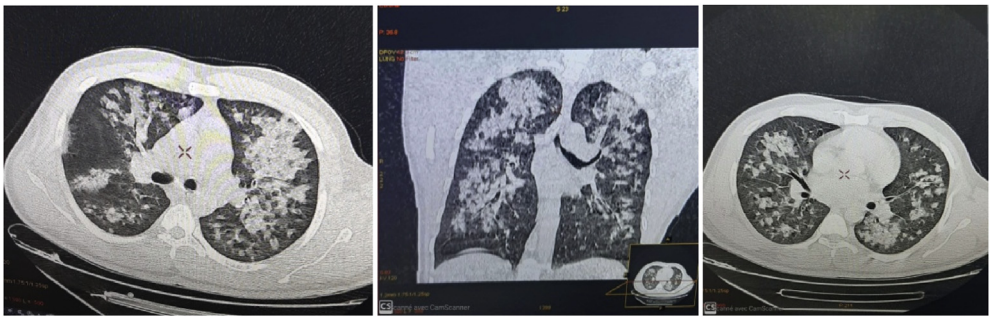

Fig. 2. Thoracic CT showing atypical form of viral pneumonia and alveolar hemorrhage.

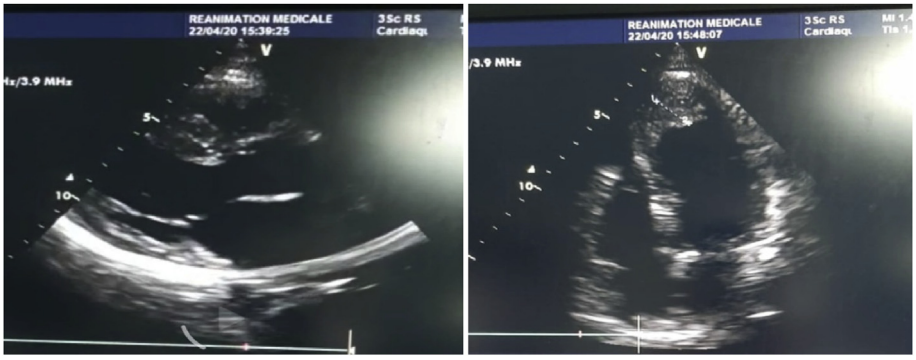

Fig. 3. TTE on the second day after admission showing decreased LVEF and a large apical thrombosis.

The prevalence of myocarditis among COVID-19 patients is undefined. This is due to the deficiency of specific diagnostic modalities to assess myocarditis [3].

Clinical presentation of SARS-CoV-2 myocarditis varies from one case to another. The most urgent clinical sign is ventricular dysfunction and cardiogenic shock [4].

Blood tests usually demanded for myocarditis show elevated levels of lactate and other inflammatory markers. Cardiac troponin I, troponin T, NT-proBNP, and BNP levels usually are elevated in myocarditis because of acute myocardial injury and possible ventricular dilation. However, a negative troponin result cannot exclude myocarditis [5].

Electrocardiogram abnormalities such as ST elevation may be observed. ECG modifications are neither sensitive nor *specific* in detecting the myocarditis [6].

The echocardiogram signs are chamber dilatation and ventricular systolic dysfunction. Yet cardiovascular magnetic resonance can be better by offering multiple advantages. The CMR results should be interpreted according to the revised Lake Louise consensus criteria. The myocardial edema and/or scarring were observed among a lot of the SARS-CoV-2 patients [7].

Endomyocardial biopsy is the finest test for myocarditis. EMB can provide an etiological diagnosis of viral infectious VS autoimmune myocarditis. So far, no case of endomyocardial biopsy or autopsy had proven myocarditis caused by COVID-19. There was no proof that COVID-19 enters cardiomyocytes nor replicating inside the myocyte and causing direct cardiomyocyte necrosis [8]. In our case there was no EMB performed.

The management protocol for cardiogenic shock includes administration of inotropes and/or vasopressors and mechanical ventilation. There was no evidence benefit of using intravenous immunoglobulin or corticosteroids in active-infection myocarditis.

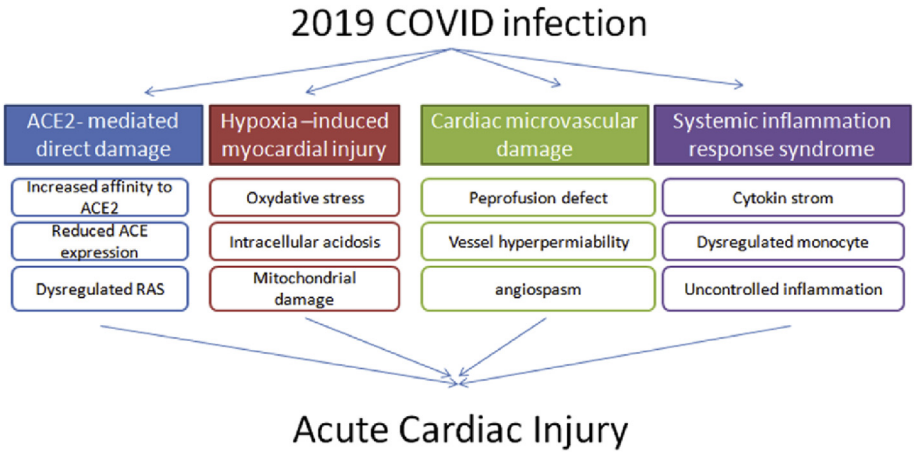

Fig. 4. Physiopathological hypotheses of cardiac injury in the COVID19 infection.

Moreover, Multiples pharmacologic drugs being purposed for use in COVID-19 patients are understudies.

Tocilizumab tested in COVID-19 patients with raised IL-6 levels, might be helpful in the setting of cytokine storm syndrome and reducing myocardial injury [9].

Chloroquine is considered a very controversial drug, multiples trials to assess its efficacy in treating COVID-19 patients with the severe respiratory syndrome. However, Chloroquine may cause QTc interval prolongation; yet their effects seem to be modest. Nevertheless, many pharmacologic agents used empirically to treat COVID-19, including ritonavir/lopinavir and azithromycin.

4. Conclusion

During this pandemic, several cases of "myocarditis" have been reported. The pathophysiology hypothesis is an association of viral injury to cardiomyocytes and immune response to infected myocardium. ECG and cardiac biomarkers can raise suspicion. Cardiac imaging is used to orient diagnosis. The gold exam is an endomyocardial biopsy. So far, no EMB or autopsy series found histological myocarditis.

Our understanding of this virus and corresponding treatment strategies continues to expand, and as cardiologist community, we need more guidance regarding cardiac complications from this virus.

Author contribution

Conception and design of Study: Sasbou L, El boussaadani B, Fellat I, Cherti M. Literature review: Sasbou L, El boussaadani B, Fellat I. Drafting of manuscript; Revising and editing the manuscript critically for important intellectual contents: Sasbou L, El boussaadani B. Supervision of the research: Fellat I, Cherti M.

Conflicts of interest

No conflict of interest exists.

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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