



Acute pericarditis in a patient with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection: a case report and review of the literature on SARS-CoV-2 cardiological manifestations

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Abstract: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), also known as coronavirus disease 2019 (COVID-19) is known to cause a cluster of flu-like illnesses and pneumonia with evolving understanding of other systemic manifestations. Currently, the known cardiac manifestations of COVID-19 include myocardial injury, acute coronary syndrome, and arrhythmias. In this report, we describe a case of pericarditis—an unusual cardiac manifestation observed in a patient with COVID-19. A 63-year-old male presented with history of fever, cough and chest pain. Electrocardiogram (EKG) demonstrated diffuse ST-T wave changes on all the leads, with normal troponin-T levels. Echocardiograph showed mild pericardial effusion without any regional wall motion abnormality. Subsequent chest radiograph and coronary angiography were normal. In view of ongoing COVID-19 pandemic, nasopharyngeal swab was performed, which was positive. Detailed etiological workup for pericarditis, including infectious and inflammatory causes were unremarkable. Viral pericarditis (possibly caused by COVID-19) was diagnosis of exclusion and patient was treated with hydroxychloroquine 200 mg twice a day, colchicine 0.5 mg twice a day, and lopinavir/ritonavir 200 mg/50 mg tablet twice a day for 10 days during admission. He was discharged with hydroxychloroquine 200 mg twice daily and colchicine 0.5 mg once daily for 15 days. On subsequent follow-up clinic visit, he reported resolution of symptoms. The purpose of this report is to add a potential cardiovascular complication of COVID-19 to the literature. Awareness of this manifestation can lead to timely laboratory and imaging examinations with potential to provide correct treatment and good outcome.

Keywords: Coronavirus disease 2019 (COVID-19); cardiovascular manifestations; case report; pericarditis; severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)

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Introduction

Coronavirus disease 2019 (COVID-19) is a rampant infectious disease of global public health concern, affecting over 15.5 million people affected in a span of 8 months (1). The WHO declared COVID-19 a pandemic in March 2020 (2). The International Committee on Taxonomy of Viruses named it severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) as it is similar to the coronavirus responsible for severe acute respiratory syndrome (SARS-CoV) (3). The SARS-CoV-2 is a novel enveloped RNA beta-corona virus which belongs to the same subgenus as the severe acute respiratory syndrome (SARS) virus but in a different clade (4). This virus binds with high affinity to the angiotensin-converting enzyme 2 (ACE2) receptor in humans like SARS-CoV (5).

The spectrum of symptomatic infection ranges from mild to critical (6). The clinical course of SARS-CoV-2 infection is usually characterized by fever, fatigue, myalgia and respiratory tract symptoms that include cough, pharyngodynia, and can progress to pneumonia and acute respiratory distress syndrome (6). Cardiovascular involvement in COVID-19 has recently been identified in literature, which includes myocardial injury, cardiomyopathy, myo-pericarditis and one reported case of cardiac tamponade as well (7-22). However, data in regards to impact of COVID-19 on the cardiovascular symptom is still evolving. In this report, we describe a case of pericardial complication in a patient affected with COVID-19. Though we were able to find several cases of myo-pericarditis in our review of literature, we did not come across any case with pericarditis as seen in our patient.

We present this case in accordance with the reporting CARE checklist (available at <http://dx.doi.org/10.21037/acr-20-90>).

Case presentation

A 63-years-old male patient presented to the emergency department with a history of fever, dry cough and malaise for a week, and chest pain for a day. He described chest pain as substernal, crushing type, moderate to severe in intensity, and not associated with exertion. His past medical history was notable for fairly controlled hypertension for the past 12 years for which he was on lisinopril 5 mg twice daily. He reported no history of type II diabetes mellitus, coronary artery disease or any other chronic illness. No family history

