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Case Presentation

Covid-19 and Acute Pericarditis: A Case Report and Review of the Literature

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Abstract

Cardiovascular manifestations of Coronavirus 2019 infection include venous thromboembolism, acute coronary injury associated or not-associated with obstructive coronary artery disease, and acute inflammatory heart diseases (myocarditis and pericarditis). All these complications may occur in the presence or not of lung involvement, well known as the most common presentation in symptomatic patients.

We report the case of a 61-years-old female, with a positive swab for SARS-CoV-2 and acute pericarditis, in the absence of lung involvement.

In addition, we reviewed the available literature on pericardial involvement in patients with COVID-19, searching on the most common electronic databases/ search engines.

Keywords: Acute Pericarditis; SARS-CoV-2; COVID-19

Introduction

The Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is the causative agent of the respiratory disease later named Coronavirus Disease 2019 (COVID-19), remaining a world public health emergency at present. The disease is mild in most people, but in some, especially the elderly and those with comorbidities, it may progress to more severe pictures, often represented by pneumonia, until Acute Respiratory Distress Syndrome (ARDS) and Multiple Organ Dysfunction Syndrome (MODS) [1].

Although the major part of symptomatic patients presents with respiratory disease, extra-pulmonary manifestations have been described, including neurological, cardiac, and hypercoagulable complications [2].

Cardiovascular manifestations of SARS-CoV-2 infection are represented by venous thromboembolism, acute coronary injury associated or not associated with obstructive coronary artery disease, and acute inflammatory heart diseases (myocarditis and/or pericarditis). The set of such disorders has been renamed as Acute COVID-19 Cardiovascular Syndrome (ACovCS), including also heart failure until cardiogenic shock and arrhythmia, that are a consequence of a cardiac damage of different origins [2].

On the basis of the current evidences, two clinical patterns of ACovCS have been proposed: 1) "mixed pulmonary and cardiac", more common, occurring in 10-25% of patients admitted to hospital, associated to typical pulmonary predominate symptoms [2,3]; 2) "predominate cardiac", observed in <5% of patients hospitalized with COVID-19, in which pulmonary involvement is mild or absent [4,5].

About acute inflammatory diseases of the heart associated to SARS-CoV-2 infection, many cases of acute myocarditis have been reported. All of them showed the presence of a significant systolic cardiac dysfunction, sometimes with fulminant course, troponin elevation and generally absence of pericardial effusion [6-10]. In a retrospective study assessing the clinical predictors of mortality on 150 patients from China, acute myocarditis was recognized as the cause of death in some COVID-19 patients, among the 40% died for cardiovascular failure associated or not to respiratory one [4]. More than one review articles have been recently published, analyzing the possible pathophysiology of COVID-19-related acute myocarditis, and proposing guidelines for its diagnosis and management [2,11,12].

Less it known on Acute Pericarditis (AP) associated to COVID-19. Etiology of AP has been categorized as infectious or noninfectious. Tuberculosis is a common causative agent in developing countries, but accounts for < 5% of cases in developed ones, where presumed viral causes are involved in 80%-90% [13,14]. The established clinical criteria for the diagnosis of AP are showed in Table 1.

In this article, we presented a new case of AP in a patient with SARS-CoV-2 infection, and we reviewed the available literature on this topic. We searched 3 electronic databases/search engines including PubMed, Web of Science, and Scopus until June 2020. We utilized the following search string: ("COVID-19" or "SARS-CoV-2" or "Coronavirus 2019" or "Coronavirus 2") and ("Pericardial" or "Pericarditis" or "Myopericarditis" or "Tamponade"). Furthermore, we conducted a manual search by checking the bibliography within each of the included studies, and the related references in PubMed and Google Scholar.

Case Presentation

A 61-years-old Caucasian woman presented to the emergency room on April 5, 2020, with fever for three weeks, associated with pharyngitis and worsening dyspnoea, orthopnoea, and asthenia for the following week. Medical history was remarkable for hypothyroidism and recurring episodes of kidney stones.

