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CASE REPORT

Myocardial lesions during Severe Acute Respiratory Syndrome Coronavirus 2 infection

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ABSTRACT
The Severe Acute Respiratory Syndrome Coronavirus 2 virus has a strong tropism for the cardiovascular system, with direct or indirect interactions mediated by inflammation. The virus can cause cardiovascular lesions that can compromise the prognosis. We report the case of a 43-year-old patient with no particular medical history, admitted with acute respiratory distress syndrome due to coronavirus disease 2019 pneumonia complicated by myocardial injury, whose condition gradually improved with apyrexia, normalization of troponins, inflammatory markers, and restoration of left ventricular function. This case report demonstrates the need to identify cardiac involvement through widespread use of echocardiography and close monitoring of cardiac and inflammatory biomarkers during coronavirus disease 2019 infection, given the prognostic implications of such involvement.

Introduction

The Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection, also known as Coronavirus Disease 2019 COVID-19, originated in China in December 2019. It can cause potentially fatal pneumonia. The World Health Organization (WHO) declared the infection a pandemic on March 11, 2020. As of April 1, 2020, the WHO reported 911,541 confirmed cases of COVID-19 and 45,532 deaths[1,2].

Clinical manifestations are dominated by respiratory symptoms ranging from mild cough or laryngeal discomfort to Acute Respiratory Distress Syndrome (ARDS). Cardiac involvement in COVID is present and severe, as well as the elevation of biomarkers, which is an important characteristic of COVID-19 and is associated with a poor prognosis. The virus has a dual effect on the cardiovascular system: the infection is more intense in individuals with cardiovascular comorbidities, and on top of that, the virus itself can cause potentially deadly cardiovascular lesions[3]. The most frequently observed cardiovascular complications in COVID-19 infections include myocardial injury (elevated troponins), myocarditis, acute coronary syndromes, arrhythmias, venous thromboembolic complications, and heart failure[4].

We present the case of a 43-year-old female patient with no significant medical history who received proper vaccination against SARS-CoV-2. However, she developed acute myocardial injury in the context of acute respiratory distress following a COVID-19 infection. This rare occurrence highlights the need for monitoring cardiac biomarkers and conducting regular echocardiographic evaluations to diagnose and track the progression of myocardial involvement in individuals with no prior cardiovascular history during a COVID-19 respiratory infection.

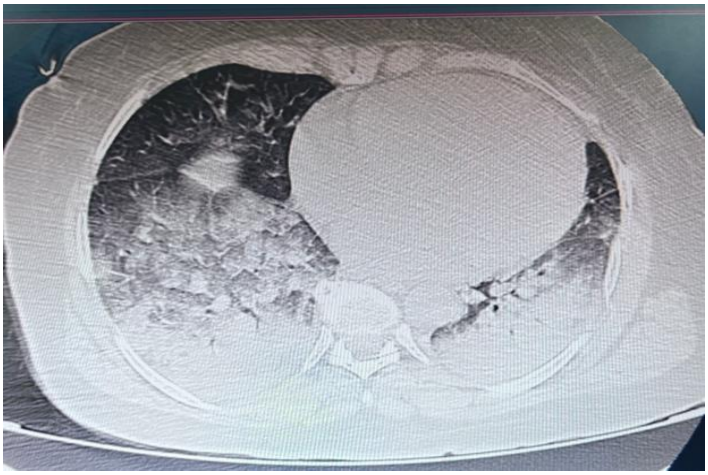
Case Report:

