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CASE REPORT

CARDIAC INVOLEMENT IN A 20-YEAR-OLD PATIENT WITH COVID-19

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ABSTRACT

This case report describes pericardial effusion and myopericarditis in a 20-year-old female patient who had a positive RT-PCR test for SARS-CoV-2. A few cases of this clinical presentation in COVID-19 have been described in the world. A COVID-19 positive patient was admitted to the Clinic for Infectious Diseases due to difficulty breathing, chest pain, fever, cough, vomiting and diarrhea. Imaging methods confirmed that the patient had initial pneumonia and pericardial effusion. In laboratory findings, she had elevated values of cardiac necrosis markers. On admission, the patient was dyspnoic, hypotensive, and tachycardic. Despite all measures of intensive treatment, the outcome was lethal.
It is necessary to recognize more severe forms of COVID-19 with involvement of other organ systems in order to start treatment in time and reduce the number of poor outcomes.

Key words: COVID-19, pericardial effusion, myopericarditis

INTRODUCTION

Myopericarditis and myositis are usually associated with cardiotropic viruses [1], multisystem inflammatory diseases [2], immunotherapy [3], although sometimes they can also be idiopathic [4]. Evidence shows the virus can affect the other organs, including the cardiovascular system with a wide spectrum of clinical presentation as the acute cardiovascular injury, myocarditis, thromboembolic presentation including cerebrovascular accident, acute coronary syndrome, pulmonary thromboembolism and also small reports of pericardial effusion and clinical tamponade [5-8]. There is limited study regarding Coronavirus disease 2019 (COVID-19) and myopericarditis [9].
So far, very few cases of cardiac manifestations in COVID 19 have been documented, especially in such young patients with a progressive course and fatal outcome.
Mechanisms responsible for complications in COVID-19 are: direct myocardial injury, systemic inflammation, immune response, altered ratio of myocardial demand and supply, plaque rupture and coronary thrombosis, adverse effects of therapy or electrolyte imbalance [10].
Pericarditis and myocarditis share common aetiologies, and overlapping forms may be encountered in clinical practice. Pericarditis with known or clinically suspected concomitant myocardial involvement should be referred to as 'myopericarditis', while predominant myocarditis with pericardial involvement should be referred to as 'perimyocarditis', according to Task Force consensus. The classical presentation is chest pain associated with other signs of pericarditis (pericardial rubs, ST-segment elevation and pericardial effusion) plus the elevation of markers of myocardial damage (ie troponins). Limited clinical data on the causes of myopericarditis suggest that viral infections are among the most common causes in developed countries, while other infectious causes are more common in developing countries. Cardiotropic viruses can cause pericardial and myocardial inflammation via direct cytolytic or cytotoxic effects and/or subsequent immune-mediated mechanisms [11-12].

CASE REPORT

We presented the case of 20 years old female patient, who was admitted to the Clinic for Infectious Diseases due to elevated temperature up to 38.5 C, cough, difficulty breathing, chest pain, vomiting, diarrhea. The symptoms lasted for 10 days before admission, but the patient did not

report to the doctor. Four days before admission, the symptoms intensified.

The patient has been healthy until now, without known chronic conditions, she was involved in sports and said that in the last 5 months she has been getting more tired during training.

During admission, the patient was pale, dyspnoic, afebrile. Vital signs: heart rate 134/minute, blood pressure 80/60 mmHg, respirations 22/minute, oxygen saturation 94% on room air.

The diagnosis of COVID-19 infection was confirmed by oropharyngeal swab using reverse transcription polymerase chain reaction (RT-PCR) diagnostic test.

Upon admission, an X-ray of the lungs was performed, which showed initial infiltration, an enlarged cardiac shadow, rounded contours, and sharp cardiophenic angles, which according to the X-ray characteristics could correspond to a pericardial effusion (Figure 1).

The electrocardiographic changes (ECG) showed sinus tachycardia, with frequency 134/minute, without signs of current ischemia or signs for pulmonary thromboembolism.

Laboratory findings on admission showed increased values of cardiac markers: creatin kinase (CK): 34 499 U/L, creatin kinase-MB (CK-MB): 969 U/L, troponin: 1 161.80 pg/ml.

An ultrasound of the heart showed increased dimensions of the right heart cavities, right atrium 6.4 cm, right ventricle 4.5 cm, hypokinesia of the inferior, posterior and lateral walls. Pericardial effusion with a diameter of about 1.7 cm in the projection of the right heart with a moderate degree of influence on hemodynamics. Pleural effusion is also present.

In the further course of hospitalization, the condition worsens. In the laboratory findings, there is an increase in cardiac necrosis markers.

A control ultrasound of the heart was performed, which describes severe dilatation of the right heart cavities with septal dyssynergy, weakened systolic function (left ventricular ejection fraction LVEF 30-35%). Small pericardial effusion in the area of the right ventricle.

On the control X-ray of the lungs, persistent lung inflammation is observed with an enlarged heart shadow (Figure 2).