of cardiac disease. On arrival, he was febrile with oral temperature of 38.4 °C (101.2 °F), pulse was 100/min and blood pressure was 96/62 mmHg. Respiratory rate (14/min) and oxygen saturation (99% on room air). Cardiopulmonary and abdominal examinations were unremarkable. The electrocardiogram (EKG) on presentation showed diffuse concave ST-segment elevations and PR segment depression (*Figure 1*). The troponin-T level was within a normal range. Bedside transthoracic echocardiography (TTE) showed small, circumferential (7 mm) pericardial effusion and mild left ventricular hypertrophy with normal left ventricular ejection fraction and no wall motion abnormality (*Figure 2*). The chest radiograph showed no abnormality. Arterial blood gas showed a pH of 7.40, the partial pressure of oxygen (PO₂) of 90 mmHg, the partial pressure of carbon dioxide (PCO₂) of 40 mmHg, bicarbonate of 22 mmol/L, and lactate of 1.4 mmol/L (mildly elevated).

Initial blood workup including complete blood count, electrolytes, blood glucose, renal and liver functions were within normal limits. Erythrocyte sedimentation rate was 11 mm/h and the C-reactive protein level was 5 mg/dL.

The patient was admitted to an intensive care unit and managed for the provisional diagnosis of acute pericarditis. Coronary angiography was performed which showed no significant stenotic lesion. Work-up for other possible causes of pericarditis including HIV, bacterial, tuberculous, and auto-immune etiologies was negative. Apart from C-reactive protein, other inflammatory markers were unremarkable. The patient was initially managed with intravenous fluid, colchicine 0.5 mg twice daily, and aspirin 325 mg thrice daily for 3 days. The nasopharyngeal swab test for SARS-CoV-2 (reverse transcription polymerase chain reaction) turned out to be positive on day 3 of admission. Subsequently, the patient was diagnosed with viral pericarditis from COVID-19 infection. The patient was then initiated on a trial of Hydroxychloroquine 200 mg twice a day and lopinavir/ritonavir 200 mg/50 mg tablet twice a day from the day 3 of admission. Following initial treatment of 2 days, his fever subsided, blood pressure improved, and chest pain reduced in intensity.

Repeat testing for SARS-CoV-2 was positive on the day 6 of admission. TTE was also repeated 1 week after admission which showed a reduction in the size of pericardial effusion. During the following days of his admission, he remained asymptomatic and the hospital course was uneventful. On the day 10 of his admission, repeat COVID-19 test was negative. The patient was discharged home on the day

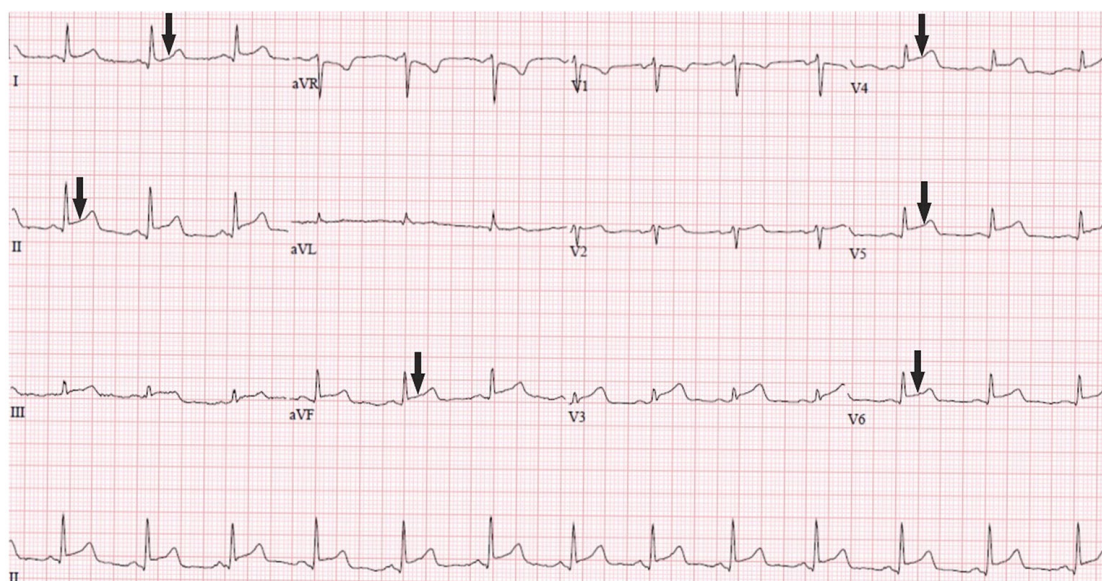


Figure 1 Electrocardiogram showing sinus rhythm, HR-84/min, normal axis, diffuse concave ST segment elevation (indicated by the arrows) with PR segment depression consistent with stage-1 pericarditis = acute phase. HR, heart rate.