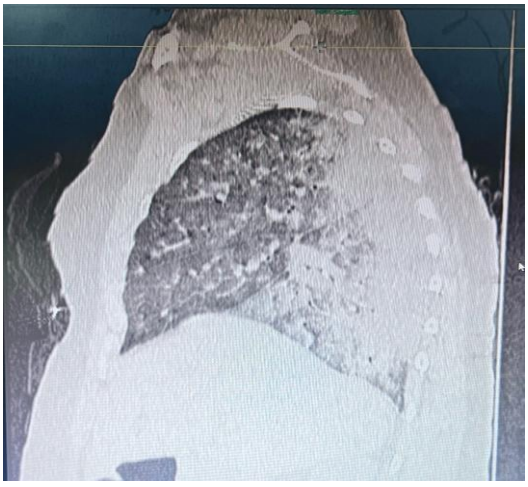
A 43-year-old woman with no significant medical history was admitted to the emergency department with acute respiratory distress. In the 7 days prior to admission, the patient had exhibited viral symptoms (dry cough, subfebrile state, and asthenia), as well as dyspnea at rest without chest pain, evolving in the context of low-grade fever and worsening general condition. On clinical examination at admission, the patient was restless with a Glasgow Coma Scale score of 14, isocoric and reactive pupils, no sensory or motor deficits, tachycardic at 120 beats per minute, blood pressure at 100/70 mmHg, polypneic at 30 cycles/minute, and oxygen saturation (SpO2) of 70% in ambient air. The chest was symmetric with signs of respiratory distress, diffuse coarse crackles were heard on auscultation bilaterally. The patient's temperature was 38.5°C. The patient rapidly deteriorated hemodynamically, developing cardiogenic shock necessitating endotracheal intubation and mechanical ventilation with the use of vasoactive drugs (dobutamine and norepinephrine).

The electrocardiogram showed regular sinus rhythm at 94 beats per minute, normal heart axis, present P wave, constant PR interval, narrow QRS complex, isoelectric ST segment, and asymmetrical positive T wave.

The initial cardiac ultrasound revealed a non-dilated left ventricle with globally impaired systolic function related to a global hypokinesia. The left ventricular ejection fraction was 20%, and left ventricular filling pressures were slightly elevated. No pericardial effusion or valvulopathy was observed.

The thoracic CT scan showed multiple areas of condensation and diffuse ground-glass opacities in both lung fields, predominantly distributed in the center, and containing aeric bronchograms.