On the advice of family physician, she underwent a nasopharyngeal swab for SARS-CoV-2 resulting positive on real-time reverse transcriptase–polymerase chain reaction assay, then she was

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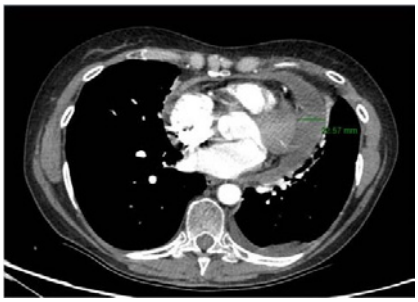

Figure 1a: The chest Computed Tomography (CT) angiography performed on arrival in the emergency room showed the pleuro-pericardial effusion.

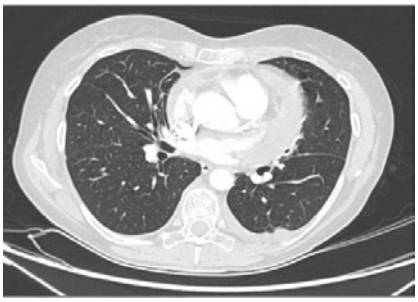

Figure 1b: In the absence of COVID-19-related lung involvement.

referred to our emergency department.

On arrival, blood pressure was 124/82 mmHg, heart rate 129 beats per minute, body temperature 37 °C, respiratory rate 18 breaths/minute, and arterial oxygen saturation (SaO2) of 95% while breathing ambient air. Physical examination was relevant for reduced heart sounds and pericardial friction rubs.

A12-lead Electrocardiogram (ECG) showed sinus tachycardia and diffuse aspecific T-wave abnormalities.

Blood tests were significant for C Reactive Protein (CRP) 137.1 mg/dL, a platelet count of 891.000/mmc, fibrinogen 887 mg/dL, IL-6 8.4 ng/mL and D-Dimer 12904 ng/mL. Procalcitonin, N-terminal prohormone of brain natriuretic peptide (NT-proBNP) and High Sensitivity troponin I were normal.

A chest Computed Tomography (CT) angiography ruled out either pulmonary embolism or pneumonia, but showed either left pleural or circumferential pericardial effusion (with maximum width of 22 mm associated with a slight thickening of the pericardial leaflets, Figure 1).

The cardiologist consultant did not perform urgent

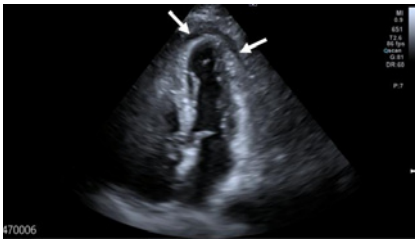

Figure 2: The cardiac ultrasound (apical window, day 10) showed pericardial effusion in the resolution phase, associated with the presence of fibrin deposits; (arrow: pericardial effusion).

echocardiography since the absence of hemodynamic instability, suggesting to assess it in the following days.

Thus, the patient was admitted to our Sub-intensive Care Unit, and a non-invasive continuous multi-parametric monitoring was started. The pharmacological treatment consisted of hydroxychloroquine 200 mg bid, azithromycin 500 mg qd, colchicine 0.5 mg bid, ibuprofen 600 mg tid, bisoprolol 1.25 mg bid PO.

Dyspnoea, orthopnoea, and asthenia quickly and stably disappeared. From day 4 to day 9 she reported several episodes of diarrhoea associated to diffuse abdominal discomfort.

Blood pressure and SaO2 maintained normal. Heart rate decreased in few days to a mean of 80 beats per minute, and no arrhythmia was detected at monitoring. Body temperature remains within the normal limits.

The control ECG resulted normal.

A nasal and pharyngeal swab for SARS-CoV-2 was repeated 9 days after admission, resulting negative.