Figure 2 Echocardiogram parasternal long-axis view (PLAX) view showing normal sized left ventricle, left atrium and aorta, mild concentric hypertrophy consistent with history of long-standing hypertension and small circumferential pericardial effusion (indicated by the arrow).

12 with empiric treatment including Hydroxychloroquine 200 mg twice a day and colchicine 0.5 mg once a day for the total duration of 15 days and was scheduled for a follow-up clinic visit.

On follow-up visit in 2 weeks, patient reported resolution of the symptoms and repeat EKG and chest radiograph

were normal.

The timeline of the important events is described in the *Figure 3*.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient.

Discussion

To the best of our knowledge, this is the first reported case of isolated pericardial involvement in COVID-19 patients without known myocardial injury or elevated troponin levels. However, it is to be noted, that this was a diagnosis of exclusion and other etiological workup including HIV, bacterial, tuberculous, and auto-immune panel was negative.

We did an extensive literature search and review and to date 157 studies on COVID-19 with cardiovascular involvement have been published. Studies focusing on non-human researches, reviews, clinical trials, laboratory investigations and comorbidities were excluded, and 17 studies (total 1,782 patients) focusing on cardiac complications were analysed (*Table 1*). Acute cardiac complications were reported in 786 patients. The most prevalent cardiac complications included myocardial

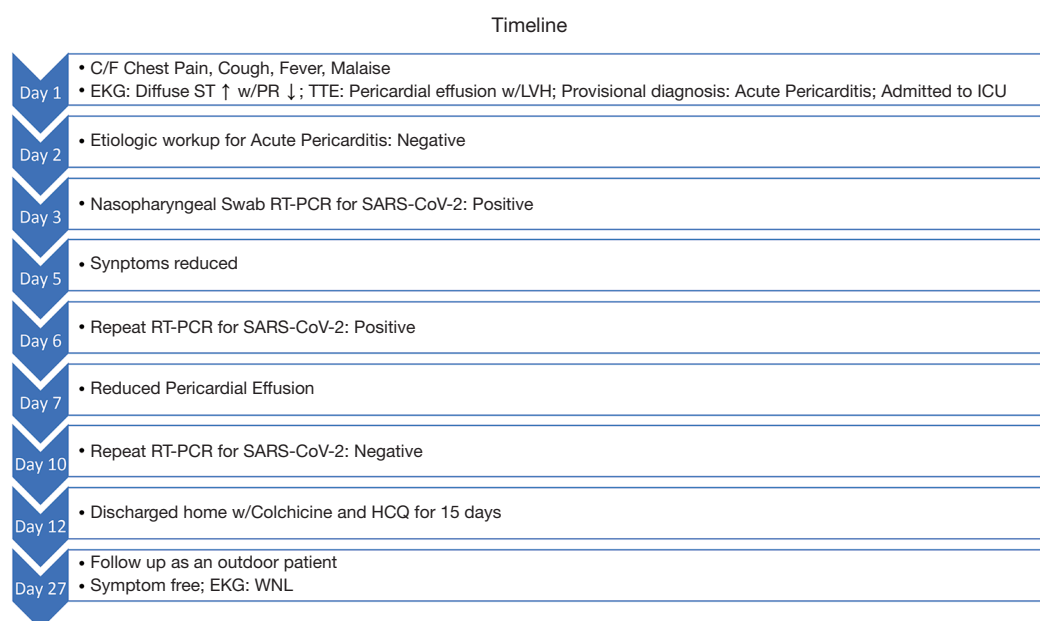


Figure 3 Timeline of the important events. C/F, clinical features; EKG, electrocardiogram; TTE, transthoracic echocardiogram; LVH, left ventricular hypertrophy; ICU, intensive care unit; RT-PCR, reverse transcription polymerase chain reaction; COVID-19, coronavirus disease 2019; HCQ, hydroxychloroquine; WNL, within normal limits.