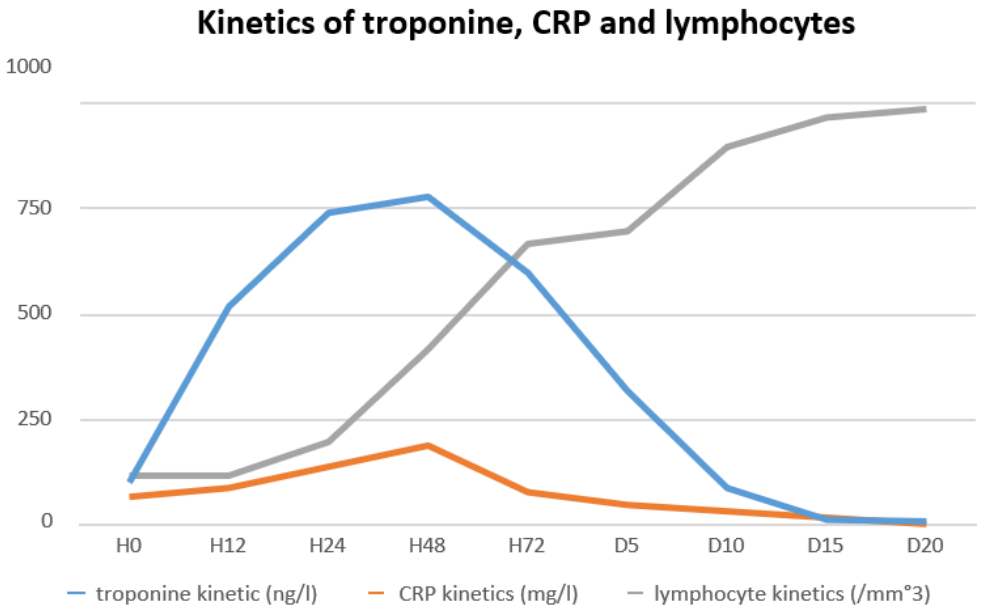


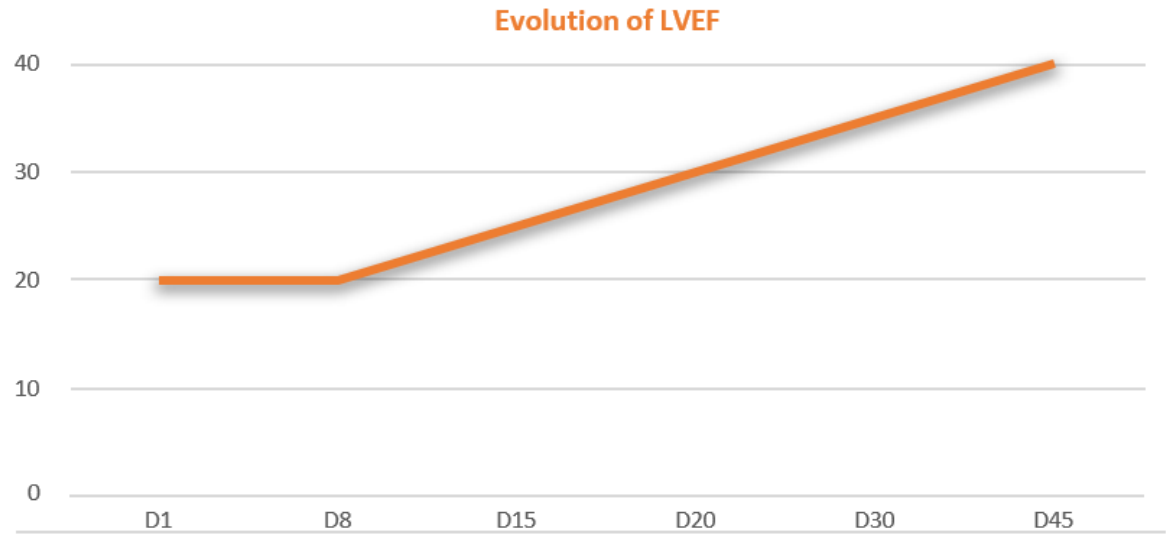


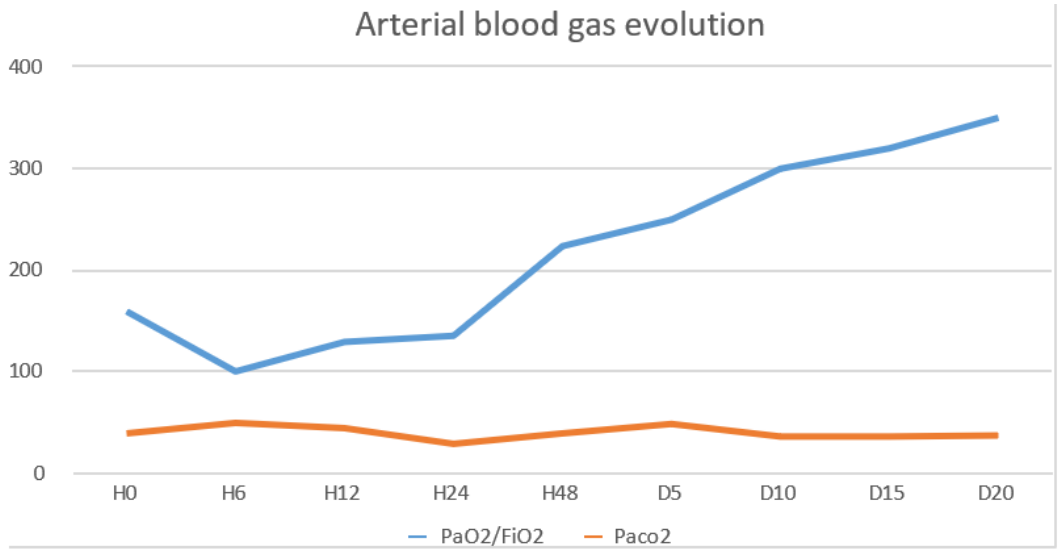
At the admission, a arterial blood gas reveled an acidosis at 7,21, hypercapnia 70mmHg with a PaO2/ FiO2 ratio decreased at 161 Laboratory tests revealed an inflammatory syndrome with elevated CRP at 69,3mg/l (N<5), troponins at 103ng/L (N<34), pro-BNP at 1541 ng/L (N<300), white blood cells at 15770.mm3, neutrophils at 13900/mm3, lymphopenia at 120/mm3, a negative PCT with a normal hepatic and renal function.

A PCR COVID test was positive. Bacteriological samples were negative, as well as the search for Mycobacterium tuberculosis in bronchial aspirate products. Therapeutically, the patient was placed on water restriction, intravenous azithromycin 500mg/day for 5 days, corticosteroids, prophylactic anticoagulation, vitamin C, vitamin D, and zinc.