Hydroxychloroquine and azithromycin were discontinued at day 7 (also because of gastrointestinal symptoms). Diarrhoea and abdominal discomfort permanently disappeared since day 12.

During hospitalization, procalcitonin and troponin remained normal, CRP (last value 57.9 mg/l) haemoglobin (last value 10.8 g/dL) and platelets count (last value 523.000/mmc) decreased, neutrophilia and relative lymphopenia normalized. Screening for either autoimmune disorders or other infectious agents was negative: ANA, anti-ENA SSB/La, anti-dsDNA, pANCA, cANCA, LAC and Rheumatoid Factor; Legionella urinary antigen, Quantiferon test and blood cultures were negative; serological tests for Influenza H1N1 and B, Parainfluenza type 1-2-3, Adenovirus, Coxsackie virus, Respiratory Syncytial virus, Legionella pneumophila, Chlamydia pneumoniae, Mycoplasma pneumoniae, Echovirus, Coxiella burnetii, Hepatitis B and Hepatitis C, Cytomegalovirus and Epstein-Barr virus.

Table 1: Clinical established criteria for the diagnosis of acute pericarditis [*].

≥ 2 of the following clinical criteria requie for the diagnosis:	
1.	Chest pain (typically sharp and pleuritic, improved by sitting up and leaning forward)
2.	Pericardial friction rubs
3.	Suggestive changes on electrocardiography (widespread ST-segment elevation or PR depression)
4.	New or worsening pericardial effusion
Elevation of markers of inflammation (eg, C-reactive protein) is an additional supportive criteria, to enforce diagnosis.	

[*] Adapted by Imazio et al, JAMA. 2015;314(14):1498-506.

Table 2: Principal characteristics of published cases of AP associated with COVID-19.

Reference	Age, Sex	Cardiac Tamponade	Acute respiratory failure	COVID-19 related lung involvement	Troponin increase	Left ventricular systolic dysfunction	Treatment for AP	Outcome
Dabbagh MF et al., JACC Case Rep	F, 67 y	Yes	No	No (CT)	No	No (EF 40% as previous control)	Yes (steroids + colchicine)	Positive
Farina A et al., Eur J Intern Med	F, 59 y	Yes	Yes	Yes (CT: ground glass - crazy paving)	Yes	No	No	Positive
Marschall A et al., Emergencias	M, 35 y	Yes	Yes	Yes (x ray: interstitial bilateral involvement)	No	No	Yes (aspirin + colchicine)	Positive
Khalid N et al., Cardiovasc Revasc Medicine	M, 35 y	Yes	No	No (x ray)	Yes	Yes (EF 25%)	Yes (steroids + colchicine)	Positive
Asif T et al., EJCRIM	F, 70 y	Yes	Yes	Yes (x ray: bilateral pulmonary infiltrates)	Not specified	No	Yes (colchicine)	Positive
Yale Tung-Chen Y et al., Med Clin	F, 35 y	No	No	Yes (US: B-lines - subpleural consolidation)	No	No	Yes (colchicine)	Positive
Cizgici AY et al., Am J Emerg Med	M, 78 y	No	Yes	Yes (CT: ground glass opacification)	Yes	Not assessed	Not specified	Positive
Inciardi RM et al., JAMA Cardiology	F, 53 y	No	No	No (x ray)	Yes	Yes (EF 35%)	Yes (aspirin + steroids)	Positive

A transthoracic echocardiography (day 10, Figure 2) showed: circumferential pericardial effusion (maximum, 8 mm postero-laterally) without signs of tamponade; normal left ventricular dimensions, wall thickness, left ventricular diastolic function, estimated left ventricular ejection fraction, without regional hypokinesis; no evidence of heart valve disease.

The patient was discharged finally at day 16, in good general conditions and with no symptoms.

