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Case Report
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Myocarditis in a Patient with COVID-19: A Persistent Troponin Elevation

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Abstract

A 52-year-old patient with chest pain and shortness of breath was admitted with the diagnosis of acute coronary syndrome due to increased troponin. Normal coronary arteries were seen in coronary angiography. During the follow-up, the patient, who developed fever and cough, COVID-19 RT-PCR test was positive. There was ground-glass opacification in the lungs on computed tomography. Cardiac magnetic resonance imaging showed heterogeneous contrast fixation in the left ventricular free wall in post-contrast series, in patient with persistent troponin increase.

Keywords: COVID-19; Myocarditis; Troponin; Magnetic resonance imaging

Introduction

The pneumonia epidemic caused by a new type of coronavirus has extended from China to the whole world since December 2019. More than 80 million people have already been infected with this virus worldwide, and the number of patients continues to increase rapidly. Coronavirus disease (COVID-19) is caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). Typically the respiratory system is affected, also cardiovascular, gastrointestinal and, neurological system involvements are common. In this case, we present a 52-year-old male patient with chest pain, shortness of breath, and fatigue and had high troponin with repolarization changes in the electrocardiography (ECG), who was subsequently diagnosed as COVID-19 related myocarditis.

Case Report

A 52-year-old male patient was admitted to the emergency department (ED) with chest pain, shortness of breath and fatigue. The symptoms of the patient started 3 days ago and continued to increase. On examination, there were no signs, including fever and hypoxia. The only cardiovascular risk factor was hypertension. High sensitivity cardiac troponin I levels were elevated (peak in ED = 1400 ng/L) with repolarization changes in the precordial ECG leads (Figure 1).

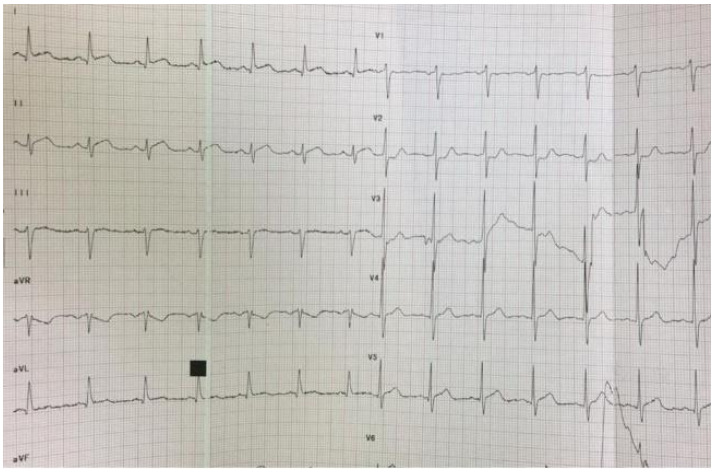

Figure 1: *Repolarization changes in the precordial ECG leads.*

Echocardiography showed normal systolic function and left ventricular hypertrophy, without pericardial effusion. The patient was admitted to the cardiology department with the diagnosis of the acute coronary syndrome. There was no occlusive coronary artery disease in the coronary angiography performed on the patient (Figure 2).

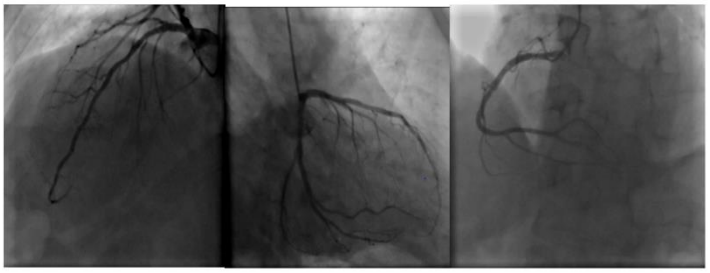

Figure 2: *Coronary artery disease in the coronary angiography performed on the patient.*

During the follow-up, the patient, who developed fever and cough, was referred to the isolated ward due to the current coronavirus outbreak and RT-PCR was performed on the nasopharyngeal swab for COVID-19. The test result was positive. Chest computed tomography (CT) was performed on the patient due to increased dyspnoea. Peripherally located multifocal ground-glass opacities were detected (Figure 3).