On the fifth day of hospitalization, the clinical condition worsened with pronounced symptomatology, mainly difficulty breathing and suffocation. On examination, she was conscious, dyspnoic, and cyanotic. Vital signs: blood pressure 212/120mmHg, heart rate 80/minute, oxygen saturation 70% in room air. Oxygen therapy was set to low flow O2 through a facial mask, and the saturation was improved to 93%.

Same day, in the afternoon, due to the repeated drop in oxygen saturation, she was switched to high flow oxygen therapy, through the nasal cannula and the saturation improved to 94%. There was a sudden drop in blood pressure, which is why inotropic support was included along with forced diuresis and volume management. In addition to the above measures, the patient was still tachypnoic, was sedated, relaxed and intubated and placed on mechanical ventilation (KMV). The next day, multiorgan failure occurs. In laboratory findings: alanine aminotransferase (ALT) 1162 U/L, aspartate aminotransferase (AST) 787 U/L, lactate dehydrogenase (LDH) 3533 U/L, ferritin 747 µg/L, myoglobin 3650 µg/L, urea 39 mmol/L, creatinine 207 µmol/L, TBIL 152 µmol/L, CK 11092 U/L, troponin 129190.80 pg/ml, C-reactive protein (CRP) 29 mg/L, procalcitonin 7.67 ng/ml. Despite inotropic support and treatment measures taken, death soon occurred.

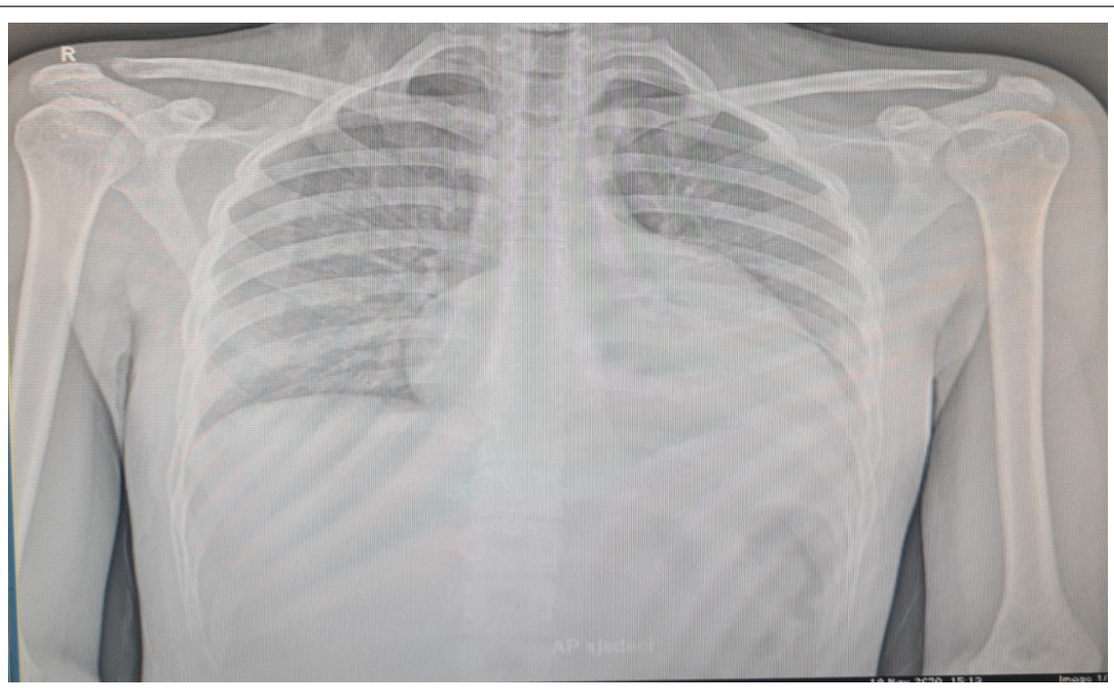

Figure 1. The intial chest X- ray showed initial infiltration, an enlarged cardiac shadow, rounded contours, and sharp cardiophenic angles, which according to the X- ray characteristics could correspond to a pericardial effusion