The patient's respiratory condition improved, as evidenced by blood gaz controls. Hemodynamically, there was improvement with progressive weaning of vasoactive drugs, a decrease in troponin levels to negative normalization of pro BNP and inflammatory markers, and gradual improvement in left ventricular ejection fraction on successive echocardiographic controls. The patient was extubated 20 days after admission and transferred to the cardiology department. We would like to note that PCR COVID test stayed positive 15 days post admission et was negative on the 20th day.







The diagnosis of myocardial injury has been made in the context of Covid-19 infection based on evolving clinical, biological, and echocardiographic data. In addition, a coronary angiography was performed and showed an intact network without significant lesions, ruling out an acute coronary syndrome.

Discussion

The world has experienced a pandemic related to the respiratory spread of SARS-CoV-2, which, beyond the well-described clinical manifestations, particularly respiratory ones, also affects the cardiovascular system[5].

In a recent literature review of 26 studies including 11,685 infected patients, the weighted prevalence of cardiac involvement was 20% [6].

The responsible virus is SARS-CoV-2, a single-stranded RNA virus that invades the cells of the body through the ACE2 receptor, which is present in the lungs as well as in the heart and kidneys [7,8].

However, little is known about the true mechanisms of cardiac injury, which are not well established but likely involve an increase in cardiac stress due to respiratory failure and hypoxemia, direct myocardial infection by SARS-CoV-2, indirect injury due to systemic inflammatory response, or a combination of these factors. Case reports of myocarditis in COVID-19 provide evidence of cardiac inflammation but do not clarify the mechanism. These mononuclear infiltrates are associated with regions of cardiomyocyte necrosis, which, according to the Dallas Criteria, define myocarditis.

However, so far, there is no data demonstrating the presence of SARS-CoV-2 in myocardial tissue [9]. Nevertheless, real-time PCR analysis of post-mortem cardiac tissue during the SARS epidemic detected viral genomes in 35% of patients (n=7/20) who died from SARS. It should be noted that these hearts also showed decreased levels of ACE2 and increased hypertrophy.

Myocardial inflammation can lead to myocarditis, heart failure, cardiac arrhythmias, acute coronary syndrome, rapid deterioration, and sudden death. The elevation of cardiac biomarkers during COVID-19 infection is associated with a poor prognosis. Moreover, there is a linear relationship between troponin elevation and CRP levels, suggesting a link between inflammation and myocardial involvement[10] Patients with unfavorable outcomes, including admission to intensive care units and mortality, had significantly higher levels of cardiac troponin. The levels of natriuretic peptides were also elevated among ICU admissions in Washington [11,12,13,14]. Consistent with our patient's case, troponin levels showed an ascending kinetics up to 20 times the normal value at 48 hours, and the natriuretic peptide level was 5 times the normal value upon admission.

In a cohort from Wuhan, myocardial lesions and heart failure contributed to 40% of deaths, either exclusively or in conjunction with respiratory failure[14]. In a Cox regression model adjusted for various factors, patients with elevated circulating cardiac injury biomarkers had a significantly higher risk of death. This is in contrast to the reported case of our patient, whose outcome was favorable, possibly explained by early diagnosis and management.

A Chinese cohort study based on the WHO definition of acute myocardial injury (i.e., elevation

of biomarkers and electrical changes) estimated that these lesions occur in 7% to 17% of infected patients and up to 59% of those who died [16]. Surprisingly, the mortality risk associated with acute cardiac injury was greater than advanced age, diabetes, chronic lung diseases, or a history of cardiovascular diseases.

Conclusion

Cardiac manifestations are not rare during COVID-19 infection and are associated with a higher risk of mortality. The underlying mechanisms are still poorly understood. The distinction between ischemic and non-ischemic causes is not systematic due to limitations in complementary exams in this particular context, which contributes to confusion in identifying these conditions. The term "acute myocarditis" should be reserved for cases proven by cardiac MRI or myocardial biopsy in order to propose appropriate short-, medium-, and long-term management. Further studies are needed regarding the management of cardiovascular involvement in COVID-19 [17,18].

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