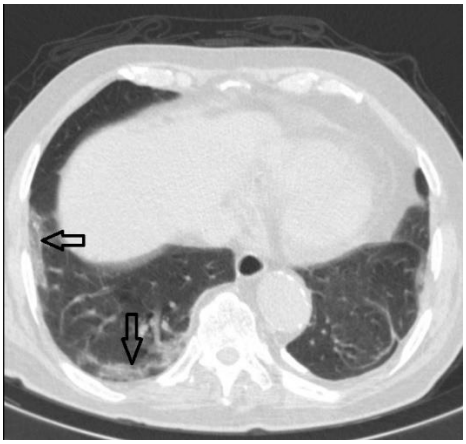

Figure 3: *Peripherally located multifocal ground-glass opacities were detected.*

Favipiravir (1600mg / 12h first day, followed by 600 mg/12h for 4 days), prophylactic Enoxaparin (4000u / 24h), and methylprednisolone (40mg/12h) treatment were initiated for the patient. Cardiac magnetic resonance imaging (MRI) was performed due to the increase in troponin values (6000 ng/L) during follow-up. Cardiac MRI showed heterogeneous contrast fixation in the left ventricular free wall in post-contrast series, which is typical of acute myocarditis. The patient was treated with of ramipril (2.5mg / 24h) and metoprolol (50mg / 24h). Two weeks later, his symptoms have decreased and his troponin levels had returned to normal (Figure 4).

Discussion

This case report demonstrates the myocarditis associated with COVID-19. COVID-19 must be considered in the ethology and pathogenesis of myocarditis under epidemic conditions [1,2]. Myocarditis is generally suspected in patients who are diagnosed with the acute coronary syndrome with ECG and laboratory tests and who do not have obstructive coronary artery disease on coronary angiography.

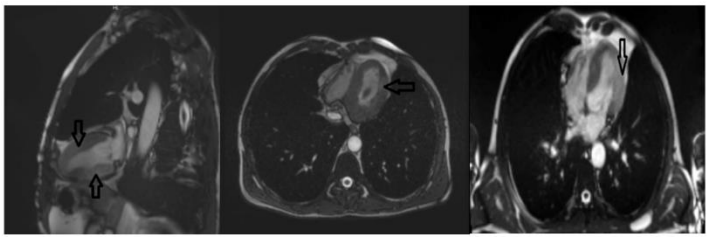

Figure 4: *Cardiac MRI showed heterogeneous contrast fixation in the left ventricular free wall in post-contrast series.*

In our case, the patient presented with chest pain, shortness of breath, and fatigue. Repolarization changes on ECG and, increased cardiac biomarkers in the laboratory indicating myocardial damage were observed. There was no occlusive coronary artery disease in the coronary angiography performed on the patient. RT-PCR and chest CT were performed since typical covid-19 symptoms were observed during the follow-up of the patient. When the patient was diagnosed with COVID-19 with swab and CT, troponin values continued to increase persistently, cardiac MRI was applied to the patient for myocardial damage. Cardiac complications such as heart failure, myocardial infarction, myocarditis, thromboembolism, and arrhythmia may accompany COVID-19 [3,4]. The mechanisms responsible for cardiovascular complications in COVID-19 are; Direct myocardial injury (binding of SARS-CoV-2 to the Angiotensin-converting enzyme 2 receptor can cause changes in signalling pathways, resulting in acute cardiac and lung damage), systemic inflammation (acute systemic inflammatory response and cytokine storm), myocardial oxygen demand-supply mismatch (increased metabolic demand and hypoxia may disrupt the myocardial oxygen demand-supply), acute coronary event (plaque rupture secondary to systemic inflammation, increased pro-coagulants, impaired fibrinolysis, and endothelial dysfunction), iatrogenic (corticosteroids and hydroxychloroquine), electrolyte imbalances (hypokalemia) [5-10]. Myocarditis should be kept in mind if there is a normal coronary artery despite the presence of troponin elevation and repolarization changes in the ECG in COVID-19 patients. Cardiac MRI is helpful to differentiate troponin elevation cardiac or non-cardiac [11].

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