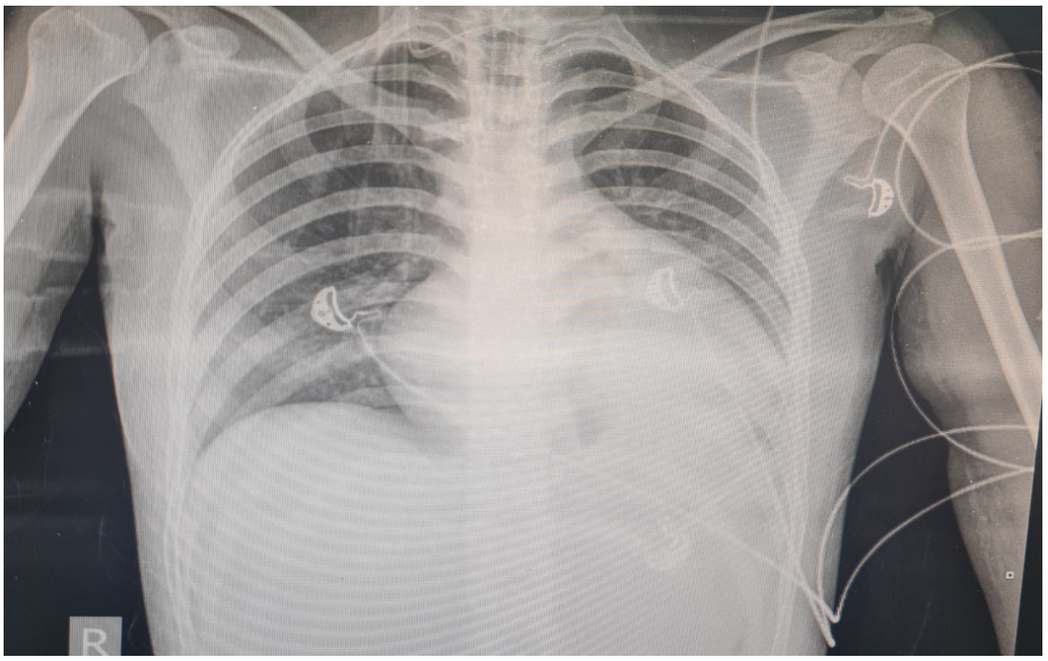

Figure 2. The control chest X – ray showed persistent lung inflammation is observed with an enlarged heart shadow

DISCUSSION

The primary clinical manifestation of COVID-19 is respiratory tract disease, but recent reports have suggested that 12% of patients had cardiac involvement. Importantly, cardiac injury has been associated with a greater risk of mortality [13].

Most of cardiac involvement seen in myocardial injury in the form of myocarditis or acute myocardial infarction, presentation with pericardial involvement is rare [14-15].

This case report describes a COVID-19 positive patient with myopericarditis. The patient initially had mild symptoms and did not see a doctor, she was treated at home for 10 days. When she was hospitalized, she was in a serious general condition, tachycardic, hypotensive, and on the ultrasound of the heart she had a pericardial effusion. In laboratory findings, she had elevated cardiac markers as well as D-dimer.

One of the possible causes of death is that the patient reported to the health facility late in a serious condition when the disease progressed and that adequate treatment was not started in time.

Viral infection has been described as one of the most common infectious causes of myocarditis, especially associated with influenza and parvovirus B-19 [16].

However, less is known about cardiac manifestations as a complication of SARS-CoV-2 infection. Our patient was not vaccinated with the vaccine for COVID-19.

The definite diagnosis of viral pericarditis requires a comprehensive workup of histological, cytological, immunohistological and molecular investigations in pericardial fluid and peri-/epicardial biopsies obtained in conjunction with pericardioscopy, allowing the evaluation of possible algorithms for a causative therapy [17].

Considering the clinical and diagnostic tests, our patient did not require a therapeutic pericardiocentesis, and it was not performed for diagnostic purposes either.

The only way to confirm myopericarditis is an magnetic resonance imaging (MRI) of the heart as well as a heart biopsy. However, Bosnia and Herzegovina is a country with a worse socio-economic status, and these tests are not performed in the same country. There was an indication to perform coronary angiography and computed tomography (CT) angiography, but due to the serious condition of the patient and the impossibility of transportation during the pandemic, those were not done.

Our patient met three of the four criteria required for the diagnosis of myopericarditis including, chest pain, sinus tachycardia and pericardial effusion. She had elevated values of cardiac markers.

The patient had previously been healthy, played sports, and had never had a systematic examination. Given the echocardiographically verified enlargement of the right heart cavities, there is a possibility that the patient had a congenital heart defect that was not observed or recognized by transthoracic echocardiography. Due to enlargement of the right heart cavities and secondary pulmonary hypertension with sinus tachycardia without clear signs of pulmonary thromboembolism on the ECG, elevated D-dimer values, there was another possibility, so pulmonary thromboembolism was also investigated. CT angiography of the pulmonary arteries was indicated, but was not performed for technical reasons. During the entire hospitalization, the patient received low-molecular-weight heparin in a

therapeutic dose, and the control D-dimer was within the reference values.

Cardiac involvement in COVID-19 patients is associated with a worse outcome of the disease. For example, patients with worse outcomes, including intesive care units (ICU) admission and mortality, had higher troponin values [18-19], as shown in our case. Our patient had a fatal outcome, and the autophsy was nit done because of the strickt policy during the pandemic.

O' Gallagher et al reported the approximate 10% incidence of pericardial effusion in COVID-19 associated myocarditis, a complication that was said to be uncommon [20]. Based on the King's College Hospital (London UK) experience, patients with previous cardiac diseases are at increased risk of developing myopericarditis. However, not find this association and the total incidence of pericardial effusion in our study population was about 1.2% and all 5 patients had no history of cardiovascular disease [5].

CONCLUSION

This case report highlights the importance of clinical surveillance and laboratory tests, including troponin, D-dimer levels, in people with COVID-19 to recognize more severe clinical forms of COVID-19 with involvement of other organ systems in order to start treatment in time and reduce the number deaths.

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