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COVID-19 a Possible Cause of Myocarditis and Pericarditis: About A Case

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Abstract **Case Report**

Severe Acute Respiratory Syndrome-Coronavirus-2 (SARS-CoV-2) is an unparalleled challenge for the healthcare community across the globe. Angiotensin-2 converting enzyme receptors play a central role in the pathogenesis of the virus. Disruption of this receptor results in cardiomyopathy, cardiac dysfunction, and heart failure. Pericarditis is described as another cause of morbidity in patients with COVID-19. The exact mechanisms of how SARS-CoV-2 can cause myocardial damage are not clearly understood. We report the case of a 72-year-old man presenting for 17 days with retrosternal chest pain suggestive of pericarditis associated with a dry cough and a decline in general condition. He has had exertional asthma and active smoking as a history. He is taken care of in the emergency room. His clinical examination found a patient hemodynamically and respiratory stable, afebrile with a normal somatic examination apart from crackling rales. The biological assessment shows an inflammatory syndrome with slightly increased troponins HS. The scan performed in the emergency room notes typical pulmonary involvement moderate to COVID-19 and low-abundance pericardial effusion confirmed by echocardiography. The COVID-19 nasopharyngeal smear was positive. We conclude in an acute peri-myocarditis with COVID-19. The clinical and biological outcome is rapidly favorable with colchicine.

Keywords: Pericarditis, Pericardial effusion, COVID-19, Case report.

INTRODUCTION

Severe acute respiratory syndrome-Coronavirus-2 (SARS-CoV-2) is an unparalleled challenge to the health care community worldwide. Respiratory involvement is the major clinical manifestation of COVID-19, ranging from mild influenza-like illness to severe pneumonia and life-threatening acute respiratory distress syndrome.

Angiotensin-2 converting enzyme receptors play a central role in the pathogenesis of the virus. Disruption of this receptor leads to cardiomyopathy, cardiac dysfunction and heart failure. Patients with cardiovascular disease are more likely to be infected with SARS-CoV-2 and are more likely to develop severe symptoms. Hypertension, arrhythmia, cardiomyopathy, and coronary artery disease are among the major cardiovascular disease comorbidities seen in severe COVID-19 cases [1]. There is a growing body of literature exploring cardiac involvement in SARS-CoV-2.

Pericarditis is described as another cause of morbidity in patients with COVID-19. The exact mechanisms of how SARS-CoV-2 can cause myocardial injury are not clearly understood.

CLINICAL CASE PRESENTATION

Patient Information:

We report the case of a 72-year-old man with a 17-day history of retro-sternal chest pain suggestive of pericarditis associated with a dry cough and a decline in general condition. He had a history of exercise-induced asthma and active smoking.

Clinical Findings:

He was taken to the emergency room. His clinical examination revealed a blood pressure of

140/80mmHg, a heart rate of 75 beats per minute, a body temperature of 36.5°C and a saturation of 92% in room air (AA). Cardiopulmonary auscultation showed diffuse and bilateral subcrepitant rales, with no heart murmur or pericardial rub.

Diagnostic Approach:

Biology objectives an inflammatory syndrome with CRP at 140 mg/L and neutrophilic polynucleosis (12.4 G/L) and fibrinogenemia at 9g/L. Peak troponin HS was 80 ng/L (norm < 30ng/L).

The ECG shows a diffuse ST-segment elevation.

The chest CT scan shows a typical moderate COVID-19 involvement and a circumferential pericardial effusion of small abundance (Figure 1) confirmed by trans thoracic echocardiography (Figure 2).

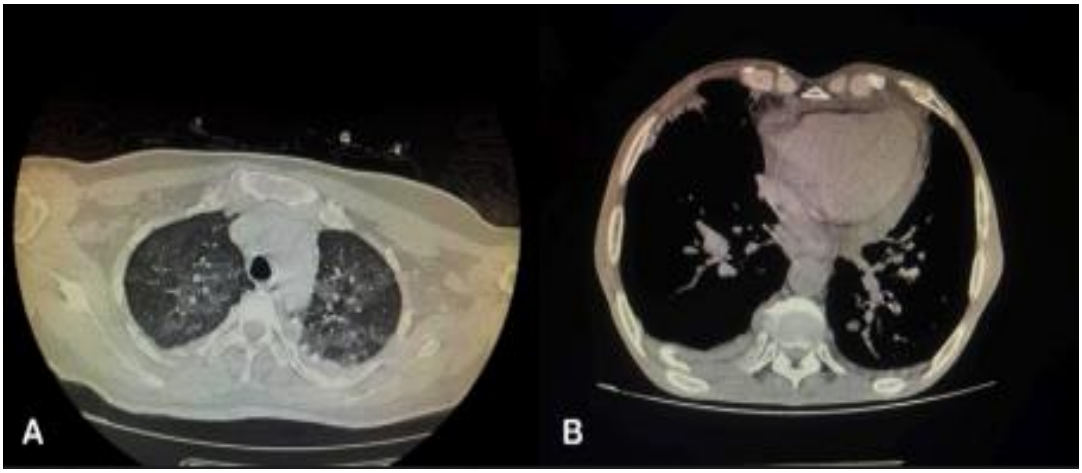

Figure 1: Noninjected chest CT scan objectifying moderate pulmonary parenchymal involvement typical of COVID (A) with low to moderate pericardial effusion (B)