Discussion and Review of the Literature

We described a new case of AP associated to SARS-CoV-2 infection in a patient without previous significant cardiovascular and systemic diseases. All known potential causes of pericarditis were investigated and excluded. Neither respiratory failure nor significant pulmonary involvements at CT scan were observed in our patient. Unfortunately analysis of the pericardial effusion was not performed, as the risks of the procedure seemed to be higher than the potential benefits. Standard treatment for AP (NonSteroidal Anti-Inflammatory Drugs - NSAIDs, and colchicine) was effective and safe in our case.

Few but interesting data are available at present about perica rdial involvement in SARS-CoV-2 infection. A study on chest CT performed at admission to hospital in 90 consecutive patients with COVID-19 showed that pericardial effusion is present in a minority of cases only (1%) [15].

Another study assessing clinical and chest CT features associated with COVID-19 pneumonia showed that 6% out of 83 patients reported chest pain and pericardial effusion was found in 4 patients (4.8%), all in the group of 25 severe/critical cases [16].

A review of autopsies of 23 patients with COVID-19 from USA showed 3 cases of lymphocytic pericarditis. Interestingly there was evidence of myocardial injury too in some patients, however without evidence of inflammatory infiltrate indicative of myocarditis except one case [17].

Other 9 case reports of AP associated to SARS-CoV-2 infection have been published at present [18-26]. The principal characteristics of these patients are summarized in Table 2.

Patients were more often female and with age < 65 years, as in our case. Six (6) out of 9 presented with or rapidly evolved towards a clinical picture of cardiac tamponade, than requiring emergent pericardiocentesis [18-23].

One or more among troponin increase, myocardial dysfunction at cardiac ultrasound, signs of myocardial inflammation at cardiac magnetic resonance, suggested concomitant myo-pericarditis, that was detectable in 5 cases [19-21,25,26].

The "predominate cardiac" pattern of ACovCS was more common in this patients series. In fact, acute respiratory failure associated to different degrees of lung involvement at chest CT, radiography or ultrasonography, was found in only 4 patients [19,21,23,25].

The pathophysiology of AP associated to COVID-19 is far to be clarified. The possible mechanisms are similar to those proposed for myocarditis. SARS-CoV-2 infects the human host by entering the airways and binding the Angiotensin-Converting Enzyme 2 (ACE2) receptors [1]. ACE2 receptors are membrane-bound aminopeptidases highly expressed in type II pneumocytes. For this reason, the virus shows a specific tropism for the lungs, with a well-known wide spectrum of clinical severity [1]. However, ACE2 receptors are expressed by several other human cells too: pericytes, cardiomyocytes, enterocytes in the small intestine and arterial and venous endothelial cells [2]. SARS-CoV-2 could pass, through the blood or via the lymphatic system, from the respiratory tract to the heart. Acute cellular injury due to ACE2 receptor-mediated SARS-COV-2 infection of cardiomyocyte, pericyte or fibroblast, leading to acute myocarditis and/or pericarditis, is a theoretical but unproven event [2]. An endomyocardial biopsy performed in a patient with COVID-19 presenting with acute myocardial injury and severe acute

systolic heart failure showed low grade myocardial inflammation in the absence of necrosis, with localization of SARS-CoV-2 within macrophages, but not cardiomyocytes [2]. Interestingly SARS-CoV-2 was detected, by rRT-PCR amplification of RNA, also in the pericardial fluid of a patients presenting with cardiac tamponade requiring emergent pericardiocentesis [19]. These findings are proof that SARS-CoV-2 can be found within the heart, but did not provide evidence for entry and replication of the virus within heart cells.