injury (59.3%), heart failure (30.5%), arrhythmia (9.3%) and cardiomyopathy (0.8%). The majority of the patients presented with myocardial injury (59.3%), only two patients (0.25%) had involvement of pericardium along with myocardium (*Table 1*).

Our report and reported literature suggest that although the patients usually present with various respiratory symptoms, cardiac involvement may occur with COVID-19 even without significant respiratory tract signs and symptoms of infection (23). The underlying pathophysiology is not yet well established, but is thought to be secondary to modulation of ACE2 by COVID-19, which is expressed in the heart, esophagus, kidney, bladder and ileum as well as the alveoli (24). Majority of the reported patients with severe SARS-CoV-2 infection were old aged and with comorbidities as in this patient. Therefore, it is difficult to consider any single comorbid condition as an independent risk factor unless more detailed analysis on the subject becomes available. Use of ACE inhibitors have also

been described to be associated with higher risk of SARS-CoV-2 infection (25).

The goal of this report is to add value to the literature about cardiovascular manifestations of the novel COVID-19, recognition of which can help in the timely management of such patients to reduce the mortality related to such complications in COVID-19. Spreading awareness of this infrequent pericardial manifestation associated with COVID-19 can be helpful in triaging patients, preventing unnecessary testing, and instituting appropriate treatment.

The main limitation of the study includes, presentation of a single case of pericarditis in a patient with COVID-19, and the causality was not well established and COVID-19 induced pericarditis was the diagnosis of exclusion. However, this is the first reported case of pericardial involvement in COVID-19 patients without known myocardial injury. The main goal of this report is to create awareness about pericarditis as a possible complication of COVID-19.

Table 1 A literature review containing 17 studies of COVID-19 patients with cardiovascular complications