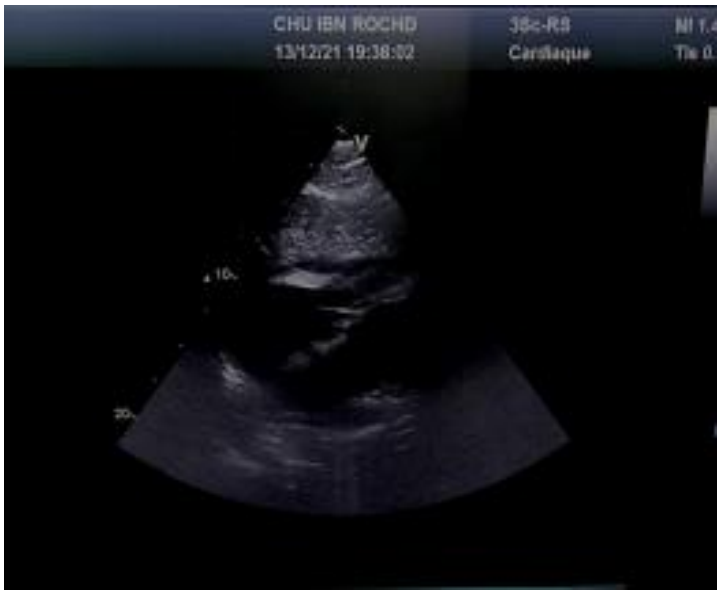

Figure 2: Trans-thoracic echocardiography in subcostal slice objectifying a circumferential pericardial effusion of low abundance

Therapeutic Intervention and Follow-up:

After the CT scan, the patient presented respiratory distress motivating an oxygen therapy at 10 L/min and a transfer to the cardiology department for adapted management.

The clinico-biological evolution was rapidly favorable with disappearance of chest pain and normalization of the biology at D7 under treatment with COLCHICINE (0.5 mg twice a day) introduced the 3rd after admission.

The serologies (HIV, HBV, HCV, EBV, CMV, Adenovirus, Picornavirus, Parvovirus B19), however, the nasopharyngeal smear COVID-19 was positive. We conclude to an acute peri-myocarditis with COVID-19.

DISCUSSION

The initial mechanism of SARS-CoV-2 infection is viral binding to the membrane bound form of angiotensin- converting enzyme 2 (ACE2) by a protein expressed in the viral coat, called SPIKE (S protein), followed by its priming by the serine protease TMPRSS2 mediating viral uptake [2]. CEA2 is a membrane-bound peptidase that is expressed in all tissues, but is particularly represented in lung, heart, vessel, kidney, brain, and intestine (Figure 3) [3].

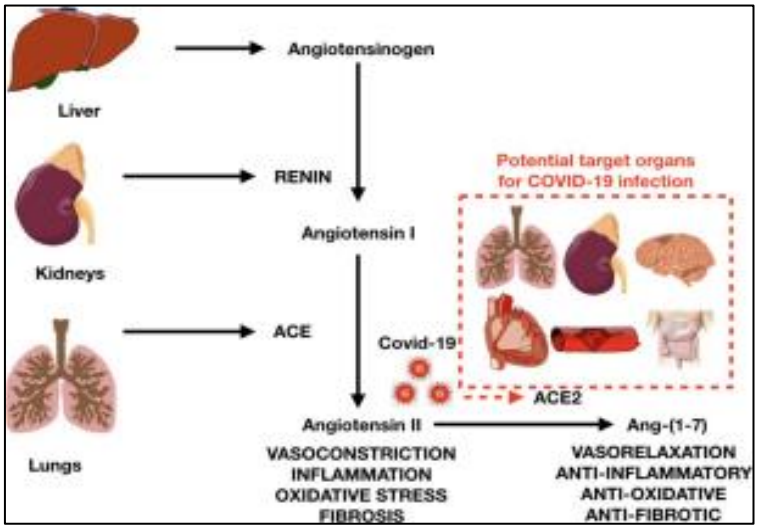

Figure 3: The renin-angiotensin system (RAS), ACE, and ACE2 (target of COVID 19) with potential target organs for COVID-19 infection (in the red dotted box)

On this basis, all of these organs are potential prime targets for the virus, which could explain several observations of pulmonary, cardiac, vascular, cerebral, and intestinal complications, as well as symptoms and signs related to the involvement of these organs during COVID-19.