A second hypothesis refers to the intense inflammatory activation and the consequent release of cytokines observed in some patients with SARS-CoV-2 infection. The pathogenesis of tissue damage, inside and outside the lungs, seems to be related to the grade of immune-inflammatory response to viral infection rather than to viral replication per se [27-30]. In fact, SARS-CoV-2 activates the innate immune system; macrophages and other innate immune cells not only capture the virus, but also release several cytokines and chemokines. Adaptive immunity is also activated by antigen presenting cells. T cells and B cells not only play an antiviral role, but also directly or indirectly promote the secretion of inflammatory cytokines [29,30]. It has been clearly demonstrated that the clinical severity of COVID-19 is strictly related to the intensity of this response. In fact, generally in more severe cases of COVID-19, a real Cytokine Release Syndrome (CRS) occurs [29,30]. CRS is a systemic inflammatory response, which can be caused by infectious or non-infectious triggers, characterized by a sharp increase of a large number of pro-inflammatory cytokines such as IL-6, IL-10, IL-2 and IFN-γ, either in the target organs, such as lungs, or in the blood stream [27-30]. The significant increase in these pro-inflammatory molecules is high probable to play a role in clinical manifestations of COVID-19, cardiovascular ones among others.

The intense systemic immune-inflammatory activation in these patients well correlates with the strong increase of inflammatory markers (CRP in first place) generally observed in acute inflammatory cardiac diseases, such as myocarditis and pericarditis.

This second potential pathophysiologic mechanism could help to explain why, in the published cases, steroidal or non-steroidal anti-inflammatory drugs, both with colchicine, when used, revealed to be effective and safe to treat AP associated to SARS-CoV-2 infection. Some considerations on treatment options for AP at the time of COVID-19 could be useful in our opinion. Anecdotal warning diffused, strongly promoted by social media, that NSAIDs could aggravate COVID-19 disease. Although currently no real scientific evidence exists on this association [31], doctors might be led to avoid these drugs in AP, although they are reported by guidelines as its first line treatment [13,14]. Unless there are absolute contraindications to their use in the single patient, NSAIDs should be the treatment of choice, and steroids only a reserve. Steroids could become a valid option when NSAIDs are contraindicated, or a concomitant ARDS occurs [13,14,32]. Colchicine, that was the most common drug administered to the cases reported in this paper, could be endowed with adjunctive advantages in AP associated to COVID-19. In fact, colchicine accumulates in granulocytes and monocytes with ensuing anti-inflammatory effects [13], thus being potentially able to modulate the systemic immune-inflammatory response to SARS-CoV-2 infection. Four randomized studies regarding colchicine in COVID-19 patients have been recently announced, in different clinical settings and with different end-points [33]. Such trials could be able to clarify if colchicine may affect clinical course of COVID-19, especially to prevent or affect pulmonary and cardiovascular complications.

Finally, therapies attempting to limit SARS-CoV-2 replication (such as hydroxychloroquine and/or lopinavir/ritonavir) have been used in some of the cases reviewed in our paper. However, there is no real evidence at present that such therapies have clinically supported efficacy for COVID-19 in general, or specifically for patients with one are more manifestations of ACovCS [2].

Conclusion

Our case and the present literature data suggest that AP, although rarely, may represent a cardiovascular complication of COVID-19, in combination or not with myocarditis and/or lung involvement. A cardiac tamponade occurred in more than half of cases. So, when faced with a patient with COVID-19 and either tachycardia or arterial hypotension, especially if acute respiratory failure or other complications have been ruled out, a severe pericardial effusion should always be assessed. Plausible pathogenic mechanisms behind the association between the SARS-CoV-2 infection and AP could be the passage of the virus from the respiratory tract to the heart with direct damage on cardiac cells, or the strong systemic immune-inflammatory response to the infection observed in some patients. Basing on actual evidences, there is no reason to do not use well known effective treatment suggested by guidelines for AP, such as NSAIDS or steroids plus colchicine. Studies on large samples of COVID-19 patients are welcome to assess the real incidence of AP, its diagnostic and prognostic characteristics, and the best treatment options.

Ethical Standards

The patient gave the informed consent prior to the inclusion in the study.

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