Publication	No. of patients	Age (years)	Comorbidities				Complications					Elevated cardiac biomarkers		Mortality (%)	Treatments given				
	Total/male/female		HTN (%)	CAD (%)	CMP (%)	DMT2 (%)	Myocardial injury (%)	Pericarditis (%)	CMP (%)	Heart failure (%)	Arrhythmia (%)	Troponin (%)	NT-pro-BNP (%)		HCQ (%)	Antiviral (%)	Antibiotic (%)	Glucocorticoid (%)	Ig (%)
Guo <i>et al.</i> (8)	187/91/96	58.5*	61 (32.6)	21 (11.2)	8 (4.3)	28 (15.0)	52 (27.8)	–	–	–	11 (5.9)	52 (27.8)	–	43 (23.0)	–	166 (88.8)	183 (97.9)	106 (56.7)	21 (11.2)
Inciardi <i>et al.</i> (9)	1/0/1	53	–	–	–	–	1 (myopericarditis) (100.0)	–	–	–	–	1 (100.0)	1 (100.0)	0	1 (100.0)	1 (100.0)	–	1 (100.0)	–
Tavazzi <i>et al.</i> (10)	1/1/0	69	–	–	–	–	1 (100.0)	–	–	–	–	1 (100.0)	–	–	–	–	–	–	–
Dong <i>et al.</i> (11)	4/4/0	43.2*	1 (25.0)	–	–	2 (50.0)	1 (25.0)	–	2 (50)	4 (100.0)	2 (50.0)	3 (75.0)	4 (100.0)	2 (50.0)	–	1 (25.0)	3 (75.0)	0 (0)	1 (25.0)
Huang <i>et al.</i> (6)	41/30/11	49**	6 (14.6)	6 (14.6)	–	8 (19.5)	5 (12.2)	–	–	–	–	5 (12.2)	–	6 (14.6)	–	38 (92.7)	41 (100.0)	9 (22.0)	–
Kim <i>et al.</i> (12)	1/0/1	21	–	–	–	–	1 (100.0)	–	–	–	–	1 (100.0)	1 (100.0)	–	–	–	–	–	–
Chadha <i>et al.</i> (13)	1/0/1	85	–	–	–	–	–	–	1 (takotsubo) (100.0)	–	–	1 (100.0)	–	0	–	–	–	–	–
Chen <i>et al.</i> (14)	274/171/103	62**	93 (33.9)	23 (8.4)	–	47 (17.2)	203 (74.1)	–	–	176 (64.2)	–	203 (74.1)	173 (63.1)	113 (41.2)	–	236 (86.1)	249 (90.9)	217 (79.2)	54 (19.7)
Fried <i>et al.</i> (15)	4/2/2	54*	2 (50.0)	–	1 (25.0)	2 (50.0)	4 (100.0)	–	1 (25.0)	2 (50.0)	2 (50.0)	4 (100.0)	2 (50.0)	0	4 (100.0)	–	2 (50.0)	–	–
Han <i>et al.</i> (16)	273/97/176	58*	–	–	–	–	34 (12.5)	–	–	–	–	27 (9.9)	34 (12.5)	24 (8.8)	–	–	–	–	–
Sala <i>et al.</i> (17)	1/0/1	43	–	–	–	–	1 (100.0)	–	1 (reverse takotsubo) (100.0)	–	–	1 (100.0)	1 (100.0)	–	1 (100.0)	1 (100.0)	–	–	–
Wang <i>et al.</i> (7)	138/75/63	56**	43 (31.2)	20 (14.5)	–	14 (10.1)	10 (7.2)	–	–	–	23 (16.7)	0	–	6 (4.3)	–	124 (89.9)	89 (64.5)	62 (44.9)	–
Shi <i>et al.</i> (18)	416/205/211	64**	127 (30.5)	44 (10.6)	17 (4.1)	60 (14.4)	82 (19.7)	–	–	–	–	–	–	57 (13.7)	–	–	–	–	–
Wang <i>et al.</i> (19)	339/166/173	71*	138 (40.7)	48 (14.2)	–	54 (15.9)	70 (20.6)	–	–	58 (17.1)	35(10.3)	–	–	65 (19.2)	–	–	–	–	–
Hua <i>et al.</i> (20)	1/0/1	47	–	–	–	–	1 (with tamponade) (100.0)	–	–	–	–	1 (100.0)	–	–	–	–	–	–	–
Dabbagh <i>et al.</i> (21)	1/0/1	67	–	–	1 (100.0)	–	–	–	1 (takotsubo) (100.0)	–	–	–	1 (100.0)	–	1 (100.0)	–	–	1 (100.0)	–
Zheng <i>et al.</i> (22)	99/51/48	49**	21 (21.2)	–	–	–	–	–	–	–	–	88 (88.9)	84 (84.8)	–	–	–	–	–	–
Total	1,782/893/889	–	492 (27.6)	183 (10.3)	27 (1.5)	215 (12.1)	466/786 (59.3)	2/786 (0.25)	6/786 (0.8)	240/786 (30.5)	73/786 (9.28)	388 (21.8)	316 (17.7)	316 (17.7)	7 (0.4)	401 (22.5)	567 (31.8)	396 (22.2)	76 (4.3)

* represents mean and ** represents median. It is to be noted that 786 out of 1,782 patients presented with cardiovascular complications. Hypertension was the most observed pre-existing co-morbidity. Most common cardiac complication was myocardial injury, only two patients showed pericardial involvement along with myocardial injury. Majority of the patients with myocardial injury had elevated cardiac biomarkers. HTN, hypertension; CAD, coronary artery disease; DMT2, diabetes mellitus type 2; CMP, cardiomyopathy; HCQ, hydroxychloroquine; Ig, immunoglobulin.

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Footnote

Reporting Checklist: The authors have completed the CARE checklist. Available at <http://dx.doi.org/10.21037/acr-20-90>

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at <http://dx.doi.org/10.21037/acr-20-90>). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient.

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