Acute myo-pericarditis refers to a clinical picture in which the myocardium is more involved than the pericardium in contrast to the term peri-myocarditis [4]. The cardiac damage is often the result not only of a viral infection that directly necroses the myocardium but also the consequence, as described above, of an excessive immune response that is originally triggered by the organism to eliminate the infectious agent.

Currently for COVID-19 we do not know the mechanism(s) that lead to the inflammation of the heart. Enteroviruses have a direct tropism for cardiomyocytes which they "lyse" and have been implicated by this mechanism in cases of fulminant myocarditis. COVID-19 could infect cardiac cells in the same way and lyse them secondarily after replication of their RNA. The discovery of the virus in cardiomyocytes supports this hypothesis. Other viruses such as parvovirus B19 target cardiac vascular endothelial cells. Corona virus is thought to have similar properties as previously explained. The pathogenic mechanisms by which B19 exerts its action are multiple and may involve direct cytotoxicity on cellular proteins, activation of interleukin-6 (IL-6), production of tumor necrosis factors, as well as stimulation of apoptosis and autolysis [5]. Infection of endothelial cells is also associated with chronic disease and later development of dilated heart disease with impaired left ventricular (LV) function. The long-term consequences of COVID-19 are obviously not yet assessable, but the similarities between the 2 infections suggest that some patients will not emerge unscathed from their myocarditis. The most typical ECG appearance of peri-myocarditis is usually the appearance of nonspecific T-wave changes (negative T-waves) as might be seen in ACS or left ventricular hypertrophy [6].

In the context of pericardial disease, there are two different possible scenarios to consider: the patient treated for pericarditis who subsequently becomes infected with SARS-CoV-2, and the patient with COVID-19 who develops pericarditis or pericardial effusion. In both cases, clinicians may question the efficacy of nonsteroidal anti inflammatory drugs (NSAIDs), corticosteroids, colchicine, and biological agents such as anti-IL1 agents [7].

Currently, limited data have been published on COVID-19 cases that develop pericarditis and pericardial effusion. Most reported cases have been associated with myocardial involvement and troponin elevation.

Although there is a warning about the use of NSAIDs in the setting of COVID-19 infection that requires further investigation, most treatments for pericarditis, including corticosteroids, colchicine do not appear to be contraindicated in the setting of COVID-19 infection (Table 1) [7].

Nevertheless, when corticosteroids are used,

careful monitoring for possible superimposed bacterial infections is warranted. On this basis, therapies for pericarditis should not be discontinued in patients on therapy when indicated to control the disease. In fact, these agents are currently being tested for efficacy in COVID-19 infection and may soon be among the recognized treatment options [7].

The major limitation of this case report is the lack of virological analysis of pericardial fluid.

Table 1: Current treatments for pericarditis and COVID-19 infection

Drugs	Attack dose	Duration	NE	Effect on COVID-19
NSAIDS	Aspirin 750-1000mg x 3/dayIbuprofen 600-800mg x 3/day Indomethacin 25-50mg x 3/day	1-2 weeks but until resolution of symptoms and normalization of CRP	A	Harmful (?)
Colchicine	0.5mg x 2/day (0.5mg 7day if <70Kg)	3 months (acute) 6 months (recurrent)	A	Potential therapy
Corticosteroids	0.2 to 0.5mg/kg/day of prednisone	Up to 1 month	B	Therapy for advanced cases
Azathioprine	Up to 2 mg/kg	> 6 months	B	Unknown
IGHN	400 to 500mg/kg/day	5 days (can be repeated after 1 month)	B	Potential therapy

NSAIDs = nonsteroidal anti-inflammatory drugs; IGHN = immunoglobulin normal human; NE = Level of Evidence for pericarditis: A for multiple RCTs or meta-analyses, B for single RCT or observational studies, C for expert consensus; CRP = C-reactive protein

CONCLUSION

In conclusion, the pericardium is a potential target for COVID-19, but at present, there are no findings that provide evidence of direct infection and replication of SARS-CoV-2 in pericardial cells. Additional pathological studies and autopsy series will be very helpful in clarifying the potential of SARS-CoV-2 to directly infect the pericardium and cause pericarditis.

Patient Consent: The patient has given consent.

Conflicts of Interest: The authors declare no conflicts of interest.

Author Contributions

All authors contributed to the drafting of the manuscript, all authors read and approved the final version of the manuscript.

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